Diseases of Fruits and Vegetables
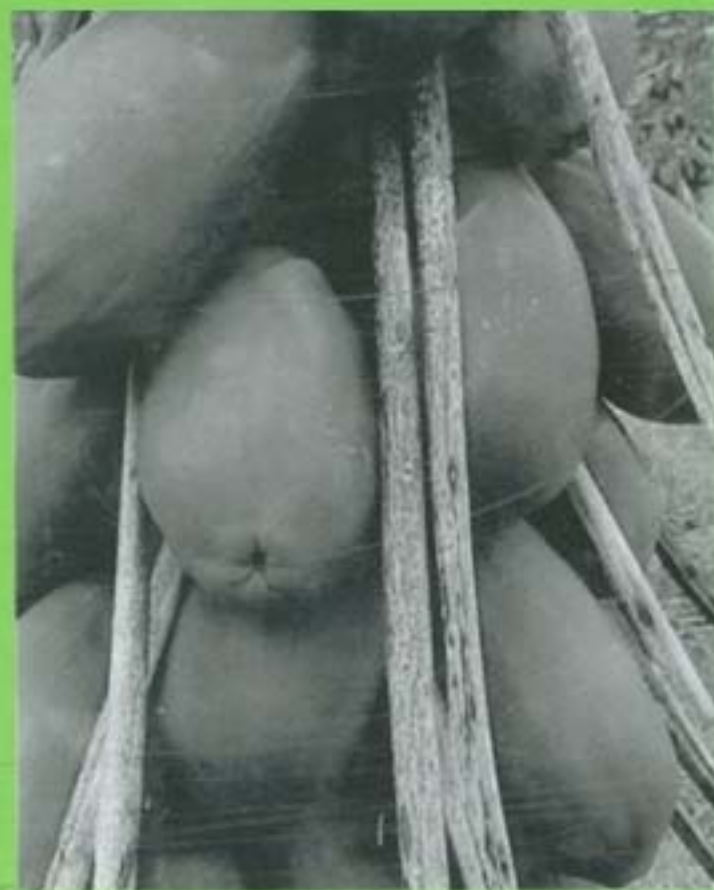
Diagnosis and Management

Volume II

Edited by S.A.M.H. Naqvi

Diseases of Fruits and Vegetables
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PREFACE

Among the Horticultural Crops, Fruits and Vegetables (FV) are of primary importance as the key source of essential components in an adequate and balanced human diet. FV have supported largely the daily food requirement of mankind since ages and even before man learned to grow cereal crops systematically. Over the years, growing FV has been the mainstay of rural economy and has emerged as an indispensable part of agriculture world over, offering farmers a wide range of crops in varied topography and climate. In certain parts of the world, FV are the major dietary staple. Apart from being a rich source of vitamins and minerals, this sector also contributes significantly in economy of the region or the nation. The increased income from per unit area of FV is far ahead and can not be compared with that of cereal crops.

A recent survey by the Economist revealed that the world population has increased by 90 % in the past 40 years while food production has increased only by 25 % per head. With an additional 1.5 billion mouth to feed by 2020, farmers worldwide have to produce 39 % more. Looking at the load of the future food requirement, the global increased production of FV during last few years has absorbed the additional food requirement and accordingly the eating habits are also changing and shifting towards more consumption of these commodities worldwide. During 2002, world fruit production excluding melons was recorded 471.377 million metric tons and that of vegetables including melons 772.71 million metric tons and thus a total world production of FV to the tune of 1244.377 million tones has substantially absorbed the additional food requirement needed for the increasing population (FAOSTAT ,2002).

Unlike cereal crops, there is a wide range of diversity available to farmers to select suitable FV crops. However, the cultivation of these crops for optimum yield and quality produce, is highly technical and needs improved technological support. Management of perennial fruit crops requires further close monitoring especially for the management of diseases that can affect production significantly and subsequently the post-harvest life of these highly perishable commodities. In given favourable conditions, even a single pathogen or disease may cause catastrophe and complete failure of the crop. The famous Irish potato famine is a well-known example where single disease could devastate the whole socio-economic fabric of the country and in fact laid a strong foundation of Plant Pathology.

Obviously with area and population to feed, China stands first in production of FV with 68.43 million tones of fruits and 368.57 million tons of vegetables in the world followed by India with 48.57 million tons of fruits and 68.06 million tons of vegetables. India leads the world in production of banana, mango, sapota and acidlime and among vegetables it is the largest producer of pea and cauliflower and second largest in onion, brinjal (egg plant) and cabbage. However, there has been huge gap in production per unit area in developing nations and developed nations though the area under cultivation is far ahead in developing nations. Hence there is an ample scope and potential in increasing production per unit area in developing nations and in certain developed nations. The low productivity and short productive life of fruit plants has been

mainly attributed to the unavailability of disease free planting stocks and among vegetable crops, inadequate plant protection measures and quality seeds. For example, China is the third largest producer of Citrus after Brazil and USA, having maximum harvested area under Citrus (1.42 million ha) with production only 8.45 MT/ha as compared to Citrus productivity 33. 33 MT / ha of USA from 0.441 million ha during 2001-2002 (FAOSTAT, 2002). Like Irish potato famine, the citrus tristeza virus has been highly destructive and this single pathogen could ravage citrus industry of many countries like Argentina, Brazil etc. amounting to billions dollars. Budwood certification programme developed to get rid of these viruses is very effective but still in countries where it has not been adopted fully are facing the problem of low productivity and short productive life of Citrus plants. Thus, effective disease management plays a key role in successful quality production of fruits and vegetables. In favourable environment conditions, the pathogen attack may reduce the productivity significantly and may also become the cause of total crop failure.

There has been a drastic deviation in global weather pattern under El Nino effect. Black Sigatoka and weak to moderate El Nino weather pattern could affect banana production. Rampant eruption of new races of banana pathogens and their rapid resistant development to new fungicides has posed a threat to banana industry in Africa that produces around 30 million tons of bananas yearly, which is mostly consumed locally. But production is already being reduced and may very well cease entirely within ten years, Scientists warn in a report published in British Weekly 'New Scientist' magazine. Because existing banana plants are reproduced from cuttings, there is little genetic diversity. Diseases, in particular fungus, rapidly can wipe out entire production regions.

With the imposition of WTO conditions in export and import of fresh fruits and vegetables, now it has become more relevant to produce disease free quality produce in order to comply strict phytosanitary conditions laid by certain countries. Recent molecular advancement in our knowledge to detect and diagnose the pathogens in commodities even at very low level made it rather mandatory to produce exportable commodities free from the pathogens.

The new millennium promises excitement and hope for the future by new advancement in eco-friendly technologies in integrated disease management of fruits and vegetables. During past twentieth century, Plant Pathology has witnessed a dramatic advancement in management of fruits and vegetable diseases through in-depth investigations of host-pathogen interactions, development of molecular diagnostic tools, integration of new concepts, principles and approaches.

My effort in bringing out this edited book is to update the achievements of twentieth century in diagnosis and management of diseases of fruits and vegetables of international trade and some under-exploited minor fruits which otherwise are widely dispersed in various scientific journals and to develop future strategies for the new millennium. The book includes latest diagnostic tools and management strategies of

almost all the economically important temperate, tropical and subtropical fruits and vegetables at one place which would be easier to refer by the students, research workers, planners, administrators, policy makers and other end users like grower of fruits and vegetables world-wide. The chapters on individual crop on various aspects of diseases like geographical distribution of disease, diagnosis, disease forecast, approaches to eliminate difficult sytemic pathogens, production of disease free planting material and integrated disease management at nursery, orchard and post-harvest level are contributed by leading Plant Pathologists having authority and significant contributions in respective fields at international level.

The diseases of economic importance caused by fungi, bacteria, viruses and virus like organisms, Phytoplasma and nematodes of each crop are covered, describing their history, distribution, losses incurred, symptoms, latest diagnostic tools, epidemiology and integrated applied management approaches including cultural, chemical, genetic resources, use of bio-control agents being adopted world-wide. The layout of each chapter includes a brief abstract, introduction and pathogen-wise description of the diseases. Each chapter is vividly illustrated with photographs of typical symptoms, graphs, tables and line drawings to make the subject more interesting and easy to understand for students, Scientists, planners, administrators, growers and other end users with latest pertinent references.

In volume I, diseases of Apple, Citrus, Grapes, Mango and Pineapple among fruits and Carrot, Celery and Cucurbits among vegetable crops with special reference to integrated diseases management practices have been included. Volume II covers Avocado, Banana, Grapes, Guava, Papaya, Passion fruit, Strawberry, Stone fruits and Minor tropical and subtropical fruits. Among vegetables, Lettuce, Pea, Pepper, Potato, Onion and Garlic have been included in this volume besides role of mycorrhiza and biocontrol agents in disease management. I am sure that these two volumes will be of immense help and use to the fruits and vegetables growers world over, students, re-search workers, planners, administrators, teachers and other end users engaged in diag-nosis and management of fruits and vegetables diseases.

I am grateful and indebted to all the learned galaxy of contributors who have spent their considerable time in contributing the chapters on various internationally important fruits and vegetables crops. I thank them for their cooperation and support during this project.

I dedicate this work to all those great Scientists who have spent their life time in diagnosis and management of diseases of fruits and vegetables world over in order to improve the quality and productivity of fruits and vegetables, to uplift the nutritional status of human diet and fight against hunger. I am thankful to my wife Dr Nikhat Sarwar Naqvi, for her constant encouragement and help in various ways while editing the book.

25th September, 2003   **S.A.M.H. Naqvi**

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COLOUR PLATES
from CHAPTER 10

Figure 1: Mature pepper fruit exhibiting multiple lesions of anthracnose (Photo courtesy T.A Kucharek)

Figure 3: *Phytophthora* blight lesion on pepper fruit (Photo courtesy R.J. McGovern)

Figure 2: *Phytophthora* blight lesion at crown of a pepper plant (Photo courtesy R.J. McGovern)

Figure 4: Powdery mildew on underside of pepper leaf (Photo courtesy K. Pernezny)

Figure 5: Postharvest decay of pepper fruit infected with bacterial soft rot, gray mold, and secondary microorganisms (Photo courtesy P.D. Roberts)

Figure 6: Bacterial spot lesions on foliage of pepper (Photo courtesy K. Pernezny)

Figure 7: Bacterial spot lesions on pepper fruit (Photo courtesy of K. Pernezny)

Figure 8: Pepper leaves and fruit exhibiting symptoms of Pepper mottle virus (Photo courtesy
   K. Pernezny)

Figure 9: Pepper leaves exhibiting symptoms of Tomato spotted wilt virus infection (Photo
   courtesy P.D. Roberts)

Avocado Diseases of Major Importance Worldwide and their Management

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Abstract: Avocado fruit has become one of the most sought after food sources worldwide. It is a nutritious source of food and is part of the staple diet of many people in third world countries where it is cultivated and where it is native. Avocado is a specialty fruit, produced on a large scale in more than 30 countries around the world and because of its appeal, it is an industry that continues to grow. It is a very lucrative industry, and with over production growers have been forced to develop overseas markets, however, the competition on these markets is very intense and growers have to ensure that their fruit are of the highest quality, unblemished and disease-free. Production costs are very high owing to the cultural requirements of the avocado tree and the presence of diseases, which can be major limiting factors to production. *Phytophthora* root rot, caused by *Phytophthora cinnamomi,* is such a disease. During the developmental stages of the fruit in the orchard and at harvesting, the fruit is also exposed to diseases such as scab (*Sphaceloma persea*), anthracnose (*Colletotrichum gloeosporioides*) and *Cercospora* spot (*Pseudocercospora purpurea),* which result in unsightly blemishes or the development of post-harvest decay; both of which are unacceptable on the export market. The objective of this chapter is two-fold: firstly, to describe the most important diseases of avocado which may be encountered in different parts of the world, and secondly, to discuss current disease management strategies used to reduce the effects of these diseases, on tree health, longevity, fruit production and fruit quality.

1. Introduction

Documentation regarding the close relatives of avocado, all in the subgenus *Persea*, as well as the occurrence of some primitive avocados, supports the theory that the region, including central Mexico through Guatemala and into Central America is the center of the origin of the avocado. The commercial avocado *(Persea americana* Mill.), which can be classified into three subspecies or botanical varieties, belongs to the family Lauraceae, and is one of 50 genera in this family. Most members of this family have aromatic foliage, these include camphor, cinnamon, sweet bay and California bay. The three subspecies, sometimes referred to as ecological races, are: americana (West Indian), guatemalensis (Guatemalan) and drymifolia (Mexican). Mexican types are generally small and cold hardy and have anise-scented leaves. The fruit are thin skinned and take about six months to mature. The West Indian race thrives in tropical areas and is not cold hardy. It produces fruit that mature in 6-7 months that are variable in size and may be very large (> 1 kg), and have skin that is intermediate in thickness between Mexican and Guatemalan cultivars. Guatemalan cultivars have small to large, generally

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oval to ovoid fruit with thick, leathery skin, are intermediate in cold tolerance, growing at moderate to high elevations (900-2400 m), and require 12-15 months to mature (Sippel 2001, Zentmyer 1998a).

In Florida the avocado industry is based on West Indian and West Indian-Guatemalan hybrid cultivars. In California, from 1911 to the 1970s, the Mexican-Guatemalan green-skinned hybrid, Fuerte, (selected in Mexico in 1911), dominated the avocado industry. By the 1980s, Fuerte was surpassed by the dark-skinned Hass, of the Guatemalan race, which was developed in California; more than 90% of California production is now Hass. Although Hass has been distributed around the world and is the principal cultivar, in many countries including South Africa, Australia, New Zealand, Spain, Chile, Israel and Mexico, Fuerte is still one of the most popular cultivars on the overseas market (Sippel 2001, Zentmyer 1998a).

There are 33 major avocado producing countries in the world, with the greatest producers (in order of production) being : Mexico (>500, 000 metric tons), the United States (>300,000 metric tons), Dominican Republic (> 200,000 metric tons), Brazil (>150,000 metric tons), Israel, Indonesia, Haiti, Venezuela, Colombia, South Africa and Australia (>100,000 metric tons). Some countries such as South Africa and Australia, have more than doubled their production over the last decade (Sippel 2001, Zentmyer 1998a).

The disease which still remains the major limiting factor in avocado production today, is *Phytophthora* root rot caused by *Phytophthora cinnamomi* Rands. In countries where avocado nursery certification programs are deficient or non-existent, diseases such as *Phytophthora* canker caused by *P. citricola* Sawada and Sun Blotch viroid disease, cause considerable losses to the avocado industries.Other diseases of varying importance in different countries are Rosellinia (Dematophora) root rot, Armillaria root rot, Verticillium wilt and several leaf and fruit spots. Because of the growing export markets, diseases which cause fruit blemishes and fruit rots are increasing in importance.

These disorders are caused by species of *Colletotrichum, Dothiorella, Phomopsis, Pseudocercospora, Spaceloma* and *Thyronectria.* Minor problems include powdery mildew, sooty mold and sooty blotch. Extensive research programs to address all of the major diseases and other important aspects of avocado production are being conducted primarily in Australia, California, Florida, Israel, Mexico, South Africa and Spain.

This chapter is devoted to the description of the most important avocado diseases and their current management. A great deal of literature is available on this subject, but the aim of this chapter is to make available, a concise and practical source of information for use by researchers who are unfamiliar with the most important diseases of avocado; extension specialists and farm advisors involved in integrated pest and disease management, and the layman.

2. Fungal diseases

2.1 *Phytophthora* root rot

Phytophthora root rot (PRR) is considered the most important and most widely distrib-

uted disease of avocados in the countries where avocados are produced, these include California, Florida, Hawaii, Texas, Australia, New Zealand, South and West Africa, Kenya, Morocco, Mexico, Central and South America, the Philippines, Taiwan , the Caroline Islands, Portugal, Spain, the Canary Islands and Israel. From a production point of view PRR is the single most important disease of avocado.

In California alone it has been estimated to affect between 60–75% of the orchards and causes a loss in excess of $40 million annually (Coffey 1992).The causal fungus *Phytophthora cinnamomi* Rands was first isolated from cinnamon trees in Sumatra in 1922 and has since been reported from over 70 countries. It has an extremely wide host range including 1000 varieties and species of plants. Major hosts include avocado, pineapple, chestnut, eucalyptus, several species of pine, sycamore, peach, pear, macadamia nut, many ornamentals (including azalea, camellia and rhododendron) and many indigenous Australian and South African plants. The first published report on PRR was from Puerto Rico in 1927. A PRR type of decline was reported from California during 1920-1930, but it was only in 1942 that *P. cinnamomi* was isolated from avocado (Zentmyer 1980, Zentmyer *et al.* 1998a).

2.1.1 Symptoms

The first signs of the disease are reflected in the tree canopy. The leaves are small, pale green, wilted with brown tips, and fall readily. In contrast to *Phytophthora* canker, new growth is usually absent, if it does develop leaves are small and of poor color. Shoots die back from the tips and eventually the tree is reduced to a bare framework of dying branches. Death of the tree may take from a few months to several years, depending on soil, cultural and environmental conditions. Declining trees may set a heavy crop of small fruit. When flowering occurs, the tree may lose all its leaves.

The small feeder roots on diseased trees may be absent in the advanced stages of decline; if present they are usually blackened, brittle and decayed in contrast to healthy trees which have an abundance of creamy-white feeder roots. Pencil sized roots or larger are seldom attacked by the fungus. In contrast, blackened , decayed feeder roots are usually absent from avocado infected by *P. citricola* Sawada, when this pathogen occurs as the sole cause of decline (*see* Fig. 4).

A weeping stem canker may occasionally be observed on the lower trunk; the exudate dries to form a white crystalline powder. These cankers are more fully described in the following section on *Phytophthora* trunk, collar and crown canker (Faber and Ohr 1999, Manicom 2001, Pegg 1991, Zentmyer 1980 and 1984).

2.1.2 Causal organism and Epidemiology

P. cinnamomi forms several different spore stages that are involved in infection , disease development and survival; these include sporangia, chlamydospores and oospores. Sporangia give rise to motile zoospores which are disseminated by free water on the surface of the soil or in films of water within the soil pores. The zoospores are attracted to the roots by root exudates, the zoospores encyst, germinate and infect the roots, invading them inter- and intracellularly. Root lesions appear within 24 hr and mycelium

can be found throughout the small absorbing roots within 72 hr.

Chlamydospores are survival structures and are normally stimulated to form by dry soil conditions and can survive for several years under extremely dry conditions. The spores are formed within the roots and are released into the soil when the roots decay. Oospores are also survival structures produced under certain soil and environmental conditions, usually low temperature. Both chlamydospores and oospores give rise to sporangia under suitable soil moisture and temperature conditions.

Soil moisture is the primary environmental factor influencing PRR development. High soil moisture stimulates the development of sporangia and improves conditions for zoospore release and movement to the infection site. Stress from excess or low moisture and excess salt can also injure roots causing them to exude root extracts which attract zoospores and incite infection.

Symptoms usually do not appear unless an upset occurs in the balance between the water requirements of the leaves and capacity of the roots to absorb water.
Where *P. cinnamomi* is not native to an area, the primary method of introduction of the pathogen into orchards is by infected nursery trees. Once in the orchard it can be spread by soil attached to shoes, tools, vehicles, picking boxes, ladders and storm water (Faber and Ohr 1999, Manicom 2001, Menge and Marais 2000a, Zentmyer *et al.* 1965, Pegg 1991, Zentmyer 1980 and 1984, Zentmyer *et al.* 1998a).

2.1.3 Disease Management

Since no definitive control measures have yet been found to control PRR , an integrated approach to managing the disease has been found to be the most effective. This approach includes prevention, cultural methods and chemical treatment. These aspects will be discussed separately below:

2.1.3.1 Site selection and soil preparation

Planting of avocado is a long term investment and therefore justifies a high capital outlay in the initial stages. Soil should be prepared well in advance of planting. Severe PRR is associated with soils that have poor internal drainage, are less than 91 cm deep , have hard pans, clay pans and high clay content. These soils are conducive to build-up of inoculum and infection of roots and should be avoided. Less hazardous soils with a clay-loam texture and depth of 92-152 cm should be deep ripped and provision made for drainage . On sloped land, the construction of interception and diversion drainage canals or provision of water tight drain pipes which drain rain water away from the orchard, will help prevent the introduction of *P. cinnamomi* into lower lying orchards. In heavy clay soils trees can be planted on mounds (100 -150 cm in diameter and 50-100 cm high) or ridges, this has been found to increase the survival rate of young trees by as much as 180%. Soil solarization, which consists of heating the soil above 45EC with clear polyethylene sheets that have been placed on the soil surface to trap the sun's radiant energy, has been found effective for reducing *Phytophthora* inoculum following tree removal in infested soil in Israel (Erwin and Ribeiro 1996). Soils with high salinity should also be avoided since, not only does salinity retard growth and reduce

yields, it exacerbates avocado root rot (Borst 1970, Menge and Marais 2000a, Zentmyer and Ohr 1978).

2.1.3.2 Soil amendments

The application of amendments such as organic mulches and inorganic gypsum contribute to improving soil structure, thereby improving drainage, helping to remove salts from the soil and have the added benefit of increasing the soil's suppressiveness to *P. cinnamomi.* The suppressive effect of calcium and organic matter was first discovered in Australia (Broadbent and Baker 1974). The beneficial effects of the organic mulch were due to the development of high populations of microorganisms in the soil which were antagonistic to *P. cinnamomi.* A study of the effects of calcium on PRR was conducted in California soils by Messenger-Routh (1996) who concluded that calcium primarily acted as a weak fungicide by reducing the size and number of sporangia produced by *P. cinnamomi.* It is recommended that applications of 1500-3000 kg /ha gypsum be made under the tree canopies, depending on the size of the trees. Mulches should be placed in layers of 15-30cm thick under the canopies of the trees (Menge and Marais 2000a,b).

2.1.3.3 Disease-free nursery trees

Historically, diseased nursery stock was the major source of spread of PRR into the avocado production areas of California . In other countries where *P. citricola* is not native, this would also be the case. Countries such as Australia and South Africa have excellent certification programs which ensure that growers obtain disease-free trees. It is particularly important to plant disease-free trees when planting new areas.

2.1.3.4 Irrigation and irrigation water

Avocado is extremely sensitive to water-logging, owing to the high oxygen requirement of avocado roots. Under these conditions root growth ceases and conditions are conducive to large scale destruction of feeder roots. The use of tensiometers or soil probes is necessary to schedule irrigation. Preferably irrigate with water from deep wells, water from reservoirs and canals can be a source of infection and should therefore be treated with chlorine or copper to eliminate the inoculum.

When an infection locus is identified in an orchard, the irrigation of these diseased trees and the margins of the diseased areas should be irrigated with caution so as not to over irrigate, careful irrigation can retard the spread of the disease and often prolong the life of affected trees (Faber and Ohr 1999, Menge and Marais 2000a,b).

2.1.3.5 Orchard sanitation

Excluding *P. cinnamomi* from a clean avocado orchard is the most economical method of controlling the disease. Movement of soil and water from diseased orchards into healthy ones should be avoided at all costs. The fungus readily moves from orchard to

orchard in moist soil on cultivation tools, vehicles, picking boxes, ladders, shoes etc. Barriers in the form of fences and warning signs, should be placed between uninfested and infested orchards. Boxes with copper sulfate should be placed at the property entrance and all workers and visitors should be requested to dust their shoes with this material before entering. Shallow, chlorinated or copper sulfate-treated water baths may also be placed at the entrance to the property for the vehicles to drive through before entering the premises. Small pieces of equipment such as shovels, soil augers and trowels should be dipped in 70% ethanol or rubbing alcohol. Always use disinfested equipment in healthy orchards before using it in a diseased orchard. Severely affected trees should be removed and the soil fumigated (Menge and Marais 2000b, Faber and Ohr 1999, Zentmyer and Ohr, 1978).

2.1.3.6 Resistant rootstocks

A great deal of research has been conducted on detecting and developing resistant rootstocks, especially in California, South Africa and Israel. Rootstocks such as Duke 7, Thomas, Barr Duke, Toro Canyon, G755 (Martin Grande) and Evstro, exhibit a greater degree of tolerance to PRR than Topa Topa. Although some resistant rootstocks may not yield as well as the traditional sensitive ones, the trees will survive under disease pressure when used in conjunction with the control measures mentioned above (Bijzet and Sippel 2001, Menge *et al.* 1992, Menge and Marais 2000b).

2.1.3.7 Crop rotation

Replanting infested soil to resistant field and fruit tree crops is one of the best ways of restoring diseased soil to a state in which it can be replanted to avocado. All varieties of citrus, many deciduous fruit tree crops, Macadamia, persimmon, berries and all type of vegetables, and most annual flower crops are not susceptible to PRR (Ohr *et al* 1994, Faber and Ohr 1999).

2.1.3.8 Chemical control

In the 1970s and 1980s systemic fungicides with specific activity against species of *Phytophthora* and related fungi revolutionized control of diseases such as PRR. The first of these compounds were metalaxyl (Ridomil®) and fosetyl Al (Aliette®) (Coffey 1987 and 1992, Erwin and Ribeiro 1996, Menge and Marais 2000b).

The phosphonates, including fosetyl Al and its active breakdown product phosphorous acid and potassium phosphite, have been effective when applied as foliar sprays, trunk paints, trunk injection, or soil application. The injection method (Fig. 1) first developed in South Africa, has given good results in South Africa and Australia (Darvas *et al.* 1984). In South Africa and Australia salts of phosphonic (phosphorous) acid, particularly potassium phosphonate (potassium phosphite) have been registered for foliar and trunk injection. A similar product has been registered for use in Israel. Trunk injection is the preferred method of application, but foliar and soil applications are made under certain conditions. Trunk injection is the best way to rejuvenate trees

severely affected by PRR. Fosetyl Al has been injected into severely affected trees and trees have recovered totally. In South Africa 1ml of a 50% neutralized phosphonic acid (neutralized with potassium hydroxide) is injected per meter square of tree drip area , this equates to 0.5 g active ingredient per meter square of tree drip area. Injections are applied twice annually, once following the hardening off of the spring flush, which occurs at flowering and the second after the hardening off of the summer flush. These

Figure. 1 : Tree injection of phosphonates to control *Phytophthora* root rot in avocado trees.

two applications coincide with the root flushes (Menge and Marais 2000b).

Metalaxyl (Ridomil®) has been applied as a granular, a drench or injected into the irrigation water and has been found to be effective in some cases. The most popular compound is however phosphonic acid. Aliette® has been found to be more effective than metalaxyl in mature orchards in California (Coffey 1992).

2.2 *Phytophthora* trunk, collar and crown canker

Three species of *Phytophthora* have been associated with trunk, collar and crown cankers on avocado trees viz. *P. cinnamomi* Rands, *P. citricola* Sawada and *P. heveae*

A Thompson. The most devastating and abundant is *P. citricola,* especially in the cooler coastal production areas of California, where it was first described on avocado in 1974. *P. citricola* is apparently present in approximately 90% of the avocado orchards in California, while the disease itself occurs in 5% of these orchards. *P. cinnamomi* the major causal organism of root rot, has infrequently been reported to cause cankers in California, South Africa, Brazil and Australia.

The only report of *P. heveae* being associated with cankers on avocado was from a small nursery planting in Guatemala (Faber *et al.* 1995, Faber and Ohr 1999, Manicom 2001, Zentmyer *et al.* 1998b).

2.2.1 Symptoms

Trunk cankers caused by *P. citricola* are normally found at the base of the trunk extending to a height of approximately 50 cm (Fig.2). The pathogen infects the crown , lower trunk and sometimes the main structural roots of avocados of all cultivars. The cankers usually originate at or below soil level, but can be found higher up if wounding has occurred. The pathogen can gain entrance through wounds caused by pruning tools, mechanical damage caused by pickers or even limbs rubbing against each other during high winds. The lesion is discolored and the cankers give rise to copious amounts of a red resinous, water soluble exudate through cracks in the bark, which dries to form a white crystalline deposit. Cutting away the superficial canker reveals an orange-tan to brown pigmented outer layer of wood and inner layer of bark, instead of the normal white or cream-colored tissues (Fig.3). The lesion has a fruity odor when exposed. The cankers have distinct reddish-brown margins from which the fungus can readily be isolated (Bender 1999).

Foliar symptoms are not unlike those caused by *Phytophthora* root rot with the exception that there always appears to be more leaf litter on the orchard floor than in the case of *Phytophthora* root rot, and there is usually an abundance of healthy, cream-colored feeder roots in the area of the root crown (Fig. 4)**.** The abundance of leaf litter is due to the fact that canker affected trees which are not severely diseased, develop a great deal more new flush during the growing period than do root rot affected trees. Moderately affected trees often appear quite healthy and can for persist this way for several years, until the canker progresses to a stage where it may start killing the cambium tissues around the trunk, girdling it. Unless the trunk is inspected for lesions and the tell tale sign of the white powdery exudate, the tree appears healthy. In some cases the disease can progress so rapidly that it will kill a tree young or old within a matter of months by killing the cambium and effectually girdling the tree. Frequently trees exhibit wilted and sparse canopies without any above ground signs of a canker, in these cases the canker has girdled the tree a few centimeters below the soil level (Bender 1999, Faber and Ohr 1999, Zentmyer *et al.* 1965).

2.2.2 Causal organism and Epidemiology

The causal organism of this destructive disease in California is *P. citricola.* Previously uncommon in this avocado producing region, it has now become second to

Phytophthora root rot in being a major limiting factor in avocado production. *P. citricola* has a wide host range and has been recorded on hosts such as walnut , cherry, cherimoya and fir trees. *Phytophtho*ra canker disease is also favored by excess moisture, which is essential for the dissemination of the spores. Stress factors resulting from water deficits, salinity, excess fertilization and root disease caused by *P. cinnamomi*, are

Figure 2: Canker caused by *Phytophthora citricola* on the lower trunk of an avocado tree. Note the white sugary exudate surrounding the crack in the canker lesion.

also conducive to infection by *P. citricola.* One of the primary ways in which the disease is spread is through infected nursery stock.. Nurseries which do not take precautions to disinfect water used for irrigation purposes, sterilize potting media and keep containers off the ground, are responsible for disseminating the disease throughout the industry. Phytophthora canker differs from the root rot pathogen in that it infects through wounds. Wounds created by gophers, pruning wounds made when removing suckers, wounds made during staking, and cold injury. It has been reported that severe pruning of canker infected trees increases the rate of demise of the trees. The pathogen can also be spread on contaminated pruning tools, harvesting equipment and vehicles, on the shoes of pickers climbing into the trees , and by wild animals such as pigs and

gophers which feed on the roots of the trees (Bender 1999, El-Hamalawi *et al.* 1995, El-Hamalawi and Menge 1994 , Faber and Ohr 1999, Manicom 2001, Zentmyer 1984, Zentmyer *et al.* 1998b).

2.2.3 Disease Management

The measures recommended for the control of *Phytophthora* canker diseases are simi-

Figure 3 : Canker on avocado tree caused by *Phytophthora citricola* showing distinct reddish brown margins and brown necrotic inner bark and wood.

lar to those described for *Phytophthora* root rot. The use of certified disease free nursery stock cannot be over emphasized. It is very difficult to cure trees once they become infected with *P. citricola*. Root rot infected trees can on the other hand be treated successfully, to a state of total remission, with chemicals such as Aliette® and phosphonates. Unfortunately some of the newer clonal rootstocks which are resistant to *Phytophthora* root rot such as Thomas, are susceptible to *P. citricola*(El-Hamalawi *et al.* 1994, Zentmyer *et al.*1998b). Chemical control of *Phytophthora* cankers caused by *P. citricola,* has not been very successful in California so far, but extensive field trials are now underway to test different chemicals and modes of application. However, cankers caused by *P. cinnamomi* have been treated with success in South Africa by

painting the trunks of affected trees with Aliette® (Manicom 2001). Greenhouse experiments at the University of California Riverside indicate that cutting away cankerous lesions on seedlings and applying Aliette® as a trunk paint, will arrest the infection (El-Hamalawi and Menge 1994).Severely pruning canker affected trees should be avoided . Pruning tools should be disinfected before moving to the next tree. It is important to remove leaf litter from crotches of trees and from around the base of the trunk. Avoid irrigation sprinklers from wetting the crotches of the trees and from spraying directly onto the trunks. Cankers frequently develop on the side of the trunk exposed to irrigation sprinklers (Faber and Ohr 1999).

2.3 Anthracnose

Anthracnose causes leaf and fruit drop and reduces the shelf life of fruit during storage and transport. Post harvest decay caused by anthracnose has been reported from Florida, Australia, South Africa, New Zealand, Mexico and Israel. Anthracnose is not normally a problem in California, but occasionally becomes a problem during periods of extended rainfall. This apparently the most important post harvest disease in South Africa, causing losses up to 37 % on fresh fruit. In South Africa anthracnose is usually found together with other fungi including *Dothiorella aromatica* (Sacc) Petr & Syd. and collectively the fruit rot caused by these pathogens is referred to as the *Dothiorella/ Colletotrichum* Complex. In Australia it has been found that Fuerte, Nabal, Wurtz and Rincon cultivars are more susceptible. Hass is also susceptible but the anthracnose spots are not clearly visible against the background of the black peel of ripe fruit (Faber and Ohr 1999, Havenga *et al.*1999, Manicom 2001, Pegg 1991, Prusky 1998).

2.3.1 Symptoms

The causal fungus can infect healthy leaves, stems and young fruit. The most distinct symptoms occur on the fruit. In the field, fallen leaves and those remaining on the tree may have large brown necrotic areas appearing in the center and on their margins (Fig. 5). Two types of symptoms occur before harvest, one consists of small lesions located around the lenticels on the peel of young fruit, which may result in reduced fruit quality and fruit drop. The second type consists of large spreading areas resulting from secondary infection during feeding of insects or mechanical damage. Following infection there is no further development until the fruit starts to ripen after harvest. The most classic symptom then observed is a large spreading lesion, dark brown to black, firm at first, penetrating the flesh below in a hemispherical pattern, down to the seed. Under moist conditions, slimy pink spore masses erupt from matured acervuli (Faber and Ohr 1999, Manicom 2001, Pegg 1991, Prusky 1998).

2.3.2 Causal organism and Epidemiology

The causal organism *Colletotrichum gloeosporioides* (Penz) Penz & Sacc., has a wide host range. It is a natural inhabitant of California avocado and citrus groves, where it grows on dead twigs and leaves and is normally of little importance owing to the

Mediterranean climate. The spores on the dead leaves and twigs and mummified fruit in the tree canopies are the primary sources of overwintering inoculum. Large dense canopies of older trees create a suitable microclimate for the production of high levels of spores. Infection can take place at any time from fruit-set to harvest. Free water in the form of dew or rain encourages spore production and enhances spore dispersal and infection of fruit. In countries such as California and Israel long periods of dew can

Figure 4 : Healthy white creamy coloured feeder roots in avocado tree affected by *Phytophthora citricola.*

enhance infection.

Quiescent infections in which the fungus infects , penetrates and then enters a dormant phase, are very common. This dormancy is caused by the presence of antifungal compounds called dienes, in the peel of unripened fruit. As the fruit ripens the concentrations of these dienes decrease below fungistatic levels, the infecting hyphae is activated and the fungus spreads into the surrounding tissues. Light brown spots appear on the peel, they rapidly enlarge, darken and induce the classic symptoms (Faber and Ohr 1999, Manicom 2001, Pegg 1991, Prusky 1998).

2.3.3 Disease Management

Cultural practices which reduce the inoculum and create conditions unsuitable for infection in particular in high rainfall areas, are *e.g.* pruning out of dead twigs and branches before flowering, removal of mummified fruit, untangling and removal of dead leaves from the tree canopy and ensuring good ventilation, by pruning lower limbs so
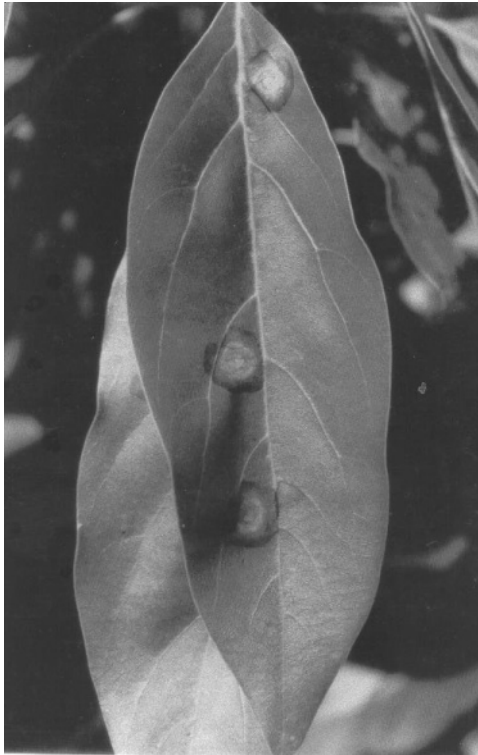

Figure 5: Leaf symptoms caused by the anthracnose fungus *Colletotrichum gloeosporioides* in avocado.

that the canopy is at least 50 cm above the ground. Preventative high volume sprays of copper fungicides will reduce infection of both anthracnose and Cercospora spot. During wet months, combinations of copper fungicides and systemic fungicides such as prochloraz and benomyl have been used in Australia and South Africa.

For post harvest control, chemicals which penetrate the peel such as prochloraz have been used in Australia and South Africa in the form of cold water fruit dips, for a period of up to 5 minutes. In South Africa a product by the name of Avogreen®, a *Bacillus* spp based biocontrol agent, is also reported to be effective. Pre-cooling of fruit as soon as possible after harvest is critical to preventing and reducing post harvest

decay. Pre-cooling to 7EC within 6 hours of harvesting has been found to be essential (Darvas and Kotzé 1987, Faber and Ohr 1999, Havenga *et al.*1999, Manicom 2001, Pegg 1991,Prusky 1998).

2.4 *Dothiorella* stem end rot

This disease does not occur as often as anthracnose but can cause significant losses under suitable conditions. This is a post harvest disease which is initiated in the field and becomes a particular problem when storage conditions are sub-optimal. This disease is an occasional but minor post harvest problem in California (Manicom 2001, Johnson and Kotzé 1998, Pegg 1991,).

2.4.1 Symptoms

Dothiorella fruit rot is distinct from stem end rot in that portions of the fruit other than the stem end are affected. Stem end rot appears within 4-7 days as a slight shrivelling of the stem end of the fruit, followed by the development of a dark brown to black rot of the fruit with a fairly defined margin. Internally the pulp is water soaked and brown, while the vascular bundles are browned in advance of the decay (Manicom 2001, Darvas and Kotzé 1987, Johnson and Kotzé 1998, Pegg 1991).

2.4.2 Causal organism(s) and Epidemiology

The most frequently isolated organism in South Africa is *Thyronectria pseudotrichia* (Berk. & M.A. Curtis) Seeler, followed by *Colletotrichum gloeosporioides, Phomopsis perseae* Zerova and *Dothiorella aromatica. Lasiodiplodia theobromae* (Pat.) Griffin & Maubl., is predominant in Israel. In Australia, New Zealand and the United States, *Botryosphaeria* spp. And their anamorphs predominate. The fungi survive on dead leaves and branches where they produce large numbers of spores . During the rainy periods , the spores are deposited on the fruit. Picking wet fruit enhances the disease. The spectrum of organisms which cause stem-end rot depends on the environmental conditions, with *L. theobromae* being promoted by hot conditions and *C. gloeosporioides* and *T. pseudotrichia* infections promoted by wet conditions. Several of the above organisms occur as endophytes in avocado stems and can infect fruit from endophytically colonized inflorescence and stem-end tissues. Symptoms of these infections are not apparent until fruit development is well advanced. Rot appears only once the fruit has matured and started to soften (Manicom 2001, Darvas and Kotzé 1987, Darvas *et al.* 1987, Johnson and Kotzé1998, Pegg 1991).

2.4.3 Disease Management

Endophytic infections in the field can be reduced by avoiding water stress and maintaining optimal nutrition in trees. Tree management practices recommended for anthracnose control combined with copper and benomyl sprays will reduce inocula of stem-end rot fungi. Controlled ripening at 16-18EC in combination with post harvest fungi-

cides such as prochloraz, will also improve control. The application of anti-oxidants in combination with ascorbic or citric acid has reportedly been effective in Israel. Fruit with a short (1-3 days) green life should be cooled promptly following harvest, to 7˚C. Lower temperatures cause chilling injury (Darvas and Kotzé 1987, Pegg 1991, Johnson and Kotzé 1998).

2.5 *Dothiorella* stem canker

Dothiorella stem canker can cause occasional losses of older avocado trees which are weakened by water stress during hot dry summers or damaged by frost during the winter period (Faber and Ohr 1999, Johnson 1998, Zentmyer *et al.* 1965).

2.5.1 Symptoms

The principal evidence of infection is the presence white powder deposits of tree exudates from splits in the bark (Fig. 6). Cankers occur on twigs (Fig. 7), branches or trunks with the bark cracking and being shed on affected limbs. The canopies of affected trees can become unthrifty and die back; in severe cases tree death may occur (Faber and Ohr 1999, Johnson 1998, Zentmyer *et al.* 1965).

2.5.2 Causal organism and Epidemiology

Dothiorella stem canker is attributed to the anamorph of *Botryospahaeria dothidea* (Moug.:Fr.) Ces & De Not, which was once regarded as *Dothiorella gregaria* Sacc.. *Dothiorella* spp. occur as endophytes in avocado stem tissue . Die-back and cankers are thought to be as a result of infections of tissues which have been weakened by water stress, sub-optimal nutrition or attack by mites and insects. Dead leaf litter and rotted fruit are a source of conidia produced by *Dothiorella* spp. and ascocarps produced on dead twigs are a source of ascospores of *Botryosphaeria spp.* Infection can occur through mechanical wounds caused by pruning, via abscission scars or directly through the epidermis. This is followed by endophytic colonization of the tissues and under stress conditions can result in the fungi becoming necrophytic. Leaf litter which accumulates in tree crotches , lower branches or around trunks creates conditions favorable for infection, especially if the litter is waterlogged (Faber and Ohr 1999, Johnson 1998, Zentmyer *et al.*1965).

2.5.3 Disease Management

Correcting moisture and nutritional stresses and the removal of diseased tree parts will reduce the incidence of infection. Mexican cultivars are more resistant to this disease than those of Guatemalan origin. Do not allow leaf litter and debris to accumulate in crotches, around lower branches or at the base of the trunks, especially if the tree is on Guatemalan rootstock or if the scion is Guatemalan and the bud union close to the ground. Application of foliar copper sprays can be beneficial but are usually not necessary if trees are maintained in a vigorous condition (Faber and Ohr 1999, Johnson 1998,

Zentmyer *et al.* 1965).

2.6 *Cercospora* spot

Cercospora spot is one of the most common diseases of avocado fruit in Florida, South Africa and other warm humid production areas where rainy conditions persist during the summer. This fruit disease is a major limiting factor in the export of avocado fruit from South Africa. This is mainly a cosmetic disorder , also known as black spot, and can result in losses of up to 70% if trees are left unsprayed. In South Africa, Fuerte and

Figure 6: Canker with white sugary exudate caused by *Dothiorella gregaria* in avocado
            trees.

Ryan cultivars are considerably more susceptible to the disease than Edranol or Hass. The disease was first reported from Florida in 1920, but in South Africa it only became a problem during the wet seasons of the late 1960s and is now prevalent throughout the avocado production regions of the country (Manicom 2001, Ruehle 1958, Pohronezny *et al.* 1998).

2.6.1 Symptoms

Symptoms occur on leaves, stems and fruit. The lesions on the leaves are 2-3 mm in diameter, and usually appear on the lower leaf surfaces first. On older leaves the lesions are angular and purplish brown in color. The angular appearance of the lesions is highly diagnostic for the disease. The lesions eventually appear on both leaf surfaces and are surrounded by chlorotic halos. Lesions can coalesce to form large necrotic areas often surrounded by smaller satellite spots. Under humid conditions spores are produced in

Figure 7: *Dothiorella* stem canker in young twigs of an avocado tree. Note the extensive white sugary exudate.

the lesions on the lower leaf surface and appear as gray, hairy mats.

The first sign of infection on fruit is a darkening of the epidermis followed by a swelling of the underlying tissue which raises the small spots. The spots on the fruit are more or less circular and become slightly sunken later on as the infected cells dry out. Frequently small cracks or fissures appear within the lesions through which the anthracnose causal organism *Colletotrichum gloeosporioides* gains entrance. Cercospora spot is normally confined to the peel of the fruit but can also invade the pulp during advanced stages (Manicom 2001, Ruehle 1958, Pohronezny *et al.* 1998).

2.6.2 Causal organism and Epidemiology

Cercospora spot is caused by the fungus *Pseudocercospora purpurea* (Cooke) Deighton. The fungus is difficult to isolate initially, but once isolated it grows readily on ordinary nutrient media, producing a tufted leathery growth, which is first greyish in color, later becoming brown or blackish-brown.

The primary source of inoculum is infected, mature leaves. The pathogen survives on old lesions and when the warm rainy period commences, spores are produced and dispersed by splashing rain, water from sprinkler irrigation systems, wind and insects. Fruit < 40 mm in diameter and those close to maturity are immune to infection. Fruit are more susceptible when the size is between 25-75% of their final size. In South Africa the fungus remains latent for approximately three months following infection, before spots are observed. In this country it has been observed that the severity of Cercospora spot is correlated with the severity of root rot caused by *Phytophthora cinnamomi* (Manicom 2001, Pohronezny *et al.* 1998).

2.6.3 Disease Management

The disease can be effectively controlled by timely spray applications of fungicides such as copper and /or benomyl. In South Africa the timing of spray applications are based on a predictive model designed by J.M. Darvas. This model uses mean weekly temperature and weekly rainfall as criteria . The equation for this model is as follows:

$$Z = -58.99 + 3.22(\text{Mean weekly temp.}) + 0.18\ (\text{Weekly rainfall in mm})$$

If Z∃ 20, the potential for infection is high. Spraying should begin when Z∃ 15. Copper oxychloride is applied as a full cover spray and repeated at 30 and 60 day intervals. If Z>20 when fruit is in the susceptible phase the second application is replaced with benomyl. It has been found vital to apply the first spray before Z values exceed 20. Biological control with *Bacillus* spp. was found to be effective in field experiments in South Africa (Havenga *et al.* 1999, Manicom 2001).

Effective fungicidal control is more difficult in late maturing cultivars such as Choquette, Lula and Monroe, and requires more frequent sprays (Pohronezny *et al.* 1998).

2.7 *Verticillium* wilt

Verticillium wilt was first described in California in 1949. It has also been reported from several other avocado producing countries, including Spain, Australia, Chile, Ecuador and South Africa. It is a disease of minor importance in these countries and is far less common than *Phytophthora* root rot. The disease is more likely to occur when avocado trees are planted on land previously cultivated to susceptible crops such as tomatoes, eggplant, potatoes, pepper, berries, apricot and certain flower crops (Faber and Ohr 1999, Manicom 2001, Pegg 1991, Zentmyer 1949).

2.7.1 Symptoms

Leaves suddenly wilt and rapidly turn brown and die on one or several branches or even the entire tree. The leaves can remain attached to the tree for several months. When the bark of affected limbs and roots is peeled, brown to grey-brown longitudinal streaks can be observed in the woody tissues (Fig. 8). Very often affected trees will send out new vigorous shoots within a few months following the initial collapse of the tree , and the tree may recover completely, with no recurrence of the disease. In Australia older trees have been known to recover, with symptoms reappearing in subsequent years (Faber and Ohr 1999, Manicom, 2001, Pegg 1991, Zentmyer 1949 and 1998b).

2.7.2 Causal organism and Epidemiology

The causal fungus *Verticillium dahliae* Kleb. is a common soil inhabitant and may survive for decades in the soil as dormant microsclerotia. It invades the tree through the roots, colonizing the water conducting tissues of the trunk and branches. In California the disease occurs sporadically but is more common in the cooler coastal regions than the warm , drier inland areas. The pathogen is endemic to California occurring on a number of indigenous hosts, and can be a minor problem on avocado planted on newly cleared areas (Faber and Ohr 1999, Pegg 1991, Zentmyer 1998b).

2.7.3 Disease Management

Preventative measures play the major role in managing this disease. Trees should not be planted on land previously cultivated to susceptible crops nor interplanted with such crops. Do not use trees that have a history of Verticillium wilt as sources of propagative material. Dead branches should be pruned out after die-back has stopped and new growth commenced. Prunings should be burnt. Continue with standard fertilization and irrigation. On sites where the disease is prevalent it is advisable to use Mexican rather than Guatemalan rootstocks; the former appear to be more resistant to the disease. If the disease has caused the death of a tree , the entire area under the canopy and planting hole should be fumigated with a fungicidal fumigant (Faber and Ohr 1999, Manicom 2001, Zentmyer 1998b).

2.8 *Armillaria* root rot

Armillaria root rot is a minor problem in most countries where avocado is cultivated. The disease has been reported on avocado in Spain, Ecuador, California, Florida and South Africa. The pathogen is a common soil inhabitant and has a wide host range worldwide. When it occurs it is limited to small areas and does not threaten the destruction of the orchard. This is in contrast to *Phytophthora* root rot which can invade large areas. Two *Armillaria* spp. are implicated viz. *A. mellea* (Vahl:Fr.) P. Kumm. also known as the oak root fungus, the shoestring fungus and the honey mushroom; and *A. socialis* (DC:Fr.) Herink. The latter species is reported to cause the disease in Florida (Faber and Ohr 1999, Manicom 2001, Ohr and Zentmyer 1998a).

2.8.1 Symptoms

The pathogen becomes well established in the root system before any visible effects appear in the tree canopy. There may be a gradual deterioration in tree vigour with yellowing of the foliage and leaf drop over part or all of the tree, or infected trees may undergo a rapid wilt and die. The most diagnostic characteristic of the disease is the white fan-shaped mycelial mats that occur just under the bark of infected roots (Fig. 9). Purplish-brown cord-like rhizomorphs that resemble feeder roots can be found growing along the root surface or for short distances into the soil. Dark structures known as pseudosclerotia, protrude through cracks in the bark. At low elevations in the tropics, rhizomorphs are not found in the soil or on roots. In winter rainfall areas where the
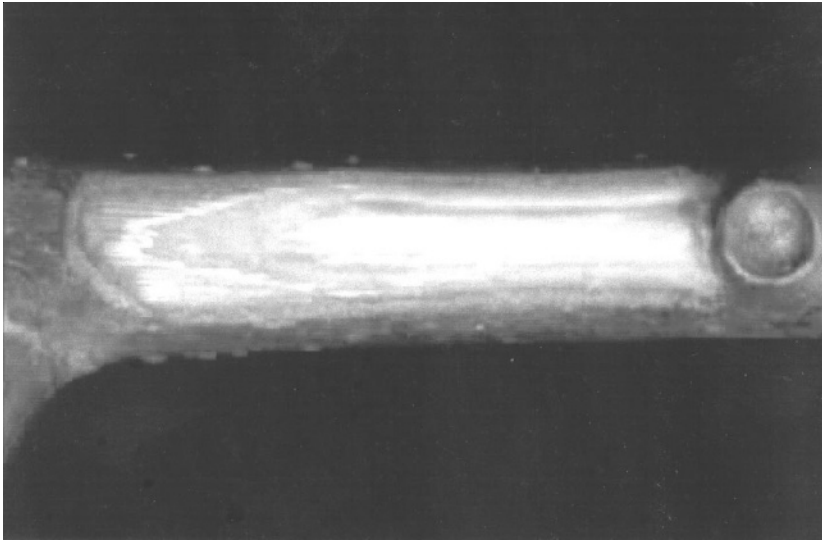
Figure 8: Vascular streaking caused by *Verticillium dahliae* beneath the bark of avocado tree. (Courtesy B.A.Faber)

temperatures are not too low, such as in the California avocado production areas, the fungus often produces mushrooms under the infected trees (Fig.10). The appearance of the mushroom cap is variable, it may range from a cream to honey-yellow to almost black and may have a covering of brown scales (Faber and Ohr 1999, Ohr and Zentmyer 1998a).

2.8.2 Causal organisms and Epidemiology

A. mellea is the major fungal pathogen implicated in Armillaria root rot. In Florida it is reportedly caused by *A. socialis.* The latter is distinguished from *A. mellea* by the

absence of annuli on its basidiomes. *A. mellea* is indigenous to many environments and is often found on hillsides and along watercourses. It has a host range in excess of 600 species, including oak and a number of other indigenous species in avocado-growing areas. The fungus inhabits the soil as a colonizer of large roots and other wood pieces, and cannot survive without such a food base. Infection takes place when a host root comes into contact with a root or food base that is colonized by the pathogen, or when it is invaded by a rhizomorph. The pathogen penetrates the host and grows rapidly up the root in the cambial region until it reaches the crown where it girdles and kills the tree. From the crown it then grows down other roots into the soil or roots of adjacent trees if they are in contact. The Armillaria fungus spreads from place to place in infected wood pieces including infected root fragments. This inoculum may be dispersed by flood water, leaf mulch gathered from underneath infected trees, by cultivating equipment, or by any activities that might move infected plant material. Trees that are planted in cleared forest areas, on hillsides or flood plains to which water has moved infected root pieces, can become infected. The disease can spread radially from an initial infection locus , killing a new ring of trees each year.

Although a great number of spores are produced by the mushrooms (basidiomes) , they are not considered to be an important source of infection in avocado orchards (Faber and Ohr 1999, Ohr and Zentmyer 1998a).

2.8.3 Disease Management

The control of Armillaria root rot using cultural methods is a very difficult task. The pathogen is sensitive to drying, and in citrus, the spread of the pathogen can be retarded by exposing the root crown and large roots to the air. In South Africa control has been achieved by removing the entire tree and larger roots followed by leaving the planting hole open for a period of 3-6 months, before replanting. The preferred method is soil fumigation with methyl bromide under soil conditions which allow free movement of the fumigant throughout the treated area (Faber and Ohr 1999, Manicom 2001, Ohr *et al.* 1973).

2.9 *Rosellinia* (Dematophora) root rot

Rosellinia (Dematophora) root rot, also known as white root rot, is most common on deciduous fruit trees and grapes. It has been reported from avocado in Israel, Spain and California. The disease has a wide geographical distribution in all temperate regions throughout the world and has a very wide host range which includes approximately 170 species in 63 genera (Khan 1955, Sztejnberg 1998).

2.9.1 Symptoms

The above ground symptoms are typical of those caused by other soil borne pathogens such as *Armillaria mellea* and *Phytophthora cinnamomi.* These symptoms include yellowing of the foliage, absence of new flush, fruit growth is arrested and fruit may shrivel. The affected trees can persist for 2-3 years and later succumb to the disease, or

be killed within a single year when root infection is severe. The symptoms below soil level are more diagnostic and are characterized by the presence of strands of white cottony mycelium on the surface of infected roots. The mycelium forms a continuous white layer of mycelium under the bark, turning greenish-grey to black with time (Sztejnberg 1998).

2.9.2 Causal organism and Epidemiology

Figure 9: Root rot of young avocado tree caused by *Armillaria mellea.* Note the white fan shaped mycelial mats. (Courtesy J.A. Menge)

The causal organism of Dematophora root rot is *Rosellinia necatrix* Prill. The pathogen can survive for extended periods on old rotted roots left in the ground, and under some conditions persist as mycelium or loose microsclerotia in the soil or adhering to root debris. The pathogen is disseminated along and within roots of infected plants to roots of adjacent trees, infested soil distributed by cultural operations including movement of nursery stock. Infection takes place by direct penetration of fine roots by active

mycelia. The hyphae then branch freely in the cortex , growing toward the larger roots whilst destroying the tissues en route. The pathogen emerges from the rotted cortical region to form the characteristic white mycelium on the roots. The mycelial webs turn greenish-grey when they reach the surface of the roots.

Optimal conditions for Dematophora root rot include high soil moisture (near to field capacity) and temperatures of 20-25EC. The disease is most common in heavy soils in which it can persist for several years. It spreads rapidly along drip irrigation

Figure 10 : Mushrooms (basidiomes) of *Armillaria mellea* at the base of the stump of a dead avocado tree. (Courtesy J. A. Menge)

lines taking advantage of the high ambient moisture which favors its rapid growth (Khan 1955, Raabe and Zentmyer 1955, Sztenjnberg 1998).

2.9.3 Disease Management

Control is based on sanitation measures , soil fumigation with methyl bromide and soil solarization. The latter procedure appears to have a long term effect on controlling the

pathogen in naturally infected soils. Sanitation measures include , removal of severely affected trees and their root systems, discontinuing irrigation of infected trees to re-duce spread of the pathogen and isolating infection foci in an orchard by trenching. All Guatemalan, Mexican and West Indian rootstocks tested, were found to be suscep-tible (Sztejnberg *et al.*1983 and 1998).

2.10 Scab

Scab is a serious problem on avocado in the humid tropics and subtropics. Severe yield
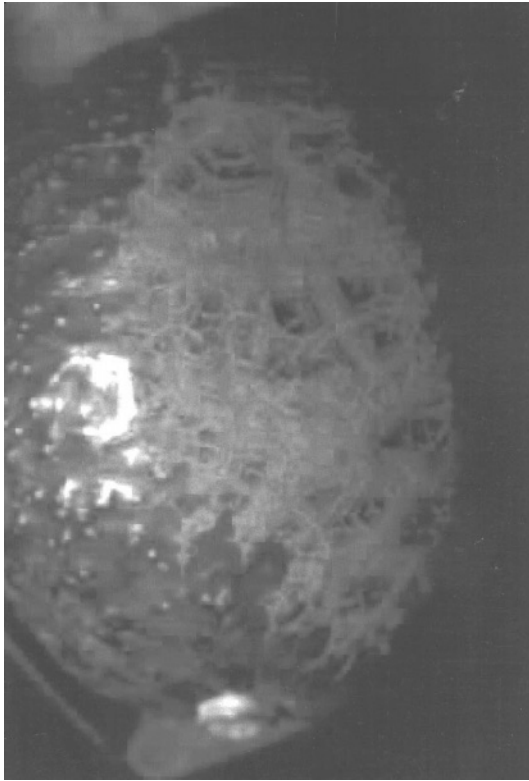
Figure 11 : Severe scab caused by *Sphaceloma perseae* on avocado fruit.

losses may result from the premature abscission of infected fruit and culling of mature fruit which is cosmetically unacceptable for export. Cultivar susceptibility varies con-siderably with Lula being extremely sensitive, followed by Booth 3, 5, 7, and 8, Monroe, Choquette and Trapp, which are moderately susceptible. The cultivars Booth 1, Collins and Pollack appear to be most resistant (Rhuehle 1958, Stevens and Piper 1941, Pohronezny and Simone 1998).

2.10.1 Symptoms

The most distinct symptoms occur on the fruit with leaf symptoms being less conspicuous as they are mostly out of sight in the upper part of the tree canopy. Fruit lesions are initially oval to irregular in shape, slightly raised and brown to purplish-brown. The lesions enlarge and coalesce as the disease progresses, forming large, rough areas over the fruit surface (Fig. 11). Invasion by secondary organisms may result in substantial fruit decay. Only young fruit and leaves are susceptible; they become resistant with age.

Foliar symptoms commence as small, discrete spots, <3.5 mm in diameter. The lesions often concentrate long the leaf veins. The leaves become crinkled and distorted as the disease progresses, lesions coalesce into star-like patterns, and shot-holes develop. Oval to elongate lesions also occur on twigs and pedicels (Manicom 2001,Pohronezny and Simone 1998).

2.10.2 Causal Organism and Epidemiology

The causal organism of avocado scab is *Sphaceloma perseae* Jenk. (Jenkins 1934a, Jenkins 1934b). The disease is exacerbated by cool moist weather and is most severe when heavy rains and fog occur during the susceptible growth stage of tissues. Emerging young tissues are most susceptible, with leaves becoming resistant to infection one month after emergence and fruit once they have attained half their normal size (Manicom 2001, Pohronezny and Simone 1998).

2.10.3 Disease Management

An integrated approach to control, which includes the use of resistant cultivars and fungicidal sprays of copper or benomyl are advocated, to reduce damage and economic losses caused by scab. When warranted, control measures should be applied during the various flowering stages. Three sprays are critical: one as flower buds appear. The second near the end of the bloom period, followed up by a third spray 3-4 weeks later (Manicom 2001,Pohronezny and Simone 1998).

3. Bacterial canker

Bacterial canker and a disorder with similar symptoms have been reported from all major avocado-producing countries of the world. Although the symptoms appear similar the causal organism of bacterial canker in California differs from that of the South African pathogen. No causal organism has been found associated with this disorder in Australia, it is believed to have an abiotic etiology. The incidence of this disease is normally low and of little consequence, but in some orchards the incidence can be very high, especially if an orchard is established from a consignment of nursery trees which was infected in the nursery (Korsten and Kotzé 1987, Cooksey *et al.* 1993, Cooksey *et al.*1998)

3.1 Symptoms

The initial visual symptom of bacterial canker on the bark is a sunken dark area with a watery necrotic pocket underneath. As the canker develops, the bark splits at the edge of one side of the lesion allowing a watery fluid to ooze out. As the fluid dries, it leaves a white powdery residue (xylitol) around the periphery and sometimes over the lesion

Figure 12: Bacterial canker lesions caused by *Xanthomonas campestris* on avocado tree in California.

(Fig. 12). Reddish brown necrotic tissue is typically present in the cortex under the canker, with streaks of the same color extending above and below the canker. In South Africa these cankers can be 20-100 mm in diameter, usually appearing at the base of the tree from where they spread upwards, mostly in a straight line. The streaks extend 300 mm above and below the canker pocket. Necrotic streaks between cankers are usually in the wood and sometimes towards the center of branches or trunks. No leaf or fruit symptoms are associated with these canker diseases. However severely affected trees exhibit retarded growth, sparse canopies, and produce low yields (Faber and Ohr 1999, Manicom 2001, Cooksey *et al.* 1993, Korsten and Kotzé 1987).

3.2 Causal organism and Epidemiology

Bacterial canker was first reported from South Africa in 1987. The causal organism found to be associated with this disorder was *Pseudomonas syringae* (van Hall) Bergey *et al*. A similar organism was isolated from canker lesions in Australia but was found to be non-pathogenic in avocado seedlings. This disorder was attributed to a boron deficiency. It is however now reported that this pathogen does cause bacterial canker in Australia and that trees which are suffering from a boron deficiency are more susceptible to the disease, in both Australia and South Africa.. In California, bacterial canker is caused by a pathovar of *Xanthomonas campestris,* and as California avocado soils are not deficient in boron, this should therefore not be a factor in determining the susceptibility of avocado trees to bacterial canker in this country. Infected nursery stock plays a major role in the spread of this disease since young cankerous lesions have been found to occur on newly planted trees. The epiphytic stage of *P. syringae* has also been detected on leaves and twigs of infected trees (Cooksey *et al.* 1993, Korsten and Kotzé 1987, Cooksey *et al.* 1998).

3.3 Disease Management

The use of disease-free nursery stock, removal of infected trees and maintaining the vigor and thriftiness of mature trees are key factors in controlling the spread of bacterial canker. Because of the systemic nature of this disease, topical therapy will not be successful (Korsten and Kotzé 1987, Cooksey *et al.*1998).

4. Avocado sunblotch

Avocado sunblotch (ASB) was first described in California in 1928 as a physiological disorder. In the 1930s it was shown to be graft transmissible and for many years it was considered to be a virus disease (Horne and Parker 1931). In the 1970s , viroids as disease causing agents, were described, and it was then speculated that ASB was caused by a viroid. It was only in 1979 that the disease was confirmed as being caused by a viroid (Palukaitis *et al.* 1979). The disease has since been reported in most avocado growing countries of the world, these include Florida, Spain, Australia, Israel, Peru, Venezuela and South Africa (Luttig and Manicom 1999, Manicom 2001, Ohr *et al.* 1998). It has been considered of minor importance in the in the past decade but may a increase in significance owing to the introduction of new canopy management practices, trunk injection to control *Phytophthora* root rot, and the use of infected seed. ASB is the only known graft transmissible disease of avocado at present.

4.1 Symptoms

Symptoms occur on leaves, twigs , fruit and bark of the trunk and larger branches. Leaves on severely infected trees appear distorted and are variegated in color (Fig.13). Symptoms on twigs include a white to yellowish , red or necrotic streaking that is often associated with a shallow indentation along the length of the twig (Fig. 14). Fruit symp-

toms are similar though the discoloration is reddish in black skin avocado cultivars (Hass), in green maturing cultivars (Bacon and Fuerte) the symptomatic areas remain white or yellowish (Fig. 15). Rectangular cracking of the bark on trunks and larger branches forming a pattern that is commonly referred to as "alligator skin" has also been associated with the disease (Fig. 16). Affected trees are often stunted exhibiting sprawling growth (Fig. 17). Symptomatic trees may have drastically reduced yields. Asymptomatic infected trees referred to as "symptomless carriers" also occur in avocado orchards; the only sign that these trees may be infected is indications of drastically reduced yields. (Desjardins *et al*., 1980, Horne and Parker 1931, Luttig and Manicom 1999, Manicom 2001, Ohr *et al.* 1998).

Figure 13: Symptoms of sunblotch on avocado leaves. (Courtesy B. A .Faber)

4.2 Causal organism and Epidemiology

The causal agent of avocado sunblotch disease is a viroid consisting of approximately 247 nucleotides with known variants ranging from 246-251 bases. The avocado sunblotch viroid (ASBVd) is the smallest of known plant viroids. The spread of ASBVd is mostly through infected nursery trees propagated from infected budwood or infected rootstock seedlings used for grafting.. Seed from symptomless carriers has been found to be 100% infected, whereas 0-5% of seed from symptomatic trees is affected. A low percentage of pollen transmission, and mechanical transmission by means of knife slashes, has been recorded under experimental conditions. There is no evidence of insect transmission. Natural root-to-root grafting can also spread the disease. As the ASBVd has been found to be mechanically transmitted by knife slashes as is the case

with exocortis viroid in citrus, it is suspected that there may be a major risk of spreading the ASBVd during mechanical pruning of tree canopies and during drilling for phosphonate injection, used for the control of *Phytophthora* root rot. Infected sap can be transferred from infected trees on the implements used, to adjacent healthy trees (Luttig and Manicom 1999, Manicom 2001, Pegg 1991, Ohr *et al.* 1998).

4.3 Disease Management

The most dangerous factor in ASBVd infection is the presence of "symptomless carri-
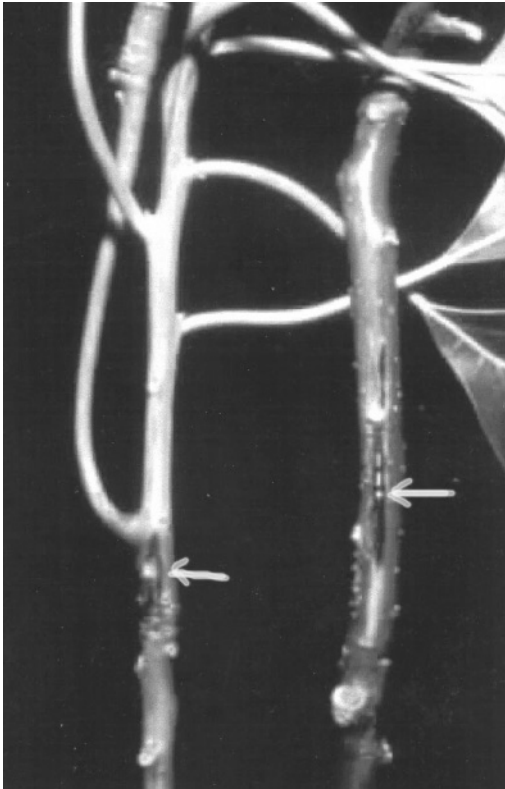
Figure 14: Sunken streaks on avocado branches caused by avocado sunblotch viroid.
(Courtesy B. A. Faber)

ers". A study in South Africa has shown that over a three year period the mean loss in yield in Fuerte trees of this type was 14 % and owing to sub-grade fruit the value of the crop was 40% lower. On symptomless Edranol trees the yield loss in a study over one year was 80%. In a study conducted over several years at Westfalia Estates, Tzaneen ,consistently good producers yielded approximately 470 kg of fruit per tree annually, whereas poor producers only yielded 9 kg. Subsequent indexing with sensitive bio-

chemical methods now available, showed that 72% of the poor producers were symp-
tomless carriers of the ASBVd.(Luttig and Manicom 1999, Manicom 2001).

     The primary control measure for ASBVd disease is the use of registered viroid-
free nursery trees. Trees with symptoms in the field should be removed and the remain-
ing stumps killed; the low yield and potential for disease spread does not justify their
retention. Pruning tools, injectors and harvesting clippers should be disinfected be-
tween trees. The most common and effective disinfectant is 1.5% sodium hypochlorite
solution. Poor yielding trees should be tested for ASBVd, and if found infected , be
removed. These trees should by virtue of the fact that they are poor producers, irre-
spective of whether the are infected or not, be removed. Usually infected trees, by
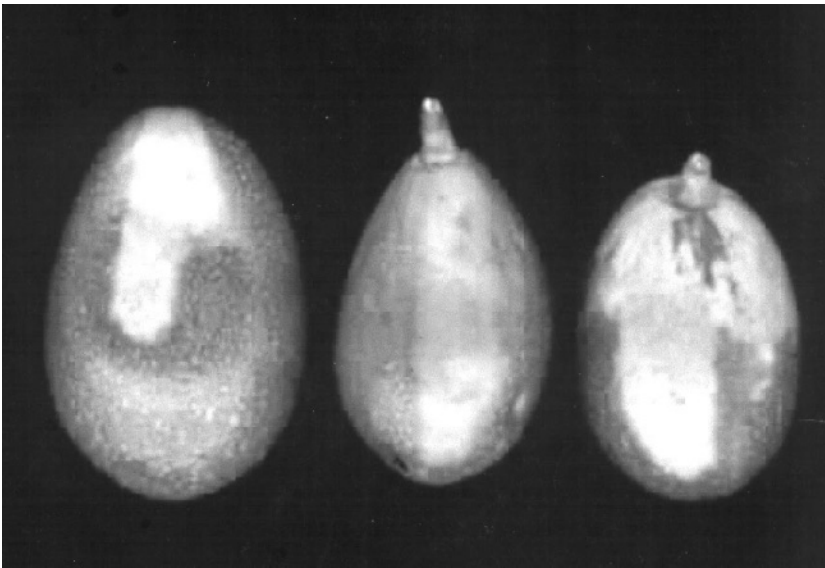
Figure15: Sunblotch symptoms on Hass avocado fruit. (Courtesy B.A. Faber)

reason of their poor growth, can be identified in the field within the first two years. The
incidence of ASB has decreased dramatically in South Africa and Australia since com-
mercial screening of nursery mother trees was implemented (Manicom 2001, Pegg 1991,
Ohr *et al.* 1998). There are indications in California that with the introduction of severe
pruning for management of canopy growth, and maintaining production in mature trees,
the incidence of ASBVd affected trees may be increasing.. This observed increase may
be as a result of the mobilization of the ASBVd in the new growth, following pruning of
symptomless carriers. A similar phenomenon has been observed in citrus in Israel
where the incidence of exocortis viroid, impietratura and citrus tristeza virus were at low
incidences (Bar-Joseph and Ben-Shalom 1982, Bar-Joseph *et al.* 1989).

5. Avocado black streak

Avocado black streak (ABS) appears to be unique to the avocado producing regions of California, stretching from San Luis Obispo County in the north, to San Diego County in the south and inland to Riverside County. It has only been observed on a single occasion in Florida and once in the Canary Islands, on trees introduced from California.

Figure 16: Rectangular cracking of bark ("alligator skin") associated with avocado sunblotch viroid disease. (Courtesy B.A.Faber)

ABS may occur wherever Guatemalan cultivars are grown in California. ABS may have been present at a very low incidence in California since 1934, but it is only since the increase in acreage of the susceptible Hass cultivar, that it has become a serious problem. Trees of all ages are affected and symptoms have been observed on trees as young as one year-old to more than 35 years old. ABS will not be present in all of the orchards in an area , with the incidence varying from orchard to orchard (Faber and Ohr 1999, Zentmyer 1951, Ohr and Murphy 1987, Ohr and Zentmyer 1998b).

5.1 Symptoms

ABS appears after prolonged periods of environmental or cultural stress such as low or it may collapse rapidly. Fruit production is usually poor on affected trees. The most characteristic diagnostic feature of ABS however is the presence of cankers on the trunk and branches. The cankers usually appear first on the lower trunk, occasionally on the upper trunk, or on the underside of a lower branch (Fig. 18). The cankers on the lower trunk can be confused with those caused by the stem canker pathogen *Phytophthora citricola*, but unlike these cankers, ABS cankers are superficial. The cankers are characterized by the accumulation of a dry powdery residue which is at first a cinnamon color, but turns white as more of it accumulates and dries. This is as a result

Figure 17: Sprawling growth in avocado tree affected by avocado sunblotch viroid disease.
(Courtesy B.A. Faber)

of a sugary substance that exudes through minute cracks in the bark and on being exposed to the air , dries into a powdery residue (xylitol). The residue is water soluble and can be washed away by rain, making it difficult to find the cankers. The cankers can be a few millimeters in diameter or large enough to encompass the entire trunk or branch.

The cankers do not favor any particular side of the tree as do those of stem canker caused by *P. citricola*, where the cankers usually develop on the side closest to the irrigation emitter, which wets the trunk at the site at which the canker develops. Scraping off the bark over the canker reveals shallow reddish brown areas that rarely extend into the cambium. These areas can often be removed easily by prying with a knife blade. Trees can die from ABS, irrespective of the number or extent of the canker lesions. The lesions appear to be a result of the disorder and not the cause of death.

Other symptoms of the disease include chlorosis, early bloom (5-10 days earlier)

branch die-back, leaf blotching, zinc deficiency, bunchy growth, wilting of foliage and rapid death of new growth (Faber and Ohr, 1999, Zentmyer 1951, Ohr and Murphy 1987, Ohr and Zentmyer 1998b).

5.2 Causal organism and Epidemiology

Figure 18: Symptoms of black streak in avocado. Note the white sugary exudate and black stained area on bark.

The causal organism is unknown. Observations of spread in the field have not yet been substantiated by artificial inoculation or graft transmission.

5.3 Disease Management

Current management of ABS is based on sound cultural practices which include optimal irrigation, fertilization and pest control to prevent stress and maintain vigor and thriftiness (Faber and Ohr 1999, Ohr and Zentmyer 1998b).

6. References

Bar-Joseph, M. and Ben-Shalom, J. 1982. Limited systemic spread of impietratura and psorosis-A in graft -inoculated grapefruit trees. Plant Dis., 66:820-821.

Bar-Joseph, M., Marcus, R. and Lee, R.F. 1989. The continuous challenge of citrus tristeza virus control. Annu. Rev. Phytopath., 27: 291-316.

Bender, G.S. 1999. Phytophthora canker or collar rot. AV-521. UC Cooperative Extension, Univ. California Davis, Davis. USA..

Bijzet, Z. and Sippel, A.D. 2001. Rootstocks. In: "The Cultivation of Avocado" (ed.. de Villiers, E.A.). Institute for Tropical and Subtropical Crops, Nelspruit, South Africa, pp.85-90.

Borst, G. 1970. Selection and management of avocado soils. Calif. Citrograph, 55:263-265.

Broadbent, P. and Baker, K.F. 1974. Behaviour of *Phytophthora cinnamomi* in soils suppressive and conducive to root rot. Aust. J. Agric. Res., 25:121-137.

Coffey, M.D. 1987. *Phytophthora* root rot of Avocado: An integrated approach to control in California. Plant Dis., 71: 1046-1052.

Coffey, M.D. 1992. *Phytophthora* root rot of avocado. In: "Plant Diseases of International Importance". Volume III. Diseases of Fruit Crops (eds. Kumar, J., Chaube, H.S., Singh, U.S. and Mukhopadhyay, A.N.) Prentice Hall, Englewood Cliffs.pp.423-444.

Cooksey, D.A., Ohr, H.D., Azad, H.R.., Menge., J.A. and Korsten L. 1993. *Xanthomonas campestris* associated with canker of avocado in California. Plant Dis.,77:95-99.

Cooksey, D.A., Ohr, H.D. and Korsten. L. 1998. Bacterial canker. In: "Compendium of Tropical Fruit Diseases" (eds Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D.), APS Press, St Paul, MN., USA, p.75.

Darvas, J.M., Toerien, J.C. and Milne, D.L. 1984. Control of avocado root rot by trunk injection with fosetyl-Al. Plant Dis., 68:691-693.

Darvas, J.M. and Kotzé, J.M. 1987. Avocado fruit diseases and their control in South Africa. S. Afr. Avocado Growers' Assoc. Yearbook, 10:117-119.

Darvas, J.M., Kotzé, J.M. and Wehner, F.C. 1987. Field occurrence and control of fungi causing postharvest decay of avocados. Phytophylactica, 19:453-455.

Desjardins, P.R., Drake, R.J. and Swiecki, S.A. 1980. Infectivity studies of avocado sunblotch disease causal agent, possibly a viroid rather than a virus. Plant Dis., 64:313-315.

El-Hamalawi, Z.A. and Menge, J.A. 1994. Avocado trunk canker disease caused by *Phytophthora citricola* : investigation of factors affecting infection and disease development. Plant Dis., 78:260-264.

El-Hamalawi Z.A., Menge, J.A. and Guillemet, F.B. 1994. Comparison of resistance to *Phytophthora citricola* in nineteen avocado rootstocks under greenhouse conditions. Avocado Yearbook California Avocado Commission**,** 78:121-129,

El-Hamalawi, Z.A., Menge, J.A. and Guillemet, F.B. 1995. Infection court and factors affecting expansion of stem canker of avocado caused by *Phytophthora citricola*. Plant Disease, 79:384-388.

Erwin, D.C. and Ribeiro, O.K. 1996. *Phytophthora* diseases worldwide. APS Press, St. Paul, MN., USA.562p.

Faber, B.A., El-Hamalawi, Z.A. and Menge, J.A. 1995. Trunk canker causing increasing damage. Calif. Grower, 19(2):38-39.

Faber, B.A. and Ohr, H.D. 1999. UC Pest Management Guidelines: Avocado. Univ. California Davis, Davis, CA, USA.

Havenga, W. De Jager, E.S. and Korsten, L. 1999. Factors affecting biocontrol efficacy of *Bacillus subtilis* against *Colletotrichum gloeosporioides*. SAAGA Yearbook, (22):12-18.

Horne, W.T. and Parker, E.R. 1931. The avocado disease called sunblotch. Phytopathology, 21:235-238.

Jenkins, A.E.. 1934a. A species of *Sphaceloma* on avocado. Phytopathology, 24:84-85.

Jenkins, A.E. 1934b. *Sphaceloma perseae* the cause of avocado scab. J. Agric. Res., 49:859- 869.

Johnson, G.I. 1998. Dothiorella Stem Canker and Fruit Rot. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, p.76.

Johnson, G.I. and Kotzé, J.M. 1998. Stem-End Rot. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D),APS Press, MN, USA, pp.81-83.

Khan, A.H. 1955. *Dematophora* root rot. Calif. Dept. Agric. Bull., 44:167-170.

Korsten, L. and Kotzé, J.M. 1987. Bark canker of avocado, a new disease presumably caused by *Pseudomonas syringae* in South Africa. Plant Dis., 71:850.

Luttig, M. and Manicom, B.Q. 1999. Application of a highly sensitive avocado sunblotch viroid indexing method. SAAGA Yearbook, (22):55-60.

Manicom, B.Q. 2001. Diseases. In: "The Cultivation of Avocado (ed. DeVilliers, E.A.), Institute for Tropical and Subtropical Crops", Nelspruit, South Africa, pp.214-230.

Menge, J.A., Guillemet, S., Campbell, S., Johnson, E. and Pond, E. 1992. The performance of rootstocks tolerant to root rot caused by *Phytophthora cinnamomi* under field conditions in Southern California. Proc. 2nd World Avocado Congress, pp. 53-59.

Menge, J.A. and Marais, L. J. 2000a. Soil Environmental Factors and their Relationship to Avocado Root Rot. Subtropical Fruit News, 8(1-2):11-14.

Menge, J.A. and Marais, L.J. 2000b. Strategies to control *Phytophthora cinnamomi* root rot of avocado. Subtropical Fruit News, Univ. of California (In Press).

Messenger-Routh, B.J. 1996. The effects of gypsum soil amendments on *Phytophthora* root rot of avocado. Ph.D. Dissertation, Univ. of Calif. Riverside.

Ohr, H.D., Munnecke, D.E. and Bricker, J.L. 1973. The interaction of *Armillaria mellea* and *Trichoderma* spp. as modified by methyl bromide. Phytopathology, 63:965-973.

Ohr, H.D. and Murphy, M.K. 1987. Blackstreak disease of avocado in California, S. Afr. Avocado Growers' Assoc. Yearbook 10:123-126.

Ohr, H.D., Faber, B.A. and Grech, N. 1994. Avocado diseases. Circular No. CAS-94/1. The California Avocado Society Inc., and The California Avocado Commission.

Ohr, H.D. and Zentmyer, G.A. 1998a. Armillaria Root Rot. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA. pp. 73-74.

Ohr, H.D. and Zentmyer, G.A. 1998b. Avocado Black Streak. In: "Compendium of Tropical Fruit Diseases" (eds. . Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN, USA, pp.74-75.

Ohr, H.D., Zentmyer, G.A. and Korsten, L. 1998. Sunblotch. In: "Compendium of Tropical Fruit Diseases" (eds. . Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D) APS Press, St. Paul, MN., USA. pp.83.

Palukaitis, P., Hatta, T., Alexander, D.M.C.E and Symons, R.H. 1979. Characterization of a viroid associated with avocado sunblotch disease. Virology 99: 145-151.

Pegg, K.G. 1991. Causes of disease. In: "Avocado Pests and Disorders" (ed. Broadley, R.H.),Queensland Dept. of Primary Industries, Brisbane, Australia, pp.1-7.

Pohronezny, K.L. and Simone, G.W. 1998. Scab. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D.), APS Press, St Paul, MN., USA, p. 81.

Pohronezny, K.L, Simone, G.W. and Kotzé, J.M. 1998. Pseudocercospora Spot (Blotch). In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, Minnesota, USA, pp.79-80.

Prusky, D. 1998. Anthracnose. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C.

Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St Paul, MN., USA, pp. 72-73.

Raabe, R.D. and Zentmyer, G.A. 1955. Susceptibility of avocados to *Dematophora* root rot. Plant Dis. Rep. 39:509-510.

Ruehle, G.D. 1958. The Florida Avocado Industry. Fla. Agric. Exp. Stn. Bull. 602.

Sippel, A.D. 2001. Origin and history of the avocado. In: "The Cultivation of the Avocado" (ed. de Villiers, E.A.) Institute for Tropical and Subtropical Crops, Nelspruit, South Africa, pp.3- 11.

Sztejnberg, A. 1998. *Rosellinia* (Dematophora) Root Rot. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, pp. 80-81.

Sztejnberg, A., Omary, N. and Pinkas, Y. 1983. Dematophora root rot on avocado trees in Israel and the development of a diagnostic method. Phytoparasitica, 11:238-239.

Stevens, H.E. and Piper, R.B. 1941. Avocado diseases in Florida. U.S. Dept. Agric. Circ. 582.

Zentmyer, G.A. 1949. Verticillium wilt of avocado. Phytopathology 39:677-682.

Zentmyer, G.A. 1951. Studies on blackstreak disease of avocado. *In:* Rep. Uni. Calif. Citrus Exp. Stn, Riverside. Proj. 944, pp.191-192.

Zentmyer, G.A. 1980. *Phytophthora cinnamomi* and the Diseases It Causes. Monogr. 10. APS Press, St. Paul, Minnesota, USA.

Zentmyer, G.A. 1984. Avocado diseases. Trop. Pest Manage. 30:388-400.

Zentmyer, G.A. 1998a. Avocado. In: Compendium of Tropical Fruit Diseases (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, pp.71-72.

Zentmyer, G.A. 1998b. Verticillium Wilt. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, pp. 83-84.

Zentmyer, G.A., Paulus, A.O., Gustafson, C.D., Wallace, J.M.. And Burns, R.M. 1965. Avocado diseases. Univ. Calif., Div. Agric. Sci., Calif. Agric. Exp. Stn. Ext. Serv. Circ. 534.

Zentmyer G. A. and Ohr, H.D. 1978. Avocado root rot. Div. Agric. Ext. Serv., Univ. of Calif., Leaflet # 2440.

Zentmyer, G.A., Menge, J.A. and Ohr, H.D. 1998a. Phytophthora Root Rot. In: "Compendium of Tropical Fruit Diseases" (eds. Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, pp.77-79.

Zentmyer, G.A., Ohr, H.D. and Menge, J.A. 1998b. Phytophthora Cankers. In: "Compendium of Tropical Fruit Diseases" (eds. . Ploetz, R.C. Zentmyer, G.A., Nishijima, W.T., Rohrbach, K.G. and Ohr, H.D), APS Press, St. Paul, MN., USA, pp.76-77.

Diseases of Banana and their Management

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Abstract: Banana is the most important fruit crop commercially grown in a number of countries worldwide for its utilization as dessert and as staple food in certain parts of world. Among the fruit crops, it is one of the most important crops in international trade for earning the foreign exchange in many African countries. The diseases caused by fungi, bacteria and viruses are the major limiting factor in successful quality production of this crop and almost all the commercial cultivars of banana are highly susceptible to certain deadly diseases. Rapid development of tolerance in pathogens to new pesticides further aggravated the problem of disease management in banana. Recently the Panama wilt pathogen has evolved new virulent races and rampant spread of black Sigatoka pathogen have left limited options to growers to grow quality exportable produce. An integrated approach appears to be feasible in management of banana disease that includes use of improved / resistant cultivars and disease free planting stocks, judicious use of pesticides, irrigation water, removal of diseased plants and plant parts, proper sanitation in plantation and a close monitoring of the diseases in relation to weather parameters. In this chapter, the diseases of international economic importance are discussed with their latest management strategies

1. Introduction

Banana is a large monocotyledonous herb and believed to be originated in Southeast Asia. Among the fruit crops, it is supposed to be the first crop domesticated by man some ten thousand years ago (Simmonds and Shepherd, 1955) and one of the most popular fruit consumed as dessert and also as staple food in certain parts of world. For example, in Uganda alone the per capita consumption of the AAA highland bananas is around 200 kg per year. After rice, wheat and milk, it is the fourth most valuable food. It is the major dietary source of carbohydrates, vitamins A, B6, C, minerals like potassium, calcium, phosphorus and fiber. Banana has around 300 or more cultivars and almost all of them are developed from two seeded, diploid species, *Musa acuminata* Colla and *M. balbisiana* Colla. and are diploid, triploid and tetraploid hybrids among subspecies of *M. acuminata*, and between *M. acuminata* and M. *balbisiana.* The haploid from these species are designated with A and B. For example, the Cavendish cultivar, the important in export trades is triploid of acuminata and thus designated as AAA (Ploetz, 1999). The total world production of Banana is about 69.51 million tones and India is the top producer of banana with 16 million tones production during 2001 and the productivity with 32.65 tones \ ha from 0.49 million ha area (FAOSTAT, 2001).

Banana production however is variously hampered due to various diseases infesting all stages of growth from seedling to fruit. To overcome this important aspect of sustainable banana production, breeding programmes and biotechnological approaches are being targeted to improve this crop against the losses caused by the diseases. Banana is attacked by a number of fungal, bacterial and virus pathogens and some of these diseases are economically important worldwide.

Africa produces nearly 30 million tons of bananas yearly, which is mostly consumed locally. But production is already being reduced mainly due to diseases. Because existing banana plants are reproduced from cuttings, there is little genetic diversity. Diseases, in particular fungus, rapidly can wipe out entire production regions. In the 1950s, Panama disease wiped out the then dominant banana variety, Gros Michel. Now, a new race of the Panama disease is back, increasingly destroying Cavendish banana plantations all over the world. Even worse is the rapidly spreading 'Black Sigatoka', which is attacking the banana plant and has already cut crop yields by 40 percent in Uganda, the world's second largest banana producer, only next to India. Certain important diseases of banana are discussed in this chapter with their effective management strategies.

2. Panama wilt

In the 1950s, Panama disease wiped out the then dominant banana variety, Gros Michel in Africa and elsewhere in the world. Now, a new race of the Panama disease is back, increasingly destroying Cavendish banana plantations all over the world. *Fusarium* wilt of bananas (Panama disease) is becoming a major problem in banana production in Malaysia, attributable to the increasing area planted with susceptible clones and the new occurrence of a highly virulent race of the pathogen, *F. oxysporum* f.sp. *cubense*, in Peninsular Malaysia (Liew, 1997).

The record by Shivas *et al.* (1995) is probably the first of *Fusarium* wilt on Cavendish bananas in Western Australia. *F. oxysporum* f.sp. *cubense* was detected in 10 out of 153 plantations surveyed at Carnarvon. It was thought to be introduced with, and spread from, diploid bananas of uncertain origin in wind-breaks, which were known to be affected by *Fusarium* wilt in Carnarvon as early as 1949. The disease is common in many parts of the world.

2.1 Symptoms

The symptoms are most apparent on at least 5 months old plants although 2-3 months old plant are also killed under highly favourable conditions for disease development. The earliest signs of the disease are faint yellow streaks in the petioles of oldest lowermost leaf. Two types of symptoms follow this stage. In the yellowing type there is progressive yellowing of the old leaves and eventual collapse at the petiole. In the non-yellowing type, the leaf collapses at the petiole without leaf chlorosis. Often all the leaves but the youngest collapses and the heart alone remain upright. Any new leaves that are produced are usually blotchy and yellow, often with wrinkling of the lamina.

The pseudostem often shows more or less conspicuous longitudinal splitting of

the outer leaf sheaths from its outer covering. But sometimes this symptom is not present. About 4-6 weeks after appearance of streaks on the petiole only the dead trunk of the pseudostem remains.

Discolored vascular strands varying from light yellow to dark brown are the distinguishing internal symptoms. Usually the discoloration appears first in the outer or oldest leaf sheath and extends up to the pseudostem. It is pronounced in the rhizome but is not common in roots. However, roots of diseased rhizomes are frequently blackened and decayed. Longitudinal sections through diseased root base show characteristic red strands passing into the rhizome stele.

2.2 Causal organism

Panama disease of banana is caused by *Fusarium oxysporum* f. sp. *cubense*. Several clones differing in pathogenicity exist in nature and may be grouped into races.

2.3 Disease cycle and environmental relations

The pathogen is a soil-borne fungus surviving in soil mainly as chlamydospores formed by the hyphal and conidial cells. Infection is always through injured roots. There is no evidence of the fungus having an ability to attack living cells of the main root of banana. Deep wounds to expose the xylem help in easy infection. Spore germination is stimulated in the vicinity of wounded roots surface while it is inhibited by intact root surface. Nematodes help in exposing to roots to infection. After entry into the roots, the fungus proceeds internally along the root to the rhizome where it develops extensively in vascular tissues before passing up the vascular system into the pseudostem and the older leaf petioles.

Recent studies to determine whether long-distance dispersal of *Mycosphaerella musicola* and *M. fijiensis* (causal organisms of yellow and black Sigatoka diseases, respectively) in banana and plantain plays a significant role in the epidemiology of these diseases are inconclusive (Burt, 1994).

2.4 Management of disease

Current research is directed to the determination of pathogenic and genetic variation in the pathogen, *Fusarium oxysporum* f.sp. *cubense* (Moore *et al.,* 1995). Corm injection of 2 per cent carbendazim plus 0.1 per cent Agallol or Aretan as well as soil drench has been claimed as a successful method (Lakshmanan and Mohan, 1989).

In general, sanitation by immediate removal of diseased plants with surrounding soil from the field, use of healthy planting stock, care during cultivation to avoid root injury, control of nematodes, and cultivation of resistant varieties in the affected areas appear to be the only practical methods. Cavendish bananas are resistant and Gros Michel bananas are susceptible. Ramkrishnan and Damodaran (1956) had reported that varieties Poovan, Moongil, Peyladen, Rajabale and Vamankeli were resistant to wilt. Carbendazim (1 g/litre) application by dipping prior to planting followed by drenching at bimonthly intervals starting from 5 months after planting is useful (Chadha, 2001).

Among the organic amendments tested, neem cake (*Azadirachta indica*) at 250 kg/ha was the most effective treatment for the control of *Fusarium oxysporum* f.sp. *cubense* on banana cv. Rasthali in field experiments in Tamil Nadu, India. It also produced the highest crop yields (Karthikeyan and Karunanithi, 1996).

Losses can be kept to an economic level by following a disease management schedule which includes the use of healthy planting material, pre-planting dipping in carbendazim (0.2%) for 45 min, soil amendment with lime/neem cake at 1 kg/pit before planting or application of urea (200 g/plant) + sugarcane trash mulch at 5 and 7 months, and eradication of infected plants, including rhizomes, as soon as they become infected, together with injections or drenches of carbendazim at 5, 7 and 9 months (Narendrappa and Gowda, 1995).

3. Black Sigatoka leaf spot

The black Sigatoka disease has become a potential threat for successful cultivation of banana worldwide because it can reduce the leaf area significantly and thus causes yield losses upto 50 % or more. The infection also causes premature ripening and defects in exportable fruit. The disease was first recognized in the Sigatoka Valley of Fiji in 1963, though it was already widespread in Southeast Asia and the South Pacific region by that time. In Western Hemisphere, it was first reported in Honduras in 1972 and in Africa in 1973 in Zambia. In most of the areas, this disease has replaced yellow Sigatoka comparatively less damaging disease (Ploetz, 1999).

3.1 Symptoms

The evident feature of the disease is the presence of abundant small discrete spots on the laminas of older leaves, with areas of 'Scorched' or brown leaf tissues. The two youngest leaves, or heartleaves of an actively growing plant are usually free from evident spotting. The first indication of infection is typically evident at close quarters to the naked eye on the 3rd or 4th leaf from the centre and sometimes on second. Black Sigatoka turns the plant's leaves a mottled yellow, brown and black, thus impeding photosynthesis, depleted of its energy reserves, the plant cuts back its fruit production, often to half (Fig. 1). For exporters, Black Sigatoka is ruinous. the disease also causes premature ripening of the fruit. Although they appear normal, bananas from affected plants spoil before arriving at markets.

3.2 Causal organism

The disease is caused by the ascomycete, *Mycosphaerella fijiensis* Morelet (anamorph: *Cercospora musae* Zimm, *Mycosphaerella musicola* Leach, *Paracercospora fijiensis* (morelet) Deighton).

3.3 Etiology

The pathogen produces elongated and multiseptate conidia in the sporodochia. One

septate, hyaline and obtuse to ellipsoid ascospores are borne in perithecia which are dark brown to black, amphigenous, erumpent and ostiolate. Sporodochia production is abundant as compared to perithecia. Both the propagules are infective, however, ascospores are produce in abundance and of smaller size and thus are more important in spreading the disease than conidia within plants and plantations. They are formed under high moisture conditions, and are disseminated with rain and irrigation water. Infected leaves often used as packing material of fruits and vegetables for distant transport are also source of long distance spread of the disease in many developing
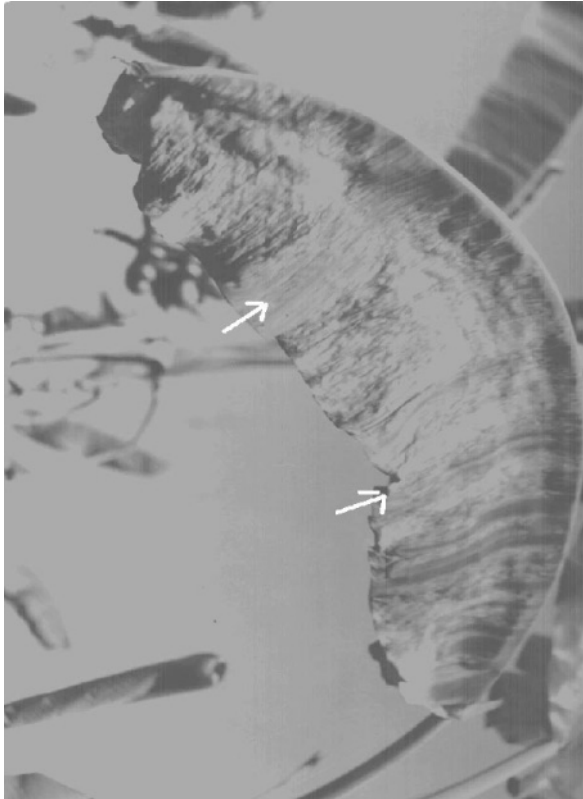
Figure 1: Black Sigatoka disease exhibiting mottled yellow, brownish black spots on old leaf

countries. Import of infected plant material with any quarantine check may also become the source of disease in virgin areas. Black Sigatoka disease is more damaging and has wider host range than yellow Sigatoka disease.

3.4 Management of the disease

All the banana cultivars having demand in export market are susceptible to the disease

and resistance to black Sigatoka among the pre-existing banana genotypes is poor. Hence the regular use of fungicides is mainstay for management of the disease. A number of fungicides and their combinations have been recommended for the control of the disease right from protective Bordeaux mixture to sterol demethylation inhibitors and strobilurins. Unfortunately, the pathogen has a tendency to develop resistance or tolerance towards the systemic fungicides, their applications is recommended with combination or alternation of protectant fungicides. However, increased tolerance of the pathogen to fungicides has resulted in increasing the applications frequencies from 25 – 40 or more in many parts of world. The annual cost of fungicide applications is estimated to be around $ 1000 per hectare for exportable produce. This high cost of production however is compensated in international trade but it is difficult for a marginal and small farmer. Thus besides the application of fungicides, other strategies like removal of older infected leaves in order to reduce inoculum level, interplanting with other nonsusceptible crops and planting in partial shade may reduce the disease severity (Oluma and Onyezili, 1995).

Improved drainage, proper weed control, removal of suckers and proper spacing for the variety grown and reducing the humidity in the plantation also help in checking the disease spread. Huq *et al.* (1994) reported that six fungicides namely Bordeaux mixture (1.0%), Bavistin 50 w.p. (Carbendazim) 0.1%, Dithane M-45 (0.2%), Mancozeb (0.25%), Calixin, Tridemorph (0.1%), and Tilt (Propiconazole) 250 EC (0.05%) were applied 6 times, initial spray when symptoms are noticed, the next spray at 20 day interval to control of black Sigatoka. Among these fungicides, Tilt (0.05%) gave best result. Saxena *et al.* (1994) reported management of the disease with the spray of Dithane M-45 (0.2%) and Bavistin (Carbendazim 0.1%) when applied alternatively every 15 day and Dipping planting suckers in Carbendazim.

The best and reliable approach in management of black Sigatoka disease would be the use of genetic resistance and improvement of varieties those have export market demand. The resistant cultivars are usually less productive or desirable than those of susceptible cultivars. Thus breeding programmes were initiated in many countries to overcome this problem and to curtail the use of pesticides in order to environmental safety and consumer concern. Among such programmes, FHIA (Fundacion Hondureana de Investigacion Agricola) in La Lima, Honduras has been successful in bring out some resistant clones (Ploetz, 1999).There is some hope from new FHIA varieties those are resistant to major diseases and pests. The FHIA banana also is highly productive and has good shipping qualities. Trial plantations in the Caribbean and Africa have proven successful, especially when it comes to cut the costs of fungicides and pesticides.

Testing in Africa showed positive results. In Tanzania and Nigeria, for example, FIAH and other varieties are now grown by more than half a million people and yields have increased from 5-20 kg a bunch to 40-110 kg a bunch. FHIA-01 would immediately double production for the 70 million Africans for whom plantains are a staple food according to FIAH project workers (http://www.fiah.hn/).

4. Leaf spot

The disease is caused by *Cordana musae* (Zimm.) Von Hohn.

4.1 Symptoms

The leaf spots caused by *Cordana musae* are usually oval in shape and pale yellow to brown in color. Concentric zones can be seen in the centre of the necrotic are of the spot surrounded with distinct halo near leaf margins. A large number of conidiophores/ conidia are developed on the underside of the leaf in the spot. In severe cases, these spots coalesce to form large strips of infected area.

4.2 Control

Usually the fungicides used for control of Sigatoka disease also control the disease. Oil and fungicidal sprays emulsion gives good control of the disease.

5. Anthracnose

The disease is caused by *Colletotrichum musae* (Berk. & Curt.) v. Arx, is wide spread in almost all the banana growing belts of world and in certain pockets of India (Thakur and Misra, 1966, Rawal and Ullasa, 1989).

5.1 Symptoms

It attacks the plants at all the stages of development. There are usually two types of infection. Latent infection usually originates in the field on uninjured green fruit where the fungus penetrates the cuticle and becomes latent as a sub-cuticular hypha. In contrast, non-latent infestation usually begins during or after harvest. Young latent infection appears as small black circular streaks on the flowers, skin and distal ends of banana heads.

The optimum temperature for disease development on ripe banana was $30-35^0$C. The disease was found to be severe from June to September when temperature remained high accompanied by showers for a number of days. The disease seems to be restricted during the cold as well as hot and dry months. On ripening, the fruit develop typical numerous small dark circular spots which enlarge, coalesce and become sunken (Snowdon, 1990). On maturity of lesions, salmon pink spore masses are produce.

5.2 Epidemiology

The fungus remains in the asexual stage, acervuli giving rise to conidial mass on leaf debris in the plantation. These spores (conidia) are liberated by rainsplash or irrigation water and dispersed by air currents to developing fruit. On moist fruit surface, the spore germinates by producing appressorium which remain attached to fruit surface in its latent / quiescent form till fruit ripening (Simmonds, 1963, Swinburne and Brown, 1983). On fruit ripening, further invasion and infection takes place. The green banana show some degree of resistance for disease development (Brown and Swinburne, 1980).

5.3 Control measures

Sanitation of plantation and packinghouse to minimize the inoculum load is necessary in order to check the disease. Correct stage of harvest and minimizing the injuries to fruit during harvest is also necessary. Post harvest treatment with systemic fungicides is more effective than pre-harvest sprays (Griffee and Burden, 1974, Ram and Vir, 1983). However, field spraying of Prochloraz (0.15%) or Chlorothalonil (0.2%) at 15 days interval was found to be quite effective. Spray of carbendazim/ Bitertanol/ Thiophenate methyl/ Calixin/ / Rovral (2 g/litre) in humid weather is also effective (Rawal and Ullasa, 1989). Chuang and Yang (1993) suggested biological control of banana anthracnose.

6. Cigar end rot

The disease is caused by *Verticillium theobromae* (Turc.) Masonet Hughes) and was first reported by Dhingra *et al.,* (1970) in India. *Trachysphaera fuctigena*, Tabor & Bunting, can also cause destructive rot in West and Central Africa (Maramba and Clerk, 1974). The disease is also reported from West Indies, Iran, Egypt and South Africa (Meredith, 1965, El-Helaly *et al.*, 1954, Ershad, 1972, Wardlaw, 1931).

6.1 Symptoms

The damage is caused on the young bunches in the form of necrosis at the pistillate end. The skin become folded and shrunken as the infection spreads slowly along the fingers, grey conidia are formed on the shriveled stalk end of the fruit *i.e.* like the ash of cigar. The pulp tissue shows typical dry rot that is the characteristic feature of the pathogen and is reduced to a dry fibrous condition. Black pitting and spotting of fruit on account of drying of the finger gives an appearance of a lighted cigar. In Trachysphaera rot the infected surface is covered with white spores which later turn pink or brown. The pulp may undergo a wet rot.

6.2 Control

Removal pistils and perianth helps in reduction of the disease. Bagging of developing fruit is also recommended (Snowdon, 1990). Spray of recommended fungicides as soon as fruits are formed significantly control the disease. Application of Benlate @ 0.5% concentration or thiophenate methyl/ Bitertanol (1 g/ litre) or Chlorothalonil is recommended.

7. Finger tip or black tip rot

The diseases is caused by *Botryodiplodia theobromae* Pat., and well known in the tropics as a wound pathogen on the banana in storage. The disease occurs as a storage problem and it has been recorded in severe forms in many parts of India (Srivastava and Tandon, 1971), Central America, Philippines, Taiwan etc.(Snowdon, 1990).

7.1 Symptoms

Surface growth of greyish black mycelial mass is the characteristic of the disease. The infection usually starts from persistent perianth (Roy and Sharma, 1952) or stem end causing progressive brownish black discoloration. The pathogen invades fruit through wound or bruised tissues and spreads rapidly in pulp turning it in black watery mass. The infected skin becomes black, soft and encrusted with pycnidial growth.

It is sometimes associated with fruit-spots and blemishes and causes an extensive tip rot in most varieties. Since the fungus grows very rapidly at tropical temperatures, it may cause considerable rotting in the course of a few days. Skin turns dark and wrinkled. Rotting of the pulp is comparatively faster than that of the skin. In maturing or mature fruits, two-thirds or even whole of the finger may be affected. In the humid weather white or light grey cottony mycelium may be seen on the affected area, and then black pycnidia becomes visible. Williamson and Tandon (1966) reported maximum damage at 25-30^{0}C. The bacteria associated with the rot were *Pediococcus sp., Propionibacterium aeruginosa*.

7.2 Control measures

Bordeaux mixture (1%) or Dithane M-45 (0.3%) may be sprayed as a preventive measures. Removal of the affected portion of the hand reduces the disease. Storage of the bunches at low temperature (10^{0}C) minimizes the incidence (Bhargava *et al.,* 1965). Dipping of fruits in benomyl or TBZ also control the rot (Sohi, 1975). Wax emulsion with benlate or Brestan delayed fruit ripening and prevented moisture loss thereby controlling the onset of rot. Ogundero (1987) reported the production of extracellular enzymes completely inhibited by Benomyl (200 ppm) treatment. Pre and post-harvest application of Carbendazim/ Bitertanol/ Thiophenate methyl/ Prochloraz (1 g/litre) and Rovral (2 g/litre) is recommended.

8. Moko disease

The diseases is also known as bacterial wilt of banana caused by *Pseudomonas solanacearum* E.F.Smith. It is widely distributed in most of banana growing regions. The disease has also been reported from Trinidad on plantain. There are several strains of the bacterium but the strains pathogenic to the commercial varieties are confined to the Western Hemisphere and the Philippines.

8.1 Symptoms

The symptoms of this disease are similar in some respects to those of Panama wilt, however, it can be differentiated based on its typical symptoms from Panama wilt. The affected plants show rapid wilting and collapse of the leaves. In the initial stages bacterial wilt is characterized by the development of a yellowish coloration of the inner leaf lamina and wilting of inner leaves. In some cases the heartleaf also collapses. In case of planting the diseased suckers, the terminal leaf becomes necrotic and plant dies.

Moko is typical vascular infection and discolored the vascular strands. The reddish tinge present in typical cases of Panama disease is usually absent although in some advance cases a reddish brown colour of vascular tissue may be visible. However the discoloration of vascular tissue in Moko is mostly confined to centre as compared to peripheral discoloration in Panama wilt. The production of bacterial ooze is the distinguishing feature of Moko disease.

8.2 Epidemiology

Large number of weeds are the collateral hosts to the pathogen. The four major strains of Moko are D, B, SFR and H. SFR and H are easily transmitted by insects visiting the flowers. Strain B has only moderate transmission through flower bracts and persists in soil.

8.3 Control measures

Phytosanitary measures and effective quarantine are essential to contain spread of the Moko disease to virgin areas. Good drainage, use of sterilized operating tools, crop rotation and effective weed management help in control of the disease.

9. Rhizome rot

The disease is caused by *Erwinia carotovora,* Jones, (Edward *et al.*, 1979) and widespread in banana growing areas of the world.

9.1 Symptoms

Discoloration and soft rot of rhizomes and suckers are typical symptoms of the disease. They have scanty roots with dark brown lesions and necrotic tip. In other cases the pseudostem tips over break across the rotted stems at the ground level. Affected plants become very weak and may be uprooted by wind. Dark brown necrosis develops on older leaves in case of young plants infection affecting the normal growth of the plant. Decayed rhizome develops dark brown water soaked cavities like root borer tunnels.

9.2 Control measures

Good drainage, adequate phytosanitary measure, use of disease free planting stocks, good soil aeration and judicious irrigation are some cultural operations help in containing the disease.

10. Root and rhizome rot

The disease is caused by burrowing nematode *Radopholus similis* (Cobb.)

10.1 Symptoms

Banana plants infected with *R. similis* first develop yellowing in the outer whorl of leaves. In 7 to 10 days yellowing extends to inner whorl also. The leaves and bunches wither and drop leading to death of the plant. Infected roots show reddish-brown cortical lesions. These lesions first appear as reddish, elongated flecks parallel to root axis, then enlarge as nematodes and their offspring's feed on the tissues. Distal portion of the root system is destroyed and root mass is reduced (Blake, 1972). This results in poor anchorage of the plants, which may topple down in high winds. In the parenchyma cavities are formed by destruction of cells. The attacked roots rot and have no laterals. Rotting tissues are also found on rhizomes. In advanced stages rotting extends to pseudostem also. Nematode damage first appears in scattered patches in the first crop, which later coalesce to form bigger patches. In about five years the entire field becomes severely infested.

10.2 Control measures

Where clean rhizomes are not available, the discoloured portions on the rhizome should be removed with a sterilized knife. The cut should be deep enough to remove not only the discoloured tissues but some of the adjoining healthy looking tissues also. The rhizomes can then be dipped in Bordeaux mixture and DBCP paste applied on the cuts. Alternatively, the rhizome can be given hot water treatment at 53^0C to 55^0C for 20 to 25 minutes. In spite of these precautions some nematodes may be left in the rhizomes and lethal populations may again built up within two to three years after planting (Thorne, 1961, Singh and Sitaramaiah, 1994). Since the nematodes can be present in deeper layers of soil, soil fumigation is not very effective, apart from being very costly. Application of D-D, EDB or DBCP at the rate of 300 l/ha at planting time results in increased yield for one year and the treatment is repeated the following year. In coconut nurseries, fensulfothion 50 kg a.i./ha phenamiphos 25 kg a.i./ha or phorate 25 kg a.i./ha applied thrice a year (September, December and March) has been found to reduce infection. Application of phenamiphos or phorate at the rate of 10 g a.i./tree to standing plants is reported to increase yields by 30 per cent. Crop rotation with sugar cane or rice found suitable. Application of neem cake (400 g/plant) once at planting and second 4 months later is also effective. Banana Elak Kibale is resistant to the nematode. Intercropping with sun help, coriander or marigold is also helpful. After remove discoloured portion of banana suckers (Paring) and dip in mud slurry with 40 g of Carbofuran on mud slurry is effective. The application of 40 g Carbofuran/plant 4 month after planting should be repeated.

11. Bunchy top

The disease was first recorded from Fiji in 1879 followed by Sri Lanka in 1913. The disease is believed to have been introduced in India (1940) through infected suckers brought from Sri Lanka into the state of Kerala. It has now spread to almost all the banana growing areas in India.

11.1 Symptoms

The primary symptom of bunchy top of banana are seen if infected suckers are planted. They put forth short, narrow leaves which are chlorotic and exhibit mosaic symptoms. The leaves arise in clusters, giving a rosette appearance (Fig. 2). They are brittle with numerous dark green dots or patches with the margins rolled upward. The plants do not usually grow taller than two to three feet and they fail to put forth any fruit.

The secondary symptoms may occur when the healthy plants in the field when freshly infected by the virus at any stage of growth. The first external symptom in secondary infection is the premature unfurling of leaves and the development of dark green spots and streaks on the blade. These symptoms are common along the secondary veins and on the midrib and petiole. In the leaves formed subsequently the symptoms are more intensified, the leaves become pale and much reduced in size and when a few more leaves develop the characteristic rosette or bunchy top symptom is evident. The affected leaves are usually more rigid than the normal ones, but they generally do not wilt. The diseased plants neither die nor recover from the infection. In the affected plants bunches may or may not develop, but sometimes bunches may develop at least partially, prematurely by bursting through the sides of the pseudostem. At times the cortical region of the corm and the roots may show decay.

When the diseased plants are cut open and examined certain characteristic abnormalities may be seen. The fibrous sheath near the phloem region of the vascular bundles is replaced by unthickened cells, filled with chromatophores. New cells may be produced in the fundamental tissues near the phloem and these may contain chloroplasts. The phloem cells contain many nuclei. The abundance of chloroplasts in the vascular tissues of leaves, midrib and petiole is responsible for the abnormally dark green patches seen in the diseased plants. The suppression of the fibrous sheath perhaps accounts for the brittleness of the leaf.

11.2 Causal organism

The disease is caused by a virus, transmissible only through an insect vector. At least two distinct strains of the virus have been established in Australia.

11.3 Disease Cycle

Being systemic in nature, the virus is present in all plant parts, including the rhizomes and the suckers. However, if the main plant is infected in advanced age, the suckers may escape the infection. The virus is transmitted by the banana aphid, *Pentalonia nigronervosa*, but not by any other insects tested. The disease symptoms appear in 35 to 45 days after inoculation by the insect. The aphid is required to feed on the infected plant for a minimum of about 17 hours before it can transmit the disease and the inoculation period inside the vector may be from about 90 minutes to 48 hours, and they retain their infective capacity for about 13 days. The vector usually attacks the host around the basal portion of the pseudostem, but at times they may be found on the upper leaf sheaths and petioles. The insect may carry the pathogen to long distances.

The same vector transmits the cardamom mosaic virus also. The bunchy top virus has also been transmitted to *Heliconia sp.*, through the insect colonies, but not to *Ravenala spp., Canna spp., and Hedychium spp.* and other plants.

11.4 Control measures

The disease is found most commonly on all the popular varieties of bananas grown in

Figure 2: Bunchy top of Banana showing chlorotic leaves in clusters, giving a rosette appearance

the region. There is no genetic resistance, except as manifested by differential intensities of the disease on different varieties. All species of banana, including edible, seedless and seeded ones are susceptible. The only method for controlling the disease is by exclusion. Thus, strict quarantine regulations in disease-free areas may be the best means to prevent its entry. Where the disease is already present, measures to check the insect vector and to plant new areas with healthy suckers will provide some control.

Systematic eradication of the diseased plants, suckers and the clumps is very essential. Injecting the herbicide Agroxone (2,4-Dichloro-phenoxyacetic acid) or MCPA (2-methyl 1-4-chloro-phenoxyacetic acid) into the diseased plant has been found effective in eradicating it in Australia. To control insect vector, fortnightly sprays of Endrin (0.02%) or Diaxinon (0.03%) are recommended. Also Thiodemeton (Solvirex), Disyston, Tindane, Dimethoate and Phorate granules @ 50 g and 25 g at plant base and leaf axil application respectively are highly effective in controlling the vector. Injection of systemic chemicals like Methyl demeton @ 0.05-0.2 ml/plant at 3-4 weeks interval also helps in reducing the vector population considerably (Chadha, 2001).

Integrated control measures recommended against this virus disease include early detection, roguing of diseased plants after spraying to eliminate vector aphids, replanting with virus-free material and careful cultivation (Zhou *et al.,* 1996).

Biotechnology is also expected to have applications in producing transgenic virus-resistant plants, using coat protein gene-mediated resistance. (Dale *et al.*, 1993).

12. References

Bhargava, S.N., Ghosh, A.K., Srivastava, M.P., Singh, R.H. and Tandon, R.N. 1965. Studies on fungal diseases of some tropical fruits VII. Effect, of temperature on the decay of mango, banana and guava caused by some important pathogens. Proc. Nat. Acad. Sci. India. 35 : 393-398.

Blake, C.D. 1972. Economic Nematology, (ed. Webster, J.M). Academic Press, New York. pp. 245-266.

Brown, A.E. and Swinburne, T.R. 1980. The resistance of immature banana fruits to anthracnose (*Colletotrichum musae* (Berk.& Curt.) Arx. Phytopathologische Zeitschrift, 99: 70-80.

Burt, P. J. A. 1994. Windborne dispersal of Sigatoka leaf spot pathogens. Grana. 33: 108-111.

Chadha, K.L. 2001. Hand Book of Horticulture, ICAR Publication, pp. 729-730.

Chuang, T. Y. and Yang, H. R. 1993. Biological control of banana anthracnose. Plant Pathology Bulletin. 2: 71-77.

Dale, J., Burns, T., Oehlschlager, S., Karan, M. and Harding, R. 1993. Banana bunchy top virus: prospects for control through biotechnology. In: "Proceedings of the workshop on biotechnology applications for banana and plantain improvement", held in San Jose, Costa Rica, 27-31 January 1992. pp.85-92.

Dhingra, O.D., Khare, M.N. and Chand, J.N. 1970. Cigar end rot disease of banana in India. Current Science, 39: 498-499.

El-Helaly, A.F., Ibrahim, I.A. and El-Arosi, H.M. 1954. Studies on some factors affecting the prevalence and distribution of cigar end disease of banana in Egypt. Alexandria Journal of Agricultural Research, 11: 9-28.

Ershad, D. 1972. The occurrence of cigar end disease of banana in Iran. Iranian Journal of Plant Pathology,8: 7-16.

FAOSTAT, 2001. Database results of Food and Agricultural Organization of United Nations, http:// fao.org.

Griffee, P.J. and Burden, O.J. 1974. Incidence and control of *Colletotrichum musae* on banana in the Windward Islands. Annals of Applied Biology, 77: 11-16.

Huq, M. I., Ahmed, H. U. and Malaker, P. K. 1994. Control of Sigatoka of banana with foliar fungicides. Bangladesh Journal of Scientific and Industrial Research. 29: 81-85.

Karthikeyan, A. and Karunanithi, K. 1996. Influence of organic amendments on the intensity of *Fusarium* wilt of banana. Plant Disease Research, 11:180-181.

Lakshmanan, P. and Mohan, S. 1989. A novel method to control Panama disease of banana incited by *Fusarium oxysporum* f. sp. *cubense*. Indian J. Mycol. Plant Pathol. 19 : 93-97.

Liew, K. W. 1997. *Fusarium* wilt disease in commercial banana production. Planter, 73: 303-308.

Maramba, P. and Clerk, G.C. 1974. Survival of conidia of Trachysphaera fructigena. Transactions of the British Mycological Society, 63: 391-393.

Moore, N. Y., Bentley, S., Pegg, K. G. and Jones, D. R. 1995. *Fusarium* wilt of banana. Musa Disease Fact Sheet. 5 : 4.

Narendrappa, T. and Gowda, B. J. 1995. Integrated management of Panama wilt of banana cv. Nanjangud Rasabale. Current Research - University of Agricultural Sciences (Bangalore). 24: 181-183.

Ogundero, V.W. 1987. Crown rot fungi of Nigerian bananas cv. Robusta and the effect of benomyl on their exogenzymes. J. of Basic Microbios Letters. 44 : 147-155.

Oluma, H. O. A. and Onyezili, F. N. 1995. Effect of cultural practices on the severity of black Sigatoka leaf spot disease of plantain and banana in the Nigerian savanna. MusAfrica. 8 : 10-11.

Ploetz, R. 1999. The most important disease of a most important fruit. APSnet Feature, April 1999. 8 p.

Ram, V. and Vir, D. 1983. Evaluation of benzimidazole and other fungicides against post-harvest spoilage of banana fruits caused by Colletotrichum musae (Berkeley & Curtis) Von Arx. Pesticides, 17: 28-29.

Ramkrishnan, T.S. and Damodaran, S 1956. Observations on the wilt disease of banana. Proc. Indian Acad. Sci. 43 B : 213-222.

Rawal, R.D. and Ullasa, B.A. 1989. Management of anthracnose of banana through fungicidal sprays. Plant Disease Research, 4: 24-26.

Roy, R.S. and Sharma, C. 1952. Diseases and pests of banana and their control. Ind. J. Hort. 9 : 39-52.

Saxena, D. R., Moly Saxena and Bhalla, P. L. 1994. Controlling Sigatoka disease of banana. Indian Horticulture. 39(1) : 20-21.

Shivas, R. G., Wood, P. M., Darcey, M. W. and Pegg, K. G. 1995. First record of *Fusarium oxysporum* f.sp. cubense on Cavendish bananas in Western Australia. Australian Plant Pathology. 24: 38-43, 26.

Simmonds, J.H. 1963. Studies in the latent phase of Colletotrichum species causing ripe rots of tropical fruits. Queensland Journal of Agricultural Science 20:373-424.

Simmonds, N.W. and Shepherd, K. 1955. Taxonomy and origin of cultivated bananas. Journal of the Lennean Society of Botany (London) 55: 302-312.

Singh, R.S. and Sitaramaiah, K. 1994. Plant Pathogens – The Nematodes. Oxford and IBH Publishing Co. Pvt. Ltd., pp. 212-213.

Snowdon, A.L. 1990. A color Altas of Post-harvest diseases and disorders of Fruits and Vegetables Vol.I: General Introduction and fruits, CRC, Press, Inc. Boca Raton, Florida, pp. 104-119.

Sohi, H.S. 1975. Anthracnose in Tropical Fruits in: "Advances in Mycology and Plant Pathology". (ed. Raychaudhuri, S.P.).

Srivastava, M.P. and Tandon, R.N. 1971. Post-harvest diseases of banana in India. Ind. Phytopath. 24 : 115-118.

Swinburne, T.R. and Brown, A.E. 1983. Appressoria development and quiescent infections of banana fruit by Colletotrichum musae. Transactions of the British Mycological Society, 80: 176-178.

Thakur, D.P. and Mishra, A.P. 1972. Varietal resistance in banana against anthracnose fungus (*Gloeosporium musorum*). Indian J. Horticulture, 23: 59-61.

Thorne, H. 1961. Principles of Nematology. McGraw-Hill.

Wardlaw, C.W. 1931. Banana diseases. 2. Notes on cigar-end (*Stachylidium theobromae* Turc.)
    Tropical Agriculture, 8: 293-298.

Williamson, D. and Tandon, R.N. 1966. Some pathological studies on *Botryodiplodia theobromae*
    Pat. causing banana rot. Mycopath. Mycol. Appl. 29 : 245-253.

Zhou, Z.J., Lin, Q.Y., Xie L.H., Chen Q.J., Wu Z.J., Huang G.S., Jiang J.F. and Zheng G.Z.
    1996. Studies on banana bunchy top. IV. Control of the disease. Journal of Fijian Agricul-
    tural University. 25: 44-49.

Management of Grape Diseases in Arid Climates

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Abstract: Diseases of grapes are important limiting factors in producing wine and table grapes. Powdery mildew is the most important disease of grapes and is managed with cultural practices and by the application of fungicides. Fungicides are used most effectively if applied according to disease forecasting models and alternated to avoid fungicide resistance. Bunch rot is an important disease on some grape cultivars and in the more humid vineyard locations. It is managed by cultural methods such as leaf removal, hedging, and shoot positioning. Its control relies on the use of fungicides often combined with those used for powdery mildew. Fungicides for Bunch rot control are generally applied four times over the growing season according to the calendar or are based on disease forecasting models. Crown gall is an important disease in cool areas where freezing damage occurs in winter and it is managed primarily by cultural methods. Rootstock selection, heat treatment, and shoot tip propagation of cuttings have been shown to exclude the pathogen from new plantings. Biological control of the pathogen appears to be promising but has not been used commercially. Postharvest decay is an important problem in stored table grapes. Decay is controlled by sulfur dioxide fumigation but alternatives to this treatment are needed because of the possibility of high sulfite residues and bleaching of the berries. Acetic acid or ozone fumigation appears to be promising alternatives.

1. Introduction

The growing of grapes is an important industry throughout the world covering an area of approximately 10 million hectares (Pearson and Goheen, 1988). In Canada two major areas are devoted to the growing of grapes. The largest area is in Eastern Canada where they are grown under a relatively humid climate and the other, relatively small, but productive area is in British Columbia (BC) where the grapes are mostly grown in an arid climate. The arid climate of BC does not foster as many diseases as are found in the more humid climates (Table 1). This chapter will focus only on diseases that occur in arid climates. The chapter will still be useful to grape growers in humid climates because the major diseases are the same with the exception of downy mildew. Growers who would like information on the many other diseases of grapes, not found in this chapter, are directed to the "Compendium of Grape Diseases" edited by Roger C. Pearson and Austin C. Goheen (1988) for this purpose. Diseases covered in depth are powdery mildew and bunch rot because they are the most widespread and damaging to both table and wine grapes. Crown gall is covered because it is an important disease in newly established vineyards. Post harvest rots are included because these diseases are important in table grapes and are often overlooked.

Table 1: Important diseases of wine and table grapes

Disease	Causal agent	Disease type	Distribution
Antrthracnose, Bird's-eye rot	*Elsinoë ampelina*, fungus	Lesions on leaves, shoots, and fruit	Rainy, humid regions
Armillaria root rot,Oak root fungus Black rot	*Armillaria mellea*, fungus, *Guignardia bidwellii*, fungus	Attack roots causing slow delcine Attack berries forming blue-black mummies	Widespread, serious in France Northeaster North America, Europe and South America
Botrytis bunch rot and blight, Pouriture grise	*Botrytis cinerea*, fungus	Attacks berries discoloring them and forming mycelial mats	Widespread, develops in humid conditions
Downy mildew, Mildiou Esca, Black measles, Apoplexy	*Plasmopara viticola*, fungus Presumably toxins from wood-rotting fungi	Attacks green leaves, shoots, and berries Causes discoloration of leaves and berries, and sudden vine death	Warm and wet regions during vine growth Warm temperate zone
Eutypa dieback, dying arm	*Eutypa lata*, fungus,	Attacks arms, cordons, or trunk	Occurs where mean annual rainfall exceeds 600 mm in warm and cool areas
Phomopsis cane and leaf spot	*Phomopsis viticola*, fungus,	Lesions on leaves, shoots and cluster stems	Widespread especially where vines may be kept wet after budbreak
Powdery mildew, Oidium	*Uncinula necator*, fungus	Attacks foliage, canes and berries causing them to dry and split tropics	Widespread occurring in most grape growing areas including the
Rust, Rouille de la vigne	*Phsopella amplopsidis*, fungus	Attacks foliage producing yellow pustules	Common in Asia and Central America
Crown gall, Black knot	*Agrobacterium vitis*, bacterium	Produces galls on lower trunk and lesions on roots	Common where climatic conditions favor freeze injury
Pierce's disease	*Xylella fastidiosa*, bacterium	Attacks vines causing them to decline and die	Occurs primarily in Southern California
Corky bark, Grapevine stem pitting	Undetermined, virus like	Attacks vines causing decline and eventually death	Occurs everywhere grapes are grown

table 1 contd ...

Fanleaf degeneration, Infectious degeneration and decline	Grapevine fan leaf virus	Attacks vines causing leaf deformation and vine decline	Occurs everywhere grapes are grown
Aspergillus rot	*Aspergillus niger*, postharvest fungus	Attacks berries causing black decay	More common in warmer countries
Blue mold rot, Penicillium rot	*Penicillium* spp., postharvest fungus	Attacks berries causing greenish-blue decay	Occurs everywhere grapes are grown
Gray mold rot, Botrytis rot	*Botrytis cinerea*, postharvest fungus	Attacks berries causing slip-skin	Occurs everywhere grapes are grown
Rhizopus rot	*Rhizopus* spp., postharvest fungus	Attacks berries causing white mold strands	Occurs everywhere grapes are grown

Source: Compendium of Grape Diseases. Edited by Roger C. Pearson and Austin C. Goheen, APS Press, St. Paul, MN. 1988.

2. Powdery mildew

2.1 Biology

Powdery mildew, also known as Oidium, is caused by the fungus *Uncinula necator* (Schwein.) Burrill (*Oidium tuckeri* Berk.). The fungus is an obligate parasite with a narrow host range attacking only members of the family *Vitaceae* (Farr *et al.*, 1989). These hosts are *Ampelopsis* Michx. (monk's-hood-vine, peppervine, and heartleaf ampelopsis), *Parthenocissus* Planch. (American ivy, five-leaved ivy, Virginia creeper, woodbine, Boston ivy, and Japanese ivy) and *Vitis* L (various grape species including American grape, European grape, and wine grape). Common powdery mildew fungi that occur on fruit and ornamental crops such as apple and roses are not able to infect grapes. The disease cycle of grape powdery mildew has two stages known as primary and secondary (Pearson, 1988). The primary infection is caused by ascospores released from a fruiting body known as a cleistothecium (Pearson and Gadoury, 1987), or by conidia produced by fungal mycelium that have overwintered in the buds (Sall and Teviotdale, 1982). Cleistothecia are produced on leaves, shoots and berries in late summer and overwinter in bark crevices (Gadoury and Pearson, 1988). Rainfall of greater than 2.5 mm appears to be a critical event in the release of ascospores and the initiation of powdery mildew epidemics in areas where cleistothecia are sources of primary inoculum (Gadoury and Pearson, 1990a). The percentage of ascospores that germinated and formed appressoria increased as temperature increased from 10 to 23° C (Gadoury and Pearson, 1990b). In order for infection to occur by released ascospores takes at least 12-15 hours of continuous leaf wetness when temperatures

average between 10 and 15° C (Weber *et al.*, 1997). Secondary infection occurs 7 to 10 days after primary infection. The secondary infection is produced by conidia released by the fungus as it grows over the surface of green shoots and leaves. The disease spreads quickly in early summer when temperatures are moderate. At temperatures above 33° C the fungus does not grow. Grape berries respond to *U. necator* by becoming resistant to infection as they mature. For example, Concord grape berries are highly
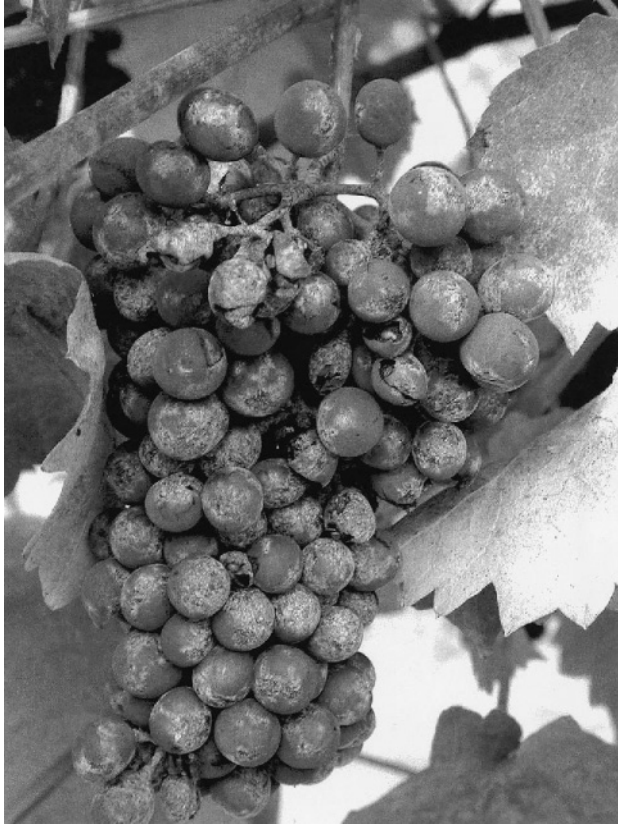
Figure 1: Grape cluster heavily infected by powdery mildew. Note the occurrence of split berries, berries that have dried up, and the netlike pattern of scar tissue on many of the berries.

susceptible to infection at prebloom and fruit set, but become almost immune to infection 2 weeks or more later (Wilcox *et al.*, 1997). In general fruit is susceptible to infection from the beginning of development until the sugar content reaches 8% (Sall and Teviotdale, 1982). Old infections become inactive and the berries become immune after the sugar content exceeds 15%. Rachises and leaves remain susceptible

much later in the season.

2.2 Symptoms

The fungus attacks leaves, berry clusters and canes (Fig.1). Leaf infection is character-
ized initially by small round colonies that develop a white color as the colony matures.
These colonies can be detected early in their development by faint yellow patches
about 6 mm in diameter and by observing them with a 10X hand lens. (Sall and
Teviotdale, 1982). The white patches coalesce to form extensive areas of white cob-
web-like growth over the entire leaf surface. The berries express early infection by
cracking and appear grayish as they mature. Infected older berries of white cultivars
may turn dull gray-green, while those of black cultivars turn pinkish red. A netlike
pattern of scar tissue sometimes forms on the surface of infected berries. Split berries
either dry up or rot. Canes are scarred by infection and develop dark brown blotches
over their surface. These canes may be used as indicators of powdery mildew infection
after fruit and leaves are gone.

2.3 Disease management

Grape cultivars vary in susceptibility to powdery mildew (Doster and Schnathorst,
1985) although most of the desirable wine varieties are susceptible (Table 2). Fungi-
cide treatment rates and application intervals should be varied according to the grape
variety that is being treated. For example, Chardonnay is very susceptible and may
need to be treated more often and at a higher rate of fungicide than Pinot gris which is
of low susceptibility.

2.3.1 Chemical control

Several classes of fungicides are used to control powdery mildew (Table 3). The fun-
gicides are applied to protect green tissue from infection and are applied from prebloom
until after veraison. Sulfur has been used to control powdery mildew and mites on
grapes since 1880 (Thomson, 1997). Sulfur comes in a wide range of formulations
however they can be categorized as either dusts, wettable powders, or flowable liq-
uids. In dry climates sulfur dust is preferred where application machinery is available,
and wettable powder and liquid formulations where rainfall is plentiful during the
growing season. The fungicidal action of sulfur on powdery mildew is due to its va-
porization (Wilhelm, 1954). The optimal range for sulfur activity is between 25 and
30° C (Pearson, 1988). Above 30° C the risk of phytotoxicity increases greatly, and
applications at 35° C or higher are not recommended. Spray intervals for sulfur may
vary from 6 to 16 days depending on weather conditions. Based on the mean of the
minimum and maximum daily temperatures in their vineyard growers can schedule
sulfur sprays according to temperature.

Ergosterol biosynthesis inhibitors or more specifically, demethylation inhibi-
tion (DMI) fungicides are more effective than sulfur against powdery mildew. This is
because DMI fungicides prevent spores of *U. necator* from growing at extremely small

doses, are locally systemic with vapor activity which makes them resistant to weather-
ing, and have lengthened spray intervals (Pearson, 1986). Spray intervals for these
fungicides are two to three times longer than the standard sulfur application schedule
of 7 to 10 days. Another advantages they have are their low application rates when
compared to sulfur. For example the amount of myclobutanil used per hectare is 200 g
compared to up to 2.4 kg per hectare of wettable sulfur (Anonymous, 2000).

   The DMI fungicides also present some problems for growers. The activity spec-
trum for these fungicides is narrow and they are ineffective in controlling bunch rot.
Although they have been used for approximately 15 years around the world, *U. neca-
tor* has developed resistance to DMI fungicides in some wine growing regions. Re-

Table 2: Susceptibility of some common wine grapes to powdery mildew

Variety	Type	Susceptibility
Chardonnay	White wine	Susceptible
Gewurztraminer	White wine	Susceptible
Chancellor	White wine	Susceptible
Bacchus	White wine	Susceptible
Riesling	White wine	Susceptible
Cabernet Sauvignon	Red wine	Susceptible
Sauvignon blanc	White wine	Susceptible
Concord	Juice	Intermediate
Pinot noir	Red wine	Intermediate
Merlot	Red wine	Intermediate
Semillon	Red wine	Least susceptible
Pinot gris	White wine	Least susceptible
Auxerrois	White wine	Least susceptible

Source: Management Guide for Grapes for Commercial Growers 2000-2001 Edition, British

ports from California (Gubler *et al.*, 1996; Ypema *et al.*, 1997) are the most compre-
hensive to date, although local reports of resistance to DMI fungicides have been made
in Portugal, Australia, and Canada. It was shown that continual application of
triadimefon leads to reduced sensitivity of *U. necator* to triadimefon and the related
DMI fungicides of myclobutanil and fenarimol. At concentrations used commercially,
myclobutanil and fenarimol may suppress the least sensitive isolates sufficiently to
prevent an epidemic, but they may select for *U. necator* less sensitive to all three DMI
fungicides if control is not complete (Ypema *et al.*, 1997). Sufficient coverage and
alternation of DMI fungicides with broad-spectrum fungicides should further delay
development of DMI resistance. In other words, fungicide resistance is checked by
making sure that label rates are followed, the DMI fungicide is never used more than
two times in succession, and it is alternated with a fungicide with a different mode of
action. These fungicides should never be used more times in the growing season than
the maximum number of times stated on the label. It is most prudent to use DMI
fungicides early in the season when their systemic activity can be used to greatest
advantage at prebloom and bloom. They also provide the optimum amount of protec-

tion at this time preventing the disease from becoming strongly established.

Several fungicides have recently been developed for the control of powdery mildew in the 1990's (Table 4). The most well known of these fungicides are the strobilurins (Leroux, 1996). Two fungicides from this class are registered on grapes in the United States, and have the common names, azoxystrobin and kresoxim methyl. These fungicides are extremely important additions to the control of powdery mildew because they have a different mode of action than the DMI fungicides. They block

Table 3: Fungicides used in grapes to control powdery mildew and bunch rot

Common name	Type	Disease spectrum	Movement in plant
Benomyl	Organic, benzimidazole compound	Powdery mildew and bunch rot	Systemic foliar
Sulfur	Inorganic, elemental compound	Powdery mildew	Contact, vapor action
Azoxystrobin	Organic strobilurin compound	Powdery mildew	Systemic foliar with translaminar effect
Kresoxim methyl	Organic strobilurin compound	Powdery mildew	Systemic foliar with translaminar effect
Tryfloxystrobin	Organic strobilurin compound	Powdery mildew	Systemic foliar with translaminar effect
Myclobutanil	Organic, DMI , triazole compound	Powdery mildew	Systemic protectant and curative
Quinoxyphen	Organic phenoxyquinoline compound	Powdery mildew	To be determined
Dichloran	Organic, chloronitroben-zene compound	Bunch rot and Rhizopus rot	Foliar and postharvest fungicide
Iprodione	Organic, dicarboximide compound	Bunch rot	Foliage-contact protectant and curative
Vinclozolin	Organic dicarboximide compound	Bunch rot	Contact and protectant
Fenheximid	Organic, cyclohexane-carboxamide compound	Bunch rot	Contact protectant
Cyprodinil	Organic, anilinopyrimi-dine compound	Bunch rot	Systemic foliar
Captan	Organic carboximide compound	Bunch rot	Contact foliar with protective and eradicating effect

Source: Agricultural chemicals Book IV – Fungicides 12th edition by W.T. Thomson, Thomson Publications, 1997.

respiration of the fungal mitochondria leading to cell death (Ypema and Gold 1999). This makes them very good candidates for fungicide resistance management in grapes. They are also very effective having locally systemic activity with high affinity for the

waxy layers of the plant surface. These fungicides are redistributed over the plant by superficial vapor movement. It is important to note that azoxystrobin will cause tissue damage to McIntosh apple in extremely minute amounts and kresoxim methyl will damage certain cherry cultivars so they should be used with extreme caution when applied near these crops. Another new powdery mildew material that is being tested is quinoxyfen. It belongs to a different chemical class than either the strobilurins or DMIs and is thought to work by inhibiting DNA synthesis. Experimental trials indicate that it is an effective control for powdery mildew in grapes.

Several alternatives to synthetic fungicides for the control of powdery mildew are known. Generally they are contact materials with very limited systemic activity. Potassium silicate has been tested with some success. When it was applied as a spray to rooted LN33 grape cuttings it reduced powdery mildew infection (Bowen *et al.*, 1992). In a field trial on *Vitis vinifera* L. grapes in British Columbia, Canada, silicon sprays reduced the incidence of powdery mildew without significant loss in yield in two of three years (Reynolds *et al.*, 1996). Efficacy of silicon was less than that of sulfur under situations of heavy disease pressure. Tasters could not distinguish between wines from sulfur and the silicon-sprayed grapes, suggesting that the mildew levels in the silicon treatments did not affect taste. Reduced severity of grape powdery mildew by silicon sprays may be partly due to a physical barrier to hyphal penetration and to a resistance response involving the lateral movement of silicon and its deposition within the leaf (Bowen *et al.*,1992).

Plant and petroleum oils are also important alternatives to synthetic fungicides for the control of grape powdery mildew. Three petroleum (mineral) oils and two glyceridic plant oils, applied as emulsions (1% v/v) in water, were tested on Chardonnay grape plants (Northover and Schneider, 1996). The plant oils showed significant action only in pre-lesion treatments and as antisporulants in treatments applied to estab-

Table 4: Percent powdery mildew in grapes treated with various fungicides

Fungicide Treatment	%Foliage infection	%Fruit infection
Myclobutanil	4 b*	6 c
Azoxystrobin	1 b	2 c
Kresoxim-methyl	0 b	2 c
Quinoxyphen	1 b	19 bc
Control	26 a	100 a

*Letters in columns followed by the same letter do not differ significantly according to Duncan's Multiple Range Test (P=0.05).

lished lesions. On the otherhand the petroleum oils were as effective as myclobutanil in suppressing powdery mildew. They showed moderate protection up to 4 days, excellent pre- and post-lesion curative action, and excellent antisporulative activity.

A complex clay found in a small geological area of Northern British Columbia has proven to be effective in the control of powdery mildew on grapes. This unique

natural oceanic clay is a heterogenous complex of minerals, ions and elements including trace and rare earth elements. Foliar sprays of this non-swelling chlorite mica clay were applied to field-grown wine grapes before natural inoculation with *U. necator* (Ehret *et al.*, 2001). Addition of 0.1% Tween 80 surfactant enhanced the ability of the foliar sprays to reduce powdery mildew severity on cucumbers. The application of clay as a spray in water was effective in reducing foliage infection of both 'Reisling' and 'Chancellor' grapes (Table 5).

The high rate of 4 kg/100 L clay in water appeared to be better than the low rate of 2 kg. The 4 kg rate of clay protected the clusters of Reisling and Chancellor grapes for approximately 6 weeks after its last application in September. When clay was applied four to five times in a program which included myclobutanil at bloom to take advantage of myclobutanil's local systemicity (Table 6), the clay was as effective as myclobutanil used consecutively for the whole season. Clay appeared to provide the added benefit of suppressing leafhoppers in this trial. It did not have any deleterious affect on grape juice quality such as pH, BRIX, or titratable acidity when compared to grapes from the standard spray program. This promising material could be an alternative to sulfur where it is important to keep sulfur residue levels lower and could have some important growth promoting properties (Ehret *et al.*,1998).

2.3.2 Biological control

Biological control of grape powdery mildew has not been very successful although several attempts have been made to make use of parasites or antagonistic microorganisms as viable alternatives to synthetic fungicides and sulfur. *Ampelomyces quisqualis* Ces. is a naturally occurring parasite of powdery mildew fungi (Falk *et al.*, 1995). A major limitation to the use of this biological control in arid climates is it requires free

Table 5: Percent powdery mildew on Reisling and Chancellor grapes treated with clay

Treatment	Rate per 100 L Water	Reisling Foliage	Chancellor	
			Foliage	Clusters
Clay program 1	2.0 kg	77 bc*	74 ab	0 b
Clay program 2	4.0 kg	71 c	43 b	12 b
Myclobutanil program	7.5 g	90 ab	41 b	5 b
Control	——	93 a	94 a	68 a

*Letters in columns followed by the same letter do not differ significantly according to Duncan's Multiple Range Test (P=0.05). Source: Ehret *et al.*, 2001

water for infection. Wetting can be provided by rain or irrigation although, even with these extra sources of water, it is unlikely that *A. quisqualis* alone will provide adequate control. *A. quisqualis* could become important in grape powdery mildew control if a strategy could be found that would use it in addition to other controls. The only other important biological control of grape powdery mildew is certain strains of *Bacil-*

lus subtilis. The QST 713 strain being developed by AgraQuest, Inc., Davis, California, USA was found to effectively control powdery mildew in both California and Chile. In these trials powdery mildew control was equal to or better than commercial standards. The biocontrol has been registered in the United States under the name of Serenade. Research continues on its formulation and use in integrated pest management programs.

Table 6: Percent powdery mildew and leafhopper damage in Pinot noir grapes treated with clay (40 kg/ha)

Treatment	% Powdery mildew		% Leafhopper
	Foliage	Fruit	Foliage
Clay program 1	72 b*	4 b	2 b
Clay program 2	68 b	4 b	12 b
Myclobutanil program	71 b	7 b	30 a
Control	97 a	70 a	34 a

*Letters in columns followed by the same letter do not differ significantly according to Duncan's Multiple Range Test (P=0.05).

2.3.3 Integrated pest management

Powdery mildew is most effectively managed by using the concepts of integrated pest management (Gadoury, 1993). First, the disease must be correctly identified. For example, powdery mildew could be mistaken for downy mildew resulting in application of ineffective fungicides and the use of incorrect cultural practices. It is important to record the dates that colonies first appear on foliage and fruit, and the severity of infection should be noted. Monitoring information could influence the selection of spray materials, and frequency of application of fungicides in the current and following year. For example, if powdery mildew colonies are observed early in the season just after bloom, and there are numerous colonies, sulfur may not provide enough control over the growing season. The grower can use this information to pick a fungicide spray program that will provide better control. In this case a DMI alternated with a strobilurin class of fungicide would provide the best control. If bunch rot is suspected because it was a problem in the previous year, this information should be used to incorporate a fungicide in the spray program that is effective against this disease. The integrated pest management approach also requires that fungicide resistance management is used in the selection of fungicides. This means that fungicides are alternated with different modes of action as much as possible.

      Biological control products should be integrated with chemical and cultural controls whenever the opportunity presents itself (Jacobsen and Backman, 1993). The integrated approach to disease control relies heavily on use of cultural practices that reduce excessive vine growth and spread of powdery mildew. Shoot removal and hedging to allow air movement within the vine canopy will slow the spread of powdery

mildew. Fertilizer and water should be used judiciously to avoid excessive growth. An open canopy will not only maintains a microclimate less favorable for disease development but also allows better penetration of fungicide (Pearson, 1988). Dormant sprays of fixed copper products or lime sulfur may reduce overwintering inoculum of *U. necator* on grape vines and should be used where powdery mildew was severe in the previous growing season.

2.3.4 Disease forecasting

An important part of powdery mildew management depends on the use of disease forecasting models. Several models for forecasting powdery mildew have been developed, but for most, information on them is either confidential or difficult to obtain. The exception is the UC model developed at the University of California, Davis, California which is widely available and thoroughly tested. A major advantage of the UC model software is that many companies that sell weather instruments make it an integral part of the instrumentation. This allows the grower to use it with ease. The model evaluates climatic conditions within the grapevine canopy and assesses the likelihood for the development of powdery mildew (Weber *et al.*, 1997). The ascospore infection forecasts are based on average temperature during the time leaves are wet, and utilizes the 0.67 value for conidial infection provided by the Mills table to predict grape infection. The Mills table was originally developed for forecasting infection of apple by *Venturia inaequalis* (Cke.) Wint. and has been revised (MacHardy and Gadoury, 1989). In general 12 to 15 hours of leaf wetness are required between 10 to 15° C for infection of grape by *U. necator*. Once infection has occurred, the model switches to the risk assessment phase and is based entirely on the affect of temperature on the reproductive rate of *U. necator.* (Thomas *et al.*, 1994). The model generates daily conidial risk indices based solely on temperature in the canopy. In order for the conidial risk index to increase, there must be 6 hours during the day when canopy temperatures are between 21 and 30° C. Once started, the index climbs 20 points on each day that meets the 6 hour temperature requirement. The index falls 10 points on days that are unfavorable for powdery mildew development. By knowing the likelihood that powdery mildew could be developing in their vineyards, growers are able to accurately time fungicide applications. During periods of low risk, fungicide application intervals can be safely stretched.

In order to use the UC model the grower must have a means for recording leaf wetness and temperature. Two types of weather stations have evolved for the use of the UC model software. The most complete system is sold by Adcon Telemetry Inc, Boca Raton, Florida. It utilizes radio telemetry to transmit weather information from field weather stations to a receiving base station. Sensors in the field collect temperature, precipitation, relative humidity and leaf wetness data, usually every 30 seconds and transmit it to a central computer. The data is averaged every 15 minutes. Powdery mildew risk indices are calculated at the base station by a personal computer containing the UC model software. Growers access the weather data by using a computer to download it from the base station computer.

A less expensive alternative is to use miniature weather stations that the grower

individually purchases. Sensors measure leaf wetness, temperature, and relative humidity in the grape canopy. Attaching a PC to the weather station downloads the weather data. UC model software that can be purchased with the weather station will automatically predict infection and degree of risk for conidial infection when used in conjunction with the weather station software. This type of configuration can be purchased for less than a thousand dollars.

Weather stations of this type sold by Spectrum Technologies, Inc. Plainfield, Illinois were tested at eight vineyard sites throughout the wine growing area of interior British Columbia (Fig. 2). The weather stations used in each vineyard were Model 450 Watch Dog Data loggers. The data loggers contained two internal sensors for relative humidity and temperature and an external sensor for leaf wetness. They were protected from rain, solar radiation, and other sources of radiation by a miniature radiation shield. The weather stations were placed near the vineyard roadway to facilitate downloading the weather data three to four times a month during the growing season. The instruments recorded every 30 minutes allowing them to operate for two weeks before permanent loss of weather data would occur. The powdery mildew disease forecasting software purchased with the unit was based on the UC model and required leaf wetness for the prediction of ascospore infection and temperature to predict risk of conidial infection. When the vineyards were visited to downloaded weather data they were also evaluated for the incidence of powdery mildew. The results of the Spectrum Technologies, Inc. software for ascospore infection and conidial risk assessment have been summarized in Table 7.

Very little powdery mildew developed on the Chardonnay grape foliage at any of the monitored sites and no berries were infected even though the model predicted that conditions for infection had been met. Furthermore the conidial index was extremely high for July and August indicating that powdery mildew should have spread rapidly from infected vines. The only explanation for the absence of powdery mildew was that the disease had not become established in these vineyards. The inoculum of *U. necator* must have been absent or too low to cause the disease. Important information learned from this study was that the Model 450 Watch dog dataloggers are reliable and easy to use. The study is being repeated in 2001 with two additional vineyards with a history of severe powdery mildew. The model will be used as a guide in the application of fungicides to control powdery mildew in these vineyards.

3. Bunch rot

Botrytis bunch rot or gray mold (Fig. 3) caused by *Botryotinia fuckeliana* (de Bary) Whetzel, of which only the conidial form, *Botrytis cinerea* Pers., is usually seen in the vineyard, is an important disease of grapes especially where cold, damp weather prevails. In wine production, the most serious damage is qualitative, from the modified chemical composition of diseased berries (Bulit and Dubois, 1988). Gray mold is sometimes confused with "noble rot", a desirable condition, that only occurs under the proper climatic conditions. These conditions are not common and most growers prefer to avoid it. *B. cinerea* frequently develops on withered calyptras, stamens, and aborted berries. From these sites it attacks the berry stalks (pedicel) and main axis of the grape

cluster (rachis). Toward the end of summer portions of the cluster below the infected area wither and drop off. From veraison grapes are infected directly through the epidermis or through wounds. The mold progressively decays the entire cluster.

3.1 Bunch rot disease management

Grape cultivars differ in susceptibility to bunch rot. For example, Chenin blanc, White

Figure 2: Miniature weather station in a vineyard in British Columbia, Canada. The Watch Dog datalogger has been removed from its protective weather enclosure so it can be seen. This particular datalogger records leaf wetness, relative humidity, and temperature for use in forecasting powdery mildew.

Reisling, and Zinfandel, sustain damage from bunch rot in most years (Savage and Sall, 1982). When rains occur before harvest or with overhead sprinkling, major losses can occur on these cultivars and more resistant cultivars such as Sauvignon blanc and Pinot noir. Serious losses can even occur without rain moisture. In these instances, *B. cinerea* infection of grape berries occurs in cultivars with dense canopies or tight berry

clusters (Gubler *et al.*, 1987).

3.1.1 Canopy management

Severe Botrytis infection was observed primarily in vineyards with vigorous canopy growth (Savage and Sall, 1983). Minor changes in air movement or light penetration in a grape cluster could alter the water status at boundary layers where the fungus

Table 7: Predicted ascospore infection dates for powdery mildew and mean conidial index for eight vineyard sites according to disease forecasting software (Spectrum Technologies Inc, Plainfield, Illinois) on Chardonnay grapes

Vineyard location	Ascospore infection		Mean conidial index*		
	Leafwetness (hr)	Date	July	August	September
Osoyoos	9.5	July 4	99.0	98.1	46.7
Oliver 1	10.3	July 4	99.0	98.4	60.0
Oliver 2	13.0	June 12	91.6	——	30.7
Keromeos	8.0	August 21	94.8	96.1	58.3
OK Falls	——	——	96.5	92.9	22.3
Penticton	12.5	June 25	95.5	92.6	20.0
Westbank	——	——	96.8	91.0	7.6
Kelowna	11.8	July 4	87.4	83.2	44.7

*Powdery mildew risk index: 0 – 30 = Light infection risk; 40 – 50 = Medium infection risk; 60 – 100 = Heavy infection risk.

exists. Incidence of *B. cinerea* infection at harvest under drought conditions was significantly influenced by trellis type and by fungicide application made at bloom. Disease was lower in the "two-wire vertical" trellis than in the "crossarm" trellised grape vines. Further studies on canopy management by hedging, shoot removal, and leaf removal, showed that leaf removal reduced bunch rot more than any other treatments including application of fungicides (Gubler *et al.*, 1987). Disease severity was reduced from 3.6% rot per cluster in the intact, nonsprayed control to 0.30% in the nonsprayed leaf removal treatment. In British Columbia, shoot positioning and shoot positioning plus hedging reduced weight of cane prunings relative to control vines but increased yield, cluster weight, and berries per cluster (Reynolds *et al.*, 1992). Higher berry temperatures were recorded in the shoot positioning plus hedging and basal leaf removal treatments that corresponded to lower fruit titratable acidity levels. Neither iprodione nor canopy manipulation appeared to have any significant impact on the percentage of bunch-rot- affected clusters in this trial. However, infection levels in the unsprayed vines were extremely low averaging just over 1% infection for the two years of the trial. This study showed that shoot positioning improved wine grape quality more than basal leaf removal. The impact of canopy manipulation on bunch rot was inconclusive.

3.1.2 Cluster management

Tight compressed clusters of grapes are often associated with the development of se-
vere bunch rot. Explanations are that the clusters do not dry as fast as loose clusters
after rain (Vail and Marois, 1991), tight clusters often cause berries to rupture (Sall *et
al.*, 1982) and berries in contact with one another are more prone to infection by *B.
cinerea* (Marios *et al.*, 1986). This was because berries in contact with one another

Figure 3: Chardonnay grape clusters infected with *Botrytis cinerea* causing bunch rot. Note
that the clusters of this variety are tightly closed predisposing them to bunch rot.

within the grape clusters developed poor epicuticular wax between their surfaces and
had numerous shallow depressions. In addition to these physical factors that promote
bunch rot, grape berries produce exudates in water trapped between the berry surfaces
providing nutrients for conidia of *B. cinerea* (Kosuge and Hewitt 1964).

It is possible to measure cluster tightness with the University of California (UC)
Firmness Tester Method (Vail and Marois, 1991). This method measures the force in
newtons (N) required to separate two contiguous berries by a distance of 2 mm. Clus-

ter tightness was measured and bunch rot was evaluated for six Chardonnay clones in each of two trials, one cane-pruned and the other spur-pruned (Vail *et al.*, 1998). The clone with the least compact clusters had the lowest disease severity levels. Bunch rot was greater in the cane-pruned trial than in the spur-pruned trial. The adoption of the UC Firmness Tester Method is encouraged for use by grape breeders. Studies on cluster tightness have shown that properly timed gibberellic acid applications elongate berry stems resulting in looser clusters (Duncan, 1999). This should result in less bunch rot.

3.1.3 Biological control

Species of the fungus *Trichoderma* have been used to control *B. cinerea* in several crops (Gullino, 1992). The fungus is applied at postbloom, bunch closing, veraison, and 3 weeks before harvest on grape. In trials in Europe it was found that *Trichoderma* spp. were as effective as the older contact fungicides such as dichlofluanid but less effective than the newer more active materials. At least three other biological controls are being tested for use against *B. cinerea* around the world. In the United States, *Bacillus subtilis,* strain QST 713 has been found to be effective against *B. cinerea* in grapes in trials conducted by AgraQuest, Davis, California. Another biological control being developed in Europe is *Ulocladium atrum.* Its high ecological competence makes it an attractive candidate for application to grapes (Elmer and Kohl, 1998). Finally, *Gliocladium roseum* Bainier has been extensively tested as a biological control for *B. cinerea* in Canada on many crops (Sutton *et al.*, 1997). The material has been applied by spraying, immersion of the plant in a suspension of the conidia, or by bees that vector it to flowers.

3.1.4 Chemical control

In North America five fungicides, benomyl, captan, cyprodinil, fenheximid, and iprodione are currently used to control bunch rot (Table 3). A program of four applications has given satisfactory results: after bloom; just before berry touch; at the beginning of veraison; and 3 weeks before harvest (Bulit and Dubos, 1988).

Benomyl, captan, and iprodione have been used for many years to control bunch rot. These products are relatively effective but have specific problems related to their use. Before benomyl became available there were no highly effective means to control *B. cinerea* in grapes. However, within two years in Northern France there were indications of a benomyl resistance problem and after four years these fungicides were no longer effective (Smith, 1988). Fungicide resistance has limited the usefulness of benomyl in many areas and even if it is not used for many years the resistance remains in the fungal population (Schuepp and Kung 1981).

The situation with dicarboximide fungicides (iprodione and vinclozolin) is much more heterogenous as far as the relation between incidence of resistance and disease control (Lorenz, 1988). There are numerous studies concerning *B. cinerea* resistant strains with conclusions that are sometimes contradictory. The resistant strains have a moderate level of resistance and low fitness if mixed with sensitive ones (Gouot, 1988).

Anti-resistance strategies with these fungicides and the low fitness of the resistant isolates have allowed these fungicides to remain effective in many areas to this day. Cross-resistance between dicarboximide fungicides to dicloran and pentachloroni-trobenzene has been demonstrated (Latorre *et al.*, 1994). In Ontario Canada the best program for use of iprodione was found to be a mixture of captan and iprodione or if iprodione treatments were alternated with captan (Northover, 1988).

Two new fungicides, cyprodinil and fenheximid, were registered in North America in the late 1990's to control grape bunch rot. The anilinopyrimidines, which includes cyprodinil and pyrimethanil, represent a new group of fungicides with strong activity against *B. cinerea* (Hilber and Schuepp, 1996). The mode of action of these systemic fungicides is not fully understood although inhibition of biosynthesis of me-thionine is likely the mode of action in the case of cyprodinil (Masner *et al.*, 1994). Pyrimethanil has a different effect on *B. cinerea* inhibiting protein secretion including reduced levels of some hydrolytic enzymes thought to be important in the infection process (Neumann *et al.*, 1992). Fenheximid is a non-systemic fungicide with strong activity against *B. cinerea.* It prevents penetration of the fungus into the plant by in-hibiting germ tube elongation, mycelial growth and spore germination.

Strategies to help prevent fungal resistance are recommended for both these fungicides. Resistance by *B. cinerea* to cyprodinil appears to present a high risk be-cause three field-resistant isolates have been detected in two Swiss vineyards and loss of control due to field resistance was experienced in an experimental plot (Hilber and Hilber-Bodmer, 1998).

Bunch rot management also requires the use of insecticides to control insects that damage the berries (Fermaud and Le Menn 1989). In the vineyard a combination of fresh wounds and new inoculum are needed for successful wound infection (Coertze and Holz, 2001). Disease management strategies should concentrate on minimizing these wounds by controlling insects that make them, and protect wounds from infec-tion by effective coverage with fungicides that control *B. cinerea*.

3.1.5 Integrated control of bunch rot

Chemical control of bunch rot must be integrated with powdery mildew control in arid climates and in more humid climates where downy mildew and black rot also occur, it is important to include fungicides for their control as well (Gadoury, 1993). Generally, iprodione or captan are combined with a fungicide that controls powdery mildew. The recent registrations of fenheximid and cyprodinil are alternatives to iprodione and captan that are used for bunch rot control and should be alternated with one another for management of fungicide resistance.

Cultural practices that reduce the risk of bunch rot development should be used in addition to chemical control (Bettiga *et al.*, 1989). To slow development of bunch rot, avoid excessive vegetation by judicious use of nitrogen fertilization; increase aera-tion by using appropriate trellising systems and by removing leaves around the fruit; and control powdery mildew and insect pests that injure the berries. Another factor that influences bunch rot is the source of inoculum. Sclerotia of *B. cinerea* are a source of primary inoculum. Conidia produced by sporogenic germination and re-sporulation

of sclerotia in spring, can infect grape flowers in the same season (Nair, 1985). Infection of developing berries can also occur as a result of conidia produced by sclerotia and the berries may be infected at all stages of maturity (Nair and Nadtotchei, 1987). Sclerotia will survive in relatively dry soil for at least eight months but are likely to survive longer on canes. Fungicides effective against *B. cinerea* applied four times from bloom to veraison will likely reduce flower infection and bunch rot caused by sclerotia.

A disease infection model for bunch rot that would predict when infection has occurred would be useful to grape growers. Disease prediction models have been developed that use weather monitoring equipment that warn when conditions are conducive to infection based on temperature, leaf wetness, relative humidity and crop stage (Broome *et al.*, 1995). Previous disease history also has been used in developing a disease model for the rational application of fungicides (Nair *et al.*, 1987,1995). For example, a 50% incidence of *B. cinerea* monitored on grapevine tissues carried over from the previous season can predict a 30% primary infection of flowers in the new season. This requires fungicide application during flowering to reduce the predicted risk. The model developed by Broome *et al.*, (1995) is based on temperature and wetness duration of mature berries and may not be accurate for flower infection. They found that infection occurred after 4 hours at 12 to 30°C. Cabernet Sauvignon flowers require only 2 hours of wetness duration at 24°C for infection (Nair and Allen, 1993). This means the Broome model would need to be modified in order to predict early infection because of the shorter wetness duration. In some vineyards, postharvest disease was significantly less when sprays were made according to the Broome model recommendations compared with the standard spray program. It appears that the Nair model (Nair *et al.*, 1995) could be used to predict the risk of inoculum carryover from one year to the next and whether blossom sprays are needed and the Broome model (Broome *et al.*, 1995) to predict the number of sprays that need to made during the period from veraison to harvest. Certainly the models should be used along with canopy management, leaf and shoot removal, hedging, judicious irrigation and fertilization, and control of insects and powdery mildew.

4. Crown gall

Crown gall also known as black knot when it occurs on the aerial part of grape vines is an important bacterial disease of *Vitis vinifera* cultivars grown in cold climates. Injuries caused by freezing are thought to allow the bacteria to enter susceptible tissue and incite galls. *Agrobacterium vitis* sp-nov (Ophel and Kerr, 1990) is highly specific to grape and has not been found on other plant species or in soils other than in vineyards (Burr 1997). Crown gall also occurs in grape nurseries. Galls develop at wounds made by disbudding, at the base of rooted cuttings, and at grafts (Burr *et al.*, 1998). On roots *A. vitis* does not typically cause galls but instead induces a localized necrosis. Certain phenolic and other compounds released by wounded cells induce the virulence genes of *A. vitis*. The bacterium can survive systemically in vines that appear healthy. Pathogenic strains have been isolated from Gewertztraminer, Pinot Chardonnay, Pinot noir, and White Reisling, but was not detected in canes taken from apparently healthy vines (Burr and Katz 1984).

The major crown gall symptom is the fleshy galls that are produced in response to infection (Burr, 1988). Galls are mostly found on the lower trunk, near the soil line. Galled vines frequently produce inferior shoot growth. It is not possible to distinguish galls from abundant callus formation at grafts. In these cases the pathogen must be isolated and identified. Indicator plants such as *Kalanchoe diagremontiana* and *Nicotiana glauca* (Fig. 4) can be used to identify *A. vitis* isolates. These plants will express gall symptoms in the greenhouse in 6 to 8 weeks.

4.1 Crown gall disease management

Grape crown gall is managed by breeding for resistant cultivars, production of *A. vitis*-free vines, biological control, and cultural control (Burr *et al.*, 1998). *V. vinifera* cultivars including Chardonnay, Riesling, Merlot, and Cabernet Sauvignon are highly susceptible. *V. vinifera* hybrid cultivars are generally more resistant. In most cases it is not practical to plant resistant cultivars because they have undesirable qualities for making wine. Use of resistant rootstocks appears to be a more promising approach. Cuttings free of *A. vitis* can be produced by hot water treatment or shoot tip propagations. Both methods have produced promising results but further research is needed before these treatments can be considered reliable.

Several methods have been used for indexing dormant grape cuttings for *A. vitis.* These methods have been based on ELISA procedure, monoclonal antibodies, and by PCR-based methods (Burr *et al.,* 1998). One of the PCR-based methods was developed in British Columbia by Eastwell *et al.*, (1995) and has been shown to be extremely sensitive.

4.1.1 Biological control

Biological control of crown gall on fruit trees, caused by a bacterium closely related to the one that causes grape crown gall, has been effectively practiced with nontumorigenic *A. rhizogenes* (Kerr, 1972) for several years. However, this biological control agent is not effective against *A. vitis*. Several researchers have shown that there are antagonistic bacteria that are effective against the causal agent of grape crown gall (Burr and Otten, 1999). Probably the first promising biological control for grape crown gall was a nontumorigenic *A. vitis* bacterium isolated in South Africa and designated F2/5 (Staphorst *et al.*, 1985). Burr and Reid (1994) found that when F2/5 was applied to wounds on potted Chardonnay and Riesling plants in the greenhouse prior to inoculation with tumorgenic *A. vitis*, it reduced gall sizes on seven of ten plants. When applied at the same time as the pathogen (co-inoculated), it significantly reduced gall size and the number of galls produced for eight of nine *A. vitis* strains. Further research on the competitiveness of F2/5 in relation to tumorigenic *A. vitis* strains are necessary to determine if it will control disease in commercial vineyards. Some other bacterial antagonists that looked promising were isolated by Bell *et al.*, (1995) from the xylem sap of grapevines grown in Nova Scotia, Canada. They identified three endophytic bacterial strains that were capable of inhibiting *A. vitis.* Unfortunately these anatagonists

were variable in their control of crown gall probably because the bacteria were unable to establish themselves in the grape vessels.

Sholberg and Eastwell (1997) used the indicator plant, *Nicotiana glauca,* to identify promising biological control agents for grape crown gall. *N. glauca* will form galls in 6 to 8 weeks when inoculated with *A. vitis*. In their trials they found five biocontrol agents that reduced galls on *N. glauca* caused by *A. vitis*. The most promising biocontrol agent was a bacterial isolate from apple identified as *Pseudomonas*

Figure 4: Galls on *Nicotiana glauca* (mustard-tree, tree tobacco) caused by inoculation with *Agrobacterium vitis*. Small holes drilled into the stem of the plant were inoculated with a water suspension of *A. vitis.* Galls developed over a period of 2 to 3 months in the greenhouse.

syringae isolate 1100-6. It reduced the number of galls from 5.0 to 0.6 when co-inoculated with *A. vitis* on wounded *N. glauca* plants. These trials are being repeated on grape plants to determine if this biocontrol could be used commercially to control crown gall.

4.1.2 Cultural control

Use of K_2O instead of nitrogen fertilizers has been reported to prevent crown gall in French vineyards (Boubals, 1987), probably because it improves resistance of the vines to freezing. Where there is a risk of freeze injury, it is advisable to earth-up the foot of the vines (about 0.25 to 0.50 m) in late fall and then remove this coverage in the spring (Burr *et al.*,1998). Also avoid wounding vines and remove and destroy all dead and infected plants. Wounds caused by machinery, insects, mites and nematodes could predispose grape vines to infection by *A. vitis* and these sources of wounds should be prevented where practical. *A. vitis* will remain in vineyard debris for several years. Burr *et al.*, (1995) recovered tumorgenic *A. vitis* from living and decaying grape roots and canes 23 months after inoculation. The effectiveness of eradicating the pathogen from vineyard sites by removing infected vines and leaving soil fallow or planting to nonhosts will vary depending on the amount of grape debris in soil and its rate of decomposition. For new plantings avoid wet heavy soils, frost-prone areas, and exposure to the north. Growers should plant cold-resistant and crown gall resistant cultivars whenever possible (Burr *et al.*, 1998).

5. Postharvest rots

Several fungi will infect ripe grape berries and cause postharvest decay. Snowdon (1990) lists Aspergillus rot caused by *Aspergillus niger* v. Tieghem, Blue mold rot caused by *Penicillium* spp., Gray mold rot caused by *Botryotinia fuckeliana* (de Bary) Whetzel (conidial state: *Botrytis cinerea),* and Rhizopus rot caused by *Rhizopus oryzae* Went & Prinsen Geerligs or *R. stolonifer* (Ehrenb. Ex Fr.) Lind as the most important postharvest diseases of grape. Blue mold rot and Gray mold rot are found regularly in all grape producing countries. Aspergillus rot is important in the warmer grape-producing countries (Snowdon, 1990).

Blue mold begins as a white mycelial growth over stems and berries of harvested grapes. Within a few days of infection the white mycelium gives rise to greenish blue powdery spores that extends over the infected berries. The rot develops from wounds or where the berry has become detached from the pedicel. Gray mold, a more common postharvest disease than Blue mold is characterized by a condition at the early stage in fruit rotting described as 'slip-skin' because the epidermis is easily detached from the berry (Nelson, 1956). This is followed by brown discoloration and the production of gray-brown spores (Snowdon, 1990; Sholberg, 2001). Aspergillus rot produces pale and water-soaked tissue. Black spores are usually produced by *A. niger* on the surface of the grape berry and the spores are easily liberated resulting in soot-like deposits on adjacent berries (Snowdon, 1990).

5.1 Postharvest disease management

Minimizing injury to grapes at all stages of production and handling and cold storage at 0 to 5° C is very important in slowing the development of these postharvest diseases. Preharvest fungicidal sprays initiated during pre-bloom phase of berry development

will reduce gray mold infection (Nair *et al.*, 1995). Fungicides used for bunch rot control applied immediately prior to harvest likely reduce Gray mold decay after harvest especially if they are systemic. Cyprodinil, a fungicide known to be effective against *B. cinerea*, was applied to apples at harvest and the fruit was stored for 3 months. These fruit developed significantly less decay than control fruit when challenged with *B. cinerea* (Sholberg and Bedford, 1999). It is likely that cyprodinil would provide postharvest decay protection to stored table grapes. The fungicide, pyrimethanil which belongs to the same chemical class, appeared to control both Blue mold and Gray mold in stored apples (Sholberg unpublished). More research is needed on the use of cyprodinil and pyrimethanil to control postharvest decay of grapes.

Stored table grapes are treated with sulfur dioxide to prevent decay. Guidelines for its safe use can be found in a bulletin published by Luvisi *et al.*, (1992). Sulfur dioxide fumigation does not eradicate fungal infection but prevents the growth of fungi in the open air and slows disease progress. Normal accumulation of sulfur dioxide residues by repeated fumigation may cause injury and lead to higher than desired residue levels. Symptoms of sulfur dioxide damage are numerous small, sunken, bleached spots on each berry, or stem-end bleaching (Snowdon, 1990). Any residue level higher than 10 ppm is not acceptable in the United States, and could result in the impoundment and holding of grapes until their residue levels decrease to less than 10 ppm (Luvisi *et al.*, 1992). Alternatives to sulfur dioxide are needed because of concern with sulfite residues, damage to berries by bleaching and browning of the rachis (Marois *et al.*, 1986a,b), and poor decay control (DeKock and Holz, 1991).

Fumigation with hydrogen peroxide was investigated for control of decay in table grapes (Forney *et al.*, 1991). Hydrogen peroxide vapors reduced the germinability of *B. cinerea* spores, inhibited berry decay, and had no detrimental effect on berry quality. However this treatment continues to be experimental and will require further study to adopt its use to commercial storage rooms.

Acetic acid vapor fumigation of stored table grapes was evaluated in British Columbia, Canada in repeated trials over a 3 year period (Sholberg *et al.*, 1996). Grapes were fumigated for 30 min with 0.27% vol/vol acetic acid and stored for 6 weeks at 5°C. Acetic acid controlled decay by both *B. cinerea* and *Penicillium* spp. and was as effective as sulfur dioxide without changing fruit composition (°Brix, titratable acidity, pH, and color). Acetic acid fumigation of table grapes is a viable alternative to sulfur dioxide for preserving grapes in cold storage. Residues of acetic acid necessary to control mold on grapes are not likely to be of concern because they are found in many food products at concentrations ranging from 0.25 (baked goods) to 9.0% (relishes) (Doores, 1990) and the Food and Agriculture Organization, recognized that acetic acid is a normal constituent of foods, has set no limit on the acceptable daily intake for humans (FAO/WHO, 1973). Wine grapes could benefit from fumigation with acetic acid because sulfur dioxide used at crush, or after pressing led to higher titratable acidity than if acetic acid was used. Acetic acid could replace sulfur dioxide at crush to allow the production of low sulfite wines. Further research will be necessary to establish the procedures necessary for the use of acetic acid vapor in wine and table grapes.

Treatment of grapes with ozone, the triatomic form of oxygen could be an

alternative to sulfur dioxide. For the postharvest treatment of fresh fruit and vegetables, ozone can be used as a relatively brief prestorage or storage treatment in air or water, or as a continuous or intermittent atmosphere throughout the storage period (Palou *et al.*, 2001). Inactivation of microflora on food by ozone depends greatly on the nature and composition of food surface, the type of microbial contaminant, and the degree of attachment or association of microorganisms with food (Kim *et al.*, 1999). Exposing berries to ozone was almost as effective as sulfur dioxide fumigation for the control of storage decay caused by *R. stolonifer* and no deleterious effects were observed on the appearance of the grape cluster (Sarig *et al.*, 1996). No phytotoxicity was observed on berries, stems, or pedicels following exposures lasting up to 40 min with 8.0 mg/L ozone. Longer periods of time led to increasingly more severe phytotoxicity in some grape cultivars.

6. Conclusions

Popular grape varieties are susceptible to diseases caused by fungi and bacteria that injure the plant reducing yield, and lowering wine quality. The most important diseases in arid climates are powdery mildew and bunch rot. Crown gall and postharvest rots are less important problems and are specific to certain vineyards or as in the case of postharvest decay are restricted to stored table grapes. Important components to the management of these diseases are detection, use of forecasting models to predict their presence and risk, and use of appropriate controls. If fungicides are used, they should be used in an integrated pest management strategy that takes advantage of cultural and biological control. Fungicides should only be used at label rates and in a way that reduces the chance of fungicide resistance.

7. Future research

More research is needed on the development of grape disease forecasting models. They need to be made more grower friendly, especially to those growers with less than 40 hectares of grapes. The models need fine tuning for different climatic areas and in association with the fungicides that are available. In apple scab disease forecasting an eradicant fungicide is used after an infection period has occurred to prevent the disease (Sutton and Jones, 1981). The advantage of this approach is that the fungicide need only be applied after the apple scab fungus is present in the host tissue. This approach usually saves several sprays each growing season. Fungicides used for powdery mildew control likely do not reach back more than 96 hours as in the case of apple scab. Areas where powdery mildew is initiated by primary ascospore infections would benefit from knowing the number of hours after an infection period that a given fungicide could be used. The forecasting models would also be improved if the presence of the pathogen was linked to them. Risk of infection in the powdery mildew model is based strictly on temperature. If the disease were not present because no primary infection occurred the UC model would still recommend fungicide sprays if temperature conditions were conducive to powdery mildew. The disease forecasting program needs to be tied to a biological source of data such as spore traps that will

indicate the presence of the causal agent. Data obtained from the spore traps could then be incorporated into the disease model providing a very accurate indication of disease risk.

8. References

Anonymous. 2000. Management guide for grapes for commercial growers 2000-2001   edition. British Columbia Ministry of Agriculture, Food and Fisheries, Victoria, British Columbia.

Bell, C.R., Dickie, G.A., Harvey, W.L.G. and Chan, J.W.Y.F. 1995. Variable response of bacteria from grapevine xylem to control grape crown gall disease *in planta*. Am. J. Enol. Vitic., 46:499-508.

Bettiga, L.J., Gubler, W.D., Marois, J.J. and Bledsoe, A.M. 1989. Integrated control of botrytis bunch rot of grape. California Agriculture, March-April pp. 9-11.

Bowen, P., Menzies, J., Ehret, D., Samuels, L. and Glass, A.D.M. 1992. Soluble silicon sprays inhibit powdery mildew development on grape leaves. J. Amer. Soc. Hort. Sci., 117:906-912.

Broome, J.C., English, J.T., Marois, J.J., Latorre, B.A. and Aviles, J.C. 1995. Development of an infection model for Botrytis bunch rot of grape based on wetness duration and temperature. Phytopathology, 85:97-102.

Bulit, J. and Dubos, B. 1988. Botrytis bunch rot and blight. In: "Compendium of grape diseases" (eds. Pearson, R.C. and Goheen, A.C.) APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp. 13-15.

Burr, T.J. 1988. Crown gall. In: "Compendium of grape diseases" (eds. Pearson, R.C. and Goheen, A.C.) APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp. 41-42.

Burr, T. J. 1997. Staying on top of crown gall disease. Vineyard & Winery Management Jan/Feb 60-61.

Burr, T.J. and Katz, B.H. 1984. Grapevine cuttings as potential sites of survival and means of dissemination of *Agrobacterium tumefaciens.* Plant Dis., 68:976-978.

Burr, T.J. and Reid, C.L. 1994. Biological control of grape crown gall with non-tumorigenic *Agrobacterium vitis* strain F2/5. Am. J. Enol. Vitic., 45:213-219.

Burr, T.J., Reid, C.L., Yoshimura, M., Momol, E.A. and Bazzi, C. 1995. Survival and tumorigenicity of *Agrobacterium vitis* in living and decaying grape roots and canes in soil. Plant Dis., 79:677-682.

Burr, Thomas, J., Bazzi, C., Sule, S. and Otten, L. 1998. Crown Gall of Grape, Biology of Agrobacterium vitis and the development of disease control strategies. Plant Dis., 82: 1288-1297.

Burr, TJ. and Otten, L. 1999. Crown gall of grape: biology and disease management. Annu. Rev. Phytopathol., 37: 53-80.

Coertze, S., Holz, G. and Sadie, A. 2001. Germination and establishment of infection on grape berries by single airborne conidia of *Botrytis cinerea.* Plant Dis., 85:668-677.

Doores, S. 1990. pH control agents and acidulants. In: "Food Additives" (eds. Branen, A.L., Davidson, P.M. and Salminen, S.). Marcel Dekker, Inc. New York and Basel, pp. 477-510.

Doster, M.A. and Schnathorst, W.C. 1985. Comparative susceptibility of various grapevine cultivars to the powdery mildew fungus *Uncinula necator*. Am. J. Enol. Vitic., 36:101-104.

Duncan, R. 1999. Managing bunch rot. Fruit Grower, April, pp. 260, and 26p.

Eastwell, K.C., Willis, L.G. and Cavileer, T.D. 1995. A rapid and sensitive method to detect *Agrobacterium vitis* in grapevine cuttings using the polymerase chain reaction. Plant Dis., 79:822-827.

Ehret, D.L., Zebarth, B.J., Portree, J. and Garland, T. 1998. Clay addition to soilless media promotes growth and yield of greenhouse crops. HortScience, 33:67-70.

Ehret, D.L., Koch, C., Menzies, J., Sholberg, P. and Garland, T. 2001. Foliar sprays of cay reduce the severity of powdery mildew on long English cucumber and wine grapes. HortScience, 36: 934-936.

Elmer, P.A.G. and Köhl, J. 1998. The survival and saprophytic competitive ability of the . *Botrytis* spp. antagonist *Ulocladium atrum* in lily canopies. Eur. J. Plant Pathol., 104:435-437.

Falk, S.P., Gadoury, D.M., Pearson, R.C. and Seem, R.C. 1995. Partial control of grape powdery mildew by the mycoparasite *Ampelomyces quisqualis.* Plant Dis., 79:483-490.

FAO/WHO. 1973. Toxological evaluation of certain food additives with a review of general principals and specifications. 17th Rep. Joint Food Agric. Org. unit Nat./World Health Org. Expert Commit. Food Addit. WHO Tech. Report Ser. No. 539. FAO Nutrition Meet. Rep. Ser. No. 53.

Farr, D.F., Bills, G.F., Chamuris, G.P. and Rossman, A.Y. 1989. Fungi on plants and plant products in the United States. APS Press, The American Phytopathological Society, St. Paul, Minnesota, USA.

Forney, C.F., Rij, R.E., Denis-Arrue, R. and Smilanick, J.L. 1991. Vapor phase hydrogen peroxide inhibits postharvest decay of table grapes. HortScience, 26:1512-1514.

Gadoury, D.M. 1993. Integrating management decisions for several pests in fruit production. Plant Dis., 77:299-302.

Gadoury, D.M. and Pearson, R.C. 1988. Initiation, development, dispersal, and survival of cleistothecia of *Uncinula necator* in New York vineyards. Phytopathology, 78:1413-1421.

Gadoury, D.M. and Pearson, R.C. 1990a. Germination of ascospores and infection of *Vitis* by *Uncinula necator.* Phytopathology, 80:1198-1203.

Gadoury, D.M. and Pearson, R.C. 1990b. Ascocarp dehiscence and ascospore discharge in *Uncinula necator*. 1990. Phytopathology, 80:393-401.

Gouot, J.M. 1988. Characteristics and population dynamics of *Botrytis cinerea* and other pathogens resistant to dicarboximides. In: "Fungicide resistance in North America" (ed. Delp, C.J.), APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp. 53-55.

Gubler, W.D., Marois, J.J., Bledsoe, A.M. and Bettiga, L.J. 1987. Control of Botrytis bunch rot of grape with canopy management. Plant Dis., 71:599-601.

Gubler, W.D., Ypema, H.L., Ouimette, D.G. and Bettiga, L.J. 1996. Occurrence of resistance in *Uncinula necator* to triadimefon, myclobutanil, and fenarimol in California grapevines. Plant Dis., 80:902-909.

Gullino, M.L. 1992. Control of Botrytis rot of grapes and vegetables with *Trichoderma* spp. In: "Biological control of plant diseases" (eds. Tjamos, E.S., Papavizas, G.C., and Cook, R.J.) Plenum Press, New York, pp. 125-132.

Hilber, U.W. and Schüepp, H. 1996. A reliable method for testing the sensitivity of *Botryotinia fuckeliana* to anilinopyrimidines *in vitro*. Pestic. Sci., 47:241-247.

Hilber, U.W. and Hilber-Bodmer, M. 1998. Genetic basis and monitoring of resistance of *Botryotinia fuckeliana* to anilinopyrimidines. Plant Dis., 82:496-500.

Jacobsen, B.J. and Backman, P.A. 1993. Biological and cultural plant disease controls: alternatives and supplements to chemicals in IPM systems. Plant Dis., 77:311-315.

Kerr, A. 1972. Biological control of crown gall: Seed inoculation. J. Appl. Bacteriol., 35:493-497.

Kim, J., Yousef, A. and Dave, S. 1999. Application of ozone for enhancing the microbiological safety and quality of foods: a review. Journal of Food Protection, 62:1071-1087.

Latorre, B.A., Flores, V., Sara, A.M. and Roco, A. 1994. Dicarboximide-resistant isolates of *Botrytis cinerea* from table grape in Chile: survey and characterization. Plant Dis., 78:990-

994.

Leroux, P. 1996. Recent developments in the mode of action of fungicides. Pestic. Sci., 47:191-197.

Lorenz, G. 1988. Dicarboximide fungicides: History of resistance development and monitoring methods. In: "Fungicide resistance in North America" (ed. Delp, C.J.), APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp.45-51.

Luvisi, D.A., Shorey, H.H., Smilanick, J.L., Thompson, J.F, Gump, B.H. and Knutson, J. 1992. Sulfur dioxide fumigation of table grapes. University of California, Division of Agriculture and Natural Resources, Bulletin 1932, Oakland, California.

MacHardy, W.E. and Gadoury, D.M. 1989. A revision of Mills's criteria for predicting apple scab infection periods. Phytopathology, 79:304-310.

Marois, J.J., Nelson, J.K., Morrison, J.C., Lile, L.S. and Bledsoe, A.M. 1986a. The influence of berry contact within grape clusters on the development of *Botrytis cinerea* and epicuticular wax. Am. J. Enol. Vitic., 37:293-296.

Marois, J.J., Bledsoe, A.M., Gubler, W.D. and Luvisi, D.A. 1986b. Control of *Botrytis cinerea* on grape berries during postharvest storage with reduced levels of sulfur dioxide. Plant Dis., 70:1050-1052.

Masner, P., Muster, P. and Schmid, J. 1994. Possible methionine biosynthesis inhibition by pyrimidinamine fungicides. Pestic. Sci., 42:163-166.

Nair, N.G. 1985: Fungi associated with bunch rot of grapes in the Hunter Valley. Australian Journal of Agricultural Research, 36:435-442.

Nair, N.G. and Nadtotchei, A. 1987. Sclerotia of *Botrytis* as a source of primary inoculum for bunch rot of grapes in New South Wales, Australia. J. Phytopathology, 119:42-51.

Nair, N.G., Emmett, R.W. and Parker, F.E. 1987. Programming applications of dicarboimides to control bunch rot of grapes caused by *Botrytis cinerea*. Plant Pathology, 36:175-179.

Nair, N.G. and Allen, R.N. 1993. Infection of grape flowers and berries by *Botrytis cinerea* as a function of time and temperature. Mycol. Res., 97:1012-1014.

Nair, N.G., Guilbaud-Oulton, S., Barchia, I. and Emmett, R. 1995. Significance of carry over inoculum, flower infection and latency on the incidence of *Botrytis cinerea* in berries of grapevines at harvest in New South Wales. Australian Journal of Experimental Agriculture, 35: 1177-1180.

Neumann, G.L., Winter, E.H. and Pittis, J.E. 1992. Pyrimethanil a new fungicide. The British Crop Protection Council. Brighton Crop Protection Conference: Pests and Deseases, Vols. 1, 2 and 3, British Crop Protection Council, Farnham, England, pp. 395-402.

Northover, J. 1988. Persistence of dicarboximide resistant *Botrytis cinerea* in Ontario vineyards. Can. J. Plant Pathol., 10:123-132.

Northover, J. and Schneider, K.E. 1996. Physical modes of action of petroleum and plant oils on powdery and downy mildews of grapevines. Plant Dis., 80:544-550.

Ophel, K. and Kerr, A. 1990. *Agrobacterium vitis* sp-nov for strains of *Agrobacterium* biovar-3 from grapevines. Int. J. Syst. Bacteriol., 40:236-241.

Palou, L., Smilanick, J.L., Crisosto, C.H. and Mansour, M. 2001. Effect of gaseous ozone exposure on the development of green and blue molds on cold stored citrus fruit. Plant Dis., 85:632-638.

Pearson, R.C. 1986. Fungicides for disease control in grapes. In: "Fungicide chemistry advances and practical applications" (eds. Green, M.B. and Spilker, D.A.) ACS Symposium Series 304, American Chemical Society, Washington, DC, pp. 145-155.

Pearson, R.C. 1988. Powdery mildew. In: "Compendium of grape diseases" (eds. Pearson, R.C. and Goheen, A.C.) APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp. 9-11.

Pearson, R.C. and Gadoury, D.M. 1987. Cleistothecia, the source of primary inoculum for grape

powdery mildew in New York. Phytopathology, 77:1509-1514.

Pearson, R.C. and Goheen, A.C. 1988. Introduction. In: "Compendium of grape diseases" (eds. Pearson, R.C. and Goheen, A.C.) APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp. 1-2.

Reynolds, A.G, Sholberg, P.L. and Wardle, D.A. 1992. Canopy manipulation of Okanagan Riesling vines for improvement of winegrape quality. Can. J. Plant Sci. 72:489-496.

Reynolds, A.G., Veto, L.J., Sholberg, P.L., Wardle, D.A. and Haag, P. 1996. Use of potassium silicate for the control of powdery mildew [*Uncinula necator* (Schwein) Burrill] in *Vitis vinifera* L. cultivar Bacchus. Am. J. Enol. Vitic., 47:421-428.

Sall, M.A. and Teviotdale, B.L.1982. Powdery mildew. In: *Grape Pest Management* (ed. D.L. Flaherty), Publication No. 4105, University of California, Berkeley, California, pp. 46-50.

Sall, M.A., Teviotdale, B.L. and Savage, S.D. 1982. Bunch rots. In: "Grape Pest Management*"* (ed. Flaherty, D.L.), Publication No. 4105, University of California, Berkeley, California, pp. 51-56.

Sarig, P., Zahavi, T., Zutkhi, Y., Yannai, S., Lisker, N. and Ben-Arie, R. 1996. Ozone for control of post-harvest decay of table grapes caused by *Rhizopus stolonifer*. Physiological and Molecular Plant Pathology, 48:403-415.

Savage, S.D. and Sall, M.A. 1983. Botrytis bunch rot of grapes: The influence of selected cultural practices on infection under California conditions. Plant Dis., 67:771-774.

Sholberg, P. 2001. Postharvest handling of pome fruits, soft fruits, and grapes.Agriculture Canada Publication 1768E, Ottawa, Canada. Electronic publication: http://res2.agr.gc.ca/ parc-crapac/ pubs/index_e.htm.

Sholberg, P.L., Reynolds, A.G. and Gaunce, A.P. 1996. Fumigation of table grapes with acetic acid to prevent postharvest decay. Plant Dis., 80:1425-1428.

Sholberg, P.L. and Eastwell, K.C. 1997. *Nicotiana glauca* as a model system for screening bacteria for biological control of *Agrobacterium vitis*. Phytopathology, 87:S90.

Sholberg, P.L. and Bedford, K.E. 1999. Use of cyprodinil for control of *Botrytis cinerea* on apples. Phytopathology, 89:S72.

Smith, C.M. 1988. History of benzimidazole use and resistance. In: "Fungicide resistance in North America*"* (ed. Delp, C.J.), APS Press, The American Phytopathological Society, St. Paul, Minnesota, pp.23-24.

Snowdon, A.L. 1990. A color atlas of post-harvest diseases and disorders of fruits and vegetables, Volume 1:General introduction and fruits. CRC Press, Inc. Boca Raton, Florida, pp.255-266.

Staphorst, J.L., vanZyl, F.G.H., Strijdom, B.W. and Groenewold, Z.E. 1985. Agrocin-producing pathogenic and nonpathogenic biotype-3 strains of *Agrobacterium tumefaciens* active against biotype-3 pathogens. Curr. Microbiol., 12:45-52.

Sutton, J.C., Li, D., Peng, G., Yu. H., Zhang, P. and Valdebenito-Sanhueza, R.M. 1997. *Gliocladium roseum* a versatile adversary of *Botrytis cinerea* in crops. Plant Dis., 81: 316-328.

Sutton, T.B. and Jones, A.L. 1981. Apple disease management. In: "Handbook of pest management in agriculture volume III*"* (ed. Pimentel, D.) CRC Press, Inc. Boca Raton, Florida, pp. 257-273.

Thomas, C.S., Gubler, W.D. and Leavitt, G. 1994. Field testing of a powdery mildew disease forecast model on grapes in California. Phytopathology, 84:1070.

Thomson, W.T. 1997. Agricultural Chemicals Book IV – Fungicides. Thomson Publications, Fresno, California..

Vail, M.E., Wolpert, J.A., Gubler, W.D. and Rademacher, M.R. 1998. Effect of cluster tightness on Botrytis bunch rot in six Chardonnay clones. Plant Dis., 82:107-109.

Vail, M.E. and Marois, J. J. 1991. Grape cluster architecture and the susceptibility of berries to *Botrytis cinerea*. Phytopathology 81:188-191.

Weber, E., Gubler, D., Thomas, C. and Derr, A. 1997. Use of a computer-based weather network and powdery mildew risk assessment model reduces fungicide use in the Napa Valley. In: "Proceedings of the fourth international symposium on cool climate enology & viticulture" (eds. Henick-Kling, T. Wolf, T.E. and Harkness, E.M.) Communications Services, New York, pp. 16-18.

Wilcox, W.F., Gadoury, D.M. and Seem, R.C. 1997. Recent findings on control of grapevine powdery mildew. Grape Research News, published by the New York State Agricultural Experiment Station, Vol.8 No.2 Summer, pp. 1-6.

Wilhelm, A.F. 1954. Über die fungizide wirkung des schwefels bei der Oidiumbekämpfung. Weinberg u. Keller, 1:124-129

Ypema, H.L., Ypema, M. and Gubler, W.D. 1997. Sensitivity of *Uncinula necator* to benomyl, triadimefon, myclobutanil, and fenarimol in California. Plant Dis., 81:293-297.

Ypema, H.L. and Gold, R.E. 1999. Kresoxim-methyl modification of a naturally occurring compound to produce a new fungicide. Plant Dis., 83: 4-19.

Guava Diseases - their Symptoms, Causes and Management

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Abstract: Guava (*Psidium guajava* Linn.) an important fruit of subtropical countries is affected by about 177 pathogens of which, 167 are fungal, 3 bacterial, 3 algal, 3 nematodes and one epiphyte. Wilt is the most important disease of guava. Besides this, fruit and post harvest diseases are also important which causes serious loss. The fruit diseases are of two types *i.e.* field diseases and post harvest diseases, which develop during transit and storage. Due to it's perishable nature number of pathogens are reported on fruits which causes different types of rots of guava fruits. In the present communication all major diseases are described with their symptoms, causal organisms and disease management practices.

1. Introduction

Guava (*Psidium guajava* Linn.) is an important fruit of subtropical countries. It is hardy crop and is cultivated successfully even in neglected soils. There are number of pathogens, mainly fungal which affect guava crop besides few bacterial, algal and some physiological disorders or deficiencies. About 177 pathogens are reported on various parts of guava plant or associated with guava, fruit of which, 167 are fungal, 3 bacterial, 3 algal, 3 nematodes and one epiphyte. About 91 pathogens are reported on fruits, 42 on foliage, 18 on twig, 18 on root and 17 fungi are isolated with surface wash of fruits. These cause various diseases viz. pre and post harvest rots of fruits (dry rots, wet rots, soft rots, sour rots, anthracnose, brown rots, ripe rots, scab, styler end rots, ring rots, pink rots and waxy fruit rots etc.), canker, wilt, die back, defoliation, twig drying, leaf spot, leaf blight, anthracnose, red rust, sooty mould, rust, seedling blight and damping off etc. (Misra and Prakash, 1990). Wilt is one of the most destructive diseases of guava in India and loss due to this disease is substantial. As the disease is of soil borne in nature, there are limitations for the control of this disease. Other important field diseases of guava are anthracnose (*Gloeosporium psidii* Declacr = *Glomerella cingulata*), canker (*Pestalotia psidii* Pat.), and algal leaf and fruit spot (*Cephaleuros virescens* Kuntze) etc. Some twig/stem diseases which are of minor importance are twig blight (*Phomopsis psidii* de Camara), stem canker and die back, drying and defoliation (*Torula* stage of *Hendersonula toruloidea* Nattrass.). There are number of foliar pathogens causing leaf blight and leaf spots. Leaf blight causing pathogens are *Phoma jolyana*, Priozy and Morg*., Alternaria alternata* (Fr.) Keissler, while leaf spot causing pathogens are *Cercospora sawada* Yamamoto, *Pestalotia jodhpurensis* Bilgrami and Purohit,

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Discosia hiptage Lib., *Curvularia siddiqui* Ahmed & Quraishi, and *Pestalotiopsis psidii* (Pat.) Mordue. Leaf fall caused by *Phytophthora hevea*, rust caused by *Puccinia psidii* Wint. and spotted anthracnose caused by *Sphaceloma psidii* are also reported. Among the root diseases, damping off of seedlings caused by *Rhizoctonia solani* Kuhn. and root rot by *Clitocybe tabascens* (Scop.) Bres., are reported. Fruit rots in field (pre harvest rots) or during transit and storage (post harvest rots) are the most serious diseases of guava, which cause maximum loss. It can be said that due to the perishable nature of the fruit and very short self life, guava suffers badly by different rot pathogens. *Phytophthora, Diplodia, Phomopsis, Gloeosporium* and *Guignardia* fruit rots which can be classified as field rots, and further damage fruits during transit and storage. Among the important storage rots, *Gloeosporium psidii*, *Pestalotia psidii*, *Phytophthora nicotianae* var. *parasitica* Dastur, *Macrophoma allahabadensis* Kapoor & Tandon, *Phomopsis psidii*, *Geotrichum candidum* Link ex Pers., *Botryodiplodia theobromae* Pat., *Fusarium solani* (Mart.)App. & Wollenw, *Pestalotiopsis psidii*, *Fusarium moniliforme* var. *intermedium*, *Phytophthora citricola* Sawada cause damage to the fruits in various ways. Diseases due to some of the deficiencies like zinc, magnesium, other nutritional deficiencies are also reported besides physiological disorders like fruit firm rot and internal break down. Thus though the guava is hardy plant, but it suffers by number of important diseases viz. wilt and fruit rots resulting in the high loss of guava production in India and abroad.

2. Wilt

Wilt of guava is one of the most important diseases of guava especially in India and loss due to this disease is substantial. As the disease is soil borne in nature, there are limitations in it's control. Misra and Pandey (1999b) reported that the disease is a challenge for coming millennium as chemical control proved unsatisfactory in field. Although, more than five decades have been passed since the disease was first reported (Das Gupta and Rai, 1947), limited progress has been made in the understanding of the disease. Comprehensive review of work has been done on wilt disease of guava by Prakash and Misra (1993), Misra (1995), Misra and Pandey (1996), Misra (2001) and Negi *et al.* (2001).

2.1 Geographical distribution

In India, wilt of guava was first reported in 1935 from Babakkarpur (Allahabad). Das Gupta and Rai (1947) recorded the disease in a severe form in orchards of Lucknow, while Dey (1948) reported it from Allahabad, Kanpur and Lucknow. During 1949-50, guava trees suffered serious losses in 11 districts of UP (Anonymous, 1949, 1950). Prasad *et al.*, (1952) estimated that guava wilt spread rapidly to cover about 20.000 sq. m area in UP, while, Mathur (1956) was of the view that 15-30 per cent trees in Allahabad, Farrukhabad and Unnao districts, 5-15 per cent in Kanpur and Jaunpur and less than 5 per cent in Gorakhpur, Ballia, Hardoi, Barabanki and Varanasi districts were affected. Edward and Srivastava (1957) reported wilt as the most serious disease threatening guava cultivation in UP. Disease was also reported from western districts of UP (Singh

and Lal, 1953), Varanasi (Pandey and Dwivedi, 1985), Kaimganj (Farrukhabad), Bithoor (Kanpur), Ganga Ghat (Unnao), Abbubakarpur (Allahabad), Lucknow, Bichpuri (Agra), Sasni (Aligarh) (Misra and Prakash, 1986, Misra, 1987). Occurrence of wilt was also reported from guava orchards in West Bengal (Chattopadhyay and Sen Gupta, 1955) where it was confined to mainly in the Gangetic alluvium of Baruipur area in the district of 24 Parganas and in the laterite zone of Jhargram and Midnapur in the districts of Midnapur and of Kashakul in the district of Bankura (Chattopadhyay and Bhattacharjya, 1968a,b). Besides U.P. and West Bengal, the disease has also been reported to occur in Haryana (Suhag, 1976, Mehta, 1987), Punjab (Chandra *et al.*, 1986), Rajasthan (Katyal, 1972), Delhi state (Anonymous, 1953), Bihar (Kumar, 1993) and Andhra Pradesh (Jhooty *et al.*, 1984). Recently the disease was also recorded in severe form from Hatod guava area of Indore (M.P.), India (Personal observation).

From other parts of world too, similar symptoms of wilt or the death of plants have been reported but with different pathogens. Disease has been reported from countries like U.S.A., Florida (Webber, 1928), Taiwan (Hsieh *et al.*, 1976, Leo and Kao, 1979), Cuba (Rodriguez and Landa, 1977), South Africa (Grech, 1985), Brazil (Tokeshi *et al.*, 1980, Rodrigues *et al.*, 1987), Pakistan (Ansar *et al.*, 1994).

2.2 Losses

Singh and Lal (1953) estimated 5-15 per cent losses every year in 12 districts of U.P., which was around Rs. 1 million during 1953. In West Bengal, it reduced the yield by 80 per cent i.e., from 113.5q / ha in healthy plantations to about 18.16-22.7q / ha in affected orchards (Chattopadhyay and Sen Gupta, 1955). Attempt to regenerate the affected trees failed and new seedlings or grafts planted in affected succumbed to wilt within a very short time (Chattopadhyay and Bhattacharjya, 1968a). In Andhra Pradesh, 7,000 acres of land was under guava cultivation and disease reduced the land value to half (Jhooty *et al.*, 1984). About 150 acres of guava orchards in Punjab and 300 acres in Haryana were uprooted during 1978-81 as these were affected by wilt (Jhooty *et al.*, 1984). Assessment of loss around Lucknow revealed that loss vary from 5-60 per cent and above 5 year old guava plants are more prone to wilt incidence (Misra and Shukla, 2002).

2.3 Symptomatology

First external symptom of the disease is the appearance of yellow colouration with slight curling of the leaves of the terminal branches. Plants, at a later stage, show un-thriftiness with yellow to reddish discolouration of leaves. Subsequently, there is pre-mature shedding of leaves. Some of the twigs become bare and fail to bring forth new leaves or flowers and eventually dry up. Fruits of all the affected branches remain underdeveloped, hard and stony. Later on, the entire plant becomes defoliated and eventually dies. Usually, fifteen days are required for complete wilting but some trees take up to one year. The finer roots show black streaks, which become prominent on removing the bark (Das Gupta and Rai, 1947). The roots also show rotting at the basal region and the bark is easily detachable from the cortex. The cortical regions of the stem and

root show distinct discoloration and damage. Light brown discoloration is also noticed in vascular tissues (Chattopadhyay and Bhattacharjya, 1968a,b). The pathogen attacks young as well as old fruit bearing trees but older trees are more prone to the disease. New seedlings and grafts also show disease symptoms (Singh and Lal, 1953 and Edward, 1960b). Mainly two main symptoms are identified: (a) Slow wilt and (b) Sudden wilt (Chakraborty and Singh, 1989).

2.4 Causal organism

The exact cause of the disease is still not fully understood but the pathogens viz. *Fusarium oxysporum* f. sp. *psidii* (Prasad, Mehta & Lal), *F. solani*(Mart.)App. & Wollenw., *Macrophomina phaeseoli* (Maubl.) Ashby, *Rhizoctonia bataticola* (Taub.) Bulter*, Cephlosporium* sp. and *Gliocladium roseum* (Link ex Fr.) and various pathogens are reported by different workers may be the incitant of the disease. Prior to 1941, wilt was considered to be caused by *Cephalosporium* sp. (Vestal, 1941). Dey (1948) also invariably isolated *Cephalosporium* from roots of wilted plants. Das Gupta and Rai (1947) reported the association of *Fusarium* sp. Later, Prasad *et al.* (1952) attributed wilt due to *Fusarium oxysporum* (Fr.) Schl. and proposed the name *Fusarium oxysporum* (Fr.) Schl. f.sp. *psidii* Prasad, Mehta and Lal. It was also supported by Edward and Srivastava (1957) and Pandey and Dwivedi (1985). Edward (1960a) also observed that *F. oxysporum* f. sp. *psidii* exist in a variety of forms, which differ in cultural and morphological characters. Besides the involvement of above pathogens, association of the other pathogens have also been suspected in inducing wilt disease of guava. In West Bengal, both *M. phaseoli* and *F. solani* were reported to incite wilt either individually or in combination.

In either case, the fungus first colonizes the surface of roots and then enters in to it's epidermal cells. There after, intercellular mycelium establishes first in epidermal cells and then spreads into cortical cells which get considerably damaged and filled up with the mycelium. *Fusarium solani* enters the xylem vessels, grows inside and blocks them. *Macrophomina phaseoli* first invades the phloem and destroys it. The xylem vessels are also attacked in a few cases (Chattopadhyay and Bhattacharjya, 1968a,b, Chattopadhyay and Sengupta, 1955). Wilt and dieback of guava are also reported to be caused by *Gloeosporium psidii* Declacr. The fungus penetrate the petiole and attack the young leaves which become distorted with dead areas at the margins or tips and in severe cases, die (Tandon and Agarwal, 1954). *F. oxysporum* f. sp. *psidii* penetrate either directly through the root piliferous layer of the guava seedlings or through openings caused by secondary roots. Hyphae are found in the xylem vessels of the roots of the inoculated plants (Edward, 1960b). Histopathological observations made by various workers in naturally wilted and artificially inoculated plants revealed the presence of *F. solani*, *F. oxysporum* and *M. phaseolina* (Tassi) Goid. in vascular tissues (Chattopadhyay and Bhattacharjya, 1968a,b, Edward, 1960c, Chandra Mohan, 1985, Pandey and Dwivedi, 1985 and Sohi, 1983a,b). *Gliocladium vermoesenii* Corda., a known saprophytic fungus, is also found associated with diseased plants (Chandra Mohan, 1985). From Varanasi *F.oxysporum* f.sp.p*sidii*, *F. solani*, *F. coeruleum*, *F. moniliforme* and *Rhizoctonia solani* Kuehn were also from rhizoplane as well as from

the soil (Dwivedi, 1991a). *Cylindrocarpon lucidum* Wollenw., *Gliocladium virens* and *Bartilinia robillardoides* Tassi. caused drooping and subsequent wilting of guava seedlings grown in Hoagland's solution on artificial testing (Misra and Pandey, 1992).

The reports from the other parts of the world are different. Similar symptoms of wilt are reported but with different pathogens. As early as in 1928, *Clitocybe tabescens* killing guava trees in Florida (USA) was reported (Webber, 1928). In Taiwan, the disease is reported to be caused by *Myxosporium psidii* Corda (Hsieh *et al.*, 1976, Leu *et al.*, 1979), while in Cuba three nematodes viz., *Meloidogyne*, *Helicotylenchus* and *Pratylenchus* have been found associated (Rodriguez and Landa, 1977). In South Africa, *Septofusidium* sp. was found associated with the rapid death of guava plants (Grech, 1985). Tokeshi isolated *Pseudomonas* sp. from wilt affected plants (Tokeshi *et al.*, 1980). Disease similar to wilt caused by *Erwinia psidii* was also observed at Sao Paulo (Brazil) in 1982 (Rodrigues *et al.*, 1987). From Pakistan (Punjab) disease is reported in the name of decline and *Fusarium oxysporum* and *Colletotrichum gloeosporioides* (*Glomerella cingulata*) are considered associated with the disease and are supposed to act synergistically when present together (Ansar *et al.*, 1994). In a recent study Misra and Pandey (1997) and Misra and Pandey (2000a) reported that *Gliocladium roseum* as a most potent pathogen, which reproduces symptom of wilt on artificial inoculation. They also developed an inoculation technique *i.e.* stem hole inoculation technique, which reproduce the wilt symptom very quickly. Misra *et al.* (2003) further reported pathogenic diversity in the cause of wilt disease of guava and advocated that there may be several cause of wilt but *G.roseum* is a very potent as pathogen of guava wilt.

2.5 Epidemiology

The severity of disease is reported high at 40-60 per cent soil saturation with *Fusarium solani* or *Macrophomina phaseoli* individually, while for both 60-80 per cent saturation is reported optimum. The pH 6.0 is reported optimum for the development of the disease, but at pH 4.0 and 8.0 the disease decrease. Incidence of the disease is reported less at 630 ppm N and is reported more both at higher or lower levels. Moderate to high concentrations of phosphates (207-345 ppm) are effective in reducing the attack. Incorporation of organic manure in affected soils reduce the disease and under green manuring there is no incidence (Chattopadhyay and Bhattachrjya, 1968b). Earlier Mehta (1951) reported severe incidence of wilt in alkaline soils at pH ranging from 7.5 to 9.0. However, high disease incidence is recorded in lateritic soils at pH 6.5 (Sen and Verma, 1954). Mehta (1987) reported more disease in clay loam and sandy loam as compared to other soil types. The wilting is observed during rainy season. It starts in August with the largest number of plants dying in September and October. The incidence of decrease is markedly reduced in November and it become negligible with advancing winter (Das Gupta and Rai, 1947). Edward (1960c) observed that the disease starts in the beginning of June but the intensity of infection and spread increases with the onset of monsoon.

The highest wilting of guava trees is, however, restricted to the month of September and October, beyond which the incidence reduces gradually. Suhag (1976) also reported that soon after rainy season, in September-October, the entire tree dries up

within a period of 3-4 weeks. But some times due to unknown reasons decline and death of tree is slow and taking 6-8 months for complete collapse. Dwivedi *et al.*, (1990) also found more pathogenic fungi during rainy and winter season surviving better in association with root bits. Studies on progressive natural wilting of guava plants during different months were done by Misra and Pandey (1999a,d), Misra and Pandey (2000b) revealed that maximum wilting takes place during the month of October. Some plants, which show slight yellowing but resist wilting, start recovering from December onwards. On analyzing the weather data, it was revealed that higher rainfall during July-September, maximum temperature around 31.3-33.5^{0}C and minimum temperature around 23-25^{0}C with high humidity of 76 per cent were conducive for wilt incidence. They further found that generally two months are required for the complete wilting of plants after the appearance of first visible symptom but some times only 16 days were required for complete wilting.

2.6 Varietal reaction

Cultivar, Chittidar, Hafsi, Safeda Riverside, Rolf and Stone acid were reported susceptible and *Psidium cattleianum* var. *lucidium* and *Syzigium cumini* (Jamun) resistant to wilt (Edward, 1961). Varieties, white guava No. 6229, Clone.32-12, Webber and Popeno from Florida (USA), Hart and Rolf from Florida but acclimatized at Allahabad, Riverside and Rolf from California (USA), Safeda from Sri Lanka, Banarasi (Andhra strain), Dholka, Sindh and Nasik (Bombay strain) were reported tolerant to wilt disease (Mathur and Jain, 1960). In Taiwan, a local variety Pei-pa has been reported resistant and *Psidium friedrichsthlianum* has been recommended as possible root stock (Leu and Kao, 1979). Singh *et al.*, (1977) reported that among 10 red-fleshed cultivars, only one of Allahabad was found infected by *Fusarium solani*. Among the 15 white-fleshed cultivars, Lucknow 49 was free from the disease and in Allahabad Safeda incidence was only 4 per cent, whereas Kerala and Behat Coconut suffer heavily (33%). None of the species, *Psidium aracae*, *P. cattleianum*, *P. cattleianum* var. *lucidium*, *P. corecium*, *P. cujavillus*, *P. quineese* and *P. fridichsthalanum* developed wilt infection. Edward and Gaurishanker, (1964) reported that *Syzigium cuminii*, *Psidium molle*, *P. quianense*, Chinese guava (*P. friedrichsthalianum*) and Philippine guava resistant to wilt organism. The strawberry guava (*Psidium cattleianm*) is reported relatively hardy species from Reunion (Normand, 1994). In South Africa, Fan Retief, a most extensively cultivated guava variety is highly susceptible to wilt (Preez , 1995).

2.7 Disease management

Time to time recommendations for the control of wilt has been given by different scientists. These are summarized as below :
Disease can be controlled by proper sanitation in the orchard. Wilted trees should be uprooted, burnt and trench should be dug around the tree trunk (Mathur, 1956). While transplanting, roots of plants should not be severely damaged. Maintenance of proper tree vigour by timely and adequately manuring, inter-culture and irrigation enable them

to withstand infection. The pits may be treated with formalin and kept covered for about 3 days and then transplanting should be done after two weeks (Edward, 1960c).

Symptoms of the disease do not appear under green manuring and the disease development is less when organic sources of nitrogen are used (Chattopadhyay and Bhattacharjya, 1968b). Organic manures, oil cakes and lime also check the disease (Das Gupta and Ghoshal, 1977). Spread of wilt could be checked by judicious amendments of N and Zn (Suhag and Khera, 1986).

Good control of wilt was achieved by the application of Chaubatia paste consisting of 4-oz red lead, 4-oz $CuCO_3$ and 5-oz lanolin (Anonymous,1949). Injection of 0.1 per cent water soluble 8-Quinolinol sulphate was found to have chemotherapeutic action against the wilt pathogen (*Fusarium oxysporum* f. *psidii*). Injection in apparently healthy guava plants in a diseased area with 0.1 per cent 8- Quinolinol sulphate provided protection against wilt for at least one year. When injected into slight wilted plants, it was beneficial for their partial recovery (Jain, 1956). Both at Allahabad and Lucknow, the wilt was controlled by soil treatment with 1.82 kg. lime or gypsum/tree, although the control mechanism was not well understood (Mathur *et al.*, 1964). Suhag, (1976) reported that it is possible to regenerate even the worst affected trees by severe pruning followed by a drench with 0.2 per cent either Benlate or Bavistin 4 times in a year and spraying twice with Metasystox and Zinc sulphate. Dwivedi, (1991b) reported that heavy metal like Hg, Cd and Cu effectively check the growth of *F. oxysporum* f.sp. *psidii.*

In Taiwan, Carbendazim (50%), Captafol (30%) and Thiabendazole proved effective against *Myxosporium psidii* under laboratory experiments but failed *in vivo* (Leu *et al.*, 1979). Disinfection of soil was achieved with DBCP at 52.8ml /10m^2 or Metham sodium at 252.5ml/10m^2 and control nematodes (Rodriguez and Landa, 1977). In Pakistan decline of guava is supposed to be caused by *Fusarium oxysporum* and *Colletotrichum gloeosporioides* and was found to be controlled by combined use of Topsin M sprays and the antagonists *Trichoderma harzianum* and *Arachniotus* sp. added in soil amended with wheat straw (Ansar *et al.*, 1994).

Use of rootstocks resistant to wilt disease could be an effective method for control of wilt. *Psidium cattleianum* var. *lucidium* and *Syzigium cuminii* (Jamun) seldom get attacked by the disease. Since interspecies and intergeneric graft compatibility is there, it may be an effective way for the control of wilt disease (Edward, 1961). Misra and Pandey (1999c) reported that though different fungicides are effective for the control of wilt pathogens in lab, but these pathogens increases it's aggressiveness, once the effect of these fungicides diminishes in soil. Eco-friendly approach of guava wilt control was suggested by Misra *et al.*, (2001) where biological control, soil amendment and intercropping were suggested. Biological control by *Aspergillus niger* strain AN17 (Misra *et al.*, 2000) and by *Penicillium citrinum* (Prakash *et al.*, 2002) are effective.

3. Anthracnose

The disease is serious problem in Uttar Pradesh, Punjab and Karnataka. It was first reported by Mehta during the year 1951 from Uttar Pradesh. In Karnataka disease was reported by Rawal during 1993 (Rawal, 1993). It causes die back, twig blight, wither tip

and fruit spots. Die back phase is caused by *Gloeosporium psidii* resulting in the death of plants was observed at Allahabad (Tandon and Agarwal, 1954) but this phase was not observed at Saharanpur (Tandon and Singh, 1969). The disease is reported from Taiwan also (Yang and Chuang,1994).

3.1 Causal organism

Gloeosporium psidii Delacroix=*Glomerella psidii* (Del.) Sheld. / *Colletotrichum psidii* Curzi.The disease is caused by *Gloeosporium psidii* Delacroix =*Glomerella psidii* (Del.) Sheld. It is now called*Colletotrichum psidii* Curzi.

Brown to dark brown coloured acervuli are formed on the affected parts of the plants. Setae and conidia are formed in the acervuli. Mycelium is intercellular, branched and light brown in colour. Conidiophores are hyaline and small, setae are long, tapering at the end, dark brown to black in colour. Conidia are formed at the tip of the conidio-phores and are sickle-shaped, unicellular, hyaline measuring 11.24 x 4.5-5µ m. They germinate by germ tube. In moist weather, acervuli appear as black dots on twigs or fruits, which later produce pinkish spore mass. Spores are disseminatedby wind or rain and initiate fresh infection.

3.2 Die back phase

Die back phase of anthracnose was reported from Minto Park, Allahabad, in 1952 and the intensity of the disease varied from half dead to complete dead plant (Tandon and Agarwal, 1954).

3.2.1 Symptomatology

The plant begins to die backwards from the top of a branch. Young shoots, leaves and fruits are readily attacked, while they are still tender. The greenish colour of the growing tip is changed to dark brown and later to black necrotic areas extending backwards causing the die back. The disease is more noticeable after a period of incubation in the infected buds and twigs. The brown spots, formed previously, change into silvery grey and ultimately develop at the junction of the diseased and healthy parts.

The fungus develops from the infected twigs and then petiole and young leaves are attacked. These may droop down or fall leaving the dried twigs without leaves. In moist condition, acervuli of the fungus may be seen as black dots scattered throughout the dead parts of the twigs (Tandon and Agarwal, 1954).

3.2.2 Disease development

After slight rain, old twigs show the symptoms of die back. In the first fortnight of August the disease causes death of small, tender growing twigs. From August to October the infected twigs wither and shrivel at their tips. The disease appears in epidemic form, during August to September. It is also noticed in December after which the older leaves and twigs remain immune from attack. During less humid weather

conditions *i.e.* from January to June further progress of the disease is not there (Tandon and Agarwal, 1954).

3.2.3 Pathogenicity

In rainy season symptoms develop after 15-20 days of inoculation. In pathogenicity test inoculation without injury give negative result. In summer, even after injury, symptom development fails. Thus, humidity is necessary for the growth of the organism. Old branches resist infection. The young leaves show dead area on the margin and the tips, which generally appear after one week of inoculation. When flowers are inoculated, unopened buds show infection within two or three days and fail to develop. Petals turn brown and buds fall off without opening. On inoculation of fruits, rot develop in the ripened fruits but they never develop mummy (Tandon and Agarwal 1954).

3.2.4 Control

Although complete control is not possible, the application of 3:3:50 Bordeaux mixture and 0.22 or 0.33 per cent Perenox give encouraging results in reducing the development of die back and mummies (Tandon and Agarwal, 1954).

3.3 Fruit and leaf infection phase

Fruit and leaf infection was reported from Saharanpur and it is generally seen in rainy season crop.

