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CAPSICUM & EGGPLANT DISEASE

FIELD GUIDE

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/// Vegetables by Bayer

Vegetables by Bayer is committed to helping our customers grow their businesses so that together we can foster a healthier, more sustainable world. We work with growers and other partners to develop innovative products that balance agronomic traits with the demands of the market. We also go beyond the seed to provide solutions for our customers — like this disease field guide which can be used as a reference for common capsicum and eggplant diseases and disorders, as well as their control.

We developed the capsicum and eggplant disease field guide for use by a wide range of professionals involved in the industry, including growers, agricultural advisors, farm managers, agronomists, food processors and members of the chemical and vegetable seed industries. It does not include every capsicum and eggplant disease, but we have included the diseases that are currently most prevalent worldwide.

The guide offers descriptions and photographs of the more common global capsicum and eggplant diseases and disorders, including the common name, causal agent, distribution, symptoms, conditions for disease development and control measures.

Even the most experienced plant pathologist relies on laboratory and greenhouse techniques to confirm a plant disease and/or disorder diagnosis. Therefore, diagnosis of capsicum and eggplant diseases and disorders using only this guide is not recommended or encouraged, and it is not intended to be substituted for the professional opinion of a producer, grower, agronomist, plant pathologist or other professionals involved in the production of these crops. Always read and follow label directions for any herbicide, fungicide, insecticide or other chemical used for treatment or control.

We are grateful to our many academic and private industry partners who contributed photographs for this guide. The photographs illustrate characteristic symptoms of capsicum and eggplant diseases and disorders; however, it's important to note that many factors can influence the appearance and severity of symptoms. A glossary can be found at the end of this guide, along with a list of references for additional information on the diseases and disorders described in this publication.

*Not all diseases affect both capsicums and eggplants.

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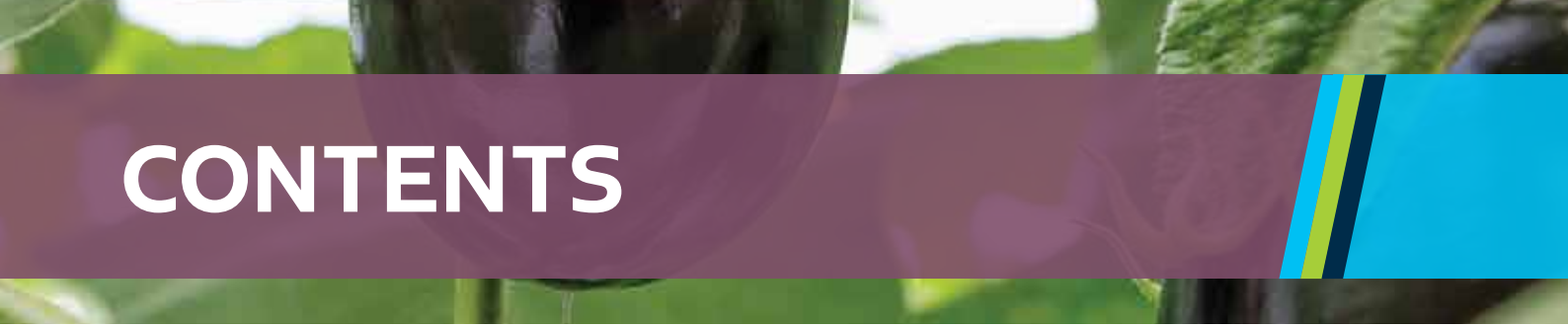