3.3.1 Symptomatology

Pin-head spots are first seen on the unripe fruits which gradually enlarge measuring 5-6 mm in diameter. They are dark brown to black in colour, sunken, circular and has minute black *stromata* in the center of the lesion which produce creamy spore masses in moist weather. Several spots coalesce to form bigger lesions. The infected area on the unripe fruits become corky and hardy, and often develop cracks in case of severe infection (Fig.1). On ripe fruits, the infection causes softening of tissues and lesions attain a diameter of 10 to 20mm (Tandon and Singh, 1969). Unopened buds and flowers are also attacked and cause their shedding. Spread of infection is very rapid on fully matured green fruits, whereas, young fruits do not normally get infected (Midha and Chohan, 1968) perhaps owing to the differences in the concentration of K- ions in the fruit tissues. On leaves, the fungus causes necrotic lesions at the tip or on the margin. These lesions are usually ashy grey and bear fruiting bodies. The tender twigs are also infected, which wither and die from the tip downwards giving it a wither tip appearance (Tandon and Singh, 1969).

3.3.2 Pathogenicity

The leaves and stems of seedlings and new leaves, buds, flowers and fruits of grown

plants are readily infected. Tandon and Agarwal (1954) were unable to infect the green fruits under artificial conditions and were of the opinion that the fungus remains latent for two and half to three months and spots appear when fruits ripen. According to them the hard tissues of young fruits prevent the fungus from growing but Tandon and Singh (1969) were able to infect both the young and ripe guava fruits. The disease was found to develop more rapidly at 30^0C and 96.1 per cent relative humidity both in ripe and unripe fruits.

Figure 1: Anthracnose of guava fruit

3.3.3 Varietal resistance

Apple guava (deep red fleshed), Apple shaped seedling, Behat Coconut, Red Chittidar, Muzaffarnagar, Bulandshahar, Lucknow-49 and species *Psidium chinensis* Lodd., *P. catteleianum* var. *lucidium* Isab. *P. quianense* and *P. molle* Bertol are susceptible to anthracnose while Apple guava (light red fleshed) has moderate resistance (Tandon and Singh, 1969). *P. chinensis* resist leaf infection whereas *P. molle* and Beumont are highly susceptible and Allahabad Safeda develop heavy infection on fruits (Anonymous, 1974).

3.3.4 Control

Effective control of anthracnose can be achieved by sprays of Bordeaux mixture (3:3:50) at 7 days interval. Copper oxychloride and cuprous oxide also significantly control thedisease (Tandon and Singh, 1969) but Bordeaux mixture and other copper fungicides cause russeting of fruits especially in cv. Allahabad Safeda and reduce their market value (Sohi and Sridhar, 1969). Monthly sprays of Difolatan (0.3%) and Dithane Z-78 (0.2%) are effective in controlling the disease (Anonymous, 1974). For post harvest treatment 20 min. dip treatment in 500 ppm tetracycline is effective (Gupta *et al.*, 1973). Pre treatment of sodium metabisulphite and stable bleaching powder is also effective under *in vivo* conditions (Singh and Sharma, 1982). Thiabendazole and Aureofungin are also effective (Sharma *et al.*, 1983). Growth and acervulus formation are inhibited by Thiophenate methyl, Benomyl and Thiabendazole at 5-50 ppm. Vitigram blue check acervulus formation at 50 ppm and hyphae is restricted by boric acid (Butt *et al.*, 1995).

4. Canker

Fruit canker caused by *Pestalotia psidii* Pat. was recorded from Bombay (Chibber, 1911) and later from Mysore (Narsimhan, 1938, Venkatakrishniah, 1952), Thane, Dharwar,Poona (Patel *et al.*, 1950), Ponta Valley, Himachal Pradesh (Verma and Sharma, 1976) and Lucknow (Misra and Prakash, 1986).

4.1 Symptomatology

The disease generally occurs on green fruits and rarely on leaves. The first evidence of infection on fruit is the appearance of minute, brown or rust coloured, unbroken, circular, necrotic areas, which in advanced stage of infection, tears open the epidermis in a circinate manner. The margin of lesion is elevated and a depressed area is noticeable inside. The crater like appearance is more noticeable on fruits than on leaves. The canker is confined to a very shallow area and does not penetrate deep into the flesh of the fruit (Fig.2). In older cankers, white mycelium consisting of numerous spores are noticeable. Canker on the green fruits of different varieties exhibit considerable differences in their appearance (Patel *et al.*, 1950). In severe cases, raised, cankerous spots develop in great numbers and the fruits break open to expose seeds. The infected fruits remain underdeveloped, become hard, malformed and mummified and drop in great numbers. Sometimes small rusty brown angular spots appear on the leaves (Venkatakrishniah, 1952). In winter the cankerous spots are common but in rainy season minute red specks are formed (Verma and Sharma, 1976).

4.2 Causal organism

Fruit canker is caused by *Pestalotia psidii* Pat. (Chibber, 1911). Narsimhan (1940) and Venkatakrishniah (1952) found *Colletotrichum psidii* Curzi, *Glomerella psidii* (Del.) Sheld. and *Pestalotia psidii* Pat. associated with canker. Venkatakrishniah (1952) advocated that *Colletotrichum psidii* is a general parasite and *Pestalotia psidii* is a special-

ized to guava. Both species are present on young green and mature fruits/leaves but *Pestalotia psidii* is considered the real cause of canker (Patel *et al.*, 1950). Pathogen develops dark black and circular pycnidia on the culture media and fruits, and these contain conidiophores and conidia. The conidia are typically 5-celled, oblong, clavate or elliptic-fusoid, erect, hardly constricted at septa, measuring 13-31 x 5-10 μm, 3 medium cells are guttulate, highly brownish; the central cell being the thickest and greatly bulged and other cells are comparatively hyaline; the apical conical or cylindrical cell grows out into 3 hyaline, slender, elongated appendages; the basal cell is obtuse, erect with a small pedicel. The mycelium of young culture is sub-aerial, serrate, thin, septate, cottony white to pinkish, irregularly branched and measuring upto 3 μm in diameter. In old cultures, the hyphae are more or less thickened (Patel *et al.*, 1950).

Figure 2: Fruit canker caused by *pestalotia psidii* Pat.

4.3 Epidemiology

The maximum disease occurs at 25-30^{0}C and at high RH (Kaushik *et al.*, 1972). The fungus grows, profusely on different media but scanty vegetative growth occurs on green guava decoction. Sporulation is quick and abundant on PDA and Richard's medium. The pathogen grows vigorously at temperature between 15-30^{0}C. Best growth and sporulation is seen at 26^{0}C. The fungus grows profusely on media containing mannitol, dextrin and sucrose, fairly on maltose and salicin, poorly on ripe guava decoction, glycerol and Richard's modified agar. Best sporulation is in mannitol, dextrin and sucrose. With different sources of nitrogen, the growth characters of *P. psidii* remain

practically constant but the sporulation is profuse in case of potassium nitrate and aspargin. The fungus grows in a wide range of H-ion concentrations, but optimum pH is 3.9-4.9 with maximum growth at pH 4.9. The spores of the fungus germinate at 10^0C and increase with rise in temperature up to 32^0C. Maximum growth is on sulphates or cystein (Tandon, 1950). On thio-urea, growth is moderate but sulphide, dithionate and persulphate are only feebly utilized. Addition of green fruit tissues and it's decoction give greater stimulation to germination than the ripe fruit tissues. The fungus remains viable in conidial stage upto 38.8^0C (Patel *et al.*, 1950). Germination of spores of *Pestalotia psidii* is maximum at 30^0C and it do not germinate below 15^0C or above 40^0C (Ramaswamy *et al.*, 1984). The best germination medium is guava fruit extract. High RH (98%) is required for germination.

4.4 Pathogenicity

Detached fruits, both ripe and unripe fail to reproduce symptoms. Fresh, young, green guava fruits (undetached) inoculated after wounding give typical severe symptoms, while in unwounded fruits symptom development take more time. Artificial infection on leaves is generally unsuccessful. The pathogen is primarily a wound parasite (Patel *et al.*, 1950).

4.5 Varietal resistance

In cv. Lucknow 49, development of canker pustule is large, more elevated and numerous. On cv. Dhokla it is not well developed. On cv Sindh, the development of pustule is insignificant and inconspicuous, while cultivar Nasik is almost immune (Patel *et al.*,1950). Safeda and Apple coloured are highly resistant cultivar to canker.

4.6 Control

The spread of disease (in early stage of infection) is controlled by 3 to 4 spraying of 1 per cent Bordeaux mixture or lime sulphur (1 in 25) at 15 days interval (Venkatakrishniah, 1952). Homeopathic drugs, Kali (Potassium iodide) at potencies of 1, 20, 24, 61 and 67; Arsenic album (Arsenic oxide) at potencies of 60, 65 and 82 completely inhibit spore germination in *in vitro* test. Potassium iodide at potencies of 1, 20, 24 and 61 and Arsenic oxide at potency of 60 inhibit growth of the pathogen. Fruit treated with some of the effective potencies before inoculation do not develop rotting (Khanna and Chandra, 1977). Leaf extract of *Azadirachata indica* and *Ocimum sanctum* inhibit the germination of spores *in vitro test*. Dipping of guava fruits in these extracts before or after inoculation is effective. Use of *O. sanctum* extract is recommended, as it does not affect fruit flavour (Pandey *et al.*, 1983).

5. Algal leaf and fruit spot

The alga causes spots on leaves and fruits and thus reduces the photosynthetic activity of the plant. The disease does not cause severe economic loss. Ruehle (1941) re-

ported *Cephaleuros virescence* Kuntze., on leaves, fruits, twigs and bark of guava from Florida. Although twig and bark infections are of little or no consequence in Florida but leaves and fruits are severely attacked. Thirumalachar (1945) collected *Cephaleuros parasiticus* from Mysore, which is responsible for blemishes of guava fruits. Yadav (1953), reported *Cephaleuros* sp. on guava from Patna (Bihar). Misra and Prakash (1986), found that the *Cephaleuros virescence* affect guava leaves in areas of Lucknow and Sitapur (U.P.) and the incidence was found as high as 30 per cent.

5.1 Symptomatology

Cephaleuros infects immature guava leaves during early spring flush. Minute, shallow brown lesions appear on leaves, and as the disease progress, the lesions enlarge to 2-3 mm in diameter. On leaves the spots may vary from specks to big patches. They may be crowded or scattered. Leaf tips, margins or areas near the mid vein are most often infected. Although the alga's zoosporangia arise from thalli on adaxial surfaces of many host species, lesions on guava frequently extend through the entire lamina and sporangia most often occur on the abaxial leaf surface. On immature fruits the lesions are nearly black. As fruits enlarge, lesions get sunken. Cracks frequently develop on older blemishes as a result of enlargement of fruits. Penetration of fruit is confined to several layers of cells beneath the epidermis. Fruit lesions are usually smaller than leaf spots. They are darkish green to brown or black in colour (Ruehle, 1941; Marlatt and Campbell, 1980 a,b).

5.2 Time of infection

Disease begins to appear from April and is more serious during May to August. In October disease is difficult to find (Ruehle, 1941). The pathogen sporulates readily during the period of highest rainfall (July-September) and the disease incidence is greatest during September. In winter, symptom is not available (Marlatt and Campbell, 1980 a, b).

5.3 Causal organism

The alga, *Cephaleuros virescens* Kuntze. (*C. mycoidae* Karst.) occurs on wide range of plants in South Florida. Nineteen species are listed as it's host in the vicinity of Homestead, Florida in 1936 (Ruehle, 1936). Ruehle (1941), reported algal leaf and fruit spot caused by *Cephaleuros virescens* Kunze (*C. mycoidea* Krast) while Thirumalachar (1945) reported that it is caused by *C. parasiticus*. Yadav (1953), Marlatt and Campbell (1980 a, b) reported it to be caused by *Cephaleuros* spp.

The pathogen extends itself between cuticle and epidermis and penetrate the epidermal cells. The affected cells eventually die. It is also called red rust because of the upper surface of the thallus produces erect, yellow to red filaments and fruiting bodies. These are flat, short, closely crowded branched filaments, beneath which, there are irregularly branched rhizoid. Most obviously fruiting bodies are consist of upright multi-cellular filaments bearing 1-8, sharply bent pedicels. Each pedicel bears a pear

shaped or nearly spherical sporangium, which eventually emits about 8-32 motile bi-flagellate spores.

On guava, contrary to other hosts, no thallus is apparent on the upper leaf surface, the lesions extend through the entire lamina soon after it appears and the sporulation occurs on the lesion surface on the underside of the leaf (Marlatt and Campbell 1980 a). The alga sporulates readily during the period of greatest rainfall.

5.4 Change in physiology

Among sugars, glucose and sucrose are less while fructose is more in alga infected leaf tissues. Cellulose and pectin are also more in infected tissues while lignin content is unaffected (Vidyasekaran and Parambaramani, 1971b). Vidyasekaran and Parambaramani (1971a) further noted that algal infection causes reduction in the total protein, ammonical nitrite, amino and amide nitrogen content of guava leaves. Glutamic acid, alanine increases considerably while glycine decreases sharply. The valine content is not affected by algal infection. In further studies they found that algal infection increases transpiration and decreases water content. While potassium and phosphorus content is less in infected tissues, sodium accumulate in them. Magnesium content slightly increase while sulphur content decreases due to infection. Among the trace element, iron and manganese accumulate while copper decrease in diseased tissues (Vidyasekaran and Parambaramani, 1972).

5.5 Varietal reaction

Large fruit peruvian variety of Florida is highly susceptible (Ruehle, 1941). The cultivars Patillo and Blitch are low disease cultivars, Ruby x Supreme 6-29 moderate disease cultivars, Webber x Supreme and Ruby x Supreme 10-30 high disease cultivar (Marlatt and Campbell, 1980a, b).

5.6 Control

The control of alga can be achieved by spray of copper oxychloride (0.3%) 3-4 times at the interval of 15 days. Spray of copper oxychloride in rainy season is more effective (Ruehle, 1941). An ascomycetous parasite closely resembling Strigula astridiza on Cephaleurous parasiticus can be used for controlling disease biologically (Thirumalachar, 1945).

6. Stem canker / bark canker

Stem canker of guava was reported from Patharchatta, Nainital (Rana, 1981).

6.1 Symptomatology

Initial symptoms of the disease are longitudinal cracks in the bark on stem or branches, which are visible during post monsoon period in October-November. Usually the dis-

ease originates from the injured bark. On scraping bark, brown to black streaks or bands are present in the sub-cortical region. The affected bark turns dark brown to greyish and develops large vertical cracks.

The disease spreads up and down from one branch to another and ultimately passes on to the main trunk and upper roots. Leaves on the engirdled portions loose their colour slowly and become purplish bronze. Completely engirdled trees decline and die gradually in course of 2-3 years.

6.2 Causal organism

The pathogen is identified as *Diplodia natalensis* Pole-Evans. Pycnidia are grey to black, erumpent, globoid to irregular, ostiolate and measure 160-600 μm in diameter. The pycnidiospores are at first hyaline, later become brown, uniseptate and striate. They are oblong to elliptical and measure 19.2-32.0 x 11.8-16.0 μm (av. 27.1 x 14.8 μm) (Rana, 1981). The bark canker disease of guava has been reported from other parts of India also but the fungus causing disease is reported as *Physalospora psidii*. Uppal (1936) observed *Physalospora psidii* in Dholka, North Gujarat and Bombay Presidency causing a severe disease of guava. Infection originates in the bark and spreads rapidly along the stem from one branch to another resulting in desiccation, cracking, decortication and death of the affected part, and finally of the whole tree. Numerous perithecia of the causal organism are scattered over the dead bark.

7. Leaf blight

The disease was first reported by Sridhar and Ullasa in 1978 although it was observed during 1975 from Hessarghatta, Bangalore, India.

7.1 Symptomatology

First symptom of blight is the appearance of small circular spots, with dark brown center, surrounded by a reddish margin. In advance stages, these spots gradually enlarge and coalesce resulting in large necrotic patches and cause blightening. The fruiting bodies (pycnidia) of the fungus appear in large numbers in the form of small, light brown to black pin heads on the necrotic spots. Both young and old leaves are susceptible to the infection and severely affected plants are completely defoliated (Sridhar and Ullasa, 1978).

7.2 Causal organism

The pathogen was identified as *Phoma jolyana* Priozy and Morg. (Sridhar and Ullasa, 1978).

8. *Cercospora* leaf spot

Leaf spot of guava caused by *Cercospora sawada* Yamamoto, was recorded from

Mauritius (Anonymous, 1965), Nainital, India (Bose and Muller, 1967) and Annamalai University (T.N.) (Raghunathan and Prasad, 1969). The disease is severe during December to February.

8.1 Symptomatology

The disease appears as water soaked, brown irregular patches on the lower surface and yellowish on the upper surface of the leaf. Old leaves are mostly and severely affected, later curl and subsequently drop off (Raghunathan and Prasad, 1969).

8.2 Causal organism

The disease is caused by *Cercospora sawadae* Yamamoto. Conidia are sub-hyaline to pale olivaceous, slightly curved, obclavate to obclavate cylindric, 1-5 septate, base obconic, tip obtuse, measuring 3-4.2 x 18-87.4 mm. Mycelial patches are hypohyllus, fruiting effuse, irregular, stromae lacking, conidiophores non fasciculate, arise singly from hyphal threads, straight or slightly curved, geniculate, 2-5 septate, pale to olivaceous brown, tip conic measuring 3-3.7 x 46 77µm (Raghunathan and Prasad, 1969).

8.3 Varietal resistance

The cultivars Red flesh, White large, Lucknow and 'Chittidar' are susceptible while the cultivars Safeda and Seedless are resistant (Raghunathan and Prasad, 1969).

9. *Pestalotia* leaf spot

A new pathogenic species of *Pestalotia i.e. Pestalotia jodhpurensis* sp. nov. Bilgrami and Purohit, which causes leaf spot of guava was reported by Bilgrami and Purohit (1971).

9.1 Symptomatology

The spots originate from the tips or the margins and gradually advance towards the base, assuming dark brown grey colour. Fruiting pustules develop profusely on the upper surface of the leaves. The pathogen is found constantly associated with such spots.

9.2 Causal organism

This leaf spot is caused by *Pestalotia jodhpurensis*. The fruiting pustule of typical pycnidium is amphigenous, globoid to ovoid, dark brown, occasionally with prominent neck, ostiolate, 167-389 µm in diameter; conidia 5 celled, narrowly elliptical, slightly constricted at septa, 17.55-24.30 x 3.51-5.40 µm, 3 intermediate coloured cells light olive, concolorous, 10.8-13.5 µm; upper two coloured cells usually smaller than lower most coloured cell, apical hyaline cell short, conic bearing 2 to 3 (usually 3) setulae, 8.1-13.5

mm long; basal hyaline cell long, conic to cylindric, pedicels oblique 2.7-7.29 μm long (Bilgrami and Purohit, 1971).

10. *Curvularia* leaf spot

Leaf spot of guava caused by *Curvularia siddiquii* Ahmed & Quraishi, was first reported from Naini, Allahabad, UP, India during 1964 (Srivastava, 1963).

10.1 Symptomatology

Dark brown spots on the leaves of guava develop during the months of September-October. The infection is restricted only on the tips and margins in the initial stages but subsequently the spots cover the whole leaf lamina. Defoliation is observed in case of severe infections.

10.2 Causal organism

The organism is identified as *Curvularia siddiquii*. The hyphae are white or olive green septate, branched, 3.0-4.0 μm wide, conidiophores light brown, variable in size, unbranched, septate, 3.5-4.5 μm wide, conidia large, brown, curved, 4 celled, two inner cells bigger than the distal cells measuring 27.5-39.0 x 13.5-21.0 μm (av. 33.0 x 16.5 μm).

11. *Pestalotiopsis* leaf spot

Pestalotiopsis psidii (Pat.) Mordue causes violate spots formed near mid rib with necrotic center and pycnidia are formed on the lower surface (Misra, 1987). *Pestalotiopsis versicolor* (Speg.) Steyaert causing leaf spot of *Anogeissus latifolia* when inoculated on guava, was also found pathogenic (Agarwal and Ganguli, 1959).

12. Rust of Guava

Rust of guava caused by *Puccnia psidii* Wint., was recorded first from Brazil in 1884 and Its distribution is limited to the western hemisphere in Central and South America, Florida and the Caribbean (Anonymous, 1949). The Pathogen attacks leaves, flowers, shoots and fruits and may cause 80 - 100 % losses. *P. psidii* has wide host range and infects about eight genera and 25 species of plants belonging to family Myrtaceae. The autoecious rust pathogen attacks and produces symptoms on guava, jamb (*Eugenia jambos*), jaboticaba (*Myrciaria jaboticaba*) and *Eucalyptus* spp.

         The disease is a serious threat to natural forests and plantations of *Eucalyptus* spp. in both Southern Africa and Australia. Description of *P. psidii* is provided in the CMI description set-6 (Anonymous, 1965).

12.1 Varietal resistance

Sweet varieties, both white and red fleshed are more susceptible to the rust than the

sour varieties. Vigorous growth varieties have high infection (Andrade, 1951).

12.2 Control

For the control of rust, spray of 1 per cent Bordeaux mixture after winter rain is recommended. Next spray is given when the tree begin their active growth, and subsequent sprays at monthly intervals. B 03818 (a combination of $NiCl_2$ and Zineb) 0.03 per cent is also effective for the control of rust and is equivalent to 1 per cent Bordeaux mixture (Andrade, 1959). The products based on chlorothalonil were the most efficient in the control of guava rust, controlling above 85% disease (Ferrari *et al.*, 1997). *Fusarium decemcellulare* (*Nectria rigidiuscula*) is found hyperparasite on the uredospores of *P. psidii* in Recife, Pernambuco, Brazil and can be used for control (Amorim *et al.*, 1993).

13. Damping off of seedlings

Damping off of guava is a serious disease and often responsible for enormous loss in nurseries. The disease was reported from Saharanpur (Tandon, 1961) and Basti (Gupta, 1978).

13.1 Symptomatology

Both pre emergence and post emergence phases of the disease are observed. In pre emergence phase the infected seeds and seedlings show water soaked discolouration, the seed becomes soft and ultimately rots. The affected young seedlings are killed before they reach the soil surface. In post emergence phase, hypocotyle at ground level or upper leaves are discoloured into yellowish to brown colour, which spreads downwards and later turn soft and finally rot and constrict. The affected seedlings ultimately topple down and die (Fig.3). Strands of mycelium may appear on the surface of the plants under humid conditions (Tandon, 1961, Gupta, 1978).

13.2 Causal organism

The pathogen is identified as *Rhizoctonia solani* Kuhn. (Gupta, 1978). Tandon (1961)reported seedling blight of guava caused by *Rhizoctonia* sp., but he did not specify the species. He reported that the seedlings over four months old are not affected.

13.3 Control

There are some old reports for the control of the disease. These are as follows. Good control of the disease was achieved by Flit 406 (0.2%) or Ferbam (0.3%) (Tandon, 1961) and with Bavistin (Carbendazim) and Brassicol (Quintozene) at 3 and 5 g/kg seed respectively (Gupta, 1979). Now for the control of the disease following recommendations may be made.
i.   Diseased guava seedlings and weeds should be removed and burned.
ii. Excessive use of water and close planting should be avoided as the organism is

moisture loving. Seed-beds should be prepared with proper drainage arrangement.
iii. As the fungus survives on several hosts. Hence planting of susceptible hosts should be avoided.
iv. Two minutes dipping of guava seeds in Captan/Thiram.
v. Drenching of soil with Copper oxychloride help in reducing the disease intensity in nursery.

14. *Clistocybe* root rot

Root rot caused by *Clitocybe tabescens* (Scop.) Bres. has been observed most fre-
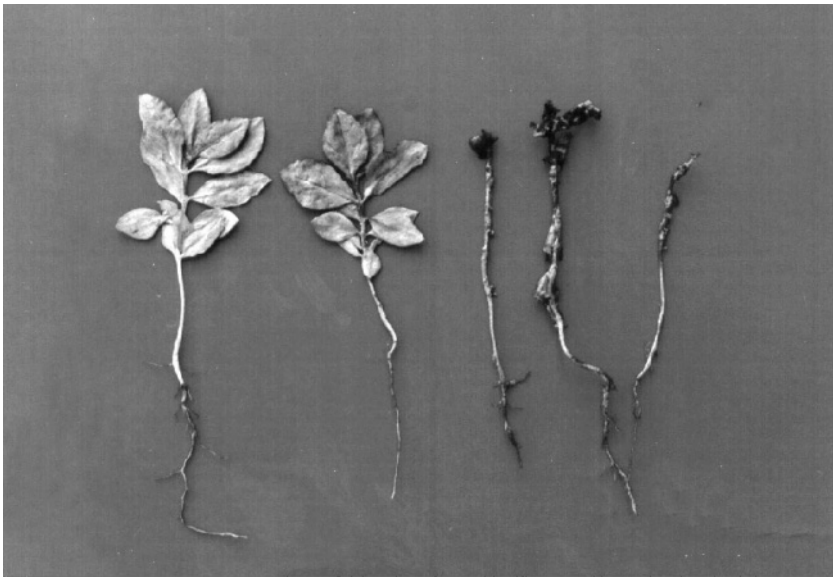
Figure 3: Damping off of seedlings

quently in guava trees, killing them in old citrus grooves in various localities of Florida (Rhoads, 1927, Webber, 1928). The disease is reported on *Psidium guajava, P. molle* and Cattley guava (Rhoads, 1927, Webber, 1928 and Rhoads, 1942).

15. *Phytophthora* fruit rot

Fruit rot caused by *Phytophthora parasitica* Dastur was reported by Mitra (1929) from Pusa, Bihar, India. He described that the disease appears in wet season (July-September). The disease is also reported to cause considerable losses in other States of India. Allahabad Safeda, Banaras, Bangalore local, Red fleshed and seedless cultivars are susceptible to the disease (Sohi and Sridhar, 1971). In Punjab, it was noticed for the first

time at Ludhiana during July 1975. The incidence of the disease varied from 5-20 per cent. The varieties, Allahabad Safeda, Apple guava, Red fleshed and pink fleshed were found affected (Singh *et al.*,1976). Fruit rot of guava caused by *Phytophthora nicotianae* var. *parasitica* Dastur, Waterhouse Syn. *Phytophthora nicotianae* Dastur was reported from Haryana (Gupta *et al.*, 1977), Poona (Rao, 1966), Bangalore (Sridhar *et al.*, 1975, Rawal,1993) and Cuba (Ariosa, 1982).

Fruit rot of guava caused by *Phytophthora citricola* Sawada was also reported by Ko *et al.*, in 1982 in an orchard of Waiakea-Uka on the island of Hawaii. Fruit hanging close to the soil surface were found affected during rainy period. The infected area appears grayish brown and water soaked with grayish black centre. Mature fruits appear to be less susceptible than mature green or green fruits. The distribution of disease appear to be limited (Ko *et al.,*1982). The fungus is self inducing (homothallic) producing oospores in single culture.

15.1 Symptomatology

The fruits especially affected are those, which have fallen on the ground or which hang near the ground level or which have been placed in storage. The disease starts at styler end. The whitish cottony growth develops very fast as the fruit ripens and is able to cover almost the entire surface within a period of about 3-4 days during humid weather (Fig. 4).

The fruits near the soil level covered with dense foliage under high relative humidity are most severely affected. The skin of the fruit below the whitish cottony growth of mycelium becomes a little soft, turns light brown to dark brown and emits a characteristic unpleasant smell. The diseased fruits generally retain their normal shape unless they are invaded by saprophytes. These fruits either remain intact or drop off. When the disease appears on young and half grown fruits, they shrink, turn dirty brown to dark brown, remain hard in texture, either remain intact as mummified fruit, or drop off (Singh, *et al.*, 1976).

15.2 Causal organism

Phytophthora parasitica Dastur (Mitra1929, Sohi and Sridhar, 1971) and *Phytophthora nicotianae* var. *parasitica* (Singh *et al.* 1976. *P. citricola* (Ko *et al.*, 1982) are reported to cause guave fruit rot.

15.3 Physiological change

Phytophthora nicotianae var. *parasitica* infected fruits had reduced free amino acids, sugars, ascorbic acids, phenol and orthodihydroxyphenol contents. The inverse correlation between disease development and phenol and ortho-dihydroxy phenol contents of the fruits suggests that phenols may not be involved in resistance mechanism against this pathogen (Mathur *et al.*, 1980). Rotted guava tissue always show more cellulolytic and pectolytic activity than healthy tissue (Prasad *et al.*, 1979).

15.4 Varietal resistance

Cultivar Lucknow-49, Banarsi Surkha, Allahabad Safeda and Mishri are highly suscep-
tible, Tehsildar is moderately susceptible and Chittidar and Apple Colour are quite
resistant (Gupta *et al.*, 1977).

15.5 Control

Diathane Z-78 (0.2 %) or Aureofungin (10 ppm) are reported effective in controlling the
disease, while Copper oxychloride is found toxic to the fruits (Sohi and Sridhar, 1971).

Figure 4: Phytophthora fruit rot

16. Dry rot

The dry rot of guava fruits was observed during 1969 at Vellayani. In some of the
infected trees more than 40 % of the fruits were infected (Rajgopalan and Wilson,
1972a).

16.1 Symptomatology

Initially the light brown spots mostly at the stalk end or at the calyx end of the fruit
develop. In few cases, infection spreads quickly and within 3-4 days entire fruit is
affected. Completely infected young and mature fruits become dark brown to almost
black in colour and ultimately dry up. A number of dry fruits can be seen on infected
trees. Numerous pycnidia of the pathogen appear as pinhead like structures on the rind

of the dried fruits. (Rajgopalan and Wilson, 1972a).

16.2 Causal organism

The dry rot of guava fruit is caused by *Diplodia natalensis* Evans. The pycnidia of the fungus produced on guava fruits are erumpent, more or less globose, dark coloured and measure 175-475 x 90-185 mm. Pycnidiospores are initially hyaline, oblong and unicellular. On maturity they become oblong to elliptical, two celled and dark brown having longitudinal striation on the wall. They measure 21.5 to 32.2 mm long and 12.2 to 16.5 mm wide (Rajgopalan and Wilson, 1972a).

16.3 Physiological changes

Sucrose, glucose and fructose concentrations increase with the age in non-inoculated fruits and sucrose of guava fruit is slowly hydrolyzed by *D. natalensis* (Ghosh *et al.*, 1964).

16.4 Control

Ziride 3000 ppm controls Diplodia dry rot of guava fruits in orchards and does not cause any phytotoxic effect on flowers and fruits of guava when it is applied at 15 days interval (Rajgopalan and Wilson, 1972b).

17. *Phomopsis* rot

The disease was reported from Sagar (Rao *et al.*, 1976). About 60 per cent of the fruits of a plant were found affected.

17.1 Symptomatology

The infected fruits show disease symptoms near the stalk. Under favourable environmental conditions, infection centers are numerous. Lesions are dark brown, at first, small and increase in size to 2 cm diameter. The tissues soften and the entire fruit rots within 8-12 days. The rotten fruits fall from the parent plants causing heavy loss in the yield of the crop (Fig.5).

17.2 Causal organism

The disease is caused by *Phomopsis destructum* Rao, Agarwal & Sakesena. The colony is white forming zones in the medium. Mycelium thin walled, hyaline, branched, septate; pycnidia dark coloured, leathery to carbonaceous, ovoid, thick walled, formed generally in 20 days old culture, 300-600 mm diameter. Sporogenous cells hyaline, simple, rarely branched, phialidic enteroblastic, arising directly from the innermost layer of cells lining the pycnidial cavity. Fungus produces two types of spores, stylospore long,

slender, often curved

17.3 Control

In *in vitro* tests, griseofulvin was found very effective in completely inhibiting the mycelial growth at 22 ppm while auriofungin and nystatin inhibited it at 100ppm. Tetracycline and Chloramphenicol are also effective (Rao and Agarwal, 1976a,b). Phenolic compounds, alpha-naphthol 50 ppm and guaicol 250 ppm cause complete inhibition of mycelial growth of *P. psidii* (Khare *et al.*, 1994)

18. *Guignardia* **fruit rot**

Figure 5 : Styler end rot caused by *Phomopsis psidii* de Camara.

Guignardia fruit rot of guava was recorded on variety Beaumont in transit as well as in field from Bangalore during the year 1980 (Ullasa and Rawal, 1984).

18.1 Symptomatology

Symptoms develop as minute depressed or flattend spots on the ripening fruits. In these spots, fungus develops in a concentric manner. Several spots later coalesce and form bigger lesions. On fruit, no fruiting structure is seen. On PDA fungus produces both ascigenous as well as pycnidial stage after 10 days of incubation under laboratory conditions. Typical symptoms appear after 6-8 days of inoculation (Ullasa and Rawal, 1984). Misra and Prakash (1986) described formation of circular black spots on the

surface of ripe fruits due to this fungus.

18.2 Causal organism

The fungus was identified as a new species *i.e. Guignardia psidii,*Ullasa and Rawal (Ullasa and Rawal, 1984). *G. psidii* was also isolated on guava fruit from Rehmankhera, Lucknow (Misra, 1987). Colonies on PDA are greenish grey which become bluish black with abundant aerial mycelium. Reverse in plate dark grey to black, submerged mycelium consists of green to brownish black hyphae are seen (Ullasa and Rawal, 1984).

19. Anthracnose / Fruit rot

Anthracnose fruit rot of guava is reported from Mysore, Bangalore, Karnataka (Narsimhan, 1938, Venkatakrishniah, 1952), Agra, Allahabad and Lucknow, U.P. (Mehta, 1951, Tandon and Agarwal, 1954, Misra and Prakash, 1986).

19.1 Symptomatology

Gloeosporium psidii remains in dormant conditions in the young infected fruits and subsequently it resumes activity and causes rot when the fruit starts ripening (Tandonand Agarwal, 1954). Non susceptibility of the young guava fruits is due to the hardness of the fruit. On Ripe fruits many small, shallow, water soaked lesions are produced on the surface of fruits and with age they enlarge and become depressed. Subsequently they coalesce to form large spots, irregular in shape and size and under humid conditions they develop salmon coloured spore masses at the center (Srivastava and Tandon, 1969a). Anthracnose caused by *G. psidii* is also common at Lucknow but in winter crop symptoms do not develop well as compared to rainy season crop (Misra and Prakash, 1986).

19.2 Causal organism

Anthracnose of guava fruit is caused by *Gloeosporium psidii* Declacr = *Colletotrichum* sp. , *Colletotrichum accutatum.*The growth of the pathogen increases with the amount of glucose in the basal medium. Absence of nitrogen source from the basal medium reduces the growth of the fungus by about 75 per cent, but the different concentrations do not affect the growth, provided the amount of glucose is kept constant. The growth of *G. psidii* is best with fructose/glucose (f/g) ratio of 0.5 at pH 4.0, a condition present in the mature green guava fruits wherein the pathogen causes maximum damage. With the f/g ratio of 1.5, 2.0 or 2.5 the growth is significantly less, a condition present in the young guava fruit wherein only incipient infection results (Midha and Chohan, 1968). *G. psidii* grows significantly better on any combination of galactose, raffinose and sorbitol. Mannitol, along with raffinose, support excellent growth of *G.psidii*. Excellent sporulation of *G. psidii* is observed on a combination of all the four carbon substances. *Gloeosporium psidii* produces maximum amount of pectinolytic enzymes in potato yeast extract medium. The optimum temperature for enzyme production is 20^0C. Maxi-

mum enzyme production is noted at pH 5.0 and maximum activity at pH 4.5. The optimum range of C/N ratio for enzyme production is 35-52 (Midha and Chohan, 1967). *Colletotrichum* sp. isolated from different fruits are either pathogenic only to their original host or to several hosts (Yang and Chuang, 1994).*Colletotrichum acutatum* – a new fruit rotting pathogen of guava was reported from Assam. In Assam 90% of the stored guava fruit rot in 5-10 days due to this fungus (Das and Bose, 1993).

19.3   Control

Storage at 10^0C prevents decay but when guava fruits are brought back to room temperature the decay is enhanced. Higher temperature favours decay (Bhargava *et al.*, 1965). The cultivar Allahabad safeda is classified susceptible, Chittidar as moderately susceptible and Apple guava as resistant to *G. psidii* (Singh and Bhargava, 1977a,b).

20. *Pestalotia* fruit rot

Pestalotia fruit rot of guava is caused by two species of *Pestalotia i.e. Pestalotia psidii* Pat. and *P. olivacea* Guba.

20.1 *Pestalotia psidii* Pat.

The disease is reported from Allahabad, Lucknow and Hissar (Srivastava and Tandon, 1969a, Misra and Prakash, 1986, Kaushik *et al.*, 1970). Misra and Prakash (1986) found the incidence of canker 10-15 per cent at Rehmankhera, Lucknow.

20.1.1 Symptomatology

The disease begins in the form of brownish discolouration, which becomes russet coloured after a week. As the spots enlarge, their central region become slightly depressed and subsequently minute, single or gregarious black acervuli with viscoid spore masses appear on the infected region (Fig.6). In partially rotted fruits, the margin of infected tissue appears ochraceous buff in the colour (Srivastava and Tandon, 1969).

20.1.2 Epidemiology

P. psidii is pathogenic on guava when fruits are injured (Kaushik *et al.*, 1970). When fruits are stored at $100^{o}C$ and brought back to room temperature, the decay is enhanced (Bhargava *et al.*, 1965). Higher temperature *favours* decay by *P. psidii.* Canker of ripe fruits, require a humid atmosphere, having higher temperature $(25-35^{o}C)$ and relative humidity (80-100%) for causing maximum disease development.

20.1.3 Physiological changes

Sucrose, glucose and fructose concentration increase with age in non inoculated fruits. Sucrose in guava is completely hydrolized by *P. psidii* in 6 days (Ghosh *et al.*, 1964).

20.1.4 Control

The infected fruits preserved at low temperature and dry atmosphere can be protected from the incidence of the disease. Post harvest wash with aureofungin (200 ppm) can also protect cent per cent guava fruits for 5 days from the canker caused by *P. psidii* (Kaushik *et al.*, 1972). Allahabad Safeda was classified as susceptible, Chittidar as moderately susceptible and Apple guava as resistant to *P. psidii* (Singh and Bhargava, 1977 a,b).

20.2 *Pestalotia olivacea* Guba

Disease was reported from Kurukshetra (Dhingra and Mehrotra, 1980).

Figure 6: Fruit rot due to *Pestalotia psidii* Pat.

20.2.1 Symptomatology

In this rot, infection on fruits start in the form of brownish coloured watery lesions, which later on changes to russet coloured spots. White fluffy growth along with black pin head like acervuli make it's appearance after one week (Dhingra and Mehrotra, 1980).

20.2.2 Causal organism

The colony of the pathogen *Pestalotia olivacea* Guba is white, hyphae hyaline, branched,

septate 2.5-3.5 mm wide; conidia 4-5 cell varied coloured with 2 end cells on either side hyaline and conical, next cells light brown and middle cells dark brown with thick wall and septa. Conidia 2.5-32.5 x 4.5-6.0 mm. Setulae 2-3 on either side of the conidia attached to hyaline cells by a short pedicel, thread like pointed ends, 23-25 mm long (Dhingra and Mehrotra, 1980).

   Pestalotia cruenta Syd. causing fruit rot of Indian gooseberry (*Phyllanthus emblica* L.) was found pathogenic on guava also (Tandon and Srivastava, 1964).

21. Stylar end rot

The stylar end rot of guava fruits is caused by *Phomopsis* sp. has been recorded in the vicinity of Lucknow and pathogenicity was proved in *in vivo* and *in vitro* test. Wounding favours the infection (Rai, 1956). Later, it was considered to be caused by *Phomopsis psidii* de Camara (Srivastava and Tandon, 1969a). The disease is also reported from Bangalore (Rawal,1993).

21.1 Symptomatology

The first visible symptom of the disease is the discoloration in the region lying just below and adjoining the persistent calyx. The discolouration area gradually increases in size and turn dark brown. Later the affected area becomes soft. Along with the discolouration of epicarp, the mesocarp tissue also shows discolouration and the diseased area is marked by being pulpy and light brown in colour in contrast to the bright white colour of the healthy area of the mesocarp. At an advanced stage due to disorganization of the inner affected tissues, size of the fruit shrinks and concentric wrinkles develop on the skin. Finally the whole fruit is affected and is covered with pycnidia. At all stages of development of the disease the affected tissue show abundant fungal hypahe which are mostly intercellular (Rai, 1956).

21.2 Causal organism

The disease is considered to be caused by *Phomopsis psidii* de Camara (Srivastava and Tandon, 1969a). The fungus produces dark carbonaceous pycnidia on rotting fruits and also in culture. Under moist conditions, masses of spores ooze out from these pycnidia through the ostiole. Pycnidia are ovoid and thick walled. They measure between 140 to 400mm in diameter. Conidia are ovate to elongate, hyaline varying between 5-9 x 2.5-4.0 mm. Stylospores long , slender, curved and vary between 16-30 x 0.8-1.4 μ m. The fungus requires a minimum temperature for growth near 10^0C, an optimum near 25^0C and maximum of about 35^0C (Rai, 1956). Change in amino acid, organic acid, sugar and ascorbic acid contents of infected fruit of guava with *Phomopsis psidii* is noticed (Lal *et al.*, 1985).

21.3 Control

Alpha-naphthol (50ppm) and guaicol (250ppm) cause complete inhibition of mycelial

growth of *P.psidii* and fruit treated with this do not show disease symptom (Khare *et al.*,1994)**.**

22. Sour rot

A fruit rot of guava caused by *Geotrichum candidum* Link ex Pers. was first recorded from Nagpur in October, 1974 and it's pathogenicity was proved on wounded ripe guava fruits. Mandarin orange, lemon, lime, sweet orange, banana, apple, tomato, grape vine, watermelon, muskmelon, cucumber fruits and tubers of potato were found to develop symptom on artificial inoculation (Shankhapal and Hatwalne, 1976).

23. Soft watery rot

It is one of the most common widely occurring disease of guava in India. This disease was recorded in India by Edward *et al.*, (1964), Srivastava and Tandon (1969a, 1969b) and Patel and Pathak (1995) from Allahabad and Udaipur. Adisa (1985) reported it from Nigeria and grouped it into soft rot causing organism and recognized the disease of high occurrence.

23.1 Symptomatology

The infection starts as a brownish discolouration mostly at the stem end and it gradually proceeds downwards in an irregular wavy manner. Finally the whole fruit may get involved. In advanced cases numerous small pycnidia are produced over the entire surface of the fruit. The rot produced by the pathogen is soft and watery.

23.2 Causal organism

Soft watery rot of guava is caused by *Botryodiplodia theobromae* Pat. The optimum temperature for the growth of the pycnidia production of *B. theobromae* is 25^0C, but conidial germination is maximum at 30^0C but at 10^0C the spores do not germinate (Srivastava and Tandon, 1969b, Patel and Pathak, 1995). No rotting takes place upto 10 days, if fruits are stored at 10-15^{0}C (Srivastava and Tandon , 1969b). Storage at 10^0C prevent decay but when guava stored at 10^0C and brought back to room temperature, the decay is enhanced (Bhargava *et al.*, 1965). Germination of spore is highest at 100 per cent R.H. and lowest at 30 per cent R.H. (Patel and Pathak, 1995).

23.3 Physiological changes

As a result of Botryodiplodia infection many amino acids decrease due to their utilization but occasionally some amino acids increase in infected tissues due to proteolysis of host proteins. Due to infection sugar content decreases considerably (Srivastava, 1969). Inoculation of fruits with *B. theobromae,* reduce protein content, vitamin C, tartaric, citric and malic acids. Succinic acid appears in inoculated fruit and reducing

sugar are abundant in inoculated fruits (Adisa, 1983).

23.4 Control

Captan is found effective (Srivastava and Tandon, 1971). Homeopathic drug arsenic oxide is effective against *B. theobromae* (Kehri and Chandra, 1986).

23.5 Varietal resistance

Variety Safeda and Apple coloured are susceptible while Pear Shaped is moderately susceptible (Srivastava and Tandon, 1969 b).

24. *Aspergillus* soft rot

Aspergillus soft rot is caused by several species of *Aspergillus* of which *A.awamori, A.wentii* and *A.niger* are important.

24.1 *Aspergillus awamori* Nakazawa

Soft rot of guava caused by *Aspergillus awamori* was reported from Allahabad (Lal *et al.*, 1980). Unlike other Aspergillus rots, which are mainly responsible for the post harvest decay, this disease occurs on unripe fruits and results in 10-15 per cent loss.

24.1.1 Symptomatology

The disease spreads as small circular water soaked spot, which enlarge and become russet brown colour with age. The diseased lesion becomes soft in middle and later develops warm sepia to clove brown coloured conidial heads surrounded by a white circular ring mixed with citron yellow coloured fungus mycelia. The disease spreads on the whole fruit with a black mouldy growth. The fruit rots completely within 10-12 days and soft pulpy tissue emits a fermented odour. Pricked fruits develop usual symptoms. High humidity and temperature greatly favour development of disease. However very young green fruits fail to show any soft rot symptoms.

24.1.2 Control

Guava fruit treated with Bavistin (Carbendazim) and Saprol (Triforine) at 1250 ppm as pre inoculation treatment completely check the rot (Arya *et al.*, 1981).

24.2 *Aspergillus wentii* Wehmer.

The infection starts as discoloured area. The spot later turns pulpy and yellow brown. The infected portion shows maceration of the tissues. Optimum temperature and R.H. for the development of fungus is 35^0C and 70% respectively (Gupta *et al.*, 1979).

24.3 *Aspergillus niger* Van Tiegh.

The fungus develops brownish soft spots, which advance in size and depth, eventually become blackish in colour. There is some shrinkage and the fruit consequently reduces in size. An intensive maceration of tissue results into oozing of yellow brown watery secretion mixed with spores and emission of disagreeable odour. Optimum RH is 70 per cent (Gupta *et al.*, 1979). With the increase in the incubation period, the Vitamin C content of both healthy and infected fruit tissue decreases but the rate of decline in the healthy fruit is much less than in infected ones (Singh and Tandon, 1971). Adisa (1985) reported soft rot caused by *A. niger* from Nigeria also.

24.4 Biochemical studies

Aflotoxin production in guava fruit by *Aspergillus* spp. is recorded. *Aspergillus flavus* Link and *A. parasiticus* Speare, produce 0.563 and 0.257 ppm of aflotoxin B_1 on guava fruits respectively. These fungi also cause considerable loss in the quantity of total reducing and non-reducing sugars, protein and ascorbic acid. Maximum decrease in sugar content is due to the infection with *A. parasiticus*, whereas, maximum depletion in protein and ascorbic acid contents is caused by *A. flavus*. Increase of 85 and 78 per cent in phenol content is recorded in fruit due to infection with *A. flavus* and *A. parasiticus* respectively (Singh and Sinha, 1983).

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25. *Pestalotiopsis* rot

Misra (1987) reported fruit rot due to *Pestalotiopsis psidii* from Rehmankhera, Lucknow. It causes reddish or brown yellow superficial spots, which cover about 50 per cent, surface of the fruit and cause soft rotting. Disease is also reported from Bangalore (Rawal,1993).

26. Fusarial rot

Adisa (1985), reported rot of guava from Nigeria by *Fusarium equiseti* (Corda) Sacc. and *F. oxysporum* Schlecht., while Misra (1987) reported *F. moniliforme* var. *intermedium* causing large dark spot with cracking of epidermal tissue from Rehmankhera, Lucknow.

27. *Rhizopus* fruit rot

Fruit rot of guava caused by *Rhizopus stolonifer* (Ehrenb. ex. Fr.) Lind. was first re-

ported by Ooka from Kauai in 1980 (Ooka, 1980). He observed soft rot affecting mature green to fully ripe fruits in orchard of Kauai island during 1978 and proved it's pathogenicity. Adisa (1985) reported *Rhizopus stolonifer* and *R. oryzae* Went. & Geerlings from Nigeria causing soft rot. In early stage of disease development the lesions appear oily and water soaked. The lesion margins are distinct and the lesions are slightly sunken at the margin. The rapidly extending lesions reduce the fruit flesh to a semi solid state in a few days.

The epidermis generally remains intact. Aerial hyphae develop at the point of infection. Although development is not extensive, it rapidly extends over the lesion and covers it with a sparse white to grey mycelium. White sporangiophores and sporangia develop which later turn black, where the aerial hyphae contact the surface. sporangiophores and sporangia develop more densely at breaks that may occur in the epidermis of the affected area and at the point of infection. Infected fruits remain attached to the tree until they are manually dislodged or fall in the course of natural maturation. Rhizopus rot is easily distinguished from Mucor rot in the field. Mucor rotted fruits are covered with abundant yellow mycelia and sporangia and Rhizopus rotted fruit show comparatively sparse aerial mycelium with dark grey to black sporangiophores and sporangia (Ooka , 1980).

28. *Mucor* fruit rot

A fruit rot of guava caused by *Mucor haemalis* Wehmer was first reported by Kunimoto *et al.*, in 1977 from island of Hawaii and Kauai. Even upto 80 per cent of mature green fruits on trees were found infected. The pathogenicity was also proved by artificial inoculation. The infected areas show water soaked lesions, which develop within a week and the entire fruit is covered with yellowish, fuzzy mass of fungal fruiting bodies and mycelia. Diseased fruits also give off and yeasty odour. The disease affects the yield of marketable fruits to considerable degree. It was found that the disease develops only when guava fruits are wounded. *Mucor haemalis* therefore is considered a wound parasite on guava fruits (Kunimoto *et al.*, 1977).

29. References

Adisa,V.A. 1983. Metabolic changes in post infected guava fruit. Fitopathologia Brasileira , 8: 81-86.

Adisa,V.A. 1985. Fruit rot diseases of guava (*Psidium guajava*) in Nigeria. Indian Phytopathology, 38: 427-430.

Agarwal, G.P. and Ganguli, G. 1959. A leaf spot disease of *Anogeissus latifolia* Wall, due to *Pestalotiopsis versicolor* (Speg.) Steyaert. Current Science, 28:295-296.

Amorim, E.P.R., Pio Ribeiro, G., Menezes, M. and Coelho, R.S.B. 1993. The pathogenicity and hyperparasitic action of *Fusarium decemcellulare* on *Puccinia psidii* in guava (*Psidium guajava*). Fitopatologia Brasileira, 18: 226-229.

Andrade, A.C. 1951. Guava rust control by means of spraying. Arq. Institute Biology, S.Paulo, 20:127-146.

Andrade, A.C. 1959. New fungicides for the control of guava rust. Biologica, 25: 178-179.

Anonymous, 1949. Distribution maps of plant diseases - Maps 169-192 issued by Common

Wealth Mycological Institute, Kew, 1949 pp. 38.

Anonymous, 1950. Annual administrative report of the department of Agriculture, United Province for the year ending 30th June, 1948, pp. 125.

Anonymous, 1953. Report of the Directorate of Plant Protection, Quarantine and Storage for the period 1946-1951, pp. 44.

Anonymous, 1965. CMI descriptions of pathogenic fungi and bacteria. Set 6, Sheets 51-60. CMI, Kew.

Anonymous, 1974. Annual Report, Indian Institute of Horticulture Research, Hessarghatta, Bangalore, pp. 47.

Ansar, M., Saleem, A. and Iqbal, A. 1994. Cause and control of guava decline in the Punjab (Pakistan). Pakistan Journal of Phytopathology, 6: 41-46.

Ariosa, T. 1982. New diseases of guava *(Psidium guajava* L.) In Sancti Spiritus Province. Centro Agricola, 9: 3-7.

Arya, A., Dwivedi, D.K., Pandey, R.S., Shukla, D.N., Bhargava, S.N. and Lal, B. 1981. Chemical control of Aspergillus rot of guava. Indian Phytopathology, 34: 359-360.

Bhargava, S.N., Ghosh, A.K., Srivastava, M.P., Singh, R.H. and Tandon, R.N. 1965. Studies on fungal diseases of some tropical fruits VII. Effect of temperature on the decay of mango, banana and guava caused by some important pathogens. Proceeding of National Academy of Science, India, Sec. B, 35: 393-398.

Bilgrami, K.S. and Purohit, D.K. 1971. A new pathogenic species of *Pestalotia*. Indian Phytopathology, 24: 211-213.

Bose, S. K. and Muller, E. 1967. Central Himalayan fungi. Indian Phytopathology, 20: 124-138.

Butt, A.A., Nasir, M.A. and Bajwa, M.N. 1995. *In vitro* evaluation of different chemicals against *Gloeosporium psidii*, the cause of anthracnose of guava. Pakistan Journal of Phytopathology, 7: 92-93.

Chakraborty, D.K. and Singh, R.N. 1989. Guava wilt correlation between variation in disease syndrome and edaphic factors. Indian Phytopathology, 42: 310.

Chandra Mohan 1985. Studies on guava decline in Punjab with special reference to wilt. Ph.D. Thesis., Punjab Agricultural University, Ludhiana, India. 90 p.

Chandra Mohan, Jhooty, J.S. and Chand, T. 1986. Prevalence of guava decline in Punjab. Plant Disease Research, 1: 77-78.

Chattopadhyay, S.B. and Bhattacharjya, S.K. 1968a. Investigation on wilt disease of guava *(Psidium guajava* L.) in west Bengal, I. Indian Journal of Agriculture Sciences, 38: 65- 72.

Chattopadhyay, S.B. and Bhattacharjya, S.K. 1968b. Investigation on wilt disease of guava *(Psidium guajava* L.) in west Bengal, II . Indian Journal of Agriculture Sciences, 38: 176-183.

Chattopadhyay, S.B. and Sengupta, S.K. 1955. Studies on wilt of guava, in West Bengal. Indian Journal of Horticulture, 12: 76-79.

Chibber, H.M. 1911. A working list of diseases of vegetable pests of some of the economic plants, occurring in the Bombay Presidency. Poona Agriculture College Magzine, 2: 180-198.

Das Gupta, M.K. and Ghosal, P.K. 1977. It is possible to control guava wilt through oil cake amendments. Science. & Culture, 43: 131-133.

Das Gupta, S. N. and Rai, J. N. 1947. Wilt disease of guava *(Psidium guajava* L.). Current Science, 16: 256-258.

Das, M. and Bose, K.N. 1993. *Colletotrichum acutatum*-a new fruit rotting pathogen of guava *(Psidium guajava* L.) in storage. Indian Journal of Mycology and Plant Pathology, 23:331.

Dey, P.K. 1948. Plant Pathology. Administrative Report of Agriculture Department, U. P. 1945-46., pp. 43-46.

Dhingra, R. and Mehrotra, R.S. 1980. A few unrecorded post harvest diseases of fruits and vegetables. Indian Phytopathology, 33: 475-476.

Dwivedi, S.K. 1991a. Population dynamic of microfungi including pathogenic forms in the beds of completely healthy, partially wilted and completely wilted guava trees grown on a line. International Journal of Tropical Plant Disease 9: 95-109.

Dwivedi, S.K. 1991b. Effect of some heavy metals on growth of *Fusarium oxysporum* f.sp. *psidii* causing guava wilt disease. International Journal of Tropical Plant Disease, 9 : 127-130.

Dwivedi, S.K., Dwivedi, R.S. and Tiwari, V.P. 1990. Studies on pathogenic fungi inciting guava wilt in Varanasi. Indian Phytopathology, 43: 116-117.

Edward, J.C. 1960a. Variation in the guava wilt pathogen, *Fusarium oxysporum* f. *psidii*. Indian Phytopathology, 13: 30-36.

Edward, J.C. 1960b. Penetration and establishment of *Fusarium oxysporium* f. *psidii* in guava root. Indian Phytopathology, 13: 168-170.

Edward, J.C. 1960c. Wilt disease of guava. The Allahabad Farmer, 34: 289 - 293.

Edward, J.C. 1961. Root stock for guava wilt control. The Allahabad Farmer, 25: 5-10.

Edward, J.C. and Srivastava, R.N. 1957. Studies on guava wilt. The Allahabad Farmer, 31: 144-146.

Edward, J.C. and Gaurishankar 1964. Root stock trail for guava (*Psidium guiajava* L.). The Allahabad Farmer, 38: 249-250.

Edward, J.C., Naim, Z. and Gaurishankar 1964. Canker and fruit rot of guava (*Psidium guajava* L.). The Allahabad Farmer, 38: 1-3.

Ferrari, J. T., Nogueira, E.M.D. and dos Santos, A. J.T. 1997. Control of rust (*Puccinia psidii*) in guava (*Psidium guajava*). Acta Hort., 452:55-58

Ghosh, A.K., Tandon, R.N., Bilgrami, K.S. and Srivastava, M.P. 1964. Post infection changes in sugar contents of some fruits. Phytopath. Z., 50: 283-288.

Grech, N.M. 1985. First report of guava rapid death syndrome caused by *Septofusidium* sp. in South Africa. Plant Disease, 69: 726.

Gupta, J.H. 1978. Damping off, a new disease of guava. Indian Journal of Mycology and Plant Pathology, 8: 224.

Gupta, J.H. 1979. Control of damping off of guava by seed treatment with systemic and non-systemic fungicides. Progressive Horticulture, 10: 53-55.

Gupta, J.P., Chatrath, M.S. and Khan, A.M. 1973. Chemical control of fruit rot of guava caused by *Colletotrichum gloeosporioides*. Indian Phytopathology, 26: 650-653.

Gupta, P.C., Madaan, R.L and Suhag, L.S. 1977. Varietal reaction of guava fruits to *Phytophthora nicotianae* var. *parasitica*. Indian Journal of Mycology and Plant Pathology, 7: 177.

Gupta, Y.K., Roy, A.N., Yadav, S. and Gupta, M.N. 1979. Investigations on post harvest diseases of guava fruits. Indian Phytopathology, 32: 623-624.

Hsieh, S.P.Y., Liang, W.J., Kao, C.W. and Leu, L.S. 1976. Morphological and physiological characters of *Myxosporium psidii*, the causal organism of guava wilt. Plant Protection Bulletin, Taiwan, 18: 309-317.

Jain, S.S. 1956. A preliminary note on the inactivation of *Fusarium oxysporium* f. *psidii* in guava plants by chemotherapeutic treatment. Indian Journal Horticulture, 13: 102-104.

Jhooty, J.S., Chand, J.N. and Krishnamurty, V. 1984. Report of committee constituted by ICAR on guava decline in Punjab and Haryana. Submitted to ICAR, New Delhi.

Katyal, S.L. 1972. Twenty-five years of research on fruit crops. Indian Farming, 22: 14-16.

Kaushik, C.D., Chand, J.N. and Thakur, D.P. 1970. Fungi associated with decay of certain fruits and potato tubers in Haryana markets. Journal of Research, Ludhiana, 7: 648-650.

Kaushik, C.D., Thakur, D.P. and Chand, J.N. 1972. Parasitism and control of *Pestalotia psidii* causing cankerous disease of ripe guava fruits. Indian Phytopathology, 25: 61-64.

Kehri, H.K. and Chandra, S. 1986. Control of *Botryodiplodia* rot of guava with a homeopathic drug. National Academy Science Letter, 9: 301-302.

Khanna, K.K. and Chandra, S. 1977. Control of guava fruit rot caused by *Pestalotia psidii* with homeopathic drugs. Plant Disease Reporter, 61: 362-366.

Khare, V., Mehta, P., Kachhwaha, M. and Mehta, A. 1994. Role of phenolic substances in pathogenesis of soft rot diseases. Journal of Basic Microbiology, 34: 323-328.

Ko, W.H., Kunimoto, R.K. and Nishijima, W.T. 1982. Fruit rot of guava caused by *Phytophthora citricola*. Plant Disease, 66: 854-855.

Kunimoto, R.K., Ito, P.J. and Ko, W.H. 1977. *Mucor* rot of guava fruits caused by *Mucor hiemalis.* Tropical Agriculture (Trinidad), 54: 185-187.

Lal, B., Arya, A., Agarwal, R. and Tewari, D.K. 1985. Biochemical changes in guava fruits infected with *Phomopsis psidii*. Acta Bot. Indica, 13:124-126.

Lal, B., Rai, R.N., Arya, A and Tiwari, D.K. 1980. A new soft rot of guava. National Academy Science Letter, 3:259-260.

Leu, L.S. and Kao, C.W. 1979. Artificial inoculation of guava with *Myxosporium psidii.* Plant Disease Reporter, 63: 1077-1079.

Leu, L.S., Kao, C.W., Wang, C.C., Liang, W.J. and Hsieh, S.P.Y. 1979. *Myxosporium* wilt of guava and it's control. Plant Disease Reporter, 63: 1075-1077.

Marlatt, R.B. and Campbell, C.W. 1980a. Susceptibility of *Psidium guajava* selections to injury by *Cephaleuros* sp. Plant Disease, 64: 1010-1011.Marlatt, R.B. and Campbell, C.W. 1980b. Incidence of algal disease (*Cephaleuros* sp.) in selections of guava (*Psidium guajava*). Proceeding of Florida State Horticulture Society, 93: 109-110.

Mathur, R. S. 1956. Guava disease in India. Indian Journal of Horticulture, 13:26-29.

Mathur, R.S. and Jain S.S. 1960. Selecting guavas for wilt resistance. Proceeding of National Academy Science, India, Sect. B. 30: 33-36.

Mathur, R.S., Jain, S.S. and Swarup, J. 1964. Chemical treatment for guava wilt. Proceeding of National Academy Science, India, Sect. B. 34: 33-36.

Mathur, S., Bhatnagar, M.K. and Mathur, K. 1980. Some chemical changes in guava fruits infected by *Phytophthora*. Philippine Agriculturist, 63: 379-383.

Mehta, N. 1987. Distribution of guava wilt in relation to age, soil type, management practices and varieties grown in Haryana. Plant Disease Research, 2: 116-119.

Mehta, P.R. 1951. Observations on new and known diseases of crop plants of Uttar Pradesh. Plant Protection Bulletin, 3: 7-12.

Midha, S.K. and Chohan, J.S. 1967. Factors affecting the production of pectinolytic enzymes by *Gloeosporium psidii*, the causal agent of fruit rot of guava (*Psidium guajava*). Indian Phytopathology, 20: 215-219.

Midha, S.K. and Chohan, J.S. 1968. Chemical basis for incipient infection caused by *Gloeosporium psidii* in guava fruits. Journal of Research, Ludhiana, 5: 395-400.

Misra, A.K. 1987. Studies on diseases of fruit crops. Annual Report, CIHNP, Lucknow. pp.124-125.

Misra, A.K. 1995. Guava Wilt. In: "Advances in Diseases of Fruits in India". (ed. Singh, S.J.), Kalyani Publishers, Ludhiana. pp. 183-190 + I plate.

Misra, A.K**.** 2001. Diseases of Guava and their management. In: **"**Diseases of Fruits and Vegetables and their Management" (ed. Thind, T. S), Kalyani Publishers, Ludhiana, pp. 128-138.

Misra, A.K. and Om Prakash 1990. Guava Diseases - (An annotated bibliography 1907-1990). Bishen Singh Mahendra Pal Singh, Dehradun , 132 P.

Misra, A.K. and Pandey, B.K. 1992. Wilt of guava and associated pathogens. Indian Journal of Mycology and Plant Pathology, 22: 85-86.

Misra, A.K. and Pandey, B.K. 1996. Present status of wilt disease of guava. In: "Disease Scenario in Crop Plants. Vol. I - Fruits and Vegetables". (eds.. Agnihotri, V.P, Om Prakash , Ram Kishun and Misra, A.K.) International Books and Periodical Supply Service, New Delhi, pp. 61-70.

Misra, A.K. and Pandey, B.K. 1997. Pathogenicity and symptom production of wilt disease of guava by a new potent pathogen *Gliocladium roseum.* Indian Phytopathological Society-Golden Jubilee International Conference.Nov. 10-15, 1997, New Delhi. pp. 319.

Misra, A.K. and Pandey, B.K. 1999a. Natural wilting of guava plants during different months. 51st Annual Meeting of Indian Phytopathological Society and National Symposium on Seed health care and Phytosanitation for Sustainable Agriculture. 17-19 Feb. 1999, IISR, Lucknow. pp 64-65.

Misra, A.K. and Pandey, B.K. 1999b. Guava wilt disease-A challenge for the coming millenium. Proceedings of the National Symposium on Challenges & Prospects of Plant Pathology in the coming millenium. Dec. 9-11, 1999 N.B.R.I., Lucknow. pp 22.

Misra, A.K. and Pandey B.K. 1999c. Pathogenicity and evaluation of fungicides against guava wilt pathogens. Journal of Mycology and Plant Pathology 29: 274-75.

Misra A.K. and Pandey, B.K. 1999d. Natural wilting of guava plants during different months. Indian Phytopathology 52: 312.

Misra, A.K. and Pandey, B.K. 2000a. Pathogenicity and symptom production of wilt disease of guava by a new potent pathogen *Gliocladium roseum*. Proceedings, Indian Phytopathological Society - Golden Jubilee, International Conference on Integrated Disease Management for Sustainable Agriculture Vol.II Pub. Indian Phytopathological Society, New Delhi, pp.749-750.

Misra, A.K. and Pandey, B.K. 2000b. Progressive natural wilting of guava plants during different months. Indian Phytopathology, 423-427.

Misra, A. K. and Prakash, Om 1986. Studies on diseases of fruit crops. Annual Report, Central Institute of Horticulture for Northern Plains, Lucknow. pp. 67-68.

Misra, A.K. and Shukla, S.K. 2002. Assessment of loss due to Guava wilt around Lucknow. National Seminar on Production and Post-Harvest Technology of Guava. Department of Horticulture, CSAUA&T, Kanpur, 9-10 January, 2002. pp 34-35.

Misra, A. K., Om Prakash and Sen, B. 2000. Biological control of guava wilt by *Aspergillus niger* strain AN17 (Pusa Mrida). National Seminar on Hi-tech Horticulture. 26-28th June, Bangalore, pp.149.

Misra, A.K., Om Prakash and Pandey, B.K. 2001. Eco-friendly approach in the management of wilt disease of guava. National Symposium on Eco-friendly Approaches for Plant Disease Management. January 22-24, 2001, CAS in Botany, University of Madras, Chennai. (ab. 66, poster session - II).

Misra, A.K., Pandey, B.K., Prasad, Babita and Shukla, S.K. 2003. Pathogenic diversity in the cause of wilt disease of guava. 55th Annual Meeting of IPS and National Seminar on Plant Pathogen Diversity in Relation to Plant Health. 16-18th Jan. 2003. Osmania University, Hyderabad. pp. 47 (abs. 61).

Mitra, M. 1929. *Phytophthora parasitica* Dast. causing damping off disease of cotton seedlings and fruit rot of guava in India. Transaction of British Mycological Society, 14: 249-254.

Narsimhan, M.J. 1938. *Pestalotia psidii* on guava in India. Annual Administrative Report , Agriculture Department, Mysore, 1936 - 37. pp.169-173.

Narsimhan, M.J. 1940. Annual Report, Mysore Agriculture Department for the year 1938 - 39. pp. 96 - 97.

Negi, S.S., Misra, A.K. and Rajan, S. 2001. Guava wilt. Proc. National Seminar on New Horizon in Production and Post Harvest Management of Tropical and Subtropical Fruits, IARI New Delhi, Held on Dec. 8-9, 1998, Special issue, Indian Journal of Horticulture, 54: 145-151.

Normand, F. 1994. Strawberry guava, relevance for reunion, Fruits-Peris, 49:217-227.

Ooka, J.J. 1980. Guava fruit rot caused by *Rhizopus stolonifer* in Hawaii. *Pl. Dis*., 64: 412-413.

Pandey, R.R. and Dwivedi, R.S. 1985. *Fusarium oxysporum* f. sp. *psidii* as a pathogen causing wilt of guava in Varanasi district, India. Phytopathologische Z., 114: 243-248.

Pandey, R.S., Bhargava S.N., Shukla, D.N. and Dwivedi, D.K. 1983. Control of *Pestalotia* fruit rot of guava by leaf extracts of two medicinal plants. Revista Maxicana de Fitopathologia, 2: 15-16.

Patel, K.D. and Pathak, V.N. 1995. Development of Botryodiplodia rot of guava fruits in relation to temperature and humidity. Indian Phytopathology, 48: 86-89.

Patel, M. K., Kamat, M. N. and Hingorani, G. M. 1950. *Pestalotia psidii* Pat. on guava. Indian Phytopath., 3: 165-176.

Prakash Om, Misra, A.K. and Shukla, S.K. 2002. *Penicillium citrinum* a potent pathogen against wilt disease of guava. Asian Congress of Mycology and Plant Pathology and Symposium on Plant Health for Food Security. Oct 1-4, 2002. University of Mysore, Mysore. pp. 180. (abs. PP-299)

Prasad, G., Bhargava, K. S. and Mehrotra, R. S. 1979. Production of pectolytic and cellulolytic enzymes *in vivo* and *in vitro* by *Phytophthora nicotianae* var. *parasitica* causing fruit rot of *Psidium guajava*. Indian Journal of Mycology and Plant Pathology, 9: 36-40.

Prasad, N., Mehta, P.R. and Lal, S.B. 1952. *Fusarium* wilt of guava (*Psidium guajava* L.) in Uttar Pradesh, India. Nature, 4305: 753.

Preez, R.J. du 1995. Guava cultivars. Inligtingsbulletin Instituut vir Tropiese en Subtropiese Gewasse, 272: 10-16.

Raghunathan, V. and Prasad, N.N. 1969. Occurrence of *Cercospora sawadae* on *Psidium guajava*. Plant Disease Reporter., 53: 455.

Rana, O.S. 1981. *Diplodia* stem canker, a new disease of guava in Tarai regions of U.P. Science & Culture, 47: 370-371.

Rao, D.P.C. and Agarwal, S.C. 1976a. Efficacy of antibiotics against *Phomopsis destructum* causing fruit rot of guava. Hindustan Antibiotic Bulletin, 18: 108-110.

Rao, D.P.C. and Agarwal, S.C. 1976b. Efficacy of fungicides against *Phomopsis* fruit rot of guava. Indian Phytopathology, 29: 345-346.

Rao, D.P.C., Agarwal, S.C. and Saksena, S.B. 1976. *Phomopsis destructum* on *Psidium guajava* fruits in India. Mycologia, 68: 1132-1134.

Rao, V.G. 1966. An account of market and storage diseases of fruits and vegetables in Bombay, Maharashtra (India). Mycopath. et. Mycol. Applicata., 28: 165-176.

Rawal, R.D. 1993. Yield loss in guava by different root rots. International Journal Tropical Plant Disease., 11: 69-72.

Rhoads, A.S. 1927. *Clitocybe* root rot of trees and other woody plants in Florida. Phytopathology, 17: 56-57.

Rhoads, A.S. 1942. Notes on *Clitocybe* root rot of bananas and other plants in Florida. Phytopathology, 32: 487 - 496.

Rodrigues, N.J., Robbs, C.F. and Yamashiro, T. 1987. A bacterial disease of guava (*Psidium guajava*) caused by *Erwnia psidii* sp. nov. Fitopathologia Brasileira, 12: 345-350.

Rodriguez, F.M.E. and Landa, J.B. 1977. Chemical soil disinfection against parasitic nematode in guava nurseries. Centro Agricola de la Facultod de Ciencias Agricolos, 4: 57-77.

Ruehle, G.D. 1936. An epiphytotic of algal spot in South Florida. U.S. Deptt. Agric., Bureau of Plant Industry. Plant Disease Reporter, 20: 221-222.

Ruehle, G.D. 1941. Algal leaf and fruit spot of guava. Phytopathology, 31: 95-96.

Sen, P.K. and Verma, B.S. 1954. Studies on die back disease of guava (*Psidium guajava* L.). A survey of the incidence of the disease in the Jhargram area (West Bengal). 41st Proceedings of Indian Science Congress, Sec. X, Agric. Sci., pp. 258.

Shankhapal, K.B. and Hatwalne, V.G. 1976. Sour rot of guava in India. Current Science, 45: 565-566.

Sharma, S.K., Singh, J.P. and Chand, J.N. 1983. Chemical control of anthracnose of guava caused by *Glomerella cingulata*. Haryana Agriculture University Journal of Research, 13: 325-326.

Singh, A. and Sinha, K.K. 1983. Biochemical changes and aflotoxin production in guava fruits by *Aspergillus flavus* and *A. parasiticus.* Indian Phytopathology, 36: 365-366.

Singh, A.P. and Bhargava, S.N. 1977a. Benlate as an effective post harvest fungicide for guava fruit. Indian Journal of Horticulture, 34: 309-312.

Singh, A.P. and Bhargava, S.N. 1977b. Storage and transit studies in apple guavas. Indian Journal of Horticulture, 34: 362-363.

Singh, B. and Lal, S.B. 1953. Wilt of guava. Agriculture and Animal Husbandry, 3: 78-79.

Singh, G., Chohan, J.S. and Mann, S.K. 1976. Fruit rot of guava - a new disease problem in Punjab. Indian Journal of Mycology and Plant Pathology, 6: 77.

Singh, J.P. and Sharma, S.K. 1982. Controlling anthracnose of guava caused by *Glomerella cingulata* by fumigation. Indian Phytopathology, 35: 273-276.

Singh, R.H. and Tandon, R.N. 1971. Vitamin C content of guava fruits infected with *Aspergillus niger.* Indian Phytopathology, 24: 807-809.

Singh, U.R., Dhar, L. and Singh, G. 1977. Note on the performance of guava cultivars and *Psidium* spp. against wilt disease under natural field conditions. Haryana Journal of Horticulture Science, 6: 149-150.

Sohi, H.S. 1983a. Studies on wilt disease of guava. Annual Report , Indian Horticulture Research Institute, Hassarghatta, Bangalore, pp.102.

Sohi, H.S. 1983b. Diseases of tropical and subtropical fruits and their control. In: Recent advances in Plant Pathology. (ed. Husain, A., Singh, K., Singh, B. P. and Agnihotri, V. P.) Print House, Lucknow, pp. 73-86.

Sohi, H.S. and Sridhar, T.S. 1969. Injurious effect of copper sprays on guava fruits. Indian Journal of Horticulture, 26: 155.

Sohi, H.S. and Sridhar, T.S. 1971. Controlling fruit rot of guava. Indian Horticulture, 16: 9-10.

Sridhar, T.S. and Ullasa, B.A. 1978. Leaf blight of guava, a new record. Current. Science, 47: 442.

Sridhar, T.S., Ullasa, B.A. and Sohi, H.S. 1975. Occurrence of a new disease on grape seedlings caused by *Phytophthora nicotianae* var. *parasitica* (Dasture) Waterhouse from India. Current Science, 44: 406.

Srivastava, H.P. 1963. Some leaf spot fungi. Proceedings of National Academy of Sciences, India, 34: 188-198.

Srivastava, M.P. 1969. Biochemical changes in certain tropical fruits during pathogenesis. Phytopathologische. Z., 64: 119-123.

Srivastava, M.P. and Tandon, R.N. 1969a. Post harvest diseases of guava in India. Plant Disease Reporter, 53: 206-208.

Srivastava, M.P. and Tandon, R.N. 1969b. Studies on *Botryodiplodia* rot of guava. Indian Phytopathology, 22: 268-270.

Srivastava, M.P. and Tandon, R.N. 1971. Efficacy of certain fungicides and an antibiotic against four isolates of *Botryodiplodia theobromae.* Indian Phytopathology, 24: 396-397.

Suhag, L.S. 1976. Observations on guava decline in Haryana and it's control. Pesticides, 10: 42-44.

Suhag, L.S. and Khera, A.P. 1986. Studies on the variation nutritional level of wilted regenerated and healthy trees of guava cultivar Banarasi Surkha. Indian Phytopathology, 39: 90-92.

Tandon, I.N. 1961. A new seedling blight of guava and it's control. Indian Phytopathology, 14: 102-103.

Tandon, I.N. and Singh, B.B. 1969. Studies on anthracnose of guava and it's control. Indian Phytopathology, 22: 322-326.

Tandon, M.P. 1950. Sulphur requirement of *Pestalotia malorum* and *Pestalotia psidii.* Proceedings of Indian Academy of Sciences, Sce. B., 32: 7-11.

Tandon, R.N. and Agarwala, R.K. 1954. Pathological studies of *Gleosporium psidii* causing die back of guava. Proceedings of Indian Academy of Sciences., Sec. B., 40: 102-109.

Tandon, R.N. and Srivastava, M.P. 1964. Fruit rot of *Embilica officinalis* Gaertn. caused by *Pestalotia cruenta* Syd. in India. Current Sciences, 33: 86-87.

Thirumalachar, M.J. 1945. An Ascomycetous parasite of *Cephaleuros*. Proceedings of Indian Academy of Sciences., Sec. B., 22: 374-377.

Tokeshi, H, Valdebenito, R.M. and Dias, A.S. 1980. Occurrence of a bacterial disease of guava in Sao Paulo state. Summa Phytopathologica, 6: 85-87.

Ullasa, B.A. and Rawal, R.D. 1984. *Guignardia* fruit rot of guava - a new disease from Bangalore. Current Science, 53: 435-436.

Uppal, B.N. 1936. *Physalospora psidii* on guava - a serious disease of guava in Bombay. International Bulletin of Plant Protection, 10: 99.

Venkatakrishniah, N.S. 1952. *Glomerella psidii* (Del.) Sheld. and *Pestolitia psidii* Pat. associated with cankerous disease of guava. Proceedings of Indian Academy of Sciences., Sec. B., 36: 129-134.

Verma, B.R. and Sharma, S.L. 1976. Seasonal variation in symptoms caused by *Pestalotia psidii* on guava fruits. Indian Journal of Mycology and Plant Pathology, 6: 97-98.

Vestal, E.F. 1941. A text Book of Plant Pathology. Kitabistan, Allahabad and Karachi. 645 P.

Vidyasekaran, P. and Parambaramani, C. 1971a. Carbon metabolism of alga infected plants. Indian Phytopathology, 24: 369-374.

Vidyasekaran, P. and Parambaramani, C. 1971b. Nitrogen metabolism of alga infected plants. Indian Phytopathology, 24: 500-504.

Vidyasekaran, P. and Parambaramani, C. 1972. Mineral metabolism of alga infected plants. Indian Phytopathology, 25: 86-90.

Webber, G.F. 1928. Plant Pathology, Annual Report, Florida Agriculture Experiment Station for the fiscal year ending June 30, 1928. 65 R-78 R.

Yadav, A.S. 1953. Some new hosts of *Cephaleuros* from Bihar. Current Science, 22: 280.

Yang, H.R. and Chuang, T.Y. 1994. Pathogenicity and zymogram of antrhacnose fungi isolated from some fruits. Memoires of the College of Agriculture, National Taiwan University, 34: 1-8.

Lettuce Diseases and their Management

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Abstract : Lettuce is the world's most popular leafy salad vegetable. Various types of lettuce are cultivated across the globe, primarily for human consumption of their fresh, succulent leaves. Over 75 lettuce disorders of diverse causes and etiologies have been described. While some diseases are limited in their importance and distribution, a significant number are present wherever *Lactuca sativa* L. is grown. Many are capable of causing devastating losses in yield and quality under favorable conditions. In this chapter, we have divided lettuce diseases broadly into infectious and non-infectious disorders. Of the important infectious diseases covered, fungi and viruses account for the bulk. Nine fungal diseases are discussed, including anthracnose, bottom rot, Cercospora leaf spot, damping-off, downy mildew, drop, gray mold, Septoria leaf spot, and southern blight. Five viral diseases are covered, and these are: beet western yellows, lettuce big-vein, lettuce infectious yellows, lettuce mosaic, and tomato spotted wilt. The sole phytoplasmic lettuce disease, aster yellows, is also discussed. Of five important bacterial diseases detailed, four are foliar disorders: bacterial leaf spot, marginal leaf blight, soft rot, and varnish spot. Corky root is the one bacterial root disease included. In contrast, all nematode pathogens discussed, lesion, needle, and root-knot nematodes, infect lettuce roots. Three important non-infectious disorders are included in this chapter, namely brown stain, pink rib, and tipburn. These are mainly disorders of mature or postharvest lettuce.

1. Introduction

1.1 Lettuce origin

Cultivated lettuce (*Lactuca sativa* L.) is a direct descendent from wild lettuce, probably *Lactuca serriola* L., originating in or around the Mediterranean basin. Evidence for its early cultivation dates back to the Middle Kingdom of the Egyptian Empire some 4,500 years ago (Lindqvist 1960). Lettuce was also among the first vegetables brought to the New World by Columbus. Today, it is one of the world's most popular and widely distributed vegetables.

1.2 Lettuce types

There are a number of different types of lettuce, including: crisphead (iceberg), romaine (cos), leaf, butterhead (Boston and bibb), and numerous others of limited popularity. These include Latin, Batavia, stem, and oilseed types (Ryder 1979).

1.3 Growth stages

Lettuce for fresh market undergoes three major growth phases prior to harvest: seed-ling development, a rosette period, and heading (Table 1). Lettuce for seed production has three additional phases, namely: seed stalk formation, flowering, and seed production.

Table 1: Developmental growth stages of crisphead lettuce

Growth stage	Process	Comments on activity
Seedling	Germination	Radicle emerges, generally in 3-7 days.
(20-36 days)	Cotyledon dev.	Cotyledons emerge and expand.
	True leaf increase	First true leaves emerge and expand.
	Thinning	Crop is thinned to final stand at 4-5 leaf stage.
Rosette (14-28 days)	Rosette dev.	Leaves develop in flattened to upright structure. Not yet curved.
Heading (26-56 days)	Early-mid heading	Curved leaves emerge and expand, becoming successively more curved.
	Mature heading	A sufficient number of interior leaves have developed that a firm spherical head forms. May require 60-120 days, depending on the season

1.4 Lettuce production

In most regions, lettuce is direct-seeded in fields using mechanically-operated precision seeders. However, in some areas it is first raised as transplants which are then planted into raised beds or flat ground. Lettuce is usually planted on 2-row, raised beds with a 30-cm row-spacing atop the beds formed on 1-m centers. Plant spacing is usually 25-30 cm, achieved by manually thinning overseeded rows. Optimum temperatures for germination and growth are 20-25° C, with temperatures exceeding 30° C being inhibitory. Adequate water and oxygen are required for germination, which usually occurs 3-7 days after planting. Thinning is usually 3-4 weeks subsequent to planting. Harvest maturity, depending upon the lettuce type and season, usually ranges from 60-120 days after seeding for crisphead lettuce. Once harvested, lettuce heads

are trimmed, packed into cartons, vacuum or water cooled to 1.0-1.1° C, and are shipped at 1.1-2.2° C.

2. Diseases caused by fungi and oomycetes

Fungi and oomycetes are organisms that lack chlorophyll and have vegetative structures that are generally threadlike and branched. Fungi are also capable of producing various types of asexual and/or sexual reproductive structures. These enable many fungal pathogens to spread rapidly over considerable distances. Some of these structures, such as oospores and sclerotia, facilitate long term survival. All fungal diseases are profoundly influenced by environmental conditions, most importantly, temperature and moisture. The manipulation of these and other environmental factors frequently aids in fungal disease control.

About 20 different fungi and oomycetes are reported to cause serious problems on lettuce (Davis, 1997). Nine are included in this chapter. Fungal diseases reported but not included due to limited distribution or economic importance are: Alternaria leaf spot (*Alternaria* spp.), Fusarium wilt (*Fusarium oxysporum* Schlechtend.:Fr. emend. W. C. Snyder & H. N. Hans. f. sp. *lactucum* Hubbard & Gerik), Mycocentrospora spot (*Mycocentrospora acerina* R. Hartig), Phytophthora rot (*Phytophthora* de Bary spp.), Pythium rot (*Pythium aphanidermatum* (Edson) Fitzp.), Pythium wilt (*Pythium uncinulatum* Plaats-Niterink & Blok and other *Pythium* spp.), rust (*Puccinia opizii* Bubak), Stemphylium leaf spot (*Stemphylium botryosum* f. *lactucum* Wallr.), Texas root rot (*Phymatotrichopsis omnivorum* (Duggar) Hennebert), and Verticillium wilt (*Verticillium dahliae* Kleb.).

2.1 Anthracnose

Worldwide in distribution, anthracnose is frequently referred to as shot-hole, ring spot, and rust. Yield losses may be significant, particularly during cool, moist periods. Lettuce anthracnose is caused by the fungus *Microdochium panattonianum* (Berl.) Sutton Galea & Price, formerly known as *Marssonina panattoniana* (Berl.) Magnus (Galea *et al.* 1986). Symptoms initially appear as small, tan water-soaked lesions on exterior leaves. Foliar lesions often expand to 2-3 mm in diameter, become straw-colored, and have their centers fall out as they age, hence the name 'shot-hole'. Anthracnose is typically most severe along the midrib of the lower leaf surface. Infections directly on the midrib typically become elongated and sunken, but do not fall through. Damaged tissues may cause poor head formation and increase lettuce susceptibility to secondary invaders such as soft rotting bacteria.

The pathogen survives as mycelium within infested debris or as microsclerotia (60-100 μm) in the soil. Under favorable conditions, microsclerotia give rise to hyphae or conidiophores capable of spore production (Patterson and Grogan 1985a). Although periods of extended leaf wetness favor infection, free moisture periods as short as 2-4 hrs may be sufficient if temperatures are optimal. Longer periods at cool temperatures are even more conducive.

Having a limited host range which includes only lettuce, escarole and endive,

crop rotation has proven to be an effective anthracnose control measure. Plowing to destroy or bury debris likewise destroys initial inoculum. While fungicides may be effective in some cases (Inglis *et al.* 1999), cultural practices designed to minimize leaf wetness should be used as much as feasible (Patterson *et al.* 1986).

2.2 Bottom rot

Bottom rot is caused by the soilborne fungus *Thanatephorus cucumeris* (A. B. Frank) Donk., however, the pathogen is more commonly referred to by its well-known anamorph, *Rhizoctonia solani* Kuhn. (Pieczarka and Lorbeer 1975). Bottom rot occurs on all types of lettuce and may be found wherever lettuce is grown. Favored by warm, wet conditions, the disease can cause losses as high as 70%. Under severe disease pressure, heads are frequently rendered unmarketable. Even under less favorable conditions, extra trimming may be required to remove affected leaves, reducing head size and weight.

 R. solani is a common soil inhabitant and therefore lesions typically appear on the lowest leaves in direct contact with the soil. Initial infections appear as small, rust-colored to chocolate brown spots, primarily on the underside of midribs. While these may be evident as early as four weeks after direct seeding, advanced symptoms of the disease are usually not seen until heading. Under favorable temperature and moisture conditions, bottom rot lesions may expand rapidly, quickly rotting midribs and leaf blades. Wilting of outer wrapper leaves is one of the first symptoms observed when infected plants are viewed from above. Decaying heads are at first slimy and brown but become almost black as they collapse and dry. The tan to brown mycelium that characterizes the fungal pathogen is frequently observed without magnification within infected head tissues. Irregularly shaped, cinnamon brown to dark brown sclerotia are produced during later stages of the disease.

 Hyphae of *R. solani* are white to brown and septate. Hyphae typically branch at near right angles with a crosswall appearing just after branching. Branches are conspicuously constricted at the juncture. Asexual fruiting structures and spores are lacking but the perfect state produces basidia measuring 18-23 x 8-11 μm. Basidiospores are hyaline and single-celled, measuring 7-13 x 4-7 μm. As mentioned previously, the pathogen also forms small tan to brown sclerotia among connecting mycelial threads. *R solani* exhibits extensive biological specialization and a number of different groups, separated on the ability of field isolates to anastomose, have been recognized. Anastomosis occurs between isolates from the same group but not between isolates from different groups. The relative importance of the various anastomosis groups (AGs) on lettuce is still under investigation, but research has demonstrated that AG-4 groups seem to be predominant (Herr 1992).

 R. solani survives between crops as sclerotia and mycelium associated with infected debris (Herr 1993). Under favorable moisture and temperature conditions, sclerotia germinate and produce mycelial growth capable of extending 7-10 cm to reach a food source. The pathogen is capable of direct penetration, entering the plant through healthy or wounded tissues. Colonization is both inter-and intracellular. Warm, moist conditions are most conducive to disease development, with

optimal temperatures being reported as 25-28°C. At such temperatures, the interval from infection to initial symptom development may be as short as 36-48 hr. Incubation periods lengthen considerably as temperatures vary from the optimal range.

Optimum control of bottom rot is achieved by using various cultural practices in combination with fungicidal control. Plowing instead of disking before planting effectively buries sclerotia.Crop rotation and effective weed control also reduce inoculum potential. Growing lettuce on high beds improves aeration and minimizes foliar contact with the soil, the point at which infection typically occurs. Planting lettuce with more erect architecture may also reduce losses and irrigation should be minimized close to harvest, as the canopy closes and retains moisture. Standard bottom rot fungicides such as iprodione and vinclozolin have proven to be effective (Mahr *et al.* 1986), and the newer strobilurin fungicides as a class show promise. Placement and timing are extremely important.

2.3 *Cercospora* leaf spot

Cercospora leaf spot, although rarely destructive, is found in many parts of the world. Favored by warm temperatures, it is most common in the tropics and subtropics. The disease has been reported as being economically significant in the Ivory Coast (Savery 1983) and has caused losses in Florida in recent years.

The disease normally appears first in the older, lower leaves. Lesions enlarge from minute brown spots surrounded by chlorotic tissue to become irregular or angular spots that vary in color from tan to brown. Fully developed lesions are slightly sunken and necrotic, exhibiting a characteristic white dot in the center. The disease spreads progressively upward, and extensive areas of leaf tissue may be killed when infections are numerous.

The disease is caused by the fungus *Cercospora longissima* Cugini ex Traverso non Cooke & Ellis, nom. illeg. No sexual stage or teleomorph has been reported. Spread of the disease appears to be entirely by means of conidia and is heavily dependent upon long periods of leaf wetness. The pathogen survives between cropping seasons on crop debris or in association with wild lettuce or closely related hosts.Conidia are hyaline and cylindrical to obclavate, with an obconically truncate base and a tapering obtuse apex. They range from 11 to 170 µm in length, with average basal and apical widths of 7.5 and 3.8 µm, respectively. Conidia are produced on unbranched, olivaceous brown conidiophores arising from stromata embedded in infected tissues. Spore germination occurs only in the presence of free moisture, with leaf wetness durations exceeding 24 hrs being required for successful penetration. At the optimal temperature of 25°C, susceptible tissues are rapidly colonized by the advancing hyphae and symptoms may become evident within 3 days after inoculation. At suboptimal temperatures of 15, 20, and 30° C, incubation periods of 7, 5, and 5 days, respectively, have been reported. Conidia, borne singly on conidiophores, become airborne during periods of low relative humidity and are transported by the wind. Spores are also spread by rain or irrigation splash.

Cultural practices for the control of Cercospora leaf spot include crop rotation,

complete destruction of crop debris and lettuce-related weeds, and good drainage. Overhead irrigation or other practices which produce prolonged leaf wetness should also be avoided. Foliar fungicides may be justified in some cases with the newer strobilurin fungicides exhibiting the most efficacious control of currently registered compounds (Raid and Nagata 2003). These should be used in a program with a broad spectrum protectant fungicide to avoid the development of resistant strains of the pathogen.

2.4 Damping-off

Seedling damping-off of lettuce occurs wherever the crop is grown. The disorder is favored by excessive soil moisture, poor soil tilth, and temperatures that are unfavorable for lettuce seed germination and growth. Symptoms may be expressed as seed decay, pre- or postemergence seedling rots, or infections of seedling roots and stems.

A number of different oomycetes and fungi have been reported as being associated with lettuce damping-off (Hine *et al.* 1991). Among oomycetes, *Pythium irregulare* Buisman, *P. sylvaticum* W. A. Campbell & Hendrix, and *P. ultimum* Trow have been most closely linked with pre- and postemergence damping-off, while *P. dissotocum* Drechs., *P. uncinulatum* Plaats-Niterink & Blok, and *P. violae* Chesters & C. J. Hickman result in root tip necrosis and the inhibition of lateral root formation. Among fungi, *Rhizoctonia solani* Kuhn has been most frequently linked to post-emergence death. *R. solani*, also the causal agent of bottom rot, typically attacks the stem at or near the soil surface. The stem tissue in this area may be marked by a distinct margin between the infected and healthy tissue.

Primary inoculum for damping-off incited by *Pythium* spp. consists of thick-walled oospores or sporangia. These spores are stimulated to germinated by nutrients contained in seed or root exudates. Secondary infections arise from motile zoospores released from germinating sporangia. Capable of swimming only short distances in water, zoospores may be transported greater distances by the movement of surface water, such as that which occurs with furrow irrigation. Damping-off incited by *Pythium* spp. is typically highest in fields with poor drainage.

A ubiquitous soil-inhabitant, *R. solani* survives as mycelium in infested crop debris or as sclerotia. A detailed description of the fungus and its mode of infection is given in the section on bottom rot. Incidence of damping-off caused by both *Pythium* spp. and *Rhizoctonia* is enhanced by suboptimal growth conditions. Cool or excessively warm temperatures, improper fertilization, and excessive moisture all serve to slow lettuce germination and seedling growth. This widens the window of vulnerability of succulent, non-hardened seedling tissues to these soilborne pathogens.

Controls for damping-off should include cultural practices which favor rapid seed germination and seedling vigor. High-quality seed should be planted under favorable environmental condition in well-drained, loose soil. Chemical seed or soil treatments may also prove useful in preventing infection.

2.5 Downy mildew

Caused by the fungus *Bremia lactucae* Regel, downy mildew is a major disease of

lettuce worldwide. Favored by cool temperatures and moist foliar conditions, downy mildew may be a significant problem in both field and greenhouse production systems. High levels of infection have resulted in entire fields being destroyed, while even relatively low levels of infection may cause significant trimming and handling losses at harvest. The disease may also promote postharvest decay, resulting in significant transit and storage losses (Crute and Dixon 1981).

Downy mildew lesions typically start out light green, turning increasingly chlorotic over time. With age, lesions eventually become angular, and turn necrotic after the onset of sporulation. Lesions are quite variable in size but are frequently delineated by the larger veins. Under conditions favoring development (cool temperatures, long dew periods), sporulation structures visible to the naked eye may be evident. While sporulation predominates on the lower leaf surface, it may also appear on the upper leaf surface. All growth stages of lettuce are susceptible to downy mildew. Seedlings may become infected to the point that cotyledons and primary leaves are completely covered by sporangiophores. This frequently results in seedling death. Early infections may also lead to systemic infection, this resulting in a dark brown discoloration of vascular stem tissues (Crute and Dixon 1981).

B. lactucae is an obligate parasite. The mycelium is nonseptate or coenocytic, although sporangiophores, which arise through the stomates, are often septate. Typically, sporangiophores are dichotomously branched, with the tip of each branch producing a sterigma, on which a single sporangium (12-31μm x 11-28 μm) is formed. *B. lactucae* may also produce oospores. While the pathogen is mostly heterothallic, meaning that two compatible mating types (B1 and B2) are needed for oospore production, a number of homothallic isolates have also been reported. However, oospores are seldom seen in field-grown lettuce or wild hosts and there is controversy surrounding their role in the epidemiology of the disease. Other potential sources of primary inoculum for this disease are infected lettuce seed, inoculum from wild hosts, or sporangia from nearby lettuce fields.

The one thing that is certain regarding *B. lactucae* epidemiology is that it is capable of rapid secondary spread by means of airborne sporangia. Produced under darkness during still, humid nights, sporangia are easily dislodged for wind-dissemination during less-humid early post-dawn hours. While sporulation is optimum at 20ºC, it may occur over the relatively wide range of 5-24º C. Sporangia germination and infection may take place in as few as 3 hrs in the presence of favorable temperatures (10-22º C) and free moisture. Temperature is highly influential regarding latent periods, with latent periods ranging from 4-7 days being reported at 20-22º C, and from 24-34 days at 6º C.

However, under fluctuating temperature regimes, latent periods tend to be shorter at lower temperatures and longer at higher temperatures (Scherm and van Bruggen 1994).

Downy mildew management should utilize a number of different control strategies. The use of resistant cultivars should be strongly considered, but this is complicated by the presence of genetic variants of the pathogen. Specificity between lettuce cultivars and *B. lactucae* isolates is thought to be controlled by a gene-for-gene interaction involving at least 13 pairs of dominant resistance genes in the host and match-

ing virulence genes in the pathogen. For many years, single-gene resistance provided high levels of control, but strains developed capable of overcoming single-gene resistance. This lead to the classification of *B. lactucae* into four pathotypes, these being distinguished based upon their virulence on an established differential set of lettuce genotypes. However, the continued development of additional pathotypes has clouded the issue. Resistance strategies are currently aimed at developing cultivars with multigenic resistance in an effort to make it more durable. Identification of the pathotypes present in an area would be key to varietal selection.

Since the downy mildew pathogen is so heavily dependent upon free moisture, irrigation and cultural practices which minimize leaf wetness should be adopted (Scherm and van Bruggen 1995). This includes using drip irrigation in place of furrow or sprinkler irrigation where feasible and timing irrigation to prevent extension of early morning leaf wetness periods.

Another useful control strategy is fungicidal control. A number of broad-spectrum protectants and systemic fungicides are available for lettuce downy mildew. These should be utilized together in a program that prevents disease buildup and minimizes the risk of selecting for fungicide-insensitive strains of the pathogen (Raid and Datnoff 1990). Isolates insensitive to metalaxyl are now widespread (Raid *et al.* 1990, Schettini *et al.* 1991) and there are reports of increasing insensitivity to fosetyl-al. Computerized forecasting programs or disease warning systems have also been developed and are being evaluated for their effectiveness (Hovius and McDonald 1999, Scherm *et al.* 1995).

2.6 Drop

Named for the rapid collapse of infected plants, drop is one of the most destructive diseases of lettuce worldwide (Patterson *et al.* 1986). The disease is noted as a cool season disease and it is caused by two species of the fungus *Sclerotinia*, *S. minor* Jagger and *S. sclerotiorum* (Lib.) de Bary. Disease incidence may range from a few percent to nearly the entire field.

Initial symptoms are a wilting of the lower, outermost leaves. This gives the plants a faded stressed appearance. As the infection advances, the entire plant wilts. Collapsed outer leaf layers flatten, turn chlorotic and then necrotic. A soft watery decay may ensue on both above- and below-ground parts, resembling that caused by soft-rotting bacteria in the field. However, with drop, black sclerotia are frequently produced beneath the leaves in contact with the soil, in rotted crown area, and in the subterranean taproot. These may be accompanied by snowy white mycelium under most conditions. Sclerotia of *S. minor* are mostly spherical and uniform in diameter (approx. 0.5-2.0 mm), while those of *S. sclerotiorum* are more irregular in shape and are considerably larger, measuring 2-20 x 3-7 mm in diameter. Entire plants may collapse from drop in as few as 2 days.

Both lettuce drop pathogens may survive as sclerotia for up to 8-10 years in the soil (Imolehin and Grogan 1980), but they frequently differ in their mode of infection (Abawi and Grogan 1979). Sclerotia of *S. sclerotiorum* within the top 2-3 cm of the soil surface germinate under favorable conditions to produce one to several apothecia.

These asci-producing structures are typically cup-shaped to flat with a central dimple. Usually white, yellow or tan, apothecia give rise to cylindrical asci containing eight nonseptate, hyaline, elliptical ascospores (9-13 x 4-5 μm). Conditions favoring asco-carp formation are high soil moisture and temperatures of 11-15°C. Over a period of 2-3 weeks, ascospores are forcibly ejected from apothecia during periods of low humid-ity and are carried on air currents to adjacent plants and fields. In the presence of free moisture, ascospores germinate to infect senescent or dead lettuce foliage, giving rise to secondary disease spread. Sclerotia of *S. sclerotiorum* seldom germinate eruptively to produce mycelial infection, and given the specific requirements for apothecial for-mation and ascospore release, drop incited by this *Sclerotinia* species may be unpre-dictable.

S. minor, on the other hand, seldom germinates carpogenically to produce apoth-ecia. Under moist soil conditions, *S. minor* sclerotia within 2 cm of the taproot or 8 cm of the soil surface germinate to produce infective mycelia. Once contact has been made with roots, stems, or senescent leaves, infection takes place. Drop caused by *S. minor* is much more consistent than that caused by *S. sclerotiorum* due to less specific requirement for infection. Fields infested with *S. minor* sclerotia will nearly always produce drop unless management strategies are implemented.

While sclerotia of the drop pathogens may be long-lived, crop rotation may provide adequate control if non-host rotational crops, such as small grains, are se-lected. However, *Sclerotinia* has a rather broad host-range, limiting crop selection. Deep-plowing to bury sclerotia deep in the soil has also proven to be effective under low inoculum densities (Subbarao *et al*. 1996). Irrigation methods which avoid wet-ting the top 5-8 cm of soil, such as subsurface drip or seepage irrigation, assist in controlling this disease.

Fungicidal control of drop has been achieved using dicarboximide fungicides and several new fungicides look promising (Matheron and Porchas 1999b, Matheron and Porchas 2000a). Fungicides should be applied in 12-15 cm bands over the plant-ing surface to form a protective barrier between the soil surface and lower leaves. Timing is important and multiple applications may be necessary. For *S. minor*, the initial application should be made immediately after thinning. Two additional applica-tions may be necessary at 10-day intervals if inoculum levels are high (Patterson and Grogan 1985b). For *S. sclerotiorum*, applications at or subsequent to the rosette stage (30-40 days pre-harvest), rather than earlier, have proven more effective.

Roguing of plants exhibiting drop, if feasible, may be successful in limiting sclerotial densities of S. minor, thereby reducing disease incidence (Patterson and Grogan 1985b). However, this strategy may not be effective for *S. sclerotiorum,* where ascospores may blow in from nearby fields. Efforts to develop drop resistant cultivars are ongoing but have met with only limited success at present.

2.7 Gray mold

Gray mold is typically of minor importance in the field but it may cause significant losses in the greenhouse or during post-harvest transit and storage (Ceponis *et al.* 1985). It is worldwide in distribution.

Incited by the fungus *Botrytis cinerea* Pers.:Fr., gray mold infected tissues initially appear water-soaked. Senescent tissues in contact with the soil are most susceptible. Lesions eventually turn different shades of grayish-green to brown, with surrounding areas developing an orange to red cast. Depending upon environmental conditions, a gray to brown fuzz, characteristic of the pathogen's sporulation, may develop on infected tissues. These may rapidly develop into a watery rot. Gray mold may be particularly severe on transplanted seedlings, girdling stems and causing them to collapse.

B. cinerea may survive as sclerotia in the soil, as a saprophyte on organic substrates, or as a pathogen on many crops (Coley-Smith *et al*. 1980). It has an extensive host range. The fungus produces copious quantities of asexual conidia, which are easily wind-disseminated, serving as secondary inoculum for the disease. Although it can develop over a rather broad temperature range (-2 to 25° C), optimal growth occurs between 20 and 25°C. Since sporulation is poor in complete darkness, *B. cinerea* infections present on lettuce in transport may not be immediately visible upon arrival at their destination. In addition, tissues colonized by the fungus are frequently invaded by other decay organisms, contributing to a rapid breakdown of lettuce held under improper postharvest storage conditions.

Management of gray mold begins with maintaining the crop in a healthy condition. Efforts should be made to minimize damaging tissue and to reduce the duration of leaf wetness. Greenhouses should be properly ventilated to reduce humidity, and overhead irrigation should be avoided wherever possible.

Fungicides have proven effective in reducing gray mold, but care should be exercised to prevent development of insensitive strains of the pathogen. This has occurred with benzimidazole and dicarboximide fungicides in some areas (Wang *et al.* 1986). Postharvest management strategies for gray mold should focus on reducing damage during harvest and shipping and rapidly precooling the harvested product. Lettuce should be shipped at temperatures between 1.1 and 2.2°C. Increasing CO_2 levels during shipment and storage may further inhibit gray mold development (Stewart 1978).

2.8 Powdery mildew

Powdery mildew, caused by the fungus *Erysiphe cichoracearum* DC., is generally considered to be a minor disease of lettuce. Found through out the world, it may cause significant losses under certain conditions.

The disorder is characterized by a white powdery fungal growth on both the upper and lower leaf surface. Older, outer leafs are principally affected, becoming chlorotic over time and taking on a brown, scorched appearance. Small, black specks (perithecia) may appear on such tissues. Early powdery mildew infections may reduce head size and quality.

An obligate parasite, *E. cichoracearum* may survive in the conidial state on old lettuce or closely related weeds. However initial infections may also arise from ascospores released from perithecia persisting from the precious crop (Schnathorst 1959). Ascospore release is triggered by free moisture and temperatures of 15-22°C. Conidia

of *E. cichoracearum* are easily dislodged during periods of low humidity and are wind-disseminated over long distances. Unlike those of most fungi, conidia of *E. cichoracearum* are inhibited from germinating by the presence of free moisture, with optimal germination occurring between 95 and 98% relative humidity (Schnathorst 1960). Germination and mycelial growth are optimized at 18ºC. Thus, powdery mildew thrives in climates that are somewhat cool and dry.

Sulfur applied at the initial appearance of the disease and at subsequent intervals is quite effective in controlling powdery mildew. Many newer compounds are also demonstrating efficacy and are worthy of investigation (Matheron and Porchas 1999a, Matheron and Porchas 2000b).

2.9 *Septoria* leaf spot

Although having worldwide distribution, Septoria leaf spot is usually of minor importance. However, under conditions of extended leaf wetness, it may result in significant foliar necrosis.

Caused by *Septoria lactucae* Pass., the fungal leaf spot usually develops on the oldest, outer leaves. Initial symptoms are small (less than 5 mm), irregular chlorotic spots that are marginally vein-delimited. Lesions enlarge over time, turning brown and necrotic. Small, black specks (pycnidia) may be evident under low magnification on the tan to brown necrotic tissue. Chlorotic halos may encircle leaf spots, which coalesce under favorable condition to form large areas of dead or necrotic tissue. The pathogen may also infect flower stalks, bracts, flowers, and seed of plants grown for seed production.

Septoria frequently survives on seed, and is in this way distributed across great distances. The fungus may also survive as pycnidia in infested debris that remains in the field. Upon exposure to free moisture, conidia are extruded from active pycnidia in the form of spore tendrils. Rain or irrigation splash then disseminates conidia to susceptible lettuce foliage.

Management of Septoria leaf spot should begin with pathogen-free or treated seed. Hot-water seed treatments at 47ºC for 30 minutes can significantly decrease seedborne inoculum (Smith 1961). Having a rather limited host range, crop rotation and allowing time for the complete decomposition of infected plant residues may also reduce primary inoculum. Sprinkle irrigation should be avoided to limit secondary spread of the leaf spot.

Although strobilurin fungicides show the most promise in helping to control Septoria leaf spot outbreaks, these should be used in conjunction with broad spectrum protectants to minimize development of insensitive strains.

2.10 Southern blight

Southern blight has been reported from nearly every country between the northern and southern latitudes of 38ºC. Favored by warm temperatures, this disease is especially severe in the subtropics and tropics. The fungal pathogen has an extremely wide host range (over 200 species), including both monocots and dicots. Fortunately, low to

moderate temperature requirements by the crop limit the impact of this pathogen on lettuce. Other common names for southern blight include southern stem rot and Sclerotium stem rot.

While the causal agent of southern blight is more widely regarded by its anamorph, *Sclerotium rolfsii* Sacc., the teleomorph has been identified as *Athelia rolfsii* (Curzi). It may be readily cultured on most general media, producing abundant, white mycelium. The coarse hyphae consists of cells 2-9 x 150-250 μm, with clamp connections readily apparent. Basidial formation is influenced by nutrient status, light intensity, and age of culture. Small, spherical (0.5-1.5 mm diam) sclerotia are also produced. These are initially white and turn light tan to brown with age.

Sclerotia of *S. rolfsii* are extremely durable, capable of surviving for years in the soil. Strongly saphrophytic, the fungus is also capable of producing growth on various host substrates. Volatile compounds produced by senescent plant tissues appear to stimulate sclerotial germination and the pathogen is capable of directly penetrating non-wounded host tissues. The prevalence of the disease in warm climates is reflective of its relatively high optimum temperature range of 27-30°C. Growth is strongly inhibited at temperatures below 15°C. Southern blight is favored by moist soil conditions and acidic, rather than alkaline, calcareous soils. Typical of many soilborne diseases, southern blight is usually clustered in the field, with cultivation and tillage equipment, movement of soil and infected debris, and movement in irrigation water providing for dissemination.

In subtropical areas, planting should be planned to take advantage of cooler growing seasons (Palti and Katan 1997). Although crop rotation by itself is not an effective or control measure, rotating from lettuce to small grains or corn may result in less disease in subsequent years. Deep plowing that buries sclerotia and infected debris in the soil reduces their viability as inoculum. While soil fumigation is generally cost prohibitive for lettuce, soil solarization has proven successful in reducing soil inoculum levels in some regions (Katan and DeVay 1991). Disease incidence may also be reduced by the use of ammoniacal nitrogen sources and fertilizers containing plant-available calcium (Punja 1985, Rodriguez-Kabana and Kokalis-Burelle, 1997).

3. Diseases caused by bacteria

Bacteria are microscopic unicellular organisms that differ from higher organisms in lacking a true nucleus. Seven genera, *Agrobacterium*, *Clavibacter*, *Erwinia*, *Pseudomonas*, *Rhizomonas*, *Streptomyces*, and *Xanthomonas* account for most, but not all of the plant diseases incited by bacteria. However, as bacteriological taxonomy has changed drastically in recent years, certain species of bacteria have undergone reclassification and subsequently, name changes. One must therefore be mindful of this when reviewing the scientific literature, for it can pose problems.

Most plant pathogenic bacteria are rod-shaped, motile, non-obligate parasites that do not form spores. Filamentous *Streptomyces* spp. are the only exception. These are nonseptate, branched filaments that produce conidia on aerial hyphae. Positive identification of bacterial species relies on a number of morphological, physiological, and serological tests to characterize certain distinguishing properties. Bacteria are

commonly disseminated on soil, by insects, splashing rain fall and irrigation water, and on workers, tools, and farm equipment. These microbes rely on wounds or natural anatomical openings to enter plant tissues, with toxin or enzyme production by the pathogens resulting in water-soaked lesions or chlorosis. Cell death (necrosis), abnormal plant growth (galls and tumors), blockage of water conduction (wilts), or collapse of plant tissues (soft rots) are other symptoms of bacteriological infection. While infections are frequently localized, bacteria may migrate throughout the plant, becoming systemic. Bacteria survive in crop debris, soil, and seeds, and on perennial or volunteer plants as epiphytes. There are five bacterial diseases of lettuce that are important world-wide.

3.1 Bacterial leaf spot

Caused by the bacterium *Xanthomonas axonopodis* pv. *vitians* (formerly *X. campestris* pv. *vitians* (Brown) Dye) , bacterial leaf spot was first described in the United States in 1918 and has since been reported in Australia, Europe, and Japan. Although usually of minor impact, it may produce catastrophic loses under certain environmental conditions (Pernezny *et al.* 1995).

The pathogen is a motile, aerobic, Gram-negative rod with a single polar flagellum. In culture, colonies are mucoid, producing a yellow pigment. Leaf spot symptoms initially start off as small, angular, water-soaked lesions occurring on mature, fully expanded leaves. Lesions take on a dark, almost-black, greasy appearance, frequently coalescing as they expand and develop. Large areas of the leaf may turn necrotic and die. Leaves in advanced stages of the disease become papery in texture and may crack and tear. Under favorable conditions, the disease may spread from outer leaves to cap leaves and subsequently to inner leaf tissues. Infected plants that are harvested and packed in cartoons may cause significant postharvest losses incited by the pathogen and infection by secondary organisms.

While bacterial spot may exhibit highly visible local lesions, recent research suggests that the bacterial pathogen may have the capacity to enter and translocate within the vascular system of lettuce plants without inducing visible symptoms (Barak *et al.* 2002). Such a capacity lends itself to seed dissemination. Since the disease may be seedborne, only lettuce seed that has been assayed as free of the presence of the pathogen should be planted in disease-prone areas (*i.e.* heavy rainfall or overhead irrigation). The bacterial spot pathogen has also been reported as capable of surviving in infested plant residues and saprophytically on certain weeds (*Barak et al.* 2001). Such residues and weeds should be plowed into the soil well in-advance of the crop, allowing them time to decompose and lessening prospects for spread of the pathogen to subsequent crops. Although chemical bactericides such as copper-based compounds may assist in suppressing the disease, these are rendered ineffective under highly favorable conditions.

3.2 Corky root

Caused by the bacterium *Rhizomonas suberifaciens* van Bruggen, Jochimsen, and

Brown, corky root has been observed throughout the lettuce growing regions of North America, Australia, New Zealand, and Europe. Losses ranging from 30-70% have been reported in heavily infested fields in California and Florida (O'Brien and van Bruggen 1992, Datnoff and Nagata 1992), primarily as a result of reduced head size. Corky root initially appears as discrete yellow lesions on the taproot and main lateral roots. These lesions develop into greenish brown bands and long, corky ridges may appear on taproots and main laterals. Infected root sections appear swollen when compared to healthy root sections. When severe, the root pith becomes discolored and hollow, with roots becoming brittle and occasionally pinching off. Interveinal chlorosis and necrosis may develop in lower leaves and heads are noticeably stunted.

The corky root pathogen grows extremely slowly on most common bacteriological media, making isolation difficult. Reported hosts include: lettuce, endive, prickly lettuce and common sowthistle (van Bruggen *et al.* 1990). Although *R. suberifaciens* has been recovered from the roots of a number of plant species, indicating that it may be a common rhizosphere inhabitant, observations suggest that populations decline in the absence of a susceptible lettuce or endive cultivar. Therefore, crop rotation can be an effective control measure (Alvarez *et al.* 1992). Disease severity has been reported as being related to heightened rates of nitrogen fertilizer applications, with ammonium rather than nitrate sources being the least detrimental. Turning in a non-host cover crop such as rye or applying slow-release nitrogen (*i.e.* sulfur-coated urea) rather than sidedressing with ammonium nitrate or ammonium sulfate may further reduce disease severity (van Bruggen *et al.* 1990). Host plant resistance to corky root has been identified and has proven itself useful (Datnoff and Nagata 1992). Corky root may also be reduced by growing lettuce on well-drained beds and by using drip irrigation rather than furrow irrigation when feasible. The use of lettuce transplants rather than direct seeding has likewise proven effective in reducing losses due to corky root in severely infested fields (van Bruggen and Rubatzky 1992).

3.3 Marginal leaf blight

Caused by the bacterium *Pseudomonas marginalis* pv. *marginalis* (Brown) Stevens, marginal leaf blight is worldwide in distribution. It is, however, typically of minor importance except under high moisture conditions. The disease is characterized by an initial wilting of localized areas along leaf margins (Brown 1918). Leaves of like-age or in the same whorl may be similarly affected. Wilted areas frequently coalesce, advancing inward along the veins several for several centimeters. The pathogen commonly enters host tissues through wounds or natural openings such as hydathodes during periods of leaf wetness. Commonly, leaf veins within affected areas may exhibit a distinctive darkening in advance of the wilting of interveinal tissues. Infected tissues eventually become brown, dry, and papery (Fahy and Persley 1983).

The pathogenic agent is a strict aerobe, Gram-negative, rod-shaped, possesses one to three polar flagella, and is pectolytic. It produces slimy yellow colonies on King's medium B, along with a diffusible yellow-green pigment that fluoresces green under ultraviolet light. Although considered ubiquitous, inoculum levels in the soil may be effectively reduced by crop rotation and destruction of crop debris prior to

planting. Vegetable crops listed as hosts include: lettuce, cabbage, chicory, cucumber, endive and onion. Cultural practices which reduce leaf wetness may also slow the rate of dissemination and infiltration. These include drip or furrow irrigation rather than overhead irrigation, and practices which encourage well-drained soil.

3.4 Soft rot

Although bacterial soft rot is one of the most serious transit and market diseases of lettuce, heavy losses may also occur in the field, where it causes a rapid wilt and collapse of crisphead and other lettuce types. Incited by *Erwinia carotovora* subsp. c*arotovora* (Jones) Bergey *et al.,* losses are frequently related to wet growing conditions. In high rainfall regions, losses as high as 90% have been reported (Cho 1977).

In the field, soft rot initially appears as a rapid wilting of outer wrapper leaves. Plants at or near harvest are most susceptible. Wilting is caused by a collapse of vascular tissues, which develop a pinkish to brown discoloration. As the disease progresses, the pith of the stem becomes water-soaked, macerated, and greenish. The basal pith in advanced stages of the disease becomes gelatin-like, giving rise to the popular reference "jelly butt". The entire head may eventually become slimy due to the extensive disintegration of foliar tissues. Bacterial soft rotted heads may be differentiated from those caused by lettuce drop by the absence of fungal mycelium and/or sclerotia.

Soft-rot infections that occur postharvest are closely associated with wounds of outer leaves (Ceponis *et al.* 1970). Wrapper leaves become wilted and discolored and heads become slimy as a result of the pectolytic breakdown foliar tissue. If infection is not extensive, rotted wrapper leaves may be stripped from the head, leaving the innermost foliage intact and marketable.

Soft-rotting bacteria are considered ubiquitous and are capable of survival in soil, in aquatic environments, and on crop residues. They are easily disseminated by insects, splashing water and contaminated equipment. Generally gaining entrance into plant tissues through wounds or natural plant openings, soft-rotting bacteria are capable of rapid multiplication under favorable temperature conditions (25-30ºC) and latent periods may be as short as 24 hr. Conversely, at cool temperatures, latent periods may range from two to three weeks. Lettuce at or near maturity appears to be much more susceptible to soft rot than lettuce at an early growth stage. Presumably, this is because of more extensive moisture periods.

Control strategies for soft rot in the field include crop rotation, improved soil drainage, drip or furrow irrigation rather than sprinkler irrigation, and proper application of fertilizers and pesticides to keep the crop growing vigorously (Cho 1977). Controls should be aimed at avoiding crop injury and excessive moisture within the canopy. Post-harvest control measures should include the most recent technological advances in shipping, handling and storage. Rapid removal of field heat through vacuum-cooling, transit temperatures below 5ºC, and extensive use of refrigeration by wholesalers and retailers have all proven successful in reducing losses due to this disease. Soft-rot resistant or tolerant varieties have been reported (Cho and Talede 1976) but are currently of limited interest in most lettuce producing regions.

3.5 Varnish spot

Nearly worldwide in distribution, varnish spot can be a very destructive disease of mature crisphead lettuce. Symptoms are generally internal and may be difficult to spot prior to harvest. Entire fields have reportedly been abandoned due to this disease. Caused by the bacterium *Pseudomonas cichorii* (Swingle) Stapp, early varnish spot infections are characterized by shiny, dark-brown, firm necrotic spots several milli-meters in diameter. Lesions frequently run lengthwise along the veins, enlarging and coalescing to infect large areas of internal foliar tissues. Varnish spot infected tissues become moist and dark, with older lesions drying out and becoming papery. Outer-most leaves are seldom affected and symptoms may not be evident without cutting the lettuce heads open (Grogan *et al.* 1977).

The varnish spot pathogen is capable of infecting a number of vegetable and other hosts, including lettuce, cabbage, cauliflower, celery, chicory, endive, and to-bacco. Soilborne, it survives in residual infected plant debris. The bacterium is dis-seminated by water and insects with wounds and natural plant openings serving as points of ingress. Therefore, control measures should be aimed at reducing periods of leaf wetness, and wounding of lettuce tissues. Reportedly, crop rotation has also dem-onstrated some effectiveness.

4. Diseases caused by viruses and phytoplasmas

Viruses are submicroscopic, obligate entities that multiply only in living or-ganisms. They are composed of nucleic acid (RNA or DNA) surrounded by a protein coat. Plant viruses are transmitted by insects, mites, fungi, nematodes, mechanical means, grafting, dodder, and seed. As a pathogen group, their importance on lettuce is rivaled only by the fungi. At least twenty viruses have been described on *L. sativa*, and six of these are detailed below. Other viruses reported on lettuce include: *Alfalfa mosaic alfamovirus* (AMV), *Beet yellow stunt closterovirus* (BYSV), *Bidens mottle potyvirus* (BiMoV), *Broad bean wilt fabavirus* (BBWV), *Dandelion yellow sequivirus* (DYMV), *Lettuce chlorosis closterovirus* (LCV), *Lettuce mottle virus* (LMoV), *Lettuce necrotic yellows rhabdovirus* (LNYV), *Sonchus yellow net rhab-dovirus* (SYNV), *Sowthistle yellow vein nucleorhabdovirus* (SYVV), *Tobacco rattle tobravirus* (TRV), *Tobacco ringspot nepovirus* (TRSV), *Tobacco streak ilarvirus* (TSV), and *Turnip mosaic potyvirus* (TuMV) (Davis *et al.* 1997). In contrast, only one phytoplasma-induced disease, aster yellows, is reported on lettuce, and that is discussed within.

4.1 Beet western yellows

Beet western yellows virus (BWYV) is a *luteovirus*, an important group of viruses affecting many of the world's major crops. Initially referred to as "June yellows", BWYV has caused lettuce yield losses throughout lettuce growing regions of the world. More than eight aphid species are capable of transmitting BWYV in a persistent (cir-culative) manner, with the green peach aphid (*Myzus persicae* Sulzer) being the most

important. Minimum acquisition and inoculation periods are 5 and 10 minutes, re-spectively. The latent period is 12-24 hrs, with the virus persisting in the host for as long as 50 days. This, in addition to an extensive host range including some 146 spe-cies in 23 families, helps to explain BWYV's wide distribution. While difficult to control, new lettuce planting should be spatially and temporally separated from crops exhibiting infection as much as feasible.

4.2 Cucumber mosaic

Cucumber mosaic cucumovirus (CMV) is the type member of the *cucumovirus* group. Although usually a minor disease on lettuce, it can be a significant problem in some temperate areas. In New York state, CMV is considered to be the most important viral disease of lettuce (Bruckhart and Lorbeer 1975). Distributed worldwide (Brunt *et al.* 1996), symptoms on lettuce include plant stunting, yellow mottling, leaf distortion, and necrotic spots on leaves (Thompson and Proctor 1966). CMV symptoms may be very difficult to differentiate from those incited by lettuce mosaic virus. CMV has one of the most extensive host ranges of any plant pathogen, including over 800 species of monocots and dicots, including many weeds commonly found in and surrounding lettuce production fields. CMV is transmitted in a non- persistent (non-circulative) stylet-borne fashion by more than 60 aphid species, including the green peach aphid (*Myzus persicae* Sulzer) and the cotton aphid (*Aphis gossypii* Glover). The virus may also be mechanically transmitted via sap. While CMV seed transmission has not been reported in lettuce, it has been detected in other hosts.

The non-persistent mode of aphid transmission and the extensive host range of CMV make it extremely difficult to control. Host plant resistance offers perhaps the most potential as a means of control. CMV resistance has been identified in *Lactuca saligna* L. and this resulted in the release of the CMV-resistant cv. Saladcrisp (Robinson and Provvidenti 1993). Although the resistance in Saladcrisp was soon overcome by new strains, additional sources of resistance have been identified, and it is hoped that more durable resistance will be forthcoming.

Other management practices which offer some benefit but not absolute con-trol are to avoid planting new lettuce near old or recently harvested lettuce or cucur-bit fields, proper weed management, and the application of mineral oils which may interfere with virus transmission. Such applications have been reported to slow CMV progress, but the need for frequent application may severely limit there feasibility.

4.3 Lettuce big-vein

Lettuce big-vein (LBV) may be the most ubiquitous lettuce disease for which there is presently no control (Falk 1997). The most obvious symptom is a clearing along the veins, making them appear larger than normal. Affected leaves frequently pucker and leaf margins may appear ruffled. Leaves of infected head lettuce often have an upright growth habit, making it more closely resemble leaf lettuce. Lettuce infected at an early growth stage with LBV may be severely stunted or delayed in their matu-rity, while plants infected in the late rosette stage may possess conspicuous vein-

clearing symptoms yet remain marketable.

There are questions concerning the exact etiology of LBV, but the cause is believed to be a virus (Vetten *et al.* 1987). What is known is that LBV is associated with the soilborne chytrid fungus, *Olpidium brassicae* (Woronin) P. A. Dang (Campbell and Grogan 1963). This organism has a wide host range and can persist in the soil for extended periods of time. The fungus infects the epidermal cells of lettuce roots and remains there, producing sporangia under moist conditions. These release motile zoospores into the soil, which may infect other plants. If the fungus had acquired the LBV agent, LBV may then be transmitted by the viruliferous zoospores. In the absence of the fungal vector, the LBV agent can be graft-transmitted from an infected plant to a healthy plant.

LBV is favored by cool temperatures and moist soils. Seedlings may become infected as early as 8 days after planting and symptoms are apparent in as few as 18 days following infection. Disease incidence of 100% has been observed in some cases. Management of LBV has focused on the fungal vector. While fumigation may reduce the incidence, it remains impractical. Tolerant, but not resistant varieties have been identified.

4.4 Lettuce infectious yellows

Vectored by the sweet potato whitefly, *Bemisia tabaci* (Gennadius), *lettuce infectious yellows closterovirus* (LIYV) became well-established in the southwestern U.S. during the early 1980s. Disease incidence reached 100% in some fields of desert lettuce (Duffus *et al.* 1986). Epidemics have subsided due to a change in whitefly populations and changing cropping patterns, but it is still observed occasionally. LIYV is characterized by leaf yellowing, rolling, brittleness and plant stunting.

LIYV transmission is specific to *B. tabaci*, which vectors the virus in a semi-persistent manner. Both LIYV and its whitefly vector have extensive host ranges, making the epidemiology of the disease complex. Management strategies which prevent the buildup of whitefly populations and cropping practices which discourage their ensuing migration from a LIYV susceptible host to lettuce should be employed.

4.5 Lettuce mosaic virus

Lettuce mosaic is one of the most common and potentially devastating diseases of lettuce. Caused by the *Lettuce mosaic potyvirus* (LMV), the disease was first reported in Florida, but has since been reported worldwide (Dinant and Lot 1992). LMV is the most important viral disease in California, the world's foremost lettuce growing region (Grogan 1980). Symptoms may vary widely, depending upon a number of factors, including: cultivar or lettuce type, crop age when infected, and environmental conditions. Initial leaves of plants originating from LMV-infected seed are irregularly shaped, exhibiting a light green mottle or mosaic. Mottling and mosaic are usually more pronounced on leaf lettuce than on crisphead cultivars. Leaf vein-clearing and necrotic flecking may also appear. Growth of infected plants is stunted and frequently, distorted. Early infection of crisphead varieties may result in small, unmarketable heads.

Infected heads may appear dull green, with outer leaves developing a characteristic downward curling. The downward curling symptom may help to distinguish LMV infections from CMV infections in the field.

LMV has a rather wide host range, including as many as 25 genera in 12 families (Brunt *et al.* 1996). The virus is transmitted in a non-persistent manner by a number of aphid species, including the green peach (*Myzus persicae* Sulzer) and the cotton aphid (*Aphis gossypii* Glover). LMV may also be mechanically transmitted in sap, and is the only economically important virus of lettuce that is seed transmitted. Four distinct pathotypes of the pathogen have been identified based upon their ability to infect a series of lettuce differential cultivars (Zerbini *et al.* 1995). Pathotypes I and II are unable to overcome the *mo* and *g* LMV resistance genes, while pathotypes III and IV can infect cultivars containing *mo* and *g* resistance genes. Pathotype II is prevalent in the U.S., while pathotypes III and IV are predominant in Europe.

Management of LMV has been successfully accomplished through the use of virus-indexed seed and/or host-plant resistance. In the U.S., a threshold of 0 infected seeds in 30,000 tested has been established, while for the Netherlands, it is 0 in 2,000 seeds tested. Indexing is commonly done by certified labs using Enzyme-Linked Immunosorbent Assay (ELISA). To complement the planting of indexed seed, a lettuce-free period of at least two weeks is recommended, along with adequate weed management. The success of this integrated pest management (IPM) program using seed certification has made it an IPM model system.

LMV resistant cultivars are more heavily relied upon for control of LMV in Europe, South America, and lettuce-growing regions other than the U.S.

4.6 Tomato spotted wilt

Tomato spotted wilt, a serious disease of numerous important food and ornamental crops worldwide, also infects lettuce. Early infections can cause plant death, with older infections producing marginal wilting, chlorosis, and necrotic spotting on leaves and petioles. Infections frequently affect one side of the plant, resulting in a twisted appearance.

Caused by *Tomato spotted wilt tospovirus* (TSWV), the disease is transmitted by several species of thrips in a circulative-propagative manner. Three genera of thrips, *Frankliniella, Scirtothrips*, and *Thrips*, have been linked to transmission, with the western flower thrips, *Frankliniella occidentalis* (Pergande), being the most important vector species (German *et al.* 1992). The insect acquires the virus as it molts, and undergoes a period of 4-12 days prior to becoming infective. Thrip larvae may transmit TSWV before or after they pupate, with winged adults disseminating the disease to other areas. Adult thrips cannot acquire the virus and TSWV-infected adults cannot pass it on to their offspring. The pathogen can only be acquired by the larval stage.

A multidisciplinary approach to TSWV management was devised by Cho *et al.* (1989) in Hawaii, where TSWV has caused significant lettuce losses. Cultural management strategies are grouped in relation to the crop cycle. Pre-crop strategies include: 1) crop rotation with nonsusceptible crops to reduce inoculum buildup, 2) crop deployment to avoid planting susceptible crops adjacent to one another, and 3) control

of alternate weed hosts. Crop phase strategies consist of: (i). use of virus-free seed-lings, (ii). insecticide applications, and (iii) reduced in-field cultivation to minimize thrip disturbance. Post-crop management includes: (i). fallowing fields for 3-4 weeks to allow thrips to emerge and disperse from the area, and (ii). soil fumigation to elimi-nate thrips associated with crop debris. Host-plant resistance is under investigation but is not yet available.

4.7 Aster yellows

First described on lettuce in 1916, *aster yellows* affects more than 300 plant species (Frazier and Severin 1945). Symptoms include blanching and chlorosis of young heart leaves. Center leaves fail to develop normally, remaining as shortened stubs in the central rosette. Outer leaves become chlorotic and twisted, and pinkish-tan latex drop-lets may form on the underside of midribs. In seed crops, the disease results in sterile, aborted flowers (Severin and Frazier 1945).

 Caused by the *aster yellows* phytoplasma, the disease is widespread but yield losses on lettuce are only sporadic. Phytoplasmas (formerly known as mycoplasma-like organisms or MLOs) are pleomorphic, unicellular prokaryotes. Bound by a unit membrane, they lack a true cell wall. Phytoplasmas colonize phloem sieve and paren-chyma cells and reproduce by binary fission, budding or fragmentation in the plant host or vector.

 The principal vector of the *aster yellows* phytoplasma is the aster leafhopper, *Macrosteles quadrilineatus* Forbes, although the disease can be transmitted by other leafhopper species. The pathogen overwinters in the body of its insect vector and in perennial or biennial weeds. Leafhoppers may remain inoculative for more than 100 days.

 Control of aster yellows in lettuce is seldom warranted. However, the destruc-tion of weed reservoirs and management of insect vectors can assist in keeping the disease in check (Davis 1997). Aster yellows resistant varieties have not yet been reported.

5. Diseases caused by nematodes

Nematodes are nonsegmented worms that inhabit plants, animals, soil, and water, with plant parasitic nematodes usually being microscopic (15-35 x 300-1,000 µm). Nema-todes that attack lettuce are typically root pathogens using a needle-like stylet to with-draw nutrients from the plant. A number of nematode species have been demonstrated to reduce lettuce growth and yield. These include the lesion nematode (*Pratylenchus penetrans* (Cobb) Filipjev & Schuurmans Stekhoven), the needle nematode (*Longidorus africanus* Merny), and the root-knot nematode (*Meloidogyne* spp.). Several additional nematodes have been associated with lettuce but are not covered in this chapter due to limited range or significance. These include: the reniform nematode (*Rotylenchulus reniformis* Linford & Oliveira), the spiral nematode (*Rotylenchus robustus* (de Man) Filipjev), the stunt nematode (*Tylenchorhynchus clarens* (Allen) and *Merlineus* spp.) and the stubby root nematode (*Paratrichodorus minor* (Colbran) Siddiqi).

5.1 Lesion nematode

The lesion nematode has been reported as a serious pest of lettuce in the northeastern United States and Canada. *Pratylenchus penetrans* is the lesion nematode most closely associated with lettuce (Olthof and Potter 1973). This nematode species causes narrow reddish brown root lesions consisting of dead or damaged cells. High nematode populations may produce above-ground symptoms such as chlorosis and stunting. As with many nematode diseases, environmental stress factors such as drought, low fertility, or cool temperatures accentuate lesion nematode damage. Lesion nematodes have a typical life-cycle of 35-42 days with both juvenile and adult nematodes capable of migrating in and out of lettuce roots.

Lesion nematodes may survive from season to season in old root tissue. Having an extensive host range exceeding 150 plant species, the nematode is seldom controlled by crop rotation. Populations may be reduced through the use of fumigants or nematicides but these measures are seldom of economic benefit. Fallowing may assist in reducing initial densities to below the 2-6 nematodes per gram of soil damage threshold (Potter and Olthof 1974).

5.2   Needle nematode

The needle nematode, *Longidorus africanus*, has been reported in California , in Africa, and Israel. An ectoparasite, the needle nematode remains outside the root tissue, feeding on root tips. This feeding causes a swelling and distortion of root tissues, which die prematurely. Reduction in the numbers of functional feeder roots frequently results in proliferation of lateral roots as a means of compensation. Lettuce root systems in heavily infested fields are branched, and significantly shortened. Above ground symptoms resemble drought stress, with plants as early as the cotyledon stage exhibiting wilting. Even in the presence of adequate moisture, leaves may turn a dull grayish green with chlorotic to necrotic margins. Early damage may reduce head size and delay maturity.

Like the lesion nematode, the needle nematode has a wide host range (Kolodge *et al.* 1987), limiting the effectiveness of crop rotation. Weeds should be eliminated in infested fields, since many of these may be host species. Planting when soil temperatures are below 22°C may also significantly reduce needle nematode damage to seedlings.

5.3 Root-knot nematode

Several different species of root-knot nematodes have been reported as parasites on lettuce. These include *Meloidogyne hapla* Chitwood, *M. incognita* (Kofoid & White) Chitwood, *M. javanica* (Treub) Chitwood, and *M. arenaria* (Neal) Chitwood.   All produce characteristic round to spindle-shaped galls or swellings on the roots. Root galls hold mature female nematodes, which appear as tiny white pearls (1.5 mm diam.) when galls are cut open.   Galled lettuce roots are typically shortened and may have fewer root hairs than healthy roots. Above ground symptoms include plant stunting,

yellowing, and even wilting. Lettuce stands in severely infested fields may appear as being very erratic and uneven.

Economic damage due to root-knot nematodes may be significant and is directly related to the size of the initial nematode population at planting (Wong and Mai 1973a). Crop rotation or periods of weed-free fallow may be effective in reducing root-knot nematode populations, however the nematode's broad host range limits the number of rotation choices. Soil treatment with fumigants or nematicides has proven effective in reducing nematode populations prior to planting (Radewald *et al.* 1969). Damage due to root-knot nematodes is usually more severe in muck and coarse-textured soils than in clay soils, presumably due to the nematode's greater mobility in these soils (Wong and Mai 1973b). While soil temperature is important in the phenology of root-knot nematodes, the optimum range varies depending on species. Reported optimum ranges are 15-25°C for *M. hapla* and 25-30°C for *M. arenaria*, *M. incognita*, and *M. javonica* (Wong and Mai 1973c).

6. Abiotic or non-infectious biotic disorders

Many disorders and injuries of lettuce are due to abiotic or non-infectious biotic causes. Due to space limitations, only three are covered in this chapter. Primarily disorders of mature or postharvest lettuce, these are brown stain, pink rot, and tipburn. Other significant abiotic or non-infectious biotic disorders not covered include: air pollution, birds, bolting, genetic abnormalities, mineral deficiencies and/or toxicities, pesticide-related injury, rib blight, soil crack, soil pH and salinity, and weather-related injuries (Davis *et al.* 1997). Individual field losses due to such causes may be total, but they are frequently confined to isolated events or locales.

6.1 Brown stain

Caused by exposure of crisphead lettuce to elevated levels of CO_2, brown stain lesions are oval, brown, and slightly sunken. Chlorophyll-free midribs at the base of lettuce leaves are most susceptible. Brown stain severity increases as CO_2 concentrations in storage atmospheres increase from 1 to 5%, and as O_2 concentrations decrease from 21 to 1% (Ke and Saltveit 1989).

Brown stain can be avoided by maintaining CO_2 levels below 2% and O_2 levels above 10%. Lettuce harvested at the proper maturity and selected varieties are more resistant to brown stain.

6.2 Pink rib

Pink rib is a postharvest disorder of crisphead lettuce only, most commonly occurring in overmature heads. Although the cause is unknown, low O_2 concentrations and elevated storage temperatures are thought to promote its development (Marlatt and Stewart 1956). Like brown stain, it is most prominent on the chlorophyll-free midrib tissues at the base of outer leaves (Snowdon 1992). Pink rib may extend into the large veins of all but the youngest of leaves when severe. Its occurrence may be reduced by

harvesting at the proper maturity and preventing CO_2 accumulation.

6.3 Tipburn

Tipburn is an abiotic disorder characterized by marginal collapse and necrosis at or near the margins of rapidly expanding inner leaves. It is associated with low tissue concentration of calcium, and an imbalance of other nutrients (Sonneveld and Mook 1983). Tipburn predisposing factors include: warm temperatures, excessive fertility (particularly nitrogen), and an increase in light intensity. Tipburn frequently manifests itself near harvest. Crop losses of 100% can occur.

Management strategies for tipburn include production practices that avoid rapid and excessive growth. Foliar sprays of calcium may provide some reduction of tipburn incidence in open-head lettuce types but not crisphead lettuce. Calcium sources added to the soil are usually ineffective due to slow translocation. Relatively resistant varieties are presently being developed (Nagata and Ryder 1997).

7. References

Abawi, G. S. and Grogan, R. G. 1979. Epidemiology of disease caused by *Sclerotinia* species. Phytopathology, 69:899-904.

Alvarez, J., Datnoff, L. E. and Nagata, R. T. 1992. Crop rotation minimizes losses from corky root in Florida lettuce. HortScience, 27:66-68.

Barak, J. D., Koike, S. T. and Gilbertson, R. L. 2001. The role of crop debris and weeds in the epidemiology of bacterial leaf spot of lettuce in California. Plant Disease, 85:169-178.

Barak, J. D., Koike, S. T. and Gilbertson, R. L. 2002. Movement of *Xanthomonas campestris* pv. *vitians* in the stems of lettuce and seed contamination. Plant Pathology, 51:506-512.

Brown, N. A. 1918. Some bacterial diseases of lettuce. Journal of Agriculture Research, 13:367-388.

Bruckhart, W. L. and Lorbeer, J. W. 1975. Recent occurrences of cucumber mosaic, lettuce mosaic, and broad bean wilt viruses in lettuce and celery fields in New York. Plant Disease Reporter, 59:203-206.

Brunt, A. A., Crabtree, K., Dallwitz, M. J., Gibbs, A. J. and Watson, L. (eds.) 1996. Viruses of Plants. CAB International, Wallingford, UK. 1484 p.

Campbell, R. N. and Grogan, R. G. 1963. Big-vein virus of lettuce and its transmission by *Olpidium brassicae*. Phytopathology, 53:252-259.

Ceponis, M. J., Cappellini, R. A. and Lightner, G. W. 1985. Disorders in crisphead lettuce shipments to the New York market, 1972-1984. Plant Disease, 69:1016-1020.

Ceponis, M. J., Kaufman, J. and Butterfield, J.E . 1970. Relative importance of gray mold and bacterial soft rot of western lettuce on the New York Market. Plant Disease Reporter, 54:263-265.

Coley-Smith, J. R., Verhoeff, F. and Jarvis, W. R. (eds.) 1980. The Biology of Botrytis. Academic Press, New York.

Cho, J. J. 1977. Control of bacterial soft rot of crisphead type lettuce in Hawaii. Plant Disease Reporter, 61:783-787.

Cho, J. J. and Talede, K. Y. 1976. Evaluation of lettuce varieties for resistance to bacterial soft rot. Horticulture Digest, 35:4-5.

Cho, J. J., Mau, R. F. L., German, T. L., Hartmann, R. W., Yudin, L. S., Gonsalves, D. and Provvedenti, R. 1989. A multidisciplinary approach to management of tomato spotted wilt

virus in Hawaii. Plant Disease, 73:375-383.

Crute, I. R. and Dixon, R. 1981. Downy mildew diseases caused by the genus *Bremia* Regel. In: "The Downy Mildews" (ed. Spencer, D. M.) Academic Press, London. pp. 422-460.

Datnoff, L. E. and Nagata, R. T. 1992. Relationship between corky root disease and yield of crisphead lettuce. Journal of the American Society of Horticultural S c i e n c e , 117:54-58.

Davis, R. M. 1997. Aster yellows. In: "Compendium of Lettuce Diseases" (eds. Davis, R. M., Subbarao, K. V., Raid, R. N., and Kurtz, E. A.) APS Press, St. Paul, MN. pp. 51.

Davis, R. M., Subbarao, K. V., Raid, R. N. and Kurtz, E. A. (eds.) 1997. Compendium of Lettuce Diseases. APS Press, St. Paul, MN. 79 p.

Dinant, S. and Lot, H. 1992. Lettuce mosaic virus: A review. Plant Pathology, 41:528-542.

Duffus, J. E., Larsen, R. C. and Liu, H. Y. 1986. Lettuce infectious yellows virus – A new type of whitefly-transmitted virus. Phytopathology, 76:97-100.

Fahy, P. C. and Persley, G. J. 1983. Plant Bacterial Diseases: A Diagnostic Guide. Academic Press, New York.

Falk, B. W. 1997. Lettuce Big-Vein. In: "Compendium of Lettuce Diseases" (eds. Davis, R. M., Subbarao, K. V., Raid, R. N. and Kurtz, E. A.) APS Press, St. Paul, MN. pp. 41-42.

Frazier, N.W. and Severin, H. H. P. 1945. Weed-host range of California aster yellows. Hilgardia, 16:619-650.

Galea, V. J., Price, T. V. and Sutton, B. C. 1986. Taxonomy and biology of the lettuce anthracnose fungus. Transactions of the British Mycological Society, 86:619-628.

German, T. L., Ullman, D. E. and Moyer, J.W. 1992. *Tospoviruses*: Diagnosis, molecular biology, phylogeny, and vector relationships. Annual Review of Phytopathology, 30:315-348.

Grogan, R. G. 1980. Control of lettuce mosaic with virus-free seed. Plant Disease, 64:446-449.

Grogan, R. G., Misaghi, I. J., Kimble, K. A., Greathead, A. S., Ririe, D. and Bardin, R. 1977. Varnish spot, a destructive disease of lettuce in California caused by *Pseudomonas cichorii*. Phytopathology, 67:957-960.

Herr, L. J. 1992. Characteristics of *Rhizoctonia* isolates associated with bottom rot of lettuce in organic soils in Ohio. Phytopathology, 82:1046-1050.

Herr, L. J. 1993. Host sources, virulence and overwinter survival of *Rhizoctonia solani* anastomosis groups isolated from field lettuce with bottom rot symptoms. Crop Protection,12:521-526.

Hine, R., Matheron, M. and Byrne, D. 1991. Diseases of lettuce in Arizona. Univ. of Arizona College of Agriculture Publication 191050.

Hovius, M. H. Y. and McDonald, M. R. 1999. Field evaluation of forecasting systems to optimize fungicide applications for downy mildew of lettuce, 1998. American Phytopathological Society, Fungicide and Nematicide Tests, 54:146-147.

Imolehin, E. D. and Grogan, R. G. 1980. Factors affecting survival of sclerotia, and effects of inoculum density, relative position, and distance of sclerotia from the host on infection of lettuce by *Sclerotinia minor*. Phytopathology, 70:1162-1167.

Inglis, D., Derie, M., Gundersen, B. and Vestey, E. 1999. Evaluation of fungicides for lettuce anthracnose control, 1998. American Phytopathological Society, Fungicide and Nematicide Tests, 54:149.

Katan, J. and DeVay, J.E. (eds.) 1991. Soil Solarization. CRC Press, Boca Raton. 267 p.

Ke, D. and Saltveit, M. E. 1989. Carbon dioxide-induced brown stain development as related to phenolic metabolism in iceberg lettuce. Journal of the American Society of Horticultural Science, 114:789-794.

Kolodge, C., Radewald, J. D. and Shibuya, F. 1987. Revised host range and studies on the life

cycle of *Longidorus africanus*. Journal of Nematology, 19:77-81.

Lindqvist, K. 1960. On the origin of cultivated lettuce. Hereditas, 46:319-350.

Mahr, S. E. R., Stevenson, W. R. and Sequeira, L. 1986. Control of bottom rot of head lettuce with iprodione. Plant Disease,70:506-509.

Marlatt, R. B. and Stewart, J. K. 1956. Pink rib of head lettuce. Plant Disease Reporter, 40:742-743.

Matheron, M. E. and Porchas, M. 1999a. Comparative efficacy of fungicides for management of downy and powdery mildew on lettuce, 1998. American Phytopathological Society, Fungicide and Nematicide Tests, 54:150-151.

Matheron, M. E. and Porchas, M. 1999b. Evaluation of a new fungicides for management of Sclerotinia leaf drop of lettuce, 1998. American Phytopathological Society, Fungicide and Nematicide Tests, 54:152.

Matheron, M. E. and Porchas, M. 2000a. Evaluation of new fungicides for management of Sclerotinia leaf drop of lettuce, 1999. American Phytopathological Society, Fungicide and Nematicide Tests, 54:167.

Matheron, M. E. and Porchas, M. 2000b. Management of powdery mildew on lettuce with fungicides, 1999. American Phytopathological Society, Fungicide and Nematicide Tests, 54:168.

Nagata, R. T. and Ryder, E. J. 1997. Tipburn. In: "Compendium of Lettuce Diseases" (eds. Davis, R. M., Subbarao, K. V., Raid, R. N., and Kurtz, E. A.) APS Press, St. Paul, MN. pp. 67-68.

0'Brien, R. D. and van Bruggen, A. H. C. 1992. Yield losses to iceberg lettuce due to corky root caused by *Rhizomonas suberifaciens*. Phytopathology, 82:154-159.

Olthof, T. H. A. and Potter, J. W. 1973. The relationship between population densities of *Pratylenchus penetrans* and crop losses in summer-maturing vegetables in Ontario. Phytopathology, 63:577-582.

Palti, J. and Katan, J. 1997. Effect of cultivation practices and cropping systems on soilborne disease. In: "Soilborne Diseases of Tropical Plants" (eds. Hillocks, R. J., and Waller, J. M.) CAB International, University Press, Cambridge. pp. 377-396.

Patterson, C. L. and Grogan, R. G. 1985a. The source and survival of primary inoculum produced by *Microdochium panattoniana*, the causal agent of lettuce anthracnose. Phtyopathology, 75:1319.

Patterson, C. L. and Grogan, R. G. 1985b. Differences in epidemiology and control of lettuce drop caused by *Sclerotinia minor* and *S. sclerotiorum*. Plant Disease, 69:766-770.

Patterson, C. L., Grogan, R. G. and Campbell, R. N. 1986. Economically important diseases of lettuce. Plant Disease, 70:982-987.

Pernezny, K., Raid, R. N., Stall, R. E., Hodge, N. C. and Collins, J. 1995. An outbreak of bacterial spot of lettuce in Florida caused by *Xanthomonas campestris* pv. *vitians*. Plant Disease, 79:359-360.

Pieczarka, D. J. and Lorbeer, J. W. 1975. Micoroorganisms associated with bottom rot of lettuce grown on organic soil in New York State. Phytopathology, 65:16-21.

Potter, J. W. and Olthof, T. H. A. 1974. Yield losses in fall-maturing vegetables relative to population densities of *Pratylenchus penetrans* and *Meloidogyne hapla*. Phytopathology, 64:1072-1075.

Punja, Z. K. 1985. The biology, ecology, and control of *Sclerotium rolfsii*. Annual Review of Phytopathology, 23:97-127.

Radewald, J. D., Mowbray, P. G., Paulus, A. O., Shibuya, F. and Rible, J. M. 1969. Preplant soil fumigation for California head lettuce. Plant Disease Reporter, 53:385-389.

Raid, R. N. and Datnoff, L. E. 1990. Loss of the EBDC fungicides: Impact on control of lettuce downy mildew. Plant Disease, 74:829-831.

Raid, R. N., Datnoff, L. E., Schettini, T. M. and Michelmore, R. W. 1990. Insensitivity of *Bremia lactucae* to metalaxyl on lettuce in Florida. Plant Disease, 74:81.

Raid, R. N. and Nagata, R. T. 2003. Evaluation of fungicides for control of Cercospora leaf spot and downy mildew on lettuce, 2002. APS Fungicide and Nematicide Tests, 58: (*in press*).

Robinson, R. W. and Provvidenti, R. 1993. Breeding lettuce for viral resistance. In: "Resistance to Viral Diseases of Vegetables" (ed. Kyle, M. M.) Timber Press, Inc., Portland, OR. pp. 61-79 .

Rodriguez-Kabana, R. and Kokalis-Burelle, N. 1997. Chemical and biological control. In: "Soilborne Diseases of Tropical Plants" (eds. Hillocks, R. J., and Waller, J. M.) CAB International, University Press, Cambridge. pp. 397-418.

Ryder, E. J. 1979. Leafy Salad Vegetables. AVI, Westport, CT.

Savery, S. 1983. Epidemiology of Cercospora leaf spot of lettuce in Republic of the Ivory Coast. Agronomie, 3:903-909.

Scherm, H. and van Bruggen, A. H. C. 1994. Effects of fluctuating temperatures on the latent period do lettuce downy mildew (*Bremia lactucae*). Phytopathology, 84:853-859.

Scherm, H. and van Bruggen, A. H. C. 1995. Comparative study of microclimate and downy mildew development in subsurface drip- and furrow-irrigated lettuce fields in California. Plant Disease, 79:620-625.

Scherm, H., Koike, S. T., Laemmlen, F. F. and van Bruggen, A. H. C. 1995. Field evaluation of fungicide spray advisories against lettuce downy mildew (*Bremia lactucae*) based on measured and forecast morning leaf wetness. Plant Disease, 79:511-516.

Schettini, T. M., Legg, E. J. and Michelmore, R. W. 1991. Insensitivity to metalaxyl in California populations of Bremia lactucae and resistance of California lettuce cultivars to downy mildew. Phytopathology, 81:64-70.

Schnathorst, W. C. 1959. Spread and life cycle of the lettuce powdery mildew fungus. Phytopathology, 49:464-468.

Schnathorst, W. C. 1960. Effects of temperature and moisture stress on the lettuce powdery mildew fungus. Phytopathology, 50:304-308.

Severin, H. H. P. and Frazier, N. W. 1945. California aster yellows on vegetable and seed crops. Hilgardia,16:573-596.

Smith, P. R. 1961. Seedborne *Septoria* in lettuce: Eradication by hot water treatment. Journal of Agriculture, 59:555-556.

Snowdon, A. L. 1992. Color Atlas of Post-harvest Diseases and Disorders of Fruit and Vegetables. Vol. 2, Vegetables. CRC Press, Boca Raton, FL.

Sonneveld, C. and Mook, E. 1983. Lettuce tipburn as related to the cation contents of different plant parts. Plant and Soil, 75:29-40.

Stewart, J. K. 1978. Influence of oxygen, carbon dioxide, and carbon monoxide levels on decay of head lettuce after harvest. Scientia Horticulture, 9:207-213.

Subbarao, K. V., Koike, S. T. and Hubbard, J. C. 1996. Effects of deep plowing on the distribution and density of *Sclerotina minor* sclerotia and lettuce drop incidence. Plant Disease, 80:28-33.

Thompson, A. D. and Proctor, C. H. 1966. Cucumber mosaic virus in lettuce. New Zealand Journal of Agriculture Research, 9:142-144.

van Bruggen, A. H. C., Brown, P. R., Shennan, C., and Greathead. A. S. 1990. The effect of cover crops and fertilization with ammonium nitrate on corky root of lettuce. Plant Disease,74:584-589

van Bruggen, A. H. C. and Rubatzky, V. E. 1992. Use of transplants instead of direct seeding to reduce corky root severity and losses due to corky root in iceberg lettuce. Plant Disease, 76:703-708.

Vetten, H. J., Lesemann, D. E. and Dalchow, J. 1987. Electron microscopical and serological detection of virus-like particles associated with lettuce big-vein disease. Journal of Phytopathology, 120:53-59.

Wang, Z.-N., Coley-Smith, J. R. and Wareing, P. W. 1986. Dicarboximide resistance in *Botrytis cinerea* in protected lettuce. Plant Pathology, 35:427-433.

Wong, T. K. and Mai, W. F. 1973a. Pathogenicity of *Meloidogyne hapla* to lettuce as affected by inoculum level, plant age at inoculation and temperature.. Journal of Nematology, 5:126-129

Wong, T. K. and Mai, W. F. 1973b. *Meloidogyne hapla* in organic soil: Effects of environment on hatch, movement and root invasion. Journal of Nematology, 5:130-138.

Wong, T. K. and Mai, W. F. 1973c. Effect of temperature on growth, development and reproduction of *Meloidogyne hapla* in lettuce. Journal of Nematology, 5:139-142.

Zerbini, F. M., Koike, S. T. and Gilbertson, R. L. 1995. Biological and molecular characterization of *lettuce mosaic potyvirus* isolates for the Salinas Valley of California. Phytopathology, 85:746-752.

Management of Diseases of Onions and Garlic

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Abstract: Onions and garlic are the most popular cultivated forms of the family Alliaceae and consumed by almost every culture on earth. According to the 2001 statistics of the FAO, onions are second only to tomatoes in value of vegetable crops cultivated worldwide. There are a number of pathogens attack onions and garlic throughout their developmental stages since the developmental process is important in the progression of diseases. Onions are normally propagated from seed, planted directly into the field, although onion sets and transplants are also used. Garlic has been vegetatively propagated for millennia, by planting cloves. Vegetative propagation results in additional disease management problems. This chapter covers disease management of many of the major bacterial, fungal and viral diseases of onions and garlic, with an emphasis on those diseases that have been the topic of disease management research or for which effective disease management systems have been put into practice. Short descriptions of the pathogens and symptoms are included to assist in identification of the specific diseases.

1. Introduction to Onions and Garlic

Onions and garlic are the most popular cultivated forms of the family Alliaceae, although many other *Allium* species are grown for food and as ornamental crops. The bulb onion (*Allium cepa* L.) is the most widely cultivated species of the genus *Allium,* and is grown from Scandinavia to the humid tropics, although the great majority of the production occurs in temperate and sub-tropical regions (Goldman 2001). According to the 2001 statistics of the FAO, onions are second only to tomatoes in value of vegetable crops cultivated worldwide. Indeed, almost every culture on earth consumes onions.

The edible portion of both the onion and garlic bulb consists of expanded leaf scales. In garlic the storage tissues of the bulb are specially modified bladeless leaves called "bulb scales". In onion the outer swollen sheaths of the bulb derive from bladed leaves, while the inner ones are bulb scales (Brewester 1994). The association between bulb scales and leaves is especially important in the development of many bacterial diseases of onion, since infections that begin on the leaves often travel down the leaves to the bulb.

Pathogens attack onions and garlic throughout their developmental stages. When onion seeds germinate, the single cotyledon emerges in a "knee" or "loop" form, with both ends in the ground. The haustoria end then pulls out of the ground to form a "flag" or "whip" shape. The first true leaf emerges from a pore that forms near the base of the cotyledon, and subsequent leaves emerge from the center of the plant through similar pores at the base of the next oldest leaf. The developmental process is important in the progression of onion smut and neck rot within the plant. The true stem is a flattened cone at the base of the bulb. Roots radiate out from this basal plate.

Onions are normally propagated from seed, planted directly into the field, although onion sets and transplants are also used. Garlic has been vegetatively propagated for millennia, by planting cloves. Vegetative propagation results in additional disease management problems. Commercial production of garlic continues to depend on vegetative propagation, although true seed has been developed (Etoh *et al.* 1988, Pooler and Simon 1994).

This chapter will cover disease management of many of the major diseases of onions and garlic, with an emphasis on those diseases that have been the topic of disease management research or for which effective disease management systems have been put into practice. Short descriptions of the pathogens and symptoms are included to assist in identification of the specific diseases. There are several good references for the identification and control of diseases of onions and garlic. These include: Allium Crop Diseases (Rabinovitch and Currah 2002), The Color Atlas of Pos-Harvest Diseases and Disorders of Fruits and Vegetables Volume 2: Vegetables (Snowdon 1992), Compendium of Onion and Garlic Diseases (Schwartz and Mohan 1995), Diseases and Pests of Vegetable Crops in Canada (Howard *et al.* 1994) and Vegetable Crop Diseases (Dixon 1981).

2. Bacterial Diseases

Bacterial diseases on onions are of major economic importance, with losses occurring in the field, in transport and in storage. Losses occur in the field, reducing yield and quality. Rots can also develop in transit and storage. Any level of bacterial infection of a bulb will render it unmarketable. Often the greatest financial losses occur where the onion bulbs appear disease free on the surface, but when they are cut open a bacterial infection is found that may damage or discolour one or two scales within the onion bulb. While consumers may complain if onions with this type of infection are found in onions for fresh consumption, they are of particular concern for processing, since processing procedures cannot effectively sort out discolored rings or onion pieces. Thus, processors may turn down truckloads of onions if this symptom is found, or refuse to purchase onions from a particular field or grower.

Bacteria species have been associated with rots, necrosis, and blights of onion, garlic, leeks and chives as either primary pathogens or as secondary colonizers following infection by fungal pathogens such as *Botrytis* spp., *Aspergillus niger*, *Fusarium oxysporum* f. sp. *cepa*, and *Alternaria porri*.

Bacterial diseases normally develop during wet periods and prefer warm tem-

peratures, free water and damaged tissue, before or during harvest. Effective management of bacterial diseases of onion depends primarily on avoidance and cultural practices since there are few effective bactericides available. Crop rotation and removing crop debris, culls and volunteer plants may reduce sources of inoculum in the fields. Where overhead irrigation is used, irrigation should be scheduled to prevent over watering and avoid favourable conditions for the development of the disease. For instance, irrigation should be done at night when temperatures are cooler. It is also important to avoid overhead irrigation late in the season, keep leaf and bulb damage to a minimum and conduct fieldwork when the onion canopy is dry. Harvest onions when the bulbs are fully mature, the leaves and neck dry and when the weather is dry. Bacterial diseases can spread quickly during harvest if the necks of onion or galic plants are green and moist or if bulbs are damaged. Efforts should be made to reduce sunscald, bruising and mechanical damage during harvest. Rapid and thorough curing will also reduce disease development and spread. The high temperatures associated with artificial curing may temporarily encourage rotting, but heat curing can be important for successful long-term storage. Subsequent cold storage will halt further multiplication of the bacteria. Onion bulbs should be stored at 0ºC and 65 to 75% relative humidity. Adequate ventilation must be provided during storage so moisture is not allowed to condense on the bulbs.

There are few, if any onion cultivars that are resistant to bacterial diseases; however, Spanish onions tend to be more susceptible to many bacterial diseases than common onion (Tesoriero *et al.* 1982).

A preventive program with copper bactericides, where registered, may be recommended as part of an integrated pest management (IPM) program. Products containing copper, or copper mixed with mancozeb, have been recommended to reduce the spread of bacterial diseases in the field. The effectiveness of these products is variable, and there is an on-going debate as to their usefulness. However, when no other products are available these may be seen as a better alternative than doing nothing.

The following is a summary of the main bacterial diseases of onion and garlic. The common names used for the bacterial diseases are those used for the American Plant Pathology Society (APS).

2.1 Slippery Skin

The disease is caused by *Burkholderia gladioli* pv. a*lliicola* (formerly *Pseudomonas gladioli* pv. *alliicola*). This disease is a problem in the southern United States and other areas with warm climates, and also has been observed in temperate regions. At harvest, there are often no external symptoms of the disease and the onion bulbs must be cut to detect symptoms. Affected bulbs may be unmarketable, especially for processing. *B. gladioli* is not a problem on other *Allium* crops.

2.1.1 Symptoms

In the field, infected onion plants may have one or two wilted leaves in the middle of the leaf cluster. Later, the wilted leaves turn light yellow to off-white and tips die back. Older

and younger leaves generally remain green. The bulbs of these onions are usually soft and watery. When pressure is applied to the base of the bulb, the rotted scales slide out through the neck, suggesting the reason for the common name (Tesoriero *et al.* 1982).

In many cases, the disease develops slowly and affected bulbs appear disease free at harvest, until they are cut open. The necks of these bulbs may become softened, after which rot spreads from the neck and the inner scales of disease bulbs are brown and water-soaked, and the bulbs turn soft and rotten (Tesoriero *et al.* 1982).

2.1.2 Causal organism

The causal agent of Slippery Skin, *B. gladioli,* forms a diffusible, non-fluorescent, pale yellow to yellow-green pigment on nutrient-dextrose agar and King's B medium. It is a motile, Gram-negative rod that has one to several polar flagella (Ballard *et al.* 1970, Burkholder 1942).

2.1.3 Disease Cycle

Slippery Skin occurs frequently during seasons with high rainfall or in fields that are heavily irrigated. Infection usually takes place while the crop is actively growing, shortly before harvest, or when the onions are topped. The pathogen is soil-borne and is probably transferred to the leaves by splashing water. Infection of the seed stalks and leaves has been reported in Hungary and India (Tesoriero *et al.* 1982, Fahy *et al.* 1981-1982). Wounds on the leaves may be important points of entry for the pathogen. Matured bulbs are highly susceptible to infection. Accumulation of water in the neck of the onion also may promote infection. Bacteria enter through the leaf tissue and spread down the leaf to the corresponding bulb scale, then progresses down the scale to the base of the bulb before spreading throughout the bulb. High temperatures and slow drying of the bulbs increase infection (Tesoriero *et al.* 1982). The bacteria grow in the range of 5-41°C, with an optimum temperature of 30°C. In warm weather, a bulb can decay in 10 days. Infected bulbs begin to rot within four weeks when stored at 25°C, but not at 15°C. In storage, the disease may take up to three months to destroy a bulb completely (Kishun and Swarup 1981).

Suggested management practices are similar to those outlined above for bacterial diseases.

2.2 Sour Skin

The disease is caused by *Burkholderia cepacia* (also known as *Pseudomonas cepacia*)*,* is a serious disease of processing onions; severe outbreaks can result in rejection of entire lots. Sour Skin was first discovered in 1950 in New York where it has become prevalent (Burkholder 1950). This disease affects only onions.

2.2.1 Symptoms

In the field, softening is observed at the base of the leaf, and a rot develops from small

areas to cover the external scale of the bulb. Affected onions usually have 1 or 2 tan or brown partially rotted leaves. Later, a watery rot develops at the base of the leaf in the bulb neck. Affected leaves can easily be pulled out of the bulb.

In advanced stages of the disease, yellow deposits are observed between the infected scalesand look slimy but not watery. The central scales and scales near the outside of the bulb may be infected in the absence of symptoms on the bulb. Infected scales may separate from adjacent healthy ones. Squeezing the base of an infected bulb may push the central portion out through the neck; however, it is not watery and remains firm unlike bulbs affected by Slippery Skin. Infection in storage becomes evident at the neck end and progresses down in the same manner as the field infection. Secondary organisms such as yeasts and fungi are often associated with this disease and may be contributed to the acrid, vinegar-like odour from which the name "Sour Skin" was derived.

2.2.2 Causal organism

The causal agent, *B. cepacia,* is a motile, Gram-negative rod with rounded ends. It has one to three polar flagella and does not form spores. These bacteria do not form fluorescent pigments, but they do produce a variety of yellowish and greenish pigments, which may diffuse into the culture medium or remain bound to the cells. *B. cepacia* is an obligate aerobe, oxidase positive.

2.2.3 Disease Cycle

B. cepacia has been isolated from organic soils and irrigation water and therefore can be easily spread by overhead irrigation and splashing rain. The bacteria enter plants through wounds or the axils of young leaves or gain entry to bulbs when onions are topped. Infection progresses down the leaf to the corresponding bulb scale. *B. cepacia* does not infect unwounded plants. High rainfall, moderately high temperatures (over 30ºC) and nitrogen before harvest may accelerate the infection.

In New York State, the disease always occurs in conjunction with rainstorms and warm weather. Infected leaves develop a light brown, watery rot in the onion neck. The bacteria spread more quickly in water-soaked tissues than in those that are not congested. Disease is more severe when the weather is warm and humid and the leaves have not completely dried down. There is no evidence that the disease spreads between onions in storage.

2.2.4 Management

Methods used to reduce losses caused by Sour Skin are similar to those for other bacterial diseases, such as Soft Rot and Slippery Skin. The irrigation system seems to be an important factor to consider in managing this disease since the pathogen has been isolated from water and soils. Overhead irrigation should be avoided from bulbing until harvest where possible, since it has been shown to encourage Sour Skin development. Furrow or drip irrigation is less conducive to the development of Sour Skin.

2.3 Bacterial Bulb Decay

It is caused by *Enterobacter cloacae* (Jordan) Hormaeche & Edwards. The disease was first reported in California (Bishop and Davis 1990) and was recently reported in Colorado (Schwartz and Otto 2000a). It appears to be associated with high temperature stress (Bishop and Davis 1990). Mature bulbs exhibit a brownish to black discoloration, loss of turgor and breakdown of inner scales (Schwartz and Otto 2000a). No evidence of disease is present on the exterior of the bulbs.

2.3.1 Causal organism

The causal agent, *E. cloacae* is a Gram negative, rod shaped, cream coloured bacterium in nutrient agar and forms yellow-orange mucoid colonies on MS. This bacterium utilizes glucose in an oxidative and fermentative manner and is catalase positive and oxidase negative. *E. cloacae* is considered to be an opportunistic pathogen of onions, since strains of this bacteria are common components of the microflora in different environments such as water, soil, hospitals, plant surfaces, meat, and intestinal tracts of human and animals, etc. (Bishop and Davis 1990, Hadar *et al.* 1983, Richard 1984).

2.3.2 Disease Cycle

The epidemiology of the disease is poorly understood. The disease has been observed in mature bulbs following a period of extreme heat, 40-45°C (Bishop and Davis 1990). Previous reports have implicated *E. cloacae* in a high temperature internal yellowing of papayas; losses in mung bean sprout production and breakdown of onions in storage (Cother and Dowling 1986). Bishop and Davis (1990) found that at 22°C the disease incidence was less than at 37°C, providing more evidence that temperature is an important factor for the development of this disease. It is believed that the disease usually appears during the last half of the growing season, since symptoms were observed in mature bulbs. The plants may become more susceptible to infection with age. The pathogen is probably transferred to the leaves by splashing water. Wounds or abrasions may be important points of entry for thepathogen.

2.3.3 Management

A three-year crop rotation with no host crops is an important part of the management procedure of this disease as is the sanitation of onion crop debris, culls and volunteer plants to reduce sources of inoculum in the fields. Treatment of seed with streptomycin and only using transplants that are free of pathogens will reduce the risk of infection. If field infection is found, the onions should be air cured at ambient temperature until the neck and outer bulb scales are thoroughly dried. Equipment, trucks and storage bins should be disinfected to reduce the spread of the disease.

2.4 Bacterial Soft Rot

The disease is caused by *Erwinia carotovora* subsp *carotovora* (also known as

Pectobacterium carotovora subsp. *carotovorum* or *Erwinia rhapontici*). Bacterial Soft Rot is one of the major postharvest diseases of onion bulbs causing serious problems to growers and packers. Soft Rot bacteria are a secondary infection pathogen but nevertheless can cause major losses. *E. carotovora* affects many vegetable crops including most cultivated *Allium* species.

2.4.1 Symptoms

The bacteria enter onion necks through wounds or abrasions, injured or dying leaves and may spread directly into one or more of the bulb scales, moving from scale to scale. Affected scales first become spongy, water-soaked and pale yellow to light grey. As the rot develops, the scales become progressively softer and the whole interior of the onion breaks down to form a sticky mass inside the dry outer scales. When the bulb is squeezed, it is soft and watery. A foul-smelling liquid usually oozes out at the neck. When infection occurs at the site of an injury, the rot can progress through several scales from the infection site and the bulb will decay.

2.4.2 Causal organism

E. carotovora is a rod-shaped, Gram-negative bacterium, with peritrichous flagella. It does not form spores and is a facultative anaerobe. Most strains do not produce acid from maltose (Bradbury 1977).

2.4.3 Disease Cycle

Soil is a major source of bacterial contamination. The pathogen survives on infested crop residues in the soil and may be transferred to neighbouring plants by splashing water. Direct contact with infested soil can also result in infection. Soft Rot may develop in the field quickly, especially after heavy rains, when the leaves are dying near the end of the season and there is warm weather. The bacteria require a wound or an infection site of another organism to gain entry to the plant. Onion maggot damage to bulbs is an important entry point for Soft Rot bacteria. Once established, Soft Rot can cause greater damage than the original injury or disease.

Once the bacteria have entered a bulb, they multiply very rapidly at high temperatures. The temperature range for growth is 6 to 37°C, with the optimum between 24 and 32°C in culture and between 18 and 27°C in plants. In storage and transit, the bacteria continue to multiply at temperatures greater than 3°C. High relative humidity and free water also promote the reproduction and spread of Soft Rot bacteria when temperatures are favourable. One infected bulb can stain surrounding bulbs in a pallet box or bag, thereby reducing onion marketability.

2.4.4 Management

General disease management practices for bacterial diseases should be followed. Wherever possible, diseased or damaged onions, especially those with onion maggot dam-

age, should be not stored.

2.5 Bacterial Stalk and Leaf Necrosis

The disease is caused by *Pantoea agglomerans* (also known as *Erwinia herbicola* or *Pseudomonas marginalis* pv *marginalis*). Bacterial stalk and leaf necrosis has only been reported in South Africa (Hattingh and Walters 1981).

2.5.1 Symptoms

Necrosis of the seed stalks is the most reliable symptom; however, leaves can also become infected. The most notable features are the rapid breakdown of the chlorophyll and the green spots that remain in the necrotic areas (Hattingh and Walters, 1981). The infected stalks frequently become weak and then collapse from the weight of the seed heads. Diseased stalks dry out or are broken down by secondary organisms, and seed fails to develop (Hattingh and Walters 1981).

2.5.2 Causal organism

The causal agent, *P.agglomerans*, is a Gram negative, peritrichously flagellated, rod-shaped, yellow-pigmented bacteria (Hattingh and Walters 1981). This bacterium has the ability to invade and grow in healthy plant tissue.

2.5.3 Disease Cycle

Little is known about the disease cycle, but outbreaks appear to result from the combination of high inoculum concentrations of the pathogen and favourable environmental conditions (Hattingh and Walters 1981). Plants inoculated with 10^4 cells of the pathogen produced disease in only 17.5% of the inoculated stalks compared with 100% infection when stalks were inoculated with 10^8 cells. Symptoms started to develop within 3-10 days of inoculation (Hattingh and Walters 1981).

2.5.4 Management

Methods used to reduce losses caused by bacterial stalk and leaf necrosis are similar to those used for other bacterial diseases such as *Xanthomonas* Leaf Blight and Bacterial Bulb Decay.

2.6 Bacterial Internal Browning

The disease is caused by *Pseudomonas aeruginosa* (Schroeter) Migula. Bacterial Internal Browning was first reported in storage in New South Wales, Australia. This disease causes considerable losses in field and storage. The symptoms of the disease are remarkably similar to those of Slippery Skin (Cother *et al.* 1976).

2.6.1 Symptoms

Apparently healthy bulbs show symptoms as yellow-brown to chocolate-red-brown firm necrosis in one or two scales, often extending only one third to half way down the bulb. In some bulbs necrotic tissue may be completely surrounded by healthy scales (Cother and Dowling 1986). The necrotic scales are generally firm except in the neck region where a soft rot can be observed due to secondary infection. At advanced stages of the infection the tissues collapse and disease spreads to the neighbouring bulbs (Cother *et al.* 1976). The symptoms of this disease are remarkably similar to those of Slippery Skin.

2.6.2 Causal agent

P. aeruginosa is known as a widespread soil and plant inhabitant (Schroth *et al.* 1977) and is considered to be an opportunistic pathogen that may infect the plants after adverse conditions (Schroth *et al.* 1977). Bacteria are motile with 1-4 polar flagella, pale dirty-white colony on nutrient agar, that turns the agar to reddish grey and the colonies become translucent and flat with irregular edges after 3 days (Cother *et al.* 1976). The bacteria are unable to utilize sucrose. An important characteristic of this bacterium is the ability to utilize erythritol and creatinine (Cother *et al.* 1976).

2.6.3 Disease Cycle

Extreme conditions such as high temperatures, high humidity and high inoculum concentrations are necessary to infect the plant (Lund 1983). The stage of plant growth at which it enters onion bulb tissue is not known (Cother and Dowling 1986), but fully mature bulbs are more susceptible to infection than immature scales (Cother *et al.* 1976). No infection or hypersensitive reaction was observed in inoculated leaves (Cother *et al.* 1976). The disease frequently occurs in the field before harvest. The bacteria have been isolated from onion field soil and surface drainage water, which are considered sources of inoculum (Kawamoto and Lorbeer 1974). The bacterium is unable to break down the constituents of the cuticle and thus is limited to the scales it initially infects (Cother *et al.* 1976).

2.6.4 Management

Follow the recommended practices for reducing infection and spread of bacteria. Minimizing leaf and bulb damage during growing season will reduce the potential for infection by *P. aeruginosa* and other pathogens.

2.7 Bacterial Leaf Spot and Necrosis

The disease is caused by *Pseudomonas syringae*. The disease occurs in onion producing regions of the southern United States. *P. syringae* is more of a problem in leek, chives and garlic than in onion.

2.7.1 Symptoms

The initial symptoms on seedlings are water-soaked, longitudinal lesions starting at leaf tips and developing down the leaf. The lesions develop chlorotic borders as they expand and elongate resulting in brown, striped lesions with yellow margins. Infected areas initially appear translucent or water-soaked; due to a massive leakage of water out of cells. Eventually the leaves wilt and die. In the field, lesions develop that extend into the sheath, causing the leaves to curl, split and later desiccate. Seriously infected plants could be misshapen and stunted and therefore unmarketable.

2.7.2 Causal organism

The causal agent is a blue fluorescent *Pseudomonas*, which is negative for cytochrome oxidase and arginine dihydrolase and is positive for the production of levan from sucrose. The host range of *P. syringae* may include onion, leeks, chives, and garlic (Samson *et al.* 1998).

2.7.3 Disease Cycle

In some cases the infection remains fairly limited, if optimum conditions are present the disease spreads throughout the whole plant, causing extensive blights and necrosis. Disease development is optimized by prolonged periods of rain, high relative humidity, dew, warm temperatures and damaged tissue. Wounds or abrasions provide access sites for the bacteria. Overhead sprinkler irrigation promotes bacterial dissemination. It appears that contaminated seed has introduced the disease into some areas.

2.7.4 Management

Testing of seed lots for *P. syringae* and the use of seed treatments to reduce or eradicate seed contamination are recommended practices for the management of this disease.

2.8 Bacterial Leaf Streak and Bulb Rot

Bacterial leaf streak and bulb rot is caused by *Pseudomonas viridiflava* (also known as *Pantoea ananatis* or *Erwinia ananas*). Bacterial Leaf Streak and Bulb Rot has been reported in Georgia (Gitaitis and Gay 1997) and in Colorado (Schwartz and Otto 2000b). This disease has caused significant losses in the field and in postharvest storage in the southern and western United States. Infected onions show foliar blight and bulb rots after bulb initiation.

2.8.1 Symptoms

P. ananatis infection results in a leaf blight that starts as whitish to tan lesions, which rapidly combine to cause a general wilt, discoloration, and death of affected foliage. Infected seed stalks become bleached and rotted while the bulbs develop a yellow -

cream to light orange discoloration in rotting neck tissue and between scales. The symptoms of *P. viridiflava* infection are water soaked lesions and streaks on leaves and a softening of basal areas of leaves above the neck of the bulb (Gitaitis *et al.* 1991, Gitaitis and Gay 1997). The streaks on the leaves become darker with age and are nearly black by the time the leaves dry up and collapse. Inner scales of infected bulbs are yellow at first and become reddish brown and decayed. The rot is restricted by the scales and develops in a ring. The rot is normally firmer than that observed with Sour Skin, Slippery Skin or Soft Rot caused by *Erwinia* spp.

2.8.2 Causal organism

P. ananatis (*ananas*) is Gram negative and rod shaped, and develops yellow colonies on nutrient agar. It can utilize glucose in an oxidative and fermentative manner. This bacterium does not produce phenylalanine deaminas, or nitrate reductase (Schwartz and Otto 2000b, Gitaitis and Gay 1997). *P. viridiflava* is a Gram negative aerobic, rod shaped fluorescent bacterium that forms white colonies that turn yellow with age. It is negative for oxidase and arginine dihydrolase, and slowly produces acid from sucrose after 7 days, as well as from rotted carrots and potatoes (Gitaitis *et al.* 1991). This bacterium is considered an opportunistic or weak pathogen.

2.8.3 Disease Cycle

The disease has been observed on onions post occurrence of bulb initiation and after heavy rain, storm damage and cool conditions (Gitaitis *et al.* 1998, Schwartz and Otto 2000b). In the field the onions are predisposed by environmental conditions, wounds, or other unknown factors that are favourable for the infection of this opportunistic pathogen. The lack of reports of disease incidence in other areas led to suspect a local source of inoculum. Gitaitis *et al.* (1991) did a survey to identify the source of inoculum of *P. ananatis* where the bacterium was detected on the foliage of weeds found in the Vidalia onion production area. However, it is not known if the pathogen survives in seed or soil associated with other hosts.

No evidence of surviving bacterium was found in soil, water or in association with soybeans, the rotation crop of onions in Georgia. This disease predisposes onion bulbs to other soft rot pathogens as well as secondary or opportunistic organisms associated with post-harvest decays.

2.8.4 Management

Crop rotation and the sanitation of onion crop debris, culls and volunteer plants may reduce sources of inoculum in the fields, however, the sources of inoculum are not known. Considering the association of *P. viridiflava* with weeds found in the onion production areas of Georgia, weed control may be beneficial for reduction of incidence or control of this disease (Gitaitis *et al.* 1991).

Many strains of *P. viridiflava* are copper tolerant; therefore the use of copper bactericides is not effective (Gitaitis *et al.* 1991).

2.9 *Xanthomonas* Leaf Blight

The disease is caused by the bacteria *Xanthomonas campestris*. *Xanthomonas* Leaf Blight has been reported in Texas (Isakeit *et al.* 2000), Colorado (Schwartz and Otto 2000c), California (Nunez *et al.* 2002), Hawaii (Alvarez *et al.* 1978), and South Africa (Serfontein 2001). It has now been reported in other tropical and subtropical areas, causing severe losses in yield due to bulb size reduction.

2.9.1 Symptoms

The first symptoms to appear on older leaves are numerous chlorotic lesions (white flecks) that normally degenerate into holes. The leaf blighting begins as linear tan to brown, water soaked lesions that rapidly elongate to form extended chlorotic areas, causing a general discolouration and tip dieback of affected foliage. Leaves often collapse at the point of infection.

2.9.2 Causal organism

X. campestris is a Gram negative, rod shaped, yellow bacteria with single polar flagella. This obligate aerobe uses glucose in an oxidative manner is catalase positive, oxidase negative, negative for tetrazolium salt tolerant test, and does not utilize asparagine as a sole source of carbon and nitrogen (Paulraj and O'Garro 1993, Serfontein 2001).

2.9.3 Disease Cycle

The ecological and epidemiology of the causal agent of Onion Leaf Blight is poorly understood. The disease generally occurs after periods of heavy rainfall or storms. High soil and air temperatures (25-30°C) and high relative humidity (85-95%) combined with overhead irrigation may increase risk of development and spread of the disease (O'Garro and Paulraj 1997, Serfontein 2001). Bulb size is severely reduced, with records of 10-15% loss of yield. Disease progress into the bulb has not been observed, but bulbs of infected plants never grow to full size (Nunez *et al.* 2002). The pathogen has been isolated from seed samples originating from a diseased seed production site, but not from healthy fields (Roumagnac *et al.* 2000).

2.9.4 Management

Seed sanitation is a major component of disease control management since the pathogen has been isolated from seed. Testing of seed lots for *X. campestris* and the use of seed treatments to reduce or eradicate seed contamination is suggested (Roumagnac *et al.* 2000). Efforts to control Leaf Blight with sprays of copper or zinc bactericides, including cuprasan, manzate, champion, and vandozeb was ineffective in Barbados (O'Garro and Paulraj 1997.) Failure of chemical controls has resulted in attention on alternative control measures such as resistant cultivars and crop rotation. O'Garro and Paulraj (1997) found that French bean, soybean, winged bean, field pea, moth bean, and

lima bean are hosts of *X. campestris* isolated from onions. Therefore, crop rotation with these crops should be avoided (O'Garro and Paulraj 1997). In order to reduce the losses by *Xanthomonas* Leaf Blight, resistant cultivars such as H-942 and H-508 should be used. Also follow the other recommended management practices.

3. Fungal Diseases

3.1 *Fusarium* Basal Plate Rot

The disease is also known as basal rot and caused by *Fusarium oxysporum* Schlechtend .: FR. f. sp. *cepae* (H.N. Hans) W.C. Snyder & H.N.Hans. Basal Rot occurs in most onion and garlic growing areas of the world and is most prevalent where these crops are grown under high-temperature conditions (Cramer 2000). The fungus attacks cloves and seedlings, causing pre- and post-emergence damping-off, root rot of older plants, and steam plate discoloration and basal rot of bulbs in the field and in storage (Abawi and Lorbeer 1972). Yield losses by Basal Rot can be more than 50% (Everts *et al.* 1985). *Fusarium* species are soil inhabitants, and infection is usually believed to follow injury, as such caused by maggots, cultivation, or pink root (Walker and Tims 1924). Most yield losses result from disease development in the field, but Basal Rot can also progress in storage. The pathogen attacks only members of the genus *Allium*. Onion, garlic, shallot, chives and leek are susceptible, but the disease is economically important only in onion and garlic (Abawi and Lorbeer 1972).

3.1.1 Symptoms

The first symptoms on onion in the field are tip dieback and yellowing of the leaves. The disease may progress until all foliage is yellow and withered, the aerial parts may die in 1 or 2 weeks or decay may extend over longer period of time (Everts *et al.,* 1985, Kehr *et al.,*. 1962). When the disease is evident above ground, decay has already started at the stem plate. Infected onions can be pulled easily out of the ground and often appear lopsided because only one side of the basal plate is infected (Walker and Tims 1924). Infected roots decay and a firm, pinkish-brown rot develops at the base of the bulb and later progresses upward, eventually the entire root system disappears. Under moist conditions, white mycelium develops on the rotted area. Under dry conditions, the tissue gradually dries and shrivels, and the dry outer scales crack open or fall off. In the case of early infection, decay may be almost completed by harvest time. Infections occurring about harvest time may continue in transit and storage (Walker and Tims 1924). Early signs of infection may be detected by cutting the bulb vertically and looking for the brown discoloration of the basal plate that begins at the outer layer and extends upward (Havey 1995).

3.1.2 Causal organism

F. oxysporum produces chlamydospores, microconidia and macroconidia, both on the host and in culture. Isolations from onion bulbs and soil indicate that the pathogen

exists in soil primarily as a sporodochial type. The many *formae speciales* of *F. oxysporum* are differentiated by the ability of the specific pathogen to infect a host (Abawi and Lorbeer 1971, 1972).

3.1.3 Disease Cycle

The fungus persists in soil in the form of chlamydospores that can be spread in water, soil, air, or carried on insects and equipment (Everts *et al.* 1985). It can penetrate roots directly or infect roots and bulbs previously injured by other onion diseases or by onion maggots. It may also invade the stem plate through natural wounds or infected roots. Disease development occurs between 15 and 30 °C with an optimum temperature of 29 °C (Walker and Tims 1924). Soil moisture levels that will support onion growth are adequate for infection and disease development. Onion plates can be infected any time during the growing season. High relative humidity in the field or storage promotes disease development. Chlamydospores form in and on infected roots; a few form in the stem plate or the leaf base of the bulb. The pathogen survives between onion crops as dormant hyphae or chlamydospores in decaying plant debris (Latham and Watson 1966).

Field symptoms usually do not appear until the soil becomes warm. At first there is a progressive yellowing and dying-back from the tips of the leaves; the aerial parts may die in 1 or 2 weeks, or decay may extend over a much longer period. Plants that are infected when young may continue to grow until harvest time. In storage or transit, the rot develops rapidly between 20 and 30°C. At temperatures below 15°C, the rate of decay is very slow but premature sprouting is still likely to occur. High relative humidity in storage promotes rotting. Spread of the disease from bulb to bulb during storage is not significant.

3.1.4 Management

The most important management practice for *Fusarium* Basal Rot on onions is the use of resistant cultivars. Many commercial onion cultivars have partial resistance to *Fusarium* Basal Rot, and this information is available from the seed companies (Abawi and Lorbeer 1971, Cramer 2000). Since the incidence and severity of *Fusarium* Basal Rot can increase in fields cropped frequently with onions (Abawi and Lorbeer 1972) a three or four year rotation with non-susceptible crops is recommended. As always, use disease free planting material. It is best to grow onions and garlic in well-drained soils (El Shabrawy *et al.* 1987) and follow recommended production practices to reduce plant stress. Reducing mechanical bulb damage during the growing season, avoiding overwatering and controlling nitrogen levels can also reduce disease incidence (El Shabrawy *et al.* 1987).

Soil solarization has proven to be a satisfactory control method, bringing soil populations of the pathogen to negligible levels in areas with appropriate weather conditions (Thornton *et al.* 1994). The use of *P. cepacia* as a seed treatment has protected seedlings against damping off (Kawamoto and Lorbeer 1976). Dipping onion seedlings in fungicide before they are transplanted has been very effective to reduce

the incidence of the disease. This approach should also reduce the spread of *Fusarium* on garlic cloves. Tebuconazole and benomyl have been reported to be effective for the control of Fusarium Basal Rot (Soares and Kurozawa 1998).Bulbs should be cured properly before harvest and damaged or diseased bulbs should be culled before storage. Maintain storage temperature at 0°C and relative humidity at 65 to 70% (Cramer 2000).

3.2 *Fusarium* Basal Rot of Garlic

The disease is caused by *Fusarium culmorum* (Wm.G.Sm) Sacc. and *F. roseum* Lk.emend. Snyd.& Hans. var. *culmorum* (Schawbe) [sic] Synd. & Hans.). The disease is caused by a specialized strain of *F. culmorum* that infects garlic and cereal crops. Fusarium basal rot of garlic that was first identified in 1976 and has since been found in the western United States and Canada. *Fusarium roseum* produces stout, thick walled, curved macroconidia with a distinctive basal cell that varies from foot-shaped to slightly notched. There are no microconidia. Chlamydospores occur singly or in chains and clumps. The fungus grows rapidly on potato dextrose agar, producing dense white to tan mycelium on the surface and a carmine red colour underneath. Orange to red-brown sporodochia form as the culture ages (Crowe 1995).

3.2.1 Symptoms

Garlic can be infected at any stage of growth but symptoms are not always diagnostic. Cloves may decay before emergence. Seedling infection of the basal plate causes chlorosis and necrosis of the leaves. The bulbs of actively growing plants may develop a firm dry rot characterized by a reddish fringe on the lesions. In other cases there may be no symptoms of infection, and discoloration on the bulb is not necessarily diagnostic (Crowe 1995).

3.2.2 Disease cycle

There are many unknowns in the epidemiology and development of this disease. Seed with a high incidence of infection may produce a crop with no symptoms for one or more generations, ,then in another season disease will develop in the field. It is not known what environmental or other conditions result in disease expression.

The fungus does survive in the soil. Initial infection may occur at the stem plate from soil-borne inoculum (Crowe 1995), and remain localized for varying periods so time, which may explain the lack of symptoms.

3.2.3 Management

Fungicide treatment of seed garlic cloves is the most effective method of disease management. Benomyl) applied in hot as water , as recommended for the control of *Penicillium* clove rot, is a very effective seed treatment and controls both seed borne and soil borne disease. (Crowe 1995). Benomyl is no longer available, but other benzimida-

zole fungicides or fungicides with systemic activity similar to that of benomyl may also effectively control this disease. Hot water treatment of garlic seed, with or without formaldehyde, provides some control, but not at levels that are acceptable for commercial production. Hot water treatment of garlic cloves is not recommended where fusarium basal rot is suspected, since this treatment may increase the amount of disease that develops, in some cases.

Crop rotations with cereal crops should be avoided, since garlic strains of *F. culmorum* will infect cereals, and this could increase the inoculum. . However, *F. culmorum* isolated from cereals not grown in rotation with garlic will not cause disease on garlic. Avoid spreading the disease in infested soil, plant debris or irrigation water runoff (Crowe 1995).

3.3 *Botrytis* Leaf Blight

The disease is caused by the fungus *Botrytis squamosa* J.C. Walker (teleomorph *Botryotinia squamosa* Vien.-Bourg). This is one of the major foliar diseases of onions in cool climate areas, and has been reported in many parts of the world, including North America, Britain, Europe, Japan and New Zealand. The severity of epidemics depends on local weather conditions. *Botrytis* Leaf Blight is an economically important disease only on common onion. Levels of disease affecting less than 11% of leaf area do not decrease yield, but when disease is severe and leaves die back, the bulbs may be small and fail to mature properly. Severely affected bulbs may not dry down enough for proper storage. They may also have fleshy leaf tissue at the neck rather than dry papery scales and are therefore more susceptible to storage rots. Rapid senescence of the leaves may also interfere with the application of sprout inhibitors, thus reducing the storage life of bulbs.

3.3.1 Symptoms

The first symptoms to develop are discrete, circular to elliptical lesions. These are grayish white, about 1 by 3 mm, and later become brownish-white and desiccated. A characteristic silvery-white "halo" with uneven margins usually develops around newly formed lesions as a result of enzyme activity, which separates the leaf epidermis from the underlying tissue. The gray mould fungus, *B. cinerea,* may also infect onion leaves, but the resulting lesions are smaller, do not penetrate to the inside of the leaf nor do they develop halos. *B. allii,*(or *B. aclada,* the causal agent of Neck Rot) can cause limited foliar spotting, but it usually remains in a latent state until the bulb is mature or the leaf has senesced.

Under favourable weather conditions, the number of lesions on a leaf increases, the lesions expand and merge and the leaves begin to die back. Dieback usually begins at the leaf tip and may extend down the entire leaf. The lower, older leaves are more susceptible to infection and are also the first to die. Sporulation occurs on necrotic leaf tips and occasionally on large lesions. Several species of *Botrytis* are associated with neck rot symptoms; the one caused by *B. squamosa* is known as Small Sclerotial Neck Rot.

3.3.2 Causal organism

The causal agent, *B. squamosa,* has characteristic conidiophores that shrink back into accordion-like folds after sporulation. The conidia are obovoid to globose and are larger (9 to 18 by 14 to 24 µm) than those of other *Botrytis. spp*. found on onion. *B. cinerea* has smaller conidia, 4-11 by6-18 µm, and has long conidiophores that are darker than those of *B. squamosa*. *B. allii* has the smallest conidia, 4-8 by 6-16 µm.

Sclerotia of *B. squamosa* form on the surface of onion bulbs and occasionally on dead leaves. They are flat, scale-like structures, 0.5 to 4 mm, which are white at first, but turn black with age. The sclerotia often produce tufts of conidiophores and, in certain locations, apothecia in the spring. The apothecia are stipitate, cupulate and 0.5 to 2.5 mm in diameter.

3.3.3 Disease Cycle

B. squamosa is a polycyclic pathogen with the potential to cause rapid disease development under favourable environmental conditions. Sclerotia overwinter in the soil, on onion debris, and on bulbs in cull piles. In the spring, the sclerotia produce conidia at temperatures between 3 and 27EC, with maximum production at 9EC. In some cases apothecia may form on the sclerotia. Conidia are the most important initial inoculum, but ascospores can also contribute to the epidemic.

Sclerotia may survive at least 21 months when buried in organic soil, but conidia usually survive for less than three months. The myceliummycelium of *B. squamosa* does not survive long in plant residues. Infection of seedlings from seedborne conidia is probably rare.

The conidia of *B. squamosa* are released during the daytime with peaks of spore release between 0900 and 1200 hours promoted by declining relative humidity. A smaller peak may occur in the evening and large releases of spores are associated with rain showers. The conidia are dry and are dispersed in turbulent air. Disease distribution in a field is usually general rather than focal. Germination of conidia and infection require liquid water and temperatures over 6EC. The optimum conditions for infection are 12 hours of leaf wetness at 15 to 18EC. Infection is reduced above 27EC (Sutton *et al.* 1986).

3.3.4 Management

The most successful management of botrytis leaf blight involves weather based disease forecasting to predict disease development and time the application of fungicides. In the absence of pest management programs or scouting, a regular spray program with protectant or systemic fungicides can also be effective, although there is a high probability that more sprays than needed will be applied. Good control has been achieved through tank mixes of a broad-spectrum fungicide and a single mode of action fungicide, especially if it has systemic activity.

Three different systems are used in North America to forecast *Botrytis* Leaf Blight and to time fungicide applications. These are the sporulation index incorporated

into the PREDICTOR (also called the PESTCASTER) developed by M.L. Lacy (1987); BOTCAST developed by J.C. Sutton *et al.* (1986); and BLIGHT-ALERT developed by P.C. Vincelli and J.W. Lorbeer (1989). Each of these programs predicts the need for fungicide applications by different means.

The PREDICTOR is based on vapour pressure deficit (VPD), which is determined using relative humidity and temperature over the previous 72 hours to arrive at an index that indicates the probability and intensity of spore release. This program utilizes an instrument that measures temperature and relative humidity and can also record leaf wetness, rainfall and soil temperature. It provides a read-out of the sporulation index for that day. A grower or scout examines the sporulation index each day and determines whether the risk is sufficient to warrant chemical control. Fungicides need not be applied more than once every five to seven days. This system is used in Michigan and Quebec. It can be grower-operated and does not depend on field scouting. In Quebec, good control of *Botrytis* Leaf Blight has been achieved by spraying when the sporulation index is over 50.

The other two programs are based on starting the fungicide spray program when a critical disease level of one lesion per leaf is reached. BOTCAST (Sutton *et al.* 1986) is designed to use accumulated microclimatic data to predict when this critical level has been reached in the field. A data logger or weather station is used to record the duration of leaf wetness, temperature, relative humidity and rainfall. Temperature and leaf wetness data are combined to provide an infection value of 0, 1 or 2. Leaf wetness duration of less than 6 hours will result in an infection value of 0. These values are multiplied to give a disease severity index for each day. Cumulative disease severity indices (CDSI) indicate two thresholds. Threshold 1 (CDSI=20-30) triggers the recommendation to spray before the next rainfall. Threshold 2 (CDSI=30-40) indicates that a fungicide should be applied as soon as possible. Once the control program has started, fungicides should be applied every 7 to 10 days. Research in Ontario has shown that using microclimatic data to lengthen the spray interval when weather is not favourable for *Botrytis* Leaf Blight only saves an average of one spray.

The BLIGHT-ALERT program (Vincelli and Lorbeer 1989) is designed specifically to time fungicide sprays following the first application. Under this system, pest management scouts walk the fields and count lesions on leaves. The threshold is one lesion per leaf. A sequential sampling method is used and scouts count lesions on the three oldest leaves (80% green) leaves of 16 to 50 plants per field. Once the one lesion/ plant threshold is reached and growers begin spraying, subsequent sprays are recommended on the eighth day after spraying if the probability of rain is greater than 30% or if the AInoculum Production Index@ is greater than 7. An index of less than 7 indicates that spore release is unlikely. Fungicide protection is considered to last for seven days. If no fungicide application is recommended for eight days, the index and probability of precipitation are checked for day nine and each day thereafter until the next spray is needed.

The Inoculum Production Index, which is based on weather conditions conducive for spore production, is calculated using average temperature, hours of relative humidity above 90% and days after planting. The index ranges from 0 to 25. Significant spore releases usually occur on days where the index is greater than 7. A probability of

precipitation greater than 30% indicates that the conditions will be favourable for infection if spores are released. In New York State, growers using the spray threshold and BLIGHT-ALERT program have saved two to three sprays per season.

Cultural practices can also be an important component of an overall disease management program, two to three year crop rotations with crops unrelated to onion can reduce the number of sclerotia that survive in the soil and thus reduce the initial inoculum. The removal of onion cull piles and the reduction of overwintering cull onions in the field also reduce inoculum levels.

3.4 Downy Mildew

The disease is caused by the Oomycete, *Peronospora destructor* (Berk.) Casp. in Berk. and disease occurs sporadically in many temperate onion-producing areas, and can also be found in the tropics where onions are grown during the cool season in subtropical countries (Snowdon 1992). The disease is rare where weather conditions are hot and dry but is a constant threat where onions are grown under cool, humid conditions. Downy mildew has the potential to destroy all of the foliage in three to four disease cycles within just four or five weeks. Severe downy mildew reduces bulb size and can reduce the storability of the crop because the bulbs will not cure properly, and the necks of diseased onions will remain succulent. Downy mildew can affect onions, garlic and several other *Allium* species.

3.4.1 Symptoms

The first symptom of downy mildew is the velvet-like growth of the pathogen on otherwise green leaves. The sporulation can occur in round to elongated lesions, or over much of the leaf. When seen early in the morning, the fungal growth appears purplish due to the colour of the sporangia , which form overnight just above the leaf surface. Later in the day the sporangia disperse in the air but the whitish sporangiophores remain on the leaf. Following sporulation, the diseased area gradually turns pale green, then yellow, and finally collapses and dies. Other fungi, especially *Stemphylium botryosum* Wallr. and *Alternaria porri* (Ellis) Cif., may colonize the infected tissue, giving the diseased area a dark appearance . Necks of affected plants remain succulent. Sporulation also occurs on seed stalks, where the affected areas tend to be circular to oval and to remain yellow. Infected stalks bend over and break when the seed head enlarges. The pathogen may also invade flowers and seeds.

3.4.2 Causal organism

Peronospora destructor is an obligate parasite. It produces aseptate mycelium, asexual sporangia borne on sporangiophores, and sexual oospores. Sporangia form on sporangiophores that emerge through the stomata of green host leaves. The sporangiophores and sporangia are characteristic and are important in identification of the pathogen. The sporangiophores have dichotomous primary and secondary branches and curved sterigmata. Mature sporangia are lemon or teardrop shaped and are thin-

walled, sub-hyaline, pyriform, papillate at the distal end and measure 18-29 by 40-72 μm in diameter. Oospores form in onion leaf tissue. They are thick-walled, spherical and 40 to 44 μm in diameter (Mukerji 1975). Oospores survive in the soil for four or five years and germinate by a germ tube (Dixon 1992) .

3.4.3 Disease Cycle

The downy mildew pathogen survives in living plants, or plant parts, including onion bulbs in the field or cull piles and in perennial onions. The pathogen can also persist as oospores in onion tissue or the soil, but the importance of oospores in initiating downy mildew epidemics is unknown. Studies indicate that mycelium surviving in overwintering onions is the most important source of inoculum (Dixon 1981). Leaves that sprout from these bulbs will be become systemically infected by the pathogen and will produce sporangia. Onion leaves can be infected at any stage.

Sporangia form on leaves or seed stalks of infected plants and are dispersed in air currents. Downy mildew develops rapidly under cool, humid conditions. Sporulation takes place at night, between midnight and sunrise, when there is high humidity (95%) and temperatures are in the range of $4 – 24$ °C, with an optimum of 12- 13 °C. Free water, whether dew or rain, will inhibit sporulation. Daytime temperatures over 24 °C inhibit sporulation (Hildebrand and Sutton 1984). Sporangia are released as the relative humidity in the air drops during the morning. Sporangia are short-lived, but can survive for 2 to 3 days in the field, especially under low light conditions.

Infection of a susceptible leaf requires a minimum of 3 to 6 hours of leaf wetness at temperatures between 6 and 27 °C, with an optimum of 10-12 °C (Hidlbrand and Sutton 1984). Once infection takes place, the pathogen colonizes the leaf for a period of 9 to 16 days before sporulations occurs. There are no visible symptoms during this stage of disease development. Following this latent period, sporulation occurs when weather conditions are conducive. Once the sporangia are released, the underlying tissue turns pale green, then chlorotic and necrotic. The pathogen can continue to colonize the leaf and produce concentric flushes of sporangia on green tissue surrounding the original lesion, of the entire leaf may die within a few days. When weather conditions are predominantly cool and humid, disease will develop rapidly and can result in the death of all the onion leaves within 3 or 4 cycles over 34 to 45 days (Hildebrand and Sutton 1984).

A downy mildew epidemic usually begins with one or a few small foci in a field. These may be difficult to detect, even with field scouting, because the first lesions can be small, localized and the tissue remains green. If the epidemic advances unchecked, the bulbs of infected plants will fail to enlarge, and the necks will remain succulent. In storage, these bulbs may become soft and shrivelled with an outer scale that will be watery, wrinkled and amber in colour. Other infected bulbs may remain firm but sprout prematurely (Snowdon 1992).

3.4.4 Management

Cultural controls including sanitation and crop rotation are very important in prevent-

ing epidemics of Downy Mildew. Cull onions should be destroyed and not left in piles near onion fields. It is very important not to grow winter onion and other host crops in fields and home gardens near summer production fields. Seed onion should be grown far away from any bulb crops.

Foliar fungicides are essential for the control of this disease. Fungicides should be used when mildew is present in the area and when weather is conducive to an increase in disease. Protectant fungicides can be used effectively is they are applied before infection takes place and on a regular schedule to ensure that the leaves are adequately covered at all times.

The Downy Mildew forecaster DOWNCAST developed by Hildebrand and Sutton (1982) may be used to identify periods when weather favours sporulation and infection by the mildew fungus. DOWNCAST requires weather monitoring in the field and indicates optimal times to look for early signs of mildew and to apply fungicide sprays. The weather data needed for the system are hourly air temperatures for the preceding day, air temperature, relative humidity and leaf wetness duration. In summary, if air temperatures were above 24 °C on one day, no sporulation will occur the following night. For sporulation to occur, there must be six hours between midnight and dawn where the relative humidity was 95% or over, temperatures between 4 and 24 °C and no leaf wetness. If environmental conditions are favourable for sporulation, and conditions for infection occur in the next 24 to 48 hours (6 hours leaf wetness at temperatures between 6 and 25 °C, then there is a high probability that infection will take place. (Jesperson and Sutton 1987). If the leaves are not protected by fungicide when these conditions occur, a systemic fungicide with activity against the Oomycetes should be applied as soon as possible. To avoid the development of fungicide resistance in *P. destructor*, systemic fungicides should be used on a preventative, rather than a curative basis, whenever possible, used in combination with a protectant fungicide, and alternated with fungicides with different modes of action.

DOWNCAST has been implemented in many regions of the world to manage downy mildew on onions. In Australia, DOWNCAST was implemented in 1998 and reduced the number of sprays applied during onion production by 4 or 5. Fungicides application was avoided early in the season when temperatures were too high for downy mildew development. During the growing season, there were several times when sporulation occurred but the leaf wetness required for infection was not present (Jackson *et al.* 1999).

3.5 Neck Rot (Gray Mould Neck Rot)

The disease is caused by *Botrytis allii* Munn (syn *Botrytis aclada* Fresen). Similar, related diseases may also be present: mycelial neck rot caused by *Botrytis byssoidea* Walker (teleomorph *Botryotinia allii* (Sawada) Y. Yamamoto) and small sclerotial neck rot *Botrytis squamosa* Walker. Neck rot is one of the most common storage diseases of onion, and can also occur on shallot, leek, garlic and chives. Infection levels exceeding 50% of the bulbs in storage have been reported. Additional losses can result from secondary infections of bacterial soft rot. Neck Rot is most commonly found on bulbs after harvest, although in wet seasons it may appear before harvest.

3.5.1 Symptoms

The first symptom is the softening of the affected neck scale tissue, which takes on a sunken, cooked appearance. There is a definite margin between the diseased and healthy tissue, which remains distinct even when the rot is advancing toward the base of the bulb. As the lesions age, the tissue becomes grayish and later a dense, grayish, cottony growth of mycelium appears on the surface of the scales. This growth gives rise to a gray-brown powdery mass of conidia that are released into the air. As the disease develops further, small, whitish kernel-like sclerotia, less than 3 mm in length, appear in the mycelium and soon turn black and hard. Symptoms usually appear first in the neck and spread downward to the base of the bulb, but symptoms may develop first around the basal plate and progress toward the neck. Infected bulbs often have secondary bacterial infections, especially soft rot. When this happens, the moist bacterial exudates may stain surrounding bulbs, increasing the losses in storage. In other instances, the infected bulb may become dry and mummified.

Mycelial Neck Rot can be distinguished from Gray Mould Neck Rot by the presence of more surface mycelium and sparse sporulation. Small Sclerotial Neck Rot is more prevalent on white onions, and as the name implies, is characterized by small sclerotia that form around the neck.

3.5.2 Causal organism

B. allii, the most common species, may produce a dense mat of conidiophores near the sclerotia on infected bulbs. *Botrytis* conidiophores are straight, alternately branched and may proliferate. The conidia are usually one-celled, gray to brown, and globose to ovoid (teardrop) in shape. In culture, *B. byssoidea* produces few conidia and they are larger, 5 to 11 by 8 to 20 μm, than those produced by *B. allii*, which measure 4 to 8 by 6 to 16 μm. *B. allii* conidia sometimes overlap the size range of *B. cinerea* conidia (10-13 by 6-10 μm) (Ellis and Waller 1974, Kritzman and Netzer 1978).

3.5.3 Disease cycle

The sclerotia that form on infected bulbs serves as the primary overwintering structures. These may survive for several years in cull piles or in some soils. The sclerotia germinate under suitably moist conditions to produce successive crops of conidia that spread on air currents.. In Britain, *B allii* was shown to survive on debris in a sandy loam soil for two years, but sclerotia failed to germinate and produce conidia after only six months in soil (Bottcher 1987). In Canada and the northeastern United States, sclerotia in cull piles and on onion debris and unharvested bulbs in the field are an important source of initial inoculum.

B. allii is also seed-borne and can survive over 3 years in seed lots (Maude and Presely 1977). Direct seedling infection has been observed with mycelium from the seed coat penetrating the tip of the cotyledon leaf. No symptoms can been seen and conidia do not form until the tissue becomes necrotic. Successive leaves become invaded, as the fungus grows systemically within the plant, with still no visible symptoms

of infection. By the time the onion is mature, the pathogen has colonized the neck tissue of the bulb(Dixon 1981).

The air-borne spores of *B.allii* can infect onion leaves during periods of cool (15 to 20EC), wet weather, but symptoms may not be visible. Either the fungus can grow within the leaf without causing symptoms or it remains quiescent in the epidermis and grows only when the leaf starts to senesce. Conidiophores are produced only on senescent or necrotic leaf tissue. The pathogen can spread rapidly during wet growing seasons and can spread downwind from a diseased to a healthy crop. Cool, wet weather during the curing and harvest periods can increase the incidence of Neck Rot. Infection can take place during harvest if the bulbs are bruised or damaged. Onions with thick necks or broken outer scales may be infected through these sites. Neck Rot rarely spreads from bulb to bulb in storage, although it may become more severe the longer onions are stored because bulbs that were infected, but symptomless when harvested, eventually develop symptoms.

3.5.4 Management

The elimination of onion cull piles and overwintering onions in the field will help to reduce the overwintering inoculum. Crop rotations of at least two years, with crops unrelated to onion, are also recommended, but because the conidia can travel considerable distances in the air, crop rotation is most effective where onion fields are widely separated from each other.

Proper management of the onion crop can reduce the opportunity for infection to take place. High levels of nitrogen fertilizer can promote vegetative growth that will delay maturity. Irrigation too close to harvest and leaf injury from herbicides, insects or other causes can provide infection sites for the pathogen. Increased spacing between plants, which can result from stand loss due to onion maggot, onion smut, or other causes, can result in thick necks in the remaining onions.

Proper curing and storage of mature bulbs is the most important cultural control for reducing Neck Rot. Onions with thick necks, nicks or bruises are susceptible to infection. Onions should have two or three layers of dry outer scales and the neck should be narrow and composed of papery-dry scales at harvest to reduce the opportunity for infection to occur. Onion bulbs should be mature at harvest. Further curing is often required for successful long-term storage. Undercutting or lifting the onions prior to harvest can aid the drying process. Leaving the lifted bulbs in windrows until the necks are thoroughly dry can be sufficient in dry climates. Artificial curing, the passing of warm, dry air through piles of onions in storage is used to cure onions that are produced in humid areas (Harrow and Harris 1969). The air should be heated sufficiently to reduce the relative humidity to 60% for 14 days or until the onion skins turn a rich golden brown. Once onions are cured thoroughly, they should be cooled slowly and stored at temperatures just above 0EC at a relative humidity of 60 to 70%.

Most commercial onion cultivars are susceptible to Neck Rot, especially white-skinned and Spanish onions. Foliar applications of systemic fungicides have been used to reduce the incidence of Neck Rot. Usually several sprays throughout the season are needed, beginning at the time that the cotyledon is senescing.

3.6 Pink Root

The disease is caused by *Phoma terrestris* E. M. Hans. (syn. *Pyrenochaeta terrestris* (E.M. Hans.) Gorenz, J.C. Walker & R.H. Larson). This is a common disease that occurs on *Allium* crops in North America, Australia, Europe, and South Africa. Yield losses as a result of Pink Root are minimal under cool growing conditions, but the disease can be very damaging in warmer regions. Losses can be difficult to document because this disease only reduces bulb size. The pathogen is a common soil inhabitant and can infect the roots of onion, garlic, other *Allium* spp. and many other crops.

The pathogen is widely distributed within fields but diseased plants often appear in patches, either near the headlands, where the soil is shallow, on knolls or over drainage tiles where the soil is poorer or drier. In some regions, the disease appears so late in the season that it has the beneficial effect of reducing moisture uptake by the plant, thereby allowing the bulbs to dry and mature in preparation for harvest.

3.6.1 Symptoms

Pink Root symptoms are frequently seen on onion roots. The distinctive, dark pink to maroon colour of infected roots identifies the disease most easily. As the disease progresses infected roots partially to totally collapse and later turn reddish brown as the root dies. In contrast, healthy roots are cylindrical and white. Diseased roots break off easily when the bulb is pulled from the soil. Under low disease pressure, onion bulbs may have only two or three pink roots and no above ground symptoms. Plants with severe Pink Root are stunted, have a high proportion of infected roots, are easily pulled from the soil, and fail to produce marketable bulbs. When disease is moderate, the leaves die back from the tip, turning yellow-white or yellow-red. The colour is more reddish than when the leaves die from environmental stress. Infected plants rarely die back completely; they usually remain stunted with only a small area of green leaf tissue remaining near the neck of the bulb.

3.6.2 Disease cycle

P. terrestris is a soil inhabitant that can survive and multiply indefinitely in soil. It is weakly pathogenic and attacks the roots of plants that are under stress or that have previously been injured. On onion roots inoculated with conidia, symptoms can be seen within 7 to 21 days. The hyphae penetrate the roots and grow through the cortical tissue; pycnidia form in both the epidermal and cortical cells. The pycnidia erupt through the epidermal tissue and release the conidia into the soil.

The fungus can develop at all soil moisture levels that will support the growth of onion. The optimum temperature for infection is 26EC. In many temperate regions these soil temperatures are associated with drought conditions, which cause further stress to the plant. Conversely, root damage by *P. terrestris* results in greater drought stress.

The fungus is spread in infested onion sets and soil. Farm machinery, dust storms and surface run-off can move soil from one area to another and spread the pathogen. However, in many regions the fungus is so widespread that is futile to take

precautionary measures to prevent its spread from one field to another.

3.6.3 Management

Many commercial onion cultivars have tolerance or partial resistance to Pink Root. The use of these cultivars is the best management method, where available. Leek and chives are resistant to Pink Root.

Long crop rotations with unrelated crops may help to reduce the soil borne inoculum, however, cereals, grasses, parsnip, radish and spinach are all potential hosts of *P. terrestris* and should not be used in rotation. One of the best cultural controls is to use crop production recommendations to supply adequate moisture, fertilizer and crop protection to encourage vigorous growth. Healthy, actively growing plants are better able to tolerate the disease by producing abundant roots.

Soil fumigation is generally considered not economical, though it will reduce the population of *P. terrestris* and may result in increased onion yields. Fumigation is more effective on sandy soils.

3.7 Purple Blotch

The disease is caused by *Alternaria porri* (Ellis) Cif., is a common disease of onion and garlic. It can be destructive alone or as a secondary pathogen, affecting leaves that already have been attacked by other pathogens or damaged by herbicides, hail or ozone. *Alternaria porri* can also infect several other *Allium* species

3.7.1 Symptoms

The initial symptoms are small, whitish, sunken and elongate lesions, which may have purplish centres. As the disease progresses, the lesions expand to become large, oval, purple blotches with concentric rings, characteristic of *Alternaria* infections. Sporulation occurs on the lesions and diseased leaves eventually die. Older leaves are more susceptible to Purple Blotch than younger leaves. Purple Blotch can cause heavy losses in onion seed crops by infecting and destroying the flower stem. The fungus also may infect the onion bulb at harvest through wounds or fleshy neck tissue. In storage, *A. porri* causes a dark yellow or deep-red spongy bulb rot.

3.7.2 Causal organism

Conidiophores of *A. porri* form singly or in clusters on diseased leaves. They are pale to medium brown, 10 to 15 µm thick, up 10 120 µm long, and may have from one to several well-defined conidial scars. Conidia are smooth or minutely verrucose, pale to golden brown, and are borne singly. When they detach, they leave a distinct scar. The conidia are straight or slightly curved, 100-300 µm long, 15-20 µm thick, and club shaped with a long beak. The beak is often as long as the main body of the conidium, and is flexible and tapered. The conidia have 8 to 12 transverse septa and none to several longitudinal or oblique septa. Each cell is capable of germination. A teleomorph is not

known (Ellis and Holliday 1970).

3.7.3 Disease cycle

The mycelium and conidia of *A. porri* overwinter on infested onion residues or in cull piles. Conidia are produced in the spring and disperse by wind or splashing water to onion leaves. Liquid water is necessary for the conidia to germinate. Germination can occur in 45 to 60 minutes at 28-36EC. Penetration can take place directly through uninjured epidermis, stomata or through wounds. Lesions may appear one to four days after penetration and conidia are produces shortly thereafter. Long periods of leaf wetness increase the likelihood of infection. Almost no infection occurs at or below 13EC. In culture, the fungus grows between 6 and 34EC. The optimum for sporulation is 25EC at 90% relative humidity. On calm days, the maximum number of conidia is trapped between 0800 and 1400 hours. Wind, rain, irrigation and spraying may promote spore release.

3.7.4 Management

Crop rotation and sanitation to remove overwintering sites of the fungus will reduce the initial inoculum. To prevent disease development in storage, onions should be harvested during dry weather when the bulbs are fully mature and the tops are dry. Bulbs should be stored at 0EC and 65 to 75% relative humidity. Broad-spectrum protective fungicides applied to onion foliage prior to spore deposition provide effective control of Purple Blotch. Fungicide seed treatments were not effective in reducing inoculum on the seed. Hot water seed treatments did reduce inoculum but also reduced seed germination and vigour (Aveling *et al.* 1993).

3.8 Blue Mold of garlic

The disease is caused by various *Penicillium* spp. (Sumner 1995, Maude 1990). The later stage of the disease at harvest and during storage is called Blue Mold and the earlier seedling stage is commonly called *Penicillium* Decay (Sumner 1995). Therefore *Penicillium* causes both field and storage disease problems. Primary decomposition of garlic bulbs in storage is usually due to infection by *Penicillium* rather than other pathogenic organisms (Bodner *et al.,* 1998, Bottcher and Gunther 1994)

Cloves with signs of disease are usually discarded before planting, so the seedling stage of the disease is usually of minor importance (Sumner 1995). If infected cloves are planted, the subsequent seedlings may appear wilted, chlorotic and/or stunted in growth (Sumner 1995). The development of new roots is usually reduced in number and the roots themselves are often stunted in growth. If the plants continue to survive they remain weak in growth and new bulb production is poor (Sumner 1995).

3.8.1 Symptoms

The harvest and storage stage of the disease is called Blue Mold (Sumner 1995). Early

symptoms are visible as pale yellowish blemishes, watery soft spots or purplish red stains on the out side of the bulb or on single cloves (Sumner 1995). Initial infections are visible at the base of the bulbs as masses of blue-green spores (Bodner *et al.,* 1998, Sumner 1995). Later infection may be hidden within the tissue and therefore are not noticeable until the dry scale around the infected clove is removed (Sumner 1995). Cloves that are infected with *Penicillium* are usually light in weight (Smalley 1954). If infections by secondary pathogens, especially bacteria, do not occur, then *Penicillium* infected tissue dries and toughens up or becomes punky (Sumner 1995). Tissue decaying as a result of infection by *Penicillium* usually has a musty odour (Sumner 1995). Affected garlic bulbs may show little external evidence of disease until decay is advanced (Sumner 1995).

3.8.2 Causal organism

Penicillium aurantiogriseaum Dierckx, *P. expansum* Link, *P. citrinum* Thom, *P. digitatum* (Pers.:Fr.) Sacc., *P. hirsutum* Dierckx, *P. funiculosum* Thom and *P. oxalicum* Currie and Thom are all species of *Penicillium* that can cause Penicillium Decay and Blue Mold of garlic (Sumner 1995). Long conidiophores arise singly from branched septate, hyaline mycelium (Sumner 1995, Raper and Fennell 1965). Conidiophores are branched near the apex, pencillate and end in a group of conidia (Sumner 1995, Raper and Fennell 1965). The conidia are globose to ovoid, in long chains and green or blue in colour (Sumner 1995, Raper and Fennell 1965).

3.8.3 Disease cycle

Penicillium spp. survive and grow on growing plants and senescing plant and animal debris on or within soil (Sumner 1995). *Penicillium* is encountered almost everywhere and is usually the most abundant genus of fungi found within soils (Jay 2000, Bodner *et al.,* 1998). The fungus does not survive long in the soil (Smalley and Hansen 1962). Therefore the largest source of *Penicillium* inoculum in garlic production is most likely infected cloves (Bodner *et al.,* 1998, Sumner 1995, Smalley and Hansen 1962). In the absence of moisture, the fungus cannot enter healthy tissue (Dillon Weston and Taylor 1943). *Penicillium* infects tissue through wounds but wounds that have healed over with callus tissue are not penetrable by *Penicillium* (Sumner 1995, Dillon Weston and Taylor 1943). When infected bulbs are cracked to separate the cloves for planting, the spores become air-borne increasing the risk for non-infected wounded cloves to become diseased (Bodner *et al.,* 1998).

After the initial infection occurs, the mycelium grows throughout the fleshy tissue (Sumner 1995). *Penicillium* sporulates on lesion surfaces and in wounds and entire garlic cloves may disintegrate and are replaced by masses of spores (Sumner 1995). The optimum temperatures for conidia germination and growth are between 21°C to 25°C, though germination and growth will occur at temperatures from 15°C to 32°C (Bertolini and Tian 1996, Sumner 1995, Smalley and Hansen 1962). Moisture is necessary for optimum growth of the pathogen (Sumner 1995).

3.8.4 Management

Cloves need to be protected from physical damage in the field, during harvest and in storage regardless of what causes the physical damage (Sumner 1995). Reducing physical damage to cloves will reduce wounding and therefore should reduce the potential for infection by *Penicillium*. Harvesting bulbs should be done carefully to minimize bruising and wounding (Sumner 1995). Free moisture on bulbs at harvest and during storage should be removed through prompt drying (Sumner 1995). Garlic bulbs should be stored at temperatures below 5°C (Smalley and Hansen 1962, Bertolini and Tian 1996) and low relative humidity (Sumner 1995, Bottcher and Gunther 1994). Avoid planting infected cloves. Plant garlic cloves as late as possible in the summer when soil temperatures are no longer in the optimal range for *Penicillium* conidia germination and growth of the mycelium (Bodner *et al.,* 1998). Treating cloves with fungicides and disinfecting solutions such as sodium hypochlorite may be recommended in some countries (Sumner 1995). Iprodione, a dicarboximide fungicide, is the most common product registered for treatment of cloves. The recommended rate is dipping cloves for 30 minutes in a solution of 2 g of iprodione per litre of water. No fungicide treatments are recommended if the cloves are going to be consumed directly, although consumption of infected cloves is not recommended since some *Penicillium* spp. produce substances toxic to humans (Jay 2000).

3.9 Garlic Rust

The garlic rust is caused by various *Puccinia* spp. (Jennings *et al.,* 1990). *Puccinia* has been considered an important pest of onion in many areas of the world but not garlic (Hill 1995). Infections on onion and garlic have been observed in most of the temperate regions in the northern hemisphere, in a few areas in South America, in the Middle East, Australasia and in China (Koike *et al.,* 2001, Hill 1995). Recently *P. porri* became a more important pest of garlic (Koike *et al.,* 2001). Serious out breaks of garlic rust occurred in the United States in California and Arizona from 1998 to present (Koike *et al.,* 2001) and 2001 in Bloemfontein, South Africa (Pretorius and Dunhin 2001). First reports of rust on garlic have occurred recently in Oregon, United States (Griesbach *et al.,* 2001) and in the late 1980's in the Punjab State, India (Singh and Basandrai 1988). The first ever report of rust was on onions in England in 1809 (Hill 1995).

3.9.1 Symptoms

Regardless of the *Allium* spp. being infected, the initial symptoms appear as small (<2 mm), white flecks on both sides of leaves (Koike *et al.,* 2001, Hill 1995, Gardner 1940). The small white spots expand somewhat over time (1 to 3 mm) into oblong lesions that rupture (Koike *et al.,* 2001, Hill 1995). The ruptured lesions expose masses of orange urediospores that are visible as pustules (Koike *et al.,* 2001, Hill 1995, Gardner 1940). The pustules usually occur between leaf veins (Hill 1995). Later in the season, teliospores develop on the leaves that urediospore pustules are visible, resulting in black pustules that remain covered by leaf epidermis until maturity (Koike *et al.,* 2001, Hill 1995, Gardner

1940). Severely infected leaves and fields will turn prematurely yellow and then dry up to a brown colour (Koike *et al.,* 2001, Hill 1995, Gardner 1940).

3.9.2 Causal organism

Puccinia porri G. Wint. and *P. allii* (F. Rudolphi) are considered the fungal pathogens that cause garlic rust (Koike *et al.,* 2001). *P. allii* is characterized by two-celled teliospores and telia containing groups of fused paraphyses (Koike *et al.,* 2001. Teliospores from *P. porri* are both one- and two-celled and are without paraphyses (Koike *et al.,* 2001). Infections by *P. asparagi* D.C. have been found on onions near infected asparagus resulting in symptoms on the plants similar to those caused by *P. porri* and *P. allii* (Walker 1921a). In all species, the yellowish orange urediospore (20 to 24 X 23 to 29 µm) is globoid to ellipsoid and has a thick (1 to 2 µm) echinulate wall with five to 10 pores. The teliospores were described above. The pycnial and aecial stages of the fungus have been found only rarely in Europe, Japan and China and no reports have been found in the Western Hemisphere (Chupp and Sherf 1960). The pycnial and aecial stages are also rare in nature (Hill 1995).

3.9.3 Disease cycle

The urediospores and teliospores both overwinter and therefore, both serve as primary inoculum in the spring on the emerging garlic crop (Hill 1995). Urediospores are more important in rust survival (Hill 1995). Urediospores and teliospores may travel by the wind over long distances (Hill 1995, Chupp and Sherf 1960). Urediospores need at least four hours of 97% relative humidity to germinate and infect but do not survive when immersed in water (Hill 1995). Optimum temperatures for infection are between 10°C to 15°C (Hill 1995). Temperatures above 24°C and below 10°C inhibit infection (Hill 1995). The cyclical production of urediospores during the growing season serves as a continuous inoculum source throughout the life cycle of the crop. When plants are physiologically stressed the disease tends to be more severe (Hill 1995).

3.9.4 Management

Healthy cloves should be planted in well-drained soils (Chupp and Sherf 1960, Hill 1995). Crop spacing to increase air flow around and within the canopy may decrease humidity levels within the canopy and decrease disease incidence. Crop rotation and control of wild *Allium* spp. within and surrounding the field may help to reduce disease incidence (Chupp and Sherf 1960, Hill 1995). A regular fungicide (*e.g.* maneb or zineb) spray schedule should result in adequate protection although Koike *et al.,* (2001) found that none of the registered materials including maneb, gave satisfactory control. Tebuconazole and azoxystrobin, not registered for control, provided good protection against rust when sprayed at 10-day intervals (Koike *et al.,* 2001). Although resistance among *Allium* spp. has been reported no acceptable levels of resistance have been found among garlic cultivars (Koike *et al.,* 2001, Hill 1995).

3.10 Smut

The smut is caused by the Basidiomycete fungus, *Urocystis cepulae* Frost (syn *Urocystis magica* Pass. in Thum and *Urocystis colchici* var. *cepulae* Cooke). This pathogen is worldwide in distribution. Stand reductions of 50 to 80% and higher have been reported where onion is grown in infested fields and the seed had not been treated with a systemic fungicide (Hoepting *et al.* 2000).

U. cepulae infects only *Allium* species. Common and Welsh onion, leek and shallot are very susceptible, whereas garlic and wild leek (*A. tricoccum* Ait.) are only moderately susceptible. Several *Allium* species are resistant to smut.

3.10.1 Symptoms

The first symptoms of Smut are visible on the cotyledon (flag leaf) and first true leaf of the onion seedling. A thickening and darkening of these leaves is visible as soon as they emerge from the soil. Long, dark blisters or pustules containing powdery black spores of the Smut fungus form within the leaves. The leaves may become bloated and distorted as they grow and often split open releasing the spores.

Smut infection may kill seedlings before they emerge from the ground or during the first three to four weeks after emergence. Some times, however, the pathogen remains isolated in the cotyledon and when this first leaf dies, the plant continues to grow free of infection. When the seedling is not killed, the pathogen then continues to grow systemically within with the plant, progressively invading the leaf bases of successive leaves. Some plants die during the growing season, while others survive and produce bulbs with the characteristic elongated dark pustules.

3.10.2 Causal organism

The pathogen*, U. cepulae,* is heterothallic and produces distinctive multi-celled teliospores, which are an important taxonomic feature of this pathogen. Each teliospore consists of a central; smooth; spherical to ellipsoidal; thick-walled cell that is dark brown and 12 to 15 μm in diameter. The central cell is surrounded by numerous colourless, thin-walled accessory cells that are 4 to 6 μm in diameter (Walker 1921b). There are conflicting reports in the literature as to whether the accessory cells can germinate or if only the central cell is capable of germination.

3.10.3 Disease cycle

The teliospores of *U. cepulae* can persist in soil for at least 15 years and are the primary overwintering and infective form of this fungus. Spore germination may be stimulated by root exudates from *Allium* species (Fig. 1). The seedling is susceptible to infection from about the second day after germination until the first true leaf emerges, a period of about 12 to 15 days, depending on weather conditions. Infection takes place before the cotyledon emerges from the soil. Each new onion leaf goes through a growth phase during which it is susceptible to infection. However, developing onion leaves remain
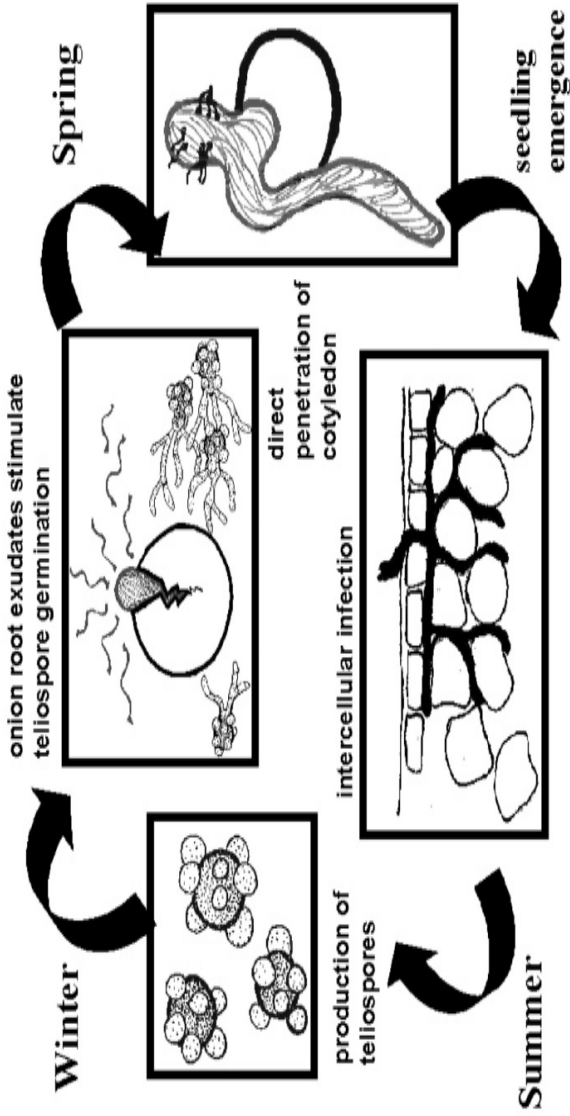
Figure 1 : Life cycle of *Urocystis cepulae*, the causal agent of onion smut. (Courtesy of C. A. Hoepting).

enclosed within the preceding leaf until they emerge from the ground, so if the preceding leaf has not been infected, the succeeding one will not become infected. Thus, if the cotyledon and the first true leaf emerge uninfected, the plant is then resistant to infection. There are two mating types of this pathogen; teliospores of both must be present for infection to take place, although this does not appear to be a limiting factor in the field.

Smut spores germinate at 13 to 22EC. The optimum range for germination on agar is 20 to 24EC. Soil temperatures of 29EC completely inhibit fungal growth. Low soil temperatures result in increased infection levels by slowing seedling emergence, thereby lengthening the time that the seedling is susceptible to infection. Deep planting of onion seed can also result in increased infection levels by delaying seedling emergence (Steinstra and Lacy 1972). Soil moisture levels have no direct effect on the germination of teliospores or infection.

The pathogen can be spread in infested soil and water and in infected plant parts. In the field, the Smut pustules burst and release the teliospores, recontaminating the surrounding soil. Onion smut was most likely introduced into many growing regions through infected onion sets. There are no reports of seed transmission.

3.10.4 Management

Growers should avoid contaminating smut-free fields with infested soil or crop residues. If onion sets are to be grown, they should be closely inspected and any that are diseased should be discarded. Smut-free sets or seedling onions for transplanting are immune to infection and can safely be grown in Smut-infested fields. Some success in breeding for resistance has been achieved by hybridizing the common onion with the Welsh onion, but in general the commercial onion cultivars are very susceptible.

Where onion is grown from seed, seed dressing with a suitable systemic fungicide is effective in protecting seedlings. Individual growers can apply the fungicide to raw seed or purchase seed that has received fungicide in the coating process from commercial seed companies. When applying fungicide to raw seed, the use of a sticker, such as a 1% methyl-cellulose solution, increases the retention of the fungicide on the seed. A drench or granular application of non-systemic fungicide into open seed furrow at planting can also provide protection from Smut. In fields where very high levels of inoculum is present growers have resorted to using treated seed in combination with an in-furrow fungicide (Hoepting *et al.* 2000)

3.11 Allium White Rot (WR)

The disease is caused by the soil-borne fungus *Sclerotium cepivorum* Berk, is a common and sometimes destructive disease of onion, garlic, shallot, and leek throughout the world (Utkhede 1982), and is reported as a limiting factor for continued commercial production of *Allium* species (Entwistle 1990b, Crowe *et al.* 1994). There are no reports of WR in wild species of *Allium* except occasionally near cultivated areas. This suggests that the widespread occurrence of WR results from transportation of infected planting material, such as garlic cloves (Campacci 1946), seedlings (Tims 1948, Coley-Smith 1990) or onion sets (Coley-Smith 1990). WR threatens the onion and garlic

industries of Canada (Utkhede and Rahe 1978, Banks and Edginton 1989), USA, (Adams 1981, Adams and Ayers 1981), New Zealand (Kay and Stewart 1994a), Brazil (Cruz *et al.* 1973), UK (Mordue 1976), Egypt (Mikhail *et al.* 1974) and Mexico (Laborde 1987, Perez-Moreno *et al.* 1990).

3.11.1 Symptoms

Infected plants may die before emergence, and the symptoms are similar to those caused by damping-off fungi, allowing the disease to go undetected (Entwistle 1990b). The first aboveground symptoms of WR may become visible within 20 days of infection. Wilting, yellowing and dieback of leaf tips progress down the leaf blades until the affected leaves collapse (Walker and Tims, 1924, Tims 1948). The roots are gradually destroyed, and there is a watery decay of the scales followed by luxurious growth of superficial, white fluffy mycelium and semi-watery decay around the base of the bulbs (Walker and Tims, 1924, Tims 1948). Masses of tiny black sclerotia form on the mycelium in the roots and bulb tissue of onions. In the advanced stages of infection, diseased plants are easily pulled due to the rot of the roots and bulbs. When the root system is large and the water supply abundant, foliar symptoms may not be evident even though the pathogen is active. Affected plants often die before harvest or rot in storage; even small amounts of damage decrease the market value of the crop (Tims 1948).

3.11.2 Causal organism

S. cepivorum is a soil borne fungus in the division Ascomycota (Alexopoulus *et al.* 1996). The fungus produces sclerotia that are the major means of survival and the principal means of dissemination. (Tims 1948, Mordue 1976). Sclerotia of *S. cepivorum* consist of a narrow rind, whose cells have thickened pigmented walls, surrounding a medulla of closely interwoven, moderately thick-walled hyphae approximately 300 μm in diameter with interhyphal spaces filled with medullary tissue (Tims 1948, Mordue 1976).

3.11.3 Disease cycle

Sclerotia are the primary inoculum of WR. The pathogen can persist as dormant sclerotia in the soil for many years in the absence of *Allium* hosts. (Coley-Smith 1960, Coley-Smith *et al.* 1987b). Viable sclerotia have been observed after 18 years in the field in the absence of host plants (Coley Smith and Sansford 1987). They are subject to exogenous dormancy, which is imposed by the fungistatic influences of the soil microflora, but they germinate readily in sterile soil (Coley-Smith 1960, Coley-Smith and Holt 1966, Bristow and Lockwood 1975). The period of dormancy lasts for 1-3 months (Brix and Zinkernagel 1992), and during this time sclerotia rarely germinate even in the presence of host plants (Coley-Smith 1987b). Sclerotia are difficult to eliminate from soil because they are adapted to survival under severe environmental conditions. They germinate any time after onions or other *Allium* hosts have been seeded (Howard *et al.* 1994) and

after the sclerotia overcome dormancy (Coley-Smith 1987b, Brix and Zinkernagel 1992). Germination of sclerotia in soil is triggered by the presence of *Allium* species (Coley-Smith and Holt 1966, Esler and Coley-Smith 1983), specifically root exudates (Coley-Smith and King 1969, Coley-Smith and Cooke 1971). These exudates are metabolized by the soil microflora to yield volatile sulphur compounds that stimulate dormant sclerotia to germinate (King and Coley-Smith 1968, Coley-Smith and King 1969). Primary infection is by hyphae that originate from germinating sclerotia (Scott 1956). The mycelium has almost no survival ability and if infection does not occur within 10-14 days, the germinated sclerotia die and decay (Coley-Smith 1960, Crowe and Hall 1980).

Typically *S. cepivorum* enters and invades the host from the roots (Fig. 2 a) or the base of the bulb (Walker and Tims 1924) , but penetration of the stem base has been observed (Stewart *et al.* 1989a). The soil temperatures (10°-18°C) that contribute to the
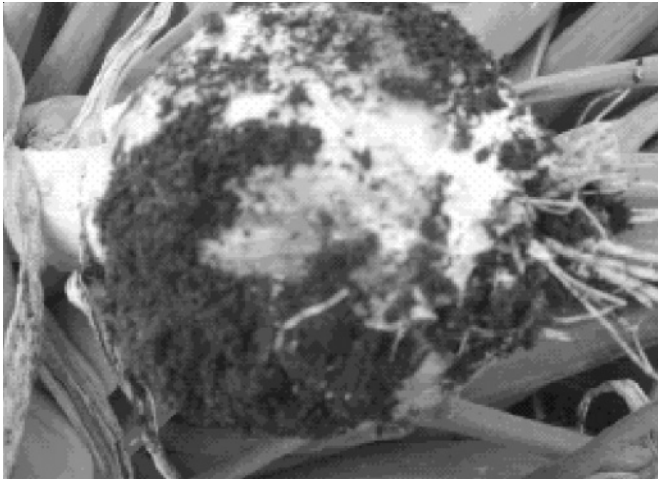
Figure 2a: Symptoms of Allium white rot on onion. Note white mycelium at the base of the bulb

development of this pathogen may occur for only part of the *Allium* growing season and this governs the time when the symptoms appear (Crowe and Hall 1980, Coley-Smith 1990, Entwistle 1990b). Moderate soil moisture levels (-45 mbars to -3 bars) encourage WR development. In dry conditions and at high moisture levels, WR development is restricted (Coley-Smith 1979, Crowe and Hall 1980).Secondary infection occurs by growth of hyphae to the roots or stem bases of adjacent plants (Scott 1956). A colonized bulb develops a watery soft rot, and thousands of sclerotia are formed, imbedded inside the bulb (Fig. 2b) and root tissue and in the surrounding soil (Tims 1948). These sclerotia can persist in soil until another host crop is grown, at which time they germinate and incite disease. WR occurs in all soil types on which *Allium* species are grown and is found on mildly acidic and alkaline soils (Walker and Tims 1924, Howard *et al.* 1994). WR often develops in patches within a field due to the non-uniform distribution of sclerotia (Adams 1981). Infested soil is soon spread within a field and

also to nearby fields by wind, water and equipment (Walker and Tims 1924, Crowe *et al.* 1980). The disease can also be spread to non-infested areas when infected transplants and onion sets are used (Scott 1956, Howard *et al.* 1994).

3.11.4 Management

There are several methods that can contribute to the effective management of *Allium* White Rot. Most researchers advocate an integrated approach to the management of this disease. Control measures include avoidance, cultural methods, chemical control, biological control, resistant varieties, sclerotial germination stimulants, and the incorporation of crucifer leaves or leaf extracts. Sites with a history of WR should be avoided because even small numbers of affected plants produce large numbers of

Figure 2b: Symptoms of Allium white rot on onion showing small black sclerotia that form in the mycelium

sclerotia (Crowe *et al.* 1990). Soil samples can be tested for the presence of sclerotia but this is more successful on mineral, rather than organic soils. Planting material should be produced only in areas where the disease is absent and the material should be inspected. At the same time, the use of clean and disinfected machinery, pallet boxes, tools, and irrigation water free of sclerotia should be considered to avoid the increase and spread of sclerotia of *S. cepivorum* (Howard *et al.* 1994, Anon. 2000).

There is continued emphasis on the development of cultural methods for reducing the incidence of WR. The removal and destruction of infected plants during the

growing season (roguing) can slow the build up of soil borne inoculum (Laborde 1987, Howard *et al.* 1994, Anon. 2000). Ideally, plants should be removed at an early stage when sclerotia are still firmly attached to the plant. The effectiveness of roguing is improved when adjacent soil and neighbouring plants are also removed. Incineration, aerobic composting (Bollen *et al.* 1989, Entwistle 1990b, Melero-Vara *et al.* 2000), fumigation, solar heating (Entwistle 1990b) and microbial control (Entwistle 1988) are effective ways of disposing of rogued plants and soil materials.

Crop rotation is not effective for the control of *S. cepivorum* because the sclerotia are extremely long lived and may remain dormant in the absence of a host for as long as 20 years (Coley-Smith *et al.* 1990). The small population of viable sclerotia remaining in the field after crop rotation can be sufficient to induce disease in the next season that an *Allium* crop is grown (Banks and Edgington 1989). The time of planting has been tested as a cultural control of WR, based on the fact that temperature is one of the environmental factors, which is critical for germination of sclerotia and growth of *S. cepivorum* (Entwistle and Munasinghe 1978, Adams 1987, Gerbrandy 1989, Entwistle 1990a). The worst effects of WR can sometimes be avoided by taking advantage of temperatures adverse to *Allium* White Rot. Pinto *et al.* (1998) found that early (warmer) planting of garlic in January was effective as a management strategy to avoid WR in Brazil. Early harvest of onions has been suggested as an alternative practice to reduce the incidence of WR in areas where disease tends to appear late in the season. In Ontario, Canada, this is starting to become a regular practice to avoid or reduce the incidence of disease. Growers also find that artificial curing with heated forced air (25-30°C and 25-35% RH) can reduce the development of White Rot symptoms in storage.

Soil solarization has proven to be a satisfactory control method, bringing soil populations of the pathogen to negligible levels in areas with appropriate weather conditions (Coley-Smith *et al.,* 1987a, Basallote and Melero 1993), and has provided partial control in other areas (Porter and Merriman 1985, Entwistle 1990b, Pereira *et al.* 1996). Disease incidence was markedly lower in solarized soil, and garlic and onion yields were improved (Porter and Merriman 1985, Satour *et al.* 1989, Basallote and Melero 1993, Cunha *et al.* 1993, Basallote *et al.* 1994). The proportion of sclerotia killed depended on the magnitude of the temperature rise, its duration and penetration into the soil (Porter and Merriman 1983, Melero-Vara *et al.* 2000).

Flooding of infested fields for up to three weeks can reduce sclerotial populations (Banks and Edgington 1989, Coley-Smith *et al.* 1990). This control measure may be effective because flooded soil undergoes various chemical transformations under anaerobic conditions (Banks an Edgington 1989). It also causes changes in microbial populations, which can promote parasitism of sclerotia by naturally occurring microorganisms (Leggett and Rahe 1985). This treatment provided 70% control of WR (Banks and Edgington 1989). Flooding for three months during the winter significantly reduced the viability of aged sclerotia but not young sclerotia (Banks and Edgington 1989). Although flooding promotes sclerotial decay, a portion of the population of sclerotia of *S. cepivorum* (1-5%) was not affected by flooding (McDonald *et al.* 1994). This remaining inoculum is sufficient to induce disease if onions are planted the following season. As a result, flooding is only recommended as part of an integrated disease management program (Banks and Edgington 1989). Host plant density has been shown to affect

disease levels in a number of other pathosystems (Crowe and Hall 1980a, Crowe *et al.* 1980), and several reports in the literature have suggested that increased levels of WR could be associated with increased host density (Scott 1956). This suggests that by decreasing plant density in fields, a reduction in disease incidence may be obtained, however, decreasing host plant density also can result in decreased yield.

There have been several attempts to control WR by the use of antagonistic microorganisms with varying results. The application of fungal and bacterial antagonists to the soil opens the possibility of control without chemicals. Among the microorganisms tested were: *Bacillus spp*. (Backhouse and Stewart 1989, Dickinson and Coley-Smith 1970), *Trichoderma harzianum* (Abd-El-Moity and Shatla 1981, Kay and Stewart 1994b), *Chaetomium globosum*, *Sporidesmium sclerotiorum* (Adams and Ayers 1982, Adams and Ayers 1981, Adams and Ayers 1979) and *Coniothyrium minitans* (Ahmed and Tribe 1977, Turner and Tribe 1976). Further study of these biological controls is needed. Although they appear to be potential agents for WR control, no large scale field trials have been undertaken with some of these agents (Coley-Smith 1990) and there are no reports of season-long disease control. Vesicular arbuscular mycorrhizae fungi (VAM) have shown potential as biological control for White Rot (Torres-Barragan *et al.* 1996, Jaime 2001). Pioneer research using VAM fungi (*Glomus* spp.) for the control of WR found that mycorrhizal colonization delayed WR epidemics by two weeks and provided significant protection against the disease for 11 weeks after transplanting in mineral soils (Torres-Barragan *et al.* 1996). These results imply limited protection of onions against *S. cepivorum* by *Glomus* spp. A reduction in the incidence of White Rot at harvest was found in transplanted onions using two commercially available VAM products MIKRO-VAM® (MIKRO-TEK, Timmins, Ontario), PRO-MIX PGX® with MYCORISE® 1000 (Premier Tech, Rivière-du-Loup, Quebec). White Rot incidence was reduced by almost 50% and was equivalent to the disease control provided by two banded sprays of Folicur 3.6F® (430 g a.i./L tebuconazole, 1 L/ha, Bayer) when roots were colonized by VAM before the plant was transplanted in infested fields (Jaime 2001). Also there was a significant correlation between lower levels of disease and higher levels of mycorrhizal colonization of the roots (Jaime 2001).

Many efforts have been made to detect resistance to *S. cepivorum* in edible species of *Allium* in field and greenhouse tests. Early studies reported that very little genetically resistant germplasm could be identified in *Allium cepa* (Coley-Smith and Esler 1983, Esler and Coley-Smith 1983, 1984), and later studies found that the resistance was not consistent (Utkhede and Rahe 1978, Rahe 1986). Promising results have been reported more recently, and some resistance has been identified in available breeding lines and cultivars (Hovius and McDonald 1998a, 1998b, Earnshaw *et al.* 2000, Jaime 2001). Attempts have been made to find resistance in other species of *Allium*, and in general, garlic is more susceptible and leek appears to be more resistant to WR than onion (Adams 1981, Coley-Smith and Esler 1983, Van der Meer *et al.* 1983, Coley-Smith 1986). A resistance breeding program has been ongoing for the last 20 years at the University of Wisconsin, Madison (Dr. I. Goldman) as well as in Israel (Dr. Kik) and several European countries, and possible sources of resistance have been found in ornamental and wild *Allium spp.* (Kik, personal communication). The major seed companies should be able to provide information about onion cultivars with partial resis-

tance to White Rot.

Irradiation has been used to induce mutation to increase resistance to WR, resulting in successful disease reduction in onions (Utkhede and Rahe 1982); similar results have been obtained in garlic (Perez-Moreno *et al.* 1995, Al-Safadi *et al.* 2000). However, some problems have been found in the agronomic performance and marketable characteristics of these *Allium* lines.

Fungicides can be effective for the control of WR (Coley-Smith 1990). Application of fungicides is difficult due to the soil-borne nature of the pathogen (Entwistle 1990b). Treatment of planting material (seed, seedlings, sets) is a convenient method of incorporating fungicides into the soil. Applying fungicides to the soil surface before emergence or to the base of the plant after emergence may extend the period of protection (Entwistle 1990b).

The fungicides tested, Iprodione (Rovral FLO® 250g a.i./kg, Rhone-Poulec) and Vinclozolin (Ronilan FL®, 500g a.i./kg, ICI), provided complete control when Iprodione was applied as a seed dressing and in mid-season as a stem base drench. Unfortunately, the dicarboximide fungicides Iprodione and Vinclozolin failed to control White Rot adequately in subsequent years due to enhanced degradation in previously treated soils (Entwistle and Munasinghe 1980).Procymidone (Sumisclex 25F®, ICI) has been used to control WR in New Zealand, Australia and Europe (Entwistle and Marian 1983a, b, Fullerton and Stewart 1991, Stewart and Fullerton 1991). Placement of Procymidone in a band below the seed or soil treatments (Sumisclex® 50g a.i./kg seed, technical grade) combined with aerial applications (Sumisclex 25F®, ICI, 0.75kg a.i./ha), provided effective control in bulb onions (Wong and Maynard 1986, Porter *et al.* 1991, Fullerton *et al.* 1995). Tebuconazole (Folicur® 500g a.i./ha, Bayer) was effective against WR when applied to soil (Dennis 1997, Jaime *et al.* 1999, Jaime 2001) or to garlic cloves (Jackson *et al.* 1997). Folicur 3.6F® (430g a.i./L tebuconazole, 1 L/ha, Bayer, Canada) reduced the incidence of White Rot by almost 50% in transplanted onions when two basal sprays 5 & 10 wks after transplant were applied (Jaime 2001). Tebuconazole can be highly phytotoxic, causing seed and seedling mortality when used as a seed treatment for onions at rates greater than 1g a.i./kg seed (Raxil®, Bayer, UK) (Fullerton *et al.* 1995, Davies *et al.* 1998).

Metham sodium, a partial soil sterilant, has been used as a drench for many years in the UK to reduce sclerotium populations in small plots (Coley-Smith 1985). Recent studies in Mexico have found metham sodium and thiocyanomethylbenzothiasol (TCMTB) effective for control of WR on garlic by reducing survival and viability of sclerotia (Perez-Moreno *et al.* 1996). The high cost of fumigation may be justified only for seed beds or perennial *Allium* crops (Entwistle 1990b). However, disease control can be variable, probably because of adsorption, a lack of penetration, or persistence of the active ingredient in soil (Adams and Johnston 1983, Wong and Maynard 1986).

Methods for decreasing sclerotial populations in the soil have also been evaluated. These are based on the specific response of sclerotia of *S. cepivorum* to germinate when exposed to the flavour and odour compounds of certain *Allium* species (King and Coley-Smith 1968, Coley-Smith and King 1969) and the inability of germinated sclerotia to survive in the absence of the host. Onion oil or synthetic garlic oil (diallyl disulphide {DADS}) incorporated into the soil in the absence of a host plant, stimulates the

germination of sclerotia which makes them predisposed to starvation and susceptible to antagonism, lysis and predation (Merriman *et al.* 1980, Coley-Smith *et al.* 1981, Entwistle *et al.* 1982). The use of sclerotial germination stimulants is of interest because it is possible to achieve control through a reduction in sclerotial populations (primary inoculum) in infested soils and not merely by protecting plants in ways that may leave the populations intact (Coley-Smith and Parfitt 1986).

In Australia, artificial onion oil (Bush Boake Allen Pty Ltd, London, England commodity code C7713) at a 5% concentration in water, injected 10 cm into soil, at least 4 weeks before seeding, reduced sclerotial numbers at 0-10 and 10-20 cm by 77 and 91%, respectively (Merriman *et al.* 1980). On organic soils synthetic garlic oil (diallyl disulphide or DADS, United Agri-Products) and to a lesser extent, di-N-propyl disulfide, effectively reduced White Rot when applied to the soil twice before onions were grown (Hovius and McDonald 2002). Dehydrated garlic powder was evaluated for the ability to reduce populations of sclerotia (Crowe *et al.* 2000). Food-grade, commercially dehydrated garlic powder (from several processing companies in California) induced germination levels of 97-99%, which was similar to the synthetic garlic oil, DADS. Research on organic soils has also demonstrated that garlic powder and natural garlic oil are potential controls for WR on onions (Jaime *et al.* 2000).

The use of cruciferous plant residues to reduce the amount of sclerotia of *S. cepivorum* and chlamydospores of *Fusarium oxysporum* f.sp. *lycopersici* in soil was investigated (Smolinska 2000). This study showed that cruciferous plants have potential to decrease the survival of long-lived fungal propagules in the soil. It was suggested that the toxic compounds that are released during decomposition of plants might kill or weaken the propagules and make them more sensitive to the activity of mycoparasitic microorganisms (Smolinska 2000).

A comprehensive program of integrated control was developed for Mexican garlic crops (Perez-Moreno, personal communication). The integrated approach involves the field sampling of soils to measure the population of sclerotia, formaldehyde treatment of cloves used as planting material, the use of protective fungicides such as dicarboximides, roguing of infected plants showing early symptoms of the disease, solarization of infested areas and the cleaning of farm equipment when it is moved from infested to clean areas. Sclerotial germination stimulants (SGS) have now been added to this integrated program (Perez-Moreno, personal communication). In Ontario, Canada, Banks and Edgington (1989) proposed an integrated control program that involved crop rotation with carrots, winter or spring flooding, seed dressing with vinclozolin (Ronilan 50WP, BASF, 50g a.i./kg seed) and stem-base sprays during the growing season. The integrated programs have shown considerable promise (Coley-Smith 1990, Entwistle1990b).

The following Integrated Disease Management (IDM) program is suggested for *Allium* White Rot on organic soil. First, select a cultivar with partial resistance. Cv. Fortress has shown consistent significant resistance to disease (Jaime 2001). The soil should be treated with SGS twice, once during the fall after onions are harvested and again in the spring before a next rotation crop is seeded (Hovius and Mc Donald 2002). The use of SGS will decrease the populations of sclerotia of *S. cepivorum* in soil, resulting in a decrease of disease pressure that will allow for improved performance of

the other mechanisms of control. Also, SGS may contribute to a decrease in the populations of teliospores of Onion Smut (*Urocystis cepulae*) and subsequently reduce the incidence of both diseases in direct seeded onions (Hoepting 2001).

For the protection of the host in the field, two options could be used: VAM or the fungicide, Folicur 3.6F®. The presence of VAM may also contribute to a reduction in the incidence of other diseases, such as Pink Root in onions caused by *Pyrenochaeta terrestris* (Becker 1976). VAM benefits the onions, and may also be beneficial to other crops, such as carrots and lettuce, that are normally used for crop rotation at the Bradford Holland Marsh, since high mycorrhizal dependency has been reported for both crops (Hamel 1996). The fungicide Folicur 3.6F® could be considered for the IDM program to reduce the spread of the disease through late season sprays in foci where symptoms of disease appear. The practices described above should be coordinated with the cultural practices available for the management of disease, such as removal and destruction of infected plants, early harvest, and artificial curing to maintain the disease damage below the economical injury level.

4. Viral Diseases

Viral diseases of garlic are worldwide, and losses in yield and quality as a result of virus infection are serious problems. The incidence of virus diseases in onions is relatively low. Garlic is normally produced by vegetative propagation, which accounts for the heavy viral infection; most plants are infected with a mixture of up to five different viruses (Dovas *et al.* 2001).

Onion Yellow Dwarf Virus (OYDV) and Leek Yellow Stripe Virus (LYSV) are the most important viruses of garlic and are reported to occur worldwide (Van Dijk 1993a). Yield losses due to viruses have been reported ranging from 25-50%. Other viruses such as carlaviruses and allexivirus are latent in the leaves and cloves and do not appear to affect yield significantly (Van Dijk *et al.* 1991).

4.1 Viruses by means of transmission

Aphids transmit several potyviruses including Onion Yellow Dwarf Virus (OYDV), Leek Yellow Stripe Virus (LYSV), and Shallot Yellow Stripe Virus (SYSV). In garlic crops the virus infection observed symptoms are mainly caused by OYDV and LYSV (Van Dijk 1993a). LYSV and OYDV are transmitted by aphids in a non-persistent manner, and their spread is mainly attributed to non-colonizing aphid species (Bos *et al.* 1978). The effect of these viruses on yield of different cultivars were studied and a reduction of bulb weight was reported ranging from 39% to 60% for OYDV, and from 17% to 54% for LYSV.

The symptoms of OYDV on infected onions are yellow and light green striping, chlorotic streaks or stripes, leaf curling and pronounced stunting. The striping is clear on older leaves but less distinct on younger leaves. Infected bulbs store poorly and sprout prematurely. Plants infected with LYSV show light yellow stripes on the tips of older and intermediate leaves. The symptoms are harder to distinguish on younger leaves and infected plants are more susceptible to frost damage (Lot *et al.* 1998). OYDV causes severe symptoms and great reduction in bulb weight.

Potyviruses can also cause mosaic virus symptoms. Turnip mosaic virus (TuMV), classified into the potyviruses, has occasionally been reported to infect wild *Allium* species (Dovas *et al.* 2001).

The carlaviruses transmitted by aphids are Garlic Common Latent Virus (GCLV) and Shallot Latent Virus (SLV). Garlic Latent Virus (GLV), when isolated from garlic, is considered identical to SLV. GCLV occurrence has been reported in most countries, but it is not endemic in Japan, Taiwan or Thailand. SLV has been detected predominantly in Asia and Europe. These carlaviruses are also transmitted by aphids in a non-persistent manner (Van Dijk 1993b, Barg *et al.* 1994, Barg *et al.* 1997) and have a wide host range with in *Allium* species. It is also possible that carlaviruses are mainly transmitted by vegetative propagation.

There is currently no available data concerning the effect of garlic carlaviruses on symptomatology, or yield loss of various garlic cultivars, however it is likely the co-infection with potyviruses, may further induce yield reduction as reported for SLV and LYSV in leek (Dovas *et al.* 2001, Lee *et al.* 1979).

Mites transmit several viruses. These belong to the group of Allexivirus (Mite-borne viruses) and include Onion Mosaic Virus (OMV), Onion Mite-Borne Latent Virus (OMbLV), Shallot Mite-Borne Latent Virus (SMbLV), Shallot Virus X (ShVX), Garlic Virus A (GarV-A), Garlic Virus B (GarV-B), Garlic Virus C (GarV-C), and Garlic Virus D (GarV-D). Mite-Borne Viruses cause faint short stripes, mild mosaic or no symptoms in *Allium* species (Yamashita *et al.* 1996, Van Dijk 1991) and are transmitted by the erio-phyid mite *Aceria tulipae* (Van Dijk 1991). Their economic significance to crop production has not yet been determinated. In *Allium* species allexiviruses occur in complexes, interfering in the virus identification. The spread of allexiviruses seems to take place primarily during bulb storage, where mites can easily spread and rarely in the field (Lange and Mann 1960). GarV type viruses are not reported to be associated with any particular symptom.

The topsovirus transmitted by thrips is Iris Yellow Spot Virus (IYSV). IYSV is transmitted by the thrips *Frankliniella occidentalis* and *Thrips tabaci*. The infection of this virus has often reached 50-60% yield reduction in onions (Gera *et al.* 1997). The symptoms in onions are straw coloured, chlorotic and necrotic lesions on leaves (Kritzman *et al.* 2001). IYSV is not a seedborne virus; therefore neither seeds nor infected plants are source of IYSV.

4.2 Management

Considering the significant impact on yield and quality of the virus infections and their rapid spread by the vectors such as aphids, mites or thrips, we emphasize the need to use certified seed bulbs produced under an appropriate certification scheme. The use of onion plants raised from seeds is an important practice for the reduction of virus infection of onions, since *Allium* seed does not transmit viruses. In cultural practices the prevention of or total avoidance are the most important strategies to use.

Tissue culture provides a useful technique for eliminating viruses from infected plantlets and for producing virus free garlic seedlings. The tissue culture could be produced from shoot tip. Various tissue culture techniques have been reported such as

stem disc, shoot-tip, cloves, bulbs or bulbils. Elimination efficiencies of viruses by meristem tips cultures of garlic have been reported between 54 to 100% depending on the type of viruses (Verbeek *et al.* 1995).

Thermotherapy has been suggested to eliminate virus from garlic; 90-100% of OYDV has been eliminated from garlic seed (Ucman *et al.,* 1998). For the management of virus diseases in onion and garlic the identification of the vectors provides valuable information and the control of these vectors with registered insecticides, will considerably reduce the incidence of the disease (Ebi *et al.* 2000). Follow IPM recommendations for the spray schedule.

In Western Europe, researchers had been able to reduce the economic impact of OYDV due to such control measures as the introduction of a period free of an onion crop to prevent an overlapping cultivation of diseased crops and new plantings.

5. References

Abawi, G.S. and Lorbeer, J.W. 1971. Reaction of selected onion varieties to infection by *Fusarium oxysporum* f.sp. *cepae*. Plant Disease reports,55:1000-1004.

Abawi, G.S., and Lorbeer., J.W. 1972. Several aspects of the ecology and pathology of *Fusarium oxysporum* f. sp. *cepae*. Phytopathology,61:1042-1048.

Abd-El-Moity, T. H. and Shatla, M. N. 1981. Biological control of white rot disease of onion (*Sclerotium cepivorum*) by *Trichoderma harzianum*. Phytopathologische Zeitschrift, 100:29-35.

Adams, P. B. 1981. Forecasting onion white rot disease. Phytopathology, 71:1178-1181.

Adams, P. B. 1987. Effects of soil temperature, moisture, and depth on survival and activity of *Sclerotinia minor*, *Sclerotium cepivorum*, and *Sporidesmium sclerotivorum*. Plant Disease, 71:170-174.

Adams, P. B. and Ayers, W. A. 1979. Ecology of *Sclerotinia* species. Phytopathology, 69:896-899.

Adams, P. B. and Ayers, W. A. 1981. *Sporidesmium sclerotivorum*: distribution and function in natural biological control of sclerotial fungi. Phytopathology, 71:90-93.

Adams, P. B. and Ayers, W. A. 1982. Biological control of *Sclerotinia* lettuce drop in the field by *Sporidesmium sclerotivorum.* Phytopathology, 72:485-488.

Adams, P. B. and Johnston, S. A. 1983. Factors affecting efficacy of metham applied through sprinkler irrigation for control of *Allium* white rot. Plant Disease, 67:978-980.

Ahmed, A. H. M. and Tribe, H. T. 1977. Biological control of white rot of onion (*Sclerotium cepivorum*) by *Coniothyrium minitans*. Plant Pathology, 26:75-78.

Alexopoulus, C. J., Mims, C. W. and Blackwell, M. 1996. Introductory Mycology. John Wiley & Sons. Toronto.

Al-Safadi, B., MirAli, N. and Arabi, M.I. 2000. Improvement of garlic (*Allium sativum* L.) resistance to white rot and storability using gamma irradiation induced mutations. Journal of Genetics and Breeding, 54:175-181.

Alvarez, A.M., Buddenhagen, I.W., Buddenhagen, E.S. and Domen, H.Y. 1978. Bacterial blight of onion a new disease caused by *Xanthomonas* sp. Phytopathology,73:1132-1136.

Anonymous, 2000. Vegetable Production Recommendations 2000-2001. Edited by Editor. Vol. Publication 363. Ontario Ministry of Agriculture, Food and Rural Affairs. Toronto.

Aveling, T.A.S., Snyman, H.G. and Naude, S.P. 1993 Evaluation of seed treatments for reducing *Alterneria porri* and *Stemphylium vesicarium* on onion seed. Plant Disease, 77(10):1009-1011.

Backhouse, D. and Stewart, A. 1989. Interactions between *Bacillus* species and sclerotia of

Sclerotium cepivorum. Soil Biology and Biochemistry, 21:173-176.

Ballard, R.W., Palleroni, N.J., Doudoroff, M., Stanier R.Y. and Mandel, M. 1970. Taxonomy of aerobic pseudomonads: *Pseudomonas cepacia, P. marginata, P. alliicola and P. caryophylli*. Journal General Microbiology,60:199-214.

Banks, E. and Edgington, L. V. 1989. Effect of integrated control practices on the onion white rot pathogen in organic soil. Canadian Journal of Plant Pathology, 11:268-272.

Barg, E., Lesemann, D.E., Vetten, H.J. and Green, S.K. 1994. Identification, partial characterisation and distribution of viruses infecting *Allium* crops in south and south-east Asia. Acta Horticulturae,358:251-258.

Barg, E., Lesemann, D.E., Vetten, H.J. and Green, S.K. 1997. Viruses of *Allium*s and their distribution in different *Allium* crops and geographical regions. Acta horticulturae, 433:607-616.

Basallote, U. M. and Melero, V. J. 1993. Control of garlic white rot by soil solarization. Crop Protection, 12:219-223.

Basallote, M. J., Bejarano, J., Blanco, M. A., Jimenez, D. R. and Melero, J. M. 1994. Soil solarization: a strategy for the control of diseases caused by soilborne plant pathogens and reducing of crop rotations. Investigación Agraria, Producción y Protección Vegetales, 2:207-220.

Becker, W. N. 1976. Quantification of onion vesicular arbuscular mycorrhizae and their resistance to *Pyrenochaeta terrestris*. PhD Thesis, University Illinois, Illinois.

Bertolini, P. and Tian, S.P. 1996. Low-temperature biology and pathogenicity of *Penicillium hirsutum* on garlic in storage. Postharvest Biology and Technology, 7:83-89.

Bodner, J., Schumacher, B. and Uyenaka, J. 1998. Garlic Production. Ontario Ministry of Agriculture and Food Factsheet. Agdex:258/13. 7 pp.

Bottcher, H. 1987. Studies on the occurrence of neck rot (*Botrytis allii*) in stored onions and its effective control. Arch. Phytopathol. 3:227-240.

Bottcher, H. and Gunther, I. 1994. Quality changes of dry garlic (*Allium sativum* L) during long-term storage. I. External quality. Nahrung-Food, 38:61-69.

Bishop, A. L. and Davis, R. M. 1990. Internal breakdown of onions caused by *Enterobacter cloacae*. Plant Disease, 74:692-694.

Bollen, G. J., Volker, D. and Wijnen, A. P. 1989. Inactivation of soil-borne plant pathogens during small-scale composting of crop residues. Netherlands Journal of Plant Pathology, Suppl 1: 19-30.

Bos, L., Huijberts, N., Huttinga, H. and Maat, D.Z. 1978. Leek yellow stripe virus and its relationship to onion yellow dwarf virus; characterization, ecology and possible control. Netherlands Journal of Plant Pathology, 84:185-204.

Bradbury, J.F.1977. *Erwinia carotovora* var *carotovora*. CMI Description of Pathogenic Fungi and Bacteria. No. 552. Commonwealth Mycological Institute. Kew, Surrey, England. 2 pp.

Brewster, J.L. 1994. Onions and other vegetable Alliums. CAB International , wllingford, U.K. 236 pp.

Bristow, P. R. and Lockwood, J. L. 1975. Soil fungistasis: role of spore exudates in the inhibition of nutrient-independent propagules. Journal of General Microbiology, 90:140-146.

Brix, H. D. and Zinkernagel, V. 1992. Screening for resistance of *Allium* species to *Sclerotium cepivorum* with special reference to non-stimulatory resistance. Plant Pathology, 41:308-316.

Burkholder, W.H. 1942. Three bacterial plant pathogens: *Phytomonas carophylli* sp. n., *Phytomonas alliicola* so. n., and *Phytomonas manihotus* (Arthand, Berhet & Bondar) Vilgas. Phytopathology, 32:141-149.

Burkholder, W.H. 1950. Sour skin, a bacterial rot of onion bulbs. Phytopathology, 40:115-117.

Campacci, C. A. 1946. Podridao branca do alho e da cebola. Biologico, 12:279-281.

Chupp, C. and Sherf, A.F. 1960. Vegetable Diseases and Their Control. John Wiley and Sons, Inc. 693 p.

Coley-Smith, J. R. 1960. Studies of the biology of *Sclerotium cepivorum* Berk. IV. Germination of sclerotia. Annals of Applied Biology, 48:8-18.

Coley-Smith, J. R. 1979. Survival of plant pathogenic fungi in soil in the absence of host plant. In: "Soil Borne Plant Pathogens". (eds. Schippers, B. and Gams, W.) Academic Press. New York, pp. 39-57.

Coley-Smith, J. R. 1985. Methods for the production and use of sclerotia of *Sclerotium cepivorum* in field germination studies. Plant Pathology, 34:380-384.

Coley-Smith, J. R. 1986. Interactions between *Sclerotium cepivorum* and cultivars of onion, leek, garlic and *Allium fistulosum*. Plant Pathology, 35:362-369.

Coley-Smith, J. R. 1990. White rot disease of *Allium*: Problems of soil-borne diseases in microcosm. Plant Pathology, 39:214-222.

Coley-Smith, J. R. and Holt, R. W. 1966. The effects of species of *Allium* on germination in soil of sclerotia of *Sclerotium cepivorum* Berk. Annals of Applied Biology, 65:56-63.

Coley-Smith, J. R. and King, J. E. 1969. The production by species of *Allium* of alkyl sulphides and their effect on germination of sclerotia of *Sclerotium cepivorum* Berk. Annals of Applied Biology, 64:289-301.

Coley-Smith, J. R. and Cooke, R. C. 1971. Survival and germination of fungal sclerotia. Annual Review of Phytopathology, 9:65-92.

Coley-Smith, J. R., Esler, G. A., New, C. M., Clark, W. S., McPherson, G. M., Ryan, E. W. and Doyle, J. A. 1981. New approaches to the control of soil-borne pests and diseases. In: "Proceedings of the 11th British Insecticide and Fungicide Conference, Edited by. at Brighton, England", pp.459-501.

Coley-Smith, J. R. and Esler, G. 1983. Infection of cultivars of onion, leek, garlic and *Allium fistulosum* by *Sclerotium cepivorum*. Plant Pathology, 32:373-376.

Coley-Smith, J. R. and Parfitt, D. 1986. Some effects of diallyl disulphide on sclerotia of *Sclerotium cepivorum*: possible novel control method for white rot disease of onions. Pesticide Science, 17:587-594.

Coley-Smith, J. R. and Sansford, C. E. 1987. Survival of sclerotia of *Sclerotium cepivorum*. In: "Proceedings of the Third International Workshop on *Allium* White Rot, Edited by Entwistle, A. R. at Wellsbourne, UK", pp.11-16.

Coley-Smith, J. R., Reese, R. A. and Georgy, N. I. 1987. Differential stimulation of germination of sclerotia of *Sclerotium cepivorum* by cultivars of onion and its effect on white rot disease. Plant Pathology, 36:246-257.

Coley-Smith, J. R., Mitchell, C. M. and Sansford, C. E. 1990. Long-term survival of sclerotia of *Sclerotium cepivorum* and *Stromatinia gladioli*. Plant Pathology, 39:58-69.

Cother, E.J., Darbyshire, B. and Brewer, J. 1976. *Pseudomonas aeruginosa*: Cause of internal brown rot of onion. Phytopathology, 66:828-834.

Cother, E.J. and Dowling, V. 1985. Association of *Pseudomonas cepacia* with internal breakdown of onion- a new record for Australia. Australasian Plant Pathology, 14:10-12.

Cother, E.J. and Dowling, V. 1986. Bacteria associated with internal breakdown of onion bulbs and their possible role in disease expression. Plant Pathology, 35:329-336.

Cramer, C.S. 2000. Breeding and genetics of *Fusarium* basal rot resistance in onion. Euphityca, 115:159-166.

Crowe, F.J. 1995. Fusarium Basal Rot of Garlic. In: "Compendium of Onion and Garlic Diseases". (eds. Schwartz, H.F. and Mohan, S.K.), APS Press, Minnesota. 11p.

Crowe, F. J. and Hall, D. H. 1980. Soil temperature and moisture effects on sclerotium germination and infection of onion seedlings by *Sclerotium cepivorum*. Phytopathology, 70:74-78.

Crowe, F. J., Hall, D. H., Greathead, A. S. and Baghott, K. G. 1980. Inoculum density of

Sclerotium cepivorum and the incidence of white rot of onion and garlic. Phytopathology, 70:64-69.

Crowe, F. J., Vomocil, J., Somerville, P. A. and Debons, J. 1990. Use of synthetic stimulants of sclerotial germination as a method of inoculum management of the *Allium* white rot fungus, *Sclerotium cepivorum*. In: "Proceedings of the Fourth International Workshop of *Allium* white rot", (ed. Weinstrasse). at Neustadt, Germany. pp.140-159.

Crowe, F. J., Debons, J., Darnell, T., McGrath, D., Koepsell, P., Laborde, J. and Redondo, J. 1994. Control of *Allium* white rot with DADS and related products. In: "Proceedings of the Fifth International Workshop on *Allium* white rot" (eds. Entwistle, A. R. and Melero-Vara, J. M.), Cordoba, Spain. pp.15.

Crowe, F. J., Davis, M., Nunez, J., Smith, R. and Darnell, T. 2000. Dehydrated garlic powder used to reduce *Sclerotium cepivorum* in field soil. Phytopathology, 91:S183.

Cruz, B. P. B., Teranishi, J., Bernardi, J. B. and Silveira, S. G. P. 1973. White rot of garlic: Behaviour of varieties. Biologico, 39:151-157.

Cunha, M.-G. D., Zambolim, L., Vale, F., Chaves, G. M. and Alves, H. 1993. The effect of soil solarization on the survival of sclerotia of *Sclerotium cepivorum* in the soil. Fitopatologia Brasileira, 18:55-61.

Davies, J. M. L., Hiron, L. C., Peterson, C. D. and Watling, M. 1998. Evaluation of tebuconazole and prochloraz seed treatment for the control of *Allium* white rot in salad and bulb onions. In: "Proceedings of the Sixth International Workshop on *Allium* white rot. Fungicide control"., (eds. Entwistle, A. R. and Perez-Moreno, L.), Irapuato, Guanajuato, Mexico. pp.6-10.

Dennis, J. J. C. 1997. Progress towards an integrated control strategy for onion white rot disease, including the use of artificial germination stimulants. In: "Proceedings of the Second International Symposium on edible *Alliaceae*, at Adelaide, Australia". pp.39.

Dickinson, D. J. and Coley-Smith, J. R. 1970. Stimulation of soil bacteria by sclerotia of *Sclerotium cepivorum*. Soil Biology and Biochemistry, 2:157-162.

Dillon Weston, W.A.R. and Taylor, R.E. 1943. Development of *Penicillium* on the cut surfaces of certain vegetables. Nature, 151:54-55.

Dixon, G. R. 1981. Vegetable Crop Diseases. AVI Publishing Co. Westport, Connetticut.

Dovas, I.C., Hatsiloukas, E., Salomon, R., Barg, E., Shiboleth, Y. and Katis, N.I. 2001. Incidence of viruses infecting *Allium* spp. in Greece. European Journal of Plant Pathology, 107:677-684.

Earnshaw, D. M., McDonald, M. R. and Boland, G. J. 2000. Interactions among isolates and mycelial compatibility groups of *Sclerotium cepivorum* and cultivars of onion (*Allium cepa*). Canadian Journal of Plant Pathology, 22:387-391

Ebi, M., Kasai, N. and Masuda, K. 2000. Small inflorescence bulbils are best for micropropagation and virus elimination in garlic. HortScience, 35:735-737.

Ellis, M.B. and Holliday, P. 1970. *Alternaria porri*. CMI descriptions of Pathogenic Fungi and Bacteria, No. 248. Commonwealth Mycological Institute. Kew, Surrey, England 2 p.

Ellis, M.B. and Waller, J.M. 1974. *Botrytis allii*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 433. Commonwealth Mycological Institute. Kew, Surrey, England 2 p.

El-Shabrawy, A.M.,Amein, A.M., Hussein, F.N. and Ali, A.A. 1987. Cultural practices in relation to garlic storage diseases. Assiut- Journal of Agricultural Science, 18:5-16.

Entwistle, A. R. 1988. Opportunities for the microbial control of *Allium* white rot. Bulletin Supersedes Organization Europeenne et Mediterraneenne Pour la Protection des Plantes, 18:19-28

Entwistle, A. R. 1990a. Onions and Allied crops: Root diseases. In: "Onion and Allied crops". (eds. Rabinowitch, H. D. and Brewster, J. L.) CRC Press. Florida, USA,pp. 103-154.

Entwistle, A. R. 1990b. *Allium* white rot and its control. Soil Use and Management, 6:201-209

Entwistle, A. R. and Munasinghe, H. L. 1978. Prospect of an effective check for white rot in

onions. Grower, 89:701-702.

Entwistle, A. R. and Munasinghe, H. L 1980. The effect of seed, furrow and stem base spray treatment with iprodione on white rot disease (*Sclerotium cepivorum*) in spring-sown salad onions. Annals of Applied Biology, 94:215-224.

Entwistle, A. R., Merriman, P. R., Munasinghe, H. L. and Mitchell, P. 1982. Diallyl-disulphide to reduce the numbers of sclerotia of *Sclerotium cepivorum* in soil. Soil Biology and Biochemistry, 14:229-232.

Entwistle, A. R. and Marian, S. E. 1983a. Evaluation of carboxin, oxycarboxin, triadimefon and tolclofos-methyl for the control of white rot in spring-sown salad onions. Annals of Applied Biology, 102:64-65.

Entwistle, A. R. and Marian, S. E. 1983b. Evaluation of thiabendazole for the control of white rot in spring-sown salad onions. Annals of Applied Biology, 102:66-67.

Esler, G. and Coley-Smith, J. R. 1983. Flavour and odour characteristics of species of *Allium* in relation to their capacity to stimulate germination of sclerotia of *Sclerotium cepivorum*. Plant Pathology, 32:13-22.

Esler, G. and Coley-Smith, J. R. 1984. Resistance to *Sclerotium cepivorum* in *Allium* and other genera. Plant Pathology, 33:199-204.

Etoh, T., Noma, Y., Nishitarumizu, Y., and Wakamoto, T. 1988. Seed productivity and germinability of various garlic clones collected in Soviet Central Asia. Memoirs of the Faculty of Agriculture of Kagoshima University. 24:129-139.

Everts, K.L., Schwartz, H.F., Epsky, N.D., and Capinera, J.L. 1985. Effects of maggots and wounding on occurrence of *Fusarium* basal rot of onions in Colorado. Plant Disease, 69:878-882.

Fahy, P.C., Tesoriero, L.A. and Gun, L.V. 1981-1982. First record in Australia of bacterilasoft rot on onion (*Allium cepa* L. var *cepa*) caused by *Pseudomonas gladioli* pv.*alliicola*. Plant survey pp 32.

Fullerton, R. A. and Stewart, A. 1991. Chemical control of onion white rot (*Sclerotium cepivorum* Berk.) in the Pukekohe district of New Zealand. New Zealand Journal of Crop and Horticultural Science, 19:121-127.

Fullerton, R. A., Stewart, A. and Slade, E. A. 1995. Use of demethylation inhibiting fungicides (DMIs) for the control of onion white rot (*Sclerotium cepivorum* Berk.) in New Zealand. New Zealand Journal of Crop and Horticultural Science, 23:121-125.

Gardner, M.W. 1940. Garlic rust in California. Plant Disease Reporter, 24:298-299.

Gera, A., Leseman, D.E., Cohen J., Franck, A., Levy, S. and Salomon, R. 1997. The natural occurrence of turnip mosaic virus in *Allium ampeloprasum*. Jorunal of Phytopathology, 145:289-293.

Gerbrandy, S. J. 1989. The effects of various temperatures during storage in soil on subsequent germination of sclerotia of *Sclerotium cepivorum*. Netherlands Journal of Plant Pathology, 95:319-326.

Gitaitis, R.D., Baird, R.E., Beaver, R.W., Sumner, D.R., Gay, J.D. and Smittle, D.A. 1991. Bacterial blight of sweet onion caused by *Pseudomonas viridiflava* in Vidalia, Georgia. Plant Disease, 75:1180-1182.

Gitaitis, R.D. and Gay, J.D. 1997. First report of a leaf blight seed stalk rot, and bulb decay of onion by *Pantoea ananas* in Georgia. Plant Disease, 81:1096.

Gitaitis, R.D., MacDonald, G., Torrance, R., Hartley, R., Sumner, D.R., Gay, J.D. and Johnson, W.C. III. 1998. Bacterial streak and bulb rot of sweet onion. II. Epiphytic survival of *Pseudomonas viridiflava* in association with multiple weed hosts. Plant Disease, 82: 935-938.

Goldman, I.L., Schroeck, G. and Havey, M.J. 2001. History of public onion breeding programs in the U.S. Plant Breeding Reviews, 20:67-103.

Griesbach, J.A., Putnam, M.L. and Driesner, D. 2001. First report of garlic rust caused by *Puccinia allii* in Oregon. Plant Disease, 85:919.

Hadar, Y., Hamar, G.E., Taylor, A.G. and Norton, J.M. 1983. Effects of pregermination of pea and cucumber seeds and of seed treatments with *Enterobacter cloacae* on rots caused by *Pythium* spp. Phytopathology, 73:1322-1325.

Hamel, C. 1996. Prospects and problems pertaining to the management of arbuscular mycorrhizae in agriculture. Agriculture, Ecosystems and Environment, 60:197-210.

Harrow, K.M. and Harris, S. 1969. Artificial curing of onions for control of neck rot (*Botrytis allii* Munn.). New Zealand Journal of Agricultural Research, 12:592-604.

Hattingh, M.J. and Walters, D.F. 1981. Stalk and leaf necrosis of onion caused by *Erwinia herbicola*. Plant Disease, 65:615-618

Havey, M.J. 1995. Fusarium Bals Plate Rot . *In* Compendium of Onion and Garlic Disease. Schwartz, H.F. and Mohan, S.K. eds. APS Press, Minnesota. pp. 10-11.

Hildebrand, P.D. and Sutton, J.C. 1982. Weather variables in relation to an epidemic of onion downy mildew. Phytopathology 72:219-224.

Hill, J.P. 1995. Rust. In: "Compendium of Onion and Garlic Disease". (eds. Schwartz, H.F. and Mohan, S.K.) APS Press, Minnesota. pp. 24-25.

Hoepting, C. A. 2001. Pesticide interactions in the management of onion maggot, *Delia antiqua* (Meigen) and onion smut, *Urocystis cepulae* Frost, in Ontario. Master Thesis, Environmental Biology, University of Guelph, Guelph.

Hoepting, C.A., Scott-Dupree, C.G., Ritcey,G., Harris, C.R. and McDonald, M.R. 2000. Evaluation of insecticide and fungicide combinations for the control of onion maggot, *Delia antigua* (Meig.) and onion smut, *Urocystis cepulae* Frost, in Ontario. Proceedings of the Brighton Crop Protection Conference - Pest and Diseases - 2000 pp. 287-291.

Hovius, M. H. Y. and McDonald, M. R. 1998a. Evaluation of commercial yellow cooking onion cultivars and breeding lines for resistance to white rot using a scale inoculation technique. In Biological and Cultural Tests for Control of Plant Diseases. pp.164-165

Hovius, M. H. Y. and McDonald, M. R.. 1998b. Field evaluation of commercial yellow cooking onion cultivars and breeding lines for resistance to the white rot pathogen, *Sclerotium cepivorum* Berk. In Biological and Cultural Tests for Control of Plant Diseases. American Phytopathological Society pp.166.

Hovius M.H.Y. and McDonald M.R. 2002. Management of *Allium* white rot (*Sclerotium cepivorum*) in organic soil with soil applied diallyl disulfide and di-N-propyl disulfide. Canadian Journal of Plant Pathology, 24:287-291.

Howard, R. J., Garland, J. A. and Seaman, W. L. (eds.) 1994. Disease and pests of vegetable crops in Canada., Printing Ltd. Ottawa.

Isakeit, T., Miller, M.E., Barnes, L.W., Dickstein E.R. and Jones, J.B. 2000. First report of leaf blight caused by *Xanthomonas campestris* in the continental United States. Plant Disease, 84:201.

Jackson, K. J., Duff, A. A. and O'Donell, W. E. 1997. Tebuconazole (Folicur) shows potential in the control of white rot (*Sclerotium cepivorum*) in garlic in subtropical Queensland, Australia. In: "Proceedings of the Second Symposium on edible Alliaceae", Adelaide, Australia. pp.42.

Jackson, K., Henderson, C., Duff, A. and Lovatt, J. 1999. Growing Onions. Before you start. DPI note. www. Dpi,gld.gov.on.au/horticulture/5387.html.

Jaime, M.D.L.A. 2001. Biological control of *Allium* white rot caused by *Sclerotium cepivorum* Berk, in onions in organic soil. Master Thesis, Plant Agriculture, University of Guelph, Guelph.

Jaime, M. D. L. A., Hovius, M. H. Y. and McDonald, M. R. 1999. Field evaluation of tebuconazole to control onion white rot, In: "Fungicide and Nematicide Tests", pp.22.

Jaime, M. D. L. A., Hsiang, T. and McDonald, M. R. 2000. Evaluation of sclerotia germination stimulants for the control of white rot, *Sclerotium cepivorum* Berk. on onions. In: "Muck Vegetable Cultivar Trial Research Report". (eds. McDonald, M. R., Janse, S. and Vander Kooi, K.) University of Guelph. Kettleby, Ontario, pp. 83-84.

Jay, J.M. 2000. Modern Food Microbiology. 6th ed. Aspen Publishers, Gaithersburg, Maryland.

Jennings, D.M, Ford-Lloyd, B.V. and Butler, M. 1990. Morphological analysis of spores from different *Allium* rust populations. Mycological Research, 92:230-232.

Jesperson, G.D. and Sutton, J.C. 1987. Evaluation of a forecaster for downy mildew of onion (*Allim cepa* L.). Crop Protection 6:95-103.

Kawamoto, S.O. and Lorbeer, J.W. 1974. Infection of onion leaves by *Pseudomonas cepacia*. Phytopathology, 64:1440-1445.

Kawamoto S.O. and Lorbeer J.W. 1976. Protection of onion seedlings from *Fusarium oxysportum* f.sp. *cepae* by seed and soil infestation with *Pseudomonas cepacia*. Plant Disease, 60:189-191.

Kay, S. J. and Stewart, A. 1994a. Evaluation of fungal antagonists for control of onion white rot in soil box trials. Plant Pathology, 43:371-377.

Kay, S. J. and Stewart, A. 1994b. The effect of fungicides on fungal antagonists of onion white rot and selection of dicarboximide-resistant biotypes. Plant Pathology, 43:863-871.

Kehr, A.E., Obrien,M.J. and Davis, E.W. 1962. Pathogenicity of *Fusarium oxysporum* f. sp. *cepae* and its interaction with *Pyenochaeta terrestris* on onion. Euphytica, 11:197-208.

King, J. E. and Coley-Smith, J. 1968. The effect of volatile products of *Allium* species and their extracts on germination of *Sclerotium cepivorum* Berk. Annals of Applied Biology, 61:407-411.

Kishun, R. and Swarup, J. 1981. Growth studies on *Pseudomonas gladiolli* pv. *alliicola* pathogenic to onion. Indian Journal of Mycology and Plant Pathology, 11:247-250.

Koike, S.T., Smith, R.F, Nunez, J.J. and Voss, R.E. 2001. Characterization and control of garlic rust in California. Plant Disease, 85:585-591.

Kritzman, A., Lampel, M., Raccah, B. and Gera, A. 2001. Distribution and transmission of Iris yellow spot virus. Plant Disease, 85:838-842.

Kritzman, G. and Netzer, D. 1978. A selective medium for isolation and identification of *Botrytis* spp. from soil and onion seed. Phytoparasitica, 6:3-7.

Laborde, J. A. 1987. Coexistence of garlic white rot in commercial production in Central Mexico. In: "Proceedings of the Third International Workshop on *Allium* white rot", (ed. Entwistle, A. R.) at Wellesbourne, UK. pp. 24-40.

Lacy, M.L. 1987. Timing of sprays for onion diseases with automated field weather stations. In: "Proceedings of the National Onion Research Conference, 1987. Denver, Colorado" (eds. Schwartz, H.F. and McBride,T.M.), pp. 52-54.

Lange, W.H. and Mann, L.K. 1960. Fumigation controls microscopic mite attacking garlic. California Agriculture December, 1960:9-10.

Latham, A.J. and Watson, R.D. 1966. Effect of specific crop residues on soil fungi, onion infection and bulb rotting. Plant Disease Reports, 50:469-472.

Lee, Y.W., Yamazaki, S., Osaki, T., and Inouye, T. 1979. The elongated viruses in garlic, garlic latent virus and garlic mosaic virus. Annals of the Phytopathological Society of Japan, 45:727-734.

Leggett, M. E. and Rahe, J. E. 1985. Factors affecting the survival of sclerotia of *Sclerotium cepivorum* in the Fraser Valley of British Columbia. Annals of Applied Biology, 106:255-263.

Lot, H., Chovelon, V. , Souche, S. and Delecolle, B. 1998. Effect of yellow dwarf and leek yellow stripe viruses on symptomatology and yiled loss of three French garlic

cultivars. Plant Disease, 82:1381-1385.

Lund, B.M. 1983. Bacterial spoilage: Post harvest pathology of fruits and vegetables. Colin Dennis. London: Academic Press. p.219-257.

Maude, R.B. 1990. Storage Disease of Onions. In: "Onions and Allied Crops".(eds. Rabinowitch, H.D. and Brewster, J.L.) CRC Press. Florida, USA. pp. 273-296.

Maude, R.B. and Presly. 1977. Neck rot (*Botrytis allii*) of bulb onions I. seed-borne infection and its relationship to the disease in the onion crop. Ann. Appl. Biol. 86:163-180.

McDonald, M. R., Janse, S. and Goldman, I. L. 1994. Evaluation of onion lines for resistance to white rot and onion maggot. In: "Proceedings of the Fifth International Workshop on *Allium* white rot", (eds. Entwistle, A. R. and Melero-Vara, J. M.), Cordoba, Spain. pp.8-14.

Melero-Vara, J. M., Prados-Ligero, A. M. and Basallote-Ureba, M. J. 2000. Comparison of physical, chemical and biological methods of controlling garlic white rot. European Journal of Plant Pathology, 106:581-588.

Merriman, P. R., Isaacs, S., Macgregor, R. R. and Towers, G. B. 1980. Control of white rot in dry bulb onions with artificial onion oil. Annals of Applied Biology, 96:163-168.

Mikhail, S., Stewart, D. M., El-Haggagy, M. K. and Wilkinson, R. E. 1974. The role of grazing animals in the dissemination of the onion white-rot pathogen in Egypt. FAO Plant Protection Bulletin, 22:37-41.

Mordue, J. E. M. 1976. *Sclerotium cepivorum*. In: "CMI Descriptions of pathogenic fungi and bacteria". Commonwealth Mycological Institute. Kew, Surrey, 2 p.

Mukerji, K.G. 1975. *Peronospora destructor*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 456. Commonwealth Mycological Institute. Kew, Surrey England. 2p.

Nunez, J.J., Gilbertson, R.L., Meng, X. and Davis, R.M. 2002. First report of *Xanthomonas* leaf blight of onion in California. Plant Disease, 86:330.

O'Garro, L.W. and Paulraj, L. 1997. Onion leaf blight caused by *Xanthomonas campestris*: alternative hosts and resistant onion genotypes. Plant Disease, 81:978-982.

Paulraj, L. and O'Garro, L. W. 1993. Leaf blight of onions in Barbados caused by *Xanthomonas campestris*. Plant disease, 77(2):198-201.

Pereira, J. C. R., Chaves, G. M., Zambolim, L., Matsuoka, K., Acuna, R. S., Vale, F. D. and Vale, F. R. 1996. Control of *Sclerotium cepivorum* by the use of vermicompost, solarization, *Trichoderma harzianum* and *Bacillus subtilis*. Summa Phytopathologica, 22:228-234.

Perez-Moreno, L., Lopez-Munoz, J., Pureco-Munoz, A. and Hinojosa-Rangel, J. C. 1990. Generation of variability in garlic (*Allium sativum* L.) for resistance to white rot (*Sclerotium cepivorum* Berk.) by the radioinduced mutagenesis method. Revista Mexicana de Fitopatologia, 8:153-159.

Perez-Moreno, L., Salinas-Gonzalez, J. G. and Sanchez-Pale, J. R. 1995. Performance and yield of garlic (*Allium sativum* L.) germplasm tolerant to white rot (*Sclerotium cepivorum* Berk.), generated through gamma irradiation mutagenesis. Revista Mexicana de Fitopatologia, 13:18-25.

Perez-Moreno, L., Sanchez-Pale, J. R. and Entwistle, A. R. 1996. Effects of metham sodium, diallyl disulphide and thiocyanomethylbenzothiasol on *Sclerotium cepivorum* sclerotia in the Bajio region of Mexico. Annals of Applied Biology, 128:6-7.

Pinto, C. M. F., Maffia, L. A., Berger, R. D., Mizubuti, E. S. G. and Casali, V. W. D. 1998. Progress of white rot on garlic cultivars planted at different times. Plant Disease, 82:1142-1146.

Pooler, M.R. and Simon, P.W. 1994. True seed production in garlic. Sexula Plant Reprotduction 7:282-286.

Porter, I. J. and Merriman, P. R. 1983. Effects of solarization of soil on nematode and fungal pathogens at two sites in Victoria. Soil Biology and Biochemistry, 15:39-44.

Porter, I. J. and Merriman, P. R. 1985. Evaluation of soil solarization for control of root diseases of row crops in Victoria. Plant Pathology, 34:108-118.

Porter, I. J., Maughan, J. P. and Towers, G. B. 1991. Evaluation of seed, stem and soil applications of procymidone to control white rot (*Sclerotium cepivorum* Berk.) of onions. Australian Journal of Experimental Agriculture, 31:401-406.

Pretorius, Z.A. and Dunhin, B.J. 2001. Garlic Rust. http://www.sun.ac.za/agric/ plant/Page1A.html

Rahe, J. E. 1986. Detection and selection for field resistance to onion white rot. In: "Proceedings of the Third International Workshop on *Allium* white rot", (ed. Entwistle, A. R.), TW. Printing Associates Ltd. at Wellesbourne UK. pp.11-17.

Raper, K.E. and Fennell, D.I. 1965. The Genus *Aspergillus*. The Williams and Wilkins Co., Baltimore.

Richard, C. 1984. *Enterobacter*. In: "Bergey's Manual of Systemic Bacteriology. Vol 1." (eds. Krieg, N.R. and Holt, J.G.) Williams & Wilkins, Baltimore, pp. 465-469.

Roumagnac, P., Gagnevin, L. and Pruvost, O. 2000. Detection of *Xanthomonas* sp., the causal agent of onion bacterial blight, in onion seed using a newly developed semi-selective isolation medium. European Journal of Plant Pathology, 106: 867-877.

Samson, R., Shafik, H., Benjama, A. and Gardan, L. 1998. Description of the bacterium causing blight of leek as *Pseudomonas syringae* pv. *porri*. Phytopathology 88: 844-850.

Satour, M. M., Abdel, R. M., El-Yamani, T., Radwan, A., Grinstein, A., Rabinowitch, H. D. and Katan, J. 1989. Soil solarization in onion fields in Egypt and Israel: short and long-term effects. Acta Horticulturae, 255:151-159.

Schroth, M N., Cho, J J., Green, S K. and Kominos, S D. 1977. Epidemiology of *Pseudomonas aeruginosa* in agricultural areas [Soil and plants serve as the natural and permanent reservoirs for the bacterium]; In: "*Pseudomonas aeruginosa*: Ecological aspects and patient colonization" (ed. Young, V.M.), pp.1-29.

Schwartz, H.F. and Mohan, S.K. 1999. Compendium of Onion and Garlic Diseases. APS Press, Minnesota. 54 p.

Schwartz, H. F. and Otto, K. 2000a. First report of a bulb decay of onion by *Enterobacter cloacae* in Colorado. Plant Disease Note 84:808.

Schwartz, H. F. and Otto, K. 2000b. First report of a leaf blight and bulb decay of onion by *Pantoea ananitas* in Colorado. Plant Disease Note 84:808.

Schwartz, H. F. and Otto, K. 2000c. First report of a leaf blight of onion by *Xanthomonas campestris* in Colorado. Plant Disease Note 84:922.

Scott, M. R. 1956. Studies of the biology of *Sclerotium cepivorum* Berk. I. Growth of the mycelium in the soil. Annals of Applied Biology, 44:576-583.

Serfontein, J.J. 2001. *Xanthomonas* blight of onion in South Africa. Plant Disease, 85:442.

Singh, P.J. and Basandrai, A.K. 1988. New report of garlic rust from Punjab-State. Current Science, 57:266-267.

Smalley, E.B. 1954. *Penicillium* clove rot of garlic. Phytopathology, 44:506.

Smalley, E.B. and Hansen, H.N. 1962. *Penicillium* decay of garlic. Phytopathology, 52:666.

Smolinska, U. 2000. Survival of *Sclerotium cepivorum* sclerotia and *Fusarium oxysporum* chlamydospores in soil amended with cruciferous residues. Journal of Phytopathology, 148:343-349.

Snowdon, A.L. 1992. Color Atlas of Post-harvest Diseases and Disorders of Fruits and Vegetables. Volume 2: Vegetables. CRC Press. pp. 236-262.

Soares, R.M. and Kurozawa, C. 1998. Chemical control of associayed fungi of garlic cloves. Summa-Phytopathologica, 24:279-283.

Stewart, A., Backhouse, D., Sutherland, P.W. and Fullerton, R.A. 1989. The development of

infection structures of *Sclerotium cepivorum* on onion. J. Phytopath. 126:22-32.

Stewart, A. and Fullerton, R. A. 1991. Additional studies on the chemical control of onion white rot (*Sclerotium cepivorum* Berk.) in New Zealand. New Zealand Journal of Crop and Horticultural Science, 19:129-134.

Stiensta, W.C. and Lacy, M.L. 1972. Effect of inoculum density, planting depth, and soil temperature on *Urocystis colchici* infection of onion. Phytopathology, 62:282-286.

Sumner, D.R. 1995. Penicillium Decay of Garlic and Blue Mold. In: "Compendium of Onion and Garlic Disease". (eds. Schwartz, H.F. and Mohan, S.K.), APS Press, Minnesota. pp. 27-28.

Sutton, J.C., James, T.D.W. and Rowell, P.M. 1986. Botcast: A forecasting system to time the initial fungicide spray for managing *Botrytis* leaf blight on onions. Agriculture, Ecosystems and Environment, 18:123-143.

Tesoriero, L.A., Fahy, P.C. and Gun, L.V. 1982. first record of bacterial rot of onion in Australia caused by *Pseudomonas gladioli* pv. *allicola* and association with internal browning caused by *Pseudomonas aeruginosa*. Australasian Plant Pathology, 11:56-57.

Thornton M.K., Mohan, S.K., Knott, E. and Torell, J. 1994. Control of soil borne onion diseases with soil solarization, fumigation and resistant varieties. Biological and Cultural Test, 39.

Tims, E. C. 1948. White rot of shallot. Phytopathology, 38:378-394.

Torres-Barragan, A., Zavaleta-Mejia, E., Gonzales-Chavez, C. and Ferrera-Cerrato, R. 1996. The use of arbuscular mycorrhizae to control onion white rot (*Sclerotium cepivorum* Berk.) under field conditions. Mycorrhiza, 6:253-257.

Turner, G. J. and Tribe, H. T. 1976. On Coniothyrium minitans and its parasitism of *Sclerotinia* species. Transactions of the British Mycological Society, 66:97-105.

Ucman, R., Zel, J. and Ravnikar, M. 1998. Thermotheraphy in virus elimination from garlic: influence on shoot multiplication from meristems and bulb formation *in vitro*. Scientia Horticulturae, 73:193-202.

Utkhede, R. S. 1982. Biology and control of onion white rot. Zeitschrift fuer Pflanzenkrankheiten und Pflanzenschutz, 89:291-301.

Utkhede, R. S. and Rahe, J. E. 1978. Screening world onion germplasm collection for resistance to white rot. Canadian Journal of Plant Science, 58:819-822.

Utkhede, R. S. and Rahe, J. E. 1982. Reduction in white rot incidence by seed irradiation in *Allium* cepa. Plant Disease, 66:723-725.

Van der Meer, Q.P., Van Bennekom, J.L. and Van der Giessen, A.C. 1983. Screening for resistance to white rot caused by *Sclerotium cepivorum* Berk. in onions (*Allium cepa* L.) and leeks (*Allium porrum* L.). Euphytica, 32:697-701.

Van Dijk P. 1991. Mite borne virus isolates from cultivated *Allium* species and their classification into two new Rymoviruses in the family Potyviridae. Netherlands Journal of Plant Pathology, 97:381-399.

Van Dijk P. 1993a. Survey and characterisation of potyviruses and their strains of *Allium* species. Netherlands Journal of Plant Pathology, 99(suppl 2):1-48.

Van Dijk P. 1993b. Carlaviruses isolates from cultivated *Allium* species represent three viruses. Netherlands Journal of Plant Pathology, 99:233-257.

Verbeek, M., Van Dijk, P. and Van well, P.M.A. 1995. Efficacy of eradication of four viruses from garlic (*Allium sativum*) by meristem-tip culture. European Journal of Plant Pathology, 101:231-239.

Vincelli, P.C. and Lorbeer, J.L.1989. Blight-alert: A weather-based predictive system for timing fungicide application on onion before infection periods of *Botrytis squamosa*. Phytopathology, 79:493-498.

Walker, J.C. 1921a. Rust of onion followed by a secondary parasite. Phytopathology, 11:87-90.

Walker, J.C. 1921b. Onion smudge. Journal of Agricultural Research 20:685-721.

Walker, J.C. and Tims, E.C. 1924. A *Fusarium* bulb rot of onion and the relation of environment

to its development. Journal of Agricultural Research, 28:683-694.

Walkey, D.G.A. 1990. Virus Diseases. In: "Onions and *Allium* Crops", Volume II. (eds. Rabinowitch, H. D., and Brewster, J. L.), CRC Press, Boca Raton, FL., pp. 191-212

Wong, J. A. L. and Maynard, J. R. 1986. Application of procymidone to seed and fertlizer for the control of *Allium* white rot in bulb onions. In: "Proceedings of the Third International Workshop on *Allium* white rot", (ed. Entwistle, A. R. T.W.), Printing Associates Ltd. at Wellesbourne, UK. pp.86-92.

Papaya Diseases and Integrated Control

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Abstract: Diseases are a significant limiting factor for papaya production. The nature and frequency of these diseases depend on local conditions and effective management depends on a thorough knowledge of the pathogen, host plant, environment, and their interaction. The precise identity of the causal agent is of paramount importance, and disease management options must be economical. Assessment of disease incidence or severity, and fruit loss are the key factors in determining the economics of disease management. In general, disease management strategies involve different practices that include plant resistance, and prophylactic and curative measures. The papaya diseases related have diverse etiologies, divided into those with biotic (infectious) and abiotic (noninfectious) etiologies that affect the fruit and those affecting the plant. We have attempted to emphasize procedures for diagnosis and control with detailed information on each. Descriptions of pathogens are provided in sufficient detail to assist professional diagnosticians in making accurate diagnosis. Fungicides used to control many of the diseases described in this chapter are mentioned for information proposes only. Legal restrictions and regulations of fungicide use vary among countries, and regulations within any country are subject to change over time. The information reported has been extracted from the available scientific literature and the authors' experience, and we hope that this publication will provide a helpful reference to growers, students, and professionals working with papaya.

1. Introduction

Diseases of papaya stand out economically because their presence can cause severe economic losses in production, sale, and exportation of fresh fruit that may reach in some cases 100%. In Brazil the principle diseases are viruses (Papaya ringspot virus and "meleira") in the field, and anthracnose and peduncular rot, in postharvest. Foliar diseases, such black spot, leaf spot and greasy spot, can also cause significant damage to fruits and reduce their commercialization when not controlled adequately.

In general, the importance of diseases of papayas varies with the region where they are grown, in function of climatic conditions; management of the orchard (cultural management practices) vector populations, inoculum density and destination of production for internal or external markets.

Post-harvest diseases are principally of three types: superficial rots, peduncular rots, and internal fruit rots, that reduce the quality of fruits and cause severe losses, reaching in some cases more than 90% depending on the conditions of harvest, transport and packing, and that may make totally impossible sale in importing markets.

2. Fungal diseases

2.1 Anthracnose

Anthracnose is considered one of the principle postharvest diseases of papaya, occurring in all production regions in the world, having been noted to cause large losses in Brazil, Hawaii (USA) and Mexico. The fungus infects various parts of the plant but its greatest importance occurs in fruits, which become unfit for commercialization.

The latent infection, not detected at harvest, develops after harvest principally during transport of the fruits for export or local markets.

The absence of control measures and use of inappropriate postharvest procedures can result in production of up to 100% diseased fruits from some orchards.

2.1.1 Etiology

The causal agent of anthracnose, the fungus *Colletotrichum gloeosporioides* (Penz.) Penz. Sacc. in Penz., (teleomorphic phase *Glomerella cingulata* (Ston.) Spauld. & Schrenk., is a pathogen common in diverse tropical fruit plants, having been reported in the teleomorphic form in fruits and leaves (Ram, 1984; Costa *et al.*, 2001; Holliday, 1980).

The fungus forms subcuticular and subepidermal acervuli, separate or confluent with pinkish to orange conidial masses that cover the lesion centers, with setae, conidiophores septate, pale brown with hyaline conidia with obtuse ends or ellipsoidal with a rounded apex and a narrow, truncate base, aseptate, more or less guttulate and cylindrical.

Costa *et al.*, (2001) reported the occurrence of the perfect stage (*Glomerella cingulata*), with perithecia on various parts of the host (dead leaves and fruits), solitary or aggregated, globose to obpyriform, dark brown to black, and asci with 8 spores, clavate to cylindrical, with ascospores narrowly oval to cylindrical to fusiform.

It has not been possible to separate isolates of the fungus obtained from different symptoms frequently observed in the fruits and leaves of the papaya plant by morphological characteristics, however, the existence of ecotypes of *C. gloeosporioides* in relation to temperature was shown in other pathosystems in spite of the similarity of the DNA (Estrada *et al.*, 2000).

The occurrence of a new anthracnose disease on papaya fruits caused by *Colletotrichum capsici* was found at Miyako Island, Okinawa prefecture, located in the south-western part of Japan in 1994. These lesions were initially water-soaked, light brown spots. They gradually enlarged, became sunken and turned blackish brown. Numerous acervuli of anthracnose were produced on the fruit lesions. Conidia were falcate, and abundant black setae were found (Yagushi *et al.*, 1998).

2.1.2 Symptoms

The fungus may initially be established in flowers, penetrating through stigmas and scars left by petals and principally by superficial wounds on these tissues.

The greatest damage is on fruits where the fungus causes small, irregular, watersoaked spots that later enlarge and become dark in color. Infection by *C. gloeosporioides* can occur in any stage of development and remain quiescent until the fruits become mature, penetration being direct, through an infection "peg" or by wounds (Chau and Alvarez, 1983a).

When infected fruits begin ripening, small drops ("beads") of latex exuded on the surface are formed. The lesions in the fruits are round and deep, and can attain up to 5cm diameter. With development of the lesions, a pink growth can be observed, arranged in concentric rings of gelatinous aspect that later turn dark, consisting of the reproductive structures of the fungus. The internal tissue of the infected area is firm, with a white-grayish discoloration that turns brown (Fig.1).

The lesions spread easily to healthy tissues, leaving depressions in the fruit,

Figure1: Anthracnose lesion caused by *C. gloeosporioides* on papaya fruit, with the presence of a typical gelatinous mass of pinkish orange color.

and can coalesce forming a large irregular to circular lesions area on the surface of the fruit, up to 10 mm in diameter, sharply defined, and occasionally depressed. In the absence of control and/or under highly favorable climatic conditions, the presence of these lesions can be observed in fruits in the initial stages of maturity, including in the field.

Mature or damaged fruit is more susceptible to infection than immature fruit. Leaf infection does occur and initially appears as small, watersoaked spots of irregular shape. These lesions become brown, with gray-white centers that often fall out. The black, pin-cushion-like fruiting structures of the pathogen can be observed in these old

lesion centers (Simone, 2002).

On petioles, dark lesions are formed with abundant formation of acervuli. The perithecia of the teleomorphic stage of the fungus also develop on the petioles (Fig. 2). Lesions on leaves are less frequent and when they occur, they are circular, with irregular borders and grayish centers, where "black spots", which are the fruiting bodies (structures) of the fungus can be observed. In certain conditions the fungus does not penetrate deeply in the flesh of the fruit, In these cases superficial lesions of yellow brown
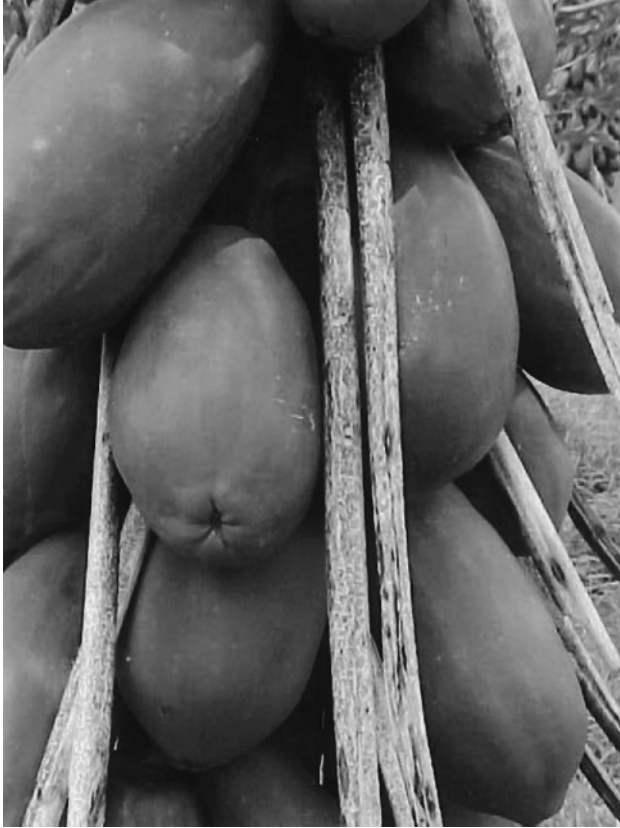
Figure 2: Lesions of anthracnose on petioles of papaya leaves with formation of perithecia of *Glomerella cingulata*, which serve as sources of inoculum of *C. gloeosporioides* underfield conditions.

color, often with a water soaked appearance on the margins occur, which are referred to as "chocolate spot" (Fig. 3).

2.1.3 Epidemiology

The infection generally occurs in the field, during the initial stages of development of

the fruits, the pathogen remaining quiescent until the maturation phase of the fruit, when the symptoms become visible. Fruits and senescent leaves infected by *C. gloeosporioides*, principally the petioles, both on the plant as well as those that fall on the ground, represent important sources of inoculum of the pathogen. The production of ascospores in the petioles of dry leaves has been frequently observed but the importance of the sexual phase in the cycle of the disease still is not clear. In the field, the production of conidia occurs in large quantity principally in the petioles of senescent leaves and in mature fruits, these being considered the source of inoculum. However, additional studies to understand their epidemiological importance are required. Drops of rain and splashes of irrigation water are the principle sources of dissemination of inoculum of the pathogen.

When the conidia germinate (6-8 hours), they form appressoria (10-12 hours), on

Figure 3: Symptoms of chocolate spot on papaya fruit cv. Sunrise Solo.

the surface of the fruits and by enzymatic action penetrate the cuticle, the hyphae remaining subcuticular in a latent state until the beginning of maturation of the fruits, when growth resumes and symptoms of the disease appear.

Conidia dispersed in the initial phases of development of the fruits have less chance of survival than those that are deposited on developed fruits and that remain and form the quiescent infections or infect the fruits after harvest.

Termination of quiescence of the fungus is related to the action of antifungicidal compounds (Prusky, 1996), or to the production of ethylene in the maturation of the fruits that favors the germination and formation of appressoria of *C. gloeosporioides*

(Dickman, and Alvarez, 1983; Dickman et al, 1982 and 1983; Flaishman and Kolattukudy, 1994).

The mechanism of quiescence appears to represent a case of co-evolution of the pathosystem, bringing advantages to both the pathogen and host in the ecosystem. To the pathogen, it represents adaptation to the physiology of the host, and to the host, resistance to the pathogen, paralyzing the evolution of infection of the tissues of the fruit. This has the advantage of making possible development of the seeds, with liberation of the seeds occurring after maturation of the fruit and disintegration of the pulp by the pathogen and saprophytes (Arauz, 2000).

Climatic conditions that favor the incidence of anthracnose are temperatures near 28ºC, ranging from 20 to 30ºC, and relative humidity of the air more than to 95%. The conidia require water in the free state to germinate and are liberated from the acervuli only when the relative humidity is greater than 95% (Quimio, 1973). Severity of the disease depends on environmental conditions, being less severe in dry periods and lower temperatures. In the north of the Espírito Santo, Brazil, these conditions normally occur from April to August. In research conducted by *Incaper,* where monitoring of the disease in fruits during the period of July 1997 to March 1998 was conducted, the incidence of anthracnose was greater than 70% from September to March. The greatest incidence was reported in the months of November (94,44%), December (97,22%) and January (100%), the months of November and December having the greatest rainfall, with precipitation of 292 and 194mm, respectively (Costa *et al.*, 2002; Tatagiba *et al.*, 2002).

2.1.4 Control

The management of anthracnose in the field should begin with the choice of the field (planting) site, taking into consideration the prior history (old plantings), avoidance of excess humidity and conditions that favor the development of the disease, as well as cultural practices, the reduction of inoculum, chemical control and genetic resistance. Measures adopted during the phases of production and post-harvest processing of the fruits (care in handling, cleanliness of packaging and of the environment, control of storage temperatures, use of heat treatments and chemical control) influence the incidence and severity of the disease and when well managed, significantly reduce losses (Ventura, 1995, Ventura and Costa, 2002).

2.1.4.1 Cultural practices

i. Balanced fertilization and management of irrigation: Plants with unbalanced nutrition and water stress become predisposed to an increase in the severity of anthracnose. Studies conducted by *Incaper* verified that doses of boron and calcium greater than the amount required by plants, 0.77g of B and 50g of Ca/plant, contributed to an increase of approximately 70% in the incidence of anthracnose in fruits compared to doses of 0.06g of B and 2.5g of Ca/plant (Tatagiba *et al.*, 1998a). The incidence in fruits reached 100% with a content of boron in the plant of 50mg/kg; the average content generally required being 23mg/kg.

In another experiment conducted in the northern region of Espírito Santo by *Incaper*, where irrigation by microaspersion was utilized, a negative relation between the amounts of water utilized and the incidence of anthracnose was observed, demonstrating the possibility of irrigation management for control of the disease. The amount of 120mm reposition the evapo-transpiration (class A pan evaporation), besides contributing to the greatest production of fruits, was where the least incidence of anthracnose was observed. In the check without application of water, the incidence of the disease in fruits reached 100% (Tatagiba *et al.*, 2001).

ii. Elimination of sources of inoculum: For cleanliness of the orchards, removal of mature fruits and especially infected fruits and senescent leaves both on the plant and those fallen on the ground, is an important procedure. Elimination of infected fruits and leaves can contribute to the reduction of the initial inoculum and to the incidence of anthracnose in the fruits.

iii. Handling of the fruits: Papaya fruits should be harvested when the color of the skin changes from dark green to light green and when one yellow streak begins development from the base upwards. Fruits in this condition will continue to ripen normally after harvest. Those fruits harvested before this stage will fail to show complete ripening, and those harvested after are more susceptible to damage and bruising during handling. After harvest, the fruit are placed in single layers into shallow, light colored field crates, preferably containing a foam layer for cushioning. All stems should be trimmed after harvest to ensure that no stem to fruit rubbing occurs during transport to the packing facilities. Fruit should never be thrown or dropped. Field crates containing the fruit should be left in shaded conditions protected from the sun and rain while awaiting collection for delivery to a packing facility.

Avoid to the maximum causing injuries to the fruits during harvest, transport and storage. These injuries become ports of entry not only to anthracnose but also to other post-harvest diseases. Putting many fruits in the boxes of harvest in the field should also be avoided, the maximum being two layers.

Grading should be carried out as soon as possible after harvest, and fruit left under ambient conditions to continue ripening or placed at 10° to 12°C for cooling and storage. On arrival in the packing facility, fruit should be washed in water to remove latex and debris, then treated for postharvest disease control. Washing, treatment and grading can be carried out using mechanized or manual systems, depending on the volumes of fruits. Storage of unripe papaya below temperatures of 10°C will result in chilling injury. The symptoms are indicated by surface pitting, discoloration of the peel and flesh, incomplete ripening, poor flavor and increased susceptibility to disease.

iv. Sanitation of installations and equipment: Disinfestation of containers, equipment and storage installations is a practice very important to eliminate sources of inoculum. In the packinghouse a solution of chlorine (70-100 ppm) should be used for the required cleaning, and the fruits from the orchards should be washed and disinfested with a similar solution of chlorine adjusted to 70-100 ppm and pH of 6.0-7.5.

2.1.4.2 Chemical control in the field

Anthracnose can be controlled or reduced by pre-harvest sprays. Although symptoms

of anthracnose are observed principally during the phases of transport and storage, control of the disease should begin in the field, with sprays applied during the period of fruiting, reaching flowers, and new and more developed fruits, combined subsequently with the treatments of post-harvest. The interval of application depends of the predominant climatic conditions of the region of production, varying from 7 to 28 days, being generally of 14 days. The fungicides most utilized are chlorothalonil, mancozeb and thiophanate-methyl. The best control is achieved by protective fungicides (chlorothalonil and mancozeb) sprays on the entire fruit and flower column, every 7-14 days during rainy periods and 14-28 days during dry conditions. In the north of the Espírito Santo, Brazil, bi-weekly and monthly applications of these fungicides, in a period of a year, provided levels of control of anthracnose that varied from 16 to 73%, 12 to 51% and 8 to 48%, respectively (Tatagiba *et al.*, 1997 and 1999). These results are similar to those obtained in other production regions of the world, where mancozeb was most effective, but no different statistically to captan and chlorothalonil (Solano and Aruz, 1995).

Few fungicides are registered officially for use on papaya. The use of fungicides on the planting should obey national legislation and also the demands of the importing countries, which normally follow the norms of the Codex (FAO/WHO) and of the EPA (USA).

Dithiocarbamate fungicides, including mancozeb, are effective in the control of the disease, but owing to the production of ethylenethiourea (ETU), are considered restricted in some countries, principally in the United States. A methyl dithiocarbamate like ferbam that does not produce ETU may be an alternative control but requires research to prove its effectiveness in the control of anthracnose.

Other fungicides like benomyl and prochloraz have been used in schedules of control of anthracnose, alternating with protective fungicides. The resistance of *C. gloeosporioides* to benomyl has been detected in the north of Espírito Santo (Ventura and Balbino, 1995) compromising the utilization of this fungicide. There are still no reports of resistance of *C. gloeosporioides* to prochloraz, but resistance in other fungi has been reported, and considering that this fungicide can be used in post-harvest treatment of fruits, its generalized use is not recommended in applications in the field in order to minimize the risk of selecting populations of the pathogen resistant to the fungicide. Other fungicides with potential to control the disease are being evaluated experimentally, but still have no official registration for papaya. As papaya is a sensitive plant, some fungicides can cause phytotoxicity, manifested by injuries to the leaves, on the skin of the fruits and, in some more extreme cases, in slowing of development, or even the death of plants. There have been reported occurrences of phytotoxicity with the fungicides pyrazophos (severe defoliation), and dinocap (causing foliar lesions) (Marin and Gomes, 2000). Fungicides of the triazol group can also cause phytotoxicity in plants and fruits of papaya when used in high doses (Table 1).

2.1.4.3 Post-harvest treatment

Beyond sprays in the orchard, post-harvest treatment of the fruits should proceed in complementary form, as a means of controlling the quiescent mycelium in the fruits and

Table 1: Evaluation of fungicides used by growers to control papaya diseases in relation to phytotoxicity to papaya plants.

Active Ingredient	Commercial Formulation	Dose (% p.c.)	Injury to the Leaves [1]
1. azoxystrobin	Azoxystrobin 80 WG	0,01	0
2. benomyl	Benlate 50 WP	0,08	0
3. captan	Captan 50 WP	0,20	0
4. chinomethionat	Morestan 25 WP	0,06	1
5. chlorothalonil	Daconil 75 WP	0,20	0
6. copper	Reconil 588	0,40	0
oxychoride	WPRecop 84 WP	0,25	0
7. dinocap	Karathane 25 WP	0,10	1
8. diniconazole	S3308L 12,5 WP	0,04	0
9. sulfur	Kumulus 80 WP	0,40	0
10. fenarimol	Rubigan 12 EC	0,06	0
11. mancozeb	Dithane M 45	0,20	0
12. propiconazole	Tilt 250 EC	0,07	2
13. pyrazophos	Afugan 30 EC	0,06	3
14. tebuconazole	Folicur 200 EC	0,25	2
15. thiabendazole	Tecto SC 450	0,10	0
16. thiophanate-methyl	Cercobin 70 WP	0,07	0
17. triforine	Saprol	0,10	0

Source: Liberato *et al.* (1999); Marin (1988); Marin and Gomes (2000).
[1] Scale of rating varying from 0 the 3, being 0=plants without symptoms of phytotoxicity; 1=injury light with small burned areas; 2=leaves chlorotic, with borders and apexs burned; and 3=leaves strongly injured and/or severe defoliation.

protection from secondary infections during the storage and transport to consumer markets.

i. Cleaning of the fruits in water: The fruits should be washed in water to remove soil and other adhering residues, and can also receive a treatment with chlorine for disinfestation. The peduncles should be trimmed and the fruits should be carefully packed when dry with the part of the peduncle down and wrapped in dry paper. This procedure can reduce infection by *C. gloeosporioides* and other fungi that cause post-harvest rots. After cleaning, the fruits should be carefully submerged in tanks of hot water for hydrothermal treatment.

ii. Hydrothermal treatment: Heat treatments have been used to control fungal diseases and insect infestation of papaya fruits for many years. The primary obstacle to the widespread use of heat to control postharvest fruit diseases is the sensitivity of the fruits to the temperatures required for effective treatment with no visible fruit damage. Specialized equipment for temperature maintenance and water circulation is necessary as fluctuations in temperature will reduce the effectiveness of the treatment and may damage the fruit (Couey, 1989).

Hydrothermal or hot-water treatment consists of the immersion of the fruits in hot water of 48°C (± 1°C) for 20 minutes, followed immediately by immersion in cold water of 8°C (± 1°C) for an equal period (Fig. 4). Other combinations of temperature-time of immersion are being tested for effectiveness in the control of post-harvest rots of papaya, as for example the combinations 54°C for 3 minutes and 66°C for 20 seconds.

However, the treatment with hot-water, described initially is that which meets the phytosanitary demands for exportation, since it also includes the capability to kill the eggs and larvae of fruit flies. The principal disadvantage of this treatment is the necessity of a system of heating with precision to maintain the temperature of the water constant during the twenty minutes, because temperatures less than 47°C do not exert the control desired and greater than the 49°C can cause scalding in the fruits. In spite of the existence of research associated with this method and gamma radiation to control post-harvest diseases, only the hydrothermal treatment has been utilized in commercial scale. A treatment by double immersion (30 minutes at 42°C, followed by 20 minutes at 49°C) was used in the Hawaii with excellent results, when combined with regular application of fungicides in the field (Alvarez and Nishijima, 1987). Excessive heating or delay in the cooling of fruits can inhibit the normal process of maturation of fruits, provoking in many cases scalding, facilitating colonization by other fungi, and affecting the commercial quality of the fruits. In Hawaii (Lay -Yee *et al.*, 1998) the effect of disinfestation of fruits cv. Waimanalo Solo was studied under commercial conditions using forced air treatment of different temperatures followed by application of the fungicide prochloraz. Fruits treated with temperatures between 47.5°C and 48.5°C caused the least loss of firmness of the pulp. Temperatures greater than 49.5°C tended to cause a loss of the firmness of the pulp of the fruits.

This effect appears to be associated with a decrease in the enzyme polygalacturonase, responsible for the catalyzation of the hydrolysis of pectins, components of the cell wall (Chan *et al.*, 1981; Lay –Yee, *et al.*, 1998). Scalding of the skin also tends to increase with increase in temperature of treatment especially when lasting more than 60 minutes. In general, the thermal treatment did not alter the incidence of peduncular rots, but was associated with an increase of other rots, reflecting the reduction of the resistance of the tissues of the fruits to infection of secondary microorganisms. The immersion of the fruits in prochloraz, 0.25%, after the thermal treatment, reduced the incidence of rots without affecting the quality of the fruits (Lay -Yee *et al.*, 1998).

iii. Post-harvest chemical treatment: In fruits for exportation, principally by ship, after hydrothermal treatment, a treatment with fungicide is recommended, with the objective of increasing the effectiveness of control of the fungal diseases. The use of the two treatments combined (hydrothermal and chemical) is an alternative more advantageous in the control of anthracnose in post-harvest, respecting always the limits of tolerance for chemical residues.

The existence of latent infections in the fruits explains the post-harvest appearance of the disease even when protection of the fruits is provided, since the fungicides apparently protect the fruits from new infections, but do not eradicate the subcuticular hyphae that are quiescent (Alvarez and Nishijima, 1987).

The most effective fungicides utilized actually are prochloraz (50ml p.c./100 L of water), and thiabendazole (200g p.c./100 L of water). This fungicide was used in Hawaii,

applied in a concentration of 4-8 g/L with wax of carnaúba (Couey *et al.*, 1984). Concentrations greater than those recommended should not be used to avoid the occurrence of phytotoxicity on the surface of the fruits and conform to limits of tolerance for residues established by the importing countries (Liberato *et al.*, 1999). Prochloraz in the EC formulation can present phytotoxicity in the fruits when used in concentrations greater than 250 mg l^{-1} a.i.

The fungicide benomyl has been used extensively for control of anthracnose of papaya in post-harvest, but studies conducted in the Espírito Santo proved its low effectiveness owing to the occurrence of populations of *C. gloeosporioides* with resistance to this fungicide (Ventura and Balbino, 1995).

After the chemical treatment, the fruits can be immersed in solution of water with wax, in the proportion of 1/1, for approximately 4 seconds.
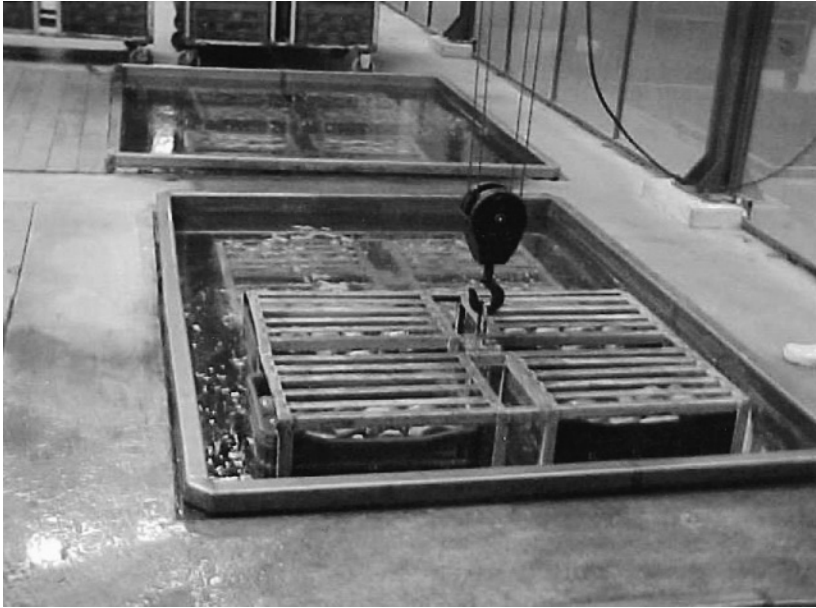
Figure 4 : Hot-water treatment of papaya fruits to control postharvest diseases

Recently the use of 1-methyl cyclopropene (1-MCP), a blocker of ethylene receptor sites, has demonstrated great effectiveness in slowing the maturation of fruits and reducing the incidence and severity of anthracnose during the storage.

iv. Genetic resistance: The commercial cultivars of papaya actually planted are susceptible to anthracnose. However, preliminary results of laboratory research demonstrated that the cv. Golden was more susceptible than other cultivars of the groups Solo and Formosa (Rodrigues *et al.*, 2001). Recent attempts to use genetic engineering to transfer resistance genes into papaya show promise.

2.2 Black spot

Black spot or leaf spot is the most common disease of papaya, occurring in both commercial and domestic orchards, as well as in isolated plants located in backyards and road margins. When it occurs with great severity in the leaves the disease can affect the development of the plants, especially those that are younger. The economic importance of the disease however is most noted when it occurs in the fruits, on which it causes lesions, reducing their value commercially, as verified in some orchards in the north of Espírito Santo and south of Bahia. It is the principal foliar disease of the culture, particularly when it occurs in the initial phase of the establishment of the orchard.

2.2.1 Etiology

The etiological agent of black spot is the fungus *Asperisporium caricae* (Speg.) Maubl., which presents subepidermal mycelium and well developed stroma, erumpent and subepidermal, forming short conidiophores closely packed together and covering the surface of the stromata, unbranched, hyaline to olive brown, smooth, with several prominent conidial scars at the apex. The conidia are solitary, dry, ellipsoidal, pyriform or clavate, almost always bicellular (uni-septate), hyaline to pale brown, distinctly verrucose, and measuring 14-26mm x 7-10mm (Holliday, 1980).

2.2.2 Symptoms

The disease occurs on the leaves and on the fruits. On upper surface of leaves, characteristic symptoms consist of round, light-brown (tan) necrotic spots, encircled by a yellow halo (Fig.5). On the lower surface of the leaves, in the areas corresponding to the spots, the powdery growth of the fungus of gray to black color can be observed (Fig. 6). In some cases, over these, a pale mycelium produced by a fungal hyperparasite of the pathogen may be observed. When it occurs, coalescence of the lesions is a common cause of leaf senescence and defoliation of the plants. Abundant spotting causes defoliation and over 50% leaf fall can occur. Young leaves generally do not present symptoms.

In the fruits, the presence of circular areas of watery aspect are observed initially, that, with evolution of the disease become brown in color, prominent, with pale points, and that may attain 5mm of diameter (Fig. 7). These lesions generally are epidermal and do not reach the pulp of the fruit, causing only a hardening of the skin of the part affected.

2.2.3 Epidemiology

Black spot occurs with greatest intensity under conditions of temperatures between 23 to 27ºC, with strong winds and high rainfall or overhead irrigation. The incidence is seasonal, and most infection occurs in late winter and spring. These conditions favor development of the lesions and dispersion of spores from older leaves, considered the principal sources of inoculum, and where the disease occurs initially, being dissemi-

nated subsequently to the younger leaves. The penetration of the fungus is stomatal and macroscopic symptoms are visible between 8-10 days after inoculation (Holliday, 1980). Fruits can be infected when still green, and the lesions resulting from the eruption of the stromas will emerge completely at the beginning of maturation, liberating new spores when the fruit is totally mature.

An epidemiological curve of the disease in different cultivars showed it to be least in *C. cauliflora* and greatest in the cv. Sunrise Solo line 72/12, while a cultivar of the group Formosa and the cvs. Baixinho of Santa Amália, Sunrise Solo, and Santa Bárbara had an intermediate comportment.

2.2.4 Control

Figure 5: Symptoms of black spot caused by *Asperisporium caricae* on the upper surface of papaya leaves, showing the characteristic yellow halo.

2.2.4.1 Cultural practices

Aimed at the reduction of the source of inoculum, older leaves with high severity of the disease should be eliminated. This practice can be carried out at the same time as the operation of sprout removal, which is begun generally 30 days after transplanting the seedlings.

2.2.4.2 Chemical control

Applications of fungicides to control black spot should be begun as soon as the first

symptoms of the disease are observed, when the plants still are in the initial phase of growth. In monitoring of the plants, older leaves on which the lesions initially occur should be observed. The first five months after planting, when the plants are most susceptible, is the period most critical for control of the disease in the leaves, which is influenced by climatic conditions.

The fungicides normally recommended for anthracnose also have presented effective control of black spot. However, during periods of climatic conditions highly favorable for the disease, principally periods of prolonged rain, these fungicides have not been effective for control of the disease.

In these cases, fungicides in the triazole and strobilurin groups should be utilized based on their greater effectiveness observed in experimental evaluations. The interval of application of these fungicides varies with climatic conditions and manage-
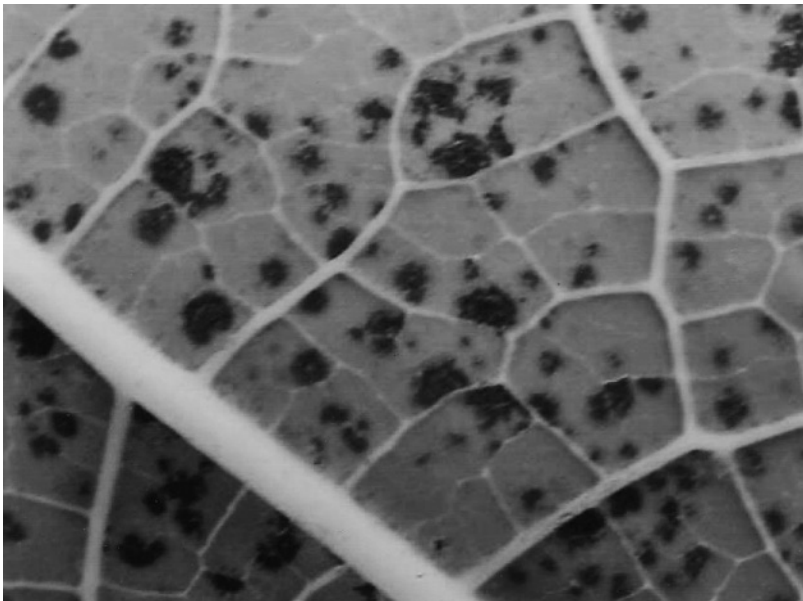
Figure 6: Black spot lesions on the lower leaf surface with the presence of fruiting structures of the fungus *Asperisporium caricae*

ment of the orchard. In drier periods of the year the interval between applications (sprays) may be as much as 30 days. If effective control of the disease in the leaves is not achieved its control in the fruiting phase becomes difficult, with losses in the commercial quality of the fruits occurring.

2.3 Powdery mildew

Papaya powdery mildew is a disease of general occurrence, especially in very shady nurseries and in colder months of the year, as noted in the regions north of Espírito

Santo and south of Bahia, Brazil, between the months of June and September. When it occurs with high severity, the disease can cause damage in the leaves, affecting photosynthesis and consequently the commercial quality of the fruits as well as slowing the development of the plants. In plants in the nursery a total loss of leaves may occur resulting in death of the plants.

2.3.1 Etiology
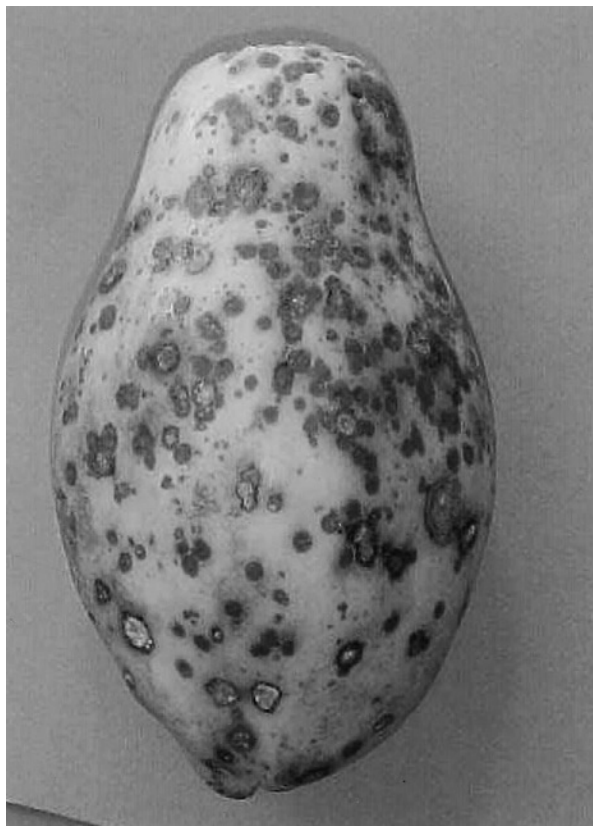
Figure 7: Black spot lesions on papaya fruits caused by *Asperisporium caricae*

Three species of *Oidium* have been reported and described as causing papaya powdery mildew: *O. caricae* (conidia elliptical, 24-30μm x 17-19 μm), *O. indicum* Kamat (conidia barrel shaped, 31-47μm x 12-33μm) and *O. caricae-papayae* Yen (conidia 36-44.4μm x 15.6-21.6μm). Another powdery mildew *Ovulariopsis papayae* van der Bijl. (teleomorph: *Phyllactinia* sp.), with conidia 14-23μm x 60-90μm, has also been described.

O. caricae known in Brazil since 1898 and reported in other production areas of papaya in the world, presents mycelium hyaline, septate, with haustoria developing in

the interior of the epidermal cells of the host. The conidia are hyaline and granular, barrel shaped, and formed in chains of three to five or more spores. *O. papayae*, observed in the north of Espírito Santo, Brazil, presents erect, multiseptate conidiophores, originating from cylindrical hyphae (Liberato *et al.*, 1995 and 1996). The conidia are large, subclavate and isolated in the apex of the conidiophores.

2.3.2 Symptoms

Symptoms caused by *O. caricae* begin in the leaves, with a light yellow-green discoloration of irregular outline and of dark-green margins. White powder-like growth of the causal fungus can develop on leaf undersides. This is usually not a severe problem but some leaf drop can occur (Simone, 2002). With evolution of the disease, the discolored patches become covered with a powdery white mass made up of structures of the fungus (mycelium and conidia), present on the upper and lower leaf surfaces. The leaves may become yellowish (chlorotic), present a generalized drying and subsequently fall. With the exception of the younger leaves, all of the leaves may be affected by the fungus, the older leaves being more susceptible. Beyond the leaves, the pathogen can in certain conditions also be observed in the stem, flowers, pedicels and fruits.

The fungus *O. papayae* causes symptoms similar to those incited by *O. caricae*. On the upper surface of the leaf chlorotic areas that evolve to yellowish spots delimited by principle veins, round (or with irregular margins), of approximately 0.5 cm diameter and that coalesce reaching great foliar area, may be observed. On the lower surface of the leaf a powdery mass of pale color, corresponding to the yellowish spots on the upper surface, is observed (Fig. 8). In contrast to *O. caricae* these signs of the fungus occur normally on the lower surface of the leaf, and are rarely observed on the upper surface. Sometimes the white patches appear on the fruits. In the nurseries seedling plants are especially susceptible to attack and may be seriously affected. Young infected leaves of the seedlings dry up prematurely and drop down.

2.3.3 Epidemiology

The disease occurs principally in the colder and drier months of the year, which for the northern region of Espírito Santo, Brazil, corresponds to the months of May to September, when the average temperature varies from 21- 24ºC and the relative humidity of the air is lower than 70% and the weather is cloudy (Anonymous, 2002).

For germination of the conidia, a brief period of high relative humidity is required, but not the presence of water in the free state. The great masses of spores produced on infected leaves are readily spread by wind currents to healthy plants. Year-round production of papaya permits uninterrupted reproduction of the fungus and continuous presence of the disease in an active state (Pernezny and Litz, 1993). The disease is more serious in orchards with systems of drip (or micro) irrigation, since sprinkle irrigation is unfavorable for the fungus.

2.3.4 Control

For both genera of fungi that cause powdery mildew, control is achieved by the applica-

tion of specific fungicides. The sprays should be applied when conditions are favorable for the occurrence of the disease, principally if these conditions occur for prolonged periods. The product most utilized is wettable sulfur or sulfur dust, applied at biweekly intervals after the start of the appearance of the first symptoms. When climatic conditions are highly favorable, the interval may be less, generally weekly. Sprays with wettable sulfur have not been effective when symptoms in the plants are severe, or that is, when the area of the leaf has signs of the fungus of more than 25%. This fact reinforces the importance of carrying out constant monitoring of the disease in the orchard. Sulfur application should be avoided in the hottest periods of the day (temperature greater than 24º C) to prevent phytotoxicity.Fungicides of the benzimidazole group, like thiophenate-methyl, benomyl and carbendazim, also have been utilized by producers to control powdery mildew in the northern region of Espírito Santo; however,
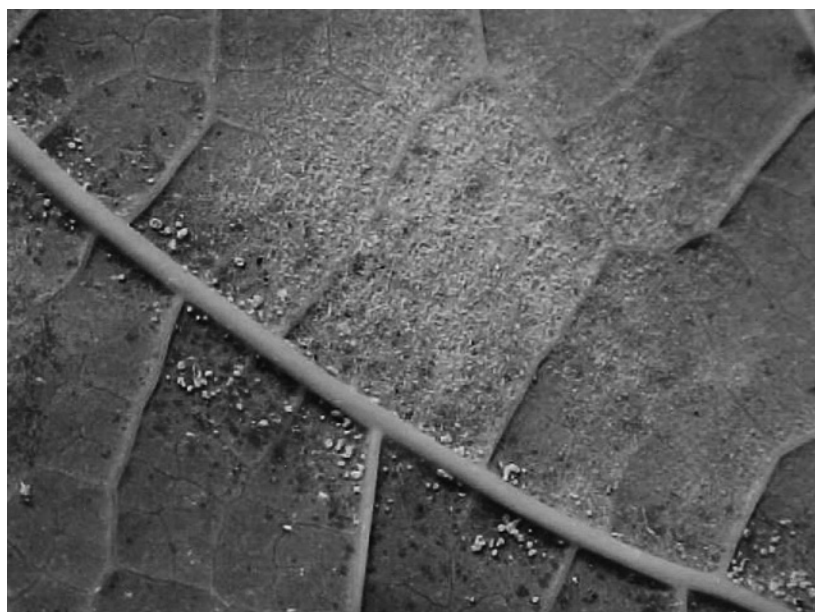
Figure 8: Symptoms of powdery mildew caused by *Ovulariopsis papayae* on the lower surface of the a papaya leaf

they have not been effective.

In the nursery, the fungicide triflumizole (15g c.p./100 l), an imidazol, was effective for control of papaya powdery mildew. In the field, triflumizole was less effective, but not significantly different than sulfur (Tatagiba *et al.*, 1998b).

2.4 *Ascochyta* leaf spot, dry rot and stem end rot of fruits

Ascochyta leaf spot, or leaf blight is a disease that can cause losses of approximately

30% in yield, as observed in some orchards of the northern region of Espírito Santo, principally during periods of high frequency of rain and when outbreaks of mites (*Tetranychus urticae*; two-spotted spider mite) which cause lesions in the leaves that serve as ports of entry for the fungus occur.

2.4.1 Etiology

The disease is caused by the fungus *Phoma caricae-papayae* (Tarr.) Punithalingam (teliom.= *Mycosphaerella* sp. (sin.= *Ascochyta caricae-papayae*), that can infect both the leaves and the fruits.

The etiological agent was for a long time reported as being of the genus *Ascochyta*, but taxonomic studies of the pathogen led to the transfer to the genus *Phoma*, by virtue of the predominance of unicellular conidia and presence of conidiogenous cells phialidics. In *Ascochyta* the conidia are septate (bicellular), and show (exhibit) annellidic ontogeny. The difficulties in the classification of fungi like *Phoma* were discussed by Holliday (1980) and Punithalingam (1980).

In the lesions, both on the leaves and fruits (dry rot), are formed smooth pycnidia and perithecia (100-180µm x 70-200µm), dark brown to black and flask shaped to oval, characteristic of the genera *Mycosphaerella*, this being the teleomorph (Alvarez and Nishijima, 1987).

The genera *Phomopsis* also can occur in papaya with similar symptoms on the fruits, but this fungus forms pycnidia in which two types of conidia are produced, the a conidia (6.4-8.0 x 2.7-3.1µm), hyaline, fusiform and unicellular, and the filiform b conidia (13.7-20.0 x 1.0-1.8µm), while *P. caricae-papayae* does not form the b conidia.

2.4.2 Symptoms

The fungus preferentially infects younger leaves and fruits but also was reported to cause spots on flowers and young fruits.

Penetration is highly favored by the presence of injuries on the borders of leaves, where the round lesions that present a dark-brown coloration and pycnidia arranged in concentric rings are generally observed (Fig. 9). As they evolve, the lesions may attain 4cm diameter, develop a blighted appearance and become broken (or wrinkled), principally in the older leaves. In the more advanced stage, when the conditions are highly favorable, the disease can cause a severe blight of the leaves in the apex of the stem leaving the plants unproductive and in some cases provoking the death of the apex. Under humid conditions, it is common to see light-colored tendrils of pycnidiospores oozing from pycnidia (Nishijima, 1998b).

Infections on flowers and young fruits are initially brown, becoming dark and sunken. Initial infection of stem-end rot is through the broken peduncle, where early symptoms are a slight browning. With growth, the infected area is delineated by a narrow, light brown, translucent margin and as the infected tissue ages, it becomes wrinkled, dry, and black.

In the fruits, the lesions generally occur in the phase of maturation, near the peduncle, turning the tissue black, wrinkled and dry, and frequently covered by a

spongy mass of gray color that tends to increase in size, with the formation of pycnidia separate and embedded in the diseased tissue as the lesions age. The lesions evolve rapidly from the base of the peduncle to the pericarp and mesocarp of the fruits, principally after the beginning of maturation, reaching and affecting the seeds (Fig. 10).

The stem-end rot caused by *Mycosphaerella* sp. begins with a translucent zone around the peduncle that becomes brown. With the evolution of the disease the lesion becomes dark (black), with dry aspect, while maintaining translucent margins. In the internal tissues of the fruit the formation of white mycelium that reaches to and colonizes the seed cavity can be observed.

After *C. gloeosporioides*, the fungus *P. caricae-papayae* is considered the most important post-harvest pathogen of papaya.
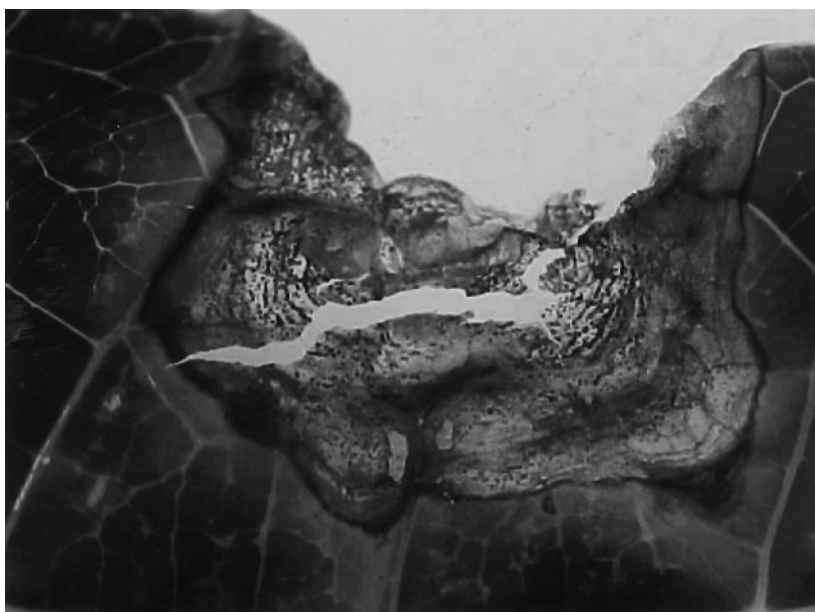
Figure 9: Lesion of *Phoma caricae-papayae* on a papaya leaf, showing the concentric rings of pycnidia.

2.4.3 Epidemiology

Climatic conditions that favor the incidence of ascochyta spot are temperatures between 21 and 26ºC and high relative humidity (>90%). In the leaves the disease is favored by the occurrence of strong winds. In fruits, handling in harvest and post-harvest causes injuries that enable the penetration of the pathogen. The fungus colonizes senescing leaves and petioles, and produces abundant fruiting structures (pycnidia) in dead leaves that serve as a source of primary inoculum in the field.

In older leaves, the presence of the burn of the leaves associated with the

damage caused by tetranychid mites, principally the two-spotted spider mite (*Tetranychus urticae* Koch), owing to the wounds that they cause to the leaves, generally can be observed.

Infection by *P. caricae-papayae* in mature fruits (more susceptible), requires a superficial wound to begin infection and a minimum of 6 hours of continual wetness (Sanchez *et al.*, 1991). In green fruits, the optimal temperature for infection is 21ºC, in the presence of water in the free state; and in the mature, the range of 18 to 24ºC enables the greatest development of lesions.

The epidemiology of the disease needs to be better studied to determine the importance of the teleomorph, and obtain data of the quantity of pseudothecia and perithecia in samples collected from orchards, both in lesions of the leaves, and, principally, in the fruit. In isolates of the fungus cultivated *in vitro* (Costa and Ventura,
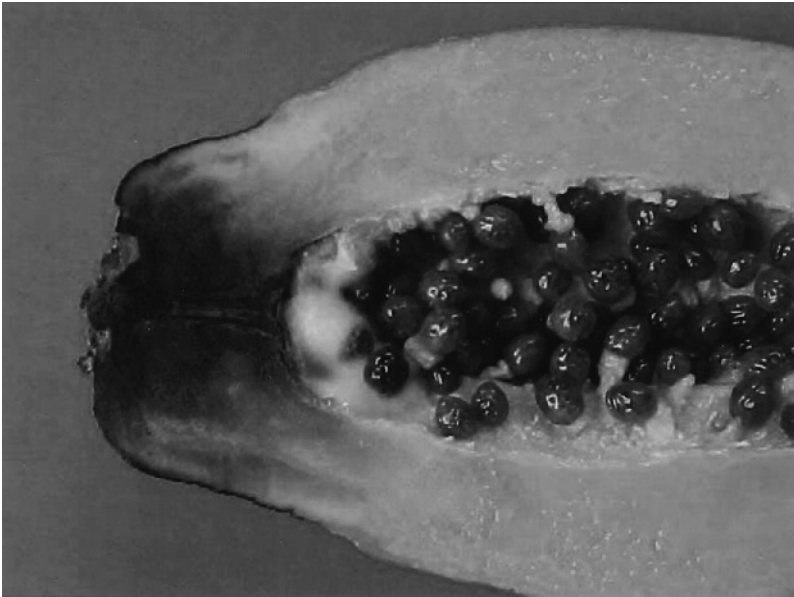
Figure 10: Stem end-rot of papaya fruit (cv. of group Solo) caused by *Phoma caricae-papayae*. Longitudinal cross section of papaya fruit showing the colonized tissues.

INCAPER, unpublished), the formation of perithecia has been observed, confirming the homothalic characteristic of the fungus, conforming to that described by Honda and Aragaki (1983).

2.4.4 Control

Wounds created during harvest and postharvest handling are quickly colonized during storage if conditions for infection exist. Also because fruit are susceptible to chilling injury at temperatures below 7°C, normal storage and shipping temperatures are suitable for spore germination and infection (Nishijima, 1998b). The chemical control mea-

sures and principally, management, recommended to control anthracnose and black spot have presented effectiveness for the control this disease. Fungicide sprays decrease inoculum levels in the field. The fungicide prochloraz showed effective postharvest control of the disease in experimental studies, when applied by immersion of the fruits.

In Hawaii, Nishijima (1998b) reported that hot water treatment at 48°C for 20 min. also reduced stem-end rot, and the double hot water dip and vapor heat quarantine treatments also control the disease on fruits.

Based on epidemiological characteristics of the disease, the use of sprinkle irrigation favors the sporulation of the fungus and its dissemination in the orchard. The removal of infected leaves, petioles, and fruits is recommended for management of the disease. Equally, the prevention of injuries to the fruits during harvest is recommended. In an evaluation of genetic resistance, the germplasm of *C. papaya* and *C. cauliflora* was susceptible to the fungus. The species *C. gaudotiana* (Tr. Et Pl.) was resistant to *P. caricae-papayae* but presented susceptibility to the fungus *C. gloeosporioides* (Sanchez *et al.*, 1991).

2.5 Greasy or Brown spot

The disease greasy spot or brown spot, also known as *Corynespora* spot, can occur on the stem, fruit, petiole, and leaf of papaya. It is caused by a widespread fungus that also causes the disease often referred to as "target spot" in various hosts. The greatest incidence of the disease is observed in plants more than four months old and in the colder months of the year, when a great number of lesions in the leaves are observed, especially the older leaves, that often yellow and fall. Heavy infection results in premature defoliation with losses in yield and possibly fruit quality.

Lesions on fruits and stems are much less frequent than on the leaves, but in recent years the incidence in fruits has increased in all the cultivars in Espírito Santo, Brazil, lowering sales of the fruits.

2.5.1 Etiology

The disease is caused by the fungus *Corynespora cassiicola* (Berk. & Curt.) Wei., which produces conidiophores erect, simple or occasionally branched, straight or slightly curved, pale to brown, smooth, septate, monotretic, with up to nine successive cylindrical proliferations (110-850mm x 4-11mm). The conidia are solitary or catenate, variable in shape, obclavate to cylindrical, straight or curved, subhyaline to rather pale olive brown having generally 4-20 pseudosepta (40-220mm x 9-22mm) with a dark "hilum" in the form of a kidney (Holliday, 1980). In culture, the fungus produces mycelium gray to olive-brown with growth superficial and into the media, with an absence of stromatas. *C. cassiicola* from infected papaya leaves in Espírito Santo, was cultured on potato dextrose agar (PDA). On PDA the isolates exhibited variability in colony color and growth rates and the optimum temperature for growth was 28°C.

The pathogen rapidly colonized dead papaya leaves and produced ca. 9,000 conidia per g (dry weight) of leaf tissue on the plant. An average of 44,500 conidia per

g of leaf tissue was produced on leaf debris from the soil beneath papaya plants (Kingsland, 1985). The fungus is reported as a pathogen in more of 70 species of plants. Isolates of different hosts are morphologically similar. However, they present significant polymorphism when the DNA is analyzed by RAPD-PCR, enabling separation into a genetic group isolates of papaya in relation to those of other hosts, with this group also not pathogenic to tomato and eggplant (Sliva *et al.*, 1998).

2.5.2 Symptoms

Older leaves are most likely to be affected. The symptoms differ greatly depending on climatic conditions and the part of the plant infected. On leaves, the infection occurs principally on the lower surface (Oluma and Amuta, 1999) and the lesions are generally small, of 2 to 3mm diameter, circular, white and encircled by a yellow halo (Fig. 11). In conditions of high humidity, on older leaves, larger lesions reaching 6mm diameter of irregular shape and of light-brown color may arise (Fig. 12). *C. cassiicola* forms conidia on both upper and lower leaf surfaces, but masses of spores are most evident on the latter. Examination of lower leaf surfaces with a hand lens for a dark growth of the fungus hyphae and spores is important for diagnosis of this disease (Pernezny and Litz, 1993).

On the petiole and stem, the lesions are red-brown with the center dark, elliptical, measuring 3 to 5mm in length, with width constant of about 2mm. In the fruits, even green, very small circular spots (1mm) appear, that may evolve rapidly (Fig. 13). The lesions are depressed and with, a dark center where the structures of the fungus are observed. The lesions may coalesce reaching a great area of irregular form on the fruit.

2.5.3 Epidemiology

Temperatures between 20 to 24ºC and high relative humidity favor infection of the pathogen, which is disseminated by the wind. *In vitro* the optimum temperature for mycelial growth is 28ºC, with a drastic reduction of radial growth occurring at temperatures greater than 32ºC, and no growth of the fungus occurring at 8ºC and 40ºC (Melendez and Pinero, 1970).

Plants with water or nutritional stress become more predisposed to the occurrence of the disease, principally the stem of the plants. The use of sprinkle irrigation generally favors the severity of the disease by forming a microclimate highly favorable for pathogen infection.

Papaya cultivars vary in their responses to infection by *C. cassiicola*. In Brazil cv. Tainung 01 develops very high levels of greasy spot during the months of January and February, when the temperature is greater than 25ºC and humidity is high (Andrade *et al.*, 2002).

The fungus *C. cassiicola* has a wide geographic distribution and has been reported in different plants including tomato, soybean, bean, cassava, rubber and cucurbits. *Commelina benghalensis* a weed of wide geographic distribution and frequently present in papaya orchards, was reported as a host of *C. cassiicola* in Brazil, and may constitute an important source of inoculum, principally when climatic condi-

tions become favorable (Silva and Souza, 1999).

2.5.4 Control

To reduce the quantity of inoculum in the orchard, the removal of senescent and highly infected leaves is recommended. No specific fungicides registrations exist for the disease in Brazil. However, the fungicides mancozeb and chlorothalonil, employed to control anthracnose, reduce inoculum levels, and provide satisfactory control.

2.6 Foot rot and *Phytophthora* rot of fruits

Foot rot, also known as root rot, stem rot, *Phytophthora* blight or *Phytophthora* rot, is

Figure 11: Lesions of greasy leaf spot (*Corynespora cassiicola*) on papaya leaves, showing the characteristic white center and with yellow halo of the lesions.

reported in all of the regions of cultivation of papaya, occurring principally in rainy periods and in heavy, excessively wet and poorly drained soils. The damage can be observed in the roots, collar, fruits and apical region of the plant. Besides causing the death of plants when it causes severe rot in the collar, *Phytophthora* rot can also cause great losses when occurring in the fruits during periods of intense rain.

2.6.1 Etiology

The etiological agent of the disease is the fungus *Phytophthora palmivora* (Butler)

Butler, which has a wide host range. The pathogen produces abundant, terminal, prominently papillate sporangia (50-33μm), in well-developed sympodia. Sporangia when mature fall away from the sporangiophores, within which are formed biflagellate zoospores (10-40). Chlamydospores produced in infected papaya tissues are thick walled (Ko, 1998).

2.6.2 Symptoms

The disease appears with greatest frequency in the collar region of the plants, where watery spots that subsequently coalesce and which may have a gummy exudation in the lesionous area, may be observed. Water soaked lesions appear on the bark just above the ground level. The parenchyma tissues are destroyed, but the vascular tis-

Figure 12 : Large irregular leaf spot symptoms caused by *C. cassiicola* on papaya leaves.

sues remain intact. In poorly drained soils, the pathogen attacks lateral roots and later extends to the taproot. The whole root system may becomes brown, soft, and shredded, and trees become stunted. The root decay has a foul odor. Leaves turn yellow, wilt and hang limply around the stem, living only a few small leaves at the apex of the plant, and the tree dies. As a result of infection in the collar, symptoms of yellowing of leaves, premature fall of fruits, wilt of the top, lodging, and death of the plant may be observed (Fig. 14). In advanced stages of the disease the plants may be knocked down by strong winds. If conditions are not favorable, the lesion may not develop, and the plant may recuperate, but production is compromised. Normally associated with the rot that occurs in the collar, symptoms of rot in the roots of the papaya can be observed, occurring

with greatest frequency up to three months after emergence of the plants. Symptoms similar to those caused by the root rot, are observed in rot of the collar. With age, plants become more resistant to infection of the roots.

Lesions may also occur on the apical region of the plants and on the column of fruits. Initially, small, discolored areas arise on the stem, principally where fruits are located, which frequently fall prematurely. These areas increase in size, encircling the stem, killing the upper part of the plant, and in periods of high rainfall, causing the death of the plant. In the fruits, principally mature and near mature, a rot of the tissues which become covered by white, cottony mycelium may be observed, the disease receiving the name of "Papai Noel" (Santa Claus) in the Espírito Santo, Brazil (Fig. 15).

2.6.3 Epidemiology

Figure 13: Symptoms of greasy spot on papaya fruits

Disease is more severe on young plants. Cool, wet environmental conditions with high soil moisture favor disease development. *P. palmivora* may be introduced into the orchard from infected seedlings produced in nurseries where phytosanitary care has not been observed, by irrigation water, and sporangia disseminated by wind. High humidity, and temperature from 28-32°C, poorly drained soils, and injuries constitute factors important for initiating the disease. Sporangia and zoospores can survival in the soil for short periods while the chlamydospores, which are the principle structures of resistance, can survive for long periods, in the soil and in fallen fruits. When in water, these germinate to produce sporangia and liberate zoospores, serving as the principal source of inoculum for the infection of plant roots in subsequent plantings.

Rain and the wind are important for dissemination. Drops of rain are necessary for the liberation of the sporangia from the infected plant material or soil. The mobility of the zoospores and their attraction to roots of the papaya plant increase with free water, which also augments the severity of the disease. The pathogen produces a great number of sporangia on the surface of diseased tissues, principally when the temperature is near 25ºC, and this is an important source of inoculum in the development of epidemics. Generally, production of sporangia does not occur when the temperature is

Figure 14: Orchard of papaya cv. Sunrise Solo with high incidence of plants infected by *Phytophthora*. Note, wilt of the canopy of the plants and abscission of the fruits.

less than 15ºC or more than 35ºC. The microbial population of the soil, principally protozoa, significantly reduces the rate of growth of the pathogen (Ramirez *et al.*, 1998).

In experimental studies to evaluate resistance under controlled conditions, the least development of lesions in fruits occurred on the cv. Sunrise Solo, when compared with the genotypes CMF002, CMF007, CMF083 and CMF033, the last two being the most susceptible (Lima *et al.*, 2000).

2.6.4 Control

In the control of foot rot, principally the measures of escape, exclusion, and eradication of the disease are recommended: i. avoid planting in excessively clay soils, with poor drainage and in regions with high rainfall. ii. planting on mounds reduces the incidence of the disease; iii. cultivate papaya in soil where the pathogen has not been reported; iv. utilize sterilized soil for seedlings; v. utilize healthy seeds treated with fungicides; vi. avoid injuries to the plants during cultural treatments; vii. remove diseased plants and fruits from the orchard.

The use of organic material and the enrichment of the soil with microorganisms, particularly in nurseries, is an important practice to prevent and reduce the reproduction of the fungus.

Figure 15: Symptoms of *Phytophthora palmivora* on fruit cv. Sunrise Solo, with growth of white mycelium.

When conditions are highly favorable for the disease, apply preventative sprays directed to the region of the collar of the plants, as well as to the column of fruits, with fungicides based on chlorothalonil or metalaxyl; and proceed with surgery to the stem, removing the tissue affected and treating the local with cupric paste (1 kg of copper sulfate + 2 kg of lime + 10 liters of water). This measure is only recommended for small and superficial lesions. In case of attack only on the apical region that affects the fruits, sprays with specific fungicides should be realized immediately to reduce the loss of fruits. Fruit rot can be controlled with fungicides (Simone, 2002).

Root rot of seedlings caused by *P. palmivora* can be controlled in replanted

fields with the virgin soil technique, in which soil from areas where papaya has never been grown is placed in planting holes that are about 30 cm in diameter and 10 cm deep, The roots of plants are protected by the virgin soil during the susceptible stage (Ko, 1987). Cultural practices are also important in the management of root rot. The disease incidence on mature plants during rainy periods can be greatly reduced by improving drainage in orchards.

2.7 Damping off

"Damping off" occurs generally in sporadic form in plants in seedbeds or sacks in nurseries, but may also occur in seedlings recently transplanted in the field. In the nurseries, papaya seedling roots are very susceptible during the first three months after seedling emergence. Infection of roots in this period resulted in yellowing of the leaves, defoliation, and death.

2.7.1 Etiology

Various soil fungi are responsible for the appearance of the disease, most prominently the genera *Pythium, Phytophthora, Sclerotium, Rhizoctonia* and *Fusarium*, which may occur individually or in association. Two species of *Pythium* were recorded on papaya: *P. aphanidermatum* (Edson) Fitzp. with a rapid growth on agar media at 34-36°C, and *P. ultimum* Trow with optimal growth in vitro at 28°C or less (Holliday, 1980). Knowledge of the etiological agent associated with the disease is important in the choice of effective fungicide or the tactic of appropriate control.

2.7.2 Symptoms

Symptoms of the disease are noticed initially in the form of a watery spot in the region in the collar of the plants that increases in size provoking the destruction of the tissues and, in consequence, lodging and death of the seedlings occurs. In the south of Bahia, Brazil, the fungus *Pythium* sp. with white mycelium, well developed and wooly, infected plants between 4 and 36 months of age. Symptoms were underdevelopment, yellowing of the leaves and subsequently, abscission of the older leaves with newer leaves remaining on the extremities of the plants and mummification of the fruits also occurring. In these plants, the root system presented a death of the lateral roots, with a zone of reddish coloration occurring in the region of transition between the healthy and diseased parts (Ram *et al.*, 1983).

2.7.3 Epidemiology

The incidence of damping off is favored by clayey, poorly drained soils with inadequate aeration, and by seeding densely and deeply. High temperatures and rainy periods contribute to an increase in the severity of the disease. The use of mulch also can significantly favor the incidence of the disease in young plants, independent of the cultivar (Elder *et al.*, 2000).

2.7.4 Control

Various measures should be adopted to control damping off, beginning with the choice of the local of establishment of the nursery, which should be in a well ventilated location, free of flooding, with good exposure to the sun, away from roads and fields of papaya, and with water for irrigation, following technical recommendations for construction of the nursery (Marin and Gomes, 2000). Plastic sacks (transparent or dark) preferentially should have the dimensions of 9 to 15 cm width by 12 to 18 cm height, with a minimum thickness of 0.06 cm and with holes in the lower part for drainage of water (Marin and Gomes, 2000). Sacks with height less than 12 cm are not recommended since that can cause deformation of the roots in the bottom of the sack, subsequently compromising the development of the plants.

The preparation of the substrate to fill the sacks is of fundamental importance. It should be composed of two to three parts of surface soil (humus) sieved and one part well-composted stable manure, also sieved (Marin and Gomes, 2000). The origin of the humus should be of sandy-clay and organic soils, avoiding clayey soils that easily become waterlogged during irrigation, prejudicing the development of the plants and predisposing them to the action of the pathogens responsible for damping off. It is important to make a chemical analysis of the substrate and enrich the mixture with the addition of fertilizer if needed, generally simple superphosphate and potassium chlorate. The soil of seedbeds or for filling of sacks should be sterilized by fumigation or heat treatment, and the seeds should be treated with fungicides. Irrigation should be moderate with water free of contamination. Temperature has a very important influence on the time required for germination of the seeds, being generally 9 to 18 days with a temperature average of 25.4ºC and of 12 to 21 days at 23.3ºC. In conditions of the north of Espírito Santo germination occurred between 7 and 12 days during the months of October to March and between 12 and 17 days during the colder months from April to September (Marin and Gomes, 2000). Seeds that germinate 20 days after seeding present low vigor and development of the plants, and are more susceptible to the diseases.

If mulch is used it should be kept at least 30 cm away from the stem of the plants (Elder *et al.*, 2000). Organic fertilizer should be well composted and its application should precede the addition of mineral fertilizer. Stable manure presents the risk of being contaminated with herbicides, like 2,4-D, which is highly phytotoxic to the papaya plant (Marin and Gomes, 2000). When the first symptoms of damping off appear, application of a specific fungicide to the plants is recommended.

2.8 Other fungal diseases and rots in postharvest

2.8.1 *Lasiodiplodia* Fruit Rot or Stem End-Rot

Besides anthracnose, other postharvest rots may cause great damage to fruits of papaya. Principal of them is lasiodiplodia fruit rot, stem end-rot or peduncular rot, which arises after harvest on the region of the cut of the peduncle, affecting the basal part of the fruit, generally at the beginning of maturation. Stem-end rot was attributed principally to the fungus *Phoma caricae-papayae*, and subsequently has also been associated with other genera of fungi like *Lasiodiplodia* (Syn. = *Botryodiplodia*), *Phomopsis*

and occasionally *Fusarium*.

The main etiological agent of this disease is the fungus *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. (syn.: *Botryodiplodia theobromae* (Pat.), which presents rapid growth, and which generally rots and totally mummifies the fruit. Actually, the involvement of other fungi with the disease is also known, including the species *Colletotrichum gloeosporioides*, *Mycosphaerella* sp. (anam.: *P. caricae-papayae*), *Alternaria alternata*, *Stemphylium lycopersici* and more frequently, *Rhizopus stolonifer*. Depending on the fungus involved, variable symptoms develop at the stem end as the fruit ripen. The fungi responsible for peduncular rot can also incite lesions in other regions of the fruit, producing spots or rots with symptoms characteristics to each species.

Lesions caused by *Lasiodiplodia theobromae* are dark with a wide margin of watery tissue and a surface wrinkled owing to the eruption of the pycnidia. Pockets of growth of mycelium occur in the tissues of the infected parenchyma. In a longitudinal cut of the fruit, the vascular tissue is dark, differing from infections of *Mycosphaerella,* which are translucent and those of *Stemphylium,* which are reddish-brown.

With exception of *R. stolonifer*, all of the other fungi can begin the process of infection in the field, causing peduncular rot alone or in different combinations, after the harvest, and during the transport and storage of the fruits. For *L. theobromae* a temperature of 30ºC, and high relative humidity, are the conditions favorable for infection by the pathogen, which colonizes the peduncular region of the fruits, developing abundant superficial mycelium of gray color with intense sporulation. The pycnidia are ostiolate, globose to sub-globose and the conidia are initially hyaline, unicellular, thick walled, and oval, with granular protoplasm. Mature conidia are two celled and light brown, generally measuring 21.7-30.1 x 11.7-18.3 µm with a central septum that turns dark.

In PDA culture media, the fungus has rapid growth, with the formation of aerial mycelium of cottony aspect, initially white and that subsequently becomes greenish-gray to black, with the formation of pycnidia solitary or grouped over dark stromas (Queiroz *et al.*, 1997). The fungus *L. theobromae* has an extensive range of hosts and in papaya, besides stem-end rot, it was also reported as the etiological agent of a rot of the stem of the papaya 'Sunrise Solo' in the state of Alagoas, Brazil (Queiroz *et al.*, 1997). No specific control measures have been developed, but orchard sprays with protective fungicides should reduce inoculum levels and disease incidence. A hot-water postharvest dip (48°C for 20 min) also is effective in controlling this disease (Nishijima, 1998a).

2.8.2 *Alternaria* fruit spot

Alternaria fruit spot is characterized by production of black, circular to oval lesions covered by a mass of black conidia and/or by the white mycelium of the pathogen. The causal agent of this disease is the fungus *Alternaria alternata* (Fr.:Fr.) Keissler, which can cause significant damage, especially in dry environments and after refrigeration, the temperature optimal for development of the disease being 25ºC.

Conidiophores are brown and up to 50 mm x 3-6mm. The conidia are also brown, formed in long chains, measuring 20-60 mm x 9-18 mm, with surface smooth or verruculose,

and with a short beak. The fungus can be encountered colonizing senescent petioles, from which the spores can contaminate the fruits during the harvest, being in this way a principal source of inoculum. The lesions are generally restricted to the surface of the fruits and do not cause extensive rotting of the flesh.

Chemical treatments in the orchards and thermal in postharvest generally are sufficient to the control the disease. The disease rarely is observed in fruits that do not undergo refrigeration, but fruits that are kept in cold storage (*e.g.*, 10ºC for 14 days), may experience high disease incidence (Nishijima, 1998a).

2.8.3 *Fusarium* fruit rot

Fusarium fruit rot is associated with various species of *Fusarium*, (*F. verticillioides*

Figure 16: Lesion of *F. solani* on the surface of a papaya fruit. Note the mycelium that has formed on the surface of the lesion.

and *F. equiseti*), but the most common of them is *Fusarium solani* (Mart.) Appel & Wollenw. emend. Snyder & Hansen, that occurs sporadically in the fruits of papaya when these begin to mature, producing small, superficial and dry lesions, subsequently forming a mass of white and very compact mycelium.

F. solani is considered a weak pathogen in fruits of papaya, requiring a stressing factor or injury for its establishment. It is frequently a secondary invader, generally associated with the lesions of *Colletotrichum*, *Corynespora* and *Phoma*.

The lesions of *Fusarium* on the fruit surface (Fig. 16) are generally 1.5-2.0 cm in diameter, and depressed, with the formation of white mycelium occurring on the surface

of the lesion (Quimio,1976; Rodrigues *et al.*, 2001; Saxena and Sharma, 1981). The fungus is also known to cause a rot of young (3-5 cm long) papaya fruits especially during wet weather. The fungus enters the seed cavity through the blossom end where it quickly spreads within the fruit and causes the fruit to abort and fall from the tree. In PDA medium, colonies of *F. solani* are white to cream colored, usually with sparse aerial mycelium. Abundant macroconidia are produced in cream colored sporodochia, from long monophialides or branched conidiophores with a distinct notched basal cell. Microconidia are present, generally single-celled, oval to ellipsoidal formed in false-heads on monophialides. Chlamydospores also are formed in specific medium and host tissues, singly and in pairs (Ventura, 2000).

The disease is favored by temperatures near 25°C with rainy weather when conditions of high humidity prevail. In Mexico *F. solani* has been reported to cause a seedling collar rot. It is a weak pathogen requiring some kind of predisposing factor that stresses or injures the fruit before it becomes established (Quimio,1976). No specific control measures have been developed, but preventive fungicide field sprays reduce inoculum levels and are effective in controlling the incidence of the disease.

2.8.4 Wet Fruit Rot

Watery rot, caused by the fungus *Phomopsis carica-papayae* Petr. & Cif. is not frequent in papaya, but when occurs it causes severe losses. The fruit rot occurs most frequently as a stem-end rot, but any part of the fruit can be infected. Initially, a wrinkling of the tissue is observed, that later becomes translucent, of color light-yellow-green color. There is a yellowing of the infected area advancing rapidly to the interior of the fruit, reaching the seed cavity (Nishijima, 1998c). The pathogen requires wounds to infect the fruits, and the disease usually develops on ripened fruits. The part infected can be removed from the fruit easily. With the evolution of the infection, there is a formation of black pycnidia on the surface, beginning in the center of the lesion. In isolates of *Phomopsis* sp. from papaya, two types of conidia are produced in the pyncidia: α-conidia (6.4-8.0 μm x 2.7-3.1 μm), hyaline, fusiform and unicellular, non-septate; and β-conidia (13.7-20.0 μm x 1.0-1.8 μm). The conidial dimensions differ from those of *P. caricae-papayae*, which also does not form β-conidia on host (Nishijima, 1998c).

The fungus sporulates on dried petioles that remain attached to the tree, and during rainy periods, conidia are discharged and deposited on fruit surfaces (Nishijima,1998c).

Control of wet fruit rot like other papaya postharvest diseases, must begin in the field with management procedures (Ventura and Costa, 2002). Senescent and dead leaves should be removed from trees, because they become an inoculum source. Nishijima (1998c) recommends that the removal of leaves is best accomplished by periodically cutting the petioles of leaves that drop below horizontal about 30 cm from the stem and removing them about a week later after the abscission zone forms but before the petiole stub has dried. Fungicide field sprays reduce inoculum levels and are effective in controlling the disease.

2.8.5 *Stemphylium* fruit rot

Stemphylium rot, caused by the fungus *Stemphylium lycopersici* (Enjoji) W. Yamamoto (syn.: *S. floridanum* Hannon & G.F. Weber), normally colonizes and is restricted to tissues of the fruit where wounds are found. Lesions incited by the pathogen are initially small, circular, dark brown color, and subsequently evolve acquiring oval forms with the margins reddish to purple. In the center of the lesion white-gray mycelium and dark-green conidia are formed. Infection by *S. lycopersici* is favored by the presence of injuries in the fruits often times caused by thermal treatment or storage at low temperatures (Chau and Alvarez, 1983b).

The optimal temperature for mycelial growth is 26°C, and sporulation occurs at 10-14°C, *in vitro* under continuous fluorescent light, but 8-10 hr of darkness is required for conidia to form. No sporulation occurs above 30°C (Nishijima, 1998a). Wounding and prolonged cold storage should be avoided. Fungicide field sprays reduce inoculum levels and are effective in controlling the disease.

2.8.6 *Rhizopus* soft rot

Rhizopus rot is a common disease in post-harvest, observed during storage and transport of the fruits in more advanced stages of maturation, but rarely detected in the field. The disease, caused by *Rhizopus stolonifer* (Ehrenb. ex Fr.) Vuill. (syn.: *R. nigricans* Ehrenb.), can cause losses of more of 50% in some shipments.

The pathogen presents great saprophytic capability and only penetrates the fruit through injuries that occur during harvest and post-harvest handling. Colonization of the tissues occurs rapidly, causing a soft and watery rot that affects all of the fruit.

On breakage of the cuticle, the fruit is covered by a mass of prominent, gray mycelium, with black, macroscopic sporangia. Conditions favorable for development of the disease are temperature around 20 to 25ºC and relative humidity of 70 to 90%. *Rhizopus*, in contrast to other fungi that cause peduncular rots, disseminates rapidly between the fruits in boxes or even within containers, provoking rot of the fruits in a few days (Alvarez and Nishijima, 1987). The treatments suggested for rhizopus control are the same as those recommended for control of anthracnose, most prominent being sanitation of installations in the packinghouse (Alvarez and Nishijima, 1987).

2.8.7 Internal Rot or Blight

Internal rot of the fruits, also called blight, is a disease that has sporadic occurrence and is caused by fungi that colonize the seed cavity and form a mass of mycelium and spores.

The principle fungi associated with this disease are *Cladosporium* sp., *Penicillium* sp., *Fusarium* sp. and *Alternaria* sp. that penetrate to the seed cavity through the stylar canal in the fruit that remains open after flowering and development of the fruits (Alvarez and Nishijima, 1987; Liberato *et al.*, 1996; Ventura and Santos, 1981). The infected fruits generally present precocious and irregular maturation and are generally

discarded in the operations of selection/packaging in the packinghouse.

Considering that the origin of the entry of the fungi is the lack of closure of the stylar canal, which may be a disorder of genetic origin, collection of seeds of plants that present the problem is not recommended.

F. solani is also know to cause a rot of young papaya fruits (3-5cm long) especially during wet weather. The fungus enters the seed cavity through the blossom end, spreads within the fruit, and causes the fruit to abort and fall from the tree (Nishijima, 1998a). Sprays with fungicides in the orchards reduce the inoculum of the fungi, ensuring that the disease has a sporadic occurrence and is of little economic importance (Alvarez and Nishijima, 1987).

3. Diseases caused by viruses

Papaya viruses cause serious reductions in fruit production, even totally destroying affected orchards. Although more than ten different viruses have been reported in papaya worldwide (Table-2), only four are considered of most importance: Papaya ringspot virus, Papaya lethal yellowing virus, Papaya droopy necrosis virus, and the Meleira or sticky disease virus, which is currently being characterized.

3.1 Papaya Ringspot Virus or Mosaic

Papaya ringspot virus also known as papaya mosaic and papaya distortion ringspot, is

Table 2: Viruses of papaya, by family and genus, in main growing regions worldwide.

Family	Genus	Virus species
Bunyaviridae	*Tospovirus*	*Tomato spotted wilt virus*, TSWV
	Tenuivirus	*Papaya mild yellow leaf virus*, PMYLV
Geminiviridae	*Begomovirus*	*Papaya leaf curl virus*, PaLCuV
	Bigeminivirus	*Croton yellow vein mosaic virus*, CYVMV
Potyviridae	*Potyvirus*	*Papaya ringspot virus*, PRSV-p
	Potyvirus	*Papaya leaf distortion mosaic virus*, PLDMV
Rhabdoviridae	*Rhabdovirus*	*Papaya apical necrosis virus*, PANV
Tombusviridae[1]	*Carmovirus*[1]	*Papaya lethal yellowing virus*, PLYV
NE[2]	*Potexvirus*	*Papaya mosaic virus*, PapMV
NE[2]	NE[2]	*Papaya meleira virus* (PMeV)

[1] Molecular research indicates homology with the Family *Sobemoviridae* and genus *Sobemovirus* (Silva *et al.*, 2000). [2] Not established. Molecular characterization of virus genome is in development

one of the most important and destructive diseases of papaya, being one of the factors limiting development of its cultivation worldwide. In Brazil, the disease was recorded in 1935 (Bitancourt, 1935). Mosaic became the principle phytosanitary problem of papaya in the state of São Paulo, which was responsible for most of the papaya produced in Brazil in the late 70's and early 80's, but accounted for less than 1% of the total papaya production by the mid 90's, leading to migration of papaya cultivation to other states. The disease is widespread in the Central and South America, Asia, Australia and is found in the papaya-growing areas of the U.S.A., including Hawaii, Florida and Texas. Losses are very high when rouging of infected plants is not carried out.

3.1.1 Etiology

Papaya ringspot virus is caused by a virus of the family *Potyviridae*, Genus *Potyvirus*, whose flexuous rod-shaped particles are about 760-800nm long by 12nm diameter. The nucleic acid consists of a linear positive single stranded RNA with 10.326pb and the genomic organization proposed consists of the VPg-5' leader, 63K *NT*, 52K *HC-Pro*, 46K, 72K *CI*, 48K *NIa*, 59K *NIb*, 35K cp, 3'-ncr and poly (A) tract (Yeh *et al.*, 1992).

There are two distinct biotypes of papaya ringspot virus: Papaya Ringspot Virus – Papaya biotype (*Papaya ringspot virus* - PRSV-p), and Papaya Ringspot Virus – biotype of cucurbits or watermelon (*Papaya ringspot virus*- PRSV-w). The papaya biotype infects papaya, cucurbits and *Chenopodiaceae* family, while the watermelon biotype infects only cucurbits and does not infect papaya, being previously known as *Watermelon mosaic virus 1* (WMV-1).

PRSV biotypes P and W cannot be distinguished on the basis of divergence in their *cp* sequences. When the nucleotide sequence of geographically different PRSV isolates of P and W types were compared, *cp* sequences which diverged more than 12% at the amino acid level were not found (Souza Jr. and Gonsalves, 1999). Both viruses cause local lesions in *Chenopodium quinoa* and *C. amaranticolor*, plants used as indicators of the viruses. PRSV-p has been transmitted experimentally by sap inocula-tion, grafting, and by more than 25 aphids species.

3.1.2 Symptoms

Early symptoms appear first on young leaves in the crowns of plants. The leaves exhibit a yellowing and later present an aspect of mosaic, green areas mixed with areas of yellow shade of variable form and size and well defined outline, resulting in a diminution in the rate of growth of the plants, loss of vigor, and decreased fruit yield.

Mottling and intense deformations and blisters may occur characterized by elevated areas of normal green color in contrast with remaining yellow areas (Fig. 17). In plants infected by severe strains of the virus, the leaves are greatly distorted and may develop a fingerlike or shoestring appearance._In the region of the stem, irregular watersoaked spots with an oily appearance may be present, these symptoms being greatly characteristic of the disease. Streaks on the petioles are usually lighter colored and blend into normal color of the petiole. The canopy of the diseased plants becomes smaller with the progress of the disease, the leaves remaining being of reduced size and

often deformed.

The fruit surface may present indistinct small concentric rings, very distinct, with the center green (Fig. 18). The first ringspots are dark green to tan and about 1mm in diameter. In the later stages, several spots may join to form irregular shaped spots up to 8mm in diameter and the rings may become necrotic and pale. Fruit yields in infected plants are markedly lower and fruits that mature after the tree is infected may appear normal but are of much low quality and generally unmarketable.Care must be taken to avoid confusing the symptoms of mosaic with those caused by broad mite attack, which are similar. In the case of broad mite infestation, the leaves present thicker veins, are deformed and rugose and reduced to almost veins only, and, on the leaf lamella, the green "islands" typical of mosaic are not observed (Fig. 17). Tree yellowing may be caused by low nitrogen levels, which cause a general loss of color. Thus, some growers

Figure 17: Symptoms of PRSV-p infection on papaya leaves showing the mosaic and green "islands".

mistakenly believe that fertilizer will cure the yellow color occurring on infected trees. It is important to note that before a tree is tagged as being infected with the virus, specific symptoms must be first identified. The yellowing associated with the PRV is a veinal chlorosis, which is very different from the general yellowing associated with nitrogen deficiency. PRV-p symptoms are most severe on younger leaves as compared to N deficiency, which is most severe on older leaves.

3.1.3 Epidemiology

Papaya ringspot virus can be transmitted mechanically and by grafting. However, it is

thought that aphid transmission is the most important mechanism for disease spread in the field.

PRSV-p may be transmitted naturally from infected plants to healthy ones by more than 20 species of aphids that feed on the leaves, in a non-persistent form. In other words, the virus is acquired and transmitted by the vectors in seconds. In Brazil, at least six species have been proven to transmit this virus under experimental conditions: *Myzus persicae* (green peach aphid), *Aphis gossypii* (cotton aphid), *A. fabae* (black bean aphid), *A. coreopsidis*, *Aphis* sp. and *Toxoptera citricidus* (black citrus aphid).

Worldwide, twenty-three aphid species have been tested experimentally and are considered to be vectors of the virus (Mark Culik, INCAPER/CNPq, personal communication, 2002). Although aphids do not colonize the papaya plant, they are attracted by
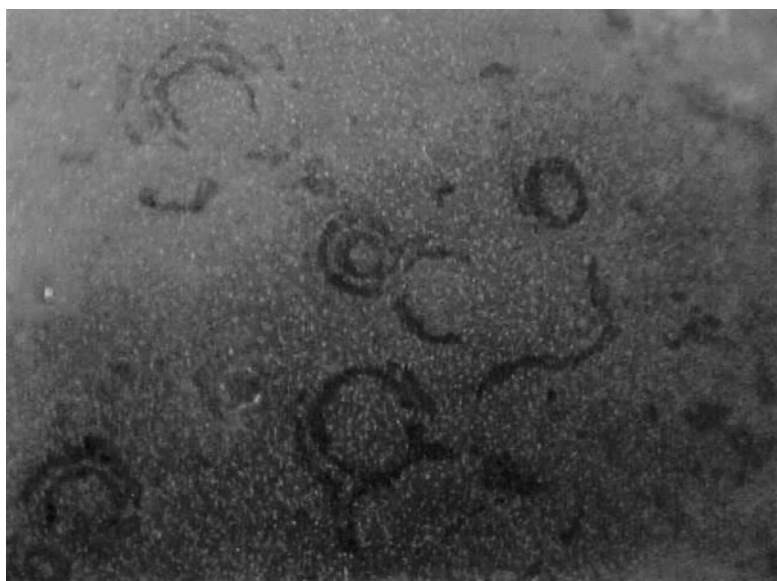
Figure 18: Small concentric rings on the surface of fruits, characteristic of infection by papaya ringspot virus (PRSV-p).

the color of the leaves and the virus is transmitted at the moment of the host recognition or "test probe", when the aphids are searching for a desirable host. By feeding for only a few seconds, an aphid can pick up the virus or infect a plant and in this way the disease can spread rapidly once infected aphids enter a papaya orchard (Nishijima *et al.*, 1989).

The aphids are usually found more numerous on alternate hosts of the virus than on papaya, which is not normally a host plant of aphids. The insects usually move into papaya fields as a result of high populations on alternate hosts, following prevailing wind directions. The disease can spread quickly if there is a source of infected plants in or around the orchard. Rain and the wind are factors important in the move-

ment of the aphids within the orchard or between neighboring orchards, facilitating the dissemination.

In fertilization of the orchards, balanced nutrition of the plants is very important, especially the relation N/K. Deficiency of N significantly favors incidence of the disease (Vallejo, 1999). The virus can also be spread by mechanical means, such as tools contaminated with sap of infected plants, but this is not considered an epidemiologically important method of transmission. Tools can be cleaned of virus by dipping them in chlorine bleach solution 10% (Nishijima *et al.*, 1989). PRSV-p may be transmitted mechanically to other species of the genus *Carica* and also to different species in the family *Cucurbitaceae*, but there is no evidence that it is transmitted in seeds.

Papaya ringspot virus presents a very rapid speed of dissemination from the focal point of the disease, and all of the plants of an orchard may be infected after a period of 3-7 months, as may be observed in areas with high winged aphid populations. The plants may be infected in any phase of vegetative development, and the symptoms appear between 2-3 weeks after inoculation. However, research in India demonstrated a significant effect of the season of planting on the incidence of disease in the plants (Singh and Singh, 1998). Plants infected at a very young age never produce fruit but rarely die prematurely. There are reports, however, that some isolates from Taiwan cause wilting and sometimes death of the plants (Nishijima *et al.*, 1989).

3.1.4 Control

Once established in a papaya orchard, the virus is rapidly disseminated and difficult to control as a consequence of its nonpersistent mode of transmission by aphids. Attempts to reduce disease levels by applying insecticides have not been successful. To control papaya ringspot virus the use of roguing and a combination of different methods is required (Costa *et al.*, 2000; Ventura and Costa, 2002). Considering that commercial varieties resistant to mosaic do not exist and control attempts by use of tolerant varieties and cross protection with attenuated strains of the virus have not shown success in the control of the disease, it has been possible to live economically with the virus using preventative methods and management of the culture aimed at reducing its dissemination:

i.   Carry out periodic visits to the orchard and cut down all infected plants (roguing) as soon as symptoms of mosaic can be recognized. After plants dry or die they no longer will attract aphids;

ii. The trees should be cut as low as possible and do not allow regrowth from the stumps of infected trees. If necessary spray the stumps with a herbicide (glyphosate) to prevent regrowth;

iii. Install nurseries and orchards as far as possible from other orchards, especially if mosaic occurs in the region;

iv. Eliminate the presence of cucurbits (such as pumpkin, melon, watermelon, cucuber, squash or others), which are hosts of the virus as well as hosts of the aphid vectors, in and near the orchard. Aphids in combination with an infected plant in an orchard will result in the rapid spread of the virus;

v.   Realize balanced fertilization, and maintain the orchard clean to prevent the forma

-tion of aphid colonies in weeds;

vi. Avoid planting rows in the same direction as predominant winds, which can favor dissemination of aphids into and within the orchard and to nearby orchards.

vii. Destroy abandoned orchards, principally those with plants infected by the virus. One diseased tree can infect a whole neighborhood;

viii.Install the orchard in regions where lower populations of aphids, vectors of PRSV p, occur;

ix. Buy only papaya plants that have been grown under conditions that will prevent infection of seedlings or grow your plants from certified seeds.

Some growers delay cutting infected trees to enable harvest of fruits on the fruit column Although it is a financial loss to growers when they cut down infected trees, it is the only way to manage the disease. Once a tree is infected with the virus, there is no cure. The longer an infected tree is left standing, the greater is the likelihood that the virus will spread to adjacent trees. This will certainly only result in a greater financial loss to the grower in the long run. The virus can easily spread not only within ones own orchard but also to neighboring orchards. It is critical for growers and field workers to learn to identify the early symptoms of the virus. Those in the field daily will have the greatest opportunity to identify infected trees early. Delay for someone else to identify and destroy infected trees will enable the virus to spread to additional trees. The best way to manage the virus is quick identification and immediate destruction of infected trees.

The measures related above, principally the systematic roguing of plants with mosaic, when employed by all growers in a production region, can give very satisfactory results, as has been noted. This has been demonstrated in the north of Espírito Santo, Brazil, since 1994 when an "Eradication of Papaya Mosaic" campaign supported by the Ministry of Agriculture was implemented in this state. Legislation provides for closing, partially or totally, farms where the presence of papaya ringspot virus has been officially diagnosed and proven; with the immediate eradication of all reported foci of disease, and elimination of the infected plants. Also, it obligates property owners, renters or occupants of whatever title, to eliminate at their expense abandoned orchards, and host plants of aphids, in and near commercial orchards, as well as to install nurseries and orchards as far as possible from other orchards where the disease has been found.

The campaign initially presented an educative character, informing the growers of the importance of the elimination of plants with PRSV-p as soon as symptoms were observed and what was mandated by the legislation. In orchards where the virus was detected, the grower received an official notification of the occurrence of PRSV-p in the orchard, establishing a time for eradication.

After the specified time, officers of the Ministry of Agriculture returned to the farm notified to verify if the grower had complied with the legislation. If not, an interdiction was issued, prohibiting the movement of any papaya vegetative material from the farm. If, the grower still did not adhere to the requirements imposed, compulsory eradication of the orchard was carried out by police action. Considering that non-compliance contributes to the maintenance and diffusion of disease, the producer is subject to

the penalties of Brazilian law, for the crime of diffusion of diseases.

The positive result of the campaign, which made possible the continuation of commercial production of papaya in Espírito Santo, was a function of the frequency of inspections of orchards by Ministry of Agriculture officers, in view of the difficulty of some growers to adhere to requirements of roguing to control of the disease, principally in periods of high papaya prices.

In 1998, of 161 farms inspected, 58 (35%) were notified, 21 (13%) interdicted and 3 were eradicated compulsorily. Of the 5.052ha of papaya cultivated in this year, 771ha (15,3%) were eradicated. In 1999, a greater consciousness of the producers to comply with the legislation was noted, in that, of 168 farms inspected, 37 (22%) were notified and only 5 (3%) were interdicted. Sources of resistance to PRSV-p in cvs. of *C. papaya* have not been found, but other species like *C. cauliflora*, *C. pubescens and C. quercifolia* have resistance to the virus. However the interspecific hybridization of these species with *C. papaya* have not been viable due to the interspecific reproductive barriers. Pre- and post-zygotic barriers have severely limited the use of this approach in breeding programs (Souza Jr and Gonsalves, 1999).

In Australia, hybrids of *C. papaya* x *C. cauliflora* have been evaluated in the greenhouse and field, and present resistance to two Australian strains of the virus, but have however problems of survival and sterility in some plants, possibly due to aneuploidia (Magdalita *et al.*, 1997). Papaya cultivars tolerant to PRSV-p were also developed in breeding programs in Florida (USA) and Taiwan, using in both cases, polygenic tolerance, originating from the dioecious papaya cv. Cariflora, selected by R. Conover (Conover *et al.*, 1986). These tolerant genotypes were susceptible to PRSV-p, but fruit and leaf symptoms were milder, and infected trees produced reasonable quantities of fruits. Solo-type papaya cultivars with resistance or tolerance have not been developed (Nishijima *et al.*, 1998a).

Cross protection is a technique attempted in different countries with reports of different degrees of success (Rezende and Costa, 1993; Lima *et al.*, 2001). This technique involves the use of a mild virus strain to protect plants against the infection of a severe strain of the same virus that causes economic damage (Rezende and Costa, 1993). Mutant mild strains showed promising results in greenhouse experiments, but when tested in field plots success was limited under severe disease pressure. The level of protection is variable and depends on the geographic region from where the virus used was obtained. Cross protection has been reported to be moderately successful in Hawaii and Taiwan, but not successful in Brazil and Thailand (Nishijima *et al.*, 1998a; Rezende and Costa, 1993).

In recent years, there has been intensified research with molecular biology aimed at producing transgenic papaya plants expressing the PRSV coat protein gene (*cp*), that resistance to PRSV-p under field conditions, which has opened up a new control measure. The first studies to develop a transgenic papaya plant resistant to the disease were conducted in the beginning of the decade of 1990, through a cooperative project between Cornell University, the University of Hawaii and the UpJohn corporation of the USA, resulting in the line of plants known as 55-1, that express the gene of the coat protein (cp) of an isolate of the virus obtained in Hawaii (Fitch *et al.*, 1992).

The RO clones of the 55-1 line maintain their resistance to PRSV-p, and the

quality of the fruits and characteristics of the plants are similar to those of 'Sunset' from which they originated (Gonsalves *et al.*, 1998).

In the field evaluations, the transgenic genetic materials were denominated 'UH SunUp' (line 55-1 homozygous for the gene of the coat protein) and 'UH Rainbow' (hybrid from the cross of 'UH SunUp' and the non-transgenic cv. Kapoho,), which retained resistance to the virus, obtained approval from official American organs (APHIS, EPA and FDA) for cultivation and commercialization, and received good acceptance by the papaya producers in Hawaii (Gonsalves *et al.*, 1998).Despite resistance to the virus of Hawaii, however, when these plants were inoculated with virus of other geographic regions, including Brazil, they demonstrated susceptibility (Tennant *et al.*, 1994; Souza Jr., 2000).

In Brazil, EMBRAPA, through of the Center of Cassava and Fruits in Bahia and more recently the Center of Genetic Resources and Biotechnology, established a part-nership with Cornell University to develop transgenic plants resistant to the Brazilian virus, and these plants have also showed resistance to isolates of the virus of Hawaii and Thailand (Souza Jr., 2000). The mechanism of resistance of the papaya plants that express the non-translatable version of the *cp* gene of the virus is mediated by the RNA and not by the protein, and is influenced by the stage of development of the plants and by the concentration of inoculum; the influence of these factors being minimal, how-ever, relative to the effects of the genetic dose and of the degree of homology between the (trans) *cp* gene and the *cp* gene of the virus used in the inoculation (Souza Jr., 2000).

The experience with transgenic papaya in Hawaii is discussed from the technical and the grower/consumer perspective by Ferreira (2001).

Currently there is much concern over the potential ecological impact of transgenic plants. One concern is the risk of viral recombination. Discussion of possible risks associated with to the use of genetically modified organisms (GMOs) has intensified in recent years, raising the concern of bio-safety, principally of food and environmental order. The genetically engineered virus gene in papaya may mix with the genome of other viruses which infect the papaya, creating new and more potent disease-causing viruses. Scientists do not fully understand such interactions, and it is therefore still unclear how readily, if at all, new viruses would arise (Kaesuk-Yoon, 1999; Swain and Powell, 2001).

In the case of transgenic papaya developed in Brazil, the genes utilized have been the *cp* gene of the virus that causes the disease, and the marker gene *nptII*, and the analysis of risks should therefore consider the interaction *C. papaya* x *cp* x *nptII* (Souza Jr., 2000). As the transgenic papaya plants of the Brazilian program express the gene of the coat protein in a non-translatable form, that does not produce the protein, heteroencapsidation of the incoming virus with coat protein produced by transgenic papaya, should not occur, however, as no studies exist with other viruses that occur in the papaya plant, this possibility should be addressed and evaluated.

In the same way, gene flow or vertical transfer of the (trans) gene to other cultivars and hybrids should be considered, principally in the programs of genetic improvement and production of seeds.

Some farmers are reluctant to plant genetically engineered papayas because of market concerns. Typically 35 to 40 per cent of the Hawaiian papaya harvest is exported

to Japan. Genetically engineered papayas have not yet been approved for sale in Japan (Kaesuk-Yonn, 1999; Swain and Powell, 2001).

EMBRAPA Genetic Resources and Biotechnology has developed papaya plants resistant to Brazilian isolates of PRSV-p, but because of questions of bio-safety and patent of the genotypes, these cultivars still are being evaluated and their commercial utilization is not permitted. Possibly, in the future, these transgenic cultivars will be available to growers and will offer a more permanent solution for control of papaya ringspot virus.

3.2 "Meleira" or Sticky Disease

Meleira also known sticky disease, is considered the most important disease of papaya and the major phytosanitary problem for its cultivation in Brazil. The first reports on this disease date from the late 1980's, when it was observed in orchards in the south of Bahia and north of Espírito Santo, Brazil (Rodrigues *et al.*, 1989a). Since then, the disease has also been found in the states of Ceará, Minas Gerais, Paraíba and Rio Grande do Norte.

Initially, meleira was localized in small areas, but today, it can be detected in all orchards of papaya in the north of Espírito Santo and in some municipalities of the states of Bahia, Minas Gerais, Ceará, and Pernambuco. The disease is characterized by intense exudation of latex in fruits that oxidizes and darkens making them totally unsuitable for commercialization, as well as compromising their flavor. In the north of Espírito Santo, while the percentage of plants eradicated with mosaic (PRSV-p) is about 2% in well-managed orchards, meleira typically infects at least 20% during the economic cycle of culture. In some orchards where rouging was not carried out, an incidence of the disease of up to 100% was recorded by the time the plants reached the harvest phase and were only 12-15 months old.

Although the occurrence of meleira had been noted by producers since the decade of 1970, this problem worried no one for the most part, until the middle of the following decade, when it was reported causing losses in commercial orchards in Teixeira de Freitas, in the south of Bahia. Meleira had been observed since 1984 in the state of Espírito Santo, but was only reported officially in 1989, when it was proven by means of epidemiological studies to be in fact a disease with biotic etiology (Rodrigues *et al.*, 1989a and 1989b).

3.2.1 Etiology

Initially, symptoms of the disease were attributed to a deficiency in the absorption of calcium or boron resulting from a lack of water or imbalance of these elements in the soil (Nakagawa *et al.*, 1987).

The biotic etiology of this disease was confirmed after the development of the anticipated typical symptoms in papaya plants followed inoculation of healthy plants with latex collected from symptomatic fruits (Rodrigues *et al.*, 1989a; Kitajima *et al.*, 1993; Ventura *et al.*, 2001a). Also, the association meleira with a biotic agent was studied by the monitoring dispersal of the disease in commercial orchards, which indicated

involvement of a pathogen (Rodrigues *et al.*, 1989b; Maffia *et al.*, 1993). Transmission electron microscope studies of the latex of leaves, stems, and fruits of plants with symptoms of meleira indicate that the disease is of viral etiology, with the presence of large numbers of isometric particles of approximately 50nm diameter, and the occurrence of a double strand of RNA (dsRNA), of approximately 6×10^6 daltons, in latex and extract of leaves and fruits of affected plants. Ultrafine tissue sections revel that these isometric particles are restricted to lactiferous vesicles (Kitajima *et al.*, 1993).

The efficiency of the virus purification protocol was confirmed after successful purification of the viral particles, and the RNA of approximately 12 kb long was observed after removing the proteins from the virus particles. The double stranded nature of the RNA was confirmed in a 1% agarose gel, as 6×10^6 daltons dsRNA purified directly from leaves (Souza Jr. *et al.*, 2002; Zambolim *et al.*, 2000). Virus purification from infected plants confirmed the viral etiology of the disease, with the inoculation of healthy papaya plants that developed symptoms after inoculation (Zambolim *et al.*, 2000). Studies demonstrated that the dsRNA is found in leaves, stem, fruits, flowers and roots of papaya.

In 2001, researchers of thr EMBRAPA Genetic Resources and Biotechnology Center, Department of Plant Pathology/Federal University of Viçosa-UFV, INCAPER and the Federal University of Espírito Santo-UFES, joined efforts and initiated collaborative work towards the characterization of the virus genome, as well as the development of protocols for early and widespread diagnosis.

3.2.2 Symptoms

Exudation of latex from fruits, spontaneous or provoked by injuries, which appears to be more fluid than the typically milky latex found in healthy plants, and that oxidizes and becomes dark, is the most common symptom of meleira (Fig. 19).

In severe cases, the extensive exudation gives a sticky aspect to the fruit, from which the name of the disease originates. The latex of fruits of a plant with meleira presents a clear watery aspect, due to its lower viscosity and lack of coagulation, that darkens with greater facility than that of healthy fruits (Ventura *et al.*, 2001b). The exudation of latex also occurs from edges of young leaves in the top of the plant and with oxidation provokes small light-brown necrotic lesions on the leaf tips (Fig. 20). These symptoms, although not always observed, permit the identification of the disease in young plants. However, they should not be confused with the lesions caused by the blight of the leaves caused by the fungus *P. caricae - papayae*.

In infected fruits, in advanced stages of the disease, irregular light-green areas are observed on the surface, slightly resembling symptoms of deficiency of micronutrients, principally boron. Farmers must be careful not to confuse symptoms of meleira in fruits with those caused by mechanical damage. Thus, it is important to note the viscosity of the latex to obtain a secure diagnosis of the disease.

3.2.3 Epidemiology

The causal agent of meleira was efficiently transmitted by injection of the latex of

diseased plants into the stem of healthy papaya, obtaining disease symptoms approximately 45 days after inoculation. Mechanical transmission by friction using latex was not effective in papaya and other plant species, requiring a continuation of research regarding transmission and alternative hosts.

Recent studies provide strong evidence of the existence of an aerial vector associated with the disease. The silverleaf whitefly (*Bemisia argentifolii* Bell. & Perring), also known as *B. tabaci* biotype B, has been associated experimentally, in controlled

Figure 19: Typical symptoms of the meleira (sticky disease) on fruits with the characteristic flow of oxidized latex

conditions, with the transmission of meleira (Vidal *et al.*, 2000).

In the plants subjected to infestation with the silverleaf whitefly, monthly monitoring for the presence of dsRNA particles was carried out, with detection occurring six months after infestation and the appearance of the characteristic symptoms of exudation of latex in the fruits in 8 months (Vidal *et al.*, 2000).

The involvement of leafhoppers, principally of the genera *Solanasca* is also

being investigated. The presence of dsRNA, with molecular weight similar to that associated with meleira was detected in samples of gramineae species *Trichacne insulares* and *Brachiaria decunbens*, present in orchards of papaya affected by meleira in the south of Bahia (Barbosa *et al.*, 1998).

Field observations, based on careful monitoring and monthly mapping of the distribution of diseased plants, demonstrated that the dispersion of meleira was along the lines of planting, presenting an aggregated arrangement of diseased plants. This evidence suggests the possibly that cultural practices could be responsible for dissemination of the disease within the orchard, because these practices are normally executed following the direction of the plant rows. Fruits of papaya from of plants infected by the virus present greater predisposition to infestation by fruit flies, principally the species *Ceratitis capitata*, possibly by the alteration of the levels of benzyl
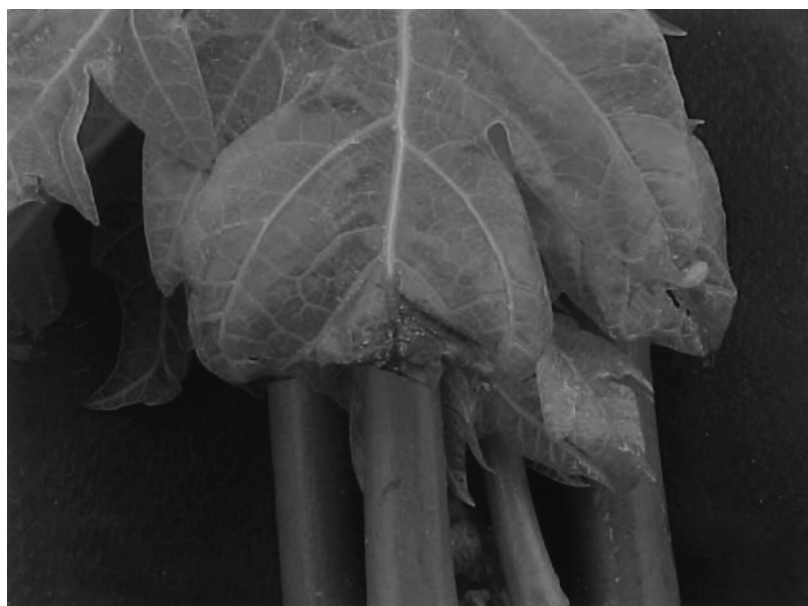
Figure 20: Early symptoms of necrotic lesions caused by meleira virus on young leaves

isothiocyanate (BITC), present in the fruits (Martins, *et al.*, 1999). Research developed by INCAPER demonstrated that the fly only occurs in fruits of plants more than three months after the appearance of disease symptoms, and is absent in areas where systematic rouging is carried out. The correct execution of a "systems approach" for production in the form carried out in the state of Espírito Santo, and which has become an important measure for the production of fruit for exportation, shows effectiveness even in orchards where meleira occurs.

3.2.4 Control

The roguing of infected plants is recommended until more specific control procedures

have been developed. The following measures are also recommended, with the objective of reducing dissemination of the disease as well as preventing or delaying its introduction into areas where the disease has not been noted:

i.   Carry out weekly inspections in orchards and eliminate diseased plants (roguing), as soon as the first symptoms of meleira are detected (Ventura *et al.*, 2001b);
ii.   Install nurseries and new orchards as far as possible from other orchards, especially those with a history of disease;
iii. Do not collect seeds from diseased plants and orchards with high disease incidence;
iv.   Disinfect all material used in the process of thinning and harvesting fruits; try to reduce injuries to plants during cultural treatments;
v.   Manage the vegetation under the plants, maintaining rows clean and the area between rows trimmed to diminish the variety of weed species;
vi.   Eliminate all orchards (diseased and healthy) at the end of the economic cycle of production to eliminate sources of inoculum;
vii. Destroy abandoned orchards, principally those with plants infected by the virus.

One diseased tree can infect a whole neighborhood. The Brazilian Ministry of Agriculture, by a Federal decree, specifies that farms of the state of Espírito Santo where official diagnosis has proven the presence of papaya meleira virus will be notified of the disease occurrence, and infected plants will be immediately eliminated as recommended for control of papaya ringspot virus.

3.3 Papaya Lethal Yellowing Virus

Lethal Yellow of Solo papaya (*Papaya lethal yellowing virus* – PLYV) was initially reported in papaya plants in the state of Pernambuco, Brazil, in 1983. Later, the disease was reported in the states of Bahia, Paraíba, Rio Grande do Norte and Ceará, being a virus little known but that is spreading in northeastern Brazil (Lima *et al.*, 2001). Because most cases of this virus have been encountered in individual plants or small orchards, little information exists about its economic importance for production. In a survey in Rio Grande do Norte, a disease incidence of 40% was reported (Kitajima *et al.*, 1982a and 1982b).

However, based on reports made by those who have observed the problem, it is believed that papaya lethal yellowing virus may bring great damage to production if it is disseminated to major, commercial growers.

3.3.1 Etiology

The disease is caused by *Papaya lethal yellowing virus* (PLYV), a virus with isometric particles, of diameter in the range of 29-32nm, and that occurs in high concentration in the tissues of affected plants.

Studies conducted with an isolate obtained from papaya plants from Natal and Rio Grande do Norte, demonstrate that the virus is made up of a piece of single stranded RNA of ca. $1,6 \times 10^6$ Da, with a single protein capsid of 36 kDa (Kitajima *et al.*, 1982b; Kitajima, 1999).

The virus was initially considered a possible member of *Tombusviridae* Family, genera *Carmovirus*, but recent molecular research shows a homology of ca. 51% of the gene polymerase nucleotidic sequences and the VPg with the Family *Sobemoviridae* and genera *Sobemovirus*. Also the sequences of *cp* gene show homology of ca. 44% with *Sobemovirus* (Silava *et al.*, 2000).

3.3.2 Symptoms

The symptoms in Solo papaya begin with a yellowing of young leaves of the top third of the stem, which later may fall. Longitudinal depressions and necrotic lesions may be observed on the leaf petioles and on the bottom side in the leaf veins, respectively. Fruits have an intense exudation of latex and round chlorotic spots. With evolution of the disease, the stem becomes twisted and the leaves chlorotic. Subsequently, the yellowed leaves wilt and dry, leading to plant death. In plants of the cv. Caiano, the foliar symptoms develop in the same manner as in the cvs. of the Solo group. However, twisting of the stem and death of the plant do not occur.

In fruits, circular spots are observed on the skin, initially light green and later yellowing. Maturation of the pulp is delayed and appears generally stony.

3.3.3 Epidemiology

The virus is transmitted mechanically from papaya to papaya. Seeds of fruits from infected plants have been tested by ELISA, indirectly verifying the presence of the virus on the surface of the seeds (Camarch *et al.*, 1997). These results suggest the recommendation to not utilize seeds of diseased plants, as well as the risk of introducing the disease in uninfested production areas through seeds.

Transmission between plants appears to depend on insect vectors still not identified. However, its form of dispersion indicates a low effectiveness of these vectors. The virus has the capability of surviving in soils of the rhizosphere of infected plants and infecting healthy seedlings when planted in these soils (Camarch and Lima, 1997). The virus also is dispersed by irrigation water (Camarch *et al.* 1997).

3.3.4 Control

Although this is a disease that has received little study, there are recommendations for control, including measures of general scope, employed to minimize dissemination within those states where it has been found, as well as to avoid or delay its introduction into areas where it still has not been noted.

Avoid movement of seedlings and seeds between states, principally those originating from those states where the disease occurs. Efforts to control the disease consists of roguing and a combination of other management methods:

i. Systematically eradicate affected plants through periodic inspections in orchards where the disease is present;

ii. Buy only papaya plants that have been grown under conditions that will prevent infection of seedlings or grow your plants from certified seeds.

iii. Install nurseries and orchards as far as possible from other orchards, especially if the disease is known to occur in the area;

iv. Manage soil and irrigation of orchards taking into consideration survival of PLYV in the soil and water

v. Tools used on infected plants may be cleaned of virus by dipping them in chlorine bleach solution 10%;

vi. Eradicate old papaya plants so they will not serve as a source of inoculum of the pathogen.

3.4 Papaya Droopy Necrosis and Papaya Apical Necrosis

Papaya droopy necrosis virus has been reported in Florida (USA) and a similar virus disease causing apical necrosis was reported in Venezuela. The viruses have similar particle morphology, and both can cause the same symptoms (Wang and Conover, 1983; Lastra and Quintero, 1981).

3.4.1 Etiology

The diseases are caused by rhabdoviruses with similar sized particles of 87-98nm x 180-254nm, for *Papaya droopy necrosis virus* (PDNV) and 80-84nm x 210-230nm for *Papaya apical necrosis virus* (PANV), aggregated and detected in the parenchyma cells in vascular tissues (Lastra and Quintero, 1981; Zettler and Wang, 1998).

3.4.2 Symptoms

The earliest symptoms of PDNV are the drooping and curvature of the leaves in the plant upper crown, which is rounded and has a distinct bunchy appearance with short internodes. The youngest leaves at the apex are pale yellow, sharply cupped downward, do not expand properly and may also be marginally necrotic. The petioles are shorter and more stiff than normal. Flowers of diseased plants abort, and fruit set ceases. The stem tip becomes very short, necrotic, and the plant eventually dies (Pernezny and Litz, 1993).

3.4.3 Epidemiology

The disease incidence is relatively low when compared with other papaya viruses, and is considerably reduced by avoiding overlapping and successive crops and by promptly roguing infected plants (Wang and Conover, 1983; Zettler and Wang, 1998). The disease usually increases in severity in the winter.

The diseases is mechanically transmissible. No natural vector or alternative host has been identified for PDNV, but papaya seedlings exposed to the leafhopper *Empoasca papaya* that had fed on PANV infected plants developed symptoms. However particles of the rhabdovirus were not detected in these plants (Zettler and Wang, 1998).

3.4.4 Control

Since field incidence is low at present, the only practical means of control is to avoid overlapping and successive crops and realize prompt roguing of symptomatic plants.

4. Diseaes caused by Phytoplasma

4.1 Dieback and Yellow Crinkle

In Australia papaya may be infected by a phytoplasma causing the disease known as by "dieback", "yellow crinkle" and "papaya mosaic" (Gibb *et al.*, 1996 and 1998). Papaya dieback disease has become one of the most serious factors limiting papaya production in Australia (Queensland), with annual plant losses often greater than 5%, and up to 100% during epidemics. The disease was first recorded in Queensland in the early 1920s (Cook, 1975; Glennie and Chapman, 1976), about the time the crop was first grown commercially.

4.1.1 Symptoms

Diseased plants present apical leaves with a slightly chlorotic appearance that soon after begin to shrivel. The older leaves develop a chlorosis and may fall.

The development of translucent areas in the extremities of the leaves of the crown and necrosis reaching the principle veins resulting in a "claw like" aspect may also be seen.

Dieback-affected papaya plants were characterized by a discoloration of the contents of laticifers, while the anatomy of sieve elements was healthy in appearance until the necrotic stages of the disorder were reached. Laticifer discoloration was not always associated with the presence of phytoplasma in affected tissue, as determined by polymerase chain reaction (PCR), using primers based on the 16S rRNA gene and 16S-23S intergenic spacer region (Siddique *et al.*, 1998).

The production of fruits is affected by the development of a greening of the floral parts and appearance of phyllody (transformation of the flowers into leaf-like structures). Plants with the "mosaic" symptoms present wet lesions in the stem, petioles and fruits.

4.1.2 Etiology

Over the years, many investigations failed to conclusively link nutrient deficiencies/ toxicities, nematode damage or a pathogenic microorganism to the disease. Although viruses, bacteria and fungi were not consistently detected by conventional techniques, the progression of disease symptoms did seem to indicate a plant pathogen affecting the phloem tissues of diseased plants.

In 1998, three phytoplasma-related diseases of papaya (*Carica papaya*), dieback, yellow crinkle, and mosaic, were recognized within Australia (Guthrie *et al.*, 1998). Phytoplasma DNA was detected using the polymerase chain reaction (PCR) with prim-

ers specific for phytoplasmas in general, and for the stolbur group of phytoplasmas. Phytoplasma DNA was detected in a range of plant tissues, including roots, but not in mature leaves, which would act as photoassimilate sources (Siddique *et al.*, 1998). Positive results for PCR tests for phytoplasmas were consistently obtained for plants affected by dieback and yellow crinkle, and no amplification was obtained from DNA extracts from a number of asymptomatic papaya plants. This was the first consistent evidence for the association of a pathogenic organism with both dieback and yellow crinkle. Similar results were obtained by other research groups and confirmed that it was phytoplasma DNA that was being amplified from tissues of diseased plants (Gibb *et al.*, 1996; Davis, *et al.*, 1996; White *et al.*, 1997). In these studies, although PCR tests indicated the presence of phytoplasma DNA, phytoplasma cells were not observed in transmission electron microscopy studies of mature sieve elements of dieback affected leaf, stem, or fruit tissue from plants at various stages of symptom expression.

In some cases membrane-bound structures, similar in shape and size to phytoplasma cells, were observed within vacuoles of cells in the phloem tissue of leaves displaying tissue breakdown in the form of a water-soaked appearance to veins ("X-Y" patterning). But, the structures were interpreted as autophagic vesicles or latex vesicles in immature laticifers. In contrast, phytoplasmas were readily observed in papaya leaves displaying symptoms of yellow crinkle. Siddique *et al.* (1998), concluded that phytoplasma cells are present in very low titer in dieback-affected tissues and that, while the plant appears to limit proliferation of the dieback-associated pathogen, this defense strategy is ultimately unsuccessful because it is associated with a rapid decline of the papaya plant.

4.1.3 Epidemiology

Phytoplasmas are spread from plant to plant by phloem-feeding insects, typically leafhoppers and planthoppers. It was transmitted by the leafhopper *Orosius argentatus* Evans, but attempts to transmit the disease by mechanical inoculation have been unsuccessful. The pathogen was also transmitted by dodder (*Cuscuta australis* Hook.), but not by grafting from papaya to papaya (Cook, 1975).

The dieback-associated phytoplasma was detected 1 week prior to or the same week as symptom expression, while phytoplasma DNA was detected between 3 and 11 weeks prior to expression of mosaic symptom. Lateral shoot regrowth on the lower stem of plants that had suffered dieback disease failed to generate stolbur-specific PCR products (Guthrie *et al.*, 1998).

In the field young plants are more susceptible than older plants, and most extensive losses are experienced in the first 12 months following planting (Cook, 1975).

4.1.4 Control

To minimize losses that can range from 20 to 100% of production, producers in Australia utilize roguing and a practice of the cutting the plants at approximately 0.75m of height (ratooning) as soon the first symptoms of the disease appear.

This strategy of management is based on the principle of that the phytoplasma

is located in the upper part of the plant, and ratooning and rouging reduce its potential dissemination (Guthrie *et al.*, 1998). Therefore, ratooning of dieback-affected plants and removal of yellow crinkle or mosaic-affected plants are suggested for the management of the disease.

4.2 Bunchy Top

Papaya bunchy top has been observed throughout much of the Caribbean region, from Cuba southward to Trinidad, since it was first reported in Puerto Rico in 1931 (Cook, 1975). Although the occurrence of the disease appeared to be restricted to the Caribbean islands, recent observations suggest that it is present in Central America and the northern part of South America. The disease has not been reported in Hawaii and Brazil. Bunchy top is a devastating disease and can severely limit commercial papaya production (Nishijima, *et al.* 1998a).

4.2.1 Symptoms

The first symptom of bunchy top is a faint mottling of the upper leaves. The laminae of infected leaves progressively become more chlorotic, especially in interveinal areas, and may eventually exhibit marginal necrosis. Leaves and petioles show reduced growth, and become rigid. Internodes become progressively shortened, and petioles tend to assume a more horizontal position. Oily appearing spots are often present in the upper parts of stems and in petioles. Apical growth ultimately ceases, which, with the shortening of internodes, imparts a "bunchy top" appearance to affected plants. With the cessation of apical growth and decline of plant vigor, the oldest leaves progressively abscise, leaving fewer, more stunted leaves at the top. Eventually, plants may be entirely killed; however, new shoots may sprout from the lower, non-affected portion of stems.

If fruits are set on infected plants, their flavor may be bitter due to the disease (Cook, 1975, Nishijima, 1998a). In plants with the dieback syndrome the absence of latex exudate from wounds was observed (Cook, 1975, Webb and Davis, 1987) and failure of latex to exude from wounds on diseased leaves, petioles, stems or fruits is a common characteristic of this disease.

4.2.2 Etiology

Although bunchy top was originally thought to be caused by a virus, the disease is most likely caused by a phytoplasma. The pathogen has been observed in the phloem of infected plants using transmission electron microscopy, and infected plants treated with tetracycline-based antibiotics exhibited a remission of symptoms (Rezende and Costa, 1993; Story and Halliwell, 1969). Although the absence of latex flow from fresh puncture wounds in leaves, petioles, stems, and fruits was considered diagnostic for bunchy top, a more recent study indicates that this is not a reliable diagnostic characteristic. The presence of phytoplasma in plants with foliar symptoms of bunchy top and normal latex flow was confirmed by electron microscopy.

4.2.3 Epidemiology

Two leafhoppers, *Empoasca papayae* Oman and *E. stevensi* Young, transmit the bunchy top agent, and the occurrence of the disease coincides largely with that of these vectors (Haque and Parasram, 1973; Seín and Adsuar, 1947; Story and Halliwell, 1969). *Empoasca papayae* has most frequently been associated with the disease, but in Trinidad, where *E. papayae* is not established, *E. stevensi* was found to be a vector. *Empoasca papayae* has rarely been found on other plant species and is the only leafhopper known to breed on papaya. *Empoasca stevensi* was originally described from specimens collected from papaya in Florida in 1940, but little is known about the ecology and distribution of this species. Interestingly, both bunchy top and *E. papayae* are not known to occur in Florida (Haque and Parasram, 1973; Story and Halliwell, 1969; Webb and Davis, 1987).

Bunchy top also can be also transmitted by grafting. Symptoms usually begin to appear 30 to 45 days after inoculation. Some papaya cultivars are more tolerant to the disease than others, but immunity is not known. The degree of tolerance affects the rate and severity of symptom expression. However, it is not known whether vector preference and the ability to transmit the bunchy top phytoplasma are affected by cultivar differences.

4.2.4 Control

Currently, the use of tolerant cultivars is the only practical means of control of bunchy top, but it has limited application. Where bunchy top occurs, tolerant cultivars can only be grown commercially in locations with low disease pressure. Disease pressure varies with geographic location, presumably due to insect vector ecology. Application of insecticide to reduce the incidence of leafhopper vectors may be beneficial (Nishijima *et al.*, 1998a).

In the past, the disease has been managed through a program involving leafhopper control throughout the productive life of the crop and the removal of sources of inoculum. The latter was attained by rouging infected trees or the topping of infected plants below the point where latex exudes after wounding. Axillary shoots that develop after topping are often free of the disease (Cook, 1975).

Antibiotic therapy is an effective control measure from an experimental standpoint but has not been applied commercially. Drenching the soil around infected plants with chlortetracycline or tetracycline hydrochloride and drenching combined with root-dip treatments were both successful. The efficacy of foliar application or trunk injection has not been established (Cook, 1975; Rezende and Costa, 1993).

5. Diseases caused by bacteria

5. 1 Bacterial Leaf Spot

Bacterial leaf spot, was first reported in the state of Rio de Janeiro, Brazil, in 1955 but since then has appeared in other countries (Cook, 1975), and more recently in the state

of Paraná, Brazil, in the region of Paranavaí, in plants both in the nursery and in the field (Funada *et al.*, 1998).

The causal agent is the bacterium *Pseudomonas carica-papayae* Robbs, gram-negative, rod-shaped, with three to six polar flagella. Colonies on nutrient agar medium are circular, flat, gray white, and fluorescent. Neither ammonia, indol or hydrogen sulfide is produced, and nitrates are not reduced, and the optimum temperature for growth *in vitro* is 23-29°C (Robbs, 1956).

Initially the lesions caused by the pathogen are minute, circular to angular and

Figure 21: Symptoms of bacterial leaf spot, with coalescence of spots

of dark-green color, and water-soaked spots first appear on the lower surface of the leaves (Robbs, 1956; Funada *et al.*, 1998). These lesions later became light brown, or of chestnut shade, translucent, 3 to 6mm in diameter, and coalesce to form irregular necrotic areas on affected leaves (Fig. 21). On the lower surface of the leaf, in the local corresponding to the lesion and under conditions of high humidity, a milky exudation made up of bacterial exudate may be seen.

The disease has irregular occurrence and is of no economic importance, but

when it occurs in young plants it can cause the death of the plants, principally in the nursery. Control should be preventative by means of sprays with products based on copper fungicides. Removal of affected plant parts and the roguing of diseased plants are also recommended. Utilization of seeds from orchards where the disease occurs is not recommended.

5.2 Purple Stain Fruit Rot and Internal Yellowing

In Hawaii, two bacteria (*Erwinia herbicola* (Loehnis) Dye and *Enterobacter cloacae* (Jordan) Horn. & Ed.) can cause rot of the pulp of fruits that affect the internal flesh of ripening fruits without causing easily noticeable external symptoms. Only purple stain fruit rot associated with *E. herbicola* occurs in sporadic form in the north of Espírito Santo, Brazil.

Internal yellowing is caused by *E. cloacae,* a gram negative rod-shaped bacterium, with peritrichous flagella, oxidase negative, catalase positive, and facultatively anaerobic. Both pathogens remain quiescent during fruit development until fruit are ripe, when symptoms are expressed.

When harvested, fruits infected by purple stain do not present external symptoms that easily differentiate them from healthy; however, when cut, a softening of the pulp with intense red coloration is observed, with decomposition of the tissues occurring, and with in some cases the red color accentuated in the vascular tissue next to the seed cavity (Fig. 22). Infected tissues of the flesh of the fruit become soft, translucent, and rotted as the fruit ripens (Nelson and Nishijima, 1980). Purple stain appears to be most common during the winter months, although incidence of the disease is usually very low and sporadic (Nishijima *et al.*, 1987). In the field, before harvest, it is possible to identify the infected fruits, which present a precocious maturation (out of position or normal order) on the plant and irregular distribution of yellow color on the skin, frequently associated a light deformation of the fruit.

The internal yellowing rot caused by *E. cloacae* causes a softening of the pulp, around the seed cavity, usually near the calyx and middle sections of the fruit, that becomes translucent or with a bright yellow to lemon-green color, with a rotting odor. The bacterium is associated with oriental fruit fly *Dacus dorsalis* Hendel, which is attracted to the bacterium and possibly transmits it to papaya flowers (Jan and Nishijima, 1990; Nishijima, 1998d). This disease still has not been reported in Brazil.

Based on their sporadic occurrence, these bacteria have little economic importance, however, infected fruits can contaminate the thermal treatment tanks, as well as installations of the packinghouse. Therefore, the elimination of the diseased fruits and the sanitation of the installations are recommended (Nelson and Alvarez, 1980; Nishijima, 1998d).

6. Diseases caused by nematodes

In the culture of papaya, gall nematodes (*Meloidogyne* spp.) and the reniform nematode (*Rotylenchulus reniformis* Linford & Oliveira), tiny soil-inhabiting worms, are the most important nematode species, principally when the orchards of papaya are planted

in sequence with other hosts of these species, which can affect the development of the plants and cause the formation of galls in the radicular system, reducing the capability for absorbtion of nutrients and water. In Espírito Santo, the damage until present is minor and reports of these nematodes are restricted to limited foci without economic importance.

6.1 Root-knot nematodes

Damage to the radicular system of the plants depends on the level of infestation of the soil. The larvae penetrate into the radicular system causing hypertrophy of tissues (giant cells) forming galls and consequently a reduction of the root system and stunting of plants, which may be more sensitive than normal to water stress, with fruits
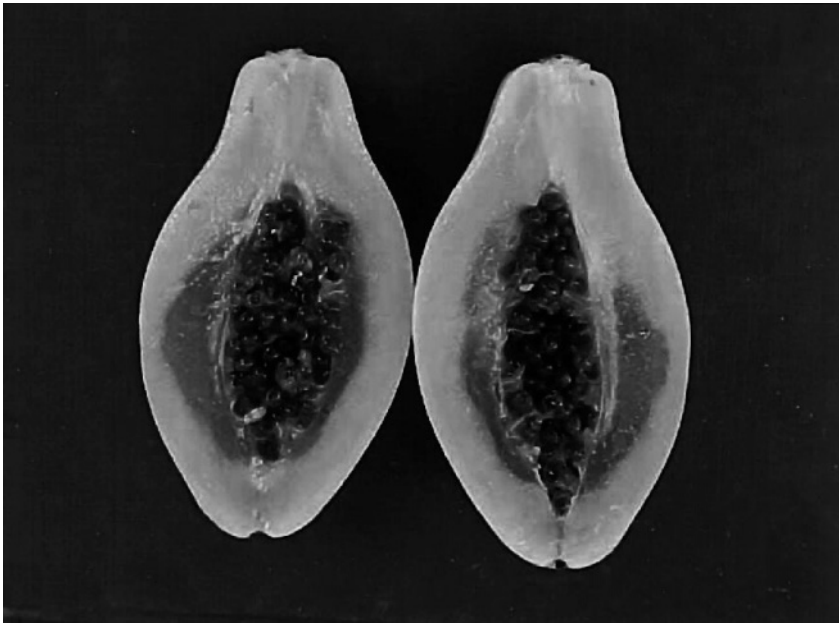
Figure 22: Longitudinal cross section of papaya fruit showing symptoms of purple stain with severe internal red coloration of the flesh.

smaller than normal and more likely to have an off-flavor. The symptoms are much less severe in loam or clay soils.

The species *Meloidogyne incognita* (Kofoid & White) Chitwood has been identified most frequently provoking galls in papaya plants.

Other species, however, like *M. javanica* (Treub) Chitwood, *M. arenaria* (Neal) Chitwood and *M. hapla* Chitwood, also have been reported infesting papaya in different countries, but occur less often. *M. hapla* prefers cool temperatures and may damage papaya trees grown at higher elevations. The larvae of these nematodes can travel

short distances in soil, finding and attacking papaya roots. Root penetration is by second-stage juvenile, generally occurring near the root tip, and when the female begins feeding in the central cylinder region of the root, giant cells are formed. During the process of formation of the galls or knots, the females remain sedentary, embedded in the tissues of the root. Each females may produce more than 350 eggs, which are maintained in oothecas, and can give rise to of 14 to 17 generations in a year. To confirm a diagnosis, one can cut into the galls and observe pearly looking, pear-shaped female nematodes embedded in the tissue.

Owing to the biology and mode of dispersion of the nematodes, preventative control associated with management practices is most recommended.

Often seedlings infested in the nursery only manifest symptoms of the nematodes later in the field, depending on climatic conditions, principally temperature and humidity, and physical and chemical characteristics of the soil.

The use of organic material, non-host plants, and antagonistic or suppressive plants are alternatives that should be used to reduce the population of nematodes in the soil. The effects of root knot can be partially alleviated by maintaining plants at optimum water and nutrition levels. No nematicides are registered for use on papaya plants in Brazil and USA, and some common systemic nematicides are phytotoxic to papaya.

6.2 Reniform nematodes

Rotylenchulus reniformis Linford & Oliveira infests many plants from the tropics to temperate regions and on papaya causes leaf chlorosis and in some cases, stunting of plants. The plants may show some wilting during periods of peak transpirational stress on plants and the fruit produced on diseased plants are smaller than normal and may be slightly soft (Holtzmann and McSorley, 1998). The nematodes also cause lesions in the cortical tissue of the roots, which can serve as ports of entry for other pathogens.

Careful observation of the washed roots with a lens (10x), makes possible identification of small lesions and the presence of points of sand like bodies in the roots, which are egg masses of the nematode.

Dissemination of the nematodes occurs principally through contaminated seedlings, by cultural treatments, and by the surface runoff of rain or through irrigation water. The juvenile phases penetrate into the cortex of the roots and remain sedentary with part of the body outside of the roots, acquiring the form of a kidney (hence the name reniform). The nematode feeds near the phloem in papaya roots, sometimes inducing the formation of giant cells and a complete life cycle requires about 25-30 days. Adults produce a gelatinous matrix containing egg masses with the appearance of "grains of sand" used in the field diagnosis.

Preplant soil treatments have been used to reduce nematode populations and have resulted in increased vigor and yields. Nematicides registered for use on papaya orchards do not exist in Brazil, and recommendations for control have to be based on management practices. Some common systemic nematicides are phytotoxic to papaya plants.

7. Miscellaneous and abiotic diseases

7. 1. "Vira cabeca" or Apical necrosis

"Vira cabeça" or apical necrosis is a devastating disease that can limit commercial papaya orchards and in recent years has increased in incidence in the south of Bahia and north of Espírito Santo, Brazil principally in the first year of development of the plants. It is very similar to bunchy top disease that was first reported in Puerto Rico in 1931 and now occurs in Central and South America.

Transmission electron microscopy has been used to observe the cells and tissues of infected plants, and PCR work with primers used for phytoplasm diagnosis indicated negative and inconsistent results (Kitajima, USP-personal communication). So, the identity of the disease etiology still is currently in doubt (unknown etiology), but the epidemiological evidence suggests the involvement of an infectious agent possibly transmitted by insects. Evaluation of mechanical transmission by insects and by grafting is being evaluated by researchers of Incaper.In Australia a disease with similar symptoms and epidemiological characteristics has been associated with a phytoplasm (*see* phytoplasma diseases).

7.1.1 Symptoms

The initial symptom of "vira cabeça" is a faint mottling of the upper leaves involving one or more young leaves. The leaves become chlorotic, especially in the interveinal areas, and eventually exhibit marginal necrosis. The petioles are reduced and assume a horizontal position, with the internodes shortened with fewer, stunted leaves. All of the leaves of the apex of the plant become chlorotic, with poor growth, and subsequently they dry and fall (Fig. 23). Sometimes oily spots appear in the upper, apical part of the plant stem. Apical growth ultimately ceases, necrosis and curvature of the apex occurs, and the plant apex dies.In the more advanced stages of the disease the plants loose their leaves, develop extensive apical necrosis and finally die.

7.1.2 Epidemiology and Control

Roguing of symptomatic plants is recommended to reduce the inoculum source and disease incidence.

7.2 Freckles or Physiological fruit spot

The quality of papaya fruits produced in the north of Espírito Santo and in the state of Bahia, Brazil, have been seriously compromised by a disturbance known as freckles or physiological fruit spot, also referred to in the international literature as "freckle spot" (Ishii and Holtzmann, 1963; Nishijima *et al.*, 1998a).

The incidence and severity of freckles, which affect the external appearance of the fruit skin, have caused great damage for exportation, with losses that reach 50% of the fruits destined for exportation, principally in the period of when sunny days prevail

(May to October), The freckles are a cosmetic disorder, since are superficial and do not affect the flesh of the fruits that become depreciated visually for the fresh market.

The etiology of freckles still is not well known, but experimental evidence points to a physiological disturbance (Eloisa *et al.*, 1994; Nishijima, 1998a). Temperature, associated with water deficiency, solar radiation, and the physiological state of the plant, appear to influence the incidence and intensity of the spots. These conditions are present during the period of greatest occurrence of freckle in the north of the Espírito

Figure 23: Symptoms of "vira cabeça" on papaya tree.

Santo.

In other fruits, low temperatures and the limitation of nocturnal transpiration in the fruits increases the potential turgor in the pericarp causing cracks (Aloni *et al.*, 1998). Physiological disorders have been associated with an imbalance of calcium in vegetative cells (Bangerth, 1979; Zambolim and Ventura, 1993), principally in the tissues with a more elevated respiratory rate, having an accumulation of Ca^{++}.

In the north of Espírito Santo, it can be observed that more vigorous plants, with

better foliation, and apparently without problems of water deficiency have presented the least intensity of spots in the fruits, suggesting that probably these plants have greatest resistance to the effect of the temperature on the fruits, suffering less from solar exposure. It is known that heat stress can cause a reduction in photosynthesis and respiration of the cells as well as the liberation of electrolytes.

The side of fruits exposed to the greatest solar radiation consistently presents the greatest intensity of spots. This occurrence may be associated with the temperature on the surface, causing cellular lesions and consequently exudation of latex below of the epidermis of the fruits. The increase in turgor pressure under conditions of thermal amplitudes (fluctuations of temperature) also can cause the rupture of lactiferous vessels in the mesocarp of the fruits, provoking the spots.

The symptoms are small superficial spots that range from points to 10mm of diameter, with appearance of gray or brown freckles on the skin of the fruits, varying in the form and number on the fruits, that coalesce and cause the fruit surface to appear russetted (Fig. 24).

They are observed only in the fruits of more than 40 days of age and intensify in the final phase of development of the fruits, principally near the point of harvest. The larger spots generally present darker coloration with miniscule cracks, showing in some cases, in the margins, a soaked appearance, and can coalesce, affecting up to 50% of the exposed surface of the fruits. In post-harvest, when the fruits mature the freckles present a greenish coloration, contrasting with the yellow of the skin, reducing commercialization of the fruits.

For the growers, while research studies are not conclusive, carrying out the correct management of irrigation, preventing the plants from suffering water stress, as well as proper fertilization, with an adequate balance of nutrients, to reduce physiological stress to the plant, is recommended.