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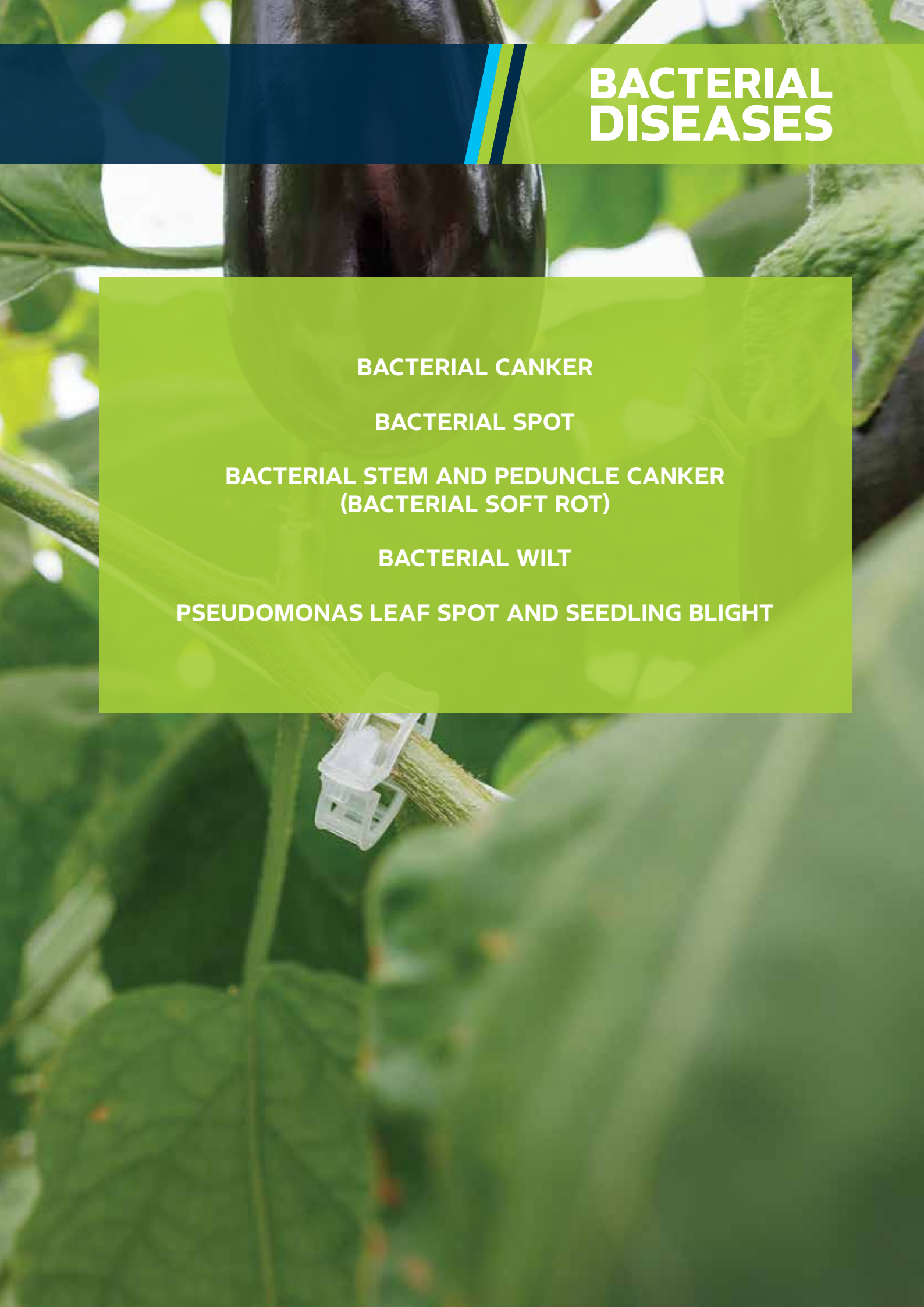


BACTERIAL DISEASES

BACTERIAL CANKER

BACTERIAL SPOT

BACTERIAL STEM AND PEDUNCLE CANKER (BACTERIAL SOFT ROT)

BACTERIAL WILT

PSEUDOMONAS LEAF SPOT AND SEEDLING BLIGHT

Causal Agent

Clavibacter michiganensis subsp. *michiganensis*

Distribution

Australia, Brazil, China, Israel, South Korea and USA (California, Indiana, Michigan and Ohio)