Mechanical protection of the fruits on the plants with sacks of paper or plastic was reported as a practice that reduced the incidence of the spots (Eloisa *et al.*, 1994), but still requires complementary research for its recommendation on commercial scale in conditions such as those of Espírito Santo, Brazil. Genetic improvement should also include the selection of plants with resistance to this disorder.

7.3 Bumpy fruit

Bumpy fruit is a physiological disorder common in all papaya-growing regions of the world, and is associated with a deficiency of boron, considered among the micronutrients most limiting to cultivation of papaya (Oliveira, 1999; Wang and Ko, 1975).

The lack of this element induces the plant to produce fruits with bumpy appearance, with the drainage of latex in different points of the skin frequently occurring as a result of the localized deficiency, which paralyzes the growth of the fruit and causes the rupture of the lactiferous vessels. Adjacent unaffected tissue continues to grow, resulting in a misshapen, bumpy appearance (Figure 25).

This symptom however, should not be confused with the drainage of latex caused by "Meleira". The seeds of fruits with symptoms generally are aborted or poorly developed, enabling a darkening of the vessels to be observed. Under severe deficiency

situations, growth of trees may be affected causing a slight rosette effect and an associated stunting in height.

In Hawaii, plants with fruits showing the symptoms of deformation generally present levels of boron in the petioles of the leaves (calculated based on dry weight) lower than 20 ppm, and normal boron level are 25ppm (Nishijima, 1998a). In Brazil, INCAPER-Instituto Capixaba de Pesquisa, Assistência Técnica e Extensão Rural, conducted nutritional studies in papaya fields in the north of Espírito Santo, and defined

Figure 24: Symptoms of freckles or physiological spot in fruits of papaya cv. Sunrise Solo

the standards of macro and micronutrients in the papaya petiole and leaves, with a mean B content in the petiole of 23.10 ppm in the wet season and 25.20 ppm during the dry season, and in the leaf lamina, 42.68 ppm and 44.26 ppm in the wet and in the dry seasons, respectively (Costa, A. N., INCAPER, personal communication). Applications of 0.25% borax ($Na_2B_4O_7.10H_2O$) or a mixture of 0.25% of boric acid (H_2BO_3) with 17% of B in a spray on the foliage have corrected the deficiency of this element and the young fruits discontinue manifestation of the symptoms (Oliveira, 1999; Nishijima, 1998a).

Deformed fruits cannot be brought back to original shape but fruits produced after treatment have normal shape and size.

Applications of boric acid to the soil (approximately 6g) on the area of the projection of the canopy also corrected B deficiency and increased petiole boron levels to prevent of deficiency symptoms on fruits subsequently formed, (however, complementary leaf sprays are also recommended). After application of boron and observation of the disappearance of symptoms in new fruits, a new leaf analysis should be made to

Figure 25: Bumpy symptoms on fruits of papaya cv. Sunrise Solo line 72/12 caused by boron deficiency.

determine the level adequate _for the specific conditions of production and cultivar used, to serve as a reference standard for subsequent analyses that should be conducted periodically.

7.4 Carpellody or Cat-face

Carpellody or "Cat-face" occurs when the flowers develop in an abnormal form as a

result of a transformation of the stamens into carpel-like structures, and the fusion of these to the ovary early in the development of the flower (Marin and Gomes, 2000). After opening, the flowers become distorted producing deformed fruits known as "catface" or carpellody fruits.

Before blooming the carpellodic flowers present a form similar to feminine flowers (with the appearance of a "candle flame"), but formed on the axil of the leaves, which is a characteristic of the hermaphroditic flowers.

The appearance of carpellodic fruits can range from those that resemble female fruits to those that are severely deformed with longitudinal ridges or seams. Carpellodic fruits are generally rounded rather than the more typical pyriform shape and are unmarketable.

It is believed to be an abnormality of genetic origin, and influenced by environmental factors, like low night temperature, elevated moisture, and excess nitrogen in the soil (Marin and Gomes, 2000; Nishijima, 1998a).Since carpellody is an inherited trait, careful seed selection and orchard management can reduce its occurrence. The Solo cultivars have a lower incidence of carpellody. These cultivars, because of many years of inbreeding, have lower incidence of carpellody but it does occur occasionally under conditions described above (Nishijima, 1998a; Arkle and Nakasone, 1984).

7.5 Pentandry

Pentandry is a genetic anomaly known in hermaphroditic plants that may be influenced by the environment, and can occur, occasionally, in plants with masculine flowers. The flower develops five short stamens internally with the filaments inserted in furrows on the wall of the ovary. The fruits develop rounded or oval, very similar to those formed of female flowers, but present five deep, longitudinal grooves that them gives an unmistakable characteristic, lowering sale of the fruits that loose their commercial value (Marin and Gomes, 2000).

8. References

Aloni, B., Karni, L., Rylski, I., Cohen, Y., Fuchs, M., Moreshet, S. and Yao, C. 1998. Cuticular cracking in pepper fruit. I – Effects of night temperature and humidity. Journal Hort. Sci. Biotechnology, 73:743-749.

Alvarez, A.M. and Nishijima, W.T. 1987. Postharvest diseases of papaya. Plant Disease 71 8:.681-686.

Andrade, J.S, Ferreira, A R, Tatagiba, J.S., Ventura, J.A. and Costa, H. 2002. Avaliação da mancha de *Corynespora* em diferentes genótipos de mamoeiro. Fitopatologia Brasileira 27

(supl,): 78-78.

Anonymous, 2002. Diseases of crops: papaya. Disponivel em: <http://www.krishiworld .com/ html.diseases_crops.html>. Access: 30 dez. 2002.

Arauz, L. F. 2000. Mango anthracnosis: economic impactand and current options for integrated management. Plant Disease 84 (6): 600-611.

Arkle,T.D. and Nakasone, H.Y. 1984. Floral differentiation in the hermaphroditic papaya. HortScience 19:832-834.

Bangerth, F.1979. Calcium related physiological disorders of plants. Annual Review Phytopathology 17: 97-102.

Barbosa, C. de J., Meissner Filho, P.E., Habibe, T.C., Patrocicio, E., Ventura, J.A. and Marques, O.J.1998. Distribuição de formas replicativas de vírus em plantas de mamoeiro afetadas pela meleira. In: "Congresso Paulista de Fitopatologia, 21, Botucatu-SP: FCA/GPF, 1998. Anais. Botucatu-SP: FCA/GPF", 1998. p.64.

Bitancourt, A.A. 1935. Mosaico do mamoeiro. O Biológico, 1: 41.

Camarch, R.F.A. and Lima, J.A.A. 1997. Sobrevivência do "Papaya Lethal Yellowing Virus" em solos da rizosfera de plantas infectadas. Fitopatologia brasileira, 22 (supl.): 332-333.

Camarch, R.F.A., Lima, J.A.A., Pio-Ribeiro, G. 1997. Presença de "Papaya Yellowing Virus" em sementes de frutos infectados de mamoeiro, Carica papaya. Fitopatologia brasileira 22 (supl.): 333.

Chan, H. T. , Tam, Y. T. and Seo, S. T. 1981. Papaya polygalacturonase and its role in thermally injured ripening fruit. Journal Food Sci. 46:190-191.

Chau, K.F. and Alvarez, A.M.1983a. A histological study of anthracnose on Carica papaya. Phytopathology 73:1113-1116.

Chau, K. F. and Alvarez, A.M.1983b. Postharvest fruit rot of papaya caused by Stemphylium lycopersici. Plant Disease, 67: 1279-1281.

Cook, A.A. 1975. Diseases of tropical and subtropical fruits and nuts. New York: Hafner Press, 317p.

Conover, R.A., Litz, R.E. and Malo, S.E. 1986. 'Cariflora'- a papaya ringspot virus-tolerant papaya for South Florida and the Caribbean. HortScience, 21: 1072.

Costa, H., Ventura, J. A. and Tatagiba, J. S. 2000. Mosaico do mamoeiro: Uma ameaça à cultura no Espírito Santo. Vitória: INCAPER, 4p. (Documentos n. 69).

Costa, H., Ventura, J. A., Rodrigues, C. H. and Tatagiba, J. S. 2001. Ocorrência e patogenicidade de Glomerella cingulata em mamão no Norte do Estado do Espírito Santo. Fitopatologia Brasileira, 26(supl.): 328-328.

Costa, H., Ventura, J. A. and Tatagiba, J. S. 2002. Severidade da antracnose e podridão peduncular do mamão no estado do Epírito Santo. Fitopatologia Brasileira 27 (supl.) 98.

Couey, H.M. 1989. Heat treatment for control of postharvest diseases and insects pests of fruits. HortScience 24(2): 198-202.

Couey, H.M., Alvarez, A.M. and Nelson, M.G. 1984. Comparison of hot-water spray and immersion treatments for control of postharvest decay of papaya. Plant Disease 68: 436-437.

Davis, M. J., Kramer, J. B., Ferwerda, F. H. and Brunner, B. R. 1996. Association of a bacterium and not a phytoplasma with papaya bunchy top disease. Phytopathology 86: 102-109.

Dickman, M.B., Patil, S.S., Kolattukudy, P.E. 1982. Purification, characterization, and role in infection of an extracellular cutinolitic enzyme from Colletotrichum gloeosporioides Penz. on Carica-papaya, L. Physiology Plant Pathology, 20: 333-347.

Dickman, M.B. and Alvarez, A.M.1983. Latent infection of papaya caused by Colletotrichum gloeosporioides. Plant Disease, 67: 748-750.

Dickman, M.B., Patil, S.S., Kolattukudy, P.E.1983. Effects of organophosphorous pesticides on cutinase activity and infection of papayas by Colletotrichum gloeosporioides. Phytopathology 73: 1209-1214.

Elder, R.J., Macleod, W.N.B., Reid, D.J. and Gillespie, R.L. 2000. Growth and yield of 3 hybrid papaya (*Carica papaya* L.) under mulched and bare ground conditions. Australian Journal of Experimental Agriculture, 40:747-754.

Eloisa, M., Reyes, Q. and Paull, R.E. 1994. Skin freckles on solo papaya fruit. Scientiae Horticulturae, 58: 31-39.

Estrada, A.B., Dodd, J.C., Jeffries, P. 2000. Effect of humidity and temperature on conidial germination and appressorium development of two Philippine isolates of mango anthracnose pathogen Colletotrichum gloeosporioides. Plant Pathology, 49:608-618.

Ferreira, F. 2001. Rainbow and sunup for papaya ringspot virus control: The Hawaiian experience. Phytopathology 91:S168.

Flaishman, M.A. and Kolattukudy, P. E. 1994. Timing of fungal invasion using host's ripening hormone as a signal. Proc. Natt. Acad. Sci. USA, 91: 6579-6583.

Fitch, M., Manshardt, R., Golsalves, D., Slightom, J. and Sanford, J. 1992. Virus resistant papaya plants derived from tissues bombarded with the coat protein gene of papaya ringspot virus. Bio/Technology, 10: 1466-1472.

Funada, C.K., Yorinori, M.A., Rodrigues, A., Auler, A.M., Leite Jr., R. P. and Ueno, B. 1998. Ocorrência da mancha foliar bacteriana causada por *Pseudomonas syringae* pv. *carica papayae* em mamoeiro no Estado do Paraná. Fitopatologia Brasileira, 23: (supl.): 209.

Gibb, K.S., Persley, D.M., Schneider, B. and Thomas J.E. 1996. Phytoplasmas associated with papaya diseases in Australia. Plant Disease, 80: 174-178.

Gibb, K.S., Schneider, R, B. and Padovan, A.C. 1998 Differential detection and genetic relatedness of phytoplasmas in papaya. Plant Pathology, 47: 325-332.

Glennie, J.D. and Chapman, K.R. 1976. A review of dieback - a disorder of the papaw (*Carica papaya* L.) in Queensland. Queensl. J. Agric. Anim. Sci. 33: 177-188.

Gonsalves, D., Ferreira, S., Manshardt, R., Fitch, M. and Slighton, J. 1998. Trasngenic virus resistant papaya: new hope for controlling papaya ringspot virus in Hawaii. St. Paul: APS net, 5p. (http:\\www.scisoc. org/feature /papaya/ Top.html).

Guthrie, J.N., White, D.T., Walsh, K.B. and Scott, P.T. 1998. Epidemiology of phytoplasma – associated papaya diseases in Queensland, Australia. Plant Disease, 82: 1107-1111.

Haque, S.Q. and Parasram, S. 1973. *Empoasca stevensi*, a new vector of bunchy top disease of papaya. Plant Disease Reporter, 57: 412-413.

Holtzmann, O.V. and McSorley, R. 1998. Papaya diseases caused by nematodes. In: "Compendium of tropical fruit diseases" (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.). 2nd. Edition. St. Paul: APS Press, pp. 68-69.

Honda,Y. and Aragaki, M. 1983. Light-dependence for fruiting body formation and its inheritance in *Phoma carica-papayae*. Micologia 75: 22-29.

Holliday, P. 1980. Fungus diseases of tropical crops. Cambridge: Cambridge University Press, 607 p.

Ishii, M. and Holtzmann, O.V. 1963. Papaya mosaic disease in Hawaii. Plant Disease Reporter, 47: 947-95.

Jan, E. E. and Nishijima, K.A. 1990. Identification and attractancy of bacteria associated with *Dacus dorsalis* (Diptera: Tephritidae). Environ, Entomology 19: 1726-1731.

Kaesuk-Yoon, C. 1999. Stalked by deadly virus, papaya lives to breed again. New York: The New York Times. <http://www.purefood.org/ge/papayas.cfm>. July 20.

Kingsland, G.C.1985. Pathogenicity and epidemiology of Corynespora cassiicola in the republic of the Seychelles. Acta Horticulturae, 153: 229- 230, (http://www.actahort.org/ books/153/ 153_31.htm

Kitajima, E,W. 1999. Viroses de fruteiras tropicais no Brasil. Summa Phytopathogica, 25: 78-83.

Kitajima, E. W. , Oliveira, F.C., Pinheiro, C.R.S., Soares, L.M., Pinheiro, K., Madeira, M.C., Chagas, M. 1982a. Amarelo letal do mamoeiro solo no estado do Rio Grande do Norte. Fitopatologia Brasileira, 17: 282-285.

Kitajima, E.W., Rezende, J.A.M., Veja, J., Oliveira, F.C. 1982b. Confirmada identidade do virus isométrico encontrado em mamoais do Rio Grande do Norte como sendo o do amarelo letal do mamoeiro solo. Fitopatologia Brasileira, 17: 336-338.

Kitajima, E.W., Rodrigues, C.H., Silveira, J.A., Ventura, J.A., Aragao, F.J.L. and Oliveira, L.H.R. 1993. Association of isometric virus like particles, restricted to laticifers, with meleira (sticky disease) of papaya (*Carica papaya*). Fitopatologia Brasileira, 18: 118-122.

Ko, W. –H. 1987. Biological control of Phytophthora root rot of papaya with virgin soil. Plant Disease 66: 446-448.

Ko, W. H. 1998. Papaya diseases caused by fungi: Phytophthora fruit rot and root rot. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.), 2nd ed., St. Paul: American Phytopathological Society, pp. 61-62.

Lastra, R. and Quintero, E. 1981. Papaya apical necrosis, a new disease associated with a rhabdovirus. Plant Disease Reporter, 65: 439-440.

Lay-Yee, M., Clare, G. K., Petry, R. J., Fullerton, R. A. and Gunson, A. 1998. Quality and disease incidence of 'Waimanalo Solo' papaya following forced heat treatments. HortScience, 33 (5): 878-880.

Liberato, J.R., Barreto, R.W., Rodrigues, C.H., Costa, H. 1995. Ocorrência de oídio (*Ovulariopsis* sp.) em mamoeiro no Espírito Santo. Fitopatologia Brasileira, 20 (Supl.): 322.

Liberato, J.R., Costa, H. and Ventura, J.A.1996. Índice de Doenças de Plantas do Estado do Espírito Santo. Vitória-ES: EMCAPA, 1996. 110 p.

Liberato, J.R., Tatagiba, J.S., Zambolim, L. and Costa, H. 1999. Fitotoxidez de fungicidas triazoles ao mamoeiro. Fitopatologia Brasileira, 24: 192.

Lima, J. F. de, Oliveira, A. A. R. and Dantas, J. L. L. 2000. Reação de Genótipos de Mamoeiro à inoculação com *Phytophthora palmivora*. In: "Congresso Brasileiro de Fruticultura, Fortaleza-CE: SBF/EMBRAPA,2000, CD-ROM. SBF/EMBRAPA: SBF/EMBRAPA",.

Lima, R.C.A., Lima, J.A.A., Souza Jr., M.T., Pio-Ribeiro, G. and Andrade, G.P. 2001. Etiologia e estratégias de controle de viroses do mamoeiro no Brasil. Fitopatologia Brasileira, 26: 689-702.

Magdalita, P.M., Persley, D.M., Godwin, I.D., Drew, R.A. and Adkins, S.W. 1997. Screening *Carica papaya* x *C. cauliflora* hybrids for resistance to papaya ringspot virus – type P. Plant Pathology, 46: 837-841.

Maffia, L.A., Rodrigues, C.H. and Ventura, J.A. 1993. Significância epidemiológica do conhecimento do arranjo espacial de plantas doentes em campo. I - Meleira do Mamoeiro. Fitopatologia Brasileira, 18 (Supl.): 315.

Martins, D. S., Uramoto, K., Malavasi, A. 1999. Mosca - das - frutas nos Estados brasileiros: Espírito Santo In: "Malavasi, A. Mosca - das - frutas de Importância Econômica no Brasil: Conhecimentos Básicos e Aplicado. Ribeirão Preto": Holos.

Marin, S.L.D. 1998. Toxidade de inseticidas, acaricidas e fungicidas ao mamoeiro cv. Solo. In: "Simpósio Brasileiro Sobre a Cultura do Mamoeiro, 2, Jaboticabal-SP: FCAV/UNESP, 1988. Anais. Jaboticabal-SP: FCAV/UNESP". pp.219-228.

Marin, S. L. D. and Gomes, J. A. 2000. Técnicas de Cultivo do Mamão. Fortaleza-CE: FRUTAL, 57 p.

Melendez, P.L. and Pinero, J.B. 1970.Corynespora leaf spot of papaya (*Carica papaya* L.) in Puerto Rico. Journal of Agriculture of University of Puerto Rico, 54: 411-425.

Nakagawa, J., Takayawa, Y. and Suzukawa, Y. 1987. Exsudação do látex do mamoeiro: estudo da ocorrência em Teixeira de Freitas, BA. In: "Congresso Brasileiro De Fruiticultura, 9, 1987, Campinas-SP: SBF. Anais. Campinas-SP: SBF/UNICAMP", pp. 555-559.

Nelson, M.N. and Alvarez, A.M. 1980. Purple stain of *Carica papaya*. Plant Disease, 64: 93-95, 1980.

Nishijima, K.A., Couey, H.M. and Alvarez, A.M. 1987.Internal yellowing, a bacterial disease of

papaya fruits caused by *Enterobacter cloacae*. Plant Disease 71: 1029-1034.

Nishijima, M.S., Nishijima, W.T., Zee, F., Chia, C.L., Mau, R. F. L. and Evans, D. O. 1989. Papaya ringspot virus (PRV): a serious disease of papaya. Honolulu: HCES/University of Hawaii at Manoa, . 4p. (Commodity fact sheet PA-4, A).

Nishijima, W.T. 1998a. Miscellaneous papaya diseases. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.) 2nd ed., St. Paul: American Phytopathological Society,.pp. 69-70.

Nishijima, W.T. 1998b Papaya diseases caused by fungi: dry rot. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W., Rohrbach, K.G., Ohr, H.D.) 2nd ed., St. Paul: American Phytopathological Society, pp. 59-60.

Nishijima , W.T. 1998c. Papaya diseases caused by fungi: wet fruit rot. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.) 2nd ed., St. Paul: American Phytopathological Society, pp.64.

Nishijima, W.T. 1998d. Papaya diseases caused by bacteria. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.)2nd ed., St. Paul: American Phytopathological Society, pp.65.

Oliveira, A.M.G. 1999. Boro: um micronutriente importante para o mamoeiro. Cruz das Almas-BA: EMBRAPA/CNPMF, 2p. (Mamão em Foco, 6).

Oluma, H.O.A. and Amuta, E.U. 1999. *Corynespora cassiicola* leaf spot of papaw (*Carica papaya* L.) in Nigeria. Mycopathologia, 145: 23-27.

Pernezny, K. and Litz, R.E. 1993. Some Common Diseases of Papaya in Florida. Gainesville: UF/IFAS.

Prusky, D. 1996. Pathogen quiescence in postharvest diseases. Ann. Rev. Phytopathology 34: 413-434, 1996.

Punithalingam, E. A. A. 1980. New combination in *Phoma* for *Ascochyta carica-papayae*. Trans. Brit. Mycology Soc. 75: 340, 1980.

Queiroz, F.M., Muniz, M. de F.S. and Menezes, M. 1997. Podridão da haste do mamoeiro 'Sunrise Solo' causada por *Botryodiplodia theobromae* no Estado de Alagoas. Summa Phytopathologica, 23(1): 44-45.

Quimio, T.H. 1973. Temperatura as a factor for growth and sporulation of anthrracnose organism of papaya. Philippine Agriculturist, 57: 245-253.

Quimio, T.H. 1976. Pathogenicity and cultural characteristics of *Fusarium solani* from papaya. Kalikasan Philippine J. of Biol. 5:241-250.

Ram, A., 1984. Forma perfeita de *Colletotrichum gloeosporioides* em frutos e folhas de mamoeiro na Bahia. Fitopatologia Brasileira, 9: 319.

Ram, A., Oliveira, M.L. de and Sacramento, C.K. 1983. Podridão das raízes do mamoeiro associada com *Pythium* sp. na Bahia. Fitopatologia Brasileira 10 (2): 581.

Ramirez, L., Duran. A. and Mora, D. 1998. Combate integrado de la podricion radical de la papaya (*Phytophthora* sp.) a nivel de vivero. Agronomia Mesoamericana, 9(1): 72-80.

Rezende, J.A.M. and Costa, A.S. 1993. Doenças de vírus e micoplasmas do mamoeiro. Summa Phytopathologica, 19(2): 73-79.

Robbs, C. F. 1956. Uma nova doença bacteriana do mamoeiro. Revista da Sociedade Brasileira de Agronomia 12: 73-76.

Rodrigues, C.H., Ventura, J.A. and Marin, S.L.D. 1989a Ocorrência e sintomas da meleira do mamoeiro (*C. papaya*) no Estado do Espírito Santo. Fitopatologia Brasileira, 14 (Supl.): 118.

Rodrigues, C.H., Ventura, J.A. and Maffia, L.A. 1989b. Distribuição e transmissão da meleira em pomares de mamão no Estado do Espírito Santo. Fitopatologia Brasileira, 14: (Supl.): 118.

Rodrigues, L., Ventura, J.A. and Costa, H. 2001. Podridão seca em frutos de mamão em condições de campo no Norte do Espírito Santo. Fitopatologia Brasileira, 26 (supl.): 406-407.

Sanchez, M., Dianese, J.C. and Costa, C.L. 1991. Fatores determinantes do dano de *Phoma caricae-papayae* ao fruto do mamoeiro (*Carica papaya*) e detecção de resistência ao fungo em

Carica gaudotiana. Fitopatologia Brasileira 16 (1): 121-129.

Saxena, R.M. and Sharma, K.D. 1981. Deterioration of papaya fruits by fungi. Agricultural Science Dig. 1: 140-142.

Seín, F. and Adsuar, J. 1947. Transmission of the bunchy top disease of papaya (*Carica papaya* L.) by the leaf hopper *Empoasca papayae* Oman. Science, 106 (27450): 130.

Siddique, A. B. M. , Guthrie, J. N., Walsh, K. B., White, D. T. and Scott, P. T. 1998. Histopathology and Within-Plant Distribution of the Phytoplasma Associated with Australian Papaya Dieback. Plant Disease 82:1112-1120.

Silva, A.M.R., Kitajima, E.W. and Resnde, R.O. 2000. Nucleotide and amino acid analysis of the polymerase and the coat protein genes of the papaya lethal yellowing virus. Virus Review and Research, 11: 196.

Silva, G. S. and Souza, I. M. R.1999. Mancha de *Corynespora* em trapoeraba *(Commelina benghalensis)*. Fitopatologia Brasileira, 24 (Supl.): 328-329.

Simone, G. W. 2002. Disease Control in Papaya (*Carica papaya*). Gainesville: UF/IFAS, 2002. (Plant Disease Management Guide, v. 3, Department of Plant Pathology, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida, <http://edis.ifas.ufl.edu>).

Singh, A.L. and Singh, G. 1998. Effect of time of planting on growth fruiting behaviour and sex relation of papaya (carica papaya). Indian Journal of Agricultural Science 68(12): 769-772.

Solano, V. and Arauz, L.F. 1995. Combate de antracnosis em frutos de papaya mediante aplicaciones de fungicidas em el campo em la zona atlantica de Costa Rica. Agronomia Costarricense, 19(2): 25-30.

Souza Jr., M.T. 2000. Mamão transgênico: uso da engenharia genética para obter resistência ao vírus da mancha anelar. Biotecnologia Ciência e Desenvolvimento, 2(3): 132-137.

Souza Jr., M.T. and Gonsalves, D. 1999. Genetic engineering resistance to plant virus disease, an effort to control *Papaya ringspot virus* in Brazil. Fitopatologia Brasileira, 24: 485-502.

Souza Jr., M.T., Tavares, E.T., Maciel-Zambolim, E. and Ventura, J.A. 2002. Studies aiming molecular characterization, early diagnosis, and control of papaya stick disease. Virus Reviews and Research, 7: 43.

Story, G.E. and Halliwell, R.S. 1969. Association of a mycoplasma-like organism with bunchy top disease of papaya. Phytopathology, 59: 1336-1337.

Swain, S. and Powell, D.A. 2001. Papaya Ringspot Virus Resistant Papaya: A Case Study. Technical report. Disponible: http://www.plant.uoguelph.ca/safefood/gmo/papayarep .htm.

Tatagiba, J.S., Liberato, J.R., Ventura, J.A. and Mendonca, L.F. 1997. Eficiência de fungicidas no controle da antracnose e da podridão peduncular em pós-colheita em frutos de mamão. Fitopatologia Brasileira, 22 (Supl.): 314.

Tatagiba, J.S., Costa, A.N., Ventura, J.A. and Costa, H. 1998a. Efeito do boro e cálcio na incidência da antracnose em frutos de mamoeiro. Fitopatologia Brasileira, 23 (Supl.): 285-286.

Tatagiba, J.S., Ventura, J.A., Liberato, J.R., Mendonca, L.F. and Costa, H. 1998b. Chemical control of Papaya Powdery Mildew. In: "International Congress of Plant Pathology, 7, Edinburgh, Scotland: ISPP. 1998. Abstracts. Vol.3. Edinburgh, Scotland: ISPP", Abstract 4.9.17.

Tatagiba, J.S., Liberato, J.R., Zambolim, L., Costa, H., Ventura, J.A. 1999. Avaliação de fungicidas no controle da antracnose e da podridão peduncular do mamoeiro. Summa Phytopathologica, 24: 57.

Tatagiba, J. S., Silva, J. G. F. , Costa, H., Ventura, J. A. 2001. Influência da irrigação na incidência da antracnose em frutos de mamão. Fitopatologia Brasileira, 26 (supl.): 329.

Tatagiba, J.S., Liberato, J.R., Zambolim, L., Ventura, J.A., Costa, H. 2002. Controle e condições climáticas favoráveis a antracnose do mamoeiro. Fitopatologia Brasileira, 27(2): 186-192.

Tennant, P.F., Gonsalves, C., Ling, K.S., Fitch, M.M.M., Manshardt, R., Slinghtom, J.L.and

Gonsalves, D. 1994. Differential protection against papaya ringspot virus isolates in coat protein gene transgenic papaya and classically cross-protected papaya. Phytopathology, 84(11): 1359-1366.

Vallejo, G. G. 1999. Efectos de la fertilizacion com nitrogeno em la produccion de papaya (*Carica papaya* L.) y en la incidencia de virosis. Rev. Fac. Nal. Agric. Medellin, 52: 515-526.

Ventura, J.A. 1995. Controle de doenças em pós-colheita de frutos tropicais. Fitopatologia brasileira 20(supl.): 273.

Ventura, J. A. 2000. Taxonomia de *Fusarium* e seus segregados. Parte II- Chaves para identificação. Revisão Anual de Patologia de Plantas, 8: 303-338.

Ventura, J.A. and Santos, A.F. 1981. dos Podridão interna em frutos de mamoeiro (*Carica papaya* L.) no Estado do Espírito Santo. In: "Congresso Brasileiro de Fitopatologia, 14, Porto Alegre-RS, 1981. Programa e resumos. Porto Alegre-RS: SBF/UFRS", 1981.

Ventura, J.A. and Balbino, J.M.S. 1995. Resistência do agente etiológico da antracnose do mamoeiro ao Benomil, no Estado do Espírito Santo. Fitopatologia Brasileira, 20 (Supl.): 308.

Ventura, J. A., Costa, H., Rodrigues, C. H., Prates, R.S. and Cardoso, A. A. B. 2001a. Meleira do Mamoeiro: uma ameaça à cultura. Vitória: INCAPER, 4p. (Folder).

Ventura, J. A., Costa, H. and Tatagiba, J. S. 2001b. Sintomatologia da meleira do mamoeiro e sua importância para o "roguing". Fitopatologia Brasileira, 26(supl): 536-536.

Ventura, J. A. and Costa, H. 2002. Manejo integrado das doenças de fruteiras tropicais: abacaxi, banana e mamão. In: "ZAMBOLIM, Laercio. (Org.). Manejo integrado de doenças e pragas: fruteiras tropicais. Viçosa-MG", pp.279-352.

Vidal, C.A. ,Nascimento, A.S., Barbosa, C.J., Marques, O.M. and Habibe, T.C. 2000. Experimental transmission of "sticky disease" of papaya by *Bemisia argentifolii* Bellows & Perring – In: "International Congress of Entomology, 21, Foz do Iguaçu-PR: SEB/EMBRAPA, 2000. Abstract book 2, Foz do Iguaçu-PR: SEB/EMBRAPA", p. 819 (abstract).

Wang, D.N. and Ko, W.H.1975. Relationship between deformed fruit diseases of papaya and boron deficiency. Phytopathology 65: 445-447.

Wang, S.-H. and Conover, R.A. 1983. Incidence and distribution of papaya viruses in southern Florida. Plant Disease, 67: 353-356.

Webb, R.R. and Davis, M.J. 1987. Unreliability of latex-flow test for diagnosis of bunchy-top of papaya caused by a mycoplasma like organism. Plant disease, 71: 192.

White, D. T., Billington, S.J., Walsh, K. B. and Scott, P. T. 1997. DNA sequence analysis supports the association of phytoplasmas with papaya (Carica papaya) dieback, yellow crinkle and mosaic. Australasian Plant Pathology 26: 28-36.

Yagushi,Y, Uehara, K., Nojima, H. , Kobayashi, T. and Nakamura, S. 1998. Occurrence of a new anthracnose in Japan, caused by *Colletotrichum capsici.* Edinburgh, Scotland: ISPP/ICPP, 1998. Abstract 6.11.

Yeh, S.H., Jan, F.J., Chiang, C.H., Doong, T.J., Chen, M.C., Chung, P.H. and Bau, H.J. 1992. Complete nucleotide sequence and genetic organization of papaya ringspot virus RNA. Journal Gen. Virolgy 73: 2531-2541.

Zambolim, L. and Ventura, J.A. 1993. Resistência a doenças induzida pela nutrição mineral das plantas. Revista Anual de Patologia de Plantas, 1: 275-318.

Zambolim, E.M., Barros, D.R., Matsuoka, K., Kunieda, S., Carvalho, M.G. and Zerbini, F.M. 2000. Purification and partial characterization of papaya "Meleira" virus. Fitopatologia brasileira, 25 (supl.): 442.

Zettler, F.W. and Wang, S.H.1998. Papaya diseases caused by viruses: papaya droopy necrosis and papaya apical necrosis. In: "Compendium of tropical fruit diseases", (eds. Ploetz, R.C., Zentmeyer, G.A., Nishijima, W.T., Rohrbach, K.G., Ohr, H.D.) 2nd ed., St. Paul: American Phytopathological Society, 1998. pp.66-67.

Virus and Phytoplasma Diseases of Passion Fruit

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Abstract: Commercial *Passiflora* species are vigorous woody perennial vines. The edible commercial species of passion fruit are probably originated on the edges of South American rainforests in the Amazon region of Brazil and possibly in Paraguay and northern Argentina. The purple passion fruit crops best at higher altitudes above 2000 m in the tropics while the golden passion fruit and hybrids are superior in the low lands.Several virus and virus- like diseases have been reported on passion fruit world- wide. These include, passion fruit woodiness virus, ring spot virus, latent carlavirus, mosaic virus, chlorotic spot, yellow mosaic tymovirus, purple granadilla mosaic virus, vein clearing rhabdovirus, tomato ringspot virus, maracuja mosaic tobamovirus and passion fruit mottle potyvirus etc. A phytoplasma disease causing witches' broom disease has also been reported. Diagnosis and management of these diseases of passion fruit are discussed in this chapter.

1. Introduction

The edible commercial species of passion fruit originated on the edges of South American rainforests in the Amazon region of Brazil and possibly in Paraguay and northern Argentina. The purple passion fruit (*Passiflora edulis* Sims.) is adopted to the cooler subtropics or at high altitudes in the tropics, while the golden passion fruit (*P. edulis* f. *flavicarpa*) is more suited to tropical and low conditions. Brazil, Venezuela, South Africa, Sri Lanka, Australia, Papua New Guinea, Fiji, Hawaii, Taiwan and Keniya account for 80-90% of the worlds passion fruit production. Other countries where passion fruits are grown commercially are New Zealand, West Samoa, Cook Islands, Malaysia, Israel, Congo, Angola, Ivory Coast, Cameroon, Peru, Colombia, and West Indies.

The family Passifloraceae includes 550 species in twelve genera and is represented by more fruits species than probably any other plant family. The most important genus *Passiflora* has about 400 species which are mostly native to tropical America but include about 40 in Asia, Australia and the South Pacific. Killip (1938) has studied the classification of Passiflora in detail and their commercial significance reviewed by Morton (1967), Martin and Nakasone (1970) and Howell (1976).

Of the 60 species of *Passiflora* which bear edible fruit, only about 10 are well known outside limited areas where they grow wild or are cultivated commercially. Tropical American *Passiflora* species which are utilized as fruiting plant or as sources of cold/ disease tolerance in breeding programmes are *P. quadrangularis, P. lingularis, P. laurifolia, P. maliformis, P. mollissima, P. antiguiensis, P. incarnata* and *P. caerulea.* In addition, there are some minor tropical *Passiflora* species cultivated for their edible

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fruit. These include: *P. alata, P. coccinea, P. mixta, P. popenovii, P. semanni* and *P. serrato-digitata* (Menzel *et al.,* 1990). Commercial *Passiflora* species are vigorous woody perennial vines with medium to large leaves (8-30 cm. long) with toothed margin and usually bearing glands on the petioles (Bailey, 1949, Purseglove, 1974). The purple passion fruit crops best at higher altitudes above 2000 m in the tropics while the golden passion fruit and hybrids between the two forms are superior in the low lands.

Several virus and virus- like diseases have been reported on passion fruit worldwide (Brunt *et al.,* 1990). These include, passion fruit woodiness virus, passion fruit ring spot virus, passion fruit latent carlavirus passion fruit mosaic virus, passion fruit chlorotic spot, passion fruit yellow mosaic tymovirus, purple granadilla mosaic virus, passion fruit vein clearing rhabdovirus, tomato ringspot virus, maracuja mosaic tobamovirus and passion fruit mottle potyvirus.

A potyvirus associated with mosaic of passion fruit has been reported from Puerto Rico and the Dominican Republic (Escudero *et al.,*1988). Seneviratne and Wickramasingha (1973, 1974) reported passion fruit crumple and passion fruit yellow blotch diseases from Sri Lanka. Cucumber mosaic virus on passion fruit has been reported from Taiwan (Chang *et al.,*1981). Passiflora leaf mottle disease caused by a whitefly- transmitted geminivirus has been recorded in Puerto Rico (Brown *et al.,*1993).

A phytoplasma disease causing witches' broom disease has been reported from Brazil (Kitajima *et al.,*1981,1986). Details of these diseases have been given in this chapter.

2. Virus diseases

2.1 Passion fruit woodiness virus (Potyvirus)

2.1.1 History

According to Cobb (1901) the woodiness disease of passion fruit was known in New South Wales (Australia) at least as early as 1891. He reported that the disease appeared to a serious extent about 1897, however, its identification was confirmed from 1953 (Talor and Greber, 1973). The epiphytotic incidence of the virus within plantations reported by Cobb over 100 years ago is of considerable interest. Since passion fruit woodiness virus (PWV) and cucumber mosaic virus (CMV) causes woodiness symptoms, the authenticity of records prior to that of McKnight (1953) is in doubt.

The PWV was rare in *Passiflora edulis* in Queensland in 1927 and widespread in 1932 (Simmonds, 1959) suggesting that it was introduced in to that state about 1927. Experimentally, a mixed infection by the two viruses (PWV and CMV) in *P. edulis* causes a very severe disease (Taylor, 1959, Taylor and Kimble, 1964) but naturally occurring mixed infections are not always so virulent. Details of woodiness virus disease has been given by Taylor and Greber (1973). Chagas *et al.* (1981) and Bezerra *et al.* (1995) reported a severe disease in golden passion fruit in the state of Bahia (Brazil) caused by an isolate of PWV.

2.1.2 Geographical distribution

The disease is wide spread and has been reported from various countries. Australia (McKnight, 1953, Greber, 1966), Brazil (Chagas *et al.,* 1981, Bezerra *et al.,* 1995, Trindade *et al.*, 1999), South Africa (Storey, 1940, Malan, 1957, Kuhne, 1968, Da Graca, 1976), Italy (Quacquarelli and Martelli, 1969), Kenya (Nattrass, 1944), Japan (Hirata and Kono, 1965), Malaysia (Ong and Ting, 1973), Nigeria (Martini, 1962), Sri Lanka (Senanayake, 1972).

In Brazil, this disease was initially reported by Yamashira and Chagas (1979) causing very serious losses in Bahia state, being identified and characterized as PWV in 1981 by Chagas *et al.* After this initial outbreak in Bahia, PWV was later reported in Pernambuco, Ceara and Sao Paulo states (Loreto and Vital, 1983, Lima *et al.* 1993, Bezerra *et al.* 1995, Inoue *et al.* 1995).

2.1.3 Symptomatology

There is a great variation in the symptoms produced by the woodiness virus and in addition there is seasonal variation in the expression of individual type of symptoms.

Thirteen types of symptoms have been reported (McKnight, 1953). These include malformed fruit, typical foliage mottle, yellow spot mottle, terminal bunchiness, translucent areas, foliage ringspot, fruit ringspot, stippled fruit, fern leaves, tip chlorosis, stem mottle, vein hypertrophy and seedling terminal reaction. The first 11 symptoms occur in field and last 2 in greenhouse.

Woodiness in fruit is detectable by their uneven shape when the fruit measure only 1/4 inch to1/2 inch along their longest dimension. A mottle on the calyx accompanies the fruit symptoms.

2.1.3.1 Typical foliage mottle

This is a broad bullet mottle with dark green area localized on the raised or blistered portions of the leaf. The blisters are raised from the top surface of the leaf. The leaf colour between the dark green, raised area is yellow green. The leaves present a puckered appearance and are frequently misshapen. This mottle can be readily detected from March until November (in Australia) but its expression is most marked during winter and spring months. A fleeting mottle can be seen in the summer months just behind the terminals.

The yellow spot mottle may be associated with the typical mottle. Occasionally a leaf may be seen with both of these mottles present but with each distinctly confined to definite areas of the leaf.

2.1.3.2 Yellow spot mottle

This mottle is produced by numerous, small, yellow spots on the leaf. Occasionally the spot may be few in number, discrete, and occur unmixed with any other leaf mottle. The yellow spots are generally circular in shape, but when large, are irregular in out-

line and occasionally have a necrotic centre. This mottle occurs on the vines from April to November. It is commonly associated with the typical mottle and may also occur on fern leaves. It is best expressed on older leaves.

2.1.3.3 Terminal bunchiness

This symptom is lack of growth at the terminal accompanied by a bunchiness of harsh-textured terminal leaves. It may be detected on systemically infected vines in any month of the year but is best expressed in winters, when the terminal leaves are markedly down curled along the axis of the main vine.

2.1.3.4 Translucent areas

Infected vines show a vein clearing in the terminal leaves accompanied by translucent or cleared areas.

2.1.3.5 Foliage ringspot

It is a relatively rare symptom. Ringspots are associated with the yellow spot mottle and are caused, apparently, by the rearrangement of the yellow spot mottle into rings. Tissue bounded by the rings is green and does not become necrotic.

2.1.3.6 Fruit ringspot

Ringspot may occur on normal fruit or on fruit that are misshapen and have hardened and thickened pericarps. They may occur as circular, green, discrete ringspot with a purple cluster, up to 3/4 cm. in diameter, and as small as 3 mm. in diameter. They may fuse during development to give large, irregularly shaped outlines. They can occur on all portions of the fruit but may be confused to one half of it. Frequently their occurrence is more intense on the side of the fruit.

As fruit ripen and shriveled the ringspots gradually become obscure. When ringspots occur in fruit is still green, their center remains a "greasy green" as the fruit ripens in place of the purple in a ripen fruit. In this case, as the fruit ripens, the ringspots again become obscure and the final results in the presence of a number of large or smaller grey-green dots or spots on the normal green of the fruit.

2.1.3.7 Stippled fruit

Marking occur on the fruit in the form of an irregular dotted and stippled portions below the epidermis. On green fruit the pattern appears, in contrast as a gray colour. When they are small, the swelling may be roughly circular but they are generally fused to give an irregular pattern.

2.1.3.8 Fern leaves

Fern leaves are distorted leaves, the individual leaf lobes being filiform and having

irregular or serrated margins. In the field and in the greenhouse the typical woodiness mottle is not found associated with fern leaves, but yellow spot mottle may be associated with this symptom.

2.1.3.9 Tip chlorosis

The terminal leaves are chlorotic, with a trace of green vein banding, associated with tip chlorosis in the stem mottle described below. Small, brown, necrotic, slightly depressed lesions appear on the stem near the terminal and the terminal leaves and tendrils die-back. Tip chlorosis is present in the field during the months of August to October.

2.1.3.10 Stem mottle

A light-green mottle may occur on the stems and petioles of infected plants and is prominent in the field in September to October.

2.1.3.11 Vein hypertrophy

This consists of a raised main vein, which commonly pursues a zigzag path.

2.1.3.12 Seedling terminal reaction

This is the first symptom in greenhouse by seedling passion vine after inoculation with the woodiness virus. The terminal reactions consists of a marked downward curling of the first unfolded terminal leaflet, followed by spiral twisting and cupping under the leaf edges. Vein clearing occurs and translucent areas appear. All these symptoms may also appear in the 2nd unfolded leaflet, while in the 3rd unfolded leaflet, marginal waving is commonly the only symptom present.

2.1.4   Transmission

The virus is easily transmitted by mechanical sap inoculation and through grafting to passion fruit and other hosts. The virus is also transmitted from the contaminated blades of pruning knife. However, no seed transmission has been reported.

2.1.4.1 Insect vector

Different species of aphids transmit this virus in a non-persistent manner namely, *Myzus persicae* (Noble and Noble, 1939, Taylor, 1959, DaGraca, 1976), *Aphis gossypii* (Magee, 1948, Greber, 1966) and *Aphis fabae* (Bezerra *et al.,* 1995).

2.1.5   Host range

Passiflora edulis, P. foetida, P. alba, P. suberosa, P. casenules.The PWV can be trans-

mitted to *Phaseolus vulgaris* var. Bountiful and from this host back to passion fruit and 25 other varieties of beans (Taylor and Kimble, 1964). The virus also occurs naturally in some tropical legumes, including *Arachis hypogea, Centrosema pubescens, Crotolaria usaramoensis, Glycine max* and *Phaseolus atropurpureus,* but is of minor importance in these species.

The virus infects 44 dicotyledonous plant species in five families (Taylor and Kimble, 1964, Teakle and Wildermuth, 1967). Hosts include 10 species of *Passiflora* and 18 in the Leguminosae. Bezerra *et al.* (1995) reported that PWV was able to infect 22 dicotyledonous plant species, the majority belonging to family Leguminosae.

2.1.6 Diagnostic species

Passiflora edulis (Passion fruit) shows mosaic, rugocity and distortion with yellow spots on older leaves. The woody fruit symptoms may also be caused by insect feeding and is of limited diagnostic value. *Passiflora edulis* f. *flavicarpa* (golden passion fruit) and *P. edulis* x *P. edulis* f. *flavicarpa* (hybrid passion fruit). Paled green to chlorotic ringspots occur on spring growth together with leaf mosaic.

Phaseolus lathyroides when inoculated, leaves show chlorotic to necrotic local lesions, followed by systemic infection with leaf epinasty, vein necrosis, leaf abscission and terminal necrosis. The symptoms on bean (*Phaseolus vulgaris* (french bean) varies with the variety infected and the strain of PWV. Inoculated primary leaves of some varieties (*e.g.* Bountiful and White wax) develop mild chlorotic spots, but in others (*e.g.* Burpee's stringless Green pod, Morsepole No. 191, Sutter Pink and San Benito Pink) develop into a spreading vein necrosis. Systemic symptoms varies from vague mottling to severe blister mottling. The varieties Sutter Pink and San Benito Pink develop necrosis and mottling of the pods.

The symptoms on *Sesbania exaltata* consists of necrotic local lesions on the inoculated primary leaves. Da Graca (1976) reported that PWV produces mosaic symptoms in mechanically inoculated cowpea. *Chenopodium amaranticolor* and *C. quinoa* produce local lesions 4-5 days after inoculation (De Wijs, 1974b, Martini, 1962, Da Graca, 1976). *Vigna unguiculata* show mosaic pattern 14 days after inoculation (Da Graca, 1976). *Petunia hybrida* is a symptomless carrier of the PWV.

2.1.7 Propagation species

P. edulis may be used to maintain all except the tip blight strain which may be maintained in the more tolerant host *P. suberosa, Phaseolus vulgaris* cv. Bountiful is a good source of virus for purification. Macroptilium lathyroides is best host for maintenance and propagation of the virus for purification (Bezerra *et al.*, 1995).

2.1.8 Assay species

Small countable, rusty-red ringspots are produced on inoculated primary leaves of *Dolicus biflorus*. Chlorotic lesions which may be counted with difficulty are produced in *Phaseolus vulgaris* cv. Bountiful. Very small seedlings of *Phaseolus edulis* are

useful for testing transmission by vectors.

2.1.9 Strains

Several strains can be distinguished by severity of symptoms in *P. edulis*. Greber (1966) described a strain which is mild in *Passiflora suberosa* but causes blight and complete loss of crop in *P. edulis* . Mild strains effectively cross protect against virulent strains in *P. edulis*.

2.1.10 Physical properties of the virus

In *Phaseolus vulgaris* sap, the thermal inactivation point (10min.) is 55-60°, dilution end point 10^{-4} to 10^{-5} and inactivation *in vitro* occurs in 3-4 days at 18°C.

2.1.11 Serology

Strongly in immunogenic. Antisera with titres up to 1/1024 were obtained by Taylor and Kimble (1964) by subcutaneous injections of virus in freund's adjuvant at several sites followed 10 days later by an intravenous injections. Serological precipitates in mixed liquids occur in a few min. and are of the flocculent type. Brazilian PWV isolates are closely related serologically to Canavalia acronecrosis virus. PWV is also distantly related serologically to Bean common mosaic virus, Black eye cowpea mosaic virus, carnation vein mottle virus, Cassia yellow spot, Cowpea green vein banding , Cowpea rugose mosaic, Papaya ringspot virus-W, Soybean mosaic virus, Watermelon mosaic virus-2 and Wisteria vein mosaic viruses (Lovisolo and Kitajima, 1992).

2.1.12 Relationships

All strains tested appear to be closely related serologically. The virus is similar to bean yellow mosaic virus in shape, physical properties and symptoms in *Phaseolus vulgaris,* it does not appear to be serologically related to this virus or to the pea mosaic strain of it (Taylor and Kimble, 1964, Teakle and Wildermuth, 1967). Its biological and physical properties relate it in the potyvirus group. Bezerra *et. al.* (1995) reported that PWV is serologically related to cowpea aphid borne mosaic, clitoria mosaic and siratro mosaic potyviruses.

2.1.13 Particle morphology

Particles are flexuous filaments about 12nm wide. In partially purified preparations, Taylor and Kimble (1964) found a normal length of 670nm, whereas Teakle and Wildermuth (1967) reported a figure of 745nm. The virus particles have molecular wt. of 36k. A smaller protein of 29k was also detected in minor concentration which probably corresponds to an enzymatic degradation product of the capsidial protein of 36k (Bezerra *et al.,* 1995 ,Inoue *et al.* 1995).

2.1.14 Relations with cells and tissues

Pinwheel inclusions and particles occur frequently in the mesophyll and vascular pa-
renchyma of *Phaseolus vulgaris*. Disorganization of chloroplasts in parenchyma cells
of infected plants has been observed (Inoue *et al.,* 1995). They do not contain virions.

2.1.15 Etiology

Passion fruit woodiness disease is caused by virus belonging to the Potyvirus
(Potyviridae) and has been named as "Passion fruit woodiness virus" (PWV) (Taylor
and Kimble, 1964). Bezerra *et al.* (1995) reported that PWV is serologically related to
cowpea aphid- borne mosaic, clitoria mosaic and sirato mosaic potyviruses.

     A woodiness disease of passion-fruit was described by Cobb (1901) but as both
passion fruit woodiness and cucumber mosaic viruses may cause woody fruit and leaf
mosaic (Taylor, 1959), the authenticity of records prior to that of McKnight (1953) is
in doubt. However, passion fruit woodiness virus was rare in *P. edulis* in Queensland
in 1927 and widespread in 1932 (Simmonds, 1959) suggesting that it was introduced
into that state about 1927. Its absence from cool, temperate areas of Australia and the
rare occurrence of cucumber mosaic virus in Queensland suggest that ecological fac-
tors influence the distribution of both viruses. Experimentally, a mixed infection by
the 2 viruses in *P. edulis* causes a very severe disease (Taylor, 1959), but naturally
occurring mixed infections are not always so virulent.

     To differentiate the 2 viruses, it is best to use the electron microscope. The
filamentous particles of passion fruit woodiness virus are usually readily found in the
sap of infected plants (Bezerra *et a1.,*1995, Inoue *et al.,*1995). The diagnosis may then
the confirmed by transmissions to diagnostic hosts. Negative results suggest the pres-
ence of cucumber mosaic virus which, except in mixed infection with passion fruit
woodiness virus, exists in very low concentration in seedlings of *Passiflora edulis* and
is extremely difficult to transmit to diagnostic host by aphid or sap inoculation. How-
ever, when *Passiflora edulis* scions infected with cucumber mosaic virus are grafted
onto *Passiflora caerulea* stock the scions develop a severe necrotic disease and often
ultimately die. The virus reaches a high enough concentration in both stock and scion
for transfers to be made from either to diagnostic hosts.

     Passion-fruit woodiness virus could be confused with passion fruit latent virus
(Schnepf and Brandes, 1962), which has slightly flexuous particles about 650nm long,
unless care is taken to check particle shape and length. Mixed infections of the two
viruses have not been reported.

     A virus reported in passion fruit in Nigeria (Martini, 1962) may be related but
serological evidence is not available.

2.1.16 Detection

The virus can be detected serologically by agar- gel diffusion test, ELISA and Western
blot.

2.1.17 Properties of the virus

The size of the coat protein is estimated as 33 k Da to 36 k Da (Inoue *et al.* ,1995, Bezerra *et al.*,1995). Molecular wt. estimates on SDS- PAGE suggest that PWV coat protein contains 275 amino acid residues. Sequence homology between the three strains of PWV, tip blight (PWV-TB), severe (PWV-S) and mild (PWV-M) and published sequences for the coat protein of 8 distinct potyviruses range from 43- 71 % (av. 57 %) (Shukla *et al.*, 1988). Gough and Shukla (1992) reported that the deduced coat protein sequence of K strain of PWV potyvirus, isolated from infected hybrid passion fruits in Queensland , Australia, differed significantly , particularly in the terminus, from the sequences of TB, M and S strains of PWV. An antiserum that reacted strongly with the TB, M and S strains reacted poorly with the K strain.

2.1.18 Management

The use of mild strain to protect plants from more virulent ones in the field conditions was suggested by McKnight (1953) and applied by Simmonds (1959), it appears to be one of the few instances of this method of control having been commercially useful. Partial control has been obtained by the use of the hybrid passion fruit which, like its *P. edulis* f. *flavicarpa* parent, produces distorted fruit only under adverse conditions or when infected with an unusually virulent strain of the virus.

2.2 Passion fruit woodiness (Cucumovirus)

2.2.1 History

Magee (1948) reported that passion fruit "woodiness" was caused by cucumber mosaic virus (CMV) in New South Wales. McKnight (1953) found that the "woodiness" disease is caused by two different viruses *i.e.* CMV and PWV. Subsequently the CMV was isolated from passion fruit showing woodiness symptoms by different workers (Hollings, 1956, Teakle *et al.,* 1963, Nattrass, 1939,1940,1944, Taylor, 1959, Taylor and Kimble, 1964, Yonaha *et al.,* 1979, Colariccio *et al.*, 1987).

2.2.2 Geographical distribution

Australia (Magee, 1948, Taylor, 1959, Taylor and Kimble, 1964, Taylor and Greber, 1973), Kenya (Nattrass, 1939,1940,1944, Martini, 1962), England (Hollings, 1956), California, USA (Teakle *et al.,* 1963), Japan (Yonaha *et al.,* 1979), Brazil (Colariccio *et al.*, 1987, Barbosa *et al.*, 1999).

2.2.3 Symptomatology

Infected plants exhibit mosaic and striking coalescent yellow rongs on the leaves with occasional slight foliar deformation (Colariccio *et al.*, 1987).

2.2.4 Transmission

The virus is easily sap transmitted from *P. edulis* to *P. edulis* and other species of *Passiflora*. Several species of aphids have been reported to transmit CMV strain of passion fruit woodiness virus. Among them, *Aphis gossypii* and *Myzus persicae* are important ones.

2.2.5 Physical properties

The thermal inactivation point of the virus lies between 60°-65°C, the dilution end point is 1:10,000 and the longevity *in vitro* is between 15 and 17 days at room temperature. The virus is destroyed by drying.

2.2.6 Host range

The virus infects a wide range of host plants (Smith, 1972). CMV was readily transmitted from *P. caerulea* seedlings by aphids, mechanical and graft inoculations but was transmitted only rarely from *P. edulis* seedlings (Taylor and Kimble, 1964).

The virus infects *Spinacia oleracea* L., *Nicotiana glutinosa* (L.) Black., *Cucumis sativus* L. var. Marketur, and *Datura stramonium* L. characteristic of CMV. Differential hosts are *Phaseolus lathyroides* L. and *Dolichos biflorus* L. The host range of passiflora strain of CMV is wide and includes *Gomphrena globosa* L. which reacts with a symptomless local infection, *Chenopodium album* L. and *C. amaranticolor* with chlorotic local lesions. Several species of leguminosae are susceptible, *Medicago scutellata* Mill. and *Trigonium suberraneum* L. react with a systemic yellow vein banding. In addition to passion fruit, three other species of *Passiflora* are susceptible, as well as *N. clevelandii*, *N. tabacum* and *Petunia hybrida* in the Solanaceae (Teakle and Wildermuth, 1967).

Taylor and Kimble (1964) reported that CMV was readily transmitted from spinach to *P. edulis* and *P. caerulea* seedlings by aphids but only rarely by sap inoculation. Initial symptoms developed in 4 to 6 days, and consisted of a very striking epinasty of the tip leaves.

2.2.7 Strains

Yonaha *et al.* (1979*)* isolated 3 strains of CMV from *P. edulis* and *P foetida.* These strains infected 31 plant species after mechanical inoculation. CMV caused chlorotic spotting and yellow mottle on passion fruit leaves and the terminal shoot leaves were often curled and twisted. One isolate induced a severe streak in tomato.

2.2.8 Particle morphology

The virus particles are polyhedric shape measuring about 26-28 nm in diam.

2.2.9 Management

A percentage of small seedlings of both *P. edulis* and *P. caerulea* were freed from CMV by heat therapy for a period of 14 days at 37^0 C. Other control measures include control of aphid vectors by systemic insecticides and keeping away the alternate and collateral hosts of the virus from the main passion fruit field.

2.3 Passion fruit " Tip Blight"

The disease was first reported by Greber (1966) from Queensland (Australia).

2.3.1 Symptomatology

The characteristics of this disease are epinasty, vein clearing and abscission of the terminal leaves, followed by a quick necrosis of the stem often extending for several inches back from the tip of all vigorously growing shoots . Sunken brown lesions often develop on the stem behind the wholly necrotic portion and affected plant show little tendency to climb but remain stunted, have mottled leaves and bear a few woody fruit only. Small plants may be killed.

2.3.2 Transmission

The virus is easily transmitted by mechanical sap inoculation. The virus is also transmitted by aphid vectors, *M. persicae* and *A. gossypii* in a non – persistent manner. Graft transmission is also reported. *P. suberosa* scion showing mottle symptoms when grafted to *P. edulis* plants induced a reaction identical with the natural disease.

2.3.3 Host range

P. edulis var. Flavicarpa*, P. alba, P. foetida, P. quadrangularis, P. subeltata, P. suberosa, P. caerulea, Phaseolus vulgaris* and *Sesbania exaltata.* The symptoms on *S. exaltata* consists of necrotic local lesions on the inoculated primary leaves. Da Graca (1976) reported that woodiness virus produced mosaic symptoms in mechanically inoculated cowpea. *Chenopodium amaranticolor* and *C. quinoa* produces local lesions 4-5 days after inoculation (Martini, 1962, Da Graca, 1976). *Vigna unguiculata* was found to show mosaic pattern 14 days after inoculation (Da Graca, 1976). *Petunia hybrida* as a symptomless carrier of the PWV.

2.3.4 Physical properties

The virus has thermal inactivation point between 55-60ºC, longevity *in vitro*, 3-4 days, dilution end point $1x10^{-5}$.

2.3.5 Purification

The virus was purified by Taylor and Kimble (1964). N-butanol (Morton, 1955) chlo-

roform-butanol (1:1) method described by Steer (1956) resulted in complete loss of infectivity.

2.3.6 Particle morphology

The virus particles are long flexuous rods with 600-800 nm in length and 16 ± 2 nm in width (Taylor and Kimble, 1964, Da Graca, 1976).

2.3.7 Etiology

Based on the host reaction, physical properties, transmission, aphid vector and particle morphology, it is concluded that the "Tip Blight" disease of passion fruit is caused by a strain of passion fruit woodiness virus (PWV). The virus is also serologically related to PWV.

2.3.8 Management

Same as described for the management of PWV.

2.4 Passion fruit ringspot

In 1968, virus-like symptoms were observed on leaves of *Passiflora edulis* var. Flavicarpa in the Ivory Coast, shriveled fruits with a reduced pulp cavity were also found, however, without "woodiness" (De Wijs, 1974a). De Wijs and Mobach (1975) isolated passion ringspot virus from *Adenia lobata* in Ivory Coast.

2.4.1 Symptomatology

In passion fruit two types diseased plants can be recognized: a few plants show malformed leaves with a severe mosaic and other leaves of normal shape showing spotting or mottling. The plants with an advanced stage of the disease appear stunted and bear little or no fruit. Such fruits remain small, immature and in many instances, shriveled. Typical woodiness *i.e.* abnormal thickening and hardening of the pericarp tissue and reduction of the pulp cavity is not found. More commonly the plants are not stunted and the leaves are not malformed but are spotted or mottled with 'ringspots' on the younger leaves, especially where these are shaded. Fruits do not show any symptom. On mechanical inoculation, plants of *P. edulis* show, spotting, mottling and ringspots but not malformation or severe mosaic.

2.4.2 Transmission

The virus is mechanically sap transmitted to *P. edulis* and other herbaceous hosts. Two aphid species *i.e. Aphis gossypii* and *A. spiraecola* transmit this virus in a non-persistent manner (De Wijs, 1974a). No seed transmission has been reported.

2.4.3 Physical properties

The dilution end point of the virus lies between 10^{-6}-10^{-7}, but the greatest change in infectivity occur at 10^{-3}-10^{-4}. The thermal inactivation point lies between 65-70°C and the longevity *in vitro* is 12-14 days at 24°C.

2.4.4 Host range

The virus infects a number of hosts belonging to different families (De Wijs, 1974a). These are as follows:

2.4.4.1 Passifloraceae

Passiflora edulis cv. Flavicarpa : Young developing leaves show vein yellowing and epinasty 5-20 days after inoculation. Mottling and rugocity sometimes occur. After about two months, spotting, mottling and ringspot symptoms develop. Fruits and flowers remain symptomless. *P. foetida*: Young leaves develop vein yellowing and epinasty before the secondary symptoms of dotting and mosaic appear on the non-well developed leaves. At times, inoculated leaves show vein banding and mottling. *P. quadrangularis* Giant granadilla : Local lesions 1-2mm in diam., develop in 4-6 days on inoculated leaves followed by leaf necrosis, stem necrosis and death of the young plants. Older plants are not infected. This plant is not suitable as a local lesion host. The following hosts show systemic symptoms: *Passiflora alba, P. caerulea, P. cincinnata, P. eichleriana, P. ligularis, P. seemannii, P. suberosa* and *P. vespertilio. Adenia glaua, A. lobata* and *Pascsiflora adenopoda* are symptomless carriers of the virus.

2.4.4.2 Chenopodiaceae

Chenopodium amaranticolor and *C. album* show faint spotting on inoculated leaves. *C. ambrosoides*, *C. botrys* and *C. quinoa* are symptomless carriers. *C. foetidium* exhibit faint spotting on the inoculated leaf when grown at 24°C.

2.4.4.3 Leguminosae

Phaseolus vulgaris var. Triomphe de Farcy: Inoculated leaves reacts with faint chlorotic spots, light green vein banding and slight necrosis of the veins. Cultivar Beka reacts with necrotic local lesions followed by vein necrosis. *Cassia occidentalis* : Systemically infected leaves develop ringspots.

The following plants are infected but without any symptoms: *Cassia hirsuda, Crotolaria retusa, Dolichus axillarius, Indigofera hirsuta, I. triata, Lathyrus odoratus, Phaseolus calcaratus, P. lathyroides, P. limensis, P. vulgaris* cvs. Bountiful and Nain fin de Bagnol, *Sesbania sesban* and *Tephrosia vogalii*. The following plants shows infection on inoculated leaves but uninoculated leaves remain symptomless-*Canavalia ensiformis, Dolichos lablab, P. vulgaris* cvs. Burpees stringless green pod, Torrent d'

or and widusa and *Vigna sinensis* cv. Deschine.

2.4.4.4 Other families

Symptomless but virus is recovered from systemically infected leaves: *Nicotiana benthamiana, N. megalosiphon* and *Portulaca oleracea*. Symptomless but virus could be recovered from inoculated leaves of *Cucurbita pepo, Helichrysum bracteatum, Gomphrena globosa, Vinca rosea* and *Zinia elegans*.

2.4.5 Purification and Particle morphology

Purification method described by De Wijs (1974a). Virus particles are flexuous rods (not enveloped) measuring 15 x 750-95 nm with a model length of 810 –830 nm (De Wijs, 1974a). Axial canal obscure. Basic helix obscure. Leaf sap contain few particles.

2.4.6 Properties of the virus

Particles sediment as 1 component A_{260}/A_{280} ratio of unfractionated preparation 1.22. Nucleic acid 6%. Protein 94%. Lipid 0%. One species of protein has been found with a molecular weight of 33,000.

2.4.7 Serology

The virus is strongly immunogenic. Particles do not react in standard agar-gel diffusion tests. The virus is serologically related but not identical to passion fruit woodiness virus. The virus seems to belong to the potato virus Y (PVY) group and has the cryptogram x / x/ x/ (6): E/E S/Ap (De Wijs, 1974a).

2.4.8 Epidemiology

Field survey and testing of leaves samples have given and impression of the distribution of passion fruit ringspot virus in two *Adenia* and two *Passiflora* spp. in Ivory Coast (De Wijs, 1975). De Wijs and Mobach (1975) found a virus on *A. lobata* causing ringspot symptoms. The causal virus is passion fruit ringspot virus. It is therefore confirmed that *A. lobata* is good host for the perpetuation of PRV. It was concluded that *Adenia lobata* is the original host plants of the virus (De Wijs, 1974a). *Aphis spiraecola* is the potential of this virus. A direct positive correlation has been established between the aphid population and transmission of passion fruit ringspot virus (De Wijs, 1974b).

    Aphis spiraecola usually occur in dense colonies on *Eupatorium conyzoides* Vahl. (Compositae). This perennial herb is found everywhere at the forest border in ORSTOM fields, Ivory Coast. The amount of rainfall and number of aphids trapped are significantly correlated with an internal of at least two weeks and that the effect of a period can last5 for several (4-6) weeks. The periods of rain are followed by periods of aphids. The chance that an infective aphid alights and feeds on one of the trap plants

depends on several factors, the abundance of winged aphids, the amount and proximity of virus source plants and the size of trap plants (Nelson and Tuttle, 1969).

2.4.9 Management

Use of virus free planting material and control of aphid vectors.

2.5 Passion fruit mottle

2.5.1 Geographical distribution

This disease was first observed on Golden passion fruit (*Passiflora edulis* Sim. f. *flavicarpa* Degner) during 1987 in Puli areas in Taiwan (Chang, 1992). A similar virus disease was also reported from Sri Lanka (Dassanayake and Hicks, 1992).

2.5.2 Symptomatology

Naturally infected plants show only mild mottle symptoms with no woody and mis-shapen fruits (Chang, 1992). Artificially inoculated plants of passion fruit (*P. edulis*, f. *flavicarpa*) no symptoms within two weeks after inoculation. Initially they exhibit vein yellowing and mild crinkling of young, non-inoculated leaves. Later, both mature and young leaves develop a green yellow mottle, with the production also of numerous small (115 mm) chlorotic leaf spots. Infected plants have reduced vigor (Dassanayake and Hicks, 1992). Infected fruits exhibit green mottle symptoms.

2.5.3 Transmission

The virus is mechanically sap transmitted. The virus can also be transmitted by pruning knife contaminated with the virus. The virus is readily transmitted by aphid vectors *e.g. Aphis craccivora, Aphis spiraecola* and *Myzus persicae*.

2.5.4 Host range

On artificial inoculation, the plant species got infection are shown in Table 1.

2.5.5 Purification of the virus

The virus has been purified by differential centrifugation (Chang, 1992) and by the method described for potyviruses by Hammond and Lawson, 1988. Purified virus preparation showed ultraviolet absorption spectrum typical of potyviruses with maximum and minimum absorption peak at 260 and 247 nm respectively.

2.5.6 Etiology

Passion fruit mottle is caused by a virus belonging to potyvirus group (Chang, 1992,

Dassanayake and Hicks, 1992).

2.5.7 Physical properties of the virus

The thermal inactivation point of the virus lies between 70-75°C, dilution end point between 10^{-5}–10^{-6} and infectivity *in vitro* up to 6 days at 20-23°C. The virus can remain infective up to one year when infected leaf tissue are stored at - 20°C over silica gel or anhydrous calcium chloride.

The virus particles are flexuous rods measuring between 600-900 nm in length and 13.1 $\pm$ 0.4 nm in width. Purified virus preparation show ultraviolet absorption spectrum typical of potyviruses with maximum and minimum absorption peaks at 260 and 247 nm respectively. The virus is immunogenic. Antiserum has been prepared against purified virions and virus – induced cylindrical inclusion proteins (CIP) (Chang, 1992). One major component of mol. wt. c. 33,200 and a minor component mol. wt. c. 27,700 of viral protein have been reported by polyacrylamide gel electrophoresis. Some components possess mol. wt. of 68,200 possibly a dimmer or inclusion protein.

2.5.8 Cytopathology

Darkly staining crystalline cylindrical inclusion are seen in the cytoplasm of infected epidermal cells of *P. foetida* (Dassanayake and Hicks, 1992). In thin sections cylindrical inclusions in the form of pinwheels as well as tubes (scrolls) and laminates have been seen in the cytoplasm of infected *P. edulis* f. *flavicarpa* leaf cells.

2.5.9 Detection

The presence of the virus can be detected serologically by ELISA, ISEM and double gel diffusion tests. The virus can also be detected by electron microscopy of purified virus preparations and thin sections.

Dassanayake and Hicks (1992) found a single band of nucleic acid, resistant to digestion in ribonuclease in distilled water, in extracts from *P. foetida* and *P. quadrangularis* . This RNA species is considered to be the double- stranded RNA form of SLPEMV genomic RNA.

2.5.10 Management

Use of virus free planting material and control of aphid vectors.

2.6 Passion fruit yellow mosaic

2.6.1 Geographical distribution

This disease has been found at Papucaia in Cashoeira de Macacu County, State of Rio de Janeiro, Brazil, during 1983 (Kitajima *et al.,* 1986). The disease incidence was less than 5% (Crestani *et al.,* 1986). Another isolate of passion fruit yellow mosaic virus

Table 1: Host range of passion fruit mottle virus

Plant species	Reactions	
	Inoculated leaves	Non-inoculated leaves
Amaranthaceae		
Gomphrena globosa	NL	--
Chenopodiaceae		
Chenopodium album	CL	--
C. amaranticolor	CL	--
C. foetidum	CL	--
C. murale	CM	--
C. quinoa	CM	--
Leguminosae		
Cassia occidentalis	CM	VY,CM
C. tora	NL	--
Crotalaria usaramoensis	CM	CM
Phaseolus vulgaris		
cv. The Prince	NL	--
cv.Canadian Wonder	NL	--
cv. Kentuky Wonder Green	NL	--
cv. Top Crop	NL	--
Vigna unguiculata		
cv. Bushita Mae	NL	--
Passifloraceae		
Passiflora edulis f. *edulis*	CM	CM,LD
P. edulis f. *flavicarpa*	CM	CM,LD,VY
P. foetida	CM	CM,VY,CL
P.lingularis	CM	CM
P. incarnata	M	
P. corcinae	SLI	
P.mollisima	CM	CM,CF,FB
P.quadrangularis	CM	CM,CF,VY
P. van-volxemii	CM	CM,CL
Solanaceae		
Nicotiana benthamiana	M	--
N. clevelandii	CL	CL
Petunia hybrida	SLI	--

CM = Chlorotic mottle/mosaic; CF = Chlorotic flecking; CL = Chlorotic lesions; NL= Necrotic lesions; LD =Leaf distortion; VY = Vein yellowing; FB = Flower break; SLI= Symptomless infection; - = No infection.

(PYMV) has been reported from Pernambuco State (Brazil) suggesting that PYMV has a larger geographical distribution (Kitajima *et al.,* 1986).

2.6.2 Symptomatology

The characteristic symptoms of the disease are bright yellow mosaic, yellow net, and leaf crinkle. Artificially inoculated passion fruit seedlings show faint vein clearing.

The vein clearing evolve to a general yellowing of the veins in a typical yellow net pattern. Otherwise, the chlorosis extend from the vein to the adjacent interveinal area, causing yellow patches. In extreme cases, the patches coalesce together, leaving only a few green areas along the main veins. At times, the chlorotic regions become whitish. These symptoms are accompanied by a slight leaf crinkle, but leaf size is not affected.

2.6.3 Transmission

The virus is transmitted by mechanical sap inoculation. The virus can also be transmitted by pruning knife used previously to cut twigs from passion fruit yellow mosaic virus (PYMV) infected plants. No seed transmission has been found. PYMV is transmitted by chrysomelid beetle namely, *Diabrotica bivitula* Kir. although it does not feed well on passion fruit. The beetles require 48 hr. acquisition feeding for virus transmission.

2.6.4 Physical properties of the virus

The dilution end point of the virus lies between 10^{-4}–10^{-5}, thermal inactivation point between 55-60°C, and aging *in vitro* for 8 days. The virus can remain infective up to 60 days in infected golden passion fruit leaves kept in freezer. Infectivity persist up to 180 days, when infected leaves are desiccated on silica gel under vacuum and kept in the freezer. The virus has been purified by differential centrifugation, the method described by Bozarth *et al.* (1977) for the purification of okra mosaic virus, with slight modification (Crestani *et al.,* 1986). Yield of PYMV in purified preparation could be obtained from 6-8 mg./100g of leaf tissue. The virus particles are isometric, not enveloped, measuring 28 nm in diam. Profiles not clearly angular or round, capsomere arrangement not readily seen. Leaf sap contains many particles.

2.6.5 Cytopathological changes

In ultra thin sections of infected leaf tissues, dense, roughly circular particles 20-25 nm in diam. scattered in the lumen of the vacuole have been observed (Crestani *et al.,* 1986). Sometimes, similar particles have been seen within the xylem vessels, however, they have not been detected in nucleoplasm. The most remarkable cytopathological effect observed is the presence small vesicles with a double membrane, commonly containing a fibrillar material, at the periphery of the chloroplasts. In chlorotic areas of the leaves, the plastids appear rounded, with a disorganized lamellar system.

2.6.6 Serology

The virus is strongly immunogenic. A highly specific antiserum with a titre of 1:2,048 has been produced (Crestani *et al,* 1986). Particles react in standard gel diffusion tests. No special conditions are required for serological tests.

2.6.7 Etiology

The yellow mosaic of golden passion fruit, is of viral etiology, and the causal virus PYMV, is a member of tymovirus group. The virus has been named "Passion fruit yellow mosaic virus" (Crestani *et al.,* 1986). Serological tests with particles of other members of the group show okra mosaic, kennedya mosaic, clitoria yellow mosaic, cocoa yellow mosaic and turnip yellow mosaic to be related.

2.6.8 Properties of the virus

Particles sediment as two components. Sedimentation coefficient is 62 and 125 S. Nucleic acid 25% or less. Viral genome single stranded RNA, linear, in one part Mr of sediment (S) $2x10^6$. One species of protein found. Mr of protein (S) 21000 and it is coat protein. Genome replicates possibly in the peripheral vesicles of the chloroplasts. mRNA coat protein translated possibly in the cytoplasm. Does not depend on another virus for replication.

2.7 Purple granadilla mosaic

Purple granadilla (*Passiflora edulis*) mosaic virus was originally described in the Cotia county the State of Sao Paulo, Brazil (Chagas *et al.,* 1984). This disease has not been reported primary anywhere else
.

2.7.1 Symptomatology

The disease is characterized by slight mosaic and vein clearing of the line pattern type. The fruits are deformed and hardened. The infected plants are stunted and fruit production is reduced (Chagas *et al.,* 1984).

2.7.2 Transmission

The virus is transmitted by mechanical sap inoculation. However, the chrysomelid beetle *Diabrotica specicola* transmits this disease with 20% efficiency when at least 10 individuals are employed in transmission. The virus is not aphid transmitted.

2.7.3 Host range

The virus is transmitted artificially to 12 species of *Passiflora e.g.* purple granadilla (*P. edulis*), yellow passion fruit (*P. edulis* f. *flavicarpa*), *P. gilberti* N. E. Brown, *P. maliformis* Vell, *P. serrato digitata* L., *P. caerulea* L. , *P. alata* (Dryland) Ait., "*Maracuja acu*", "*Maracuja-do diabo*", "*Maracuja do-mato*"," *Maracuja- chifre-de-veao*" and *P. sylvestris.* Among these plants, the last three is only be infected by grafting (Oliveira *et al.,* 1994). Purple granadilla is the only infected host which show symptoms, as previously described (Chagas *et al.,* 1984). Though *P. edulis* and *P. edulis* f. *flavicarpa* are easily infected in transmission tests, including by a pruning

operations, other passion fruit types are infected only in low percentage. *P. suberosa, P. incarnata, P. macrocarpa* and "*Maracuja- mirim*" are not infected by PGMV by mechanical means or grafting.

2.7.4 Physical properties

Thermal inactivation point of the virus lies between 60-60ºC, dilution end point between 10^{-5}–10^{-6} and longevity *in vitro* in the sap for 7 days at room temperature. PGMV remains active in dried leaves at –18ºC up to 150 days (Chagas *et al.,* 1984, Oliveira *et al.,* 1994).

2.7.5 Virus purification

The virus is purified by butanol clarification, precipitation by polyethylene glycol, differential and sucrose density gradient centrifugation (Oliveira *et al.,* 1994). The purified preparations of PGMV produce an absorption spectrum typical of nucleoprotein with an estimated yield of 0.3-2 mg of virus/100g of leaf tissue.

2.7.6 Serology

Polyclonal antiserum against PGMV with a dilution of 1:512 has been prepared.

2.7.7 Properties of the virus

Estimated sedimentation coefficient of PGMV is 129 S and the extinction coefficient, 8.4 mg/ml/cm. The electrophoresis analysis has revealed a single polypeptide in a capsid of 35.5 kDa and the genome is a single stranded RNA (ssRNA) of 1.83×10^6 Da. The virus particles are isometric, around 28-30 nm in diam., occurring in high concentrations in the tissues.

2.7.8 Cytopathological changes

 Electron microscopy of ultra thin sections of infected tissues revealed the presence of masses of virions in the cytoplasm or within the vacuoles. Virus particles, were detected in the cytoplasm and vacuoles, fibrilar material within the mitochondrial christae (Vega and Chagas, 1983). These fibrils may represent replicative forms of the viral RNA. The crystals found in nuclei may represent normal component (Oliveira *et al.,* 1994). Chagas and Vega (1988) also reported cytoplasmic intrusions into the chloroplasts in chlorotic areas of infected leaves.

2.7.9 Etiology

The disease is caused by a virus, however, the taxonomic position of PGMV is still uncertain. Its molecular characteristics place it close to either tombus-or carmovirus group, although the size of RNA is slightly larger and the capsid protein somewhat

smaller than the average values of these groups. The virus is not serologically related to several viruses tested so far.

2.7.10 Management

The management includes, use of virus free planting material and control of chrysomelid beetle.

2.8 Passiflora latent carlavirus

This disease was first reported on *Passiflora cerulea* in the Federal Republic of Germany (Schnepf and Brandes, 1962).

2.8.1 Symptomatology

No apparent symptoms are found on *P. caerulea*.

2.8.2 Transmission and host range

The virus is transmitted by mechanical sap inoculation and by grafting. The virus infects several host plants when inoculated artificially.

2.8.3 Diagnostic host species

Chenopodium album and *C. quinoa* produce leaf chlorosis. Diagnostically unsusceptible are *Nicotiana tabacum, N. glutinosa, Gomphrena globosa, Phaseolus vulgaris* and *Cucumis sativus*. For maintenance, propagation host species and assay *Chenopodium quinoa* is used.

2.8.4 Physical properties

Thermal inactivation point of the virus is 65ºC in sap from infected *Chenopodium quinoa*. Virus particles are filamentous, not enveloped, usually straight, 648 nm long. Axial canal obscure. Leaf samples contain few particles.

2.8.5 Serology

Purified preparations strongly immunogenic. Particles do not react in standard gel diffusion tests. No special condition required for serological tests. Mixed liquid tests give flocculent precipitates.The virus belongs to carlavirus group. Serological tests with particles of other members of the group show potato virus S, potato virus M, carnation latent, and chrysanthemum B and cactus 2 viruses to be related.

St. Hill *et al.*(1992) reported Passiflora latent virus from Florida. This virus induced local chlorotic lesions and systemic chlorosis in manually inoculated plants of *Chenopodium amaranticolor* and *C. quinoa*. In *Passiflora edulis* f. *edulis*, *P. edulis* f.

flavicarpa, *P. foetida*, and *P. incarnata*. This virus induced inconspicuous systemic foliar mosaic symptoms. This virus did not infect any of the following plants: *Beta vulgaris* L. 'Detroyt Dark Red', *C. occidentalis*, *C. melo* 'Smiths', *C. pepo* 'Early Prolific Straight Neck', *C. sativus* 'Poinsett', *Gomphrena globosa*, *Luffa acutangula*, *Macroptelium lethyroides* (L.) Urb., *Nicotiana benthamiana*, *N. tabacum* 'Burley', 'Havana 4-5', 'Samsun NN', 'Samsun Turkish', 'Xanthi NC', *Phaseolus vulgaris* 'Top Crop', *Solanum melongena* L., *Vigna unguiculata* 'California Black Eye', 'Zipper Cream'. Cytoplasmic inclusions were seen in infected epidermal tissues of *Passiflora* x Incense and *C. quinoa* stained in Azur A. These were red violet and resembled the "Vaccuolate", "para crystalline", and "fusiform-banded". The purified preparations revealed the presence of flexuous rod-shaped virus particles measuring between 531-772 nm in length. The virus can be detected serologically using ELISA test. The virus is serologically related to PLV isolate from Germany (St. Hill *et al.*, 1992).

2.8.6 Management

Use of virus free planting material.

2.9 Maracuja mosaic virus

2.9.1 Geographical distribution

This disease was first reported from Coastal area of Northern Peru on *Passiflora edulis* Sims. '*Flavicarpa*' (Spanish name: Maracuja) (Fribourg *et al.,* 1987).

2.9.2 Symptomatology

Infected plant exhibit symptoms of leaf mosaic mottle and reduced leaf size.

2.9.3 Transmission

The disease causing virus is readily transmitted by mechanical sap inoculation. The virus can also be transmitted by contact. Brushing one systemically infected *Nicotiana benthamiana* plant against leaves of three or four healthy plants of the same species, plants developed local lesions and systemic symptoms (Fribourg *et al.,* 1987).

2.9.4 Host range

The virus produces local infection in 27 species belonging to the Amaranthaceae, Balsamiaceae, Chenopodiaceae, Compositae, Cucurbitaceae, Labiatae, Passifloraceae, Scrophulariaceae and Solanaceae but induces systemic infection in *Passiflora edulis* and *N. benthamiana* (Table. 2). The most useful hosts are *C. amaranticolor, C. quinoa* and *Luffa acutangula* which develop necrotic local lesions. *Nicotiana tabaccum* react with necrotic local lesions, incomplete necrotic rings and lines. Leaves of *N. benthamiana* exhibit systemic necrotic lesions, line patterns, crinkling and mosaic.

Table 2: Symptoms of maracuja mosaic virus in indicator plants

Family/species	Symptoms
Amaranthaceae	
Amaranthus edulis	CLL
Gomphrena globosa	RLL
Balsamiaceae	
Impatiens balsamina L.	CLL
Chenopodiaceae	
Chenopodium amaranticolor Coaste & Reyn.	NLL
C. murale L.	NLL
C. quinoa Wild.	NLL
Compositae	
Helianthus annuus L.	SLI
Hechrysum sp.	CLL
Tagetes erecta L.	SLI
Zinnia elegans Jacq.	SLR
Cucurbitaceae	
Cucumis melo L.	NLL
C. sativus L.	NLL
Cucurbita maxima Duch.	NLL
C. moschata Duch.	--
Lagenaria siceraria (Molina) Standl.	NLL
Luffa acutangula Roxb.	NLL
Labiatae	
Salvia splendens Ker Gawl.	SLI
Passifloraceae	
Passiflora edulis Sims.	M
Scrophulariaceae	
Antirrhinum majus L.	SLI
Solanaceae	
Datura stramonium L.	CLL
Lycopersicon chilense Dun.	SLI
L. esculentum Mill.	SLI
Nicandra physaloides	NLL
N. benthamiana Domin.	NLL,SNI,CNCP,M
N. gultinosa L.	NLL
N. tabacum L.	NLL,LINR
Physalis floridana Rybd.	SLI

C = Crinkling; CLL= Chlorotic local lesions; M= Mosaic; NLL= Necrotic local lesions; LINR = Local incomplete necrotic ring; NLP = Necrotic line pattern; RLL = Reddish local lesions; SLI= Symptomless infection; SNL = Systemic local lesions; SLR = Sunken local rings

2.9.5 Physical properties

The thermal inactivation and the dilution end points of the virus lies between 90-95°C and between $10^{-10} – 10^{-11}$ respectively. The virus has been purified by the procedure

described for potato virus Y (Fribourg and Nakishima, 1984) with modification (Fribourg *et al.*, 1987). The virus has also been purified by St. Hill *et al.* (1992). The A_{260}nm/$_{280}$ nm ranged between 1.15 to 1.22. The purified virus preparations revealed two protein bands of 33-35 k Da for PLV-FL. Purified virus preparations have a A_{260}/A_{280} ratio of 1:1 and an extinction coefficient of 2.4 (mg/ml)$^{-1}$cm^{-1} at 260 mm, both corrected for scattering. The percentage of nucleic acid, assumed to be RNA, is calculated at about 7%, S erl obtained by centrifugation with marker viruses in sucrose density is 192.

2.9.6 Serology

The virus is highly immunogenic, homologous antiserum with a titre of 1:1024 has been prepared. The virus is serologically related to cucumber green mottle mosaic virus (CGMMV), frangipani mosaic virus (FMV) , and tobacco mosaic virus (TMV) but not with other tobamoviruses. Maracuja mosaic viruses from Peru and Florida are serologically related.

2.9.7 Particle morphology

Virus particles are rigid rods with an internal canal and structure of typical tobamovirus. The particle length is 304 nm and width about 15 nm (Fribourg, 1987). However, St. Hill *et al.* (1992) reported particle length between 283-320 nm.

2.9.8 Etiology

The maracuja mosaic is caused by a new virus belonging to tobamovirus group (Fribourg *et al.*, 1987).

2.9.9 Cytopathology

Fribourg *et al.* (1987) reported that systemically infected leaves of *N. benthamiana* contained numerous 300 nm thick, plate like aggregates of virus particles in the epidermis, leaf parenchyma and vascular parenchyma. These virus aggregates were highly ordered and showed a crystal-like pattern and sectioned perpendicular to the long axis of the particles. The ends of the particles were often regularly arranged to form nearly a straight line when sectioned parallel to the long axis of the inclusion in cross section. Scattered virus particles were seldom seen and were only conspicuous in virus containing xylem vessels. In addition to virus particles aggregates, cytoplasmic electron dense granular inclusions, rounded or irregularly shaped, not delimited by membranes, and with diameters of 1-3 mμ, were found consistently but sparsely in infected cells. As with other tobamoviruses (Francki *et al.*, 1985), vessels budding from the tonoplast in to the vacuoles were 60-70 nm in diameter and contained fibrillar material that resembled nucleic acid chloroplasts of infected tissues contained abnormally large starch grains. Crystalline cytoplasmic inclusions appearing as "hexagonal crystals" and "rounded plates" (characteristic of tobamoviruses, Christie and Edwardson, 1977) were seen in the epidermis of *P. edulis* f. *edulis*, *P. edulis* f. *flavicarpa*, and *P. incarnata* plants infected with

the virus (St. Hill *et al.,* 1992).

2.9.10 Detection

The virus can easily detected by serological techniques like ELISA. Immunosorbent electron microscopy (ISEM) and electroblot immuno assay are another good and reliable techniques for detection of MrMV (Fribourg *et al.,* 1987, St. Hill *et al.,* 1992).

2.10 Maracuja mosaic virus-Florida strain

St. Hill *et al.* (1992) reported a serologically distinct strain of maracuja mosaic virus in *Pasiflora* spp. in Florida.

The host range of this virus was similar to MrMV reported from Peru. Symptoms of MrMV-P were more pronounced than those of MrMV-FL in *N. benthamiana*. These two viruses are so differed in that MrMV-FL infected *P. incarnata*, whereas *P.quadrangularis*, and *P. suberosa* L., which were not tested with MrMV-P were susceptible to MrMV-FL. *P. quadrangularis* developed local necrotic lesions and systemic shoot necrosis after inoculation with MrMV-FL, whereas only mosaic symptoms are observed on plant of *Cassia occidentalis* and *P. suberosa*.

2.11 Soybean mosaic on Passion fruit

Benscher *et al.* (1994,1996) reported the infection of soybean mosaic virus (SMV) in *Passiflora* spp. in Colombia. The disease is characterized by severe mosaic, epinasty, defoliation, and premature death of *Passiflora* spp. Vines of maracuja infected with the virus showed intense chlorosis, mosaic, and epinasty followed by hardening of the leaves, severe defoliation and premature death. Ring spots on maracuja fruit were also observed. Granadilla exhibited mosaic and anthocyanin pigmentation of the leaves , and fruits showed ring spots.

The virus is transmitted by *Aphis gossypii* and *Toxoptera citricida*. The virus is also transmitted mechanically by sap inoculation but not by seeds.The virus can be detected serologically and by dot- hybridization with c DNA probes. The virus is serologically related to SMV. The deduced coat protein amino acid sequences for all of the Colombian isolates were virtually identical and shared 98% similarity with SMV. The virus belongs to potyvirus group.

2.12 Passion fruit vein clearing (Syn.Enfenzameto)

2.12.1 Geographical distribution

Batista *et al.* (1981) described at Lagarto, SE, a new disease, of possible viral etiology, designated locally as "Enfezameto" (a type of bushy stunt) relatively widespread in golden passion fruit plantations. This disease has also been reported from other regions throughout Brazil i. e. Serra da Ibiapina, CE, Bonito, PE, Feira de santana, BA, Mendes, RJ, Araguari, MG and Mo-retes, PR (Kitajima and Chagas, 1984, Kitajima and

Crestani, 1985, Chagas *et al.,* 1987). It is also reported from Viamao and Guaiba, RS (Prata, 1984).

2.12.2 Symptomatology

The disease is characterized by plants with short internodes, brittle leaves with typical vein clearing, stunting, deformed and sometimes woody fruits. The yield is reduced significantly (Kitajima and Crestani, 1985).

2.12.3 Transmission

The disease is transmitted by grafting, although grafted root stocks usually take a very long time, sometimes up to two months, to develop characteristic vein clearing symptoms. Mechanical and aphid transmission have been negative so far (Chagas *et al.,* 1983).

2.12.4 Etiology

Rhabdovirus-like particles have been found in thin sections of infected leaf tissues. These are found in the perinuclear cavity of some vascular parenchyma, although at times mesophyll cells also contain particles, however, the concentration is low. Another feature of some of the nuclei in these tissues, either containing particles or not, is the presence of an irregular mass of fine granular material, similar to the nucleoplasm reported in association with the infection by some rhabdoviruses (Francki *et al.,* 1981). Rhabdovirus particles found in PVC- affected passion fruit leaf tissues measure Ca. 300 nm x 70 nm.

3. Green spot of passion fruit

A severe outbreak of this disease occurred in orchards of Vera Cruz County, State of Sao Paulo, Brazil (Kitajima *et al.*, 1997). The disease was characterized by necrotic lesions and green spots in mature fruits and senescent leaves, usually resulting in death of the plant. Growers referred to this condition as 'green spot disease'. This condition was associated with heavy infestation by *Brevipalpus phoenicis*, mite and the death of the plant was caused by the girdling of the stem by the confluence of the necrotic lesions. In extreme cases, few orchards were completely destroyed.

   Electron microscopic examination of tissues from stem lesions and green spots on leaves and fruits revealed the presence of particles with round (60-70 nm in diameter) to bacilliform (60-70 x 130-150 nm) profile contained in membrane-bounded cavities and dense masses in the cytoplasm, similar to those found in tissues infected by the citrus leprosis virus (CitLV). Masses of amorphous, finely granular and electron dense material with circular to elliptical profile, without surrounding membrane with diameter ranging from 5-10 mμ occurred also in the cytoplasm. These affects are seen mostly in mesophyll parenchyma and occasionally in epidermal cells but not in the vascular region and are basically similar to what was reported in tissues infected

with CitLV (Kitajima *et al.*, 1974, Colariccio *et al.*, 1995).

Mites collected from affected orchards transferred to passion fruit seedlings produced small dark ring spots in the older leaves. These leaves rapidly became chlorotic except for the green spots, especially along the vein, similar to what has been observed in the field plants. Thin sections from these spots showed the same particles and dense masses as described above. The disease was not mechanically transmitted.

The exact causal organism of this disease is yet to be confirmed, however, it is suggested that this disease is caused by a virus similar or related to CitLV (Kitajima *et al.*, 1997).

4. Phytoplasma disease

4.1 Passion fruit witches' broom

4.1.1 Geographical distribution

This disease has been reported from Rio de Janeiro State and Pernambuco State, Brazil (Kitajima *et al.,* 1981,1986). This disease devastated the crops in the States of Rio de Janeiro, Pernambuco and Parana (Kitajima *et al.,* 1981, Leao, 1980, Loreto and Vital, 1983, Lima *et al.,* 1993, Kitajima and Chagas, 1984).

4.1.2 Symptomatology

The characteristic symptoms of this disease are shortening of the internodes, general chlorosis, falling of the flowers and fruits, fruit malformation and cracking and giant calyx.

4.1.3 Etiology

Electron microscopic evidence suggests that witches' broom of passion fruit may be caused by phytoplasma ((Kitajima *et al.,* 1981,1986).

4.1.4 Management

Use of disease free planting material is recommended.

5. References

Bailey, L. H. 1949. Manual of cultivated plants. McMillan Co., New York. pp.690-691.

Barbosa, C. J., Stenzel, N. M. C. and Jacowino, A. P. 1999. Occurrence of CMV in passion fruit in the state of Parana, Brazil. Fitopatol. Bras., 24: 193.

Batista, F. A. S., Gomes, R. C. and Ramos, V. F. 1981. Acorrencia de uma anomatia de possivel causa viriticaou semelhante a virus, provocando entezyamento do maracuyazeiro. An VI Cong. Bras. fruitcult., 1048-1413.

Benscher, D., Pappu, S. S., Niblett, C. L., Provvidenti, R. and Varon, de Agudelo, F. 1994.

Characterization of potyviruses from Passiflora by host range, molecular hybridization and sequence homology (Abstr.). Phytopathology, 84: 866.

Benscher, D., Pappu, S. S., Niblett, C. L., Varon, de Agudelo, F., Morales, F., Hodson, E., Alvarez, E., Acosta, O. and Lee, R. F. 1996. A strain of soybean mosaic virus infecting *Passiflora* spp. in Colombia. Plant Dis., 80: 258-262.

Bezerra, D. R., Albertso, J., Lima, A. and Xivier, Filho, J. 1995. Purification and characterization of an isolate of passion fruit woodiness potyvirus from Ceara State, Brazil (in Portuguese). Fitopatol. Bras., 20: 553-560.

Bozarth, R. F., Lana, A. O., Koenig, R. and Reese, J. 1977. Properties of the Nigerian and Ivory Coast strains of the okra mosaic virus. Phytopathology, 67: 735-737.

Brown, J. K., Bird, J. and Fletcher, D. C. 1993. First record of the passiflora leaf mottle disease caused by whitefly transmitted geminiviruses. Plant Dis., 77: 1264.

Brunt, A., Crabtree, K. and Gibbs, A. 1990. *Viruses of Tropical Plants*. Wallingford, UK: Commonwealth Agricultural Bureaux International 707pp.

Chagas, C. M., Kitajima, E. W., Lin, M. T., Maria I. C. S. and Yamashiro, T. 1981. Grave Molestia em Maracuja amarelo (*Passiflora edulis* f. *flavicarpa* Deg.) no Estado da Bahia causada por um isolado do virus do "woodiness" do Maracuja. Fitopatol. Bras., 6: 259-268.

Chagas, C. M., Colariccio, A. and Kitajima, E. W. 1983. Estudos de transmissibilidade do enfezamento do maracujazeiro. Fitopatol. Bras., 8: 620 (Res.).

Chagas, C. M., Catroxo, M. H., Moraes de Oliveira, J. and Furtado, E. L. 1987. Occurrence of passion fruit vein clearing virus in the State of Sao Paulo, Brazil (in Portuguese). Fitopatol. Bras., 12: 275-278.

Chagas, C. M., Colariccio, A., Kudamatsu, M., Lin, M. T., Brioso, P. S. T. and Kitajima, E. W. 1984. Estripe incomum do virus do mosaico do pepino (CMV) isolado de maracuja amarelo (*Passiflora edulis* f. *flavicarpa*). Fitopatol. Bras., 9: 402 (Abstr.).

Chagas, C. M. and Vega, J. 1988. Novos aspectos citoplatologicos observatos em maracuja roxo (VMMR). Fitopatol. Bras., 13: 132 (Abstr.).

Chang, C. A. 1992. Characterization and comparison of passion fruit mottle virus, a newly recognized potyvirus, with passion fruit woodiness virus. Phytopathlogy, 82: 1358-1363.

Chang, C. A., Wang, H. L., Chen, Y. L. and Chou, T. Y. 1981. The investigation and identification of virus diseases of passion fruit in Taiwan (Abstract in Chinese). Plant Prot. Bull., (Taiwan, R. O. C.) 23: 267.

Christie, R. G. and Edwardson, J. R. 1977. Light and electron microscopy of plant virus inclusions. Fla. Agric. Exp. Stn. Monogr. Ser., 9: 150.

Cobb, N. A. 1901. Woodiness of the passion fruit. Agric. Gaz. N. S. W., 12: 407-418.

Colariccio, A., Chagas, C. M., Mizuki, Vega, J. and Cereda, E. 1987. Infeccao natural do maracuja amarelo pelo virus do mosaico do pepino no estado de Sao Paulo. Fitopatol. Bras., 12: 254-257.

Colariccio, A., Lovisolo, O., Galletti, S. R., Rossetti, V. and Kitajima, E. W. 1995. Mechanical transmission and ultrastructural aspects of citrus leprosis disease. Fitopatol. Bras. 20: 208-213.

Crestani, O. A., Kitajima, E. W., Lin, M. T. and Marinho, V. L. A. 1986. Passion fruit yellow mosaic virus, a new tymovirus found in Brazil. Phytopathology, 76: 951-955.

Dassanayake, E. M. and Hicks, R. G. T. 1992. Sri Lankan passion fruit mottle virus, a potyvirus infecting golden passion fruit in Sri Lanka. Ann. Appl. Biol., 120: 459-469.

Da Graca, J. V. 1976. Studies on woodiness disease of passion fruit, *Passiflora edulis,* in South Africa. Phytophylactica, 8: 37-40.

De Wijs, J. J. 1974a. A virus causing ring spot of *Passiflora edulis* in the Ivory Coast. Annls. Appl. Biol., 77: 33-44.

De Wijs, J. J. 1974b. The correlation between the transmission of passion fruit ringspot virus and

populations of flying aphids. Neth. J. Pl. Path., 80: 133-144.

De Wijs, J. J. 1975. The distribution of passion fruit ringspot virus in its main host plants in Ivory Coast. Neth. J. Plant Path., 81: 144-148.

De Wijs, J. J. and Mobach, J. D. 1975. Passion fruit ringspot virus isolated from Adenia lobata in Ivory Coast. Neth. J. Plant path., 81: 152-154.

Escudero, J., Bird, J., Monllor, A. C. and Zettler, F. W. 1988. Mosaic of passion fruit (*Passiflora edulis*) in Puerto Rico. Phytopathology, 78: 857.

Francki, R. I. B., Kitajima, E. W. and Peters, D. 1981. Rhabdoviruses In: "Kurstak, E. ed. Hand Book of plant virus infections and comparative diagnosis". Amsterdam, Elsevier- North Holland. p. 455-489.

Francki, R. I. B., Milne, R. G. and Hatta, T. 1985. An Atlas of Plant Viruses, vol. 1. CRC Press, Boca Reton, F. L.

Fribourg, C. E. and Nakashima, J. 1984. Characteristics of a new potyvirus from potato. Phyto-pathology, ? 1363-1369.

Fribourg, C. E., Koenig, R. and Lesemann, D. E. 1987. A new tobamovirus from *Passiflora edulis* in Peru. Phytopathology, 77: 486-499.

Gough, K. H. and Shukla, D. D. 1992. Major sequence variations in the N-terminal region of the capsid protein of a severe strain of passion fruit woodiness potyvirus. *Arch. Virol.,* 124: 389-396.

Greber, R. S. 1966. Passion fruit woodiness virus as the cause of passion fruit vine tip blight disease. Qd. J. Agric. and Animal Sci., 23: 533-538.

Hirata, S. and Kono, A. 1965. Some studies on diseases of papaya and passion fruit in the warm region of Japan. Bull. Fac. Agric. Univ. Miyazaki, 11: 273-295.

Hollings, M. 1956. *Chenopodium amaranticolor* as a test plant for plant viruses. Plant Path., 5: 57-60.

Howell, C. W. 1976. Edible fruited Passiflora adopted to South Florida growing conditions. Proc. Fla. State Hort. Soc., 89 : 236-238.

Inoue, A. K., Mello, R. N., Nagata, T. and Kitajima, E. W. 1995. Characterization of passion fruit woodiness virus isolates of Brasilia and surrounding region, Brazil. Fitopatol. Bras., 20: 479-487.

Killip, E. P. 1938. Field Mus. Nat. Hist. Bot. Ser., 19 : 407-408.

Kitajima, E. W., Blumensehein, A. and Costa, A. S. 1974. Rodlike particles associated with ringspot symptoms in several orchid species in Brazil. Phytopath. Z., 81: 280-286.

Kitajima, E. W., Robbs, C. F., Kimura, O. and Wanderely, L. J. G. 1981. O 'Irizado' do chuchuzeiro E O superbrotamento do maracuja: Duas enfermidades associadas a microrganismos do tipo micoplasma constatadas nos estados do rio de janeiro e pernambuco. Fitopatologia Bras., 6: 115-122

Kitajima, E. W. and Chagas, C. M. 1984. Problems de viroses ou de etiologic micoplasmatica na cultura do maracuazeiro no Brazil. Fipatol. Bras., 10: 1681-1688 (Res.).

Kitajima, E. W. and Crestani, O. A. 1985. Association of a rhabdovirus with passion fruit vein clearing in Brazil. Fitopatol. Bras., 10: 681-688.

Kitajima, E. W., Chagas, C. M. and Crestani, O. A. 1986. Enfermidades de etiologia viral e associadas a organismos do tipo micoplasma em maracujazeiro no Brasil. Fitopatol. Bras., 11: 409-432.

Kitajima, E. W., Rezende, J. A. M., Rodrigues, J. V. C., Chiavegato, L. G., Piza Jr, C. T. and Morozini, W. 1997. Green spot of passion fruit, a possible viral disease associated with infestation by the mite *Brevipalpus phoenicis*. Fitopatol. Bras., 22: 555-559.

Kuhne, F. A. 1968. Cultivation of granadillas part 3. Fmg. S. Afr., 44: 17-19.

Leao, J. A. C. 1980. Algumas consideracoes sobre doencas e pragas que ocorremna microregiao homogena do agreste meridional do est. Pernambuco, particularmente nos municipios de

Bonito, Camocin, Barra da Guabiroba, Saire, Cortez e S. Jaaguim do monte. A nais IO Enc. Est. Cult. Marcuja, EMATER/ SE, p. 67-76.

Lima, No. V. C., Lima, M. L. R. Z. C., Souza, V. B. C., Malucclli N. H. 1993. Superbortameat do maracujazeiro associado a microorganismos do tipo micoplasma em culturas do municipiode Morretes, PR Rev. Sector Cien. Agri. U. F. Pr., 5: 83-86.

Lovisolo, O. and Kitajima, E. W. 1992. Nomenclature and relationships of some Brazilian leguminous potyviruses related to bean common mosaic and /or passion fruit woodiness viruses. Arch. Virol., 51: 307-310.

Loreto, T. J. G. and Vital, A. F. 1983. Viroses e micoplasmes do maracuja em Pernambuco. Informe SERDV, ano IV, 23p.

Magee, C. J. 1948. Woodiness or mosaic disease of passion fruit. Agric. Gaz. N. S. W., 59: 199-202.

Malan, E. F. 1957. Granadillas. In: *"Hand Book, Fmrs. S. Afr."* Vol. 2 Govt. Printer, Pretoria. pp. 828-834.

McKnight, T. 1953. The woodiness virus of the passion vine (*Passiflora edulis* Sims.). Qd. J. Agric. Sci., 10: 4-35.

Martin, F. W. and Nakasone, H. Y. 1970. The edible species of *Passiflora*. Econ. Bot., 24 : 333-343.

Martini, C. K. H. 1962. Some properties of the virus causing woodiness of passion fruit in western Nigeria. Ann. Appl. Biol., 50: 163-168.

Menzel, C. M., Winks, C. W. and Simpson, D. R. 1990. Passion fruit. In: "Fruits, Tropical and sub-tropical" (eds. Bose, T.K. and Mitra, S.K.) Naya Prakash Publishers, Calcutta. India. pp. 690-721.

Morton, J. F. 1967. Yellow passion fruit ideal for Florida some gardens. Proc. Fla. State Hort. Soc., 80: 320-330.

Morton, R. K. 1955. Method of extraction of enzymes from animal tissues. In: "Methods in Enzymology" Vol. 1. (Polwick, S.P. and Kaplan, W.O.) Acad. Press: New York.

Nattrass, R. M. 1939. A preliminary note on the 'woodiness' disease of passion fruit in Kenya. E. Afr. Agric. J., 6: 54.

Nattrass, R. M. 1940. Further notes on the "woodiness" disease of passion fruit in Kenya. E. Afr. Agric. Jour., 6: 54 (Rev. Appl. Mycol. 19: 664-665).

Nattrass, R. M. 1944. The transmission of the virus of the "woodiness" disease of passion fruit (*Passiflora edulis*) by single leaf grafts. Ann. Appl. Biol., 31: 310-311.

Nelson, M. R. and Tuttle, D. M. 1969. The epidemiology of cucumber mosaic virus and watermelon mosaic virus-2 of cantaloupe in an arid climate. Phytopathology, 59: 849-856.

Noble, R. J. and Noble, N. S. 1939. Aphid vectors of the virus of woodiness or bullet disease of passion fruit. J. R. Soc. N. S. W., 72: 293-317.

Oliveira, C. R. B., Maninho, V. L. A., Astolfi, S., Azevedo, M., Chagas, C. M. and Kitajima, E. W. 1994. Purification, serology and some properties of the purple granadilla (*Passiflora edulis*) mosaic virus. Fitopatologia Bras., 19: 455-462.

Ong, C. A. and Ting, W. P. 1973. Two virus diseases of passion fruit (*Passiflora edulis* f. *flavicarpa*). MARDI Research Bull., 1: 33-50.

Prata, C. H. S. 1984. Molestias do maracuja amarelo (*Passiflora edulis* f. *flavicarpa* Deg.) em dois pomares insta lados nos municipios de Viamao e Guaiba no Est. Rio Grande do Sul. Dissertacao de Mestrado, Univ. Fed. Rio Grande do Sul, 75p.

Purseglove, J. W. 1974. Tropical crop-Dicotyledons, Longman Group Ltd., London, pp 420-429.

Quacquarelli, A. and Martelli, G. P. 1969. *Passiflora coerulea* L. e *Nicotiana glauca* R. Grah. ospiti del virus del mosaico del Cetriolo (CMV). Atti de Primo Congr. dell' Unione. Fitopathol. Medit., 1: 224-257.

Schnepf, E. and Brandes, J. 1962. Uber ein Virus aus *Passiflora* sp. Phytopath. Z., 43: 102-105.

Senanayake, A. E. 1972. A disease of passion fruit (*Passiflora edulis* f. *flavicarpa*). Tropical Agriculturist No., 1&2: 47-49.

Seneviratne, S. N. de S. and Wickramasingha, D. L. 1973. Passion crumple, a new virus disease of passion fruit (*Passiflora edulis* f. *flavicarpa*). Proc. of the Ceylon Assoc. for the Advancement of Science, 1:66.

Seneviratne, S. N. de S. and Wickramasingha, D. L. 1974. Passion fruit yellow blotch, a new virus disease of passion fruit (*Passiflora edulis* f. *flavicarpa*).Proc. of the Ceylon Assoc. for the Advancement of Science, 1: 65.

Shukla, D. D. , Mc Kren, N. M. and Ward, C. W. 1988. Symptomatology, serology, and coat protein sequences of three strains of passion fruit woodiness virus. Arch. Virol., 102: 221-232.

Simmonds, J. H. 1959. Mild strain protection as a means of reducing losses from the Queensland woodiness virus in the passion vine. Qd. J. Agric. Sci., 16: 371-380.

Smith, K. M. 1972. Atext book of plant virus diseases. *3rd Ed. Longman Group Limited. London,* 684 pp.

St. Hill, A. A., Zettler, F. W., Elliot, M. S., Peterson, M. A., Li, R. H. and Bird, J. 1992. Presence of Passiflora latent virus and a serologically distinct strain of maracuja mosaic virus in *Passiflora* spp. in Florida. Plant Dis., 76: 843-847.

Storey, H. A. 1940. In: "Ann. Rep. E. Afr. Agric. Res. Station ",Amani, *1939*: 8-10.

Taylor, R. H. 1959. An investigation of the viruses which cause woodiness of passion fruit (*Passiflora edulis* Sims.). J. Austr. Inst. Agric. Sci., 25: 71 (Thesis Abstract).

Taylor, R. H. and Kimble, K. A. 1964. Two unrelated viruses which cause woodiness of passion fruit (*Passiflora edulis* Sims.). Austr. J. Agric. Res., 15: 560-570.

Taylor, R. H. and Greber, R. S. 1973. Passion fruit woodiness virus. Descriptions of Plant Viruses CMI/AAB No. 122.

Teakle, D. S., Gill, C. C., Taylor, R. H. and Raabe, R. D. 1963. Cucumber mosaic virus in *Passiflora* in California. Plant Dis. Reptr., 7: 677-678.

Teakle, D. S. and Wildermuth, G. B. 1967. Host range and particle length of passion fruit woodiness virus. Qd. J. Agric. Animal Sci., 24: 173-186.

Trindade, D. R., Poltronieri, L. S., Albuquerque, F. C., Rezende, J. A. M., Novaes, Q. and Kitajima, E. W. 1999. Occurrence of passion fruit woodiness virus (PWV) in the state of Para, Brazil. Fitopatol. Bras., 24: 196.

Vega, J. and Chagas, C. M. 1983. Citopitologia causada porum virus isolado de maracuja roxo (*Passiflora edulis* Sims.). Fitopatologia Bras., B.621 (Abstr.).

Yamashira, T. and Chagas, C. M. 1979. Occurrencia de grave virose em maracuja amarello (*Passiflora edulis* f . *flavicarpa* Deg.) no Estado da Bahia. Anais do V Cong. Bras. Fruitic., Pelotas , 915-917.

Yonaha, T., Tamori, M., Yamanoha, S. and Nakasone, T. 1979. Studies on passion fruit virus diseases in *Passiflora edulis* and *Passiflora foetida* plants. Sci. Bull. of the Col. Agric. Univ. Ryukyus, No. 26: 29-38.

Pea Diseases and their Management

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Abstract: Peas are grown worldwide as either dry peas used for human or animal consumption or as fresh, processing peas. One particular characteristic of pea agronomy that has implications for disease management is that peas are usually the first crop sown in spring and thus are often planted into cold and wet soils. Peas are affected by a number of bacterial, fungal, viral, and nematode diseases. These diseases, under the right conditions, can significantly decrease both yield and quality. In this chapter we divide pea diseases broadly into soilborne, airborne and viral diseases, a convenient classification when it comes to managing these different diseases. Important groups of organisms causing soilborne diseases on pea include fungi, bacteria, and nematodes. Soilborne fungal diseases discussed in this chapter include seedling diseases, root rots and wilts. The pea cyst, the root knot and the root-lesion nematodes cause soilborne nematode diseases. Fungi and bacteria cause major foliar diseases of pea. Foliar diseases caused by fungal pathogens include white mold, powdery mildew, downy mildew and *Ascochyta* blight. Important bacterial diseases include bacterial blight as well as brown spot. Finally, major diseases caused by viruses include *Alfalfa mosaic virus, Bean leafroll virus, Pea enation mosaic virus, Pea streak virus, Red clover vein mosaic virus* and *Pea seedborne mosaic virus.*

1. Introduction

Peas (*Pisum sativum* L.) are cultivated as a dry, edible crop or as fresh, processing peas. Dry peas are usually grown in rotation with cereals in extensive farming systems. Production of dry peas is expanding in developed countries such as France, Canada and Australia, where they are often used as protein supplements. Processing cultivars are harvested when green and succulent, while dry peas are harvested when seed reaches a moisture content of 12% or less. Fresh peas are often grown in rotation with other processed vegetables. Peas are usually the first crop sown in the spring and thus are often planted into cold and often wet soils.

Peas are affected by a number of bacterial, fungal, viral, and nematode diseases. These diseases, under the right conditions, significantly decrease both yield and quality. Important groups of organisms causing soilborne diseases on pea include fungi, bacteria and nematodes. In this chapter we divide pea diseases broadly into soilborne, airborne and viral diseases, a convenient classification when it comes to managing these different diseases. Soilborne fungal diseases discussed in this chapter include seedling diseases caused by *Pythium spp.* and *Rhizoctonia solani* Kühn; root rots caused by *Aphanomyces euteiches* Drechs., *Fusarium solani* f. sp. *pisi* (Jones) Snyd.

& Hans. and *Thielaviopsis basicola* (Berk. and Broome) Ferraris; and wilts caused by *Fusarium oxysporum* f. sp. *pisi* (van Hall) Snyd. & Hans. races 1, 2, 5, and 6. Major nematode diseases are caused by the pea cyst (*Heterodera goettingiana* (Liebscher)), the root knot (*Meloidogyne* spp.) and the root-lesion nematodes (*Pratylenchus penetrans* (Cobb) Filip. & Schuur.-Stek.). Fungi and bacteria cause major foliar diseases of pea. Foliar diseases caused by fungal pathogens include white mold caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, powdery mildew caused by *Erysiphe pisi* DC., downy mildew caused by *Peronospora viciae* (Berk.) Casp., gray mold caused by *Botrytis cinerea* Pers.:Fr., pea rust caused by *Uromyces fabae* (Grev.) Fuckel, and *Ascochyta* blight caused by a complex of three fungi including *Ascochyta pisi* Lib., *Mycosphaerella pinodes* (Berk. & Bloxam) Vestergr., and/or *Phoma medicaginis* var. *pinodella* (Jones) Boerema. Important bacterial diseases include bacterial blight caused by *Pseudomonas syringae* pv. *pisi* (Sackett) Young, Dye and Wilkie as well as brown spot caused by *Pseudomonas syringae* pv. *syringae*. Major diseases caused by viruses include *Alfalfa mosaic virus, Bean leafroll virus, Pea enation mosaic virus, Pea streak virus, Red clover vein mosaic virus* and *Pea seedborne mosaic virus.*

2. Soilborne diseases of peas

Root and seedling diseases damage crops and can result in significant yield losses. This damage is often not recognized because root diseases are, for a large part, hidden from view. The habitat in which roots grow is complex and provides significant challenges to researchers. Given the complexity of soilborne disease, substantial progress has been made in understanding the biology and management of these diseases (Hall 1996), and much of this general knowledge is transferable to peas. Three soilborne groups of organisms cause important diseases of pea, namely fungi, oomycetes and nematodes. One difficulty with all soilborne diseases is that their management is made difficult because pathogens survive in the soil and their eradication is not economically feasible. Resistance is one major component of disease management; however, gene-for-gene resistance is only available for *Fusarium* wilt and rate-reducing resistance, if available at all, is generally not enough for disease management (Table 1). Much of the management of soilborne diseases relies on use of clean seed, seed treatments, crop rotation, suppressiveness, management of the soil habitat through cover cropping and increase of organic matter, and if economically feasible, use of soil solarization or fumigation (Fritz *et al.,* 1995, Grünwald *et al.,* 2000 a, b, Hall 1996, Kraft *et al.,* 1988, van Bruggen and Grünwald 1996).

2.1 Fungal and Oomycete diseases

2.1.1 Seed and Seedling diseases

Establishment of healthy seedlings is the basis for a healthy pea crop. A number of abiotic and biotic factors can reduce seedling health and lead to poor stand establishment. Peas are a short-term, early-season crops. Thus, poor seed vigor and cold, wet soils result in poor seedling emergence, even when plant disease is not involved.

Several pathogens can cause seed and seedling diseases. The two most important pathogen groups are the Oomycetes in the genus *Pythium* and the fungi in the genus *Rhizoctonia* (Martin and Loper 1999, Anderson 1982). *Pythium ultimum* Trow var. *ultimum* (Van der Plaats-Niterink 1981) and sporangial forms similar to *P. ultimum* are the most frequently described *Pythium* species affecting peas. Other *Pythium* species affecting pea include *P. aphanidermatum* (Edson) Fitzp., *P. irregulare* Buisman, *P. splendens* Braun, *P. debaryanum* Hesse, *P. dissotocum* Drechs., *P. acanthicum* Drechsler, *P. spinosum* Sawada apud Sawada & Chen, and *P. oligandrum* Drechs. *Rhizoctonia solani* Kuehn assigned to the AG-4 grouping (Anderson 1982) causes seed, epicotyl, and hypocotyl rot.

Diseases of pea caused by *Pythium* and *Rhizoctonia* spp. are referred to as seed rot, damping-off, or root rot diseases. Damage by *Pythium* tends to be more severe when soil moisture is high and soil temperatures are in the range of 10 to 15ºC (Kraft *et al.,* 1988). Disease caused by *R. solani*, on the other hand, is most severe where conditions are warm and moist, particularly in lighter or sandy soils. *Pythium* damage is characterized by a watery, soft rot and the infection of juvenile tissues, such as root tips. Symptoms of infection by *R. solani* include a water-soaked appearance of the

Table 1: Sources and nature of resistance available for managing major soilborne fungal dis eases of pea. Only a selection of references on the source and/or genetics of resis tance are presented and no attempt was made to be exhaustive.

Disease	Pathogen	Genetic nature of resistance	References on sources and/or genetics of resistance
Aphanomyces root rot	*Aphanomyces euteiches*	Major and minor QTL	Pilet-Nayel *et al.,* 2002
Damping-off/ Seedling rot	*Rhizoctonia solani; Pythium* spp.	Not known	Kraft and Roberts 1970
Fusarium root rot	*Fusarium solani* f. sp. *pisi*	Not known	Grünwald *et al.,* 2003 in press, Kraft 1975, Kraft and Roberts 1970
Fusarium wilt	*Fusarium oxysporum* f. sp. *pisi*	Single, dominant genes	Haglund 1989, Haglund and Kraft 2001, Kraft 1994,

hypocotyl and epicotyl, and reddish-brown to brown lesions above and below the cotyledonary node. *Pythium* spp. form oospores and *Rhizoctonia* form sclerotia as survival structures.

Most seedborne diseases caused by fungi or oomycetes can be chemically controlled with fungicide seed treatments or biological control agents (Martin and

Loper 1999, Parmeter 1970). Fungicide-treated seed preempts seed infection and delays seedling infection by *Rhizoctonia* or *Pythium* species and is the single most important management technique for establishment of a good seedbed. Smooth seeded peas do not exude as much carbohydrate and inorganic salts as do wrinkled seed peas and are thus less susceptible to seed and seedling infection (Kraft 1991). Planting in wet, cold, heavy and compacted soils should be avoided. There are no resistant pea cultivars, but those which are vigorous, have thick epicotyls and emerge rapidly, can escape serious damage (McCoy and Kraft 1984). Rotation with cereals, clean tillage prior to planting, and fungicidal seed treatments provides some protection (Parmeter 1970, van Bruggen *et al.,* 1996).

2.1.2 *Aphanomyces* **root rot**

Aphanomyces root rot is one of the most destructive and widespread pea diseases. A field planted to pea can be completely destroyed by this disease. Aphanomyces root rot is caused by *Aphanomyces euteiches* Drechs., an oomycete water mold currently classified as being more closely related to golden-brown algae than to true fungi. The first symptoms appear on roots about 2 weeks after infection. Initial lesions appear light brown and tissue turns soft and darker as disease progresses. Microscopic observation often reveals oospores in diseased tissue. *A. euteiches* produces both oospores and zoospores. Zoospores are motile spores that can swarm towards a root if enough free water is available. Oospores serve as survival structures and are able to overwinter several years.

A. euteiches has a wide host range and can infect pea roots in any stage of plant development. Planting in wet soils exacerbates the disease. Infection can occur over the entire range of temperatures conducive to pea growth.

Aphanomyces remains a difficult disease to manage. No commercial cultivars with tolerable levels of resistance to Aphanomyces root rot exist. However, good levels of resistance have been identified and several quantitative trait loci for resistance to Aphanomyces root rot have been mapped (Pilet-Nayel *et al.,* in press). An effective means of avoiding losses is to assess infestation levels of fields before planting and avoiding moderately to highly infested fields (Kraft *et al.,* 1990). Oat residue has been found to reduce the severity of Aphanomyces root rot (Fritz *et al.,* 1995, Williams-Woodward *et al.,* 1997). Avoiding infested land and preventing buildup of inoculum through long crop rotations of non-host crops are crucial.

2.1.3 *Fusarium* **root rot**

Fusarium root rot of peas occurs in most pea fields throughout the world. The disease was first described on pea in Minnesota in 1918 (Bisby 1918). *Fusarium* root rot is caused by the fungus *Fusarium solani* f. sp. *pisi*. *F. solani* f. sp. *pisi* is unique among the forma specialis of *F. solani*, in that it affects several hosts including pea, mulberry, ginseng and chickpea (Matuo and Snyder 1972, Kraft 1969). The perfect stage of *F. solani* is *Nectria haematococca* Berk. and Broome and has only been reported in Japan as causing branch blight of mulberry trees (*Morus alba* L.) (Matuo and Snyder 1972).

Early symptoms of *Fusarium* root rot appear as a reddish-brown discoloration of the roots. Lesions become darker as disease progresses. Roots eventually become shrunken and constricted by lesions, and vascular tissues acquire a red discoloration.

The disease causes little damage in unstressed plants. *Fusarium* root rot is generally more severe during hot, dry seasons, when high soil temperatures favor infection and development of the pathogen. Close rotations, compacted soil and low fertility also increase severity of this disease. Chemical seed treatment, plant resistance, wide rotations, and management practices that avoid root stress and water stress can achieve partial control. Sources of resistance to *Fusarium* root rot have recently been described for Plant Introduction accessions of *Pisum* (Grünwald *et al.,* 2003 in press) that show promise for improving levels of resistance currently available in commercial cultivars. Biological control of *Fusarium* root rot has been attempted through incorporation of cover crops or organic materials such as barley, white mustard, lettuce or chitosan. Biological control has also been attempted with fungal antagonists, mycorrhizal fungi, or *Pseudomonas* bacteria. However, none of these methods of biological control are currently being used in practice (Kraft *et al.,* 1988).

2.1.4 *Thielaviopsis* root rot

Thielaviopsis root rot is also known as black root rot because of the large, black lesions typical of the disease. *Thielaviopsis* is considered to be a disease of local importance and is a minor disease on a global scale. *Thielaviopsis* root rot is caused by *Thielaviopsis basicola*. Typical symptoms include dark brown to black elongated lesions on the roots. Lesions coalesce as disease progresses and taproots undergo necrosis. *T. basicola* forms thick-walled chlamydospores in diseased host tissue as well as endoconidia. The disease is favored by periods of high soil moisture and high temperature (Lloyd and Lockwood 1963). Chlamydospores can survive in soil for several years and germinate by forming germtubes that grow towards host roots.

As with Aphanomyces and *Fusarium* root rot, the most effective control measure is avoidance of fields with high inoculum potential. Rotation is an important management tool. A rye crop has shown suppressiveness to *Thielaviopsis* root rot (Harman and Dillard 2001). Use of fungicides is not economically feasible. Development of varieties resistant to this disease has not been given much importance. Movement of infested soil, infected host debris, water and contaminated farm implements should be avoided as they result in spread of the disease.

2.1.5 *Fusarium* wilt

Fusarium wilt of peas is a global problem occurring anywhere peas are grown. The disease can be very severe, particularly when short rotations are used. *F. oxysporum* f. sp. *pisi* is the causal agent of *Fusarium* wilt. The organism is disseminated through movement of soil, plant residue, wind and water. Several races of the pathogen are recognized. Races 1 and 2 are economically important in most areas of the world (Kraft *et al.,* 1988). Disease caused by race 2 is referred to as "near wilt", because symptoms appear later in the growing season, often at bloom. Races 3 and 4 were originally

described in the Netherlands and Canada, but have since been reclassified as being more virulent isolates of race 2 (Kraft and Haglund 1978). Races 5 and 6 have only been reported for northwestern Washington State, the US, and British Columbia, Canada (Haglund and Kraft 2001). Plant symptoms caused by races 1, 5 and 6 on pea consist of chlorotic leaflets, which curl downward and become flaccid. In advanced stages the plant wilts and turns yellowish-brown in color. Often, the aboveground vascular system turns a light yellow to brick-red color. Symptoms of race 2 are similar to those of races 1, 5 and 6, but occur later and are less pronounced.

The genetics of resistance to the different races have been well characterized and a set of differential pea lines is available (Haglund 1989, Kraft 1994). Four single, dominant genes confer resistance to individual races 1, 2, 5, and 6 of *F. oxysporum* f. sp. *pisi* (Haglund and Kraft 2001).

Host plant resistance is the single most important tool available to manage *Fusarium* wilt of pea. Resistance to race 1 is commercially available and has all but eliminated this race as a constraint to commercial production. Resistance to race 2 is also available commercially, but is not widely deployed. Resistance to races 5 and 6 is available for production in the northwestern US and Canada.

2.2   Nematode diseases

Nematodes are small, unsegmented round-worms, some of which attack plants. More than 20 different genera of nematodes can attack pea roots. Economically important nematodes affecting pea include the pea cyst nematode *H. goettingiana*, the root-knot nematode (*Meloidogyne* species), particularly *M. incognita* (Kofoid & White) Chitwood, as well as the root-lesion nematodes in the *Pratylenchus* genus of which *Pratylenchus penetrans* (Cobb) Filip. & Schuur-Stek is the most common (Johnson and Fassuliotis 1984, Riggs and Niblack 1993). In general, economical control practices for control of nematodes on pea are limited. *Meloidogyne* and *Heterodera* are managed through crop rotation, as the host range is limited. Crop rotation is not effective for control of *Pratylenchus* (Inglis 2001). Plant parasitic nematodes, particularly root-knot nematodes, are often associated with other diseases as they predispose the plant to infection by root rot or wilt pathogens (Johnson and Fassuliotis 1984).

2.2.1 Pea cyst nematode

The pea cyst nematode, *Heterodera goettingiana* Liebs., can cause severe yield losses. The disease occurs throughout many regions of Europe and to a limited extent in the USA (Di Vito and Greco 1986, Di Vito 1991). Symptoms in the field include patches of stunted and brightly yellowed plants. These patches are small and circular, and later on extend throughout larger areas in a field (Di Vito and Greco 1986). Cysts, typically formed by cyst nematodes and recognizable as lemon-shaped, leathery bodies formed by adult females, appear embedded in the root. Pea, broad bean (*Vicia faba*), Austrian winter pea (*Pisum sativum* var. *arvense*), gross-pea (*Lathyrus cicera*) and vetch (*Vicia* spp.) are major hosts of the pea cyst nematode.

Disease caused by the pea cyst nematode is often accompanied by secondary

infection with fungal pathogens (Stone and Course 1974), particularly *F. oxysporum* f. sp. *pisi*. Management of pea cyst nematode is limited to (i). avoiding establishment of this nematode in new fields and (ii). to minimizing the buildup of soil populations once they are established. Critical levels of nematode infestation for peas are around 0.5 eggs per g soil (Greco *et al.,* 1991). Because the host range of this nematode is limited, crop rotation helps maintain populations at low levels. Rotations of 4 to 5 years without faba bean or pea can serve to maintain populations at low or non-detectable levels. Resistance to pea cyst nematode is currently not used as a method of control.

2.2.2 Root-knot nematode

Root knot nematodes are worldwide in distribution and have an extensive host range (Barker   *et al.,* 1985, Sasser and Johnson 1985). *Meloidogyne* spp. and particularly *Meloidogyne incognita* can infect pea, other legumes including bean and lima bean (Santo and Ponti 1985), as well as other non-leguminous hosts. Yield losses are most severe in sandy or well-drained soil. Disease by other root rot or wilt pathogens is predisposed by the root-knot nematode and, as such, good management practices for root rots and wilts should be followed. Most pea cultivars are tolerant to root-knot nematodes; nonetheless, a good rotation schedule with crops that do not increase root-knot nematode problems should be implemented.

2.2.3 Root-lesion nematode

Several species of *Pratylenchus* can infect pea roots and *P. penetrans* is one of the most common. While root-lesion nematodes are distributed worldwide, they occur most frequently in temperate climates. The nematode is disseminated by movement of soil, root debris and water. Data on population densities necessary for causing significant yield losses is limited. Soil infestation is managed primarily by use of nematicides. Usefulness of rotation as a management tool depends on the host range of each *Pratylenchus* species and can be useful if non-host crops are used as an alternative (Inglis 2001, Jensen 1972, Mai   *et al.,* 1977).

3. Airborne diseases of peas

Management of aerial diseases is conceptually quite different from that of soilborne diseases. For many of the foliar diseases resistance is available as a management strategy, as is the case for powdery mildew where gene-for-gene resistance is available, or for *Ascochyta* blight, where resistance is of a quantitative nature. Foliar diseases can also be managed with fungicides or bactericides, which are not generally effective for management of soilborne diseases.

Two important groups of organisms cause aerial diseases on pea and include fungi and bacteria. These fungal and bacterial diseases affect peas worldwide. Among the fungal diseases are the *Ascochyta* blight complex; downy and powdery mildew; white and Botrytis gray mold; and pea rust. Only two bacterial diseases, namely bacterial blight and brown spot are of importance in pea.

3.1 Fungal diseases

3.1.1 *Ascochyta* Blight Complex

Ascochyta blight of pea is a disease complex occurring in many parts of pea-growing regions, particularly in temperate regions of Europe, North America, Australia, New Zealand and the USSR. Serious yield losses ranging from 10% to 40% may result from *Ascochyta* blight (Bretag *et al.*, 1995a, Warkentin *et al.*, 1995, Tivoli *et al.*, 1995). *Ascochyta* blight and bacterial blight may exhibit similar foliar symptoms, but can be differentiated by holding the lesions to the light; *Ascochyta* blight leaf lesions are opaque, whereas bacterial blight leaf lesions appear translucent. Three pathogens are involved and often more than one of these pathogens occur together (Table 2).

The three pathogens that cause the *Ascochyta* blight complex of pea include *Ascochyta pisi* which causes spots on leaves and pods; *Ascochyta pinodes* (teleomorph *Mycosphaerella pinodes*), which causes blight; and *Phoma medicaginis* var. *pinodella*, previously *Ascochyta pinodella,* which causes foot rot. Although *M. pinodes* is generally the most damaging among the three pathogens, the three pathogens often occur together and can be difficult to separate from one another (Wallen 1974). Differences exist in conidia morphology among the three species. However, identification based on microscopic examination is not reliable because of variation in morphological traits even among isolates. Biochemical and molecular markers can assist in the identification process (Faris-Mokaiesh *et al.,* 1996). *M. pinodes* and *P. medicaginis* var. *pinodella*

Table 2: Characteristics of the three pathogens involved in the *Ascochyta* blight complex of pea.

Pathogen	Disease symptoms	Sexual spores	Asexual spores	Resistant chlamydospores	Main sources of primary inoculum
Ascochyta pisi	Leaf and pod spot	Not known	Conidia in pycnidia	Not known	Seedborne conidia
Mycosphaerella pinodes	Blight	Ascospores in perithecia	Conidia in pycnidia	Yes	Ascocspores and chlamydospores
Phoma medicaginis var. *pinodella*	Foot rot	Not known	Conidia in pycnidia	Yes	Chlamydospores

differ from *A. pisi* in that they produce resistant chlamydospores that can survive more than 10 years in soil. Table 2 summarizes the main differences among the three pathogens involved in the *Ascochyta* blight complex.

In most pea production areas, *Ascochyta* blight remains a persistent problem. Seed infection is prevalent and is the main source of primary inoculum (Bretag *et al.,* 1995b, Moussart *et al.,* 1996, Michall *et al.,* 1998). Ascospores of *M. pinodes* can be

airborne and can result in long distance dispersal and as an additional source of primary inoculum. Chlamydospores of *M. pinodes* and *P. medicaginis* var. *pinodella* can survive in soil for years and serve as inoculum as well. Their conidia are dispersed by rain splash within the canopy and are an important source of secondary inoculum. Optimum temperature for disease development is about 20ºC, but infection can occur in temperatures ranging from 5 to 30ºC (Roger *et al.,* 1998). Prolonged leaf wetness periods are very critical for spore germination and infection. All plant parts from the underground seed zone to the pods are susceptible to the *Ascochyta* blight complex. Symptoms caused by *A. pisi* are slightly different from those caused by the other two pathogens of the *Ascochyta* blight complex. *A. pisi* lesions are almost always found on above-ground parts, and are slightly sunken and defined by a sharp, dark circular border on leaves and pods or elongated on stems and usually contain black pycnidia. Symptoms caused by *M. pinodes* and *P. medicaginis* var. *pinodella* are often difficult to distinguish. Although *P. medicaginis* var. *pinodella* is usually associated with severe symptoms on below ground parts, especially near the seed zone, *M. pinodes* causes damage to above-ground parts including leaves, stems and pods, but rarely to underground parts. Resistance to *Ascochyta* blight exists in pea but the mechanisms are still not clear (Clulow *et al.,* 1991). It appears that resistance is inherited as a quantitative trait (Wroth 1999) and that resistance to stem and leaf infections by *M. pinodes* is controlled by different genes (Clulow *et al.,* 1991). *Lathyras* spp (*L. sativus*, *L. ochrus* and *L. clymenum*) are very resistant to *Ascochyta* blight and should be exploited for developing resistant pea cultivars (Gurung *et al.,* 1999).

To manage the *Ascochyta* blight complex of pea, clean seed that is free of the pathogens should be used. Seed should thus be produced in dry areas where precipitation will not favor development of *Ascochyta* blight. Infested seed should be treated with thiabendazole and thiram or carboxin (Bretag 1985). Overhead irrigation should be avoided to reduce spread of conidia and to avoid periods of prolonged leaf wetness (Roger and Tivoli 1996). Rotation is generally not an effective means for controlling a disease complex caused by *M. pinodes* and *P. medicaginis* var. *pinodella* due to the fact that chlamydospores can survive in soil for several years. Resistant or tolerant cultivars should be used when available (Bretag 1989). However, due to the potential for evolution of new races in the pathogen population (Nasir and Hoppe 1991, Wroth 1998), cultivars should be tested for resistance to the prevalent local races of the pathogen before they are selected for planting. In Australia, late planting (after mid June) can preclude exposure of young plants to high levels of inoculum (Bretag *et al.,* 2000). Fungicides, such as chlorothalonil and benomyl, are also used for managing *Ascochyta* blight epidemics (Warkentin *et al.,* 1995).

3.1.2 Gray mold

Gray mold occurs under humid conditions on all aboveground parts of peas and causes rot of infected tissue. It may also occasionally cause problems in fresh market peas during shipping. Gray mold of pea is caused by *Botrytis cinerea*, whose perfect (sexual) state is *Sclerotinia fuckeliana* (de Bary) Fuckel. Disease lesions usually turn gray in color, due to the asexual spores of the pathogen, which gives the disease its name.

Gray mold usually begins at the lower leaves where humidity is higher. Infected leaves can dry out resulting in severe defoliation. Stem lesions usually start near the stipule resulting in wilt of the upper portions of the plant. The most damaging phase of gray mold occurs when the organism moves from the senescing flower directly onto newly forming pods (Ford and Haglund 1963). Senescing flowers provide an ideal, humid environment as well as nutrient sources for this mostly saprophytic fungus. Although the pathogen can generally survive as conidia and sclerotia on plant debris or on growing plants, sclerotia are the main survival means under dry and cold conditions. Under warm and humid conditions, conidia can be readily spread by wind, rain splash or machinery. The sexual stage of the pathogen is not considered as important in contributing to epidemic development. Because resistance to gray mold is not known, fungicide application is the primary method for controlling gray mold, especially when the weather is wet during flowering and pod set. Fungicide applications are necessary to protect developing pods from infection and to prevent pathogen reproduction on dropped flowers (Biddle and Yeatman 1986). Caution is in order because resistance to benomyl is widespread in *Botrytis cinerea*, and thus protectant fungicides should be applied in rotation or mixture with benomyl to reduce the chance of developing a benomyl-resistant pathogen population. A balanced mineral nutrition of the pea crop is important in maintaining healthy pea plants. Potassium deficiency can predispose pea plants, especially pods, to infection by *B. cinerea* and excess mineral N fertilizer can lead to a lush canopy that is more susceptible to gray mold.

3.1.3 Powdery mildew

Powdery mildew of pea occurs wherever pea is grown. All aboveground parts of pea plants are susceptible to powdery mildew (Dixon 1978b). Pod infection may discolor seeds to a gray brown color. The powdery look of the disease is caused by the profuse production of conidia on the upper leaf surface (Fallon *et al.,* 1989). The disease is caused by the ascomycte *Erysiphe pisi*, an obligate parasite that grows only on living tissues of peas, as well as on other legumes including alfalfa, vetch, lupine and lentils. The disease usually first appears on surfaces of lower leaflets or stipules as small diffuse spots. Under ideal conditions, as provided by warm and dry weather and night temperatures at or below the dew point, the fungus can completely cover the whole plant. Such heavy infections turn the plant color to a gray or light blue. Conidia overwinter in alternate host plants and also on pea seed. Conidia can be spread over long distance by wind. Black globose cleistothecia, the sexual fruiting body of the pathogen in which ascospores differentiate, may develop in dead or dying host tissues. Ascospores develop within cleistothecia and are not important in the powdery mildew disease cycle, because the sexual stage of *Erysiphe pisi* is rarely observed. Severe infections, especially those started early in the growing season, can significantly reduce yield and plant height or result in death of the plant. Seed quality is adversely affected as seeds often acquire a bitter flavor or characteristics that are otherwise undesirable for processing. The disease may cause crops to mature at unscheduled times, thus interrupting carefully planned pea harvesting schedules of pea processing companies. Heavily infected crops may also cause health problems, as the fungal spores

that are also potent allergens can cause respiratory problems in farm workers.

Wherever possible, resistant cultivars should be selected, especially in late sown crops, which are likely to experience high disease pressure. Resistance is conditioned by two recessive genes (*er-1* and *er-2*) along with two or more modifying genes (Timmerman *et al.,* 1994, Tiwari *et al.,* 1997). Resistance in cultivars homozygous for *er-2* is expressed mostly in leaves and this resistance can be rendered ineffective under high disease pressure (Viljanen-Rollinson *et al.,* 1998). The disease is often less severe in areas where overhead irrigation is applied regularly because long periods of free water on host leaves reduce conidium viability and wash conidia from host tissue. Other control measures include fungicide sprays of sulfur and/or demethylation inhibitors such as cyproconazole, fenarimol and triadimenol. Fungicide spray should be applied at least two weeks before harvest to avoid residue on peas.

3.1.4 Downy mildew

Downy mildew occurs under cool, moist environmental conditions. The oomycete *Peronospora viciae* is actually more closely related to golden-brown algae and the *Pythium* and *Aphanomyces* pathogens discussed above, than to fungi, and is placed in the kingdom Stramenopila (Alexopoulos *et al.,* 1996). It can also infect peas, broad beans, alfalfa, and vetch (Farr *et al.,* 1989). Pathogenic variation exists among natural populations, and there can be host species-specific strains usually referred to as pathotypes or races (Taylor *et al.,* 1989).

Symptoms of downy mildew in pea can be either systemic or local (Pegg and Mence 1972). Systemic infection, which is the more severe form of the disease, results in stunting and distortion of the plants, usually killing the plants before flowering. The pathogen produces abundant inoculum in the form of sporangia on infected plant surfaces. Foliar infections are usually local, and begin on the lower parts of leaves. Local infections produce various shapes of brown blotches on the upper surfaces, and form profuse mildew like sporangia on the lower side of the lesions (Fallon and Sutherland, 1996). *P. viciae* may also infect and sporulate on the inflorescences and tendrils. Pod infection can occur under conditions of high relative humidity even in the absence of foliar infection. Infected pods become deformed and exhibit surface blistering. Pod infection may also stimulate proliferation of pod endocarps, giving an appearance of felt-like growth (Dixon 1978a). Later in the season, sexual oospores develop on or in senescent tissues and can survive in soil for up to 15 years (Van der Gaag and Frinking 1997). Optimal conditions for downy mildew development consist of cool humid weather, in contrast to the warm and dry conditions required for infection by powdery mildew. Secondary spread of the disease occurs solely by means of sporangia. Sporangial production requires high levels of relative humidity for at least 12 hours. Sporangia germinate best at low temperatures between 4 and 8 C and require rain for dispersal. The longevity of sporangia decreases significantly as temperature increases.

There appears to be no immunity among pea cultivars although tolerance to downy mildew is available among European cultivars. However, the downy mildew pathogen also shows pathogenic variation, and at least six races have been reported (Hubbeling 1975). Different races may occur together, and evaluation of resistance

should be monitored throughout the growing season from the seedling to mature stages. Evaluation of resistance to systemic infection is the most important criterion, and resistance to pod infection is secondary.

Deep plowing and long-term rotation of five years can also be employed to control downy mildew. Oomycete-specific fungicides such as metalaxyl can be applied to control the disease, but fungicide resistance in the downy mildew pathogen has been widely reported and can develop quickly. Thus, systemic fungicides such as metalaxyl should be applied in rotation with other protectant fungicides such as chlorothalonil.

3.1.5 White mold

White mold of pea occurs worldwide under wet conditions on foliage and stems with excessive vine growth. White mold is caused by *Sclerotinia sclerotiorum*, a fungal pathogen that has a wide range of host plants including many cultivated crops such as beans, *Brassica* spp., vegetables, potato, sunflower, soybean, and pea (Farr *et al.,* 1989).

The pathogen infects stems, leaves and flowers that are in contact with the ground due to logging (Kraft *et al.,* 1996). It produces prominent black sclerotia within mycelium and in cavities of infected stems. Sclerotia can germinate and produce mycelium for infection, but they most often develop apothecia, a sexual fruiting body produced after a period of dormancy. Within the apothecia, asci develop which eventually release ascospores into the air. The disease is most severe at the late growth stage when plants are flowering and the canopy is closed. Under these conditions, a humid microclimate in the lower pea canopy is produced, that is conducive to disease development. Senescent flowers are considered to be the initial nutrient source for infection. Once infection has started, the pathogen can quickly infect all plant parts (Biddle 2001).

To control white mold, seed used for planting should be free of sclerotia. Dicarboximide can be used as a protectant fungicide during the flowering stage in fields with known risk of white mold. Pea cultivars with semi-leafless traits may offer advantage for reducing white mold (Coyne *et al.,* 1974, Fuller *et al.,* 1984). Rotation with cereal crops, but not with many of the broad leaf crops such vegetables, beans, soybean and oilseed rape, can reduce inoculum build-up in the soil. No resistant cultivars of pea are available although resistance in some plant introduction accessions has been reported (Blanchette and Auld 1978).

3.1.6 Pea rust

The pathogen that causes pea rust occurs in many parts of the world and can seriously reduce yields. Pea rust is an important disease in south Asia, the southern regions of Russia, and has most recently emerged in North America (Xue and Warkentin 2002). The disease is caused by *Uromyces fabae*. This rust is autoecious, because it completes its life cycle on a single host. The host range includes pea, lentil, faba bean, sweat pea, and *Lathyrus* spp. (Farr *et al.,* 1989; Kraft 2001). Considerable pathogenic variation exists among isolates of *U. fabae* (Xue and Warkentin 2002). To date, six

pathotypes of the pathogen have been identified (Singh and Sokhi 1980). The pathogen produces three types of spores: aeciospores, urediospores and teliospores. Teliospores, produced near the end of the growing season, are resistant to adverse conditions and serve as primary inoculum. Aeciospores and urediospores are produced during the middle of the growing season and serve as secondary inoculum. Optimal conditions for infection consist of periods with high relative humidity and a temperature near 20ºC.

Resistance to pea rust is an important management strategy. Although there are no pea cultivars that are immune to pea rust, resistant cultivars are available (Xue and Warkentin 2002). Germplasm accessions have been reported to be resistant to both rust and powdery mildew (Pal *et al.,* 1980). Research has shown that resistance to rust in pea is controlled by a single dominant gene (Tyagi and Srivastava 1999). Because the pathogen is an obligate parasite, implementation of rotations that exclude volunteer plants and other legume hosts in the same geographic area is crucial. Systemic fungicides, such as triadimefon, hexaconazole, flusilazole, thiabendazole and benomyl, are very effective tools for managing pea rust (Sugha *et al.,* 1994, Gupta and Shyam 2000).

3.2 Bacterial diseases

There are two major bacterial diseases of pea, namely bacterial blight and brown spot (Table 3). These diseases are best managed by planting disease-free seed and using cultivars that are resistant.

3.2.1 Bacterial blight

Bacterial blight was first described in Colorado in 1915 by Sackett (1916). The disease has since been found in many parts of the world and on every continent. Under wet spring conditions or in overhead-irrigated systems, the disease can be severe and significantly reduce yields. Losses are generally proportional to disease severity or incidence (Roberts *et al.,* 1995, Mansfield *et al.,* 1997). Bacterial blight is caused by the bacterium *Pseudomonas syringae* pv. *pisi*. *P. syringae* pv. *pisi* can be differentiated from the brown spot pathogen *P. syringae* pv. *syringae*, based on biochemical and molecular assays (Hollaway *et al.,* 1997) or by inoculations with the pathogen to susceptible hosts. *P. syringae* pv. *pisi* initially produces water soaked lesions on inoculated leaves, whereas *P. syringae* pv. *syringae* produces necrotic lesions. There are seven races of *P. syringae* pv. *pisi* based on pathogenicity on differential cultivars (Bevan *et al.,* 1995). Race differentiation is also correlated with DNA fingerprints (Hollaway *et al.,* 1997). A gene-for-gene relationship appears to exist between pathogen avirulence and host resistance genes. Polymerase chain reaction (PCR) assays with two sets of four specific primers can differentiate *P. syringae* pv. *pisi* into two types based on amplified fragment sizes (Arnold *et al.,* 1996). In addition to providing a specific means for detecting the bacterial blight pathogen, separation of the two DNA types correlates with some of the races and/or phenotypes of *P. syringae* for fluorescence or non-fluorescence.

The pathogen is seedborne and is very prevalent in seed stocks (Roberts *et al.,*

1991, Reeves *et al.,* 1996). It resides in seed both externally and internally, and can survive for three years in infected or infested seed. The seedborne nature provides the pathogen with a primary means of long distance dispersal. The bacterium can be spread from infested/infected seeds to healthy seeds at harvesting by machinery or during the milling process. The bacterial blight pathogen can survive in infected pea debris for over a year, but does not survive well in soil, especially in organic soil (Hollaway and Bretag 1997). The bacterial blight pathogen proliferates profusely on plant surfaces of susceptible and resistant pea plants (Grondeau *et al.,* 1996) and is spread from plant to plant by rain (Roberts 1997). The pathogen has also been found on surfaces of more than 60 species of crops and weeds. Walking in fields during the growing season when plants are wet spreads the pathogen and should be avoided. Resistant cultivars should be grown wherever possible. Cultivars resistant to specific races are available and offer a very effective means of controlling bacterial blight. However, the race of the pathogen prevalent in a location should be determined before a resistant cultivar is selected for planting. A 2-year rotation in combination with pathogen-free seed or seed disinfested with 1% sodium hypochlorite is appropriate.

Table 3: Symptoms and control strategies for bacterial blight and brown spot, two major bacterial diseases affecting pea.

Disease	Pathogen	Symptoms	Control strategies
Bacterial blight	*Pseudomanas syringae* pv. *pisi*	Initially water soaked lesions on all aboveground tissue; lesions eventually turn brown and necrotic. Lesions on leaves may take on an angular shape; stem infections appear around nodes, stipules and leaflets. Infected flowers shrivel and decay and lead to pod and seed infection.	Plant pathogen-free seeds; deploy race-specific resistant cultivars; disinfest equipments
Brown spot	*Pseudomonas syringae* pv. *syringae*	After initial water soaked appearance, lesions on leaves resemble burning or physical injury; on stems, lesions are sunken and elongated, distorting stems, petiole and growing points.	Plant resistant cultivars; rotate with crops other than beans and soybeans; disinfest seed harvest-ing equipment.

3.2.2 Brown spot

Brown spot of pea occurs in many parts of the world. Although it is not as serious as bacterial blight, it is very common in fall or winter sown pea. Brown spot disease of pea is caused by the bacterium *Pseudomonas syringae* pv. *syringae*. The pathogens that cause brown spot and bacterial blight are very similar. Biochemical, serological and

molecular tests are required to distinguish them (Hollaway *et al.,* 1997, Malandrin and Samson 1998, Pankova and Kokoskova 1999). Pathogenicity tests on pea and beans can also be employed to differentiate the two organisms. The brown spot bacterium is pathogenic to both pea and bean, whereas the bacterial blight pathogen does not infect beans. Symptoms caused by brown spot and bacterial blight are also very similar. The main differences are that water soaking is apparent with bacterial blight infection, whereas browning, necrotic and sunken lesions become apparent with brown spot.

The brown spot pathogen is seedborne, primarily as a colonizer of the seed surface (Hoitink *et al.,* 1967). Six-month storage can significantly reduce contamination by the brown spot pathogen, although the pathogen could still be detected after storage for 1-year. The pathogen can survive in soil saprophytically for up to eight months. Although the pathogen can also colonize a wide range of other crops and weeds (Legard and Hunter, 1990), only beans and soybeans are important alternate host plants and should be specifically avoided in rotational sequences. Planting seed free of the brown spot pathogen is an important means for control of the disease. Extra care should be taken during seed harvest to avoid seed contamination from infected pea fields. Infested seed could be used after storage for at least one year as the pathogen does not survive well on seeds. Because brown spot is very prevalent in peas physically injured by wind, frost and hail, fall and winter planting should be avoided in areas with frequent brown spot epidemics (Butler and Fenwick, 1970).

3.3 Viral diseases of pea

Pea crops have been reported to be susceptible to over 50 different virus diseases. However, with the availability of improved molecular and serological techniques, many of the diseases have been determined to be caused by one or more strains of a particular virus, thereby reducing the number of actual viruses affecting pea to approximately 25. Only about six viruses occur at rates persistently high enough internationally to be of economical significance. The ecology of these viruses is diverse because of the numerous methods of transmission.

Weed species as well as other nearby legume crops including alfalfa and different clover species usually serve as natural inoculum reservoirs. Most viruses of pea are transmitted in a nonpersistent manner by aphids while a few are transmitted by nematodes, thrips and beetles. Several viruses are also seed-transmitted in pea and have resulted in significant yield losses. Seed-transmissible viruses including those affecting weeds and other susceptible crops have the potential to be transmitted over great distances and as such, pose a serious international threat.

Symptoms of infected plants can vary greatly for the same viral disease due to symptom variability among strains of the same virus. Thus, visual diagnosis can be difficult or inaccurate. Conversely, a single virus strain may be capable of producing a wide range of symptoms in different pea cultivars. Mixed infections often occur in pea and usually exacerbate symptoms on the plant (Schroeder *et al.,* 1959). The major virus diseases and control strategies are discussed in this chapter, although other viruses may be of particular importance in specific regions or countries**.**

3.3.1 Alfalfa Mosaic Virus

Alfalfa mosaic virus (AMV) has the largest alternate host range of any virus that infects pea crops, and includes a host range of approximately 150 plant species that have been reported to be naturally infected (Bol and Jaspers 1994). Symptoms in pea can vary greatly depending on the virus strain, cultivar, and environment. Infection in pea is revealed by symptoms such as general chlorosis, leaf curling, and the presence of brown necrotic lesions on leaves of plants. Purple or brown necrotic streaks typically occur in the vascular system of the stem and leaf veins. Plants exhibiting the latter symptoms usually die as a result of plugging of the vascular system. Pods may be misshapen or malformed, discolored, and exhibit brown necrotic lesions (Zaumeyer 1938). Ford and Baggett (1965a) reported that AMV consistently caused severe stunting in all pea cultivars tested. Not all strains result in stunting, and the strain used in their tests was probably highly virulent. Symptoms on pea resulting from infection with AMV can be confused with those caused by pea streak virus (Ford and Baggett 1965b) discussed below.

AMV is transmitted in a nonpersistent manner by at least 14 different aphid species (Crill *et al.,* 1970). The pea aphid (*Acyrthosyphon pisum* Harris) and the green peach aphid (*Myzus persicae* Sulz.) are considered the most common and widespread vectors of AMV in peas. Several other aphid species that vector AMV include the bean aphid (*Aphis fabae* Scopoli), the foxglove aphid (*Aulacorthum solani* Kaltenbach), and the potato aphid (*Macrosiphon euphorbiae* Thomas). Several species of leafhopper, although of little significance on pea, have also been reported to transmit the virus (Crill *et al.,* 1970). The most important perennial reservoir of AMV worldwide is considered to be alfalfa (*Medicago sativa* L.) and clovers (*Trifolium* spp.), although the wide range of other annual and perennial hosts provide additional inoculum sources from which aphids may acquire the virus.

Numerous plant introduction (PI) accessions with resistance have been identified but no resistant commercial cultivars are currently available. Accurate screening of lines is difficult because an immense number of AMV strains or isolates exist in nature. Ford and Baggett (1965b) reported that 31 of 900 pea lines tested exhibited some resistance against a single isolate of AMV from white clover (*Trifolium repens* L.). In contrast, Hagedorn (1968) tested 397 accession lines against an isolate from red clover (*T. praetense* L.) and found none of the PI lines was immune to the virus. Most of the lines reported as resistant to AMV by Ford and Baggett were retested by Hagedorn using the red clover AMV strain and found to be susceptible to the virus.

3.3.2 Bean leafroll virus

Bean leafroll virus (BLRV) is also known as pea leafroll virus and is commonly referred to by growers and fieldmen as "top yellows". The virus was first reported on faba bean and pea in Germany in 1954 (Quantz and Volk 1954), but because it was identified first in faba bean (*Vicia faba* L.), the virus was named bean leafroll virus. The virus was later detected in England (Tinsley 1959), New Zealand (Smith 1996), Iran (Kaiser 1972), the United States (Thottappilly *et al.,* 1977, Hampton 1983), Africa and India (Ashby 1984),

and more recently in Italy (Larsen and Webster 1999). The virus is now considered to be distributed worldwide. Symptoms on pea include, leafroll, stunting and bronzing or chlorosis, which may occur only in the apical region or over the entire plant. Stunting can be severe when plants are infected early in their growth stage and can also result in premature death.

BLRV is a member of the genus *Luteovirus* in the family *Luteoviridae* (Domier *et al.,* 2002), and is related to other yellowing viruses including *Beet western yellows virus* (BWYV), *Barley yellow dwarf virus* (BYDV), and *Soybean dwarf virus* (SDV). The virus infects several annual and perennial legumes including lentil, chickpea, faba bean, alfalfa and many clover species. Reports exist of infection in bean (*Phaseolus vulgaris* L.) (Ashby and Bos 1979, Ashby 1984, Thottappilly *et al.,* 1977), although the disease on this host is of minor importance in the US and Europe.

The virus is transmitted in a persistent manner by 12 aphid species (Edwardson and Christie 1991). The pea aphid however, is considered the most important vector and the virus is not mechanically transmissible. Aphids can acquire the BLRV in 2 hr or less, and transmit the virus in 1hr or less after a 3-day acquisition access period (Thottappilly *et al.,* 1977). The latent period of the virus in the vector is 16-20 hr but times may vary depending on the virus isolate. Baggett and Hampton (1991) examined host response to 11 BLRV isolates collected from Idaho on a wide range of pea genotypes and observed no differences amongst the isolates. Consistent with other phloem-limited viruses, Kaiser (1972) found that BLRV was not transmissible by seed. BLRV has been shown to share extensive serological relationships with several other luteoviruses including SDV, BWYV, BYDV-PAV strain, and *Carrot red leaf viru*s (Smith *et al.,* 1996, Martin and D'Arcy 1990, D'Arcy *et al.,* 1988, Waterhouse *et al.,* 1988).

Incorporation of resistance to BLRV has been a success for breeding programs, and many commercial cultivars are currently available that make BLRV management possible. Resistance in pea is inherited as a single recessive gene designated *lr* (Drijfhout 1968). Crampton and Watts (1968) also reported that resistance to BLRV is an additive system of inheritance. Moderate resistance was found to be a dominant gene for susceptibility and high resistance. A gene for tolerance, designated *lrv*, was described by Baggett and Hampton (1991) on the basis of symptom expression in selected pea cultivars. Additional research is needed to investigate the relationship between the *lrv* and the *lr* gene.

3.3.3 Pea enation mosaic virus

Pea enation mosaic virus (PEMV) is a unique virus in that it is associated with the genera *Enamovirus* and *Umbravirus* based on the two distinct RNAs in its genome, and is referred to as *Pea enation mosaic virus-1* and *Pea enation mosaic virus-2,* respectively (Demler *et al.,* 1996). Both RNAs must be present to cause the disease. Losses due to infection by the virus can be severe in pea, lentil and chickpea in epidemic years. PEMV was first described on faba bean by Osborne (1935) and subsequently in Europe, (Cockbain and Gibbs 1973), Iran, and Sicily (Peters 1982). The virus infects few plants outside the family *Leguminosae.* Foliar symptoms on pea include conspicuous chlorotic, translucent flecks, small brown necrotic lesions, and vein clear-

ing. Leaves and stipules are typically malformed, and plants are usually severely stunted and distorted. Small outgrowths or enations are often formed on the underside of leaves but this varies by cultivar. Pods are severely misshapen and produce raised wart-like outgrowths or proliferations on the surface. The virus causes death of plants in young, susceptible cultivars, although symptoms may be attenuated when plants are infected after the onset of bloom. The overwintering perennial hosts of PEMV are not well documented, although clover and vetch are suspected. Alfalfa has long been considered to be the primary, perennial host and inoculum source for PEMV (McWhorter and Cook 1958). However, work by Larsen *et al.,* (1996) demonstrated that PEMV could not be detected by ELISA or dot blot hybridization in alfalfa samples collected in Washington State between 1988 and 1994, nor could alfalfa be infected by mechanical inoculation or by aphid transmission with the virus.

PEMV is most commonly and efficiently transmitted by the pea aphid, although at least seven other aphid species have been reported to transmit the virus (Demler and de Zoeten 1994). Toros *et al.,* (1978) demonstrated that when pea aphid nymphs were given an acquisition access period of 3 hr, the virus had a latent period of 10 hr. Following the latent period, the aphid was able to transmit the virus during brief probes into the cytoplasmic tissue. Specific strains of PEMV have not been identified, although some isolates may exhibit slightly different biological properties such as variations in aphid transmission properties (Bath an Tsai 1969).

Resistance in pea to PEMV is determined by a dominant gene *En* and a recessive gene *en/en* (Schroeder and Barton 1958). Tolerance to the disease is available in selected pea cultivars and breeding lines. Progress has been made by the use of molecular markers for screening pea germplasm. A marker locus for PEMV resistance in pea has been identified as *Adh-1* (Weeden and Provvidenti 1988). In addition, Random Amplified Polymorphic DNA (RAPD) markers have been identified and mapped near *En* and *Adh-1* (Yu *et al.,* 1995). The complete nucleotide sequence for PEMV has been published by Demler and de Zoeten (1991) and Demler *et al.,* (1993), which has allowed the study of the genomic strategy of the virus. In addition, coat protein-mediated resistance to PEMV has been met with some success by Chowrira *et al.,* (1998). Experiments with transformed pea resulted in delayed or attenuated symptoms in the transgenic plants.

3.3.4 Pea streak virus

Pea streak virus (PeSV), a member of the large genus *Carlavirus,* is occasionally referred to as "streak" or "pea streak" because of the necrotic streaking symptoms occurring in the vascular system of pea. Specific symptoms include purple to brown necrotic streaks on stems and petioles, brown necrotic lesions on leaves, and wilting of the plant. Pods exhibit sunken brown necrotic lesions and fail to fill properly if plants are infected at an early age (Hagedorn and Walker 1949). While symptoms may closely resemble those caused by AMV, plants infected with PeSV generally do not show chlorosis in leaves as observed in plants infected by AMV. The effects of PeSV are compounded when plants are also infected with AMV.

PeSV has only been reported to occur in the United States (Zaumeyer 1938,

Hagedorn and Walker 1949), Canada, and Germany (Bos 1973). The host range is relatively small and affects lentil, faba bean, chickpea (Kaiser *et al.,* 1993), and the forage legumes alfalfa and clover. The virus is transmitted in a non-persistent manner by the pea aphid. Alfalfa is considered to be the most important perennial reservoir source of PeSV (Hampton and Webster 1983). Several strains of the virus have been identified and some may be transmitted more efficiently than others. Kim and Hagedorn (1959) demonstrated that pea aphids transmitted the Wisconsin strain of PeSV at a low percentage rate, while a western strain from Idaho could be readily transmitted. Data is lacking on transmission variability by different biotypes of the pea aphid.

Sources of resistance to PeSV are limited and no pea cultivars currently contain resistance or tolerance to the virus. Four PI accessions including PI 193845, 203066, 212029, and 261677 were found by Ford and Baggett (1965b) to be resistant to PeSV while Hagedorn (1968) reported resistance in PI lines 116944, 140297, and 195405. Two breeding lines, OSU B442-15 and OSU B445-66, derived in Oregon have been demonstrated to exhibit moderate resistance. Although PeSV continues to be responsible for losses in pea primarily in North America, virtually no research is being conducted to develop pea cultivars containing resistance to the virus.

3.3.5 Red clover vein mosaic

Red clover vein mosaic virus (RCVMV) is also a *Carlavirus* and is an important virus of pea and other legume crops. The virus is also referred to as "pea stunt" and was first described in pea in Wisconsin by Hagedorn and Walker (1949), although it was reported earlier on red clover by Osborne (1937). The natural host range is limited to leguminous hosts including clover, alfalfa, faba bean, and more recently chickpea (Larsen *et al.,* 1993) and lentil (Larsen and Myers 1998). Bean (*Phaseolus vulgaris* L.) is not generally considered a host to RCVMV, although Bos *et al.,* (1972) identified a strain that induced vein necrosis. Infection in pea by RCVMV results in symptoms of vein clearing and mosaic in leaves, proliferation of axillary buds, and a pronounced shortening of internodes that results in terminal rosetting. Pod formation can be severely affected when plants are infected before flower set. Plants are often severely stunted or killed if infected at an early stage of growth.

The chief vector of RCVMV is the pea aphid and the virus is transmitted in a nonpersistent manner. Alternate hosts on which the aphid can acquire the virus include alfalfa and several species of clover, in particular, red clover (*Trifolium pratense* L.), crimson clover (*T. incarnatum* L.), and alsike clover (*T. hybridum* L.) (Hagedorn and Hanson 1951). Strains or isolates of the virus have been reported (Bos et al 1972, Hampton *et al.,* 1974) but few have been well characterized. RCVMV was reported earlier to be closely related to PeSV (Veerisetty and Brakke 1977). However, recent evidence has shown that the two viruses are instead very distantly related serologically. Alignment of the putative coat protein amino acid sequences of RCVMV and PeSV resulted in identities of only 53% (Larsen, R. C. *unpublished*).

Suitable germplasm with tolerance to RCVMV is currently limited to only six PI accessions (Hagedorn 1968). RCVMV in pea frequently occurs as a virus complex with PEMV where peas are grown in the western region of Oregon and Washington State in

the U.S. As a result of the virus pressure in these areas, breeding lines OSU 663, OSU 668, and OSU 677 were developed that are resistant to RCVMV and PEMV (Baggett 1984).

3.3.6 Pea seedborne mosaic

Pea seedborne mosaic virus (PSbMV) belongs to the genus *Potyvirus* in the family *Potyviridae*. The virus was reported first in Europe in 1966 (Musil 1966) and subsequently in Japan in 1967 (Inouye 1967). PSbMV was then discovered in the United States in 1968 (Stevenson and Hagedorn 1969) where the virus was demonstrated to be seed transmissible at levels of 10%. Mink *et al.,* (1969) later reported that the virus produced symptoms on up to 90% of plants germinated from seed under growth chamber conditions at 29° C. The virus was readily distributed around the world due to dissemination of contaminated accessions from the USDA germplasm collection, breeding lines, and commercial cultivar seed. Hampton and Braverman (1979) found that 23% of the 1835 PI lines, originating primarily from India, were infected with PSbMV. Because of the high percentages of contaminated seed, intensive screening assays of all PI lines were carried out in 1975-1976 in an effort to rid the accessions of PSbMV. Pea seed continues to be tested on a regular basis and currently less than 1% of all seed tested is positive for the virus.

Symptoms in susceptible lines may include leaf roll, mosaic, leaf and stem malformation, and stunting, which can be severe depending on the accession or cultivar and environmental conditions. Field plants often display fewer obvious symptoms than plants grown in the greenhouse or in growth chambers (Hampton *et al.,* 1976). A terminal rosetting appearance that arises from reduced internode growth is common. Symptoms are often most severe on plants that emerge from infected seed although symptoms can also be severe if spread to young healthy seedlings in the field. Pods on infected plants are frequently distorted or misshapen. Seed may exhibit cracked seed coats although this condition may occur as a result of non-virus-related physiological conditions as well. Stevenson and Hagedorn (1970) observed that cracks in the seed coat transmitted PSbMV at 33%, compared to 4% in seed with normal intact seed coats.

The host range of PSbMV is fairly extensive and infects 47 plants in 12 families (Aapola *et al.,* 1974). The virus is seed-borne in pea, lentil, and faba bean, but it is not known to be seed-borne in chickpea. Plant species useful as diagnostic indicators for PSbMV include *Chenopodium quinoa* Willd., and *C. amaranticolor* Corte and Reyn. (Hampton and Mink 1975). *Phaseolus vulgaris* is also generally included as a negative non-host control. PSbMV exists as several strains or pathovars. Alconero *et al.,* (1986) reported strains P-1 and P-4 infecting pea, and the lentil strain L, which produces more severe symptoms in pea than do P-1 and P-4. Additional isolates have been reported by Hampton *et al.,* (1981), who demonstrated that seven different isolates could be separated on a set of pea germplasm differentials. PSbMV is readily detected by ELISA in seed coats, but seed transmission typically does not occur from these seed. Seed transmission results from direct invasion of immature embryos where the virus multiplies in the embryonic tissues and persists during seed maturation (Wang and Maule 1992).

PSbMV is transmitted in a nonpersistent manner primarily by *Acyrthosiphon pisum* Harris, *Myzus persicae* Sulz., and *Aphis craccivora* Koch. However, 18 other aphid species have been reported to transmit the virus (Khetarpal and Maury 1987). The potato aphid (*Macrosiphon euphorbiae* Thom.) was reported by Gonzales and Hagedorn (1971) to be the most efficient vector, with the pea aphid being the least efficient, when transmitting a single sub-isolate of PSbMV. The aphid can acquire the virus in five minutes or less.

Resistance to PSbMV was characterized by Hagedorn and Gritton (1973) as being determined by a single recessive gene *sbm*. The gene *sbm-1* was found to confer resistance to the P-1 pathotype and is linked with *wlo* on chromosome 6 (Gritton and Hagedorn 1975). Provvidenti and Alconero (1988a) demonstrated that two recessive genes *sbm-2*, linked to *mo* on chromosome 2, and *sbm-3* conferred resistance to pathotypes L and L-1. A fourth gene, *sbm-4*, is monogenic recessive and confers resistance to the P-4 pathotype (Provvidenti and Alconero 1988b). Several commercial cultivars with resistance to PSbMV are available. Molecular approaches utilizing techniques for inducing resistance by gene silencing (Jones *et al.,* 1998) and marker-assisted selection (MAS) have been investigated. Timmerman *et al.,* (1993) demonstrated that marker GS185 could be used to identify *sbm-1* in plant breeding programs and that a strong association exists with a specific hybridization pattern and resistance to PSbMV.

3.3.7 Control of virus diseases

Preventative measures are the most effective methods of virus control in pea fields. Growers must be diligent in the use of seed known to be free of PSbMV. Once introduced into a field, it can be spread rapidly if aphids are abundant. Planting near alfalfa fields or areas with high clover populations should be avoided wherever possible. As perennial crops, both alfalfa and clover can serve as reservoirs of many viruses and their insect vectors that infect pea.

Reduction of local aphid populations can temporarily reduce the spread of virus. Vectors are capable of transmitting viruses for varying time periods that are categorized as persistent, semi-persistent, and non-persistent transmission. Each type of transmission is defined by the time required for the vector to acquire the virus once in contact with the infected plant, and the length of time a vector then remains viruliferous. Persistently-transmitted viruses (such as BLRV) can be transmitted by the aphid for four days but the aphid can frequently remain viruliferous for longer periods up to the life of the vector. Non-persistently transmitted viruses, also considered stylet-borne, are only transmitted for 3-4 hours or less before they must re-acquire the virus. Non-persistently transmitted viruses include AMV, PeSV, RCVMV, PSbMV, and most other aphid-transmitted viruses of pea. It should be mentioned that time periods for levels of persistence and non-persistence are approximate and may vary by the aphid type and dynamics or environmental conditions. For example, the ability of an aphid to transmit a stylet-borne virus may be reduced to only 15 minutes if the aphid is actively probing plants compared to one that is inactive after acquisition.

Movement of viruses into a pea field by aphids can be influenced by several

factors. When an aphid enters a field it seeks a plant on which to feed. During this process, it may briefly probe several plants before settling down. It is during this period of initial probing that nonpersistent virus transmission occurs and this phenomenon is referred to as primary virus spread (Kennedy 1976). The aphid may have originated from other fields or hosts in the area. If the vector is not controlled and is allowed to colonize plants, a secondary population and subsequent spread of the virus may occur from plant to plant within the field. In the case of nonpersistently transmitted viruses, transient non-colonizing aphids may also be responsible for secondary spread. Secondary spread may appear in the field as small groups or loci of infected plants. Insecticides can be used to control the spread of plant viruses but must be used judiciously. Overuse is harmful to the environment and may not be economically viable. In addition, their application for control of nonpersistently-transmitted viruses is generally not effective, but can be effective in the control of viruses transmitted in a persistent manner due to the increased acquisition, latent, and incubation periods necessary for the aphid to place the virus in or near the phloem area (Jayasena and Randles 1985). Although the aphids eventually die from insecticide exposure, short-term plant-to-plant spread is increased after an insecticide application because of increased vector agitation.

The use of resistant or tolerant pea cultivars is by far the most effective method for managing losses due to viruses. Numerous commercial cultivars, breeding lines and PI accessions are available or have been identified with various levels of resistance to specific viruses. By far the most progress in incorporating resistance has been made for BLRV, PEMV and PSbMV. Success has been limited for PeSV and RCVMV because few PI accessions with resistance or tolerance have been identified. Resistance can break down because of the emergence of new virus strains that can overcome identified resistance or tolerance. Plant breeders and virologists must continue to work together so that knowledge of new virus strains and sources of resistance can be effectively used together to hold field losses to a minimum.

4. Conclusions

This chapter has described several major diseases affecting peas. Certainly, not all diseases have been discussed in this relatively small amount of space. There are many other minor diseases as well as diseases that may be of significant local importance in pea crops that have not been discussed here. In addition, new diseases will likely be discovered or introduced that will affect peas. For example, a new disease of peas has recently been discovered in the Pacific Northwest of the U.S. *Rhizoctonia oryzae* was recently isolated from pea roots (Paulitz 2002). This newly recognized disease might become important as peas are grown in rotation with wheat and barley. *R. oryzae* is virulent on both of these cereal crops as well as on pea. Work is currently in progress to elucidate the etiology, epidemiology and importance of this novel disease. Pea disease management is a moving target and agricultural researchers, breeders, extension agents and growers must continually adjust disease management strategies to adapt to new developments.

5. References

Aapola, A.A., Knesek, J.E., and Mink, G.I. 1974. The influence of inoculation procedure on the host range of pea seed-borne mosaic virus. Phytopathology, 64:1003-1006.

Alconero, R., Provvidenti, R., and Gonsalves, D. 1986. Three pea seedborne mosaic virus pathotypes from pea and lentil germplasm. Plant Disease, 70:783-786.

Alexopoulos, C.J., Mims, C.W. and Blackwell, M. 1996. Introductory Mycology, fourth edition, John Wiley & Sons, Inc, New York. 868 p.

Anderson, N.A. 1982. The genetics and pathology of *Rhizoctonia solani*. Annual Review of Phytopathology, 20:329-347.

Arnold, D.L., Athey-Pollard, A., Gibbon, M.J., Taylor, J.D. and Vivian, A. 1996. Specific oligonucleotide primers for the identification of *Pseudomonas syringae* pv. *pisi* yield one of two possible DNA fragments by PCR amplification: Evidence for phylogetic divergence. Physiological and Molecular Plant Pathology, 49:233-245.

Ashby, J. W. 1984 Bean leafroll virus. Descriptions of Plant Viruses, No. 286, Commonwealth Mycological Institute, Kew, Surrey. UK. 5p.

Ashby, J.W., and Bos, L. 1979. Bean (pea) leafroll virus. Bean Improvement Cooperative. p. 22.

Baggett, J.R. 1984. Cultivar differences in susceptibility to *Ascochyta* stem blight, enation mosaic, and red clover vein mosaic. Pisum Newsletter, 16:4-5.

Baggett, J.R., and Hampton, R.O. 1991. Inheritance of viral bean leafroll tolerance in peas. Journal of the American Society of Horticultural Science, 116:728-731.

Barker, K.R., Carter, C.C., and Sasser, J.N. 1985. An advanced treatise on *Meloidogyne*. Vol II: Methodology. North Carolina State University Graphics, Raleigh, USA. pp. 223.

Bath, J.E. and Tsai, J.H. 1969. The use of aphids to separate two strains of pea enation mosaic virus. Phytopathology, 59:1377-1380.

Bevan, J.R., Taylor, J.D., Crute, I.R. Hunter, P.J. and Vivian, A. 1995. Genetics of specific resistance in pea (*Pisum sativum*) cultivars to seven races of *Pseudomonas syringae* pv. *pisi*. Plant Pathology, 44:98-108.

Biddle, A.J. 2001. Sclerotinia white mold. In: "Compendium of Pea Diseases" (eds. Kraft, J.M. and Pfleger, F.L.). American Phytopathological Society Press, St. Paul, MN, pp. 30-31.

Biddle, A.J. and Yeatman, C. 1986. Control of *Botrytis cinerea* in combining peas. In: "Pests and Diseases, Vol. 3". British Crop Protection Council, Surrey, U.K., pp. 1021-1025.

Bisby, G.R. 1918. A *Fusarium* disease of garden peas in Minnesota. Phytopathology, 8:77.

Blanchette, B.L., and Auld, D.L. 1978. Screening field peas for resistance to white mold. Crop Science, 18:977-979.

Bol, J.F. and Jaspers, E.M.J. 1994. Alfalfa mosaic virus and ilarviruses. In: "Encyclopedia of Virology", Vol. 1. (eds. Webster, R.G. and Granoff, A.) Academic Press, San Diego, CA, pp. 30-35.

Bos, L. 1973. Pea streak virus. Descriptions of Plant Viruses, No. 112. Commonwealth Mycological Institute, Kew, Surrey, UK. 4p.

Bos, L., Maat, D. Z. and Markov, M. 1972. A biologically highly deviating strain of red clover vein mosaic virus usually latent in pea *(Pisum sativum* L.) and its differentiation from pea streak virus. Netherlands Journal of Plant Pathology, 78:125-152.

Bretag, T.W. 1985. Chemical control of *Ascochyta* blight of field peas. Australasian Plant Pathology, 14:42-43.

Bretag, T.W. 1989. Resistance of pea cultivars to *Ascochyta* blight caused by *Mycosphaerella pinodes*, *Phoma medicaginis* and *Ascochyta pisi*. Annals of Applied Biology, 114:156-157.

Bretag, T.W., Keane, P.J. and Price, T.V. 1995a. Effect of *Ascochyta* blight on the grain yield of field peas (*Pisum sativum* L.) grown in southern Australia. Australian Journal of Experimental Agriculture, 35:531-536.

Bretag, T.W., Price, T.V. and Keane, P.J. 1995b. Importance of seed-borne inoculum in the etiology of the *Ascochyta* blight complex of field peas (*Pisum sativum* L.) grown in Victoria. Australian Journal of Experimental Agriculture, 35:525-530.

Bretag, T.W., Keane, P.J. and Price, T.V. 2000. Effect of sowing date on the severity of *Ascochyta* blight in field peas (*Pisum sativum* L.) grown in the Wimmera region of Victoria. Australian Journal of Experimental Agriculture, 40:113-1119.

Butler, L.D. and Fenwick, H.S. 1970. Austrian winter pea, a new host of *Pseudomonas syringae*. Plant Disease Reporter, 54:467-470.

Chowrira, G.M., Cavileer, T.D., Gupta, S.K., Lurquin, P.F. and Berger, P.H. 1998. Coat protein-mediated resistance to pea enation mosaic virus in transgenic *Pisum sativum* L. Transgenic Research, 7:265-71.

Clulow, S.A., Mathews, P. and Lewis, B.G. 1991. Genetic analysis of resistance to *Mycosphaerella pinodes* in pea seedlings. Euphytica, 58:183-189.

Cockbain, A.J. and Gibbs, A.J. 1973. Host range and overwintering sources of bean leafroll and pea enation mosaic viruses in England. Annals of Applied Biology, 73:177-187.

Coyne, D.P., Steadman, J.R. and Anderson, F.N. 1974. Effect of modified plant architecture of great northern dry bean varieties (*Phaseolus vulgaris*) on white mold severity and components of yield. Plant Disease Reporter, 58: 379-382.

Crampton, M.J. and Watts, L.E. 1968. Genetic studies of pea leaf-roll (top-yellows) virus resistance in *Pisum sativum*. New Zealand Journal of Agricultural Research, 11:771-783.

Crill, P., Hagedorn, D.J. and Hanson, E.W. 1970. Incidence and effect of alfalfa mosaic virus on alfalfa. Phytopathology 60, 1432-1435.

D'Arcy, C. J., Martin, R.R. and Torrance, L. 1988. Monoclonal antisera for detection and diagnosis of luteovirus infections. Phytopathology, 78: 1537.

Demler, S.A. and de Zoeten, G.A. 1991. The nucleotide sequence and luteovirus-like nature of RNA 1 of an aphid non-transmissible strain of pea enation mosaic virus. Journal of General Virology, 72:1819-1834.

Demler, S.A., Rucker, D.G. and de Zoeten, G.A. 1993. The chimeric nature of the genome of pea enation mosaic virus: the independent replication of RNA 2. Journal of General Virology, 74:1-14.

Demler, S.A. and de Zoeten, G.A. 1994. Pea enation mosaic virus. In: "Encyclopedia of Virology" (eds. Webster, R.G. and Granoff, A.) Acedemic Press, San Diego, USA, pp. 1083-1089.

Demler, S.A., de Zoeten, G.A., Adam, G. and Harris, K.F. 1996. In: "The Plant Viruses, Vol. 5, Polyhedral Virions and Bipartite RNA Genomes", (eds. Harrison B.D. and Murant, A.F.) Plenum Press, New York, USA, pp. 303-304.

Di Vito, M. 1991. The pea cyst nematode, *Heterodera goettingiana*. Nematology Circular No. 188. Florida Department of Agriculture and Consumer Services, Division Plant Industry, 3 p.

Di Vito, M. and Greco, N. 1986. The pea cyst nematode. In: "Cyst Nematodes. Series A: Life Sciences", Vol. 121 (eds. Lamberti, F., and Taylor, C. E.) Plenum Press, New York, USA, pp. 321-332.

Dixon, G.R. 1978a. Downy mildews of peas and beans. In: "The Downy mildews" (ed. Spencer, D.M.) Academic Press, London, pp.487-511.

Dixon, G.R. 1978b. Powdery mildews of vegetable and allied crops. In: "Powdery Mildews" (ed. Spencer, D.M.) Academic Press, London, pp. 502-506.

Domier, L.L., McCoppin, N.K., Larsen R.C. and D'Arcy, C.J. 2002. Nucleotide sequence shows that bean leafroll virus has a Luteovirus-like genome organization. Journal of General Virology, 83:1791-1798.

Drijfhout, E. 1968. Testing for pea leafroll virus and inheritance of resistance in peas. Euphytica, 17:224-235.

Edwardson, J.R. and Christie, R.G. 1991. Handbook of viruses infecting legumes. CRC Press, Inc., Boca Raton, FL. 504 p.

Fallon, R.E., Sutherland, P.W. and Hallett, I.C. 1989. Morpholgy of *Erysiphe pisi* on leaves of *Pisum sativum*. Canadian Journal of Botany, 67:3410-3416.

Fallon, R.E. and Sutherland, P.W. 1996. *Peronospora viciae*: Morphology of asexual reproductive structures. Mycologica, 88:473-483.

Faris-Mokaiesh, S., Boccara, M., Denis, J.B., Derrien, A. and Spire, D. 1996. Differentiation of the "*Ascochyta* complex" fungi of pea by biochemical and molecular markers. Current Genetics, 29:182-190.

Farr, D., Bills, G.F., Chamuris, G.P. and Rossman, A.Y. 1989. Fungi on Plants and Plant Products in the United States. The American Phytopathological Society Press, St. Paul, USA. 1252 p.

Ford, R.E. and Haglund, W.A. 1963. *Botrytis cinerea* blight of peas associated with senescent blossoms in north-western Washington. Plant Disease Reporter, 47:483-485.

Ford, R.E. and Baggett, J.R. 1965a. Relative severity of legume viruses in peas measured by plant growth reduction. Plant Disease Reporter, 49:627-629.

Ford, R.E. and Baggett, J.R. 1965b. Reactions of plant introduction lines of *Pisum sativum* to alfalfa mosaic, clover yellow mosaic, and pea streak viruses, and powdery mildew. Plant Disease Reporter, 49:787-789.

Fritz, V.A., Allmaras, A.A., Pfleger, F.L. and Davis, D.W. 1995. Oat residue and soil compaction influences on common root rot (*Aphanomyces euteiches*) of peas in a fine-textured soil. Plant and Soil, 171:325-244.

Fuller, P.A., Steadman, J.R., and Coyne, D.P. 1984. Enhancement of white mold avoidance and yield in dry bean by canopy elevation. HortScience, 19:78-79.

Gonzales, L.C., and Hagedorn, D.J. 1971. The transmission of pea seed-borne mosaic virus by three aphid species. Phytopathology, 61:825-828.

Greco, N., Ferris, H., and Brandonisio, A. 1991. Effect of *Heterodera goettingiana* populations on the yield of pea, broad bean and vetch. Revue de Nematologie, 14:619-624.

Gritton, E.T. and Hagedorn, D.J. 1975. Linkage of the genes sbm and wlo in peas. Crop Science, 15:447-448.

Grünwald, N. J., Hu, S. and Van Bruggen, A.H.C. 2000a. Short-term cover crop decomposition in organic and conventional soils: Characterization of soil, C, N, microbial and plant pathogen dynamics. European Journal of Plant Pathology, 106:37-50.

Grünwald, N. J., Hu, S. and Van Bruggen, A.H.C. 2000b. Short-term cover crop decomposition in organic and conventional soils: Soil microbial and nutrient cycling indicator variables associated with different levels of soil suppressiveness to *Pythium aphanidermatum*. European Journal of Plant Pathology, 106:51-60.

Grünwald, N. J., Coffman, V.A. and Kraft, J.M. 2003. Sources of resistance to *Fusarium* root rot in the *Pisum* core collection. Plant Disease, in press.

Grondeau, C., Mabiala, A., Ait-Oumeziane, R. and Samson, R. 1996. Epiphytic life is the main characteristic of the life cycle of *Pseudomonas syringae* pv. *pisi*, pea bacterial blight agent. European Journal of Plant Pathology, 102:353-363.

Gupta, S.K. and Shyam, K.R. 2000. Post-infection activity of ergosterol biosynthesis inhibiting fungicides against pea rust. Journal of Mycology and Plant Pathology, 30:414-415.

Gurung, A.M., Pang, E.C.K. and Taylor, P.W.J. 1999. Examination of *Pisum* and *Lathyrus* species as sources of *Ascochyta* blight resistance for field pea (*Pisum sativum*). Australasian Plant Pathology, 31:41-45.

Hagedorn, D.J. 1968. Disease reaction to *Pisum sativum* plant introductions to three legume viruses. Plant Disease. Reporter, 52:160-162.

Hagedorn, D. J., and Walker, D.J. 1949. Wisconsin pea streak. Phytopathology, 39:837-847.

Hagedorn, D.J. and Hanson, E.W. 1951. A comparative study of the virus causing Wisconsin pea

stunt and red clover vein mosaic. Phytopathology, 41:813-819.

Hagedorn, D.J. and Gritton, E.T. 1973. Inheritance of resistance to the pea seed-borne mosaic virus. Phytopathology, 63:1130-1133.

Haglund, W.A. 1989. A rapid method for inoculating pea seedlings with *Fusarium oxysporum* f. sp. *pisi*. Plant Disease, 73:457-458.

Haglund, W.A. and Kraft, J.M. 2001. *Fusarium* wilt. In: "Compendium of Pea Diseases and Pests", (eds. Kraft, J.M., and Pfleger, F.L.) American Phytopathological Society Press, St Paul, USA, pp. 14-16.

Hall, R. 1996. Principles and practice of managing soilborne diseases. APS Press, St. Paul, USA. 330 p.

Hampton, R.O. 1983. Pea leafroll in northwestern pea seed. Plant Disease, 67:1306-1310.

Hampton, R.O., Knesek, J.E. and Mink, G.I. 1974. Particle-length variability of the pea seedborne mosaic virus. Phytopathology, 64:1358-1363.

Hampton, R.O. and Mink, G.I. 1975. Pea seed-borne mosaic virus. In: "Descriptions of Plant Viruses", No. 146, Commonwealth Mycological Institute, Kew, Surrey, UK.

Hampton, R.O., Mink, G.I., Hamilton, R.I., Kraft, J.M. and Muelbauer, F.J. 1976. Occurrence of pea seedborne mosaic virus in North American pea breeding lines, and procedures for its elimination. Plant Disease Reporter, 60:455-458.

Hampton, R.O. and Braverman, S.W. 1979. Occurrence of pea seedborne mosaic virus and new virus immune germplasm in the plant introduction collection of *Pisum sativum*. Plant Disease Reporter, 63:95-99.

Hampton, R.O., Mink, G.I., Bos, L., Inouye,T., Musil, M. and Hagedorn, D. 1981. Host differentiation and serological homology of pea seed-borne mosaic virus isolates. Netherlands Journal of Plant Pathology, 87:1-10.

Hampton, R.O. and Weber, K.A. 1983. Pea streak virus transmission from alfalfa to peas: Virus-aphid and virus-host relationships. Plant Disease, 67:305-307.

Harman, G.E. and Dillard, H.R. 2001. *Thielaviopsis* root rot. In: "Compendium of Pea Diseases and Pests", (eds. Kraft, J.M., and Pfleger, F.L.) American Phytopathological Society Press, St Paul, USA, pp. 17-18.

Hoitink, H.A.J., Hagedorn, D.J. and McCoy, E. 1967. Survival, transmission, and taxonomy of *Pseudomonas syringae* Van Hall, the causal organism of bacterial brown spot of bean (*Phaseolus vulgaris* L.). Canadian Journal of Microbiology, 14:437-441.

Hollaway, G.J. and Bretag, T.W. 1997. Survival of *Pseudomonas syringae* pv. *pisi* in soil and on pea trash and their importance as a source of inoculum for a following field pea crop. Australian Journal of Experimental Agriculture, 37:369-375.

Hollaway, G.J., Gillings, M.R. and Fahy, P.C. 1997. Use of fatty acid profiles and repetitive element polymerase chain reaction (PCR) to assess the genetic diversity of *Pseudomonas syringae* pv. *pisi* and *Pseudomonas syringae* pv. *syringae* isolated from field peas in Australia. Australasian-Plant-Pathology, 26:98-108.

Hubbeling, N. 1975. Resistance of peas to downy mildew and dinstinction of races of *Peronospora pisi* Syd. Mededelingen van de Faculteit Landbouwwetenschappen Rijksuniversiteit Gent, 40:539-543.

Inglis, D.A. 2001. Diseases caused by nematodes. In: "Compendium of Pea Diseases and Pests", (eds. Kraft, J.M., and Pfleger, F.L.) American Phytopathological Society Press, St Paul, USA, pp. 18-21.

Inouye, T. 1967. A seed-borne mosaic virus of pea. Annals of the Phytopathological Society of Japan, 33:38-42.

Jayasena, K.W. and Randles, J.W. 1985. The effect of insecticides and a plant barrier row on aphid populations and the spread of bean yellow mosaic potyviurs and subterranean clover red leaf luteovirus in *Vicia faba*. South Australian Annals of Applied Biology, 107:355-364.

Jensen, H.J. 1972. Nematode pests of vegetable and related crops. In: "Economic Nematology", (ed. Webster, J M.) Academic Press, New York, USA, pp. 377-409.

Johnson, A.W. and Fassuliotis, G. 1984. Nematode parasites of vegetable crops. In: "Plant and Insect Nematodes" (ed. Nickle, R.), Marcel Dekker, New York, USA, pp.323-372.

Jones, A.L.L., Johansen, I.E., Bean, S.J., Bach, I. and Maule, A.J. 1998. Specificity of resistance to pea seed-borne mosaic potyvirus in transgenic peas expressing the viral replicase (Nlb) gene. Journal of General Virology, 79:3129-3137.

Kaiser, W.J. 1972. Diseases of food legumes caused by pea leafroll virus in Iran. FAO Plant Protection Bulletin, 20:127-133.

Kaiser, W.J., Klein, R.E., Larsen, R.C. and Wyatt, S.D. 1993. Chickpea wilt incited by pea streak carlavirus. Plant Disease, 77:922-926.

Kennedy, G.G. 1976. Host plant resistance and the spread of plant viruses. Environmental Entomology, 5:827-832.

Khetarpal, R.K. and Maury, Y. 1987. Pea seed-borne mosaic virus: a review. Agronomie, 7:215-224.

Kim, W.S. and Hagedorn, D.J. 1959. Streak inciting viruses of canning pea. Phytopathology, 49:656-664.

Kraft, J.M. 1969. Chickpea, a new host of *Fusarium solani* f. sp. *pisi*. Plant Disease Reporter, 53:110-111.

Kraft, J.M. 1975. A rapid technique for evaluating pea lines for resistance to *Fusarium* root rot. Plant Disease Reporter, 59:1007-1011.

Kraft, J.M. 1991. Pea diseases. Aspects of Applied Biology, 27:313-319.

Kraft, J.M. 1994. *Fusarium* wilt of peas. Agronomie, 14: 561-567.

Kraft, J.M. 2001. Foliar diseases of local importance. In: "Compendium of Pea Diseases and Pests", (eds. Kraft, J.M., and Pfleger, F.L.) American Phytopathological Society Press, St Paul, USA, p. 32.

Kraft, J.M., and Roberts, D.D. 1970. Resistance in peas to *Fusarium* and Pythium root rot. Phytopathology, 60:1814-1817.

Kraft, J.M. and Haglund, W.A. 1978. A reappraisal of the race classification of *Fusarium oxysporum* f. sp. *pisi*. Phytopathology, 68:273-275.

Kraft, J.M., Haware, M.P. and Hussein, M.M. 1988. Root rot and wilt disease of food legumes. In: "World Crops: Cool Season Food Legumes" (ed. Summerfield, R.J.), Kluwer Academic Press, Boston, U.S.A, pp. 565-575.

Kraft, J. M., Marcinkowska, J. and Muehlbauer, F.J. 1990. Detection of *Aphanomyces euteiches* in field soil from northern Idaho by a wet-sieving/baiting technique. Plant Disease, 74: 716-718.

Kraft, J.M., Larsen, R.C. and Inglis, D.A. 1996. Diseases of Pea. In: "The Pathology of Food and Pasture Legumes" (ed. Allen, D.) CAB International, Wallingford, UK, pp. 325-370.

Larsen, R.C., and Kaiser, W.J., and Wyatt, S.D. 1993. First report of a virus disease of chickpea caused by a strain of red clover vein mosaic virus. Plant Disease, 77:922.

Larsen, R.C., Kaiser, W.J. and Klein, R.E. 1996. Alfalfa, a non-host of pea enation mosaic virus in Washington State. Canadian Journal of Plant Science, 76:521-524.

Larsen, R.C. and Myers, J.R. 1998. First report of red clover vein mosaic carlavirus naturally infecting lentil. Plant Disease, 82:1064.

Larsen, R.C. and Webster, D.M. 1999. First report of bean leafroll luteovirus infecting pea in Italy. Plant Disease, 83:399.

Legard, D.E. and Hunter, J.E. 1990. Pathogenicity on bean of *Pseudomonas syringae* pv. *syringae* recovered from the phylloplane of weeds and from bean crop residue. Phytopathology, 80:938-942.

Lloyd, A.B. and Lockwood, J.L. 1963. Effect of soil temperature, host variety, and fungus strain

on *Thielaviopsis* root rot of peas. Phytopathology, 53:329-331.

Mai, W.F., Bloom, J.R. and Chen, T.A. 1977. Biology and ecology of the plant parasitic nematode *Pratylenchus penetrans*. Pennsylvania State University College of Agriculture Agricultural Experiment Stations Bulletin 815.

Malandrin, L. and Samson, R. 1998. Isozyme analysis for the identification of *Pseudomonas syringae* pathovar *pisi* strains. Journal of Applied Microbiology, 84:895-902.

Mansfield, P.J., Wilson, D.W., Heath, M.C. and Saunders, P.J. 1997. Development of pea bacterial blight caused by *Pseudomonas syringae* pv. *pisi* in winter and spring cultivars of combining peas (*Pisum sativum*) with different sowing dates. Annals of Applied Biology, 131:245-258.

Martin, F.N. and Loper, J.E. 1999. Soilborne plant diseases caused by *Pythium* spp.: ecology, epidemiology and prospects for biological control. Critical Reviews in Plant Science, 18:111-181.

Martin, R.R. and D'Arcy, C.J. 1990. Relationships among luteoviruses based on nucleic acid hybridization and serological studies. Intervirology, 31:23-30.

Matuo, T. and Snyder, W.C. 1972. Host virulence and the hypomyces stage of *Fusarium solani* f. sp. *pisi*. Phytopathology, 62:731-735.

McCoy, R.J. and Kraft, J.M. 1984. Resistance in *Pisum* to epicotyl rot caused by *Rhizoctonia solani*. Plant Disease, 68:491-493.

McWhorter, F.P. and Cook, W.C. 1958. The hosts and strains of pea enation mosaic virus. Plant Disease Reporter, 42:51-60.

Michall, S.H., Abd El-Rehim, M.A., Abo Taleb, E.M. and Metwally, S.M. 1998. Effect of level of *Ascochyta* seed-borne infection on pea plants grown in cultivated and virgin soils. Seed Science and Technology, 26:125-130.

Mink, G. I., Kraft, J., Knesek, J. and Jafri, A. 1969. A seed-borne virus of peas. Phytopathology, 59:1342-1343.

Moussart, A., Tivoli, B., Lemarchand, E., Deneufbourg, F., Roi, S. and Sicard, G. 1996. Role of seed infection by the *Ascochyta* blight pathogen of dried pea (*Mycosphaerella pinodes*) in seedling emergence, early disease development and transmission of the disease to aerial plant parts. European Journal of Plant Pathology, 104:93-102.

Musil, M. 1966. Über das Vorkommen des Viruses des Blattrollens der Erbse in der Slowakei (Vorläufige Mitteilung). Biologia (Bratislavia), 21:133-138.

Nasir, M. and Hoppe, H.H. 1991. Studies on pathotype differentiation within *Mycosphaerella pinodes* (Berk. & Bloxam) Vestergren, a component of the *Ascochyta*-disease-complex of peas (*Pisum sativum* L.). Journal of Plant Disease Protection, 98:619-626.

Osborne, H.T. 1935. Incubation period of pea mosaic in the aphid, *Macrosiphum pisi*. Phytopathology, 25:160-177.

Osborne, H.T. 1937. Vein mosaic virus of red clover. Phytopathology, 27:1051-1058.

Pal, A.B., Brahmappa, Rawal, R.D. and Ullasa, B.A. 1980. Field resistance of pea germplasm to powdery mildew (*Erysiphe polygoni*) and rust (*Uromyces fabae*). Plant Disease, 64:1085-1086.

Pankova, I. and Kokoskova, B. 1999. Serological and biochemical distinguishing of *Pseudomonas syringae*: pathovars on peas. Plant Protection Science, 35:79-84.

Parmeter, J.R. 1970. *Rhizoctona solani*, biology and pathology. University of California Press, Berkeley, USA. 255 p.

Paulitz, T.C. 2002. First report of *Rhizoctonia oryzae* on pea. Plant Disease, 86:442.

Pegg, G.F. and Mence, M.J. 1972. The biology of *Peronospora viciae* on pea: The development of local systemic infections and their effect on vining yield. Annals of Applied Biology, 71:19-31.

Peters, D. 1982. Pea enation mosaic. In: "Descriptions of Plant Viruses", No. 257, Commonwealth

Mycological Institute, Kew, Surrey, UK.

Pilet-Nayel, M.L., Muehlbauer, F.J., McGee, R.J., Kraft, J.M., Baranger, A. and Coyne, C.J. 2002. A major and minor QTLs control field aphanomyces root rot resistance in pea. Theoretical Applied Genetics, in press.

Provvidenti, R. and Alconero, R. 1988a. Inheritance of resistance to a lentil strain of pea seed-borne mosaic virus in *Pisum sativum*. Journal of Heredity, 79:45-47.

Provvidenti, R. and Alconero, R. 1988b. Inheritance of resistance to a third pathotype of pea seed-borne mosaic virus in Pisum sativum. Journal of Heredity, 79:76-77.

Quantz, L. and Volk, J. 1954. Die Blattrollkrankheit der Ackerbohne und Erbse, eine neue Viruskrankheit bei Leguminosen. Nachrichtenblatt des Deutschen Pflantzenchutzdienstes, 6:177-182.

Reeves, J.C., Hutchins, J.D. and Simpkins, S.A. 1996. The incidence of races of *Pseudomonas syringae* pathovar *pisi* in UK pea (*Pisum sativum*) seed stocks, 1987-1994. Plant Varieties and Seeds, 9:1-8.

Riggs, R.D. and Niblack, T.L. 1993. Nematode pests of oilseed crops and grain legumes. In: "Plant parasitic nematodes in temperate agriculture", (eds. Evans, K., Trudgill, D.L., and Webster, J.M.), CAB International, Wallingford, UK, pp. 209-258.

Roberts, S.J. 1997. Effect of weather conditions on local spread and infection by pea bacterial blight (*Pseudomonas syringae* pv. *pisi*). European Journal of Plant Pathology, 103:711-719.

Roberts, S.J., Reeves, J.C., Biddle, A.J., Taylor, J.D. and Higgins, P. 1991. Prevalence of pea bacterial blight in UK seeds stocks, 1986-1990. Aspects of Applied Biology, 27:327-332.

Roberts, S.J., Phelps, K., McKeown, B.M., Heath, M.C. and Cockerell, V. 1995. Effect of pea bacterial blight (*Pseudomonas syringae* pv. *pisi*)on the yield of spring sown combining peas (*Pisum sativum*). Annals of Applied Biology, 126:61-73.

Roger, C. and Tivoli, B. 1996. Spatio-temporal development of pycnidia and perithecia and dissemination of spores of *Mycosphaerella pinodes* on pea (*Pisum sativum*). Plant Pathology, 45:518-528.

Roger, C., Tivoli, B. and Huber, L. 1998. Effects of temperature and moisture on disease and fruit body development of *Mycosphaerella pinodes* on pea (*Pisum sativum*). Plant Pathology, 48:1-9.

Sackett, W.G. 1916. A bacterial stem blight of field and garden peas. Colorado Agricultural Experimental Station Bulletin, 218:3-43.

Santo, G.S. and Ponti. R.P. 1985. Host suitability and reaction of bean and pea cultivars to *Meloidogyne chitwoodi* and *M. hapla*. Journal of Nematology, 17:77-79.

Sasser, D.A. and Johnson, A.W. 1985. An advanced treatise on *Meloidogyne*. Vol I: Biology and Control. North Carolina State University Graphics, Raleigh, USA. 422 p.

Schroeder, W. T. and Barton, D. W. 1958. The nature and inheritance of resistance to the pea enation mosaic virus in garden pea, *Pisum sativum* L. Phytopathology, 48:628-632.

Schroeder, W.T., Provvidenti, R. and McEwen, F.L. 1959. Pea streaks naturally incited by combinations of viruses. Plant Disease Reporter, 43:1219-1226.

Singh, S.J. and Sokhi, S.S. 1980. Pathogenic variability in *Uromyces viciae-fabae*. Plant Disease, 64:671-672.

Smith, H.G., Barker, I., Brewer, G., Stevens, M. and Hallsworth, P.B. 1996. Production and evaluation of monoclonal antibodies for the detection of beet mild yellowing luteovirus and related strains. European Journal of Plant Pathology, 102:163-169.

Stevenson, W.R. and Hagedorn, D.J. 1969. A new seed-borne virus of peas. Phytopathology, 59:1051-1052.

Stevenson, W.R. and Hagedorn, D.J. 1970. Effect of seed size and condition on transmission of pea seed-borne mosaic virus. Phytopathology, 60:1148-1149.

Stone, A.R. and Course, J.A. 1974. *Heterodera goettingiana*. CIH Descriptions of Plant Parasitic

Nematodes, Set 4, No. 47, Commonwealth Institute of Helminthology, St. Albans, UK.

Sugha, S.K., Chauhan, R.S. and Singh, B.M. 1994. Sensitivity of aeciospores and uredospores of the pea rust pathogen to selected systemic fungicides. Tropical Agriculture, 71:27-30.

Taylor, P.N., Lewis, B.G. and Mathews, P. 1989. Pathotypes of *Peronospora viciae* in Britain. Journal of Phytopathology, 127:100-106.

Thottappilly, G., Ya-Chu, J.K., Hooper, G.R., and Bath, J.E. 1977. Host range, symptomology, and electronmicroscopy of a persistant, aphid-transmitted virus from alfalfa in Michigan. Phytopathology, 67:1451-1459.

Timmerman, G.M., Frew, T.J., Miller, A.L., Weeden, N.F. and Jermyn, W.A. 1993. Linkage mapping of sbm-1, a gene conferring resistance to pea seed-borne mosaic virus, using molecular markers in *Pisum sativum*. Theoretical Applied Genetics, 85:609-615.

Timmerman, G.M., Frew, T.J., Weeden, N.F., Miller, A.L. and Goulden, D.S. 1994. Linkage analysis of er-1, a recessive *Pisum sativum* gene for resistance to the powdery mildew fungus (*Erysiphe pisi* DC.). Theoretical and Applied Genetics, 88:1050-1055.

Tinsley, T.W. 1959. Pea leaf roll, a new virus disease of legumes in England. Plant Pathology, 8:17-18.

Tivoli, B., Beasse, C., Lemarchand, E. and Masson, E. 1995. Effect of *Ascochyta* blight (*Mycosphaerella pinodes*) on yield components of single pea (*Pisum sativum*) plants under field conditions. Annals of Applied Biology, 129:207-216.

Tiwari, K.R., Penner, G.A. and Warkentin, T.D. 1997. Inheritance of powdery mildew resistance in pea. Canadian Journal of Plant Science, 77:307-310.

Toros, S., Schotman, C.Y.L. and Peters, D. 1978. A new approach to measure the LP50 of pea enation mosaic virus in its vector. Virology, 90:235-240.

Tyagi, M.K. and Srivastava, C.P. 1999. Inheritance of powdery mildew and rust resistance in pea. Annals of Biology,15:13-16.

Van Bruggen, A.H.C. and Grünwald, N.J. 1996. Tests for risk assessment of root infection by plant pathogens. In: "Methods for Assessing Soil Quality" (eds. Doran, J.W., and Jones, A.J.), Soil Science Society of America, Madison, WI, U.S.A, pp. 293-310.

Van Bruggen, A.H.C., Grünwald, N.J. and Bolda, M. 1996. Cultural methods and soil nutrient status in low and high input agricultural systems, as they affect *Rhizoctonia* species. In: "*Rhizoctonia* species: Taxonomy, molecular biology, ecology, pathology and disease control" (eds. Sneh, B., Jabaji-Hare, S., Neate, S., Dijst, G.), Kluwer Academic Press, Dordrecht, The Netherlands, pp. 407-421.

Van der Gaag, D.J. and Frinking, H.D. 1997. Survival characteristics of oospore populations of *Peronospora viciae* f. sp. *pisi* in soil. Plant Pathology, 46:978-988.

Van der Plaats-Niterink, A.J. 1981. Monograph of the genus *Pythium*. Studies in Mycology No. 21, Centraalbureau Voor Schimmelcultures, Baarn, The Netherlands. 242p.

Veerisetty, V. and Brakke, M.K. 1977. Differentiation of legume carlaviruses based on their biochemical properties. Phytopathology, 84:226-231.

Viljanen-Rollinson, S.L.H., Gaunt, R.E., Frampton, C.M.A., Fallon, R.E. and McNeil, D.L. 1998. Componenets of quantitative resistance to powdery mildew (*Erysiphe pisi*) in pea (*Pisum sativum*). Plant Pathology, 47137-147.

Wallen, V.R. 1974. Influence of three *Ascochyta* diseases of peas on plant development and yield. Canadian Plant Disease Survey, 54:86-90.

Wang, D. and Maule A.J. 1992. Early embryo invasion as a determinant in pea of the seed transmission of pea seed-borne mosaic virus. Journal of General Virology, 73:1615-20.

Waterhouse, P.M., Gildow, F.E. and Johnstone, G.R. 1988. The luteovirus group. Descriptions of Plant Viruses, No. 339 In: Commonwealth Mycological Institute, Kew, Surrey, UK.

Warkentin, T.D., Rashid, K.Y. and Xue, A.G. 1995. Fungicidal control of *Ascochyta* blight of field pea. Canadian Journal of Plant Science, 76:67-71.

Weeden, N.F. and Provvidenti, R. 1988. A marker locus, Adh-1, for resistance to pea enation mosaic virus in *Pisum sativum*. The Journal of Heredity, 79:128-131.

Williams-Woodward, J.L., Pfleger, F.L., Fritz, V.A. and Allmaras, A.A. 1997. Green manures of oat, rape and sweet corn for reducing common root rot in pea (*Pisum sativum*) caused by *Aphanomyces euteiches*. Plant and Soil, 188:43-48.

Wroth, J.M. 1998. Variation in pathogenicity among and within *Mycosphaerella pinodes* populations collected from field pea in Australia. Canadian Journal of Botany, 76:1955-1966.

Wroth, J.M. 1999. Evidence suggests that *Mycosphaerella pinodes* infection of *Pisum sativum* is inherited as a quantitative trait. Euphytica, 107:193-204.

Xue, A.G. and Warkentin, T.D. 2002. Reactions of field pea varieties to three isolates of *Uromyces fabae*. Canadian Journal of Plant Science, 82: 253-255.

Yu, J., Gu, W.K., Provvidenti, R. and Weeden, N.F.1995. Identifying and mapping two DNA markers linked to the gene conferring resistance to pea enation mosaic virus. Journal of the American Society of Horticultural Science, 120:730-733.

Zaumeyer, W.J. 1938. A streak disease of peas and its relation to several strains of alfalfa mosaic virus. Journal of Agricultural Research 56, 747-772.

Diseases of Pepper and their Management

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Abstract: Diseases of pepper caused by biotic (infectious) and abiotic (non-infectious) agents interfere with the production of pepper. Biotic agents of disease of pepper include fungi, bacteria, nematodes, and viruses. Abiotic disorders include a number of unfavorable cultural or climatic conditions, such as sunlight, nutrient deficiency, and temperature excesses. Diseases affect all parts of the pepper plant including the foliage, stems, roots, fruit, and young seedlings. Fungi and bacteria cause a variety of symptoms such as leaf and fruit spotting, wilting and plant death. Typical symptoms of viral infections are stunting, mottling of foliage and fruit, and small, misshapen fruit. Abiotic disorders such as sunscalding or blossom end rot greatly interfere with the fruit quality and reduces yield. Management of diseases begins prior to planting of the crop and many techniques are used to minimize losses due to disease problems. Control methods include, but are not limited to, the use of cultivars with resistance to diseases, pathogen-free seeds, sterilized equipment for transplant production, rouging and eradication of diseased plants and alternative hosts, field sites that are pathogen-free and isolated from other solanaceous crops, soil fumigation, and application of pesticides and other disease-suppression compounds. Post-harvest disease management techniques include growing healthy pepper plants to produce fruits free from predisposing injuries, using field sanitation practices, avoiding the wounding of fruit, and storing of fruit under optimum environmental conditions.

1. Introduction

Many diseases and disorders can interfere with pepper production. These may have a biotic (living) or abiotic (non-living) origin. Biotic agents are infectious agents that include fungi, bacteria, nematodes, and viruses. Abiotic disorders include a number of unfavorable cultural or climatic conditions, such as temperature extremes, water imbalances, sunlight, nutrients, soil pH, air pollutants and chemicals.

Management of diseases begins prior to planting of the crop. In general, the following practices should be followed to promote plant health and limit losses due to diseases. These practices frequently apply to the control of more than one disease. Recommendations for the control of specific diseases are discussed in the management section in the individual disease sections.

Cultivars of peppers should be selected whenever possible that contain resistance to diseases. Many pathogens including fungi, bacteria, and virus are seed-borne and seeds should be pathogen-free. The location of transplant production sites ideally should be isolated from major production fields of pepper and other solanaceous crops

such as tomato and potato. Transplant production should utilize growing media, trays, and production structures that are pathogen-free. Volunteer plants and weeds that can harbor pathogens must be destroyed. Workers and equipment should be disinfected when moving between houses or from diseased to healthy plants. Diseased plants should be rouged and discarded quickly.

Field sites should be selected that are free from soil-borne diseases. The presence of some pathogens may require crop rotation between plantings. Fields that are isolated from other solanaceous crops are preferred. Fields should be well-drained and free from low lying areas that retain water. Proper irrigation is needed. The soil is prepared by adjusting the pH, if needed, and applying appropriate amounts of fertilizer. Soil fumigation and raised beds covered with plastic mulch are desirable in many situations. Weeds and alternate hosts need to be eliminated. Insect pests must be controlled. For many disease problems, pesticides and other microbicidal compounds can be used preventively in the field and post-harvest. Biological amendments can improve plant vigor and suppress disease.

Post-harvest diseases are managed by growing healthy pepper plants to produce fruits free from predisposing injuries, using field sanitation practices, avoiding the wounding of fruit which creates sites for pathogen ingress, and storing of fruit under optimum environmental, and having sanitary conditions in the packinghouse.

The remainder of the chapter describes many of the important disease problems on pepper. The diseases caused by fungi and bacteria are listed alphabetically in section 2 and 3 respectively. Diseases caused by viruses are listed by genera in section 4. Nematodes are discussed, but their life cycles and management is universal for most crops, therefore nematodes are only briefly presented in section 5. Other sources on nematodes should be consulted for detailed information. Abiotic disease problems are discussed in section 6.

2. Diseases caused by fungi and oomycetes

The diseases of pepper under this head are described in alphabetical order.

2.1 *Alternaria* leaf spot and fruit rot

The fungus, *Alternaria alternata,* (Fr.) Keissler causes fruit rot on pepper. One of the early reports of the disease was the black spot of pepper in 1917 (Melchers and Dale 1917). *A. alternata* is considered weakly pathogenic and fruit rot symptoms occur usually in association with a predisposing injury due to sunscald, frost, or blossom end rot. However, internal mold of fruit develops without any external sign or damage on fruit. *A. solani* Sorauer, (*Macrosporium solani* Ellis & Martin) the causal agent of early blight, is an important disease on tomato and potato and will upon occasion cause a minor leaf spot of foliage and stem lesions on pepper. However, *A. solani* on pepper is generally unimportant and control measures are unnecessary. These fungi are widespread in both the tropics and temperate zones. Affected fruit reached 45% in commercial planting. (Melchers and Dale 1917)

2.1.1 Symptoms

Alternaria fruit rot lesions are slightly sunken, dark brown to black spots that can occur at any location on the fruit. The fungus is evident in the lesion as a dark brown to black growth (Melchers and Dale 1917). The fungus typically infects through wounds such as those made during the harvest and postharvest handling. Natural wounds around the peduncle and calyx are common sites of infection and damage at these locations may extend to the interior of the fruit.

Internal mold of the fruit causes necrosis of the pericarp, placenta, and seeds of pepper fruit. Fruit may not have any external sign of the disease and the damage is frequently only detected by cutting it open. The surfaces of infected seeds are covered with brown lesions of variable sizes. The necrosis in seeds is usually accompanied by necrosis of the blossom end, but the reverse is not always true (Halfon-Meiri and Rylski 1983).

2.1.2 Causal Organism

Alternaria leaf spot and fruit rot is caused by *Alternaria alternata* (Fr.)Keissler and *Alternaria solani* Sorauer (Macrosporium solani Ellis & Martin). *Alternaria* alternata have conidiophores that arise singly or in small groups, simple or branched, straight or flexuous, and pale to dark brown in color measuring up to 50 µm long by 3-6 µm thick with 1 or several conidial scars. Conidia are formed in long, often branched chains, obclavate or ovoid, often with a short beak that is up to, but not more than, one-third its length. The conidia are 20- 63 µm long × 9-18 µm at the widest point and have up to 8 transverse and several longitudinal septa. The color is pale to light brown (Ellis 1971).

2.1.3 Disease Cycle

The fungus commonly colonizes wounded or senescent tissue. Fruit damaged by cold temperatures, sunscald, or blossom end-rot (calcium deficiency) are particularly susceptible (Sherf and McNab 1986). Conidia, produced on infected tissue, are dispersed by wind or rain splashing to new infection sites. Conidia require moisture to germinate and optimal mycelial growth occurs at 26-28ºC. The strains of *A. alternata* that cause internal mold invade at the flowering stage via the stigma and the style causing necrosis of live tissue. Optimum temperature for internal mold disease development ranges from 24 –28ºC.

2.1.4 Management

Damage to fruit by other disorders should be avoided. Pepper cultivars that shade fruit well should be used in order to reduce damage by sunscalding. Diseases that defoliate the plant, such as bacterial leaf spot, should be controlled by the use of resistant varieties or protective sprays. Plants need adequate moisture and calcium to prevent blossom end.

Weeds and volunteer plants should be eliminated in and near fields. Differences

in susceptibility to *Alternaria* sp. have been detected among pepper varieties, however, resistance is not a practical control method at this time. Applications of fungicides in the field are not necessary but post-harvest fungicidal applications or hot water treatments may decrease fruit rot. Fruits should be cooled promptly to 10ºC with high humidity. Storage facilities should be kept clean and disinfected. Reduced decay and a longer shelf life was achieved experimentally by dipping fruit in a commercial solution of hydrogen peroxide and 0.05% silver ion (Fallik *et al.,* 1994).

2.2 Anthracnose

Anthracnose, caused by several species of the fungus *Colletotrichum*, is a minor disease of pepper foliage but causes serious losses to pepper fruit. The disease was first reported in New Jersey, U.S.A., in 1890 and is currently found throughout North America, Asia, Australia and Africa. *Colletotrichum* isolates typically have a wide host range and may also infect other solanaceous crops such as tomato, potato and eggplant and members of Cucurbitaceae, Leguminosae and Malvaceae. Anthracnose incidence on fruit in pepper ranged from 5 to 75% (Hadden 1989).

2.2.1 Symptoms

Isolates of *Colletotrichum* are capable of affecting all parts of the pepper plant at any stage of plant growth; however, fruit lesions are the most common and serious disease manifestation. In pepper transplants, the pathogen causes damping-off, dieback, and leaf spots. Small lesions, approximately 1-2 mm, develop on cotyledons shortly after emergence. Lesions are light brown to yellow and may contain dark fruiting bodies (acervuli).

Leaf symptoms, although rare on mature plants, appear initially as light tan lesions that eventually turn necrotic. Shoot infections cause wilting of the branches and eventually leads to plant dieback. Anthracnose lesions on stems are elongated, white and demarcated from healthy green tissue by a black line.

Anthracnose symptoms on fruit may develop while still in the field or latent infections develop postharvest. Initial symptoms are small, water-soaked lesions that later become soft, slightly sunken and turn tan in color. Multiple lesions may coalesce but remain distinct and cover large areas of the fruit surface (Fig. 1). Within a lesion are numerous, sub-epidermal acervuli which become erumpent when the conidia are mature and spores are released in droplets. Spore masses are usually orange in color although, depending upon the species of *Colletotrichum*, the spore masses and lesions may be brown and then black due to formation of setae and sclerotia. Concentric rings of acervuli, which may or may not have black setae depending upon species involved, are highly diagnostic features of this disease. On mature, colored pepper, the lesions may bleach the color from the fruit. Lesions on the fruit surface can extend to the seed cavity and severely infected seeds are brown and discolored.

2.2.2 Causal Organism

Many different species of *Colletotrichum* cause anthracnose of pepper. *Vermicularia*

capsici Syd [anamorph = *Colletotrichum capsici* (Syd) Butler and Bisby] and *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk [anamorph= *C. gloeosporioides* (Penz) Penz. & Sacc. in Penz] are the two most often reported species causing anthracnose on pepper in North America and Asia. Other species reported are *Vermicularia atramentaria* Berk. & Br [anamorph = *C. coccodes* (Wallr.) Hughes] and *C. acutatum* Simmonds (Hadden 1989, Manandhar *et al.*, 1995a).

Some species of *Colletotrichum* are pleomorphic and have a readily detected sexual stage (perithecia containing ascospores in asci) while others have only the asexual stage (acervuli giving rise to conidia) and a resting state (sclerotia). The following descriptions are examples of the fungal structures from selected species.

Acervuli produced by *V. capsici* are rounded or elongated, approximately 350 μm in diameter, and have brown setae that are 1-5 septate. Acervuli are usually subepidermal and erumptant (Mordue 1971b). Conidia of *V. capsici* are nonseptate, hyaline,
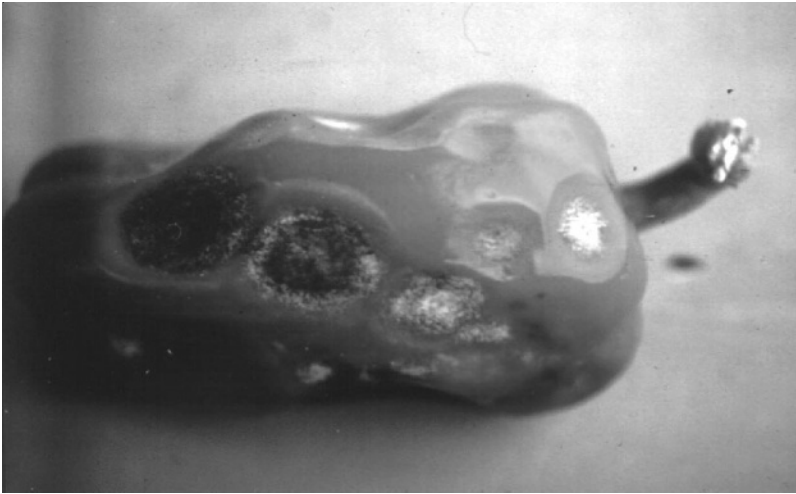
Figure 1: Mature pepper fruit exhibiting multiple lesions of anthracnose. (Photo courtesy T.A. Kucharek) *See also Colour Plates, pp. xv-xix.*

16-30 μm × 2.4-4 μm, falcate with an acute apex and narrow truncate base. The conidiophores are septate and sometimes branched (Mordue 1971a). Conidia of *G. cingulata* and *V. atramentaria* are cylindrical and *C. acutatum* conidia are fusiform (Hadden 1989). Sclerotia of *V. atramentaria* on potato dextrose agar are initially smooth and gray, but become dark and setose. Appressoria produced by *C. coccodes* are cinnamon buff, ovate or obclavate to elliptical, occasional irregularly lobed, approximately 5-15 × 4-11 μm, and borne on hyphae (Mordue 1967). Perithecia of *G. cingulata* are round, dark brown to black, measure 85-300 μm, with a lightly papillate ostiole. Asci are cylindrical containing 8 ascospores and interspersed with sterile paraphyses at the base of the perithecium. Ascospores are oval to cylindrical to fusiform, hyaline, and one-celled.

2.2.3 Disease Cycle

The fungus is commonly seedborne and survives internally as mycelium inside the seedcoat and externally as acervuli. The acervuli and mycelium remain viable in or on seed for at least 9 months (Smith and Crossan 1958). The fungus overseasons on weed hosts or in the soil either in association with plant debris or freely as sclerotia.

Infested debris in soil is an important primary source of inoculum in addition to infested seeds. Conidia produced on the debris are dispersed to infection sites. The disease becomes polycyclic when acervuli within the lesions of infected tissue produce new spores that are dispersed by rain splashing or by wind-blown rain to secondary infection sites. Spores may also be disseminated via equipment or by workers handling infected plants. The disease may be introduced into the field on infected transplants produced from contaminated seed or otherwise inoculated in the transplant house.

Disease usually occurs during warm, wet weather. The optimum temperature for infection is 27°C although infection can occur over a range of temperatures of 10-30 °C. Severe losses occur during rainy weather because of extensive dispersal of spores to other fruit.

Anthracnose is more likely to develop on mature fruit that is present for longer periods of time on the plant, but immature fruit are also susceptible. Appressoria form from germinated conidia and attach to the fruit epidermis. Infection hyphae directly penetrate the host surface. Although all fruit is susceptible to infection, more mature, red pepper fruit are infected more frequently than green, immature fruit, which may be due to physical and chemical differences in the cuticle and exocarp (Kim *et al.,* 1999, Manandhar *et al.,* 1995b). Immature fruit that are infected may not express symptoms until the fruit matures.

2.2.4 Management

Only seeds that are pathogen-free should be planted. Transplants should be kept free of the disease by controlling weeds and solanaceous volunteers around the transplant houses. Diseased transplants should be discarded. In the field, infected plant debris should be removed promptly or deep plowed to completely cover. If disease was previously present, crops should be rotated away from solanaceous plants for at least 2 years. Insects should be controlled to prevent damage to fruit which provides entry wounds for the fungus. Weeds and volunteer host plants should be eliminated.

Resistance is commercially available for some chili pepper cultivars and is reported for sweet peppers (Ullasa *et al.,* 1981). Pepper cultivars should be selected that produce fruit with a shorter ripening period to escape anthracnose infection. Fungicide labeled for this disease are applied either preventively or at first sign of the disease to reduce disease losses.

2.3 *Cercospora* leaf spot (Frog-eye spot)

Cercospora leaf spot is a fungal disease that most commonly occurs in tropical and subtropical pepper production regions. The major problem associated with the disease

is defoliation that reduces plant vigor and yield. The disease occurs in Africa, Asia, North and South America, the East and West Indies. The host range is limited to sweet and chili peppers.

2.3.1 Symptoms

The disease affects only the aerial plant parts except of fruit. Symptoms on leaves consist of circular spots that are whitish, gray or tan in the center surrounded by narrow, dark brown margins. Lesions are usually 5-10 mm in diameter and often appear zonate. Large lesions become dry, brittle, crack and pieces drop out leaving a shot-hole appearance. Severely infected leaves turn yellow and abscise which reduces plant vigor and decreases yield. Stems and peduncles of the fruit can be infected and exhibit circular or oblong lesions. Fruit may be smaller than normal and misshapen (Sherf and McNab 1986).

2.3.2 Causal organism

Cercospora capsici Heald and F.A. Wolf is the causal organism of the disease. The conidia of *C. capsici* are borne singly and are straight or slightly curved, colorless, with 5-9 septa and measure 40-135 μm in length $\times$ 3-5 μm wide and are slightly wider at base, with a thickened scar at the base. Conidiophores are clustered, unbranched, straight or slightly curved, pale brown, and 30-80 μm in length $\times$ 4-6 μm wide (Kirk 1982).

2.3.3 Disease cycle and Epidemiology

The fungus survives primarily on plant debris. Seed-borne survival and transmission of the fungus is not confirmed. The spores are spread mainly by wind but also by wind-splashed rain. Mechanical transmission of the fungus is through contaminated equipment and personnel.

The fungus enters the plant tissue through the stomata after germinating on the surface of the leaf. After the tissue dies, secondary infections arise from spores disseminated from sporulating lesions. Wet weather and moderate temperatures favor disease infection.

2.3.4 Management

Fields should not be worked when plants have wet foliage. A fungicide should be applied to plants in the transplant bed at the first sign of the disease. Resistance is reported but not widely available in commercial cultivars (Ullasa *et al.*,1981).

2.4 Damping-off and root rot

Damping-off is a common problem on pre-emergent seeds or seedlings in both greenhouse and field production. Minor damage may occur on older crops. A number of fungi cause the disease including *Rhizoctonia solani*, *Pythium* spp., *Phytophthora*

spp., *Colletotrichum* spp., and *Fusarium*. The disease occurs worldwide in temperate and tropical climates. Entire plantings of seeds or seedlings may be wiped out.

2.4.1 Symptoms

Symptoms vary with the developmental stage of the pepper plant. Infected seeds do not germinate and turn brown, are shrunken, soft, and decompose in the growing media. The number of seedlings that emerge is reduced. Infected seedlings have brown, water-soaked lesions both above and below the soil line. The bottom portion of the stem may be rotted and constricted and no longer supports the seedling, which falls over, withers, and dies (Sherf and McNab 1986). The root system is reduced and may be brown with little to no secondary roots. Stands of transplants will have missing plants in either patches or randomly scattered throughout. Infected mature plants are chlorotic, stunted, and less vigorous. Roots are usually brown and rotted.

Symptoms of damping-off may be confused with plant injury caused by excessive fertilization, high soluble salts, water stress, temperature extremes, or chemical injury. This type of damage typically shows on the aerial parts of leaves and upper stem of the plant before lesions on the root are seen.

2.4.2 Causal organisms

Several species of fungi and bacteria cause damping off and root rot in nurseries. Thus one or more of the several pathogenic organisms may be associated with damping-off and root rot. The most common are *Rhizoctonia solani* Kuhn, *Pythium* spp., *Fusarium* spp., and rarely *Phytophthora* spp. and *Colletotrichum* spp. Fungi are identified by morphological characteristics usually after isolation on semi-selective or selective media.

In general, species of *Pythium* and *Phytophthora* are more likely to cause damping-off in cool, wet soils whereas *R. solani* and *Fusarium* spp. cause damping-off under warmer and drier conditions and generally caused post emergence damping-off by killing the seedling at the soil line. *Pythium* spp. attacks below the soil line, often at root tips. Infection of roots by *Pythium* spp. and *Fusarium* spp. occurs on mature plants (Chellemi *et al.,* 2000).

2.4.3 *Disease Cycle and Epidemiology*

Pythium has a broad host range, is soilborne, and survives in the field on alternate hosts including weeds. *Pythium* spp. can survive independently of a host in the soil for very long periods (>1 year) by producing oospores. Spread of the pathogen occurs by movement of contaminated soil or through irrigation and ground water. Germinating seeds and root exudates stimulate oospore to germinate and infect plants. High soil moisture generally increases disease severity. Crowded plants and excess of nitrogen favor infection.

Rhizoctonia solani is a common, endemic soil-borne pathogen with a very wide host range. *R. solani* survives indefinitely in soils by colonizing organic

material and producing sclerotia. The fungus can be moved in contaminated soil or on equipment.

2.4.4 Management

Soil used in transplant production should be sterilized by steam pasteurization that maintains a temperature of 71°C for 30 minutes throughout the soil volume. Soilless medium that is pathogen free should not be contaminated by other sources. Pots and transplant trays should be new or disinfested before reuse by hot water or steam (71°C for 30 minutes) or placed in a 0.5% sodium hypochlorite solution for 30 minutes. Fungicide-coated seeds with a high germination rate should be planted at the correct depth. Transplant houses should maintain moderate temperatures to promote seed germination and growth and adequate air circulation, light and ventilation. Plants should not be overcrowded and have adequate water, preferably applied in the late morning. Workers should clean hands and tools with soap and water before handling healthy plants.

In the field, plant in well-drained soil. Fumigation of the soil reduces losses to damping off. Only healthy transplants should be planted and at adequate planting spacing. Plant when temperatures are favorable for rapid plant growth. Movement of soil between fields should be avoided and workers should disinfest before moving from diseased to healthy plants. A labeled fungicide can be banded in beds by mixing in transplant water during setting or applied as a drench or heavy spray as soon as the first symptoms of damping-off are observed, however identification of the causal organism should be obtained so that proper chemicals can be applied. Several applications of the fungicide may be necessary. The biological agent *Trichoderma* and *Gliocladium*, added to the soil demonstrated suppression of damping-off (Lewis *et al.,* 1998).

2.5 Downy mildew

Downy mildew very rarely infects the solanceous crops of tomato, eggplant and pepper. The disease was usually seen on these crops when they were grown near tobacco plants that were heavily infected with blue mold in southern regions of the USA. Pepper and tomato were reported hosts in the USA, Russia, and Argentina, however, some isolates only infected pepper and tobacco and not tomato in other locations (Armstrong and Albert 1933, Gayed 1984, Hindi *et al.,* 1965). Although extensive losses in transplants have occurred, the disease is of minor, infrequent importance.

2.5.1 Symptoms

Downy mildew produces its characteristic symptoms on the leaf underside where the fungus sporulates through the leaf stomata. The sporulation is seen as fuzzy, white to grayish patches. On the upper surface of the leaf, pale green to yellow spots may appear opposite the areas of sporulation. Older leaves are affected first and often turn completely brown from infection (Sherf and McNab 1986).

2.5.2 Causal organism

Downy mildew is caused by the fungus *Peronospora tabacina* Adam. Sporangio-phores are dichotomously branched at acute angles, tapered, and curved with pointed tips bearing sporangia. Sporangia are oval.

2.5.3 Disease Cycle and Epidemiology

Sporangia are wind-dispersed over long distances. Wind-driven rain or splashing water can transport sporangia short distances. The fungus survives on infected plant debris in the soil and on related weed species. Environmental conditions for infection are high humidity, leaf wetness, and moderately cool weather (daytime temperatures ranging from 15 to 24° C).

2.5.4 Management

Transplants infected with downy mildew should be removed from the transplant house and discarded. Watering of transplants should be performed with a minimum of splash-ing and done early in the morning to allow foliage to dry. Increased plant spacing and reduced humidity levels should also help to keep foliage dry. Rouging in the field may be useful if a small number of plants are infected. Fields and transplant production areas should be kept free of weeds and isolated from tobacco production. Fungicides labeled for this disease can be used preventively to control the disease.

2.6 *Fusarium* Wilt

Fusarium wilt of pepper was reported in Argentina and Italy and in many states in the USA including Louisiana, New Mexico, Colorado, New Jersey and Ohio. However, the most in depth work describing the pathogen and its etiology was done in Louisiana (Rivelli 1989, Black 2003). Other *Fusarium* spp. associated with pepper roots cause decline symptoms and root rots but not a vascular wilt. They also have a broader host range compared to the pepper *Fusarium* wilt pathogen. Losses due to *Fusarium* wilt were severe, up to 35% of plants were affected in a single field.

2.6.1 Symptoms

The symptoms described were present on plants in Louisiana, USA. The vascular wilt symptoms appear initially as a slight yellowing and wilting of the upper leaves. The plant becomes permanently wilted within a few days and dies. The dried foliage remains attached to the plant. Brown discoloration of the vascular tissue can be observed by cutting the stem lengthwise (Rivelli 1989).

Other reports of *Fusarium* wilt describe dark brown, sunken, external cankers at the crown area of the pepper and girdling of the stem that culminates in rapid collapse and plant death. Infected roots are brown and soft (Sherf and McNab 1986). However these symptoms were not evident in the plants affected in Louisiana and are more

typical of *Phytophthora* blight caused by *P. capsici* Leon.

In several cases, pathogenic *Fusarium spp.* have been associated with a general plant decline. The symptoms appear as gradual chlorosis of the foliage, thinned canopy, and the plant appears slightly stunted and unthrifty compared to healthy plants.

2.6.2 Causal organism

The proposed name for the causal organism of *Fusarium* wilt in pepper is *Fusarium oxysporum* (Schlect.) emend. Snyd. & Hans. f. sp. *capsici* f. sp. nov. (Rivelli 1989) Earlier reports identified it as *Fusarium oxysporum* var. *vasinfectum* (Sherf and McNab 1986). The fungus produces microconidia, macroconidia and thick-walled chlamydospores. Macroconidia are septate and average 41-46.8 μm length $\times$ 4.5-4.7 μm width. Terminal chlamydospores form in hyphal strands and intercalary chlamydospores form in hyphae and macroconidia (Rivelli 1989).

2.6.3 Disease cycle and Epidemiology

In general, *Fusarium* wilt pathogens survive in the soil in the absence of the host for periods of up to 2 years. Spores are disseminated by the wind, in ground water, or by movement of contaminated soil, stakes, or equipment. Plant to plant movement happens by contact of roots on contiguous plants.

The temperature range for growth of *F. oxysporum* f. sp. *capsici* forma specialis nov is 12–32 °C with maximum growth at 28°C. Optimal growth range for temperature is 23 to 33° C. Excessive soil moisture greatly enhances disease development (Rivelli 1989). Alternate hosts are not reported. The pathogen does not infect cotton, okra, tomato, eggplant, watermelon, cantaloupe, cucumber or cabbage in greenhouse experiments (Rivelli 1989).

2.6.4 Management

Fields should be well-drained and free of low-lying wet areas. Excessive irrigation should be avoided. Culture of plants on raised beds with plastic mulch improves drainage. Plants should be grown to maintain high vigor. Crop rotation of 2 to 6 years is needed to reduce the population of the fungus. Although weed hosts are not reported, weed and volunteer plants should be controlled and infested plant material destroyed after harvest to maintain good field sanitation. Infected plant material and soil should not be moved to uninfected fields on equipment or personnel. Soil fumigation will probably reduce populations of *Fusarium*. Resistance is reported in some *Capsicum* accessions but is not available in commercial varieties at present (Jones 1992).

2.7 Gray leaf spot

Gray leaf spot, caused by the fungus *Stemphylium solani* Weber, is a relatively minor foliar disease on pepper that is most damaging on seedlings in transplant production. The fungus causes significant disease problems on tomato, eggplant, and potato. The

disease on pepper was first reported in 1930 is reported most frequently from the USA although the fungus is distributed worldwide. Gray leaf spot caused severe defoliation and reduced plant growth on pepper in Louisiana in 1958 and the disease appeared regularly in Florida in 1966 through 1971 (Blazquez 1969 and 1971, Sinclair *et al.,* 1958).

2.7.1 Symptoms

Symptoms on foliage appear initially as round, dark brown lesions. Older lesions are sunken with a light tan to white center surrounded by dark, reddish-brown margins. Spots are small, typically 3 to 5 mm in diameter. The centers of some older spots fall out leaving a shot-hole appearance. Lesions may occur on stems, petioles, and fruit peduncles, sepals, and the calyx but not on flower petals or fruit. Seedlings are particularly susceptible to defoliation and stem lesions can cause the stem to break. Severely infected seedlings are stunted and severely defoliated.

Lesions are distinguishable from bacterial leaf spot (*Xanthomonas*) or the early stages of frogeye spot (*Cercospora capsici*) by the dark gray masses of conidiophores on the edge of the lesion on both sides of the leaf.

2.7.2 Causal Organism

The pathogen on pepper was identified as *Stemphylium solani* although it was also identified as *S. vesicarium* (Wallr.) and *S. lycopersici* (Enjoji) Yamamto (=*S. floridanum*) (Blazquez 1971). *Stemphylium lycopersici* from tomato was reported to infect peppers in the Philippines. Another species, *S. botryosum* f. sp. *capsicum*, is pathogenic on pepper but the lesions are larger (10-20 mm) and concentrically ringed (Braverman 1968).

Stemphylium solani has hyphae that are variously branched, septate, and occur intercellularly. The conidiophores are dark, septate, and slightly larger than the sterile hyphae, measure $130\text{-}200 \times 4\text{-}7\ \mu m$ with swollen tips and irregular bases. The conidia are muriform, dark, and oblong-rectangular with one end rounded and the other somewhat pointed. Conidia have 3-6 traverse septae and several longitudinal and are constricted near the middle, with an average size of $48 \times 22\ \mu m$ (Ellis and Gibson, 1975).

2.7.3 Disease cycle and Epidemiology

The fungus survives on volunteer plants, other hosts such as tomato or potato, solanaceous weeds, and crop debris. Conidia are spread by wind or moved with splashing rain. Periods of leaf wetness are required for germination of conidia. Warm temperatures of 24°C and high humidity, including fog, favor disease development.

2.7.4 Management

Weeds and volunteers should be removed from field site to reduce inoculum. Field and transplant production sites should not be located near other host crops, particularly tomato and potato. Residual crop debris should be buried or destroyed. Chemical con-

trol on preventative spray schedule is effective to control the disease.

2.8 Gray mold

Gray mold disease affects all aerial parts of the pepper plant and is a significant cause of postharvest losses. The disease occurs in field in greenhouse production. The fungus is a common saprophyte and is considered a weak pathogen even though it infects a very wide host range of plants that includes lettuce, tomato, eggplant and snap bean. The disease occurs worldwide in temperate and sub-tropical climates. Gray mold as a postharvest disease problem was found in 25% of all pepper shipments to New York during 1972-1984 (Ceponis *et al.,* 1987).

2.8.1 Symptoms

The fungus infects young leaves, new stems, and flowers. Lesions on leaves begin as water-soaked spots that enlarge rapidly and are whitish tan in color. Numerous, dark conidiophores at the center of the lesion can be observed without the aid of magnification. Seedlings are infected on tips of leaves or on the stem near the cotyledons resulting in dieback. Flowers and senescent tissue are susceptible to colonization by the fungus. Plants infected on a basal stem exhibit a dark brown to black lesion that may lead to plant wilting and death. Old, diseased tissue is covered with gray sporulating conidiophores that appear dark brown and felt-like, and release clouds of spores when shaken.

Postharvest fruit decay frequently develops in wounds near the blossom end or calyx and quickly expands to encompass the entire fruit. The lesion is slightly shrunken, soft and is a darker olive green compared to healthy tissue. The fruit skin remains intact while the tissue underneath is soft and rotted. Conidiophores and conidia also develop on the older lesions.

2.8.2 Causal organism

Botryotinia fuckeliana (de Bary) Whetzel [Syn *Sclerotinia fuckliana* (de Bary) Fuckel] [anamorph= *Botrytis cinerea* Pers.:Fr.] is very common both as a common pathogen and saprophyte.

The conidiophores are 1–2 mm in length by 16–30 µm thick and dichotomously branched at the tip. Conidia are borne at the tips of the numerous short branches and resemble a cluster of grapes. The conidia are hyaline or slightly gray or brown colored, single-celled, round to oval, and measure $10\text{-}13 \times 6\text{-}10$ µm. Sclerotia are flat or convex, hard, black, 2-5 mm long, and usually formed in host tissue. Sclerotia rarely give rise to apothecia and ascospores (Ellis and Waller 1974).

2.8.3 Disease cycle and Epidemiology

Survival of the fungus occurs either in the soil as sclerotia or in infested plant material. The sclerotia are produced in infected, fully colonized tissue and are able to produce

conidia immediately or later after a dormant period. Sclerotia may persist in the soil for months or years. The fungus occurs commonly on a wide host range that includes many perennial and annual weeds and cultivated plants from which the conidia easily move by wind or splashing of rain.

Lesions on plants or fruit are more likely to occur close to the ground. The fungus infects flowers, soft tissue, or enters plant tissue through natural and other wounds. Fruits are susceptible to infection if in direct contact with soil or through wounds. The fungus is weakly pathogenic and most frequently colonizes dead or dying tissue. Conidia germinate in the presence of water to produce an appressoria and infection tube that penetrates the cuticle within hours. The optimum temperature for disease development is 20°C and high humidity favors disease. The fungus produces a soft rot of infected tissue by dissolving pectin in the middle lamella. Secondary infection cycles occur, especially in wet weather.

2.8.4 Management

Infected plant debris and discarded plant material should be removed from the vicinity of production. Seedlings and pepper fruit should not be injured during transplanting and storage. Chilling injury is especially important in the predisposition of fruit to gray mold and fruit should be stored at optimum conditions of 7-13°C and 90-95% relative humidity.

Biological control agent (T39 strain of *Trichoderma harzianum*) suppressed disease in greenhouse crops (Elad 2000). Fruit damage was reduced by optimum calcium nutrition provided to the plant during growth (Elad *et al.,* 1993). Fungicide or disinfectant, such as H_2O_2, applied postharvest to fruit reduces decay development on fruit and extends shelf life (Fallik *et al.,* 1994). Hot water dips of fruit at 50°C for 3 minutes also reduces fruit decay development (Fallik *et al.,* 1996). Shortwave ultraviolet radiation (UV-C) reduces the number of infections that occur during storage, apparently act by inducing disease resistance in addition to killing surface conidia (Mercier *et al.,* 2001).

2.9 *Phytophthora* blight

Phytophthora blight is caused by the Oomycetes pathogen, *Phytophthora capsici* Leonian. *P. capsici* has a very wide host range that includes tomato, pepper, watermelon, eggplant, cantaloupe, chayote, cucumber, honeydew melon, pumpkin, and squash. The disease on pepper was first reported in New Mexico, USA in 1922 (Leonian 1922). The disease is distributed worldwide throughout North and South America, Europe, and Asia. Extensive losses of pepper occur during an epidemic.

2.9.1 Symptoms

The pathogen is capable of infecting all parts of the pepper plant. It causes seedling death, crown and stem lesions, root rot, leaf blight and fruit rot. Seedling death causes the entire young plant to wilt and die. Plants may be infected in the transplant tray or

after transplanting. Crown and stem infection at the soil line causes sudden wilting and death. Stem lesions, whether at the soil line or higher, initially appear dark green and water-soaked, but turn dark brown to black and girdle the stem (Fig. 2). Infected roots are dark brown and rotten.

Leaves are blighted in an aerial phase of the disease. Leaf spots initially appear water-soaked, and are small, irregular to round shaped. As it ages, the lesion becomes dry, papery, and turns a light tan to grayish brown. During warm, wet weather, the

Figure 2: *Phytophthora* blight lesion at crown of pepper plant (Photo courtesy R. J. McGovern) *See also Colour Plates, pp. xv-xix.*

infected areas may be bordered by white fungal growth. New leaves and shoots are susceptible to rapid blighting. Entire branches may wilt and die from infection at the forked part of stems.

Pepper fruit are frequently infected through wounds. Cracks that develop along the peduncle and mechanical injuries are common entry sites. Lesions maintain a water-soaked margin around the infected tissue as it enlarges. The fruit tissue becomes dark,

shrunken, and white fungal growth of mycelium and sporangia is apparent on its surface (Fig. 3). Infected fruit remain attached to the stem.

2.9.2 Causal organism

Phytophthora capsici Leonian is the causal pathogen. *Phytophthora capsici* reproduces asexually by producing ellipsoid to pyriform sporangia with papilla. The sporangium is usually oblong, average 30-60 µm × 25-30 µm and has a length to width ratio of 1.3 to 2.1. The sporangium may produce 15-25 zoospores that are flagellated spores that swim and are chemotacticallly attracted to plant and root exudates. Hyphae are fairly coarse, 5 to 15µm wide, and irregularly branched. Sporangiophores are 1.5 –2 µm wide and sometimes widening slightly at the base of the sporangium. *P. capsici* is
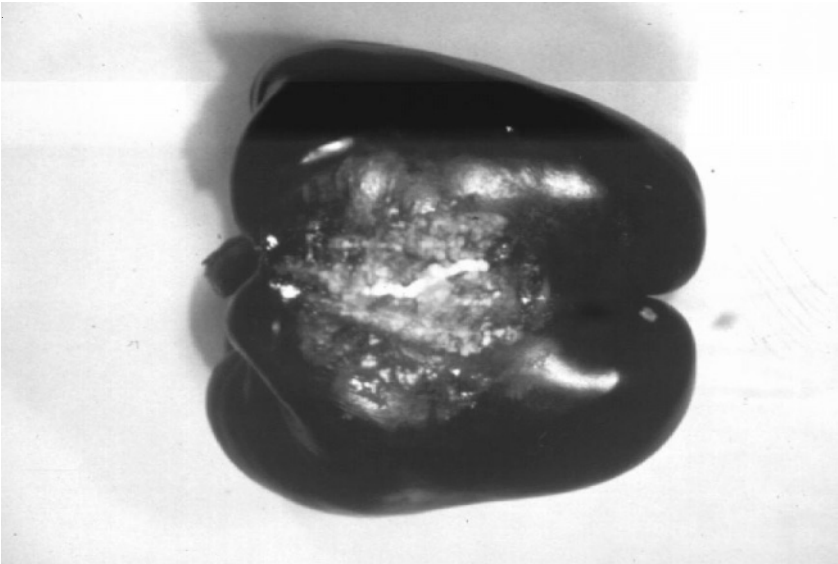
Figure 3: *Phytophthora* blight lesion on pepper fruit (Photo courtesy R. J. McGovern)
          See also Colour Plates, pp. xv-xix.

heterothallic, it requires two mating types (A1 and A2) or compatibility types to produce the sexual spore, the oospore. The oospore results from the pairing of an oogonium with an antheridium. Oogonia average 30 µm (18-50 µm) in diameter. The antheridia are on average 17 × 14 (14-19 × 13-15) µm long. The resulting oospore averages 26 µm (15-40 µm) in diameter and has a wall thickness of 2-6 µm. The pathogen does not produce chlamydospores.

2.9.3 Disease cycle and Epidemiology

Phytophthora capsici survives on host plant debris and in the soil as oospores. Both heterothallic mating types are frequently recovered within the same field (Ristaino

1990). Oospores are probably the primary structure for overseasoning and remain viable in the soil for up to 12 weeks in the summer (Bowers *et al.,* 1990a). Mycelium survives in moist soil from up to 5 months to a year (Satour and Butler 1967). Sporangia and zoospores persists the least amount of time, about 4-8 wks (Bowers *et al.,* 1990a).

    Phytophthora blight is a polycyclic disease in which the pathogen reproduces and infects new plants several times within a single season. Zoospores are produced within the sporangia and when released, swim in a film of water to reach host tissue. Periodic flooding and saturated soil conditions from either rainfall or irrigation stimulates the release of zoospores from sporangia (Bowers and Mitchell 1990). The rate of plant infection is increased under those conditions. Inoculum is spread by several means. Sporangia are dispersed by splashing rain and wind or overhead irrigation. The pathogen is spread with water movement in the field with includes irrigation water, surface run-off, or rainfall (Bowers *et al.,* 1990b, Ristaino 1991). Dissemination is also through infected transplants and contaminated soil and equipment. Root to root contact spreads the disease.

    Conditions that favor disease development are high soil moisture and warm temperatures. Therefore, wet waterlogged soil conditions coupled with warm temperatures are ideal for disease onset and development. The application of water made frequently through drip irrigation causes earlier onset of the disease compared to applications of drip irrigation made less frequently (Ristaino 1991, Ristaino *et al.,* 1993). Wet, waterlogged soil and mild to warm temperatures conditions are ideal for epidemics. The optimum temperature for oospore germination is 24°C (Hord and Ristaino 1991). Temperatures ranging from 26-32°C are optimum for zoospore production, the infection process and vegetative fungal growth. In pathogenicity studies on several weeds commonly found in squash fields, *P. capsici* was isolated from *Portulaca oleracea* (common purslane) roots (Ploetz and Haynes 2000). The potential of this and other solanaceous weeds to serve as alternate hosts can allow for survival of *P. capsici* during and after the production season.

2.9.4 Management

Transplant producers should use fungicide-treated seed and sterile potting media. Flats, plug trays, benches, seeding equipment and plant house structures should be disinfested using a solution of sodium hypochlorite or other disinfestants. Steam sterilization of flats and plug trays is useful. Transplant trays with infected plants should be discarded immediately. Workers should disinfest their hands after contact with infected plants or when moving to new plant houses.

    Fields should be well drained and free of low-lying areas to minimize waterlogged conditions. The field should be kept free of weeds and volunteers, particularly cucurbit and solanaceous plants. Pepper should be rotated with non-host crops. Soil should be fumigated before planting. The use of black plastic mulch can aid in disease prevention by acting as a barrier to dispersal of inoculum by splashing, or it may increase movement of the pathogen in water caught on the surface of the plastic. As an alternative, a no-till wheat cover provides a barrier to inoculum dispersal by splashing water and reduce surface run-off (Ristaino *et al.,* 1997). Equipment and machinery

should not be moved from field to field or they must be decontaminated between infested and noninfested fields. Infected fruit should be culled to prevent spread in the packing house and during shipment. Effective, labeled fungicides, both contact and systemic, should be applied to the foliage and used preventatively according to label instructions, however, some isolates are insensitive to mefenoxam and the fungicide should not be used where these isolates occur.

Soil solarization may reduce soil populations of the pathogen. Soil solarization with a clear polyethylene sheet (0.03 mm thick) and soil temperatures reaching 47°C at 5 cm and 35° C at 30 over a 8 week period significantly reduced disease incidence compared to chemical fumigated plots (Yucel 1995). Soil solarization also retarded the onset of the disease by 4 to 9 weeks (Polizzi *et al.,* 1994).

Soil amendments, such as compost and manure, are a possibility for disease management, as they may be responsible for developing suppressive soils over time. Further, they may improve plant growth but can also suppress plant growth as well. Sources of resistance in pepper to *P. capsici* were reported as early as 1960 and since then many sources of resistance gene(s) have been incorporated into cultivars. (Barksdale *et al.,* 1984, Kimble and Grogran 1960, Ortega *et al.,* 1992). Resistant varieties should be planted where available.

2.10 Powdery mildew

Powdery mildew on pepper, caused by the fungus *Leveillula taurica* (Lev) *Arnaud* [anamorph = *Oidiopsis sicula* Scalia *syn. Oidiopsis taurica* (Lev) Arnaud], occurs in worldwide in Africa, Asia, the Mediterranean and Caribbean regions, and North America. Isolates of *O. taurica* pathogenic to pepper have a wide host range and are usually cross-infective to tomato and eggplant. The disease is a common problem on peppers in greenhouse production. Severe losses are reported when plants are heavily defoliated.

2.10.1 Symptoms

Powdery mildew symptoms appear as powdery white to light gray-colored spots on the aerial parts of the plant including leaves, stems, buds or flower tissue. However, the white, talcum powdery-like growth that is readily apparent for many other powdery mildew diseases is not nearly so obvious on pepper (Fig. 4). The signs of the pathogen are best observed on the underside of the oldest lesions. On the top surface of leaves, lesions are yellow with brown necrotic centers. Leaves curl upwards. Premature senescence of the leaves results in defoliation. Both the number of fruit and the size of fruit are reduced in heavily infected plants.

2.10.2 Causal organism

The fungus *Leveillula taurica* (anamorph *Oidiopsis taurica*) causes powdery mildew of pepper. Similar to other powdery mildew fungi, *O. taurica* is an obligate parasite but unlike other powdery mildews, it appears to have a wide host range. Host specificity of

some isolates is observed (Ayesu-Offei 1998, Correll *et al.,* 1987).

O. taurica has endophytic mycelium. Conidiophores are long and multi-branched. Conidia are dimorphic (pyrifrom and cylindrical) and borne singly or in short chains. The size of the conidia vary according to the isolate. Mean measurements range from 49-71 μm $\times$ 16-24 μm and 44.6-65 μm $\times$ 16-23 μm for the pyriform and cylindrical conidia, respectively (Correl *et al.,* 1987).

It should be noted that *O. taurica* has been known for many years as the causal agent of powdery mildew of tomato in the western USA but was not the cause of powdery mildew of field tomato in Florida. However, *O. tautica* was identified as the causal organism of powdery mildew on pepper in greenhouse peppers in Florida.

Figure 4 : Powdery mildew on underside of pepper leaf (Photo courtesy K. Pernezny)
      See also Colour Plates, pp. xv-xix.

2.10.3 Disease cycle and Epidemiology

Conidia are windblown or dispersed by splashing rain. Hyphae and conidia on the surface of the leaf tissue give it a whitish appearance. Temperatures of 25-30ºC favor germination of conidia. Some isolates of powdery mildew that infect pepper can apparently develop infection under a wider range of dry to humid conditions compared to other powdery mildews. Relative humidity that is higher at night than during the day and temperatures of less than 30ºC are conducive to disease. Free moisture on leaves inhibit spore germination. Throughout the growing season, new infections may develop new, succulent shoots of plants.

2.10.4 Management

In the greenhouse, increased plant spacing, avoidance of overhead irrigation, and

increased the air circulation can be used to reduce relative humidity. Tolerance in some commercial cultivars is reported (Ullasa *et al.,* 1981).

In both greenhouse and the field, registered fungicides are used to prevent and control powdery mildew. Several systemic and contact fungicides provide good powdery mildew control (Keshwal and Choubay 1983). Preventative spray programs in greenhouses should be established prior to infection. Applications of mono-potassium phosphate as a foliar spray inhibit the fungus on plant leaves and reduce yield losses (Reuveni *et al.,* 1998). Bicarbonates ($NaHCO_3$ or $KHCO_3$) applied to the foliage reduce disease severity, leaf defoliation, and postharvest decay of fruit (Fallik *et al.,* 1997). Auxins (indoleacetic acid, indole butyric acid) applied to infected pepper leaves inhibit defoliation (Reuveni *et al.,* 1976). Practical application of some of these compounds in pepper production needs to be demonstrated.

2.11 Southern blight

Southern blight is a common and destructive disease of peppers and many other vegetable, agronomic and horticultural crops. The host range includes more than 500 plant species in 100 families. Southern blight is found primarily in the warm, humid regions of the world through the American continent, Australia, the Mediterranean, south Europe, Africa, Asia, and Pacific islands (Weber 1931, Punja 1985).

2.11.1 Symptoms

On pepper, the fungus most frequently attacks the crown area of the pepper plant. The first symptoms are wilting and yellowing of the foliage. The infected stem turns brown and rots both above and below the soil line. The lower part of infected plant stem remains intact while the rest of the plant wilts and dies. The fungus grows in a white mycelial mat that covers the stem and adjacent soil and is easily seen without magnification. Sclerotia, which are embedded on the mycelium, initially appear white and fuzzy, but gradually turn into hard, smooth round objects that are light to dark brown and resemble a mustard seed (Jenkins and Averre 1986). Lesions on fruit are watersoaked, rotted, and filled with mycelium and sclerotia.

2.11.2 Causal Organism

Southern blight is caused by the sclerotial state of the fungus *Sclerotium rolfsii* Sacc. The teleomorph is *Athelia rolfsii* (Curzi) Tu & Kimbrough. Synonyms of *Sclerotium rolfsii* are *Corticium rolfsii* (Curzi) and *Pellicularia rolfsii* is the teleomorph.

The fungus grows very rapidly in culture and may cover a petri plate within 48 hours. Both in culture and in plant tissue, the mycelium grows outward in a fan shape. Two types of hyphae are produced. At the leading edge of the colony, hyphae are large, 4.5-9 μmm × 350 μmm, straight cells with one or more clamp connections at each septa. The secondary and tertiary hyphae are slender, 1.5-2.5μm in diameter, grow irregularly and lack clamp connections (Mordue 1974).

Sclerotia form on the surface of the mycelial mat. Initially, sclerotia are fuzzy,

balls of white mycelium that gradually melanize to a dark brown coloration when mature. The mature sclerotium (1-2 mm) has a hard, protective rind.

A sexual stage that has two or four thin-walled colorless basidiospores that are borne on short sterigmata at the ends of slightly enlarged short basidiophores is rarely reported.

2.11.3 Disease cycle and Epidemiology

The sclerotia survive in the soil for extended periods of time. Sclerotia can remain viable for long periods of time, conservatively estimated at 10 months or less, in the soil (Beute and Rodriguez-Kabana 1981). The fungus survives on plant debris including crop refuse, volunteers, and weeds. The fungus is disseminated by both sclerotia and mycelium in infested soil, or on contaminated tools and machinery, by infected transplants, irrigation water, and possibly on seeds.

Environmental conditions of warm temperatures and high moisture are favorable for the fungus and disease development. The fungus rarely occurs where daily winter temperatures are below freezing. Optimal temperatures for growth and sclerotial formation are 25 to 35°C and little to no growth occurs at 10°C or 40°C. Sclerotia can survive at temperatures at least 10 degrees lower (-10°C) than the mycelium (Beule and Rodriquez-Kabana 1981). The optimum pH for is 3.0 to 5.0. Germination of the sclerotia occurs between pH 2.0 and 5.0 and is inhibited at higher than pH 7.0. Southern blight is usually not a problem on calcareous soils with a high pH.

Infection by *A. rolfsii* is usually restricted to any plant part in contact with the soil. Root, stem, leaves, and fruit are susceptible to infection. Fruits of pepper are infected after contact with infested soil or by splashing soil. A mass of mycelium is usually produced on the plant surface 2 to 10 days prior to infection. The pathogen penetrates the host tissue by producing an enzyme that deteriorates the outer cell layer of the host tissue and causes tissue decay. Further production of mycelium and the formation of sclerotia complete the cycle.

2.11.4 Management

Control of southern blight is difficult and an integrated approach to its control should be used. Avoidance of the disease by selecting fields that are free of *A. rolfsii* is the best method of control and the disease should not be introduced into the field on diseased plants or by movement of infested soil (Jenkins and Averre 1986).

Sanitation and cultural control methods may help to limit the disease. Diseased plants should be rouged and weed host eliminated. A dense canopy increases disease incidence, thus increasing plant spacing can help keep infection to a minimum. Damage may be avoided by planting at times in the season when temperatures are cooler and less moisture is available. Soil pH may be adjusted by liming. The use of ammonium fertilizers decreases disease incidence. Crop rotation must be done with a non-host such as a monocot (grass, corn, or wheat).

The fungus is highly aerobic and plant debris and sclerotia should be buried by deep plowing (at least 20 cm) with a moldboard extension that inverts top soil. Buried

soil should not be re-surfaced by other cultural operations.

Black plastic mulch and row cover reduces disease severity and incidence by proving a barrier between the plants and soil (Brown *et al.,* 1989). Incorporation or compost or straw to the soil limits disease incidence (Gurkin and Jenkins 1985). The addition of such an amendment may help to increase the populations of antagonistic soil microorganisms.

Solarization of soil performed by covering the soil with a transparent polyethylene sheet can generate heat sufficient to kill sclerotia to depths of 6-20 cm depending upon soil type and season. A number of antagonistic fungi have been shown to provide suppression against *A. rolfsii.* Organisms used are: *Trichoderma harzianum*, *T. viride*, *Bacillus subtilis*, *Penicillium* spp., and *Gliocladium virens* (Peeples *et al.,* 1976). Soil solarization combined with the addition of *Trichoderma harzianum* was more effective to decrease disease incidence than either treatment alone (Elad *et al.*, 1980, Mihail and Alcorn 1984).

Pre-plant chemicals and fumigants such as metam sodium (Vapam), Vorlex, methyl bromide, and chloropicrin, when applied to soil can reduce pathogen population and provide control (Elad *et al.,* 1980).

2.12 *Verticillium* Wilt

Verticillium wilt is caused by the fungi *Verticillium albo-atrum* Reinke & Berthold and *V. dahliae* Kleb. These fungi attack a wide range of plant species, including cultivated crops and weeds. *Verticillium* wilt is also important on strawberry, raspberry, certain stone fruits, mango, cotton, maple, tomato, eggplant, and potato. The disease is distributed in Europe and North America in Canada and the western part of the USA. Severe losses can occur in due to this disease.

2.12.1 Symptoms

The first symptom of *Verticillium* wilt is often diurnal flaccidity followed in time by permanent wilting. The lower leaves express wilting first and turn yellow, curl, and the tips and margins dry out and turn brown. Leaf wilting is followed by necrosis and stunting. Infection of only a few vascular bundles results in sectoring of symptoms to infected branches. The infection in the vascular system causes a reddish-brown discoloration that is observed by cutting into the stem longitudinally.

2.12.2 Causal organism

Verticillium wilt is caused by the fungi *Verticillium albo-atrum* Reinke & Berthold and *V. dahliae* Kleb. The conidiophores are erect, hyaline, and vertically branched with 3-4 phialides at each node. Phialides measure from 16-35 μm $\times$ 1-2.5 μm. Single conidia are produced at the tips of the phialides and are ellipsoidal to irregular, hyaline, and mostly entire but occasionally one septate. Conidia of *V. dahliae* measure an average of 2.5-8 μm $\times$ 1.4-3.2 μm. Conidia of *V. albo-atrum* may be slightly larger than *V. dahliae*. *V. dahliae* has true microsclerotia that are dark brown to black, oval to irregularly round,

and very variable in size ranging from 15-50 μm in diameter. *V. albo-atrum* do not form microsclerotia (Hawksworth and Talboys 1970a and b).

2.12.3 Disease cycle and Epidemiology

The pathogen persists in field and greenhouse soils for many years. Infected plant debris is an important survival site. Microsclerotia of *V. dahlaei* persists in soil in the absence of host tissue. Roots are infected after contact with microsclertia. Weeds can serve as alternative, asymptomatic hosts. Dissemination of the fungi occurs with infected plants or infested soil. Damage to roots by plant parasitic nematodes aid in fungal ingress. Conidia on conidiophores are formed on plant debris.

 V. albo-atrum is a cool weather organism that grows best when soil temperatures are between 18 and 24°C, whereas, *V. dahliae* generally prefers warmer temperatures and is more active between 24°C and 28°C. Even though more infection occurs at lower temperatures, the disease symptoms are more severe at higher temperatures due to restricted water movement in the plant brought about by plugging in the xylem elements earlier in the growing season. The two *Verticillium* species are frequently found within the same field. Races for the two species that vary in virulence and host specificity occur (Bhat and Subbaro 1999).

2.12.4 Management

A crop rotation of 4 to 6 years with non-host plants, primarily monocots (cereals and grasses), should be used to reduce inoculum levels of these fungi (Benson and Ashworth 1976). Plant debris should be removed and destroyed at the end of the season. Fields should be maintained weed-free. Adequate nutrition and water is essential to maintain plant vigor. Overwatering should be avoided. Soil fumigation should be used to reduce both *Verticillium* and nematode populations. Resistant pepper cultivars should be used.

2.13 Wet rot (*Choanephora* blight)

Wet rot is not a common disease of pepper. It does occur occasionally, especially in hot, humid environments. The majority of reports of this disease are from India and southeastern United States. A severe outbreak affecting 100% of plants in a commercial occurred in Florida in 2003.

 The wet rot fungus, *Choanephora cucurbitarum* (Berk. & Ravenel) Thaxter, has a wide host range. Many cucurbits, especially the summer squashes, are very susceptible. Other hosts are okra, cotton, eggplant, snap bean, and papaya.

2.13.1 Symptoms

In Florida, wet rot of pepper occurs most frequently on when temperatures and relative humidities are high and rainfall is abundant (Dougherty 1979). The disease is almost unknown in the winter and spring months of pepper production.

Symptoms are most evident on buds and flowers. Tissue becomes watersoaked, dark green, and soft. The fungus develops rapidly on these tissues, appearing as a mass of tiny black-headed pins sticking in a pincushion. Young, succulent leaves and stems may also be attacked, usually after the fungus becomes established on flower petals or buds. As the disease progresses, flowers and buds wilt and young plants may completely collapse (Sinha 1940).

2.13.2 Casual Organism

Wet rot is caused by C*hoanephora cucurbitarum*, (Berk. & Ravenel) Thaxter a member of the fungal phylum Zygomycota, Class Zygomycetes. The mycelium is generally speaking coenocytic.

Reproduction of *C. cucurbitarum* is by modified sporangia called sporangiola. The sporangiola (sometimes called conidia) result from the evolutionary reduction of large sporangia that contain hundreds of spores to a small sporangium that is monosporous. The spore and the sporangial wall appear fused, hence serving as a single asexual reproductive unit similar to a conidium. Masses of these sporangiola give the fungus its characteristic pincushion appearance. Viewed under the microscope, detached sporangiola have a clearly observed series of longitudinal striations. Sporangiola measure 8-30 μm long by 5-18 μm wide (Alexopoulas *et al.,* 1995).

C. cucurbitarum is heterothallic, with zygospore production possible if both mating types are present in a field. However, long-term survival is more likely by chlamydospores which form in chains within hyphae of older fungal growth. Occasional septation may develop as hyphae age.

2.13.3 Disease cycle and Epidemiology

C. cucurbitarum is especially aggressive under hot, wet conditions. Sporangiola are the most likely form of both primary and secondary inoculum. The fungus may survive in soil, primarily as chlamydospores, and produce mycelium with sporangiola for infection of current season crops. The sporangiola are spread by wind and wind-driven rain to pepper. Flowers seem particularly susceptible and probably represent the initial infection court for most diseased pepper plants.

2.13.4 Management

It is fortunate that wet rot is not an important disease of pepper worldwide, as control is difficult when conditions for development are favorable. Wider plant spacings will help manage this disease by allowing better air movement and more rapid drying of plant surfaces. Avoid overhead irrigation whenever possible. Excessive fertilization, especially with nitrogen, can make plants more succulent and, subsequently more susceptible to wet rot. As with bacterial spot, planting dates can be manipulated, if economically practical, to avoid excessively hot, wet weather. Fungicides applied for other foliar diseases may provide some control of wet rot. No labels exist specifically for wet rot control.

2.14 White mold (*Sclerotinia* stem rot)

The fungus causes a wide range of diseases on more than 350 different host species. It causes recurring, serious losses in tomato, snap bean, and cabbage. It occurs sporadically on pepper, and does not appear to be a major problem on pepper at this time. The traditional names for Sclerotinia disease on pepper are white mold and Sclerotinia stem rot. White mold is found throughout the world wherever cool, damp growing conditions prevail.

2.14.1 Symptoms

White mold is usually first seen on pepper about the time of flowering. When flower petals senesce and die, they fall and lodge in crooks of the pepper branches. The S*clerotinia* fungus invades this non-living petal tissue, allowing the pathogen to develop and advance into the plant itself. Watersoaked spots appear where *Sclerotinia* invades stems. These diseased areas enlarge and portions of the plant above these lesions are killed.

Signs of the fungus usually become apparent as the disease progresses and are highly diagnostic. White cottony growth appears on the outside of stems. Relatively large, black sclerotia may be produced in mycelial mats as well as within stems (Pernezny and Purdy 1981).

Some initial attack may also occur at the soil line, if senescent tissue falls about the base of the plant, providing the needed organic tissue for initial colonization. The resulting symptom is the rapid, complete wilt and collapse of the infected plant. Fruits may also become infected. Initial symptoms consist of watersoaked, dull green spots on fruit. Later, a cottony mycelium develops with embedded sclerotia within fruit (Abawi and Grogas 1979).

2.14.2 Causal Organism

White mold is caused by the Ascomycete fungus, *Sclerotinia sclerotiorum* (Lib.) de Bary. It may be that *S. minor* Jagger may cause some outbreaks of white mold on pepper, but little research has been done to confirm this etiology.

Sclerotia of *S. sclerotiorum* are hard and quite variable in shape. They are usually black on the outside and off-white on the inside. Typically, they measure 3-10 × 3-7 mm. One or more apothecia may form from a sclerotium. The apothecia (sporocarps) are usually cup-shaped with a definite stipe attachment. However, some are quite elongated and almost oppressed to the sclerotial surface. Instead of a clearly recognizable cup, there may be just a small, central dimple. Apothecia are usually white but yellow and light brown have been observed. Within apothecia, asci are cylindric-clavate 130 × 10 μm, with eight ascospores. Ascospores are nonseptate, hyaline, and elliptical (9-13 × 4-5 μm). Hyaline paraphyses occur on the hymenium.

2.14.3 Disease cycle and Epidemiology

S. sclerotiorum is a fungus that prefers cool, moist weather, causing disease of great intensity when temperatures range from 15-21°C. Disease development and spread is

greatly enhanced when humidity is high and dew and misty rains are common.

The fungus overseasons as sclerotia. These sclerotia may be moved to previously uninfested areas with infested soil, in running water, or with infested seed lots or planting stock.

Virtually all pepper inoculum is ascosporic. Sclerotia at or near the soil surface are the source of airborne ascospores. Sclerotia become carpogenic after a reconditioning period of cool, damp weather. It is important to note that sclerotia may survive in soil for many years.

Once sufficiently wetted, asci split and ascospores are ejected and carried by the wind to susceptible pepper. These ascospores require an exogenous carbon source (*i.e.*, senescent pepper flower petals) for initial growth. Once firmly established on the senescent tissue, invasion of the healthy plant can take place (Purdy 1979).

Growing conditions that lead to long periods of moisture retention on shoot surfaces favor white mold development. The disease is always worse when canopies are dense and pepper growth is lush.

2.14.4 Management

In greenhouses and in many areas of fresh-market pepper production, application of broad-spectrum fumigants kills many sclerotia and significantly reduces disease. Windborne ascospores may enter pepper crops from distant points where sclerotia escape destruction by fumigants. Seed free of sclerotia should be planted. Plant spacing and fertilizer regimes should be managed to avoid unnecessarily dense plant canopies.

Flooding of fields for periods of 4-5 weeks may reduce viable numbers of sclerotia (Moore 1949). Sprinkler irrigation generally enhances white mold development compared to drip and other irrigation methods that do not wet the foliage. Proper ventilation of greenhouse pepper crops is warranted to minimize build-up of free moisture on foliage.

3. Diseases caused by Bacteria

3.1 Bacterial soft rot

Soft rot of peppers, caused by subspecies of the bacteria *Erwinia carotovora*, is the most significant postharvest disease problem of pepper fruits. Although the disease is primarily a postharvest problem, it also causes loss of seedlings, stem rot, wilting, and plant death in the field and greenhouses. The disease occurs worldwide in North America, Israel, and Africa. These bacteria have an extremely wide host range including other solaneceous crops such as potato, eggplant and tomato. Soft rot was the predominant parasitic disorder affecting 70% of all shipments of fresh pepper to New York during 1972-1984 (Ceponis *et al.,* 1987).

3.1.1. Symptoms

Symptoms on seedlings in transplant production are stem rot, wilt and collapse (Hadas

et al., 2001). The first appearance of symptoms is a watersoaked lesion at the tip of the cotyledon that extends towards the stem. The crown area develops a brownish soft rot that leads to seedling collapse. Symptoms can also appear as a dark brown to black coloring of veins in the first true leaves prior to rotting. Mature plants in greenhouse production exhibit symptoms of wilting at the time of first fruit harvest or later. Lesions are apparent at the crown and on lower stems that are wet, slimy, black and slough off (Schuerger and Batzer 1993).

Fruit are the primary plant organ affected and soft rot most commonly occurs during post harvest storage and shipment. Infected fruits have water-soaked lesions that are soft and slightly shrunken and dark olive green. The tissue under the fruit skin is macerated which leaves the fruit skin wrinkled and fragile. The rot spreads quickly and the entire fruit may collapse within days. The rotting tissue is susceptible to inva-
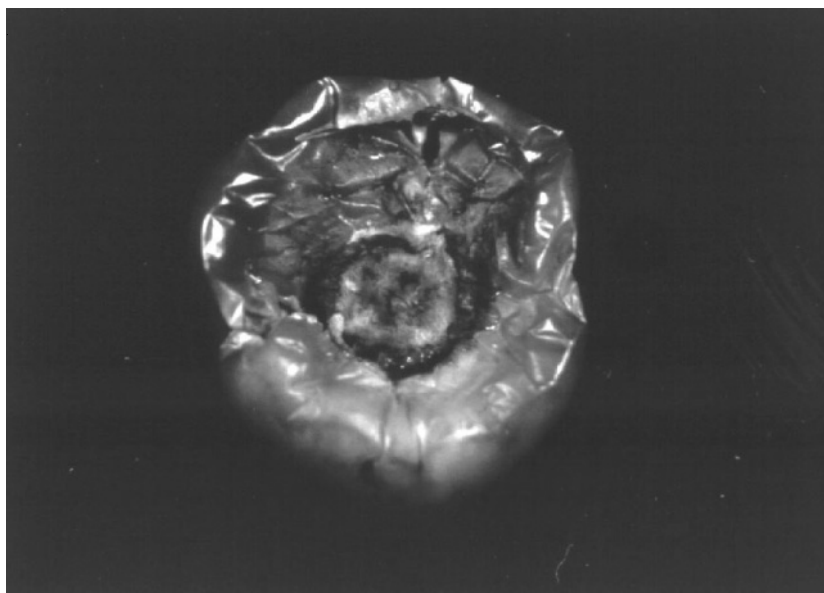
Figure 5: Postharvest decay of pepper fruit infected with bacterial soft rot, gray mold, and secondary microorganisms (Photo courtesy P.D. Roberts)
See also Colour Plates, pp. xv-xix.

sion by numerous other microorganism and fungal mycelium may be present (Fig. 5). In packing boxes, a nest of infected fruit occurs after secondary spread from diseased fruit. Soft rot damage to fruit may also occur in the field especially during rainy periods. Natural wounds at the peduncle and calyx of the fruit provide entry sites for the bacterial cells and symptoms arising at these sites occur the most. Soft rot from these areas typically advances downward into fruit tissue. Infections that begin from wounds made anywhere else on the fruit also will typically spread lengthwise throughout the fruit. Soft rot may advance quickly within a few days of infection. Diseased fruits remain on plants in the field, collapse, and hang loosely from the peduncle. Fruits eventually

desiccate and only a dried shell of the fruit is left intact on the plant.

3.1.2 Causal organism

Both pathovars or subspecies of *Erwinia carotovora* pv. *carotovora* (Jones) Bergey and *E. c. s*ubsp. *atroseptica (*van Hall) Dye *et al.* cause soft rot. The bacterium is gram negative, facultatively anaerobic, strongly pectolytic, non-spore-forming, rod-shaped (0.7×1.5 μm) and motile by peritrichous flagella. The cells occur singly, in pairs, or occasionally in short chains. A differential characteristic of the two species is growth at 37°C; *E. c.* subsp. *atroseptica* is unable to grow at 37°C whereas *E. c.* subsp. *carotovora* grows at 36-37°C. *E. chrysanthemi* also causes a soft-rot at temperatures similar to *E. c.* subsp. *carotovora* (Smith and Bartz 1990, Strommell *et al.,* 1996).

3.1.3 Disease cycle

The bacteria infest the surface of pepper seeds providing a primary and important source of inoculum and significantly reduce seed germination. Bacterial population declines within 6 and 8 months on seeds stored at 23°C and 6°C, respectively (Hadas *et al.,* 2001).

Strains of *Erwinia* spp. commonly occur on the surface of many different plants, on roots, and free-living in the soil and water. The bacteria can be transmitted in water or by mechanical means on workers or contaminated equipment. In the greenhouse, contaminated re-circulating water can rapidly distribute the bacteria throughout the crop.

Fields previously cropped to potato or cabbage have higher populations of *Erwinia* sp. in the soil (Coplin 1980). Fruit are contaminated in the field with the bacteria and post-harvest washing of the fruit effectively spreads the bacteria and provides it with easy access to wounds. Natural wounds such as those at the stem and wounds made during harvest or cultural activities provide sites of entry. Feeding insects transmit the soft rot bacteria from fruit to fruit. Injury to fruit by sunscald predisposes it to soft rot. Severe outbreaks of foliar diseases that expose fruit to sunscald injury result in increased incidence of soft rot. High humidity and rainfall favor disease development.

The storage of fruit at 23°C resulted in much greater fruit decay by *E. c.* subsp. *carotovora* compared to *E. c.* subsp. *atroseptica*. In contrast, storage of fruit at 10°C caused greater losses by *E. c.* subsp. *atroseptica* than by *E. c.* subsp. *carotovora* (Strommel *et al.,* 1996). However, all amounts of fruit decay were greatly reduced at 10°C compared to 23°C

3.1.4 Management

Seeds should be surface sterilized with sodium hypochlorite or 10% solution of trisodium phosphate to reduce bacteria on the seed surface (Hadas *et al.,* 2001). In the field, damage by insects should be controlled to reduce the spread of bacteria and its entry into wounds. Foliar diseases should also be controlled to maintain sufficient foliage to prevent sunscald injury that provides entry for bacteria. Fruits from plants fertilized

with higher rates of nitrogen (266 kg/ha) and potassium applied by fertigation had less incidence of bacterial soft rot in experimental trials (Carballo *et al.,* 1994). Weeds and volunteer plants around the field should be eliminated. Avoid wounding fruits during harvest and packinghouse operations and only harvest fruits when plants are dry. Harvesting containers and the interior of packinghouses should be kept clean and disinfested.

In the packinghouse, avoid washing fruit whenever possible. Fruit that must be washed should be by chlorinated water only and wash-water must be maintained pathogen-free. Peppers picked during the heat of the day should not be subjected to cold water since this allows water and surface contaminants like bacteria to be drawn into the fruit. Packinghouse culling operation must be carefully monitored to ensure removal of diseased or damaged fruit. Fruit should be dried by forced air prior to packing to mitigate likelihood of soft rot. Storing fruit at temperatures lower than 23°C reduces disease incidence. Although hot water treatment may reduce bacterial soft rot, subsequent hydrocooling of infected fruit leads to greater decay.

Differences in tolerance of pepper fruit were reported but resistance in commercial cutivars is not generally available (Bartz and Stall 1974). There are no good chemical controls that can be used in the field to prevent Erwinia soft rot.

3.2 Bacterial Spot

Bacterial spot of pepper was first reported in 1918 (Sherbakoff 1918) and later determined to have the same as the causal agent as bacterial spot of tomato, incited by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye (Gardner and Kendrick 1923, Higgins 1922) Bacterial spot is present in most pepper-growing regions; however, it is most severe in tropical and subtropical regions where significant precipitation and high temperatures occur during production. The disease is most important in the southeastern United States, especially in Florida where it is a major problem. Crop loss results both from the actual yield reduction occurring from defoliation and from severely spotted fruit that renders the fruit unfit for market. Furthermore, sunscald and resulting fruit decay occurs often times as a result of defoliation and fruit decay.

3.2.1 Symptoms

The disease is associated with all above-ground plant parts. Spots begin as small, brown lesions that may be water-soaked in appearance and become brown. The spots may become dark brown (Fig. 6). A water-soaked appearance is more readily observed during a rainy period or when dew is present. Lesions may enlarge to 10 mm in diameter. The lesions are generally sunken on the top surface of the leaf and slightly raised on the bottom. When conditions are optimal for disease development, spots on the leaves coalesce and have a blighted appearance. A general yellowing may occur on infected leaflets and often leads to premature defoliation .

Stem lesions are narrow, elongated and raised expanding up to 6 mm. The lesions become light brown and rough in appearance. Fruit lesions are prominent beginning as green spots. As a spot enlarges, it becomes brown in color, and raised with a

cracked, roughened, wart-like appearance (Fig. 7). The lesions range in size, up to 5 mm in diameter. During periods of high moisture, fruit rot may develop around the lesions.

3.2.2 Causal organism

Bacterial spot is incited by two major groups of bacteria, which represent two bacterial species (Bouzar *et al.,* 1994, Stall *et al.,* 1986). These are *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye, designated the A group, and *Xanthomonas vesicatoria* (Doidge). The bacteria are motile, strictly aerobic, Gram-negative rod, and possess a single polar flagellum. On nutrient agar the bacteria grow relatively slowly, with the colonies being circular, wet, shiny, yellow, and entire in appearance. The A group consists of strains which are generally non-amylolytic and non-pectolytic, whereas the B group consists of strains which are positive for both characteristics (Bouzar *et al.,*

Figure 6 : Bacterial spot lesions on foliage of pepper (Photo courtesy K. Pernezny)
         See also Colour Plates, pp. xv-xix.

1994). Eleven pepper races have been identified based on the differential reactions to four resistance genes (Minsavage *et al.,* 1990). Strains occur that are pathogenic on pepper only, tomato only, and on both pepper and tomato.

3.2.3 Disease cycle and Epidemiology

The bacterium is seed-borne being present within or on the surface of the seed (Bashan *et al.,* 1982). Dissemination of the bacterium on seed and transplants is important. The bacterium is also able to survive on crop residue up to several months (Peterson 1963). Temperatures between 24°C and 30°C, high precipitation, and high relative humidity

(>85%) favor disease development. Low relative humidity is unfavorable to the disease and can greatly inhibit disease development following infection. The bacterium is disseminated within a field by wind-driven rain droplets, clipping of transplants, and aerosols (McInnes *et al.,* 1988). The pathogen penetrates directly through stomates and hydathodes and through wounds created by wind-driven sand, insect punctures, or mechanical means.

3.2.4 Management

Bacterial spot is extremely difficult to control using conventional bactericides (Marco and Stall 1983). The antibiotic streptomycin has been effective for controlling bacterial spot when sensitive strains are present; however, as a result of exposure to the antibiotic, resistant strains develop quickly and disease control is reduced (Thayer and Stall

Figure 7: Bacterial spot lesions on pepper fruit (Photo courtesy K. Pernezny)
      See also Colour Plates, pp. xv-xix.

1961). Copper bactericides have provided control of copper-sensitive strains, although the presence of copper-tolerant strains makes control with copper compounds extremely difficult. Copper-tolerant strains are more effectively controlled by the combination of a copper bactericide and mancozeb or maneb (Marco and Stall 1983, Stall *et al.,* 1986).

    As a result of bactericides providing mediocre control of bacterial spot when environmental conditions are optimal for disease development, other control strategies are necessary to reduce disease losses. First of all, cultural practices must be modified to minimize the chances for a disease outbreak. In situations where transplants are used, they should be produced in an area where field production is not in close proxim-

ity. Pathogen-free seed should be used in production of disease-free transplants to minimize the introduction of exotic strains. Seed treatment should be used to reduce possible transmission of the bacterium. In situations where overhead watering of transplants is used, this should be limited to minimize the incidence of disease on transplants used in field production. Crop rotation must be practiced in an attempt to avoid carryover on volunteers and crop residue. Never follow peppers by peppers or tomatoes. Cull piles are a source of inoculum and should be avoided near greenhouse or field operations.

Seed treatment to reduce the bacterial populations may be an effective method for reducing disease problems in the field. Several methods have been reported for disinfesting and/or disinfecting seed of the bacterial spot pathogens. First, hot water treatment may be used in which the seed are submerged in water heated to 50° C for 25 minutes. This method may result in reduced seed germination and is not completely effective although incidence is drastically reduced. A second method consists of soaking the seeds in a copper sulfate solution (1 ounce $CuSO_4$ in 4 gallons water) for 10 minutes, followed by drying the seed and dusting with finely ground lime before sowing. A third treatment involves disinfesting the seed surface in which the seed are soaked for 1 minute in 1.3% sodium hypochlorite (1 part 5.25% Clorox plus 3 parts water).

Control of bacterial spot by plant resistance is promising. Three single dominant genes have been identified in *Capsicum*, which confer resistance to *Xanthomonas* strains pathogenic on pepper (Bonas *et al.,* 1989, Cook and Stall 1963, Cook and Guevara 1984, Kim and Hartmann 1985). The genes, designated *Bs1*, *Bs2*, and *Bs3* were first found in PI 163192 (*C. annuum*), PI 260435 (*C. chacoense*), and PI 271322 (*C. annuum*), respectively. None of the genes result in resistance to all strains of *Xanthomonas* pathogenic on pepper, however. Recently another source of resistance has been found in PI 235047 (*C. pubescens*). Eleven races have been distinguished using 'Early Calwonder' near-isogenic lines containing one of the three single resistance genes and the resistance discovered in *C. pubescens*. Resistant varieties containing the *Bs1*, *Bs2*, and/or the *Bs3* are available and may provide excellent control, when pepper races with the corresponding avirulence gene are present in the field. However, pepper races exist that are aggressive on varieties containing one, two or all three resistance genes (Dahlbeck and Stall 1979, Kousik and Ritchie 1996, Pohronezny *et al.,* 1992).

3.3 Bacterial wilt (Southern bacterial wilt)

Bacterial wilt or Southern bacterial wilt, caused by *Ralstonia* [*Pseudomonas*] *solanacearum*, (Smith 1896) Yabuuchi *et al.,* 1995 is a limiting disease problem in tropical, subtropical, and some warm temperate climates. The disease occurs in greenhouses in cooler climates. Bacterial wilt causes disease on more than 200 plants species belonging to over 40 families. Major agronomic hosts include pepper, potato, tobacco, tomato, eggplant, and banana. Reports of complete crop loss in the field are not uncommon.

3.3.1 Symptoms

Initial symptoms of bacterial wilt on pepper plants are sudden wilting of foliage on the

youngest leaves. Plants remain green and may recover during the night. Only one side or part of the plant may be affected. Eventually, the plant does not recover from wilting and dies. The vascular system in affected plants is discolored a pale yellow to a dark brown color. A simple diagnostic test is to place the lower part of the cut stem into water. A milky, white exudate of bacteria will be seen within minutes streaming from the cut tissue.

3.3.2 Causal organism

The disease is caused by *Ralstonia solanacearum* (Smith 1896) Yabuuchi *et al.,* 1995. *Ralstonia solanacearu,* formerly known as *Pseudomonas solanacearum*, is a gram-negative rod, $0.5 \times 2.0\,\mu m$, aerobic, and motile by one to four polar flagella.

Strains of *R. solanacearum* differ in host range, geographical distribution, pathogenicity, and physiological properties. Strains are subdivided informally into five races based upon host range or differentiated into five biovars based upon the utilization and/or oxidation of three disaccharides and three sugar alcohols as carbon sources (Buddenhagen *et al.,* 1962, Hayward 1991). Race 1 has a very wide host range of crops and weeds and strains are predominately biovars 1, 3 and 4. Race 1 strains cause bacterial wilt on pepper and other solanaceous crops of potato, tobacco, tomato, and eggplant. Race 2 strains cause Moko disease on banana and diseases on heliconias. In general, race 3 and biovar 2 strains are the same and affect mainly potato but occasionally pepper and other solanaceous crops and weeds. Race 4 and 5 have host ranges limited to mulberry and ginger, respectively (He *et al.,* 1983). Molecular analysis by restriction length fragment polymorphism and 16S rRNA grouped all the strains tested into two major divisions that were related to geographical origin of the strains (Cook *et al.,* 1989, Poussier *et al.,* 2000). Two separate groups of strains apparently evolved independently. The 'Asiaticum' division probably originated in Asia and contains all members of race 1 biovars 3,4, and 5. The 'Americanum' division probably originated in Central and South America and contains all members of race 1 biovar 1 and races 2 and 3. (Cook *et al.,* 1989).

3.3.3 Disease cycle and Epidemiology

The bacteria are able to survive in moist soil for longs periods of time in moist soil in the absence of host material. The length of survival period depends on the strain of bacteria, soil type, and soil moisture. Survival is longest in moist soil with low to moderate pH although the disease occurs in almost all soil types or soil pH where host plants normally grow. The bacteria also survive in water for long periods of time. The bacteria overseason in plant debris. The two biovars that typically cause bacterial wilt of pepper have a wide host range which means that *R. solanaceous* is widely present. It is not unusual to find reports of it causing disease on crops planted the first time in previously uncultivated land. Dissemination of the bacteria occurs with irrigation or rain water, on infected plants, infested seeds of tomato and eggplant, and the soil (Goto 1990).

Entry into the plant is via roots through growth cracks or wounds incited from nematode damage, insects or farm equipment. The bacteria colonize the xylem and

spread throughout the plant, multiplying to a high population density. Wilting of the plant may be observed within days of infection under favorable environmental conditions. The bacteria invade the intercellular spaces of the parenchyma cells in the cortex and pith and disintegrate the cell walls resulting in pockets filled with bacterial cells and polysaccharide and cellular debris. Bacteria are released back into the soil from infected plants roots and decaying plant material (Bruehl 1987).

The bacterial wilt pathogen generally does not survive where the mean temperature is below 10°C although survival of some strains of *R. Solanacearum* occurs in temperate climates (van Elsas *et al.,* 2000). The disease usually develops when mean temperatures are above 20°C and more severe wilting is seen at higher temperatures of 30°C or higher and high soil moisture.

3.3.4 Management

The most effective and often only means of control is to avoid planting in infested soil. The disease should be avoided by using pathogen-free seeds grown in sterile soil and transplant trays. A long rotation with non-host crops, particularly grasses, may reduce the population in infested fields. The grafting of susceptible tomato and eggplant scions onto resistant rootstocks has been used and may be an option for pepper also. Fields should be kept free of weeds and volunteer plants. Nematodes in soil should be eradicated since their presence increases bacterial wilt incidence. Soil should not be moved from infected fields on equipment or workers. Workers and equipment should be disinfested. There is no effective chemical control for bacterial wilt although soil fumigation may reduce the population of *R. solanacearum* in soil. Amendments to the may reduce disease incidence by creating suppressive conditions.

4. Diseases caused by viruses

Plant viruses are obligate parasites that can cause significant economic losses in peppers. Major viral pathogens of peppers are found in the *Potyvirus*, *Tobamovirus*, *Tospovirus*, *Cucumovirus* and *Begomovirus* genera. A more extensive list of viruses infecting peppers is presented in Table 1. Insects are important vectors for many of the pepper viruses and include various species of aphids, thrips and whiteflies. Mechanical and seed transmission are highly efficient means of spread for some pepper viruses. Symptoms induced by viruses infecting peppers can vary significantly with cultivar, plant age, virus isolate and environment. Therefore, a reputable diagnostic laboratory should be used to ensure accurate identification of the virus.

4.1 *Potyvirus* genus

Pepper mottle virus (PepMoV), *Tobacco etch virus* (TEV) and *Potato virus Y* (PVY) are members of the *Potyvirus* genus. TEV and PVY are distributed worldwide while PepMoV is currently limited to the Americas and Southeast Asia. Potyviruses are significant pathogens of many solanaceous crops including pepper, potato, tomato and tobacco. TEV is one of the most important viral diseases of pepper worldwide and incidence of

Table 1: Viruses infecting solanaceous crops.

Virus	Virus Genus	Distribution	Transmission	Host Range/Reservoir
Tobacco mosaic virus (TMV)	Tobamovirus	Worldwide	Mechanical Seedborne	Solanaceae/Solanaceous weeds Crop debris
Tomato mosaic virus (ToMV)	Tobamovirus	Worldwide	Mechanical Seedborne	Solanaceae/Solanaceous weeds Crop debris
Pepper mild mottle virus (PMMoV)	Tobamovirus	Worldwide	Mechanical Seedborne	Solanaceae/Crop debris
Potato virus Y (PVY)	Potyvirus	Worldwide	Aphid	Solanaceae/*Solanum and Physalis spp.*
Tobacco etch (TEV)	Potyvirus	China, N and S America	Aphid	Solanaceae/*Datura,* virus *Cassia* spp., tomato, *Solanum*
Pepper mottle virus (PepMoV)	Potyvirus	N and C , America, Asia	Aphid	Solanaceae/Solanaceous weeds
Chilli veinal mottle virus (ChiVMV)	Potyvirus	Asia	Aphid	Solanaceae/Eggplant, tobacco, tomato
Pepper veinal mottle virus (PVMV)	Potyvirus	Africa, Asia	Aphid	Solanaceae/tomato, pepper
Tomato spotted virus (TSWV)	Tospovirus	Worldwide	Thrips	Wide/ Solanaceous wilt ornamentals,crops, weeds,
Tobacco rattle virus (TRV)	Nepovirus	N, C and S America, Asia, Europe Australia	Nematode,	> 9 families/Weeds, crops
Cucumber mosaic virus (CMV)	Cucumovirus	Worldwide	Aphid	Wide /Perennial weeds
Alfalfa mosaic virus (AMV)	Alfalfamo-virus	Worldwide	Aphid	Wide /Alfalfa, clover
Beet curly top virus	Curtovirus	Worldwide, especially in irrigated & arid regions	Leafhopper	Moderate 300 species/ Russian thistle, sugar beet
Pepper hausteco virus (PHV)	Begomovirus	Mexico, USA	Whitefly	Pepper, tomato/Tobacco, Datura stramonium

table 1 contd...

Pepper mild tigre virus	Begomovirus	Mexico, USA	Whitefly	Pepper/ Tomato
Serrano golden mosaic virus (SGMV)	Begomovirus	N & C America	Whitefly	Pepper,tomato, tobacco / *Datura, Nicotiana and Physalis spp.*
Tomato yellow leaf curl virus (TYLCV)	Begomovirus	Worldwide	Whitefly	Solanaceae/*Datura Malva,stramonium, nicaeensis*
Sinaloa tomato leaf curl virus (STLCV)	Begomovirus	C America	Whitefly	Pepper, tomato/*Datura spp., Malva parviflora*

TEV reached 96% in naturally infected pepper fields during a single growing season (Benner *et al.,* 1985). PepMoV is a serious pathogen in the southern United States, Mexico and Central America. The *Potyvirus* genus currently contains the largest number of members, including some of the most economically important plant viruses. Other potyviruses infecting pepper include *Pepper severe mosaic virus, Chilli veinal mottle virus* and *Pepper veinal mottle virus.*

4.1.1 Symptoms

Symptoms induced by potyviruses in pepper are greatly influenced by pepper cultivar and virus strain. In general, a mosaic or mottle is observed on the leaves of plants infected with PepMoV, PVY and TEV and is frequently accompanied by dark green vein-banding. Symptoms of PepMoV are shown in on foliage and fruit in Figure 8. Leaves are commonly distorted and sometimes fruit is also. TEV may induce large concentric rings and line patterns on both leaves and fruit. Fruit from TEV-infected plants are often misshapen with brown to black, flattened or sunken blotches in the fruit wall. Plants infected early in the season with any of these three viruses will be stunted and have both reduced fruit set and size. Some pepper cultivars wilt and die following TEV infection resulting in total crop loss. PepMoV, TEV and PVY are frequently found in mixed infections, sometimes with each other, which often results in severe symptoms.

4.1.2 Causal agent

PVY is the type member of the *Potyvirus* genus of RNA viruses, which also includes PepMoV and TEV. All potyviruses form flexuous, rod-shaped particles ranging from 680-900 nm in length. Potyvirus genomes consist of one positive-sense, single-stranded RNA (De Bokx, J.A. and Huttinga 1981, Nelson *et al.,* 1982, Sanchez and Grogan 1970). TEV has a fairly wide host range infecting both solanaceous plants and members of at least nine other plant families (Purcifull and Heibert 1982). PVY and PepMoV are prima-

rily restricted to solanaceous plants. Potyviruses frequently induce distinctive pinwheel or cylindrical cytoplasmic inclusions.

4.1.3 Disease cycle and Epidemiology

Potyviruses are transmitted by any of at least ten species of aphids in a non-persistent manner. The green peach aphid, *Myzus persicae,* is a common and efficient vector for PepMoV, TEV and PVY (Lopez-Moya and Garcia 1999). Adjacent fields of solanaceous crops (especially potato, tomato and tobacco) are common sources of potyviruses and their vectors.

  Potatoes are perhaps the most common source of initial PVY inoculum in temperate climates. A number of weeds, solanaceous and other, provide initial inoculum for

Figure 8: Pepper leaves and fruit exhibiting symptoms of Pepper mottle virus (Photo
   courtesy K. Pernezny). *See also Colour Plates, pp. xv-xix.*

PepMoV, TEV and PVY, especially in subtropical and tropical climates where they also serve as oversummering hosts. TEV occurs naturally in many weeds including *Solanum* spp. (as horsenettle and nightshade), *Cirsium vulgare* (thistle), *Chenopodium album* (lambsquarters), *Nicandra physalodes* (Apple of Peru), and *Datura stramonium* (Jimsonweed). PVY also occurs naturally in many solanaceous weeds (as *Solanum nigrum* and *Solanum atropurpureum*) and *Physalis* spp. (Anderson 1958, Chagas *et al.,* 1977). PepMoV, TEV, and PVY commonly occur as mixed infections in pepper. PepMoV is frequently found with tobamoviruses while TEV and PVY often occur together.

4.1.4 Management

Distancing pepper plantings from potato, tomato, tobacco and other solanaceous crops is the best way to limit potyvirus infection. Solanaceous weeds that can harbor potyviruses and aphids should be removed, when possible, from around production fields to further reduce inoculum. Plant residue from previous solanaceous crops should also be removed or incorporated into the soil before new plantings.

Cultivars resistant to TEV and PVY are available and can help control these viruses. Cultivars resistant to PVY can also be effective against TEV because resistance to both viruses is linked. However, a few strains of TEV can overcome PVY resistance genes. Plants can be sprayed with mineral oil to the reduce frequency of virus acquisition and transmission by aphids. Insecticide applications to control the vector are usually inefficient and not recommended. Border crops taller than pepper can impede aphid entry into fields and weekly insecticide treatment of these border crops can help reduce aphid populations. The use of reflective mulches can reduce the number aphids landing on crops. When practical, pepper production can be timed to coincide with periods of low aphid population.

4.2 *Tobamovirus* genus

Tobacco mosaic virus (TMV), *Tomato mosaic virus* (ToMV) and *Pepper mild mottle virus* (PMMoV) are members of the *Tobamovirus* genus. All three viruses are distributed worldwide and can infect many field and greenhouse crops including pepper. TMV and ToMV infect tomato and tobacco while PMMV is restricted to *Capsicum* spp. Common strains of TMV and ToMV can often be controlled in pepper through the use of resistant cultivars and therefore is not generally a problem in commercial plantings. TMV was the first virus of any type ever discovered.

4.2.1 Symptoms

Pepper cultivar and virus strain greatly affect the symptoms induced by tobamoviruses in pepper. In general, a mosaic or mottle of dark and light green or yellow is observed on the leaves of plants infected with these viruses, especially TMV and ToMV (Hollings and Huttinga 1976).

PMMoV usually induces a less dramatic foliar color change (as its name implies) although it frequently causes leaves to pucker. Plants infected at an early age by any of these three viruses are stunted. Some combinations of cultivars and TMV or ToMV strains cause more severe symptoms including defoliation, premature flower drop, wilting, stem death and necrosis of tissue inside the fruit wall. In some cultivars, PMMoV causes deformation of fruit (lumpy and mottled), brown streaks on fruit and undesirable fruit color at maturity. High temperature and low light intensity can increase symptom severity and disease development.

4.2.2 Causal agent

TMV is the type member of the *Tobamovirus* genus of RNA viruses, which also in-

cludes ToMV and PMMoV (Lewandowski and Dawson 1999). All tobamoviruses form rigid, rod-shaped particles approximately 300 nm in length. Tobamovirus genomes consist of one positive-sense, single-stranded RNA (Wetter and Conti 1988, Zaitlin and Israel 1975). TMV has a wide host range while ToMV and PMMoV have more limited host ranges. The Solanaceae family contains many species susceptible to infection by all three viruses. Tobamovirus particles frequently form large hexagonal crystals visible by light microscopy.

4.2.3 Disease cycle and Epidemiology

Tobamoviruses form very stable particles making them easy to spread and difficult to eliminate. They are readily transmitted by mechanical means including workers' hands, clothing or tools (Gibbs 1977). No insect vectors are required for tobamovirus transmission nor are any generally involved. Tobamoviruses are carried on the seed coat although the embryo is not infected. Plant debris can harbor infectious virus particles for many years. Hence, infested seed and debris from previous crops are the most common sources of primary inoculum. Infected weeds and nearby plantings of infected crops are also sources of inoculum. Secondary spread within a field or greenhouse occurs by contact between plants, which is frequently mediated by workers handling plants or using virus-contaminated tools during staking or tying. The nutrient solution in hydroponic pepper production spreads the virus from plant to plant (Pategas *et al.,* 1989).

4.2.4 Management

Virus-free seeds should be used and care should be taken to not infect transplants during production to ensure that only uninfected transplants are set in the field. Seed may be disinfested with heat, acid or trisodium phosphate treatment. Sanitation is important during both transplant and field production phases. Workers handling plants should avoid concurrent use of tobacco products as they may contain tobamoviruses. Workers should also wash their hands with strong soap or 70% alcohol prior to handling plants. Common strains of TMV and ToMV can often be controlled in peppers through the use of resistant cultivars, frequently containing a single dominant resistance gene. However, certain strains of TMV and ToMV can overcome this resistance. Although genes for PMMoV resistance are known, very few resistant cultivars are commercially available. When tobamovirus infection has already occurred, plant handling should be minimized to reduce virus spread. Infected fields or greenhouses should be worked at the end of the day to avoid introducing the virus to uninfected areas. Tools and equipment should be cleaned with trisodium phosphate or household bleach.

4.3 *Tospovirus* genus

Tomato spotted wilt virus (TSWV) infects peppers and a wide range of other crops across temperate, subtropical and tropical regions worldwide. Spotted wilt was first reported in 1915 in Australia on tomatoes and later demonstrated to be of viral origin. In addition to pepper and tomato, major crops susceptible to TSWV infection include

lettuce, potato, peanut and tobacco (German *et al.,* 1992). Infection rates of 50-90% lead to major losses in commercial vegetable crops making TSWV one of the most economically destructive plant viruses of recent times (Cho *et al.,* 1986).

4.3.1 Symptoms

The appearance and severity of spotted wilt symptoms in pepper vary widely depending on the cultivar, virus isolate, stage of plant growth at time of infection, and environmental conditions (German *et al.,* 1992). Plants infected at the transplant stage usually are severely stunted throughout the growing season and frequently yield no fruit. Leaves and stems on plants infected later in the season may develop chlorotic or necrotic flecking and/or chlorotic or necrotic ringspots (Fig. 9). TSWV infection can

Figure 9 : Pepper leaves exhibiting symptoms of Tomato spotted wilt virus infection (Photo courtesy P.D. Roberts) *See also Colour Plates, pp. xv-xix.*

cause flower and leaf drop in some cultivars. Fruit of infected plants may develop chlorotic or necrotic spots, ring patterns and/or mosaic that are especially evident as an undesirable fruit color at maturity.

4.3.2 Causal agent

TSWV is the type member of the *Tospovirus* genus of RNA viruses (Adkins 2000). TSWV forms 80-120 nm pleomorphic particles with surface projections composed of two viral glycoproteins. The genome consists of one negative and two antisense single-stranded RNAs, each encapsidated by multiple copies of the viral nucleocapsid protein

to form ribonucleoprotein structures also known as nucleocapsids. The nucleocapsids are enveloped in a host-derived membrane bilayer along with the viral L protein, the putative RNA-dependent RNA polymerase. More than 800 plant species, both dicots and monocots, in more than 80 plant families, are susceptible to TSWV. The Solanaceae and Compositae families contain the largest numbers of susceptible species.

4.3.3 Disease cycle and Epidemiology

TSWV is transmitted from plant to plant almost exclusively by several species of thrips (Thysanoptera:Thripidae) (Sherwood *et al.,* 2000). The western flower thrips (*Frankliniella occidentalis*) and the tobacco thrips (*F. fusca*) are major vector species although *F. schultzei*, *F. intonsa*, *F. bispinosa*, *Thrips tabaci* and *T. setosus* can also transmit TSWV. Only larval thrips can acquire TSWV, while both the larval and adult thrips can transmit the virus in a persistent, though often sporadic fashion. TSWV replicates in its thrips vectors in addition to its plant host. Virus and vector are commonly spread through transport of vegetable transplants and ornamentals. Adjoining fields of agronomic crops (often tobacco or peanut) and nearby weeds are also common sources of both virus and vector (Goldbach and Peters 1994).

4.3.4 Management

Control of TSWV is difficult because of the extremely wide and overlapping host range of the virus and its thrips vectors. A scarcity of host plant resistance genes and a large number of weed and ornamental hosts providing between-crop virus reservoirs exacerbate the situation. Multicomponent management approaches are most effective. The use of virus-free transplants is a necessity. In greenhouse production, thrips-proof screens can prevent or delay infection. Chemical control of the vector is generally not recommended, as it is difficult to achieve good coverage of the thrips and to kill them before they transmit the virus. There are few, if any, commercial pepper cultivars with TSWV resistance although there are promising developments in pathogen-derived (transgenic) resistance in tomato and tobacco.

4.4 *Cucumovirus* genus

Cucumber mosaic virus (CMV) is one of the most important viral pathogens of pepper worldwide. The host range contains over 1200 plant species in 100 families. CMV occurred in infected pepper fields with a disease incidence of 20-50% (Benner et al 1985). Major crops such as tomato, cucumber, melon and squash are susceptible to CMV infection as are numerous weed species.

4.4.1 Symptoms

Symptoms caused by CMV are quite variable, in large part due to differences between virus strains and host reactions. Plants infected early in the season have more severe symptoms and reduced fruit quality and set. Stunting is a common symptom of peppers infected young and is frequently accompanied by a lighter than normal shade of green

in and an overall thickening of the leaves. Leaves may also be elongated and narrowed or have chlorotic or necrotic ringspots or oakleaf patterns. Chlorotic or necrotic ringspots may also develop on the fruit. Plants that are older when infected may have very mild to no visible symptoms (Roossinck 1999).

4.4.2 Causal agent

CMV is the type member of the *Cucumovirus* genus of RNA viruses. CMV forms icosahedral particles of approximately 29 nm in diameter. The genome consists of three positive-sense, single-stranded RNAs. The largest two genomic RNAs are packaged independently while the third genomic RNA is packaged together with a subgenomic messenger RNA for the coat protein. Neither the coat protein nor its messenger RNA is required to initiate infection (unlike *Alfalfa mosaic virus*) (Roossinck 1999). Satellite RNAs are often associated with CMV and can either intensify or ameliorate host symptoms (Francki *et al.,* 1979).

4.4.3 Disease cycle and Epidemiology

CMV is transmitted by any of at least 75 species of aphids in a non-persistent manner. The most efficient vectors are the aphids *Aphis gossypii* and *Myzus persicae*. Transmission efficiency varies with the aphid species, the virus strain, host plant species, environmental conditions and the season (Francki *et al.,* 1979). CMV survives between crops in perennial weeds. Adjacent fields of susceptible crops (especially cucurbits) are also common sources of both virus and vector.

4.4.4 Management

Greenhouses for transplant production should be isolated from field sources of CMV. Weeds should be eradicated from around greenhouses and infected plants should be discarded. In field production, weeds (especially perennials) that harbor aphids or virus should be removed when possible. Crop debris should also be removed or buried. A barrier of a taller crop (such as sugar cane or sweet corn) can be planted around the field margins to interfere with aphid flight into the field. Weekly insecticide sprays of the barrier crop can reduce aphid populations. Reflective mulches that confuse or impair aphids from landing on plants can be used. Mineral oil sprays may also be used to interfere with the acquisition and transmission of CMV and other aphid-transmitted pepper viruses. Acceptable levels of tolerance to CMV are available in a few commercial pepper cultivars, although the resistance may be limited to certain, not all, strains of CMV. Viral satellite-mediated protection with a mild strain of CMV or by a mild strain of CMV alone shows promise for cross-protection against more virulent strains of CMV (Montasser *et al.,* 1998).

4.5 *Begomovirus* genus

Pepper huasteco virus (PHV), *Chino del tomato virus* (CdTV) *Pepper mild tigré virus*

(PMTV) and *Serrano golden mosaic virus* (SGMV) and *Sinaloa tomato leaf curl virus* (STLCV) are the members of the *Begomovirus* genus. SGMV and Texas pepper viruses are currently recognized as synonyms for the same virus. Begomoviruses are significant pathogens of major crops worldwide including tomato, bean and cotton. Occurrence of these viruses in pepper has generally been documented in subtropical and tropical regions of North and Central America and has become an important pathogen of pepper in this area (Idris *et al.,* 1999a,b, Lotrakul *et al.,* 2000, Rojas *et al.,* 2000). Begomoviruses infecting pepper are often restricted to solanaceous hosts and have not yet become a worldwide production limitation. Other begomoviruses infecting pepper include *Tomato yellow leaf curl virus* and *Sinaloa tomato leaf curl virus* although these two viruses are usually found in tomato (Brown *et al.,* 1993, Idris and Brown 1998 and Idris *et al.,* 1999a and b). New viruses will probably continue to be identified and the relationship and groupings of these viruses within the *Begomovirus* genus will occur as sequences are compared to each other.

4.5.1 Symptoms

Symptoms induced by begomoviruses in pepper are influenced by pepper cultivar and virus isolate. A distinct, bright yellow mottle or mosaic is observed on leaves of plants infected with PHV or SGMV while interveinal chlorosis is induced by PMTV. STLCV causes leaf curl and mild yellow mosaic. CdTV causes very mild symptoms on pepper plants and stripes on fruit. Other symptoms that may occur are leaf curling, crinkled and/or distorted leaves, bright yellowing of veins, and stunting. Fruits may be misshapen, smaller, discolored and have necrotic ends. Plants infected early in the growing season are frequently stunted have reduced yields.

4.5.2 Causal agent

PHV, CdTV, SGMV, and STLCV are members of the *Begomovirus* genus (type member *Bean golden mosaic virus*) in the family *Geminiviridae* of DNA viruses. Typical of the geminiviruses, they form geminate (twinned) particles of about 18×30 nm. Each virus particle contains one circular, single-stranded genomic DNA molecule. There may be one or two types of genomic DNA molecules depending on the virus. Most begomoviruses have narrow, dicotyledonous host ranges (Buck 1999, Rybicki *et al.,* 2000, Stenger *et al.,* 1990).

4.5.3 Disease Cycle

All begomoviruses are transmitted exclusively by the B biotype of the whitefly, *Bemisia tabaci*, which is also known as the silverleaf or sweet potato whitefly. Some entomologists have characterized this biotype as a separate whitefly species, *B. argentifolii*. An increase in whitefly population generally precedes the appearance of the begomoviruses. Adjacent fields of tomato and other solanaceous crops frequently provide initial inoculum and whitefly for infection of pepper, especially in areas with overlapping tomato and pepper production. Solanaceous weeds can also provide initial inoculum for

begomoviruses by serving as oversummering or between-crop reservoirs. Mixed infections of geminiviruses have been observed in pepper and frequently lead to induction of more severe symptoms and greater yield loss than the individual viruses.

4.5.4 Management

Distancing pepper fields from tomato and other solanaceous crop production areas is probably the best way to limit begomovirus infection. Transplants should be produced free of the virus and plants must be free of the whitefly vector. Another important control strategy is to reduce or eliminate whitefly populations. Removal of solanaceous weeds that can harbor begomoviruses and whiteflies is advised when practical. Pepper and tomato crop residue should be removed promptly and destroyed at the end of the season. Begomovirus diseases of pepper have not yet reached worldwide epidemic proportions but given the increasing importance of begomoviruses in tomato, it is probably only a matter of time. Therefore, management strategies developed for tomato production may be considered including the use of insecticides, oils and/or soaps to reduce whitefly transmission of viruses and rouging of infected plants. Establishment of whitefly-free periods may also be warranted in the future. Resistant pepper cultivars are not available (Polston and Anderson 1997).

4.6 *Curtovirus* genus

Beet curly top virus (BCTV) is an important viral pathogen of pepper worldwide, especially in irrigated arid and semi-arid regions like western North America and the eastern Mediterranean basin. Crops such as sugar beet, tomato, bean, cucurbits and crucifers are susceptible to BCTV, as are common weeds such as Russian thistle (*Salsola australis*).

4.6.1 Symptoms

BCTV was named for the symptoms induced upon infection. In pepper, leaves generally roll upwards or curl from the margins while the petioles curve downward. Plants infected early in the season are severely stunted and chlorotic. Infected plants produce only a few, misshapen fruit that generally ripen prematurely.

4.6.2 Causal Agent

BCTV is the type member of the *Curtovirus* genus in the family *Geminiviridae* of DNA viruses. Typical of the geminiviruses, BCTV forms geminate (twinned) particles of about 18×30 μm. The genome consists of one single-stranded DNA molecule, which is packaged within each geminate particle (Buck 1999, Thomas and Mink 1979). More than 300 plant species in 44 plant families are hosts for BCTV. Multiple strains of BCTV have been identified that vary in virulence, host range and symptoms induced.

4.6.3 Disease cycle

BCTV is transmitted by two leafhopper species, the beet leafhopper, *Circulifer tenellus* (in North America and the Mediterranean basin) and *C. opacipennis* (in the Mediterranean basin). Transmission by the leafhoppers occurs in a persistent although nonpropagative manner. The leafhopper vectors of BCTV prefer conditions of high light intensity, warm to hot temperatures, low humidity, and places on the plant leaves where evaporation is rapid. Leafhopper activity is reduced where plants are shaded. BCTV is phloem-limited and difficult to mechanically transmit. Virus reservoirs include weeds and other crops, especially sugar beets. Migration of leafhoppers has been extensively studied in the western USA (Kendrick *et al.,* 1951, Zitter 1991a,b).

4.6.4 Management

Removal of weed hosts prior to planting and isolation of pepper fields from other crop hosts, especially sugar beets, may reduce BCTV inoculum. Plantings may be timed to avoid the peak flights of leafhoppers. Once infected, plants will not be productive. Removal of initially infected plants may reduce secondary infections. However, since most damage occurs from primary infections, the value of rouging may be limited. Chemical control of leafhoppers is sometimes used but the weeds and other host plants must be sprayed prior to leafhopper migration into peppers. In small gardens, plants may be shaded during early growth since the leafhoppers are more active in sunny locations. Pepper cultivars resistant to BCTV are not currently available.

4.7 *Alfamovirus* genus

Alfalfa mosaic virus (AMV) has a wide host range but is generally only found in pepper when grown nearby alfalfa or other forage legumes. AMV also infects other crops including potato, tomato and bean. The virus occurs throughout the world. AMV is usually not an economically important pathogen of peppers.

4.7.1 Symptoms

Leaves of AMV-infected plants frequently show striking yellow or white blotches or mosaic. Chlorotic ring spots or patterns and severe leaf necrosis may also occur. Extensive early season infections may lead to stunted plants and distorted fruit with necrotic spots. The specific strain of AMV involved and environmental conditions affect symptom expression.

4.7.2 Causal Agent

AMV is the type member of the *Alfamovirus* genus of RNA viruses. AMV forms five particle types all of which have a diameter of 19 nm. The smallest particle type is icosahedral while the four larger types are bacilliform with lengths of 30, 35, 43 and 56 nm. The genome consists of three positive-sense, single-stranded RNAs which are

independently packaged in the three largest particle types. A subgenomic messenger RNA for the coat protein is packaged in the two smallest particle types. The coat protein or its messenger RNA are required along with the three genomic RNAs to initiate infection. More than 150 plant species in at least 22 plant families are naturally infected by AMV. The Fabaceae family contains a large number of susceptible species (Bol 1999, Jaspers and Bol 1980).

4.7.3 Disease cycle

AMV is principally transmitted from plant to plant by any of at least 15 species of aphids in a non-persistent manner (Zitter 1991a,b). Common vectors include the pea aphid (*Acyrthosiphon pisum*), the blue alfalfa aphid (*A. kondoi*) and the green peach aphid (*Myzus periscae*). Although not always symptomatic, fields of AMV-infected alfalfa and other forage legumes growing adjacent to pepper plantings provide a reservoir of the virus.

4.7.4 Management

Distancing pepper plantings from alfalfa and other forage legumes is the best way to avoid AMV infection. Plant debris from infected crops should be removed or buried at the end of the season to reduce potential inoculum for the following season. Barriers (*e.g.* a 50-foot strip of a non-susceptible crop such as corn) can be planted around pepper fields to trap flying aphids until they become non-infective. The barrier crop can be sprayed with a suitable insecticide at least weekly to reduce the population of insect vectors. Oil applied to pepper interferes with the acquisition and/or transmission of a virus by the aphid in the field. Resistance is not available.

5. Diseases caused by nematodes

Nematodes, also known as roundworms or eelworms, are classified in the animal phylum Nematoda. Although wormlike in appearance, nematodes are quite distinct taxonomically from true worms. Nematodes are ubiquitous in fresh and salt water and soil and live by feeding on microorganisms, plants and animals, and are important in decomposition and recycling of nutrients. Parasitic nematodes attack animals, humans and several hundred species feed on plants. Plant pathogenic nematodes are distinguished by presence of a stylet (a needle-like mouth part) used to obtain food.

Several species of nematodes cause disease on peppers. Root-knot (*Meloidogyne* spp.) and sting (*Belonolaimus* spp.) are two of the most important and others such as stubby (*Trichodorus* spp.), lesion (*Pratylenchus* spp.) and others are considered less important (Netscher and Sikora 1990, Potter and Olthof 1993).

Symptoms of damage by nematodes appear on aerial plant parts and plant roots Symptoms on above ground plant parts are typical of root-damaged such as stunting, yellowing, symptoms of nutrient deficiencies, wilting and reduced yields. Symptoms on roots are root knots or galls, lesions, excessive branching, injured roots and secondary infections by other microorganisms causing root rots.

Life cycles of most nematodes are quite similar and the root-knot nematode is presented as a typical example. Root-knot disease is most frequently caused by *Meloidogyne incognita* although others species may also cause damage. The life cycle is complete in 25 days at 27°C and involves four juvenile stages. The first stage begins in the egg. The second stage juvenile hatches from the egg, moves in the soil, and this is the stage that infects the plant. After entering the plant, the nematode becomes sedentary and feeds on cells with its stylets. The secreted saliva stimulates the cell enlargement. The nematode molts rapidly to the third and fourth stage and finally the pear-shaped adult female or males. The female lays eggs in a gelatinous mass either inside or outside of the root (Agrios 1997).

A number of control methods may be useful in managing nematodes on peppers. All materials in transplant productions should be free from nematodes. Sterilization of soil by fumigation or solarization can be used to reduce populations of nematodes. Granular nematicides may be used for the same purpose. Crop rotation with non-hosts, particularly grasses, is recommended, although the nematode present and its host range must be known prior to selecting the alternating crop. Weeds and volunteers in the fields must be eradicated or populations will persist on these hosts. Resistant cultivars are available for some races of root-knot nematodes, but not generally for other types of nematodes, and should be used when available.

6. Abiotic disorders

6.1 Blossom end rot

Blossom-end rot is a common physiological disorder believed to be associated with calcium deficiency and fluctuations in soil moisture. The injury commonly occurs on the blossom end of the pepper fruit. Tomato, eggplant and watermelon fruits are also susceptible to the disorder. Losses of up to 50% can occur in some seasons.

6.1.1 Symptoms

Blossom end rot damage is most frequent around the blossom end of the fruit, however it can occur on the sides of the pepper fruit, but usually near the blossom end. The first symptom appears as a small, watersoaked area that becomes larger and quickly expands as the fruit matures. The affected tissue turns black, leathery and shrinks until the lesion is flat or concave. The flesh is frequently colonized and decayed by secondary fungi. In severe cases, it may completely cover the lower half of the fruit. Peppers exhibit the problem when about one-third to one-half mature although damage can occur during any stage of fruit development.

6.1.2 Causal agent

Blossom-end rot is a physiologic disorder associated with a low concentration of calcium in the fruit. Growing cells normally require high levels of calcium. If the fruit cells have an inadequate supply of calcium, the tissue breaks down resulting in blossom end

rot lesions (Snowdon 1992). Calcium deficiency in fruit may be caused for a number of reasons. These reasons include too low levels of calcium in the soil, drought stress, high amounts of competitive cations in the soil, excessive soil moisture fluctuations that reduces plant uptake of calcium and its movement in the plant, or excessive nitrogen fertilization leading to lush vegetative growth.

6.1.3 Management

Adequate calcium should be supplied through liming and fertilization. Irrigation should be monitored to maintain moisture of the soil as uniformly as possible. Black plastic and organic mulches can help to stabilize soil moisture. Nitrate nitrogen is the preferred nitrogen source in fertilizer because ammoniacal nitrogen supplies ammonium ions that reduce calcium uptake by plants. Excess fertilization as side dressings during early fruiting should be avoided, especially with ammoniacal forms of nitrogen. The soil pH should be maintained around 6.5. Liming will supply calcium as well as increase the ratio of calcium ions to other competitive ions in the soil. Foliar applications of calcium, which are often advocated, are of little value because of poor absorption and movement to fruit where it is needed.

6.2 Chilling injury

Chilling injury occurs on pepper fruit stored at above freezing temperatures to 5°C for 14 days or longer. Injury on fruit appears as sheet pitting where small pits occur over a large area on fruit and as scald which seen as surface discoloration of fruit to grayish-brown (Snowdon 1992). The calyx area deteriorates. Secondary invasion of other microorganisms can cause rotted tissue. Damage can be avoided by storage of peppers at temperatures above 7.5°C with adequate humidity for periods of less than 28 days.

6.3 Sunscald

Sunscald of peppers occurs on parts of the fruit that are directly exposed to sunlight. Fruits are typically exposed after defoliation or wilting of foliage due to other disease such as bacterial leaf spot or powdery mildew. The symptoms appear initially as a light-colored, soft, and slightly wrinkled area on the fruit. As the injured tissue dries, it becomes sunken and has a white, paper-like appearance. In many cases, secondary microorganisms invade and enter through these areas. Damage can be avoided by growing cultivars that shade fruit well and controlling foliar disease such as powdery mildew and bacterial spot that cause defoliation.

7. References

Abawi, G. S. and Grogas, R. G. 1979. Epidemiology of diseases caused by *Sclerotinia* species. Phytopathology, 69:899-904.

Adkins, S. 2000. Tomato spotted wilt virus-positive steps towards negative success. Molec. Plant Pathol., 1:151-157.

Agrios, G. N. 1997. Plant Pathology, 4th ed. Academic Press, San Diego. 635 p.

Anderson, C.W. 1958. A study of field sources and spread of five viruses of peppers in central Florida. Phytopathology, 49:97-101.

Armstrong, G.M. and Alberts, W.B. 1933. Downy mildew of tobacco on pepper, tomato, and eggplant. Phytopathology, 23:837-839.

Ayesu-Offei, E.N. 1998. Formae speciales of *Leveillula taurica* (Lev.) Arn. infecting peppers and eggplants in Ghana. Trop. Agric., 66: 355-360.

Barksdale, T.H., Papvizas, G.C. and Johnston, S.A. 1984. Resistance to foliar blight and crown rot of pepper caused by *Phytophthora capsici*. Plant Dis., 68:506-509.

Bartz, J.A. and Stall, W.M. 1974. Tolerance of fruit from different pepper lines to *Erwinia carotovora*. Phytopathology, 64: 1290-1293.

Benner, C.P., Kuhn, C.W., Demski, J.W., Doboson, J.W., Colditz, P. and Nutter, F.W., Jr. 1985. Identification and incidence of pepper viruses in northeastern Georgia. Plant Dis., 69:999-1001.

Benson, D.M. and Ashworth, Jr., L.J. 1976. Survival of *Verticillium albo-atrum* on nonsuscept roots and residues in field soil Phytopathology, 66:883-887.

Beute, M.K. and Rodriguez-Kabana, R. 1981. Effects of soil moisture, temperature, and field environment on survival of *Sclerotium rolfsii* in Alabama and North Carolina. Phytopathology, 71:1293-1296.

Bhat, R.G., and Subbarao, K.V. 1999. Host range specificity in *Verticillium dahliae*. Phytopathology, 89:1218-1225.

Black, L.L. 2003. Fusarium Wilt. In: "Compendium of Pepper Diseases". American Phytopathological Society Press, St. Paul, MN, pp. 14-15

Blazquez, C.H. 1969. Occurrence of gray leafspot on peppers in Florida. Plant Dis. Report, 53:756.

Blazquez, C.H. 1971. Gray leafspot of pepper. Proc. Fl. State Hort. Soc., 84:171-177.

Bol, J.F. 1999. Alfamovirus and Ilarviruses (Bromoviridae). In: "Encyclopedia of Virology" (eds. Granoff, A. and Webster, R.G.). San Diego: Academic Press. Pages 38-43.

Bonas, U., Stall, R. E. and Staskawicz, B. 1989. Genetic and structural characterization of the avirulence gene *avrBs3* from *Xanthomonas campestris* pv. *vesicatoria*. Mol. Gen. Genet., 218:127-136.

Bouzar H., Jones, J. B., Stall, R. E., Hodge, N. C., Minsavage, G. V. ,Benedict A. A. and Alvarez, A.M. 1994. Physiological, chemical, serological, and pathogenic analyses of a worldwide collection of *Xanthomonas campestris* pv. *vesicatoria* strains. Phytopathology, 84: 663-671.

Bowers, J.H. and Mitchell, D.J. 1990. Effect of soil-water matric potential and periodic flooding on mortality of pepper caused by *Phytophthora capsici*. Phytopathology, 80: 1447-1450.

Bowers, J.H., Papavizas, G.C. and Johnston, S.A. 1990a. Effect of soil temperature and soil-water matric potential on the survival of *Phytophthora capsici* in natural soil. Plant Dis., 74: 771-777.

Bowers, J.H., Sonoda, R.M. and Mitchell, D.J. 1990b. Path coefficient analysis of the effect of rainfall variables on the epidemiology of *Phytophthora* blight of pepper caused by *Phytophthora capsici*. Phytopathology, 80: 1439-1446.

Braverman, S.W. 1968. A new leaf spot of pepper incited by *S. botryosum* f. sp. *capsicum*. Phytopathology, 58:1164-1167.

Brown, J.E., Stevens, C., Osborn, M.C. and Bryce, H.M. 1989. Black plastic mulch and spunbonded polyester row cover as a method of southern blight control in bell pepper. Plant Dis., 73:931-932.

Brown, J.K., Idris, A.M. and Stenger, D.C. 1993. Sinaloa tomato leaf curl virus, a newly described geminivirus of tomato and pepper in west coastal Mexico. Plant Dis., 77:1262.

Bruehl, G.W. 1987. Soilborne plant pathogens. Macmillan Publishing Company, 866 Third Avenue, New York, USA.

Buck, K.W. 1999. Geminiviruses (Geminiviridae). In: "Encyclopedia of Virology" (eds.Granoff, A. and Webster, R.G.). San Diego: Academic Press. pp. 597-606.

Buddenhagen, I.W., Sequeira, L. and Kelman, A. 1962. Designation of races of *Pseudomonas solanacearum.* Phytopathology, 52:726.

Carballo, S.J., Blankenship, S.M. and Sanders, D.C. 1994. Drip fertigation with nitrogen and potassium and postharvest susceptibility to bacterial soft rot of bell peppers. J. of Plant Nutrition, 17: 1175-1191.

Ceponis, M.J., Cappellini, R.A. and Lightner, G.W. 1987. Disorders in fresh pepper shipments to the New York market, 1972-1985. Plant Dis., 71: 380-382.

Chagas, C.M., Vicente, M., Alba, A.P.C. and July, J.R. 1977. *Solanum atropurpureum* Schrank, a natural reservoir of potato virus Y (PVY). Phytopathol. Z ,90:147-151.

Chellemi, D.O., Mitchell, D.J., Kannwischer-Mitchell, M.E., Rayside, P.A. and Rosskopf, R.N. 2000. *Pythium* spp. associated with bell pepper production in Florida. Plant Dis., 84: 1271-1274

Coplin, D.L. 1980. *Erwinia carotovora* var. *carotovora* on bell peppers in Ohio. Plant Dis., 64: 191-194.

Cook, A. A. and Stall, R.E. 1963. Inheritance of resistance in pepper to bacterial spot. Phytopathology, 53:1060-1062.

Cook, A. A. and Guevara, Y.G. 1984. Hypersensitivity in *Capsicum chacoense* to race 1 of the bacterial spot pathogen of pepper. Plant Dis., 68:329-330.

Cook, D., Barlow, E. and Sequiera, L. 1989. Genetic diversity of *Pseudomonas solanacearum*: Detection of Restriction fragment length polymorphisms with DNA probes that specify virulence and the hypersensitive response. Molecular Plant-Microbe Interactions., 2:113-121.

Correl, J.C., Gordon, T.R. and Elliot, V.J. 1987. Host range, specificity, and biometrical measurements of *Leveillula taurica* in California. Plant Dis., 71: 248-251.

Dahlbeck, D. and Stall, R.E. 1979. Mutations for change of race in cultures of *Xanthomonas vesicatoria*. Phytopathology, 69:634-636.

De Bokx, J.A. and Huttinga, H. 1981. Potato virus Y. CMI/AAB Descriptions of Plant Viruses, No. 242.

Dougherty, D. E. 1979. Bud rot of pepper. Proc. Fla. State Hortic. Soc., 92:103-106.

Elad, Y. 2000. *Trichoderma harzianum* T39 preparation for biocontrol of plant diseases- Control of *Botrytis cinerea, Sclerotinia sclerotiorum* and *Cladosporium fulvum*. Biocontrol Sci. and Tech., 10:499-506.

Elad, Y., Katan, J., and Chet, I. 1980. Physical, biological, and chemical control integrated for soilborne diseases in potatoes. Phytopathology, 70:418-422.

Elad, Y., Yunis, H. and Volpin, H. 1993. Effect of nutrition on susceptibility of cucumber, eggplant, and pepper crops to *Botrytis cinerea*. Can. J. Bot. 71: 602-608.

Ellis, M.B. 1971. Dematiaceous hyphomycetes. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 608 p.

Ellis, M.B. and Waller, J.M. 1974 *Scerotinia fuckeliana* (conidial state: *Botrytis cinerea*). CMI Descriptions of Pathogenic Fungi and Bacteria. No. 431. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 p.

Ellis, M.B. and Gibson, I.A.S. 1975. *Stemphylium solani*. CMI Descriptions of Pathogenic Fungi and Bacteria. No. 472. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 pp

Fallik, E., Aharoni, Y., Grinber, S., Copel, A. and Klein, J.D. 1994. Postharvest hydrogen peroxide treatment inhibits decay in eggplant and sweet red pepper. Crop Protect., 13:451-454.

Faillik, E., Grinberg, S., Alkalai, S. and Lurie, S. 1996. The effectiveness of postharvest hot water dipping on the control of grey and black moulds in sweet red pepper (*Capsicum anuum*). Plant Pathology., 45: 644-649.

Fallik, E., Ziv, O., Grinberg, S., Alkalai, S. and Klein, J.D. 1997. Bicarbonate solutions control powdery mildew (*Leveillula taurica*) on sweet red pepper and reduce the development of postharvest fruit rotting. Phytoparasitica, 25:41-43.

Francki, R.I.B., Mossop D.W. and Hatta T. 1979. Cucumber mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 213.

Gardner, M. W. and Kendrick, J.B. 1923. Bacterial spot of tomato and pepper. Phytopathology, 13:307-315.

Gayed, S.K. 1984. The response of pepper and tomato cultivars to challenge inoculation by *Peronospora tabacina*. Can. J. Plant Sci., 64:225-228.

German, T.L., Ullman, D.E. and Moyer, J.W. 1992. Tospoviruses: diagnosis, molecular biology, phylogeny, and vector relationships. Annu. Rev. Phytopathol., 30:315-348.

Gibbs, A.J. 1977. Tobamovirus group. CMI/AAB Descriptions of Plant Viruses, No. 184.

Goldbach, R. and Peters, D. 1994. Posssible causes of the emergence of tospovirus diseases. Sem. Virol., 5:113-120.

Goto, M. 1990. Fundamentals of bacterial plant pathology. Academic Press, Inc. 1250 Sixth Avenue, San Diego, CA, USA.

Gurkin, R.S. and Jenkins, S.F. 1985. Influence of cultural practices, fungicides, and inoculum placement on southern blight and Rhizoctonia crown rot of carrot. Plant Dis., 69:477-481.

Hadas, R., Kritzman, G. and Manulis, S. 2001. Detection, quantification and characterization of *Erwinia carotovora* ssp. *carotovora* contaminating pepper seeds. Plant Path., 50: 117-123.

Hadden, J. H. 1989. The etiology and control of pepper anthracnose. Ph.D. dissertation. Louisiana State University, Baton Rouge. 77 pp.

Halfon-Meiri, A., and Rylski, I. 1983. Internal mold caused in sweet pepper by *Alternaria alternata*: Fungal ingress. Phytopathology, 73:67-70.

Hayward, A.C. 1991. Biology and epidemiology of bacterial wilt caused by *Pseudomonas solanacearum*. Ann. Rev. Phytopathology, 29:65-87.

Hawksworth, D.L. and Talboys, P.W. 1970a. *Verticillium albo-atrum.* CMI Descriptions of Pathogenic Fungi and Bacteria. No. 255. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 pp.

Hawksworth, D.L. and Talboys, P.W. 1970b. *Verticillium dahliae.* CMI Descriptions of Pathogenic Fungi and Bacteria. No. 256. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 pp.

He, L.Y., Sequeira, L, and Kelman, A. 1983. Characteristics of strains of *Pseudomonas solanacearum* from China. Plant Dis., 67:1357-1361.

Higgins, B. B. 1922. The bacterial spot of pepper. Phytopathology, 12:501-516.

Hindi, E., Dishon, I. and Nevo, D. 1965. Observations on tobacco blue mold in Israel. Plant Dis. Rep., 49:154-156.

Hollings, M. and Huttinga, H. 1976. Tomato mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 156.

Hord, M.J. and Ristaino, J.B. 1991. Effects of physical and chemical factors on the germination of oospores of *Phytophthora capsici* . Phytopathology, 81:1541-1546.

Idris, A.M. and Brown, J.K. 1998. Sinaloa tomato leaf curl geminivirus: biological and molecular

evidence for a new subgroup III virus. Phytopathology, 88:648-657.

Idris, A.M., Lee, S.H. and Brown, J.K. 1999a. First report of Chino del tomate and pepper huasteco geminiviruses in greenhouse-grown tomato in Sonora, Mexico. Plant Dis., 83:396.

Idris, A.M., Rivas-Platero, G., Torres-Jerez, I. and Brown, J.K. 1999b. First report of Sinaloa tomato leaf curl geminivirus in Costa Rica. Plant Dis., 83:303.

Jaspers, E.M.J. and Bol, L. 1980. Alfalfa mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 229.

Jenkins, S.F. and Averre, C.W. 1986. Problems and progress in integrated control of southern blight of vegetables. Plant Dis., 70:614-619.

Jones, M.M. 1992. *Fusarium* wilt of pepper: Response of *Capsicum* spp. accessions to *Fusarium oxysporum* f. sp. *capsici* and analysis of vegetative combatility. M.S. thesis, Louisiana State University, Baton Rouge. 69 p.

Kendrick, J.B., Anderson, L.D. and Dickson, R.C. 1951. Source and seasonal spread of certain viruses in peppers in southern California. Phytopathology, 41:20.

Keshwal, R.L. and Choubay, P.C. 1983. Studies on control of powdery mildew of chillies. Pesticides, 17:30,32.

Kim, B. S. and Hartmann, R.W. 1985. Inheritance of a gene (Bs3) conferring hypersensitive resistance to *Xanthomonas campestris* pv. *vesicatoria* in pepper (*Capsicum annuum*). Plant Dis., 69:233-235.

Kim, K.D., Oh, B.J. and Yang, J. 1999. Differential interactions of a *Colletotrichum gloeosporioides* isolate with green and red pepper fruits. Phytoparasitica, 27:2.

Kimble, K.A. and Grogran, R.G. 1960. Resistance to *Phytophthora* root rot in pepper. Plant Dis., Rep. 44:872-873.

Kirk, P.M. 1982. *Cercospora capsici.* CMI Descriptions of Pathogenic Fungi and Bacteria. No. 723. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 pp.

Kousik, C.S. and Ritchie, D.F. 1996. Race shift in *Xanthomonas campestris* pv. *vesicatoria* within a season in field-grown pepper. Phytopathology, 86:952-958.

Leonian, L.H. 1922. Stem and fruit blight of pepper caused by *Phytophthora capsici* species nov. Phytopathology, 12:401-408.

Lewandowski, D.J. and Dawson, W.O. 1999. Tobamoviruses. In: "Encyclopedia of Virology" (eds.Granoff, A. and Webster, R.G.,). San Diego: Academic Press. Pages 1780-1783.

Lewis, J.A., Larkin, R.P. and Rogers, D.L. 1998. A formulation of *Trichoderma* and *Gliocladium* to reduce damping-off caused by *Rhizoctonia solani* and saprophytic growth of the pathogen in soilless mix. Plant Dis,. 82:501-506.

Lopez-Moya, J.J. and Garcia, J.A. 1999. Potyviruses (Potyviridae). In: "Encyclopedia of Virology" (eds.Granoff, A. and Webster, R.G.). San Diego: Academic Press. pp. 1369-1375.

Lotrakul, P., Valverde, R.A., De la Torre, R. and Sim, J. 2000. Occurrence of a strain of Texas pepper virus in Tabasco and Habanero pepper in Costa Rica. Plant Dis., 84:168-172.

Manandhar, J.B., Hartman, G.L. and Wang, T.C. 1995a. Semiselective medium for *Colletotrichum gloeosporioides* and occurrence of three *Colletotrichum* spp. on pepper plants. Plant Dis., 79:376-379.

Manandhar, J.B., Hartman, G.L. and Wang, T.C. 1995b. Anthracnose development on pepper fruits inoculated with *Colletotrichum gloeosporioides*. Plant Dis., 79:380-383.

Marco, G.M. and Stall, R.E. 1983. Control of bacterial spot of pepper initiated by strains of *Xanthomonas campestris* pv. *vesicatoria* that differ in sensitivity to copper. Plant Dis., 67:779-781.

McInnes, T. B., Gitaitis, R.D., McCarter, S.M., Jaworski, C.A. and Phatak, S.C. 1988. Airborne dispersal of bacteria in tomato and pepper transplant fields. Plant Dis., 72:575-579.

Melchers, L.E. and Dale, E. E. 1917. Black spot of pepper. Phytopathology, 7:63.

Mercier, J., Baka, M., Reddy, B., Corcuff, R. and Arul, J. 2001. Shortwave ultraviolet irradiation

for control of decay caused by *Botrytis cinerea* in bell pepper: Induced resistance and germicidal effects. J. Amer. Soc. Hort. Sci., 126:128-133.

Mihail, J.D. and Alcorn, S.M. 1984. Effects of soil solarization on *Macrophomina phaseolina* and *Sclerotium rolfsii*. Plant Dis., 68:156-159.

Minsavage, G. V., Dahlbeck, D., Whalen, M.C., Kearney, B., Bonas, U., Staskawicz, B. and Stall, R.E. 1990. Gene-for-gene relationships specifying disease resistance in *Xanthomonas campestris* pv. *vesicatoria*-pepper interactions. Mol. Plant-Microbe Interact., 3:41-47.

Montasser, M.S., Tousignant, M.E. and Kaper, J.M. 1998. Viral satellite RNAs for the prevention of cucumber mosiac virus (CMV) disease in field-grown pepper and melon plants. Plant Dis., 82:1298-1303.

Moore, W. D. 1949. Flooding as a means of destroying the sclerotia of *Sclerotinia sclerotiorum*. Phytopathology, 39:930-927.

Mordue, J.E.M. 1967. *Colletotrichum coccodes*. CMI Descriptions of Pathogenic Fungi and Bacteria. No. 131. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 p.

Mordue, J.E.M. 1971a. *Colletotrichum capsici*. CMI Descriptions of Pathogenic Fungi and Bacteria. No. 317. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 p

Mordue, J.E.M. 1971b. *Glomerella cingulata*. CMI Descriptions of Pathogenic Fungi and Bacteria. No. 315. Commonwealth Mycological Institute, Ferry Lane, Kew, Surrey. England. 2 pp

Mordue, J.E.M. 1974. *Corticium rolfsii*. CMI Descriptions of Pathogenic Fungi and Bacteria. No. 410. Commonwealth Mycological Institute, Surrey, England.

Nelson, M.R., Wheeler, R.E. and Zitter, T.A. 1982. Pepper mottle virus. CMI/AAB Descriptions of Plant Viruses, No. 253.

Netscher, C. and Sikora, R. A. 1990. Nematode parasites of vegetables. In: "Plant Parasitic Nematodes in Subtropical and Tropical Agriculture" (eds. Luc, M. Sikora, R. A. and Bridge, J.) CAB International, Wallingford, UK.

Ortega, R.G., Espanol, C.P. and Zueco, J.C. 1992. Genetic relationships among four pepper genotypes resistant to *Phytophthora capsici*. Plant Breed., 108: 118-125.

Pategas, K.G., Schuerger, A.C. and Wetter, C. 1989. Management of tomato mosaic virus in hydroponically grown pepper (*Capsicum annuum*). Plant Dis., 73:570-573.

Peeples, J.L., Curl, E.A. and Rodriguez-Kabana, R. 1976. Effect of the herbicide EPTC on the biocontrol activity of *Trichoderma viride* against *Sclerotium rolfsii*. Plant Dis. Report, 60:1050-1054.

Pernezny, K. and Purdy, L.H. 1981. Sclerotinia diseases of vegetable and field crops in Florida. Univ. Fla. Plant Path. Rep. No. pp-22.

Peterson, G. H. 1963. Survival of *Xanthomonas vesicatoria* in soil and diseased tomato plants. Phytopathology, 53:765-767.

Ploetz, R.C. and Haynes, J.L. 2000. How does *Phytophthora capsici* survive in squash fields in southeastern Florida during the off-season? Proc. Fla. State Hort. Soc., 113:211-215.

Pohronezny, K., Stall, R. E., Canteros, B. I., Kegley, M., Datnoff, L. E. and Subramanya, R.. 1992. Sudden shift in the prevalent race of *Xanthomonas campestris* pv. *vesicatoria* in pepper fields in southern Florida. Plant Dis., 76:118-120.

Polizzi, G., Agosteo, G.E. and Cartia, G. 1994. Soil solarization for the control of *Phytophthora capsici* on pepper. Acta Horticulturae, 336: 330-335.

Polston, J.E. and Anderson, P.K. 1997. The emergence of whitefly-transmitted geminiviruses in tomato in the Western Hemisphere. Plant Dis., 81:1358-1369.

Potter, J. W. and Olthof, T. H. A. 1993. Nematode pests of vegetable crops In*: Plant Parasitic Nematodes in Temperate Agriculture.* (eds. K. Evans, D. L. Trudgill, and J. M. Webster).

CAB International, Wallingford, UK.

Punja, Z.K. 1985. The biology, ecology, and control of *Sclerotium rolfsii*. Ann. Rev. Phytopathol., 23:97-127.

Purcifull, D.E. and Hiebert, E. 1982. Tobacco etch virus. CMI/AAB Descriptions of Plant Viruses, No. 258.

Purdy, L. H. 1979. *Sclerotinia sclerotiorum*. History, diseases and symptomatology, host range, geographic distribution, and impact. Phytopathology, 69:875-880.

Poussier, S., Vandewalle, P. and Luisetti, J. 1999. Genetic diversity of African and worldwide strains of *Ralstonia solanacearum* as determined by PCR-Restriction fragment length polymorphism analysis of the *hrp* gene region. Appl. Environ. Microbiol. 65:2184-2194.

Reuveni, R., Perl, M. and Rotem, J. 1976. Inhibition of shedding of pepper leaves infected with powdery mildew (*Leveillula taurica*) by application of auxins. Phytoparasitica, 4: 197-199.

Reuveni, R., Dor, G. and Reuveni, M. 1998. Local and systemic control of powdery mildew (*Leveillula taurica*) on pepper plants by foliar spray of mono-potassium phosphate. Crop Protect., 17:703-709.

Ristaino, J.B. 1990. Intraspecific variation among isolates of *Phytophthora capsici* from pepper and squash fields in North Carolina. Phytopathology, 80:1253-1259.

Ristaino, J.B. 1991. Influence of rainfall, drip irrigation, and inoculum density on the development of *Phytophthora* root and crown rot epidemics and yield in bell pepper. Phytopathology, 81: 922-929.

Ristaino, J.B., Larkin, R.P. and Campbell, C.L. 1993. Spatial and temporal dynamics of *Phytophthora* epidemics in commercial bell pepper fields. Phytopathology, 83: 1312-1320.

Ristaino, J.B., Parra, G. and Campbell, C.L. 1997. Supression of *Phytophthora* blight in bell pepper by a no-till wheat cover crop. Phytopathology, 87: 242-249.

Rivelli, V. 1989. A wilt of pepper incited by *Fusarium oxysporum* f.sp. *capsici* forma specialis nova. M.S. thesis, Louisiana State University, Baton Ronge. 72 p.

Rojas, A., Kvarnheden, A. and Valkonen, P.T. 2000. Geminiviruses infecting tomato crops in Nicaragua. Plant Dis., 84:843-846.

Roossinck, M.J. 1999. Cucumoviruses (Bromoviridae). In: "Encyclopedia of Virology" (eds.Granoff, A. and Webster, R.G.,). San Diego: Academic Press. pp. 315-324.

Rybicki, E.P., Briddon, R.W., Brown, J.K., Fauquet, C.M., Maxwell, D.P., Harrison, B.D., Markham, P.G., Bisaro, D.M., Robinson, D. and Stanley, J. 2000. Geminiviridae. In: "Virus Taxonomy: Classification and Nomenclature of Viruses, Seventh Report of the International Committee on Taxonomy of Viruses" (eds. van Regenmortel, M.H.V, Fauquet, C.M., and Bishop, D.H.L.). San Diego: Academic Press. pp. 285-297.

Sanchez, S. and Grogan, R.G. 1970. Potato Virus Y. CMI/AAB Descriptions of Plant Viruses, No. 37.

Satour, M.M. and Butler, E.E. 1967. A root and crown rot of tomato caused by *Phytophthora capsici* and P. *parasitica*. Phytopathology, 57: 510-515.

Schuerger, A.C. and Batzer, J.C. 1993. Identification and host range of an erwinia pathogen causing stem rots on hydroponically grown plants. Plant Dis., 77: 472-477

Sherbakoff, C. D. 1918. Report of the associate plant pathologist. Fla. Agr. Exp. Sta. Rpt., 1916-1917: 66R-86R.

Sherf, A.F. and McNab, A.A. 1986. Vegetable diseases and their control. 2nd Edit. John Wiley & Sons. 728 p.

Sherwood, J.L., German, T.L., Whitfield, A.E., Moyer, J.W. and Ullman, D.E. 2000. Tomato spotted wilt. In: "Encyclopedia of Plant Pathology" (eds. Maloy, O.C. and Murray, T.D.,). New York: John Wiley and Sons. pp. 1030-1031.

Sinclair, J.B., Horn, N.L. and Tims, E.C. 1958. Unusual occurrence of certain diseases in Louisiana. Plant Dis. Report, 42:984-985.

Sinha, S. 1940. On the characteristics of *Choanephora cucurbitarum* Thaxter on chillies (*Capsicum* spp.) Ind. Acad. Sci., Sec. B. (Proceedings), 11:162-166.

Smith, C. and Bartz, J.A. 1990. Variation in the pathogenicity and aggressiveness of strains of *Erwinia carotovora* subsp. *carotovora* isolated from different hosts. Plant Dis., 74: 505-509.

Smith, R.W. and Crossan, D.F. 1958. The taxonomy, etiology and control of *Collectotrichum piperatum* (E&E) and *Collectotrichum capsici* (Syd.) B&B. Plant Dis. Rep., 42: 1099-1103.

Snowdon, A.L. 1992. Color atlas of post-harvest diseases and disorders of fruits and vegetables. Vol. 2 : Vegetables. BPCC Hazell Books, Aylesbury, England. 416 p.

Stall, R. E., Loschke, D. D. and Jones, J. B. 1986. Linkage of copper resistance and avirulence loci on a self-transmissible plasmid in *Xanthomonas campestris* pv. vesicatoria. Phytopathology, 76:240-243.

Stenger, D.C., Duffus, J.E. and Villalon, B. 1990. Biological and genomic properties of a geminivirus isolated from pepper. Phytopathology, 80:704-709.

Strommel, J.R., Goth, R.W., Haynes, K. G. and Kim, S.H. 1996. Pepper (*Capsicum annuum*) soft rot caused by *Erwinia carotovora* subsp. *atroseptica*. Plant Dis., 80:1109-1112.

Thayer, P. L. and Stall, R. E.. 1961. A survey of *Xanthomonas vesicatoria* resistance to streptomycin. Proc. Fla. State Hort. Soc.,75:163-165.

Thomas, P.E. and Mink, G.I. 1979. Beet curly top virus. CMI/AAB Descriptions of Plant Viruses, No. 210.

Ullasa, B.A., Rawal, R.D., Sohi, H.S., Singh, D.P. and Joshi, M.C. 1981. Reaction of sweet pepper genotypes to anthracnose, *Cercospora* leaf spot, and powdery mildew. Plant Dis., 65:600-601.

van Elsas, J.D., Kastelein, P., van Bekkum, P., van der Wolf, J.M., de Vries, P.M. and van Overbeek, L.S. 2000. Survival of *Ralstonia solanacearum* biovar 2, the causative agent of potato brown rot, in field and microcosm soils in temperate climates. Phytopathology, 90:1358-1366.

Weber, G.F. 1931. Blight of carrots caused by *Sclerotium rolfsii*, with geographic distribution and host range of the fungus. Phytopathology, 21:1129-1140.

Wetter, C. and Conti, M. 1988. Pepper mild mottle virus. CMI/AAB Descriptions of Plant Viruses, No. 330.

Yucel, S. 1995. A study on soil solarization combined with fumigant application to control *Phytophthora* crown blight (*Phytopthora capsici* Leonian) on peppers in the East Medditerranean region of Turkey. Crop Protect., 14:653-655.

Zaitlin, M. and Israel, H.W. 1975. Tobacco mosaic virus. CMI/AAB Descriptions of Plant Viruses, No. 151.

Zitter, T.A. 1991a. Curly Top In: "Compendium of Tomato Diseases". (eds. Jones, J.P., Jones, J.B., Stall, R.E., and Zitter, T.A.) APS Press, St. Paul, MN. pp 36.

Zitter, T.A. 1991b. Alfalfa Mosaic Virus In: "Compendium of Tomato Diseases". (eds. Jones, J.P., Jones, J.B., Stall, R.E., and Zitter, T.A.) APS Press, St. Paul, MN. pp 34-35.

Potato Viruses and their Management

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Abstract : The potato is propagated vegetatively through seed tubers. Vector aphids are mainly responsible for spread of common viruses, viz. PVY, PLRV, PVA etc. Besides, the common viruses, early autumn and spring potato crops in the Indian plains are being threatened by potato stem necrosis tospovirus and apical leaf cure gemini virus. In warm subtropical areas due to enough vector (aphids/thrips/white flies) population/activity, rapid degeneration of the seed stocks due to viruses and allied pathogens necessitates frequent replacement of seed for economical yields. Therefore, knowledge about the potato viruses, their detection/elimination and methods of indirect control to avoid their spread in nature *i.e.* raising healthy seed crops starting from nucleus seed by way of isolation, sanitation, adjustment of planting and harvesting dates, haulms cutting, crop rotations, roguing, certification etc. will be helpful for the growers/agencies. All possible details on the above aspects including integrated disease management schedule for viruses have been touched.

1. Introduction

The potato (a wholesome food) ranks fourth in production *i.e.* after wheat, rice and corn. As food crop potato promises to meet requirement of ever increasing world population on account of its higher dry matter and protein production per unit time and area as well as its versatility to adopt to a wide range of climate. Potato crop is raised through the 'seed' tubers which may carry a number of viruses from parent plant/crop. Losses due to the virus(es) and allied pathogens are not confined to the year of infection. They continue to increase progressively up to crop's saturation with one or more viruses *i.e.* every time the seed from an infected crop is used. 'Degeneration' of seed potatoes mainly due to viruses is marked by stunting and curling and overall chlorosis of plants or atleast the foliage. It is responsible for fewer and rather small sized tubers with reduction in yields by 50% or still higher. It was established in UK and other European countries during 18th century, even without knowing anything about the viruses that once the potato seed gets saturated with certain viruses (under certain conditions) it yields lesser hence needs replacement with healthy seed. It is now known to occur wherever potatoes are grown and is well established to be due to complex of a few or several of almost 40 viruses (Table 1) infecting potatoes (Nagaich *et al.*, 1974; Hooker, 1981; Khurana and Singh, 1986; Salazar, 1990). Viruses infecting potato can be placed into two distinct groups based on their dependence or/not on the potato (Table 1).

Table 1: Potato viruses grouped on the basis of their virion morphology and transmission mode

Virus (Group)	Acronym	Vector/Transmission mode
A. Isometric		
1. Andean Potato latent(Tymovirus)	APLV	Flea beetle /Pollen,TPS
2. Andean potato mottle (Comovirus)	APMV	Beetle / Contact
3. Arracacha-B(Oca strain) (Nepovirus)	AVB-O	Unknown / Pollen, TPS
4. Potato black ring[U] (Nepovirus)	PBRV	Nematode / Pollen,TPS
5. Tomato black ring[ND] (-do-)	TmBRV	Nematode / -do-
6. (Tobacco ringspot)[ND] (-do-)	TRSV	Nematode / -do-
7. Potato U (-do-)	PUV	Nematode / Contact
8. Cucumber mosaic[ND] (Cucumovirus)	CMV	Aphid
9. Sowbane mosaic[ND] (Sobemovirus)	SMV	(Leafhopper / (Pollen,TPS) leafminer) / Contact
10. Beet curly top[ND] (Geminivirus)	BCTV	Leafhopper/ -
11. Solanum apical leafcurl[ND] (-do-)	SALCV	
12. Potato deforming mosaic[U] (-do-)	PDMV	*Bemisia tabaci*
13. Potato yellow mosaic[ND] (-do-)	PYMV	*B. tabaci*
14. Tobacco necrosis (Necrovirus)	TNV	Fungus / (*Olpidium* sp) Mechanical
15. Tomato spotted wilt[ND] (Bunyavirus)	TSWV	Thrips / Mechanical
16. Tomato yellow mosaic[ND](Gemini)	TYMV	*B. tabaci*
17. Tobacco streak[ND] (Ilarvirus)	TSV	Thrips / Mechanical
B. Anisometric		
a) Filamentous		
18. Potato latent - (Carlavirus)	PotLV	Aphid/Contact
19. Potato M(-do-)	PVM	-do-
20. Potato S(-do-)	PVS	-do-
21. Potato P(-do-)	PVP	-do-
22. Potato rough dwarf (-do-)	PRDV	-do-
23. Potato T (Trichovirus)	PVT	Pollen,TPS/Contact
24. Potato V (Potyvirus)	PVV	Aphid[np]
25. Potato X (Potexvirus)	PVX	- / Contact
26. Potato Y (Potyvirus)	PVY	Aphid[NP] / Mechanical
27. Potato A (Potyvirus)	PVA	Aphid[NP] / Mechanical
28. Wild potato mosaic(Potyvirus)	WPMV	Aphid[NP]/ -
29. Potato aucuba mosaic(Potexvirus)	PAMV	Aphid[HC] / Contact
30. Wild aucuba mosaic (-do-)	WAMV	- do -
b). Fragile Rods		
31. Potato moptop (Furovirus)	PMTV	Fungus (*Spongospora* sp.)/ Mechanical
c). Rods		
32. Tobacco mosaic[ND] (Tobamovirus)	TMV	TPS (ext.)/Contact
33. Tobacco rattle[ND] (Tobravirus)	TRV	Nematode[P]/TPS/Mechanical
34. Tomato mosaic[ND] (Tobamovirus)	TmMV	Pollen, TPS(Ext.)/Contact
d). Bacilliform		
35. Alfalfa mosaic[ND] (Alfamovirus)	AMV	Aphid[NP]/Pollen, TPS

table 1 contd......

36. Potato yellowing[ND] (-do-)	PYV	Aphid[SP]/TPS
37. Potato yellow dwarf[ND](Rhabdovirus)	PYDV	Leafhopper[P]/Mechanical
38. Eggplant mottled dwarf[ND] (-do-)	EMDV	Aphid/Mechanical
C. Not characterized yet		
39. Potato yellow vein (Closterovirus)	PYVV	Glasshouse-whitefly[P]/-
40. Saq'O[U] (Luteovirus + Phytoplasma)	Saq'O	Aphid+leafhopper/tubers
D. Viroid		
41. Potato spindle tuber[U] (Viroid)	PSTVd	Pollen,TPS/Aphids[CI]/contact

TPS = Transmission through botanical (true) potato seed.; [*P/NP] = Persistently/ Non-persistently aphid transmitted.; SP =Semi-persistently aphid transmitted.; HC = Co-infections in the source host essential for vector acquisition, *i.e.* Helper component involved for transmission.; CI = Co-infection of PLRV essential for aphid transmission of viroid (Transencapsidation involved).; ND = Not dependent on potato for survival, and U-undetermined origin.

2. Economic importance

A lower incidence (say upto 5%) of the viruses, either singly or even upon combined infections, in the current season or coming from the previous season/crop, hardly reduces the yields (Reestman, 1970; Hooker, 1981; Khurana and Singh, 1988). But a higher virus incidence causes serious depressions (Tables 2 and 3) in the tuber yield (Hooker, 1981; Garg, 1987). The severe strains of potato virus Y (PVY) and leafroll (PLRV) may take a heavy toll *i.e.* upto 60-75% reduction in yield while the mild viruses like potato viruses X (PVX), S (PVS), A (PVA) and M (PVM) can depress the yields only up to 10-30% (Nagaich *et al.*, 1974; Khurana and Singh, 1988). But even losses due to PLRValso vary greatly according to the season, viz. only 7-15% in the autumn as against 40-60% in the spring crop (Khurana and Singh, 1988; Salazar, 1996). Even though potential losses by severe virus infections (even singly) can greatly reduce the tuber yields, the field data do not actually warrant that, mainly because of the limited incidence and compensation by neighbouring plants (Nagaich *et al.*, 1974; de Bokx and van der Want, 1987; Khurana, 1999; Salazar, 1996). Besides, potato spindle tuber viroid (PSTVd), known to be common in Europe and N. America, lowers the yields greatly (16-64%) depending on the viroid strain/potato variety and warm weather (Singh, 1983, 1988a). Yet another severe stem/leaf necrosis disease, caused by a tospovirus, heavily infects the early crop of potato in plains/plateaux of central/western India (Khurana *et al.*, 1989a). Similarly, a whitefly transmitted begomovirus, causing apical leafcurl and yellowing of plants, has been found seriously affecting the rabi crop in North Indian plains (Garg *et al.*, 2001).

2.1 Evaluation of yield losses

As mentioned earlier, yield losses caused by viruses, however, depend very much on a number of factors mainly the variety, growing conditionsetc. Thus it is not really easy

to estimate yield losses due to individual or combined virus infections. Naturally the methods to be used for the purpose need to take into consideration all possible factors that may affect such an estimation. Mere comparison of yields of only diseased plants *vis-a-vis* the same number of healthy plants alone gives the potential yield losses (Lal and Khurana, 1983; Khurana and Singh, 1988) and not actual estimates of depressions in tuber yield (Khurana, 1992, 1998). In fact, incidence of different viruses, infecting the plants singly or in combination, would vary and when the secondary infection is less than 10% in an otherwise healthy crop/plot, the losses will be rather negligible or small (Table 3). Mostly the secondary infections due to severe viruses and/or a high incidence of virus induce higher yield losses than late age primary infection. This is mainly because of the compensation occurring due to increased yields of surrounding healthy plants or even mildly diseased plants next to severely infected ones yield higher. Increased yield of such healthy plants can be compared to the effect of gaps or missing hills. Such compensation is pronounced in case of PLRV and even PVY infections but not that of PVX or PVS. However, primary infections at early stages of crop, particularly that of severe viruses causing foliage necrosis etc., can also result in heavy losses. Notwithstanding the above, generally 'uncared and bad seed' crops have per cent

Table 2: Yield loss potential of PVX and PVY in Indian potato cultivars* (from Khurana and Singh, 1988).

Cultivar	Percent reduction in yield due to					
	PVX			PVY		
	I	II	III yr	I	II	III yr
Kufri Chandramukhi	11	23	35	48	38	56
Kufri Jyoti	18	22	29	40	45	49
Kufri Sindhuri	17	24	36	36	40	61
Kufri Badshah	16	-	-	25	-	-
Kufri Bahar	10	-	-	23	-	-

*Average of 150 plants mechanically inoculated (under field). ; Seed with known infection carried on for 2nd/3rd year. ; - Not tried.

yield depressions equal to the total per cent incidence of the viruses.

The losses caused due to the potato virus(es) are not confined to the season/ year when the infection occurs but continue to affect the crop as long as the diseased seed tubers are used and only half to one-third yield (Table 3; de Bokx, 1972, Khurana, 1992). Generally tuber yield losses are 5-15% if all plants are secondarily infected with PVX and PVS; 15-30% for 100% secondary infection of PVY[n] and 40-70% due to PLRV (de Bokx, 1972; Lal and Khurana, 1983). It may be noted that these figures are only indicative of the range of losses often observed under summer conditions of the crop in the temperate zone (Khurana, 1992; Banttari *et al.*, 1993). Any adversity of climatic conditions, especially high temperatures and the drought stress, result in much higher losses.

3. Distribution of viruses

Majority of the potato viruses are widely, though uneven, distributed throughout the world (Hooker, 1981; de Bokx and van der Want, 1987; Jefferies, 1998). Naturally spread of the individual viruses varies from place to place depends on the climate, varietal pattern, vector population, seed source, etc. The important viruses causing mosaics in potatoes are usually PVY, PVA and PVM as well as severe strains of PVX, either singly and/or in different combinations (McDonald, 1986; Khurana and Garg, 1992; Khurana, 1999). Potato virus S being contagious is pandemic yet given the least importance on account of its being symptomless and causing only a little reduction in yield. PVA and PVM are not that common in India like in Europe. Similarly, PVY-N and PSTVd are not known in Indian potatoes. Potato mosaics and leafroll are important and occurring widely in almost all varieties (Nagaich *et al.*, 1974; Khurana, 1992). Incidence of PLRV and PSTVd is normally higher in warm-areas/season because of high vector activity for the former and higher multiplication of the viroid in the tropical weather. Under sub-tropical conditions in North Indian plains the seed can be maintained in good health for

Table 3: Degeneration of Indian potato cultivar Kufri Jyoti due to PVX,Y and PVX+Y
(Pooled for 1981-82) (from Khurana and Singh, 1988).

Seed stock	% infection with			Total % infection	Kg yield/plot* (%depression)
	PVY	PVY	PVX+Y		
Fresh breeder's Seed (control)	3	0.5	1.5	5	33.25 (0.0)
2nd year (1 year old)	15	3	2	20	30.25 (9.8)
3rd year (2 year old)	36	6	6	45	26.20(18.2)
4th year (3 year old	52	11	8	71	17.8 (46.5)

*The actual yield / plot of 150 plants.

several years (Vashisth *et al.*, 1981; Verma *et al.*, 1998).

Incidence of Aucuba and Calico mosaics was relatively higher in old Indian and exotic varieties (Vasudeva and Azad, 1952) but they have, however, become less common in present day Indian commercial potato cultivars (Singh, 1980; Khurana, 1999) hence economically not important. Spread of different viruses, even from the source host with two or more viruses, occurs differently and part by part depending on the nature and mode of virus transmission, say PVX and PVS spread readily through contact as well as most daughter tubers carry them. The horizontal spread of PVY, PVA and PLRV, is mainly through aphid vectors *i.e.* why their combined infections may or may not be lower than their individual infections or with that of contagious ones, viz. PVX,PVS, depending on the age of seed stocks and/or rate of degeneration in the

variety and the area where being cultivated (Khurana and Singh, 1988; Garg *et al.*,1999). Mosaics and leafroll are the most common in occurrence and also severe in the sub-tropical/tropical climate prevailing in plateaux and South Indian plains. It is due to non-frequent replacement of seed stocks, high frequency of the virus sources within and around the crop coupled with large population buildup and greater activity of aphid vectors in such areas (Singh *et al.*, 1981, 1982, 1984a).

4. Symptoms

The commonest among viral disease symptoms are mosaics (Table 4). But the term 'mosaics' includes a larger number of distinct viruses (Khurana and Garg, 1992, 1998) *i.e.* a wide range of overlapping symptoms are invoked by different viruses individually and/or in various combinations, such as super-mild, transient, typical (green) or yellow, mild/severe mosaics or mottle, to general chlorosis of plants coupled with various types of foliage discolouration/ distortion.The yellow mosaics, like that in aucuba, calico, mop-top, or even potato virus U, which show faint/bright yellowing or leafspots, do not affect the plant vigour but may invoke necrotic brown blotches in tubers' flesh in some cultivars (Hooker, 1981).

An intense yellowing of entire plant, but initially the veins and later the entire lamina, is known to be caused by potato yellow vein virus, so far restricted to some Latin American countries. In case of veinal mosaics as in APLV, APMV, chlorosis is confined along the midribs and often chlorotic areas are irregular in distribution, shape and even extend to the adjacent inter-veinal regions forming irregular patterns. Thus, it is often not easy to distinguish it from the common mosaic. Inter-veinal mosaic (as caused by PVX) is marked by characteristic grayish mottle of flat lamina and plants have slightly reduced vigour.

Severe and crinkle mosaics show strongly mottled and severely puckered, small leaves with wavy or indented margins on stunted plants with highly reduced vigour. In contrast, the rugose mosaic is characterized only by mild, inter-veinal puckering of leaves with or without veinal necrosis. Such plants also lack vigour. In case of plants with leafdrop streak, the vigour is less, chlorosis is prominent, leaflet's margins turning downwards and leaf rugosity as well as necrosis on underside veins resulting in leaf-drying. Dried leaves remain hanging along the stems. Such plants only have a 'crown' of green stunted leaves. Potato deforming mosaic causes extreme reduction/ deformation of leaflets (Salazar, 1998). Either combination of several common viruses (say PVX, S, Y and M) or even beet curly top virus or Solanum apical leafcurl virus (singly) result in a localized, severe disease wherein plants remain dwarfed with terminal rosetted leaves (Khurana, 1992; 1999; Salazar, 1996).

PSTVd symptoms are difficult to recognize in field in young plants. They vary according to strain/variety but the severe strains result in dwarfing and uprightness with dark green and rugose leaves and tubers become misshapen, spindly with heavy eye brows and cracks.Tuber symptoms on surface occur as arcs for common viruses but are prominent in case of either PVY-ntn or moptop or TRV and even TNV. They are also observed in case of PAMV but only in certain cultivars after storage at 20-24°C. Potato moptop virus invokes necrotic rings both on and within the tubers. NTN iso-

lates of PVY cause severe but superficial, slightly raised necrotic ringspots/lesions while TRV induces corkiness of tubers showing a few to several necrotic rings or arcs both on the surface and deep in the flesh. TBRV also results in tubers with severe

Table 4 : Symptomatological Key helpful for rouging plants with common potato viral diseases

Latent or Faint Mosaic

PVX	-	Latent in many vars. or interveinal to barely perceptible Mosaic Var. Craigs Defiance shows top necrosis.
PVS	-	Usually latent or very mild or barely perceptible mottle and faint vein banding.
PVT	-	Often symptomless but may produce mild mottle or slight vein necrosis/chlorotic spots.

Mild Mosaic

PVM	-	Latent in some varieties or mild to severe mosaic, slight leaf chlorosis, deformations and wavy margins of leaflets.
PVA	-	Mosaic faint mottling; sometimes leaf distortion, top necrosis in some vars., rarely rugosity, shiny leaves.
PSTVd	-	Very obscure and unusual. Plants rather erect with spindly stunted growth, often with curling leaves darker green than healthy. Leaflets twisted, petioles subtended at 45° angle from the main stem. Tubers cylindrically long with tapered ends. Eyes numerous, conspicous and with distinct eye brows.
APLV	-	Mild mosaic or mottle and chlorotic/necrotic netting of minor veins. Leaf deformation and mosaic in sensitive cultivars under cool conditions.

Severe mosaic/leafroll/dwarfs

Green type

PVY	-	Symptoms vary with strains/variety. Mild or severe mosaic and venial necrosis and leafdrop streak. Plants stunted Rugosity, twisting of leaves with slight inverted cupping of leaflets. Older leaves often colapse and drop or show mild mosaic.
APMV	-	Mild mottle (primary symptom); severe mottle or mosaic (secondary symptom); top necrosis and stunting, leaf deformation and systemic necrosis.

Yellow types

PAMV	-	Bright yellow spots interspersed in the green lamina and stunting.
PMTV	-	Primary symptoms are brown rings on tuber surface extending into tuber flesh as arc. Foliar symptoms occur in secondary infection : Old leaves with bright yellow (aucuba) patches, yellowish 'V' (Chevron) marks on top leaves; stunting of top leaves (simulating a mop) and tuber cracking in sensitive varieties.
PLRV	-	Top young leaves standing upright with pale rolling leaflets, often with pink-reddish margins. Stunted, chlorotic plants with older leaves stiff, dry, leathery and

table 4 contd ....

		rolled like paper with pink to brownish margins.
PYDV	-	Dwarfed and brittle vines with overall yelow appearance. Leaflets have upward rolled margins but the midrib curves downward. Pith necrosis of stems, begins from top. Tubers are usually few, small, deformed and have internal necrosis, often fail to germinate.
TRV	-	Mottling to severe mosaic of leaves, often with deformation and stunting usually on oneor a few out of many stems. Foliage shows yellow 'V' marks or ring-like patches. The tubers may develop necrotic rings/arcs (called spraing) on surface and in flesh.
AMV	-	Alfalfa mosaic virus Calico-Bright yellow marking or complete yellowing of leaflets.
GBNV/	-	Scattered, necrotic local lesions on 'silvery' leaves PSND followed by that on petioles/stems. The plants turn chlorotic tospovirus and blighted.
Yellow mosaic and APCLV geminivirus	-	Dwarfing of plants with curling and bunchiness of apical leaves along with mosaic and chlorosis

cracking and distortion. The stem necrosis tospovirus causes localized lesions in a few stems mainly on internodes but even on point of leaf attachment (Khurana *et al.*, 2001). However, the tospovirus in Europe/Americas causes consequent infection of tubers with some necrotic damage in flesh of tubers of very susceptible cultivars. PLRV induces net necrosis as spots/specs in the tuber flesh (seen only after storage in certain cultivars) but neither arcsnor rings develop as in case of spraing (TRV).

5. Detection

In nature, the above described foliage/stem/tuber symptoms (Table 4) are usually in-voked by infection of single or mixed viruses. It is not easy to identify the causal virus(es) merely on the basis of the visual symptoms because the host (variety), virus(es)/strains and the environment interact to govern the symptom expression. Fac-tors like light intensity and temperature are very important. Other factors like nutrient-deficiency, insect-feeding, etc. may also complicate the visual diagnosis of viral disease(s) during field inspection. Therefore, the symptoms KEY (guide) can be of some help, mainly in field, for tentative diagnosis of the virus(es) involved.

Initially the production of healthy seed potato was based on the selection of apparently healthy plants of desired varieties through field inspection to have 'elite' tubers for clonal multiplication. A good 'crop walker' (person involved in rouging of infected plants) needs enough experience of recognizing virus infections, a well trained eye, good power of concentration and plenty of stamina for crop inspection. Later, the practice of growing single eyes from selected 'clones' was adopted for rejecting the latently infected ones. In 60's use of serological testing (chloroplast agglutination) test

for PVX, S(M); biological testing for PVY/A on 'A6' leaflets, and histological test for PLRV were also adopted for ensuring seed health by CPRI for seed production (Lal and Khurana, 1983). There has been progressive development in the virus detection techniques mainly applied for to maintain and produce virus free nucleus/breeders' seed.

Detection methods used for different viruses and viroid have been indicated with their descriptions. However, the subject has been briefly touched below. Table 5 indicates the detection limits of the two currently employed virus detection tests, viz. DAS-ELISA and NASH (Salazar, 1996).

5.1 Viruses

Biologically the viruses can be readily detected through their reaction on the indicator hosts. Currently, serological methods like ELISA (enzyme linked immunosorbant assay) is in vogue for mass scale indexing while ISEM is used for final confirmation of low concentration of viruses in nucleus stocks/mericlones, etc. (Khurana, 1990a, Torrance, 1992; Salazar, 1996). ELISA tests are highly virus-specific (Koenig and Paul, 1982; Singh and Khurana, 1994; Banttari and Khurana, 1998) as against the fact the many viruses (including unknowns) can be detected on a single indicator host (Franc and Banttari, 1986). Both nucleic acid probes (Querci and Salazar, 1998; Singh, 2000), and electron microscopy including that of ultrathin sections (Roberts, 1986; Khurana and Garg, 1993 and Garg *et al.*, 1993) are highly sensitive and specific. The latter are also useful in determining the virus morphology.

5.1.1 Bioassay

Important indicator plants for potato viruses and their reactions have been shown in Tables 6 and 7. Careful selection of indicator is a must since differences have been reported in susceptibility between accessions of the same species to the virus infection. Inoculum may be prepared by grinding leaf tissue either in tap or distilled or deionised water. One may however use phosphate buffer eg 0.02M phosphate buffered saline (PBS) pH 7.4+2% w/v polyvinyl pyrrolidone (PVP, MW 10000). Test plants are inoculated by rubbing plant sap on leaves lightly dusted with carborundum (400-600 mesh). It is important that each potato must be tested separately. Normally samples from plants should be taken from two different positions. Leaflets from the same plant may be bulked. Symptom development on indicator is often affected by the condition of the test plant, cultural and environmental conditions. They are highly susceptible when young and actively growing; shading them (eg by covering with paper) prior to and after inoculation may enhance their susceptibility and symptom expression. Two different tests, such as bioassay and ELISA, need to be conducted at least to be sure of the conclusion from a bioassay.

5.1.2 ELISA

Different formats of ELISA, viz. Double antibody sandwich (DAS), triple antibody sandwich (TAS) or indirect alkaline phosphatase enzyme label are commonly used for

indexing (Torrance, 1992) or accompany the reagents in case of commercial kits. Comparison of latex agglutination and ELISA (Franc and Banttari, 1986) showed that ELISA is about 4 to 10 times more sensitive in detection of PVX/PVS and has the ability to detect ultralow concentration viruses, like PLRV even in crude extracts. Several variants of ELISA are available now yet the classical direct, double antibody sandwich format in polystyrene microtitre plate (Clark and Adams, 1977) is widely used in routine testing (Casper, 1977; Maat and de Bokx, 1978; Gugerli, 1979; Singh and McDonald, 1981; Khurana and Garg, 1993; Singh and Khurana, 1994). ELISA has made it possible to quantify the relative virus (antigen) concentration using ELISA readers to measure the enzyme colour reaction.

A penicillinase based ELISA was standardized at CPRI (Singh *et al.*, 1989). It employs the indigenously available enzyme and is equally sensitive in virus detection and non-hazardous and economical substrate (Singh *et al.*, 1989, Singh and Barker,

Table 5: Comparative detection range of DAS-ELISA and NASH for some potato viruses (after Salazar, 1996)

Method	Virus[o]		
	PVX*	PVY	PLRV
DAS-ELISA	0.5-10ng (25-500 pg RNA)	0.5-25ng (25-1,250 pgRNA)	50pg-10ng (10-2,000 pgRNA)
NASH	1-10 pg RNA	0.05-3 pg RNA	1.5-6 pg RNA

[o]For DAS-ELISA concentration ranges are expressed in ng for virions, with the equivalent amount of RNA in parentheses.; *PVX, potato virus, X; PVY, potato virus Y; PLRV, Potato leafroll virus.

1991). For most viruses, the sap extract is prepared in standard sample buffer (PBS-Tween +2% PVP).

dot-ELISA on nitrocellulose membranes (NCMs) and other (filter) papers has been successfully adopted with relatively higher sensitivity (Banttari and Goodwin, 1985; Kumar and Khurana, 1989; Kumar and Singh, 1999). The test is similar to that of standard das-ELISA in microtitre plates except that the enzymes substrates to be used must produce insoluble colour precipitate which develop as colour dots on the membrane.

Use of fluorogenic substrates, like 4-methyl umbeliferyl phosphate (MUP) in place of p-nitrophenyl phosphate helped to increase the test's sensitivity 2-4 times (Torrance and Jones, 1982) for detecting PLRV in tubers and viruliferous *Myzus persicae*. Besides, Stanley et al (1985) employed an enzyme amplification system with a primary activator enzyme to activate a secondary enzyme-substrate which was used to detect PLRV in single viruliferous aphids (Banttari and Khurana, 1988-unpublished).

It has been possible to detect one out of several virus(es) in a single or simultaneous test using polyvalent (combined) antisera (Banttari and Franc, 1982; Perez *et*

al., 1988; Kumar and Singh, 1999) but the sensitivity is reduced and the background signals may be higher. Even short duration protocols of either one or half day for the assay are available now (Singh and Khurana, 1999). Further, Reichenbacher *et al.* (1984) evolved a micro-das-ELISA which requires only 5-10% of the reagents. Stobbs and Barker (1985) devised a rapid cocktail ELISA wherein simultaneous incubation of virus samples with the conjugate reduced the assay time and labour. A quick and highly sensitive magnetic microsphere ELISA for PVX and PLRV was developed by Banttari *et al*. (1991) wherein the analyte, conjugate and the antibody coated microspheres are

Table 6 : A suggested list of diagnostic indicator hosts for biological detection of ten important potato viruses

Host	VIRUS									
	PVX	PVY	PVA	PLRV	PSV	PVM	PVT	APMV	APLV	PMTV
Nicotiana tabacum	L,S	S	S	*	-	-	-	S	S	*
N.clevelandii	S	S	S	-	-	-	-	S	S	S
N.glutinosa	S	S	S	-	-	-	-	S	S	-
N.debneyi	S	S	S	-	S	L	-	S	S	S
Physalis floridana	L,S	L,S	S	S	-	-	-	-	-	-
Lycopersicon esculantum	S	S	S	-	-	S	-	-	-	-
Datura stramonium	S	-	-	S	-	-	-	S	-	-
D.metel	S	S	S	-	L	L	-	-	-	-
Gomphrena globosa	L	-	-	-	-	-	-	-	-	-
Chenopodium amaranticolor	L	-	-	-	-	-	S	-	LS	L
C.quinoa	L	-	-	-	-	-	S	-	L	LS
Phaseolus vulgaris	-	-	-	-	L	L	LSR	-	-	-
Potato clone A6	L	L	L	-	L	-	-	S	S	-

* - = absence of symptoms. Only local (L) and systemic (S) symptoms with diagnostic value are indicated for each virus. Host susceptibility without symptoms and insusceptibility are not taken into consideration. PLRV is inoculated through aphids or grafting, whereas for all other viruses mechanical inoculation with infective sap is used. Bold type indicates the most sensitive hosts. R= Recovery from symptoms.

incubated together just for 10 minutes.

Production of Monoclonal antibodies (M/Abs) is highly sophisticated yet they have been produced against several potato viruses and useful in detection and diagnosis of viruses esp. that are difficult to purify(van Regenmortel, 1984; Karande *et al.*, 1998). These are highly specific and can be of great use in particular studies viz. Epidemiology of specific strains of a virus. They can also help in studying the architecture of a virus. However for detection and diagnosis the McAb are likely to recognize almost all the serotypes of a given virus/group, eg the Universal PVY analyser McAb. Recently ADGEN,UK has marketed a McAb based rapid but virus specific field ELISA

Table 7: Diagnostic symptoms/reaction of potato viruses of quarantine   importance on potato
and recommended biological indicators

Virus	Symptoms on potato	Reaction on indicator hosts
APLV	Latent/MM and Chl netting of Pr. Veins and slight rugosity	*N. bigelovii* - LL/mosaic, *N. clevelandii* -severe mosaic/vein netting *N.benthamiana*- Chl./Necr LL / Syst. Rugosity, mottle.*N.occidentalis* - 'P1' - Chl/ Necr. LL.
APMV	Chl. Blotches/S.Mottle rugosity and dwarfing	*N.bigelovii/N.clevelandii*-systemic mosaic
AVB-O	Symptomless	*C. amaranticolor* - Necr. Lesions, syst. Mild mosaic and then recovery.*C. murale* - Syst. Chl. Mottle, necrosis of tip/upper leaves.
EMDV	Stunting, epinasty, Dwarfing, chlorosis and syst. necrosis	*Datura stramonium/Phaseolus vulgaris* - chlorosis and wilting/Local lesions. *N. glutinosa /N./rustica* - Chl. LL and and/syst. conspicious chlorosis.
PBRV	Syst. necrotic spotting	Ch.quinoa-Necr.LL and Syst apical necrosis/death.
(TRSV-Ca)	Bright yellow blotches	*N.glutinosa/N.rustica* -Chl.LL and/syst. conspicious chlorosis.
(TBRV)	Black necr. spots/rings	
PDMV	Severe leaf deformation and yellow mosaic	On grafting *D.stramonium* and tomato 'Rutgers' show yellow mosaic and leaf distortion.
PotLV*	Symptomless	? (*N. bigelovii* - Faint syst. mottle) ? (*C. murale* - Local chl. spots)
PMTV*	Yellow blotching, 'chevron' markings and extreme stunting; Tuber flesh with lines/rings or brown arcs	? (*C. amaranticolor*- Conc. Brown LL) ? (*C. quinoa*- LL and necr along veins ? (*N. debneyii* - Syst. Chl. mottling)
PVT	Usually systomless	*Ch. amaranticolor/C.quinoa* / Local chl.Spots, syst. Leaf necrosis and mosaic.
PVU^SU	Symptomless/(bright Yellow leaf markings)	*C.amaranticolor/C.quinoa*-Chl.& necr. local lesions, syst.mottle and leaf deformation
PVV^SU	Symptomless in most cvs/ some cv may show mosaic and necrosis	*N.debneyii*-Chl./LL and syst. Vein clearing/mosaic/chl. Rings. *S.demissum* 'A' - Syst. Necrosis.
PYM	Bright yellow mosaic, dwarfing stunting	*Petunia hybrida* - Chl.spots in leaf distortion and inoculated leaves and syst.golden yellow mottle/deformation/
PYV	Symptomless but causes	*Capsicum annuum*-Syst. Leaf disstor-

table 7 contd.....

	mosaic rarely yellowing and early senescence	tion /yellow mosaic. *N.tabacum* "Samsum" - Syst. Line patterns/mosaic and chl./spots.
TNV	Only tubers show light/ dark brown rings which darken, crack or sunk upon storage	*Phaseolus vulgaris* cv."Dubbele Witte" or "D.W. zonder Draad" - Necr. LL.
TRV[su]	Often show tuber 'Spraing' or brown arcs in flesh. Foliage symptoms-mottling of one or few stems and stunting	*C. amaranticolor* - Necr. LL.*C.quinoa* - Spreading LL.*P. vulgaris* - Local pinpoint lesions.
TSV	Necr.conc. rings/lesions on leaves/stems in pr. Infection red/chl. Lesions/syst.necrosis.	*N.tabacum* 'White Burley' - Local necr. spots/rings. *Vigna unguiculata* ssp *cylindrica*- Local
WPMV**	Severe chl.mosaic/ deformation rugosity/ enation in wild spp.	*N.bigelovii/N clevelandii* - Systemic mosaic.

LL = Local lesions (Chl. - chlorotic; Syst. -systemic, Necr. Necrotic/ necrosis.
* = Use of ELISA recommended for detection; ? = Biol. Test on indicator hosts is unreliable
** = Test using Potyvirus specific a/b ELISA.; Su = Serological detection unavailable/unreliable

under the trade name of "SPOT-CHECK". It is based on a test strip which is precoated with specific antibody and allowed to react briefly with the sample solution, probe antibody, enzyme conjugate and finally the substrate in a test tip connected to a syringe. Nevertheless, there are problems at times due to high specificity and sensitivity of ELISA. Clarke *et al.* (1980) observed a relatively higher background even for healthy potato tubers in ELISA for PLRV. Tuber ELISA also did not detect low concentrations of both PVY and PLRV, that could be detected later upon ELISA of sprouts/foliage (Khurana *et al.*, 1991; unpublished).

5.1.3 Electron Microscopy

Association of the virus particles in a sample may be best done by viewing preparations either by direct or immunosorbent electron microscopy (ISEM) (Roberts, 1986). ISEM is a highly sensitive method for virus detection (Khurana and Garg, 1993). It is 100 times sensitive than the conventioal leafdip electron microscopy PLRV (Garg *et al.*, 1989; Singh *et al.*, 1990).

Various factors such as pH of the extraction buffer, pH and titer of the trapping antisera and their combinations, 'virus-acquisition time'on EM grids and the virus host, may affect ISEM for virus detection (Garg and Khurana, 1992). The method of preservation and storage of virus infected leaf tissue or extract also adversely affects ISEM (Khurana *et al.*, 1993). Combined antisera, esp. that of PVS/ PVM, influence virion morphology and may hamper trapping of potyvirions and even dislodging the

trapped particles (Garg and Khurana, 1993,1994). ISEM for potato viruses X/S was found to be ideal with sample extracts in phosphate buffer (0.1M, pH 7.0) while supplementing the extraction buffer with 0.1M EDTA helped in preventing loss of PVY due to aggregation (Garg and Khurana, 1992). Higher titres of the antiserum used for 'decoration' of PVY in ISEM caused deterioration/degradation of the virions (Garg and Khurana, 1993). Such an effect was not observed in case of PVX and PVS. The decoration was heavy under all conditions except at pH 6.0 and dilution 1:0.5 of antiserum when only a small proportion of virions got decorated. Besides, the pH of the extraction buffer and trapping antiserum affected virion trapping (Garg and Khurana, 1992) while in case of PVY it was also affected by the host species (Garg and Khurana, 1992; Garg et al., 2000).

An effective simpler ISEM technique has also been standardized for detecting PLRV in the vector aphids and tuber sprouts (Garg et al., 1989; Singh et al., 1990). In case of PLRV, only a few virus particles are trapped upon routine ISEM and required not only prolonged incubation on the grids but also EM screening time per sample. The problem has been resolved by solid phase protein A-ISEM wherein the clumps of PLRV particles and homologus antibodies are trapped on grids coated with protein-A (Garg and Khurana, 1991). For PSND tospovirus, though ISEM was not effective yet the virus was readily detected through EM in dip-preparations provided the samples (virus) of potato stem necrosis were prefixed with glutaraldehyde (Khurana et al., 1990a).

5.2 Viriods

PSTVd spreads readily through contact and also the pollen and true potato seed or TPS (Singh, 1970). It is, therefore, essential to check the true seeds, seedlings for freedom from the viroid that can be detected either through bioassay either on tomato 'Rutgers' or Scopolia sinensis. It is not only time, labour and space consuming, inoculated plants have to be provided temperature above 25°C and continuous fluorescent light intensity to help express symptoms within 2-4 weeks (Grasmic and Slack, 1985). Some isolates of PSTVd were detected in India from potato plants and true seeds on biological indicator tomato cv. Rutgers. They proved to be mild isolates and did not produce lesions on S. sinensis. Yet another distinct PSTVd isolate from wild vein-necrosed tuber-bearing Solanum spp. was established (Khurana et al., 1990b; Owens et al., 1992).

5.2.1 PAGE

Since the viroid is a free RNA, it was easily detected using an electrophoretic procedure (Morris and Smith, 1977) and till recently, a simple one-day-procedure was extensively employed, even for mild strains (Salazar, 1989). The combination of tomato-electrophoresis procedure confirms presence of PSTVd even in single TPS (Grasmick and Slack, 1986, 1987). The "Return" gel electrophoresis has also been standardized for reliable detection of PSTVd in small quantities of test tissues (Singh and Boucher, 1987). But the use of nucleic acid probes (Owens and Diener, 1981) of PSTVd replaced the PAGE method. Salazar et al. (1988) observed that as minmum as

0.33 pg of PSTVd could be detected using RNA transcripts. The nucleic acid spot hybridization (NASH) test is 1000 times sensitive than PAGE. It is possible to detect even single infected seed among 80 healthy ones (Salazar, 1989, Singh *et al.* 2000).

5.2.2 Nucleic acid hybridisation

Although cDNA or cRNA probes are available for many potato viruses, their use is still limited to certain laboratories (Singh, 2000). Cold (Non-radioactive) probes, based on digoxigenin, etc are now available with similar sensitivity but longer shelf life (Querci and Salazar, 1998). To avoid sample deterioration in transit, dot/squash blots or NASH (nucleic acid spot hybridisation) methods are used by first trapping the virus particles/ nucleic acid on membranes which are then mailed them for probing by a central laboratory.

Besides, viruses can also be detected through use of the specific primers, developed recently for use in reverse transcription polymerase chain reaction (RT-PCR) protocols (Innis *et al.*, 1990). An evaluation of NASH and RT-PCR for detection of PVY in dormant tubers and its comparison with visual symptoms and ELISA in plants of several Canadian varieties showed no significant difference between them, suggesting that any one of these could be used (Singh *et al.*, 1999a). Phenolic compounds in plant-tissues inhibit RT-PCR. Multi-step protocols, using several additives to inhibit polyphenolic compounds during nucleic acid extraction, are common but time consuming and laborious. Use of sodium sulphite (0.65 to 0.70%) in the extraction buffer minimizes the pigmentation and improves detection (Singh *et al.*, 2002). Recently, a multiplex reverse transcription polymerase chain reaction (m-RT-PCR) was developed for the simultaneous detection of five potato viruses and a viroid (Nie and Singh, 2000, 2001). This helps save on both the costs and time.

The tuber necrotic ring spot disease, a serious problem in some parts of Europe and because it can not be distinguished serologically from PVY[n], ADGEN, UK has come up with a diagnostic test combining serology and PCR. In this the distinguishing sequence differences between the strains are detected through primers specifically designed to detect PVYntn from PVY-o and PVY[-n]. RT-PCR may replace common serological tests for viruses etc., if simplified and robust protocols are developed for routine indexing purpose (Seal and Coates, 1997; Singh, 2000).

6. Pathogen descriptions

Virus combinations are most common in field causing synergistic severe disease(s). Therefore, to be sure of diagnosis, many characteristics, serological relationship and/or virus particle morphology have to be checked. The following descriptions of most common and important viruses, and the data have been summarized in Table 8, to help people involved in potato viruses research or routine detection, etc.

6.1 True seed transmitted

Potato Spindle Tuber Viroid Potato spindle tuber viroid (PSTVd) is an infectious, most

contagious and highly stable, circular single stranded RNA. PSTVd is the only viroid known to be naturally infecting potatoes. It is commonly having 359 nucleotides but rarely 358 and 360 (Herold *et al.*, 1992; Lakshman and Tavantzis, 1993) in potato. Various isolates have only very minor differences in homology yet they cause different types of symptoms in potato.

Symptoms of PSTVd are often obscure due to naturally combined virus infections in field. Normally, PSTVd infected plants remain stunted with upright growth, dark green and rugose leaves. Tubers produced are small, spindling with conspicuous eye brows, even having cracks (Singh, 1983, 1988a,b). Extensive veinal necrosis has been observed in leaves of a few wild *Solanum* spp. (Owens *et al.*, 1992). The natural host range of PSTVd is narrow. Primarily found infecting potato, tomato, avocado, wild *Solanum* spp. (Owens *et al*., 1992; Salazar, 1996). Widespread in North America, parts of Eastern Europe, the former USSR, China, etc and readily detected in germplasm from such countries (Mukherjee *et al.*, 2000). PSTVd incidence has now come far lower due to constant efforts for its eradication over the last 10-20 years in North America and parts of Eastern Europe (Salazar, 1996).

Yield losses upto 64% have been observed due to PSTVd (Singh,1983) depending on the isolate, cultivar and growing conditions. Botanical seed and breeding programmes are most vulnerable to viroid infection (Singh, 1970). PSTVd spreads rapidly through pollen and botanical seed (Singh, 1970; Singh *et al.*, 1992b) and even contact, but mainly by machinery in the field. Experimental transmission by aphids, *Myzus persicae* from plants co-infected by PLRV has also been observed (Querci *et al.*, 1997).

Biological detection of PSTVd was done for long on tomato varieties Sheyenne or Rutgers. But the samples for viroid testing need to be grown at higher temperatures (>18°C). The symptoms are best at 25-28°C and high light intensity after at least two weeks (Grasmick and Slack, 1985), yet mild strains remain latent hence can be missed.

The viroid was mostly detected by Return- PAGE (polyacrylamide gel electrophoresis) with silver staining (Singh and Boucher, 1987) not only for its detection but also separation of a severe from mild strain. NA probes are more sensitive using P^{32} - or digoxigenin-labelled DNA (Harris and James, 1987) or RNA probes (Salazar and Querci, 1992; Querci and Salazar, 1998). Highly sensitive RT-PCR methods are also available now (Levy *et al*., 1994; Shamloul *et al*., 1997).

6.2 Contact transmitted

Latent or Faint mottle/Inter venial mosaic (Potato virus X) Potato virus X (PVX) (Potexvirus) has 515 x 13 nm filamentous, It is stable and occurs in high concentration serologically related to white clover mosaic and aucuba mosaic viruses. Occurrence of serotype PVX^0 is the most common occurrence world-wide. Serotype PVX^A has restricted distribution while cp strain is restricted to South central Andes, Peru. Strain HB occurs in Bolivia breaking the extreme resistance of Rx (immunity genes) (Moreira *et al.*, 1980) It was only once encountered in India (Kumar *et al.*, 1987). Recently, 'MS' strain from Argentina, was also found to break the Rx gene resistance in cultivar Serrana Inta (Tozzini *et al*., 1994).

PVX normally causes inter venal mosaic or faint mottle in young plants. Some strains may incite mosaic while rugose mosaic occurs upon combination with PVY. PVX has a narrow natural host range mainly infecting potato, tomato, *Nicandra physaloides, Amaranthus virdis* and *Portulaca oleracea* are, however, important weed hosts common in the potato fields. Experimentally PVX infects over 240 species in 16 families. PVX is economically important as its incidence is high despite lower potential for yield reduction, usually 15-20% (Khurana and Singh, 1988). Combined infection with other viruses, particularly PVA and PVY, cause severe diseases and higher yield losses.

PVX is highly contagious, spreading upon cutting of tubers or mechanically and also upon plant-to-plant contact or through farm machine or otherwise contaminated implements.

Bioassay of PVX is most effective. It is detected easily through sap inoculation on biological indicators like *Gomphrena globosa* and *Capsicum pendulum. Nicotiana benthamiana, N glutinosa* and *N. tabacum* cvs Samsun and White Burley) give necrotic spots/ringspots on inoculated leaves and systemic necrotic mottle, mosaic, or veinal chlorosis due to two serotypes PVX° (includes PVX-MS) and PVX^A (includes strains cp and HB).

Serologically PVX is readily detected through chloroplast latex, agglutination tests in field etc. ELISA using polyclonal antibodies is highly effective (Singh *et al.*, 1989; Singh and Khurana, 1994).

McAb based ELISA can distinguish between the strains (Torrance *et al.*, 1986;Karande *et al.*, 1998). ISEM is resorted to detect very low concentration of the virus in mericlones (Khurana and Garg 1993; Khurana and Sane, 1998). Nucleic acid probes though helpful (Querci *et al.*, 1993; Querci and Salzar, 1998) are not much used in detection of PVX.

6.3 Contact and non-persistently aphid transmitted

6.3.1 Latent/Faint mosaic (Potato virus S)

Potato virus S (PVS) (Carlavirus) has slightly flexuous 660 x 12 nm filaments. The virus occurs in low concentration yet most easy to detect biologically. It is serologically related to PVM and carnation latent virus. Two strain groups have been recognized, designated PVS° (ordinary) and PVS^A (Andean) (Foster, 1991). PVS is normally latent and infected plants of most of the potato varieties look almost healthy but for occasional/transient leaf symptoms of faint rugosity, vein deepening and leaf bronzing; PVS^A may invoke severe symptoms (Salazar, 1996).

Host range of PVS is highly restricted both naturally and experimentally. It infects potato and pepino (*Solanum muricatum*) and only some species of Chenopodiaceae and Solanaceae.

PVS occurs worldwide and is the most frequently found virus in potato. An incidence of 8.5 to 99.5% has been recorded in Indian potatoes at different locations (Singh *et al.*, 1994). Reduction in tuber yield is very low but may go upto 10-20% or more when in combined with PVX. It has been found breaking late blight resistance in some potato cultivars (Khurana, 1992). PVS is most contagious, readily spreading through

Table 8: Characteristics of viruses infecting potatoes

Pathogen	Acronym	Genus	Virus properties								
			1	2	3	4	5	6	7	8	9
Alfalfa mosaic	AMV	Alfamo	Bac virus	4-5	19	30-58	Aphids	Non-presistent	Self eliminating	Widespread	Low
Andean potato latent virus	APLV	Tymovirus	Iso	1	28-30	N/A	Beetles	-	Contact, TPS	S. America	Low
Andean potato mottle	APMV	Comovirus	Iso	1	28	N/A	*Beetles*	-	Contact	S.America	Low
Arracacha B (ocastrain)	AVB	Nepovirus	Iso	1	26	N/A	-	-	TPS	S. America	Low
Beet curly top	BCTV	Gemini virus	Seg	2	18-20	32-35	Leaf hopper	Persistent	-	MiddleEast N. America	Low
Cucumber mosaic	CMV	Cucumo virus	Iso	1	30	N/A	Aphids persistent	Non-persistent	-	Widespread	Low
Potato A	PVA	Potyvirus	Fil	1	--	730	Aphids	Non-persistent	-	Widespread	Medium
Potato aucuba	PAMV	Potexvirus	Fil	1	11	580	Non-persistent transmission when PVY or PVA present			Widespread	Low
Potato black ringspot	PBRSV	Nepovirus	Iso	1	26	N/A	*Nematodes*	-	-	S. America	Low
Potato deforming mosaic	PDMV	Gemini virus	Seg	2	18	30	-	-	-	S. America	Medium
Potato leafroll	PLRV	Luteovirus	Iso	1	24	N/A	Aphids	Persistent	-	Widespread	Large

Potato M	PVM	*Carlavirus*	Fil	1	12	650	Aphids	Non persistent	-	Widespread	Medium
Potato mop-top	PMTV	Furovirus	Rod	2	18-20	100-150 250-300	Fungus	Persistent	Self-eliminating	Cooler climes N & S. hemisphere	Medium
Potato S	PVS	Carlavirus	Fil	1	12	660	Aphids	Non-persistent	Contact	Widespread	Low
Potato spindle tuber	PSTVd	Viroid	None	Circular ssRNA only			Aphid-borne when plants co-infected with PLRV	TPS		Widespread	Medium
Potato T	PVT	Trichovirus	Fil	1	12	640	- TPS	Contact,		S. America	-
Potato V	PVV	Potyvirus	Fil	1	12-13	700-	Aphids 720	Non-persistent	-	S. America, Europe	Low
Potato U	PVU	Nepovirus	Iso	1	28	N/A	Nematode-		-	S. America	-
Potato X	PVX	Potexvirus	Fil	1	13	515	-	-		Widespread	Medium
Potato Y	PVY	Potyvirus	Fil	1	11	740	Aphids	Non-persistent	Contact	Widespread	Large
Potato yellow dwarf	PYDV	Rabdo virus	Bac		75	380	Leaf hoppers	Persistent	Self-eliminating	N. America	Low
Potato yellow mosaic	PYMV	Gemini virus	Seg	2	18-20	?	Whitefly	-	-	S. America	Unknown
Potato yellow vein	PYVV	Clostero virus	Fil	-	-	-	Whitefly	-	-	S. America	Large
Potato yellowing	PYV	Alfamo virus	Bac		21	60	Aphids	Semi-persistent	TPS	S. America	Unknown
Red la Soda	RLSV	*Carla virus**	Fil	1	12	625	Aphids	*Non-persistent*	-	N. America	Low

table 8 contd....

Virus	Abbr	Group					Vector			Distribution	Significance
Solanum apical leaf curling	SALCV	*Geminivirus*	Seg	3	18	50	-	-	-	S. America	Unknown
Tobacco black ring	TBRV	Nepovirus	Iso	1	28	N/A	Nemato-todes	Persistent	TPS, self-eliminating	Widespread	Low
Tobacco mosaic	TMV	Tobamovirus	Rod	1	18	300	-	-	Contact	Widespread	Low
Tobacco necrosis	TNV	Necrovirus	Iso	1	26	N/A	Fungus	Persistent	-	Widespread	Low
Tobacco rattle	TRV	Tobravirus	Rod	2	22	50-115 190	Nematodes	Persistent	-	Mainly N. hemisphere	Medium
Tobacco streak	TSV	Ilarvirus	Quasi iso	1	22-35	NA	*Thrips*	-	Self-eliminating	S. America	Low
Tomato mosaic	ToMV	Tobamovirus	Rod	1	18	300	-	-	Contact	Widespread	Low
Tomato spotted wilt or Peanut bud necrosis Potato stem necrosis	TSWV PBNV	Tospovirus(es)	Env	1	70-110	N/A	Thrips	Persistent	-	Widespread	Large in localised areas
Wild potato mosaic	WPMV	*Potyvirus*	Fil	1	??	685 800	Aphids	Non-persistent	-	S. America	Low

Key to columns of virus properties:

1. Particle morphology (Bac = bacilliform; Iso = isometric; Seg = segmented (*i.e.* geminiviruses that occur as bi – or tri – geminate particles in addition to the single particles, bigeminate are usually the predominant the most stable form); Fil = filamentous; Rod = rod or tubular shaped; Quasi – iso = quasi-isometric; Env = Enveloped particles.

2. No. of distinct particle sizes; **3.** Particle diameter; **4.** Particle length for rods, filaments, segmented (Geminiviruses) and bacilliform particles. **5.** Vector type; **6.** Persistence of virus in vector; **7.** Transmission through botanical true seed (TPS), self-elimination (SE) from tubers, and by transmission from plant-to-plant (contact); **8.** Geographical distribution; **9.** Significance to potato production taking into account severity of symptoms and frequency of infection. NB : Entries in italics are based on provisional data.

seed cutting and even plant-to-plant contact. Some isolates (mainly PVS[A]) also spread in a non-persistent manner by aphids, particularly *Myzus persicae* (Slack, 1983). *Portulaca oleracea* has been found to be the common collateral host though symtomless and may not play much role in epidemiology of the virus.

Biological detection of PVS is easy on *Chenopodium murale* and more effective on *C. quinoa*. PVS[o] isolates result in chlorotic local lesions while PVS[A] invokes systemic chlorosis and necrosis. Latex agglutination, ELISA (Fribourg and Nakashima, 1974; Singh and Khurana, 1994) and ISEM (Garg and Khurana, 1992; Khurana and Garg, 1993) for PVS are easy. NASH is also effective while McAb help in specific detection of PVS[A] (Cerovska and Filigarova, 1995).

6.3.2 Leaf-rolling mosaic or paracrinkle (PVM)

Potato virus M (PVM) (Carlavirus) serologically related to PVS, has slightly curved filaments of 650 x 12 nm. PVM generally involves only transient mosaic, crinkling and waviness, rolling of margins of leaflets (paracrinkle) with leaves tending to roll and stunting of shoots.

It also has a very narrow host range, like PVS, infecting mainly potato and only a few species of Solanaceae. Despite worldwide distribution, it has utmost significance in eastern European varieties but not common in the Andean region of South America (Salazar, 1996).

PVM normally causes only less reduction of potato yield that may go up to 15-45% as observed in eastern Europe and Russia where some cultivars may be 100% infected (de Bokx and van der Want, 1987). PVM is experimentally sap transmissible but it naturally spreads through infected tubers and aphids in a non persistent manner.

Biological detection is possible through sap inoculation on *Datura metel* (first showing chlorotic/necrotic lesions and then systemic, rugose chlorotic mottle), or *Phaseolus vulgaris* cv. Red Kidney developing pin head size necrotic local lesions for most of the isolates. PVM can be best detected through latex agglutination (Fribourg and Nakashima, 1974); ELISA (Khurana and Garg, 1993; Singh and Khurana, 1994) and ISEM (Garg and Khurana, 1992, 1993).

6.3.3 Streak or Severe/Rugose mosaic (PVY)

Potato virus Y (PVY) (Potyvirus) has flexuous, 740 x 11 nm long filaments, occuring in low concentration in potato and serologically related to PVA. PVY isolates have been grouped into different strain groups on the basis of local and systemic symptoms on tobacco, *Physalis floridana* and potato cultivars: C, the stipple streak strain; O, the common strain; N, the tobacco veinal necrosis strain (including NTN, the tuber necrotic strain of N) (Khurana, 1992; Salazar, 1996). A new straingroup, 'Z' has also been suggested (Jones 1990). Some distinct isolates of PVY[C]-GL/AB have been reported to be serologically related but biochemically distinct and named as PVV (Fribourg and Nakashima, 1984).

PVY[o] normally involves severe mosaic symptoms on most cultivars. But mild rugosity, drying and dropping of leaves (leaf drop streak) and severe systemic necrosis

are also frequent uncommon. (Khurana *et al*., 1975). PVY^C usually causes stipple streaks or necrotic 'stars'/veins. N isolates usually cause only slight leaf symptoms. NTN isolates cause severe superficial tuber necrosis (potato tuber necrotic ring spot disease) and may also cause necrotic foliar symptoms (van den Heuvel *et al*., 1994; Salazar, 1996). Besides potato, PVY has a very wide natural host range infecting many important solanaceous crops including pepper (*Capsicum* spp.), tobacco (*Nicotiana* spp.), tomato (*Lycopersicon esculentum*), and a large number of weeds. Experimental host range also wide, covering more than 400 species in 30 families. PVY takes heavy toll of yield usually above 30% and up to 80%. Losses are naturally higher in plants coinfected with PVX (Khurana and Singh, 1988; Khurana, 1992). PVY infected plants are rendered less susceptible to late and early blights (Kalra *et al*.,1989, Singh and Khurana, 1993).

PVY-O strain group occurs world-wide, PVY-C strain group in Australia, Ecuador and India; N strain group in Africa, Europe, South America, the former USSR and localised areas in North America. PVY^N strains have been known since 1940 from Europe, South America, and also New Zealand for some time. However, the PVY^{NTN} isolates were detected later and only in the last decade it has been found in most countries in Europe, Lebanon, Israel, Portugal, Italy and South America with some symptoms variations. Oshima et al (2000) observed that distinct geographical sequence variants may exist.

Aphid species (mainly *Myzus persicae and Aphis gossypii*) vector PVY in a nonpersistent manner. Plant-to-plant contact transmission for some Y^N isolates is known. Sap inoculation either to clone *Solanum demissum* 'Y' or 'A6' *(Solanum demissum x S. tuberosum* cv. Aquila) results in local lesions. *N. tabacum* cv. White Burley is the most useful host to differentiate the Y^N strains (systemic vein banding and severe veinal necrosis) from the strains Y^o/Y^c (vein banding and mottle).

Detection is effective through ISEM and ELISA with polyclonal antibodies against any strain group (Khurana and Garg,1993). Peptide profiling of tryptic digests of coat proteins of PVY strains by HPLC helped in differentiating Y$^°$ strain isolates (Bhat *et al*., 1997). It is also possible to detect virus in aphids by duplex reverse transcription-PCR (Singh, *et al*., 1996).

McAbs specific to or detecting most isolates of PVY^C, PVY^N and PVY$^°$ with or without little cross reaction to other stains are available (Ellis *et al.*, 1996; McDonald and Singh, 1996). Nucleic acid probes and PCR have been effectively used for detection of PVY and differentiation of strains (Baulcombe and Fernandez-Northcote, 1988; Barker *et al*., 1993; Weidemann and Maiss, 1996).

6.3.4 Super Mild mosaic (Potato virus A)

Potato virus A (PVA) (Potyvirus) has flexuous 730 x 11 nm long filaments and is serologically closer to PVY. It also   has distinct strain groups, based on hypersensitive response in potato (Valkonen *et al*., 1995).

PVA normally incites super mild mosaic, often with shining or rough leaf lamina, yet there are many varieties that do not develop any symptoms upon infection with common strains. PVA naturally infects only potato and even experimentally, it infects only a few Solanaceaeous species. PVA also has a World-wide distribution, yet rare in

South America, Andes (Salazar, 1996)and not at all common in Indian potatoes. PVA has a lower incidence, rarely occurs alone *i.e.* it coinfects potato with PVX or PVY. Yield reduction due to PVA may go up to 30-40% but naturally higher in combination with PVX and/or PVY (Dedic, 1975; Khurana, 1992). PVA is mainly aphid transmitted non-persistently through *Myzus persicae* and some aphids.

ELISA is the best for detection of PVA (Gugerli, 1979; Vetten *et al.*, 1983; Browning *et al.*, 1995) and equally well with ISEM (Khurana and Garg, 1993). Biologically PVA is detected by sap inoculation to *Nicotiana tabacum* cvs Samsun or White Burley, developing systemic symptoms while *Solanum demissum* clone 'A' or 'A6' (*Solanum demissum x S. tuberosum* cv. Aquila). *Physalis angulata* and *S. microdontum* (PI 558098) react with local lesions.

6.4 Persistently Aphid Transmitted Virus

6.4.1 Potato leafroll virus (PLRV) (Luteovirus)

PLRV has isometric virions of 24 nm diameter. It occurs in extremely low concentration, confined to the phloem cells (de Bokx and van der Want, 1987; Rowhani and Stace-Smith, 1979; Singh *et al.*, 1990; Garg and Khurana, 1991). It is serologically related to beet western yellow virus while Tomato yellow top virus is possibly having seroaffinity to PLRV (Salazar, 1996).

Leafroll, the most severe viral malady of potato, upon primary infection causes chlorosis of top (young) leaves with an erect habit of the infected plant while secondary symptoms include stunting of shoots, upward rolling of older leaves turning chlorotic, leathery and brittle. Some cultivars also show reddening or purpling of leaves. Water logging, excessive calcium in soil, or even drought stress and maturity characteristics in some varieties, result in pseudo (leafroll) symptoms which, however, are without thickening and consequent brittleness. Temperatures below 10 °C and early rains in winter season or *rabi* crop Indian plains delay the emergence and crop growth. Severe stem infection of *Rhizoctonia solani* (black scurf) also results in slightly yellowed and rolled top leaves disguising the primary symptoms of PLRV. PLRV is also having a narrow host range both naturally and experimentally infecting mostly *Solanum* spp.

PLRV has world wide distribution. Incidence of PLRV, however, remains low when seed stocks are frequently replaced and systemic insecticides are used. It causes heavy yield losses (up to 90%) and also reduction in tuber quality due to net necrosis in the flesh.

Aphids transmit PLRV in a persistent/circulative manner of which *Myzus persicae* is the most important and efficient vector; *Macrosiphum euphorbiae, Aphis gossypii, Acyrthosiphom pisum* and *Aulacorthum solani* also poorly transmit PLRV (Singh *et al*., 1981, 1982).

PLRV was earlier detected biologically through grafting on to *Datura stramonium* (systemic interveinal yellowing), *Physalis pubescens L.*(=*P.floridana* Rydb.) (systemic interveinal chlorosis, older leaves slightly rolled, plant stunted) and susceptible *Solanum tuberosum* lines (eg DTO-33, Maris Piper, Russet Burbank. Histochemical test for callose

deposit due to PLRV in tuber tissue was *in vogue* (de Bokx, 1972) but has been abandoned since development of ELISA. PLRV is now easily detected through ELISA (Smith and Banttari, 1987; Van der Heuvel *et al*., 1989; Singh *et al*., 1990; Banttari *et al*., 1991). Various additives to the extraction buffer help improve ELISA detection of PLRV (Singh and Khurana, 2000). 'Cocktail' ELISA has higher sensitivity. Detection of PLRV may be difficult in some potato genotypes because of "restricted virus multiplication" (Barker and Harrison, 1985) where either ISEM (Garg and Khurana, 1991) or Nucleic acid probes are helpful (Robinson and Romero, 1991; Loebenstein *et al*., 1997). The virus can also can be detected in single aphids by RT-PCR for epidemiological investigations (Singh *et al.*, 1995, Singh 2000).

6.5 Thrip and whitefly transmitted viruses

6.5.1 Stem Necrosis tospovirus

Potato stem (and foliar) necrosis disease (PSND) is rather common in early crop of potatoes in Central/Western plains and plateaux of India. A tospovirus was found to cause PSND. The virus has large roughly spherical, enveloped particles, ranging from 70-110 nm in diameter, covered with knob-like surface projections (Best, 1968; Khurana *et al*.,1990; Garg *et al*, 2000)).

Infected plants show extensive necrosis with formation of concentric rings or spots on leaves and stems. Shoots that are not killed have stunted/rosetted appearance and chlorotic necrotic ring spots on leaves. Tubers on such plants may be few, sometimes small and rarely deshaped yet without the virus (Khurana *et al*., 1989a and b, 1990,1997; Garg *et al*., 2000).

The tospovirus has a wide natural host range including crops like tomato (*Lycopersicon esculentum*), tobacco (*Nicotiana* spp.), peanut (*Arachis hypogaea*), soyabean (*Glycine max*) and cotton (*Gossypium* spp.). Experimentally also wide covering over 30 monocotyledonous and dicotyledonous families.
Distribution of the tospovirus is worldwide. It is known to naturally infect potatoes from Argentina, Australia, Brazil (Best, 1968; Salazar, 1996) and central India (Khurana *et al*., 1989a and b; 1997).

PSND is important only in localized areas where both the vector (thrips) and virus sources occur. It is also prevalent on some other hosts. Viruliferous thrips move to early crop of potatoes from other preceding crops. They cause losses upto 29% (Singh *et al*., 1997a,b). Planting after end of October is helpful in reducing the disease incidence almost to negligible by avoiding crop exposure to the vector. It was also achieved through use of systemic insecticide either as tuber dressing or/and foliar sprays (Singh *et al*., 1999b).

The tospovirus infecting Indian potatoes in Central/Western India was found serologically related to peanut bud necrosis tospovirus (Khurana *et al*., 1989a,'97; Jain *et al*., 2000). Several thrips species belonging to the genera *Thrips* and *Frankliniella* act as the virus vectors in a persistent manner but they can acquire the virus only as nymphs, with 4-9 days of latency and about 1 hr inoculation access (Wijkamp *et al*., 1996). Detection is readily possible either by ELISA or electron microscopy (Khurana *et*

al., 1990b). The virus could also be detected in the single viruliferous thrips by dot-ELISA. The best method for virus detection is by whole mount immunofluorescent staining (T. Nagata and D. Peters, 1999. Pers. comm.). Can be biologically detected on test plants like Cowpea C-152, *Nicotiana benthamiana*, on *N.occidentalis*, *N.tabacum* cultivars Samsum and White Burley giving large, slightly sunken necrotic local lesions/systemic necrosis. Tospoviruses are highly unstable *in vitro*. Reliable infection has been obtained using a buffer of 0.02M phosphate buffered saline pH 7.4 and 2% polyvinylpyrrolidone (PVP, MW 10000). Detection through nucleic acid probes (Huguenot *et al.*, 1990) and RT-PCR is reliable (Adam *et al.*, 1996; Mumford *et al.*, 1996).

6.5.2 Yellow mosaic/leaf curl geminiviruses

Four geminiviruses, namely, potato deforming mosaic (PDMV), yellow mosaic (PYMV), *Solanum* apical leaf curl (SALCV) occurring in South America while beet curly top (BCTV) gemini virus known to occur in Middle East and Americas are well documented. Yet another gemini virus causing potato apical leaf curl has only recently been recorded from India.

The potato yellow mosaic virus recorded from Venezuela is characterized by causing bright yellow mosaic, leaf distortion and dwarfing. It also induces appreciable yield loss. The virus has narrow host range limited to potato, tomato and some *Solanum* spp. It is readily transmitted by tobacco white fly and is easily detected through ELISA. It has typical geminate (bisegmented) virions measuring 18-20 nm in dia.Another important white fly transmitted geminivirus causes deforming mosaic on potato in Brazil. Yet another similar and severe on potatoes is Solanum apical leaf curling begomo-virus is known from South America. It is also transmitted through white flies. Similarly the beet curly top virus is pre-dominant in the Middle East. Based on the DNA sequence analysis, a number of geminiviruses infecting tomato from Caribbean Islands have been found close to potato yellow mosaic virus.

A recently observed apical leaf curl virus on potatoes in India is also readily transmitted through white flies. The virions size, however, is bigger *i.e.* 17-28nm in dia. It is rather serious due to early infection and high incidence of the virus, hence the yield losses are significant, well over 20-25%.

7. Interaction among viruses and other pathogens

Previous infection of either PVS and/or PVM can reduce the number of PVX and PVY infected tubers if the plants get successively infected by either or both of them regardless of host age (Pietrak, 1981). On the other hand, in case of combined infection of PVX and PVY, only the PVX multiplication/concentration is enhanced (Garg and Khurana, 1989-90, unpublished) causing severe disease symptoms and significant yield losses (Singh and Khurana, 1995). Potato aucuba mosaic virus is aphid transmitted only from plants having combined infection of PVY/PVA. Under experimental conditions, PSTVd is also acquired and transmitted by aphid, *Myzus persicae* but only from plants co-infected with PLRV *i.e.* involving heterologous encapsidation of the viroid in the virus particles (Querci *et al.,* 1997).

High incidence of PVS was observed to be responsible for breaking the late blight resistance of important cultivars like Kufri Jyoti (Singh *et al.*, 1992a; Khurana, 1992). Infection of potato plants with viruses X, S, Y and M and also leafroll renders them less susceptible to both late and early blights (Muller and Munro, 1961; Nagaich and Prasad, 1970; Pietkiewiczs, 1975, Kalra *et al.,* 1989 and 1992). On the other hand, various other reports indicate that PVY and latent/mild mosaics predispose potato plants to both late and early blights (Khurana and Raychaudhuri, 1988). Similarly, late blight was found to be acute on PLRV infected foliage. Komkov (1975) also reported less infection of PVX or PVM infected potatoes to black scurf (*Rhizoctonia solani*).

8. Epidemiology

The natural mode of transmission of most potato viruses is through the infected seed tubers, more so on cutting of tubers into pieces and/or mechanical injury of tubers, sprouts, foliage, etc. This is most common in case of contagious viruses (PVX,S) and viroid (PSTVd) which easily spread through workers' contaminated hands, clothes, implements, farm machinery, seed trays, etc.

In case of severe mosaic (PVY) and leafroll (PLRV), the aphids, mainly *Myzus persicae* and *Aphis gossypii*, act as vectors and spread them both far and within the fields. A number of other natural virus-vector sources are the ground keeper (volunteer/self sown potatoes), many weeds and also the cultivated crops/ornamental plants in the vicinity of the potato fields (Singh, 1988b). But for potato aucuba mosaic virus, both mechanical/contact and aphid transmission are important yet the latter occurs only either if the virus source has combined infection of PVY, PVA and PAMV or if vector aphids have had acquisition on plants infected with some potyvirus (Kassanis and Govier, 1971). AMV (Calico) is readily transmissible through various aphid species but not at all common in new Indian potato varieties except when vector aphids migrate from alfalfa fields to neighbouring potato fields (Khurana and Garg, 1992).

PVX infection spreads rather rapidly (upto 50%) in uncared crops raised from the seed successively for 3-4 years (Khurana and Singh, 1988). Like PVX, PVS is also contagious hence ubiquitous in occurrence. Some strains of PVS may also spread non-persistently through aphids (*M. persicae*). PVS being contagious and latent, in almost all cultivars under diverse environments occurs as most frequent natural contaminant along with PVX, and/or PVY and PVM.

PSTVd is readily transmitted through pollen and true potato seed (Singh and Fernow, 1981). PSTVd being contagious and highly stable also spreads through contaminated seed tubers, farm machinery and tools. About 50% transmission of PSTVd occurs through contact of bruised sprouts while 80-100% infection was recorded on rubbing of healthy and diseased plants.

A number of weeds harbouring the viruses and/or aphids seem to have epidemiological significance by way of help in over-wintering aphids and consequent virus spread (Singh and Boiteau, 1988; Turl (1983) reported that after severe winters, the vector populations and consequent incidence of virus(es) were lower in Scotland.

In the NW/NE plains of India M. persicae begins to appear on potato crop from end of November to beginning of December. The aphid population builds up from mid-

December onward and crosses the critical limit of 20 aphids/100 compound leaves by 25th December to 10th January in NW/NE plains, respectively (Chaudhuri, 1955; Nagaich *et al.*, 1969; Singh *et al.*, 1982).

M. persicae acts as the potential vector followed by *A. gossypii* in spreading both PVY and PLRV during autumn and spring seasons. Their activity is higher in spring and consequently the virus incidence is higher maximum being in cv. Kufri Sindhuri followed by Kufri Chandramukhi and least in Kufri Jyoti (Singh *et al.*, 1981). This is correlated to optimum weather (temperature) for aphid build up and their movement/activity (Singh *et al*., 1981, 1982, 1984a). Further, a combination of high humidity (80-90%) and moderate temperature (25-30°C) increase virus transmission by aphids but the transmission of these viruses is almost half when RH is low (50%) (Singh *et al*., 1988).

Usually the virus infected plants do not recover and the virus(es) reach all parts of the plant except the true potato seed (TPS) which is used for developing new varieties. Nevertheless, some uncommon potato viruses get transmitted through the TPS, viz. APLV, PVT, TRSV-ca, and Arracacha virus-B (AVB-oca). Besides, potato yellowing virus (PYV) has also been suspected to be seed borne. PSTVd is most readily true seed borne (Singh, 1970; Hooker, 1981; Khurana, 1990b). Despite the existence of a band of above mentioned true seed borne viruses, they do not seem to have spread far and wide beyond their probable site of origin (Jones, 1983). However, with the large scale international exchange of germplasm (both tubers and true seeds) in the recent past, PSTVd has spread over to far and wide areas (Salazar, 1989; Khurana, 1990a, 1990c).

9. General control

Because of high economic importance of the potato viral diseases it is helpful to manage them for better crop production by preventing viruses from infecting the seed stocks. Effective management practices for potato viruses include quarantine, sanitation, eradication, avoiding vectors, seed certification, resistance breeding, etc. They are briefly touched below. An Integrated Schedule for Management of viruses right is also given (Table 10).

9.1 Quarantine

The quarantine regulations exist for import of potato seed tubers and even TPS, *in vitro* plants etc. not only in India but in most developed countries. Quarantine testing and rejection of infected stocks has helped prevent entry of many potato viruses such as Andean potato latent, tobacco ringspot, potato virus T[*], viroid (PSVTd)[*], etc. (Singh and Khurana, 1993). The table 9 summarises biological/biophysical characteristics to help detection/diagnosis of the endemic viruses under quarantine.

9.2 Sanitation

Contagious viruses like PVX, S and viroid (PSTVd) spread readily both in stores and fields through contaminated farm machinery/tools, hands, clothes of workers. Use of

either high pressure steam or disinfectants help clean the equipments and stores just prior to the crop harvest. Use of chemicals like 3% trisodium phosphate, or household bleach (sodium hypochlorite) solution diluted 1:50 can be used for inactivating viruses on hands, tools, other surfaces. It is however important to first clean or remove the dust etc. An isolation of 10m between fields of seed and ware crops, starting seed crop with reliable quality/healthy seed and following proper sanitation/disinfection procedures greatly check any chances of the spread of virus/viroid in the seed crop (Banttari *et al.*, 1978, 1993; Upreti *et al.*, 1979).

9.3 Meristem tip culture

Viruses have to be eradicated if they are to continue in commercial production. Virus elimination can be achieved to a certain extent by heat treatment of tubers (Kassanis, 1950; Thirumalachar, 1954). When no virus-free stocks are available, for certain cvs/ hybrids, *e.g.* PLRV can be inactivated in potato tubers at/or around 37 °C for 8 weeks. Heat therapy of tubers may not be successful in many instances still it provides a valuable pretreatment of the infected material and enhances chances of getting virus-free stocks (Nyland and Goheen, 1969) though meristem tip culture. Morel and Martin's (1952) hypothesis that it would be possible to isolate the apical meristems from a sytemically infected plant and culture them *in vitro* to get virus free plants, was first applied to potato by Norris (1954). Virus free plants, regenerated from meristem tips, are genetically stable and yield true-to-type plants (Pennazio,1971; Quak, 1972; Dhingra *et al.*, 1992;Khurana *et al.*,1996). Elimination of virus(es) through meristem tip culture occurs possibly because:

i). Viruses move readily through the plant's vascular system *i.e.* absent in the non differentiated meristematic tissue.

ii). High metabolic activity in the actively dividing meristem cells does not allow virus replication.

iii). High endogenous auxin level in shoot apices may also inhibit the virus replication and the virus inactivating system, if any, have a higher activity in the meristems (Wang and Hu, 1982; Khurana *et al.*, 1996).

For details about the culture medium etc, the readers may refer to relevant literature (Slack and Tufford, 1995;Khurana and Sane, 1998). Many other factors also influence the meristem culture and help in virus elimination (Fig. 1). Generally meristems smaller than 0.1mm fail to grow while the longer ones continue with the virus. Meristematic domes with one or two leaf primordia measuring 0.2-0.3mm generally give higher percentage of virus free plantlets. Success of virus elimination depends much on the cultivar and the virus(es) involved; seasonal variations, viz. meristems excised in spring and early summer rooted easily than later in autumn etc.

Multiplication rate of potato meristem tips is about fifty fold when the virus free meristems are induced to produce multiple shoots in shake culture (Roca *et al.*, 1978). Plantlets thus regenerated can be multiplied further through nodal cuttings. It is important to note that meristem culture can be achieved even on an illuminated shelf in a clean room at most ambient temperatures (Dhingra *et al.*, 1992; Slack and Tufford, 1995; Khurana and Sane, 1998). Different light regimes do not help rooting yet leaf and

root development were better under high light intensity. Potato meristems taken from sprouts developed better under constant light.

9.4 Thermotherapy

Heat treatment of the explants reduces the virus concentration in the infected plant hence there are better chances of recovery of virus free plants (Stace-Smith, 1985). Different viruses have a varying response in their sensitivity to heat, viz. PVY and PVA are easily eliminated at 36/39 °C (Quak, 1977). Viroid PSTVd on the other hand, is eliminated after chilling at 5-6 C for 8-12 weeks treatment (Lizarraga *et al.*, 1980). The plant age, treatment duration and season(s) strongly influence the survival of infected

Virus Elimination

```
Infected Potato tuber/Plant
        │
        ▼
Sprout/Shoot-tip culture
        │
        ▼
    Virus Test ──(- ve)──►  Virus-free
        │                    starting
      (+ ve)                 material for
        │                    potato seed
        ▼                    production
Thermo-/Chemotherapy + Meristem culture       ▲
        │                                   (- ve)
        ▼                                     │
    Mericlones ──────────►  Virus Test
```
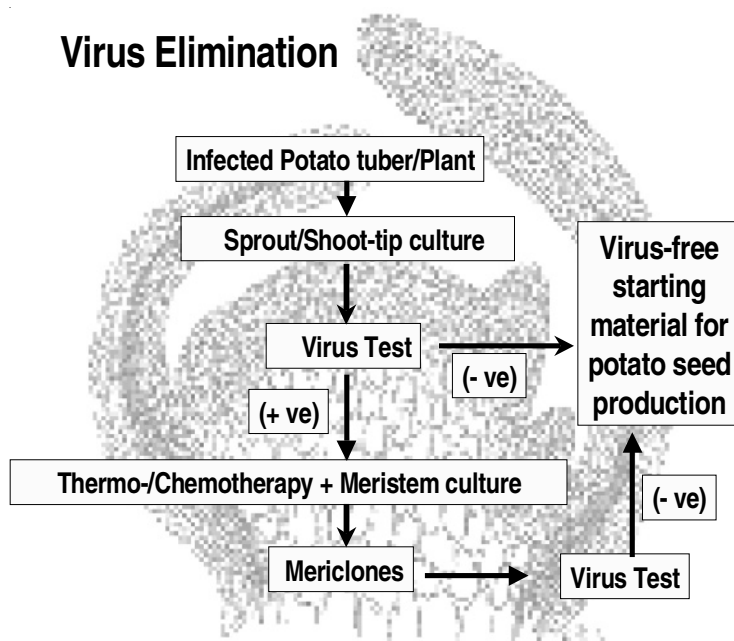
Figure 1: Outline of the sequence of events for obtaining virus free mericlones and their being maintained in vitro as the prenucleus or buffer seed stocks

plants upon heat exposure. Normally, well established plants shall be treated. Heat treatment invokes temporary damage or abnormalities of colour and shape of foliage and plant growth (Nyland and Goheen, 1969). These changes may not be important since the plants start turn normal a few weeks after the heat treatment is over.

Thermotherapy prior to meristem culture helps in elimination of the viruses otherwise difficult to eliminate. Lozoya-Saldana and Dawson (1982) compared the effect of constant temperature (37 °C) with alternating temperature regimes to obtain PVS

free plants. Temperatures fluctuating between 35 and 43 °C are most favourable for plant survival for several months than 38 °C constant (Khurana *et al.*, 1996; Khurana and Sane, 1998). Preconditioning of plants at 27-35 °C for a few days to one week, prior to treatment at 38 °C is helpful. In our experience 20-24 weeks treatment of 16h day at 36 C and 8h night at 29 °C is ideal for eradication of PVX and PVS and yet plants remain in sound health. The temperature and length of treatment vary with the heat tolerance of the cultivar. Most potato cultivars can withstand 37 °C up to a few weeks.

Different potato viruses have been listed hereafter in order of increasing difficulty of their eradication, viz. PLRV, PVA, PVY, Aucuba mosaic, PVX, PVM, PVS and PSTVd. In fact PVS and PSTVd are the most difficult to eradicate whereas PVA and PVY are often eliminated by meristem culture alone without prior heat treatment. It has been observed that PVA and PVY were eliminated from 85-90% of the meristem cultures while

Table 9: Field tolerance (no. of affected plants in %) for basic and certified seed in India, as compared to four developed countries

Disease(s)	Seed grade	India[2]	Canada[1]	France[1,3]	Holland[1,3]	UK[2]
Severe mosaic	BS(FS)*	1.0	0.25	0.33	0.09	0.1
leafroll etc.	CS	3.0	1.0	1.0	0.25	2.0
Mild mosaic	BS	0.5	0.25	0.33	0.09	0.5
	CS	1.0	1.0	1.0	2.0	5.0
Ring/Brown rot**	BS	None	0.1	0.5	None	0.5
	CS	-do-	2.0	1.0	5 plants/ha	2.0
Varietal mixture	BS	0.05	0.1	0.1	0	0.05
	CS	0.10	0.1	0.2	0	0.5

1,2.Three/two official inspections for BS (basic seed) and two for CS (certified seed).
3.The data presented for basic and certified seed concern those for the grades E and A respectively (grades E and A are generally marketed)*(FS) = In India Breeder's seed from CPRI is multiplied on the State Govt. Farms and given over as foundation seed.
**Ring rot is not found in India. Instead brown rot/bacterial wilt is a problem in certain pockets (Deccan plateau, mid hills, etc.

PVX and PVS, being stable, were eliminated from less than 10%. Some strains of PVA are also resistant for virus elimination through meristem culture as PVA persisted in all (39/39) plantlets developed from buds that were subjected to 37 °C for 12-20 days. Both PVA and PVS even persisted in plants that were freed from PVX (Gregorini and Lorenzi, 1974).

Recovery of virus free plants upon thermotherapy largely depends on the meristem size than the duration of heat treatment (Zaklukicwicz, 1983; Sajid *et al.*, 1986). The number of virus particles was directly proportional to the increasing size of the meristem (Faccioli *et al*., 1982, 1988). Heat treatment of sprouts on tubers before meristem culture gave rise to 16 out of 18 PVS free plantlets. Combination of meristem tip culture and thermotherapy was very effective in eradicating PVX (Dhingra *et al*., 1988).As already mentioned, PSTVd is the most stable and difficult pathogen to eradicate from potato cultivars. Growing potato plants at lower temperatures reduces viroid replica-

tion. Lizarraga *et al*.(1980) successfully eradicated a severe strain of PSTVd from a potato clone by growing viroid infected plant at 5-8 °C for six months followed by meristem tip culture. Prolonged cold treatment, however, severely damages a large number of meristem tips.

9.5 Chemotherapy

Use of antiviral agents either given to the infected plant before bud excision or incorporated into the culture medium, help decrease the virus concentration. Besides, the use of different growth promoting substances in the *in vitro* culture medium can also help virus elimination (Quak, 1961).

PVS is eliminated when the mother plants were first treated with antimetabolites (Quak, 1961) but incorporation of riboside in the medium greatly helps in eradication of PVX, PVY, PVM, PLRV (Cassells and Long, 1982; Dhingra *et al*., 1987; Khurana *et al*., 1996). The synthetic riboside, Ribavirin[*] is very effective when used *in vitro* as it resulted in elimination of one or more potato viruses. It is the best to use the heat treatment of *in vitro* plants and/or supplement the medium with the antiviral agent(s). Kim *et al.* (1996) synergistically eradicated PVS by a combination of thermo-(33°C) and chemotherapy (20ppm ribavirin) of intact nodal shoots of potato. The Fig. 1 outlines the sequence of events for obtaining virus free mericlones and their being maintained in vitro as the prenucleus or buffer seed stocks.

9.6 Therapy for virus/viroid eradication

9.6.1 Viruses

A combination of thermo/chemotherapy and apical meristem is the most efficient way for eradication of most of the viruses. If 10 plantlets are used, more than 50% should test virus-free (above 80% virus-freedom can be expected for most viruses) following the combined therapy. The level of success depends on the virus/isolate/ *Solanum* spp./cultivar (Kassanis and Varma, 1967; Dhingra *et al*., 1987,1988; Griffiths *et al*., 1990; Slack and Tufford, 1995; Khurana *et al*., 1996;Khurana and Sane, 1998).

Plantlets are established on Murashige and Skoog's (1962) medium at 22-25°C, giving 16 h light + 8 h dark (light intensity 75 micromol $M^{-2} S^{-1}$ is ideal). Nodal cuttings are subcultured on media amended with 20 mg/1 ribavirin (Virazole) and after establishment heat treatment is initiated. A four hour alternating 35°C light and 31°C dark cycle for four weeks is recommended (reduced light of 25 micromol $M^{-2} S^{-1}$ has been found most effective). For leafroll virus, alternating temperatures of 40°C and 25°C are better giving 100% elimination even without Virazole.

Plantlets are also tested by ELISA before and after therapy as this approach permits quantitative monitoring of reduction in the virus concentration and/or its elimination. Virus- free plantlets are selected and apical nodes excised to culture them onto MS medium. After 4-6 weeks, top parts of each such plant are retested. If virus negative (test after subculturing for retention), they are grown in a glasshouse or growth room and again subjected to testing (two three times over a complete vegetative cycle).

9.6.2 Viroid

PSTVd elimination is not easy but possible through low temperature treatment of *in vitro* plantlets (6-8°C for 3-4 months) (Lizarraga *et al*., 1980). Follow the protocol below:

Plantlets are first established on Murashige and Skoog's (1962) medium at 22-25°C, 16 h light + 8 h dark (light intensity 75 micromol $M^{-2} S^{-1}$ is ideal) for 2-3 weeks. Then the temperature is reduced to 6-8°C and the plantlets incubated for 3-4 months, meristems removed (with no more than one leaf primordium) and cultured onto MS medium and incubated at 22-25°C, 16 h light. The top part of each plantlet is tested for PSTVd after 4-6 weeks. If viroid negative, the base part is allowed to regrow and tested again after another 4 weeks. Strict aseptic conditions must be maintained at each step and between cutting of different plantlets. If still negative, plantlets are allowed to regrow (after taking subcultures for retention and storage) and then the plants are grown under containment (in a glasshouse or growth room) and retested several weeks later for latent or low concentration of PSTVd. The plants must be subjected to repeated testing for viroid, preferably through NASH, during summer months when viroid multiplication is higher.

9.7 Tuber Indexing and Clonal multiplication

Indexing of seed tubers for viruses is done to discard the infected ones. Detection of viruses is affected by many factors like temperature, physiological stage of tubers (dormant or sprouted) and the technique employed (Singh *et al*., 1984b, 1987). ELISA detection of PVX and PVS was higher in physiologically advanced or non-dormant material, when grown and tested 12 weeks after harvest at 6.0-29.5°C than the dormant stocks of same age at 12.5-28.5°C (Singh and Somerville, 1983). Verma *et al*. (1998) however found an erratic trend of the incidence of different viruses during tuber index-ing over a span of five years.