Symptoms

Symptoms of bacterial canker in capsicum include leaf and fruit spots and, less frequently, systemic wilt. In localised infections, symptoms first appear as small blisters or raised white spots on leaves and stems. Later, the centres of the leaf spots become brown and necrotic, and develop a white halo. Stem lesions often develop a crusty appearance and elongate to form cankers. Symptoms on fruit first appear as very small, round, slightly raised spots. These spots gradually increase in size and may develop a brown centre and a white halo. When these are numerous, spots merge and take on a crusty appearance. In systemic infections, a gradual wilting occurs followed by plant death as a result of the plant being girdled by the canker.

Conditions for Disease Development

The bacterium enters the plant via wounds (most commonly due to pruning in an agricultural setting) and stomata. *Clavibacter* may be seed-borne and may infest the seed externally or under the seed coat. High relative humidity and daytime temperatures between 25° and 30° C generally favour the disease. Dense plant populations and overhead irrigation also provide an ideal environment to spread the bacterium. Insects, tools and human contact may also aid the spread.

Control

Sow only tested seed and certified transplants. Do not transplant capsicums into ground used for tomatoes during the previous season. Clean cultivation equipment before entering a new field, avoid entering fields when foliage is wet, and incorporate plant debris immediately after harvest to help reduce losses. Never harvest fruit from symptomatic plants. Rogue all symptomatic and adjacent plants. Rotate to a non-host for a minimum of three years if the disease is found in a field or greenhouse.

Lesions may merge and appear crusty.

Foliar symptoms in field.

Raised lesions on leaf undersurface.

Bacterial canker lesions on green fruit.

Bacterial canker lesions on red fruit.

Causal Agent

Xanthomonas spp.

X. euvesicatoria (synonym = *X. campestris pv. vesicatoria)*

X. vesicatoria

X. gardneri

X. perforans

Distribution

Worldwide in warm, humid areas

Symptoms

Symptoms develop on leaves, stems and fruit of sweet capsicum and are less severe in chilli. Symptoms first appear on leaf undersurfaces as small, irregular, water-soaked areas. Later, lesions enlarge, turn dark-brown to black with a pale tan centre and may develop a thin, yellow halo. Generally, lesions on upper leaf surfaces are slightly sunken, and those on lower leaf surfaces are slightly raised. Leaves that are severely infected often turn chlorotic and appear ragged. Defoliation occurs under heavy disease pressure. Stem lesions appear as narrow, light-brown, longitudinally raised cankers. Fruit spots begin as water-soaked areas that later turn necrotic. These spots are rough in appearance and crack as they develop.

Conditions for Disease Development

Xanthomonas can be seed-borne on the seed surface and within the seed. Infected seed and transplants moved over long distances can be the initial source of inoculum for epidemics. The bacterium also survives in crop debris, volunteer plants and in solanaceous weeds. High relative humidity and heavy dew formation on leaves, together with warm weather, favour infection and development of Bacterial Spot. The bacterium is readily water-splashed from infected transplants or debris to healthy plants. Fruit are infected through growth cracks, abrasions, insect punctures and other wounds. Secondary fruit rots often develop around Bacterial Spot lesions during damp weather.

Control

Use only tested and treated seed and certified transplants. Once present, the disease is difficult to control. Commercial varieties with one or more genes for resistance to X. *campestris* pv. *vesicatoria* are available. Copper-based sprays can help reduce the rate of disease development. Clean cultivation equipment before entering a new field, avoid entering fields when foliage is wet, and incorporate plant debris immediately after harvest to help reduce losses due to Bacterial Spot. Rotation to non-host crops and controlling weeds and volunteer plants are good preventive practices. When the disease is present, avoid overhead irrigation.

Water-soaked lesions on leaf undersurface.

Longitudinal stem cankers.

Foliar symptoms in the field.

Infected seedlings.

Dark-brown lesions with tan centres.

Large, rough lesions on green fruit.