The clonal selection from stage I instead of stage III resulted in an overall improvement in the disease freedom of seed stocks being multiplied (Singh *et al*., 1984). Thorough roguing of seed crop helps an effective check of the natural spread of vi-ruses etc. (Upreti *et al*., 1987). ELISA is now routinely employed for tuber indexing. It has replaced biological, chloroplast agglutination testing (Singh *et al*., 1994; Khurana, 1990a) that required more space/time and labour as well as allowed some latent infec-tions of PVS/M, PVY etc. going unchecked.

9.8 Avoidance of Vectors

Several potato viruses (viz. PVY, PLRV, PVA, PVM) spread mainly through one or more aphid vectors. Therefore, the seed crop in NW Indian plains is raised only in aphid-free periods or locations in the designated seed producing areas through the Seed Plot Technique (Pushkarnath, 1967; Khurana *et al*., 2000). Growing of seed crop during aphid-free period (October-December/January) involves use of healthy seed, applica-tion of systemic insecticides, field inspection for roguing all infected/offtype plants, and dehaulming the crop as soon as the aphids cross the critical limit of 20 aphids/100

compound leaves (Fig 2).

Vashisth *et al*. (1981); Verma and Vashisth (1985) found that incidence of the viral diseases was maintained within permissible limit of 1 per cent for several years if the haulms were cut as soon as the aphid build up started reaching critical limit (Verma *et al*., 1998). Singh *et al*. (1999) and Chaudhari *et al.* (2000) were able to effectively manage PSND by manipulating planting dates and use of systemic insecticides for managing thrips.

9.9 Control of aphids

Normally aphids do not attain pest status on potato crop yet their vectorial activity for
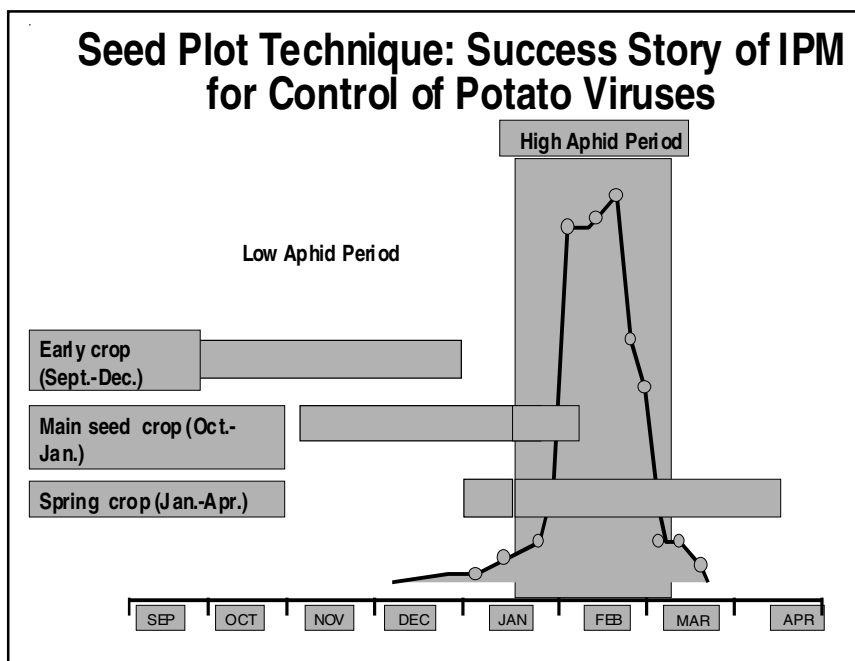
Figure 2: Seed plot technique to understand aphid free period for growing seed crop in north western Indian plains.

viruses in seed crop calls for their chemical control. Even then insecticides can help to reduce aphid population buildup which in turn prevents only persistently spread leafroll virus. Systemic soil insecticides are applied at planting while foliar sprays of certain contact cum systemic insecticides help checking aphid population on seed crop but only for a short period until dehaulming. Therefore, other cultural practices, use of aphid resistant varieties and essentials of integrated management for viruses are recommended.

9.10 Forecasting for viruses

Large number of interactions occur among host plants, viruses, insect vectors and the

environment hence they make the situation very complicated. Naturally, the environ-
ment has the maximum influence among all the factors, viz. the temperature directly
affects the vector behaviour and also virus multiplication and its translocation within
the host. Forecasting for the aphid borne viral diseases has been tried mainly in case of
PVY and PLRV especially in N.Europe (Sweden etc) based on the relationship between
aphid migration and spread of PVY which has been studied by exposing bait plants to
the aphid vectors in the fields (Sigvald, 1998). Winged aphids have also been collected
and percentage of the viruliferous ones determined by using biological test plants
under glasshouse condition (de Bokx and Pirone, 1984; Harrington *et al.,* 1986). Great
variations occur in nature in virus transmission efficiency of different aphid species
(Khurana and Singh, 1995). For developing some forecasting methods for potato vi-
ruses, following variables have been used:

Number of alate aphids and their vector efficiency, the time of aphid migration in
relation to plant growth, and also the availability of virus source (Sigvald, 1985, 1986)
Simulation models have also been used to describe the epidemiology of non-persis-
tently transmitted viruses taking care of aphid behaviour in the epidemiology of PVY.
The data from field experiments were used to calculate the latency after aphid inocula-
tion of PVY and before an infected plant can serve as the virus source in field condi-
tions. In southern Sweden, aphid migration normally occurred in July and potato plants
became infected with PVY. It took almost three weeks for the virus(es) infecting the
daughter tubers (Sigvald, 1986). The studies indicated that mainly the alates (and not
apterae) were responsible for spread of PVY (Sigvald, 1990).

In another simulation model, relationship between important variables and pa-
rameters like plant disease-vector dynamics at the level of individual field, was worked
out. Some of the most important variables are alates as virus vectors, PVY infected
plants as virus sources, the susceptibility of the crop based on the planting date, date
of haulm killing and PVY infected daughter tubers. The model output successfully
predicts the extent to which the proportion of progeny tubers infected with PVY will
increase during late summer. The present model differs from the earlier one (Sigvald,
1998) in adding/changing the following parameters/ variables:

Newly PVY infected plants, totally (PVY) infected potato plants, spread of PVY,
latent period, cultivar susceptibility, date of haulm killing, roguing of PVY infected
plants and possible risk of virus spread from off the field virus sources.

The model output and the real data showed high degree of agreement. How-
ever, changing the proportion of PVY diseased plants serving as a virus source greatly
influenced the proportion of potato tubers infected with PVY: for example from 0.1 to
0.5% caused the latter to increase from 20 to 60%. Besides, the pattern of aphid migra-
tion differs over years and in regions. Therefore, such changes can influence the model
output in case of aphid species but the variation for *Myzus persicae* was slight. The
predicted values matched well with actual data from seed potato fields for PVY infected
tubers and confirmation of virus infection in tubers under glasshouse conditions. In
years, with low vector numbers (1977), most of the seed potato fields had low incidence
of PVY (2%) as predicted, and even if vector population was high, viz. 1976, the model
successfully predicted a high incidence of PVY (actual data = 93% v/s predicted inci-
dence of 96%), showing its high flexibility and accuracy.

Vector efficiency is determined by vector behaviour and its mobility hence models were tried to predict the relationship between vector activity and the incidence of vector PVY, viz. Van Harten (1983) calculated vector pressure from Relative Efficiency Factors (REF) of related aphids species prevalent in the area.

Gabriel (1961, 1981) calculated contamination index 'W' from the number of aphids trapped or observed on 100 potato leaves over 80 days and the efficiency with which tubers got infected with PVY. REF values assigned to different aphid species vary and depend on vector efficiency, relative abundance, time of migration, age of potato crop, etc. Thus, *Brachycaudas helichrysi* had a higher REF than *M. persicae* at Harpenden during 1984 due to higher efficiency and larger number (Harrington *et al.*,1986); *Rhopalosiphum padi* was more important in Sweden (Sigvald, 1987) and the Netherlands (de Bokx and Pirone 1990) due to early migration in larger numbers when the potato crop is young and more susceptible to PVY.

Occurrence of young PVY infected source plants early in the season, when alates are moving about, lead to greater risk for PVY infection in the progeny tubers than if alates migrate late in the season because of mature plant resistance. The simulation model of Sigvald (1998) can also be used in other countries despite large variations in virus spread during different years. When a high virus incidence is predicted *i.e.* a field may get rejected, despite early dehaulming, the crop produce may be sold for ware purpose without dehaulming the crop.

Aphid migration can also be predicted by the use of suction traps, 12 m above ground level (Wiktelius, 1982). A close correlation between suction trap counts and virus spread was observed in Sweden when the incidence of virus source plants, potato variety and crop age (plant resistance) were also considered.

Attempts have also been made on similar lines to predict the incidence of PLRV. A link between the number of aphids trapped in suction trap, and the winter temperature, spring migration of aphids and spread of PLRV in the field was observed (Sparrow, 1976). Winter temperature during February and dates of first catches of an holocyclic specimens of *M. persicae* had a positive correlation (Turl, 1980) hence it was used to forecast the spring migration of *M. persicae* (Turl, 1983) as well as to advise farmers whether or not to use the pesticide for aphid control while planting seed crop.

Delayed planting (beyond 25th October) and application of systemic insecticide helped in preventing the crop from thrips-borne stem necrosis in Central/Western India (Khurana *et al.*, 1997).

Based on the aphid freedom in growing period for seed crop, the growers in areas fit for seed production (and also in secondary seed areas are able to maintain home grown potato seed stocks for 3-5 years following the basic essentials of seed plot technique (Verma *et al.*, 1998). The farmers need to initially multiplying fresh seed in two stages *i.e.* firstly on 1/25th and then on to 1/5th area of the ware crop to be planted.

9.11 Seed Certification

Diseased seed (tubers) used for raising the crop is the main cause for astronomical spread of potato viruses. The best method for containing potato viruses is to remove all infected tubers, *i.e.*, multiply only the selected virus-free seed in a manner that

minimum reinfection occurs upon field operations. Healthy seed stocks may be multiplied into 2-3 stages to produce foundation and certified seed. In India three inspections of crop in fields in the hills and two in the plains are mandatory to certify the seed health and finally after the harvest, for grading of seed (Khurana, 1992,1998). Indian potato seed certification standards are quite stringent and comparable to that in developed countries (Table 9; Khurana and Garg, 1998; Khurana 1999).

9.12 Resistant and Transgenic Varieties

Virus resistance in potato varieties is the most practical approach for managing viral diseases. But not many varieties are really available, at least in India (Khurana, 1999). Many old exotic, mostly cultivars, namely, Craig's Defiance, Up-to-date, Great Scot, Saco, Tawa, Katahdin, having virus resistance, are no more in cultivation. Sources of resistance to important potato viruses are wide spread in *S. tuberosum* spp. *andigena, S.berthaultii, S.acaule*, etc. Multiple disease resistant lines were derived from *S.tuberosum* x *S.andigena* crosses (Nagaich and Singh,1982) having resistance to both PVX and PVY. Varieties like Kufri Ashoka, Kufri Badshah, Kufri Jyoti, Kufri Jawahar, Kufri Kanchan, Kufri Lalima, Kufri Pukhraj, Kufri Sindhuri, etc. have resistance/tolerance to one or the other viruses and have slow rate of degeneration (Singh *et al.*, 1994).

Resistance to vector-aphid is also there in (Indian) CPRI potato cultivars: Kufri Jyoti, Kufri Shakti, Kufri Badshah, SLB Z/405-a (Singh *et al.*, 1983) Resistance against the tospovirus causing stem necrosis and/or vector thrips has been found in a number of genotypes (Singh, *et al.*, 1997a).

In developing countries, where farmers cannot afford to buy or do not get enough seed, development of cultivars with resistance to viruses X,S,Y and leafroll, need to be given priority. Breeding for virus tolerance is not desirable in view of the difficulty it can cause for seed production still it has a practical value for the areas where seed production is not effective and frequent replacement of seed stocks is not easy. The preferred forms of resistance is either hypersensitivity or extreme resistance/immunity to the viruses and vectors (Beekman, 1987; Banttari *et al.*, 1993). It has been rather easy to breed varieties with combined resistance to PVX and PVY and PVA but not leafroll virus. Resistance and immunity to both PVX and PVY (+PVA) are controlled by single dominant genes (Beekman, 1987). In contrast it is understood to be polygenic for PLRV and both additive and non-additive gene effects are involved. However, PLRV resistance is ineffective in clones susceptible to PVX and PVY (Jayasinghe *et al.*, 1989). High degree of PLRV resistance has been detected in *S.brevidens* accessions against virus multiplication, and for vector aphids in *S.tuberosum* x *S.berthaultii* hybrids bearing glandular trichomes. Cultivars such as Katahdin, Penobscot, remained free from PLRV despite abundant aphid colonization while some crosses with low aphid preference were found to be severely infected by the virus.

A highly productive crop like potato is always at risk from several biotic and abiotic stresses and many transgenic cultivars have been produced to take 'care' of majority of biotic and abiotic constraints (Ghislain and Golmirzaie, 1998) esp for the viruses. Therefore, initial efforts in potato genetic engineering focussed at imparting virus resistance in cultivars of choice (Ghosh *et al.,* 2000, 2002; Khurana and Chakraborty,

2000).

One of the most popular transgenic strategy to control virus(es) in potato is through coat protein (CP)-mediated resistance (Beachy *et al.*, 1990). The CP protein, when expressed constitutively in a transgenic plant, interferes with the virion disassembly, multiplication, expression and spread of freshly infected virus (Clark *et al.*, 1995; Baulcombe, 1996). It is not even strain specific. One CP gene can also provide protection against other related viruses, having more than 60% homology in their CP gene sequences (Stark and Beachy 1989; Mandahar and Khurana, 1998). Hoekema *et al* (1989) who first field tested transgenic potato expressing the CP gene of PVX for the European varieties Bintje and Escort, observed significant reduction in PVX incidence in tubers from mechanically field-infected transgenic lines (Jongedijk *et al.,* 1992).

Similarly, Malnoe *et al.* (1994) evaluated the resistance of potato variety Bintje expressing CP gene of PVY to natural spread of two PVY strains, Y^N and Y^O. Potato plants expressing the CP gene of PVY^N exhibited complete resistance to PVY^N and some degree of protection to PVY^o under natural aphid transmission in the field. Kaniewski et al (1990) also observed resistance to mechanical infection of PVX and PVY in potato plants expressing the CP genes of both PVX and PVY. Transgenic potato plants expressing CP gene of potato leafroll luteovirus (PLRV) have also been developed (Barker *et al.*, 1992; Kawchuk *et al.*, 1990; Brown *et al.*, 1995), showing high levels of virus resistance to aphid inoculations.

The major drawback of CP-mediated resistance is that the resistance is not functional in most cases when plants are inoculated either with naked viral RNA or with a high concentration of intact virus. Moreover, coat protein has the ability to non-specifically bind to other RNAs including the host's cellular RNAs. Furthermore, a chance recombination can occur between transgenically expressed CP RNA and other viral RNAs present in the same host (Allison *et al.*, 1996). Commercial release of CP-engineered potato is still withheld, probably because of such reasons. Nevertheless, the success of CP-mediated potato transgenics is so far the best documented approach in plant genetic engineering.

9.13 Movement protein (MP)-mediated resistance

Knowledge of movement protein (MP), helping in cell to cell movement of plant viruses, has also provided suitable transgenic strategies, viz. a defective MP competes with its wild type during virus infection process. Once the viral genome is blocked by dysfunctional MP, intra- and inter-cellular movement of the homologus virus (and other viruses) will be curtailed (Malyshenko *et al.*, 1993). The mutant MP of TMV also mediates resistance against potex, cucumo and tobraviruses (Cooper *et al.*, 1995) or transgenic potato plants expressing a movement protein of PLRV show resistance to PVX, PVY as well (Tacke *et al.*, 1996).

Viral infection can also be contained through exploitation of either antisense RNA (Nicholson, 1996) or the catalytic RNAs like ribozymes. However, *in vitro* success of ribozyme in virus degradation does not match with its *in vivo* performance hence requires refinement for field application. Antisense constructs targeted against the viral coat protein as well as replicase have been successfully used against PVX, PVS

and PLRV (Kawchuk *et al.*, 1991) inducing virus resistance almost at par with that by CP.

9.14 Homology dependent gene silencing

Resistance to plant viruses can also be achieved by transferring pathogen-derived untranslatable sequences that are not translated into any protein products inside the transgenic plant (Goodwin *et al.*, 1996) *i.e.* the viral genome has some sequence identity to the transgene (English *et al.*, 1996). The replicase-mediated resistance also has a similarity with such gene silencing. Transgenic Russet Burbank potato, expressing PLRV replicase gene, has been demonstrated to be field resistant against PLRV. Replicase-mediated resistance, however, is highly strain specific and essentially dependent on RNA sequence homology between the inoculated virus and the transgene.

9.15 Non-pathogen derived resistance

Transgenic potato clones expressing a gene isolated from pokeweed encoding a ribosome inactivating protein (RIP) have high degree of virus resistance (Lodge *et al.*, 1993). Similarly, a mammalian oligonucleotide synthetase gene into potato has been reported to confer extreme resistance to PVX in field grown plants (Truve *et al.*, 1993). Besides *Solanum acaule* has a dominant gene (Rx) conferring extreme resistance, almost approaching immunity. Once the resistance conferred by Rx is elicited by coat protein of the infecting virus, it can suppress replication of a completely unrelated virus(es) hence may take care of important potato viruses. Similarly, a dominant gene (Y) conferring immunity to PVY has also been identified in the tetraploid *Solanum tuberosum subsp. andigena* alone (Hamalainen *et al.*, 1997).

10. Integrated management of potato viruses

Due to the differences in mode of spread and perpetuation of viruses, different indirect and direct control measures have to be adopted. It is normally done through an integrated package (Table 10). Indirect measures of control of viruses and viroid are more important while direct control measures depend mainly upon cultivation of virus resistant varieties. The best policy for management is to prevent virus infection of seed crop/stocks. Reliable detection methods have a great significance in the production of high quality (virus free) seed potatoes. To achieve this goal, detailed information on various aspects about: (a) nature of virus; (b) the mode of transmission; (c) health standards of the planting materials, which may act as internal source of the virus spread in the crop; (d) weed hosts which may act as external sources of the viruses; and (e) factors affecting the build up of the vector and virus diseases must be available.

11. Conclusions

An attempt has been made to review the pertinent information on potato viruses and viral diseases, with special reference to subtropicalpotato cultivation as in India, along with the therapy and control measures. Detailed description of only a few important

viruses has been given while properties of all viruses infecting potatoes have been tabulated. There is a need for continuing intensified research on virus distribution and epidemiology; breeding for resistance either by conventional breeding or genetically modifying the available desired varieties. The seed technology is developing very fast

Table 10 : Schedule of integrated control of potato mosaic viruses after Khurana and Garg, 1992)

Control measures

1.	Previous Crop:	Inspection of seed production areas to ascertain the health standard of the seed crop; reject the fields in which incidence of mosaic is higher than the prescribed level. Kill the vines of seed crop at the prescribed date or even earlier as and when critical aphid level is reached and that there is no regrowth of vines.
		Destroy volunteer potato plants and weeds in or around the seed crop to eliminate possible reservoirs of viruses.
		Apply insecticides to keep the aphid vectors below the critical level.
		Use properly disinfected tools for the harvest of seed crop.
ii.	Between Crops:	Destroy potato volunteers.
iii.	Pre-plants:	Remove weed hosts of viruses and vectors and volunteer potato plants. Apply insecticides in the soil.
iv.	At Planting:	Maintain proper isolation of the seed crop from virus sources.
		Apply systemic insecticides to soil or tubers to control vectors.
		Ensure good sanitation measures during planting. Plant the best quality certified seed tubers. Avoid use of cut tubers as seed for seed crop. Plant seed crop at a specified period to avoid exposure of the crop to the vectors.
v.	Pre-emergence:	Apply pre-emergence herbicides.
vi.	Post-emergence:	Minimise chances of virus spread through farm machinery by controlling weeds with herbicides. Monitor the population of vectors. Apply insecticides when necessary to suppress vector. Rogue out the virus diseased plants as early as they are detected along with tubers, if any.
vii.	Pre-harvest:	Kill vines at a specific date or earlier if the vector population tends to reach critical level. Do not allow re-growth of the vines.
		Stop irrigation 10-15 days before harvest to allow skin curing.
viii.	Harvest and Storage :	Disinfect the tools before harvest. Sort out the seed tubers, grade and give them the required treatment and shade dry before cold storage. Use proper sanitation in storage. Control the sprout aphids in stores. Avoid rubbing of chitted/sprouted tubers in stores or seed trays while taking them to field.

hence the seed production agencies must keep pace by imparting required training to the personnel involved in virus detection and crop inspection to maintain high seed/ crop health standards.

Acknowledgements

12. References

Adam, G., Peters, D. and Goldbach, R.W. 1996. Serological comparison of tospovirus isolates using polyclonal and monoclonal antibodies. Acta_Horticulture, 431: 135-158.

Allison, R.F., Schneider, W.L. and Greene, A.E. 1996. Recombination in transgenic plants. Seminars in Virology, 7: 417-422.

Banttari, E.E., Anderson, N.A., Jevning, J. and Sushak, R. 1978. Reinfection rates and performance of disease tested potato seed stocks. In: "Proceedings Potato Research Plan and Report Conference/RRVPGA", East Grand Forks, Minnesota, 1978, 31 pp.

Banttari, E.E. and Franc, G.D. 1982. ELISA with single and combined antisera for viruses S and X in potato tubers and plants. American Potato Journal, 59:375-383.

Banttari, E.E. and Goodwin, P.H. 1985. Detection of potato viruses S, X and Y by ELISA on nitrocellulose membrane (dot-ELISA). Plant Disease, 69: 202-205.

Banttari, E.E., Clapper, D.L., Hu, S.P., Daws, K.M. and Khurana, S.M.Paul. 1991. Rapid magnetic microsphere enzyme immunoassay for potato virus X and potato leafroll virus. Phytopathology, 81: 1039-1042.

Banttari, E.E., Ellis, P.J. and Khurana, S. M. Paul. 1993. Management of diseases caused by viruses and virus like pathogens. In: Potato Health Management (ed. Rowe, R.C.) PAA and APS Press, St. Paul, MN (USA), pp. 127-133.

Banttari, E.E. and Khurana, S.M.Paul.1998. The Potato Viruses and their Management. In: Pathological Problems of Economic Crop Plants and their Management (ed. Khurana, S.M. Paul) Scientific Publishers Jodhpur (India). pp.489-509.

Barker, H., Reavy, B., Kumar, A., Webster, K.D. and Mayo, M.A. 1992. Restricted virus multiplication in potatoes transformed with the coat protein gene of potato leafroll luteovirus: similarities with a type of host gene-mediated resistance. Annals of Applied Biology, 120: 55-64.

Barker, H., Webster, K.D. and Reavy, R. 1993. Detection of potato virus Y in potato tubers : a comparison of polymerase chain reaction and enzyme-linked immunosorbent assay. Potato Research, 36:13-20.

Baulcombe, D.C. 1996. Mechanisms of pathogen-derived resistance to viruses in transgenic plants. The Plant Cell, 8: 1833-1844.

Baulcombe, D.C. and Fernandez-Northocote, E.N. 1988. Detection of strains of potato virus X and of a broad spectrum of potato virus Y isolates by nucleic acid spot hybridization (NASH). Plant Disease, 72 : 307-309.

Beachy, R.N., Loesch-Fries, S. and Tumer, N.E.1990. Coat protein-mediated resistance against virus infection. Annual Review Phytopathology, 28: 451-474.

Beekman, A.G.B. 1987. Breeding for resistance. In: Viruses of Potatoes and Seed Potato Production (eds. de Bokx, J.A. and Vanderwant, J.P.H.), PUDOC, Wageningen. pp 162-174.

Best, R.J. 1968. Tomato spotted wilt virus. Advances in Virus Research, 13: 65-146.

Bhat, I., Varma, A., Jain, R.K. and Khurana, S.M.Paul. 1997. Differentiation of potato virus Y strains by N-terminal serology and HPLC peptide profiling. Indian Phytopathology, 50: 89-96.

Brown, C.R., Smith, O.P., Damsteegt, V.D., Yang, C.P., Fox, L. and Thomas, P.E. 1995. Suppression of PLRV titer in transgenic Russet Burbank and Ranger Russet. American Potato Jour-

nal, 72: 589-597.

Browning, I.A., Burns, R., George, E.L. and Darling, M. 1995. Development and evaluation of ELISA assays incorporating monoclonal antibodies for the detection of potato A potyvirus. European Plant Protection Bulletin, 25: 259-268.

Casper, R. 1977. Detection of potato leafroll virus in potato and in *Physalis floridana* by enzyme linked immuno-sorbent assay (ELISA). Phytopathology Z, 90: 364-368.

Cassells, A.C. and Long, R.D. 1982. The elimination of potato viruses X,Y,S and M in meristem and explant cultures of potato in the presence of virazole. Potato Research, 25: 165-173.

Cerovska, N. and Filigarova, M. 1995. Specific detection of the Andean strain of potato virus S by monoclonal antibodies. Annals of Applied Biology, 127: 87-93.

Chaudhuri, R.P. 1955. Some aspects of insect transmission of plant viruses. Indian Journal of Entomology, 17:40-48.

Chaudhari, S.M., Khurana, S.M.Paul, Patel, P.K. and Patel, R.N. 2000. Management of potato stem necrosis disease through manipulation of planting dates and application of insecticides at Deesa. In: Potato, Global Research and Development, Vol.1(eds. Khurana S.M. Paul, Shekhawat, G.S., Singh, B.P. and Pandey, S.K.). Indian Potato Association, CPRI, Shimla, pp 393-397.

Clark, M.F. and Adams, A.N. 1977. Characteristics of the microplate method of enzyme linked immunosorbent assay for the detection of plant viruses. Journal of General Virology, 34:475-483.

Clark, W.G., Fitchen, J.H. and Beachy, R.N. 1995. Studies of coat protein-mediated resistance to TMV. Virology, 208: 485-491.

Cooper, B., Lapidot, M., Heick, M., Dodds, J.A. and Beachy, R.N. 1995. A defective movement protein of TMV in transgenic plants confers resistance to multiple viruses whereas the functional analog increases susceptibility. Virology, 206: 307-313.

de Bokx, J.A. (ed.) 1972. Viruses of Potatoes and Seed Potato Production, PUDOC, Wageningen, 233p.

de Bokx, J.A. and Pirone, P.G.M. 1984. Aphid trapping in potato fields in the Netherlands in relation to transmission of PVY[n]. Mededelingen Fac. Landbouw. Rijksuniversiteteit- Gent. 49/2b: 443-452.

de Bokx, J.A. and van der Want, J.P.H. (eds.) 1987. Viruses of Potatoes and Seed-potato Production. PUDOC, Wageningen, Netherlands. 259p.

de Bokx, J.A. and Pirone, P.G.M. 1990. Relative efficiency of a number of aphid species in the transmission of potato virus Y[o]-in the Netherlands. Netherlands Journal of Plant Pathology, 96:237-246.

Dedic, P. 1975. The effect of virus A (PVA) on yield in some potato varieties. Ochrana Rostlin. 11: 127-133.

Dhingra, M.K., Khurana, S.M.Paul and Lakhanpal, T.N. 1987. Effect of virazole and 2,4-D on the growth and potato virus X content of potato leaf callus. Journal of Indian Potato Association, 14:100-103.

Dhingra, M.K., Khurana, S.M. Paul, Lakhanpal, T.N. and Chandra, R.1988. Thermotherapy in potato tissue culture. Journal of Indian Potato Association, 15 :53-59.

Dhingra, M.K., Naik, P.S., Chandra, R. and Randhawa, G.J. 1992. Tissue culture techniques for potato health, conservation, micropropagation and improvement. Technical Bulletin No.39, CPRI, Shimla, 26p.

Ellis, P., Stace-Smith, R., Bowler, G. and Mackenzie, D.J. 1996. Production of monoclonal antibodies for detection and identification of strains of potato virus Y. Canadian Journal of Plant Pathology, 18:64-70.

English, J.J., Nueller, E.E. and Baulcombe, D.C. 1996. Suppression of virus accumulation in transgenic plants exhibiting silencing of nuclear genes. The Plant Cell, 8:179-188.

Faccioli, G. and Rubies-Autonell, C. 1982. PVX and PVY distribution in potato meristem HPS

and their eradication by the use of thermo-therapy and meristem tip culture. Phytopathology Z, 103:66-76.

Faccioli, G., Rubies-Autonell, C. and Resca, R. 1988. Potato leafroll virus distribution in potato meristem tips and production of virus-free plants. Potato Research, 31:511-520.

Foster, G.D. 1991. Molecular variation between the ordinary and Andean strain of potato virus S. Research in Virology, 142: 413-416.

Franc, G.K. and Banttari, E.E. 1986. Comparison of latex agglutination, enzyme-linked immunosorbent assay and indicator plants for detection of potato viruses S and X in potatoes. American Potato Journal, 63:357-362.

Fribourg, C.E. and Nakashima, J. 1974. An improved latex agglutination test for routine detection of potato viruses. Potato Research, 27:273-279.

Fribourg, C.E. and Nakashima, J. 1984. Characterization of a new potyvirus from potato. Phytopathology, 74:1363-1369.

Gabriel, W. 1961. La importance de certainers especes de pucerons pour la propagation des maladies a virus de la pomme de terre en Pologne. In: "Proceedings 4th Conference on Potato Virus Diseases, Braunschweig 1960, pp.126-137.

Gabriel, W. 1981. Essais d' amelioration de la previsin del'infection des tubercules des pommes de terre par la virus Y. Potato Research, 24:301-308.

Garg, I.D. 1987. Degeneration of potato varieties in western Maharashtra. Journal of Indian Potato Association, 14: 127-128.

Garg, I.D., Khurana, S.M.Paul and Singh, M.N. 1989. Immuno electron microscopy for the detection of potato leafroll virus in *Myzus persicae*. Journal of Aphidology, 3:196-200.

Garg, I.D. and Khurana, S.M.Paul. 1991. Protein-A supplemented immune electron microscopy for diagnosis of potato viruses X, S, Y and leafroll. In: Horticulture - New Technologies and Applications. (eds. Prakash, J. and Pierik, R.L.M.) Kluwer Academy Publication, Dordrecht. pp.329-336.

Garg, I.D. and Khurana, S.M. Paul. 1992. Factors influencing immune electron microscopy of flexuous potato viruses. Acta Virologica, 36:435-442.

Garg, I.D. and Khurana, S.M. Paul. 1993. Morphological changes in the flexuous potato viruses upon decoration in immunosorbent electron microscopy. Acta Virologica, 37:407-411.

Garg, I.D. and Khurana, S.M. Paul. 1994. ISEM: Dislodging of PVY/PVA virions upon incubation with antisera to PVS/PVM. In: "Potato: Present and Future" (eds. Shekhawat,G.S., Khurana, S.M. Paul and Chandra,R.). Indian Potato Association, Shimla, pp.325-327.

Garg, I.D., Singh, M.N., Khurana, S.M. Paul and Gopal, Jai. 1999. Virus analysis of indigenous potato germplasm collection. Journal of Indian Potato Association, 26:111-117.

Garg, I.D., Hegde, V. and Khurana, S.M. Paul. 2000. Optimal parameters for the immune electron microscopic diagnosis of potato viruses. In: "Potato, Global Research and Development", Vol.1 (eds. Khurana, S.M. Paul, Shekhawat, G.S., Singh, B.P. and Pandey, S.K.). Indian Potato Association, CPRI, Shimla, Vol.1, pp 405-412.

Garg, I.D., Khurana, S.M.Paul, Shiv Kumar and Lakra, B.S. 2001. Association of a geminivirus with potato Apical Leaf Curl in India and its immuno-electron microscopic detection. Journal of Indian Potato Association, 28:227-32.

Ghislain, M and Golmirzaie, A. 1998. Genetic engineering for potato improvement. In: Comprehensive Potato Biotechnology, (eds. Khurana, S.M. Paul, Ramesh,C.and Upadhya,M.). Malhotra Publication House, New Delhi. pp. 115-162.

Ghosh, S.B., Ussuf, K.K., Khurana, S.M. Paul and Mitra, R.K. 2000. The analysis of significance of DNA sequence of coat protein gene of an Indian isolate of potato virus Y. In: Potato, Global Research and Development, Vol.1 (eds. Khurana, S.M.Paul, Shekhawat, G.S., Singh, B.P. and Pandey, S.K.). Indian Potato Association, CPRI, Shimla, pp.268-270.

Ghosh, S.B., Nagi, L.H.S., Ganapathi, T.R., Khurana, S.M.Paul and Bapat, V.A. 2002. Cloning and sequencing of potato virus Y coat protein gene from an Indian isolate and development

of transgenic tobacco for PVY resistance. Current Science, 82:855-859.

Goodwin J., Chapman, K., Swaney, S., Parks, T.D., Wernsman, F.A. and Dougherty, W.G. 1996. Genetics and biochemical dissection of transgenic RNA-mediated virus resistance. Plant Cell, 8: 95-105.

Grasmick, M.E. and Slack, S.A. 1985. Symptom expression enhanced and low concentrations of potato spindle tuber viroid amplified in tomato with high light intensity and temperature. Plant Disease, 69:49-51.

Grasmick, M.E. and Slack, S.A. 1986. Effect of potato spindle tuber viroid on sexual reproduction and viroid transmission in true potato seed. Canadian Journal of Botany, 64:336-340.

Grasmick, M.F. and Slack, S.A. 1987. Detection of potato spindle tuber viroid in true potato seed by bioassay on Rutgers tomato. American Potato Journal, 64:235-244.

Gregorini, G. and Lorenzi, R. 1974. Meristem tip culture of potato plants as a method of improving productivity. Potato Research, 17:24-33.

Griffiths, H.M., Slack, S.A., Dodds, J.H. 1990. Effect of chemical and heat therapy on virus concentrations in *In vitro* potato plantlets. Canadian Journal of Botany, 68:1515-1521.

Gugerli, P. 1979. Potato virus A and potato leafroll virus: purification, antiserum production and serological detection in potato and test plants by ELISA. Phytopathology Z., 96:97-107.

Harrington, R., Katis, N. and Gilson, R.W. 1986. Field assessment of the relative importance of different aphid species in the transmission of potato virus Y[n]. Potato Research, 29:67-76.

Hamalainen, J.H., Watanabe, K.N., Valkonen, J.P.T., Arihara, A., Plaisted, R.L., Pehu, E., Miller, L. and Slack, S.A. 1997. Mapping and marker-assisted selection for a gene for extreme resistance to potato virus Y. Theoretical and Applied Genetics, 94: 192-197.

Harris, P.S. and James, C.M. 1987. Exclusion of viroids from potato resources and the modified use of a cDNA probe. European Plant Protection Bulletin, 17:51-60.

Herold, T., Haas, B., Singh, R.P., Boucher, A. and Sanger, H.L. 1992. Sequence analysis of five new field isolates demonstrates that the chain length of potato spindle tuber viroid (PSTVd) is not strictly conserved but is variable as in other viroids. Plant Molecular Biology, 19:329-333.

Hoekema, A., Huisman, M.J., Molendijk, L., van den Elzen, P.J.M. and Cornelissen, B.J.C. 1989. Genetic engineering of two commercial potato cultivars for resistance to potato virus X. Bio/Technology, 7:273-278.

Hooker, W.J. 1981. Compendium of potato diseases. American Phytopathological Society, St. Paul, MN. (USA), 125p.

Huguenot, C., van den Dobbelsteen, G., de Haan, P., Wagemakers, C.A.M., Drost, G.A., Osterhaus, A.D.M.E. and Peters, D. 1990. Detection of tomato spotted wilt virus using monoclonal antibodies and riboprobes. Archives Virology, 110:47-62.

Innis, M.A., Gelfand, D.H., Sninsky, J.J. and White, T.J. eds. 1990. PCR Protocols: A guide to methods and applications Academic Press, San Diego. USA.482p.

Jayasinghe, U., Chuqullanqui, C. and Salazar, L. F. 1989. Modified expression on virus resistance in mixed virus infections. American Potato Journal, 66 : 137-144.

Jefferies, C. 1998. FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm. No.19. Potato. FAO/IPGRI, Rome. 177pp.

Jones, R.A.C. 1983. Andean potato viruses and virus strains, and potato quarantine. In: Exotic Plant Quarantine Tests and Procedures for Introduction of Plant Materials, CIP, Lima (Peru), pp.11-17.

Jones, R.A.C. 1990. Strain group specific and virus specific hypersensitive reactions to infection with potyviruses in potato cultivars. Annals of Applied Biology, 117:93-105.

Jongedijk, E., de Schutter, A.A.J.M., Stolte, T., van den Elzen, P.J.M.and Cornelissen, B.J.C. 1992. Increased resistance to potato virus X and preservation of cultivar properties in transgenic potato under field conditions. Bio/Technology, 10:422-429.

Kalra, A., Grover, R.K., Rishi, N. and Khurana, S.M. Paul. 1989. Interaction between *Phytophthora infestans* and potato viruses X and Y in potato. Journal of Agricultural Sciences, Cambridge,

112 : 33-37.

Kalra, A., Grover, R.K., Rishi, N. and Khurana, S.M. Paul. 1992. Influence of different factors on the reduced susceptibility of potato virus X infected potato leaves to *Alternaria solani*. Journal of Agricultural Sciences, Cambridge, 119:185-190.

Kaniewski, W., Lawson, C., Sammons, B., Haley, L. and Hart, J. 1990. Field resistance of transgenic Russet Burbank potato to effects of infection by potato virus X and potato virus Y. Bio/Technology, 8:750-54.

Karande, A.A., Savithri, H.S. and Khurana, S.M. Paul. 1998. Monoclonal antibodies- Production and Application for the Detection and Diagnosis of Potato Viruses. In: Comprehensive Potato Biotechnology (eds. Khurana, S.M. Paul, Ramesh, C. and Upadhya, M.), Malhotra Publication House, New Delhi, pp.163-186.

Kassanis, B. 1950. Heat inactivation of leaf roll virus in potato tubers. Annals of Applied Biology, 37:339-41.

Kassanis, B. and Varma, A. 1967. The production of virus-free clones of some British potato varieties. Annals of Applied Biology, 59:447-450.

Kassanis, B. and Govier, D.A. 1971. The role of helper virus in aphid transmission of potato acuba mosaic virus and potato virus C. Journal of General Virology 13:221-228.

Kawchuk, L.M., Martin, R.R. and McPherson, J. 1990. Resistance in transgenic potato expressing the potato leafroll virus coat-protein in Russet Burbank potato plants. Molecular Plant-Microbe Interaction, 3:301-307.

Kawchuk L.M., Martin, R.R. and McPherson, J. 1991. Sense and antisense RNA-mediated resistance to potato leaf roll virus in Russet Burbank potato plants.Molecular Plant-Microbe Interaction, 4:247-253.

Kim, H.S., Jeon, J.H., Choi, K.H., Joung Y. and Park, S.W. 1996. Eradication of PVS (Potato Virus S) by thermo- and chemo-therapy in potato tissue culture. Journal of Korean Society Horticulture Science, 37:533-536.

Khurana, S.M. Paul. 1990a. Modern approaches for detection and management of the potato viruses and viroid. In: "Current Facets in Potato Research" (eds. Grewal, J.S., Shekhawat, G. S., Singh, R.A.) Indian Potato Association, Shimla, pp98-108.

Khurana, S.M. Paul. 1990b. True Potato Seed: Certification and Quality control. In: "Commercial adoption of true potato seed technology-prospects and problems". (ed. Gaur, P.C.) pp.120-127, CPRI, Shimla.

Khurana, S.M. Paul. 1990c. Detection of potato viruses and viroid in India: In: International Potato Center (CIP) Control of virus and virus-like diseases of potato and sweet potato. Report of 3rd Planning Conference, Lima, Peru, November 20-22, 1989, pp.61-64.

Khurana, S.M. Paul. 1992/1999. Potato viruses and viral diseases. Technical Bulletin No. 35 (Revised) CPRI, Shimla, 93p.

Khurana, S.M. Paul. 1998. The potato viruses and their management. In: "Pathological Problems of Economic Crop Plants and their Management" (ed. Khurana, S.M. Paul) Scientific Publishers (India) Jodhpur. pp.489-509.

Khurana, S.M. Paul, Singh, V. and Nagaich, B.B. 1975. Five strains of potato virus Y affecting potatoes in Shimla hills. Journal of Indian Potato Association, 2:38-41.

Khurana, S.M. Paul and Raychaudhuri, S.P. 1988. Interaction between plant pathogens with special reference to the viruses. International Journal of Tropical Plant Diseases, 6:43-58.

Khurana, S.M. Paul and Singh, M.N. 1988. Yield loss potential of potato viruses X and Y in Indian potatoes. Journal of Indian Potato Association, 15: 27-29.

Khurana, S.M. Paul, Phadtare, S.G., Garg, I.D., Singh, M.N., Bhardwaj, V.P. 1989a. Potato stem necrosis epidemic due to tomato spotted wilt virus in India. In: "Proceedings IV Interna-

tional Plant Virus Epidemiology Workshop", Montpellier, France, September 3-8, 1989, 10:30(Abstr.).

Khurana, S.M. Paul, Singh, M.N., Garg, I.D. and Agarwal, H.O. 1989b. PSTV and other viroid-like infections in Solanaceous plants in Shimla hills. In: "Proceedings Indo-US Workshop on Viroids and Diseases of uncertain etiology", IARI, New Delhi, November 15-18, 1989, pp.8-9 (Abstr.).

Khurana, S.M. Paul, Garg, I.D., Behl, M.K. and Singh, M.N. 1990a. Potential reservoirs of tomato spotted wilt virus in the North West Himalayas. National Academy of Sciences-Letters, 13:297-299.

Khurana, S.M. Paul, Singh, M.N., Owens, R.A. and Garg, I.D. 1990b. The wild potato viroid is distinct from spindle tuber viroid. In: Proceedings 6th Annual Convention, Indian Virological Society, December 17-19, 1990, Pune, pp.96.

Khurana, S.M. Paul and Garg, I.D. 1992. Potato mosaics. In: Plant Diseases of International Importance, Vol. II (eds. Chaube, H.S., Kumar, J., Mukhopadhyay, A. N. and Singh, U.S.), Prentice Hall, New Jersey, pp.148-164

Khurana, S.M. Paul and Garg, I.D. 1993. New techniques for detection of viruses and viroids. In: Advances in Horticulture, Vol. 7 Potato (eds. Chadha, K.L. and Grewal, J.S.). Malhotra Publication House, New Delhi, pp.529-566.

Khurana, S.M. Paul, Garg, I.D., Singh, M.N. and Kumar, Shiv. 1993. Storage and preservation affect immuno electron microscopy of flexuons potato viruses. Asian Potato Journal, 3:33-36

Khurana, S.M. Paul and Singh, M.N. 1995. Factors governing aphid transmission of the potato viruses, In: "Proceedings 2nd Agrilcultural Sciences Congress" (eds. Swaminathan, M.S.,Randhawa,N.S., Ananthakrishnan,T.N. and Narain,P.) NaTIONAL Academy of Agri-cultural Sciences, New Delhi.pp. 233-252.

Khurana, S.M. Paul, Chandra, R. and Dhingra, M.K. 1996. Potato viruses and their eradication for production of virus-free seed stocks. In: Disease Scenario in Crop Plants. Fruits and Vegetables, Vol.I (eds. Agnihotri,V.P., Prakash,O,Kishun,R., and Mishra,A.K.). IBPSS, New Delhi, pp. 195-218.

Khurana, S.M. Paul, Pandey, S.K., Singh, R.B. and Bhale, Usha M. 1997. Spread and control of the potato stem necrosis. Indian Journal of Virology, 13:23-28.

Khurana S.M. Paul and Garg, I.D. 1998. Present status of controlling mechanically and non-persistently aphid-transmitted potato viruses. In: "Plant Virus Disease Control" (eds. Hadidi, A., Khetrapal,R.K.and Koganezawa,H.) APS Press, St. Paul, USA, pp.593-615.

Khurana, S.M. Paul and Sane, Anuradha. 1998. Apical meristem culture - a tool for virus elimination. In: "Comprehensive Potato Biotechnology" (eds. Khurana, S.M. Paul, Ramesh,C. and Upadhya,M.). Malhotra Publishing House, New Delhi, pp 207-232.

Khurana, S.M. Paul and Chakraborty, S.K. 2000. Genetically engineered improvement of root and tuber crops. In: "Biotechnology in Horticulture and Plantation Crops" (eds. Chadha , K.L., Ravindra , P.N .and Sahijram, L.) Malhotra Publication House, New Delhi, pp.422-451.

Khurana, S.M. Paul, Verma, K.D. and Chandla, V.K. 2000. Potato virus vectors and their management. In: "Potato, Global Research and Development", Vol.1 (eds.Khurana, S.M.Paul, Shekhawat,G.S., Singh,B.P. and Pandey,S.K.). Indian Potato Assoc., CPRI, Shimla, pp 352-362.

Koenig, R. and Paul, H.L. 1982. Variants of ELISA in plant virus diagnosis. Journal of Virological Methods, 5: 113-125.

Komkov, Dya. 1975. On the question of correlation between mosaic virus and *Rhizoctonia solani* Kuhn on potato plants. Nauch.- Tr- NII Kartof- Kh- Va. 21 : 154-157.

Kumar, Shiv, Garg, I.D., Khurana, S.M. Paul and Singh, M.N. 1987. A resistance breaking strain

of potato virus X from Kufri Jyoti potatoes in Shimla hills, India. Proc. 3rd ann. Conv. Indian Virological Society, Calcutta, Abstract No. R.R. 13.

Kumar, Shiv and Khurana, S.M. Paul. 1989. Dot-ELISA or DIBA for potato viruses X, S, Y and leafroll. Proceedings of 5th Annual Indian Virological Society Convention, CPRI, Shimla, Oct. 17-19, 1989. SP-VIII/8, PP. 95.

Kumar, Shiv and Singh, Sarjeet. 1999. Sensitivity of polyvalent DOT-ELISA in comparison to DOT and DAS ELISA for detection of potato viruses. Journal of Indian Potato Association, 26: 58-62.

Lakshman, D.K. and Tavantzis, S.M. 1993. Primary and secondary structure of a 360-nucleotide isolate of potato spindle tuber viroid. Archives Virology, 128 : 319-331.

Lal, S.B. and Khurana, S.M. Paul. 1983. Detection and identification of viruses and mycoplasma and evaluation of yield losses. In: *Potato Production, Storage and Utilization* (eds.Nagaich,B.B.) CPRI, Shimla, pp. 334-348.

Levy, L., Lee, I.M. and Hadidi, A. 1994. Simple and rapid preparation of infected plant tissue extracts for PCR amplification of virus, viroid, and MLO nucleic acids. Journal of Virological Methods, 49:295-304.

Lizarraga, C.E., Salazar, L.F., Roca,W.M. and Schilde-Rentschler, L. 1980. Elimination of potato spindle tuber viroid by low temperature and meristem culture. Phytopathology, 70 : 754-755.

Lodge, J.K., Kaniewski, W.K. and Tumer, N.E. 1993. Broad-spectrum virus resistance in transgenic plants expressing pokeweed antiviral protein. Proceedings of National Academy of Science USA 90: 7089-7093.

Loebenstein, G., Akad, F., Filatov,V., Sadvakasova, G., Manadilova, A., Bakelman, H., Teverosky, E. , Lachmann, O. and David,A. 1997. Improved detection of potato leafroll luteovirus in leaves and tubers with digoxigenin-labeled cRNA probe. Plant Disease, 81: 489-491.

Lozoya-Saldana, H. and Dowson, W.O. 1982. The use of constant and alternating temperature regimes and tissue culture to obtain PVS free potato plants. American Potato Journal, 59: 221-229.

Maat, D.Z. and de Bokx, J.A. 1978. Enzyme-linked immunosorbent assay (ELISA) for the detection of potato virus A and Y in potato leaves and sprouts. Netherland journal of Plant Pathology, 84: 167-173.

Malnoe, P., Farinelli, L., Collet, G.F. and Reust, W. 1994. Small-scale field tests with transgenic potato cv. Bintje, to test resistance to primary and secondary infections with potato virus Y. Plant Molecular Biology, 25: 963-975.

Malyshenko, S.K., Kondakova, O.A., Nazarova,J.V., Kaplan, I.B., Talinasky, N.E. and Atabekov, J.G. 1993. Reduction of tobacco mosaic virus accumulation in transgenic plants producing non-functional viral transport proteins. Journal of General Virology, 74: 1149-1156.

Mandahar, C.L. and Khurana, S.M. Paul. 1998. Role of biotechnology in controlling plant diseases. In: Pathological Problems of Economic Crop Plants and their Management (ed. Khurana, S.M. Paul) Scientific Publishers Jodhpur (India). pp. 637-648.

McDonald, JG. 1986. Differences in mosaics disease virus profiles between three potato cultivars. Canadian Plant Disease Survey, 65 : 51-52.

McDonald, J.G. and Singh, R.P. 1996. Host range, symptomatology and serology of isolates of potato virus Y (PVY) that share properties with both the PVY[N] and PVY° strain groups. American Potato Journal, 73: 309-315.

Moreira, A., Jones, R.A.C. and Fribourg, C.E. 1980. Properties of a resistance-breaking strain of potato virus X. Annals of Applied Biology, 95 : 93-103.

Morel, G. and Martin, C. 1952. Guerison de dahlias atteints d'une maladie a virus. C.R. Academy Science. Paris 235: 1324-1325.

Morris, T.J. and Smith,E.M. 1977. Potato spindle tuber disease : procedures for detection of viroid RNA and certification of disease free potato tubers. Phytopathology,67 : 145-150.

Mukherjee, K., Singh, M.N., Khurana, S.M. Paul and Sandhu, S.K. 2000. Status of potato spindle tuber viroid in the field-grown germplasm collection determined by cRNA. In: Potato, Global Research and Development, Vol.1 (eds.Khurana, S.M.Paul, Shekhawat,G.S., Singh,B. P. and Pandey,S.K.). Indian Potato Assoc., CPRI, Shimla, pp 587-490.

Muller, K.O. and Munro, J. 1961. The reaction of virus-infected potato plants to *Phytophthora infestans*. Annals of Applied Biology, 38 : 765-773.

Mumford, R.A., Barker, H. and Wood,M.K.R. 1996. An improved method for the detection of tospoviruses using the polymerase chain reaction. Journal of Virological Methods, 57 : 109-115.

Murashige, T. and Skoog, F. 1962. A revised medium for rapid growth and bioassays with tobacco tissue culture. Physiologica Plantarum, 15: 473-497.

Nagaich, B.B., Pushkarnath, Bhardwaj,V.P., Giri, B.K. and Upreti,G.C. 1969. Production of disease free seed potatoes in the Indo-Gangetic plains. Indian Journal Agricultural Sciences, 39 : 238-243.

Nagaich, B.B. and Prasad, B. 1970. Interaction between *Alternaria solani* and potato viruses X and Y. Indian Journal of Experimental Biology, 9: 88-90.

Nagaich, B.B., Shekhawat, G.S., Khurana, S.M. Paul and Bhattacharyya, S.K. 1974. Pathological problems of potato cultivation. Journal of Indian Potato Association, 1 : 32-44.

Nagaich, B.B. and Singh, V. 1982. Andigena crosses for improving potato production by utilizing their disease resistance potential. In : "Potato in Developing Countries" (eds. Nagaich, B.B., Shekhawat, G.S. and Verma, S.C.). IPA, CPRI, Shimla, pp 306-312.

Nicholson, A. 1996. Structure, reactivity, and biology of double-stranded RNA. Progress in Nucleic Acid Research and Molecular Biology. 52: 1-65.

Nie, X. and Singh,R.P. 2000. Detection of multiple potato viruses using an oligo (dT) as a common cDNA primer in multiplex RT-PCR. Journal of Virological Methods, 86: 179-185.

Nie, X. and Singh, R.P. 2001. A novel usage of random primers for multiplex RT-PCR detection of virus and viroid in aphids, leaves and tubers. Journal of Virological Methods,91: 37-49.

Norris, D.O. 1954. Development of virus-free stock of green maintain potato by treatment with malachite green. Australian Journal Agricultural Research, 5: 658-663.

Nyland, G. and Goheen, A.C. 1969. Heat therapy of virus diseases of perennial plants. Annual Review of Phytopathololy, 7: 331-354.

Oshima, K., Sako, K., Hiraishi, C., Nakagawa, A., Matsuo, K., Ogawa,T., Shikata, E. and Sako, N. 2000. Potato tubers necrotic ringspot disease occurring in Japan: its association with potato virus Y necrotic strain. Plant Disease, 84:1109-1115.

Owens, R.A., and Diener, T.O. 1981. Sensitive and rapid diagnosis of potato spindle tuber viroid disease by nucleic acid hybridization. Science, 213 : 670-672.

Owens, R.A., Khurana, S.M. Paul, Smith, D.R., Singh, M.N. and Garg,I.D. 1992. A new mild strain of potato spindle tuber viroid isolated from wild *Solanum* spp. in India. Plant Disease, 76 : 527-529.

Pennazio, S. 1971. Potato therapy: meristem tip culture combined with thermotherapy. Rivista della Ortoflorofrut-ticolutura Italiana. 55:446-452.

Perez, de San Roman, C., Legorburu, F.J., Pascualena, J. and Gil, A. 1988. Simultaneous detection of potato viruses Y, leafroll, X and S and by DAS-ELISA technique with artificial polyvalent antibodies (APAs). Potato Research, 31: 151-158.

Pietkiewiczs, J. 1975. Effect of viruses on the reaction of potato to Phytophthora infestans, II. Mechanism of changes in reaction of *Phytopthora infestans* in virus-infected plants. Phytopathology Z., 82 : 49-65.

Pietrak, J. 1981. Influence of the presence of viruses M and S in potato plants on tuber infection by viruses X and Y. Biuletyn Instytutu Ziemniaka, 26 : 25-31.

Pushkarnath. 1967. Seed potato production in the sub-tropical plains of India. American Potato Journal, 44 : 429-441.

Quak, F. 1961. Heat treatment and substances inhibiting virus multiplication in meristem culture to obtain virus-free plants. Advances in Horticulture Science, 1:144-148.

Quak, F. 1972. Therapy. In: Viruses of potatoes and seed potato production. Centre for Agric. Pub. And Doc., Wageningen. pp. 158-166.

Quak, F. 1977. In : Plant Cell Tissue and Organ Culture (eds.Reinert, J. and Bajaj, Y.P.S.) Springer-Verlag, Berlin, pp. 598-615.

Querci, M., Salazar, L.F. and Fernadez-Northcote, E.N. 1993. Detection of Andean potato virus X isolates by radioactive and non-radioactive nucleic acid spot hybridization tests. Phytopathology, 83 : 171-176.

Querci, M., Owens,R.A., Bartolini, I., Lazarte, V. and Salazar,L.F. 1997. Evidence for heterologous encapsidation of potato spindle tuber viroid in particles of potato leafroll virus. Journal of General Virology, 78 : 1207-1211.

Querci, M. and Salazar, L. 1998. Molecular probes for detection of viruses/viroid in potato. In Comprehensive Potato Biotechnology (eds. Khurana, S.M. Paul, Ramesh,C. and Upadhya,M.) Malhotra Publ. House, New Delhi., pp. 187-206.

Reestman, A.J. 1970. Importance of the degree of virus infection for the production of ware potatoes. Potato Research, 13: 248-268.

Reichenbacher, D., Kalinnia, I., Schultze, M., Hom, A. and Kleinhemple, H.1984. Ultramicro-ELISA with a fluorogenic substrate for detection of potato viruses. Potato Research, 27: 353-364.

Roberts, I.M. 1986. Practical aspects of handling, preparing and staining samples containing plant virus particles for electron microscopy. In: Developments and Applications in Virus Testing (eds.Jones, R.A.C. and Torrance, L.). Association of Applied Biologists, Wellesbourne, U.K. pp 213-243.

Robinson, D.J. and Romero,J. 1991. Sensitivity and specificity of nucleic acid probes for potato leafroll luteovirus detection. Journal of Virological Methods, 34: 209-219.

Roca, W.M., Espinoza, N.O., Roca, M.R. and Bryan,J.E.. 1978. A tissue culture method for rapid propagation of potatoes. American Potato Journal, 55: 691-701.

Rowhani, A. and Stace-Smith, R. 1979. Purification and characterization of potato leafroll virus. Virology, 98 : 45-54.

Sajid, G.M., Quraishi, A. and Salim, M.. 1986. Thermotherapy and meristem tip culture of *S. tuberosum* for elimination of potato viruses X,S and Y. Pakistan Journal of Botany, 18: 249-253.

Salazar, L.F. 1989. Potato spindle tuber. In : Plant Protection and Quarantine, Vol. II (ed. Kahn, R.P.) CRC Press, Fla., pp. 155-167.

Salazar, L.F. 1990. Main virus diseases of potato. In : International Potato Center (CIP). Control of virus and virus like disease of potato and sweet potato. Report of 3rd Planning Conference, Lima, Peru. Nov. 20-22, 1989,pp. 9-12.

Salazar, L.F. 1996. Potato Viruses and their Control. International Potato Center, Lima. Peru. 205 pp.

Salazar, L.F., Balbo, I. and Owens,R.A. 1988. Comparison of four radioactive probes for the diagnosis of potato spindle tuber viroid by nucleic acid spot hybridization. Potato Research, 33 : 323-328.

Salazar, L.F. and Querci, M. 1992. Detection of viroids and viruses by nucleic acid probes. In Techniques for the Rapid Detection of Plant Pathogens. (eds. Duncan, J.M. and Torrance, L.). Blackwell Sci. Publ. Oxford, UK, pp. 129-144.

Seal, S. and Coates, D. 1997. Assaying by PCR. In: *Plant Virus Protocols from virus isolation to transgenic resistance* (eds. Foster, G.D. and Taylor, S.). Humana Press, Totowa, New Jersey, USA.

Shamloul, A.M., Haddidi, A., Zhu, S.F., Singh, R.P. and Sagredo, B. 1997. Sensitive detection of potato spindle tuber viroid using RT-PCR and identification of a viroid variant naturally

infecting pepino plants. Canadian Journal of Plant Pathology, 19 : 89-96.

Sigvald, R. 1985. Mature plant resistance of potato plants against potato virus Y (PVY). Potato Research, 28: 135-143.

Sigvald, R. 1986. Forecasting the incidence of potato virus Y. In: Plant Virus Epidemics - Monitoring, Modelling and Predicting Outbreaks (eds. McLean, G.D., Farret,R.G.and Ruesink,W.G.). Academic Press, Australia. pp 419-441.

Sigvald, R. 1987. Aphid migration and the importance of some aphid species as vectors of potato virus Y (PVY) in Sweden. Potato Research, 30: 267-283.

Sigvald, R. 1990. Aphids on potato foliage in Sweden and their importance as vectors of potato virus Y. Acta Agriculturae Scandinavica, 40: 53-58.

Sigvald, R. 1998. Forecasting aphid borne virus diseases. In: Plant virus disease control (eds. Hadidi, A., Khetrapal, R.K. and Koganezawa, H.) APS Press, St. Paul, USA. pp. 172-187.

Singh, B.P., Singh, M.N., Bhattacharyya, S.K., Khurana, S.M. Paul and Shekhawat, G.S. 1992. Potato virus S enhances susceptibility of genotypes to late blight. Proceedings Symposium Integrated Disease Management and Plant Health, UHF, Solan, Sept 25-26, 1992, Abstr. No.018.

Singh, K., Rai, R.P., Srivastava, S.N.S. and Ramphal. 1986. Dates of planting and haulm cuttings for disease-free seed potato production in eastern Gangetic plains. Journal of Indian Potato Association, 13 : 31-41.

Singh, M., Singh, R.P. and Moore, L. 1999. Evaluation of NASH and RT-PCR for the detection of PVY in dormant tubers and its comparison with visual symptoms and ELISA in plants. American Journal of Potato Research, 75: 61-66.

Singh, M.N., Khurana, S.M. Paul,Nagaich, B.B. and Agrawal, H.O.. 1981. Epidemilogical studies on potato viruses Y and leafroll in sub-tropical India. Proceedings of International Epidemiological Conference, Oxford, July 27-30, pp 89-90.

Singh, M.N., Khurana, S.M. Paul, Nagaich, B.B. and Agrawal, H.O.. 1982. Efficiency of *Aphis gossypii* and *Acyrthosiphon pisum* in transmitting potato viruses leafroll and Y. In : Potato in developing Countries ((eds. Nagaich,B.B.,Shekhawat,G.S.and Verma,S.C.) Indian Potato Association, Shimla, pp 289 293.

Singh, M.N., Khurana, S.M. Paul and Sharma, H.C.. 1983. A new technique to screen potato genotypes for aphid resistance. Journal of Indian Potato Association, 10 : 104-110.

Singh, M.N., Nagaich, B.B. and Agrawal, H.O. 1984. Spread of viruses Y and leafroll by aphids in potato fields. Indian Phytopathology, 37:241-251.

Singh, M.N., Khurana, S.M.Paul, Nagaich,B.B. and Agrawal, H.O. 1988. Environmental factors influencing transmission of potato virus Y and potato viruses. Potato Research, 31 : 501-509.

Singh, M.N., Khurana, S.M. Paul and Joshi, M. Usha. 1989. A simple penicillinase based ELISA for potato virus X. Proceedings of Indian National Science Academy, B 55 : 287-290.

Singh, M.N., Khurana, S.M. Paul and Garg, I.D. 1990. Antiserum production and immunoelectron microscopy of potato leafroll virus. Indian Phytopathology, 43 : 13-19.

Singh, M.N. and Khurana, S.M. Paul. 1994. Comparison of three ELISA variants for detecting potato viruses. In Potato : Present and Future (eds. GS Shekhawat, Khurana, S.M. Paul and Chandra,R.). Indian Potato Association, Shimla, pp. 314-317.

Singh, M.N. and Khurana, S.M. Paul. 1999. Evaluation of short reaction times for optimizing das-ELISA schedule for three flexuous potato viruses. Journal of Indian Potato Association, 26: 35-40.

Singh, M.N. and Khurana, S.M. Paul. 2000. Role of additives in sample extraction buffer for improvement of ELISA for potato leafroll virus. In: Potato, Global Research and Development, Vol 1 (eds. Khurana, S.M. Paul, Shekhawat, G.S., Singh, B.P. and Pandey,S.K.). Indian Potato Assoc., CPRI, Shimla, pp 439-443.

Singh, M.N., Mukherjee, K. Khurana, S.M.Paul, Gopal, Jai and Querci, M. 2000. Detection of potato spindle tuber viroid by NASH in exotic potato germplasm. In: Potato, Global Research and Development, Vol .1 (eds. Khurana, S.M. Paul, Shekhawat, G.S., Singh, B.P. and Pandey, S.K.). Indian Potato Assoc., CPRI, Shimla, pp 491-494.

Singh, R.A. 1980. Studies on potato aucuba mosaic. Ph D Thesis. IARI, New Delhi, 112 pp.

Singh, R.A., and Khurana, S.M. Paul. 1993. Viral and allied diseases of potato. In :Advances in Horticulture, Vol. 7. Potato (eds. Chadha, K.L. and Grewal, J.S.) Malhotra Publishing House, New Delhi. pp 491-528.

Singh, R.B., Khurana, S.M.Paul, Pandey, S.K. and Srivastava, K.K. 1997a. Screening germplasm for potato stem necrosis resistance. Proceedings of Indian Virological Society Conference, GAU, Anand Sept. 1-3, 1997, pp 24 (Abstr.).

Singh, R.B., Srivastava, K.K., Khurana, S.M. Paul and Pandey, S.K.. 1997b. Assessment of yield losses due to potato stem necrosis disease. Indian Journal of Virology, 13 : 135-137.

Singh, R.B., Srivastava, K.K. and Khurana, S.M. Paul.1999. Seed tuber treatment with Imidacloprid protects potato crop from vector spread of the stem necrosis tospovirus. In: Proceedings 51st Annual Meeting Indian Phytopathgological Society, Febr 17-19, Lucknow. p 102 (Abstr.).

Singh, R.P. 1970. Seed transmission of potato spindle tuber virus in tomato and potato. American Potato Journal, 47 : 225-227.

Singh, R.P. 1983. Viroids and their potential danger to potatoes in hot climates. Canadian Plant Disease Survey, 63 : 13-18.

Singh, R.P. 1988a. Control of viroid and contact transmitted virus diseases. In: Potato Pest Management in Canada. (eds. Boiteau G., Singh,R.P. and Parry,R.H.) Canada Agriculture, NB.pp. 309-325.

Singh, R.P. 1988b. Role of weeds in potato virus spread, In: Potato Pest Management in Canada. (eds. Boiteau G., Singh,R.P. and Parry,R.H.) Canada Agriculture, NB. pp. 355-362.

Singh, R.P. 2000. Advances in molecular detection methodologies for potato viruses and viroids. In: Potato, Global Research and Development, Vol 1 (eds. Khurana, S.M. Paul, Shekhawat, G.S., Singh, B.P. and Pandey,S.K.). Indian Potato Association, CPRI, Shimla. pp. 219-235.

Singh, R.P. and Fernow,K.H. 1981. Potato spindle tuber viroid. In : Compendium of potato diseases. (ed. Hooker, WJ). American Phytopathological Society. St. Paul, Minnesota. pp. 89-90.

Singh, R.P. and Mc Donald, J.G. 1981. Purification of potato virus A and its detection in potato by enzyme linked immunosorbent assay (ELISA). American Potato Journal, 58: 181-189.

Singh, R.P., and Somerville, T.H. 1983. Effect of storage temperatures on potato virus infectivity levels and serological detection by ELISA. Plant Disease, 67 : 1133-1136.

Singh, R.P. and Boucher, A. 1987. Electrophoretic separation of a severe from mild strains of potato spindle tuber viroid. Phytopathology, 77 : 1588-1591.

Singh, R.P. and Boiteau,G. 1988. Control of aphid-borne diseases : non-persistent viruses. In: Potato Pest Management in Canada. (eds. Boiteau G., Singh,R.P. and Parry,R.H.) Canada Agriculture, NB. pp. 30-53.

Singh, R.P., Boucher, A. and Somerville, T.H. 1992. Detection of potato spindle tuber viroid in the pollen and various parts of potato plant pollinated with viroid-infected pollen. Plant Disease, 76 : 951-953.

Singh, R.P., Kurz, J., Boiteau, G. and Bernard,G. 1995. Detection of potato leafroll virus in

single aphid by the reverse transcription polymerase chain reaction and its potential epidemiological application. Journal of Virological Methods, 55: 133-143.

Singh, R.P., Nie,X., Singh, M., Coffin R. and Duplessis, P. 2002. Sodium sulphite inhibition of potato and cherry polyphenolics in nucleic acid extraction for virus detection by RT-PCR. Journal of Virological Methods, 99: 123-131.

Singh, Sarjeet, Upreti, G.C., Bhardwaj,V.P., Vashishth, K.S., Verma,A.K., Garg V.K. and Dhingra, M. K.. 1984. Effect of temperature and physiological age of the tubers on detectability of potato virus X and S. Journal of Indian Potato Association, 11 : 12-19.

Singh, Sarjeet, Garg, V.K. and Singh,Jagpal. 1987. Presprouting for reduction of sources of infection of viral and mycoplasmal diseases in breeders' seed potatoes. Indian Journal of Virology, 3 : 147-155.

Singh, Sarjeet and Barker, H. 1991. Comparison of penicillinase-based and alkaline phosphatase-based enzyme-linked immunosorbent assay for the detection of six potato viruses. Potato Research, 34: 451-457.

Singh, Shatrughna, Kumar, Shiv and Khurana, S.M. Paul. 1994. Incidence and relative concentration of common potato viruses in five cultivars. Indian Journal of Virology, 10 : 44-50.

Singh, Shatrughna and Khurana, S.M. Paul. 1995. Interaction among three viruses in naturally infected potato cultivars. Indian Phytopathology, 48 : 70-73.

Slack, S.A. 1983. Identification of an isolate of the Andean strain of potato virus S in North America. Plant Disease, 67 : 786-789.

Slack, S.A. and Tufford,L.A. 1995. Meristem culture for virus elimination. In : Plant, Cell, Tissue and Organ Culture: Fundamental Methods. (eds. Gamborg, O.L. and Phillips,G.C.). Springer-Verlag, New York, USA, pp. 117-128.

Smith, F.D. and Banttari,E.E. 1987. Dot-ELISA on nitrocellulose membrane for detection of potato leafroll virus. Plant Disease, 71 : 795-799.

Sparrow, L.A.D. 1976. Recent trends in the activity of aphids infesting potatoes in south-east Scotland in relation to virus incidence in the crop. Scottish Horticulture Research Institute Association Bulletin, 11:8-14.

Stace-Smith, R. 1985. Virus free clones through plant tissue culture. In: Comprehensive Biotechnology, Vol.4 (eds. Robinson, C.W. and Howell, R.A.,) Pergamon Oxford UK. pp.169-179.

Stanley, C.J., Johanson, A. and Self, C.H. 1985. Enzyme-amplification can enhance both the spread and the sensitivity of immunooassays. Journal of Immunological Methods 83 : 89-95.

Stark, D.M. and Beachy, R.N. 1989. Protection against potyvirus infection in transgenic plants: evidence for broad spectrum resistance. Bio-Technology, 7: 1257-1262.

Stobbs, L.W. and Barker, D. 1985. Rapid simple analysis with a simplified ELISA. Phytopathology, 75 : 492-495.

Tacke, E., Salamini, F. and Rohde, W. 1996. Genetic manipulation of potato for broad spectrum protection against virus infection. Nature-Biotechnology, 14: 1597-1601.

Thirumalachar, M.J. 1954. Inactivation of potato leafroll by high temperature storage of seed potatoes in Indian plains. Phytopathology Z.,22:429-436.

Torrance, L. 1992. Serological methods to detect plant viruses : production and use of monoclonal antibodies. In Techniques for the Rapid Detection of Plant Pathogens (eds. Duncan, J.M. and Torrance, L.). Blackwell Scientific Publications, Oxford, UK, pp 7-32.

Torrance, L. and Jones, R.A.C.. 1982. Increased sensitivity of detection of plant viruses obtained by using a fluorogenic substrate in ELISA. Annals of Applied Biology, 91 : 205-213.

Torrance, L., Larkins, A.P. and Butcher,G.W. 1986. Characterization of monoclonal antibodies against potato virus X and comparison of serotypes with resistance groups. Journal of General Virology, 67: 57-67.

Tozzini, A.C., Ceriani, M.F., Cramer, P., Palva, E.T. and Hopp, H.E. 1994. PVX-MS, a new

strain of potato virus that overcomes the extreme resistance gene Rx. Journal of Phytopathology, 141 : 241-248.

Truve, E., Aaspoullo, A., Honkanen, J., Puska, R., Mehto, M., Hassi, A., Teeri, T.H., Kelvi, M., Seppanen, P. and Saarma, M. 1993. Transgenic potato plants expressing mammalian 2'-5' Oligoadenylate Synthetase are protected from potato virus X infection under field conditions. Bio/Technology, 11: 1048-1052.

Turl, L.A.D. 1980. An approach for forecasting the incidence of potato and cereal aphids in Scotland. European Plant Protection Bulletin, 10:135-141.

Turl, L.A.D. 1983. The effect of winter weather on the survival of aphid population on weeds in Scotland. Bulletin OEPP, 13 : 139-143.

Upreti, G.C., Bhardwaj, V.P., Garg, V.K. and Singh, Sarjeet. 1979. Natural spread of contagious viruses PVX and PVS in some potato varieties during nucleus seed production. Journal of Indian Potato Association, 6: 60-69.

Upreti, G.C., Verma, R.K. and Kang, G.S. 1987. Viral and mycoplasmal diseases spread in potato field during seed production at Mukteswar (Kumaon Hills). Journal of Indian Potato Association, 14 : 76-77.

Valkonen, J.P.T., Puurand, U., Slack, S.A., Makinen, K. and Saarma, M. 1995. Three strain groups of potato A potyvirus based on hypersensitive responses in potato, serological properties and coat protein sequences. Plant Disease, 79:748-753.

van den Heuvel, J.F.J.M., van der Vlugt, R.A.A., Verbeek, M., De Haan, P.T. and Huttinga, H. 1994. Characteristics of a resistace-breaking isolate of potato virus Y causing potato tuber necrotic ringspot disease. European Journal of Plant Pathology, 100 : 347-356.

Van Harten, A. 1983. The relationship between aphid fights and the spread of potato virus Y-n(PVY^{-N}) in the Netherlands. Potato Research, 26: 1-15.

Vashisth, K.S., Verma, A.K., Chaubey, I.P. and Nagaich, B.B. 1981. Comparative degenerative effects of viral and MLO diseases during autumn and spring seasons on Indian potato varieties in Punjab. Seeds and Farms, 7: 25-28.

Vasudeva, R.S. and Azad, R.N. 1952. Investigation of potato diseases and production of disease free seed potatoes in India. Empire Journal of Experimental Agriculture, 20 : 293-300.

Verma, A.K. and Vashisth, K.S. 1985. Manipulation of planting and haulms cutting dates for better health standards in seed potato crop. Indian Journal of Virology, 1: 54-60.

Verma, K.D., Chaubey, I.P., Jeswani, M.D. and Khurana, S.M.Paul. 1998. Present status of *Myzus periscae* in potato seed production in Meerut region. Journal of Indian Potato Association, 25: 109-112.

Vetten, H.J., Ehlers, U. and Paul, H.L. 1983. Detection of potato virus Y and A in tubers by enzyme-linked immunosorbent assay after natural and artificial break of dormancy. Phytopathology, 108: 41-53.

Wang, P.J. and Hu, C.Y. 1982. *In vitro* mass tuberization and virus-free seed potato production in Taiwan. American Potato Journal, 59: 33-39.

Weidemann, H.L. and Maiss, E. 1996. Detection of the potato tuber necrotic ringspot strain of potato virus Y (PVY^{NTN}) by reverse transcription and immunocapture polymerase chain reaction. Journal of Plant Disease Protection, 103: 337-345.

Wijkamp, I., Van de Wetering, F., Goldbach, R. and Peters, D. 1996. Transmission of tomato spotted wilt virus by *Frankliniella occidentalis*: median acquisition and inoculation access period. Annals of Applied Biology, 129: 303-313.

Wiktelius, S. 1982. Flight phenology of cereal aphids and possibilities of using suction trap catches as an aid in forecasting outbreaks. Swedish Journal Agricultural Research, 12:9.

Zaklukicwicz, K. 1983. Liberation of potato plants from viruses S and M, Potato Abstracts1984, 9: 170.

Strawberry Disease Management

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Abstract: Strawberry is affected by many diseases around the world and their economic importance often is determined by cultural systems and varieties used, local environment, and limitations on availability and effectiveness of management strategies. This chapter reviews the currently available control measures and strategies, with emphasis on use of resistant varieties and non-chemical disease management. It is in necessity that a general treatise is presented, stressing strategies that should not become obsolete and that are useful in many, if not most, strawberry-growing regions of the world. One or more disease control strategies are generally emphasized because of local needs and restrictions. A great deal of information has been included which should give the reader a fuller understanding of strawberry diseases and their control, aspects of control strategies that are not readily apparent to the casual reader, interactive effects of pathogen, strawberry variety, and environment, and a large body of references pertinent to these areas. This review should be useful to growers, consultants, and researchers interested in developing disease management strategies for strawberry culture.

1. Introduction

Initiation and development of disease in plants requires a susceptible host plant, a pathogen, and favorable environmental conditions. The genotype, or genetic makeup, of the host is most often a limiting factor in disease development, as is the genotype of the pathogen. A compatible combination of a non-resistant, or susceptible plant plus a pathogen with virulence genes for that host must act together under favorable conditions of temperature, moisture, and often other factors for disease development to proceed. In the cultivated strawberry (*Fragaria x ananassa* Duchesne), cultivars differ in their susceptibilities to many pathogens; *e.g*, *Verticillium* species (*Verticillium* wilt), *Phytophthora fragariae* Hickman var. *fragariae* (red stele, or red core, root rot), *Sphaerotheca macularis* (Wallr.:Fr.) Jacx. f. sp. *fragariae* Peries (powdery mildew), *Alternaria alternata* (Fr.:Fr.) Keissl. f. sp. *fragariae* Dingley (Alternaria black leaf spot), and *Colletotrichum* species (anthracnose) (Hancock *et al.,* 1990). These genotypic differences, and our ability to breed and select disease-resistant types, are the basis of many strategies for disease control in strawberry. Several strawberry pathogens also exist as races or pathotypes which, although indistinguishable on the species level, differ genetically in their abilities to cause disease. For example, several pathogenic races of *P. fragariae* are known to possess virulence and avirulence genes that interact in a gene-for-gene manner with genes of the host cultivar (Van de Weg 1997); this interaction apparently involves specific elicitors for pathogenicity (Scott and Maas, unpublished data).

The purpose of this chapter is to review general control measures and strategies for strawberry diseases. It is of necessity that a general treatise is presented, stressing strategies that should not become obsolete and that are useful in many, if not most, strawberry-growing regions of the world. One or more disease control strategies are generally emphasized because of local needs and restrictions. The reader is referred to local experts for pesticide use recommendations. A great deal of peripheral information has been included which should give the reader a fuller understanding of strawberry diseases and their control, aspects of control strategies that are not readily apparent to the casual reader, interactive effects of pathogen, strawberry cultivar, and environment, and a body of references pertinent to these areas. This chapter is not intended to be a picture book for disease identification; for this the reader is asked to consult other works that necessarily are directed more exhaustively toward this purpose. An excellent reference for strawberry disease identification is the Compendium of Strawberry Diseases (Maas, 1984, 1998), published by the American Phytopathological Society (*http://www.apsnet.org*) as part of their series of disease compendia on many other crops.

1.1 The Strawberry

The modern cultivated strawberry is a relatively recently developed crop. Its origin is the result of the hybridization of *Fragaria virginiana* Duch. from North America and *F. chiloensis* (L.) Duch. from South America (Darrow 1966; Hancock 1999; Wilhelm and Sagen 1974). Plants of *F. chiloensis* were imported to Europe in 1716 by the French spy Captain Amédée Frézier where chance hybridizations occurred with *F. virginiana* plants already established in Europe. By the 1750s commercial fields were established with both species, but it was not until 1817 that the first attempts were made to breed and select superior strawberry types (Darrow 1966). Rapid progress in strawberry cultivar improvement has since taken place in the 1900s, resulting in nearly 1,000 strawberry cultivars being released worldwide. World production of strawberries has risen dramatically since 1961 [75 thousand metric tones (kt)] to approximately 2,784 kt in 2000 (FAO 2001; Rieger 2001). The leading strawberry-producing countries currently are the United States (785 kt), Spain (367 kt), Japan (198 kt), Poland (171 kt), Korean Republic (155 kt), Russia (128 kt), Mexico and Turkey (110 kt), and Germany and Italy (81 kt) (FAO 2001; Rieger 2001).

Although the genetic base of the domestic strawberry is relatively narrow (Sjulin and Dale 1987), the high levels of heterozygosity in its genome has allowed breeders to continually breed and select cultivars improved for various traits. These traits include increased plant productivity, fruit size, fruit flavor, fruit nutritional content, disease resistance, adaptation to specific locality environments, adaptation to various cultural systems, mechanical harvesting, and market needs such as increased fruit firmness and shelf-life. One of the most recent advances and one having perhaps the greatest impact on worldwide expansion of strawberry production is the change-over from the photoperiod/temperature interaction sensitivity for flowering from obligate short-day (Junebearing) plants typical of older cultivars to facultative long-day and day-neutral (everbearing) plants (Bringhurst and Voth 1978; Darrow 1966, Durner *et al.,*

1984; Heide 1977; Nicoll and Galletta 1987). Many of these breeding and selection goals and achievements are discussed in Galletta and Maas (1990).

1.2 Cultural systems

Strawberries are grown in many systems; from wide, flat uninterrupted beds, narrow flat beds, elevated beds (plasticulture) with two to four rows of plants, in protected culture under low or high tunnels, in glasshouses with varying plant support systems, and in aquaculture. The matted-row system is basically perennial, kept in production for at least three years and often several additional years depending on plant health and weed control. The raised-bed systems are by nature annual systems; however, there has been increasing interest in prolonging the planting life for an additional year. The difficulty here is that cultivars adapted to raised-bed culture, such as 'Chandler', are susceptible to many diseases and become unproductive during the second fruiting year.

Tunnel or cloche cultivation, greenhouse forcing, and hydroponic cultivation systems have been developed in Europe and Asia, to provide fresh strawberries at otherwise out-of-season times, and they may be used to extend normal seasons. In the United States, production of strawberries is nearly continuous throughout the year, mostly with annual raised-bed systems. California provides fresh fruit from February to September, Florida from December to March, and other states from May to July, depending on latitude (for Junebearing cultivars), and into October or November with day-neutral cultivars (Galletta and Bringhurst 1990). World production areas have shifted in the past twenty years, from traditionally northern latitudes where matted-row culture predominated, to areas with Mediterranean climates and abundant labor where raised-bed culture predominates. Strawberry production has increased in the past ten years in countries such as Egypt, Morocco, Iran, Kuwait, Lebanon, Turkey, Spain, Mexico, Germany, and the United States, while decreasing in Poland and Japan (FAO Production Yearbook 2001). Fruit yields vary with location, climate, cultivar, and culture system. In the United States, yields with matted-row systems in the northeastern and north central regions commonly are 2-17 t/ha and yields with annual raised-bed systems in Florida in the southeast are 22-34 t/ha and on the Pacific coast in California 90-112 t/ha (Galletta and Bringhurst 1990).

The preferred cultural systems have been determined by economic factors; the highest production possible with the highest net return of investment. In most regions, the choices of cultural system are dictated by the availability and costs of land and labor as well as by market considerations. It is important to note, that with the different cultural systems, disease pressures vary as do the strategies for disease control. Thus, diseases and pests encountered in matted-row, raised-bed, and protected culture systems often are different. Consequently, the strategies for disease management in each system are also different. Interactions among culture system, cultivar, and disease also occur. For example, *Botrytis* fruit rot of 'Sweet Charlie' plants was found to be as much as 94% lower in tunnels than in the field. The incidence of *Botrytis* rot in the tunnels was found to be less than 2%, due to absence of free water on plants, and required no fungicidal control (Xiao *et al.,* 2001). In the same study, powdery mildew

of the fruit of 'Sweet Charlie' plants was exceptionally higher in the tunnels than in field plantings due to lower light intensity, higher relative humidity, and cooler temperatures in tunnels compared to in field plantings. Incidences of both diseases were reversed for plants of 'Camarosa'; *Botrytis* fruit rot was low in the field and powdery mildew of the fruit was high in tunnels. Fungicidal control of each disease was found to be dependent on cultivar and cultural system. Some generalities can be made, however. The successful management of strawberry diseases begins with pathogen-free propagation planting stock, use of cultivars resistant to the predominant diseases in a locality, sanitation, and use of chemical and biological controls of pathogens and vectors.

2. Root and crown diseases

The healthy root system of a strawberry plant consists of long primary roots, as many as 50 or more extending from the crowns of older plants, and thousands of small secondary roots branched from the primary roots. The primary roots may live for a year in commercial strawberry cultivars unless damaged by disease or other factors, such as nematodes or drought, while the secondary roots are relatively short lived. Older primary roots naturally become dark, but the central xylem-containing stele remains white and functional. Various fungi may attack and destroy secondary roots and invade primary roots, limiting the plant's ability to take up water and nutrients. Above-ground symptoms of root destruction include leaf yellowing, fruit desiccation and poor development, wilting, and plant collapse. The strawberry root in a natural soil situation co-exists with a myriad of fungi and other microflora and fauna. Fungi cohabiting strawberry roots may be confined to the outer surface of roots or may invade roots and form symbiotic relationships with the plant, deriving nutrients from root tissues and making nutrients more available for uptake by the root system. *Rhizoctonia,* for example, is commonly isolated from healthy-appearing roots. When the natural balance of root and potential pathogen is interrupted, root infection and disease may occur. (*see:* "Healthy" Soils.)

The strawberry crown is a perennial structure and may live for years if it is not damaged by pathogens or other factors such as insects or freezing temperatures. Crown infection may derive from root infection, or direct infection of the crown itself. Symptoms associated with crown infection include discoloration of the interior crown tissues, leaf yellowing, and plant collapse. Major root- and crown- infecting pathogens include species of *Verticillium, Colletotrichum, Phytophthora, Fusarium, Rhizoctonia,* and *Pythium*. Anthracnose, caused by *Colletotrichum* spp., is discussed under Fruit Diseases.

2.1 *Verticillium* Wilt

Verticillium albo-atrum Reinke & Berth. and *V. dahliae* Kleb., causes of *Verticillium* wilt in strawberry, have wide host ranges among annual and perennial crops and weeds and are capable of persisting for long periods in soil in undecayed plant debris and as microsclerotia. Although the fungus can be carried in nursery stock, especially in re-

sistant plants, disease outbreaks originating from nursery plants seldom occur. Most outbreaks of *Verticillium* wilt are related to the recent crop history of the land. Growers who alternate strawberry crops with other crops such as tomato, eggplant (*Solanum melongena* L.), potato (*S. tuberosum* L.), pepper (*Capsicum annuum* L.), alfalfa (*Medicago sativus* L.), cabbage (*Brassica oleracea* var. *capitata* L.), cauliflower (*B. oleracea* var. *Botrytis* L.), lettuce (*Lactuca sativa* L.), and watermelon (*Citrullus vulgarus* Schrader) should be aware that many cultivars of these crops are hosts to *Verticillium*, (Bhat and Subbarao 1999; Farr *et al.,* 1989). The adage "Do not follow solanaceous crops with strawberry" is very appropriate with this disease.

Initial symptoms of *Verticillium* wilt often appear first during periods of environmental stress, such as high temperatures and/or drought, that interrupt otherwise mild growing conditions. Fruiting also stresses plants and wilt may also be apparent during this time. Although plants may seem to recover following fruiting, they remain unthrifty and nonproductive. Typically, plants expressing wilt symptoms begin wilting with the outer-most leaves first while the central leaves remain upright but stunted. Upon sectioning, the vascular traces of primary roots and crowns show discoloration. The pattern of discoloration is different from that observed with winter freeze injury, red stele root rot, black root rot, and crown rot (Fig 1).

Severity of wilt depends on several predisposing factors: lush plant growth, high available nitrogen, level and distribution of inoculum in a field, previous crops or weeds in the field prior to strawberry, and the predominant isolate of *Verticillium* in the field (Maas *et al.,* 1989), and environmental conditions. Infected plants may occur at random in a field, sometimes in small groups, depending on inoculum distribution in the soil (Harris and Yang 1996). Disease spread from plant to plant is negligible, but the fungus can be carried from one location to another in infected plants and plant debris, by water movement, and by any agent that moves soil.

Excellent control of *Verticillium* wilt has been achieved through soil fumigation, especially with chloropicrin, mixtures of chloropicrin with methyl bromide or C_3 hydrocarbons, and methyl isothiocyanate (Maas *et al.,* 1991; Shaw and Larson 1999). Soil drenches with systemic fungicides such as benomyl and thiophenates during the growing season also have been successful. Where *Verticillium* wilt does occur, measures should be taken to minimize movement of soil and plant debris to unaffected locations. A large number of cultivars are available that are moderately to highly resistant to *Verticillium* wilt (Table 1).

2.2 Red Stele (Red Core) Root Rot

Red stele root rot, first observed in Scotland in 1920, now occurs in most strawberry-growing countries with cool, moist climates. The reader is referred to several reviews on the red stele disease and the pathogen, *Phytophthora fragariae* var. *fragariae* (Milholland 1994; Montgomerie 1977; Nickerson and Maas 1991). Prior to development of cultivars resistant to the red stele disease and of effective fungicides, this disease made strawberry production untenable in some areas where the fungus had been introduced. Strawberry is the only host for *P. fragariae* var. *fragariae* and genetically different races of the fungus exist with differential pathogenicities to culti-

vars. A closely related fungus, *P. fragariae* var. *rubi*, causes a severe root rot of rasp-
berry (*Rubus* spp.).

Phytophthora fragariae, referred to as a water mold, is soil-borne and is most
active under cool, wet conditions. The fungus produces both a durable propagule
(oospore) resulting from sexual recombination and an asexual propagule (zoospo-
rangium). Oospores are produced within infected strawberry roots and are released
into the soil when the roots decay. Oospores have been known to be viable after burial
for at least 15 years in soil. Oospores germinate by forming zoosporangia as primary
inoculum. Most zoosporangia, however, are produced on the surface of infected roots;
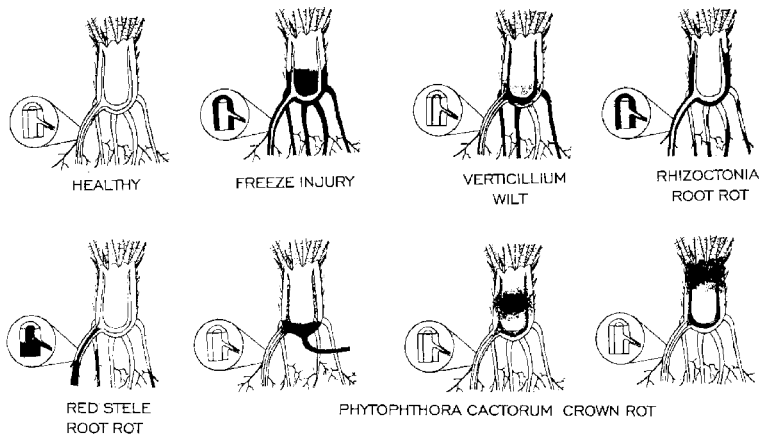
Figure 1: Diagrammatic symptom representations of freeze injury and common root and crown
diseases of strawberry in longitudinal section. Healthy crowns and roots appear white
when first sectioned. Sections of crowns and roots (represented in the circled enlarge
-ments of roots) show various patterns of discoloration of cortical, medullary, and
vascular tissues depending on the cause of injury to the plant. Freeze injury causes
browning of the crown cortex and medulla and root cortex; however, the root cortex
may also be discolored with red stele and Rhizoctonia root rot diseases. Red stele
also has a characteristic reddish-brown to blood-red of the root vascular system and
typical "rattail" appearance as secondary roots are destroyed. Verticillium wilt is
characterized by brownish discoloration of the root vascular system, which may be
evident in the crown vascular system as well. Rhizoctonia crown rot appears as brown
to black lesions in the cortex of roots and crowns. Phytophthora cactorum crown rot
symptoms are most evident at the basal stolon attachment area, or in the mid, or in the
apical portions of the crown, depending on the site of the initial infection of the
crown. (Adapted from Maas 1984; with permission of the American Phytopathologi
cal Society.)

these germinate to release motile zoospores that are attracted to secondary metabolites
exuded from healthy strawberry roots. Infection of root tips is followed by growth of

the fungus into the root toward the crown of the plant. The pathogen typically does not grow into the crown (Fig. 1). As the infection advances up the root, the stele develops a characteristic reddish brown coloration. Only darkened primary roots remain as secondary roots are destroyed, resulting in a characteristic "rattail" appearance of the root system. Above-ground symptoms of red stele root rot in plants of susceptible cultivars usually develop in spring and during fruit development. Plants show stunting and wilt-

Table 1: Disease reactions of strawberry cultivars to red stele, Verticillium wilt and powdery mildew. Information on resistance (R), susceptibility (S), or intermediate to moderately resistant (I) reactions from Hancock (1999), Hancock *et al*. (1990), Maas (1988) Maas *et al*. (1989), and cultivar release publications. Dashes indicate that data is not available; in most instances the cultivar likely is susceptible to that disease. Degree of resistance exhibited by a cultivar may vary with fungus race(s) (red stele) or isolate (Verticillium wilt) involved and with prevailing environmental conditions (powdery mildew).

Cultivar (Origin)	Red Stele	Verticillium Wilt	Powdery Mildew
Allstar (USA)	R	R	I-R
Annapolis (Canada)	—	R	—
ArKing (USA)	R	—	R
Aromas (USA)	—	—	R
Atlas (USA)	R	—	—
Benton (USA)	R	—	R
Bolero (England)	—	R	R
Brunswick (Canada)	R	—	—
Calypso (England)	—	R	S
Cardinal (USA)	S	S	R
Catalina (USA)	—	R	S
Cavendish (Canada)	R	I-R	R
Chambly (Canada)	R	—	—
Cortina (Italy)	—	—	R
Dana (Italy)	—	R	—
Delmarvel (USA)	R	R	I
Diamante (USA)	—	—	R
Earliglow (USA)	R	R	I
Emily (England)	—	S	R
Eros (England)	R	I	I
Evangeline (Canada)	—	I	—
Florence (England)	—	R	R
Gaviota (USA)	—	—	R
Glooscap (Canada)	S	—	I
Guardian (USA)	R	R	S-R
Honeoye (USA)	S	S	—
Kent (Canada)	S	S	S
Lateglow (USA)	R	R	—
Latestar (USA)	R	R	I

table 1 contd....

Laura (England)	—	I	R
Melody (England)	—	R	—
Mesabi (USA)	R	—	—
Mira (Canada)	R	—	—
Mohawk (USA)	R	—	R
Northeaster (USA)	R	R	S
Pandora (England)	S	R	R
Pegasus (England)	R	R	S
Primetime (USA)	R	R	I
Redchief (USA)	R	I-R	S-R
Rhapsody (England)	R	I-R	—
Sable (Canada)	—	I	—
Salinas (USA)	S	R	S
Shuksan (Canada)	R	R	—
Sierra (USA)	—	R	I-R
Sparkle (USA)	R	S	R
Suhong (Korea)	—	—	R
Sunrise (USA)	R	I-R	R
Surecrop (USA)	R	R	I-R
Symphony (Scotland)	R	—	S
Tango (England)	R	R	S
Totem (USA)	R	R	—
Toyonoka (Japan)	—	R	—
Tribute (USA)	R	R	R
Tristar (USA)	R	R	R
Wiltguard (USA)	—	R	—
Winona (USA)	R	—	—

ing of leaves followed by total plant collapse and death.

Phytophthora fragariae var. *fragariae* is usually introduced into new areas on infected nursery stock that did not show visible symptoms. Plant production practices in the nursery to minimize or eliminate potential risk of infection and plant inspection for low levels of infection in certification programs are essential first steps in reducing risk of introducing this disease into fruit production fields. These programs will not alleviate disease problems in soils already infested with *P. fragariae*. Since the pathogen requires free water to produce infective propagules, the risk of red stele outbreaks can be minimized by improvement of water drainage in the field. Planting on raised beds has helped to reduce the incidence of this disease in many areas. Soil fumigation may reduce inoculum in soil, but may not eradicate the fungus. Fungicides such as metalaxyl and fosetyl-Al have been used to control red stele; however, strains of the fungus have been found to develop resistance to metalaxyl (Nickerson and Maas 1991). This becomes especially troublesome in countries where fosetyl-Al is not registered for use on strawberry; otherwise the two fungicides could be used alternately to reduce the possibility of resistance developing in pathogen populations. There also is evidence to indicate that the use of metalaxyl in nursery plant production may mask root

infection symptoms without killing oospores in roots (Duncan 1985), suggesting that metalaxyl should not be used in nurseries for red stele control.

The use of resistant cultivars is by far the most reliable way to avoid red stele root rot problems. Many cultivars are available in North America and Europe that are resistant to this disease (Table 1). Disease problems can arise even with resistant cultivars. Most red stele resistant cultivars have been developed for use where only certain races of the pathogen predominate. Thus, cultivars bred and selected for resistance to a narrow spectrum of races may be highly susceptible to other races in different growing regions. Most strawberry breeding programs concerned with red stele resistance have concentrated on incorporating resistance to multiple races to extend the useful range of cultivars that may be planted in sites infested with multiple races of the red stele pathogen (Maas *et al.,* 1989). This has been complicated in the past because the nature of genetic resistance was largely unknown and crossing two red stele resistant parents often gave unpredictable results (Maas and Galletta 1989). However, recent studies have shown that resistance and pathogenicity are inherited in a gene-for-gene manner involving at least five genes for resistance (Van de Weg 1997). Molecular markers for resistance genes are being developed to assist in selection of resistance to the red stele root rot disease (Haymes *et al.,* 2000).

2.3 Black Root Rot

Black root rot has plagued strawberry growers for at least a century in nearly all strawberry growing areas of the world. Black root rot could be considered a descriptive term for symptoms associated with a number of possible causes; nematodes, fungi, cold or winter injury, herbicides, soil compaction, poor drainage, planting age, monoculture, and even fumigation (Wing *et al.*, 1994; 1995). Field symptoms of black root rot due to pathogenic causes include dwarfing or stunting of plants, shortened petioles, and small leaves, giving plants a flattened appearance that contrasts with the normal appearance of healthy tall, bushy plants. Affected plants often occur in patches, giving an uneven aspect to the planting. Affected plants often wilt at fruiting time and/or in dry weather when plants are under the greatest stress. When dug, primary roots may have small to large black cankers that involve entire roots in advanced stages of the disorder. Only the outer black root cortex is infected; it may be pulled away, leaving only the white stele present (Fig.1). There is a noticeable lack of secondary and feeder roots. These symptoms contrast with light winter injury to the crown with no root death, and those of *Verticillium* wilt, red stele root rot, and anthracnose crown rot (Maas, 1998).

Pratylenchus nematodes, especially *P. penetrans*, have been implicated as primary (*e.g.*, Chen and Rich, 1962) or as co-factors (*e.g.*, Bosher, 1954; Miller, 1956) in the development of the disease, along with *Rhizoctonia solani* Kühn and *R. fragariae*. Other fungi, such as *Pythium* (Yuen *et al.*, 1991; Wing *et al.*, 1995) and *Cylindrocarpon* species (Yuen *et al.*, 1991) have also been implicated as primary pathogens, although most reports indicate that *R. fragariae* is the main fungus pathogen involved in black root rot in North America, whereas *R. solani* has been implicated in other countries.

Complicating the involvement of *R. fragariae* in black root rot is its potential

mycorrhizal, rather than pathogenic, association with strawberry. *R. fragariae* has been shown to penetrate roots inter- and intracellularly without causing disease symptoms. However, disease rapidly developed when inoculated plants were planted in a medium less favorable for growth, suggesting a pathogenic relationship under conditions that may be more stressful to the plant (Ribero and Black 1971). In addition, at least three anastomosus groups (AGs) of *R. fragariae* may exist in the same planting and each AG strain may be active during different times of the growing year (Martin 1988; Martin 2000). In one study, for example, strain AG-I was more virulent than either AG-A or AG-G at 10°C, but strain AG-G caused the most disease at 24°C soil temperatures (LaMondia and Martin 1989).

Much of the early work on controlling black root rot centered around the use of fungicides, nematicides, and general biocides to treat black root rot soil. Soil fumigation for control of black root rot has proven to be problematic as a "boomerang" effect may occur following soil sterilization. With the lack of competitive organisms, *Rhizoctonia* or other aggressive and fast growing pathogens introduced into the sterile soil environment may proliferate and cause even more disease and more rapidly than if the fumigation had not been done (Wing *et al.,* 1995). A fumigation combination of nematicide plus fungicide has given some control of black root rot. Either fumigant used alone gave only intermediate control, suggesting that *P. penetrans* and *R. fragariae* play equal roles in the black root rot syndrome (Miller 1956).

Contemporary work on controlling black root rot is centered around the concept of "healthy" soils (*see:* "Healthy" Soils). Pertinent to the development of this concept are the following quotations: "The overall picture one obtains of the rhizosphere is that of a complex of micro-organisms, some harmless, others beneficial, and still others with pathogenic propensities, in equilibrium but susceptible to the influence of various factors, such as moisture, soil treatment and nature and age of the plant." and that "By controlling the direction of change of this equilibrium and the intensity of microbial activity in the root zone by means of appropriate soil amendment, it appears to be possible to control certain root diseases. The influence of these treatments on the nutrition and vigor of the plant itself may be an important factor contributing to its resistance to disease" (Katznelson and Richardson 1948).

We have not progressed to the point with strawberry black root rot that it can be controlled with organic soil amendments. However, some progress has been made. In one series of studies it was found that rotation with rye resulted in low recoveries of *Rhizoctonia* and *P. penetrans*. Even lower populations of *P. penetrans* were observed following rotations with sorghum-sudan grass, 'Saia' oat, or canola. When strawberry followed alternate crop rotations, the greatest recovery of *Rhizoctonia* was with canola and of *P. penetrans* with 'Garry' oat. Overall, however, the greatest recovery of *Rhizoctonia* was obtained when strawberry followed strawberry. 'Saia' oat or 'Triple S' sorghosudangrass decreased *P. penetrans* nematode numbers in strawberry roots and decreased black root rot severity. Only 'Garry' oat reduced infection of roots by *Rhizoctonia* (Elmer and LaMondia 1999; LaMondia 1994; 1999).

Attempts to identify or develop strawberry cultivars resistant to black root rot have not been successful. Part of the reason is that the causes of black root rot differ in various regions of the strawberry-growing world and that environment plays an essen-

tial role in the development of the disease. In theory, resistance to *R. fragariae* could be combined with resistance to *Pythium* spp. and also to *P. penetrans*. However, this has not been accomplished and likely will not be in the foreseeable future.

Biocontrol is a possibility for controlling black root rot. *Trichoderma harzianum*, which parasitizes *R. solani* has shown some effect in reducing black root rot in Israel (Elad *et al.,* 1981). Applied in commercial nurseries and fruit production fields, *T. harzianum* reduced disease 18-46% in nursery plots, and resulted in a 20% increase in fruit yields when applied to both nurseries and fruit production fields. A more recent biocontrol innovation is the biological fungicide Deny (Market VI, Shawnee, Kansas). Applications of Deny as a plant drench and through irrigation has provided control of black root rot due to *Rhizoctonia* infection and may also be effective against infection by *Pythium* and *Fusarium* (O'Dell 1999).

Some generalities for black root rot management that were formulated in 1924 (Coons 1926) still bear relevance today, with editorial comments in brackets: (i). Avoid soils that have previously borne grain crops [but look into some of the newer cover crops]; (ii). Strawberries should not follow strawberries [black root rot is a re-plant disorder]; (iii). Use plants with sound, bright roots [always a good idea]; (iv). Use winter protection with a proper mulch application; (v). No chemical controls are advised [but, if you fumigate, be aware of a possible fumigation boomerang effect with soil-borne pathogens]; (vi). Badly affected plantings may benefit from light applications of well-rotted [composted] manure or light applications of sodium nitrate or ammonium sulphate; (vii). The best sites are on sandy, slightly acid soil and allow for adequate drainage of water; (viii). Set fresh transplants as quickly as possible, or use cold stored plants that have been properly stored [use of plug plants should reduce the possibility of pathogen transport on roots].

2.4 *Fusarium* wilt

Fusarium wilt, also called *Fusarium* yellows, is a systemic disease caused by *Fusarium oxysporum* f. sp. *fragariae* that is reported to occur only in Australia, Korea, and Japan (Maas 1998), but possibly also in Argentina (Mena *et al.,* 1975). The strawberry form of *F. oxysporum* is pathogenic only to strawberry and the disease can be severe, causing as much as 40-50% plant loss (Kim *et al.,* 1982; Winks and Williams, 1965). *Fusarium* wilt is considered the most devastating strawberry disease in Japan where susceptible cultivars such as 'H k wase' are grown (Oda 1991). *Fusarium* wilt is a soil-borne disease that begins as an infection of roots and crowns and becomes systemic. Symptom development is favored by high temperatures and high stress during fruiting, which causes leaves of infected plants to wilt and die rapidly. Sudden plant collapse is similar to that caused by some other wilt diseases. Leaf chlorosis may develop, especially under cool weather conditions. Crowns show distinct reddish-brown discoloration that may be confused with symptoms of anthracnose crown rot (*see:* Anthracnose). However, unlike anthracnose crown rot, the lower crown tissues may be decayed completely by a dry rot, and vascular discoloration may extend into the leaf bases as the disease advances (Winks and Williams, 1965). Slimy, white or pink masses of conidia may be produced on infected plant parts. Chlamydospores are produced in

infected plant tissue and are released into the soil where they may remain viable for long periods of time. In Japan, nursery plants become infected in the field, and when transplanted to tunnel culture, the disease progresses under warm temperature conditions (Oda 1991). Other *Fusarium* spp. commonly are isolated from strawberry roots in other locations in the world and may be associated with root disease, but they do not cause wilts of strawberry plants.

Fusarium wilt has been controlled by cultural means, such as the addition of large amounts of potassium oxide or lime to the soil, or by covering fields with black vinyl or silver polyethylene films. Soil fumigation of nursery beds with methyl bromide also has been used to reduce infection levels (Oda 1991). There is evidence that biological controls such as soil amendment with chitin-degrading microbes (Ouchi *et al.,* 1992), *Trichoderma harzianum* (Moon *et al.,* 1995), and non-aggressive strawberry isolates of *F. oxysporum* (Tezuka and Makino 1991) may protect plants from infection. Since the disease is systemic, it may be carried asymptomatically in plants from one location to another. The disease also is more severe when strawberry crops are grown year after year in the same location without alternating with other non-host crops (Okayama *et al.*,1988). Apparently strains of *F. oxysporum* pathogenic to tomato, watermelon, cabbage, and passionfruit (*Passiflora edulis* Sims) are not pathogenic to strawberry and strawberry pathogenic strains are not pathogenic to the other crops (Cho and Moon, 1984; Horimoto *et al.,* 1988a, b; Okayama *et al.,* 1988; Winks and Williams, 1965).

Cultivars vary in susceptibility to *Fusarium* wilt; cultivars Fukuba, Hogoyoku, Senga Sengana, Senga Gigana, Himiko; Kurumae 38; Yachiyo, Daehak 1, Shikinari, Toyonoka, Hatsukuni, and Suhong are resistant. In addition, somaclonal variants from leaf-derived callus have been regenerated that also are resistant to *Fusarium* wilt (Toyoda *et al.,* 1991). There appears to be sufficient resistance in strawberry that this disease should be managed successfully by combining plant resistance with cultural management in the plant nursery and in tunnel and field fruit production systems.

2.5 Crown rot

Crown rot, caused by *Phytophthora cactorum*, is an important strawberry disease in Europe and other temperate to subtropical regions. The disease is apparently caused by a distinct pathotype of *P. cactorum*; only isolates from diseased strawberry crowns are pathogenic to strawberry only and isolates from plants other than strawberry are not pathogenic to strawberry (Lilja *et al.,* 1998; Lederer and Seemüller 1992; Seemüller 1998). Isolates of *P. cactorum* that cause crown rot apparently also are distinct from those that cause leather rot (*see:* Leather Rot), although both diseases may occur simultaneously in the same planting (Seemüller and Schmidle 1979). Other species of *Phytophthora* also have been implicated in causing crown rots which may be sporadic in occurrence or of local importance.

Crown rot often appears in the field as a sudden wilting, or collapse, of plants. Early symptoms of youngest leaves turning bluish-green also may be apparent. When lifted, diseased plants may easily break at the crown. Extensive internal dark-brown crown necrosis and vascular disintegration is characteristic of this disease. Crown ne-

crosis may begin at the upper part and progress basipetally, or start from the point of its stolon adhesion and progress acropetally (Fig. 1). Roots typically are not infected but die when the upper part of the plant dies. Plants that do not succumb and appear to recover remain stunted and unproductive. Stunting may be confused with symptoms of black root rot or *Verticillium* wilt *(see:* Black Root Rot and *Verticillium* Wilt). Cold-stored, or frigo, plants may be killed very quickly following transplanting or may carry latent infections into the new field (Pettitt and Pegg 1994).

Oospores in soil or in strawberry plant debris provide initial sources of inoculum. The oospores germinate to produce zoosporangia and zoospores which infect plants, usually at wound sites. The fungus requires warm temperatures and prolonged wetness periods for inoculum production and for infection to occur. Infection of freshly dug runner plants that are wounded during transplanting may occur, but many infections occur at stolon stumps. Crowns that have been damaged by low temperature injury following severe frosts prior to plant harvesting or by low temperatures during cold storage are predisposed to infection (Bell *et al.,* 1997; Pettitt and Pegg 1994). Planting time also affects symptom development; plantings established in spring to early summer are more quickly affected than those established in mid-summer, and plantings established in late summer usually do not appear affected until the following spring, especially when plants are under stress during fruiting. Disease development is accelerated by high temperatures and water stress (Molot and Nourrisseau 1974; Lederer and Seemüller 1992). Water stress often occurs following transplanting and during fruit development. Low temperatures delay disease progression, but does not halt it. Infections may become latent as temperatures become lower and especially during cold storage.

In general, site and soil conditions should be managed to ensure rapid water run-off and prevention of soil water-logging. Planting in low, wet areas should be avoided as should fields that have a history of crown rot disease. Healthy runner plants should be used for transplantation.

Avoiding transplants that have been injured during cold storage or that have latent infections is difficult. Use of transplants propagated from runner-tip plantlets (*i.e.,* plug or container-grown plants) is a reliable means to ensure freedom from infection by *P. cactorum* in plant material (Harris *et al.,* 1997). Fungicides, such as metalaxyl and fosetyl-Al, applied as plant dips or drenches have been recommended as preventative measures (Seemüller 1982). However, the same cautions should be taken in using these systemic fungicides for control of crown rot as for the control of red stele root rot and leather rot to prevent development of resistant strains of *P. cactorum* (*see* : Red Stele Root Rot and Leather Rot). Once the fungus has established in the crown it is nearly impossible to eradicate the infection (Molot and Nourrisseau 1978).

Cultivars vary in their resistance to crown rot, but the degree of resistance may be slight and depend on predisposing factors and resistance screening methods (Bell *et al.,* 1997; Hancock *et al.,* 1990). For example, 'Cesena', known to perform well in contaminated fields, was found to be highly susceptible following artificial inoculation (Colombo and Maltoni 1996). Cultivars Grandee, Senga Sengana, Yalova 104, Hapil, and Savio were reported resistant to crown rot (Bell *et al.,* 1997). However, in the same study, 'Redgauntlet', 'Tamella', and 'Cambridge Favourite' were found to

be moderately resistant, whereas in another study, these cultivars were determined to be highly susceptible to crown rot (Pettitt and Pegg 1994). In yet another study, 'Redgauntlet', Bogota', and 'Lambada' were found highly resistant, 'Senga Sengana', Karina', and 'Elsanta' were moderately resistant, and 'Avanta' and 'Tamella' were highly susceptible to crown rot (van Rijbroek *et al.,* 1997).

The old cultivar Merton Dawn is considered to be very resistant to crown rot (Pitrat and Risser 1977). Of some of the newly released cultivars from the Horticultural Research Institute in England, 'Gerida' is reported to be highly resistant, 'Pegasus', 'Pandora', 'Bolero', and 'Florence' are described as having moderate, or "useful", resistance, and 'Alice' has some resistance to crown rot. 'Catalina', 'Cal Giant 3', and 'Cal Giant 4', from the New Fruit West Corporation, California, and 'Patty', from the Istituto Sperimentale per la Frutticoltura, Forli, Italy, should also be included in a listing of potentially useful cultivars where Phytophthora crown rot may be encountered.

3. Foliage diseases

Strawberry plants are subject to many diseases of the leaf, but the most important and destructive of these are bacterial angular leafspot, powdery mildew, Alternaria black leaf spot, *Mycosphaerella* leaf spot, Phomopsis leaf blight, leaf scorch, and irregular and black leafspot phases of anthracnose *(see:* Anthracnose). Leaf pathogens can overwinter in persistent living or dead leaves and petioles, and form spores in warm weather to initiate new infections. These foliar pathogens are disseminated by wind and/or rain and irrigation water splash. Leaf diseases may occur as soon as young leaves develop in the spring and continue until dormancy occurs in the fall. Generally, these diseases do not cause economic damage; however, under epiphytotic conditions in perennial culture systems, leaf injury becomes significant and plants are weakened to the point that they may become more susceptible to other diseases and winter damage. In addition, reduced initiation of floral buds in the fall may occur. In annual culture systems, foliar diseases may reduce plant vigor, fruit yield, and fruit quality. Generally, except for powdery mildew and bacterial angular leafspot, these diseases do not directly affect fruit, and, except for bacterial angular leafspot, foliar diseases in general are managed with fungicide applications and the use of disease-resistant cultivars.

3.1 Bacterial angular leafspot

Bacterial angular leafspot, caused by the bacterium *Xanthomonas fragariae*, was first described in 1962 from Minnesota, USA and has since been established in many strawberry-producing countries of the world (reviewed in: Maas *et al.,* 1995). Its spread to other countries was likely, as with many pathogens, as systemic infections in imported planting materials. Typical symptoms of this disease appear first as small angular water-soaked lesions on the lower leaf surface. These spots enlarge, but remain angular in outline and usually delimited by small leaf veins. Although appearing green, the lesions are characteristically translucent when examined with transmitted light. Under moist conditions, a characteristic slimy bacterial ooze will develop from lesions on

under sides of leaves, which later dries to a whitish to brownish film. Translucency of lesions and presence of bacterial ooze distinguish this disease from all other common leaf diseases. Heavily infected leaves may become tattered, torn, and may eventually die. Occasionally, the calyx may become infected, causing sepals to become unattractive and reduce marketability of the fruit. Strawberry is the only natural host for this bacterial pathogen. Systemic spread of the bacterium through the vascular system to other plant parts, including developing runner-tip plantlets may occur. In some cases, the vascular system fails, resulting in wilting and death of the plant. The collapse of plants may resemble plant collapse resulting from wilts caused by *Colletotrichum* spp. and *P. cactorum*; however, no discoloration of the crown tissue is associated with bacterial angular leafspot disease.

X. fragariae bacteria are highly resistant to desiccation and can survive from season to season in old, persistent leaves and dead plant debris buried in soil. The bacteria do not move in soil, nor can they survive free in soil. Infection is favored by cool temperatures; around 17°C daytime and 2°C nighttime temperatures are ideal, with periods of moisture from rain or irrigation. Bacteria may be spread by water splash, and by animals, machinery, and humans that come into contact with bacterial ooze. Quite often, use of overhead irrigation in early spring for protection of flowers from freeze damage exacerbates the development of this disease. Importantly, bacterial angular leafspot may be spread from affected plantings and nurseries, to new locations through transport of systemically-infected transplants.

Control of bacterial angular leafspot is mainly through use of certified planting material. Copper-containing bactericides and antibiotics such as streptomycin have been used, but with less than satisfactory control, except under light infection pressure situations. In addition, repeated and heavy use of copper fungicides may result in severe phytotoxicity. No cultivars have been developed that are resistant to this disease. However, progress is being made in identifying germplasm resistant to the known genotypes of *X. fragariae* and resistant germplasm is expected to be available to world strawberry breeders in 2002 (Maas *et al.,* 2000; Maas *et al.,* unpublished).

3.2 Powdery mildew

Powdery mildew occurs on strawberry plants in all areas of the world and is one of the earliest recorded diseases of strawberry, first technically described in the 1850s. Powdery mildew can affect flowers and fruit as well as leaves. It also can be a serious problem in plant nurseries. Although powdery mildew is inhibited by rainy, wet conditions, it thrives under short day or low light intensities, and relatively cool temperatures (14-27°C) accompanied by high relative humidity. Severe foliar infection may lead to necrotic lesions and defoliation resulting in reduced photosynthetic activity which may affect fruit yields. Direct infection of flowers and fruit can result in large yield losses (Maas 1998; Peries 1962). Powdery mildew can be particularly severe in closed or protected culture systems and in cool coastal growing areas. Strawberry apparently is the only host for this form of powdery mildew, although other plant species have been experimentally infected (Peries 1962). Powdery mildews on other crops or weeds do not transfer to strawberry. The fungus, *Sphaerotheca maculata* f.

sp. *fragariae*, is an obligate parasite and survives from season to season only within living tissues of its host (Peries 1961).

Symptoms of powdery mildew infection are very distinctive. White patches of mycelium develop on lower leaf surfaces which may continue to develop to cover the entire surface. The leaf edges characteristically curl upwards. Mycelial growth on lower leaf surfaces may be accompanied by purple to reddish blotches. Flowers may become enveloped in mycelium and are either deformed or killed. Infection may also cause abnormal pollen production. Developing fruit in all stages may become infected; immature fruit become hard and fail to ripen and ripe fruit remain soft and pulpy. Profuse white, powdery sporulation may occur on leaf and fruit surfaces. Occasionally, small dark cleistothecia, or fruiting bodies, of the fungus may develop along with mycelium on leaves.

The first step in controlling powdery mildew is the use of resistant cultivars, of which there are many that are adapted to most strawberry-growing areas of the world. Failing that, nursery plant and fruit growers should establish plantings with disease-free transplants, and fungicides must be applied at the first signs of infection. Fungicidal control is problematical in tunnel, glasshouse, and annual production systems where re-entry and harvest limitations are imposed with use of most fungicides. In addition, in field plantings, the powdery mildew fungus has developed populations resistant to many formerly effective fungicides. Since the fungus grows within host tissues, only systemic fungicides will have eradicative effects. Protective fungicides will not eradicate infections, but may slow the progression of powdery mildew in a field and help prevent the occurrence of a potential epidemic. Fungicide coverage for control of flower and fruit infection should be applied as early as possible when flowering begins and should continue at regular intervals throughout the fruiting season in annual plantings and throughout the growing season in perennial plantings. Late season fungicide applications in perennial plantings may help reduce the amount of inoculum carryover from one year to the next.

3.3 *Alternaria* black leaf spot

Alternaria black leaf spot occurs in Europe, Korea, and Japan (Maas 1998; Roudeilac and Veschambre 1987; Wada *et al.,* 1996). The pathogen is a distinct pathotype of *Alternaria alternata* which has a highly selective pathogenicity to strawberry cultivars. The host range of the pathogen apparently is determined by the action of a host-specific toxin, AF-toxin, which is secreted during spore germination on host tissue (Otani *et al.,* 1989). Whether the pathogen *Alternaria alternata* f. sp. *fragariae* reported in New Zealand is the same as the AF-toxin producing strawberry pathotype, *A. alternata* f. sp. *alternata*, is not clear (Dingley 1970; Wada *et al.,* 1996); however, recent evidence suggests that the strawberry pathotype does occur in New Zealand (Takahashi *et al.,* 1997). To avoid confusion in common names of strawberry diseases, the term "black spot", as often used for this disease, should be modified with the term "Alternaria" in technical communications because anthracnose fruit rot is also often called "black spot".

Alternaria black leaf spot can occur in epidemic proportions under conditions

of heavy and constant rain, high relative humidity, and daily temperatures from 20-25°C in combination with susceptible cultivars. Although *Alternaria* species may attack fruits, this is of negligible occurrence. Spots on upper surfaces of leaves are small to medium in size (2-5 mm diameter), mostly round in shape, but becoming irregular in outline along leaf margins. Lesions are brown and surrounded by dark reddish purple (violet) halos. Lesions on the lower leaf surface appear grayish-brown. Eventually spots coalesce and large necrotic areas form, often resulting in defoliation that may become severe.

Although fungicides are recommended for control of Alternaria black leaf spot (Bal *et al.,* 1983; Roudeillac and Veschambre 1987), the principal means of control is to plant resistant cultivars. Susceptibility to the disease and to AF-toxin is determined by a single gene pair with semi-dominance (Takahashi *et al.,* 1992), thus breeding and selecting for disease resistance has been successful. In addition, older cultivars apparently selected in the absence of this disease are also highly resistant to it. Cultivars reportedly resistant to Alternaria black leaf spot include H k wase, Reiko, Akita Berry, Himiko, Apollo, Cambridge Favourite, Senga Gigana, Senga Sengana, Addie, Carezza, Clea, Cortina, Darsidor, Egla, Gea, Idea, Linda, Marmolada, Miranda, Nike, Pajaro, Sella, Selva, and Teodora, and susceptible cultivars include Dana, Sivetta, Tamella, Tenira, Elista, Bogota, Tago, Morioka-16, Miss, Cesena, and Talisman (Cavanni *et al.,* 1996; Roudeillac and Veschamber 1987; Takahashi 1993; Takahashi *et al.,* 1997). 'Gorella' and 'Redgauntlet' have been reported susceptible in Europe (Roudeillac and Veschamber 1987), but resistant in Japan (Takahashi *et al.,* 1997). 'Elsanta' has been reported both resistant (Cavanni *et al.,* 1996) in Italy and susceptible in other European countries (Roudeillac and Veschamber 1987). Thus, it remains problematical whether all Alternaria black leaf spot disease is caused by the AF-toxin producing strains of *A. alternaria*.

3.4 Leaf scorch

Leaf scorch, caused by *Diplocarpon earlianum* [anamorph *Marssonia fragariae* (Lib.) Kleb.], occurs in most strawberry-growing regions and occurs only on species of *Fragaria* (Sutton 1998a). Losses to leaf scorch may range from negligible to severe, depending on cultivar susceptibility, culture system, and environmental conditions. Symptoms on leaves are numerous small (1-5 mm diameter), irregular, purplish spots, the centers of which become brownish. Tissues between spots turn purplish to bright red, depending on cultivar. Spots may coalesce to cover leaflets, turning them purplish to reddish to brown (*i.e.*, appearing scorched). Centers of spots do not turn gray or white as with lesions of leaf spot. Scorch lesions also can develop on stolons, petioles, flower trusses, sepals, and flowers. The disease is favored by long periods of leaf wetness (12 hours or more) at 15-25°C, although it can develop at or near freezing temperatures. Progress of leaf scorch is delayed by leaf removal during crop renovation in perennial plantings and by hot and dry weather conditions. The fungus survives over winter in infected leaves and leaf debris, providing inoculum for new infections in the spring.

Cultivars resistant to leaf scorch are available for many areas, but, if suscep-

tible cultivars are grown in an area with a history of leaf scorch and the likelihood of long wetness periods, fungicide applications are necessary during the flowering period and in late summer and fall. The economic threshold for fungicide application has been suggested for when 25% of leaf area is involved with leaf scorch lesions during the peak of an epidemic in late summer and fall.

3.5 Leaf spot

Leaf spot, caused by *Mycosphaerella fragariae* (anamorph *Ramularia brunnea* Peck., syn. *R. tulasnei* Sacc.), also known as common leaf spot, Mycosphaerella leaf spot, and Ramularia leaf spot, occurs world wide only on species of *Fragaria*. Prior to development of resistant cultivars, this disease was considered the most damaging to strawberry, and it still ranks as an important disease where susceptible cultivars are favored. Leaf spot affects leaves, petioles, stolons, fruit trusses, and fruit. Plant vigor, fruit yield, and fruit quality are reduced by this disease. Lesions on leaves begin as small, deep purple, round to irregularly shaped lesions on the upper leaf surface. The lesions enlarge to 3-6 mm in diameter. On young leaves, the spots remain light brown in the center, but on older leaves, the centers of lesions change from brown to grayish and finally to white. A reddish purple to rusty brown halo surrounds the necrotic areas. Lesions on heavily infected leaves may coalesce and cause defoliation. Symptom expression depends on cultivar, pathogen strain, and day-night temperatures. During warm, humid weather uniformly rusty brown lesions lacking the typical light colored centers may predominate on young leaves. When centers of leaf spot lesions remain brown, they may be confused with those of leaf blight.

      The fungus survives over winter in perennial plantings in infected plant tissues and as sclerotia in dead infected leaves. Spore inoculum produced in spring is disseminated by wind and water movement. Timely applications of protective fungicides are necessary for susceptible cultivars and disease-free nursery transplants are recommended for control of leaf spot, in addition to use of leaf spot resistant cultivars.

3.6 Leaf blight

Leaf blight, also called *Phomopsis* leaf blight, caused by *Phomopsis obscurans* [syn. *Dendrophoma obscurans* (Ellis & Everh.) W. H. Anderson], may be a serious disease in some localities. The pathogen also may severely damage stolons and fruit trusses (Maas 1985) and causes a ripe rot of fruit. In exceptional cases, the disease may cause sufficient late-season leaf destruction in perennial plantings to weaken plants and reduce fruit yields the following spring. Large lesions formed on stolons may cause unrooted daughter plants to wither and die. Infection of fruit trusses early in the season has caused trusses and the fruit they bear to die. Leaf lesions begin as nearly circular, reddish purple spots that later develop gray centers. At this point, leaf blight lesions closely resemble those of leaf spot. Older lesions develop an outer purple, red, or yellow halo that shades into the normal green of the leaflet, a inner light brown halo surrounding a dark brown inner area. Older lesions along major leaf veins develop into large characteristic V-shaped necrotic areas; however, these lesions may also be

seen in leaves affected by anthracnose. Young leaves and stolons are more susceptible to infection than are mature structures. Disease initiation and development is favored by warm temperatures (26-32ºC) and a wetness period of at least 72 hours (Eshenaur and Milholland 1989).

In perennial culture systems, *P. obscurans* overwinters in infected living leaves. In annual systems, the fungus may be introduced into new plantings with infected nursery transplants. *Fragaria* species are the only known hosts of *P. obscurans*. Regularly scheduled fungicide applications for other diseases in spring, summer, or fall seem to keep leaf blight in check. Cultivars vary in susceptibility to this disease, but apparently none are highly resistant.

4. Fruit diseases

A number of diseases affect strawberry fruit; however, only those caused by *Botrytis cinerea, Phytophthora cactorum* and *Colletotrichum* species are addressed here. *Botrytis* rot and fruit anthracnose constitute the major strawberry fruit disease problems in the world and result in the greatest fruit losses and they also are responsible for the greatest amounts of, and expenditures for, fungicides used in most nations for strawberry fruit protection. Leather rot, although sporadic in occurrence, is included in this discussion because of its potential to be devastating to fruit producers. Other diseases of fruit, such as powdery mildew and rot, are discussed under leaf diseases.

4.1 *Botrytis* fruit rot (gray mold)

Botrytis cinerea colonizes senescent flower parts and usually remains quiescent until fruit have developed to a point that they also become physiologically senescent (*i.e.*, red ripe) at which time *Botrytis* hyphae become active in the fruit, and the production of gray, powdery spore masses is noted on the surface of the fruit lesion. Rot development is rapid and quite common in the field, but some apparently unaffected fruits may not develop lesions until after harvest. When conditions favor early development of *Botrytis* rot, flowers and green fruit may become involved in active infections, resulting in loss of flowers and young fruit.

Since 1960, recommendations for control of *Botrytis* fruit rot have emphasized applications of fungicides during bloom (Cooley *et al.,* 1996; Powelson 1960; Wilcox and Seem 1994). Except under very unusual circumstances with Junebearing strawberries, post-bloom fungicide coverage for control of *Botrytis* fruit rot is unnecessary. However, with day-neutral strawberries that bloom and develop fruit over an extended period of time (up to three months for some cultivars in California, Florida, and Spain) fungicide applications may be necessary during the entire fruiting period to control pre- and post-harvest *Botrytis* fruit rot (Blacharski *et al.,* 2001; Mertely *et al.,* 2000; Strand 1993).

Primarily a saprophyte, but also a facultative parasite, *Botrytis cinerea* is universally present in most environments. In strawberry fields however, spore inoculum originates from within the planting. Young leaves become infected as they develop and the fungus remains quiescent until the leaves begin to senesce. Therefore, the

fungus overwinters in infected leaves and produces spores during the spring bloom period. Infections of leaves occur in the summer and fall to complete the annual cycle (Sutton 1998b). Attempts have been made to reduce the presence of inoculum in leaves by defoliation and renovation following harvest (Daugaard 2000), removal of leaf trash, mowing and removing leaves in fall, and fungicide applications applied to leaves prior to bloom and in late summer or early fall (Cooley *et al.,* 1996; Mertely *et al.,* 2000; Sutton 1998b) with some effectiveness. However, in all studies on reduction of *Botrytis* inoculum and disease incidence, the recommendation to apply fungicides to flowers remains to be the most appropriate management strategy for *Botrytis* rot control.

Additional measures that can be taken to minimize losses due to *Botrytis* fruit rot include prevention of excessive vegetative growth by regulating plant density, timely nitrogen applications, removal of infected fruit from the field, harvesting fruit before it is fully ripe to avoid bruising and other injuries, prompt transfer harvested fruit to refrigerated storage reduces the incidence of *Botrytis* fruit rot.

Some variation in susceptibility to *Botrytis* rot exists among cultivars; however, any significant resistance is quickly leveled by abnormally high rainfall and humidity during flowering. Cultivars Allstar, Bounty, Canoga, Columbia, Holiday, Honeoye, Lester, Northwest, Olympus, Shuksan, Tago, Tioga, Totem, and Tyee (Hancock *et al.,* 1990), Eros (NSA Plants Ltd., 1994 Launch Bulletin), Camarosa (Xiao *et al.,* 2001), and Earliglow (Maas and Smith 1978) reportedly exhibit resistance to *Botrytis* fruit rot. Since genetic resistance in strawberry to *Botrytis* infection apparently is multigenic and has a very low general combining ability, there has been little success in breeding and selecting cultivars resistant to this disease.

4.2 Leather rot

Leather rot, caused by *Phytophthora cactorum*, usually is not a serious problem, but on occasion may cause considerable loss of fruit. In addition to direct loss of fruit, fruit with leather rot has a distinctly unpleasant odor and taste that may be imparted to processed fruit products. Therefore, the level of tolerance for leather rot in the field is very low compared to other fruit-rot diseases. *P. cactorum* also causes a serious crown rot, but apparently the fruit rotting and crown rotting forms of *P. cactorum* are caused by two distinct pathotypes, although the rots may occur simultaneously (Madden *et al.,* 1991) (*see* : also: Crown Rot).

Fruit at any stage of development may be infected by *P. cactorum*. Diseased areas on green fruit usually are dark brown, but on occasion may remain green but with a brown margin. As the rot spreads, the entire fruit turns brown and develops a rough texture. Infected fruit appear and feel leathery. Fully mature fruit when infected may change little in color or may discolor from brown to dark purple. Both green and mature fruit eventually dry to form hard, shriveled mummies containing oospores, the reproductive and winter survival structures of the fungus. The unpleasant odor and taste of infected fruit are diagnostic symptoms for this disease. In spring, the oospores germinate and produce sporangia which, in the presence of free water, produce motile zoospores. Zoospores splash or reportedly swim to developing fruit and readily infect

fruit in the presence of at least a film of water. Rapid infection may occur under optimum conditions of temperature (17-25°C) and moisture (2 hours) and a second crop of sporangia may be produced on fruit within five days, rapidly spreading the disease (Ellis *et al.,* 1998).

Control of leather rot requires an integration of several strategies; site selection and maintenance to enhance drainage, elimination of standing water, planting on elevated beds, use of rough-textured mulch around plants, and fungicide applications. Since *P. cactorum* spreads by means of motile zoospores, moving water and rain quickly spread the disease. Rough-textured mulch, such as straw, reduces leather rot incidence by holding fruit above standing water and soil and reduces the splashing of water droplets that may bear sporangia containing zoospores of the fungus (Madden and Ellis 1990). Plastic mulch may actually intensify disease incidence by puddling of water on the plastic and by increasing secondary splash dispersion of impacting water droplets.

Unfortunately, many fungicides used for control of other fruit-rot diseases such as *Botrytis* rot and anthracnose do not affect leather rot. Captan and thiram may provide some control, but they will not provide adequate control if conditions are favorable for an epidemic to occur. In addition, the use of these fungicides may be restricted during the fruit harvesting period. The systemic fungicides metalaxyl and fosetyl-Al are highly effective for the control of leather rot and a single pre-bloom application has been shown to be effective (Ellis *et al.,* 1998). Both fungicides have also been used for suppression of red stele root rot. Because resistance to metalaxyl may occur readily, it is advised to alternate its use with another, unrelated, fungicide such as fosetyl-Al to minimize the threat of *P. cactorum* developing resistance to metalaxyl (Straub 1991).

Cultivars may exhibit differences in susceptibility to leather rot (Olcott-Reid and Moore 1995); however, it is not clear whether these differences are due entirely to genotype responses of fruit to infection, or are mitigated by plant architecture and environmental effects (Madden *et al.,* 1991).

4.3 Anthracnose

Anthracnose of strawberry, described first in the United States in 1931, was determined to be caused by a new species of *Colletotrichum, C. fragariae* Brooks (Brooks 1931). The disease affects stolons and petioles (Brooks 1931), and crowns of plants (Brooks 1935). Since these early studies, anthracnose diseases of strawberry have been described as caused by *C. acutatum* (teleomorph *Glomerella acutata* Guerber & Correll), *C. gloeosporioides* (Penz.) Penz. & Sacc. [teleomorph *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk.], and *C. dematium* (Pers.) Grove (Howard *et al.,* 1992; Maas 1998). *C. dematium* only occasionally causes strawberry fruit rots; however, the former three species have become of worldwide importance in strawberry culture since around 1990.

Anthracnose fungi may attack any part of the strawberry plant. Typical lesions on petioles and fruit trusses are dark brown, elongated, and slightly sunken. In the nursery and in matted-row beds, lesions may girdle the stolon, resulting in wilting and death of the young unrooted daughter plant distal to the lesion. Infected flowers and

buds may become dry and withered. Leaf lesions can become large, irregular, and brown, but sometimes also V-shaped (irregular leaf spot), resembling those of leaf blight (caused by *Phomopsis obscurans*), or they may form smaller (0.5 to 3 mm in diameter) gray to black lesions (anthracnose leaf spot). Anthracnose leaf spot should not be confused with Alternaria black leaf spot which may exhibit similar appearing leaf and petiole lesions. Similar appearing stolon and fruit truss lesions may also be produced by other pathogens; *e.g.*, *Diplocarpon earliana*, *Mycosphaerella fragariae*, *Verticillium* spp., and *Phomopsis obscurans* (Maas 1985)*.*

Fruit infection can be devastating during periods of favorable temperature and presence of moisture, either in the form of rain or high relative humidity. Circular, firm, sunken lesions that typically become black may develop on ripening fruit. Green as well as ripe fruit may be infected. Lesions on green fruit are very similar to those caused by *Rhizoctonia* species. However, lesions on ripe fruit are easily distinguished from lesions caused by other fruit-rotting pathogens especially when slime-like masses of pinkish spore masses are produced on lesions.

Crown rot occurs when sufficient numbers of spores are washed onto the central buds, or when the fungus grows from other lesions into the crown. Transplants that became infected in the nursery may appear normal for a while, but suddenly wilt and die. Infected crowns typically have a reddish-brown, firm rot, or reddish-brown streaking in their interior; the reddish tint is diagnostic for anthracnose crown rot. Other pathogens and conditions may cause darkening of crown tissues and crown death, but the reddish hue is absent.

Root necrosis may accompany anthracnose crown rot, flower blight, and fruit rot. Severe outbreaks of root necrosis, caused by *C. acutatum*, have occurred in Israel wherein entire beds of transplanted strawberry plants showed symptoms of stunting and chlorosis followed by collapse of the plants (Freeman and Katan 1997). Affected roots appear dark brown and decayed, symptoms that could be confused with those associated with diseases like black root rot and *Verticillium* wilt and necessitating accurate pathogen determinations.

Inoculum sources of anthracnose fungi are ubiquitous. Both *C. gloeosporioides* and *C. acutatum* have large host ranges, are genetically very heterogeneous, and apparently exhibit no host specificity (*see* in: Farr *et al.,* 1989; Freeman *et al.,* 1998; 2001). Weeds and crop plants may provide sources of inoculum for infection of strawberry. *C. acutatum* apparently can survive in soil (Eastburn and Gubler 1990; Freeman and Katan 1997) and in mummies of infected fruit (Wilson *et al.,* 1992) and *C. gloeosporioides* can survive over summer in infected strawberry crowns (Ureña-Padilla *et al.,* 2001).

Anthracnose crown rot is regarded as a nursery problem wherein infected nursery plants transplanted to fruit-production fields rapidly show symptoms of the disease (Freeman and Katan 1997; Freeman *et al.,* 1997; Howard *et al.,* 1992; McInnes *et al.,* 1992). Very little spread of crown rot occurs in the production field (Howard *et al.,* 1992); however, new root infection and plant stunting can occur if new transplants are placed in the same locations of removed diseased plants (Freeman and Katan 1997). Fruit infection can spread rapidly in the field. Profuse conidial production occurs between 15 and 30°C with maximum production near 25°C (King *et al.,* 1997). Conidia

are dispersed by splashing from rain and over-head irrigation and the efficiency of dispersal depends on droplet size and intensity of rain or irrigation (Ntahimpera *et al.,* 1997; 1999).

Control of anthracnose can be difficult. Anthracnose crown rot can be minimized by the use of non-infected nursery plants, minimal use of fertilizers and water in transplanting to fruiting fields, and removal of infected plants and fruit from fruiting fields followed by immediate fungicide applications after each rain period. However, few available fungicides are effective against anthracnose of strawberry; once an epidemic of anthracnose fruit rot begins in a susceptible cultivar, it is nearly impossible to control. In addition, *Colletotrichum* species and isolates may differ in sensitivity to fungicides (Freeman *et al.,* 1998; McInnes *et al.,* 1992; Sanitation during fruit harvests must be implemented so that the disease is not spread from an affected field to a non-affected field.

The prospects for developing cultivars resistant to anthracnose are relatively good. Although the genetics of anthracnose resistance is complicated, gains from recurrent selection have been possible because of the high broad sense heritability estimates for resistance (Gupton and Smith 1991). Complications include the effects of environment on infection and symptom expression, species and isolate of *Colletotrichum* involved, plant part attacked, method of resistance evaluation, and host genotype (Delp and Milholland 1980; Denoyes-Rothan 1997; Denoyes-Rothan and Guérin 1996; Howard *et al.,* 1992; Smith and Black 1987; Smith and Spiers 1982). The following cultivars have been reported to be resistant to at least one anthracnose disease phase (where indicated in the literature C = crown and F = fruit infection): Gaviota, Elvira (C), and Pandora (C) from England; Cortina and Idea from Italy; H k wase from Japan; Suhong from Korea; Elsanta (F) from The Netherlands; and Allstar (F), Aromas, Cardinal (F), Delmarvel (F), Dover (CF), Earliglow (F), Heidi, Honeoye (CF), Pajaro (F), Pelican (CF), Selva (F), and Sweet Charlie (CF) from the United States (Chandler *et al.,* 1997; Denoyes-Rothan *et al.,* 1999; Galletta *et al.*, 1995; Olcott-Reid and Moore 1995; Simpson *et al.,* 1994; Smith *et al.*, 1998). Whereas 'Pajaro' and 'Selva' appeared resistant in France (Denoyes-Rothan *et al.*,1999) each were rated very susceptible to both crown and fruit infection in Florida, USA (Howard *et al.,* 1992), illustrating the variability that is encountered in assessing resistance to this disease.

5. Disease control strategies

5.1 Pathogen identification/ detection

Unambiguous pathogen identification leads not only to a better understanding of disease, but is also required for application of appropriate disease control strategies. Most strawberry pathogens have been known for many years and are well characterized; however, some have been somewhat misunderstood in terms of their relationships to similar pathogens of other crop plants while others, such as phytoplasmas, have been impossible to characterize without molecular methods. Molecular marker development based on polymerase chain reactions (PCR) have clarified some of these rela-

tionships. Early detection of pathogens in plants and detection of low titers of patho-gen cells in plant tissues have been greatly facilitated with the development of primers developed for specific pathogens (specific primers) or groups of related pathogens (universal primers). For example, using random amplified polymorphic DNA (RAPD) analyses, Phytophthora crown rot of strawberry in Finland was found to be caused by a pathotype of *P. cactorum* that apparently is specific to strawberry and is different from the pathotype infecting silver birch (*Betula pendula* Roth) (Hantula *et al.,* 2000; Lilja *et al.,* 1998.). PCR-based methods have been developed to detect the presence of *P. cactorum* in apparently healthy strawberry crowns and to detect early infection of strawberry roots by *P. fragariae* (Lacourt *et al.,* 1997). *Rhizoctonia* species have long been recognized as pathogens of strawberry (Maas 1998) and binucleate forms are known to belong to several anastomosis groups (AGs) (Martin 1988). However, not all *Rhizoctonia* isolates yield positive anastomosis reactions, restricting their identifi-cation for epidemiological studies (Martin, personal communication). RFLP analyses of the rDNA region have effectively differentiated isolates not clearly defined by AG reactions and have allowed estimates of the impact of each pathogen type on the de-velopment of black root rot, the influence of seasonal variations of environmental conditions on individual epidemiologies, and the interactions among the different patho-gen types in disease severity and economic losses (Martin 2000; Martin personal com-munication).

Phytoplasmas (mycoplasma-like organisms, MLOs) infecting plants are now characterized molecularly as to their phylogenetic relationships based on RFLP analy-ses of their 16S rRNA gene and ribosomal protein gene sequences (Lee *et al.,* 1998). Since phytoplasmas have not been established in artificial culture, earlier classifica-tions of phytoplasmas were based on disease symptomatology. This did not account for the possibility that two or more phytoplasmas could be associated with similar disease symptoms, or that more than one phytoplasma strain could concomitantly in-fect the same plant. Since plant-infecting phytoplasmas can not be studied in synthetic culture, their phylogenetic classification by molecular methods has enabled plant pa-thologist to learn a great deal of the nature and occurrence of phytoplasmas in various crop plants and, importantly, of their host ranges. Several new phytoplasmas, based on RFLP analyses, recently have been added to the list of pathogens associated with straw-berry diseases (Table 2).

Few diseases of strawberry are known to be caused by bacteria. Bacterial angu-lar leafspot and its causal agent, *Xanthomonas fragariae*, can be identified in pure culture by traditional methods, although PCR-based methods have shown that four pathogenic genotypes exist among strains of *X. fragariae* (Pooler *et al.,* 1996) and there is evidence to show that these genotypes differ in pathogenicity to strawberry cultivars (Maas *et al.,* 2000). Specific primers have been developed for identification of *X. fragariae* in culture and detection in host materials (Hartung and Pooler 1997). Unlike bacterial angular leafspot, marginal chlorosis, a new disease of strawberry first observed in France in 1988, is caused by a bacterium-like organism which is phloem-limited in the plant and has not been cultured in artificial media (Nourrisseau *et al.,* 1993). The pathogen was recently phylogenetically characterized by PCR methods and, in comparison with gene sequences of other similar organisms, was determined

to represent a new bacterial genus and was named "*Candidatus* Phlomobacter fragariae" (Zreik *et al.,* 1998). Specific identification of these pathogens is important because a large variety of bacteria exist in apparently healthy strawberry tissue and often are detected in isolations from diseased plant parts (Mahuku and Goodwin 1997; Pooler *et al.,* 1996) or in attempts to establish strawberry plants in tissue culture (Tanprasert and Reed 1998).

Table 2: Phytoplasmas associated with diseases of strawberry, their phylogenetic (16S rRNA) group and subgroup, and major symptoms associated with their presence.

Phytoplasma	16S rRNA group-subgroup	Major symptoms associated with phytoplasma infection	Reference
Eastern aster yellows	I-A	Chlorosis, virescence, phyllody	Clark. 1998
Western aster yellows	I-B	Chlorosis, virescence, phyllody	Clark. 1998
Clover phyllody	I-C	Petals virescent, adherent, turning red; phyllody	Clark. 1998, Harrison *et al.,* 1997
STRAWB1	I-I	Plant and leaves stunted	Jomantiene *et al.,* 1998b
STRAWB2	I-K	Plant and leaves stunted, phyllody	Jomantiene *et al.,* 1998b, Jomantiene *et al*., 1999b
Clover yellow edge	III-B	Plant stunted, chlorotic; phyllody	Jomantiene *et al.,* 1999b;
Strawberry leafy fruit	III-K	Plant stunted, chlorotic; phyllody	Jomantiene *et al.,* 2001.
Clover proliferation	VI-A	Plant stunted, chlorotic; phyllody	Jomantiene *et al.,* 1999a
Strawberry multicipita	VI-B	Plant stunted, crown proliferation, phyllody	Jomantiene *et al.,* 1998a
"*Candidatus* Phytoplasma australiense"	XII-B	Young leaf margins chlorotic; older leaves purple or bronze pigmented flower abortion	Andersen *et al.,* 1998
Mexican periwinkle virescence	XIII-B	Plant stunted; virescence; phyllody	Harrison *et al.,* 1997

Detection and identification of strawberry-infecting viruses have benefited from molecular technologies. Traditionally, viruses infecting strawberry are characterized and identified by disease symptoms in indicator plants, electron microscopy, and/or serological methods (Converse 1987). The use of indicator plants, whether species of *Fragaria* or other plants, is necessary since symptom expression due to virus infection

is rarely exhibited in commercial strawberry plants. Along with enzyme-linked immunosorbent assays and double-stranded RNA analyses, PCR has been increasingly used for detection of minute quantities of viruses in plant tissues (Clark, 1998). For example, a cDNA probe was developed to rapidly and specifically detect strawberry vein banding virus in dot blot hybridizations without the use of radioactive materials (Mráz *et al.,* 1996). Multiple virus infections in strawberry plants have been difficult to assess and virus identifications nearly impossible to determine by traditional means. Dot blot hybridizations were used to confirm electron microscopic findings of dual virus infection of strawberry and to determine the identities of the two viruses (Fránová-Honetšlegrová *et al.,* 1999).

5.2 Certification and clean planting stocks

Establishment of strawberry plantings with transplants that are essentially free of disease-causing organisms is critical for obtaining optimum fruit production from a planting (Speiegel 1998). Certification programs have been established in many countries to ensure that nursery propagation material is free of known nematodes, viruses, and fungi. In field propagation of strawberry plants, generally no more than three years elapse from the original propagation of plants under screenhouse conditions through field propagation and distribution to the fruit producer. Although field exposure of transplants is minimal and periodic inspections are made to determine the presence or absence of disease, there still exists a chance that some plants may become infected and carry pathogens to fruit production fields. An alternative to field-grown transplants is the use of container-grown, or plug plants. Plug plants rooted in soil-less media offer the advantage of being grown under controlled environmental conditions that practically ensure that the young plants are disease-free (Maas 2000; Poling and Maas 2000). Although more costly to produce, the use of plug plants has become wide-spread in many strawberry-growing regions; particularly in central Europe and in the southeastern and Mid-Atlantic regions of North America. Plug transplants, produced in 4 weeks compared to 4 or more months for field-grown transplants, also offer the advantages of lower pesticide use, reduced worker exposure to pesticides, and lower pesticide residues in the transplant stocks.

5.3 Soil sterilization and "healthy soils"

World-wide, the most prevalent strawberry disease management practices are based on the use of fungicides and other biocides. Paramount in strawberry fruit and nursery-plant production is the use of methyl bromide fumigation. Methyl bromide is regulated under the United Nations Protocol as an ozone-depleting substance (Montreal Protocol 1992). In the United States, the Protocol calls for a 50% reduction per year of methyl bromide sold to users. By 2005, most uses of methyl bromide will be banned in the United States; farmers in developing countries will have an additional 10 years before their phase-out date. Strawberry growers are being hard-hit by this phase-out. Prior to 2001, California, which produces 75% to 80% of strawberries in the United States, used approximately 7,300 metric tons of methyl bromide annually, one-half of

the nation's total annual use. In addition to the Protocol restrictions, the California Department of Pesticide Regulation has added new state-level restrictions limiting the use of methyl bromide in many highly populated areas (Carpenter *et al.,* 2001).

Preplant methyl bromide fumigation first was used to control *Verticillium* wilt in California (Wilhelm and Koch 1956). It was later discovered that increased growth and yield responses, independent of pathogen control, occurred in plantings treated with methyl bromide (Wilhelm *et al.,* 1974). The greatest plant responses occurred with methyl bromide/chloropicrin mixtures. Yield reductions of at least 10 percent are anticipated in strawberry fruit yields in plantings no longer fumigated with methyl bromide (Shaw and Larson 1999).

Potential replacements for methyl bromide are being actively sought (MBA 2000). Chloropicrin and 1,3-dichloropropene are currently approved for use in California for soil fumigation. Field studies suggest that the use of chloropicrin in field plantings and nurseries will offer strawberry nursery and fruit producers a tool for managing diseases caused by soil-born pathogens (Coffey *et al.,* 1994; Duniway and Gubler 1994; Duniway *et al.,* 1994, Rieger *et al.,* 2001; Welch and Gubler 1994). Although chloropicrin is not as effective as methyl bromide in controlling weeds and nematodes, it can be combined with other materials. Studies suggest that pre-plant treatment of soil with chloropicrin may result in yields as high as those obtained with comparable methyl bromide/chloropicrin application rates (Larson and Shaw 1994; Welch and Gubler 1994). Other soil fumigants, such as metam-sodium, dazomet, and methyl iodide are being considered for use in strawberry production (Shaw and Larson 1999). None of these fumigants are expected to produce the increased growth and yield responses associated with methyl bromide fumigation.

Organic strawberry production is also considered as an alternative to methyl bromide use. Organic strawberry production eliminates environmental stresses caused by pesticide use, thus increasing soil biotic diversity and increasing antagonistic and predacious micro-organisms that are beneficial to plant health; *i.e.*, maintenance of a "healthy soil" (Gliessman *et al.,* 1996). Lower fruit production in organic systems can be offset by higher prices paid for organically-grown fruit. Non-chemical alternatives (*e.g.*, solarization) are effective in some areas but not feasible in others. Solarization is effective where day-time temperatures are sufficiently high and periods of high temperatures are of sufficient duration to reduce pathogen populations in soil to an economic level.

Loss of effective pre-plant fumigation chemicals has highlighted the need for disease- and pest-resistant cultivars. Cultural systems that are dependent on the use of pre-plant fumigation with methyl bromide mixtures may not survive intact. The development of highly productive, but disease susceptible, cultivars may become an artifact of the past. Many public institutions and private companies around the world are devoting increased efforts to developing cultivars that are productive in the presence of one or more plant pathogens. In North America, Agriculture and Agri-Food Canada and the U. S. Department of Agriculture have long-term programs for developing strawberry cultivars resistant to several diseases that threaten strawberry production (*e.g.*, red stele root rot, *Verticillium* wilt, anthracnose, bacterial angular leafspot, and several leaf diseases). Similar programs in England, France, Germany, Italy, Russia,

and Korea have been successful in developing cultivars resistant to these and other diseases (*see:* Section 5.5 and Table 1).

5.3.1 Healthy soils

Another corollary to the organic food equation is the question of what constitutes a "healthy" soil. With years of pesticide input into soils in many production areas, and continuous cropping to strawberries, the "natural" microflora of these soils have been altered. The black root rot disease of strawberry, caused by *Rhizoctonia fragariae* Husain & W. E. McKeen, is a good example of how soil modification through years of continuous strawberry production can become a major impediment to future production. Site degradation through soil compaction, poor drainage, strawberry monoculture, inappropriate herbicide use, fumigation, presence of nematodes (*Pratylenchus* species), and planting age all have been implicated in increased incidence and severity of black root rot (LaMondia 1999; Wing *et al.,* 1994, 1995). These processes eventually reconfigure the microbiological balance of soil, decreasing the population and diversity of natural innocuous or beneficial bacteria while increasing populations of non-beneficial bacteria in the root zone of the strawberry plant. This is associated with increases in black root rot incidence (Hildebrand and West 1941; West and Hildebrand 1941). More recent research has shown that rotation with some crops such as rye (*Secale cereale* L.) tilled into the soil may result in development of increased beneficial soil microbe populations and decreased nematode [*Pratylenchus penetrans* (Cobb) Filipjev & Schuurmans Stekhoven] populations and black root rot incidence (LaMondia 1999). Not all organic soil amendments lead to similar results. Other cover crops, such as canola (*Brassica napus* L.) (LaMondia 1999), and decomposed "barnyard" manure, timothy (*Phleum pratense* L.), and red clover (*Trifolium pratense* L.) (West and Hildebrand 1941), increased the incidence of black root rot while rotation with oats (*Avena sativa* L.) increased populations of *P. penetrans* (LaMondia 1999). Thus, soil treatment by organic amendment may be beneficial in managing some disease problems in strawberry culture. But, as West and Hildebrand (1941) observed, with the use of organic soil amendments for disease control "...it is the sum of all the microbiological changes that must be taken into account in describing the influence of the soil treatments on the root rot picture."

5.4 Sanitation management practices

Sanitation can be overlooked as a integral practice in strawberry production. Sanitation and cultural method often go hand-in-hand, with different cultural methods requiring particular emphases on sanitation requirements. For example, the production of plug plants requires care to be taken to reduce over crowding and over watering of plants, removal of diseased plants and plant debris, and monitoring of plants to constantly ensure production of healthy stocks. However, the use of plug plants is not without risk; runner-tip plantlets may be infected with systemic pathogens such as phytoplasmas (*e.g.*, clover phyllody, clover proliferation, and strawberry multicipita phytoplasmas) and bacteria (*e.g.*, *Xanthomonas fragariae* Kennedy & King), but re-

main symptomless until after being transplanted. Still other diseases, such as anthracnose, may be carried with the runner-tip plantlets as incipient stolon infections, and young leaves of runner-tip plantlets may harbor latent *Botrytis cinerea* Pers.:Fr. [teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel] infections that may become active during plug-plant production or after planting in the fruit production field (Maas 2000).

In other cultural systems, such as matted-row and raised-bed systems, removal of diseased plants and fruit, plant debris, and weeds are essential. Because several root-rotting diseases prosper in water-logged soil, it is also important to ensure that excess irrigation and rain waters are rapidly drained from the field. Root and crown diseases such as red stele and crown rot, caused by *Phytophthora cactorum* (Lebert & Cohn) J. Schröt., require prolonged soil moisture for infection of plants. In many production areas drip irrigation tape placed on the soil surface or buried in the root zone is now commonly used to minimize water use and to maximize the efficiency of water delivery (with or without pesticides and nutrients) to plants. This also is an important consideration for managing foliar and fruit infection by *Botrytis cinerea*, since plant and fruit surfaces remain dry and less likely to become infected. In many cases, high humidity conditions in overly vegetative plantings with full leaf canopies and crowded plants invites considerable loss due to infection of leaves and fruit by fungal pathogens. The use of mulch around plants also contributes to management of several diseases. Straw and plastic mulch provide barriers between developing fruit and soil. Straw or similar rough-textured mulches also reduce dispersal of fungal spores (*e.g.*, of *P. cactorum* and *Colletotrichum* species) by rain impact from inoculum-production sites to healthy leaves and fruit.

5.4.1 Loss of pesticides

The high value of strawberry fruit may justify the use of relatively elaborate or expensive chemical disease prevention or management options, in addition to site modification, sanitation, and other cultural adaptations. This becomes more apparent because i. cultural practices differ in the many strawberry-growing regions of the world, ii. regulations and availability of pesticides vary with country and local use, iii. adequate replacements for some pesticides must be researched and approved as some are lost to growers (*e.g.*, the phaseout of methyl bromide for fumigation use in major strawberry-producing countries by 2005); and iv.the numerous and diverse diseases of strawberry caused by fungi, bacteria, viruses, phytoplasmas, nematodes, and arthropod pests (Maas, 1998). Currently, much strawberry research efforts and funds are being spent in major strawberry production areas to find replacements for methyl bromide soil fumigant and to redesign cultural systems to accommodate the loss of this fumigant, while maintaining high fruit productivity. Public concern over health effects of pesticide-contaminated food products and of detrimental effects of pesticides in the soil, water, and atmosphere environments has also spurred development of a considerable organically-grown fruit industry. This, too, has presented unique disease situations that must be addressed with the use of non-chemical control and management strategies.

5.5 Disease resistance

Although the genetic base of the commercial strawberry is narrow (Sjulin and Dale 1987), there exists sufficient heterogeneity in this and other *Fragaria* species to enable breeders to obtain genotypes resistant to many diseases by recurrent selection and by other less commonly used means (Hancock *et al.,* 1990). Resistance to pathogens is the simplest and surest disease control strategy. However, there are impediments to developing disease resistant cultivars: only those diseases which are of greatest economic impact and for which no other economic control strategy is available will be considered for resistance breeding. This is because the process of breeding and selecting resistant genotypes is a slow, laborious, and expensive process. Also, the popularity of any one cultivar may last only a relatively short time, so, as the spectrum of cultivars in a locality constantly changes, new disease-resistant cultivars with superior horticultural traits must continuously be in the process of development. In addition, the strawberry generally is adapted to very specific local environments, often requiring several cultivars to be developed that are resistant to the same disease. Resistance to disease is not the same as immunity; new or imported races and virulence types of pathogens and environmental effects optimum for infection and disease progression may also destabilize specific and general resistance.

5.5.1 Traditional breeding and selection

Cultivars have been developed in several breeding programs around the world that are resistant to red stele root rot, Phytophthora crown rot, *Verticillium* wilt, *Fusarium* wilt (*Fusarium* yellows, *Fusarium oxysporum* Schl.:Fr. f. sp. *fragariae* Winks & Williams), leaf scorch [*Diplocarpon earliana* (Ellis & Everh.) Wolf], common leaf spot [Ramularia leaf spot, *Mycosphaerella fragariae* (Schw.) Lind.], leaf blight [*Phomopsis obscurans* (Ellis. & Everh.) Sutton], powdery mildew, Alternaria black leaf spot, anthracnose fruit and crown rots, and bacterial angular leafspot (*Xanthomonas fragariae*). Some cultivars are less susceptible, but not highly resistant, to *Botrytis* fruit rot and others are tolerant to virus infection; *i.e.*, plants of tolerant cultivars become infected but either they do not exhibit symptoms or they remain productive for longer periods of time than do susceptible (or intolerant) cultivars. Apparent resistance to virus infection may in some instances be due to resistance to, or lack of, a compatible vector. The reader is referred to Hancock *et al.,* (1990) for a comprehensive listing of cultivars resistant or tolerant to these and other diseases, nematodes, viruses, insects, and mites.

Methods to develop disease-resistant cultivars by means other than by traditional breeding and selection have been exploited. To expand the genetic base of the strawberry, interspecific crosses with the octoploid species *F. chiloensis* from South America and *F. virginiana* from North America have been made to bring in new genes for horticultural and disease resistance traits. Notable among these interspecific crosses are those involving clones of both native species that have conferred resistance to several races of *Phytophthora fragariae* (Waldo 1953, Stembridge and Scott 1959). Potential sources of resistance in native octoploid species include virus tolerance (Darrow 1966) and aphid (virus vector) resistance (Crock *et al.,* 1982) in *F. chiloensis*

and *Verticillium* wilt resistance in *F. chiloensis* and *F. virginiana* (Bringhurst *et al.,* 1966; Newton and van Adrichem 1958). In our laboratory, we have identified resistance to *Xanthomonas fragariae* in two clones of *F. virginiana* and these are currently being examined as potential parents for conferring resistance to bacterial angular leafspot disease (Maas *et al.,* 2000). Our studies on inheritance of the resistance genes show segregation among different progenies in crosses between resistant and susceptible parent clones (Maas *et al.,* unpublished data).

In California, where the major production systems have been developed around the use of pre-plant fumigation with methyl bromide mixtures, efforts are underway by corporate breeders to develop disease-resistant cultivars to replace the widely-used, but highly disease susceptible, cultivars that have been the mainstay of the California strawberry industry. Promising examples of progress in this area of cultivar development are the cultivars 'Catalena' and 'Patty'. 'Catalena', although susceptible to powdery mildew and bacterial angular leafspot, is resistant to *Verticillium* wilt, *Rhizoctonia, Pythium,* and various *Phytophthora* species (U.S. Plant Patent PP 9,320, 1995; D. Small, California Giant, Inc., personal communication 2001). Even more encouraging is that 'Catalena' and its more recently developed cousins, 'Cal Giant 3' and 'Cal Giant 4', which are resistant to powdery mildew, are highly productive in non-fumigated soils in California (D. Small, California Giant, Inc., personal communication 2001). 'Patty' , from the Strawberry Group of the Istituto Sperimentale per la Frutticoltura, Forli, Italy (Community Plant Variety Right File no. 19991570) is also reported to perform well in non-fumigated soil, having good tolerance to soil-borne diseases and anthracnose and resistance to powdery mildew (*http://www.agraria.it/isf/news/ fragola/homepage1uk.htm*).

Development of disease-resistant cultivars, in my estimation, will be the main objective in strawberry research in the foreseeable future. The genes for these traits are believed to be available in strawberry germplasm; it is a matter of observation and recognition of these traits at all levels of strawberry development for their incorporation into commercially acceptable cultivars.

Development of disease-resistant cultivars is not a rapid process, taking an average of 13 years from parental crosses to cultivar release to the grower. Direct incorporation of resistance genes from "undeveloped" parents into commercially acceptable cultivars is not possible; resistance traits often carry with them unwanted traits such as small fruit size and susceptibility to other diseases (Scott *et al.,* 1972). Three to four generations of backcrosses to commercial cultivar parents and large populations of seedlings for selecting segregating resistance genes are required to obtain new disease resistant cultivars, followed by extensive evaluation under different environmental conditions and cultural management systems (Bringhurst and Voth 1984).

5.5.2 Biotechnology

Improvement of disease resistance characteristics in strawberry through transfer of resistance genes from other organisms is being explored. However, there has been more promise than progress in this area of strawberry biotechnology research (Hokanson and Maas 2001). As with other transgenic (*i.e.*, genetically modified through "non-

traditional" means) crops, there are concerns of the public for environmental safety and human health. The public or private plant developer also faces the potential of developing and marketing a product that may not have sufficient public acceptance to guarantee financial returns that justify the research involved. Biotechnology, however, has made great strides in other areas related to strawberry diseases. These achievements relate to rapid and accurate determination of pathogens that, here-to-fore, have been very difficult or impossible to identify (*see:* Section 5.1).

5.5.3 Somaclonal variation

Tissue-culture induced (somaclonal) variation offers potential for developing disease resistant genotypes (Larkin and Scowcroft 1981) and has been a successful strategy in a wide range of plants, including sugarcane (*Saccharum officinarum* L.), tomato (*Lycopersicon esculentum* Mill.), mango (*Magnifera indica* L.), apple (*Malus x domestica* Borkh.), peach [*Prunus persica* (L.) Batsch], and strawberry (Hammerschlag 1992). In strawberry, regenerated plants of *in vitro*-derived somaclonal variants from leaf callus tissues have been shown to be resistant to the *Fusarium* wilt disease of strawberry (Toyoda *et al.,* 1991) and to Alternaria black leaf spot of strawberry (Takahashi *et al.,* 1993). In our Laboratory, we have derived disease-resistant regenerated somaclonal variants from strawberry leaves that exhibit increased resistance, compared to parent plants, to anthracnose caused by *Colletotrichum acutatum* Simmonds (Garcés *et al.,* 2001). Interestingly, we found in this study that cultivars differ in the degree to which somaclonal variation results in regenerants resistant to anthracnose, and that resistant variants may be derived from both anthracnose susceptible (*e.g.,* 'Chandler') and resistant ('Sweet Charlie') parent plants. It is quite likely that resistance to other diseases can be obtained in this manner with somaclonal variants. No attempts have been made to select variants resistant to more than one disease, but it seems likely that this also may be possible with traditional breeding methods.

5.6 Biological control organisms

Use of biological controls in pest management has been very successful. However, there has been relatively little progress in developing biological controls for diseases caused by fungi. Some limitations are inherent in developing biologicals for management of strawberry diseases.

Microorganisms introduced to control disease must interact with the crop plant, potential pathogens, environmental variables, and indigenous organisms under prevailing microclimate conditions (Sutton 1994). Biological control organisms must be present at critical times favorable for pathogen infection, but must not themselves present disease problems. In strawberry, potential biologicals have been isolated from plants and soil under cultivation. It is assumed that these natural colonizers of strawberry fruit, leaves, and roots are either weakly or non-pathogenic, or saprophytic and are capable of interacting successfully with the plant, microbiological and other environmental conditions, and cultural systems. The isolation and testing of potential pathogen antagonists and development of delivery systems for biological control agents for

strawberry were reviewed by Sutton (1994) and Sutton and Peng (1993).

Gray mold fruit rot of strawberry, caused by *Botrytis cinerea*, has received the most attention with regard to developing biological control agents (Bhatt and Vaughan 1962; Sutton 1994; Sutton and Peng 1993; Swadling and Jefferies 1996; Tronsmo and Dennis 1977). Isolates of *Gliocladium roseum* Bainier have shown to be effective in reducing the incidence of gray mold fruit rot (Sutton 1994). Two aspects of infection are addressed in biological control of *Botrytis* fruit rot; reduction of inoculum from infected leaves and protection of flower parts. *Gliocladium roseum* is a naturally occurring epiphyte on strawberry. When applied to leaves, it has the ability to colonize green leaves without causing disease. Thus, its continual presence may inhibit leaf infection by *B. cinerea* (Sutton 1994). Since most fruit infection by *B. cinerea* is a result of the colonization of flower parts (stamens, petals, and sepals), it is imperative that protective applications of chemicals or biologicals are made during early to late bloom periods (Sutton 1998). *Gliocladium roseum* also can be delivered to flower parts, either by spray applications or by pollinating honey bees (*Apis mellifera* L.), effectively reducing fruit rot incidence (Sutton 1994). *Trichoderma* species also have shown some effectiveness in managing fruit rot caused by *Botrytis cinerea* (Gullino 1992; Elad 1993; Sesan and Teodorescu 1993; Sutton 1994; Tronsmo and Dennis 1977). Trichodex, a formulation of *T. harzianum* Rifai, is one of the first commercial products developed for control of *Botrytis* on strawberry and other crops (Elad 1993). Other commercial formulations of *Trichoderma* species also are available for control of *Botrytis* and other pathogens on several crops.

Fusarium wilt of strawberry, caused by *Fusarium oxysporum* f. sp. *fragariae*, has received attention in Japan and Korea as a target for biological control systems. *Trichoderma harzianum* has been shown to be effective in decreasing incidence of *Fusarium* wilt (Moon *et al.,* 1995). Non-pathogenic isolates of *F. oxysporum* isolated from strawberry plants also controlled *Fusarium* wilt in field tests when plants were inoculated with the non-pathogenic isolates prior to planting (Okayama, 1991; Tezuka and Makino 1991). These antagonistic fungi are common inhabitants of the strawberry rhizosphere and apparently well-adapted to the ecological niche that same niche that otherwise could be colonized by the pathogenic species.

5.7 Integrated Pest Management (IPM)

One cannot consider disease management without also addressing pest management strategies. Generally, management practices for disease control go hand-in-hand with pest control strategies. Integrated pest management (IPM) is the practice of integrating agriculture with disease and pest management in a manner that is environmentally friendly, but also economical. In other words, the ends must justify the means in agricultural production within limitations imposed by society. The fundamental concepts of IPM have been summarized as optimization of pest control in an ecologically and economically sound manner with emphasis on coordinated use of multiple tactics to enhance stable crop production and with maintenance of pest damage below injurious levels while minimizing hazards to non-targets (other plants, humans, animals, and the environment) (Dover 1985). Sustainable agriculture has been used as a term to

describe the successful use of IPM practices.

Where does IPM fit into the management of strawberry diseases? IPM includes the use of disease-resistant and stress-tolerant cultivars, and certified plants. It also includes sanitation, and cultural practices such as weed control, proper irrigation, drainage, and fertilization, renovation and maintenance of plant-stand densities, optimum site selection, improvement of aeration, reduction of inoculum, monitoring for disease, appropriate timing of fungicide applications, crop rotation, and use of biologicals (Cooley *et al.,* 1996). Using *Botrytis* fruit rot as an example, there are several points in the disease cycle where disease can be controlled effectively with reductions in fungicide use and protection of predator mites (Cooley *et al.,* 1996; Sutton 1998b; Wilcox and Seem 1994). Several studies have shown that the critical period for reduction of gray mold incidence is during flowering (Gilles 1959; Jordan 1978; Powelson, 1960). Other important management practices that may reduce incidence of *Botrytis* fruit rot include: reducing plant density to increase air circulation and reduce moisture on plant surfaces, application of nitrogen fertilizer only after harvest to control plant density prior to bloom and fruit development, and removal of infected and damaged fruit from the field. In controlling *Botrytis* fruit rot, however, cultural manipulations generally must be secondary to timely and appropriate fungicide applications (Daugaard 2000; Mertely *et al.,* 2000). Post-harvest *Botrytis* rot incidence is the consequence of pre-harvest infection that was initiated during the bloom period (Maas 1981). Post-harvest rot development is delayed by rapid removal of field heat with refrigeration and often augmented with controlled atmosphere storage (*i.e.,* high CO_2) to slow fruit metabolism and development of incipient infections.

6. References

Andersen, M.T., Longmore, J., Liefting,L.W., Wood,G.A., Sutherland,P.W., Beck, D.L. and Forster, R.L.S. 1998. Phormium yellow leaf phytoplasma is associated with strawberry lethal yellows disease in New Zealand. Plant Dis., 82:606-609.

Bal, E., Gilles, G, Creemers, P., and Verheyden, C. 1983. Med. Fac. Landbouww. Rijkuniv. Gent, 48:637-646.

Bell, J.A., Simpson, D.W., and Harris, D.C. 1997. Development of a method for screening strawberry germplasm for resistance to *Phytophthora cactorum.* Acta Hort., 439:175-179.

Bhat, R.G. and Subbarao, K.V. 1999. Host range specificity in *Verticillium dahliae*. Phytopathology, 89:1218-1225.

Bhatt, D.D. and Vaughan, E.K. 1962. Preliminary investigations on the biological control of gray mold (*Botrytis cinerea*) of strawberries. Plant Disease Reporter, 46:342-345.

Blacharski, R.W., Bartz, J.A., Xiao, C.L., and Legard, D.E. 2001. Control of postharvest *Botrytis* fruit rot with preharvest fungicide applications in annual strawberry. Plant Dis., 85:597-602.

Bosher, J.E. 1954. Root-lesion nematodes associated with root decline of small fruits and other crops in British Columbia. Can. J. Agr. Sci., 34:429-431.

Bringhurst, R.S., Wilhelm, S. and Voth, V. 1966. *Verticillium* wilt resistance in natural populations of *Fragaria chiloensis* in California. Phytopathology, 51:219-222.

Bringhurst, R.S. and Voth, V. 1978. Origin and evolutionary potentiality of the day-neutral trait in octoploid *Fragaria*. Genetics, 90:510.

Bringhurst, R.S. and Voth, V. 1984. Breeding octoploid strawberries. Iowa State J. Res., 58:371-

381.

Brooks, A.N. 1931. Anthracnose of strawberry caused by *Colletotrichum fragariae*, n. sp. Phytopathology, 21:739-744.

Brooks, A.N. 1935. Anthracnose and wilt of strawberry caused by *Colletotrichum fragariae*. Phytopathology, 25:937. (Abstract)

Carpenter, J., Lynch, L. and Trout, T. 2001. Township limits on 1,3-D will impact adjustment to methyl bromide phase-out. California Agriculture, 55(3):12-18.

Cavanni, P., Montuschi, C. and Maltoni, M.L. 1996. Alternariosi della fragola: primi risultati di saggi per la resistenza varietale. Riv. Frutticoltura Ortofloricoltura, 58:47-50.

Chandler, C.K., Albregts, E.E., Howard, C.M. and Brecht, J.K. 1997. 'Sweet Charlie' strawberry. HortScience, 32:1132-1133.

Chen, T.A. and Rich, A.E. 1962. The role of *Pratylenchus penetrans* in the development of strawberry black root rot. Plant Disease Reporter, 46:839-843.

Cho, C.T. and Moon, B.J. 1984. Studies on the wilt of strawberry caused by *Fusarium oxysporum* f. sp. *fragariae* in Korea (in Korean). Korean J. Plant Protection, 23:74-81.

Clark, M.F. 1998. Leafhopper-vectored diseases caused by phytoplasmas. In: "Compendium of strawberry diseases."(ed. Maas, J.L.), APS Press, St. Paul, Minnesota. pp. 72-75.

Coffey, M., Paulus, A.O., Schmitz, I., Rich, P., Krueger, H., Meyer-Posolsky, M., Forster, H., Vilchez, M. and Westerlund, F. 1994. Evaluation of alternative soil fumigation methods for use in strawberry production in southern California. 1994 Conference Proceedings, Annual International Res. Conf. on Methyl Bromide Alternatives and Emission Reductions. Number 1. Kissimmee, Florida.

Colombo, L. and Maltoni, M.L. 1996. Prime valutazioni della suscettibilità varietale a *Phytophthora cactorum* in fragola. Riv. Frutticoltura Ortofloricoltura, 58:63-65.

Cooley, D.R., Wilcox, W.F., Kovach, J. and Schloemann, S.G. 1996. Integrated pest management programs for strawberries in the northeastern United States. Plant Dis., 80:228-236.

Coons, G.H. 1926. Black root of strawberry. Michigan State Univ. Agr. Expt. Sta. Quart. Bull. 7:25-26.

Converse, R.H. (ed.) 1987. Virus Diseases of Small Fruits. U.S. Department of Agriculture, Agricultural Research Service, Agriculture Handbook 631p.

Crock, J.E., Shanks, C.H., Jr. and Barritt, B.H. 1982. Resistance in *Fragaria chiloensis* and *F. ananassa* to the aphids *Chaetosiphon fragaefolii* and *C. thomasi*. HortScience, 17:959-960.

Darrow, G.M. 1966. The Strawberry: History, Breeding, and Physiology. Holt, Rinehart and Winston, New York, USA.

Daugaard, H. 2000. Effect of cultural methods on the occurrence of grey mould (*Botrytis cinerea* Pers.) in strawberries. Biol. Agr. Hort.18:77-83.

Delp, B.R. and Milholland, R.D. 1980. Evaluating strawberry plants for resistance to *Colletotrichum fragariae*. Plant Dis., 64:1071-1073.

Denoyes-Rothan, B. 1997. Inheritance of resistance to *Colletotrichum acutatum* in strawberry (*Fragaria x ananassa*). Acta Hort., 439:809-814.

Denoyes-Rothan, B. and Guérin, G. 1996. Comparison of six inoculation techniques with *Colletotrichum acutatum* on cold stored strawberry plants and screening for resistance to this fungus in French strawberry collections. European J. Plant Pathol., 102:615-621.

Denoyes-Rothan, B., Lafargue, M., Guerin, G. and Clerjeau, M. 1999. Fruit resistance to *Colletotrichum acutatum* in strawberries. Plant Dis., 83:549-553.

Dingley, J.M. 1970. Recordings of fungi parasitic on plants in New Zealand. N. Z. J. Agric. Res.,13:325-337.

Dover, M.J. 1985. A better mousetrap: Improving pest management for agriculture. Study 4. World Resources Inst., Washington, D. C.

Duncan, J.M. 1985. Effect of fungicides on survival, infectivity and germination of *Phytophthora*

fragariae oospores. Trans. Brit. Myc. Soc., 85:585-593.

Duniway, J. and Gubler, W. 1994. Evaluation of some chemical and cultural alternatives to methyl bromide fumigation of soil in a California strawberry production system. 1994 Conference Proceedings, Annual International Res. Conf. on Methyl Bromide Alternatives and Emission Reductions. Number 37. Orlando, Florida.

Duniway, J., Gubler, W. and Filajdic, N. 1994. Evaluation of strawberry growth, fruit yield, and soil microorganisms in non-treated soil and in soil treated with methyl   bromide/chloropicrin, Telone II /chloropicrin, chloropicrin, or Vapam in a California strawberry production system. 1994 Conference Proceedings, Annual International Res. Conf. on Methyl Bromide Alternatives and Emission Reductions. Number 16. Kissimmee, Florida.

Durner, E.F., Barden, J.A., Himelrick, D.G. and Poling, E.B. 1984. Photoperiod and temperature effects on flower and runner development in day-neutral, Junebearing, and everbearing strawberries. J. Amer. Soc. Hort. Sci., 109:396-400.

Eastburn, D.M. and Gubler, W.D. 1990. Strawberry anthracnose: Detection and survival of *Colletotrichum acutatum* in soil. Plant Dis., 74:161-163.

Elad, Y. 1993. Implementation of biological control of foliar diseases. In: "Biological Control of Foliar and Post-Harvest Diseases." (eds. Fokkema, N.J., Köhl, J. and Elad, Y.) IOBC/WPRS Bull. Vol. 16. Montfavet, France. pp. 229-233.

Elad, Y., Chet, I. and Henis, Y. 1981. Biological control of *Rhizoctonia solani* in strawberry fields by *Trichoderma harzianum.* Plant Soil, 60:245-254.

Ellis, M.A., Wilcox, W.F. and Madden, L.V. 1998. Efficacy of metalaxyl, fosetyl-aluminum, and straw mulch for control of strawberry leather rot caused by *Phytophthora cactorum.* Plant Dis., 82:329-332.

Elmer, W.H. and LaMondia, J.A. 1999. Influence of ammonium sulfate and rotation crops on strawberry black root rot. Plant Dis., 83:119-123.

Eshenaur, B.C. and Milholland, R.D. 1989. Factors influencing the growth of *Phomopsis obscurans* and disease development on strawberry leaf and runner tissue. Plant Dis., 73:814-819.

FAO Production Yearbook 2001. FAO Statistics Series no. 156. Production vol. 53. Food and Agricultural Organization of the United Nations. Rome.

Farr, D.F., Bills, G.F., Chamuris, G.P. and Rossman, A.Y. 1989. Fungi on Plants and Plant Products in the United States. APS Press, St. Paul, Minnesota. 1,252 p.

Fránová-Honetšlegrová, J., Mráz, I, Nebesá ová and Šíp, M. 1999. Preferential banding of secondary veins in strawberry is caused by mixed virus infection. Acta Virol., 43:349-355.

Freeman, S. and Katan, T. 1997. Identification of *Colletotrichum* species responsible for anthracnose and root necrosis of strawberry in Israel. Phytopathology, 87:516-521.

Freeman, S., Katan, T. and Shabi, E. 1998. Characterization of *Colletotrichum* species responsible for anthracnose diseases of various fruits. Plant Dis., 82:596-605.

Freeman, S., Minz, D., Maymon, M. and Zveibil, A. 2001. Genetic diversity within *Colletotrichum acutatum sensu* Simmonds. Phytopathology, 91:586-592.

Freeman, S., Nizani, Y., Dotan, S., Even, S. and Sando, T. 1997. Control of *Colletotrichum acutatum* in strawberry under laboratory, greenhouse, and field conditions. Plant Dis,. 81:749-752.

Galletta, G.J. and Bringhurst, R.S. 1990. Strawberry management. In: *Small Fruit Crop Management* (eds. Galletta, G.J. and Himelrick, D.G.). Prentice Hall, Englewood Cliffs, New Jersey, USA. pp. 83-156.

Galletta, G.J. and Maas, J.L. 1990. Strawberry genetics. HortScience, 25:871-879.

Galletta, G.J., Maas, J.L., Enns, J.M., Draper, A.D., Fiola, J.A., Scheerens, J.C., Archbold, D.D. and Ballington, J.R., Jr. 1995. 'Delmarvel' strawberry. HortScience, 30:1099-1103.

Garcés, S., Hammerschlag, F. and Maas, J. 2001. Increased resistance to *Colletotrichum acutatum*

is exhibited by leaf explant regenerants derived from several strawberry cultivars. Proc. 5th North American Strawberry Conference (in press).

Gliessman, S.R., Werner, M.R., Swezey, S.L., Caswell, E., Cochran, J. and Rosado-May, R. 1996. Conversion to organic strawberry management changes ecological processes. California Agriculture, 50(1):24-31.

Gilles, G. 1959. Biology and control of *Botrytis* cinerea Pers. on strawberries. Höfchen-Briefe, 12:141-170.

Gullino, M.L. 1992. Control of *Botrytis* rot in grapes and vegetables with *Trichoderma* spp. In: "Biological Control of Plant Diseases. Progress and Challenges for the Future". (eds. Tjamos, E.S. Papavisas, G.C. and Cook, R.J.) Plenum Press, New York. pp. 125-132.

Gupton, C.L. and Smith, B.J. 1991. Inheritance of resistance to *Colletotrichum* species in strawberry. J. Amer. Soc. Hort. Sci., 116:724-727.

Hammerschlag, F. 1992. Somaclonal variation. In: *Biotechnology of Perennial Fruit Crops*. (eds. Hammerschlag, F. and Litz, R.E.). CAB International, Wallingford, UK. pp. 35-55.

Hancock, J.F. 1999. *Strawberries*. CAB International Publishers, Cambridge, New York.

Hancock, J.F., Maas, J.L., Shanks, C.H., Breen, P.J. and Luby, J.J. 1990. Strawberries *(Fragaria)*. In: "Genetic Resources of Temperate Fruit and Nut Crops, (eds. Moore, J.N. and Ballington Jr., J.R.) Acta Horticulturae, 290:491-546.

Hantula, J., Lilja, A., Nuorteva, H., Parikka, P. and Werres, S. 2000. Pathogenicity, morphology and genetic variation of *Phytophthora cactorum* from strawberry, apple, rhododendron, and silver birch. Mycol. Res., 104:1062-1068.

Harris, D.C., Simpson, D.W. and Bell, J.A. 1997. Studies on the possible role of micropropagation in the dissemination of the strawberry crown rot pathogen *Phytophthora cactorum.* J. Hortic. Sci., 72:125-133.

Harris, D.C. and Yang, J.R. 1996. The relationship between the amount of *Verticillium dahliae* in soil and the incidence of strawberry wilt as a basis for disease risk prediction. Plant Pathol., 45:106-114.

Harrison, N.A., D.E. Legard, R. DiBonito and P.A. Richardson. 1997. Detection and differentiation of phytoplasmas associated with diseases of strawberry in Florida. Plant Dis., 81:230.

Hartung, J.S. and Pooler, M.R. 1997. Immunocapture and multiplexed-PCR assay for *Xanthomonas fragariae*, causal agent of angular leafspot disease. Acta. Hort., 439:821-828.

Haymes, K.M., Van de Weg, W.E., Arens, P., Maas, J.L., Vosman, B. and Den Nijs, A.P.M. 2000. Development of SCAR markers linked to a *Phytophthora fragariae* resistance gene and their assessment in European and North American strawberry genotypes. J. Amer. Soc. Hort. Sci., 125:330-339.

Heide, O.M. 1977. Photoperiod and temperature interactions in growth and flowering of the strawberry. Physiol. Plant., 40:21-26.

Hildebrand, A.A. and West, P.M. 1941. Strawberry root rot in relation to microbiological changes induced in root rot soil by the incorporation of certain cover crops. Can. J. Res., 19-C:183-198.

Hokanson, S.C. and Maas, J.L. 2001. Strawberry biotechnology. Plant Breeding Reviews, 21:139-180.

Horimoto, K., Okayama, K., Kobatake, H. and Kodama, T. 1988a. Studies on the control of *Fusarium* wilt of strawberry by preceding crops. II. Variation in pathogenicity and cultural characteristics of *Fusarium oxysporum* isolated from strawberries (in Japanese). Bull. Nara Agr. Exp. Sta., 19:79-87.

Horimoto, K., Okayama, K., Kobatake, H. and Kodama, T. 1988b. Studies on the control of *Fusarium* wilt of strawberry by preceding crops. III. Some characteristics of *Fusarium oxysporum* f. sp. *fragariae* cultivated with microorganism-free plants (in Japanese). Bull Nara Agr. Exp. Sta., 19:89-95.

Howard, C.M., Maas, J.L., Chandler, C.K. and Albregts, E.E. 1992. Anthracnose of strawberry caused by the *Colletotrichum* complex in Florida. Plant Dis., 76:976-981.

Jomantiene, R., Davis,R.E. Dally, E.L. and Maas,J.L. 1998a. The distinctive morphology of "*Fragaria multicipita*" is due to phytoplasma. HortScience, 33:1069-1072.

Jomantiene, R., Davis,R.E. , Maas, J.L. and Dally, E.L. 1998b. Classification of new phytoplasmas associated with diseases of strawberry in Florida, based on analysis of 16S rRNA and ribosomal protein gene operon sequences. Intl. J. Syst. Bacteriol., 48:269-277.

Jomantiene, R., Maas,J.L., Dally,E.L. and Davis, R.E. 1999a. First report of clover proliferation phytoplasma in strawberry. Plant Dis., 83:967.

Jomantiene, R., Maas,J.L., Dally,E.L. and Davis, R.E. 1999b. First report of clover yellow edge and of STRAWB2 phytoplasmas in strawberry in Maryland. Plant Dis., 83:1072.

Jomantiene, R., Maas, J.L., Dally, E.L. and Davis, R.E. 2001. Molecular identification and classification of a phytoplasma associated with phyllody of strawberry fruit in Maryland. Plant Dis., 85:335.

Jordan, V.L. 1978. Epidemiology and control of fruit rot *Botrytis cinerea* on strawberry. Pflanzenschutz-Nachr., 31:1-10.

Katznelson, H. and Richardson, L.T. 1948. Rhizosphere studies and associated microbiological phenomena in relation to strawberry root rot. Scientific Agriculture, 28:293-308.

Kim, C.H., Seo, H.D., Cho, W.D, and Kim, S.B. 1982. [Studies on varietal resistance and chemical control to the wilt of strawberry caused by *Fusarium oxysporum* (in Korean)]. Korean J. Plant Prot., 21:61-67.

King, W.T., Madden, L.V., Ellis, M.A. and Wilson, L.L. 1997. Effects of temperature on sporulation and latent period of *Colletotrichum* spp. infecting strawberry fruit. Plant Dis., 81:77-84.

Lacourt, I., Bonants, P.J.M., Van Gent-Pelzer, M.P., Cooke, D.E.L., Hagenaar-De Weerdt, M., Surplus, L. and Duncan, J.M. 1997. The use of nested primers in the polymerase chain reaction for the detection of *Phytophthora fragariae* and *P. cactorum* in strawberry. Acta. Hort., 439:829-838.

LaMondia, J.A. 1994. The effect of rotation crops on strawberry black root rot pathogens in field microplots. (Abstract) J. Nematol., 26:108.

LaMondia, J.A. 1999. Influence of rotation crops on the strawberry pathogens *Pratylenchus penetrans, Meloidogyne hapla*, and *Rhizoctonia fragariae.* J. Nematol., 31:650-655.

LaMondia, J.A. and Martin, S.B. 1989. The influence of *Pratylenchus penetrans* and temperature on black root rot of strawberry by binucleate *Rhizoctonia* spp. Plant Dis., 73:107-110.

Larkin, P.J. and Scowcroft, W.R. 1981. Somaclonal variation - A novel source of variability from cell cultures for plant improvement. Theor. App. Genet., 60:197-214.

Larson, K.D. and Shaw, D.V. 1994. Evaluation of eight preplant soil treatments for strawberry production in southern California. 1994 Conference Proceedings, Annual International Res. Conf. on Methyl Bromide Alternatives and Emission Reductions. Number 24. Kissimmee, Florida.

Lederer, W. and Seemüller, E. 1992. Untersuchungen zur Prädisposition der Erdgeere für die Rhizomfäule (*Phytophthora catorum*). Zeit. Pflantzenkrankheiten Pflanzenschut ,99:225-233.

Lee, I.M., Gundersen-Rindal,D.E., Davis,R.E. and Bartoszyk, I.M. 1998. Revised classification scheme of phytoplasmas based on RFLP analyses of 16S rRNA and ribosomal protein gene sequences. Intl. J. Syst. Bacteriol., 48:1153-1169.

Lilja, A., Karjalainen, R., Parikka, K., Kammiovirta, K. and Nuorteva, H. 1998. Pathogenicity and genetic variation of *Phytophthora cactorum* from silver birch and strawberry. European J. Plant Pathol., 104:529-535.

Maas, J.L. 1981. Postharvest diseases of strawberry. In: "The Strawberry; Cultivars to Market-

ing." (ed. Childers, N.F.) Horticultural Publications, Gainesville, Florida, USA.

Maas, J.L. (ed.) 1984. Compendium of Strawberry Diseases. APS Press. St. Paul, Minnesota, USA.

Maas, J.L. 1985. New symptoms of strawberry leaf blight disease. Advances in Strawberry Research, 4:34-35.

Maas, J.L. (ed.) 1998. Compendium of Strawberry Diseases., Second Edition. APS Press. St. Paul, Minnesota, USA.

Maas, J.L. 2000. Opportunities to reduce the potential for disease infection and spread with strawberry plug plants. Acta Horticulturae, 513:409-414.

Maas, J.L. and Galletta, G.J. 1989. Germplasm evaluation for resistance to fungus-incited diseases. Acta Hort., 265:461-472.

Maas, J.L., Galletta, G.J. and Draper, A.D. 1989. Resistance in strawberry to races of *Phytophthora fragariae* and to isolates of *Verticillium* from North America. Acta. Hort., 265:521-526.

Maas, J.L., Gouin-Behe, C., Hartung, J.S. and Hokanson, S.C. 2000. Sources of resistance for two differentially pathogenic strains of *Xanthomonas fragariae* in *Fragaria* genotypes. HortScience, 35:128-131.

Maas, J.L., Pooler, M.R. and Galletta, G.J. 1995. Bacterial angular leafspot disease of strawberry: Present status and prospects for control. Advances in Strawberry Research, 14:18-24.

Maas, J.L. and Smith, W.L., Jr. 1978. 'Earliglow' a possible source of resistance to *Botrytis* fruit rot in strawberry. HortScience, 13:275-276.

Maas, J.L., Wilhelm, S. and Galletta, G.J. 1991. Pest management systems for strawberry diseases. In: "CRC Handbook of Pest Management in Agriculture, 2nd Edition, Vol. III. (ed. Pimentel, D.). CRC Press, Boca Raton, Florida. pp. 553-571.

Madden, L.V. and Ellis, M.A. 1990. Effect of ground cover on splash dispersal of *Phytophthora cactorum* from strawberry fruits. J. Phytopathol., 129:170-174.

Madden, L.V., Ellis, M.A., Grove, G.G., Reynolds, K.M. and Wilson, L.L. 1991. Epidemiology and control of leather rot of strawberries. Plant Dis., 75:439-446.

Mahuku, G.S. and Goodwin, P.H. 1997. Presence of *Xanthomonas fragariae* in symptomless strawberry crowns in Ontario detected using a nested polymerase chain reaction (PCR). Can. J. Plant Pathol., 19:366-370.

Martin, F.N. 2000. *Rhizoctonia* spp. recovered from strawberry roots in central coastal California. Phytopathology, 90:345-353.

Martin, S.B. 1988. Identification, isolation frequency, and pathogenicity of anastomosis groups of binucleate *Rhizoctonia* spp. from strawberry roots. Phytopathology, 78:379-384.

MBA, 2000. Methyl Bromide Alternatives and Emissions Reductions Annual International Research Conference. 2000. www.epa.gov/ozone/mbr/mbrpro00.html

McInnes, T.B., Black, L.L. and Gatti, J.M., Jr. 1992. Disease-free plants for management of strawberry anthracnose crown rot. Plant Dis., 76:260-264.

Mena, A.J., Palacios de Garcia, M.E. and Gonzalez, M.A. 1975. [Enfermedades raduculares de la frutilla causadas por *Fusarium oxysporum* Schl. f. sp. *fragariae* Winks y Will y *Rhizoctonia fragariae* Hussain y McKeen (in Spanish)]. Rev. Agron. N. O. Argentina, 12:299-307.

Mertely, J.C., Chandler, C.K., Xiao, C.L. and Legard, D.E. 2000. Comparison of sanitation and fungicides for management of *Botrytis* fruit rot of strawberry. Plant Dis. 84:1197-1202.

Miller, P.M. 1956. Control of black root rot of strawberries with nematicide-fungicide combinations. Plant Disease Reporter, 40:45-47.

Milholland, R.D. 1994. A monograph of Phytophthora fragariae and the red stele disease of strawberry. North Carolina Agricultural Research Service Technical Bull. 306. North Carolina State University, Raleigh, North Carolina.

Molot, P.M. and Nourrisseau, J.G. 1974. Influence de quelques substances de croissance sur la sensibilité du fraisier aux attaques de *Phytophthora cactorum*. Fruits, 29:697-702.

Molot, P.M. and Nourrisseau, J.G. 1978. Bilan des recherches réalisées par l' I.N.R.A. pour lutter contre le *Phytophtora* [sic] *cactorum* du fraisier. *P.H.M.*- Rev. Horticole, 191:23-33

Montgomerie, I.G. 1977. Red core disease of strawberry. Horticultural Review No. 5. Commonwealth Bureau of Horticulture and Plantation Crops, East Malling Research Station, Kent, UK.

Montreal Protocol 1992. The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer. United Nations Environment Programme, Ozone Secretariat. www.unep.org/ ozone/ mont_t.shtml

Moon, B.J., Chung, H.S. and Park, H.C. 1995. Studies on antagonism of *Trichoderma* species to *Fusarium oxysporum* f. sp. *fragariae* V. Biological control of *Fusarium* wilt of strawberry by a mycoparasite, *Trichoderma harzianum*. Korean J. Plant Pathol., 11:298-303.

Mráz, I., Honetšlegrová, J. and Šíp, M. 1996. Diagnosis of strawberry vein banding virus by a non-radioactive probe. Acta Virol., 40:139-141.

Newton, W. and van Adrichem, M.C.J. 1958. Resistance to *Verticillium* wilt in F_1 generations of *Fragaria*. Can J. Bot., 36:297-299.

Nickerson, N.L. and Maas, J.L. 1991. Red stele in North America. In: "The Strawberry into the 21st Century" (eds. Dale, A. and Luby, J.J.). Timber Press, Portland, Oregon. pp.195-201.

Nicoll, M.F. and Galletta, G.J. 1987. Variation in growth and flowering habits of Junebearing and everbearing strawberries. J. Amer. Soc. Hort. Sci., 112:872-880.

Nourrisseau, J.G., Lansac, M. and Garnier, M. 1993. Marginal chlorosis, a new disease of strawberries associated with a bacteriumlike organism. Plant Dis., 77:1055-1059.

Ntahimpera, N., Madden, L.V. and Wilson, L.L. 1997. Effect of rain distribution alteration on splash dispersal of *Colletotrichum acutatum*. Phytopathology, 87:649-655.

Ntahimpera, N., Wilson, L.L., Ellis, M.A. and Madden, L.V. 1999. Comparison of rain effects on splash dispersal of three *Colletotrichum* species infecting strawberry. Phytopathology, 89:555-563.

Oda, Y. 1991. The strawberry in Japan. In: "The Strawberry into the 21st Century" (eds. Dale, A. and Luby, J.J.). Timber Press, Portland, Oregon, USA. pp. 36-46.

O'Dell, C. 1999. Strawberry alert. Fruit Grower, September, p. 33.

Okayama, K. 1991. Selection [*sic*] and effect of antagonistics on *Fusarium* wilt of strawberries. Bull. Nara Agri. Expt. Sta., 22:17-22.

Okayama, K., Horimoto, K., Kobatake, H., Kodama, T. and Kitagawa, Y. 1988. [Studies on the control of *Fusarium* wilt of strawberry by preceding crops. I. Effects of preceding crops on *Fusarium* wilt of strawberry and presence of microorganisms in the soil (in Japanese)]. Bull. Nara Agr. Expt. Sta., 19:67-78.

Olcott-Reid, B. and Moore, J.N. 1995. Field reactions of strawberry cultivars and selections to anthracnose fruit rot, leather rot and gray mold in Arkansas. Fruit Var. J., 49:4-13.

Otani, H., Kohmoto, K., Kodama, M. and Nishumura, S. 1989. Role of host-specific toxins in the pathogenesis of *Alternaria alternata*. In: "Molecular Strategies of Pathogens and Host Plants" (eds. Patil, S.S., Ouchi, S. Mills, D. and Vance, C.). Springer-Verlag, New York. pp. 139-149.

Ouchi, S., Toyoda, H., Morimoto, M., Kurusu, T., Matsuda, Y., Goto, S. and Fukamizo, T. 1992. Integration of chitin-degrading microbes into biological control system for *Fusarium* wilt of strawberry. In: "Biological Control of Plant Diseases" (eds. Tjamos, E.S. *et al.*). Plenum Press, New York. pp. 335-339.

Peries, O.S. 1961. Overwintering of *Sphaerotheca humuli* on strawberry plants. Plant Pathol., 10:65-66.

Peries, O.S. 1962. Studies on strawberry mildew, caused by *Sphaerotheca macularis* (Wallr. Ex Fries) Jaczewski. I. Biology of the fungus. Ann. Appl. Biol., 50:211-224.

Pitrat, M. and Risser, G. 1977. Étude de la sensibilité variétale du fraisier à *Phytophthora cactorum* après contamination provoquée. Ann. Amélior. Plantes 27:49-60.

Poling, E.B. and Maas, J.L. 2000. Recent advances in strawberry plug transplant technology. Acta Horticulturae, 513:393-401.

Pooler, M.R., Ritchie, D.R. and Hartung, J.S. 1996. Genetic relationships among strains of *Xanthomonas fragariae* based on random amplified polymorphic DNA PCR, repetitive extragenic palindromic PCR, and enterobacterial repetitive intergenic consensus PCR data and generation of multiplexed PCR primers useful for identification of this phytopathogen. App. Environm. Microbiol., 62:3121-3127.

Powelson, R.L. 1960. The initiation of strawberry fruit rot caused by *Botrytis cinerea*. Phytopathology, 50:491-494.

Ribero, O.K. and Black, L.L. 1971. *Rhizoctonia fragariae*: a mycorrhizal and pathogenic fungus of strawberry plants. Plant Disease Reporter, 55:599-603.

Rieger, M. 2001. World Wide Web: <http://www.uga.edu/hortcrop/rieger/strawbry.htm>. May, 2001.

Rieger, M., Krewer, G. and Lewis, P. 2001. Solarization and chemical alternatives to methyl bromide for preplant soil treatment of strawberries. HortTechnology, 11:258-264.

Roudeillac, P. and Veschambre, D. 1987. La Fraise: Techniques de production (in French). Centre Technique Interprofessionel des Fruits et Légumes. Paris.

Scott, D.H., Draper, A.D. and Greeley, L.W. 1972. Interspecific hybridization in octoploid strawberries. HortScience, 7:382-384.

Seemüller, E. 1982. Versuche zur Bekämpfung der Rhizomfäule der Erdgeere mit Metalaxyl und Aluminiumfosetyl. Nachrichtenbl. Dtsch. Plfanzenschutzdienstes (Braunschweig) 34:118-122.

Seemüller, E. 1998. Crown rot. In: "Compendium of Strawberry Diseases" (ed. Maas, J. L.). APS Press, St. Paul, Minnesota. pp. 50-51.

Seemüller, E. and Schmidle, A. 1979. Einfluss der Herkunft von *Phytophthora cactorum*-Isolaten auf ihre Virulenz an Apfelrinde, Erdbeerrhizomen und Erdbeerfrüchten. Phytopathol. Z., 94:218-225.

Sesan, T.E. and Teodorescu, G. 1993. Investigations on prevention of strawberry grey mould (*Botrytis cinerea* Pers.) in Romania by using the fungus *Trichoderma viride* Pers. ex S.F. Gray. In: "Biological Control of Foliar and Post-Harvest Diseases". (eds. Fokkema, N.J. Köhl, J. and Elad, Y.) IOBC/WPRS Bull. Vol. 16. Montfavet, France. pp. 234-237.

Shaw, D.V. and Larson, K.D. 1999. A meta-analysis of strawberry yield response to preplant soil fumigation with combinations of methyl bromide-chloropicrin and four alternative systems. HortScience, 34:839-845.

Simpson, D.W., Winterbotton, C.Q., Bell, J.A. and Maltoni, M.L. 1994. Resistance to a single UK isolate of *Colletotrichum acutatum* in strawberry germplasm from Northern Europe. Euphytica, 77:161-164.

Sjulin, T.M. and Dale, A. 1987. Genetic diversity of North American strawberry cultivars. J. Amer. Soc. Hort. Sci., 112:375-386.

Smith, B.J. and Black, L.L. 1987. Resistance of strawberry plants to *Colletotrichum fragariae* affected by environmental conditions. Plant Dis., 71:834-837.

Smith, B.J., Gupton, C.L., Galletta, G.J., Maas, J.L., Enns, J.M., Ballington, J.R., Jr., Constantin, R.J., DiVittorio, T.J. and Himelrick, D. 1998. 'Pelican' strawberry. HortScience, 33:1082-1084.

Smith, B.J. and Spiers, J.M. 1982. Evaluating techniques for screening strawberry seedlings for resistance to *Colletotrichum fragariae.* Plant Dis., 66:559-561.

Speiegel, S. 1998. Virus certification of strawberries. In: "Plant Virus Disease Control" (eds.. Hadidi, A Khetarpal, R.K. and Hoganezawa, H.). APS Press, St. Paul, Minnesota. pp. 320-

324.

Stembridge, G.E. and Scott, D.H. 1959. Inheritance of resistance of strawberry to the common race of the red stele root rot fungus. Plant Disease Reporter, 43:1091-1094.

Strand, L.L. 1993. Integrated Pest Management for Strawberries. University of California, Division of Agriculture and Natural Resources Publication 3351. Berkeley, California.

Straub, E. 1991. Fungicide resistance: Practical experience with antiresistance strategies and the role of integrated use. Annu. Rev. Phytopathol., 29:421-442.

Sutton, J.C. 1994. Biological control of strawberry diseases. Advances in Strawberry Research, 13:1-12.

Sutton, J.C. 1998a. Leaf scorch. In: "Compendium of Strawberry Diseases". (ed. Maas, J.L.) APS Press, Minneapolis, Minnesota. pp. 19-20.

Sutton, J.C. 1998b. *Botrytis* fruit rot (gray mold) and blossom blight. In: "Compendium of Strawberry Diseases". (ed. Maas, J.L.) APS Press, Minneapolis, Minnesota. pp. 28-31.

Sutton, J.C. and Peng, G. 1993. Manipulation and vectoring of biocontrol organisms to manage foliage and fruit diseases in cropping systems. Annu. Rev. Phytopathol., 31:473-493.

Swadling, I.R. and Jeffries, P. 1996. Isolation of microbial antagonists for biocontrol of grey mould disease of strawberries. Biocontrol Sci. Technol., 6:125-136.

Takahashi, H. 1992. [Breeding of strawberry cultivars resistant to Alternaria black spot of strawberry (*Alternaria alternata* strawberry pathotype (in Japanese)]. Bull. Akita Pref. Coll. Agr., 19:1-44.

Takahashi, H., Furuya, H., Takai, T. and Matsumoto, T. 1997. Characteristics of *Alternaria alternata* strawberry pathotype isolated in New Zealand and the resistance of the 'Akita Berry' strawberry to the fungus. J. Japan. Soc. Hort. Sci., 65:785-790.

Takahashi, H., Matsumoto, T. and Takai, T. 1993. Somaclonal variants from strawberry cv. Morioka-16, 'M16-AR 1, 2, and 3' resistant to several isolates of *Alternaria alternata* strawberry pathotype occurring in Tohoku and Hokkaido. J. Japan. Soc. Hort. Sci., 61:821-826.

Tanprasert, P. and Reed, B.M. 1998. Detection and identification of bacterial contaminants from strawberry runner explants. *In Vitro* Cell. Dev. Biol.-Plant, 33:221-226.

Tezuka, N. and Makino, T. 1991. Biological control of *Fusarium* wilt of strawberry by non-pathogenic *Fusarium oxysporum* isolated from strawberry (in Japanese). Ann. Phytopath. Soc. Japan, 57:506-511.

Toyoda, H., Horikoshi, K. Yamano, Y. and Ouchi, S. 1991. Selection for *Fusarium* wilt disease resistance from regenerants derived from leaf callus of strawberry. Plant Cell Rpt., 10:167-170.

Tronsmo, A. and Dennis, C. 1977. The use of *Trichoderma* species to control strawberry fruit rots. Neth. J. Plant Pathol., 83:449-455.

Ureña-Padilla, A.R., Mitchell, D.J. and Legard, D.E. 2001. Oversummer survival of inoculum for Colletotrichum crown rot in buried strawberry crown tissue. Plant Dis., 85:750-754.

Van de Weg. 1997. A gene-for-gene model to explain interactions between cultivars of strawberry and races of *Phytophthora fragariae* var. *fragariae.* Theo. Appl. Genet., 94: 445-451.

van Rijbroek, P.C.L., Meulenbroek, E.J. and van de Lindeloof, C.P.J. 1997. Development of a screening method for resistance to *Phytophthora cactorum.* Acta Hort., 439-181-183.

Wada, H., Cavanni, P., Bugiani, R., Kodama, M., Otani, H. and Kohmoto, K. 1996. Occurrence of the strawberry pathotype of *Alternaria alternata* in Italy. Plant Dis., 80:372-374.

Waldo, G.F. 1953. Sources of red stele disease resistance in breeding strawberries in Oregon. Plant Disease Reporter, 37:236-242.

Welch, N. and Gubler, W. 1994. Soil fumigation experiment in strawberries in the central coast district of California. 1994 Conference Proceedings, Annual International Res. Conf. on Methyl Bromide Alternatives and Emission Reductions. Number 17. Kissimmee, Florida.

West, P.M. and Hildebrand, A.A. 1941. The microbiological balance of strawberry root rot soil

as related to the rhizosphere and decomposition effects of certain cover crops. Can. J. Res., 19-C:199-210.

Wilcox, W.F. and Seem, R.C. 1994. Relationship between strawberry gray mold incidence, environmental variables, and fungicide applications during different periods of the fruiting season. Phytopathology, 84:264-270.

Wilhelm, S. and Koch, E.C. 1956. *Verticillium* wilt controlled. California Agriculture, 10:3, 14.

Wilhelm, S. and Sagen, J.A. 1974. A History of the Strawberry. University of California Division of Agriculture Publication 4031. Berkeley, California, USA.

Wilhelm, S., Storkan, R.C. and Wilhelm, J.M. 1974. Preplant soil fumigation with methyl bromide-chloropicrin mixtures for control of soil-borne diseases of strawberry—A summary of fifteen years of development. Agr. Environ., 1:227-236.

Wilson, L.L., Madden, L.V. and Ellis, M.A. 1992. Overwinter survival of *Colletotrichum acutatum* in infected strawberry fruit in Ohio. Plant Dis., 76:948-950.

Wing, K.B., Pritts, M.P. and Wilcox, W.F. 1994. Strawberry black root rot: a review. Advances in Strawberry Res., 13:13-19.

Wing, K.B., Pritts, M.P. and Wilcox, W.F. 1995. Biotic, edaphic, and cultural factors associated with strawberry black root rot in New York. HortScience, 30:86-90.

Winks, B.L. and Williams, Y.N. 1965. A wilt of strawberry caused by a new form of *Fusarium oxysporum.* Queensland J. Agr. Animal Sci., 22:475-479.

Xiao, C.L., Chandler, C.K., Price, J.F., Duval, J.R., Mertely, C.J. and Legard, D.E. 2001. Comparison of epidemics of *Botrytis* fruit rot and powdery mildew of strawberry in large plastic tunnel and field production systems. Plant Dis., 85:901-909.

Yuen, G.Y., Schroth, M.N., Weinhold, A.R. and Hancock, J.G. 1991. Effects of soil fumigation with methyl bromide and chloropicrin on root health and yield of strawberry. Plant Dis., 75:416-420.

Zreik, L., Bové, J.M. and Garnier, M. 1998. Phylogenetic characterization of the bacterium-like organism associated with marginal chlorosis of strawberry and proposition of a *Candidatus* taxon for the organism, '*Candidatus* Phlomobacter fragariae'. Intl. J. System. Bacteriol., 48:257-261.

Stone Fruit Diseases and their Management

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Abstract: Stone fruits comprising peach, plum, almond, apricot, cherry and nectarines are attacked by various fungal and prokaryotic (*i.e* bacteria, fastidious vascular bacteria) pathogens. Amongst fungal diseases, the most important ones are peach leaf curl, brown rot, powdery mildew, rust, leaf spots, peach scab, canker and die-back, black knot, wilt and root rot. Amongst prokaryotic diseases, crown gall, bacterial spot, canker, phony peach, almond leaf scorch and plum leaf scald are important. Stone fruit diseases are successfully managed by the combined use of cultural, chemical and biological measures supplemented with the cultivation of resistant cultivars. Foliar diseases are effectively controlled by timely sprays of fungicides. Proper pruning, applying chemicals and antagonist effectively manage canker and die-back. Removal of severely infested plants and isolation of diseased patches controls wilt, crown and root rot. Crown gall, a very serious disease of stone fruits is being managed successfully by a non-pathogenic strain of the causal bacterium. Diseases caused by fastidious vascular bacteria are managed by applying antibiotics and rouging infested plants.

1. Introduction

Stone fruits such as peach, plum, apricot, cherry, almond and nectarines are grown in sub-temperate and temperate regions of the world, however, in India, these are grown in and near the Himalayas and in very small acreage in south. The area under these fruits is increasing every year but the production is not increasing corresponding to the increase in area because these fruits are subjected to a number of abiotic and biotic stresses of which fungal and bacterial diseases are of utmost importance. Since many of the diseases attacking stone fruits are common so they have been discussed together under the host on which they are of economic importance.

2. Fungal Diseases

2.1 Brown rot

The disease is found in all countries where stone fruits are grown. Besides peach, the disease affects plum, prune, cherry, apricot and almond. It reduces yield by killing blossoms and twigs, and by rotting the fruit both on the tree and after harvest. In severe

infections, 50-75 per cent of the fruit may rot in the orchard and remainder may become infected before reaching the market.

2.1.1 Symptoms

Brown rot attacks blossoms, spurs, shoots and fruits. Symptoms may develop on a few or all of these plant parts during the growing season. Blossom blight is the first symptom during the spring and the attacked parts turn grey to dark brown. The fungus spreads through the peduncle and reach branches resulting in twig blight. Stem cankers develop usually from blighted twigs or fruit spurs. Fruit rot is the most destructive phase of the disease and is more common on mature fruit than on immature fruit. Small, circular, light brown spots develop on the surface of the fruit and expand rapidly under favourable conditions, destroying entire fruit in a few hours. Rotted fruit may fall to the ground or persist as mummies on the tree (Sharma and Kaul, 1988). Under wet and humid conditions, ash-grey tuffs of fungus develop over the surface of the lesions. These structures called sporodochia, produce conidia important in spread of the disease. Appearance of conidia on lesions is the most obvious characteristic of brown rot. Gupta and Byrde (1988) found *Monilinia laxa* (Aderh. & Ruhl.) Honey, to cause blossom wilt of apricot and almond in North India.

2.1.2 Causal organism

Brown rot disease is caused by *Monilinia fructicola* (Wint.) Honey, *M. fructigena* (Aderh. & Ruhl.) Honey, *M. laxa* (Aderh. & Ruhl.) Honey, and *M. laxa* (Aderh. & Ruhl.) Honey f. sp. *mali* Wormald sensu Harrison.

The mycelium produces chains of *Monilia* -type conidia on hyphal branches arranged in groups or tuffs (sporodochia). The sexual stage (apothecium) originates from pseudosclerotia formed in mummified fruit partly or wholly buried in soil or debris.

2.1.3 Etiology

The pathogen over winters as mycelium in mummified fruit on the tree and in cankers of affected twigs or as pseudosclerotia in mummies in the ground. In the spring, the mycelium in mummified fruit on the tree and in the twig cankers produces new conidia, while the pseudosclerotia in mummified fruit buried in the ground produce apothecia, which form asci and ascospores. Both conidia and ascospores can cause blossom infections. Conidia are wind blown or may be carried by rainwater and splashes or insects to floral parts and initiate infection with in a few hours. Infected floral parts soon rot, while twig exhibit depressed, reddish brown shield shaped cankers. The surface of infected plant parts is soon covered with conidial tuffs and conidia from these tuffs initiate secondary infections. Conidia produced on twig cankers during humid weather in summer infect ripening fruit. The fungus causes the maceration of infected tissues by secreting pectolytic enzymes in invaded fruits. Infected fruits either remain hanging on tree or fall to the ground. The fungus may persist for two or more years on the

mummified fruits fallen on the ground.

2.1.4 Epidemiology

Warm, wet, humid weather is particularly favourable for brown rot. At 20°C, 3-5 hr of wetting may lead to significant infection. With 24 hr of wetting, severe blossom infection may result regardless of temperature. Optimum temperature for blossom infections of peach and cherry is 25°C (Biggs and Northover, 1988).

2.1.5 Control

Brown rot of stone fruits can be controlled best by completely controlling the blossom blight phase of the disease by spraying two or four times with an effective fungicide from the time the blossom buds show pink until petal fall. Benomyl, thiophanate methyl, iprodione, vinclozolin, captan, thiabendazole, triflumizole, dicloran, cyproconazole + dithianon and chlorothalonil are the fungicides used for brown rot control (Byrde and Willetts, 1977, Osorio *et al.*, 1993, Ruegg and Siegfried, 1993, Balardin *et al.*, 1994, Andrade and Matos, 1996). Resistant strains of the brown rot fungus have developed to the systemic fungicides, therefore, these chemicals are generally used in combination with the broad spectrum run fungicides such as captan or sulphur.

Twigs bearing infected blossoms, cankers should be removed as early as possible to reduce the inoculum available for fruit infections later in the season and for over wintering.

To control brown rot in ripening fruit, the fungicides are applied to the trees a few weeks before harvest and continue until just before harvest.

To prevent infection at harvest and during storage and transit, fruit should be picked and handled with care to avoid punctures and skin abrasions on the fruit. All fruits with brown rot spots should be discarded. Post-harvest brown rot can be reduced by dipping fruit in carbendazim, thiabendazole, benomyl and thiophanate methyl solution (Sharma and Kaul, 1988a, 1989, Sharma, 1994), before storing in controlled atmosphere storage having 11% carbon monoxide, 4% oxygen and 5% carbondioxide mixture (Kadar *et al.*, 1982).

Biological control of post-harvest brown rot has been reported by the application of *Bacillus subtilis*, *Epicocum nigrum* and *Pseudomonas* spp. but is not used commercially (Pusey and Wilson, 1984, Smilarick *et al.*, 1993, Madrigal *et al.*, 1994, Foschi *et al.*, 1995).

2.2 Powdery mildew

Different species of *Podosphaera* Kunze ex Lev. namely *P. clandestina* Wallr. Ex Fr, *P. leucotricha* Ell. & Ev*., P. oxycanthae* and *P. tridactyla* (Wallr.) de Bary and *Sphaerotheca pannosa* Wallr. & Lev. cause powdery mildew in almond, apricot, peach, plum and cherry. The disease is very serious in nurseries where seedling stocks infected early in the growing season remain stunted. Khan *et al.* (1975), Sharma (1985), Kaul (1967) and Pandotra *et al.* (1968) reported occurrence of powdery mildew on

almond and plum due to *Phyllactinia* Lev. and *Uncinula* Lev.

2.2.1 Symptoms

The fungus infects leaves, young shoots and fruits. On the young leaves the disease appears at first as blister like areas that soon become covered with grayish white patches of fungus growth. Infection causes leaf distortion, curling and premature leaf fall. White patches of fungus appear on green shoots, which may become curved at the tips. Buds and flowers may also be directly attacked. In that case buds either fail to open or open improperly while flowers are discoloured, stunted, and eventually dry up. Infected fruits first show circular spots which may spread over the whole fruits. The colour is at first pinkish and later becomes dark brown. Epicarp of the fruit becomes leathery and hard.

2.2.2 Etiology

The fungus over winters as mycelium in the buds. Cleistothecia are only rarely found on peach and apricot but are common on rose. Primary inoculum is therefore, represented by conidia produced by the overwintering mycelium, and sometimes by ascospores.

Conidia and ascospores are carried by wind to green tissues where they germinate, each producing a germtube which produces a fine hypha. This hypha penetrates the host directly through the cuticle and when it reaches the cell lumen of an epidermal cell it forms a globose haustorium by which the fungus obtains its nutrients. The aerial mycelium on the other hand produces short conidiophores each bearing a chain of egg shaped conidia. These conidia are dispersed by wind and infect expanding leaves, young shoots and fruits until they are 2-3 cm in diameter. The optimum temperature for conidia germination lies between 21 and 27°C (Weinhold, 1961).

2.2.3 Control

The disease can be effectively controlled by three sprays of wettable sulphur or carbendazim/thiophanate methyl before opening of blooms, at petal fall and two weeks later (Anonymous, 1995). New fungicides viz myclobutanil, fenarimol, flusilazole, pyrifenox, triademefon, captafol and tebuconazole provide good control of powdery mildew in stone fruits (Torre Almaraz and Ceballos Silva, 1990, Dong *et al.*, 1991, Jones *et al.*, 1993 and Huang *et al.*, 1995).

2.3 Peach Leaf Curl

The disease is prevalent in all the peach growing areas of the world. In India, it is prevalent in Himachal Pradesh, Jammu and Kashmir, Assam, Bihar and Kumaon Hills of Uttar Pradesh (Jain, 1962, Ferraris, 1928 and Sydow and Butler, 1916). Disease causes defoliation of peach trees, which may lead to small fruit or fruit drop. Different species of *Taphrina* Fr. cause leaf, flower and fruit deformation on stone fruits and

forest trees. Thus *T. deformans* (Berk) Tul. causes leaf curl on peach and nectarine, *T. communis* and *T. pruni* Tul. cause plum pocket on American and European plums, respectively, *T. cerasi* causes leaf curl and witches broom on cherries and *T. coerulescens* causes leaf blister of oak. The most common losses, however, are those caused primarily on peach, nectarine and sometimes plum.

2.3.1 Symptoms

The disease is noticeable on the leaves within a month after bloom. Infected leaves are two to three times larger than healthy leaves and show curling. They are thickened and puckered and often develop red or purple colour. Old infected leaves become necrotic, wither and have premature fall. Infected twigs become swollen and are killed. Flowers and fruits when infected, drop rapidly. The fungus also infects apricot where the affected leaves on the growing tips are easily distinguishable as they give an effect of red flowers blooming on the tree (Gupta *et al.*, 1973a).

2.3.2 Causal organism

Leaf curl is caused by *Taphrina deformans* (Berk.) Tul. Asci of the fungus break through the cuticle of the distorted leaf. They appear as a powdery grey felt like area on the thickened leaf. The mature asci on being ruptured release ovoid ascospores. During periods of spring rain, the ascospores produce large numbers of blastospores or bud conidia by budding.

2.3.3 Etiology

The fungus overwinters as ascospores or thick walled conidia on the tree, perhaps on the bud scales. In spring, these spores are washed, splashed, or blown onto young tissues, germinate and penetrate the developing leaves and other organs directly through the cuticle or through stomata. The binucleate mycelium grows between cells and invades the tissues, inducing cell elongation and cell division, which results in the enlargement and distortion of the plant organs. Later, numerous hyphae grow outwards in the area between the cuticle and epidermis, break into their component cells and produce asci. The asci enlarge and break through the cuticle to form a compact, felt like layer of naked asci. The ascospores are released into the air, carried to new tissues and buds to form conidia. All organs become resistant to infection, as they grow older.