Small lesions on peduncle.

Causal Agent

Pectobacterium carotovora, P. atrosepticum, Dickeya chrysanthemi

Distribution

Worldwide

Symptoms

This disease affects capsicum stems and fruit. Internal discolouration appears on the stem, followed by hollowing-out of the pith and wilting. As lesions expand along the stem, branches break. Foliar chlorosis and necrosis may also develop. Symptoms of post-harvest decay start as sunken, water-soaked areas around the edge of wounds or on the stem end next to the peduncle. These areas may be light or dark and become soft as they rapidly expand. Often, the epidermis splits open, releasing watery, macerated tissue.

Conditions for Disease Development

Soft rot bacteria are common inhabitants of soils. Under warm, humid conditions, infection through wounds or cut stems occurs. Splashing rain and irrigation water spread bacteria to foliage and fruit.

Control

In greenhouse operations, provide adequate air circulation to help reduce relative humidity. Avoid injuries to plants during the growing season and on fruit during harvest. Improved sanitation in the field and in packing houses is effective in reducing losses. All harvest equipment, the packing line and packing boxes should be sanitized frequently. Dump tank water and packing line washers should maintain a minimum available chlorine concentration of 150 ppm at a pH of 6.0 to 7.5. Wet fruit should be dried promptly before packing and then cooled quickly to below 10° C.

10 / BACTERIAL STEM AND PEDUNCLE CANKER (SOFT ROT)

Soft sunken lesions.

Fully macerated fruit.

Soft rot infected fruit.

Causal Agent

Ralstonia solanacearum (synonym = *Burkholderia solanacearum, Pseudomonas solanacearum)*

Distribution

Worldwide in the tropics, semi-tropics and some temperate regions

Symptoms

In tropical and subtropical regions, affected plants may wilt and die within days of infection. Leaves may appear healthy or only slightly yellow prior to plant death. Under temperate conditions, infected plants develop a slower, progressive wilt in which leaves turn yellow. The lower stems of affected plants develop dark, vascular browning that often extends into the cortical and pith tissues. When stems of symptomatic plants are cut and placed in water, milky white streams of bacteria flow from cut ends.

Conditions for Disease Development

Ralstonia Solanacearum is soilborne and can survive for long periods in the soil on roots and debris. The bacterium infects roots through wounds caused by nematode feeding, transplanting and cultivation. High temperatures and high soil moisture generally favour the disease. Bacteria is spread in irrigation water, diseased transplants and in soil moved with cultivation equipment.

Control

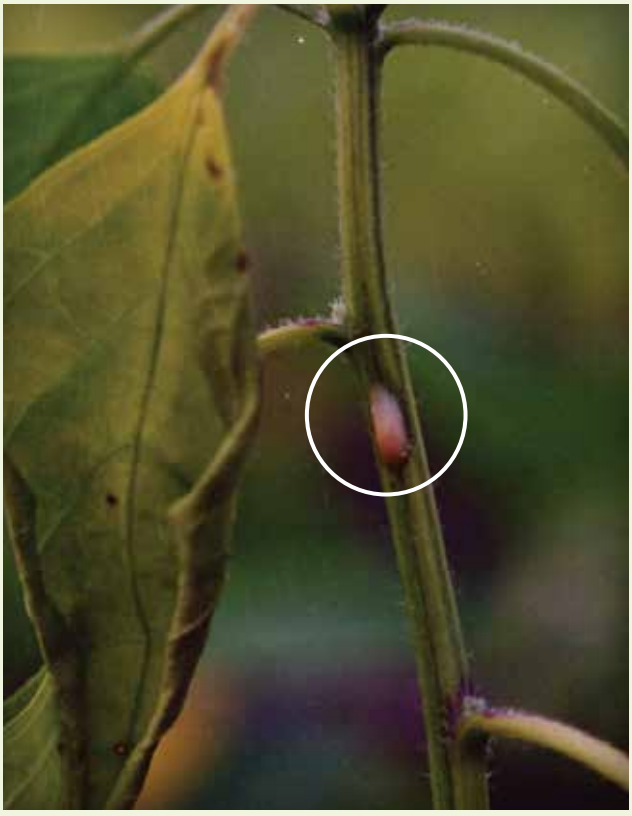
When possible, avoid land with a history of Bacterial Wilt. Commercial capsicum and eggplant varieties with Intermediate Resistance to Bacterial Wilt are available. Resistant rootstocks are also available. A soil pH between 5.5 and 7.0, good soil drainage and raised beds help alleviate disease pressure. Rotate to non-host crops in order to lower the population of bacteria in the soil.

Mild (left) and severe (right) vascular discolouration of capsicum.

Bacterial wilt of chilli.

Susceptible eggplant between rows of intermediate resistant plants.

Wilted eggplant.



Bacteria oozing from capsicum stem.

Causal Agent

Pseudomonas syringae pv. *syringae*

Distribution

Southern and Southeastern Europe, Southern USA

Symptoms

Affected leaves or cotyledons develop irregular, water-soaked lesions that later become necrotic, turning dark-brown with a light centre. Lesions may coalesce to form relatively large necrotic areas. Lesions with chlorotic halos are rare. However, under heavy disease pressure, large areas of the leaf may be affected and the whole leaf may prematurely turn yellow and drop. Infected fruit develop brownish-black, watery lesions that expand and rot. Symptoms of Syringae Seedling Blight can be confused with those caused by Bacterial Spot. However, the lower temperatures at which Syringae Seedling Blight occurs can help differentiate these two diseases.

Conditions for Disease Development

Temperatures between 16° and 24° C and high humidity favour Syringae Seedling Blight. Bacteria is generally spread by splashing water that enters the plant through natural openings or wounds.

Control

Avoid low temperature and high humidity conditions in nurseries. Inspect seedlings for symptoms before transplanting to avoid introducing the disease to the field. Avoid overhead irrigation whenever possible.

Discrete and coalescing lesions.

Coalescing leaf spots forming large lesions in eggplant.
(Courtesy A. Obradovic—© APS. Reproduced, by permission, from Pernezny, K., Roberts, P. D., Murphy, J. F., and Goldberg, N. P., eds. 2003. Compendium of Capsicum Diseases. American Phytopathological Society, St. Paul, MN.)

FUNGAL DISEASES

ANTHRACNOSE

CERCOSPORA LEAF SPOT (FROGEYE)

CHOANEPHORA BLIGHT (WET ROT)

DAMPING-OFF

FUNGAL FRUIT ROTS

FUSARIUM WILT

GREY LEAF SPOT

GREY MOULD

LEAF SPOTS

PHOMOPSIS BLIGHT

POWDERY MILDEW

SOUTHERN BLIGHT (SCLEROTIUM WILT)

VERTICILLIUM WILT

WHITE MOULD (PINK ROT, WATERY SOFT ROT)

Causal Agent

Colletotrichum truncatum (ex Colletotrichum capsici), C. gloeosporioides, C. coccodes, C. acutatum.

Distribution

Worldwide in tropical regions

Symptoms

Anthracnose affects all above-ground parts of capsicums during any stage of growth. Seedling infection may be confined to cotyledons and not spread. Necrotic grey to brown spots may develop on leaves and stems. Fruit lesions are the most economically important aspect of this disease. Fruit symptoms begin as water-soaked areas that turn tan or brown. Lesions may be small and circular, or coalesce to cover large areas of the fruit. Under moist conditions, pink, salmon or orange masses of spores are formed, usually in concentric rings. Depending on the *Colletotrichum* species present, black or brown filamentous structures may be visible in the lesion. Anthracnose can affect both green and ripe fruit, but symptoms are usually not visible until fruit ripen and turn red.