2.3.4 Epidemiology

Leaf curl is damaging when the weather is cool and moist. Rain is necessary for infection while low temperatures are considered to retard maturation of leaf tissue, thus prolonging the time of infection. The fungus can penetrate young leaves at temperatures between 10 and 21.1^{O}C but only weakly below 7.2^{O}C. The disease severity can be predicted if average maximum temperature during later half of February and March

fluctuates in between 15-20°C (18°C optimum) alongwith high humidity or precipitation (Agarwala *et al.*, 1966).

2.3.5 Control

The disease can be easily controlled by a single fungicide spray, if applied either before the bud swell in spring or after the general leaf fall in autumn (Sohi *et al.*, 1968). Such sprays can be of a fungicide belonging to Copper oxychloride, carbendazim (Sharma *et al.*, 1987, 1988a, Bhardwaj and Ved Ram, 1995) or lime-sulphur (Bose and Sindhan, 1972). Other fungicides effective against peach leaf curl are Ziram, Captafal, Thiram, Dodine, Captan and Chlorothalonil (Rana and Jain, 1992, Brunelli and Ponti, 1993, Ponti *et al.*, 1993 and Tate and Wood, 1994).

Peach cultivars Red Heaven, Favorita, Morettinijaune Gold, Fertilia-1, Eixan-1, Modeline Pouyet, Flacara X HB9-35, Flacara X Miorita and J.H. Hale X Sunbeam are either tolerant or resistant to the disease (Coroianee and Ivascu, 1981, Ritchie and Warner, 1981). Nautiyal *et al.* (1988) classified four cvs. viz., Stark Early Giant, Starking Delicious, World Earliest and Tesia Samisto as resistant. Three cvs. viz., Bed Will's Early, July Alberta and World's Earliest showed tolerant reaction to leaf curl disease (Sharma and Badiyala, 1994).

Besides above, the trees must be kept free from diseased leaves. Fallen and dried leaves should be raked and destroyed.

2.4 Rust

Stone fruits viz., Peach, Plum, Almond, Apricot and Prunes are affected by this disease and is of worldwide occurrence but is severe only is warmer areas. It often causes less damage as it appears late in the season after the trees have attained sufficient growth for the production of the following years. The disease was first reported from Australia in 1890 (Dunegan, 1938). Later on it was reported from China, Japan, Brazil, New Zealand and is now widely distributed in Europe and North America also. In India, the disease was reported by Butler and Bisby in 1931. The disease has been observed to be very serious on almonds and plum in northern parts (Sharma *et al.*, 1988b, Bhardwaj and Shyam, 1986, Kaul, 1971, Waraich and Khatri, 1977, Singh *et al.*, 1976).

2.4.1 Symptoms

On peach, the symptoms are evident on leaves, stems and fruit. Symptoms on leaves start as pale yellowish spots on both surfaces which later change into bright yellow islands prominent on the upper surface. On the lower surface cinnamon brown dusty pustules appear. Severe infection leads to defoliation.

Fruit infection occurs only when conditions are favourable for the fungus to cause infection at a relatively late stage of fruit maturity. Water soaked dark green spots appears on fruits and their growth gets arrested. The spots become sunken which are deeper green than the surrounding tissue and later turn deep yellow.

Twig infection is uncommon on peach but on plum, the bark gets infected during the early spring when buds are swelling.

2.4.2 Causal organism

Persoon (1801) first named the causal agent as *Puccinia* Pers. However, Arthur (1906) erected a new genus *Tranzschelia* Arthur on the basis of teliospore morphology. *Tranzschelia* is distinguished from *Puccinia* by virtue of the fasciculate bunching of teliospores attached by their pedicels to a common basal cell and also by the ready separation of two cells of the spore. *T. pruni-spinosae* (Pers.) Diet. is separated into two varieties according to the combination of aecial and telial hosts and differences in the morphology of the teliospores. *T. pruni-spinosae typica* was proposed for the variety found on wild spp. and *T. pruni-spinosae discolor* for those attacking the cultivated spp.

Hawksworth *et al.* (1983) have described seven species of *Tranzschelia* genus. However, in India only *T. discolor* (Kaul, 1971, Waraich and Khatri, 1977) and *T. prune-spinosae* (Waraich and Khatri, 1977, Bhardwaj and Shyam, 1986) have been recorded.

Bolkan *et al.* (1985) demonstrated specialized parasitism by various isolates of *T. discolor* and named various varieties of *T. discolor* as *T. discolor* f.sp. *dulcis* on almond, *T. discolor* f.sp. *persicae* on peach and *T. discolor* f.sp. *domestica* on prunes and other hosts are namely, plum (*P. salicina*), nectarines (*P. persica*), apricot (*P. armeniaca*) and cherries (*P. avium*).

2.4.3 Etiology

Stone fruit rust over winters in twigs as uredosori and produce uredospores in spring (Jafar, 1958). However, *Anemone coronaria* has been reported to be the alternate host. Jafar (1959) further showed that canker developed on young wood also produce spores in late spring, thus serving as source of infection. The extent of overwintering inoculum is dependent on twig infection in the late season which remain dormant till spring. Perennial mycelium in 1 to 2 year old shoots has also been shown as the perpetual sources of infection (Nadazdin and Nadazdin, 1977, Tevistdale *et al.*,1994). The infection can take place at 15-23$^{\text{O}}$C and the incubation period is 7 and 70 days at higher and lower temperatures, respectively. Leaf and fruit infection occurs under high humidity (Goldworthy and Smith, 1931) which is favoured by abundant rains during August (Vitanov, 1976). Epidemics are favoured by rainy period in summer when temperature is around 20-30$^{\text{O}}$C which is most favourable for disease development (Simeone *et al.*, 1985).

2.4.4 Control

Dippenaar (1941) showed zinc lime sulphur to be very effective against leaf rust of apricot and peach. Later on various fungicides like zineb, captan, thiram etc. were tried and Jafar (1959) suggested zineb as most effective. Captan and Thiram to be effective and superior to a combination of lime sulphur and colloidal sulphur. Decker

and Buchanan (1975) reported that trees sprayed with zineb five times during the grow-
ing season retained normal foliage upto mid November and those sprayed with sulphur
and triforine upto late October only. Fruit yield increased by 35% on trees sprayed with
zineb while triforine did not increase yields. Michailides and Ogawa (1986) found wet-
table sulphur and manocozeb effective in reducing the disease in sprayed trees.

New fungicides like Ergosterol biosynthesis inhibitors propiconazole, RH-2161
and RH-3866 (myclobutanil) also showed strong curative activity against *I. discolor*
infections in plum leaves. Foliar spray (25-50 ppm) of these fungicides applied during
the latent period upto 7 days after inoculation completely suppressed the disease. When
applications were made after the appearance of symptoms, they inhibited further le-
sion growth and sporulation. Shabi *et al.* (1990) showed that rust incidence was low in
plants treated with captan, maneb, dithianon, bitertanol and systhane. Recently, Ram
et al.(2000) reported sprays of SAN-619 (cycloconazole) or Contaf + Kavach very
effective in reducing the incidence of apricot rust.

In plum, Voromin *et al.* (1982) showed californian monopodal and Prune to
possess resistance. Corazza *et al.* (1984) studied the reaction of about 25 cvs. under
natural conditions and showed Brusca, Fracasso, Frasso and San Castrese to be mod-
erately resistant. Ram *et al.* (2000) found three cultivars of apricot namely Fragmore
Early, Kaisha and Shakarpara resistant to rust under Indian conditions. Simeone *et al.*
(1984) showed Japanese plums to be less susceptible than European plums and Santa
Rosa plums to be immune. In Almond, Thin Shelled, Badamajor and Spillo No-2 were
reported resistant, 1XL, Ribba Selection, Spillo - 7 were moderately resistant.

No attempts have been made to control this disease with biological methods
but few reports of parasitism of the pathogen with other organisms exit. *Tuberculina
persicina* has been reported to parasitize *Transzchelia pruni-spinosae* and *Anemone
coronaria* (Borkai-Colon, 1959). In India, Kaul (1967) showed that *Derluca filum*
parasitizes rust fungi in Kashmir Valley.

2.5 Leaf spots

Spots of different shapes and sizes are produced on the leaves of stone fruits due to the
infection caused by many pathogens which many remain restricted and turn necrotic.
Among various leaf spots *Coryneum* blight/shot hole, cherry leaf spot, frosty mildew,
target leaf spot etc. are major diseases.

2.5.1 *Coryneum* blight

The disease also known as shot hole, California peach blight, fruit spot, winter blight
and pustular spot) is commonly found on peach, apricot, nectarine, sweet cherry and
almond. In India, Munjal and Kulshrestha (1968) first reported the occurrence of this
disease on peach, apricot and almond and on cherry by Puttoo and Rajdan (1991) from
Jammu and Kashmir. The disease has been reported in severe form in lower hills of
Kullu Valley, stone fruit areas of Mandi, Solan and Shimla districts of Himachal Pradesh
(Gupta *et al.*, 1973b).

2.5.1.1 Symptoms

Blight symptoms appear both on twigs and fruits. On twigs small, purple and slightly raised pustules appear in late December or early January which later expand into necrotic cankers. Some of the severely attacked twigs and new shoots are killed in summer. The spots on fruits are small circular, deep purple, raised and appear after the peach fruits attain almost half size by the middle of May. These spots soon turn brown, coalesce, give scurfy appearance to the fruit. Such fruits fall an easy prey to the secondary invasion by soft rot fungi and develop soft rot. On leaves dark brown, scattered lesions enlarge rapidly and abscission of the diseased area results in shot hole.

2.5.1.2 Causal organism

The disease is caused by a fungus *Wilsonomyces carpophilus* (Lav.) Adaskaveg (Adaskaveg *et al.*, 1990). The earlier generic names for this fungus include *Clasterosporiumi* Schw*., Coryneum* Nees ex Fr. and *Stigmina* Sacc. The fungus produces black dot like fruiting bodies on the necrotic cankers and twigs. These fruiting bodies produce numerous conidia which are thick walled, ellipsoidal or fusiform, with three to five transverse, double walled septa and infect buds and twigs of trees in winter with the onset of rains.

2.5.1.3 Predisposing conditions

Disease spread is continuous as the day temperature rises and it appears in epidemic form with the occurrence of frequent rains in subsequent months of spring and summer (Gupta *et al.*, 1972/74).

2.5.1.4 Control

The disease is effectively controlled with fungicide sprays. Two sprays with captan 50WP (0.3%) first at leaf fall stage in autumn and second at bud swell stage in spring provide effective control of the disease. These sprays need to be continued for next few years to obtain complete elimination of the disease from the orchard.

Peach cultivars Early Crawford, Foster, Lovel Palora and apricot cultivar Filipol are resistant to shot hole (Angelov, 1980).

2.5.2 Cherry leaf spot

Cherry leaf spot is the most serious disease in nursery and orchard plantations and is caused by *Coccomyces hiemalis* Higgins (*Blumeriella jaapi* (Rehm. Arx). It is also known as yellow leaf since bright yellow foliage develops soon after infection. Disease appear on the upper surface of the leaf as small, circular, purple spots which turns brown and form a definite boundary. The centre of the spot may become necrotic and fall off giving a shot hole appearance. The fungus overwinters in infected leaves on the orchard floor and produces fruiting bodies (apothecia) which normally mature

during the blooming period. Upon wetting, the ascospores are released and cause primary infection. After appearance of the lesions, acervuli form near the centre on the underside of the leaf. Conidia are responsible for the secondary spread of the disease.

Cherry leaf spot can be managed with fungicides such as Chlorothalonil, Dodine, Baycor, Ancil, Punch, Score and Topas (Jones *et al.*, 1993, Frisullo *et al.*, 1993, Larsky and Necesany, 1995). A strain of Mazzard cherry is highly resistant to leaf spot and can be used in the disease management. Zhukov and Kharitonova (1982) reported that Almoz a hybrid obtained by crossing *Prunus carasus* and *P. padus* is resistant to cherry leaf spot.

Another leaf spot of cherry caused by *Cercospora circumscissa* Sacc. has been reported from India (Verma and Gupta, 1979). The disease is effectively controlled by application of Captan (0.2%) in October before leaf fall.

2.5.3 Frosty mildew

Frosty mildew of peach and apricot is caused by *Cercosporella persica* Sacc. The disease is common in neglected orchards and nurseries. Corresponding to the pale green areas on the upper leaf surface, creamy white fungal growth appears on the lower surface (Sohi *et al.*, 1964, Sharma and Paul, 1986a).

2.5.4 Target leaf spot

Target leaf spot of peach is caused by *Phyllosticta persicae* Sacc. On the upper surface of the leaf, concentric rings develop giving a characteristic target board appearance. Black, minute fungal fruiting bodies can be seen scattered throughout the necrotic areas on the lower leaf surface (Sharma and Paul, 1986b).

Fungi reported to cause leaf spots in stone fruits are: *Polystigmina rubra* (Desm.) Sacc., *Cercospora prunicola* Ell. & Ev., *C. rubrotincta* Ell. & Ev., *Alternaria pruni, A. tenuis, Polystigma rubrum* and *Gleosporium amygdalium* on Almond; *Phyllosticta prunicola, P. cerasicola, Colletotrichum gloeosporioides, Astomella mali, Phoma pomorum* on Apricot; *Cercospora rubrotincta, C. circumscissa* Sacc., *Pithomyces sacchari* and *Ginomonia erythrostoma* on Cherry; *Pestalotiopsis disseminata, Alternaria alternata, Cercospora prunicola, Sarcinella prunicola, Gloeosporium laeticolor* and *Colletotrichum gloeosporioides* on Peach; *Polystigmina rubra* on Plum.

2.6 Peach scab

Peach scab is also known as freckles or black spot. The disease occurs primarily in the warmer peach producing areas. It is worst where a good spray schedule is not followed early in the season. Besides peach, the disease occurs on apricot, nectarines and cherry. Bagghee and Singh (1960) and Baruah *et al.* (1980) reported occurrence of this disease on peach from India.

2.6.1 Symptoms

The disease occurs on the twigs and leaves but is most conspicuous on the fruit. On

fruits, olivaceous spots 2-4 mm in diameter appear which gradually become circular, dark -olivaceous lesions. In case of severe infection, the individual spots merge and form a uniform, dark-olivaceous, velvety blotch. The fruit becomes abnormal in shape and cracks, thus deteriorates the fruit quality. On twigs fungus produces dark brown, oval lesions of 3-6 mm diameter. The under surface of the leaf shows angular, pale green areas which gradually turn dark brown. Dark brown long and narrow lesions are formed on the midrib.

2.6.2 Causal organism

The disease is caused by *Venturia carpophila* Fisher, the imperfect stage is known as *Fusicladium carpophilum* (Thuem.) Oudem or *Cladosporium carpophilum* (Thum).

2.6.3 Etiology

The fungus overwinters in lesions on the twigs. Conidia are airborne and water borne and are most abundant 2 to 6 week after the shuck split stage of development. If weather conditions are favourable, infection begins to occur at about shuck fall. The fruit remain susceptible until harvested. Spores from the fruit reinfect the twigs and leaves (Lawrence and Zehr, 1982).

2.6.4 Control

The disease is controlled primarily with fungicide sprays, although pruning helps increase air circulation and reduces the chances of infection. Low lying field should not be selected as planting sites. Timely application of most of the standard fungicides can control the scab. Sprays for the control of brown rot also control late scab infections. Huang *et al.* (1993) found bitertanol spray with summer oil week before bloom followed by bitertanol spray twice at 15-20 days interval reduced scab incidence and increased fruit set.

2.7 Black Knot of Plum and Cherry

The disease occurs on cultivated and wild plums and cherries. It causes conspicuous, 2 to 25 cm long, black knotty swellings on one side of, or encircling, twigs and branches. The knots may be several times the diameter of the limbs and make heavily infected trees appear quite grotesque. Infected plants become worthless after a few years as a result of limb death and stunting of the trees. The fungus, *Dibotryon morbosum* (Sch.) Theis and Sydow, produces conidia on free hyphae and ascospores in perithecia formed on the black knots. Conidia and ascospores are spread by wind and rain, and in early spring can penetrate healthy and injured woody tissue of the current season's growth. Large limbs are also attacked at points of developing small twigs. The fungus grows into the cambium and xylem parenchyma and along the axis of the twigs. After 5 to 6 months, excessive parenchyma cells are produced and pushed outward, forming the swelling. The following spring conidia are produced on the knot surface, giving it a

temporary olive-green velvety appearance. The knots enlarge rapidly during the second summer, and in their surface layer perithecia are formed that develop during the winter and release ascospores the following spring (Wain Wright and Lewis, 1970).

The disease can be controlled by pruning and burning of all black knots and destruction of black knots or of all affected wild plums and cherries near the orchard. Spraying the orchard trees before and during bloom with sulphur, captan, chlorothalonil, fenbuconazole, thiophanate methyl or fixed copper fungicides protects trees from infection (Rosenberger and Gerling, 1984; Northover and Mcfadden- Smith, 1995).

2.8 Canker and dieback

Silver leaf, *Eutyta* dieback and *Leucostoma* canker are important diseases in this group.

2.8.1 Silver leaf

Silver leaf disease caused by *Chondrostereum purpureum* (Pers.; Fr.) is a destructive disease of stone fruit trees, and is present in most temperate zone production areas. The foliage of affected plants show a metallic luster in contrast to the normal green colour of the healthy trees. Diseased limbs show dark discolouration of the heartwood. The fungus is a wound parasite, usually enters through pruning cuts. The risk of infection and disease is enhanced if trees are pruned during late winter and early spring, when concentrations of nutrients in sap, such as nitrogen and carbohydrates are highest. Infection is also favoured when trees forced into rapid growth by heavy pruning, particularly in nurseries when rootstocks are headed to the scion buds in early spring. To reduce the risk of disease, all potential sources of infection should be destroyed or burned. Application of fungicidal paste/paints immediately after pruning on the cut ends surfaces. Application of an antagonist (*Trichoderma viride*) spores on the fresh pruning wounds provide an effective control of this disease. Other promising antagonists are *Truncatella* spp. and *Gliocladium* spp. (Dubos and Ricard, 1974, Corke, 1978). Pruned cut of the trees when sprayed immediately with 0.8 per cent captafol provide effective control. Foliar application of iron compounds has been shown to reduce the disease incidence (Patli, 1981).

2.8.2 *Eutypa* Dieback

Eutypa dieback of apricot is caused by *Eutypa armeniacae* Hansford and Carter and now designated as *Eutypa lata* (Pers.; Fr.) Tul. & C. Tul., occurs widely throughout temperate regions of the world. The disease is more prevalent where natural rainfall is 60 cm or more. Canker or dieback symptoms starts from pruning wounds. Leaves on the portion of the branch beyond an established canker may wilt suddenly. In advanced stages it produces soft rot symptoms in the xylem and makes these branches brittle and when bent, snaps easily and suddenly with a short-gained fracture (English and Davis, 1978). Almond and peach trees are less susceptible to this pathogen.

The disease can be suppressed by the combined use of benomyl and antagonist

(*Fusarium lateritium*), which is more effective than either treatment alone when applied on fresh wounds (Carter and Price, 1985).

2.8.3 *Leucostoma* canker

Leucostoma canker, also called perennial canker, *Cytospora* canker, or *Valsa* canker, is most serious on peach and other stone fruits. The diseased bark becomes dark, smelly and oozes gum. Later the bark shrivels and separates from the underlying wood and from the surrounding healthy bark. Small pimple like pycnidia appear on the dead bark. Later the shriveled bark slough off, exposing dead wood beneath. The cankers increase in size each year and become unsightly, rough swellings. Many twigs and branches dieback as a result of cankers that girdle them completely.

    Leucostoma canker result mostly from infections by conidia (*Cytospora*). Perithecia and ascospores (*Leucostoma*) are not common. During wet weather, the spores ooze out of the pycnidium and may be splashed by rain or may be spread by insects and humans. Most infections take place in late fall or early winter and in late winter or early spring. Weakened, injured trees, however, may be infected throughout the growing season. Both mycelium and the conidia of the fungus overwinters on the infected parts. Small twigs are infected through injuries or leaf scars. In larger branches, the fungus enters through wounds and form canker.

    Control measures include good cultural practices, watering and fertilization to keep the trees in good vigour, avoiding wounding and severe pruning of trees, removing cankers from trunks and large branches during dry weather and treating the wound and all pruning cuts with a disinfectant and a wound dressing, removing and burning cankers and dead branches and twigs, pruning as late in the spring as possible, and spraying with benomyl immediately after pruning and before it rains (Biggs, 1989, Jones and Aldwinkle, 1990).

    Other fungi reported to cause dieback and canker diseases in stone fruits are *Coniothyrium pyrinum, Crytosporiopsis* sp., *Diplodia sarmentorum, Fusicoccum amygdali, Fusarium oxysporum* and *Thenatophorus cucumeris* on almond; *Phoma glomerata, Sphaeropsis* sp., *Botryosphaeria dothidea, Micordiplodia pruni* and *Schizophyllum commune* on Apricot; *Fusarium amygdali* and *Botryospharia berengeriana* f.sp. *persicae* on peach.

2.9 *Verticillium* wilt

Verticillium wilt of stone fruit, also called black heart, verticilliosis, *Verticillium* hadromycosis, and vascular wilt, occurs in many parts of the world and causes serious economic losses. In India, Agarwala and Gupta (1968) reported occurrence of this disease on almond, apricot, cherry, peach and plum. The leaves of infected plants or of parts of infected plants lose turgidity, become flaccid and lighter green to greenish yellow, droop and finally wilt, turn yellow and then brown and die. In cross sections of infected stems and twigs, discolored brown areas appear as a complete or interrupted ring consisting of discoloured vascular tissues. In the xylem vessels of infected stems and roots, mycelium and spores of the fungus may be present. Some of the vessels

may be clogged with mycelium, spores, or polysaccharides produced by the fungus (Pegg, 1974).

2.9.1 Causal organism

Two species of *Verticillium, V. albo-atrum* Reinke & Berth. and *V. dahliae* Kleb cause *Verticillium* wilts in most plants. Both produce conidia that are short lived. *Verticillium dahliae* also produces micorsclerotia, whereas *V. albo-atrum* produces dark thick walled mycelium but not microsclerotia. *V. albo-atrum* grows best at 20-25°C, whereas *V. dahliae* prefers slightly higher temperatures (25-28°C). Both species, however, can overwinter as mycelium within perennial hosts, in propagative organs, or in plant debris. Fungus penetrates young roots of host plants directly or through wounds.

2.9.2 Control

Since the pathogens are soil inhabitating and infect through the roots, cannot be controlled by use of protective sprays. Plant for new orchards should be obtained from nurseries known to be free from this disease. Soil fumigation with chloropicrin or with methyl bromide and chloropicrin destroys the inoculum (Wilhelm and Ferguson, 1953). Application of chelates, such as ferrous sulphate, manganese sulphate and molybdenum considerably increase the tree resistance in apricots (Kibishauri and Tsiklauri, 1979).

2.10 Root and crown rot

2.10.1 *Armillaria* root rot

Armillaria root rot also known as shoe string root rot (*Armillaria mellea* (Vahl.) Quel.) is widely distributed through out the stone fruit growing areas. Affected trees exhibit reduced growth, smaller yellowish leaves, die-back of twigs and branches and gradual or sudden death of the tree. White mycelial fan-shaped strands of the fungus on the roots is a distinguishing feature of the disease.

The fungus overwinters as mycelium or rhizomorphs in diseased trees or in decaying roots. Control measures include removal of badly infected plants and digging a trench (1.8 m) around infected trees and kept open for several years to prevent growth of rhizomorphs to adjacent trees and fumigation of the infected area to destroy the fungus in the soil before Armillaria killed trees may be replaced. Resistant root stocks such as Myrobalan 29 and Mariana 2624 should be used (Anderson, 1956). Nicolas and Bonet (1996) recommended Myran for use in light well drained soils, whereas Ishtara (plum x peach hybrid) for heavier and moist soils to have a lower mortality than peaches grown from seed.

Another closely related fungus causing root rot similar to shoe string root rot is *Armillaria tabescens* (Scop.; Fr.) Denis *et al.* The symptoms and management practices are almost similar to those described for shoe string root rot.

2.10.2 *Phytophthora* **root and crown rot**

Phytophthora root and crown rot of stone fruits are important in cherries and peaches. *Phytophthora* spp. viz. *P. cactorum* (Leb. and Cohn.) Sch., *P. cambivora* (Petri) Busim., *P. cinnamomi* Rands, *P. cryptogea* Pethybr. & Laff., *P. megasperma* Drechsl., *P. citricola* Swada and *P. dreschleri* cause crown rot of peaches and cherries (Taylor and Washington, 1984).

2.10.2.1 Symptoms

Above ground symptoms of affected stone fruit trees often appear first as insufficient extension of shoot growth. Leaves become sparse, small and chlorotic. Fruits may remain small in size. The disease produces cankers at or below the ground line in the root-crown area. Infected bark becomes brown and is often soft and mushy or slimy when wet. Cankers may extend upto the point of origin into the root system and upto the trunk to the bud union and occasionally into scaffold branches. Girdling of the entire crown region may lead to the death of the tree. The pathogen survives in soil for several years. Pathogen population is directly influenced by soil moisture and temperature and a soil temperature of 12-20$^{\text{O}}$C with pH 5-6 is best for survival of the fungal propagules (Rana and Gupta, 1984).

2.10.2.2 Control

The disease can be best controlled by superficial heating of the localized lesions with the help of blow lamp or scarifying cankered lesions to healthy tissues followed by application of Chaubattia paste or copper oxychloride paint and irrigation of 0.3% Dithane M-45 or 0.5-1% Blitox/Fytolan/Blue copper in 30 cm radius around free trunk (Agarwala, 1970, Rana and Gupta, 1981 and 1983). Soil application of systemic fungicides *i.e.* metalaxyl and soil or foliar application of alliette reduced *P. cactorum* incidence in peach (Taylor and Washington, 1984).

Besides chemical control other strategies of *Phytophthora* root and crown rots include careful soil water management and genetically resistant rootstocks. It is advisable to keep graft union atleast 30 cm above soil level. Precaution must be taken to apply benomyl, carbendazim/ thiophanate-methyl in crown rot affected trees as it is bound to increase the severity of the disease (Byrde and Jordan, 1977).

3. Bacterial diseases

Three bacterial diseases viz., crown gall, bacterial spot and canker are the most important in stone fruits.

3.1 Crown gall

The disease is world wide in its occurrence. It affects woody and herbaceous plants belonging to 140 genera of more than 60 families (DeCleene and DeLey, 1976). It is

mostly found on pome and stone fruit trees, brambles and grapes. In India, the occurrence of disease is reported on cherry, almond, apricot, peach and plum (Durgapal, 1974, Jindal and Sharma, 1988a). The incidence of crown gall is found more in nursery plants compared to mature trees in orchards. Infected plants grow poorly and their yield is reduced. Severely infected plants may die.

3.1.1 Symptoms

Crown gall first appear as small, round, whitish, soft overgrowths on the stem and roots particularly near the soil line. As tumor enlarge, their surfaces become convoluted and other tissues become dark brown due to the death and decay of the perithecial cells. The tumor may appear as an irregular swelling and may surround the stem or root, or it may lie outside but close to the outer surface of the host. Some tumors are spongy and may crumble, others become woody and hard, looking knotty and reaching sizes upto 30 mm in diameter. Affected plants become stunted, produce small, chlorotic leaves and are more susceptible to adverse environmental conditions especially to winter injury.

3.1.2 Causal organism

The disease is caused by *Agrobacterium radiobacter* pv. *tumefaciens* (Smith & Twon.) Conn. The bacterium is rot shaped with few *peritrichous flagella*. Virulent bacteria carry one to several large plasmids. One of these plasmids carries the genes that determine the host range of the bacterium and the kinds of symptoms that will be produced. The most characteristic property of this bacterium is its ability to introduce part of the Ti-plasmid (T-DNA) into the plant cells and to transform normal plant cells to tumor cells in short periods of time. Transformed cells then synthesis specific chemicals called opines, which can be utilized only by bacteria that contain an appropriate Ti-plasmid. This property makes the bacterium a genetic parasite since a piece of its DNA parasitizes the genetic machinery of the host cell and redirects the metabolic activities of the host cell to produce substances used as nutrients only by the parasite (Zambryski, 1992).

3.1.3 Etiology

Crown gall bacterium may persist in field soil for atleast one year, or considerably longer if large amounts of infected root residues remain in soil when trees are removed. Crown gall infections are initiated at wounds at the crown or on the roots and rarely on trunks and limbs. The bacterium first attacks the wounded host cells and than transfer T-DNA into the cell. Infections are favoured by moist, alkaline, poorly drained soils and can be stimulated by the feeding damage of plant parasitic nematodes. When temperatures are above $20^{\circ}C$, gall become visible two to four weeks after infection. At low temperature, infection and disease development is delayed (Moore, 1976).

3.1.4 Control

The use of disease free nursery stock is extremely important to avoid the disease.

Susceptible nursery stock should not be planted in fields known to be infested with the pathogen. Infested fields should be planted with corn or other grain crops for several years before they are planted with nursery stock. Since the bacterium enters only through fresh wounds, wounding of the crowns and roots during cultivation should be avoided, and root chewing insects in the nursery should be controlled to reduce crown gall incidence. Nursery stock should be budded rather than grafted because of the greater incidence of galls on graft than on bud unions.

Excellent biological control of crown gall is obtained by soaking germinated seeds or dipping nursery seedlings or root stocks in a suspension of a particular strain (No 84) of *Agrobacterium radiobacter*. This strain of bacteria is antagonistic to most strains of *A. tumefaciens*. The antagonist controls crown gall initiation by establishing itself on the surface of the plant tissues, where it produces the bacteriocin agrocin 84 (Kerr, 1980). Unfortunately, some strains of *A. tumefaciens* inherited from strain 84 are resistanct to Agrocin 84. Therefore, a new strain (K-1026) is now used because it lacks the ability to transfer its resistant gene to pathogenic *Agrobacterium* strains (Jones and Kerr, 1989).

3.2 Bacterial spot

The disease is present in most areas where stone fruits are grown and may cause serious losses by reducing the marketability of the fruit and by weakening trees through leaf spots, defoliation and lesions on twigs. In India, the disease is reported on apricot, plum, peach and almond (Anonymous, 1961, Durgapal, 1974, Jindal and Sharma 1988b).

3.2.1 Symptoms

On the leaves circular to irregular, water soaked spots about 1 to 5 mm in diameter appear which later turn purple or brown. Usually the central area of the spot separates from the surrounding tissues and fall off giving a shot hole appearance. Severely affected leaves turn yellow and drop.

On the fruit, small circular, darkbrown and depressed spots develop which result in pitting and cracking. On the twigs, dark, slightly sunken lesions form usually around buds in the spring or on green shoots later in summer.

3.2.2 Causal organism

Xanthomonas campestris pv. *pruni* (Smith) Dye. The bacterium is a gram negative, motile rod measuring 0.2-0.4 X 0.8-1.0 µm.

3.2.3 Etiology

The bacteria overwinter in twig lesions and in the buds. In the spring they ooze out and are spread by rain splashes and insects to young leaves, fruits and twigs, which they infect through natural openings, leaf scars and wounds (Feliciano and Daines, 1970).

The occurrence and development of primary and subsequent secondary infections depend entirely on environmental conditions. Frequent periods of moisture during late bloom to few weeks after petal fall are very conducive to primary fruit and leaf infection.

3.2.4 Control

The disease is more severe on weakened trees than on vigorous ones, therefore, keeping trees in good vigour help them resist the disease. The disease on trees can be effectively controlled by trunk infusion of oxytetracycline about 0.8g per tree (Keil, 1979). Four sprays of streptocycline plus copper oxychloride gave more than 73 per cent reduction in leaf infection and 80 per cent in fruit gummosis incidence and resulted in a net increase of 40 per cent in green fruit yield in almond (Jindal *et al.*, 1989).

3.3 Bacterial canker

The disease is also known as gummosis, blossom blast, die-back, spur blight and twig blight, occurs in all major fruit growing areas of the world. The disease occurs in a serious form on all species of stone fruit trees throughout Himachal Pradesh, Kashmir Valley and Kumaon and Gharwal hills of Uttar Pradesh (Agarwala, 1961, Singh and Singh, 1956, Durgapal, 1974). Tree losses from 10-75 per cent have been observed in young orchards. The disease also kills buds and flowers of trees, resulting in yield losses of 10 to 20 per cent but sometimes upto 80 per cent. Leaves and fruits are also attacked, resulting in weaker plants and in low quality or not salable fruit.

3.3.1 Symptoms

The disease affects trunks, limbs, shoots, fruit spurs, blossom, dormant buds, leaves and fruits. Most destructive phase of the disease is that on the trunks and branches where gumming symptoms are evident beside canker. The girdled branches fail to develop or die. The blossom blight has been observed on plums and cherries.

On leaves, water soaked spot about 1 to 3 mm in diameter appear. Later the spots become brown, dry, brittle and fallout giving the leaves a shot hole or tattered appearance. Fruit invasion results in sunken and black lesions, 2 to 10 mm in diameter and depth. The underlying tissue become gummy or spongy.

3.3.2 Causal organism

The pathogens are *Pseudomonas syrinnas* pv. *syringae* Van. Hall and the more specialized *P. syringae* pv. *morsprunorum* (Wormald) Young *et al.*, which is restricted predominantly to cherry and plum. *P. syringae* pv. *syringae* produce the phytotoxins syringomycins, which appear to play a role in the virulence of the pathogen. *Pseudomonas amygadli* produced similar symptom (as mentioned above) on almond (Ecrolani and Ghaffar, 1985).

3.3.3 Etiology

The bacteria overwinter in active cankers, infected buds and systemically in apparently healthy tissues of the tree. Infection occurs in the autumn through leaf scars and in spring through scars left by bud scales. In spring, bacteria from cankers and infected buds are disseminated by rainfall onto emerging tissues. Infection on developing blossoms, leaves and fruit occurs through natural openings and wound. Leaves and blossoms are probably more susceptible when injured by frost. Secondary spread from primary infections occurs throughout the growing season and is important in the build up of the disease. Periods of frequent rainfall, cool temperatures and high winds are most favourable for early season infection and spread.

3.3.4 Control

No complete control of bacterial canker and gummosis of fruit trees could be obtained by any single method. Certain cultural practices and control measures help keep down the number and severity of infection. Only healthy bud wood should be used for propagation. Susceptible varieties should be propagated on root stocks resistant to the disease and should be grafted as high as possible. Orchards should not be located in areas where trees are subjected to freeze damage, water logged soils and prolonged drought.

Partial control of the canker phase of the disease both in nursery and in the orchard is obtained with sprays of fixed copper or Bordeaux mixture in the fall and in the spring before the blossoming. Cankers on trunks and large branches can be controlled by cauterization with a hand held propane burner (Hawkins, 1976). The treatment is carried out in early to mid spring and if necessary, should be repeated 2 to 3 weeks later. Mashobra paste formulated by Agarwala (1964) has been reported effective against the disease on peach, plum, cherry and apricot. Four sprays of streptocycline (200 ug/ml) + Blitox (0.2%) first immediately after the disease appearance and 3 subsequent sprays at 10 days interval gave 74.83% reduction in leaf infection and 80% in fruit infection in apricot (Jindal and Rana, 1992).

3.4 Fastidious vascular bacterial diseases

Phoney peach, almond leaf scorch and plum leaf scald are the important diseases caused by fastidious vascular inhabiting bacteria.

3.4.1 Phoney peach

Infected trees are dwarfed, have a deep green colour and present a compact appearance in comparison to healthy trees. Internodes are shortened and the foliage is flattened. Affected trees bloom earlier and retain foliage longer in the fall. Fruits are smaller in size, ripen early and are deep coloured. In addition to peach trees, apricots, nectarines, and many species of plums are natural hosts for phoney peach bacterium. The bacterium is gram negative and has been isolated on several artificial media (Mount

and Lacy, 1982). The bacterium is transmitted by sharpshooter leaf hoppers *Homalodisca insolita*, *H. Coagulata, Oncometopia orbona, O. nigricans, Graphocaphala versuta* and *Draeculacephala versuta*. Management strategies include rouging infected plants and replanting with disease tolerant cultivars. Almond is immune to phoney disease and hence use of almond root stocks may be useful in controlling the phoney peach disease. Removal of alternate host has also been recommended. Injection of antibiotics, particularly tetracycline hydrochloride into diseased tree trunks adequately reduce the disease.

3.4.2 Almond leaf scorch

The disease is caused by a xylem limited fastidious walled bacteria (Mircetich *et al.*, 1976). From India, Jindal and Sharma (1987) reported this disease for the first time. The bacterium is closely related to phoney peach bacterium and Pierce's disease of grapevine (Mount and Lacy, 1982). The margins and tips of leaves become necrotic, grey brown, bordered by yellow or reddish tissues. Necrotic areas from leaf margins progress inward resulting in scorchy appearance. Insect vectors and management practices of almond leaf scorch are similar to phoney peach disease.

3.4.3 Plum leaf scald

A gram negative bacterium with rippled cell wall is responsible for this disease (Raju *et al.*, 1982). It appears that the bacterium causing plum scald is the same as phoney peach (Mount and Lacy, 1982) but needs confirmation. Almond trees inoculated with this bacterium does not show symptoms. Bacterium is isolated from petiole, stem and root tissues of plum showing leaf scald symptoms Chang *et al.* (1987) reported suppression of leaf scald symptom in plum with trunk infection of oxytetracycline (OTC).

4. References

Adaskaveg, J.E., Ogawa, J.M. and Butler, E. E. 1990. Morphology and ontogeny of conidia in *Wilsonomyces carpophilus*, gen. nov. and comb. nov., causal pathogen of shot hole disease of *Prunus* species. Mycotaxon 37: 275-290.

Agarwala, R.K. 1961. Bacterial gummosis of stone fruits. Himachal Horticulture 2: 49-51.

Agarwala, R.K. 1964. Bacterial gummosis of stone fruits. Part II. State Regional Fruit Research Station, Mashobra. Agricultural Research, New Delhi 4: 139-140.

Agarwala, R.K. 1970. Relative importance of the control methods of *Phytophthora cactorum* collar rot disease of apple trees. In: "Plant Disease Problems". Proc. Ist Int. Symp. Pl. Path., Indian Phytopathological Society, I.A.R.I., New Delhi: 632-638.

Agarwala, R.K., Arora K.N. and Singh A. 1966. Effect of temperature and humidity variation on the development of peach leaf curl on mid hills and its control. Indian Phytopath. 19: 308-309.

Agarwala, R.K. and Gupta G.K. 1968. New records of pome and stone fruit tree diseases from Himachal Pradesh. Indian Phytopath. 21: 299-301.

Anderson, H.W. 1956. Diseases of fruit crops. New York McGraw Hill Book Company Inc. 501pp.

Andrade, E.R.D.E. and Matos C.S. 1996. Chemical control of *Monilinia fructicola* on post-

harvest peach fruits. Fitopatologia Brasileria 21: 301-303.

Angelov, A. 1980. The effectiveness of breeding in fruit growing in Bulgaria. Ovoshchavstvo 59: 12-16.

Anonymous 1961. Quarterly reports for January-March, April-June 1961 of the Plant Protection Committee for the South East Asia and Pacific Region-18pp, 16pp, FAO Publication, Bangkok, Thailand, pp. 18, 16.

Anonymous 1995. Package of Practices for fruit crops 1995, Dr. Y.S. Parmar University of Hort. and Fty. Nauni, Solan. 186pp.

Arthur, J.C. 1906. Cultures of uredineae in 1906. Jour. Mycol. 13: 189-205.

Bagghee, K.D. and Singh, V. 1960. Indian Forest Records, New Series 10: 199-348.

Balardin, R.S., Balardin, C.R.R. and Chaves, L.C. 1994. Fungicides and doses efficiency on *Monilinia fructicola* (Wint.) post-harvest control in peach (*Prunus persicae* var. *Vulgaris.* Ciencia Rural 24: 15-17.

Baruah, A., Baruah, S.N. and Bora, K.N. 1980. A fungal disease of peach plant collected from Gauhati University Campus, Assam (*Venturia carpophila*). Sci. & Cult. 46: 264.

Bhardwaj, L.N. and Shyam, K.R. 1986. Occurrence of rust on Japanese plum. Him. J. Agri. Res. 12: 62.

Bhardwaj, S.S. and Ved Ram. 1995. Incidence of apricot leaf curl and its management. In: "Integrated Disease Management and Plant Health". (eds. Gupta, V.K. and Sharma, R.C) Scientific 'Publishers, Jodhpur, pp227-279.

Biggs, A.R. 1989. Integrated approach to controlling *Leucostoma* canker of peach in Ontario. Plant Dis. 73: 869-874.

Biggs, A.R. and Northover, J. 1988. Influence of temperature and wetness duration on infection of peach and sweet cherry fruits by *Monilinia fructicola.* Phytopathology 78: 1352-1356.

Bolkan, H.A., Ogawa, J.M., Michailides, T.J. and Kable, P.F. 1985. Physiological specialization in *Tranzschelia discolor*. Plant Dis. 69: 485-486.

Borkai-Colon. 1959. *Tuberculina persicina* attacking rust fungus in Israel. Bull. Res. Conn. Israel. Sect. D **8:** 41-46.

Bose, S.K. and Sindhan, G.S. 1972. Peach leafcurl (*Taphrina deformans* (Berk.) Tul.) in Kumaon and its control. Prog. Hort. 4: 5-8.

Brunelli, A. and Ponti, I. 1993. Observations of epidemiology and control measures against leaf curl on peach. Bulletin OILB/SROP 16: 64-67.

Butler, E.J. and Bisby, G.R. 1931. The fungi of India. Imp. Council Agri. Res. (India) Sci. Monog.1

Byrde, R.J.W. and Jordan, V.W.L. 1977. In: "Systemic Fungicides" (ed. RW Marsh) Longman, London and New York. 280 pp.

Byrde, R.J.W. and Willetts 1977. The Brown Rot Fungi of Fruit: Their Biology and Control. Pergaman Press, New York 171 pp.

Carter, M.V. and Price, T.V. 1985. Biological control of *Eutypa armeniacae* III. A comparison of chemical, biological and integrated control. Aust J. Agric. Res. 26: 537-543.

Chang, C.J., Yonce, C.E. and Gardner, D. 1987. Suppression of leaf scald symptoms in plum by oxytetracycline injection. Ann. Phytopath. Soc. Japan **53:** 354-359.

Corazza, L., Simeone, A.M. and Ialongo, M.T. 1984. Relation of some apricot cultivars of rust (*Tranzschelia pruni-spinosae* Persoon) Distel var *discolor* (Fuckel Dunegan) at a location on the latium coast. Informatore Fitopatologico 34: 45-48.

Corke, A.T.K. 1978. Microbial antagonist affecting tree diseases. Ann. Appl. Biol. 89: 89-93

Coroianee, A. and Ivascu A. 1981. Investigations on the behaviour of some peach cultivars and hybrids to attack by *Taphrina deformans.* Lucr. Stiinti B24: 59-63.

Decker, R. and Buchanan, D.W. 1975. Effect of rust (*Tranzschelia discolor*) on the tree performance and fruit yield of 'Early Amber' peach and sungold nectarine. Proc. Fl. State Hort.

Soc. 86: 333-335.

DeCleene, M. and De Ley, J. 1976. The host-range of crown gall. Bot. Rev. 42: 389-466.

Dippenaar, B.R. 1941. Diseases of fruit trees caused by leaf rust, Manganese and Zinc deficien-
    cies and their control. S. Afr. J. Sci. 37: 136-155.

Dong, C.O., Kim, S.B., Jang H.I., Cho M.D. and Lee E.K. 1991. Studies on the ecological
    characteristics and control methods of peach powdery mildew. Journal of the Korean Soci-
    ety for Horticultural Science 32: 191-198.

Dubos, B. and Ricard, J.L. 1974. Curative treatment of peach trees against silver leaf disease
    (*Stereum purpureum*) with *Trichoderma viride* preparations. *Plant Dis. Rep.* **58:** 147-150.

Dunegan, J.C. 1938. The rust of stone fruit. Phytopathology 28: 411-426.

Durgapal, J.C. 1974. A preliminary note on bacterial diseases of temperate plants in India. II.
    Bacterial diseases of stone fruits. Indian Phytopath. 24: 379-382.

English, H. and Davis, J.R. 1978. *Eutypa armeniacae* in apricot. Pathogenesis and induction of
    xylem soft rot. Hilgardia 46: 193-204.

Ecrolani, G.L. and Ghaffar, A. 1985. Bacterial canker and gummosis of stone fruits. FAO Plant
    Prot. Bull. 33: 37-39.

Feliciano, A. and Daines, R.H. 1970. Factors influencing in grass of *Xanthomonas pruni* through
    peach leaf scars and subsequent development of spring cankers. Phytopathology 60: 1720-
    1726.

Ferraris, T. 1928. Agricultura ef fitopathologianel Kashmir. Curiamo. de Plante 6: 61-86.

Foschi, S., Roberti, R., Bremelli, A. and Flori, P. 1995. Application of antagonistic fungi against
    Monilinia laxa agent of fruit rot of peach. Bulletin OILB/SROP 18: 79-82.

Frisullo, S., Ciccarese, F., Ferrara, G. and Amenduni, M. 1993. Chemical control trials against
    cherry leaf spot. Petria 3: 141-146.

Goldworthy, M.C. and Smith, R.E. 1931. Studies on a rust of clingstone peaches in California.
    Phytopathology 21: 133-168.

Gupta, G.K., Agarwala, R.K. and Dutt, K. 1972/74. Stigmina blight of peach and its control in
    Himachal Pradesh. Hort. Adv. 9: 49-51.

Gupta, G.K., Agarwala, R.K. and Dutt, K. 1973a. Blight of peaches in H.P. can be controlled.
    Indian Hort. 17: 13.

Gupta, G.K., Agarwala, R.K. and Dutt, K. 1973b. Apricot leaf curl caused by *Taphrina defor-
    mans* in Kullu Valley, India. Plant Dis. Reptr. 57: 361-362.

Gupta, G.K. and Byrde, R.J.W. 1988. *Monilinia laxa* associated with blossom wilt of apricot
    and almond in Himachal Pradesh, India. Plant Pathology 37: 591-593.

Hawkins, J.E. 1976. A cauterisation method for the control of cankers caused by *Pseudomonas
    syringae* in stone fruit trees. Plant Dis. Rep. 60: 60-61.

Hawksworth, D.L., Sutton, B.C. and Ainswoth, G.C. 1983. Ainsworth and Bisby's Dictionary
    of the fungi: 7th ed. Comm. Mycol. Inst., Kew Surry

Huang, J.W., Yong, H.C. and Leu, L.S. 1993. Studies on control of Japanese apricot scab. Plant
    Pathology Bulletin 2: 7-11.

Huang, J.W., Chen, J.H., Chung, W.C. and Yang, S.H. 1995. Chemical control of powdery
    mildew on Japanese apricot. Journal of Agriculture and Forestry 44: 13-18.

Jafar, H. 1958. Studies on the biology of peach rust (*Tranzschelia pruni-spinosae* Pers.) in
    Newzealand. Investigations on the control of peach rust. N.Z.J. Agric. Res. 1: 642-651.

Jafar, H. 1959. Current Research and Investigations- Orchard. NZ 32: 17-19.

Jain, S.S. 1962. Tests of fungicides for control of peach leaf curl (*Taphrina deformans* Tul.)
    Himachal Horticulture 3: 11-15.

Jindal, K.K. and Sharma, R.C. 1987. Almond leaf scorch - a new disease from India. FAO Plant
    Prot. Bull. **35:** 64-65.

Jindal, K.K. and Sharma, R.C. 1988a. Crown gall and hairy root in fruit plant nurseries in

Himachal Pradesh. Proceedings of 5th International Congress of Plant Pathology, Kyota, Japan, August 20-29, 1988, p.88 (Abstr.)

Jindal, K.K. and Sharma, R.C. 1988b. Occurrence of bacterial leaf spot of peach incited by *Xanthomonas campestris* pv. *pruni*. Plant Dis. Res. 3: 60-61.

Jindal, K.K., Sharma, R.C. and Gupta, V.K. 1989. Chemical control of bacterial leaf spot and gummosis caused by *Xanthomonas campestris* pv. *pruni* in almond (*Prunus dulcis*). Indian J. Agric. Sci. 59: 754-755.

Jindal, K.K. and Rana, H.S. 1992. Studies on germplasm resistance and chemical control of bacterial canker of apricot. Plant Dis. Res. 7: 7-10.

Jones, A.L. and Aldwinkle, H.S. (eds.) 1990. Compendium of Apple and Pear Diseases. APS Press, St. Paul, Minnesota

Jones, A..L, Ehret ,G.R., Garcia, S.M., Kesner, C.D. and Klein, W.M. 1993. Control of cherry leaf spot and powdery mildew on sour cherry with alternate side applications of fenarimol, myclobutanil and tebuconazole. Plant Dis. 77: 703-706.

Jones, D.A. and Kerr, A. 1989. *Agrobacterium radiobacter* strain K 1026, a genetically engineered derivative of strain K-84, for biological control of crown gall. Plant Dis. 73: 15-18

Kadar, A.A., El-Goorani, M.A. and Sommer, N.F. 1982. Post-harvest decay, respiration, ethylene production and quality of peaches held in controlled atmosphere with added carbon monoxide. J. Amer. Soc. Hort. Sci. 107: 856-859.

Kaul, T.N. 1967. Diseases of stone-fruits in Kashmir. Horticulturist 2: 52-58.

Kaul, T.N. 1971. Rust of almond a new record. Proceedings of Second International Symposium on Plant Pathology, New Delhi, Indian Phytopathological Society, p.159.

Keil, H.I. 1979. Control of bacterial spot caused by *Xanthomonas pruni* in apricot trees by trunk infusion with oxytetracycline. Plant Dis. Rep. 63: 407-409.

Kerr, A. 1980. Biological control of crown gall through production of agrocin 84. Plant Dis. 64: 25-30.

Khan, M.W., Malik, K.A. and Khan, A.M. 1975. Perithecial stages of certain powdery mildews including some new records-III. Indian Phytopath. 28: 199-201.

Kibishauri, V.P. and Tsiklausi, M.S. 1979. The effect of chemical nutrition on the resistance of apricot to Verticillium wilt. Zaschity Rastenii Gruzinskoi SSR 30: 10-14.

Larsky, M. and Necesany, V. 1995. An aimed sour cherry protection against cherry leaf spot caused by the fungus (*Blumeriella jaapi* (Rehm.) V, Arx. Vedecke Pracs Ovonarske 14: 9-15.

Lawrence, E.G. Jr. and Zehr, E.I. 1982. Environmental effects on the development and dissemination of *Cladosporium carpophilum* on peach. Phytopathology 72: 773-776.

Madrigal, C., Pascual, S. and Melgarejo, P. 1994. Biological control of peach twig blight (*Monilinia laxa*) with *Epicoccum nigrum*. Plant Pathology 43: 554-561.

Michailides, T.J. and Ogawa, J.M. 1986. Chemical control of prune leaf rust (*Tranzschelia discolor* f.sp. *domestica*). Plant Dis. 70: 307-309.

Mircetich, S.M., Lowe, S.K., Moller, W.J. and Nyland. 1976. Etiology of almond leaf scorch disease transmission of the causal agent. Phytopathology 66: 17-24.

Moore, L.W. 1976. Latent infections and seasonal variability of crowngall development in seedlings of three *Prunus* species. Phytopathology 66: 1097-1101.

Mount, M.S. and Lacy G.H. 1982. Phytopathogenic Prokaryotes Vol. 1 & 2 541 & 506 P New York Academic Press.

Munjal, R.L. and Kulshreshta, D.D. 1968. Some Dematiaceae hyphomycetes from India. Indian. Phytopath. 21: 309-314.

Nadazdin, M. and Nadazdin, V. 1977. Some morphological and biological features of the pathogens of rust of peach and apricot in Hercegovina. Zastita Bilja 28: 327-333.

Nautiyal, M.C., Prakash, S. and Kumar, A. 1988. Screening of peach cultivars for their reaction

against leaf curl (*Taphrina deformans*). Indian J. Agric. Sci. 58: 575-576.

Nicolas, J. and Bornet, E. 1996. Armillaria root rot of peach trees and tolerant root stocks. Arbriculture Fruitiere No. 493: 30-32.

Northover, J. and Mcfadden Smith W. 1995. Control and epidemiology of *Apiosporina morbosa* of plum and sour cherry. Canadian J. Plant Pathol. 17: 57-68.

Osorio, J.M., Adaskaveg, J.E. and Ogawa, J.M. 1993. Comparative efficacy and systemic activity of iprodione and the systemic activity of iprodione and the experimental anilide E-0858 for control of brown rot on peach fruit. Plant Dis. 77: 1140-1143.

Patli, J. 1981. Cultural practices and infections crop diseases. Berlin Springer Verlog. 243pp.

Pandotra, V.N., Kachroo, J.L. and Sastry, K.S.M.. 1968. Six powdery mildew from Jammu and Kashmir State. Proc. Indian Acad. Sci. 67B: 119-124.

Pegg, G.F. 1974. *Verticillium* diseases. Rev. Plant Pathol. 53: 157-182.

Persoon, C.N. 1801. Synopsis Methodica Fungorum Goattingae Dieterich

Ponti, I., Brunell, A., Spada, G., Tosi, C., Garaffoni, M., Emiliani, G. and Cont, C. 1993. Activity of dodine against peach blister. Informatore Agrario 49: 75-79.

Pusey, P.I. and Wilson, C.I. 1984. Post-harvest biological control of stone fruit brown rot by *Bascillus subtilis*. Plant Dis. 68: 753-756.

Puttoo, B.L. and Razdan, V.K. 1991. Stigmina blight of cherry -a new record from India. Plant Dis. 6: 60.

Raju, B.C., Wells, J.M., Nyland, G., Brlansky, R.H. and Lowe, S.K. 1982. Plum leaf scald isolation culture and pathogenicity of the causal agent. Phytopathology 72: 1460-1466.

Ritchie, D.F. and Werner, D.J. 1981. Susceptibility and inheritance of susceptibility to peach leaf curl in peach and nectarine cultivars. Plant Dis. 65: 731-734.

Ram, V., Usha Sharma and Bhardwaj, L.N. 2000. Management of apricot leaf rust. Plant Dis. Res. 15: 113-115.

Rana, B.S. and Jain, V.B. 1992. Screening of peach varieties against leaf curl disease and its control. Indian J Mycol. Pl. Pathol. 22: 87 (Abstr.)

Rana, K.S. and Gupta, V.K. 1981. Cause and control of collar rot of apple in India. Indian Phytopath. 34: 17-19.

Rana, K.S. and Gupta, V.K. 1983. *In vitro* and *in vivo* efficacy of systemic and protectants fungicides against *Phytophthora cactorum*. Indian J Mycol. Pl. Pathol. 13: 272-276.

Rana, K.S. and Gupta, V.K. 1984. Effect of fungicides on the viability of *Phytophthora cactorum* propagules in the soil. Phytopath. Z. 110: 245-250.

Rosenberger, D.A. and Gerling, W.D. 1984. Effect of black knot incidence on yield of stackley prune trees and economic benefits of fungicides protection. Plant Dis. 68: 1060-1064.

Ruegg, J. and Siegfried, W. 1993. Integrated fruit production: news on blossom and fruit Monilia. Schweizerische zeitschrift fiir obst-und weibau 129: 620-624.

Shabi, E., Elisha, S., Birger, R. and Singer, G. 1990. Almond diseases and their control. Alow Hanolea 42: 445-450.

Sharma, A.K. 1985. A new species of *Phyllactinia* (Erysiphaceae) from India. Trans. British Mycological Society 85: 756-759.

Sharma, I.M. and Badiyala, S.D. 1994. Susceptibility of peaches to *Taphrina deformans* in relation to blooming, environmental factors and genetic inheritance. Indian Phytopath. 47: 65-71.

Sharma, R.C .and Paul, Y.S. 1986a. New diseases of peach (*Prunus persica* L.) from India. Indian J Plant Pathol. 4: 182.

Sharma, R.C.and Paul, Y.S. 1986b. Target leaf spot of peach. A new disease from India. Plant Dis. Res. 1: 90-91.

Sharma, R.C., Jindal, K.K. and Gupta, V.K. 1987. Managing fungal peach leaf curl through chemicals and host resistance. 11th Int.t Prot., Manila, October 1987. 51p

Sharma, R.C., Gupta, V.K. and Gag, R.C. 1988a. Dormant application of fungicides for the management of peach leaf curl. Indian Phytopath. 41: 259-260.

Sharma, R.C., Jindal, K.K. and Gupta, V.K. 1988b. Reaction of some almond cultivars to rust. Plant Dis. Res. 4: 80-81.

Sharma, R.L. 1994. Efficacy of post-harvest fungicidal treatments on brown rot of peach. Indian J Mycol. Pl. Pathol. 24: 60-61.

Sharma, R.L. and Kaul, J.L. 1988a. Occurrence of brown rot (*Monilinia* spp.) on stone fruits in Himachal Pradesh. Plant Dis. Res. 3: 46-47.

Sharma, R.L. and Kaul, J.L. 1988b. Efficacy of post harvest fungicidal treatment in controlling brown rot (*Monilinia* spp.) of apricot. Indian J Mycol. Pl. Pathol. 18: 92-93.

Sharma, R.L. and Kaul, J.L. 1989. Effect of post-harvest fungicides treatments on brown rot of apricot. Plant Dis. Res. 4: 54-58.

Simeone, A.M., Ialonga, M.T. and Corazza, L. 1984. The reaction of certain plum cultivars to rust (*Tranzschelia pruni spinosae*) in Latium Littoral region. Annual Dell's Institute sperimenta perla Fruitticoltura 15: 39-49.

Simeone, A.M., Ialonga, M.T. and Corazza, L. 1985. Reaction of some plum varieties of rust (*Tranzschelia pruni-spinosae*) Dietal var. *discolor*) in a locality of Latium Coast. Informatore Fitopatologico 35: 37-41.

Singh, G., Chauhan, J.S., Bhatt, A.S. and Malhi, C.S. 1976. Evaluation of germplasm collections of almond rust in Punjab. Indian J Mycol. and Pl. Pathol. 6: 31

Singh, L.B. and Singh, R.L. 1956. Peach special number. Agricultural Animal Husbandry, U.P. 6: 3-28.

Smilarick, J.L., Deris-Arrue, R., Bosch, J.R., Gonzalez, A.R., Herson, D. and Jarisiewiez, W.J. 1993. Control of post-harvest brown rot of nectarines and peaches by *Pseudomonas* species. Crop Protection 12: 513-520.

Sohi, H.S., Jain, S.S., Sharma, S.L. and Verma, B.R. 1964. New records of plant diseases from Himachal Pradesh. Indian Phytopath. 17: 42-45.

Sohi, H.S., Sharma, S.L., Verma, B.R. and Shyam, H.S. 1968. Chemical control of peach leaf curl. Indian. J Hort. 25: 48-51.

Sydow, H. and Butler, E.J. 1916. Fungi Indiae Orientalis Part IV. Annals of Mycology 14: 177-220.

Tate, K.G. and Wood, P.N. 1994. Field evaluation of fungicides for control of peach leaf curl (*Taphrina deformans*). In: "Proceedings of the Forty Seventh New Zealand Plant Protection Conference", Waitongi Hotel, New Zealand, 9-11 August, 1994. Rotorua, New Zealand, New Zealand Plant Protection Society: 289-293.

Taylor, P.A. and Washington, W.S. 1984. Curative treatments for *Phytophthora cactorum* in peach trees using metalaxyl and phosetyl-Al. Aust. J Plant Pathol. 13: 31-34.

Torre Almaraz, R.De La and Ceballos Silva, A.P. 1990. Chemical control of peach disease at Acajete, Pucbla. Revista Mexicana de Fitopathologia 8: 181-190.

Verma, K.D. and Gupta, G.K. 1979. Studies on leaf spot of cherry caused by *Cercospora circumscissa* sacc. and its control. Prog. Hortic. 10: 57-62.

Vitanov, M. 1976. On some questions of the pathogens of plum rust in Bulgaria and its control. Rastitelna Zshchita 24: 34-36.

Voromin, E.I., Kartausova, V.A. and Kopylova, N.I. 1982. Plum rust in the Crimea. Trudy Po Prikladnoi Botanike Genetskei Solktsii 71: 72-74.

Wain Wright, S.H. and Lewis, F.H. 1970. Developmental morphology of the black knot pathogen on plum. Phytopathology 60: 1238-1244.

Waraich, K.S. and Khatri, H.L. 1977. Occurrence of pink disease and rust on plum in India. Indian J Mycol. Pl. Pathol. 7: 202-206.

Weinhold, A.R. 1961. Temperature and moisture requirements for germination of conidia of

Sphaerotheca pannosa. Phytopathology 51: 699-703.

Wilhelm, S. and Ferguson, J. 1953. Soil fumigation against *Verticillium albo-atrum*. Phytopathology 43: 593-596.

Zambryski, P.C. 1992. Chronicles from the *Agrobacterium* - plant cell DNA transfer story. Annu. Rev. Plant. Physiol. Plant Mol. Biol. 43: 465-490.

Zhukov, O.S. and Kharitonova, E.N. 1982. Remote hybridization in breeding cherry and wild cherry for resistance to *Coccomyces* disease and winter hardiness. Byull Vses Nauchno-Issalad Inst. Rasteni Vavilova 123: 46-48.

Biologically-Based Alternatives to Synthetic Fungicides for the Control of Postharvest diseases of Fruit and Vegetables

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Abstract: Considerable amounts of fruits and vegetables are lost to spoilage after harvest. This loss can range from 10-50% depending on the commodity and country. Presently, synthetic fungicides are the primary means of controlling postharvest diseases of fruits and vegetables. Public concern over food safety, however, has enhanced interest to find effective alternatives to fungicides to control postharvest diseases of fruits and vegetables. Currently, several promising biological approaches that include microbial antagonists, naturally-occuring antifungal compounds, and induced resistance have been advanced as potential alternatives to synthetic fungicides for postharvest disease control. Among the proposed alternatives, the use of antagonistic microorganisms and chitosan as a pre-storage treatment of fruit and vegetables has been the most studied and substantial progress has been made in these areas. Unfortunately, the efficacy of some biological control antagonists evaluated under simulated and actual commercial conditions has been irregular unless they are combined with other treatments, such as low rates of fungicides. The fundamental basis, the potential, and the limitations of the different biological approaches and the advantages of a multifaceted strategy will be presented.

1. Introduction

Worldwide, postharvest losses of fruits and vegetables have been estimated at 25% and much of this is due to fungal and bacterial infections. In developing countries, postharvest losses are often more severe due to inadequate storage and shipping facilities. Any reasonable reduction in the postharvest losses would ease pressure on the production resources and generate much needed revenue in developing countries. The major cause of postharvest losses is often due to quality deterioration associated with high respiratory metabolism, biosynthesis and action of ethylene, transpiration, and decay. Although manipulating environmental storage conditions to delay senescence can reduce losses associated with accelerated metabolic turnover, these beneficial practices are usually not sufficient to completely protect harvested commodities from infection.

Decay of fruits and vegetables can be traced to infections that occur either between flowering and fruit maturity or during harvesting and subsequent handling and storage (Eckert and Eaks, 1989). In the former case, preharvest infections remain

quiescent until the physiological conditions of the fruit are favorable for the proliferation of the pathogen (Swinburne, 1983, Eckert and Eaks, 1989). On the other hand, postharvest infections that occur through surface wounds inflicted during harvest and subsequent handling are mainly caused by necrotrophic pathogens. Currently, synthetic fungicides are the primary means of controlling postharvest diseases of fruit and vegetables (Eckert and Ogawa, 1988).Growing health and environmental concerns over pesticide disposal and residue levels on fresh commodities, however, have generated a growing interest in the development of safer alternatives that are effective and pose no risk to human health or the environment. This has led to the de-registration of some of the more effective postharvest fungicides, and the ones considered to be safe are threatened by the development of fungicide-resistance (Spotts and Cervantes, 1986, Wilson and Wisniewski, 1994). Recently, several promising biological approaches that include antagonistic microorganisms, naturally-occuring biactive compounds, and induced resistance have been proposed as potential alternatives to synthetic fungicides for postharvest disease control (Wilson and Wisniewski, 1994, El Ghaouth *et al.*, 1997a, Janisiewicz, 1998). In this chapter the fundamental basis, the potential, and the limitations of various biological approaches for the control of postharvest diseases will be discussed with special reference to microbial antagonists and chitosan treatment.

2. Antagonistic microorganisms

As a result of the concerns over fungicides safety, real or perceived, a number of key fungicides for the control of postharvest diseases have been withdrawn from the market. Also, the trend to restrict or ban the use of current, synthetic fungicides is continuing. All this has intensified exploratory and developmental research for biocontrol agent as substitutes for synthetic fungicides for the control of postharvest decay. From these efforts, substantial progress has been made and a large body of information regarding postharvest biocontrol antagonists is now available (Wilson and Wisniewski, 1994). Presently, several antagonistic microorganisms have been identified and shown to have commercial potential against a number of postharvest pathogens on a variety of harvested commodities (Wilson and Wisniewski, 1994, Korsten *et al.*, 1994, El Ghaouth and Wilson, 1995, El Ghaouth *et al.* 1997a, Janisiewicz 1998). Antagonistic bacteria and yeasts have been shown to control decay of a variety of harvested commodities including citrus, pome, and stone fruits. Effective control of decay of citrus fruit was observed with yeasts such as *Pichia guilliermondii* Wickerham, *Candida oleophila* Montrocher**,** *C*andida *sake* Saito & Ota **,** *C*andida *famata* Meyer & Yarrow**,** *C*andida *saitoana* Nakase&Suzuki**,** *Debaryomyces hansenii* Lodder & Krejer-Van Rij**,** *Aureobasidium pullulans* (de Bary) Arnaud*,* and *Saccharomyces cerevisiae* Hansen (Droby *et al.,* 1989, Mcguire, 1994, Arras, 1996, El Ghaouth *et al.,* 1998a, Droby *et al.,* 1999a, Ippolito *et al.,* 2000). Control of decay of citrus fruit caused by *Penicillium digitatum* (Pers.:Fr.) Sacc., and *Penicillium italicum* Wehmer was also reported with bacterial antagonists such as *Bacillus subtilis* (Ehrenberg) Cohn, *Burkolderia* (*Pseudomonas*) *cepacia* Palleroni & Holmes, and *Pseudomonas syringae* Van Hall (Singh and Deverall, 1984, Smilanick and Dennis-Arrue, 1992, Huang *et al.*, 1995, Bull *et al.*, 1997). Fungal antagonists including *Myrothecium roridum* Tode.: Fries

(Appel *et al.*, 1988), and *Trichoderma viride* Persoon.:Fries., (Borras and Aguilar, 1990) were also shown to reduced decay of citrus fruit.

The biocontrol potential of microbial antagonists was also reported on pome and stone fruits. On apples and pears, control of decay caused by *Botrytis cinerea* Pers.: Fr. and *Penicillium expansum* Link was reported with bacterial antagonists *P. cepacia* and *P. syringae* (Janisiewicz *et al.*, 1991). Decay of apple was also controlled by antagonistic yeasts *C. sake* (Teixido *et al.*, 1999), *C. oleophila* (Wisniewski *et al.*, 1995), *C. saitoana* (El Ghaouth *et al.*, 1998a). Chand-Goyal and Spotts (1997) showed that *Cryptococcus infirmo-miniatus* (Okanuki) Phaff & Fell and *Cryptococcus laurentii* (Kufferath) Skinner were effective in controlling decay of apple and pear caused by *B. cinerea* and *P. expansum*. On stone fruit , antagonistic bacteria (Pusey and Wilson, 1984), yeast, and fungi (Hong *et al.*, 1998) were shown to reduce brown rot of caused by *Monilinia fructicola* (Winter) Honey. The biocontrol potential of microbial antagonists has also been demonstrated on kiwi fruit, potato, strawberry fruit, avocado, table, and cherry tomato (Burkhead *et al.*, 1995, Korsten *et al.*, 1995, Cook *et al.*, 1999, Schena *et al.*, 1999, Guinebretiere *et al.*, 2000, Yakoby *et al.*, 2001). On avocado, *B. subtilis* and an isolate of *Colletotrichum gloeosporioides* Penzig generated by insertional mutagenesis were effective against decay caused *C. gloeosporioides* (Prusky *et al.*, 1994, Korsten *et al.*, 1995). Scheena *et al.* (1999) showed that isolates of *A. pullulans* were effective against *B. cinerea*, *Rhizopus stolonifer* (Ehrenberg.:Fries) Lind, and *Aspergillus niger* van Tieghem on table grape and *B. cinerea* and *R. stolonifer* on cherry tomato.

The success of some of these microbial antagonists in laboratory studies has generated interest by several agrochemical companies in the development and commercialization of microbial antagonists for control of rots of fruits and vegetables. Several microbial antagonists have been patented and evaluated for commercial use as a postharvest treatment. Presently, four antagonistic microorganisms, two yeasts; *C. oleophila* and *Cryptococcus albidus* (Saito) Skinner*,* and two strains of the bacterium *P. syringae* are commercially available under the trade names ASPIRE, YieldPlus, and BIOSAVE-110, respectively. A few others are at different stages of commercial development and expected to reach the marketplace within two to three years.

2.1 Mode of Action

While there is a large body of information regarding the development of postharvest biocontrol antagonists, the mechanisms by which microbial antagonists exert their protection has not been fully elucidated. The biological activity of microbial antagonists has been attributed either to antibiotic production in the case of bacterial antagonist or in case of antagonistic yeast to a complex mode-of-action involving nutrient competition, direct parasitism, and induced resistance. In most of these studies, however, the putative modes of microbial antagonists has been inferred but not clearly demonstrated *in planta*. In order to fully exploit the biocontrol potential of microbial antagonists, a more fundamental understanding of their mode of action is essential. As we learn more about the mechanism by which antagonistic yeasts protect fruits from decay more effective methods of formulating, applying, and selecting antagonists will

ultimately emerge.

2.1.1 Antibiosis

Antibiosis is a commonly assumed mechanism for the biocontrol activity of bacterial species on leaf surfaces, the rhizosphere, and in fruit wounds. For instance the antagonist, *B. subtilis* (strain B-3), which controls brown rot of peaches, produces an antibiotic, Iturin, which inhibits spore germination of the brown rot fungus *M. fructicola* (Pusey, 1989). Topical treatment of fruit wounds with *Iturin* alone reduced decay caused by *M. fructicola* (Pusey, 1989), thus it is thought to play an important role in the mode-of-action of *B. subtilis*. Similarly, reduction of decay was also reported in apples, pears, and citrus treated with *B. cepacia* or its antibiotic, pryrrolnitrin (Smilanick and Dennis-Arrue, 1992, Janisiewicz *et al.*, 1991). The bacterial antagonist, *P. syringae*, which controls green molds of citrus fruit and gray molds of pome fruit, also produces an antibiotic, syringomycin, which is inhibitory to *P. digitatum in vitro* and in fruit wounds (Bull *et al.*, 1998). Although antibiotic-producing microorganisms have a potential to be used as biocontrol agents of postharvest diseases, more effort is being deployed toward the development of non-antibiotic-producing, antagonistic yeasts for the biological control of postharvest diseases (Wisniewski and Wilson, 1992).

2.1.2 Attachment

Direct parasitism by the antagonist of the pathogen's propagules has been reported to play a major role in biological control against soil-borne and foliar diseases (Elad, 1995). In the postharvest arena, very little information is available on biological control agents that directly parasitize pathogens. Wisniewski *et al.* (1991), however, have shown that the yeast cells of *P. guilliermondii* and *D. hansenii* when co-cultured with *B. cinerea* attached strongly to hyphae of *B. cinerea*. When yeast cells were dislodged from the hyphae, the hyphal surface appeared concave and partial degradation of the cell wall of *B. cinerea* was observed at the attachment sites (Wisniewski *et al.*, 1991). The partial degradation of *B. cinerea* cell walls by *P. guilliermondii* was attributed to its tenacious attachment to hyphal walls in conjunction with its production of ß-1,3-glucanase (Wisniewski *et al.*, 1991). Attachment to fungal walls was reported with *C. saitoana (*El Ghaouth *et al.*, 1998a*)*. The antagonistic yeast *C. saitoana* when co-cultured with *B. cinerea* attach strongly to hyphae of *B. cinerea* and cause swelling and, in extreme cases, the complete disruption of hyphal wall structure (El Ghaouth *et al.*, 1998a). Attachment of antagonistic yeasts to fungal cell walls was observed *in planta*. In apple wounds, *C. saitoana* prevented the proliferation of *B. cinerea* and attached to the hyphae of *B. cinerea* (El Ghaouth *et al.*, 1998a). In apple wounds, *Botrytis* hyphae in contact with yeast cells displayed severe cellular alterations that included cell wall swelling, extensive vacuolation, and cytoplasmic degeneration.

Yeast attachment to fungal cell walls appears to be blocked when the yeast cells or the pathogen hyphae were exposed to compounds that affect protein integrity or respiratory metabolism, thus indicating a lectin-type of recognition system (Wisniewski *et al.*, 1991). Investigations are underway to isolate the compounds in-

volved in attachment in antagonistic yeasts. Recently, a gene ALA1 from *Candida albicans* (Robin) Berkhout that encode for cell surface molecules, adhesins, have been isolated and shown to promote both attachment and aggregation in *S. cerevisiae* transformants (Gaur *et al.*, 1999). The adherence of *S. cerevisiae* overexpressing the ALA1 gene was unaffected by shear force and the presence of several competing molecules including salts, sugar, and detergent. The adherence, however, was inhibited by agents that disrupt hydrogen bonds (Gaur *et al.*, 1999).

As to the role played by the attachment capability of antagonistic yeasts in the observed control of lesion development, it has been suggested that attachment may affect the ability of the pathogen to initiate infection. Analysis *in planta* of the effect of treatments that negate attachment may provide further insight regarding the extent of the role played by attachment in the biocontrol activity of antagonistic yeasts. The role played by attachment will be determined once the yeast adhesive compounds that mediate attachment to fruit tissue and fungal walls has been isolated or characterized and the molecular bases of antagonistic yeast-substratum binding have not been elucidated.

2.1.3 Competition for nutrients and space

Competition for nutrients and space is probably one of the most extensively studied modes of action of biocontrol microorganisms. Competition for nutrients is believed to play a major role in the mode of action of *P. guilliermondii* against *P. digitatum* (Droby *et al.*, 1989), *Enterobacter cloacae* (Jordan) Hormaeche & Edwards against *R. stolonifer* (Wisniewski *et al.*, 1989), *C. laurentii* against *B. cinerea* (Roberts 1990), and *Sporobolomyces roseus* Kluyver & van Neil against *B. cinerea* and *P. expansum* (Janisiewicz, 1994). *In vitro* studies revealed that antagonistic yeasts and bacteria take up nutrients more rapidly than the target pathogens and thereby prevent spore germination and growth of the pathogens (Wisniewski *et al.*, 1989, Droby *et al.*, 1989, Droby and Chalutz, 1994, Droby *et al.*, 1998). Filonow *et al.* (1996) showed that the antagonist yeast *S. roseus* was more effective in removing ^{14}C glucose than the conidia of *B. cinerea. P. guilliermondii* when co-cultured with *P. expansum* in a minimal synthetic medium or in wound leachate solutions inhibited the spore germination and growth of *P. expansum* (Droby *et al.*, 1989).

Involvement of nutrient competition in the mode action of antagonistic bacteria and yeasts has also been indirectly demonstrated in fruit wounds. Droby *et al.* (1989) showed that the biocontrol activity of *P. guilliermondii* was highly dependent on the ability of the antagonist to prevent the establishment of a nutritional relationship between the pathogen and the host. The biocontrol activity of *P. guilliermondii* (Droby *et al.*, 1989), *E. cloacae* (Wisniewski *et al.*, 1989), *C. laurentii* (Roberts, 1990), and *S. roseus* (Janisiewski *et al.,* 1994) was partially or completely reversed by the addition of exogenous nutrients into the wound site. The level of control provided by antagonistic microorganisms is also highly dependent on the initial concentration of the antagonist applied in the wound and its ability to rapidly colonize the wound site (Janisiewicz and Roitman, 1988, Wisniewski *et al.*, 1989, Droby *et al.*, 1989, Mclaughlin *et al.*, 1990).

In most reports on biological control of postharvest diseases of fruits and vegetables, a quantitative relationship has been demonstrated between the concentration of the antagonist applied in the wound and the efficacy of the biocontrol agent (Wisniewski *et al.*, 1989, Droby *et al.*, 1989, Mclaughlin *et al.*, 1990, Janisiewski, 1998). Thus, a delicate balance apparently exists at the wound site between the number of antagonist cells and the pathogen propagules which affects the outcome of the interaction and determines whether or not the wound becomes a site of infection. Manipulation of the initial concentration of the antagonist cells and/or the fungal spores clearly affects infection. In general, with most harvested commodities, the biocontrol activity increased with increasing concentrations of antagonists and decreasing concentrations of the pathogen. Microbial antagonists are most effective in controlling postharvest decay when applied at 10^7 to 10^8 CFU/ml (Mclaughlin *et al.*, 1990, El Ghaouth *et al.*, 1998a, Janisiewski, 1998). This quantitative relationship, however, is highly dependent on the ability of the antagonists to multiply and grow at the wound site. This was demonstrated by using a mutant of *P. guilliermondii*, which lost its biocontrol activity against *P. digitatum* on grapefruit and against *B. cinerea* on apples, even when applied to the wound at concentrations as high as 10^{10} cells/ml (Droby *et al.*, 1991). The cell population of this mutant remained constant at the wound sites during the incubation period, while that of the wild type increased 10 to 20-fold, within 24 hours.

Currently, there is only fragmented data regarding the antagonist-pathogen interaction in terms of competitions for limiting nutrient essential for pathogenesis. Once more information regarding the specificity of competition between antagonistic and pathogens in fruit wounds is available and genes responses of antagonism of biocontrol agents have been characterized, it will be possible to develop antagonistic strains with a higher rate of transport and/or metabolism of limiting nutrient essential for pathogenesis. This may allow us to circonvent some the limitations of microbial antagonists.

2.1.4 Induced Resistance

Additional mode of action, such as induced resistance, has also been suggested and several microbial antagonists were shown to induce a wide range of defense responses in harvested commodities (Droby and Chalutz ,1994, Rodov *et al.*, 1994, Arras, 1996, El Ghaouth *et al.*, 1998a, Ippolito *et al.*, 2000). Treatment of apple wounds with *C. saitoana* induced chitinase activity and the formation of structural barriers such a papillae along host cell walls (El Ghaouth *et al.*, 1998a). Similarly in apple wounds, *A. pullulans* caused a transient increase in ?-1,3-glucanase, chitinase, and peroxidase activities (Ippolito *et al.*, 2000). Induction of disease resistance responses were also reported in pineapple, avocado, and citrus fruits (Tong-Kwee and Rohrbrook, 1980, Prusky *et al.*, 1994, Rodov *et al.*, 1994, Arras, 1996). Antagonists were shown to increase the levels of preformed antifungal diene in avocado fruits and the accumulation of the phytoalexin in citrus fruit (Prusky *et al.*, 1994, Rodov *et al.*, 1994, Arras, 1996, Yakoby *et al.*, 2001). Arras (1996) showed that scoparone accumulation could be 19 times higher when the antagonist *C. famata* was inoculated 24 h prior to *P.*

digitatum, and only four times higher if inoculated 24 h after the pathogen.

Although a causal connection between the accumulation of the host defense responses and bioprotection by antagonistic yeasts has not yet been clearly established, the occurrence of high levels of host antifungal compounds in protected tissue suggest their implication in disease resistance. Detailed studies regarding the implication of induced defense responses in the bioprotection by microbial antagonists are needed. Also, the identity and structure of the signal molecules generated by antagonistic yeast warrant further investigation. In fruit wounds, antagonistic yeasts often produce a large amount of extracellular mucilage along host cell walls (Wisniewski *et al.*, 1991, El Ghaouth *et al.*, 1998a). This extracellular mycilage is believed to be implicated in cell adhesion and may well contain active chemical elicitor that provides signals for recognition and subsequent responses. Oligosaccharide fragments of yeast cell wall polysaccharides are known to be active elicitors of host defense responses (Base *et al.*, 1992).

3. Natural antifungal compounds

Several naturally-occurring compounds are known to reduce the incidence of decay in a variety of fruits and vegetables. Reduction of postharvest decay was observed with plant extracts (Ark and Thompson, 1959, Sholberg and Shimizu, 1991), essential oils, and volatile substances (Wilson *et al.*, 1986, Mattheis and Roberts 1993, Vaugh *et al.*, 1993, Wilson *et al.*, 1997) and natural compounds derived from microbial fermentation (Gueldner *et al.*, 1988, Janisiewicz *et al.*, 1991). Wilson *et al.* (1986) showed that a number of volatile compounds produced by ripening peaches were fungicidal. Three of these compounds (benzaldehyde, methyl salicylate, ethyl benzoate) completely inhibited growth of two major postharvest pathogens, *M. fructicola* and *B. cinerea*, and reduced the incidence of decay of peaches and apples. Control of postharvest decay by volatile compounds also was reported by Vaugh *et al.* (1993). Exposure of strawberry and raspberry to either benzaldehyde, 1-hexanol, E-2-hexenal, Z-3-hexen-1-ol, or 2-nonanone completely suppressed decay caused by *B. cinerea* (Vaugh *et al.*, 1993). Similar reduction of decay was also reported for acetaldehyde vapor treated berries, apple, and citrus fruit (Stadelbacher and Prasad, 1974, Pesis and Avissar, 1990, Mattheis and Roberts, 1993).

Reduction of postharvest decay of fruits was also observed with ethanol, carbonate salts, organic acid, and hydrogen peroxide (Eckert and Eaks, 1989, Al Zaemey *et al.*, 1993, Smilanick *et al.*, 1995). Smilanick *et al.* (1995) showed that a brief immersion of citrus fruit in a solution containing ethanol (10 % wt/vol) reduced green mold incidence without significant injury to the fruit. Similar control of green mold on lemon was also obtained with a brief immersion in a heated sodium carbonate (3 % wt/vol) solution (Smilanick *et al.*, 1999a). Immersion of lemon fruits in hydrogen peroxide solutions (5 to 15 % wt/vol) was shown to significantly reduce green mold incidence, however, the risk of injury to the fruit was high (Smilanick *et al.*, 1999b). Ozone, applied either in air or water, does not stop infections development but oxidizes ethylene and thereby delays senescence that exacerbates decay problems (Smilanick *et al.*, 1999b).

Control of postharvest decay has also been reported with the sugar analog 2-

deoxy-D-glucose (Janisiewicz, 1994, El Ghaouth *et al.*, 1997b). 2-deoxy-D-glucose has been shown to interfere with the growth of several postharvest pathogens and provides partial control of apple and peach fruits decay (Janisiewicz, 1994, El Ghaouth *et al.*, 1997b). When tested *in vitro*, 2-deoxy-D-glucose inhibited the radial growth of several postharvest pathogens and caused morphological alterations in *R. stolonifer* and *B. cinerea* (El Ghaouth *et al.*, 1997b). Hyphae of *B. cinerea*, *P. expansum*, and R. *stolonifer* grown in the presence of 2-deoxy-D-glucose exhibited severe cellular injuries ranging from cell-wall disruption to cytoplasm disintegration. Cytochemical study of fungal cell wall components showed that 2-deoxy-D-glucose caused a marked increase in chitin and ß-1,3-glucan labeling in *R. stolonifer* and *B. cinerea*, thus indicating an interference of 2-deoxy-D-glucose with fungal wall biosynthesis.

The inhibitory effect of 2-deoxy-D-glucose is believed to be due to its ability to interfere with the metabolic processes implicated in cell wall biosynthesis (Moore, 1981) and can be reversed by the addition of metabolizable sugars. Reduction of postharvest decay of fruits and vegetables was also reported with chitosan coating and the potential of chitosan as an antifungal preservative has been extensively studied on a variety of commodities.

3.1 Chitosan: A potential preservative for postharvest commodities

Chitosan is a ß -(1 ,4)-D-glucosamine polymer (M.W. range 1.2 X 10^4 - 1.7 X 10^6 D) that is found as a natural constituent in the cell walls of many fungi (Araki and Ito, 1975). It is produced from a chitin (ß -(1,4)-N-acetylglucosamine polymers) component of both fungal cell walls and arthropod exoskeleton that has been deacetylated to provide sufficient free amino groups to render the polymer readily soluble in dilute organic acids (Filar *et al.*, 1978). The industrial and laboratory chitosan obtained from shellfish chitin by deacetylation carries about 70% free amino groups while the remaining 30% of the amino groups are acetylated, with a resultant pK value between 6 and 7 (Filar *et al.*, 1978). Chitosan is known to interfere with growth of a wide range of fungi (Allan and Hadwiger, 1979, Stossel and Leuba, 1984, Hirano *et al.*, 1984), to induce a multitude of biological processes in host tissue(Walker-Simmons *et al.*, 1984, Mauch *et al.*, 1984, Kendra *et al.*, 1989) and to form a semi-permeable film.

In recent years, there has been an increasing interest in the use of chitosan as a speciality chemical in several fields including agriculture, and several potential biological functions have been identified (Hirano *et al.*, 1984, Hadwiger *et al.*, 1984, Benhamou, 1992). Attempts to exploit the filmogenic and biological properties of chitosan were recently made with several postharvest commodities. Chitosan when applied as a coating was found to delay ripening by acting as a barrier to gas diffusion, reduce the incidence of decay, and stimulate several defense responses in plant tissue (El Ghaouth *et al.*, 1992a-e). Coating tomato, cucumber, bell pepper, and strawberry fruits with chitosan reduced their respiration rate, weight loss, improved their appearance and extended the shelf-life (El Ghaouth *et al.*, 1991a, 1991b, and 1992b). Coated fruits were firmer and higher in titrable acidity than control fruits indicating an overall delay of ripening. In tomato fruit, delay of ripening by a chitosan coating was associated with its ability to modify the internal atmosphere (El Ghaouth *et al.*, 1992b). The

coating provided a greater barrier for the efflux of CO_2 than for the influx of O_2. Delay of ripening by chitosan coating was also reported on apple fruits and attributed the defferential permeability of N,O-carboxymethylchitosan film (Elson *et al.*, 1985)). Elson *et al.* (1985) reported that the permeability of N,O-carboxymethylchitosan film was highly sensitive to the relative humidity of the air; at relative humidity below 70%, the films were somewhat impermeable to O_2 and CO_2, but at RH above 90% the permeability of the film to both gases increased dramatically with a higher perrneation rate for O_2 than for CO_2. While instances of interference with normal fruit ripening have been reported with other edible coatings (Elson *et al.*, 1985, Meheriuk and Lau, 1988), chitosan coatings neither altered the ripening capacity of the fruit nor caused any apparent phytotoxicity.

In addition, to mimicking the beneficial effect of modified atmosphere, chitosan coatings offer the added advantage of reducing postharvest decay. When applied as a coating, chitosan was effective in reducing decay of tomato, Bell pepper, cucumber, and strawberry caused by *B. cinerea* and *R. stolonifer* (El Ghaouth *et al.*, 1991a, 1991b, 1992a, 1992b) . In strawberry fruit stored at 13°C, a chitosan treatment was as effective as Rovral in controlling decay caused by *B. cinerea* (El Ghaouth *et al.*, 1991b). Reduction of decay by chitosan was also observed in tomato and bell pepper fruits stored at 20°C (El Ghaouth *et al.*, 1991a, 1992b). Chitosan, when applied as a stem scar treatment of bell pepper, markedly reduced lesion development caused by *B. cinerea* (El Ghaouth *et al.*, 1994). By the end of a 14-day storage period, less than 20 % of the chitosan treated fruits developed small lesions, whereas all the inoculated control had reached an advanced stage of decay.

Control of decay by chitosan coatings was also observed with apple, lemon, and oranges (El Ghaouth *et al.,* 1998b). In laboratory tests, chitosan treatment controlled postharvest decay caused by *B. cinerea* and *P. expansum on apple*, and *P. digitatum on citrus fruit*. In a series of semi-commercial tests, chitosan was very effective in controlling natural decay of major apple and citrus varieties (El Ghaouth *et al.*, 1998b).

The data accumulated so far bear witness to the film-forming ability of chitosan and to its biological activities. Chitosan, a biodegradable food fiber, offers great potential as an antifungal preservative for fresh fruit and vegetables. Detailed studies on the permeability of chitosan films under high humidity to various gases and volatiles are required in order to establish its ultimate potential applications as a coating for harvested commodities. There are some indications in the literature of its unusual differential permeability to gases as opposed to typical plastic films *i.e.*, chitosan films are more permeable to oxygen than to carbon dioxide. Above all, the antifungal and eliciting activities of chitosan present exciting possibilities for crop protection. Its dual activity gives chitosan great potential as an antifungal preservative for fresh horticultural commodities. The physico-chemical and biological functionality of chitosan which makes possible its diverse applications originate from its unique structure. Chitosan polymeric nature confers to it a film-forming property. Its polycationic nature provides the basis for its ability to interact with negatively charged biomolecules which are responsible for its biochemical properties.

3.1.1 Antifungal activity of chitosan

The antifungal property of chitosan is well established and has been demonstrated with several pathogenic fungi. Allan and Hadwiger (1979) showed that chitosan at 1000 μg/ml was more effective in inhibiting the growth of fungi than chitin. Among the 46 fungi tested, 36 of them were inhibited by chitosan. Only fungi containing chitosan as a major cell-wall component (*i.e.* Zygomycetes) were insensitive to chitosan (AIlan and Hadwiger, 1979). Hadwiger *et al.* (1984) suggested that the observed insensitivity of Zygomycetes to chitosan could be due to their ability to rapidly degrade chitosan or to an exclusion process.

    The inhibitory effect of chitosan was also demonstrated with soilbome phytopathogenic fungi such as *Pythium paraoecandrym* Drechsler, and *Aphanomyces euteidtes* Drechsler (Stossel and Leuba, 1984). At a concentration of 10 mg/ml, chitosan markedly reduced the radial growth of all tested fungi except *A. euteidtes* (Stossel and Leuba, 1984). Chitosan was also shown to not only inhibited the radial growth of major postharvest pathogens, but also induce severe morphological alterations in *R. stolonifer* and *B. cinerea,* as well as increased cellular leakage in both fungi (El Ghaouth *et al.*, 1992c, 1992d). The antifungal activity of chitosan was also observed *in planta*. In bell pepper tissue, chitosan treatment adversely affected the potential of *B. cinerea* to initiate infection and caused severe cellular alteration to invading hyphae (El Ghaouth *et al.*, 1994, 1997c). Most of the invading hyphae displayed severe cellular disorganization that ranged from vacuolation to complete protoplasm disintegration. The preservation of pectic and cellulosic binding sites in the host wall, in contact with affected hyphae, strongly suggests that chitosan might have affected the capability of *B. cinerea* to produce macerating enzymes. This was further confirmed by data showing that chitosan affected the ability of the pathogen to produce pectinolytic enzymes (El Ghaouth *et al.*, 1997c).

    While the mechanism underlying the antifungal action of chitosan is not well understood, it seems to consist of more than one mode. Chitosan was found to induce leakage of proteinaceous and other UV-absorbing materials in fungi (Leuba and Stossel, 1986, El Ghaouth *et al.*, 1992c), presumably by interacting with negatively charged phospholipid head-groups in the cell membrane. Such interaction may disturb the intermolecular bonding responsible for maintaining the membrane integrity. In addition to disturbing the membrane function by cross-linking with the phospholipid or protein components of the cell membrane, chitosan also causes severe cellular alteration in several fungi (El Ghaouth *et al.*, 1992c, 1994). Chitosan treatment induced severe morphological alterations characterized by excessive branching and wall alteration in *R. stolonifer* presumably via its action on a cell wall forming enzymes, namely chitin deacetylase (El Ghaouth *et al.*, 1992c and d). Exposure of *Rhizopus* cells to chitosan induced the activity of chitin deacetylase. *Rhizopus* cells walls appeared swollen and showed signs of deterioration (El Ghaouth *et al.*, 1992c). Similar ultrastructural and cellular alterations were also exhibited by *B. cinerea* (El Ghaouth *et al.*, 1994) and *Fusarium oxysporum* f.sp. *lycopersici* (Saccardo) Snyder & Hansen (Benhamou, 1992) grown in the presence of chitosan.

3.1.2 Eliciting activity of chitosan

Besides interfering directly with fungal growth, chitosan can induce several defense responses in plant tissue. Chitosan stimulated the formation of phenylanine ammonia Iyase, a key enzyme in the phenylpropanoid pathway (Hadwiger *et al*., 1994); induced fungal cell wall degrading enzymes such as chitinase and, ß-1,3-glucanase (Mauch *et al.*, 1988); and elicited the accumulation of the antifungal phytoalexin (pisatin) in pea pods (Kendra and Hadwiger, 1984, Hadwiger *et al*., 1994). The activities of chitinase and ?-1,3-glucanase can be co-ordinately induced in plant tissue not only by ethylene (Boller, 1988) but also by elicitors such as chitosan (Mauch *et al.*, 1988). These hydrolytic enzymes are implicated in host defense against pathogenic fungi, presumably by hydrolyzing the main components of fungal cell walls, chitin and ? ß -1,3-glucan (Schlumbaum *et al.*, 1986; Mauch *et al.*, 1988).

Chitosan treatment was also shown to stimulate defense enzymes and the formation of physical barriers in fruit tissue. In strawberry fruit, the ability of chitosan to stimulate defense enzymes such as chitinase seems to be expressed more in cut fruit than in intact fruit (El Ghaouth *et al*., 1992a). In bell pepper and tomato fruit, the activity of chitinase, chitosanase, and ß-1,3-glucanase appeared to increase upon chitosan treatment (El Ghaouth *et al*., 1994, 1997c). In chitosan-treated tissues, ß-1,3-glucanase and chitinase activities remained elevated for up to 14 days after treatment. Also, various structural barriers including formation of hemispherical protuberances along host cell walls and occlusion of many intercellular spaces with a fibrillar material were observed in chitosan-treated bell pepper tissues (El Ghaouth *et al*., 1994). While it is not possible to determine exactly the role played by these inducible defensive reactions in the control of *Botrytis* rot, they are more likely to have played a supporting role. This is indirectly supported by: (1) the fact that invading hyphae were mainly restricted to the epidermal cells ruptured during wounding and (2) the observation that fungal chitin was substantially reduced over fungal walls in contact with chitosan-treated tissue

3.1.3 Structural origin of the biological activity of chitosan

The antifungal activity of chitosan appears to increase with concentration and the degree of deacetylation of chitosan (Stossel and Leuba, 1984, El Ghaouth *et al*., 1992c). Leuba and Stossel (1984) showed that the inhibitory effect of chitosan increases with increasing deacetylation of the polymer. However, the relationship between the degree of deacetylation of chitosan, and its inhibitory activity was detectable only at intermediate chitosan concentration but not at either low or high concentrations. The biological activity of chitosan was also shown to be related to its polycationic nature, as well as, to its polymeric size. Stossel and Leuba (1984) reported that chitosan was more effective at pH 6 (the pKa value of chitosan is 6.2) than at pH 7.5, when most amino groups are in the free base form. Thus, suggesting that the antifungal activity of chitosan is due in part to its polycationic nature. The relationship between the cationic nature of chitosan and its antifungal activity was further confirmed by the results obtained by Leuba and Stossel (1984). They showed that synthetic polycations such as

polylysine and polyethylenimine were somewhat more effective in inhibiting the growth of test fungi than chitosan, while the polyanion, poly-L-glutamic acid exhibited little inhibitory activity. In addition, they noted that chitosan at pH 5.8-increased membrane permeability of *P. paraoecandrym*, while a treatment of the suspension culture of *Pythium* with either chitin or chitosan at pH 7.5 did not cause any significant leakage. Similar results were also observed with *B. cinerea* and *R. stolonifer* when exposed to chitosan (El Ghaouth *et al.*, 1992c).

In addition to the polycationic nature of chitosan, its polymeric size plays an important role in the biological activity of chitosan (Pearce and Ride, 1982, Kendra *et al.*, 1989, Hadwiger *et al.*, 1994). Kendra and Hadwiger (1984) showed that the anti-fungal and pisatin inducing activity of chitosan increased with increasing polymeric size. Maximal antifungal and pisatin inducing activity was noted for chitosan oligo-mer of seven monomer units or more, while the pentamer was less active and the smaller oligomers were totally inactive (Kendra and Hadwiger, 1984). The role of chitosan heptamer in the protection of host tissue against infection, presumably by inducing the host responses, was demonstrated by Kendra *et al.* (1989). They detected a rapid release and accumulation of chitosan oligomers (heptamer or more) from *Fusarium solani* t. sp. *phaseoli* (Burkhead) Snyder & Hansen during the pea-*Fusarium solani* interactions. Such accumulation of biologically active chitosan oligomers may influence the intensity of the host defense responses (Kendra and Hadwiger, 1984, Kendra *et al.*, 1989). The importance of the size of chitosan oligomers in relation to its biological activity was also reported by other researchers (Pearce and Ride, 1982, Walker-Simmons *et al.*, 1984). Walker-Simmons *et al.*, (1984) demonstrated the rela-tionship between the size of chitosan oligomers and proteinase-lnhibitor inducing ac-tivity in tomato leaves. They showed that chitosan pentamer and hexamer were as active as the total HCl hydrolyzed chitosan in inducing proteinase inhibitor synthesis, while the monomer was inactive.

4. Induced Resistance in Postharvest Commodities

Plants, upon recognition of a potential pathogen or its by-products, usually mount a highly coordinated cascade of defensive responses to ward off infection. These re-sponses include reinforcement of the cell wall by deposition of lignin, callose, and hydroxyproline-rich glycoproteins; accumulation of phytoalexins; synthesis of pro-teinase inhibitors and lytic enzymes such as chitinases, chitosanase, and ß-1,3-glucanases (Kuc and Strobel, 1992, Schroder *et al.*, 1992, Lawton *et al.*, 1996, Ryalls *et al.*, 1996, Van Loom *et al.*, 1998). These biochemical and structural defense pro-cesses can also be triggered by treatment with biotic and abiotic elicitors. The induc-tion of defense reactions that involves the transcriptional activation of defense genes can be restricted to tissues close to the site of stimulus or can be expressed systemati-cally throughout the tissue. In recent years, considerable attention has been placed on induced resistance in vegetative crops as an important manageable form of plant pro-tection and several active microbial and chemical elicitors have been identified (Kuc and Strobel, 1992, Schroder *et al.*, 1992, Lawton *et al.*, 1996, Ryalls *et al.*, 1996, Van Loon *et al.*, 1998). This interest has been generated by the large volume of basic and

applied research that demonstrates that plants can be rendered resistant by artificially turning on their natural defense mechanisms (Kuc, 1990). Induced resistance has been studied extensively in tobacco and cucumber and its onset was closely correlated with an increase in the expression of defense proteins (Kuc, 1990).

Although fruit and vegetables also possess a natural defensive mechanism to ward off infection, this potential has not been fully explored. Very little is known regarding the nature and the regulation of defense mechanisms of harvested fruits and vegetables. In recent years, there have been several attempts to manipulate inducible defense mechanisms of harvested commodities through pre-storage treatments with innocuous abiotic and biotic elicitors to control postharvest decay. The reduction of postharvest decay by pre-storage treatment with fungal wall components and UV-C light suggests that induction of defense mechanisms has potential in controlling postharvest decay (Droby *et al.*, 1993, Stevens *et al.*, 1996, El Ghaouth *et al.*, 1992e). For instance, non-ionizing UV-C (from 190-280 nm) radiation has been shown to reduce decay in variety of commodities and the optimum doses of UV-C appear to occur in a rather narrow range depending on the commodity, the type of cultivar, and the physiological status of tissue (Chalutz *et al.*, 1992, Droby *et al.*, 1993, Stevens *et al.*, 1996).

Pre-storage treatment of several postharvest commodities with low doses of UV-C light has been shown to reduce disease development and in some cases delaying ripening (Stevens *et al.*, 1996). UV-C treatment controlled natural infection in Walla Walla onions, sweet-potato, tomato, apple, peaches, and citrus fruit (Droby *et al.*, 1993, Mercier *et al.*, 1993, Stevens *et al.*, 1996). In several commodities, UV-C treatment triggered a gradual development of tissue resistance that coincide with the induction and accumulation of phytoalexins (Ben-Yohoshua *et al.,* 1992, Droby *et al.*, 1993, Mercier *et al.*, 1993). Challenge inoculation of UV-treated citrus and apple fruit at different times following treatment revealed that the maximum resistance of fruit tissue to infection occurred within 24 to 48 h after UV-treatment and decreased afterward (Chalutz *et al.*, 1992). The implication of antifungal compounds in the UV-C mediated resistance was demonstrated in several commodities. In UV-treated citrus fruit the onset of tissue resistance to infection by *P. digitatum* was shown to coincide with the induction of phenylalanine ammonia lyase (PAL) activity, a key enzyme in the phenylpropanoid pathway, and peroxidase (Droby *et al.*, 1993). Similarly in UV-C treated carrot and lemon a close relationship between the level 6-methoxymellen and scoparone, respectively, and the resistance of carrot slices and lemon to infection was shown (Mercier *et al.*, 1993, Rodov *et al.*, 1994).

Induction of disease resistance in harvested commodities was also reported with plant growth regulators (jasmonic acid and gibberelic acid), a signal transduction compound (salicylic acid), polypeptides such as harpin, and heat treatment (Klein and Lurie, 1991, Ben-Yohoshua *et al.*, 1992, Afek *et al.*, 1995, Lyon *et al.*, 1995, Droby *et al.*, 1999b, Qui and Wei, 2000). Jasmonates have been shown to protect potato and tomato plants from infection (Cohen *et al.*, 1993) and reduce decay of harvested commodities such as strawberries, peppers, grapefruits, and roses (Moline *et al.*, 1997, Meir *et al.*, 1998, Droby *et al.*, 1999b). Prestorage heating of fruits and vegetables was also shown to reduce postharvest decay by directly inhibiting pathogen growth, acti-

vating natural resistance, and slowing down the ripening process (Couey, 1989, Klein and Lurie, 1991, Ben-Yohoshua *et al.*, 1992). In lemon fruit, heat treatment induced the accumulation of the phytoalexin, scoparone, and increased resistance to infection (Ben-Yohoshua *et al.*, 1992). Treatment of a variety of fruits with either microbial antagonists or attenuated strains of pathogens has been shown to induce several biochemical defense responses that may help ward off infections (Prusky *et al.*, 1994, Rodov *et al.*, 1994, Arras, 1996, El Ghaouth *et al.*, 1998a). In avocado fruits, a non-pathogenic endophytic mutant of *Colletotrichum magna* was shown to penetrate the peel of fruit and prevent anthracnose symptoms caused by *C. gloeosporioides* by activating various host defense responses (Prusky *et al.*, 1994). Induction of defense responses in harvested commodities was also observed following the treatment with antagonistic yeasts (Rodov *et al.*, 1994, Arras, 1996, El Ghaouth *et al.*, 1998a) and the implication of induced resistance in the mode of action of microbial antagonists has been discussed earlier.

Success with the induction of resistance in several harvested crops by treatment with biotic and abiotic elicitors suggests potential for technology based on these principles for the control of postharvest diseases of fruits and vegetables. In most cases, the biocontrol potential of the bioelicitor is attributable to the interplay of its biological and eliciting properties. Although the use of elicitors as a disease control method has been demonstrated with several postharvest commodities, the elicitor-mediated protection tends to last a short period and declines rapidly with ripening, a period where the fruit become more susceptible to infection. More effective methods of controlling diseases through pre-storage treatment with elicitors should ultimately emerge as we learn more about: (i). the biological activity of elicitors; (ii). the signal transduction pathways that link the host perception with the expression of defense genes required to ward off infection; (iii). the regulation of defense genes associated with the induced resistance in harvested tissue; (iv). their role in resistance; and (v). the effect of the ripening process on the disease resistance potential of the harvested crop.

5. Enhancement of biocontrol efficacy with additives

Several promising biological control approaches that include the use of antagonistic microorganisms, natural fungicides, and induced resistance are available for developing safer technologies for postharvest disease control. Among the proposed alternatives, development and enhancement of antagonistic microorganisms has been the most extensively studied and substantial progress has been made in this area. Despite all the efforts that focused on the development and use of microbial antagonists, bioactive compounds, and induced resistance for the control postharvest diseases, the widespread utility of these biological approaches has not been fully realized. The level of control provided by microbial antagonists, bioactive compounds, and induced resistance often fall short of chemical control and under commercial conditions none of these biological approaches have been shown to clearly offer consistent and effective control of decay comparable to that obtained with synthetic fungicides (Wilson *et al.,* 1996, El Ghaouth *et al.*, 1997a). Because infection of fruits occur either prior to har-

vest or during harvesting and processing, biological approaches are expected to display both a protective and curative activity comparable to that observed with synthetic fungicides. None of the proposed biological alternatives (microbial antagonists, bioactive compounds, and induced resistance), however, have been shown to control previously-established infections (Roberts, 1990, Wilson *et al.,* 1996). As we learn more about the fundamental bases underlying the protective effect of microbial antagonists, bioactive compounds, and induced resistance, more effective methods of formulating combinations of complementary biological approaches for additive and/ or synergistic effects will emerge. So far, the results obtained with different combinations of biological approaches demonstrate the potential of this multifaceted approach as a viable alternative to synthetic fungicides. Combining biocontrol protectants can be expected to have greater stability and effectiveness than the use of a single biocontrol agent.

5.1 Enhancing efficacy by addition of low levels of fungicides

Because certain fungicides such as imazalil and thiabendazole have been exceptionally effective in controlling postharvest diseases it is difficult to find biocontrol agents that will perform as effectively. Special attention needs to be paid to ways of enhancing the activity of biocontrol agents. One approach has been to combine the biocontrol agent with low levels of synthetic fungicides. If effective, this procedure can be used to reduce the chemical residues on fruits and vegetables while effectively controlling postharvest decay. In large scale tests on Navel and Valencia oranges, formulated biocontrol products such as ASPIRE and BIOSAVE-110 often provide a level of control equivalent to synthetic fungicides only when combined with a low doses of synthetic fungicides (Brown and Chambers, 1996, Droby *et al.*, 1998). ASPIRE in combination with 200 µg/ml of thiabendazole controlled citrus decay at the level equivalent to the commercial fungicide treatment and reduced the variability often observed when using the antagonistic yeast alone (Brown and Chambers, 1996, Droby *et al.*, 1998). Similar results were also reported on apple and pears fruits treated with a combination of *C. infirmo-miniatus* with 264 µg/ml thiabendazole (Chand-Goyal and Spotts, 1997). Isolate L47 of *A. pullulans* gave better results on strawberries and table grapes when sprayed in combination with a low dose of a fungicide as compared to the antagonist alone (Schena *et al.*, 1999).

5.2 Enhancing efficacy by salt additives

Recently, we have explored the use of various additives to improve efficacy of ASPIRE™ the product based on the yeast *C. oleophila.* Among the additives, we examined the use of calcium propionate, sodium bicarbonate, and EDTA. When used in combination with ASPIRE™, all three additives provided some level of enhancement of efficacy of ASPIRE™ against apple decay caused by *B. cinerea* and *P. expansum* (Droby *et al.*, unpublished data). On peach, calcium propionate and EDTA actually increased the percent of infection. The use of 2% sodium bicarbonate exhibited a consistent ability to significantly enhance the performance of ASPIRE (Droby *et al.*, un-

published data). The additive effect of sodium carbonate or bicarbonate was also reported by Smilanick and coworkers (1999b). They showed that control of green mold on oranges was maximized when dip treatments in sodium carbonate or bicarbonate were followed by the application of *P. syringae* strain ESC10, the active ingredient in the postharvest biological control BioSave™ products. Similar results were also observed with lemon fruits pretreated with sodium carbonate prior to treatment with *C. saitoana* (El Ghaouth *et al.*, 2000a and b). The biocontrol activity of microbial antagonists was also shown to be enhanced by calcium chloride. On pome fruit, the addition of $CaCl_2$ was shown to increase the protective effect of some antagonistic yeast and also greatly reduce the population size of yeasts required to give effective control (Mclaughlin *et al.*, 1990, Wisniewski *et al.*, 1995, Droby *et al.*, 1997).

5.3 Enhancing biocontrol activity by addition of nutrients

The biocontrol activity of microbial antagonists on citrus and pome fruit was also shown to be augmented by the addition of nitrogenous (L-asparagine and L-proline) compounds and 2-deoxy-D-glucose, a sugar analog (Janisiewsicz, 1994, El Ghaouth *et al.*, 2000a). The combination of *C. saitoana* with a low dose of 0.2 % (w/v) 2-deoxy-D-glucose applied to fruit wounds before inoculation, was more effective in controlling decay of apple, orange, and lemon caused by *B. cinerea*, *P. expansum*, and *P. digitatum* than either *C. saitoana* or 2-deoxy-D-glucose alone (El Ghaouth *et al.*, 2000a). In large-scale pilot tests on apple and citrus fruit the combination of *C. saitoana* with 0.2 % 2-deoxy-D-glucose displayed greater stability and effectiveness in controlling natural infection than either antagonist or sugar analog alone. The level of disease control obtained with the combination of *C. saitoana* with 0.2 % 2-deoxy-D-glucose was comparable to that obtained with the recommended fungicides (Imazalil and Thiabendazole). The biocontrol activity of bioenhancer appeared to be due to the synergistic interactions between the antagonistic activity of the yeast and the antifungal property of sugar analog. This was well illustrated by the curative activity of the combination of *C. saitoana* with 0.2 % 2-deoxy-D-glucose. In laboratory tests, the combination of *C. saitoana* with 0.2 % 2-deoxy-D-glucose was also effective against infections established up to 24 h before treatment. When applied within 24 h after inoculation, the combination of *C. saitoana* with 0.2 % 2-deoxy-D-glucose was very effective in controlling blue mold of apple and green mold of oranges and lemons. The level of control of green mold was equivalent to the imazalil treatment. No apparent control of blue mold of apple and mold of oranges and lemons was observed when *C. saitoana* or 2- deoxy-D-glucose were applied within 24 h after inoculation of the fruit with the pathogen.

5.4 Enhancing activity by addition of chitosan

 Recently, we have developed a biocontrol product termed a "bioactive coating" that consists of a unique combination of an antagonistic yeast with chemically modified chitosan (El Ghaouth *et al.*, 2000b). This combination makes it possible to exploit the antifungal property of chemically modified chitosan and the biological activity of the

antagonistic yeast. In laboratory studies, the biocontrol activity of *C. saitoana* against decay of apple, lemon, and orange caused by *B. cinerea*, *P. expansum*, and *P. digitatum* was enhanced markedly by the addition of glycolchitosan (El Ghaouth *et al.*, 2000b). Under semi-commercial conditions, the bioactive coating was superior to *C. saitoana* or glycolchitosan alone in controlling decay of oranges and lemons, and the control level was equivalent to that with imazalil (El Ghaouth *et al.*, 2000b). On apple fruits, depending on the apple variety used, the bioactive coating was comparable or superior to thiabendazole in reducing decay. Unlike *C. saitoana*, which showed a poor performance on late season fruit, the bioactive coating treatments offered consistent control of decay on oranges and lemons in early and late seasons. The combination of *C. saitoana* with 0.2 % glycolchitosan also reduced the incidence of stem-end rot of `Valencia´ oranges, but control was less effective than that with imazalil. The results obtained from semi-commercial tests demonstrates the potential of the bioactive coating as a viable alternative to synthetic fungicides.

5.5 Enhancing biocontrol efficacy by integration with physical means

The enhancement of microbial biocontrol agents has been reported with physical additives such as curing and heat treatments (Barkai-Golan, 1991, Huang *et al.*, 1995, Cook *et al.*, 1999), ultraviolet light (Stevens *et al.*, 1996), and modified or controlled atmosphere and cold storage (Sugar *et al.*, 1994, Lurie *et al.*, 1995). The efficacy of *C. oleophila* against postharvest rots of nectarines increased when fruits were stored at optimal low storage temperature under controlled atmosphere (Lurie *et al.*, 1995). Integrating UV-C radiation with antagonistic yeast was shown to enhance the performance of the yeast and provide a level of control equivalent to synthetic fungicides (Stevens *et al.*, 1996). Conway *et al.* (1999) showed that 'Gala' apples heated after harvest (38°C for 4 days), pressure-infiltrated with 2% CaCl$_2$ or treated with the antagonist *P. syringae,* reduced postharvest decay caused by *P. expansum.* The least decay occurred on apples treated with a combination of calcium plus the antagonist, or calcium plus the antagonist and the heat. Huang *et al.* (1995) demonstrated that biocontrol of green mold using *Pseudomonas glathei* could be enhanced when heat was applied to retard conidial germination of *P. digitatum* while simultaneously stimulating bacterial multiplication. Porat *et al.* (2000) showed that a hot water brushing treatment at 56 °C for 20 s reduced development of natural decay on different citrus cultivars such as 'Minneola' tangerines, 'Shamouti' oranges, and 'Star Ruby' grapefruit by 45-55% after 6 weeks storage at the appropriate temperature. In a more recent work, we have shown that the incidence of decay developed on grapefruits treated with hot water and *C. oleophila* 24 h after inoculation with *P. digitatum* was dramatically reduced in comparison to each treatment alone (unpublished data).

5.6 Enhancing biocontrol efficacy by mixture of antagonists

In almost all biocontrol systems, a single biocontrol agent is applied. However, it has been suggested that different antagonists should be used to assure adequate disease control under various condition (Cook, 1993). A mixture of antagonists may have at

least three main advantages: (i). it may broaden the spectrum of activity; (ii). it may enhance the efficacy, allowing a reduction in application rate; and (iii). it may allow the combination of various biocontrol traits without resorting to genetic engineering (Janisiewicz, 1998). A greater biocontrol activity of a strain mixture (*S. roseus* and *P. syringae*) against postharvest blue mould on Golden Delicious apples, as compared with the same strains applied individually, was demonstrated in bioassays under controlled environmental conditions (Janisiewicz, 1998). Similar results were obtained by Leibinger *et al.* (1997) using mixture of two strains of *A. pullulans* and one strain of *Rhodotorula glutinis* (Fresenius) Harrison (mixture M1) and two strain of *B. subtilis* and one of *A. pullulans* (mixture M2). Leibinger *et al.* (1997) used the mixtures (M1 and M2) also for preharvest treatments but they were not compared with the individual antagonists. Interestingly, in the most effective mixture (mixture M1) the population sizes of *A. pullulans* increased more than 10 fold on apples, while populations of *A. pullulans* were substantially reduced when this yeast-like fungus was applied in combination with *B. subtilis*. The base of this incompatibility was attributed to the production of antibiotic compound by *B. subtilis*. In trials on wounded table grape berries, better results were obtained combining a yeast-like fungus (*A. pullulans*) and a yeast (*Metschnikowia pulcherrima* Pitt & Miller) as compared to the use of a mixture of two yeasts (*M. pulcherrima* and *P. guilliermondii)*; similar results were obtained also in field treatment on table grapes (Ippolito *et al,* unpublished data). This behavior could be attributed to the same nutritional profile of the two yeast species (Janisiewicz, 1998). Better microorganism mixtures could be selected on a more rational basis if we have more information about the ecology of the microorganisms (Andrews, 1992).

5.7   Enhancing of biocontrol efficacy by preharvest application of antagonists

Preharvest application of biocontrol agents as a stand alone treatment or combined with a postharvest application of the biocontrol agent may also prove to be a useful strategy in achieving improved performance against infections. This approach could be used as a tool to manipulate epiphytic populations and change patterns of surface wound colonization. Reports indicate the possibility of reducing postharvest decay caused by *C. gloeosporioides* on avocado and mangoes by a preharvest spray with *B. subtilis*, an antibiotic producing bacterium (Korsten *et al.*, 1994). Koomen and Jeffries (1993) have also demonstrated the feasibility of controlling anthracnose on mango fruit with *Bacillus cereus* Frankland & Frankland and *Pseudomonas fluorescens* Migula. Droby *et al.* (1999a) suggested the possible use of preharvest application of the yeast antagonist *P. guilliermondii* to reduce the development of postharvest decay of citrus fruit, and a reduction of postharvest *Rhizopus* rot of table grapes was also achieved by a preharvest spray of the yeast *P. guilliermondii*. More recently, Benbow and Sugar (1999) showed that preharvest application of the yeasts *C. infirmo-ministus*, *C. laurentii* and *C. oleophila* provided control of postharvest decay on two pear cultivars. *C. infirmo-ministus* gave the most consistent postharvest decay control in fruit treated 3 weeks before harvest. Preharvest introduction of antagonists in conjunction with additional postharvest applications may prove successful in providing acceptable levels of con-

trol. To fully explore the potential of this approach, however, obtaining data on the composition of epiphytic populations before and after the introduction of a single antagonist is crucial.

6. References

Afek, U., Aharoni, N. and Carmeli, S.1995. Increased celery resistance to pathogens during storage and reducing high-risk psoralen concentration by treatment with GA$_3$. *Journal of the American Society of Horticulture Science* 120: 562-565.

Allan,C.R. and Hadwiger, L.A. 1979. The fungicidal effect of chitosan on fungi of varying cell wall composition. *Experimental Mycology* 3: 285-287.

Al Zaemey, A.B., Magan, N. and Thompson. A.K.1993. Studies on the effect of fruit-coating polymers and organic acids on growth of *Colletotrichum musae in vitro* and on postharvest control of anthracnose of bananas. *Mycological Research* 12: 1463-1468.

Andrews, J.H. 1992. Biological control in the phyllosphere. *Annual Review of Phytopathology* 30: 603-635.

Appel, D.J., Gees, R. and Coffey, M.D. 1988. Biological control of the postharvest pathogen *Penicillium digitatum* on Eureka lemons. *Phytopathology* 12: 1595-1599.

Araki, U. and Ito, E.A. 1975. pathway of chitosan formation in Mucor rouxii enzymatic deacetylation of chitin. *European Journal of Biochemistry* 55: 71-76.

Arras, G. 1996. Mode of action of an isolate of *Candida famata* in biological control of *Penicillium digitatum* in orange fruits. *Postharvest Biology and Technology* 8:191-198.

Base, C.W., Bock, K. and Boller, T. 1992. Elicitors and suppressors of the defense response in tomato cells. Purification and characterization of glycopeptide elicitors and glycan suppressors generated by enzymatic cleavage of yeast invertase. *Journal of Biology and Chemistry* 267: 10258-10265.

Barkai-Golan, R. and Douglas, J.P. 1991. Postharvest heat treatment of fresh fruits and vegetables for decay control. *Plant Disease* 75: 1085-1091.

Benbow, J.M. and Sugar, D. 1999. Fruit surface colonization and biological control of postharvest diseases of pear by preharvest yeast application. *Plant Disease* 83:839-844.

Benhamou, N. 1992. Ultrastructural and cytochemical aspects of chitosan on *Fusarium oxysporum* f. sp. *radicis-lycopersici,* agent of tomato crown and root rot. *Phytopathology* 82: 1185-1192.

Ben-Yohoshua, S., Rodov, V., Kim, J.J. and Carmeli, S. 1992. Preformed and induced antifungal materials of citrus fruits in relation to the enhancement of decay resistance by heat and ultraviolet treatments. *Journal of Agriculture and Food Chemistry* 40: 1217-1221.

Boller, T. 1988. Ethylene and the regulation of antifungal hydrolases in plants. *Oxford Survey of Plant Molecular and Cell Biology* 5:145-153.

Borras, A.D. and Aguilar, R.V. 1990. Biological control of *Penicillium digitatum* by *Trichoderma viride* on postharvest citrus fruits. *International Journal of Food Microbiology* 11: 179-184.

Brown, G.E. and Chambers, M. 1996. Evaluation of biological products for the control of postharvest diseases of Florida citrus. *Proceeding of Florida State Horticulture Society* 109: 278-282.

Bull, C.T., Stack, J.P. and Smilanick, J.L. 1997. *Pseudomonas syringae* strains ESC-10 and ESC-11 survive in wounds on citrus and control green and blue molds of citrus. Biological Control, 8: 81-88.

Bull, C.T., Wadsworth, M.L.K., Sorenson, K.N., Takemoto, J., Austin, R. and Smilanick, J.L. 1998. Syringomycin E produced by biological agents controls green mold on lemons. Bio-

logical Control, 12: 89-95.

Burkhead, K.D., Schisler, D.A. and Slininger, P.J. 1995. Bioautography shows antibiotic pro-
duction by soil bacterial isolates antagonistic to fungal dry rot of potatoes. *Soil Biology and
Biochemistry* 26: 1611-1616.

Chalutz, E., Droby, S., Wilson, C.L. and Wisniewski, M. 1992. UV-induced resistance to
postharvest diseases of citrus fruit. *Journal of Phytochemistry and Photobiology* 15: 367-
374.

Chand-Goyal, T. and Spotts, R. A. 1997. Biological control of postharvest diseases of apple and
pear under semi-commercial and commercial conditions using three saprophytic yeasts.
Biological Control 10: 199-206.

Conway, W.S., Janisiewicz, W.J., Klein, J.D. and Sams, S.E. 1999. Strategy for combining heat
treatment, calcium infiltration, and biocontrol to reduce postharvest decay of 'Gala' apples.
HortScience 34: 700-704.

Cook, R.J. 1993. Making greater use of introduced microorganisms for biological control of
plant pathogens. Annual Review of Phytopathology, 31: 53-80.

Cook, D.W.M., Long, P.G. and Ganesh, S. 1999. The combined effect of delayed application of
yeast biocontrol agents and fruit curing for the inhibition of the postharvest pathogen *Botrytis
cinerea* in Kiwifruit. Postharvest Biology and Technology, 16: 233-243.

Couey, H.M. 1989. Heat treatment for control of postharvest diseases and insect pest of fruit.
HortScience 24: 198-201.

Droby, S., Chalutz, E., Wilson, C.L. and Wisniewski, M.E. 1989. Characterization of the
biocontrol activity of *Debaryomyces hansenii* in the control of *Penicillium digitatum* on
grapefruit. *Canadian Journal of Microbiology* 35: 794-800.

Droby, S., Chalutz, E. and Wilson, C.L. 1991. Antagonistic microorganisms as biological con-
trol agents of postharvest diseases of fruits and vegetables. *Postharvest News and Informa-
tion* 2: 169-173.

Droby, S., Chalutz, E., Horev, B., Cohen, L., Gaba, V., Wilson, C.L. and Wisniewski, M. E.
1993. Factors affecting UV-induced resistance in grapefruit against the green mold decay
caused by Penicillium digitatum. *Plant Pathology* 42: 418-424.

Droby, S. and Chalutz, E. 1994. Mode of action of biocontrol agents for postharvest diseases.
In: *Biological Control of Postharvest Diseases of Fruits and Vegetables-Theory and Prac-
tice* (eds. Wilson,C.L. and Wisniewski, M.E.) CRC Press, Boca Raton, Florida, pp. 63-75.

Droby, S., Wisniewski, M.E., Cohen, L., Weiss, B., Touitou, D., Elam Y. and Chalutz, E. 1997.
Influence of CaCl$_2$ on *Penicillium digitatum*, grapefruit pell tissue, and biocontrol activity
of *Pichia guilliermondii.* Phytopathology, 87: 310-315.

Droby, S., Cohen, A., Weiss, B., Horev, B., Chalutz, E., Katz, H., Keren-Tzur, M. and Shachnai,
A. 1998. Commercial testing of Aspire: A yeast preparation for the biological control of
postharvest decay of citrus. Biological Control ,12 : 97-100.

Droby, S., Lischinski, S., Cohen, L., Weiss, B., Daus, A., Chand-Goyal, T., Eckert, J.W. and
Manulis, S. 1999a. Characterization of an epiphytic yeast population of grapefruit capable
of suppression of green mold decay caused by *Penicillium digitatum*. Biological Control,
16: 27-34.

Droby, S., Porat, R., Cohen, L., Weiss, B., Shapirom, B., Philosoph-Hadas, S. and Meir, S.
1999b. Suppressing green mold decay in grapefruit with postharvest jasmonate application.
Journal of the American Society of Horticulture Science, 124: 184-188.

Eckert, J.W. and Ogawa, J.M. 1988. The chemical control of postharvest diseases: Deciduous
fruits, berries, vegetables and roots/tuber crops. Annual Review of Phytopathology, 26:
433-469.

Eckert, J.W. and Eaks, I.L. 1989. Postharvest disorders and diseases of citrus fruits. In:" The
Citrus Industry", Vol. 4 (eds. Reuther, W. Calavan and, E. Carman, G.E.) University of

California, Division of Natural Resources, Oakland, California, pp.179-187.

Elad, Y. 1995. Mycoparasitism In: "Pathogenesis and Host-Parasite Specificity in Plant Diseases: Histopathological, Biochemical, Genetic and Molecular Basis" (eds. Kohomo, K., Singh, R.P. ,Singh, U.S. and Zeigler, R.) Pergamon Press Oxford, pp. 152-160.

El Ghaouth, A., Arul, J., Ponnampalam, R. and Bollet, M. 1991a. Use of chitosan coating to reduce water losses and maintain quality of cucumber and bell pepper fruits. Journal of Food Processing and Preservation, 15: 359-368.

El Ghaouth, A., Arul., Ponnampalam, R. and Bollet, M. 1991b. Chitosan coating effect on storability and quality of strawberries. Journal of Food Science, 56: 1618-1620.

El Ghaouth, A., Arul, J., Grenier, J. and Asselin, A. 1992a. Antifungal activity of chitosan on two postharvest pathogens of strawberry fruits. Phytopathology, 82: 398-492.

El Ghaouth, A., Ponnampalam, R., Castaigne, F. and Arul, J. 1992b. Chitosan coating to extend the storage life of tomatoes. HortScience, 27: 1016-1018.

El Ghaouth, A., Arul, J., Asselin, A. and Benhamou, N. 1992c. Antifungal activity of chitosan on post-harvest pathogens: Induction of morphological and cytological alterations in *Rhizopus stolonifer*. Mycological Research, 96: 769-779.

El Ghaouth, A., Arul, J., Grenier, J. and Asselin, A. 1992d. Effect of chitosan and other polyions on chitin deacetylase in *Rhizopus stolonifer*. Experimental Mycology*,* 16: 173-177.

El Ghaouth, A., Arul, J. and Asselin, A. 1992e. Potential use of chitosan in postharvest preservation of fruits and vegetables. In: *Advances in Chitin and Chitosan* (eds. Brines, J.B., Sandford, P.A. and Zikakis, J.P.) Elsevier Applied Science, London, pp. 440-452.

El Ghaouth**,** A., Arul, J., Wilson, C.L., Asselin, A. and Benhamou, N. 1994. Ultrastructural and cytochemical aspects of the effect of chitosan on decay of bell pepper fruit. Physiological and Molecular Plant Pathology, 44:417-432.

El Ghaouth, A. and Wilson, C. 1995. Biologically-based technologies for the control of postharvest diseases. Postharvest News and Information, 6: 5-11.

El Ghaouth, A. 1997a. Biologically-based alternatives to synthetic fungicides for the control of postharvest diseases. Journal of Industrial Microbiology and Biotechnology, 19: 160-162.

El Ghaouth, A., Wilson, C. and Wisniewski, M. 1997b. Antifungal activity of 2-Deoxy-D-glucose on *Botrytis cinerea*, *Penicillium expansum*, and *Rhizopus stolonifer*: Ultrastructural and Cytochemical aspect. Phytopathology, 87: 772-779.

El Ghaouth, A., Arul, J., Wilson, C. and Benhamou, N. 1997c. Biochemical and cytochemical events associated with the interaction of chitosan and Botrytis cinerea in bell pepper fruit. Postharvest Biology and Technology, 12: 183-194.

El Ghaouth, A., Wilson, C.L. and Wisniewski, M. 1998a. Ultrastructural and cytochemical aspect of the biocontrol activity of *Candida saitoana* in apple fruit. Phytopathology, 88: 282-291.

El Ghaouth, A. 1998b. Manipulation of defense systems with elicitors to control postharvest diseases. In: "Postharvest handling of tropical fruits" (eds. Champ, B.R., Highley, E. and Johnson, G.I.) ACIAR Proceedings, Canberra, Australia 80:131-135.

El Ghaouth, A., Smilanick, J., Wisniewski, M. and Wilson, C.L. 2000a. Improved control of apple and citrus fruit decay with a combination of *Candida saitoana* with 2-deoxy-D-glucose. Plant Disease, 84: 249-253.

El Ghaouth, A., Smilanick, J., Eldon Brown, E. G., Ippolito, A. and Wilson, C.l. 2000b. Application of *Candida saitoana* and glycolchitosan for the control of postharvest diseases of apple and citrus fruit under semi-commercial conditions. Plant Disease, 84: 243-248.

Elson, C.M., Hayes, E.R. and Lidster, P.D. 1985. Development of the differentially permeable fruits coating "Nutri-Save" for the modified atmosphere storage of fruit. In:" Controlled atmosphere research" (ed. Blankership, S.M.) North Carolina State University, Raleigh, pp. 248-254.

Filar, L., Rahe, J.E. and Wirick, M.G. 1978. Bulk solution properties of chitosan. In: "Chitin and chitosan" (eds. Muzzarelli, R.A.A. and Pariser, E.R.) MIT, Cambridge, MA, pp- 169-174.

Filonow, A.B., Vishniac, H.S., Anderson, J.A. and Janisiewicz, W.J. 1996. Biological control of *Botrytis cinerea* in apple by yeasts from various habitats and their putative mechanisms of antagonism. Biological Control, 7: 212-220.

Gaur, N.K., Klotz, S.A. and Henderson, R.L. 1999. Overexpression of the *Candida albicans* ALA1 gene in *Saccharomyces cerevisiae* results in aggregation following attachment of yeast cells to extracellular matrix proteins, adherence properties similar to those of *Candida albicans*. Infection Immunology, 67: 6040-6047.

Gueldner, R.C., Reilly, C.C., Pusey, P.L., Costello C.E. and Arrendale, R.F. 1988. Isolation and identification of iturins as antifungal peptides in biological control of peach brown rot with *Bacillus subtilis*. Journal of Agricriculture and Food Chemistry, 36: 366-370.

Guinebretiere, M.H., Nguyen-the, C., Morrison, N., Reich, M. and Nicot, P. 2000. Isolation and characterization of antagonists for the biocontrol of the postharvest wound pathogen *Botrytis cinerea* on strawberry fruits. Journal of food Protection, 63: 386-394.

Hadwiger, L.A., Fristensky, B. and Riggleman, R.C. 1984. Chitosan, a natural regulator in plant-fungal pathogen interactions, increases crop yield. In: "Chitin, chitosan, and related enzymes" (ed. Zikakis, J.P.) Academic Press Inc, Orlando, pp. 291-298.

Hadwiger, L.A., Ogawa, T. and Kuyama, H. 1994. Chitosan polymer sizes effective in inducing phytoalexin accumulation and fungal suppression are verified with synthesized oligomers. Molecular Plant-Microbe Interaction, 7: 531-533.

Hirano, S., Senda, H., Yomamoto, Y. and Watanabe, A. 1984. Several novel attempts for the use of the potential functions of chitin and chitosan. In: "Chitin, chitosan, and related enzymes" (ed. Zikakis, J.P.) Academic Press Inc, Orlando, pp. 77-86.

Hong, C., Michailides, T.J. and Holtz, B.A. 1998. Effects of wounding, inoculum density, and biological control agents on postharvest brown rot of stone fruits. Plant Disease, 82:1210-1216.

Huang, Y., Deverall, B.J. and Morris, S.C. 1995. Postharvest control of green mold on oranges by a strain of *Pseudomonas glathei* and enhancement of its biocontrol by heat treatment. Postharvest Biology and Technology, 3: 129-137.

Ippolito, A., El Ghaouth, A., Wisniewski, M. and Wilson, C. 2000. Control of postharvest decay of apple fruit by *Aurobasidium pullulans* and induction of defense responses. Postharvest Bioliogy and Technology, 19:265-272.

Janisiewicz, W.J. and Roitman, J. 1988. Biological control of blue mold and gray mold on apple and pear with *Pseudomonas cepacia*. Phytopathology, 78:1697-1700.

Janisiewicz, W.J. Yourman, L., Roitman, J. and Mahoney, N. 1991. Postharvest control of blue mold and gray mold of apples and pears by dip treatment with pyrrolnitrin, a metabolite of *Pseudomonas cepacea*. Plant Disease, 75 : 490-494.

Janisiewicz, W.J. 1994. Enhancement of biocontrol of blue mold with nutrient analog 2-deoxy-D-glucose on apples and pears. Applied Environmental Microbiology, 60:2671-2676.

Janisiewicz, W.J. 1998. Biocontrol of postharvest diseases of temperate fruits: Challenges and opportunities. In: "Plant-microbe interactions and biological control" (eds. Boland, J. and Kuykendall, L.D.) Marcel Dekker, New York, pp 171-198.

Kendra, F.D. and Hadwiger, L.A. 1984. Characterization of the smallest chitosan oligomer that is maximally antifungal to *Fusarium solani* and elicits pisatin formation in *Pisum sativum*. Experimental Mycology, 8: 276-281.

Kendra, F.D., Christian, D. and Hadwiger, L.A. 1989. Chitosan oligomers from *Fusarium solani*/pea interactions, chitinase/β-glucanase digestion of sporelings and from fungal wall chitin actively inhibit fungal growth and enhance disease resistance. Physiological and Molecular Plant Pathology, 35: 215-223.

Klein, D. J. and Lurie, S. 1991. Postharvest heat treatment and fruit quality. Postharvest News and Information, 2: 15-19.

Koomen, I. and Jeffries, P. 1993. Effect of antagonistic microorganisms on the postharvest development of *Colletotrichum gloeosporioides* on mango. Plant Pathology, 42: 230-237.

Korsten, L. De Villiers, E. E., Wehner, F. C. and Kotze, J. M. 1994. A review of biological control of postharvest diseases of subtropical fruits. In: "Postharvest handling of tropical fruits (eds. Champ, B.R., Highley, E. and Johnson, G.I.) ACIAR Proceedings, Canberra, Australia 50: 172-185.

Korsten, L., De Jager, E.S., De Villers, E.E., Lourens, A., Kotze, J.M. and Wehner, F.C. 1995. Evaluation of bacterial epiphytes isolated from avocado leaf and fruit surfaces for biocontrol of avocado postharvest diseases. Pant Disease, 79: 1149-1156.

Kuc, J. and Strobel, N. 1992. Induced resistance using pathogens and nonpathogens. In: "Biological Control of Plant Diseases" (eds. Tjamos, E. and Papavisas, G.) Plenum Press, New York, pp. 295-303.

Kuc, J. 1990. Immunization for the control of plant disease. In: "Biological Control of Soilborne Plant Pathogens" (ed. Hornby, D.) C.A.B International, Oxfordshire, UK, pp 355-373.

Lawton, K., Friedrich, L., Hunt, M., Weymann, K., Kessmann, H., Staub, T. and Ryalls, J. 1996. Benzothiadiazole induces disease resistance in *Arabidopsis* by activation of the systemic acquired resistance signal transduction pathway. Plant Journal, 10: 71-82.

Leibinger, W., Breuker, B., Hahn, M. and Mendegen, K. 1997. Control of postharvest pathogens and colonization of apple surface by antagonistic microorganisms in the field. Phytopathology, 87:1103-1110.

Lurie, S., Droby, S., Chalupowicz, L. and Chalutz, E. 1995. Efficacy of *Candida oleophila* strain 182 in preventing Penicillium expansum infection of nectarine fruits. Phytoparasitica, 23:231-234.

Lyon, G. D., Reglinski, T. and Newton, A. C. 1995. Novel disease control compounds: the potential to immunize plants against infections. Plant Pathology, 44: 407-427.

Mattheis, J. and Roberts, R. 1993. Fumigation of sweet cherry (*Prunus avium* 'Bing') fruit with low molecular weight aldehydes for postharvest decay control. Plant Disease, 77: 810-814.

Mauch, F., Hadwiger, L. A. and Boller, T. 1984. Ethylene: Symptom, not signal for the induction of chitinase and ß-1,3-glucanase in pea pods by pathogens and elicitors. Plant Physiology, 76:607-612.

Mauch, F., Mauch-Mani, B. and Boller, T. 1988. Antifungal hydrolases in pea tissue II. Inhibition of fungal growth by combination of chitinase and ß-1,3-glucanase. Plant Physiology, 88: 936-942.

McGuire, R.G. 1994. Application of *Candida guilliermondii* in commercial citrus coating for biocontrol of *Penicillium digitatum* on grapefruits. Biological Control, 3: 1-7.

McLaughlin, R.J. Wisniewski, M.E. Wilson, C.L. and Chalutz, E. 1990. Effect of inoculum concentration and salt solutions on biocontrol of postharvest diseases of apple with *Candida* sp. Phytopathology, 80: 456-461.

Mercier, J., Arul, J., Ponnampalam, R. and Boulet, M. 1993. Induction of 6-methoxymellein and resistance to storage pathogens in carrot slices by UV-C. Journal of Phytopaphology, 137: 44-55.

Meir, S.S., Philosoph-Hadas, S., Lurie, S., Droby, S. and Akerman, G. 1998. Reduction of chilling injury in stored avocado, grapefruit, and bell pepper by methyl jasmonate. Canadian Journal of Botany, 74: 870-874.

Moline, H.E., Buta, J.G., Saftner, R.A. and Maas, J.L. 1997. Comparison of three volatile natural products for the reduction of postharvest decay in strawberries. Advance in Strawberry Research**,** 16: 43-48.

Moore, D. 1981. Effect of hexose analogs on fungi: Mechanisms of inhibition and resistance. New Phytologist, 87: 487-515.

Pearce, R.B. and Ride, J.P. 1982. Chitin and related compounds as elicitors of the lignification response in wounded wheat leaves. Physiological Plant Pathology, 20: 119-123.

Pesis, E. and Avissar, I. 1990. Effect of postharvest application of acetaldehyde vapour on strawberry decay, taste and certain volatiles. Journal of Science and Food and Agriculture , 52: 377-385.

Porat, R., Daus, A., Weiss, B., Cohen, L., Falik, E. and Droby, S. 2000. Reduction of postharvest decay in organic citrus fruit by a short hot water brushing treatment. Postharvest Biology and Technology, 18: 151-157.

Prusky, D. Freeman, S. Rodrigues, R.J. and Keen, N.T. 1994. A nonpathogenic mutant strain of *Colletotrichium magna* induces resistance to *C. gloeosporioides* in avocado fruits. Molecular Plant Microbe Interaction, 7: 326-333.

Pusey, P.L. and Wilson, C.L. 1984. Postharvest biological control of stone fruit brown rot. by *Bacillus subtilis*. Plant Disease, 68: 753-756.

Pusey, P.L. 1989. Use of *Bacillus subtilis* and related organisms as biofungicides. Pesticide Science, 27: 133-140.

Qui, D. and Wei, Z.M. 2000. Effect of Messenger on gray mold and other fruit rot diseases. Phytopathology, 82:62.

Roberts, R.G. 1990. Biological control of gray mold of apple by *Cryptococcus laurentii*. Phytopathology,\ 80: 526-530.

Rodov, V., Ben-Yehoshua, S., Albaglis, R., and Fang, D. 1994. Accumulation of phytoalexins scoparone and scopoletin in citrus fruits subjected to various postharvest treatments. Acta Horticulturae, 381: 517-523.

Ryalls, J., Neuenschwander, U., Willits, M., Molina, A., Steiner, H.Y. and Hunt, M. 1996. Systemic acquired resistance. Plant Cell, 8: 1809-1819.

Schena, L., Ippolito, A., Zahavi, T., Cohen, L., Nigro, F. and Droby, S. 1999. Genetic diversity and biocontrol activity of *Aureobasidium pullulans* isolates against postharvest rots. Postharvest Biology and Technology, 17: 189-199.

Schlumbaum, A., Mauch, F., Vogeli, U. and Boller, T. 1986. Plant chitinases are potent inhibitors of fungal growth. Nature, 324: 365-367.

Schroder, M., Hahlbrock, K. and Kombrink, E. 1992. Temporal and spatial patterns of β-1,3-glucanase and chitinase induction in potato leaves infected by *Phytophthora infestans*. Plant Journal, 2: 161-172.

Singh, V. and Deverall, B. J. 1984. *Bacillus subtilis* as a control agent against fungal pathogens of citrus fruit. Transactions of the British Mycological Society, 83: 487-490.

Sholberg, P.L. and Shimizu, B.N. 1991. Use of the natural plant products, hinokitiol, to extend shelf-life of peaches. Journal of Canadian Institute of Food Science and Technology, 2: 273-276.

Smilanick, J.L. and Dennis-Arrue, R. 1992. Control of green mold of lemons with *Pseudomonas* species. Plant Disease, 76:481-485.

Smilanick, J.L., Margosan, D.A. and Henson, D.J. 1995. Evaluation of heated solutions of sulfur dioxide, ethanol, and hydrogen peroxide to control postharvest green mold of lemons. Plant Disease, 79: 742-747

Smilanick, J.L., Crisosto, C. and Mlikota, F. 1999a. Postharvest use of ozone for decay control. Perishables Handling Quarterly, 99:10-14.

Smilanick, J.L., Margosan, D.A., Mlikota, F., Usall, J. and Michael, I.F. 1999b. Control of citrus green mold by carbonate and bicarbonate salts and the influence of commercial postharvest practices on their efficacy. Plant Disease, 83:139-145.

Spotts, R.A. and Cervantes, L.1986. Populations, pathogenicity, and benomyl resistance of

Botrytis spp., and *Mucor piriformis* in packinghouses. Plant Disease, 70: 106-108.

Stadelbacher, G.J. and Prasad, Y. 1974. Postharvest decay control of apple by acetaldehyde vapor. Journal of the American Society of Horticulture Science, 99: 364-369.

Stevens, C., Kahn, V.A., Lu, J.Y., Wilson, C., El Ghaouth, A., Chalutz, E. and Droby, S. 1996. Low dose UV-C light as a new approach to control decay of harvested commodities. Recent Research Developments in Plant Pathology, 1: 155-169.

Stossel, P. and Leuba, J.L. 1984. Effect of chitosan, chitin and some aminosugars on growth of various soilborne phytopathogenic fungi. Phytopathologische Zeitschrift, 111: 82-90.

Sugar, D., Roberts, R.G., Hilton, R. J., Reghetti, T.L. and Sanchez, E.E. 1994. Integration of cultural methods with yeast treatment for control of postharvest decay in pear. Plant Disease, 78: 791-795.

Swinburne, T.R. 1983. Quiescent infections in postharvest diseases. *In* : Postharvest Pathology of fruits and vegetables. In: "Postharvest Pathology of fruits and vegetables" (ed. Dennis, C.) Academy Press, London, pp. 1-25.

Teixido, N., Usall, J. and Vinas, I. 1999. Efficacy of preharvest and postharvest *Candida sake* biocontrol treatments to prevent blue mould on apples during cold storage International Journal of Food Microbiology, 50: 203-210.

Tong-Kwee, L. and Rohrbock, K. G. 1980. Role of *Penicillium funiculosum* strains in the development of pineapple fruit diseases. Phytopathology, 70: 663-665.

Van Loon, L.C., Bakker, P.A.H. and Pieterse, M.J. 1998. Systemic resistance induced by rhizosphere bacteria. Annual Review of Phytopathology, 36: 453-483.

Vaugh, S. F., Spencer, G. F. and Shasha, S. 1993. Volatile compounds from raspberry and strawberry fruit inhibit postharvest decay fungi. Journal of Food Science, 58: 793-796.

Walker-Simmons, M., Jin, D., West, C.A., Hadwiger, L. and Ryan, C.A. 1984. Comparison of proteinase inhibitor-inducing activities and phytoalexin elicitor activities of a pure fungal endopolygalacturonase, pectin fragments, and chitosans. Plant Physiology, 76: 833-836.

Wilson, L.C., Franklin, J.D., and Otto, B. 1986. Fruit volatiles inhibitory to *Molonilia Fructicola* and *Botrytis cinerea*. Plant Disease, 71: 316-319.

Wilson, C.L. and Wisniewski, M.E. (eds.). 1994. Biological Control of Postharvest Diseases of Fruits and Vegetables-Theory and Practice. CRC Press, Boca Raton, Florida.

Wilson, C.L., Wisniewski, M.E., El Ghaouth, A., Droby, S. and Chalutz, E. 1996. Commercialization of Antagonistic Yeasts for the Biological Control of Postharvest Diseases of Fruits and Vegetables. Journal of Industrial Microbiology and Biotechnology, 46 237-242.

Wilson, C., El Ghaouth, A., Solar, J. and Wisniewski, M. 1997. Rapid evaluation of plant extracts and essential oils for fungicidal activity against Botrytis cinerea. Plant Disease, 81: 204-210.

Wisniewski, M.E., Wilson, C.L. and Hershberger, W. 1989. Characterization of inhibition of *Rhizopus stolonifer* germination and growth by *Enterobacter cloacae*. Canadian Journal of Botany, 67:2317-2323.

Wisniewski, M.E., Biles, C., Droby, S., McLaughlin, R., Wilson, C. and Chalutz, E. 1991. Mode of action of the postharvest biocontrol yeast, *Pichia guilliermondii*. I. Characterization of attachment to *Botrytis cinerea*. Physiological and Molecular Plant Pathology, 39: 245-258.

Wisniewski, M.E. and Wilson, C.L. 1992. Biological control of postharvest diseases of fruits and vegetables Recent. Advances in Horticulture, 27: 94-98.

Wisniewski, M.E., Droby, S., Chalutz, E. and Eilam, Y. 1995. Effect of Ca^{+2} and Mg^{+2} on *Botrytis cinerea* and *Penicillium expansum in vitro* and on the biocontrol activity of *Candida oleophila*. Plant Pathology, 44: 1016-1024.

Yakoby, N., Zhou, R., Kobiler I., Dinoor, A. and Prusky, D. 2001. Development of *Colletotrichum gloeosporioides* restriction enzyme-mediated integration mutants as biocontrol agents against anthracnose disease in avocado fruits. Phytopathology, 91: 143-148.

Mycorrhiza in Management of Fruits and Vegetables Diseases

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Abstract: Fruits and vegetables are important source of essential components in adequate and balanced human diet. The indiscriminate use of synthetic pesticides in production system of fruits and vegetables is of great concern for health and environment safety. Research and Development strategies are presently diverted in search of suitable biological alternatives to replace the pesticide use. Substantial progress has been made in exploring the use of microorganisms in control of plant diseases in integrated plant disease management. One such strategy is the better exploitation of microbes present in soil, which contribute to soil fertility. Certain fungi colonize and form symbiotic association with roots of the plant like mycorrhiza. Significant advances have been made in last two decades to understand the potential of mycorrhizal fungi in suppression of plant pathogens especially soil borne pathogens in wide range of fruits and vegetable host plants. The role of mycorrhiza in management of fruits and vegetable diseases is discussed with their mode of action and future perspectives.

1. Introduction

Fruits and vegetables are the rich source of essential components like vitamins, minerals and carbohydrates in a balanced human diet. These commodities are being consumed largely as desserts, salads and also in certain parts of world as staple food. In commercial production system of fruits and vegetables for their quality and high productivity per unit area, the pesticides are being used indiscriminately at large scale. Since fruits are vegetables are mostly consumed fresh, there has been uprising concern among the consumers for the pesticidal residues and also for environmental safety. Recently, a number of pesticides have been withdrawn from the market and in most of the countries, there is restriction for their use in fruits and vegetable crops. The demand for organically grown pesticides free crops is gaining momentum worldwide and these crops also fetch reasonably high price in global market. The efforts are being made to discover suitable alternatives and / or substitutes for synthetic pesticides and Research and Development agencies in various parts of world have intensified their research programmes in search of such bioagents for integrated management of fruits and vegetable diseases and to reduce the use of chemicals. Substantial progress has been made in exploring the use of microorganisms in control of plant diseases and quite a good number of biocontrol agents have been registered for their commercial use.

One such strategy is the better exploitation of microbes present in soil, which contribute to soil fertility. Certain fungi colonize and form symbiotic association with

roots of the plant like mycorrhiza. Significant advances have been made in last two decades to understand the potential of mycorrhizal fungi in suppression of soil borne pathogens and thereby imparting resistance to plant disease in wide range of fruits and vegetable host plants.

The fungi forming arbuscular mycorrhiza (AM) are of increased interest due to the widespread distribution of these fungi in the soils of various climatic zones and their ability to colonize major fruits and vegetable crops thereby enhancing plant productivity and improving their mineral nutrition especially phosphorus (Smith, 2002). Number of studies have demonstrated the beneficial role of AM fungi in plant growth and health (Bethlenfalvay and Linderman, 1992, Bagyaraj, 1992) and it appears that they are essential for the survival of plant species in many ecosystems (Allen, 1991). Root pathogens are a major limiting factor for plant production. Fungi belonging to the genera *Phytophthora, Fusarium, Pythium, Rhizoctonia*, some bacteria, nematodes are the most widespread among the various fruits and vegetable crops. They kill the roots or reduce their ability to absorb water and nutrients by penetrating root tissues and producing toxins. In recent years, many reports indicate an interaction between AM fungi and plant pathogenic organisms (Jalali and Jalali,1991, Mukerji, 1999, Singh *et al.,* 2000, Abdalla and Abdel-Fattah, 2000, Abdel-Fattah and Shabana, 2002). A host plant previously inoculated with AM fungi exhibits increased resistance to several root diseases (Linderman, 2000). Because AM fungi establish in the roots of the host plants and research on mycorrhizae- pathogen interactions has been concentrated on diseases caused by soil borne pathogens. Reduction of disease symptoms and severity has been reported in different fruits and vegetables with mycorrhiza-pathogen interaction for various fungal pathogens like *Phytophthora, Fusarium, Pythium, Rhizoctonia, Verticillium, Thielaviopsis, Aphanomyces, Pyrenochaeta,* bacterial pathogens like *Pseudomonas, Erwinia*, viruses and nematodes like *Radopholus, Heterodera, Rotylenchus, Pratylenchus, Tylenchulus, Meliodogyne* (Table1, Table 2). The influence of AM fungi on root pathogens vary greatly with the type of pathogen (Sharma *et al.,* 1992).

Some reports also indicate an increase in disease severity under the influence of AM fungi (Ross, 1972, Davis *et al.,* 1978, Davis and Menge, 1980, Shaul *et al.,* 1999) while some workers have reported no effect in disease incidence by AM fungi. (Baath and Hayman,1984, Prados-Ligero *et al.,* 2002). In case of viral diseases, mycorrhizal plants always show increased intensity of disease (Nemec and Myhre, 1984, Jayaram and Kumar1995, Shaul *et al.,* 1999). In this chapter, the role of mycorrhiza in management of fruits and vegetable diseases is discussed with their mode of action and future perspectives.

2. AM fungi and plant pathogens

Many reports indicate an interaction between AM fungi and plant pathogenic organisms. To evaluate the influence of AM fungi on disease incidence and development, different factors like plant pathogen, host plant, AM fungal species/strain and environmental conditions have to be taken into account. Mostly, the interactions between pathogen and symbiont are mediated by the host. The effectiveness of AM fungi in

protecting plants against disease incidence varies according to the species, strain of AM fungus and variety of host plant involved.

2.1 Diseases caused by soil borne fungi

AM fungi colonize the roots of host plants, so most of the research on mycorrhizae – disease incidence has been concentrated on diseases caused by soil borne pathogens. Diseases caused by soil borne fungi can be influenced by the colonization of AM fungi in the root system of the plant. Most of the results indicate that mycorrhizal plants suffer less damage and the incidence of disease is decreased or development of pathogen is inhibited (Schenck and Kellam, 1978, Schonbeck, 1980). However some of the reports indicate an increased susceptibility of mycorrhizal plants to disease (Ross, 1972, Atilano *et al.,* 1976, Davis et *al.,* 1978, Davis and Menge, 1980).

Positive effect of AM fungi in reducing disease severity can be seen within a broad range of host plants and different fungal pathogens. High inoculum levels of the pathogen decrease the positive influence of the AM fungal inoculation on disease incidence. In case of *Fusarium* wilt of tomato , mycorrhizal plants had less mycelium inside the stem tissue as a result of vascular infection but increased inocula of the pathogen changed this effect (Dehne and Schonbeck, 1979).

Interactions between AM fungi , fungal root pathogens and host plants are complex and each combination should be considered individually. Effectiveness of AM fungi in protecting plants varies according to the species , strain of AM fungus and host plant involved (Schenck and Kellam, 1978). Plants of *Citrus* are protected against *Phytophthora cinnamomi* root rot when the plant is previously inoculated with AM fungus *Glomus fasciculatum* (Davis and Menge, 1980). Mycorrhizal onion roots were reported to be less susceptible to pink rot caused by *Pyrenochaeta terrestris* (Safir, 1968).

Different AM fungi confer variable tolerance or resistance to *Phytophthora parasitica*. Out of nine different symbionts used for studying interaction, maximum benefit in terms of increased growth response was conferred by *G. fasciculatum* and *G. constrictus* (Davis and Menge, 1981). Nemec(1974) found higher number of *Endogone* spores in rhizosphere soil of strawberry roots growing in a plot without root rot than in the rhizosphere of roots in a plot with root rot. His results showed that as the roots age, or are diseased, *Endogone* reacts by producing numerous spores in the rhizosphere. The order in which the AM fungus and pathogen are added in the soil during the experiment is also important in determining the effect on growth of host plants. Caron *et al.,*(1986a) studied the effect of sequence of inoculation on the interaction between *G. intraradices* and *F. oxysporum* f.sp. *radicis lycopersici* in tomatoes. Mycorrhizal root with *G. intraradices* showed reduction in root necrosis due to *Fusarium* infection independent of the sequence of inoculation of the two fungi. However this effect was more pronounced when *G. intraradices* was inoculated four weeks prior to *F. oxysporum*. In mungbean, *Glomus coronatum* reduced the severity of disease caused by *Rhizoctonia solani*.(Kasiamdari *et al.,* 2002).An orchard experiment was established to determine the long term (6 year) effects of chemical, fertilizer and biological agents including *G. intraradices* on root rot incidence of apple trees in apple-replant disease soil that

Table 1: Influence of AM fungi in control of different pathogens of fruit crops

Host	Pathogen	AM fungi	References
Fungal Pathogens			
Avocado	*Phytophthora*		Davis *et al.*, 1978.
Citrus	*cinnamomi*	*Glomus* sp.	Davis *et al.*, 1978
	P. cinnamomi		
	P. parasitica	*G.fasciculatum*	Davis and Menge,
		G. mosseae	1981.
		Gigaspora	
		margarita	
		G.constrictum	
Citrus	*Thielaviopsis*	*G.fasciculatum*	Davis, 1980
	basicola		
Pyrus	*Phtophthora*	*G.intraradices*	Utkhede and Smith,
malus	*cactorum, Pythium*		2000
	ultimum		
Papaya	*P. palmivora*		Ramirez, 1974
Strawberry	*P.cactorum*	*G.mosseae*	Vestberg *et al.*, 1994
micropropa-		*G.fistulosum*	
gated			
Nematodes			
Citrus	*Tylenchulus*		Baghel *et al.*, 1990
jambhiri	*semipenetrans*		
C.limon	*Radopholus*	*G.intraradices*	Smith and Kaplan,
	citrophilus		1982
	R. similis	*G.etunicatus*	O'Bannon and Nemec
			1979
Musa	*R. similis*	*G.fasciculatum*	Umesh *et al.,* 1988
acuminata			
Prunus	*Pratylenchus*	*G.intraradices*	Pinochet *et al.*, 1996
avium	*vulnus*		
P. domestica	-do-	*G.mosseae*	Camprubi *et al.*, 1993
P. insititia	-do-	-do-	-do-
P. persica	-do-	-do-	Pinochet, *et al.*, 1996
Pyrus malus	-do-	*G. intraradices*	-do-
Vitis vinifera	*Meloidogyne*	*G.fasciculatum*	Atilano *et al.*, 1976
	arenaria		
Musk melon	*Rotylenchulus*	*G. mosseae*	Sitaramaiah and
	reniformis		Sikora, 1981
Viruses			
Citrus macrophylla	Citrus tristeza virus	Endogone	Schonbeck, 1979
Strawberry	Arabis mosaic virus	Endogone	Daft and Okasanya,
			1973
Grapefruit	Citrus leaf rugose		Schonbeck, 1979
	virus		
Sour orange	CTV		-do-

Table 2: Influence of AM fungi in control of different pathogens of vegetable crops

Host	Pathogen	AM fungi	References
Fungal Pathogens			
Allium cepa	*Fusarium oxysporum* f.sp. cepi		Dehne, 1982
	Pyrenochaeta terrestris		Becker, 1976
	Sclerotium cepivorum	*Glo*mus sp.	Torres-Barragan *et al.*, 1996
Brassica sp.	*Rhizoctonia solani*		Iqbal *et al.*, 1988, Iqbal and Nasim, 1988
Cowpea	*Rhizoctonia solani*	*G.clarum*	Abdel-Fattah and Shabana, 2002
Tomato	*F.oxysporum*	*G.mosseae*	Al-Momany and Al-Raddad, 1988
	F.oxy. f.sp. lycopersici	G.intraradices	Caron *et al.*, 1986a,b Dehne and Schonbeck, 1979 McGraw and Schenck, 1981
	Fusarium sp.	*G.mosseae*	Ramraj *et al.*, 1988
	Pyrenochaeta lycopersici	*G.caledonium*	Bochow and Abou-Shaar, 1990
	Phytophthora nicotianae	*G. mosseae*	Cordier *et al.*, 1996 Trotta *et al.*, 1996
	P. parasitica	*G.mosseae*	Pozo *et al.*, 2002
	Verticillium albo-atrum		Baath and Hayman 1983
	V. dahliae	*G.mosseae*	Karagiannidis *et al.* 2002
	Pythium aphanidermatum	*G. fasciculatum*	Hedge and Rai, 1984
Cucumis melo	*F.oxysporum*		Schonbeck, 1979
Cucumber	*Pythium ultimum*		Rosendahl and Rosendahl, 1990
Phaseolus aureus	*Macrophomina phaseolina*	*G.mosseae*	Jalali *et al.*, 1990
Phaseolus vulgaris	*Rhizoctonia solani*	*G.intraradices*	Guillon *et al.*,2002
Pisum sativum	*Aphanomyces euteiches*	*G.fasciculatum*	Rosendahl, 1985
	-do-	*G.intraradices*	Bodker *et al.*, 1998
Solanum melongena	*Verticillium dahliae*	*G.mosseae*	Karagiannidis *et al.*, 2002
	V. albo-atrum		Melo *et al.*, 1985
Vicia faba	*F. oxysporum*	*G.macrocarpum* *G.fasciculatum*	Singh *et al.*, 1987

table 2 contd....		*G.mosseae*	
		Gigospora calospora	
Vigna unguiculata	*Macrophomina phaseolina*	*G.etunicatum*	Ramraj *et al.,*1988
V. radiata	*Rhizoctonia solani*	*G.coronatum*	Kasiamdari *et al.*, 2002
Lettuce	*Olpidium brassicae*		Schonbeck and Dehne, 1981
Potato	*Rhizoctonia solani*	*G.etunictum* *G.intraradices*	Yao *et al*., 2002
Viruses			
Tomato	Tobacco Mosaic virus	*Endogone*	Schonbeck and Spengler, 1978 Daft and Okasanya, 1973
Tomato	Potato virus X	*Endogone*	Daft and Okasanya, 1973
Bacterial pathogens			
Tomato	*Pseudomonas syringae*	*G.mosseae*	Garcia-Garrido and Ocampo, 1989a,b
Nematodes			
Allium cepa	*Meloidogyne hapla* *M. incognita*	*G.etunicatum*	Verdejo *et al.*, 1990
Bush bean	*Rotylenchulus reniformis*	*G.mosseae*	Sitaramaiah and Sikora, 1981
Cucumis melo	*M. incognita*		Heald, *et al.*, 1989
Cucumber	*Rotylenchuslus reniformis*	*G. mosseae*	Sitaramaiah and Sikora, 1981
Daucus carota	*M. hapla*	*G. mosseae*	Sikora and Schonbeck, 1975
Tomato	*M.incognita*	*G.fasciculatum*	Bagyaraj *et al.*, 1979 Nagesh *et al*., 1999
	M. incognita	*G. mosseae*	Sikora and Schonbeck, 1975
	M. incognita	*Gigaspora margarita*	Sikora and Schonbeck, 1975
	M.javanica	*G.mosseae*	Al-Raddad, 1995
	Rotylenchulus reniformis	*G.fasciculatum*	Sitaramaiah and Sikora,1982
	M.hapla	*Gigospora margarita*	Cooper and Grandison, 1986
Medicago sativa	*M. hapla*		Grandison and Cooper, 1986
Phaseolus vulgaris	*M.incognita*	*Glomus* sp.	Osman *et al.,* 1990
	M.javanica	*G.etunicatum*	Oliveira and Zambolim, 1986
Vigna unguiculata	*Heterodera cajani*	*G.fasciculatum*	Jain and Sethi, 1988a, b

table 2 contd....

	G.epigaeus	
Meloidogyne	G.fasciculatum	Jain and Sethi,
incognita	G.epigaeus	1988 a,b

was conducive to replant disease over 6 years. The application of combination of chemical, biological agents significantly reduced infection by *Phytophthora cactorum* and *Pythium ultimum*. The results suggest the potential use of *Bacillus subtilis*, *Enterobacter aerogenes* and *G. intraradices* to increase growth and fruit production of apple trees planted in soil conducive to replant disease. (Utkhede and Smith, 2000). Bodker *et al.,* (1998) studied the effects of inorganic phosphate levels and presence of AM on disease severity of *Aphanomyces euteiches* in pea roots. AMF *G. intraradices* reduced disease development in peas. The epicotyl of mycorrhizal plants showed reduction in disease severity although this part of the plant was not mycorrhizal. They concluded that an induced systemic factor may be responsible for increased resistance in mycorrhizal plants. In micropropagated potato plantlets var Gold rush, inoculation with *G. etunicatum* led to significant reduction in disease severity caused by *Rhizoctonia solani*, ranging between 60.2% and 71.2% on both shoot and crown. Compared with control plantlets, inoculation with *G. etunicatum* and *G. intraradices* reduced significantly the mortality rate by 77% and 26% respectively (Yao *et al.,* 2002). Although most of the studies have indicated reduced disease severity and less damage to the mycorrhizal plants when the plants are infected by fungal pathogens, yet few reports have shown increased disease incidence in mycorrhizal plants. In Avocado, little or no resistance to *Phytophthora parasitica* was reported in plants previously colonized by *G. fasciculatum* and more damage was done to mycorrhizal seedlings (Davis *et al.,*1978). Vestberg *et al.,* (1994) studied the effect of arbuscular mycorrhizas (*G. mosseae, G. fistulosum*) on crown rot caused by *Phytophthora cactorum* in micropropagated strawberry plants. They reported that inoculation with AMF did not decrease severity of crown rot disease in strawberry. In garlic the inoculation of plots with *G. intraradices* before planting for three consecutive years was not effective for control of white rot caused by *Sclerotium cepivorum* (Prados-Ligero *et al.,* 2002). In micropropagated potato cultivar LP89221, inoculation with *G. etunicatum* did not result in reduction of disease severity caused by *Rhizoctonia solani* and AM fungi did not significantly influence the mortality rate (Yao *et al.*, 2002).

2.2 AM fungi and foliar diseases

AM fungi present in roots of host plants alter disease resistance in the shoot and leaves. The non-mycorrhizal plants growing in the same conditions show more susceptibility towards leaf disease pathogens. Reduced degree of infection by *Erysiphe cichoracearum* in mycorrhizal cucumber plants was reported as compared to non-mycorrhizal plants (Schonbeck and Dehne, 1981).

2.3 AM and bacterial pathogens

The effects of AM fungi on pathogenic bacteria are poorly documented. Little work has

been done on bacterial pathogens in relation to mycorrhizal resistance. Experimental results show that the damage due to *Pseudomonas syringae* on tomato is significantly reduced when the plants were endomycorrhizal (Garcia-Garrido and Ocampo, 1989b). Protection of tomato plants against *Erwinia carotovora* and *Pseudomonas syringae* resulted in reduction in number of colony forming units of the pathogen in the rhizosphere of mycorrhizal plants inoculated with *Glomus mosseae* (Garcia-Garrido and Ocampo, 1989a).

2.4 AM fungi and virus pathogens

In contrast to the diseases caused by soil borne fungal pathogens and nematodes, that is reduced in mycorrhizal plants, the mycorrhizal plants show increased severity of virus diseases (Nemec and Myhre, 1984). AM fungi colonize the root system of host plants, so any interaction between mycorrhizal fungi and plant pathogen is expected to occur in roots. Viral diseases spread systemically throughout the whole plant. Reports have indicated that in all cases whether the viral infection was local or systemic, mycorrhizal host plants always showed increased disease intensity (Schonbeck and Schinzer, 1972, Green and Deng, 1985). Virus multiplication and even the sensitivity to viral contamination is increased in mycorrhizal plants. The vigorously growing plants are more susceptible to viral disease. The increased sensitivity and higher reproduction of a virus infected mycorrhizal plants may partly be due the improved nutrients status in mycorrhizal plants.

The experiments with split root system have shown that the increase in viral disease is not only due to better nutrition, but mycorrhizal roots of the same plants had higher TMV concentration than non-mycorrhizal roots. Immunofluorescent techniques indicated that the increased virus concentration in mycorrhizal host roots is mostly limited to arbuscular stage of the endophyte (Dehne, 1982, Schonbeck and Spengler, 1978). Electron microscopy showed that TMV was restricted to the host cell cytoplasm, but was more numerous in cells that contain arbuscules (Jabaji-Hare and Stobbs, 1984). This indicates that the high metabolic activity in the host cytoplasm of cell with arbuscules are favourable for the accumulation of virus particles. The increase exchange of substances from the fungus into the host cells and *vice versa* can be characterized by high phosphate metabolism and high concentration of nucleic acids and proteins (Schonbeck and Dehne, 1981).

The virus infection and multiplication may benefit from this high physiological potential in the root tissue. The increased synthesis of nucleic acids and proteins may promote virus multiplication and thus be responsible for a better spread over the whole plant. Leaves of mycorrhizal tobacco plants infected with TMV showed higher incidence and severity of necrotic lesions than those of non-mycorrhizal control (Shaul *et al.,* 1999). Bondoux and Perrin (1982) have reviewed effects of mycorrhiza on viruses, their mode of action, problems in using mycorrhizae against plant parasites, equilibrium of micro-organism in the host and use of mycorrhizae in the control of soil borne disease. The protective effects induced by AM Fungi against yellows disease caused by Phytoplasma of the Stolbur group have been investigated in tomato by morphometry and flow cytometry. Symptoms induced by the phytoplasma were less severe when

the plants also harboured AM fungi. In AM plants infected with phytoplasmas, reduced nuclear senescence was observed (Lingua *et al.,* 2002).

2.5 Diseases caused by soil borne nematodes

Both AM fungi and nematodes occur together in the roots and rhizosphere of the same plant, each having a characteristic but opposite effect on plant vigor. AM fungi may stimulate plant growth whereas plant parasitic nematodes usually suppress plant growth. AM fungi influence the parasitization of plants by nematodes. The penetration rate of parasitic nematodes into the roots of mycorrhizal plants can be decreased, reproductive rate is lowered, their development inside the root may be retarded or the degree of damage caused by the nematode may be lowered.

Most of the studies on interactions of nematode- AM fungi have been carried out in fruits like citrus (O'Bannon and Nemec, 1979, O'Bannon *et al.,* 1979), grapes (Atilano *et al.,* 1976, 1981) banana (Jaizme-Vega *et al.,*1997), vegetables like tomato (Sikora and Schonbeck, 1975). O'Bannon and Nemec (1979) found reduction in mycelium growth and vesicle formation by *Glomus etunicatus* in nematode –infected citrus roots. Reduced development of root knot nematodes or reduced formation of root galls in different plants like tomato preinoculated with *G. mosseae, G. fasciculatum,* and *G. macrocarpus* has been reported (Sikora and Schonbeck, 1975, Bagyaraj *et al.,*1979). Mycorrhizal fungi are only found near galls, not inside them. Studies have suggested that there is a competitive interaction between AM fungi and nematode. There is competition for space and food supply in the root system. Nematode-AM interactions can vary with the host cultivar, species of mycorrhizal fungi, nematode inoculum densities, susceptibility of host cultivar to nematode and soil fertility. Greater populations of root knot larvae have been reported from mycorrhizal plants than from non-mycorrhizal plants(Atilano *et al.,* 1976). Interactions between AM fungi, nematodes and plant roots appear complex and vary with each combination (Schenck and Kellam, 1978).

In studies where parasitic nematodes that reduce plant growth have been used, it has been found that plants inoculated with both nematodes and AM fungi have intermediate yields between those inoculated with either microorganism alone, indicating that the beneficial effect of AM fungi does not completely compensate for the damage caused by the nematodes (Atilano *et al.,* 1976, O'Bannon and Nemec1979, O'Bannon *et al.,*1979).

In grapes interaction between *Meloidogyne arenaria* and *G. fasciculatum* results in the stimulated growth of host plants in dual inoculated plants (Atilano *et al.,*1976).Similar positive effect were also reported in tomato(Sikora and Schonbeck, 1975). Sequence of inoculation of the parasite and symbiont also influence the net result of interaction. Preinoculation of tomato, carrot with *G. mosseae* before *M. incognita* resulted in fewer larvae penetrating and developing in roots of mycorrhizal plants as compared to non-mycorrhizal plants (Hussey and Roncadori, 1982).

O'Bannon *et al.,*1979 found that endoparasite *Tylenchulus semipenetrans* was less pathogenic on rough lemon, when co-inoculated with *G. mosseae*. Cooper and Grandison (1986) studied interaction between root knot nematodes and three AM fungi in tomato and clover. Mycorrhizal plants were found to be more resistant to root knot

nematode at all phosphate levels and growth benefits were generally greater in plants precolonized by mycorrhizal fungi. Mycorrhizal infection thus increased the plants resistance to infection by *Meloidogyne hapla.* Bagyaraj *et al.,* (1979) also advocated the use of AM fungi in controlling *M. incognita* on tomato. Green house testing to evaluate the influence of *Glomus mosseae* on penetration and development of *Rotylenchulus reniformis* on bush bean, cucumber and musk melon showed that reduction in the number of nematodes that penetrated mycorrhizal bush bean was 35 and 41 % lower than the control at 4 and 8 days respectively after nematode inoculation (Sitaramaiah and Sikora, 1981). A similar reduction in larval penetration was observed on mycorrhizal cucumber and muskmelon 8 days after nematode inoculation.

These studies have shown that the AM fungus, *G. mosseae* increases plant resistance to nematode infection. Pre-inoculation allowed the growing fungal symbiont to establish itself in the roots before exposure to *R. reniformis.* Studies have also shown that inoculation of tomato transplants or seed beds with the fungal endophytes appreciably reduced nematode juvenile penetration and development on mycorrhizal plants compared with control plants (Sitaramaiah and Sikora, 1982). The development of gelatinous matrix was delayed and fewer eggs / egg sac were produced on plants with mycorrhiza. It has been demonstrated that several phases of life *of R. reniformis* are affected adversely by *G. fasciculatum.* Cooper and Grandison (1986) showed that mycorrhizal plants were more resistant to root knot nematode (*M. hapla*) at all phosphate levels and growth benefit were generally more significant in plants pre-infected with mycorrhizal fungi.

In rough lemon, growth of mycorrhizal seedling was significantly greater than the growth of non-mycorrhizal seedlings inoculated with citrus burrowing nematode (*Radopholus citrophilus*). When seedlings were inoculated with both nematode and AM fungus, *G. etunicatum*, suppression of seedling growth caused by nematode was alleviated by *G. etunicatum* in inoculated seedlings as compared to non-mycorrhizal plants (O'Bannon and Nemec, 1979).

Inoculation with two isolates of *G. mosseae* increased the growth of banana cultivar, Musa AAA plants in comparison to non-mycorrhizal plants. *G. mosseae* increased the growth of banana plants by enhancing the plant nutrient uptake (N,P,K, Ca and Mg) and suppressing *M. incognita* reproduction and gall formation during early stages of plant development (Jaizme-Vega *et al.,* 1997). Experiments were carried out by Nagesh *et al.,* (1999) in pot conditions and in nursery beds to study the correlation between *G. fasciculatum,* spore density and root colonization and *M. incognita* infection on tomato. They reported that nematode population, egg masses / plant and root gall index were negatively correlated with spore density of AM fungi.

3. Mechanism of interaction between AM fungi and plant pathogens

Several hypotheses have been put forth to explain the mechanism involved in reduction of disease severity from pathogenic infections by mycorrhizal fungi (Linderman, 2000). Depending on the disease and the environmental conditions, any or all mechanism may be involved.

3.1 Changes in morphology of host root

Plants colonized by AM fungi develop extensive root system with higher number of lateral roots as compared to non-mycorrhizal plants (Berta *et al.,* 1993). Mycorrhizal infection enhances lignification in root tissues, particularly in stelar region which may be responsible for restricting soil borne pathogens that invade the host roots. Lignification is responsible to prevent penetration of mycorrhizal plants by *Fusarium oxysporum* (Dehne and Schonbeck, 1978). Mycorrhizal plants develop strong vascular system which increases nutrient flow, provides greater mechanical strength and reduces the detrimental effect of vascular pathogens (Hussey and Roncadori, 1982).

3.2 Physiological changes in host root

The relationship between host-pathogen is influenced through modification in basic physiology of host roots. There is increase uptake of water and nutrients by mycorrhizal fungi which affects pathogenesis. Through increased P nutrition, AM fungi enhance root growth, increase the capacity for absorption and affect cellular process in roots (Smith and Gianinazzi-Pearson, 1988). Such changes may explain increased tolerance of mycorrhizal plants towards pathogens. Mycorrhizal roots show increased respiration than non AM roots (Dehne, 1982). Increased respiration rate of AM roots indicates higher metabolic activity which might enable mycorrhizal plants to be more resistant against root pathogens. Increased ethylene and DNA methylation have also been reported in AM roots (Dugassa *et al.,* 1996). Similarly the arginine level in mycorrhizal roots was reported to be six fold more due to an increased specific activity of enzymes in ornithine cycle and blocking of cycle by arginase enzyme system in such roots (Dehne *et al.,* 1978). Mycorrhizal development enhances the production of isoflavonoids which play major role in natural defense of plants (Morandi, 1996).

3.3 Changes in root exudates

Root exudation pattern also changes in quality as well as quantity with mycorrhizal colonization which might play an import role in pathogenesis (Graham *et al.,* 1981). Root exudates directly affect the pathogen by inducing their germination or by inhibiting saprophytic and pathogenic activity. The pathogens are affected directly by competition and antibiosis by the root microflora whose activities are also mediated by root exudates. It has been proposed that AM symbiosis is regulated by the amount of organic nutrients (amino acids, reducing sugars) leaked from the roots (Graham *et al.,* 1981). Root exudation is maximum in zone of elongation, the portion of root growth that is most susceptible to AM fungal colonization (Buwalda *et al.,* 1984).

3.4 Enhanced nutritional status of host plants

Greater tolerance of mycorrhizal plants towards the pathogens is attributed to increased phosphate status and more vigorous growth of plants (Azcon-Aguilar and Barea, 1996). In addition to phosphates, AM fungi increase uptake of Ca^{+2}, Cu^{+2}, SO_4^{-2} and Zn^{+2}

(Smith and Gianinazzi-Pearson, 1988). Host susceptibility to infection and tolerance to disease is influenced by the nutritional status of the host and fertility level of the soil *e.g.* nematode infected plants are frequently deficient in B, N, Fe, Mg and Zn (Good, 1968). When plants are non mycorrhizal, P can combine with minor elements to create deficiency which would predispose plants to root knot nematodes (Smith *et al.*, 1986). However some reports indicates that improved P nutrition results in increased disease severity (West, 1995) or has no affect on disease control (Caron *et al.*, 1986b). Addition of phosphorus fertilizer did not result in duplication of benefits obtained by mycorrhizal inoculation against pathogens in tomarilla that indicates it is not due to only improved P nutrition of the host but other mechanisms are also involved (Cooper and Grandison, 1987).

3.5 Competition for space and host photosynthates

Mycorrhizal fungi, soil fungal pathogens and plant parasitic nematodes, all occupy the same root tissue, resulting in direct competition for space which might explain the disease resistance in mycorrhizal plants (Azcon-Aguilar and Barea, 1996). AM fungus colonization in the root would limit the development of pathogenic fungi to areas of root which had not been colonized, thus providing some protection (Goncalves *et al.*, 1991). Cordier *et al.*, (1996) reported that *Phytophthora* does not penetrate arbuscule-containing cells and its development is also reduced in adjacent non colonized region. However, this hypothesis does not get much support from other workers (Linderman, 1994).

Both mycorrhizal fungus and pathogen are dependent on host photosynthates for their growth. The major substrate for microbial activity in the rhizosphere or on rhizoplane is organic C released by host roots (Azaizeh *et al.*, 1995). Mycorrhizal fungi and root pathogens compete for the C compounds reaching the root (Smith, 1987). When mycorrhizal fungus has primary access to photosynthates, the higher C demand may inhibit pathogen growth (Azcon_Aguilar and Barea, 1996).

3.6 Biological interactions in the mycorrhizosphere

The concept of mycorrhizosphere is based on the fact that mycorrhizae exert the strong influence on the microflora in the rhizosphere (Bansal *et al.*, 2000). This results in microbial shift in the mycorrhizosphere which could influence the health of plants (Whipps, 2001, Barea *et al.*, 2002). The possible mechanisms involved in interaction of AM fungi and other rhizosphere micro-organisms include physical mechanism, changes in quality and quantity of root-exudates caused by AM colonization (Bansal and Mukerji, 1994), direct interaction between hyphal network of AM fungi and rhizosphere microbes (St-Arnaud *et al.*, 1995). Changes in soil microbial community brought by mycorrhizal colonization may lead to stimulation of those micro-organism which may be antagonistic to root pathogens (Azcon-Aguilar and Barea, 1996). The number and proportion of bacterial antagonists against several root pathogens increase when AM association is established. More actinomycetes antagonistic to *Fusarium solani* and *Pseudomonas solanacearum* were isolated from the rhizosphere pot cultures of *G.*

fasciculatum colonized plants than from non-mycorrhizal control (Secilia and Bagyaraj, 1987). Reduction in *Fusarium* population in soil surrounding mycorrhizal tomato roots was observed as compared with soil of non-mycorrhizal control (Caron *et al.*, 1986b). Meyer and Linderman (1986) observed that sporangium and zoospore production by root pathogen, *Phytophthora cinnamomi* was reduced in the presence of rhizosphere leachates from mycorrhizal sweet corn and *Chrysanthemum.*

3.7 Enzyme production

The symbiotic host-fungus relationship is characterized by the formation of haustoria-like intracellular arbuscules which are successively degraded. This process represents digestion of the fungus by the host. For this degradation to occur, the fungal cell wall of the endophyte has to be attacked by the host. Therefore the roots colonized by mycorrhizal fungus show high chitinolytic activity. These enzymes can be effective against other fungal pathogens as well. High chitinase activity in the mycorrhizal root tissues may also restrict the growth and progression of root pathogens in host tissues. Differential expression of genes encoding acid and basic chitinases have been reported during mycorrhizal formation (Gianinazzi-Pearson *et al.,* 1996). Dumas-Gaudot *et al.,* (1996) reported induction of new acidic chitinase isoforms in tobacco roots by AM fungi.

Increased activity of peroxidase associated with epidermal and hypodermal cells has also been reported in mycorrhizal roots (Gianinazzi and Gianinazzi-Pearson, 1992). Induction of pathogenesis related (PR) proteins has been reported in plants during resistance to pathogen infection (Stintzi *et al.,* 1993, Shaul *et al.,* 1999). These proteins have been grouped into eleven families designated PR-1 to PR- 11 (VanLoon *et al.,* 1994). PR –2, PR-3 and PR –11 protein are hydrolytic enzymes with β-1,3 glucanase or chitinase activities (Stintzi *et al.*, 1993, VanLoon *et al.,* 1994, Gianninazzi-Pearson *et al.,* 1996). Shaul *et al.,* (1999) based on their work on tobacco strongly support the existence of regulatory processes initiated in the roots of mycorrhizal plants that modify disease symptom development and gene expression in their leaves.

3.8 Molecular mechanisms

The response of plants to AM fungi involves temporal and spatial activation of different defense mechanisms (Garcia-Garrido and Ocampo, 2002), however, how these defenses affect the functioning and development of AM remains unclear. A number of regulatory mechanisms of plant defence response have been described during establishment of AM symbiosis. These include (i). Elicitor degradation, (ii). Modulation of second messenger concentration,(iii). Nutritional and hormonal plant defense regulation (iv). Activation of regulatory symbiotic gene expression. Colonization of roots by AM fungi brings about biochemical changes within host tissues. These biochemical changes include:
(i). Stimulation of the phenylpropanoid pathway (Harison and Dixon, 1993),
(ii). Changes in levels of aliphatic polyamines (El Ghachtouli *et al.,* 1995),
(iii). Activation of defense related genes (Harison and Dixon, 1993, Franken and

Gnadinger, 1994, Dumas-Gaudot *et al.,* 2000),

(iv). Enhancement of certain hydrolytic enzyme activities (Dumas-Gaudot *et al.,* 1996),

(v). synthesis of protein of unknown function (Dumas-Gaudot *et al.,*, 2000, Samara *et al.,* 1996),

(vi). Elicitation of plant chitinase and B-1,3 glucanase (Blee and Anderson, 1996) which are antifungal against pathogenic soil and root borne fungi. In a study conducted by Pozo *et al.,* (2002), the ability of two AM fungi (*G. mosseae* and *G. intraradices*) to induce local or systemic resistance to *Phytophthora parasitica* in tomato roots was compared using a split root experimental system. *G. mosseae* was effective in reducing disease symptoms produced by *P.parasitica* infection. The biochemical analysis of different plant defense- related enzymes showed a local induction of mycorrhiza related new isoforms of hydrolytic enzymes chitinase, chitosanase and B-1,3 glucanase as well as super oxide dismutase, an enzyme involved in cell protection against oxidative stress,

(vii). Induction of chitinase isoforms by mycorrhiza : These chitinases release oligosaccharide elicitors from the chitinous AM fungal cell walls which in turn stimulates the general defense responses of plants (Dumas-Gaudot *et al.,* 1996, Cordier *et al.,* 1996),

(viii). Increase ethylene production and DNA methylation by AM roots. A higher demethylation can be related to gene expression for higher resistance of plants against pathogens (Dugassa *et al.,* 1996),

(ix). Expression of chitinases, B1,3 glucanases and genes involved in the biosynthesis of isoflavanoid phytoalexins have been detected in symbiotic AM associations (Dixon *et al.,* 1993). Phytoalexins are toxic compounds released at the site of infection. Isoflavonoids, viz. glyceollin which is nematostatic and caumestrol which is fungitoxic together with coumestan isosojagol, showed greater amount of accumulation in mycorrhizal roots than in non-mycorrhizal roots of soybean (Morandi and Le Querre, 1991). The level of phytoalexin medicarpin, coumestrol, daidzein, medicarpin-malonyl glucoside, formononetin, showed transient increase in roots of *Medicago truncatula* 7 – 40 days after inoculation with *Glomus* species, thereby showing that phytoalexin and their precursors are activated by AM inoculation (Harison and Dixon, 1993),

(x). Phenolics may also play a role in protecting AM roots against pathogenic fungi (Grandmaison *et al.,* 1993). Both oxidation and polymerisation of tomato roots phenols have been reported to increase following AM association (Dehne and Schonbeck, 1979). A continuous increase of total soluble phenols has been found in mycorrhizal roots of peanuts (Krishna and Bagyaraj, 1984). Ultrastructural and immunocytochemical studies suggest that enhanced protection of mycorrhizal Ri T-DNA transformed carrot roots against infection by *Fusarium oxysporum* f.sp. *chrysanthemi* involves accumulation of electron dense deposits of phenolics and hydrolytic enzymes which restricts pathogen progression (Benhamou *et al.,* 1994). These depositions were not seen in *Fusarium* infected carrot roots that were non-mycorrhizal. These substance are reported to cause strong morphological and cytochemcial deformations of the pathogen,

(xi). AM fungi can activate a part of plant metabolic pathways associated with defense process, but they do so in an uncoordinated weak, transient and very localized fashion (Guillon *et al.,* 2002). The cellular and molecular responses induced in myc[-1]mutants triggered by AM fungi confirm that the latter produced elicitors of defense related

mechanism, and indicate that their expression is some how controlled by specific plant gene during normal symbiotic interactions so that resistance reactions do not occur in outer root cells (Gianinazzi-Pearson *et al.,* 1996).

4. Factors influencing the potential of mycorrhizae in disease management

The potential ability of mycorrhizae in disease management is governed by a number of biotic and abiotic factors like any other bio-control agent. It is utmost essential to provide the congenial conditions for growth and development of the specific mycorrhizal fungus recommended for management of plant disease. Among the abiotic factors, soil temperature, soil moisture and phosphorus content are the major factors to be monitored closely for the interaction of mycorrhizae and pathogen(s) to get the desirable results. Among the biotic factors, host and mycorrhizal fungus specificity, type of pathogen(s) and its virulence, interaction of other soil microflora, nutrient availability and space in rhizosphere, rate of colonization, level of inoculum and time of inoculation of mycorrhizal fungus play important role and become decisive for successful management of the disease. These aspects have been reviewed elsewhere (Linderman, 2000, Jalali and Jalali, 1991, Singh *et al.,* 2000).

5. Future thrust

Significant advances have been made in last two decades to understand the potential of mycorrhizal fungi in suppression of soil borne pathogens and thereby imparting resistance to plant disease in wide range of fruits and vegetable host plants. However, increased resistance or decreased susceptibility requires optimum conditions for the development of the symbiosis before the attack of the pathogen. Successful suppression of the pathogen depends on mode of parasitism, virulence of pathogen and on potential of particular AM fungi to induce resistance. Suitable host-fungus combination has to be found for improving the plant growth and inducing resistance to pathogen in given environmental conditions.

The studies on interaction between AM fungi and soil borne pathogens has no doubt proved potential of AM fungi in controlling plant pathogens. Most of the studies on role of mycorrhiza in biological control have been carried out under controlled glass house conditions. Although there are some promising results that support the conclusions of green house studies but so far examples of successful practical application in field are few. We must ensure that potentially efficient strains of mycorrhizal symbionts establish and survive in phase of competition from less efficient and naturally occurring symbionts, as well as antagonism for resident root pathogens and other rhizosphere microorganisms if we are to exploit the potential of mycorrhizal technology as biocontrol tool. AM fungi being obligate symbionts can't be grown on synthetic culture media. Several attempts have been made during last three decades to obtain mycorrhizal symbiosis in vitro. The use of root organs cultures has proved particularly useful (Fortin *et al.*, 2002). In order to exploit the mycorrhizal fungi, tech-

niques for commercial production and application of AM fungi in field must be developed and standardized. There is need to transfer mycorrhizal technology from lab to land and to find out whether mycorrhiza can work as an effective plant protection system in the field.

6. References

Abdalla, M.E. and Abdel-Fattah, G.M. 2000. Influence of the endomycorrhizal fungus *Glomus mosseae* on the development of peanut pod rot disease in Egypt. Mycorrhiza, 10: 29-35.

Abdel-Fattah, G.M. and Shabana, Y.M. 2002. Efficacy of the arbuscular mycorrhizal fungus *Glomus clarum* in protection of cowpea plants against root rot pathogen *Rhizoctonia solani*. Zeitschrift fur pflanzenkrankheiten und pflanzenschutz-Journal of Plant Diseases and Protection, 109: 207-215.

Allen, M.F. 1991. The ecology of mycorrhizae. Cambridge University Press, Cambridge, 184p.

Al-Momany, A. and Al-Raddad, A. 1988. Effect of vesicular-arbuscular mycorrhizae on *Fusarium* wilt of tomato and pepper. Alexandria Journal of Agricultural Research, 33: 249-261.

Al-Raddad, A.M. 1995. Interaction of *Glomus mosseae* and *Paecilomyces lilacinus* on *Meloidogyne javanica* of tomato. Mycorrhiza, 5: 233-236.

Atilano, R.A., Menge, J. A. and Van Gundy, S. 1981. Interaction between *Meloidogyne arenaria* and *Glumus fasciculatum* in grapes. Journal of Nematology, 13: 52-57.

Atilano, R.A., Rich, J.R., Ferris, H. and Menge, J.A. 1976. Effect of *Meloidogyne arenaria* on endomycorrhizal grape (*Vitis vinifera*) rootings. J. Nematol. 8: 278.

Azaizeh, A. Marschner, H., Romheld, V. and Wittenmayer, L. 1995. Effect of a vesicular-arbuscular mycorrhizal fungus and other soil microorganisms on growth, mineral nutrient acquisition and root exudation of soil-grown maize plants. Mycorrhiza, 5: 321-327.

Azcon-Aguilar, C. and Barea, J.M. 1996. Arbuscular mycorrhizas and biological control of soil-borne plant pathogens- an overview of mechanisms involved. Mycorrhiza, 6: 457-464.

Baath, E. and Hayman, D.S. 1983. Plant growth responses to vesicular-arbuscular mycorrhiza XIV. Interactions with *Verticillium* wilt on tomato plants. New Phytol. 95: 419-426.

Baath, E. and Hayman, D.S. 1984. No effect of vesicular-arbuscular mycorrhiza on red core disease of strawberry. Transactions of British Mycological Society, 82:532-536.

Baghel, P.P.S., Bhatti, D.S. and Jalali, B.L.1990. Interaction of VA mycorrhizal fungus and *Tylenchulus semipenetrans* on citrus. In:"Current trends in Mycorrhizal Research"(eds. Jalali, B.L. and Chand, H.), Proc. Natl. Conf. On Mycorrhiza, Haryana Agricultural University, Hissar, India, TERI, New Delhi, pp. 118-119.

Bagyaraj, D.J. 1992. Vesicular-arbuscular mycorrhiza : Application in Agriculture. Methods in Microbiol. 24: 360-373.

Bagyaraj, D.J., Manjunath, A. and Reddy, D.D.R. 1979. Interaction of vesicular-arbuscular mycorrhiza with root knot nematode in tomato. Plant Soil, 51:397-403.

Bansal, M. and Mukerji, K.G. 1994. Positive correlation between VAM-induces changes in root exudation and mycorrhizosphere mycoflora. Mycorrhiza, 5:39-44.

Bansal, M., Chamola, B.P., Sarwar, N. and Mukerji, K.G. 2000. Mycorrhizosphere: Interactions between rhizosphere microflora and VAM fungi. In: "Mycorrhizal Biology"(eds.Mukerji, K.G., Chamola, B.P. and Jagjit Singh), Kluwer Academic Publishers, NY, pp. 143-152.

Barea, J.M., Azcon, R. and Azcon-Aguilar, C. 2002. Mycorrhizosphere interactions to improve plant fitness and soil quality. Antonie van Leeuwenhoek International Journal of General and Molecular Microbiology, 81: 343-351.

Becker, W.N. 1976. Quantification of onion vesicular-arbuscular mycorrhizae and their resistance to *Pyrenochaeta terrestris*, Ph.D. thesis, University of Illinois, Urbana, pp.72.

Benhamou, N., Fortin, J.A., Hamel, C., St-Arnaud, M. and Shatilla, A. 1994. Resistance responses of mycorrhizal Ri T-DNA-transformed carrot roots to infection by *Fusarium oxysporum* f.sp. *chrysanthemi*. Phytopathology, 84: 958-968.

Berta, G., Fusconi, A. and Trotta, A. 1993. VA mycorrhizal infection and the morphology and function of root systems. Environ. Expt. Bot., 33:159-173.

Bethlenfalvay, G.J. and Linderman, R.G.(eds.) 1992. Mycorrhizae in Sustainable Agriculture. ASA Special Publication No. 54, Madison, Wis.

Blee, K.A. and Anderson, A.J. 1996. Defence related transcript accumulation in *Phaseolus vulgaris* L. colonized by the arbuscular mycorrhizal *fungus Glomus intraradices* Schenck and Smith. Plant Physiol. 110: 675-688.

Bochow, H. and Abou-Shaar, M. 1990. On the phytosanitary effect of mycorrhiza in tomatoes to the corky-root disease. Zentralbratt fur mikrobiologie, 145: 171-176.

Bodker, L., Kjoller, R. and Rosendahl, S. 1998. Effect of phosphate and the arbuscular mycorrhizal fungus, *Glomus intraradices* on disease severity of root of peas (*Pisum sativum*) caused by *Aphanomyces euteiches*. Mycorrhiza, 8: 169-174.

Bon-doux, P. and Perrin, R. 1982. Mycorrhizas and Plant protection. Comptes, Rendus des seances de I' Academie d Agric. De France. 68:1162-1177.

Buwalda, J.G., Stribley, D.P. and Tinker, P.B. 1984. The development of endomycorrhizal root systems.V. The detailed pattern of development of infection and the control of infection level by host in young leek plants. New Phytol. 96: 411.

Camprubi, A. Piaochet, J., Calvet, C. and Estaum, V. 1993. Effects of the root-lesion nematode *Pratylenchus vulnus* and the vesicular-arbuscular mycorrhizal fungus *Glomus mosseae* on the growth of three plum rootstocks. Plant Soil, 153: 223-229.

Caron, M., Fortin, J.A. and Richard, C. 1986a. Effect of inoculation sequence on the interaction between *Glomus intraradices* and *Fusarium oxysporum* f.sp. *radices-lycopersici* in tomatoes. Canadian Journal of Plant Pathology, 8:12-16.

Caron, M., Richard, C. and Fortin, J.A. 1986b. Effect of preinfestation of the soil by a vesicular-arbuscular mycorrhizal fungus, *Glomus intraradices*, on *Fusarium* crown and root rot of tomatoes. Phytoprotec. 67:15-19.

Cooper, K.M. and Grandison, G.S. 1986. Interaction of vesicular-arbuscular mycorrhizal fungi and root knot nematode on cultivar of tomato and white clover susceptible to *Meloidogyne hapla*. Ann. app. Biol. 108: 555-565.

Cooper, K.M. and Grandison, G.S. 1987. Effects of vesicular-arbuscular mycorrhizal fungi on infection of tomarillo (*Cyphomandra betacea*) by *Meloidogyne incognita* in fumigated soil. Plant Dis. 71: 1101-1106.

Cordier, C., Gianinazzi, S. and Gianinazzi-Pearson, V. 1996. Colonization patterns of root tissues by *Phytophthora nicotianae* var. *parasitica* related to reduced disease in mycorrhizal tomato. Plant Soil, 185: 223-232.

Daft, M.J. and Okasanya, B.O. 1973. Effect of *Endogone* mycorrhiza on plant growth. V. Influence of infection on the multiplication of viruses in tomato, petunia and strawberry. New Phytol. 72: 975-983.

Davis, R.M. 1980. Influence of *Glomus fasciculatum* on *Thielaviopsis basicola* root rot of citrus. Plant Disease, 64:839-840.

Davis, R.M. and Menge, J.A. 1980. Influence of *Glomus fasciculatum* and soil phosphorus on *Phytophthora* root rot of citrus. Phytopathology, 70: 447-452.

Davis, R.M. and Menge, J.A. 1981. *Phytophthora parasitica* inoculation technique and intensity of vesicular arbuscular mycorrhizae in citrus. New Phytol. 87: 705-715.

Davis, R.M., Menge, J.A. and Zentmyer, G.A. 1978. Influence of vesicular-arbuscular mycorrhizae on *Phytophthora* root rot of three crop plants. Phytopathol. 68: 1614-1617.

Dehne, H.W. 1982. Interaction between vesicular-arbuscular mycorrhizal fungi and plant patho-

gens. Phytopathology, 72: 1115-1119.

Dehne, H.W. and Schonbeck, F. 1978. The influence of endotrophic mycorrhiza on plant diseases, 3, Chitinase- activity and ornithine-cycle. J. Plant Dis. Protec. 85:666-678.

Dehne, H.W. and Schonbeck, F. 1979. The influence of endotrophic mycorrhiza on *Fusarium* wilt of tomato. Z.Pfkrankh.Schutz. 82: 630-632.

Dehne, H.W., Schonbeck, F. and Baltruschat, H. 1978. The influence of endotrophic mycorrhiza on plant diseases. 3,Chitinase- activity and the ornithine-cycle. Z.Pflanzenkrankh. pflanzenschutz, 85:666-678.

Dixon, R.A., Dey, P.M. and Lamb, C.J. 1993. Phytoalexin: Enzymology and molecular biology. Adv. Enymol. 55: 1-136.

Dumas-Gaudot, E., Gollotte, A., Cordier, C., Gianinazzi, S. and Gianinazzi-Pearson, V. 2000. Modulation of host defence systems. In: " Arbuscular mycorrhizas : physiology and function".(eds. Kapulnick, Y. and Douds Jr. D.D.) Kluwer Academic Press, pp. 173-200.

Dumas-Gaudot, E., Slezack, S., Dassi, B., Pozo, M.J., Gianinazzi-Pearson, V. and Gianinazzi, S. 1996. Plant hydrolytic enzymes (chitinases and β-1,3-glucanases) in root reactions to pathogenic and symbiotic micro-organisms. Plant and Soil, 185: 211-221.

Dugassa, G. D., von Alten, H. and Schonbeck, F. 1996. Effect of arbuscular mycorrhiza (AM) on health of *Linum usitatissimum* L. infected by fungal pathogens. Plant Soil, 185: 173-182.

El Ghachtouli, N., Paynot, M., Morandi, D., Martin-Tanguy, J. and Gianinazzi, S. 1995. The effect of polyamines on endomycorrhizal infection of wild type *Pisum sativum* cv. Frisson (nod[+] myc[+]) and two mutants (nod[-] myc[+] and nod[-] myc[-]). Mycorrhiza, 5: 189-192.

Franken, P. and Gnadinger, F. 1994. Analysis of parsley arbuscular endomycorrhiza: infection development and mRNA levels of defence related genes. Mol. Plant-Microbe Interac. 7: 612-620.

Garcia-Garrido, J.M. and Ocampo, J.A. 1989a. Interaction between *Glomus mosseae* and *Erwinia carotovora* and its effect on the growth of tomato plants. New Phytol. 110: 551-555.

Garcia-Garrido, J.M. and Ocampo, J.A. 1989b. Effect of VA mycorrhiza infection of tomato on damage caused by *Pseudomonas syringae*. Soil Biol.Biochem. 121: 165-167.

Garcia-Garrido, J.M. and Ocampo, J.A. 2002. Regulation of the plant defence response in arbuscular mycorrhizal symbiosis. Journal of experimental Botany, 53: 1377-1386.

Gianinazzi, S. and Gianinazzi-Pearson, V. 1992. Cytological, histochemistry and immunocytochemistry as tools for studying structure and function in endomycorrhiza. In: "Technique for the study of Mycorrhiza: Methods in Microbiology" vol. 24 (eds. Norris, J.P., Read, D.J., and Varma, A.K.) Academic Press, London, pp. 109-139.

Gianinazzi-Pearson, V., Dumas-Gaudat, E., Gollote, A., Tahiri-Alaoui, A. and Gianinazzi, S. 1996. Cellular and molecular defence-related root responses to invasion by arbuscular mycorrhizal fungi. New Phytol. 133: 45-57.

Goncalves, E.J., Muchovej, J.J. and Muchovej, R.M.C. 1991. Effect of kind and method of fungicidal treatment of bean seed on infection by the VA mycorrhizal fungus *Glomus macrocarpum* and by the pathogenic fungus *Fusarium solani.* I. Fungal and plant parameters. Plant Soil, 132: 41-46.

Good, J.M. 1968. Relation of plant-parasitic nematodes to soil management practices. In: "Tropical Nematology" (eds. Smart, G.C. and Perry, V.G.), Univ. of Florida, Gainsville, pp. 113-138.

Graham, J.H., Leonard, R.T. and Menge, J.A. 1981. Membrane-mediated decrease in root exudation responsible for phosphorus inhibition of vesicular-arbuscular mycorrhizae formation. Plant Physiol. 68: 548-552.

Grandison, G.S. and Cooper, K.M. 1986. Interaction of vesicular-arbuscular mycorrhiza and cultivars of alfalfa susceptible and resistant to *Meloidogyne hapla*. J. Nematol. 18: 141-149.

Grandmaison, J., Olah, G.M., van Calsteren, M.R. and Furlan, V. 1993. Characterization and localizatioin of plant phenolics likely involved in the pathogen resistance expressed by

endomycorrhizal roots. Mycorrhiza, 3:155-164.

Green, S.K. and Deng, T.C. 1985. Effect of endomycorrhizal fungus, *Glomus mosseae* on soybean mosaic virus in soybean. Plant Protection Bulletin, Taiwan, 27:353-358.

Guillon, C., St-Arnaud, M., Hamel, C. and Jabaji-Hare, S.H. 2002. Differential and systemic alteration of defence-related gene transcript levels in mycorrhizal bean plants infected with *Rhizoctonia solani*. Canadian Journal of Botany, 80: 305-315.

Harison, M.J. and Dixon, R.A. 1993. Isoflavonoid accumulation and expression of defence gene transcripts during the establishment of vesicular-arbuscular mycorrhizal associations in roots of *Medicago trunculata*. Mole. Plant Microbe Interac. 6:643-654.

Heald, C.M., Bruton, B.D. and Davis, R.M. 1989. Influence of *Glomus intraradices* and soil phosphorus on *Meloidogyne incognita* infecting *Cucumis melo*. J. Nematol. 21: 69-73.

Hedge, S.V. and Rai, P.V. 1984. Influence of *Glomus fasciculatum* on damping off of tomato. Current Science, 53: 588-589.

Hussey, R.S. and Roncadori, R.W. 1982. Vesicular-arbuscular mycorrhiza may limit nematode activity and improve plant growth. Plant Dis. 66: 9-14.

Iqbal, S.H. and Nasim,G. 1988. IV. VA mycorrhiza as a deterrant to damping off caused by *Rhizoctonia solani* at defferent temperature regimes. Biologia Pakistan, 34: 215-221.

Iqbal, S.H., Nasim, G. and Niaz, M. 1988. II. Role of vesicular-arbuscular mycorrhiza as a deterrent to damping off caused by *Rhizoctonia solani* in *Brassica oleracea.* Biologia Pakistan, 34: 79-84.

Jain, R.K and Sethi, C.L. 1988a. Influence of endomycorrhizal fungi *Glomus fasciculatum* and *G. epigaeus* on penetration and development of *Heterodera cajani* on cowpea. Indian J. Nematology, 18: 89-93.

Jain, R.K and Sethi, C.L. 1988b. Interaction between vesicular-arbuscular mycorrhiza, *Meloidogyne incognita* and *Heterodera cajani* on cowpea as influenced by time of inoculation. Indian J. Nematology, 18: 263-268.

Jaizme-Vega, M.C., Tenoury, P., Pinochet, J. and Jaumot, M. 1997. Interaction between the root knot nematode *Meloidogyne incognita* and *Glomus mosseae* in banana. Plant and Soil, 196: 2 7-35.

Jabaji-Hare, S.H. and Stobbs, L.W. 1984. Electron-microscopic examination of tomato roots coinfected with *Glomus* sp. and tobacco mosaic virus. Phytopathology, 74:277-279.

Jalali, B.L. and Jalali, I. 1991. Mycorrhiza in plant disease control. In: " Handbook of applied Mycology" Vol. I: Soil and Plants (eds. Arora, D.K., Rai, B., Mukerji, K.G. and Knudsen G.R.) Marcel Dekker Inc. New York, pp. 131-154.

Jalali, B.L., Chhabra, M.L. and Singh, R.P. 1990. Interaction between vesicular-arbuscular mycorrhiza endophyte and *Macrophomina phaseolina* in mungbean. Indian Phytopathology, 43: 527-530.

Jayaram, J. and Kumar, D. 1995. Influence of mungbean yellow mosaic virus on mycorrhizal fungi associated with *Vigna radiata* var. PS 16. Indian Phytopathology, 48: 108-110.

Karagiannidis, N., Bletsos, F. and Stavropoulos, N. 2002. Effect of *Verticillium* wilt (*Verticillium dahliae* Kleb.) and mycorrhiza (*Glomus mosseae*) on root colonization, growth and nutrient uptake in tomato and eggplant seedlings. Scientia Horticulturae, 94: 145-156.

Kasiamdari R.S., Smith, S.E., Smith, F.A. and Scott, E.S. 2002. Influence of the mycorrhizal fungus, *Glomus coronatum* and soil phosphorus on infection and disease caused by binucleate *Rhizoctonia* and *Rhizoctonia solani* on mung bean (*Vigna radiata*). Plant and Soil, 238: 235-244.

Krishna, K.R. and Bagyaraj, D.J. 1984. Phenols in mycorrhizal roots of *Arachis hypogaea*. Experientia, 40: 85-86.

Linderman, R.G. 1994. Role of VAM fungi in biocontrol. In: "Mycorrhiza and Plant Health" (eds. Pleger, F.L. and Linderman, R.G.), APS, St, Paul, pp. 1-26.

Linderman, R.G. 2000. Effect of mycorrhizas on plant tolerance to diseases. In: " Arbuscular mycorrhizas: physiology and function". (eds. Kapulnick, Y. and Douds Jr., D.D.), Kluwer Academic Press, pp. 345-366.

Lingua, G., D'Agostino, G., Massa, N., Antosiano, M. and Berta, G. 2002. Mycorrhiza-induced differential response to a yellows disease in tomato. Mycorrhiza, 12: 191-198.

McGraw, A.C. and Schenck, N.C. 1981. Effect of two species of vesicular-arbuscular mycorrhizal fungi on the development of *Fusarium* wilt of tomato. Phytopathology, 71:894.

Melo, I.S., Costa, C.P. and Silveria, A.P.D. 1985. Effect of vesicular-arbuscular mycorrhizae on aubergine wilt caused by *Verticillium albo-atrum* Reinke and Berth. Summ. Phytopathol. 11: 173-179.

Meyer, J.R. and Linderman, R.G. 1986. Selective influence on populations of rhizosphere-rhizoplane bacteria and actinomycetes by mycorrhizas formed by *Glomus fasciculatum.* Soil Biol. Biochem. 18: 191-196.

Morandi, D. 1996. Occurrence of phytoalexins and phenolics compounds in endomycorrhizal interaction and their potential role in biological control. Plant and Soil, 185: 241-251.

Morandi, D. and Le Querre, J.L. 1991. Influence of nitrogen on accumulation of isosojagol (a newly detected coumestan in soybean) and associated isoflavonoids in roots and nodules of mycorrhizal and non-mycorrhizal soybean. New Phytol. 117: 75-79.

Mosse, B. 1973. Advances in the study of vesicular-arbuscular mycorrhiza. Ann. Rev. Phytopathol. 11: 171-196.

Mukerji, K.G. 1999. Mycorrhiza in control of plant pathogens : Molecular approaches. In: "Biotechnological approaches in biocontrol of plant pathogens". (eds. Mukerji, K.G., Chamola, B.P. and Upadhyay, R.K), Kluwer Academic & Plenum Publishers, New York, pp. 135-155.

Nagesh, M., Reddy, P.P., Kumar, M.V.V. and Nagaraju, B.M. 1999. Studies on correlation between *Glomus fasciculatum* spore density, root colonization and *Meloidogyne incognita* infection on *Lycopersicum esculentum.* Zeitschrift fur pflnzenkrankheiten und pflanzenschutz-Journal of Plant Disease and Protection, 106: 82-87.

Nemec, S. 1974. Population of *Endogone* in strawberry fields in relation to root rot infection. Trans. Br. Mycol. Soc. 62: 45.

Nemec, S. and Myhre, D. 1984. Virus-*Glomus etunicatum* interactions in Citrus rootstocks. Plant Dis. 68: 311-314.

O'Bannon, J.H. and Nemec, S. 1979. The response of *Citrus limon* seedlings to a symbiont, *Glomus etunicatum* and a pathogen, *Radopholus similis*. J. Nematology,11: 270-275.

O'Bannon, J.H., Inserra, R.N., Nemec, S. and Vovlas, N. 1979. The influence of *Glomus mosseae* on *Tylenchulus semipenetrans* infected and uninfected *Citrus limon* seedlings. J. Nematology, 11: 247-250.

Oliveira, A.A.R. and Zambolim, L. 1986. Interaction between the endomycorrhizal fungus *Glomus etunicatum* and the gall nematode *M. javanica* on beans with split roots. Fitopatologia Brasilera,12:22-225.

Osman, H.A., Korayem, A.M., Ameen, H.H. and Badr-Eldin, S.M.S. 1990. Interaction of root-knot nematode and mycorrhizal fungi on common bean *Phaseolus vulgaris* L. Anzeiger fur Schadlingskunde, Pflanzenschutz, Umweltschutz. 63: 129-131.

Pinochet, J., Calvet, C., Campprubi, A. and Fernandez, C. 1996. Interaction between migratory endoparasitic nematodes and arbuscular mycorrhizal fungi in perennial crops. A review. Plant Soil, 185:183-190.

Pozo, M.J., Cordier, C. and Dumas-Gaudot, E. 2002. Localized versus systemic effect of arbuscular mycorrhizal fungi on defence responses to *Phytophthora* infection in tomato plants. Journal of Experimental Botany, 53: 525-534.

Prados-Ligero, A.M., Bascon-Fernandez, J., Calvet-Pios, C., Corpas-Hervias, C., Ruiz, A.L., Melero-Vara, J.M. and Basallote Ureba, M.J. 2002. Effect of different soil and clove treat-

ments in the control of white rot of garlic. Annals of Applied Biology, 140: 247-253.

Ramirez, B.N. 1974. Influence of endomycorrhizae on the relationship of inoculum density of *Phytophthora palmivora* in soil to infection of papaya roots. MS thesis, Univ. Florida, Gainesville, 45p.

Ramraj, B., Shanmugam, N. and Reddy, D.A. 1988. Biocontrol of *Macrophomina* root rot of cowpea and *Fusarium* wilt of tomato by VAM fungi. Mycorrhizae for Green Asia, Proc. First Asian Conf. On Mycorrhiza, Jan., 29-31,pp. 250-251.

Rosendahl, S. 1985. Interaction between the vesicular-arbuscular mycorrhizal fungus *Glomus fasciculatum* and *Aphanomyces euteiches* root rot of peas (*Pisum sativum*). Phytopathol. 114: 31-40.

Rosendahl, C.N. and Rosendahl, S. 1990. The role of vesicular-arbuscular mycorrhiza in controlling damping off and growth reduction in cucumber caused by Pythium ultimum. Symbiosis, 9:363-366.

Ross, J.P. 1972. Influence of Endogone mycorrhiza on *Phytophthora* rot of soybean. Phytopathol. 62:876-897.

Safir, G. 1968. The influence of vesicular-arbuscular mycorrhiza on the resistance of onion to *Pyrenochaeta terrestris*. M.S. thesis, Univ. Illiinois, Urbana, 36p.

Samara, A., Dumas-Gaudot, E., Gianinazzi-Pearson, V. and Gianinazzi, S. 1996. Studies of *in vivo* polypeptide synthesis in non-mycorrhizal and arbuscular-mycorrrhizal (*Glomus mosseae*) pea roots. In: " Mycorrhizas in integrated systems from genes to plant development". (eds. Azcon-Aguilar, C. and Barea, J.M.), Kluwer Academic Publishers, Dordrecht, The Netherland, pp. 263-266.

Schenck, N.C. and Kellam, M.K. 1978. The influence of vesicular-arbuscular mycorrhizae on disease development. Fla. Agric. Exp. Stn. Tech. Bull. 798.

Schonbeck, F. 1979. Endomycorrhizas in relation to plant diseases. In: "Soil-Borne Plant Pathogens". (eds. Schippers, B. and Gama, W.) Academic Press, New York, pp. 271-280.

Schonbeck, F. 1980. Endomycorrhiza : Ecology, function and phytopathological aspects. Forum Microbiol. 3: 90 – 96.

Schonbeck, F. and Dehne, H.W. 1981. Mycorrhiza and plant health. Gesunde pflanzen, 33:186-190.

Schonbeck, F. and Schinzer, U. 1972. Investigations on the influence of endotrophic mycorrhiza on TMV lesion formation in *Nicotiana tabacum* L. var. xanthi. Phytopathol. Z. 73: 78-80.

Schonbeck, F. and Spengler, G. 1978. Detection of TMV in mycorrhizal cells of tomato by immunofluoresecence. Phytopathol. Z. 94: 84-86.

Secilia, J. and Bagyaraj, D.J. 1987. Bacteria and actinomycetes associated with pot cultures of vesicular-arbuscular mycorrhizas. Canadian J. Microbiology, 33:1069-1073.

Sharma, A.K., Johri, B.N. and Gianinazzi, S. 1992. Vesicular-arbuscular mycorrhizae in relation to plant disease- review. World Journal of Microbiology and Biotechnology, 8: 559-563.

Shaul, O, Galili, S., Volpin, H., Ginzberg, I., Elad, Y., Chet, I. And Kapulnik, Y. 1999. Mycorrhiza-induced changes in disease severity and PR protein expression in tobacco leaves. Mol. Plant-Microbe Interact., 12: 1000- 1007.

Sikora, R.A. and Schonbeck, F. 1975. Effect of vesicular-arbuscular mycorrhiza (*Endogone mosseae*) on the population dynamics of the root knot nematodes *Meloidogyne incognita* and *M. hapla*. 8th Int. Cong. Plant Prot., 5:158-166.

Singh, R., Adholeya, A. and Mukerji, K.G. 2000. Mycorrhiza in control of soil borne pathogens. In: "Mycorrhizal Biology"(eds.Mukerji, K.G., Chamola, B.P. and Jagjit Singh), Kluwer Academic Publishers, NY, pp.171-196.

Singh, K., Varma, A.K. and Mukerji, K.G. 1987. Vesicular-arbuscular mycorrhizal fungi in diseased and healthy plants of *Vicia faba*. Acta Botanica Indica, 15: 304-310.

Sitaramaiah, K. and Sikora, R.A.1981. Influence of the endomycorrhizal fungus *Glomus mosseae*

on *Rotylenchulus reniformis* penetration and development on bush bean, cucumber and musk melon. Med. Fac. Landbouww, Rijksuniv. Gent. 46: 695-702.

Sitaramaiah, K. and Sikora, R.A. 1982. Effect of mycorrhizal fungus, *Glomus fasciculatum* on the host parasite relationship of *Rotylenchus reniformis* in tomato. Nematologica, 28: 412-419.

Smith, F. W. 2002. The phosphate uptake mechanism. Plant and Soil, 245: 105- 114.

Smith, G.S. 1987. Interactions of nematodes with mycorrhizal fungi. In: "Vistas on Nematology" (eds. Veech, J.A. and Dickon, D.W.), Soc. Nematol., Hyattsville, Md., pp. 292-300.

Smith, G.S. and Kaplan, D.T. 1988. Influence of mycorrhizal fungus phosphorus and burrowing nematode interactions on growth of rough lemon seedlings. J. Nematol. 20:539-544.

Smith, G.S., Roncadori, R.W. and Hussey, R.S. 1986. Interaction of endomycorrhizal fungi, superphosphate and *Meloidogyne incognita* on cotton in microplot and field studies. J. Nematology, 18: 208-214.

Smith, S.E. and Gianinazzi-Pearson, V. 1988. Physiological interactions between symbionts in vesicular-arbuscular mycorrhizal plants. Annual Review of Plant Physiology and Molecular Biology, 39: 221-244.

St-Arnaud, M., Hamel, C., Vimard, B., Caron, M. and Fortin, J.A. 1995. Altered growth of *Fusarium oxysporum* f.sp. *chrysanthemi* in an *in vitro* dual culture system with the vesicular-arbuscular mycorrhizal fungus *Glomus intraradices* growing on *Daucus carota* transformed roots. Mycorrhiza, 5:431-438.

Stinzi, A., Heitz, T., Prasad, V., Wiedemann-Meidinoglu, S., Kaufmann, S., Geoffroy, P., Legrand, M. and Pritig, B. 1993. Plant Pathogenesis-related proteins and their role in defence against pathogens. Biochimie, 75:687-706.

Torres-Barragan, A., Zavalta-Mejia, E., Gonzalez-Chavez and Ferrera-Cerrato, R. 1996. The use of arbuscular mycorrhizae to control onion white rot (*Sclerotium cepivorum*) under field conditions. Mycorrhiza, 6: 253-257.

Trotta, A., Varese, G.C., Gnavi, E., Fusconi, A., Sampo, S. and Berta, G. 1996. Interaction between the soil borne root pathogen *Phytophthora nicotianae* var. *parasitica* and the arbuscular mycorrhizal fungus *Glomus mosseae* in tomato plants. Plant Soil, 185: 199-209.

Umesh, K.C., Krishnappa, K. and Bagyaraj, D.J. 1988. Interaction of burrowing nematode, *Radophilus similis* and VA mycorrhiza, *Glomus fasciculatum* (Thaxt.) Gerd and Trappe in banana (*Musa acuminata* Colla.). Indian J. Nematology, 18:6-11.

Utkhede, R.S. and Smith, E.M. 2000. Impact of chemical, biological and cultural treatments on the growth and yield of apple in replant-disease soil. Australian Plant Pathology, 29: 129-136.

Van Loon, L.C., Pierpoint, W.S., Boller, T. and Conejero, V. 1994. Recommendations for naming plant pathogenesis related proteins. Plant Mole. Biol. Repr. 12: 245-264.

Verdejo, S., Calvet, C. and Pinochet, J. 1990. Effect of mycorrhiza on kiwi infected by the nematodes *Meloidogyne hapla* and *M. javanica*. Buletin deSanidad Vegetal, Plagas, 16: 619-624.