Conditions for Disease Development

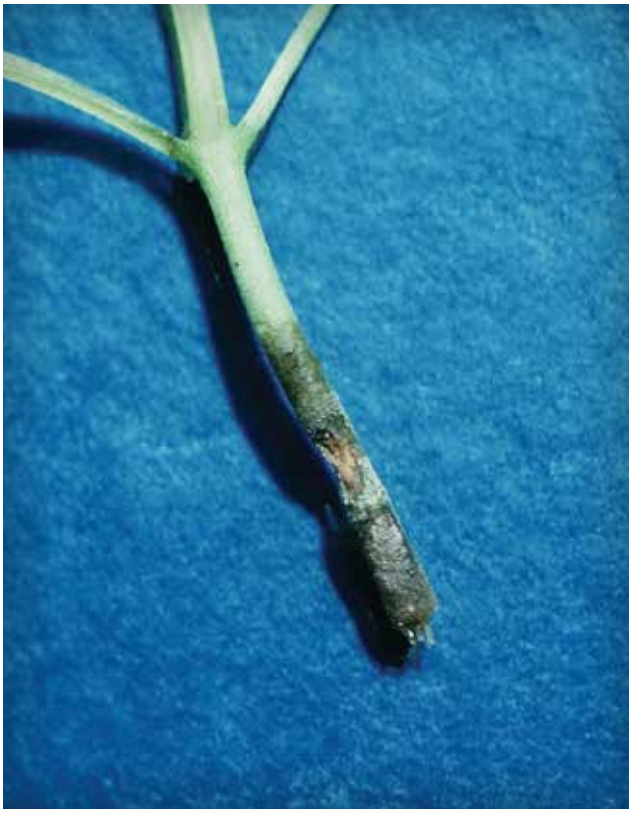
Warm, wet weather generally favours infection and development of symptoms. Depending on the species of *Colletotrichum* present, optimal temperatures for infection range from 20° to 27° C. Free moisture is necessary for infection. Fog and dew are conducive to disease development. Rain disseminates the pathogen's spores and often leads to severe losses, especially if fruit are wounded. These fungi can survive in infected seed and persist in leaf or stem lesions in plant debris for long periods of time. Anthracnose can also infect fruits on plants or as post-harvest disease. This is described in the Fungal Fruit Rots section.

Control

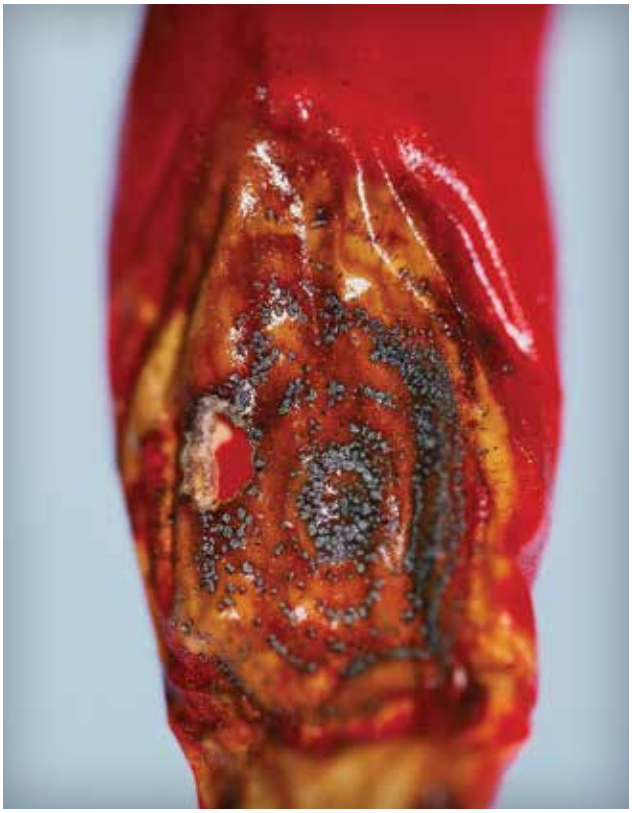
This disease can be managed by sowing good quality seed, rotating out of solanaceous crops for two to three years, removing weeds and infected debris, and choosing fields that drain well. Minimise fruit wounds by controlling insects. Copper fungicides are available, but have limited economical value for controlling this disease.