Vestberg, M., Palmujoki, H., Parikka, P. and Uosukainen, M. 1994. Effect of arbuscular mycorrhizas on crown rot (*Phytophthora cactorum)* in micropropagated strawberry plants. Agricultural Science in Finland, 3: 289-296.

West, J.M. 1995. Soil phosphate status modifies response of mycorrhizal and non-mycorrhizal *Senecio vulgaris* L. to infection by the rust, *Puccinia lagenophorae* Cooke. New Phytol. 129: 107-116.

Whipps, J.M. 2001. Microbial interactions and biocontrol in the rhizosphere. Journal of experimental Botany, 52: 487-511.

Yao, M.K., Tweddell, R.J. and Desilets, H. 2002. Effect of two vesicular-arbuscular mycorrhizal fungi on the growth of micropropagated potato plantlets and on the extent of disease caused by *Rhizoctonia solani*. Mycorrhiza, 12: 235-242.

Diseases of Minor Tropical and Sub-tropical Fruits and their Management

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Abstract : In the present chapter diseases of aonla, bael, ber, custard apple, fig jackfruit, jamun, karonda, litchi, loquat, mulberry, passion fruit, phalsa, pineapple, pomegranate, and sapota have been furnished. These fruits are considered of minor importance in tropical and subtropical area because of their overall popularity, restricted area and awareness regarding their nutritive value. As such not much information has been available to readers on disease scenario and their management. Therefore, various diseases affecting them globally have been furnished in this chapter. In most of the cases anthrocnose, dieback, leaf spots, blight and fruit rots are quite common. While many diseases originate in the orchards, fruit rots may inititates from the field and may express in storage also in view of incipient infection. In other cases fruit rots may appear in storage due to lack of proper handling and storage. The amount of losses caused by fruit diseases is tremendous and has not yet been documented properly. The diseases can be controlled by taking integrated approach which involves right selection of variety, adoption of proper cultural practices, timely application of fungicides etc. Many post harvest disease which emanates from field can also be taken care of by pre-harvest spray of suitable fungicides. However post harvest fungicidal dip has to taken care to help health hazard. Detail methods of protection have been furnished in each crop.

1. Introduction

The tropical and subtropical fruits grow in wide range of agroclimatic conditions and are subject to attack by a large number of diseases caused by fungi, bacteria, mycoplasma and viruses. Diseases are limiting factor in the production of these minor fruits throughout the world. Diseases like anthracnose, mildews, wilt, rust, leaf spots, blight, die back and rots are widely prevalent throughout the region. To reduce the losses caused by these diseases, sound knowledge of their proper diagnosis, factors affecting development and spread and their management practices are very important. With the adoption of modern methods of intensive cultivation practices, a number of diseases have assumed significant importance in recent years and require greater attention and timely adoption of control measures to reduce losses. Fruit crops are perennial in nature and often provide high cost/benefit ratio and thus adoption of suitable remedial measures is warranted. Fruits like aonla, avocado, bael, ber, custard apple, fig, jackfruit, jamun, karonda, litchi, loquat, mulberry, passion fruit, phalsa, pineapple, pomegranate, and sapota though often referred to as minor fruits yet are endowed with tremendous nutrients and medicinal ingredients and hence constitute important source

of nutrition and health support. In this chapter, work done on important diseases of these minor fruits of tropical and subtropical regions are discussed.

2. Indian Gooseberry (*Phyllanthus emblica* L.)

2.1 Anthracnose

Disease appears on leaflet and fruits during August-September (Mishra and Shivpuri, 1983). Initially disease makes its appearance in the form of minute, circular, brown to grey spots with yellowish margin on leaflets. Central area of the spot remain grayish with dot like fruiting bodies. Lesions on fruits are usually sunken and turn dark with age bearing dot like fruiting bodies – the ascervuli arranged in rings. The lesion may vary in size and shape with spore masses appearing on fruiting bodies at high humidity. Consequently, the infected fruits become shrivelled and rot.

The disease is caused by *Colletotrichum gloeosporioides* [*Glomerella cingulata* (Stonem) Spauld & Schrenk]. The disease is favoured by hot and humid weather.

Spraying carbendazim (0.1%) can check the disease. Alternatively TPM may also be employed.

2.2 Die-Back

Dieback is indicated by a general unthrifty and stunted appearance of the tree. Drying of young twigs, papery bark and wood decay of the affected branches are the main symptoms. Leaflets in the disease part showed chlorotic appearance resembling nutritional deficiencies followed by premature leaf fall. Characteristic browning of vascular elements also observed.

Botrydiplodia theobromae Pat. is reported to cause die-back disease in various orchards of UP (Arya *et al.*, 1987). However, Rai (1982) found it causing dry rot of fruits and 5-10% trees were found effected.

2.3 Rust

It is an important disease especially in Rajasthan (Tyagi, 1967). It has also been observed at Lucknow and Rajgarh, Maharashtra and Madhya Pradesh (Anonymous, 1988). Initially conspicuous brown rust pustules are formed on leaflets and fruits of infected plants and finally these pustules become dark brown to black in colour. Severely infected fruits may lead to premature fruit drop. Plants with severe attack show no symptoms on leaves and vice-versa. Disease is responsible for considerable losses in major aonla growing areas of Uttar Pradesh (Rawal, 1993).

Ravenelia emblicae Syd. is known to cause this disease. The uredia of *R. emblicae* are typically sub-epidermal erumpent, bearing smooth walled uredospores produced singly on pedicels, measuring 9-10 x 6-7 µm. Telia are sub-epidermal in origin, erumpent, bearing pedicellate compound teleospores, measuring 42-54.5 x 20-28.8 µm. The uredospores are easily blown away by air current which successively deinfect the new leaflets and fruits during the growing season.

The disease can be controlled by applying monthly sprays of wettable sulphur (0.25%) or zineb (0.2%) during July-September. Control of *R. emblicae* var. *pinnulae* and *R. emblicae* var. *fructicola* by using wettable sulphur dust and ultra sulphur was obtained by Tyagi and Pathak (1987). Banarasi and Chakaiya cultivars are believed to be relatively free from disease.

2.4 Fruit Rots

2.4.1 *Phoma* Rot

Soft rot is caused by *Phomopsis phyllanthi* Punith. Disease normally appears in December-January (Pandey *et al.* 1980b) and characterized by the appearance of small pinkish brown necrotic spots extending towards both the ends of fruits forming an eye shaped appearance. Infected fruits become dark brown and crinkled with softening of underlining tissues.

The disease is reported to be caused by *Phoma putaminum* Speg. Jamaluddin *et al.,*(1975) also reported dry fruit rot caused by *P. emblicae.*

2.4.2 Soft Rot

It is an important fruit rot of aonla occurring both in orchards and markets. (Lal *et al.,*1982) usually noticed from December to February. Circular lesions with smoke brown to black colour develop with in 2-3 days of inoculation: The infected part-later shows olive-brown-discolouration with water soaked area which covers the whole fruit with in 8 days leading to deformity of fruits. Both the young and mature fruits are infected by the fungus but mature fruits are more susceptible. Pre-injured fruits are prone to infection. Fungicides like difolaton (0.2%) mancozeb (0.2%) or carbendazim (0.1%) as pre harvest sprays are effective in minimizing soft rot during storage and market (Lal *et al.,* 1982). Difolaton, however, need not be used for foliar application for safety reason.

2.4.3 *Cladosporium* Fruit Rot

It is usually a dry rot disease responsible for 2-5 per cent loss (Jamaluddin, 1978). Cladosporium fruit rot is caused by two species viz., *Cladosporium tenuissimum* Cooke and *C. cladosporioides* (Fr.) de Vries. In case of former species, the fruit infection initiates during November to February as colourless area slightly soft and extended in a circular manner covered by light brown mycelial growth of the fungus measuring 1.0 to 1.5 cm in diameter. The latter species develops at the end of season during February-March as dark-brown and necrotic lesions (0.7-1.2 cm) with slight mycelial growth in the necrotic cavity. Injury of fruits was necessary for manifesting the infection. Earlier, Tandon and Verma (1964) observed *C. herbarum* on fruits of aonla.

2.4.4 *Nigrospora* Fruit Rot

The disease is caused by *Nigrospora sphaerica* (Sacc.) Mason. It is reported from

Kanpur, UP occurring in December causing 5-10 percent losses annually (Kamthan *et al.,* 1981). Symptoms appear as small dots of 2 mm diameter extending to a black ring spot upto 7 mm. Complete rotting of infected fruits occurred if several rings adjacent to each other coalesce.

2.4.5 *Pestalotia* Fruit Rot

The disease is caused by *Pestalotia cruenta* Syd. The disease has been reported from markets of Allahabad during the month of November by Tandon and Srivastava (1964). Rot appears initially as irregular and brown spots on fruits. The disease usually starts as brown discolouration on the fruit surface which grows slowly and later become mummified, dark brown with light brown discolouration of skin around them. Finally the infected area of fruits get covered with white mycelial growth.

The treatment of fruits with 1250 µg/ml Bavistin showed good control of the disease.

2.4.6 *Alternaria* Fruit Rot

Alternaria alternata (Fr.) Krissler is responsible for the disease. This is usually associated with dropping of fruits in Allahabad, India (Pandey *et al.*, 1984). Initial symptom of the disease appears as small brown to black, spherical necrotic spots on fruits which coalesce to cover large areas. Infected tissues central portion become soft and pulpy and affected fruits usually drop from trees.

2.4.7 *Penicillium* Rot or Blue Mold

Blue mold is caused by *Penicillium islandicum* Sopp. The occurrence of *Penicillium* sp. on aonla fruit in India was reported by Srivastava *et. al.,* (1964). Initial symptoms appears as water soaked brown areas with three distinct zones viz., bright yellow, purple brown and bluish green. With the progress of disease the whole fruits finally get covered with bluish green moldy growth of the organism. (Setty, 1959). A yellowish liquid, emitting bad odour, exudes from infected rotten areas.

Management of fruit rot lies in preharvest spray of 0.2% carbendazim or 0.2% mancozeb once or twice during the season. Proper sanitation in the field, careful handling so as to avoid injury to fruits is usually helpful in minimizing the post-harvest losses. Prestorage treatment of fruits with weak borax or sodium chloride has also been advocated in avoiding the appearance of disease on fruits.

2.4.8 *Aspergillus* Rot

Srivastava *et al.,*(1964) reported the occurrence of *Aspergillus awamori* Nakazava and *A. niger* Van Tiegh. causing surface rot of aonla. About 8 to 21% fruits of aonla rotted in storage. While studying the metabolic pattern of host plant, Jamaluddin *et al.,*(1979) revealed various changes induced during pathogenesis.

2.4.9 Other Fruit Rots

Other pathogens reported to cause fruit rot of aonla besides mentioned above include *Cladosporium herbarum* (Pers.) Link ex Fr. and *Hendersonula toruloides* Nattaras, (Sumali, 2000).

3. Wood apple (*Aegle marmelos L.* Correa)

3.1 *Alternaria* Leaf Spot

Initially brown or dark brown coloured spots of indefinite size appear on leaves with light brown or dark brown rings. Affected leaves blighten and fall. The disease is incited by *Alternaria alternata* (Fr.) Keissler. Seedlings could also get infected (Madaan and Gupta, 1985).

Spraying trees with 0.2 per cent copper oxychloride at 15 days interval helps in controlling this disease.

3.2 Stalk End Rot

Stalk end rot caused by *Fusarium solani* (Mart.) Sacc. was reported by Bhargava *et al.,*(1977). *F. semitectum* var. *majus* has been reported earlier on darkened young shoots of bael (Mitra, 1935). Immature dropping of young fruits during rainy season is the main symptom of this disease. The disease is characterized by softening of rind and under-laying pulp. The affected rind turns dark brown at a later stage of infection. Fruits neither shrivell nor loose their shape until press. Infected fruits fall when there is heavy rain or strong wind.

Fungicides like benomyl, captan, plantavax, thiram @ 1000 mg/ml and ferbam and difolaton @ 500 mg/ml were effective against the disease when tested *in vitro* (Bhargava *et al.*, 1977). However, difolation should not be taken up for foliar application for safety consideration.

3.3 *Aspergillus* Rot

During summer months, May-June, a sever post-harvest rot caused by *Aspergillus awamori* Nakazawa was observed on bael by Arya *et. al.* (1986). The disease resulted into 100% loss of fruit kept in store house, since the infection was latent. In severe cases the white outer skin of the shell become soft and rotten. Inner pulp produces black sooty growth of the fungus.

3.4 *Fusarium* Rot

During rainy season in the month of June-July, along with Aspergillus rot, a new rot caused by *Fusarium moniliformae* Shelden was also observed by Arya *et. al.,* 1986. Cottony growth of fungal mycelium was observed just beneath the hard shell. The fungus which encircled the whole fruit turned it soft and pulpy.

3.5 Bacterial Shot Hole and Fruit Canker

The disease is reported from Maharashtra (Patel *et al.,* 1953). Symptoms of the disease appears as round, water soaked spots (0.5 mm) surrounded by clear hole which later on increase in size and form brown lesions with saucer like depression in the center, surrounded by oily raised margin. On fruits, the lesions are round, raised, water soaked 4-7 mm size and without any halo later on depression are formed in the center of spots surrounded by irregular, oily, raised margin which become corky, irregular

Table 1: Some minor diseases of wood apple.

Disease	Causal organism (s)	Reference
Dry rot	*Aspergillus niger*	Verma *et al.,* (1991)
	A. fumigatus	
	A. luchuensis	
Leaf blight and leaf spot	*Myrothecium roridum*	Harsh *et al.,* (1989)
	Colletotrichum gloeosporioides	
Leaf spot	*Fusarium roseurm*	Kore and Dhande, (1973)
	Cladosporium cladosporoides	Sarbajha, (1989)
Powdery mildew	*Oidium* sp.	Giri *et al.,* (1989)
	Sphaerothcea fuliginea	Sinha and Singh (1995)
	Oidium tingitatanium	
Fruit rots	*Fusarium solani*	Sharma *et al.,* (1979)
	Phoma glomerata	Pandey *et al.,* (1980a)
	Othia spiracae	Srivastava, (1982)

and chocolate brown in colour. On twigs, the lesions are large (150 x 4 mm) and infected tissue ruptures in the center giving a rough corky appearance. Bacterial exudates are found on all infected part of plant. The disease is caused by *Xanthomonas campestris* pv *bilvae* (*Xanthomonas bilvae* Patel *et al.*, 1953).

3.6 Other Diseases

A number of other diseases have been reported on wood apple but not much work appears to have been undertaken on management. These are summarized in Table 1.

4. Ber (*Zizyphus spp*.)

4.1 Powdery Mildew

4.1.1 Occurrence

This is one of the most predominant diseases and causes significant losses due to its

infection on fruits (Kapur *et al.*, 1975). It is widely prevalent and reported from Allahabad (Mitter and Tandon, 1930), Kanpur (Mehta, 1950) and Bombay (Uppal *et al.*, 1935, Patel *et al.*, 1949). Disease is now known to appear on both cultivated and wild species. This disease is present in most of the grafted ber plantations causing heavy losses every year both qualitatively and quantitatively.

4.1.2 Symptoms

The disease generally appears by the end of October and prevails from November to April. With the rise in temperature after February, the disease subsides comparatively. The disease first appears on fruits in the form of white floury patches and later cover the whole fruit. With the passage of time, the infected area becomes slightly raised and rough. The infected fruits often become misshapen and may shed from the trees. Kumar *et al.,* (1978) have also observed symptoms on leaves and stem besides fruits in Rajasthan. The disease appears on developing young shoots in the form of white powdery mass on the leaves which results in shrinkage. Though primarily the disease appears on flowers and fruits but Gupta (1984) observed the germinating conidia of the causal fungus on leaf surfaces.

4.1.3 Causal Organism

The disease is caused by the fungus *Oidium erysiphoides* f.sp. *zizyphi* Yen and Wang which survives in bud wood and on some collateral host during the absence of flowers and fruits which serve as primary source of infection. The secondary spread takes place by air borne spores. The fungal mycelium becomes external on the host while conidiophores are upright, single, measuring 75.8-139.4x12.6 mm. Conidia are cylindrical, hyaline, catenulate measuring 25.2-37.8x16.8-21.0 mm. Powdery mildew of ber caused by *Microsphaera alphitoides* f.sp. *ziziphi* has also been reported by Mehta (1950).

4.1.4 Epidemiology

The mycelium overwinter in the new shoots and arise annually. It may also survive on some alternate host during the absence of flower and fruits, which become primary source of infection. Air borne spores become secondary source of infection. *M. alphitoides* perpetuation through dormant mycelium in buds as perithecia have not been reported so far in India (Parkash *et al.*, 1988). No collateral host of *M. alphitoides* f sp. *ziziphi* has been reported. Conidia of this fungus germinate and form appressoria after 2-4 hours at 20±2°C in most saturated atmosphere, whereas sporulation starts 96 hours after inoculation on susceptible ber leaves. Temperature range from 10-30°C and relative humidity levels of 32 per cent and above favour disease development (Parkash and Jhooty, 1987). Singh *et al.,*(1999) developed prediction model for powdery mildew disease build up based on weather parameters in Haryana. Maheshwari and Singh (1999) assessed the disease severity on fruits in relation to environment during 1991-93 at Kanpur, India.

4.1.5 Chemical Control

Sulphur dusting in December has been considered enough for satisfactory control by Mehta (1950). Several workers have reported the efficacy of dinocap, carbendazim, fenarimol and triademefon in reducing the disease (Gupta *et al.*, 1977, 1978, Reddy *et al.*, 1990; Das *et al.*, 1994; Singh *et al.*, 1995). Five sprays of dinocap one each from August to December at monthly interval are useful. Sulfex (0.2%), dinocap (0.05%) and carbendazim (0.05%) or wettable sulphur (0.2%) at ten days interval between the second week in October and December are also quite effective recommendations (Yadav *et al.*, 1980). Benlate is reported to give 92 per cent control of the disease followed by Afugan (Kapur *et al.*, 1975).

Disease can effectively be checked by spraying the fruits with carbendazim (0.1%) first when the fruits are of pea size stage followed by two or three sprays of dinocap (0.1%) at 10 to 15 days interval (Singh and Sidhu, 1985). Triadimefon, dinocap, sulphur and propiconazole have been reported much effective (Thind and Kaur, 1998). Of the six fungicides tested, triadimefon (0.1%) was most effective followed by thiophanate methyl (0.1%) and dinocap (0.1%) and also recorded highest yield (Reddy *et al.*, 1997). Both floral and fruit infection can be significantly reduced by giving six sprays of wettable sulphur involving either 1 or 2 sprays of triadimefon 0.1% at the flowering stage (Desai, 1998a). Wettable sulphur is cheaper substitute for powdery mildew control. A schedule of 5 sprays involving first two fortnightly sprays with bayleton (0.1%) commencing from third week of September at flowering and early fruit set stages and remaining three fortnightly sprays with sulphur (0.2%) at pea size and marble size fruit stages was also found very effective in controlling the disease (Anonymous, 2000).

4.1.6 Varietal Resistance

The disease can also be averted by growing vars. Safeda Rohtak, Sua, Noki, Chonchal, Sanaur-5, Kathaphal, Sanaur-1, Illachi-Jhajjar, Kakrola Gola Kala Gora, Pathani and Mirchia which have been reported resistant by several workers (Gupta *et al.*, 1978; Kapur *et al.*, 1975; Jeyarajan and Cheema, 1972). Five ber cultivars viz., Guli, Seedless, Villaiti, Darakhi and Darakhi-2 were found resistant to powdery mildew under artificial epiphytotic conditions at Rahuri, Maharashtra, India (Anonymous, 1985). As such, these varieties may be taken up for cultivation to ward off powdery mildew.

4.2 *Alternaria* Leaf Spot

Initial symptom of the disease appears with the formation of small irregular brown spots on the upper surface of leaves and dark brown to black spots on lower surface (Madaan and Gupta, 1976; Gupta and Madaan, 1977a). Later on, many spots coalesce to form large patches and such leaves later dry. The disease is caused by several species of *Alternaria* such as *A. chartarum* Preuss from Maharashtra (Rao, 1971), *A. passeriniana* Berk. from Jodhpur (Panwar and Vyas, 1974). *Alternaria* spp. from Punjab (Jeyarajan and Cheema, 1972) and *A. alternata* (Fr.) Krissler (Gupta and Madaan, 1977a) from

Hisar, India.

The disease development is favoured at 20 to 30°C with optimum at 25°C. Primarily the pathogen survives in plant debris and frequent rainfall helps in dissemination of fungal spores which causes secondary infection (Madaan and Chand, 1985).

The disease can be effectively checked by spraying 0.2 per cent Dithane Z-78 (Gupta and Madaan, 1978) or growing Bahadurgarhia, Govindagarh special, Gola Gurgaon, Popular Gola, Seo, Bahadhurgarhia, ZG 3, Safeda Rohtak, Jhajjar Special and Mirchia cvs. of ber which are reported resistant against this disease by Gupta and Madaan (1980). Out of 35 cultivars screened against this disease, 25 were found moderately susceptible (Jeyarajan and Cheema, 1972).

4.3 *Isariopsis* Mouldy Spot

Also known as black leaf spot or black mildew disease of ber . The disease starts during October-November and appears as sooty tuft-like circular to irregular black spots on lower leaf surface. As the infection advances, it covers a large area on the lower surface of leaves and upper surface shows brownish discolouration and such infected leaves may finally drop.

The disease is caused by *Isariopsis indica* Nair var. *zyziphi* in Haryana (Gupta and Madaan, 1977a). The fungus consists of synnemata of dark olivaceous colour, divergent, 220 x 102 μm composed of loose conidiophore which are erect, simple, olivaceous in colour, bears conidia terminally and laterally measuring 54.4–138.4 x 6.8 μm; conidia olivaceous, 0-3 septate, cylindrical to obclavate, sometimes pyriiform measuring 17.0-4.2 x 8.5-10.2 μm.

The fungus survives in plant debris and soil as a primary source of infection. The secondary spread occurs through spores present in air. Verma *et al.,*(1995) reported the survival of pathogen either on infected fallen leaves or leaves sticking to branches and cracks of the bark. The pathogen is disseminated by air currents. Lower epidermis may provide better adherence for conidia and moist conditions for germination compared with the smooth upper leaf surface.

The disease can be checked by spraying 0.2 per cent Bavistin (Gupta and Madaan, 1985). Though the disease has also been controlled by spraying 0.3 per cent zineb or copper oxychloride but the best results have been achieved by spraying 0.1 per cent carbendazim or thiophanate methyl (Rawal and Saxena, 1989; Singh *et al.*, 1989). *Hansfordia pulvinata* (Berk et curt) Hughes, a mycoparasite was found growing on the infected area which can keep the disease under control (Gupta and Madaan, 1979). Nine cultivars of ber viz., ZG-3, Safeda Rohtak, Mudia-Murhera, Sua, Sanaur-1, Pathani, Jhajjar Selection, Seo Bahadurgarhia and Jhajjar Special were found resistant to black leaf spot (Gupta *et al.*, 1980).

4.4 *Cercospora* Leaf Spot

Disease appears as circular to oval discoloured epiphyllous spots measuring upto 4 mm in diameter on leaves which later turn brown and get surrounded by dark brown margins. Two species of *Cercospora* (*C. ziziphi* Petch and *C. jujubae* Chowdhuri) have

been reported from different ber growing areas of India responsible for leaf spot disease (Chona *et al.*, 1959; Vasudeva, 1960; Rao, 1962; Yadav, 1963; Agarwal and Sahni, 1964; Govindu and Thirmalachar, 1964; Gupta and Madaan, 1975a).

Spraying trees with 0.2 per cent Dithane M-45 gives good control of the disease. Eight cultivars viz., ZG-3, Safeda Rohtak, Kakrola Gola, Reshmi, Bahadurgarhia, Jhajjar Selection, Seo Bahadurgarhia and Popular Gola are reported to be resistant to this disease (Gupta and Madaan, 1985).

4.5 *Cladosporium* Leaf Spot

Initial symptom of the disease appear on lower surface of leaves during October-November as small, light brown to dark brown irregular spots. *Cladosporium zyzyphi* Karst and Roum (Uppal *et al.*, 1935; Prasad and Verma, 1970; Saini and Uppal, 1981) and *C. herbarum* (Persoon) Link (Gupta and Madan, 1975b) is reported to cause leaf spot disease in ber. However, disease is generally caused by *C. harbarum*. Fungus consists of conidiophores erect, little branched, septate, brown to olive- green 5-10 μm. Conidia terminal by extension of tip falsely lateral, on short knee, elongate, oval usually one celled or cylindrical to ellipsoidal. The fungus survives in plant debris and soil and survive as primary source of infection. Secondary spread through spores present in air.

Spraying of ber trees with 0.3 per cent copper oxychloride or captan twice at two weeks interval with the first appearance of disease is recommended for effective control. Cultivars Banarsi, Villaity, ZG-3, Govindgarh Sel-3, Jhajjar Selection and Zogia show resistant reaction (Gupta and Madan, 1985).

4.6 Rust

Rust disease was first reported from Bihar, Maharashtra, West Bengal (Sydow and Sydow, 1907) and now occur in all the ber growing tracts of India (Yadav, 1963; Gupta *et al.*, 1984). Disease first appears on the lower surface of leaves as small, irregular, reddish brown urdopustules which later advance to cover the whole surface of the leave. The infected leaves finally shed from the trees. *Ziziphus* rust is the main disease in China (Zhao *et al.*, 2000). The disease which is caused by the fungus, *Uredo zizyphi* (*Phakospora zizyphi vulgaris* (P. henn.) Diet. occurs both on wild and cultivated varieties of ber during January-February.

Disease can be checked by spraying Dithane M-45 (0.2%) or Dithane Z-78 (0.2%) or Sulphur (0.2%) or Vigil (0.1%). Bordeaux mixture, lime sulphur, tridimefon (amiral) or carboxin also effectively control rust disease (Zhao *et al.*, 2000). In rust prone areas, cultivation of cultivars Banarsi, Seo, Katha Gurgaon, Laddu, Dandan, Sanaur-1, Gola, Gurgaon-2, Safeda Selection, Sanaur-3, Kishmish Narma and Safeda Rohtak may be taken up which are reported resistant (Gupta and Madaan, 1985) against rust.

4.7 Fruit Rots

Several fungi including *Pestalotia versicolor* Speg, *Phoma* spp., *Colletotrichum* spp., *Trichothecium* spp. and *Alternaria* spp. cause fruit rots by producing light grey-brown

spots to dark brown rings on fruits (Gupta and Madaan, 1975a, 1977b). Five fungi viz., *Cladosporium tenuissimum, Fusarium pollidoroseum* (Cooke) Sacc., *Pythium aphanidermatum* (Eds.) Fitz., *Phoma nebulosa* and *Rhizoctonia solani* Kuchn. associated with post harvest decay of ber fruits were reported from Rajasthan (Sharma *et al.*, 1993).

Eighteen different fungal species reported to cause post harvest decay of fruits,. However, only *Aspergillus flavus* infection was of significance which results in maximum loss of ascorbic acid and also induced aflatoxin production during pathogensis (Singh and Sumbali, 2000). 83.7% of the *A. flavus* isolates associated with decay were toxic. *Alternaria alternata*, *Phoma destructiva* and *Fusicoccum* spp. were also isolated from Chinese jujube brown cortex (Kang *et al.*, 1998).

Other fungi reported to be associated with post harvest fruit rot of ber includes *Phoma capitulum, Phomopsis ziziphina* Ponnappa*., Cladosporium oxysporum* Berk. and Curt., *Curvularia lunata* (Wakker) Boedijn, *Colletotricum gloeosporioides* (Penz.) Sacc., *Ceratocystis adiposa* (E. Butter), *C. morean* and *Trichothecium roseum* Link (Sumbali, 2000).

Spraying zineb (0.2%) or copper oxychloride (0.3%) at the initial stage of infection effectively reduced the fruit rot diseases.

4.8 Witches Broom

A witches broom disease was observed from Korea (Kim, 1965; Lin and Li, 1985) and on Chinese jujube in China (Zhu *et al.*, 1983). One third of the ber orchards were found affected in Maharashtra during 1993. The disease is also associated with dieback symptom. In nature, Phyllody is a characteristic symptom if the plants are infected one year before attaining the flowering stage and affected trees may produce small fruits but do not flower in subsequent years. Shoots of infected trees are weak and elongated and cannot stand upright. The auxiliary buds proliferate and produce many small, thin and weak secondary branches, covered with yellow leaves giving it a bushy appearance. Mosaic symptoms are caused by increase of chlorophyllagae and decrease in chlorophyll. Crinkled leaves and malformed flower buds results from histological changes in cells.

The disease is transmitted by grafting and symptoms appear after two and a half months as production of abundant small leaves. Attempts to transmit the disease through sap inoculations on *Z. mauritiana* and herbaceous hosts failed. Affected plants when sprayed with tetracycline antibiotic (1000 µg/ml) develop new shoots with normal-size green leaves (Pandey *et al.*, 1976) suggesting the involvement of mycoplasma like organism with this disease. Im *et al.,*(1985) reported that the disease is caused by MLO in mature sieve elements and further confirmed when it was found absent from phloem elements after oxytetracycline HCL injections. However, Zhao *et al.,*(2000) described witches broom in China as phytoplasma disease. Factors affecting the observation of phytoplasma infection in Chinese jujube trees were studied systematically by fluorescence microscopy (Wang *et al.*, 1999). Cheng and Liu (1994) develop new technique for treating jujube twig materials infected with witche's broom disease for electron microscopy which completes in 10-15 h whereas commercial technique

needed 100 h. In China and Korea, apart from cutting out the infected branches of zuzube, infecting a solution of tetracycline or terramycin has some effect on witches broom disease (La *et al.*, 1977; Zhao *et al.*, 2000). A method for elimination of phytoplasma causing witches broom of *Zizyphus jujube* (*Ziziphus sativa*) is described by Zhu *et al.* in 1996.

4.9 Fruit Shrink Disease

Fruit shrink is also one of the important disease of Chinese zuzube in China. The disease appears throughout the growing period in China and results in a yield reduction of 50-60%, poor quality and bitter fruits (Zheng *et al.*, 1998). *Coniothyrium olivaceum, Alternaria alternata, Dothiorella gregaria* and an unidentified bacterium were reported causal agents of this disease in China (Zheng *et al.*, 1996; Xu *et al.*, 1995). However, Zheng *et al.*,(1998) identified the causal agent as *Alternaria tenuis* (*A. alternata*).

4.10 Other diseases

Septoria leaf spot caused by *Septoria capensis* Wint., and *Pestalotia* leaf spot caused by *Pestalotia subinae* Fantrey, are reported from Hisar (Madan and Gupta, 1976). *Tandonella* leaf spot caused by *Tandonella zizyphi* Prasad & Verma, reported from Bihar (Prasad and Verma, 1970) and Punjab (Verma and Cheema, 1983). Sirosporium leaf spot caused by *Sirosporium carissae* Kapoor, from Rohilkhand (Pandey *et al.*, 1986) and *Botrydioplodia* leaf spot caused by *Botryodiplodia theobromae* from Gwalior, India (Jain *et al.*, 1983). Wilt of ber seedling is caused by *Fusarium equiseti* (Corda) Sacc. (Lodha, 1984) in Jodhpur (Rajasthan) and *Fusarium oxysporum* Schlecht (Gupta and Madaan, 1985) in Hisar, Haryana, Inida.

5. Custard apple (*Annona squamosa* L.)

5.1 *Glomerella* Fruit Rot or Anthracnose

The rot is caused by *Glomerella cingulata* (*Colletotrichum gloeosporioides*) . The disease has been reported from Assam in India (Chowdhury, 1947). Initial infection begins at blossom end in the form of a dry, blackish-brown spot which very slowly, spreads in all directions and generally covers the whole fruit, transforming it into a shrivelled mass. Affected fruits may cling to the tree or fall down.

The disease can be effectively checked by spraying with Bordeaux mixture (2:2:50). Benlate (0.05%), Topsin-M (0.2%), Dithane M-45 (0.2%) and Bavistine (0.05%) has also been found effective in controlling the disease.

5.2 *Botrytis* Rot

Botrytis cinerea Pers. was observed on seeds, and fruits on flowers of custard apple trees during February 1998 in Chile (Veronica and Ximena, 1998). The incidence of

disease started with the rainy period in May and reaches maximum in October. The incidence of *Botrytis* in post harvest fruits reached 10.6 per cent.

5.3 *Phytophthora* Blight and Damping Off

Phytophthora nicotianae Breda de Haan, is reported to cause *Phytophthora* blight and damping off disease in India (Nema and Sharma, 1996). The fungus is isolated from seedlings and lower leaves of custard apple with symptoms of blight and damping off. The disease is observed as water soaked lesions, which later turn brown in colour. In seedling stage, the lesions results in the death of the plant.

5.4 Pink Disease

Pink disease is caused by *Pellicularia solomonicolor* Berk. & Br.. The disease can be managed by removal and destruction of affected plant and spraying with Bordeaux mixture.

5.5 Other Diseases

Leaf spots (*Cercospora annonae* Muller & Chupp.*, Phyllosticta annonae-squamosae* Vasant Rao*, Diplodia natalensis* Evans.*, Drechslera rostrata (Drechs.*) Richardson & Fraser., anthracnose (*Colletotrichum anonicola* Speg.) and fruit rots (*Pestalotia bicolor* Ell. & Ev.*, Thielaviopsis paradoxa*(de Seynes) has also been reported to occur on custard apple (Saha, 2002).

6. Fig (*Ficus carica* L.)

6.1 Rust

It is serious disease of fig in all the fig growing countries. The disease occurs on all species of *Ficus* including cultivated fig. Small, round, brownish to black eruptive lesions appear mostly in the lower surface of leaves which later result in defoliation and yield reduction. The cinamon-yellow waxy, sub-epidermal, erumpent teliosori on leaves are less powdery in appearance than uredosori (Thirumalachar *et al.*, 1950). Disease is characterized by the appearance of rusty brown raised pustules on lower surface of leaf and discolouration on upper surface is also reported. Rust is caused by *Cerotelium fici* (Cast.) Arth. Disease is also reported to occur during survey of orchard in Ludhiana from Punjab (Verma and Kapur, 1995).

The spread of disease can be checked by spraying trees with zineb (0.2%) or bayleton (0.2%). Mancozeb (0.2%) and tridemorph (0.05%) can also be used effectively (Desai and Jamadar, 1997; Desai, 1998b).

6.2 Leaf Spot

Leaf spot is caused by *Cylindrocladium scoparium* Morg. and was first reported from

Uttar Pradesh, India (Mehta and Bose, 1947). Initially, minute brown spots appear on leaves which enlarge into uniform or zonate, prominent, redish brown lesions with dark brown margins. These lesions later coalesce to form irregular patches and affected leaves shed earlier. The centre of the leaf become papery and drops off. Cobweb like mycelium spread over the lower surface of leaves and later on ultimately become powdery.

The spread of the disease can be checked by spraying trees with 0.3% mancozeb or captan.

6.3 *Ascochyta* Leaf Spot

It has been first reported from Chaubattia (Uttar Pradesh) in 1984 (Singh *et al.*, 1984). Irregular, grey spots appear on upper surface of leaves after rains. It is incited by *Ascochyta caricae* Rebenth. Pycnidia of the fungus are globose, dark immersed in host tissue black septate conidiophores indistinct, conidia hyaline, ovoid to oblong, 2 celled, biguttulate, double walled, measuring 3-5x10 x 2.6 μm in size.

The disease can be effectively controlled by spraying trees with 0.1% benomyl or carbendazim (Singh and Singh, 1986).

6.4 Anthracnose

The disease is caused by *Sphaceloma fici-caricae*, Wani and Thirum. Anthracnose is common disease in certain fig growing areas of India (Wani and Thirumalachar, 1973). The disease becomes severe if humid conditions prevail. The fungus causes heavy spotting on leaves, petioles, tender shoots and fruits. In initial stages, chalky-white spots appear on leaves extend on petioles, tender shoots and fruits. In case of severe spotting, fruit crack and develop unevenly.

The spread of the disease can be checked by spraying Aureofungin (40 μg/ml) plus copper sulfate (20 μg/ml) in soap solution.

6.5 Fruit Rots

Fruit surface rot (*Alternaria* sp.) and storage rot (*Rhizopus stolonifer*) causes considerable losses of fig fruits. Fruit surface rot disease could be checked by spraying trees with 0.2% chlorothalonil (Bewaji *et al.*, 1977). Pre-harvest spray of copper sulphate (20 μg/ml) in combination with Aureofungin (40 μg/ml) in soap solution is quite effective in reducing the storage rot (Wani and Thirumalachar, 1973).

A fruit rot caused by *Choanephora cucurbitarum* (Berk. & Rav.) Thaxt. has also been first observed in Allahabad (Uttar Pradesh) on green fig in 1980 (Bhargava *et al.*, 1982). Initial symptom of disease appear as light brown lesions on fruit surface which gradually enlarge and change to dark brown-black. The infected fruits shrivel, dry and finally fall off. When such infected fruits are cut open, they are found infected from within. Fruit rot caused by *Phytopthora palmivora* (Butler) Butler, has also been reported to occur in commercial crop of fig from Argentina (Gonzalez *et al.*, 1998).

The diseases viz., bread mold, Fusarium wilt, and die back caused *by Aspergil-*

lus niger, Fusarium moniliforme and *Sclerotinia sclerotiorum* respectively also attack fig plant but the extent of damage is not much. Besides this, *Cladosporium sicophilum* and *Botrytis cinerea* are also reported to affect the fig plants. Spraying 1% ferrous sulphate in late winter or 4% Bordeaux mixture in early spring is helpful against these pathogens. However, in summers captan or zineb or ziram (0.1%) should be used.

6.6 Fig Mosaic

Mosaic of fig occurs throughout the world. In India disease was reported from Kumaon and Shimla hills (Bhargava and Bist, 1962; Nagaich and Vashisht, 1962). The disease expressed differently in wild and cultivated species of figs. On wild species symptom appears as masked mosaic with blister like patches and later on such leaves become pale yellow and wither away. Infected plants bear few under size fruits. However, on cultivated varieties, yellow green spots scattered over lamina, which sometimes coalesce to form bigger spots of various shape and size.

Besides being graft transmissible the virus is also transmitted by fig mite (*Aceria fici*) (Vashisht and Nagaich, 1968). Two types of mosaic symptoms are reported to occur in Japan (Nitta *et al.*, 1995). Light chlorotic spotting and mottling (type A) and extensive chlorosis along the veins, often with leaf malformation (type B). Type A symptom result from fig bud mite infestation while type B symptoms are induced by graft transaminable pathogen, which could also be transmitted by *A. ficus*. The causal virus can infect *F. nemosalis, F. carcia* and *Morus indica* in addition to *F. palmata* (Vashisht and Nagaich, 1965).

Gella *et al.,* (1997) suggested the elimination of fig mosaic from fig shoot tip cultures by thermotherapy. Apical bud (0.5-0.7 cm) with mosaic symptom cultured in medium and subjected to an alternating, high temperature regime with 16 h light at 37°C followed by 8 h dark at 34°C resulted in fig plants without mosaic symptom after year of cultivation in green house.

6.7 Endosepsis

Endosepsis caused by *Fusarium moniliforme* (*Gibberella fujikuroi*) is important disease in California, USA (Michailides *et al.*, 1996). Both capri figs and calimyrna figs are resistant to endosepsis infection upto 7 weeks prior to maturity (Subbarao and Michailides, 1996).

6.8 Soaring rot

Hosomi and Kusakari (1995) reported the occurrence of soaring rot caused by yeast (type O) in Japan. Fig fruit decayed and became soar, brownish and water soaked. These symptoms are different from that caused by *R. stolonifer*. The yeast is carried by adult *Drosophila melanogaster* and causes soft rot of fig. Adults were often observed on rotted fig and yeast type O was also isolated from legs of these flies.

7. Jack Fruit (*Artocarpus heterophyllus* Lamk.)

7.1 Die Back

It is one of the most destructive diseases of jack fruit. The disease is characterized by discolouration and darkening of the bark some distance from the tip. The dark area advances and young green twigs start withering first at the base and then extending outwards along the veins of leaf edges. The affected leaves turn brown and their margins roll upwards (Rawal, 1993). The diseased twigs and branches die, shrivel and fall off. The affected branches exude gum which are later infested by shoot borers. Infested twigs show internal discolouration. In early stages, epidermal and sub-epidermal cells of twigs appear slightly shrivelled and on such twigs, *Colletotrichum gloeosporioides* has also been recorded.

Die back caused by *Botryodiplodia theobromae* Pat is often has been found associated with *C. gloeosporioides.* The disease can be effectively managed by pruning out infected twigs, applying fungicidal paints on cut surfaces followed by spraying trees with 0.1% Bavistin or Topsin M or 0.2% chlorothalonil.

7.2 *Rhizopus* Rot

Rhizopus rot also known as male inflorescence rot was first reported from Assam in 1949. It is widely prevalent in India and results in premature dropping of young fruits and thereby causing considerable losses. Rotting of young fruits starts from stalk end and further extend covering whole of the fruit. The infected fruits later rot or mummified and fall prematurely. Besides young fruits, male flowers are also attacked by the causal fungus. The disease is caused by *Rhizopus artocarpi* Racib., *R. stolonifer* has also been reported to cause premature drop of young fruits (Pandey *et al.*, 1979). The fungus lives saprophytically on plant debris, manure piles and compost heaps.

Spraying 0.05% benomyl or carbendazim or 0.25% Blitox-50 at 15 days interval during fruit growth stages is effective in reducing the disease. The disease can be minimized by improved sonication near the plants.

7.3 *Phytophthora* Fruit Rot

Water soaked lesions appear on fruits which later become enlarged, light brown in colour and finally exhibit soft rot symptoms (Pathak, 1980). The fruits are infected directly or through wounds. The disease is caused by *Phytopthora palmivora* Butler which affects both the jack and bread fruits.

The disease can effectively be checked by spraying Bordeaux mixture (0.8%) or copper oxychloride (0.3%).

7.4 Leaf Spots

Leaf spots caused by *Colletotrichium lagenarium* (Pers.) Ell. & Holst. produces dark brick red spots on both the surface of leaf which upon maturity become greyish stud-

ded with dark coloured, pin headed fruiting bodies of the fungus. The margins of mature spots are dark brown. The disease can be checked by spraying 0.1% carbendazim or 0.2% captan.

Tandon and Bilgrami (1957) also reported leaf spot caused by *Phyllosticta artocarpina* Syd. & Butl. but this disease is not so serious in India. Small round yellow spot appear on leaves. The causal fungus survives for over six months on diseased leaves. The disease can be effectively controlled by spraying 0.3% copper oxychloride or Pereno.

7.5 Pink Disease

It is caused by *Botryobasidium solmonicolor* (Berk. & Br.) Venkat. The disease is wide spread in tropical and subtropical areas on jack fruit. Initial symptom appear as thin-pink colour powdery coating on lower and shaded sides of affected trees. The branches above infection sites lose their leaves and dry (Rawal, 1993). Young woody branches lose their leaves and die. Thin pink incrustations appear on lower side of leaves. Disease can be effectively managed by pruning affected branches, destroying them by burning and protecting cut surfaces by applying Bordeaux past or copper oxychloride.

7.6 Minor Diseases

Charcoal rot (*Ustulina zonata*), brown root (*Fomes noxius*), black root (*Rosellinia arcuta*), leaf spot (*Gloeosporium artocarpi*) and fruit rot (*Alternaria alternata*) are also reported as minor diseases on jack fruit (Saha, 2002).

8. Java plum or 'Jamun' (*Syzygium cuminii* Skeels)

8.1 Leaf Spot and Fruit Rot

The disease is caused by *Glomerella cingulata* (Stonem) Spauld. & Schrenk. Leaf spot and fruit rot is an important disease of jamun (Prakash *et al.*, 1975). Initially affected leaves show small scattered spots on lamina which enlarge to become oval or elongated and turn light brown or reddish brown. Black, raised fruiting bodies of the causal fungus appears on the spots. The leaf spots may coalesce to give a patchy appearance. Disease symptoms are not produced on midrib and petiole. Small water soaked circular or depressed lesions appear on affected fruits which later become dark and develop concentric rings with fruiting bodies of the pathogen arranged in the form of rings. Creamy spore masses develop on the fruiting bodies during humid conditions and affected fruits finally rot and shrivel (Bose and Mitra, 1990).

The disease can be checked by spraying 0.2% zineb or 0.8% Bordeaux mixture.

8.2 *Botryodiplodia* Leaf Spot

Leaves show oval or irregular brown coloured spots which later coalese to form bigger spots. The disease is caused by *Botryodiplodia variispora* (Died) Z. Ambettakis.

Earlier, Butler and Bisby (1931) reported this fungus as *Diplodia variispora* Died.

8.3 *Cercospora* Leaf Spot

The disease is caused by *Cercospora eugeniae* (Rangel) Chupp. The disease was reported from Nandi Hills, Mysore, India by Rangaswamy *et al.,*(1970).

Numerous irregular olinaceous brown or deep brownish spots of variable size on foliage are characteristic symptoms of the disease. The disease occur during rainy season and young leaves are more susceptible.

8.4 *Fusarium* Leaf Spot

Reddy (1975) reported the occurrence of *Fusarium acuminatum* Ell and Ev. from leaves of Jamun from Jodhpur (Rajasthan), India. Symptoms include browning of oval to irregular spots, sometime the infected portion detached from leaf giving a shot hole appearance.

8.5 *Alternaria* Soft Rot

The disease fruits showed water soaked lesions. These spots are pinkish in the beginning surrounded by white margins but in advance stages more than half of the fruit rot within 8 days and then turn black with slightly raised margins. The disease occur during July-August and is caused by *Alternaria alternata* (Fr.) Krissler. (Tewari *et al.*, 1988).

8.6 Anthracnose

The disease is caused by *Gloeosporium eugeniae*. Anthracnose is observed on fruits during July-August as small water soaked or pinkish depressions later turning blackish brown and spreading over the entire fruit causing heavy losses in India (Mishra *et al.*, 1973; Prakash *et al.*, 1975).

8.7 Post Harvest Diseases

Wadia and Manoharachary (1982) reported occurrence of post harvest diseases of Jamun fruits which include anthracnose (*Pestalotiopsis palmarum* (Cooke) Steyaert), soft rot (*Penicillium expansum* Link.), brown rot (*Rhizopus stolonifer* (Ehrenb. Ex. Fr.) Lind), Fusarium rot (*F. semitectum* Berk. & Rav.), black rot (*Aspergillus niger* Van Teigh) and rots due to *Curvularia lunata* (Wakker) Boedijn (*Cochliobolus lunatus*) and *Colletotricum gloeosporioides* Penz. (*Glomerella cingulata*).

8.8 Minor Diseases

Leafspots (*Diplodia variispora, Mycosphaerella bombycina, Phomopsis eugeniae and Salenophoma kamatii*); tarspot (*Phyllachora eugeniae*); wood rot (*Trametes*

persoonii) and sooty mould (*Meliola cladotricha*) diseases are also reported on jamun (Saha, 2002). Some minor fungal pathogen have also reported on leaves of jamun from different parts of India which are summarized Table 2 .

9. Karonda (*Carissa carandas* L.)

9.1 Anthracnose

Anthracnose is caused by fungus *Colletotrichum inamdarii*. The fungus perennates as mycelium under unfavourable conditions. The disease is widely distributed in Uttar Pradesh, India (Lal and Singh, 1953) and infection is stimulated by exudation from wounds caused by thorns. Pinhead size pinkish red spots appear on leaves which rapidly increase in size. Lesions are irregular, later turn brown and get surrounded by a red margin. Fruiting bodies develop on central region of the spots. The fungus infects uninjured leaves when inoculum is sporulated in host extract.

Spraying 0.1% copper sulphate is effective in controlling the disease (Lal and Singh, 1953). Alternatively, 0.2% copper oxychloride may also be employed for controlling the disease.

9.2 Bacterial Canker

The disease was first observed in 1962 at Poona, Maharashtra (Moniz *et al.*, 1964) later on, disease was reported widespread in Rajasthan (Kumar and Patel, 1968; Rathore, 1970). Symptoms first appear as small, round, water soaked, translucent spots on lower surface of leaves which gradually expand, turn dark brown and get surrounded by yellowish green halo. The spots on midvein cause browning and necrosis of the affected area. Spots are not generally delineated by small veins, but those near veins or midvein extending laterally are limited. The water-soaked spots on the under surface of the leaf enlarge and become raised. Spots may coalesce and turn brown. The central tissue of spots finally collapse and turn brown. The upper leaf surface over the spot may be slightly raised. Twigs show mild symptom, however, flowers and fruit do not show any symptom. The disease is caused by *Xanthomonas campestris* pv. *carissae* (Moniz *et al.*, 1964) Dye. Bacterium is a gram negative rod, motile with single polar flagellum and measuring 0.66x2.5 μm. Bacterium survive in diseased plant debris and enters the host mainly through stomata but can also enter through wounds as well. Maximum number of leaf lesions are observed in the third week of October in Jobner when temperature remains 24ºC and mean relative humidity around 56% (Rathore, 1970).

The disease can be managed by removing diseased leaves from the shrubs pruned to the height of 45 cm from ground level. Spraying the phytomycin (200 μg/ml) can also effectively check the disease (Prasada *et al.*, 1971).

9.3 Leaf Blotch

Leaf blotch incited by *Pseudocercospora* sp. is reported from Faizabad, India causing heavy damage. Symptoms include superficial dark coalescing blotches (which disap-

pear when rubbed) on the under surface of the leaf, causing chlorosis on the upper surface and defoliation.

9.4 Fruit Rots

Cochliobolus spicifer and *Rhizoctonia solani* were found on rotting fruits in Hyderabad and Secunderabad, India during post harvest survey (Wadia and Manoharachary, 1979). Fruit rots caused by *Myrothecium roridum* (Kumar and Tandon, 1978), *Pestalotiopsis versicolor* (Kumar and Tandon, 1982), *Aspergillus awamori* and *A. niger* (Garg and Gupta, 1980) are also reported from India.

Table 2: Some diseases of minor importance of Jamun

Pathogen(s)	Locality	Reference
Neocosmospora vasinfecta Smith; *Phoma glomerata* (Corda) Wr. & Hochapfel	Jobner (Raj.) India.	Goyal *et al.,*(1971)
Pestalotiopsis versicolor India.	Allahabad (UP)	Tandon and Bilgrami (Speg) stey. (1961)
Pestalotiopsis carissae Guba	Poona (Mah.) India.	Gopinath Nair (1964)
Pestalotiopsis eugeniae . Thuem	Poona (Mah.) India.	Rao (1963)
Macrophoma mangiferae Hingorani & Sharma	New Delhi India.	Waraiteh (1977)

10. Litchi (*Litchi chinensis* Sonn.)

10.1 *Armillaria* Rot

The disease causes considerable loss to litchi planting in heavy rainfall areas (Darvis, 1992). Initial symptoms of the disease include loss of vigour and absence of new growth which is followed by defoliation and subsequent death of the trees. During warm rainy summer season, the young plants often collapse suddenly and rapid girdling of trunk occurs when the pathogen is most active. Infected old trees may deteriorate for many years showing symptoms of a slow decline.

Armillaria rot is caused by *Armillaria mellea* (Vah) Quel. The pathogen enters and kill major roots first and then invades the trunk. In orchards, the disease spreads in a circle and is severe in stressed trees, such as planting in lands with a high water table

The disease can be checked within infested tree by trenching and fumigating with carbon disulphide. Excess of water should be avoided since it predispose litchi plants to rot. Infected plants should be removed and soil drenching with Bordeaux mixture and Bavistin gave good control. Biological control with *Trichoderma* spp. also provide good control of the disease.

10.2 Leaf Spots

Leaf spot caused by *Microdiploidia litchii* is a serious problem in Rajasthan (Pathak and Desai, 1971). Initial symptoms of the disease appear as yellowish brown to brick red areas mostly around the margins which gradually become light brown and show black dot like pycnidia.

Leaf spots caused by *Pestalotia pauciseta* Sacc., *Botryodiplodia theobromae* Pat. and *Colletotrichum gloeosporioides* Penz. have also been reported as mixed infection during rainy season (Prasad, 1962,1967; Srivastava *et al.*, 1964). The spots incited by these fungi measure 0.25-0.7 x 2-3 cm in early stages and lesions coalesce at the later stage. Younger leaves show less infection than older leaves. Initially, leaf spots are small but later many of them coalese and form large necrotic areas on leaf surface. These spots are common during July-December but pathogen may remain on twigs to cause infection of fruit pedicels and fruits. Pathogen survives through twigs canker, spots on leaves present on the tree and through fallen leaves.

Field sanitation, destruction of infected leaves combined with spray of 0.3% copper oxychloride can effectively manage the disease. Dithiocarbamate fungicides such as zineb and mancozeb also provide effective control of leaf spots.

10.3 Red Rust

The algal parasites *Cephaleuros mycoides* (Sood *et al.*, 1987) and *C. virescens* Kunze (Gupta, 1992) have been reported to cause characteristic symptoms of red rust. The infection reduces the vitality of the tree by hampering photosynthetic activity. Plant growth is retarded due to defoliation which indirectly effect yield. Small lesions of velvety growth appear on lower surface of young folded tender leaves. With the unfolding of leaves, these lesions enlarge on under surface later turning dark brown to brick red in colour, and the areas on upper surface just above the lesions become chlorotic. Old and thick leaves show various types of depressions, curling and ultimately become leathery and brittle. A survey of litchi orchards in the Kangra valley in Himachal Pradesh, India, revealed that red rust thought to be caused by a complex of an alga *Cephaleuros virescens*, a mite *Aceria litchii* and possibly a fungus and was most prevalent at an altitude of 750-950 m and the infestation ranged from 50 to 90% (Gupta *et al.*, 1997).

Cephaleuros sp. has also been observed occurring on the lower surface of leaves of litchi growing at Pinjore Garden near Chandigarh, India (Vinayak and Mandhar, 1994). The alga showed a typical heterotrichous habit consisting of the basal disc-like thallus on the under side and vertically upwardly rising branches from its upper side. The thallus was reddish brown in colour and had a velvety appearance. Leaf symptom appeared first on the margins and then progressed inwards affecting in severe cases, the entire lower leaf surface. Diseased leaves were characterized by inwardly directed margins, wrinkling and upwardly directed eruptions occurring irregularly and resulting in leaf distortion.

Six sprays of lime sulphur can effectively reduce the infection by over 90% (Sood *et al.*, 1987). Spraying 0.3% copper oxychloride followed by 0.2% captafol, 0.25%

ziram has also been reported quite effective when sprayed during July-September (Gupta, 1992). Four sprays of dimethoate (0.03%) or dicofol (0.05%) from February, at monthly intervals, for control of vector – *Aceria mangiferae,* pruning and burning of affected plant parts in January is recommended for the control of this disease (Gupta *et al.*, 1997).

10.4 Fruit Rots

Several fruit rot diseases have been reported by Prasad and Bilgrami (1973b) in India which includes rot caused by *Aspergillus flavus* Link. ex. Fr., *A. nidulans* (Eidam.) Wint., *A. niger* Van Tiegham*, A. quadrilineatus* Thom. & Raper, *A. varsicolor* Berk & Br., *Botryodiplodia theobromae* Pat*., Colletotricum gloeosporioides* Penz.*, Cylindrocarpon tonkinense* Bugh. and *Pestalotia* species. Prasad and Bilgrami (1969) observed that the organisms may get associated with fruits in the orchards and later may cause infection during transit or storage. Prasad and Bilgrami (1973a) examined effect of temperature and humidity on decay of fruits and found no infection occurred at 10°C. However, at 30% relative humidity, the rotting percentage was nominal. Infection during transit and storage occur through wounds. In some cases latent infection of fruits from the tree also. Litchi fruit rot caused by *Geotrichum candidum* Link ex. Pers. was also observed in Allahabad by Jamaluddin *et al.*, (1975).

Post-harvest disease can be reduced through precautions during picking and storage. One or two pre-harvest sprays of captan (0.2%) or copper oxychloride (0.1%) are effective in decreasing fruit rot after harvest. Pre-treatment of fruits with Aureofungin (500 µg/ml) is effective against all the fruit rots (Prasad and Bilgrami, 1975). Fruit dipped in water at ambient temperature and then in hot benomyl (0.05-0.2%) for 2 to 16 minutes is recommended for post harvest rot caused by *Aspergillus* and *Rhizopus* spp. and browning of litchi fruits (Huang and Scott, 1985). These fruits are air dried and packed loosely in sealed PUC bags. Two minute dip of fruits in hot water at 52°C control the rot caused by *Aspergillus* and *Rhizopus* (Barekai-Golan and Phillips, 1991). Fruit rot caused by *Colletotricum* sp. is reduced by dose of 75 and 300 Gy irradiation of fruits with no adverse effect on quality when stored for three weeks at 5°C (McLauchan *et al.*, 1992).

Storage rot caused by *Geotrichum candidum* controlled by dipping fruits for 10 minutes in nickel chloride (0.1%), nickel sulphate (0.1%) or captan (0.2%) and then storing them at 3.8-5.5°C (Yadav *et al.*, 1984). However, such a treatment need not be adopted in view of health hazards. Dipping litchi fruits in hot benomyl (0.1%) at 48°C for 1-3 minutes and 50°C for 1-2 minutes gave best control of fruit rot in Australia (Wong *et al.*, 1991) and most acceptable fruit appearance with least skin colour loss. In another report from Australia, treatment of fruits with hot benomyl at 52°C for 2 min and air dried prior to PUC wrapping and storage at 5°C also found effective in controlling the rots caused by *Cladosporium* and *Fusarium* spp. Dipping the fruits in solution of 0.25 g/litre prochloraz at room temperature (25°C) for 5 seconds to 5 minutes reduced the rotting (Brown *et al.*, 1984). Similarly, reduction in post-harvest decay was observed with SO_2 + prochloraz 0.05% + packing in Everfresh (Swart, 1990).

Good control of fruit decay was obtained with Nustar (flusilazol), Punch C

(flusilazol 250 g + carbendazim 125 g/litre) and Punch X (flusilazol 125 g + carbendazim 250 g/litre). However, browning is problem when flusilazol is used. The use of two slow releasing paper sheets impregnated with sodium metabisulphate, placed in outer edge of litchi fruits within the polythene bags, gave complete control of decay. Fruit dip in 1% semperfresh (a sucrose easter preparation) + 600 μg/ml benomyl + 250 μg/ml prochloroaz in combination with one foam sheet (as buffer) and one slow release sheet impregnated with sodium metabisulphite and storage at 3.5°C gave excellent control of decay and browning (Schutte *et al.*, 1990).

Treatment of fruits with SOPP (0.25%) followed by coating with wax emulsion (6%) increases the storage life of fruits by 4 days (Prasad and Bilgrami, 1973b). Litchi fruits treated with a culture solution of *Bacillus subtilis* and Duokangmeisu (an anti-fungal agent) maintained high quality for one month after treatment, with only 4.2% rotted fruit being recorded (Jiang *et al.*, 1997a). Jiang *et al.,*(1997b) reported the control of post harvest browning of litchi fruit by sodium bisulphite and hydrochloric acid. Fruits were soaked in 1% NaHSO$_3$ containing 0.1, 0.5 and 1% HCl for 2-30 minutes. Treatment of NaHSO$_3$ and HCL is an alternative method of preserving litchi red colour.

10.5 Brown Root Rot

Brown rot caused by *Phellinus noxius* (Corner) G. Cunn. was first reported from Taiwan during survey from 1990-96 (Ann *et al.*, 1999). Most trees with decline symptoms in central and southern Taiwan had brown rust with ages ranging from young seedling to 10 year old trees. Symptom include leaf discolouration, unthrifty appearance and eventual death. Fungus produce fruiting bodies artificially on sawdust medium that are similar to those produced on the lower stems of declining trees. Fruiting bodies have rarely found in the field.

10.6 Sour Rot

Geotrichum ludwigii and *G. candidum* are reported to cause sour rot of litchi in Taiwan (Tsai and Hsieh, 1998, 1999). Colonies of *G. candidum* were effuse, farinose and lanose. Hyphae showed dichotomous or trichotomous branching. Arthrospores were cylindrical, ellipsoidal, hyaline, smooth, 0-1 septate and 5-15 x 4-8 μm. The optimum temperature for arthospore germination were 29-32°C. Colonies of *G. ludwigii* were effuse and whitish. Arthrospores were cylindrical, ellipsoidal, hyaline, smooth and 5-35x5-10 μm. The optimum temperatures, for arthrospore germination were 28-32°C. The optimal temperatures for the growth of both fungus were 28-32°C and the optimal pH was 6-9.

10.7 Downy Mildew

Downy mildew causes heavy loss in China on cv. Huaizhi (Ou *et al.*, 1999). It is caused by fungus *Peronophythora litchi*. However, *P. litchi* isolate with downy blight symptom were also collected from various parts of Taiwan (Chen *et al.*, 1998) which cause disease symptoms on mature fruit via both wounds and non-wound inoculation suggesting that the disease could develop in field without any mechanical injury. Sporan-

gia were lemon-shaped with apparent papilla. The sprongiophores are dichotomously branched and the sporangia formed on a single sporangiophore mature simultaneously. The temperature for mycelial growth of the fungus ranged from 8 to 32°C with optimal temperature 24°C.

The disease is effectively controlled by spraying mancozeb 80 W.P. 6 times at 10-15 days interval with first spray commencing 7-10 days before flowering (Ou *et al.*, 1999). The disease is also controlled by using agricultural antibiotic 904 and Pulike, which provided 93.5-94.2% control (Yu *et al.*, 1995).

10.8 Dieback and Leaf Blight

Kang and Singh (1991) reported this disease caused by *Pestalotiopsis mangiferae* (Henn.) Steyaert. from Punjab. More than 85% litchi plants in and around Chandigarh got infected by this disease (Misra and Pandey, 2001) The twigs at the top show a naked look and plant dry from top downwards and leaves are blighted. Manicom (1995), also reported dieback of litchi caused by *Armillaria* sp. which may also appear in combination with other fungi such as *Fusarium, Botryosphaeria and Phomopsis* and causing loss of 6000 trees worth 3 million Rand in South Africa.

Not much work has been done on the control of this disease. Spraying of copper and dithiocarbonate fungicides early in the season may helps in reducing the disease.

10.9 Fruit Drop

Fruit drop is a serious problem in litchi. Besides other factors, disturbance in the endogenous hormonal level may be responsible for this problem.
Foliar sprays with zinc sulphate at 0.5, 1.0 and 1.5% concentrations of zinc can reduce the fruit drop (Awasthi *et al.*, 1975).

10.10 Fruit Cracking

This is physiological disorder where fruit starts cracking under dry conditions. This may be due to high temperature and relative humidity and these factors in combination with rapid flesh growth and relatively less elasticity of fruit skin. As a result of this problem fruits become unfit for marketing.

Proper irrigation during fruit development helps in reducing fruit cracking. Mulches applied around the tree trunk help in preserving moisture. Spraying with 2-4,5-T or NAA 35-100 µg/ml has been found effective in controlling fruit cracking and also increase the size of fruit.

10.11 Leaf Curl

Leaf curl disease is of considerable economic importance. The symptom of the disease is characterized by appearance of convex spots on upper surface and concave spots on under surface. The trouble has been attributed to mite attack. Constant association of *Ceuthospora litchii* sp. nov. have been reported with the diseased areas (Chona and

Munjal, 1956). However, role of fungus in causation of the disease remains to be investigated.

11. Loquat (*Eriobotrya japonica* Lindl.)

11.1 Wither Tip or Anthracnose

The disease is incited by *Colletotrichum gloeosporioides* Penz. Wither tip is characterized by withering of branch tips and shedding of leaves (Lele and Ram, 1969). Initial symptoms appear as green spots on leaves which later turn brown and such spots are generally produced and are pronounced at margins or tips of leaf lamina. In moist weather, acervuli appear as dot like bodies on twigs and leaves from which pinkish masses of spores ooze out on the surface.

Pruning of infected branches and protection of cut surfaces with chevastelon solution (6% cold solution of potassium dichromate mixed with 6% cold solution of copper sulphate for ready use) are effective in managing the diseases (Lele and Butani, 1975). Alternatively, pruned surface may be applied with Bordeaux paste. Application of Benomyl (600 µg/ml) also effectively controlled the disease.

11.2 Collar Rot

The infected area on stem base gets discoloured which becomes brown colour and later results in its complete girdling during March-June but not in July-September. Collar rot is primary caused by *Sclerotium rolfsii* Sacc. (Tandon, 1965; Morita, 1995). However, *Diplodia natalensis* Evans also infects invariably as a secondary organism (Tandon, 1961). The discolouration caused by *S. rolfsii* is brownish and lighter in colour than that caused by *D. natalensis*. The later attack the bark at collar region which turns brown, cracks and sometime peels off. The entire tree may be killed in due course of time.

Disease can be effectively managed by scrapping of infected bark followed by painting of exposed healthy portions with fungicidal paints (Randhawa and Singh, 1970).

11.3 Twig Blight and Canker

The affected trees give sickly and blighted appearance and devoid of normal foliage (Lele and Ram, 1969; Morita, 1991). The bark of the infected twigs is loosened and such twigs bear embossed, globose and cankerous pimples. Amber yellow coloured exudation becomes evident on the infection sites, which later dry to become horny. Disease is common in Delhi and Western Uttar Pradesh (Lele and Ram, 1969).
Twig blight and canker is caused by *Cytospora chrysoperma* Pers.

Twig blight caused by *Phytopthora cactorum* was also observed on young trees of loquat in Apulia, Italy in 1993 (Frisullo *et al.*, 1997). The aerial attacks of this typically soil borne pathogen probably occurred as consequence of persistent rain and wind. Pruning of infected twigs and point cut surfaces with fungicidal paints effectively control the disease (Lele and Bhutani, 1975).

11.4 Root and Foot Rot

In 1995, loquat trees growing in central Taiwan were infected with a disease causing wilt and death of plants due to severe foot and root rot caused by *Phytophthora parasitica* (*P. nicotianae* var. *parasitica*) (Chern *et al.*, 1998). The vascular tissues of all infected plants turned brown.

10.5 Die Back

Die back is caused by *Cytospora eriobotryae* Curzi and Barbaini. Singh *et al.,*(1969) have reported die back as an important disease of loquat. The disease is characterized by drying and death of the infected twigs. The bark of infected twigs became soft and sunken and later, epidermis peels off and twigs finally dry up. The infection then spread downwards, and may sometimes reach the main branch causing its drying.

10.6 Soft Rot

Two different soft rots caused by *Pestalotiopsis versicolor* (Speg.) Steyart and *Fusarium semitectum* Berk. were reported by Joshi *et al.,*(1983). In case of former small, dark brown and water soaked spots are formed on fruits which later become very soft and spore mass is found over the infected surface. However, in case of latter, though the lesions are smaller but completely destroy the fruits within 15 days and the entire soften surface of fruits is covered by pinkish white cottony growth of pathogen. Fruit rot of loquat in Amami Islands is caused by fungi such as *Pestalotiopsis* and *Colletotrichum* (Nojima *et al.*, 1995).

The occurrence of *P. eriobotryicola* in loquat causing reduction in yield and quality of fruit was reported by Liu (1995) in Taiwan. Disease becomes severe at 25-29°C from May-September in Taiwan. However, with disease severity >50%, plants defoliate prematurely, had fewer leaves and flowers on each branch and produce fruit with less weight and sugar content.

Hall (1983) reported benomyl fungicide effective in controlling the post harvest disease of loquat like Diplodia rot and rots caused by *Pestalotia* sp. and *Colletotrichum* sp. However, Koga (1999) reported the occurrence of benomyl fungicide low sensitivity strains of *P. eriobotryicola* and *P. neglecta* in Japan.

11.7 Grey Spots

The disease is common occurring in Taiwan. It causes grey spotted leaves, buds, stem, flowers and fruits. Liu *et al.,*(1991) have established *Pestalotia eriobotrycola* as the cause of this disease. The optimum temperature for germination and hyphal growth of *P. eriobotrycola* was between 20-28°C. Disease occurred most serious during June-October in Taiwan.

Spraying of 0.2% chlorothalonil or 0.05% carbendazim can effectively check the disease.

11.8 Fire Blight

Fire blight caused by *Erwinia amylovora* is reported to be serious disease in Israel (Miriam *et al.*, 1999). Artificial inoculation of fruiting orchards growing in the costal area of Israel showed that flowers were less sensitive than the wounds caused by thinning the flower clusters. Wounds were susceptible to the bacteria within first 24 hours after thinning.

Starner at 0.15% (Oxyolinic acid 20% WP) and Glycocide 2% (Hydroyacetic acid) were found most effective in controlling fire blight in artificially and naturally infected trees (Miriam *et al.*, 1999).

11.9 Brown Root Rot

Brown root rot incited by *Phellinus noxius* was reported from central and southern Taiwan during 1990-96 survey of wilted and dead plants (Ann *et al.*, 1999). Most trees with decline symptom had brown root rot with ages ranging from young seedling to 10 year old trees. Characteristic symptom of the disease include leaf discolouration, unthrifty appearance and eventual death. Fruiting bodies were rarely found in the field. Disease can be induced by replanting seedlings in infested soil or by contact with the diseased or dead roots.

11.10 'Tate-boya' Disease

A new disease is reported to occur on loquat fruit with Tate-boya symptoms, several longitudinal lines with splashed patterns on the surface of fruit from bract to tips, resembling an abrasion effect, hence the name Tate-boya (Morita, 1997). SEM examination of symptomatic fruit surfaces showed signs of the presence of the loquat rust mite *Aceria* sp. and conidia and mycelia of various filamentous fungi. No such symptoms were found on fruit from which bracts had been removed at the early stage of development. In bracteate cavities on diseased fruit, many loquat rust mites could be seen. *Botrytis cinerea* was most frequently isolated from the inside of bracts and from diseased parts of fruit.

Tate-boya symptoms were found only when both the *B. cinerea* and loquat rust mite were found together within bracteate cavities. The symptoms could be reproduced when conidial suspensions of *Botrytis* sp. were injected into bracteate cavities which were inhabited by the loquat rust mite. No other species of isolated fungi could reproduce the symptom.

11.11 Minor Diseases

The following diseases have also been reported on loquart from different parts of world which are summarized in Table 3.

Table 3: Some minor diseases of Luquat.

Name of the diseases	Pathogen(s)	Reference(s)
Leaf and fruit sports	*Pestalotiopsis guepini* from Argentina	Perello & Larren, (1999)
Leaf blight	*Diploearpon mespilli* from Srinagar (India)	Dar and Zargar, (1986)
Anthracnose	*Colletotrichum acutatum* from Kagothima and Chiba prefectories (Japan)	Sato *et al.,* (1997)
Stem canker	*Pseudomonas syringae pv. eriobotryae*	Kamiunten, (1995)
Leaf spot	*Alternaria erybotryae*	Saha, (2002)
Fruit rot	*Monochaetia indica, Pestalotia longiaristate, Aspergillus niger*	Saha, (2002)

12. Mulberry (*Morus* species)

12.1 Powdery Mildew

12.1.1 Occurrence

Powdery mildew is common in all the mulberry areas of the India and causes considerable damage to mulberry plants. (Gangwar *et al.,* 1994; Biswas *et al.*, 1995b) Leaf yield loss to the extent of 12.1-32.5% have been reported from Karnataka during 1995-96 and 24.02% coefficient of disease index in West Bengal. (Srikantaswamy *et al.*, 1998). The disease cause severe damage during rainy and winter season in Karnataka, India, causing white powdery patches on the abaxial surface of leaves making them unsuitable for feeding to silkworms.

12.1.2 Causal organism

The disease is caused by fungus *Phyllactinia corylea* (Pers.) Karst. Surface of the clavate, hyaline conidium is ornamental with evenly distributed spine like protrusions (Kumar *et al.*, 1998a). The conidia germinate 4 h after inoculation producing a single germ tube mostly from a little behind the distal end of conidium. The hyphae soon produce special branches, stomatopodia, which enter leaf through stomata. The stomatopodia are produced singly or in pairs, and 1 or 2 stomatopodia enter in a stoma. The branched superficial hyphae forms a mycelial mass within 72 h after inoculation. The disease becomes apparent at the conidial stage. The conidiophores are straight at early stages, but appear spirally coiled when the conidia are mature, which may aid for conidial detachment.

12.1.3 Epidemiology

Disease assessment keys for three major foliar diseases (powdery mildew, leaf rust and leaf spot) of mulberry that reduce leaf yield by 10-30% during rainy season in Mysore were prepared which will allow the precise quick and consisted assessment of disease in the field (Gunasekhar and Govindaiah, 1994). Vidyasagar and Rajasab (1997) indicated that dark brown spots caused by *Phoma mororum* on mulberry leaves occurred only when the leaves were also infected by *P. corylea*. In Darjeeling hills, during 1991-92 maximum disease intensity occurred in August-September followed by May-July with lowest in spring (March-April) (Misra and Das, 1998). Disease intensity increase with increasing temperature and humidity.

12.1.4 Chemical Control

The disease control is not practically advisable using Bordeaux mixture or lime sulphur since the fungicide affect the health of silkworms (Ramakrishnan and Sundaram, 1954). However, where rearing of silkworm is not the consideration, use of fungicides known to be effective against powdery mildew can be resorted too. Of these, Bavistin 0.2% can be successfully employed (Srikantaswami *et al* 1998). Bavistin applied once as a spray at 0.15%, 35-40 days after pruning appeared most effective (Biswas *et al.*, 1995b). Two sprays of dinocarp or carbendazim were found most effective and residual toxicity of these fungicides on silkworms and cocoons after 3, 6 and 9 days of spraying had no adverse effect (Govindaiah *et al.*, 1994).

12.1.5 Mycorrhiza

Disease incidence of powdery mildew, rust, leaf spot, bacterial blight and blight was reduced in plants inoculated with VA-mycorrhizal fungi, *Glomus fasciculatum* and *G. mosseae* in combination with 60 or 90 kg P/ha/year (Sharma *et al.*, 1995). Both fungi are equally effective for reducing disease incidence.

12.1.6 Botanical pesticides

Extracts of *Adhatodo zeylanica* was most effective in reducing the disease followed by extracts of *Azadirachta indica*, *Launaea coromandelica* and *Oxalis corniculata* (Biswas *et al.*, 1995a). Extracts of plants leaves of *Calotropis jigantea, Ocimum sanctum* (*O. tenuiflorum*) and *Tageles patula*, onion bulbs and ginger rhizomes can be used without causing any adverse effects (Kumar and Vijayan, 1999).

12.1.7 Varietal Resistance

Among the 10 varieties screened, in West Bengal, India, S-799 is most tolerant followed by C-1729 variety (Biswas *et al.*, 1995b). Among the 58 genotypes of mulberry screened, 19 varieties viz., Chinese white, Ichinose, Sanish 5, Kairy-oronezaemigaeshi (K.N.G.), Tsukasakawa, Limoncina, Serpentina, Italian Sarnal, Brantul Kashmir,

Zagatul, Chattatul, Botatul, Zanzabud, Lajward, Nadigam, Kokuso 21, Kairyorosa, Kasuga, Enshatakasulle (tropical region) were field resistant to powdery mildew (Fotadar *et al.*, 1998).

12.2 Yellow-net Vein

The disease was first observed in Kalimpong, India. Later, it was observed in other mulberry growing tracts of West Bengal. The characteristic symptoms of the disease appear in March-April as yellowing of leaf veins. However, interveinal areas remain green. The disease is easily transmissible by pen grafts and inarch grafts (Raychaudhuri *et al.*, 1961). Several *Morus* species, varieties and clones are susceptible to yellow net vein infection (Raychaudhuri *et al.*, 1965). The vector of the disease is whitefly, *Bemisia* sp. (Raychaudhuri *et al.*, 1966). The control of the disease therefore lies in the management of white flies.

12.3 Mosaic

A disease with typical mosaic symptoms, often associated with curling and puckering of leaves without any appreciable reduction in leaf size was first observed in Kalimpong, India (Raychaudhuri, 1962) but now, the mosaic is more commonly distributed in north Bengal. The mosaic can be transmitted by mechanical inoculations and by aphids *Rhopalosiphum maidis* and *Myzus persicae* and *Aphis gossypii* (Raychaudhuri *et al.*, 1962, 1965; Chatterjee and Raychaudhuri, 1963, 1965). The virus can withstand an exposure upto 50°C for 10 minutes but not at 60°C. Dilution end point is between 1:2000 and 1:4000 and longevity *in vitro* is 10 and 15 days at room temperature of 28-30°C (Raychaudhuri *et al,* 1965).

      The virus infects a large number of mulberry varieties and clones, however, varieties such as Inchinose and Kairyo-sezumequarshi of *Morus alba* L. and Oshinasho and Kosen of *M. latifolia* are resistant to mosaic disease (Raychaudhuri *et al.*, 1965). Control of the disease lies in the control of vector.

      A new disease with green mosaic symptom was observed on leaves of variety Kairyorosa at Mysore (Gupta, 1999). On establishment of grafts and new growth, 72.7% of 18 buds on diseased stocks showed typical symptoms, whereas all shoots from cutting of diseased plants exhibited symptom. Mechanical inoculation did not transmit the disease. An antibiotic treatment did not inhibit symptom development, suggesting that causatic agent was a virus and not a phytoplasma (Gupta, 1999).

12.4 *Cercospora* Leaf Spot

The disease is caused by *Cercospora moricola.* A leaf spot complex caused by *Myrothecium rodidum* in combination with *C. moricola* is also reported from India. (Qadri *et al.,* 1999).

      Cercospora leaf spot is most severe disease in Khowang in Assam according to Boruah *et al.,* (1998). Incidence of disease remains invariably high during August-September and low during May-June crop season. The mean leaf yield loss in south

India estimated for 3 years varied between 3.30-13.14% (1993-95) in the rainy season and 2.29-10.80% in winter season (Srikantaswamy *et al.*, 1999). Both incidence and severity of the disease were positively correlated with leaf yield loss. The cocoon yield loss at the highest leaf spot disease severity was estimated to be 55.58% in India (Qrdri *et al.*, 1999). The monitory loss in term of cocoon price due to quality deterioration was estimated to be 61.28% at the highest disease severity. Mulberry leaf spot also reduced fecundity and the hatching percentage of eggs.

The effect of cultivars, plant spacing and fertilizer dose on disease incidence was studied in Mysore, all cultivars showed equal infection and disease intensity was greater within close spacing (Sharma *et al.*, 1996). Biochemical alteration in protein, phenol, sugar, mineral and chlorophyll content induced by *C. moricol*a were studied by Srikantaswamy *et al.,* (1996). Implications of alterations on feeding value and quality of leaves to silkworm are also discussed.

Philip and Govindaiah (1996) found that resistant genotypes Kaliakuttai and Belidevalaya had more cuticular wax, thicker surface layer, few stomata per unit area, thick palisade layer and higher palisade index values and suggested that these factors play important role in resistance to *C. moricola* in mulberry. Kazoli and MS-2 genotypes also show resistant reaction (Philip *et al.*, 1995A).

Among twenty genotypes screened by Philip *et al.,* (1996) only 10 genotypes showed resistance, while remainder showed moderately resistant reaction. *Trichoderma harzianum* and *T. viride* were most antagonistic to *C. moricola* (Shivapratap *et al.*, 1996).

12.5 Brown Ring Spot

Brown Ring spot of mulberries caused by *Cephalosporium strictum* was first reported from West Bengal, India (Maji *et al.*, 1999). Of the 162 mulberry varieties tested by them, Kurimota, Rokouaso, Ichimse, Rosa and Mizusuwas were found highly susceptible to the disease.

12.6 Anthracnose

It is characterized by production of oval or circular light brown spots with reddish violet margin. Often few spots coalesce to cover larger areas followed by development of dot like fruiting bodies, the acervuli.

The disease is caused by *Colletotrichum dematium* and *C. gloeosporioides.* From Japan the disease has been reported due to *C. acutatum* (Yoshida and Shirata, 1999b). Fungus can over winter in infected or latently infected leaves and these leaves can be a source of primary infection the following year (Yoshida and Shirata, 1999a). Conidia in soil were not a significant form of over-wintering inoculum. Hence, elimination of fallen leaves in the field in autumn may be an effective way to prevent occurrence of the disease the following year. *C. dematium* produces phytotoxins and some components in mulberry leaves may be indispensable substrates for producing the toxins (Yoshida *et al.*, 2000).

12.7 Glucy Spot Disease

Initial symptom of the disease appear as spots measuring 1-6 mm diameter and in the later stages of infection the diseased portion often falls out leaving holes in the leaves. The disease is reported to be caused by *Cladosporium* sp.. Conidiophores are 40.5-67.5 μm long and 3.75-6.25 μm in diameter; conidia are ovoid, 5.00-6.25 μm in diameter and 5.00-9.38 μm long. Infection of isolated leaves in plastic bags at 18ºC was apparent after 25 h with 0.5 mm diameter spots evident.

12.8 Trunk Blight and Canker

Trunk blight and canker disease reported to occurs on 1-3 year old nursery trees and ornamental mulberry trees in Hungary (Vajna, 1999, 2000). The disease is caused by *Fusarium lateritium* f. sp. *mori* (*Gibberella baccata*) and its symptom, morphology, identification and control measures are described by Vajna (1999).

12.9 Root Rot

Fusarium solani causes root rot disease in mulberry. *F. oxysporum* and *Aspergillus niger* were also isolated from field of Kerala, Tamil Nadu, Karnataka and Andhra Pradesh, India during 1992 (Philip *et al.*, 1995b). Of 86 garden sampled, 43 were infected and percent infection ranged from 3 to 30 with average infection level of 14%. Carbendazim and mancozeb were very effective in controlling the various root diseases. Treating mulberry sapling with 0.1% carbendazim or dusting with 3-4 g of mancozeb is recommended for these pathogens (Philip *et al.*, 1995c). *T. harzianum* completely inhibit *R. solani* within 6 days (Kumar *et al.*, 1998b).

12.10 Rust

Rust disease of mulberry caused by *Periopsora mori* reported to occur in Karnataka, India during 1988-90 (Prasad *et al.*, 1997). In West Bengal and Mysore, India leaf rust disease is also reported to be caused by *Cerotelium fici* (Sharma *et al.*, 1995; Gunasekhar *et al.*, 1995).

Two sprays of Foltaf (captafol) or Kavach (chlorothalonil) at 0.2% reduced leaf rust severity by upto 50% and increased leaf yield by 28% (Gunasekar *et al.*, 1995). Treated leaves could be used for feeding silk as early as the first day after spraying, without any toxic effect. However, captafol should not be resorted for foliar spray in view of restrictions imposed by Central Insecticide Board.

The disease severity on leaves was significantly reduced by mulching and covering the soil surface with green manure crop of *Crotalaria juncea* (Prasad *et al.*, 1997). Extracts of *Adhatoda zeylanica*, *Launaea cormandelica, Oxalis corniculata, Celosia argentia* and *Chromolaena odorata* significantly reduced both leaf rust and leaf spot caused by *Pseudocercospora mori*. (Biswas *et al.*, 1995a).

Among the 25 genotypes screened under natural conditions cultivar, China Peking, Cottaneo, MS-2 and MS-6 were resistant, however, following artificial inocula-

tion only China Peking and Cattlaneo demonstrated resistant (Philip *et al.*, 1994a). Among the 20 genotypes tested against this disease at Karnataka during 1990-93 none of the genotypes were resistant to leaf rust but all showed moderate resistance (Philip *et al.*, 1996).

12.11 Leaf Blight and Spots

Leaf blight has been reported to be caused by *Fusarium solani* on sprouted cuttings, *F. pallidoroseum* and *F. lateritium* (*Gibberella baccata*) on leaves and buds respectively and *Alternaria alternata* in meristem and on established plants (Philip *et al.*, 1994b). *F. pallidoroseum* and *F. moniliforme* var. *intermedium* causes leaf blight and *F. oxysporum* causes leaf spot diseases. Leaf blight symptom caused by *A. alternata* were also observed in Punjab (Verma and Kapur, 1995).

Leaf extract (1.5) of *Parthenium pinnata* were highly fungitoxic to *F. pallidoroseum* and *F. moniliforme* var. *intermedium* inhibiting mycelial growth by 78.2 and 84.3%, respectively whereas *Calotropis gigantea* and *Azadirachta indica* were most effective against *F. oxysporum* inhibiting its mycelial growth by 78.5 and 73.2%, respectively (Gupta *et al.*, 1996). Under green house conditions, aqueous leaf extracts (25%) of *P. pinnata* reduced the incidence of leaf blight caused by *F. pallidoroseum* and *F. moniliforme* var. *intermedium* by 63.6 and 67.1% respectively whereas in the case of leaf spot caused by *F. oxysporum, C. gigantea* and *A. indica* were both effective in reducing leaf spot incidence by 60.2 and 57.2% respectively.
Recommended control measures include field sanitation, wide spacing (3 ft x 3 ft) and drench applications of green leaves covering with soil between the rows to encourage the growth of antagonists such as *Trichoderma*. Chemical control should be carried out 15 days after each pruning or leaf harvest, spraying 0.2% Foltaf or Dithane M-45 on both leaves and surrounding soil (Philip *et al.*, 1995b). Philips *et al.,* (1994c) also suggested the control measures against leaf blight disease.

12.12 Bacterial Diseases

A severe bacterial blight of mulberries was observed in eastern Anatolia region of Turkey (Sahin *et al.*, 1999). The pathogen was identified as *Pseudomonas syringae* pv. *mori*. Pathogen multiplied on mulberry leaf surface and particularly colonized the cystoliths and growth of vein (Gupta *et al.*, 1995). Bacterium invaded leaf tissues through cystoliths and did not enter through stomata and trichoms.

Yoon *et al.,* (1994) also reported *Erwinia carotovara* infecting leaves, shoots and branches of mulberry and *Pseudomonas syringae* infecting leaves, shoots but not branches from rotted branches following harvesting for silkworm feed in autumn in Korea.

12.13 *Botryodiplodia* Root Rot

A new root disease caused by *Botryodiplodia theobromae* was observed in Tamil Nadu, India (Radhakrishan *et al.*, 1995). Affected tissue show dark discolouration and

young tissue showed blackening and necrosis of root system.

12.14 Black Leaf Spot

In June, 1992, black leaf spot a new disease of mulberries was observed in Karnataka, India caused by *Colletotrichum gloeosporioides* (*Glomerella cingulata*). This is the first report of this pathogen infecting mulberries (Philip *et al.*, 1994d).

12.15   Black Mildew

A black incrustation was observed on the foliage of mulberry during the winters of 1991-92 in Berhampore, West Bengal, India (Teotia, 1995). Symptoms were character-ized by the presence of a thick, black velvet coating on the upper leaf surface. The disease is caused by *Meliola amphitricha* and is new record in India.

12.16 Dwarf Disease

This disease is caused by mulberry dwarf phytoplasma. Dai and Sun (1995) observed the suppressive effect of N-triacontanol on symptoms of mulberry dwarf disease.

12.17 Other Minor Diseases

The occurrence of foliar disease of mulberries, caused by *Pestalotiopsis disseminata* (Thum). Steyalrt was first noticed in Kerala, India during 1992 (Philip and Govindaiah, 1995).

      Phoma multirostrata and *Glomerella cingulata* are reported on mulberry roots at Anantapur and on mulberry leaves at Mysore, India, in 1992 (Philip *et al.*, 1994b). *Coniothyrium foedans* on mulberry at Kalimpong, India during 1991 was described by Misra *et al.,* (1994).

      Fungi, *Botryodiplodia theobromae, Fusarium solani, Phoma mororum* and *P. sorghina* causes serious diseases like stem canker, cutting rot, collar rot and die back on mulberries. Soil solarization increased the soil temperature by 8-11°C and results in the reduction in the inoculm level of these pathogens in the soil, which reduced disease incidence by 64.5% (Gupta *et al.*, 1999). The survival and growth of sapling were improved by 41.6 and 38.6%, respectively.

13. Passion fruit (*Passiflora edulis* Sims.)

13.1 Leaf Blight and Damping Off

The disease has been reported by Ullasa and Sohi (1975). The damping off symptoms are seen in the nursery seedlings which show irregular, water soaked areas at the collar region resulting in damping off. During prolonged wet weather conditions disease becomes severe and causes defoliation in grown up plants and death of the seedlings.

Initially symptoms appear on the lower most leaves and then on upper leaves, flowers and young fruits. The affected areas turn discoloured to dull green and finally leading to defoliation and shedding of flowers and fruits. Leaf blight and damping off is incited by *Phytophthora nicotianae* Br. DeH.

13.2 Powdery Mildew

The occurrence of the disease was recorded on leaves by Sundaram (1961). The affected leaves show irregular, pale yellow discolouration on the upper surface and whitish growth of the fungus on the lower leaves. The disease is incited by *Leveillula taurica* (Lev.) Arn.

13.3 Fruit Rot

Penicillium expansum, Aspergillus niger, Fusarium oxysporum and *Rhizopus stolonifer* are reported to cause decay of fruits in storage. Packaging and storage of fruits in polythene bags and wooden crates treated with 5% Lysol can check the fruit decay caused by these fungus.

13.4 Collar Rot

Collar rot is serious disease of passion fruit in Uganda and development of passion fruit production industry in Uganda is seriously hindered by the outbreak of this disease (Ssekyewa *et al.*, 1999b). The disease is limited to warm low land areas of Uganda. The disease is caused by *Fusarium solani*. Fungus grows best at 25-30ºC temperature and virulent isolates in soil results in the limited yield and production of small purple passion fruits. Factors like soil pH, inoculm density and grown species could influence the disease progress (Ssekyewa *et al.*, 1999b).

Ssekyewa *et al.,* (1999a) developed sustainable environmental friendly collar rot control package. Chemical control was found economical when copper oxychloride (60 g/20 litre) was applied as drench. Rovral 70 WP (iprodiome) and Cercobin 50 L fungicides were also found effective. Two species, *P. edulis* f. *flavicarpa* and *P. maliformis* were partially resistant to collar rot and can be recommended for use as root stock to susceptible mulberries.

From India, Sawant *et al.,* (1995) reported the *Rhizoctonia solani* as the causal organism of collor rot of passion fruit in Karnataka. *Trichoderma harzianum* controlled the disease while *Gliocladium virens* had no effect. Tolclofos-methyl (Rizolex) and carbendazim (Bavistin) fungicide effectively controlled the disease.

13.5 Wilt and Collor Rot

Wilt and reddish-brown collor rot caused by *Phytophthora parasitica* (*P. nicotianae* var. *parasitica*) of yellow passion fruit (*P. edulis* ssp. *flavicarpa*) has been reported from central Venezuela during 1991-98 (Gonzalez *et al.*, 2000). Wounded and unwounded uninfected passion fruits seedlings developed chlorosis and collar necrosis 15 days

after exposure to fungal suspension, and mortality reached 50% after one month.

13.6 Base Rot

Base rot disease is caused by *Fusarium oxysporum* f. sp. *passiflorae* and the disease develops between March and December in Fujian (Zheng and Huang, 1997). Thiophanate-methyl (70 WP) was found best in controlling the disease.

13.7 Anthracnose

Anthracnose is an important disease of passion fruit. Symptoms on fruit initially appear as light brown spots that later become necrotic due to acervuli emergence and mycelial growth emerging from these structures.

Disease is caused by *Colletotrichum gloeosporioides* (*Glomerella cingulata*)Two strain of *G. cingulata* were identified as the cause of anthracnose in Venezuela. These strains are perithecial type strain (PTS) and sexual (conidial) chromogenic type strain (ACTS). Mycelial disc on the wounded fruit epidermis is most efficient inoculation method for evaluation of isolates pathogenicity and more than two inoculation methods must be used for isolate evaluation (Rocha *et al.*, 1998). Oat meal agar with yeast extract culture media appeared best for sporulation (Francisco *et al.*, 1994).

Highest conidial germination occurred at 30 and 33ºC. Fungus usually produce perithecia on media and also formed accruuli (Cedeno *et al.*, 1993). Conidia immersed in pink or orange-coloured masses. Ascospores measured 14-20 x 4-6 µm. On infected fruit produce only dark-coloured, subepidermic and setous acervuli in which chromogenic masses were not detected, probably due to the scarce conidia formation. Occasionally some setae were seen in the fruiting body.

Trichoderma koningii (Ti2, Ti17, Ti25) and *T. harizianum* (T-25) were found effective against *C. gloeosporioides in vitro* and under field and post harvest conditions (Rocha and Olivera, 1998a,b). All the *Trichoderma* isolates controlled the pathogen in fruit, limiting the necrotised area around wounds, however, Ti17 remained in the phylloplane for 30 days.

13.8 Web Blight

Web blight was reported to cause leaf drying on yellow passion fruit trees in orchards at Brazil in 1998 (Poltronieri *et al.*, 1999). Initially small, round, light green water soaked spots are observed on the leaves. These spots increased in size and coalesce to form irregular necrotic areas, surrounded by a chlorotic halo. Fungal hyphae grow on the lesions and extend very quickly to healthy tissue, as leaves are held together by a mycelial thread, on which microsclerotia were found.

The fungus, *Thanatephorus cucumeris*, perfect state of *Rhizoctonia solani* causes the disease. Fungal mycelium is light brown and thin walled hyphe branched at 90ºC angle.

13.9 Bacterial Blight

A bacterial blight disease was found on passion fruit in Cauca valley state Colombia (Castillo and Granada, 1995). The disease caused by *Xanthomonas campestris* pv. *passiflorae*, is also the most important disease of this crop in Brazil and is considered a serious restriction for its commercial cultivation (Beriam *et al.*, 1998). Genotypic characterization of *Xanthomonads* strains isolated from passin fruit and their relatedness to different *Xanthomonas* species was studied by Goncalves and Rosato (2000).
Spraying before and after inoculation of Agrimycine 100, Agri-step 500 (1 g/litre) and Kocide 101 (3g/litre) gave some preventive control (Castillo and Granada, 1995).

13.10 Cucumber Mosaic Virus (CMV)

Barbosa *et al.,* (1999) reported the occurrence of cucumber mosaic virus (CMV) in passion fruit from Parana, Brazil. Golden passion fruit plants with bright yellow spots were found in fields of Parana. Disease incidence was usually 30-40%, and >90% in severe cases during 1988-95 in China (Xu *et al.*, 1999). Infected plants had ring spot, mosaic, ring spotted mosaic and leaf distortion, tip necrosis and woody fruits. *Passiflora* spp. infected with CMV were sub grouped by the host reaction to indicative plant AAS-ELISA using monoclonal bodies. Thirty five isolates belong to CMV sub group I and 1 isolate belonged to CMV subgroup II (Xu *et al.*, 1999). Isolates of CMV subgroup I were predominant in the field.

13.11 Passion Fruit Woodiness Virus (PWV)

Passion fruit woodiness potty virus was first isolated from hybrid cv. Ohdama in Japan (Iwai *et al.*, 1996). The disease showed systemic mosaic symptoms on the leaves and produced severely malformed and woody fruits. The isolate designated as PWV-AO was transmitted by *Myzus persicae* in non-persistent manner. This Amami Ohshima isolate (PWV-AO) was subjected to CDAA cloning and the nucleotide sequence of its coat protein gene was determined (Iwai *et al.*, 1997).

Bezerra *et al.,* (1995) also identified a strain of passion fruit woodiness virus (PWV) from Brazil which induced mosaic in golden passion fruits, without causing woodiness of fruits. This virus was transmitted by *Aphis faba* from passion fruit to *Macroptilium lathroides* in a non-persistent manner. PWV was found infecting about 20% of passion fruits in plantation of two counties of Para and Brazil was introduced through seedlings imported from states of Minas Gerais and Bahia (Trindade *et al.*, 1999). Possible use of indirect DAS-ELISA for screening of passion fruit tolerant to passion fruit woodness virus was suggested by Novaes and Rezende (1999).

13.12 Crinkle Poty Virus (PCV)

A new virus, designated as passion fruit crinkle poty virus (PCV) was isolated from passion fruit cultivar Tainling No. 1 causing crinkle symptoms on foliage (Chang *et al.*, 1996). It was identified as potyvirus on the basis of particle morphology, aphid trans-

mission and the ability to induce cytoplasmic cylindrical inclusions (CI) in infected cells. It has wide host range than passion fruit mottle virus (PaMV) and PWV.

13.13 Passiflora Latent Carlavirus (PLV)

Occurrence of carlavirus was wide spread in commercial passion fruit planting in New South Wales and Queensland and present in Australian passion fruits for more than 10 years. (Pares *et al.*, 1997).

The particles of virus were flexuous rods with mean diameter of 651 x 12 nm. The particles often occurred in cells as aggregates but were never associated with pinwheel inclusion bodies, as it typical with PWV.

13.14 Green Spot

Kitajima *et al.,* (1997) reported green spot of passion fruit, a possible viral disease associated with infestation of mite, *Brevipalpus phoenicis* in Brazil. The disease is characterized by necrotic lesions in stem and green spots in mature fruits and senescent leaves, usually resulting in death of the plants. This condition is associated with heavy infestation of *B. phoenicis* and the death of plant was caused by girdling of stem by the confluence of the necrotic lesions. In extreme cases orchards were completely destroyed. Electron microscopy examination suggested that disease is caused by virus similar or related to citrus leprosis rhabdovirus (Cit LV). Spraying miticides in the early phase of *B. phoenicis* infestation gave good control.

14. Phalsa (*Grewia asiatica* L.)

14.1 *Cercospora* Leaf Spot

The disease appears during the month of July-August as tiny lesions on the both sides of the leaf which later get covered with a white crust of fungal growth. The patches rapidly enlarge and turn blackish in the centre. Gradually, the black growth become cusion-like consisting of mycelium and spores.

The disease is caused by *Cercospora grewiae* (Srivastava and Mehta, 1951). *Fusarium semitectum* is reported as mycoparasite on *C. grewiae* which causes leaf spot in Uttar Pradesh (Rathaiah and Pavgi, 1973)

Two or three spraying of Dithane M-45 (0.2%) is effective in controlling the disease. Spraying Aureofungin (30 μg/ml) and benlate (600 μg/ml) is also effective in reducing number of lesions and extending period of retention of leaves on plants.

14.2 *Phyllosticta* Leaf Spot

Phyllosticta grewiae fungi have been reported to cause leaf spot disease (Chona and Munjal, 1955). Occasionally the disease comes in severe form and causes considerable damage to this fruit crop. Symptoms appear in the form coloured spots, which become circular measuring 3.5-9.0 mm in diameter, amphigenous, scattered, sometime coalesc-

ing and involving the whole leaf which become purplish brown in colour changing to dark brown. On the upper surface of leaf, their center are white ash coloured. Small round gregarious pycnidia which are hypophyllous form on these spots. No effective remedial measures against this disease have yet been found.

14.3 Rust

Dasturella grewiae (Pat. & Har.) Thiram. fungi have been reported to cause rust disease from Bombay (Payak, 1953). The disease occur during the month of October-November in very severe form in Hisar, Haryana. However, Verma and Kapur (1995) have encountered rust caused by *Cerotelium fici* in Ludhiana, Punjab.

The disease appears in the form of yellow to light brown colour pustules having uredospores. The severely affected leaves start drying followed by defoliation. Two or three sprays of Dithane M-45 or Dithane Z-78 (0.2%) at weekly interval effectively control the disease (Gupta and Madaan, 1985). Dithane M-45 was also found best of the 6 fungicides tested in western Uttar Pradesh (Gupta, 1986).

14.4 Bushy stunt disease

A bushy stunt disease of phalsa caused by mycoplasma is reported by Rishi and Raychaudhary (1981). The twigs of the plants show stunty and bushy appearance with smaller internode, leaves and flowers forming a cluster. Either there is no fruit setting on these twigs or remain very tiny and do not ripe. The suppression of symptom by tetracycline and observation of mycoplasma like bodies within the sieve tube cells has been observed.

14.5 Other diseases

Some other diseases reported on *Grewia asiatica* L. are Powdery mildew caused by *Oidium grewiae* sp. nov*., Rust caused by *Phakospora grewiae (Pat and Har.)* Cumm. from Jabalpur, India, Sharma and Jain, (1981), Sclerotium soft rot caused by *Sclerotium rolfisii* (Saxena *et al.,* (1981) and Leaf spot caused by *Phoma sorghina* from Rajasthan,India (Shivpuri and Mishra, (1982).

15. Pineapple (*Ananas comosos* (L.) Mer.)

15.1 Base Rot, Fruit Rot and Leaf Spot

15.1.1 Occurrence

Base rot of pineapple is a serious disease prevalent in area having humid and tropical to subtropical climatic condition. In India, the disease frequently occur in Assam (Chowdhury, 1945a). The pathogen causes three types of symptoms *i.e.* base rot, leaf spot and fruit rot. Base rot is the most serious phase which destroy upto 10% of the crop whereas, fruit rot phase affects 3-15% of the fruits in the field. This phase attains

serious status during transit and storage. However, leaf spot is of less economic impor-
tance. In Brazil, it is reported to be one of the most important diseases (Costa and
Chalfoun, 1998).

15.1.2 Symptom

Initial symptoms appear as large grey spots on leaves with dark margins which turn
olive brown or white. As the disease develop, tissues dry and leaves become distorted.
Infection on suckers do not allow them to develop normally after planting and such
plants do not respond to normal functioning of roots.

Leaves on such plants/suckers remain under developed, show yellowing and
start withering. Plant exhibiting such type of symptoms show rotten black area on the
base of stem. Eventually, the plant/sucker starts dying due to extensive rotting of the
base. This is the base rot phase which is most serious as it does not allow the proper
stand of the crop.

Water soaked lesions appeared on fruits which turn yellowish, dark and affected
tissue rot. The stem end rot and soft rot of pineapple have been described by Mehta
(1940). At times, the disease develop so fast that the entire plant may turn dark and rot
within 2-3 days. It is covered with a mass of macrospores of fungus.

15.1.3 Causal Organism

The disease is caused by *Ceratocystis paradoxa* (Dada)*C. Morean* [(*Thielaviopsis
paradoxa*(Deseynes) Hohnel)]. The mycelium is hyaline or light brown. The fungus
produces two types of conidia-macroconidia and microconidia.

Microconidia are produced endogenously and are pushed out of the micro
conidiophors in chains. These are of two types – hyaline and light brown. Hyaline ones
are smooth, cylindrical to oval and thin walled and brown ones are barrel shaped and
thick walled. The micro-conidiophores are slender, septate, arising laterally from the
hypae, hyaline to pale brown, phialidic, tapering towards the apex, with short cells at
the base and a long terminal cell.

The macroconidia are produced on shoot lateral macro conidiophores in long
chains and are obovate to oval, smooth, dark brown and thick walled. Perithecia are
partly or completely immersed, light brown, globose, ornamental with numerous stellate
or coralloid appendages and the neck is long upto 1400 μm, tapering, black at the neck
and pale brown towards the tip. Ascopores are ellipsoid, 1-celled, often with unequally
curved sides and smooth walled.

15.1.4 Epidemiology

The pathogen is capable of surviving in the soil for a long period and invades the host
tissue through wounds and cut ends of suckers. It then spreads inter and intra-cellularly.
Suckers collected from the infested area carry disease with them to new ones. Seprophytic
activity of *C. paradoxa* was decreased with increasing soil depth (Milanes and Herres,
1994).

15.1.5 Management

Destruction of affected plants and planting disease free suckers can check the spread of disease. Exposure of harvested fruits to sun for two hours (Pathak, 1980) and dipping the cut end of fruit stalk in 10% benzoic acid in alcohol checked the disease. Packing cases and baskets should be sprayed with 3% solution of formalin well in advance. Since the disease is soil-borne, disinfection of nursery soil with Brassicol or Vapam and dipping of suckers in Bordeaux mixture (2:2:50) or borax before planting has been advocated by Raychaudhuri and Lele (1970). Post harvest dipping of fruits for 5 minutes in benomyl (200 µg/ml) or TBZ (1000 µg/ml) has been reported to reduce storage decay (Sridhar, 1975). Dusting of fruits with benzoic acid (0.1%) coated in kaoline also reduces this disease in transit (Mallikarjunaradhya *et al.*, 1979).

Among the 16 plants extracts tested against the causal agent of fruit rot, *Xanthium strumarium* extract was most effective followed by garlic extract (Damayanti *et al.*, 1996). Other extracts effective in decreasing order were *Meriandra bengalensis, Mentha piperita, Curcuma longa, Phylogacanthus thyrsiflorus, Toona ciliate, Vitex negundo, Azadirachta indica, Eupatorium birmanicum, Ocimum sanctum* and *Leucas aspera*. Post harvest gamma irradiation of fruits is highly effective against the fruit rot but is not practically feasible.

15.2 Sclerotium Wilt

15.2.1 Symptom

Sclerotium wilt is an important disease which causes considerable damage to pineapple crop in India. Singh (1972) reported this disease for the first time from Bangalore. It is characterized by yellowing and wilting of the affected plants. The infection appears on stem near the soil level which becomes water soaked, soft and later turn brownish. Mycelial growth appears on affected stem and on the soil around the plant which later disappears leaving behind masses of small, round to ovoid, light to dark brown coloured, hard sclerotia near the collor region. The disease spreads both upward and downwards covering the plant parts with mycelial growth and farming sclerotia. Root zone invasion leads to wilting and ultimately death of the plant.

15.2.2 Causal Organism

Disease is incited by *Sclerotium rolfsii* Sacc., which produces abundant, white, fluffy, branched radiating mycelium. Aerial hyphae appear as dense tufts and sometimes aggregate into strands or rhizomorph like structure. But individual hyphae are hyaline, thin walled, sparsely septate when young. The broader hyphae may have clamp connections at the septa whereas slender hyphae often lack clamp connections. Number of nuclei per cell is variable. Sclerotia are formed from hyphal strands which consists of 3-12 hyphae lying parallel. A spherical shape is soon assumed even if it is only a loose mass of hyphal network. With further hardening, differentiation takes place and the sclerotium shows an outer layer of polyhedral cells surrounding the compacted hy-

phae. Rind, cortex and medulla are differentiated as the sclerotium darkens. Basid-
iospores occur occasionally under humid conditions. The fungus is polyphagous and
has been reported to have very wide host range.

15.2.3 Epidemiology

The fungus is soil borne and survive as sclerotia for long period and also as mycelium
in infected tissues or plant debris. The fungus enter the host either directly or through
wounds and its subsequent spread and production of mycelium and sclerotia is rapid
particularly during high moisture and temperature conditions. The fungus seems to
grow, survive and attack plants best near the soil level, which may be due to prevalence
of favourable microclimate in the region.

15.2.4 Management

Disinfection of nursery soil with formaldehyde accompanied by dipping of suckers in
Bordeaux mixture (2:2:50) a little before planting helps in checking the spread of disease.
Keeping the plants healthy and providing good drainage and other optimum soil con-
ditions helps to avoid the disease. Deep ploughing of the fields to burry the plant into
the soil also helps in the reduction of inoculum. The management of sclerotium wilt is
difficult and depends partly on cultural practices and partly on application of fungi-
cides.

15.3 Heart or Stem Rot and Wilt

15.3.1 Occurrence

The disease was first reported from India by Chowdhury (1945b,1946) and is prevalent
in the lower Pulney hills, causing severe losses in regions with high rainfall and low
temperature where pineapple is grown as mixed crop with mandarin (Prakasam and
Subbaraja, 1994). Although sporadic in the nature of its occurrence in Assam (India),
the disease causes 7-25% mortality of plants in the affected area. Heart rot generally
appear in newly planted pineapples and occasionally in developing plants.

15.3.2 Symptom

Initial infection appears at the apical end of stem, at the base of leaf, at the basal end of
the stem or at the roots when it causes loss of turgidity and a slight twisting of ventral
leaves. However, at later stages, withering and yellow or brownish discolouration are
observed. Inner whorl of leaves can be readily detached from the stem by slight pull.
The affected stem show brownish discolouration of the margin between healthy and
diseased tissue.
     The wilt is common during the period of heavy rain and occurs in 1-2 years old
plantings. The growth of plants cease and leaves look like almond coloured, flaccid and
dropping followed by wilt. Roots are affected even before the leaf symptoms appear

which is cause of above ground symptoms. The growth of fruit ceases and its colour also changes before ripening. The affected fruits are spongy and salty in taste.

15.3.3 Causal Organism

The fungus, *Phytophthora nicotianae* var. *parasitica* causes this disease and survives in soil. In poor drainage areas the disease incidence is more. Plants propagated from slips are more susceptible than those from sucker.

15.3.4 Management

Removal of infected plants, planting of the suckers in well drained soil can check the disease spread. Dipping and spraying of plant material in Bordeaux mixture (1%) or copper oxychloride (0.25%) is also effective in controlling the disease (Parkasan and Subbaraja, 1994). Application of fosetyl Al as a pre-plant dip protects the plants against heart rot for 18 months (Cohen and Coftey, 1986). Spraying with 2-(chlorophenoxy) propionic acid 37 days before harvesting helps in reducing black heart (Kruger *et al.*, 1998). Some species of *Ananas* are resistant to the disease while *Psudanas* is immune (Pathak, 1980). Relatives to C13 (Cayenne Queensland Clone 13), intergroup hybrids 73-50 and 53-116 had some level of field resistance to black heart disease in subtropical Queensland (Sanewski and Giles, 1997). Exposing pineapple crown buds, later buds, suckering buds and rhizomes to sunlight for 4-8 hours at a temperature of 33°C also reduce heart rot.

Reducing the storage temperature of pineapple by 3°C by per day from harvesting temperature showed the greatest promise for reducing the black heart (Kruger *et al.*, 1998). Black inhibitor No. 1 and No. 2 was effective in controlling black heart disease and also have positive effect on quality of treated fruits of pineapple (Luo and Zhong, 1998). Effect of soaking treatment on black heart and storage quality of winter pineapple stored at 12°C are described by Tang (1996).

15.4 Leaf Blotch

Leaf Blotch disease was reported to occur in Karnataka (Rao and Mahaskar, 1973). The disease is characterized by appearance of pale brown circular to irregular small necrotic lesions on the leaves mostly originating from the tip of central part which soon enlarge and coalesce, turn dark brown, rough with irregular outlines and slightly depressed at the centre. At this stage, the leaves turn slightly yellowish and exhibit blotch appearance and numerous black sub erumpent fructification of the casual fungus, *Pestalotia microspora* develop on affected areas.

The disease has been found associated with cool humid weather. Its other host plants are oil palm, apple and guava. Optimum temperature for sporulation of the fungus in culture was 25-28°C with optimum humidity between 75 and 100%.

The disease can be controlled by spraying 0.2% Dithane M-45 or 0.2% chlorothalonil or 0.4% Blitox.

15.5 Anthracnose

The disease has been reported from Maharashtra (Garud, 1968) and initial infection appears in the form of irregular blotches with minute fruiting bodies originating mostly from the apex or tip of leaves. Marginal spotting is also seen occasionally. It results in withering and dropping of leaves and ultimately the growth of infected culms and development of fruits is seriously affected.

The disease is caused by fungus *Colletotrichum ananas* and favoured by high humidity and takes about fortnight to develop on young leaves. Black specks on leaves are also observed during June-July which leads to withering. The disease can be effectively checked by spraying 0.1% Bavistin or 0.1% Topsin M or 0.2% chlorathalonil.

15.6 Pineapple Wilt

15.6.1 Occurrence

Pineapple wilt of viral etiology is known to occur in all the areas of world wherever pineapple is cultivated. From India, the disease was first recorded by Singh and Shastry (1974). Association of mealy bug with the wilt was suggested by Illingworth (1931) and later Carter (1933) reported definite evidence of the relationship between mealy bug feeding and the wilt disease on pineapple.

Severe infection results in root collapse and the wilted plants topple invariably have poor root system which results in considerable loss due to poor yield.

15.6.2 Symptom

Initially leaves develop characteristic browning starting from third to fourth whorl on-wards and leaves show bright pink colour and browning of the tips with downward curling of the margins. The pink colour becomes more pronounced and drying of leaves from top downwards becomes more evident.

Finally, the tips dry up completely, the bright pink colour becomes dull and root system collapse. There is no fruit formation if the plants are infected in the early stages. Variation in symptom may occur depending upon the season.

15.6.3 Causal Organism

The disease is caused by pineapple wilt virus, a capillovirus which has non enveloped flexuous filament 1200-1500 mm long and 12 mm wide particle having dsRNA (Rohrbach *et al.*, 1988). *Mealy bugs* (*Dysmicocus brevipes* CKL.) transmitted the disease from wilting plant to healthy one in semi-persistent manner by mechanical transmission.

Dassanayake *et al.,* (1994) used ELISA for detection of pineapple wilt virus in Sri Lanka. Hu *et al.,* (1995) also detected pineapple clostero virus particles (PCV) in pine-apple plants and from mealy bug collected from wilted plants. Hu *et al.,* (1997) examine the distribution of pineapple closterovirus in Hawaii using tissue blotting immunoas-say by and suggested that PCV may be involved in mealy bug wilt of pineapple. Two

species of mealy bugs *Dysmicoccus neobrevipes* and *D. brevipes* were also found transmitting PCV (Sether and Hu, 1997). Closterovirus like particles are also associated with pineapple plants affected with pineapple mealy bug wilt in Cuba (Borroto *et al.*, 1998). Foliar contents of phenolic compounds in pineapple as affected by wilt, were studied by Goncalves *et al.,* (1999).

15.6.4 Epidemiology

Relationship between mealy bugs and ants occurring on pineapple plant is very important factor in the epidemiology of disease. The mealy bugs are dependent on the activity of ants for their vigorous growth and reproduction and thereby affects their transmission behaviour. Percentage of mealy bugs infected pineapple plants was highly correlated with number of ants caught in pitfall traps (Beardsley *et al.*, 1982) which confirm that the incidence of mealy bug wilt was highest in field, edges, reflecting the greater abundances of mealy bugs and ants in these area. Mealy bugs reared on the healthy pineapple do not transmit any virus but healthy mealy bugs fed on diseased plants for 24-28 hours when transferred onto healthy pineapple plants, the initial disease symptoms appeared after 40-50 days (Singh and Shastry, 1974). In field the amount of wilt depends on the average number of mealy bugs per plant and the average length of time these mealy bugs feed on each plants.

15.6.5 Management

Higher levels of nitrogen decrease the wilt disease incidence. Minimum incidence of disease was observed in plants supplied with highest dosage of N, P and K and plots having higher plant populations (Singh and Shastry, 1975). Disease suckers can be recovered within 30-50 days by subjecting them to hot water or heat treatments at 50°C for 3 hours (Singh and Shastry, 1974). Pineapple wilt infected suckers when treated with hot water at 40°C and 50°C survived upto 80-100% and ELISA demonstrated that 60-100% of these were free from virus (Ullman *et al.*, 1991).

The disease can be managed to a considerable extent by the use of insecticides against mealy bug vector and ants.

15.7 Pineapple Bacilliform Virus (PBV)

Presence of some bacilliform particle belong to Badnovirus group were seen in partially purified preparation from both asymptomatic and wilting pineapple leaves from Australia (Wakman *et al.*, 1995). Pineapple bacilliform virus (PBV) was also detected using the polymerase chain reaction (PCR) in extracts from infected plants from Australia (Thomson *et al.*, 1996). The nucleotide sequence of the cloned PCR product suggested that PBV was related to, but distinct from, other badna-viruses. PBV appeared to be present in all pineapple growing areas along the east coast of Australia. PBV was detected in plants grown from seeds, plants propagated through meristem tip culture and in mealy bug which were collected from infected plants. PBV was detected in crown, leaf and root tissue from infected pineapple plants.

15.8 Fruitlet Rot

Fruitlet rot is an important cause of loss in pineapples intended for fresh market or for canning. The disease has also been described as fruitlet brown rot, eye rot and black spot rot. The disease is characterized by appearance of light to brown soft rot of the axis of an individual fruitlet. In cross-section, rot appears as a brown spot in the centre of the fruitlet while in longitudinal sections the affected area is elongated and extended towards the heart of the fruit. Sometimes diseased tissues become black, wet or dry according to the humidity (Edmonstone, 1958).

Large number of microbes including bacteria and fungi cause fruitlet rot. Important pathogens which produce this disease are *Gibberella fujikuroi* var. *subglutinans* Edward (*Fusarium moniliforme* var. *subglutinans* Wallenw & Reinking) (Perez *et al.*, 1994), *Penicillium funiculosum* Thom. and *Pantoea ananas* pv. *ananas* (Serrano) Mergaret *et al.,* Pathogens survive on debris in the soil and is disseminated by water and insects. Bacteria and fungal spores are carried into floral cavities by water splashes. Spraying acaricide and insecticide to control mites and mealy bugs is the best mean to control this disease (Mourichon, 1983).

15.9 *Botryodiplodia* Rot

Botryodiplodia rot caused by *Botryodiplodia theobromae* Pat. is a serious market disease in India (Tandon and Bhargava, 1962). Infection takes place through cut stems. The fungus spread in the pulp upto axis which later becomes dark brown and soft. Pycnidia develop in the peal. Rapid decay occurs at 25°C but storage of fruits at 10°C can check the spread of disease.

A leaf blight disease caused by *B. theobromae* was also identified as new disease of pineapple in Kerala, India in 1989 (Suharban and Rajan, 1993).

15.10 Bacterial Soft Rot

Bacterial soft rot is a serious disease in Malaysia. Initial infection occurs at flowering stage but no symptoms appear till ripening. Rotting proceeds with great rapidity and fruits undergo complete rotting within day or two. *Pectobacterium chrysanthemi* (*Erwinia chrysanthemi* Barcholder *et al.,*) is the causal fungus of this disease.

Soft rot caused by *Pseudomonas marginalis* was also reported in high altitude area of Crespo in the state of Lara, Venezuela but incidence (0.8-6.2%) was lower (Marcano *et al.*, 1993; Cardona *et al.*, 1994). Temperature and cultivation conditions in that area appeared to favour the development of the disease.

15.11 *Rhizopus* Rot

The disease is reported to occur in Nigeria. In tropical areas, decay is rapid and infected fruit become soft and covered with fungal strands. *Rhizophus oryzae* Went. & P. Geerlings and *R. stolonifer* (Ehrenb. ex. Fr.) Lind are responsible for rhizopus rot of pineapple (Lim, 1985).

15.12 Leathery Pocket

Leathery pocket disease is reported to occur in South Africa and Hawaii. The disease is characterized by cork formation deeper in the tissues. The disease is caused by fungus *Penicillium funiculosum* Thom. and disseminated by mites. Spraying acaricide and fungicides helps in controlling the disease.

15.13 Pink Disease

Pink disease causes heavy losses in Hawaii, Philippines, Australia, Mexico and Tanzania (Mabagala and Maerere, 1998; Cha *et al.*, 1997b). Several bacteria are reported to cause this disease, including *Gluconobacter, Acetobacter* and *Enterobacter* (Rohrbach and Pfeiffer, 1976). Some strains induce a brownish pink discolouration of the pulp causing losses in fresh pineapples. Other strains infect without producing any symptoms, however, when fruits are cooked during canning discolouration occurred. The symptoms are not discernible externally and the exact nature expressed within fruits viz., colour intensity and aroma, depends largely on bacterial genus responsible for pink disease. The flesh colour vary from light pink to brown white aroma, from normal to the odour of ripe cantaloupe. The discolouration on fruit may be entire or restricted to fruitlets. Infected flesh invariably turn brown after cooking. The bacteria enter the fruit via open flowers and various insect and mites were responsible for disseminating the bacteria.

The causal agent of pink disease was identified as *Pantoea citrea* in Philippines. Bacterium induces the production of compounds in pineapple which become pink to reddish brown upon cooking the fruit, pulp or juice. Identification and characterization of *P. citrea* gene encoding glucose dehydrogenase that is essential for causing pink disease was done by Cha *et al.,* (1997a). The pathogen and their symptomatology is also described by Kontaxis and Hayward (1978).

Field experiments have shown that the disease could be reduced by hand dusting Disyston 10G (disulfoton) four times on developing pineapple inflorescences (Kontaxis, 1977).

15.14 Marbled Fruit

Marbling or marbled fruit of pineapple is bacterial disease caused by *Pantaea ananatis* pv. *ananatis* (*Erwinia ananas* pv. *ananas*) and is also known as fruitlet brown rot and bacterial fruitlet rot. The disease is reported to occur in Philippines, Mexico and causes heavy losses (Linford, 1952; Lim, 1983).

No external symptoms are discernible. At early stage, a tangential section of the fruit would reveal brown discolouration of one or all three placental lobs of the fruitlets. Fruitlets at the middle of fruit or the base, are usually more affected than towards the crown. Extensive rot reaches to the core and more often restricted to one or more of the locules. Brown specking is most diagnostic feature and in severely affected fruits, transverse section of diseased portions radiating around the core giving a variegated appearance. The disease is spread by wind, rain and insects and is most serious when

fruits are developing during warm dry weather when fruit acidity is low. Bacteria enter the fruit through open flowers and transmitted by insects to the stigmatic fluid or nectar of open blossoms (Linford, 1952).

Fortnightly spray with Bordeaux mixture on fruits for three to four months give satisfactory control. Application of potassium sulphate at 500 kg/ha at 10 and 13 month increased fruit acidity from pH 4.0 to 3.8 resulting in decreased diseases incidence (Serrano, 1934). Increasing the planting density to reduce fruit size and thus increase fruit acidity is recommended control measure (Linford, 1952; Tay, 1974).

15.15 Fruit collapse

Pineapple fruit collapse is caused by *Pactobacterium chrysenthemi* (*Erwinia chrysenthemi*) and causes losses ranging from 2-40% (Thompson, 1937; Johnston, 1957; Lim, 1978; Lim and Lowings, 1983). Symptoms develop at about two to three weeks before ripening of fruits. Infection is characterized by copious exudation of fluid from the inter fruitlet tissues accompanied by babbles of gas. Skin of the infected fruit changes from dark purple to olivaceous green.

The pathogen enters fruit through open flowers. Ants could transfer the pathogen to the flowers during search for nectar. Winds and rain splash also play important role in disease dissemination.

In ratoon crop, economical method of control is to time the forcing interval so that the opening of flowers from forcing does not coincide with collapsing fruits from the previous round. Forcing intervals of four months reduced disease incidence significantly (Lim, 1978). Cultivars Smooth Cyenne and Sarawak exhibited a higher degree of tolerance (Lim and Lowing, 1979). Spraying insecticides against and vectors also reduced the disease (Lim and Lowing, 1982). Sanitation by timely removal of diseased fruit and plants also helps in reducing the disease.

15.16 *Fusarium* Wilt

Fusarium wilt incited by *Fusarium subglutinans* (*Gibberella fujikuroi* var. *subglutinans*) is most important disease in Brazil (Martelleto *et al.*, 1998; Costa and Chalfoun, 1998). The mycellial growth of the fungus was in the 10-30°C range, with 25°C being the optimal temperature for mycelial development. Sporulation occurred between 15 and 30°C and its increase was directly proportional to temperature increase. The formation and expansion of the lesions in the foliar tissue occurred between 10 and 39°C, although it was more marked in the 15-25°C range (Martelleto *et. al.,* 1998).

15.17 Root Knot Nematode

The root knot nematode, *Meloidogyne incognita* (Kofoid and White, 1919) Chitwood, 1949 is a serious problem on pineapple and causes more than 40% loss in fruit yield to the first crop (Reddy and Nagesh, 2000).

Yield increase in pineapple has been observed when planting is done in a field having pigeonpea in the previous season sowing to decrease in the infestation by *M.*

incognita. Crop rotation with pangola grass (*Digitaria decumbens*) helps in minimizing the losses. *Ananas ananasoides* and three of its hybrids are resistant to this nematode.

15.18 Reniform Nematode

The reniform nematode (*Rotylenchulus reniformis* Linford. & Oliveira) infects pineapple and is responsible for decline of pineapples. The plant population is reduced by more than 83% in the field (Reddy and Nagesh, 2000).

Rotation with sugarcane or pangola grass is effective in controlling this nematode. Nematode population is also reduced by keeping land fallow after irrigation. Application of carbofuran (22.5 kg/ha), oxamyl (9 kg/ha) or fenamiphos (45 kg/ha) are effective in controlling the nematode and increasing fruit yield (Reddy and Nagesh, 2000). Pineapple varieties/species, Venezolana, *Ananas ananasoides* and several of its hybrids are resistant to reniform nematode. Do not allow the initial infestation to be maintained.

16. Pomegranate (*Punica granatum* L.)

16.1 *Phomopsis* Leaf Spot and Fruit Rot

Symptoms of the disease appear on leaves as buff brown coloured marginated to diffused spots having black dot like fruiting bodies (pycnidia) near centres (Shreemali, 1972). However, on fruits the disease starts as discolouration from calyx end, gradually spreads over entire fruits having pin head sized pycnidia on the lesions (Mehta, 1950). The twigs are also infected.

The disease is caused by the fungus *Phomopsis aucubicola*. Hypae are hyaline, poorly branched, closely septate, 1.4-3.8 μm wide, pycnidia densely gregarious black, immersed than erumpent, globose to sub globose, 201.6-489 μm in diameter, dark brown to black, ostiolate, conidiophores simple, short, unbranched, hyaline 4.6 x 8.2 μm long, alpha spores hyaline, ovoid to oblong or rarely fusoid to sub fusoid, often biguttulate, 7.4-11.8 x 2.4-3.1 μm, beta spores filiform curved or unicinate, 19.8-34.4 x 0.54-1.4 μm.

The disease can be prevented by removing and burning the infected plant parts. Spraying 0.05% benomyl or carbendazim or 0.3% copper oxychloride or zineb are effective against the disease. Jeolicola Local variety was less susceptible, whereas Country Large Red and Muscat White were most susceptible to fruit rot (Verma *et al.*, 1982).

16.2 *Glomerella* Leaf Spot and Fruit Rot

Minute dull violet to black spots appear on leaves with their surrounding region turning yellow. The spots later enlarge covering major part of leaves which finally fall off (Chandra and Tandon, 1965). On fruits, discolouration starts from lower part or sometimes from the sides. The discoloured areas later become brown to black covering whole of the fruit and causing complete softening within a week of infection. Severe

spotting and rot of fruits is also reported from Venezuela (Mazzani, 1994).

The disease on fruit is caused by *Glomerella cingulata* (Stonem) Spauld & Schrenk and on leaves by its asexual stage *Colletotrichum gloeosporioides* Penz. Sprays of 0.05% thiophanate methyl or carbendazim or 0.3% captan is effective in reducing the disease.

16.3 Canker and Dieback

Initially elliptical black spots appear on the twigs. With advances of infection, the affected areas become flattened and depressed with raised edges. The bark dries and upon its cracking the underlying wood shows abnormal dark brown to black discolouration. Twigs beyond lesions dry up and in severe cases, the whole tree may die. The fungus *Ceuthospora phyllosticta* causes canker and die back disease (Pathak, 1980). Disease can be effectively managed by pruning of infected twigs, protecting cut surfaces by fungicidal paints and spraying 0.3% mancozeb or captan.

Tang *et al.,* (1998) also reported the occurrence of pomegranate canker caused by *Zythia versoniana* which usually attack fruit, branches and trunk in China. Conidia germinate vigorously at 25°C, while 30°C is the optimum temperature for hypal growth. Pathogen mainly over winter in the 1 to 2 year old branches. Spraying a solution of 40% carbendazim emulsion or 50% thiophanate-methyl (Topsin M), twice in late June and early July, could successfully control the disease.

16.4 Bacterial Blight

Bacterial blight is prevalent in all the pomegranate growing states of country (Hingorani and Mehta, 1952; Kanwar, 1976; Sohi *et al.*, 1964). It is the most devastating disease of pomegranate and is the major constraint in commercializing its cultivation (Chand and Kishun, 1991).

Initial symptom of the disease appears as small, irregular translucent water soaked spots from 2 to 5 mm in diameter with necrotic centres on leaves. Gradually these spots turn light to dark brown and are surrounded by prominent water soaked margins. Numerous spots may coalesce to form large patches. Severely infected leaves became yellow and are easily shed. On stem, the disease starts as brown to black spot around the node. In advance stage of nodal infection, girdling and cracking of nodes are found which finally lead to break down of branches. Brown to black spots appear on pericarp with L or Y shaped cracks.

Disease is incited by *Xanthomonas axonopodis* pv. *punicae*. It is a gram negative rod, motile with single polar flagellum and measures 0.5x1.0-2.5 µm in size. The bacterial cells are capable of surviving in soil for more than 120 days. They are dispersed by the wind and lodged on leaves where they cause infection through wounds and stomata. Within tree, the pathogen spreads from leaf lamina to petiole and then to stem and node (Chand and Kishun, 1992). In India, disease spread fast due to high temperature and low humidity during March-July.

Copper fungicides or Bordeaux mixture provided some protection against this disease. Effective control of the disease has been observed (Suriachandraselvan *et al.*,

1993) by three sprays of Paushamycin (0.05%) plus copper oxychloride (0.2%) at 15 days interval. Rani and Verma (2001) also found three sprays of streptocycline (200 µg/ml) best in controlling the disease followed by Bordeaux mixture (1.0%) + streptocycline (100 µg/ml) at fortnightly intervals.

16.5 *Fusarium* Leaf Spot

The disease is characterized by appearance of minute specks towards the leaf margin which are brownish and circular to irregular in outline. With the advancement of the disease, the spots coalesce to form big dark brown necrotic blotch.

The disease is reported to be caused by *Fusarium fusarioides* (Frag. & Cif.) Booth from Maharashtra by Sherkar and Utikar (1982b). 5°, 27-30° and 45°C are minimum, optimum and maximum temperature requirement of fungus respectively on media.

Difolatan and Miltox were found effective in inhibiting the growth of the fungus in bioassay. However, due to ban on spraying of Difolaton and non-availability of Miltox, tank mix of copper oxychloride and zineb can be used for spray.

16.6 Fruit Spot

The disease is characterized by appearance of black circular spots which gradually enlarge and coalesce to form big dark black spots leading to necrosis. The margin of spots vary from reddish to brown in colour. The infection is restricted to the rind of the fruit and, to some extent, to underside of the pulp.
The disease is reported to be caused by *Beltaraniella humicola* (Sherkar and Utikar, 1982a).

Inhibiting effect of benlate and copper oxinate are reported *in vitro* (Sherkar and Utikar, 1982a). Leaf and fruit spots are controlled through the sprays of Baycor (0.1%), Antracol (0.2%) and Benlate (0.1%) when sprayed at 15 days interval during July-September months (Anonymous, 2000).

16.7 *Drechslera* Fruit Spot

This kind of fruit spot on pomegranate was first reported in 1972 on Muscat variety (Utikar *et al.*, 1976) and fruits at all the stages are attacked. Appearance of numerous, small black spots scattered all over the fruits are the characteristic symptom of the disease. The margin of the spots varies from dark green to orange in colour which later gradually enlarge and coalesce to form big dark spots of various sizes. Mild infection is confined to rind of the fruit but severe infection extends to the inner tissues and even to the seeds, showing ashy discolouration. Such infected fruits are not suitable for consumption.

The disease is caused by *Drechslera rostrata*. The conidia of the fungus are pale to dark olivaceous brown, cylindrical to rostrate, somewhat less curved, measuring 43.06-124.94 x 9.24-17.63 µm in size with 5-13 transverse septa and a minute hilum protruding from the base. The other end is bluntly tapered. The septa from basal and apical cells are prominent being thicker and darker than the intermediate septa. Spray-

ing with 0.3% mancozeb or captan after fruit formation is recommended for reducing losses due to this disease.

16.8 Leaf and Fruit Spots

Most common fungi causing leaf and fruit spots of pomegranate in Maharashtra are *Cercospora* sp. *Colletorichum gloeosporioides* (*Glomerella cingulata*) *Alternaria alternata* and *Drechslera rostrata*. Environmental conditions are very favourable for the development of leaf spots and fruit spots during June and July (Navale *et al.*, 1998).

Fungicides, copper oxychloride (0.4%), ziram (0.25%), Bordeaux mixture (0.1%), mancozeb (0.3%), captan (0.2%) and carbendazim (0.1%) give effective control of leaf and fruit spots when one prophylactic spray of respective fungicide is given during flowering/fruit setting stage and thereafter nine sprays at 10 days intervals (Navale *et al.*, 1998).

16.9 Flower and Fruit Lesion

Thankamma (1983) reported lesions on flowers and fruits caused by *Phytophthora nicotianae*. Infected flowers are shed prematurely and fruits remains on plant but carry infection and scrapings from the fruit surface show plenty of sprongia of *Phytophthora*. In culture, fungus is uniformly fluffy without zoning. Hyphae are fairly uniform in diameter (4.8 mm) with hyphal swelling. Chlamydospores measuring 36 μm are produced in plenty and are both intercalary and terminal and readily germinate in water. Sporangia are broadly turbinate with spherical basal portions and apical part prolonged into beak, papillate and measure 63x44 μm. Sporangia do not usually shed.

16.10 Fruit Rots

16.10.1 *Rhizopus* Soft Rot

Rhizopus soft rot is an important storage and market disease of pomegranate. Initially small spots appear which later coalesce and cover large areas. Apparently the rind portion of fruit seems to be diseased but internally entire fruit decays into a pulpy mass. The appearance and spread of the disease is favoured by high temperature and high relative humidity (Kanwar *et al.*, 1973).

The disease is caused by two species of *Rhizopus*, namely *R. arrhizus* Fischer and *R. stolonifer* (Ehrenb ex. Fr.) Link. The former shows black hairy growth on fruit surface. Pre-storage treatment of fruits with linseed, mustard or castor oil and treatment of packing straws with SO_2 producing chemicals are helpful in protecting fruits from soft rot (Kanwar and Thakur, 1972).

16.10.2 *Cladosporium* Fruit Rot

The disease is characterized by the production of orange red to dull brown circular spots on fruits with their grains becoming olive brown (Panwar and Vyas, 1974). In

advance stage, the whole fruit rots. *Cladosporium oxysporum* Berk and Curt. cause fruit rots of pomegranate. The mycelium is dark olive green colour, hyphae are septate, light olive green, 2.5-3.0 µm in width, conidiophores are light brown, simple and conidia are light brown to green, 1 celled, fusoid, 1-20x3.4-4.5 µm.

16.10.3 *Aspergillus* **Fruit Rot**

This is common rot of pomegrate fruit affected by fruit flies and usually occurs in the market. Brown discolouration of the fruit surface is the beginning of rot. The affected area gradually turns black and sticky. Numerous greenish conidia of fungus cover the whole fruit which shows soft rot and gives fermented smell. Three species of *Aspergillus* viz. *A. flavus* Link., *A. niveus* Blotchw and *A. versicolor* (Vuill) Tiraboschi have been reported to cause fruit rot in pomegranate (Srivastava and Tandon, 1971; Kanwar and Thakur, 1973; Sharma *et al.*, 1981).

    Aspergillus flavus causes brownish discolouration, which gradually becomes blackish and a little slimy. Subsequently, it gets slightly depressed and is later covered by green conidial heads of the incitant. The disease causes soft rot and emits fermented odour (Srivastava and Tandon, 1971).

    Aspergillus niveus produces almost circular and light yellow patch. The infected tissue turn soft, darker in colour and soon gets covered by mycelial growth. Secretion of yellow watery substance and emittance of foul smell are usually associated with it (Sharma *et al.*, 1981).

    Aspergillus versicolor produces small brownish patch which increases in diameter and turn darker with blackish tinge in the centre. The diseased tissue shrinks and ultimately disintegrate especially in the central region, followed by irregular depression and exudation of slimy mass emitting foul odour (Sharma *et al.*, 1981).

16.10.4 *Alternaria* **Rot**

Post harvest rot of pomegranate fruits is caused by *Alternaria alternata* which causes qualitative damage to fruits (Majumdar and Singh, 1997). Injury to the rind of the fruit, such as punctures caused by fruit flies, provide avenues for entry of fungi. Control of fruit flies is therefore, one of the most important steps in management of the rot. Tilt (propiconazole) dip in both pre and post inoculation treatment at 0.10% solution for 5 minutes is most effective in reducing fruit spoilage followed by Dithane M-45 (Majumdar and Singh, 1997). *In vitro* test indicated fungicidal properties in aqueous leaf extract of *Calotropis procera, Azadirachta indica, Lantana camara* and *Ocimum basilicum* against *A. alternata* isolated from fruits of pomegranate (Srivastava and Lal, 1997). *In vivo* study revealed 64 to 85% control of fruit rot.

16.10.5 **Dry Fruit Rot**

Pomegranate fruit rot caused by *Coniella granati* was studied at Udaipur (Rajasthan), during 1983-84 by Lukose and Singh (1997). Good rainfall, high humidity (around 80%) and a temperature range of 22-32°C favoured disease development. Under these opti-

mum conditions, the infection covered the entire fruit within a week causing complete rotting of the fruit. This fungus also reported to causes dry rot of pomegranate in Himachal Pradesh (Sharma, 1998).

16.11 Decline

Pomegranate decline mostly observed in newly established 3 to 7 year old orchards in Iran and its etiology of the disease is described by Banihashemi, (1998). The first disease symptom appears as yellowing, wilting and death of some branches usually appeared in the early summer. Sudden drop in temperature in late autumn and early winter in the milder areas where the trees are not fully dormant is the main contributing factor to decline in Iran.

16.12 Wilt

Wilt of pomegranate caused by *Ceratocystis fimbriata* was first reported from India during survey of 44 location from 1995 to 1998 and showed crop loss of Rs. 30 lakhs and 7.5% of the crop (Somasekhara, 1999).

17. Sapota (*Achras sapota* L.)

17.1 Sooty Mould

Sooty mould is an important disease of sapota in India and affects the normal development of plants. The mould grows on the honeydew secreted by hoppers, scales and coccids on leaves and twigs and produces mass of black spore which stick to the leaf surface forming a film over the foliage. Although no direct damage is caused by the fungus since it neither enters the host nor derives nutrient from the host. However, the photosynthetic activity of the leaf is adversely affected because of black cover of spore mass over the leaf area and disfiguring of the fruits.

The fungus responsible for sooty mould is *Capnodium* sp. which also affects a number of other hosts, such as citrus, mango etc. The fungal growth can be checked by spraying plants with 0.25% wettable sulphur (Singh and Singh, 1972). It can be controlled by spray of 100 g starch in 18 litres of water. On drying it forms thick flakes which drop off along with mouldy cover. Spraying with 40 g zineb in 18 litre of water also checks the disease.

17.2 Flat Limb

Flat limb disease was first reported from coastal regions of Gujarat and Maharashtra (Khurana and Singh, 1972) and is on increase in various sapota growing areas of country. The affected branches of tree become flattened and twisted with severe bunching of leaves which become small, thin and yellow. The affected branches bear small, dry, hard and shrivelled fruits and these affected branches give rise to normal branches during summer months. Flat limb affects the yield adversely.

Although fungi such as *Botryodiplodia theobromae* Pat. and *Phoma* spp. have been isolated from the different tissues, precise etiology of the disease is unknown (Pathak *et al.*, 1988). However, Patel and Chauhan (1993) reported *B. theobromae* as the causal agent of sapota twig blight in Gujarat.

Pruning of affected branches followed by sprays of 0.3% captan or zineb can manage the disease effectively. Bavistin and Agrozim were also found significantly superior for checking twig blight disease (Patel and Chauhan, 1997). Low temperature storage is reported to help in minimizing the losses.

17.3 *Pestolotia* Leaf Spots

Disease symptoms appear as numerous, small, reddish brown specks on leaf lamina, which gradually enlarge to form more or less circular spots measuring 1 to 3 mm in diameter. Enlarged spots have greyish centre and reddish to dark brown margins. On the grey coloured regions of these spots minute, black fruiting bodies of the pathogen may be seen.

Pestolotia leaf spot is caused by *P. versicolor* Speg. Though *P. versicolor* has been reported from Kerala (Wilson *et al.*, 1970) to cause this disease but from Karnataka another species, *P. scirrofaciens* has been reported (Rajendran, 1971). Aceruveli of *P. versicolor* are 80-175 μm wide. Conidia are 5 celled, clavate fusiform, erect or somewhat curved, 22-27 x 7.5-9.5 μm in size and bear 3, rarely 4, apical, flexucus, widely divergent settullae which are 17-27 μm long. The fungus survive on fallen leaves and fruits around the trees.

Spraying zineb (0.25%) or copper oxychloride (0.3%) is recommended for the effective control of the disease.

17.4 Leaf Spots

The leaf spot caused by fungus, *Phaeophleospora indica* Chinnappa is first reported from Dharwar and characterized by the appearance of numerous, small circular, pinkish to reddish brown conspicuous spots with whitish centres on maturity (Chinnappa, 1968). Affected leaves finally, turn yellow and shed prematurely resulting in yield reduction. Maximum incidence of the disease is observed in the months of October to December and is widespread in Mysore state. Monthly sprays with 0.3% copper oxychloride are effective in managing the disease (Sohi and Sridar, 1972, 1973).

Leaf spots caused by *Gloeosporium rubi* reported from Allahabad (Srivastava *et al.*, 1964), *Nigrospora oryzae* (Berk. et. Br.) Petch and *Chaetomium* sp. from Allahabad & Poona (Tandon, 1967) and *Curvularia leonata* (Wakker) Boedijn var. *aeria* from Delhi (Kore and Bhide, 1977) also reported on sapota.

17.5 Anthracnose

Anthracnose is caused by *Glomerella cingulata* (Stonem.) Spauld and Schrenk. Which appears initially as yellow irregular lesions on both the leaf surfaces. In case of severe attack, the whole leaf turn yellow and later the lesions turn greyish to dark olive and

numerous fruiting bodies of the pathogen appear in centre of the spot. This pathogen is most destructive on sapota in Guangdong province in China (Zhang and Qi, 1994). Two new species, *Laphodermium sapoti* and *Didymella sapotae* are also reported to occur on sapota.

Spraying trees with 0.2% chlorothalonil or zineb at monthly interval is quite effective in checking the disease. Spraying of Topsin M, Bavistin and Dithane M-45 is also reported effective in reducing the disease.

17.6 Leaf Blight

Chinnappa and Rao (1970) reported *Fusicoccum sapoticola* from Maharashtra producing small brownish irregular specks along the margins of leaves which later coalesce to form irregular patches. Eventually the leaf is blighted. Pycnidia in the form of black pin point bodies may appear in these spots. The pycnidia are unilocular, ostiolate, variously shaped, and measure 95-100 x 38-144 µm. Coniodophore are hyaline, clamate, and 3.6-5.4 x 2 µm in size. The conidia measure 5.5-9.2 x 1.8-3.6 µm. Certain species of *Fusicoccum* have their perithecial stage in *Botryospaeria*.

17.7 Fruit Rots

Fruit rots caused by *Botryodiplodia theobrome* Pat., *B. dothedia* Moug. ex Fr., *Phytophthora palmivora* (Butler) Butler, *Pestalotiopsis versicolor* (Speg) Steyart, *Pestalotia sapotae* P. Henn, *Hendersonula toruloides* and *Fusarium roseum* Link are prevalent in India (Srivastava *et al.*, 1964; Gupta and Sehgal, 1974; Jain *et al.*, 1981) but not much work has been done in these diseases.

18. References

Agarwal, G.P. and Sahni, V.P. 1964. Fungi causing disease at Jabalpur. Mycopath et Mycol. Applicata 22: 245-247.

Ann, P.J., Lee, H.L. and Huang, T.C. 1999. Brown root rot of 10 species of fruit trees caused by *Phellinus noxius* in Taiwan. Plant Disease 83(8): 746-750.

Anonymous 1985. Proceeding of 3rd Workshop on Arid Zone Fruit Research held at Rahuri.

Anonymous 1988. Annual Report, Central Institute of Horticulture for Northern Plains, Lucknow. pp. 74-92.

Anonymous 2000. Annual Report, 1999-2000. Indian Institute of Horticultural Research (ICAR) Hessarghatta, Bangalore, pp. 40.

Arya, A., Lal, B. Agarwal, R. and Srivastava, R.C. 1986. Some new fruit rot diseases – II: Symptomatology and host range. Indian Journal of Mycology and Plant Pathology 16(3): 265-269.

Arya, A., Pandey, R.S. and Lal, B. 1987. Stem canker and die back disease of certain fruit trees. Acta Botanica Indica 15: 141-42.

Awasthi, R.P., Tripathi, B.R. and Singh, A. 1975. Effect of foliar sprays of zinc on fruit drop and quality of litchi (*Litchi chinensis* Sonn.). Punjab Horticultural Journal 15: 14-16.

Banihashemi, Z. 1998. Etiology of pomegranate decline in Fars province of Iran. Phytopathologia Mediterranea 37(3): 127-132.

Barbosa, C.J., Stenzel, N.M.C. and Jacomino, A.P. 1999. Occurrence of cucumber mosaic virus (CMV) in passion fruit in the state of Parana, Brazil. Fitopatologia Brasileira 24(2): 193.

Barekai-Golan, R. and Phillips, D.J. 1991. Post harvest treatment of fresh fruits and vegetables. Plant Disease, 75: 1085.

Beardsley, J.W., Su, T.H., McEween, E.L. and Gerling, D. 1982. Field investigations on the interrelationships of the big-headed ant, the grey pineapple mealy bug, and the pineapple mealy-bug wilt disease in Hawaii. Proceedings Hawaii Entomological Society, 24: 51-67.

Beriam, L.O.S., Malavolta, V.A., Jr., Rosato, Y.B. and Yano, T. 1998. Serology applied to the study of *Xanthomonas campestris* pv. *passiflorae*, a causative agent in bacteriosis of the passion fruit tree (*Passiflora* spp). Arquivos do Instituto Biologico (Sao Paulo), 65: 25-33.

Bewaji, O. English, H. and Shick, F.J. 1977. Control of Alternaria surface rot of Kadota figs. Plant Disease Reporter, 61: 351-355.

Bezerra, D.R., Lima, J.A.A. and Xavier Filho, J. 1995. Purification and characterization of an isolate of passion fruit woodiness potyvirus. Fitopatologia Brasileira, 20: 553-560.

Bhargava, K.S. and Bist, N.S. 1962. Fruit tree virus disease investigation in Uttar Pradesh. Proceedings 4th Symposium on Virus Diseases of Fruits and Trees in Europe. Lyngby (1960), Tidsskr. –Planteavl, 65, Saemwnmor, pp. 196-203.

Bhargava, S.N., Shukla, D.N. and Singh, A.P. 1977. Stalk-end rot of *Aegle marmelos* – a new disease. Indian Phytopathology, 30: 120-121.

Bhargava, S.N., Pandey, R.S., Shukla, D.N. and Dwiwedi, D.K. 1982. New fruit rot of fig. National Academy of Science Letters, 5: 251.

Biswas, S., Das, N.K., Qadri, S.M.H. and Saratchandra, B. 1995a. Evaluating different plant extracts against three major diseases of mulberry. Indian Phytopathology, 48: 342-346.

Biswas, S., Mandal, S.K., Teoria, R.S., Nair, B.P. and Sengupta, K. 1995b. Intensity of mulberry powdery mildew in West Bengal with some measures to control. Indian Journal of Sericulture, 34: 114-117.

Bora, S.S. 1994. Avocado rust – a new record from India. Progressive Horticulture, 26: 85-86.

Borroto, E.G., Cintra, M., Gonzalez, J., Borroto, C. and Oramas, P. 1998. First report of a closterovirus-like particle associated with pineapple plants (*Ananas cosmosus* cv. Smooth Cayenne) affected with pineapple mealybug wilt in Cuba. Plant Disease, 82: 263.

Boruah, P., Sarma, P.C. and Dutta, S. 1998. Incidence of *Cercospora* leaf spot disease of mulberry (*Morus alba* L.) in Upper Brahmputra Valley (Assam). Advances in Forestry Research in India, 18: 239-244.

Bose, T.K. and Mitra, S.K. (eds) 1990. Fruits: Tropical and Subtropical. Naya Parkashan, Calcutta, 88 pp.

Brown, B.L., Scot, K.J. and Mayer, D.G. 1984. Control of ripe fruit rot of guava, Lyche and custard apple by post harvest prochloraz dip. Singapore Journal Primary Industries, 12: 40-44.

Butler, E.J. and Bisby, G.R. 1931. The fungi of India. Imperial Council of Agricultural Research India, Science Monograph, I: 17,237 pp.

Cardona, R., Carrasco, A. and Camino, J.M. 1994. New bacterial disease present in pineapple plantations in Lara State, Venezuela. Fitopatologia Venezolana, 7: 54-55.

Carter, W. 1933. The pineapple mealybug, *Pseudococcus brevipes* and wilt of pineapple. Phytopathology, 23: 207-242.

Castillo, N.M. and Granada, G.A. 1995. Studies on bacteriosis of passion fruit in the valley of Cauca: aetiology, hosts and control. Fitopatologia Columbiana 19(1): 55-61.

Cedeno, L., Mohali, S. and Palacios-Pru, E. 1993. Anthracnose caused by 2 strains of *Glomerella cingulata* on fruits of passion fruit. Fitopatologia Venzolana, 6: 30-33.

Cha, J.S., Pujol, C. and Kado, C.I. 1997a. Identification and characterization of a *Pantoea citrea* gene encoding glucose dehydrogenase that is essential for causing pink disease of pineapple.

Applied and Environmental Microbiology, 63: 71-76.

Cha, J.S., Pujol, C., Ducusin, A.R., Macion, E.A., Hubbard, C.H. and Kado, C.I. 1997b. Studies on *Pantoea citrea*, the causal agent of pink disease of pineapple. Journal of Phytopathology, 145(7): 313-319.

Chand, R. and Kishun, R. 1991.   Studies on bacterial blight (*Xanthomonas campestris* pv. *punicae*) of pomegranate. Indian Phytopathology, 44: 370-372.

Chand, R., and Kishun, R. 1992. Transmission of nodal blights stain of *Xanthomonas campestris* pv. *punicae*. Indian Journal of Plant Pathology, 10: 74-75.

Chandra, S. and Tandon, R.N. 1965.   Control of leaf spot of pomegranate with fungicides. Science and Culture, 31: 536.

Chang, C.A., Chen, C.C., Deng, T.C. and Zettler, F.W. 1996. Characterization of passion fruit crinkle potyvirus – a newly found virus infecting passion fruit. Plant Protection Bulletin (Taipei), 38: 339-354.

Chatterjee, S.N. and Raychaudhuri, S.P. 1963. Additional vectors of mulberry mosaic. Indian Phytopathology, 16: 243-244.

Chatterjee, S.N. and Raychaudhuri, S.P. 1965. A note on aphid transmission of mosaic disease of mulberry. Indian Phytopathology, 18: 319-320.

Chen, L.C., Lai, S.C., Lee, C.C., Chung, Y.W. and Ann, P. 1998. Effect of environmental factors on mycelial growth of *Peronophythora litchii*. Plant Pathology Bulletin, 7: 128-133.

Cheng, G.Y. and Liu, J. M. 1994. A new technique treating jujube twig materials infected with witches' broom disease for electron microscope examination. Plant Protection, 20: 36.

Chern, L.L., Ann, P.J. and Young, H.R. 1998. Root and foot rot of loquat in Taiwan caused by *Phytophthora*. Plant Disease 82(6): 651-656.

Chinnappa, B. 1968. A new species of *Phaeophleospora* affecting sapota. Current Science, 37: 566.

Chinnappa, B. and Rao, V.G. 1970. A new species of *Fusicoccum* on *Achras sapota*. Science and Culture, 36: 295-296.

Chona, B.L. and Munjal, R.L. 1955. Notes on miscellaneous Indian fungi-II. Indian Phytopathology, 8: 184-198.

Chona, B.L. and Munjal, R.L. 1956. Notes on miscellaneous Indian fungi-III. Indian Phytopathology, 9: 53-66.

Chona, B.L., Lall, G. and Munjal, R.L. 1959. Some *Cercospora* species from India. Indian Phytopathology, 12: 76-84.

Chowdhury, S. 1945a. Ceratustomella disease of pineapple. Indian Journal of Agricultural Science, 15: 135.

Chowdhury, S. 1945b. Heart or stem rot of pineapple. Indian Journal of Agricultural Science, 15: 139-140.

Chowdhury, S. 1946. Wilt of pineapple. Current Science, 15: 82.

Chowdhury, S. 1947. A *Glomerella* rot of nuna. Current Science, 16: 384.

Cohen, Y. and Coftey, M.D. 1986. Systemic fungicides and control of Oomycetes. Ann. Rev. Phytopathology, 24: 311.

Costa Santa-Cecilia, L.V. and Chalfoun, S.M. 1998. Pests and diseases which affect pineapple. Informe Agropecuario (Belo Horizonte), 19: 40-57.

Dai, Q. and Sun Z. H. 1995. Suppressive effects of N-triacontanol on symptoms of mulberry dwarf disease and on the causal phytoplasma.   Plant Pathology, 44: 979-981.

Damayanti, M., Susheela, K. and Sharma, G.J. 1996. Effect of plant extracts and systemic fungicide on the pineapple fruit rotting fungus *Ceratocystis paradoxa*. Cytobios, 86: 155-165.

Dar, G.H.Z. and Zargar, M.Y. 1986. Occurrence of *Entomosporium maculantum* Lev. on Loquat in India. Indian Journal of Mycology and Plant Pathology, 16: 342.

Darvis, J.M. 1992. Armillaria rot of litchi trees in South Africa. South African Litchi Growers Association Yearbook, 4: 2-4.

Das, N.D., Rao, M.S. and Shankar, B.R.M. 1994. Efficacy of two new fungicides for the control of powdery mildew of ber and their compatibility with insecticides. Annals of Plant Pathology Science 2: 76-78.

Dassanayake, E.M., Wickremasingha, D.L. and Perera, W.G.S. 1994. The use of enzyme linked immunosorbent assay (ELISA) for the detection of pineapple wilt virus in pineapple (*Ananas comosus*). Sri Lankan Journal of Agricultural Sciences, 31: 50-58.

Desai, S.A. and Jamadar, M.M. 1997. Fungicides for the control of fig rust under field conditions. South Indian Horticulture 45: 70-71.

Desai, S.A. 1998a. A note on the spray schedule for the control of powdery mildew of ber in Karnataka. Karnataka Journal of Agricultural Sciences, 11: 242-243.

Desai, S.A. 1998b. Chemical control fig of rust. Karnataka Journal of Agricultural Sciences, 11: 827-828.

Edmonstone-Sammons, C.P. 1958. Some aspects of black spot in pineapple. South African Journal of Agriculture Science, 1: 111-120.

Fotadar, R.K., Dhar, A. and Dandin, S.B. 1998. Screening of mulberry genotypes against major fungal diseases and insect pest under field conditions. Indian Journal of Sericulture, 37(1): 40-43.

Francisco, N.E., Nakamura, K. and Oliveira, J.C. 1994. Influence of some factors on the mycelial growth, sporulation, and conidial germination of *Colletotrichum gloeosporioides*, the causal agent of passion fruit anthracnose. Summa Phytopathologica, 20: 96-100.

Frisullo, S., Cacciola, S.O. and Pane, A. 1997. Twig blight of loquat caused by *Phytophthora cactorum*. Informatore Fitopatologico, 47: 30-32.

Gangwar, S.K., Sinha, P.S., Dutta, A.K., Singh, B.D. and Sinha, S.S. 1994. Incidence of disease and pests of mulberry in Chotanagpur (Bihar). Karnataka Journal of Agricultural Sciences, 7(4): 487-489.

Garg, P.K. and Gupta, M.N. 1980. Some new fruit rot diseases of *Carissa*. Indian Phytopathology, 33: 481-482.

Garud, B. 1968. An Anthracnose disease of pineapple in India. Plant Disease Reporter, 52: 436-437.

Gella, R., Marin, J.A., Corrales, M.L., Toribio, F. 1997. Elimination of fig mosaic from fig shoot tip cultures by thermotherapy. In: Proceedings of the First International Symposium on Fig, Izmin, Turkey, 24-28 June, 1997. (eds. Aksoy, U., Ferguson, L. and Hepakson, S.) Acta Horticulture, 480: 173-177.

Giri, D., Banerjee, K., Laha, S.K. and Khatua, D.C. 1989. Some diseases of horticultural and field crops. Environment and Ecology, 7: 821-825.

Goncalves-Gervasio, R. De C.R. and Santa-Cecilia, L.V.C. 1999. Foliar contents of phenolic compounds in pineapple as affected by wilt, cultivar and plant cycle. Revista Brasileira de Fruticultura, 21: 232-234.

Goncalves, E.R. and Rosato, Y.B. 2000. Genotypic characterization of *Xanthomonad* strains isolated from passion fruit plants (*Passiflora* spp.) and their relatedness to different *Xanthomonas* species. International Journal of Systematic and Evolutionary Microbiology, 50: 811-821.

Gonzalez, M.S., Suarez, Z., Rosales, C. and Parra, D. 2000. Collar rot and wilt of yellow passion fruit in Venezuela. Plant Disease, 84:103.

Gonzalez, V., Ramallo, J., Ramallo, N.E.V. De., Ploper, L.D. and Torres Leal, G. 1998. Fig fruit rot caused by *Phytophthora palmivora* in fig crops in Tucuman, Argentina. Fitopatologia, 33: 228-231.

Gopinath Nair, K.R. 1964. Some new records of fungi imperfecti from Bombay, Maharasthra.

University Journal, Poona, 26: 109-111.

Govindaiah, Gunasekhar, V., Gowda, P. and Thiagrajan, V. 1994. Field evaluation of fungicides against *Phyllactinia corylea* causing powdery mildew in mulberry (*Morus alba* L.). Indian Journal of Sericulture, 33: 160-162.

Govindu, H.C. and Thirmalachar, M.S. 1964. Notes on some Indian *Cercospore* XI. Sydowia, 18: 18-22.

Goyal, J.P., Desai, B.G., Bhatnagar, L.G. and Pathak, V.N. 1971. Fungal collection from Rajasthan State of India. Sydowia 25: 172-175.

Gunasekhar, V. and Govindaiah 1994. Disease assessment keys for three major diseases of mulberry. Indian Journal of Sericulture, 33: 122-125.

Gunasekhar, V., Govindaiah and Himantharaj, M.T. 1995. Efficacy of fungicides in controlling mulberry leaf rust caused by *Cerotelium fici*. Indian Journal of Sericulture, 34: 60-62.

Gupta, D., Bhatia, R., Sharma, N.K., Chandel, J.S. and Sharma, R. 1997. Incidence and management of red rust of litchi in lower hills of Himachal Pradesh. Pest Management in Horticultural Ecosystems, 3: 70-74.

Gupta, J.H. 1984. Germination of conidia of *Oidium erysiphoides* f sp. *zizyphi* causing powdery mildew of ber. Indian Journal of Mycology and Plant Pathology, 14: 294.

Gupta, J.H. 1986. Fungicidal control of phalsa rust. Indian Journal of Mycology and Plant Pathology, 16: 311.

Gupta, J.H. 1992. Chemical control of algal rust of litchi caused by *Cephaleuros virescens*. Progressive Horticulture, 24: 109-110.

Gupta, I.J. and Sehgal, S.P. 1974. A new Pestalotiopsis fruit rot of sapota from India. Indian Phytopathology, 27: 616.

Gupta, J.H., Ram Nath and Srivastava, V.P. 1977. Chemical control of powdery mildew of ber. Progressive Horticulture, 9: 81-83.

Gupta, P.C. and Madaan, R.L. 1975a Diseases of fruits from Haryana. I. A New fruit rot of *Zizyphus mauritiana* Lamk. Current Science, 44: 908.

Gupta, P.C. and Madaan, R.L. 1975b Two leaf spot disease of ber from Haryana. Current Science, 44: 248.

Gupta, P.C. and Madaan, R.L. 1977a Diseases of fruits from Haryana – a new leaf spot disease of ber. Current Science, 46: 37-238.

Gupta, P.C. and Madaan, R.L. 1977b Fruit rot diseases of Ber (*Zizyphus mauritiana* Lamk). from Haryana. Indian Phytopathology, 30: 554-555.

Gupta, P.C. and Madaan, R.L. 1978. Occurrence of Alternaria leaf spot disease of (*Zizyphus mauritian* Lamk.) and its control. Abstract, Symposium Advancing Frontiers in Plant Biology, BHU, Varanasi, pp. 45.

Gupta, P.C., Madaan, R.L. and Grover, R.K. 1978. Occurrence of powdery mildew of ber in Haryana and its control. Indian Phytopathology, 31: 440-443.

Gupta, P.C. and Madaan, R.L. 1979. *Hansfordia pulvinata* – A mycoparasite on *Isariopsis indica* var. *zizyphi*. Current Science, 48: 121-122.

Gupta, P.C. and Madaan, R.L. 1980. Varietal resistance against leaf spot of ber. HAU Journal of Research, 10: 75-76.

Gupta, P.C., Madaan, R.L. and Chauhan, K.S. 1980. Varietal resistance of ber against *Isariopsis* mouldy leaf spot disease. Indian Phytopathology, 33: 140-141.

Gupta, P.C., Madaan, R.L. and Yamadagni, R. 1984. Three rust fungi on fruit crops from Haryana. Indian Phytopathology, 37: 407.

Gupta, P.C. and Madaan, R.L. 1985. Report on pathological aspects of fruit crops (1975-83). Department of Horticulture, HAU, Hisar, pp. 1-42.

Gupta, V.P. 1999. On the occurrence of green mosaic disease on mulberry in India. Indian Phytopathology, 52: 154-155.

Gupta, V.P., Tewari, S.K. and Datta, R.K. 1995. Surface ultrastructural studies on ingress and establishment of *Pseudomonas syringae* pv. *mori* on mulberry leaves. Journal of Phytopathology, 143: 415-418.

Gupta, V.P., Govindaiah and Datta, R.K. 1996. *Plant extracts: a non chemical approach to control* Fusarium diseases of mulberry. Current Science, 71: 406-409.

Gupta, V.P., Sharma, D.D., Govindaiah; and Chandrashekar, D.S. 1999. Soil solarization for the control of nursery disease in mulberry. Indian Journal of Sericulture, 38: 44-47.

Hall, D.J. 1983. Fungicides for post harvest decay control in loquats. Proceedings Florida State Horticultural Society, 96: 366-367.

Harsh, N.S.K., Tiwari, C.K. and Nath, V. 1989. Foliage diseases in forest nurseries and their control. Journal of Tropical Forestry, 5: 66-69.

Hingorani, M.K. and Mehta, P.P. 1952. Bacterial leaf spot of pomegranate and its control. Indian Phytopathology, 5: 55-56.

Hosomi, A. and Kusakari, S. 1995. Yeast associated with soft rot of fig fruit in Japan. Proceedings of the Kansai Plant Protection Society, 37: 9-10.

Hu, J.S., Sether, D.M. and Ullman, D.E. 1995. Detection of pineapple closterovirus in pineapple plants and mealybugs using monoclonal antibodies. Phytopathology, 85: 1137.

Hu, J.S., Sether, D.M., Lin, X.P., Wang, M., Zee, F. and Ullman, D.E. 1997. Use of tissue blotting immunoassay to examine the distribution of pineapple closterovirus in Hawaii. Plant Disease, 81: 1150-1154.

Huang, P.U. and Scott, K.J. 1985. Control of rotting and browning of litchi fruits after harvest at ambient temperature in China. Tropical Horticulture, 62: 2-4.

Illingworth, J.F. 1931. Preliminary report on evidence that mealybugs are an important factor in mealybug wilt. Journal of Economic Entomology, 31: 291-298.

Im, H.B., La, Y.J., Lim, U.K., Chang, T.J., Shin, J.D. and Lee, S.H. 1985. Histopathological studies of witches broom infected jujube trees treated by oxytetracycline. Korean Journal of Plant Pathology, 1: 101-100.

Iwai, H., Ohmori, T., Kurokawa, Y., Muta, T. and Arai, K. 1996. New record of passion fruit woodiness virus in Japan. Annals of the Phytopathological Society of Japan, 62: 459-465.

Iwai, H., Sakai, J., Hanada, K. and Arai, K. 1997. Nucleotide sequence of the coat protein gene and 3'-noncoding region of the passion fruit woodiness virus-amami ohshima isolate. Annals of the Phytopathological Society of Japan, 63: 475-478.

Jain, S.K., Saxena, A. and Saxena, S.B. 1981. Two new fruit rot diseases of *Achras sapota*. Indian Phytopathology, 34: 403.

Jain, S.K., Saxena, A. and Saxena, S.B. 1983. A new leaf spot disease of *Z. jujube* Lamk. (*Botryodiplodia theobromae* on *Z jujube*). National Academy Science Letters, 5: 22.

Jamaluddin, 1978. Cladosporium rot of fruits of *Phyllanthus emblica*. Proceedings of National Academy of Science, India, 43: 62.

Jamaluddin, Tandon, M.P. and Tandon, R.N. 1975. A fruit rot of aonla caused by *Phoma*. Proceedings of National Academy of Science, India B., 45: 75-77.

Jamaluddin, Tandon, M.P. and Tandon, R.N. 1979. The nature of some metabolic changes infected aonla fruit. Proceedings of National Academic Science, India, 49(B): 113-115.

Jeyarajan, R. and Cheema, S.S. 1972. Screening of ber varieties for resistant to leaf spot and powdery mildew diseases. Indian Journal of Horticulture, 29: 353.

Jiang, Y.M., Cheng, F., Li, Y.B. and Liu, S.X. 1997a. A preliminary study on the biological control of post-harvest diseases of litchi fruit. Journal of Fruit Sciences, 14: 185-186.

Jiang, Y.M., Liu, S.X., Chen, F., Li, Y.B. and Zhang, D.L. 1997b. The control of post harvest browning of litchi fruit by sodium bisulphite and hydrochloric acid. Tropical Science, 37: 189-192.

Johnston, A. 1957. Pineapple fruit collapse. Malaysia Agricultural Journal, 40: 253-263.

Joshi, I.J., Saxena, A. and Saksena, S.B. 1983. Two new soft rot diseases of loquat from India. Current Science, 52: 610.

Kamiunten, H. 1995. Involvement of a plasmid in the expression of virulence in *Pseudomonas syringae* pv. *eriobotryae*. Annals of the Phytopathological Society of Japan, 61: 376-380.

Kamthan, K.P., Misra, R. and Shukla, A.K. 1981. Nigrospora fruit rot of *Emblica officinalis,* a new disease record. Science and Culture, 47: 371-372.

Kang, M.S. and Singh, I. 1991. Die back and leaf blight incited by *Pestalotiopsis mangiferae* (Henn.) Steyaert. Plant Disease Research, 6: 103-104.

Kang, S., Di, D.P., Li, X.H., Peng, S.Q., Mao, Y.M. and Zhou Z.Y. 1998. Identification of the pathogen of Chinese jujube brown cortex. Acta Phytopathologica Sinica, 28: 165-171.

Kanwar, Z.S. 1976. A note on bacterial disease of pomegranate (*Punica granatum* L.) in Haryana. Haryana Journal of Horticultural Science, 5: 171-180.

Kanwar, Z.S. and Thakur, D.P. 1972. Controlling post harvest soft rot of pomegranate fruits by treatment of pack of straw. Science and Culture, 38: 450-452.

Kanwar, Z.S. and Thakur, D.P. 1973. Some new fungal rots of pomegranate in Haryana. Science and Culture, 39: 274-276.

Kanwar, Z.S., Thakur, D.P. and Kadian, D.P. 1973. A note on effect of temperature and RH on the development of soft rot of pomegranate due to *Rhizopus arrhizus*. Indian Phytopathology, 26: 742-743.

Kapur, S.P., Chema, S.P. and Singh, M.P. 1975. Occurrence and control of powdery mildew of ber (*Zizyphus mauritiana* Lame.) in Punjab. Journal of Research PAU, 12: 26-29.

Khurana, S.M.P. and Singh, R.A. 1972. A note on flat limb of sapota in south India. Zeitschrift Pflanzenhrankheitin und Pflanzenschutz, 79: 310-312.

Kim, C.J. 1965. Witches broom of jujube tree (*Zizyphus jujube* Mill. Var. inermis Reld). IX. Effect of low temperature in winter upon the appearance of symptoms. Korean Journal of Plant Protection, 4: 7-10.

Kitajima, E.W., Rezende, J.A.M., Rodrigues, J.C.V., Chiavegato, L.G., Piza Junior, C.T. and Morozini, W. 1997. Green spot of passion fruit, a possible viral disease associated with infestation by the mite *Brevipalpus phoenicis*. Fitopatologia Brasileira, 22: 555-559.

Koga, K. 1999. Outbreaks of fruit rot on loquat and occurrence of benomyl fungicide low sensitivity strains of *Pestalotiopsis eriobotrifolia* and *Pestalotiopsis neglecta*, the causal fungi of loquat gray leaf spot, in Nagasaki Prefecture, Japan. Proceedings of the Association for Plant Protection of Kyushu, 45: 38-44.

Kontaxis, D.G. 1977. Chemical control of pink disease of pineapple fruit in the Philippines (abstract). Proceedings of American Phytopathology Society, 4: 207-208.

Kontaxis, D.G. and Hayward, A.C. 1978. The pathogen and symptomatology of pink disease of pineapple fruit in the Philippines. Plant Disease Reporter, 62: 446-450.

Kore, S.S. and Bhide, V.P. 1977. New hosts for *Curvularia lunata* var. *aeria*. Indian Phytopathology, 30: 409.

Kore, S.S. and Dhande, G.W. 1973. A new leaf spot of bel (*Aegle marmelos* Corr.) caused by *Fusarium roseum* Link from India. Punjabrao Krishi Vidyapeeth Research Journal, 1: 223-224.

Kruger, F., Rabie, E., Wesson, K. and Tustin, H. 1998. The fight against blackheart in 'Queens'. Neltropika Bulletin, 304: 18-20.

Kumar, A., Bhansali, R.R. and Arya, H.C. 1978. A note on the occurrence of powdery mildew of ber (*Zizyphus* spp.) in Indian arid zone. Annals Arid Zone, 17: 323-325.

Kumar, P.M.P. and Vijayan, K. 1999. Effects of extracts of different plants on seed germination and seedling growth of mulberry *Imorus indica* L.). Indian Journal of Plant Physiology, 4: 343-345.

Kumar, S. and Tandon, M.P. 1978. A new fruit rot of *Carissa*. Indian Phytopathology, 31: 105.

Kumar, S. and Tandon, M.P. 1982. A new fruit rot of *Carissa*. Current Science, 51: 1119.

Kumar, V. and Patel, P.N. 1968. Symptoms and host range in a bacterial disease on *Carissa carandas* L. Indian Phytopathology, 21: 315-317.

Kumar, V., Babu, A.M., Sharma, D.D. and Datta, R.K. 1998a. Penetration and infection process of *Phyllactinia corylea* on mulberry leaf causing powdery mildew-I. Asexual stage. Journal of Phytopathology, 146: 469-472.

Kumar, V., Sharma, D.D., Babu, A.M. and Datta, R.K. 1998b. Studies on the hyphal interactions between a biocontrol agent *Trichoderma harzianum* and a mycopathogen *Fusarium solani* causing root rot disease in mulberry. Indian Journal of Sericulture, 37: 17-20.

La, Y.J., Brown Jr. W.M. and Moon, D.S. 1977. Control of witches broom disease of jujube with oxytetracycline injection. Korean Journal of Plant Protection, 15: 107-110.

Lal, A. and Singh, R.P. 1953. Anthracnose disease of *Carissa carandas* Linn. caused by *Coleototrichum inamdarii*. Journal of Indian Botanical Society, 32: 54-63.

Lal, B., Arya, A., Rai, R.N. and Tewari, D.K. 1982. A new soft rot of aonla caused by *Phomopsis phyllanthi* and its chemical control. National Academy of Science Letters, 5: 183-185.

Lele, V.C. and Ram, A. 1969. Two new disease of loquat in India. Indian Phytopathology, 22: 502-504.

Lele, V.C. and Butani, D.K. 1975. Trends in plant disease control in India with particular reference to fruit crops. Pesticides, 9: 75-95.

Lim, W.H. 1978. Studies on the etiology, epidemiology, ecology and control of pineapple fruit collapse. Ph.D. Thesis, University of Cambridge, England, 409 pp.

Lim, W.H. 1983. Marbled fruit, a potentially serious disease of Hybrid I pineapples. MARDI Fruit Br. Info. Leaflet No. 15, 9 pp. (Mimeo.).

Lim, W.H. 1985. Bacterial diseases of pineapple. In: "Review Tropical Plant Pathology", Today and Tomorrow's Printers and Publishers, New Delhi, 2: 127-140.

Lim, W.H. and Lowings, P.H. 1979. Pineapple fruit collapse in Peninsular Malaysia: symptoms and varietal susceptibility. Plant Disease Reporter, 63: 170-174.

Lim, W.H. and Lowings, P.H. 1982. Some ecological aspects of the transmission of fruit collapse disease in pineapple. Acta Oecologica, 3: 71-77.

Lim, W.H. and Lowings, P.H. 1983. Pathogenic of *Erwinia chrysanthemi* from pineapple with strains from other hosts. MARDI Research Bulletin, 11: 342-350.

Lin, K.J. and Li, G.X. 1985. Observations on symptom development of jujube witch's broom disease. Shanxi Fruit Trees, 19: 35-37.

Linford, M.B. 1952. Pineapple diseases and pests in Mexico. FAO Planta Protection Bulletin, 1: 21-25.

Liu, T.C., Huang, S.H. and Yang, L.J. 1991. Occurrence and control of loquat gray spot. Bulletin Taichung Agriculture Information Station, 30: 43-51.

Liu, T.D. 1995. The occurrence of *Pestalotiopsis eriobotryicola* in loquat and effects on yield and quality of fruits. Bulletin of Taichung District Agricultural Improvement Station, 47: 59-66.

Lodha, S. 1984. Wilt of jujube caused by *Fusarium equiseti*. FAO Plant Protection Bulletin, 31(2): 95.

Lukose, C. and Singh, R.D.1997. Climatic factors affecting the severity of pomegranate fruit rot. Journal of Mycology and Plant Pathology, 27: 48-50.

Luo, H.H.. and Zhong, S.Z. 1998. Effects of black-inhibitor for controlling black heart disease in pineapple. Journal of Guangxi Agricultural University, 17: 233-240.

Mabagala, R.B. and Maerere, A.P. 1998. First report of pink fruit disease of pineapple in Tanzania. Fruits (Paris), 53: 235-240.

Madaan, R.L. and Gupta, P.C. 1976. Diseases of fruits from Haryana-II. Two new leaf spot diseases of ber. Indian Phytopathology, 29: 328.

Madaan, R.L. and Chand, J.N. 1985. Epidemiological studies on Alternaria leaf spot and fruit rot disease of ber. Indian Journal of Mycology and Plant Pathology, 15: 26-27.

Madaan, R.L. and Gupta, P.C. 1985. A leaf spot disease of *Aegle marmelos* L. caused by *Alternaria alternata* (Fr.) Keissler. Indian Journal of Plant Pathology, 3: 239.

Maheshwari, S.K. and Singh, S.B. 1999. Effect of temperature and relative humidity on development of powdery mildew of ber (*Ziziphus mauritiana* Lamk.). Annals of Planta Protection Sciences, 7: 105-107.

Maji, M.D., Qadri, S.M.H., Gangwar, S.K., Kumar, P. M.P. and Saatchandra, B. 1999. A new brown ring leaf spot disease of mulberry. Indian Phytopathology, 52: 203.

Majumdar, V.L. and Singh, J. 1997. Management of Alternaria rot in pomegranate by chemicals. Golden Jubilee International Conference on "Integrated Plant Disease Management for Sustainable Agriculture", November 10-15, 1997, New Delhi, Indian Phytopathological Society (Abstract), p. 322.

Mallikarjunaradhya, A.V., Bhat, A.V., Gown, J.K., Rao, A.R.V., Ramana, K.V.R. and Narasimhan, P. 1979. Control of fungal stem end rot during transport of pineapple. Journal of Food Science & Technology, 16: 232-234.

Manicom, B.K. 1995. Litchi dieback. Year Book – South Africa Litchi Growers Association, 7: 3-4.

Marcano, D.A. De., Bravo, I.M. De., Gallardo, E. and Prays, R. 1993. Distribution and incidence of soft rot on pineapple in the municipality of Crespo in the state of Lara. FONAIAP Divulga, 10: 8-9.

Martelleto, L.A.P., Castilho, A.M.C. and Goes, A. De 1998. Influence of incubation temperature on mycelial growth, sporulation and pathogenicity of *Fusarium subglutinans*, the causal agent of Fusarium wilt in the pineapple plant. Summa Phytopathologica, 24: 242-246.

Mazzani, C. 1994. *Colletotrichum gloeosporioides* causing a severe spotting and rot of pomegranate fruits in Venezuela. Fitopatologia Venezolana, 7(1): 28.

McLauchan, R.L., Mitchell, G.E., Johnson, G.I., Nottingham, S.M. and Hammerton, K.M. 1992. Effect of disinfection dose of irradiation on the physiology of Taiso Lychee. Post harvest Biology and Technology, 1: 273-281.

Mehta, P.R. 1940. Stem end rot and soft rot of pineapple in United Province. Current Science, 9: 330.

Mehta, P.R. 1950. Some new diseases of plants of economic importance in Uttar Pradesh. Plant Protection Bulletin, New Delhi, 2: 50-51.

Mehta, P.R. and Bose, S.R. 1947. A leaf spot disease of fig caused by *Cylindrocladium scoparium*. Indian Journal of Agricultural Science, 17: 219-221.

Michailides, T.J., Morgan, D.P. and Subbarao, K.V. 1996. An old disease still a dilemma for California growers. Plant Disease, 80: 828-841.

Milanes, V.P. and Herrera, I.L. 1994. Over wintering of *Thielaviopsis paradoxa* (de Seynes) Hohn on sugarcane and pineapple residues in a typical carbonated soil. Centro Azucar, 21: 66-72.

Miriam, Z., Shula, M. and Frieda, K. 1999. Etiology and control measures of fire blight in Loquat. In: "Proceedings of the Eighth International Workshop on Fire Blight, Kusadasi, Turkey, 12-15 October, 1998" (eds. Momol, M.T. and Saygili, H.). Acta Horticulturae, 489: 495-497.

Mishra, A. and Shivpuri, A. 1983. Anthracnose – a new disease of aonla. Indian Phytopathology, 36: 406-407.

Mishra, B., Prakash, O. and Misra, A.P. 1973. *Gloeosporium eugeniae* causing anthracnose disease on *Syzygium cuminii* Skeels from India. Indian Journal of Horticulture, 30: 448-450.

Misra, A.K., Das, B.K. and Ahsan, M.M. 1994. A new record of *Coniothyrium foedans* on mulberry. Indian Phytopathology, 47: 439.

Misra, A.K. and Das, B.K. 1998. Incidence of powdery mildew disease in mulberry in Darjeeling hills as influenced by some abiotic factors. Indian Agriculturist, 42: 49-53.

Misra, A.K. and Pandey, B.K. 2001. Diseases of litchi and their management. In: "Disease of Fruits and Vegetables and their management". (ed. Thind, T.S). Kalyani Publishers, Ludhiana, pp. 150-153.

Mitra, A. 1935. Investigations on the wound-paracitism of certain *Fusaria*. Indian Journal of Agricultural Sciences, 5: 632-637.

Mitter, J.N. and Tandon, R.N. 1930. The fungus flora of Allahabad. Journal of Indian Botanical Society, 9: 197.

Moniz, L., Sabley, J.E. and More, W.D. 1964. A new bacterial canker of *Carisa congesta* in Maharashtra. Indian Phytopathology, 17: 256.

Morita, A. 1991. Seasonal occurrence of loquat canker and analysis of its affecting factors. Proceeding of Association Plant Protection Kynshu, 37: 63-71.

Morita, A. 1995. Occurrence of southern blight disease caused by *Sclerotium rolfsii* on loquat. Annals of the Phytopathology Society of Japan, 61: 197-201.

Morita, A. 1997. Causal factors of Tate-boys, a new disease of loquat fruit. Annals of the Phytopathological Society of Japan, 63: 44-50.

Mourichon, X. 1983. Fruitlet core rot or leathery pocket of banana caused by *Penicillium funiculosum*. Fruits, 38: 601-609.

Nagaich, B.B. and Vashisht, K.S. 1962. Mosaic disease of fig in Himachal Pradesh. Current Science, 31: 166-167.

Navale, A.M., Padule, D.N. and Kaulgud, S.N. 1998. Efficacy of different fungicides against leaf and fruit spots of pomegranate in Mrig Bahar. Journal of Maharashtra Agricultural Universities, 23: 251-253.

Nema, S. and Sharma, N.D. 1996. *Phytophthora* blight and damping-off of custard apple from India. Indian Phytopathology, 49: 407.

Nitta, H., Imada, J., Kano, T., Nakamoto, K. and Ogasawara, S. 1995. Occurrence and causes of fig mosaic symptoms in Hiroshima Prefecture. Bulletin of the Hiroshima Prefectural Agriculture Research Centre, 62: 53-65.

Nojima, H., Kiku, T., Kumamoto, D. 1995. Fruit rot of loquat in the Amami Islands. Proceedings of the Association of Plant Protection of Kyuschu, 41: 36-42.

Novaes, Q.S. and Rezende, J.A.M. 1999. Possible use of indirect DAS-ELISA for screening of passion fruit tolerant to passion fruit woodiness virus. Fitopatologia Brasileira, 24: 76-79.

Ou, Z.J., Deng, W.S. and Wu, C.T. 1999. Experiment of control of litchi downy mildew disease by using 80% mancozeb wetted powder. China Fruits, 3: 32.

Pandey, B.N., Misra, U.S., Yadav, S., Pandey, R.R. and Dwivedi, R.S. 1986. A new leaf spot of ber (*Zzyphus jujuba*) from Rohilhand region of India. Acta Botanica Indica, 14: 236-242.

Pandey, R.S., Bhargava, S.N., Shukla, D.N. and Khati, D.V.S. 1979. Control of Rhizopus rot of Jackfruit. Indian Phytopathology, 32: 479-480.

Pandey, R.S., Khati, D.V.S., Shukla, D.N. and Bhargava, S.N. 1980a. A new Phoma rot of bael fruit. Indian Phytopathology, 33: 319.

Pandey, R.S., Shukla, D.N., Khati, D.V.S. and Bhargava, S.N. 1980b. A new fruit rot of *Phyllanthus emblica*. Indian Phytopathology, 33: 491.

Pandey, R.S., Bhargava, S.M., Shukla, D.N. and Dwivide, D.K. 1984. Two new fruit diseases of aonla caused by *Alternaria* species. International Journal of Tropical Plant Disease, 2: 79-80.

Pandey, P.K., Singh, A.B., Nimbalkar, M.R. and Marathe, T.S. 1976. A witches' broom disease of jujube from India. Plant Disease Reporter, 60: 301-303.

Panwar, K.S. and Vyas, N.L. 1974. *Cladisporium oxysporum* causing fruit rot in pomegranate and ber. Indian Phytopathology, 27: 121-122.

Pares, R.D., Gunn, L.V., Keskula, E.N., Martin, A.B. and Teakle, D.S. 1997. Occurrence of passifloroa latent carlavirus in cultivated and wild *Passiflora* species in Australia. Plant Disease, 81: 348-350.

Parkash, V. and Jhooty, J.S. 1987. Epidemiology of powdery mildew of *Ziziphus mauritiana* caused by *Microsphaera alphitoides* f.sp. *ziziphi*. Indian Phytopathology, 40: 491-494.

Parkash, V., Kaur, G.P. and Jhooty, J.S. 1988. Perpetuation of *Microsphaera alphitoides* f.sp. *ziziphi* causing powdery mildew of ber. Indian Phytopathology, 41: 141-143.

Patel, M.K., Kamat, M.N. and Bhide, V.P. 1949. Fungi of Bombay. Supplement I. Indian Phytopathology, 2: 142-155.

Patel, M.K., Allayyanavaramath, S.B. and Kulkarni, Y.S. 1953. Bacterial shot hole and fruit canker of *Aegle mormelos* Correa. Current Science, 22: 216-217.

Patel, P.B. and Chauhan, H.L. 1993. Twig blight of sapota caused by *Botryodiplodia theobromae* – a new record. Indian Journal of Mycology and Plant Pathology, 23: 219.

Patel, P.B. and Chauhan, H.L. 1997. Effect of different fungicides for the control of twig blight in sapota. Indian Phytopathological – Golden Jubilee International Conference on "Integrated Plant Disease Management for Sustainable Agriculture", November 10-15, 1997, New Delhi (Abstract), p. 323.

Pathak, V.N. 1980. Disease of fruit crops. Oxford and IBH Publishing Co., New Delhi, 309 p.

Pathak, V.N. and Desai, B.G. 1971. A new species of *Microdiplodia* on *Nephelium litchi* Camb. Sydowia, 24: 73-74.

Pathak, V.N., Doshi, A. and Mathur, S. 1988. Management of diseases in tropical fruits. In: "Tree Protection" (eds. Gupta, V.K. and Sharma, N.K.) ISTS, Solan, pp. 62-67.

Payak, M.M. 1953. Some new records of fungi from the Bombay State. Science and Culture, 18: 342-343.

Perello, A.E. and Larren, S. 1999. First report of *Pestalotiopsis guepini* on loquat in Argentina. Plant Disease, 83: 695.

Perez, M.C., Borras, O., Arzola Gonzalez, M. and Rodriguez, Y. 1994. Report of *Fusarium moniliforme* var. *subglutinans* as a pathogen of pineapple in Cuba. Centro Agricola, 21: 88-90.

Philip, T., Govindaiah and Datta, R.K. 1994a. Sources of resistance in mulberry to leaf rust. Indian Phytopathology, 47: 201-202.

Philip, T., Govindaiah and Saramangala, H.S. 1994b. Two new host records in mulberry. Indian Phytopathology, 47: 214.

Philip, T., Gunasekhar, V., Govindaiah and Datta, R.K. 1994c. Measures to control leaf-blight of mulberry. Indian Farming, 43: 9-10.

Philip, T., Janardhan, L., Govindaiah and Bajpai, A.K. 1994d. Black leaf spot disease of mulberry – first report. Indian Journal of Sericulture, 33: 186-187.

Philip, T. and Govindaiah 1995. *Pestalotiopsis disseminata* (Thum.) Steyaert – a new pathogen on mulberry. Indian Journal of Sericulture, 34: 159-160.

Philip, T., Bajpai, A.K., Govindaiah and Sarkar, A. 1995a. Screening of mulberry genotypes for resistance to *Mycophaerella mori* leaf spot. International Journal of Tropical Plant Diseases, 13: 199-204.

Philip, T., Janardhan, L., Govindaiah, Mallikarjuna, B., Mandal, K.C. and Bajpai, A.K. 1995b. Some observations on the incidence, associated microflora and control of root-rot disease of mulberry in south India. Indian Journal of Sericulture, 34: 137-139.

Philip, T. and Govindaiah 1996. Studies on the factors contributing for disease resistance in mulberry against *Cercospora moricola*. Indian Journal of Sericulture, 35: 39-42.

Philip, T., Sarkar, A. and Govindaiah 1996. Screening of some promising genotypes of mulberry for leaf spot and rust resistance. Indian Journal of Sericulture, 35: 158-159.

Poltronieri, L.S., Trindade, D.R., Albuquerque, F.C. and Benchimol, R.L. 1999. Web blight

(*Thanatephorus cucumeris*) of passion fruit in the state of Para, Brazil. Fitopatologia Brasileira, 24: 92.

Prakasam, V. and Subbaraja, K.T. 1994. Manage heart-rot of pineapple. Indian Horticulture, 39: 14.

Prakash, O., Singh, S.J. and Tewari, R.P. 1975. An anthracnose disease of *Syzygium cuminii* from India. Indian Phytopathology, 28: 267-268.

Prasad, K.V., Yadav, B.R.D. and Sullia, S.B. 1997. Effect of mulching on severity of mulberry rust disease. Iran Agricultural Research, 16: 139-146.

Prasad, N. and Verma, A.P. 1970. A new genus of moniliates from India. Indian Phytopathology, 23: 111-113.

Prasad, S.S. 1962. Two new leaf spot disease of *Nephelium litchi* Camb. Current Science, 31: 293.

Prasad, S.S. 1967. Leaf spot diseases of *Nephelium litchi* Camb. Indian Phytopathology, 20: 50-53.

Prasad, S.S. and Bilgrami, R.S. 1969. Investigation on diseases of litchi-I. Phyllosphere mycoflora of *Litchi chinensis* in relation to fruit rot. Indian Phytopathology, 22: 507-510.

Prasad, S.S. and Bilgrami, R.S. 1973a. Investigation on diseases of litchi-II. Influence of temperature and humidity on the decay of fruits caused by nine virulent pathogens. Indian Phytopathology, 26: 517-522.

Prasad, S.S. and Bilgrami, R.S. 1973b. Investigation on diseases of litchi-III. Fruit rots and their control by post harvest treatments. Indian Phytopathology, 26: 523-527.

Prasad, S.S. and Bilgrami, R.S. 1975. Investigation on diseases of litchi. VII – Antibiotics for control of Aspergillus fruit rot. Proceedings of National Academic Science India B, 45: 40-42.

Prasada, R., Pathak, V.N., Goyal, J.P. and Sharma, H.C. 1971. Proceedings Second International Symposium Plant Pathology, New Delhi. Indian Phytopathological Society, New Delhi, pp. 145-146.

Qadri, S.M.H., Gangwar, S.K., Kumar, P.M.P., Elangovan, C., Das, N.K., Maji, M.D. and Saratchandra, B. 1999. Assessment of cocoon crop loss due to leaf spot disease of mulberry. Indian Journal of Sericulture 38(1): 35-39.

Radhakrishan, N.U., Ramabadran, R. and Jayaraj, J. 1995. *Botrydiplodia* root rot – a new diseases of mulberry. Indian Phytopathology, 48: 492.

Rai, R.N. 1982. Pathological and physiological studies of certain fungi causing fruit rot disease. D.Phil Thesis University of Allahabad, 217 p.

Rajendran, V. 1971. Leaf spot of sapota. Lal Bagh, 16: 21-22.

Ramakrishnan, T.S. and Sundaram, N.V. 1954. Notes on some fungi from south India-III. Indian Phytopathology, 7: 61-68.

Randhawa, G.S. and Singh, R.K.M. (eds) 1970. The Loquat in India, ICAR, New Delhi, 62 p.

Rangaswamy, G., Seshadri, V.S. and Lucy Channamma, K.A. 1970. Fungi of India. University of Agricultural Sciences, Bangalore, 193 p.

Rani, U. and Verma, K.S. 2001. Field evaluation of different chemotherapeutants against black spot of pomegranate. Plant Disease Research, 16: 87-88.

Rao, R.N. 1962. Some *Cercospora* species from Hyderabad. Indian Phytopathology, 15: 112-140.

Rao, V.G. 1963. Some new records of folicolous fungi imperfecti from India. Bulletin Botanical Society College of Science, Nagpur, 42:5-57.

Rao, V.G. 1971. An account of fungus genus Alternaria Nees from India. Mycopath. Et. Mycol. Applicata, 43: 361-74.

Rao, V.G. and Mhaskar, D.N. 1973. Rivista Di Patol, Veg. IX(iv): 131-137.

Rathaiah, Y. and Pavgi, M.S. 1973. *Fusarium semitectum* mycoparasitic on *Cercosporae*. Phytopathologische Zeitschrift, 77: 278-281.

Rathore, R.S. 1970. Further studies on bacterial leaf spot of *Carissa carandas* L. M.Sc. Thesis, University of Udaipur, Campus-Jobner.

Rawal, R.D. 1993. Fungal diseases of tropical fruits. In: "Advances in Horticulture Vol. 3 Fruit crops" (eds. Chadha, K.L. and Pareek, O.P.). Malhotra Publication House, New Delhi, pp.

Rawal, R.D. and Saxena, A.K. 1989. Evaluation of different fungicides against black leaf spot of ber. Indian Journal of Horticulture, 46: 413-414.

Raychaudhuri, S.P., Chatterjee, S.N. and Dhar, H.K. 1961. Preliminary note on the recurrence of yellow-net vein disease of mulberry. Indian Phytopathology, 14: 94-95.

Raychaudhuri, S.P., Chatterjee, S.N. and Dhar, H.K. 1962. A mosaic disease of mulberry. Indian Phytopathology, 15: 187-189.

Raychaudhuri, S.P., Ganguli, B. and Basu, A.N. 1965. Further studies on the mosaic disease of mulberry. Plant Disease Reporter, 49: 981.

Raychaudhuri, S.P., Ganguli, B. and Basu, A.N. 1966. Virus disease of mulberry in India. Plant Disease Proceedings Ist Symposium on Plant Pathology, pp. 274-277.

Raychaudhuri, S.P. and Lele, V.C. 1970. Producing disease free planting materials of fruits. Indian Farming, 20: 11-14.

Reddy, S.M. 1975. Some new leaf spot diseases caused by Hyphomycetes. Proceedings National Academy of Science, India, 45B: 97-100.

Reddy, M.M., Rao, D.M. and Reddy, G.S. 1990. Preliminary field evaluation of some fungicides in the control of powdery mildew (*Oidium erysiphoides* Fr.) on ber in alfisol of arid zone of Andhra Pradesh. Madras Agricultural Journal, 77: 229-330.

Reddy, M.M., Reddy, G.S. and Madhusudan, T. 1997. Evaluation of some ber (*Zizyphus mauritiana* L.) varieties and fungicides against powdery mildew. Journal of Research ANGRAU, 25: 19-26.

Reddy, P.P. and Nagesh, M. 2000. Nematode diseases of tropical and sub-tropical fruits. In: "Diseases of Fruit Crops" (eds. Gupta, V.K. and Sharma, S.K.). Kalyani Publishers, Ludhiana, pp. 251-265.

Rishi, N. and Raychaudhuri, S.P. 1981. Some studies on bushy stunt diseases of *Grewia asiatica* Linn. Mant. In: "Proceedings XVII IUFRO World Congress, Kyoto, Japan 1981. Pass", pp. 347-350.

Rocha, J. De. R. De S. and Oliveira, N.T. De. 1998a. Biocontrol of *Colletotrichum gloeosporioides*, anthracnose agent on passion fruit (*Passiflora edulis*), with *Trichoderma koningii*. Summa Phytopathologica, 24: 272-275.

Rocha, J.De., R. De S. and Oliveira, N.T. De 1998b. *In vitro* antagonistic potential of *Trichoderma* spp. against *Colletotrichum gloeoporioides* agent of anthracnose in passion fruit (passiflora). Boletin Micrologico, 13: 103-110.

Rocha, J.De R. De S., Oliveira, N.T. De and Menezes, M. 1998. Comparison of inoculation methods efficiency for evaluation of *Collletotrichum gloeosporioides* isolates pathogenicity on passion fruits (*Passiflora edulis*). Brazilian Archives of Biology and Technology, 41: 145-153.

Rohrbach, K.G. and Pfeiffer, J.B. 1976. The interaction of four bacteria causing pink disease of pineapple with several pineapple cultivars. Phytopathology, 66: 396-399.

Rohrbach, K.G., Beardley, J.W., German, T.L., Reimer, N.J. and Sanford, W.G. 1988. Mealybug wilt, mealybugs and ants on pineapple. Plant Disease, 72: 558-565.

Saha, L.R. 2002. Hand Book of Plant Diseases. Kalyani Publishers, Ludhiana, India, 302 p.

Sahin, F., Kotan, R. and Donmeiz, M.F. 1999. First report of bacterial blight of mulberries caused by *Pseudomonas syringae* pv. *mori* in the eastern Anatolia region of Turkey. Plant Disease, 83: 1176.

Saini, S.S. and Uppal, H.S. 1981. Nitrogen nutrition of *Cladosporium zizyphi* the causal organism of leaf spot disease of ber. Indian Journal of Mycology and Plant Pathology, 11: 263-65.

Sanewski, G.M. and Giles, J. 1997. Blackheart resistance in three clones of pineapple (*Ananas comosus* (L.) Merr.) in sub-tropical Queensland. Australian Journal of Experimental Agriculture, 37: 459-461.

Sarbajha, K.K. 1989. New host records of some hyphomycetous fungi from Indian. Indian Phytopathology, 42(4): 600-601.

Sato, T., Uematsu, S., Mizoguchi, H., Kiku, T. and Miura, T. 1997. Anthracnose of prairie gentian and loquat caused by *Colletotrichum acutatum*. Annals of the Phytopathological Society of Japan, 63: 16-20.

Sawant, I.S., Sawant, S.D. and Ganapthy, M.M. 1995. Collar rot of passion fruit caused by *Rhizoctonia solani* and its control. Indian Phytopathology, 48: 202-205.

Saxena, A.K., Saksena, S.B. and Jain, S.K. 1981. Sclerotium rot of *Grewia asiatica* Linn. Mant. Science and Culture, 47: 440.

Schutte, G.C., Botha, T. and Kotze, J.M. 1990. Post harvest control of decay and browning of litch fruit by fungicide dips and paper sheets impregnated with sodium metabisulphite. Litchi Year Book – South Africa Litchi Grower association, 3: 10-14.

Serrano, F.B. 1934. Fruitlet black rot of pineapple in the Philippines. Philippines Journal of Science, 57: 29-62.

Sether, D.M. and Hu, J.S. 1997. Transmission of pineapple closterovirus (PCV) by two species of mealybug. Phytopathology, 87 (Supplement): S 88.

Setty, K.G.H. 1959. Blue mould of aonla fruits. Current Science, 27: 27-28.

Sharma, D.D., Govindaiah, Katiyar, R.S., Das, P.K., Janardhan, L., Bajpai, A.K. and Choudhury, P.C. 1995. Effect of VA-mycorrhizal fungi on the incidence of major mulberry diseases. Indian Journal of Sericulture, 34: 34-37.

Sharma, D.D., Govindaiah, Ghosh, A., Philip, T., Ambika, P.K. and Choudhury, P.C. 1996. Effect of seasons, spacings, host genotypes and fertilizer doses on the incidence of major foliar diseases in mulberry. Indian Journal of Sericulture, 35: 57-61.

Sharma, M., Majumdar, U.L. and Mukesh, M. 1993. Some new post harvest diseases of ber fruits in India. Indian Phytopathology, 46: 415.

Sharma, N.D. and Jain, A.C. 1981. *Oidium grewiae* sp. nov. and *Phakopsora grewiae* (Pat. & Har.) Cumm. – two new diseases of *Grewia asiatica* L. from Jabalpur. Current Science, 50: 133-134.

Sharma, R.B., Roy, A.N. and Verma, R.K. 1979. A soft fruit rot of *Aegle marmelos*. Current Science, 48: 22-23.

Sharma, R.B., Sinha, B.P. and Ray, A.N. 1981. Post harvest fruit rots of pomegranate. Indian Phytopathology, 34: 69-70.

Sharma, R.L. 1998. Occurrence of dry rot of pomegranate in Himachal Pradesh. Plant Disease Research, 13: 175-176.

Sherkar, B.V. and Utikar, P.G. 1982a. *Beltraniella humicola* – A new fruit spotting fungus on pomegranate. Indian Journal of Mycology and Plant Pathology, 12: 50.

Sherkar, B.V. and Utikar, P.G. 1982b. *Fusarium fusarioides* – A new leaf spot disease of pomegranate. Indian Journal of Mycology and Plant Pathology, 12: 51.

Shivapratap, H.R., Philip, T. and Sharma, D.D. 1996. *In vitro* antagonism of *Trichoderma* species against mulberry leaf spot pathogen, *Cercospora moricola*. Indian Journal of Sericulture, 35: 107-110.

Shivpuri, A. and Mishra, A. 1982. Occurrence of *Phoma sorghina* on phalsa. Indian Journal of Mycology and Plant Pathology, 12: 83.

Shreemali, J.N. 1972. Some new members of Sphaeropsidales from India. Indian Phytopathology, 25: 58-60.

Singh, B.B. and Mukerjee, P. 1979. *Pseudocercospora* causing leaf blotch of *Carissa carandas*. Indian Phytopathology, 32: 468-470.

Singh, D., Andotra, P.S. and Singh, D. 1989. Fungicidal control of Isariopsis leaf spot of ber. Indian Journal of Mycology and Plant Pathology, 19: 105-106.

Singh, G., Singh, R.N. and Bhandari, T.P.S. 1984. Ascochyta leaf spots on fig and hops in India. Indian Journal of Mycology and Plant Pathology, 14: 100.

Singh, G. and Singh, R.N. 1986. Control of leaf spot disease of fig caused by *Ascochyta caricae*. Indian Phytopathology, 39: 462-463.

Singh, H.P. and Sidhu, S.S. 1985. Control of ber powdery mildew. Indian Horticulture, 29: 27.

Singh, R., Niwas, R., Singh, R. and Gupta, P.C. 1999. Prediction model for powdery mildew disease build up in ber based on weather parameters. Annals of Agri-Bio Research, 4: 11-15.

Singh, S.B., Upadhyay, J. and Prasad, B. 1969. Dieback of loquat by *Cytospora eriobotryae*. Indian Phytopathology, 22: 525-526.

Singh, S.B., Maheshwari, S.K. and Singh, P.N. 1995. Field evaluation of fungitoxicants against powdery mildew of ber. Annals of Plant Protection Science, 3: 168-169.

Singh, S.J. 1972. A sclerotium wilt of pineapple from India. Sydowia. Annales Mycologiae, 26: 204-206.

Singh, S.J. and Sastry, K.S.M. 1974. Wilt of pineapple – A new virus disease in India. Indian Phytopathology, 27: 298-303.

Singh, S.J. and Sastry, K.S.M. 1975. Effect of different fertilizer and spacing on the incidence of pineapple wilt virus. Indian Journal of Mycology and Plant Pathology, 5: 156-160.

Singh, S.P. and Singh, R.K. 1972. Studies on disease of mango and other fruit plants. Proceedings Third International Symposium on Subtropical Horticulture, Bangalore, 121 p.

Singh, Y.P. and Sumbali, G. 2000. Ascorbic acid status and aflatoxin production in ripe fruits of jujube infected with *Aspergillus flavus*. Indian Phytopathology, 53: 38-41.

Sinha, J.N. and Singh, A.P. 1995. Some new host records from India. Indian Phytopathology, 48: 111.

Sohi, H.S., Jain, S.S., Sharma, S.L. and Verma, B.R. 1964. New records of plant diseases from Himachal Pradesh. Indian Phytopathology, 17: 42-45.

Sohi, H.S. and Sridhar, T.S. 1972. Chemical control of leaf spot disease of sapota caused by *Phaeophleospora indica*. Indian Journal of Agricultural Science, 42: 1139-1142.

Sohi, H.S. and Sridhar, T.S. 1973. Chemical control of leaf spot disease of sapota caused by *Phaeophleospora indica*. Indian Journal of Horticulture, 30: 553-557.

Somasekhara, Y.M. 1999. New record of *Ceratocystis fimbriata* causing wilt of pomegranate in India. Plant Disease, 83: 400.

Sood, A.K., Sharma, R.D. and Singh, B.M. 1987. Red rust-emerging threat to litchi cultivation in Kangra Valley of Himachal Pradesh. Indian Horticulture, 32: 19-20.

Sridhar, T.S. 1975. Black rot of pineapple – a new record from south India. Current Science, 44: 869.

Srikantaswamy, K., Govindaiah., Reddy, M.M., Bajpai, A.K. and Raveesha, K.A. 1996. Effect of *Cercospora moricola* on the leaf quality in mulberry. Indian Journal of Sericulture, 35: 144-146.

Srikantaswamy, K., Gupta, V.P., Gunasekhar, V. and Renukeswarappa, J.P. 1998. Evaluation of fungicidal control of leaf spot and powdery mildew diseases of mulberry at farmers' fields. Indian Journal of Sericulture, 37: 159-162.

Srikantaswamy, K., Gupta, V.P. and Renukeswarappa, J.P. 1999. Incidence, severity and yield loss due to leaf spot of mulberry caused by *Cercospora moricola*. Indian Journal of Sericulture, 38: 8-11.

Srivastava, A.K. and Lal, B. 1997. Studies on biofungicidal properties of leaf extract of some plants. Indian Phytopathology, 50: 408-411.

Srivastava, H.C. and Mehta, P.R. 1951. A new species of *Cercospora* on *Grewia asiatica*. Indian Phytopathology, 4: 67-70.

Srivastava, M.P., Tandon, R.N., Bilgrami, K.S. and Ghosh, A.K. 1964. Studies on fungal diseases of some tropical fruits. I-A list of fungi isolated from fruits and fruit trees. Phytopathologische Zeitschrift, 50: 250-261.

Srivastava, M.P. and Tandon, R.N. 1971. Aspergillus rot of pomegranate. Indian Phytopathology, 24: 173.

Srivastava, R.C. 1982. Fungi causing plant diseases at Jaunpur (U.P.). Indian Journal of Mycology and Plant Pathology, 12: 244-246.

Ssekyewa, C., Opio, A.F., Swinburne, T.R., Damme, P.L.J.Van and Abubakar, Z.M. 1999a. Sustainable management of collar rot disease of passion fruits in Uganda. International Journal of Pest Management, 45: 173-177.

Ssekyewa, C., Swinburne, T.R., Damme, P.L.J.Van and Abubakar, Z.M. 1999b. Passion fruit collar rot disease occurrence in major growing districts of Uganda. Fruits (Paris), 54: 405-411.

Subbarao, K.V. and Michailides, T.J. 1996. Development of phenological scales for figs and their relative susceptibilities to endosepsis and smut. Plant Disease, 80: 1015-1021.

Suharban, M. and Rajan, K.M. 1993. A new blight of pineapple. Journal of Tropical Agriculture, 31: 126.

Sumbali, G. 2000. Fungal diseases of Aonla, Ber and Guava. In: "Diseases of Fruit Crops" (eds. Gupta, V.K. and Sharma, S.K.). Kalyani Publishers, Ludhiana, pp. 28-35.

Sundaram, N.V. 1961. Notes on some fungi from south India. Indian Phytopathology, 14: 202-209.

Suriachandraselvan, M., Jayasekhar, M. and Aubu, S. 1993. Chemical control of bacterial leaf spot and fruit spot of pomegranate. South Indian Horticulture, 41: 228-229.

Swart, D.H. 1990. The post harvest treatment of Madras Litchis. Litchi Year Book – South Africa Litchi Grower's Association, 3: 21-22.

Sydow, H. and Sydow, P. 1907. Fungi Indiae Orientalis Part II. Annals of Mycology, 5: 485-515.

Tandon, I.N. 1961. Diplodia collar rot and root rot of loquat. Horticulture Advancement, 5: 115.

Tandon, I.N. 1965. Sclerotium collar rot of loquat and its control. Indian Phytopathology 18: 240-245.

Tandon, R.N. 1967. Final Technical report PL-480 Scheme (FG-1N-133). University of Allahabad.

Tandon, R.N. and Bilgrami, K.S. 1957. Phyllosticta leaf spot of jackfruit in India. Proceedings of National Academy Science, India, 27: 204-209.

Tandon, R.N. and Bilgrami, K.S. 1961. Some new leaf spot diseases. Current Science, 30: 348.

Tandon, R.N. and Bhargava, S.N. 1962. Botryodiplodia rot of pineapple (*Ananas camosusmerr*). Current Science, 31: 344-345.

Tandon, R.N. and Srivastava, M.P. 1964. Fruit rot of *Emblica officinalis* caused by *Pestalotia cruenta* in India. Current Science, 33: 86-87.

Tandon, R.N. and Verma, A. 1964. Some new storage diseases of fruits and vegetables. Current Science, 33: 625-627.

Tang, Y. 1996. Effect of waxing treatment on black-heart and storage quality of winter pineapples stored at low temperature. Acta Phytophylacica Sinica, 23: 371-372.

Tang, Y.H., Win, T.H. and Liu, S.L. 1998. The occurrence of pomegranate canker disease and its control. China Fruits, 3: 36.

Tay, T.H. 1974. Studies on the nutrition of *Anamas comosus* cv. Singapore Spanish on Malaysian peat. M.Sc. thesis, University of Singapore.

Teotia, R.S. 1995. Black mildew – a new disease of mulberry (*Morus alba*). Indian Phytopathology, 48: 489.

Tewari, D.K., Srivastava, R.C., Katiyar, N., Arora, S. and Lal, B. 1988. Post harvest diseases of

fruits. Proceedings National Academy Sciences, India, 58(B): 345-346.

Thankamma, L. 1983. *Phytophthora* species on eight indigenous host species in south India and their pathogenicity on rubber. Indian Phytopathology, 36: 17-23.

Thind, S.K. and Kaur, N. 1998. Screening of promising ber varieties against powdery mildew and its control. Plant Disease Research 12: 194-196.

Thirumalachar, M.J., Babbarao, D.V. and Ravindernath, V. 1950. Telia of rust on cultivated figs. Current Science, 19: 27-28.

Thompson, A. 1937. Pineapple fruit rot in Malaya. A preliminary report on fruit rots of the Singapore canning pineapple. Malaya Agricultural Journal, 30: 407-420.

Thomson, K.G., Dietzgen, R.G., Thomas, J.E. and Teakle, D.S. 1996. Detection of pineapple bacilliform virus using the polymerase chain reaction. Annals of Applied Biology, 129: 57-69.

Trindade, D.R., Poltroneri, L.S., Albuquerque, F.C., Rezende, J.A.M., Novaes, Q. and Kitajima, E.W. 1999. Occurrence of the passion fruit woodiness virus (PWV) in the state of Para, Brazil. Fitopatologia Brasileira, 24: 196.

Tsai, J.N. and Hseih, W.H. 1998. Occurrence of litchi sour rot and characteristics of the pathogens *Geotrichum candidum* and *G. ludwigii*. Plant Pathology Bulletin, 7: 10-18.

Tsai, J.N. and Hseih, W.H. 1999. A selective medium for the isolation of *Geotrichum candidum* and *Geotrichum ludwigii* from litchi and soil. Plant Pathology Bulletin, 8: 9-14.

Tyagi, R.N.S. 1967. Morphological and taxonomical studies on the genus *Ravenalia* occurring in Rajasthan. Ph.D. thesis, University of Rajasthan, Jaipur, 120 p.

Tyagi, R.N.S. and Pathak, V.K. 1987. Control of aonla (*Emblica officinalis* (Gaerth) rust (*Ravenelia emblicae* Syd.) through fungicides. Indian Journal of Mycology and Plant Pathology, 17: 218-219.

Ullasa, B.A. and Sohi, H.S. 1975. A new Phytophthora leaf blight and damping off disease of passion fruit from India. Current Science, 44: 593-594.

Ullman, D.E., German, T.L., McIntosh, C.E. and Williams, D.D.F. 1991. Effect of heat treatment on a closterovirus like particles associated with mealybug wilt of pineapple. Plant Disease, 75: 859-861.

Uppal, B.N., Patel, M.K. and Kamat, M.N. 1935. The fungi of Bombay B, 1-56. Bulletin Department of Rec. Agriculture, Bombay, 1934 viii+56 pp.

Utikar, P.G., Lande, P.S. and More, B.B. 1976. *Drechslera rostrata* – a new pathogen of pomegranate. Indian Phytopathology, 29: 189.

Vajna, L. 1999. Trunk-blight and canker of mulberry (*Morus alba* var. *pendula*) caused by *Fusarium lateritium* f. sp. *mori*. Novenyvedelem, 35: 619-622.

Vajna, L. 2000. First report of trunk blight and canker of *Morus alba* var. *pendula* caused by *Fusarium lateritium* f. sp. *mori* in Hungary. Plant Disease, 84: 372.

Vashisht, K.S. and Nagaich, B.B. 1965. *Morus indica* – an additional host of fig mosaic. Indian Phytopathology, 18: 135.

Vashisht, K.S. and Nagaich, B.B. 1968. *Aceria ficus* (Colte) as vector of fig mosaic in India. Indian Journal of Entomology, 30: 322.

Vasudeva, R.S. 1960. Report of the Division of Mycology and Plant Pathology, Science Report, Indian Agricultural Research Institute, Delhi (1957-58), pp. 111-130.

Verma, K.S. and Cheema, S.S. 1983. Tandonella leaf spot – a new disease of ber in Punjab. Current science, 52: 437.

Verma, K.S. and Kapur, S.P. 1995. Some new disease records of fruits from Punjab. Plant Disease Research, 10: 64-65.

Verma, K.S., Kumar S. and Kumar, S. 1995. Survival and dispersal of *Isariopsis indica* var. *zizyphi* causing mouldy leaf spot of ber. Indian Phytopathology, 48: 45-48.

Verma, R.R., Lal, B. and Tripathi, K.C. 1982. Evaluation of some pomegranate varieties to the

Anar borer and fruit rot in Garhwal hills. Indian Journal of Forestry, 6: 237-238.

Verma, S., Gupta, S., Singh, R.V., Abidi, A.B., Verma, S. and Gupta, S. 1991. Changes in biochemical constituents of bael fruits infected with *Aspergillus* species. Indian Phytopathology, 44: 405-406.

Veronica, C.Z. and Ximena, B.C. 1998. Epidemiological aspects of *Botrytis cinerea* in custard apple-tree (*Annona cherimola* Mill.). Boletin Micologico, 13: 85-91.

Vidyasagar, G.M. and Rajasab, A.H. 1997. Predisposition of powdery mildew infected mulberry leaves to *Phoma mororum* infection (Phoma leaf spot). Indian Journal of Sericulture, 36: 65-66.

Vinayak, A. and Mandhar, C.L. 1994. *Cephaleuros* infection on leaves of litchi plant. Research Bulletin of the Panjab University, Science, 44: 165-172.

Wadia, K.D.R. and Manoharachary, C. 1979. The new post-harvest diseases of *Carissa carandas* L. fruits. Geobios, 6: 126-127.

Wadia, K.D.R. and Manoharachary, C. 1982. New post-harvest diseases of jambolan fruits. Indian Phytopathology, 35: 707-708.

Wakman, W., Teakle, D.S., Thomas, J.E. and Dietzgen, R.G. 1995. Presence of a clostero-like virus and a bacilliform virus in pineapple plants in Australia. Australian Journal of Agricultural Research, 46: 947-958.

Wang, X.L., Liu, M.J., Liu, L.J., Zheg, L.K., Zhou, J.Y. and Liu, P. 1999. Fluorescence microscopic diagnosis of Chinese jujube tree infected with phytoplasma. Journal of Hebei Agricultural University, 22: 46-49.

Wani, D.D. and Thirumalachar, M.J. 1973. Control of anthracnose disease of figs by fungicides and antibiotic aureofungin. Hindustan Antibiotiotics Bulletin, 15: 79-80.

Waraiteh, K.S. 1977. Additions to Pyrenomycetes fungi of India. Proceedings National Academy of Science, India, 46th Annual Session, p. 23.

Wilson, K.L., Balakrishnan, S. and Nair, N.G. 1970. Leaf spot of sapota in Kerala. Science and Culture, 36: 109-110.

Wong, L.S., Jacobi, K.K. and Giles, J.E. 1991. The influence of hot benomyl dips on the appearance of cool stored Lychee (*Litchi chinensis* Sonn.). Scintie Horticulturae, 46: 245-251.

Xu, P.D., Li, M. and Ke, C. 1999. Occurrence of virus diseases of *Passiflora* spp. and subgrouping of cucumber mosaic Cucumovirus in Fujian. Acta Phytophylacica Sinica, 26: 50-54.

Xu, Y., Zheng, X.L., Zhao, G.Y. and Mao, Z.C. 1995. Pathogenetic studies on fruit shrink of jujube. Acta Agriculturae Boreali-Sinica, 10(Suppl.): 139-143.

Yadav, A.S. 1963. Addition to the microfungi II. Indian Phytopathology, 16: 167-170.

Yadav, G.R., Nirwan, R.S. and Prasad, B. 1980. Powdery mildew of ber in Uttar Pradesh and its control. Progressive Horticulture, 12: 27-32.

Yadav, G.R., Prasad, B. and Upadhyay, J. 1984. Effect of post harvest treatment on storage rot of litchi fruits. Progressive Horticulture, 16: 351-352.

Yoon, H.J., Kim, Y.T., Jin, K.S. and Park, I.G. 1994. Isolation and identification of pathogenic bacteria from branch rot of mulberry. RDA Journal of Agricultural Science Crop Protection, 36(2): 331-336.

Yoshida, S. and Shirata, A. 1999a. Survival of *Colletotrichum dematium* in soil and infected mulberry leaves. Plant Disease, 83: 465-468.

Yoshida, S. and Shirata, A. 1999b. The mulberry anthracnose fungus, *Colletotrichum acutatum*, overwinters on a mulberry tree. Annals of the Phytopathological Society of Japan, 65: 274-280.

Yoshida, S., Hiradate, S., Fujii, Y. and Shirata, A. 2000. *Colletotrichum dematium* produces phytotoxins in anthracnose lesions of mulberry leaves. Phytopathology, 90: 285-291.

Yu, S.Q., Fang, L.X.., Tang, W.Q. and Liu, J. F. 1995. Technology for controlling major pests on

litchi. Guangdong Agricultural Science, 3: 38-40.

Zhang, C.F. and Qi, P.K. 1994. Studies on pathogenic fungi on *Achras sapotae* L. in Guangdong Province. Journal of South China Agricultural University, 15: 31-36.

Zhao, Z.J., Lin, Z.M. and Zhao, X.J. 2000. The jujube main disease in Shanxi province and its control. China Fruits, 1: 45.

Zheng, J.X. and Huang, Y. 1997. Base rot disease of passion fruit and its control in Fujian. Journal of Fujian Academy of agricultural Sciences, 12: 40-43.

Zheng, X.L., Qi, Q.S., Li, X.J. and Jiang, Z.J. 1996. Identification of pathogens of jujube fruit shrink disease and induction of their fruit body. Plant Protection, 22: 6-8.

Zheng, X.L., Zhao, G.Y., Qi, Q.S., Hou, Q.S. and Li, P. 1998. Occurrence and symptoms of jujube fruit shrink. Plant Protection, 24: 17-19.

Zhu, B.M., Chen, Z.Y., Zheng, D.X., Wang, S., Yu, B.W., Zhou, P.Z. and Jiang, X.Y. 1983. Preliminary studies on the pathogen of Chinese jujube mosaic diseases. Nature, 5: 77-78.

Zhu, W.Y., Du, X.M., Guo, H.P., Zhao, Y. and He, X.H. 1996. Virus elimination and tissue culture of Jun Jujuba. Acta Horticulturae Sinica, 23(20: 197-198.

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