Natural field infection.

Grey necrotic spots on cotyledons.



Stem lesion.



Concentric rings of spores.

Advanced anthracnose lesion on fruit.

Coalescing lesions with salmon-coloured spores.

Causal Agent

Cercospora capsici, C. melongenae

Distribution

Worldwide in tropical regions

Symptoms

This disease affects the leaves, petioles, stems and peduncles of capsicum and eggplant. Symptoms first appear as small, circular to oblong chlorotic lesions. Lesions later turn necrotic with a sporulating light-grey centre and a dark-brown margin. Concentric rings may be observed as individual lesions expand. These lesions often resemble frog eyes, giving this disease its common name. As the lesions dry, the centres crack and drop out. When the disease is severe, defoliation and reduction in fruit size occur.

Conditions for Disease Development

These fungi can survive for at least one year in infected plant debris. Wet, warm weather conditions favour disease development. Spores are spread by wind, rain and irrigation water or mechanically by equipment and people.

Control

A calendar-based protectant fungicide spray program combined with cultural practices can help reduce losses from Cercospora Leaf Spot. Turn under or remove all plant debris and rotate to non-host crops to lower field inoculum levels. Mulch and furrow or drip irrigate to help reduce spread of the pathogen from splashing water.

20 / CERCOSPORA LEAF SPOT (FROGEYE)

Centres of lesions dry and crack on capsicum.

Lesions with concentric rings on capsicum.

Necrotic lesions on eggplant.

Sporulating centre with chlorotic halo on capsicum.

Casual Agent

Choanephora cucurbitarum

Distribution

Worldwide in tropical regions

Symptoms

Symptoms are visible on apical growing points, flowers and fruits. Initially, water-soaked areas develop on leaves, and apical growing points become blighted. Later, the fungus grows rapidly downward, causing dieback. Dark-grey fungal growth can be seen on some lesions. Close inspection will reveal silvery, spine-like fungal structures and dark spores. In seedlings, symptoms may be confused with Phytophthora Blight. A black soft rot can also develop in fruit.

Conditions for Disease Development

The fungus is found throughout the tropics on many crops including beans, peas, squash, cucumber, eggplant and capsicum. Extended periods of rain, high humidity and high temperatures generally favour fungal sporulation and disease development. The fungus is generally spread via wind and splashing water, and on clothing, tools and cultivation equipment.

Control

Fungicide sprays may help reduce disease damage.

22 / CHOANEPHORA BLIGHT (WET ROT)

Blight and dieback of capsicum foliage and fruit.

Necrotic dieback and fruit rot of capsicum.

Wet rot of capsicum seedlings.

Sporulation along capsicum stem.

Sporulation on eggplant fruit.

Casual Agent

Rhizoctonia solani

Distribution

Worldwide

Symptoms

Pre-emergence Damping-Off: Seeds may rot before germinating or seedlings may die prior to emergence.

Post-emergence Damping-Off: Young seedlings develop a rot at the crown. Later, the tissue becomes soft and constricted, and the plants wilt and fall over.

Rhizoctonia solani: Pre-emergence symptoms include reddish-brown lesions on hypocotyls and death of growing tips. Post-emergence Damping-Off is characterised by reddish-brown to black lesions that develop on roots and hypocotyls at or below the soil line. Later, hypocotyls collapse and seedlings wilt and die.

Conditions for Disease Development

These fungi have a wide host range and can survive for long periods in soil, plant debris and weeds. Damping-Off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather. Water-splashing moves infested soil from diseased to healthy plants.

Control

Improving drainage and moisture regulation to help prevent soil saturation reduces Damping-Off. Fungicidal soil drenches and seed treatments are available that help manage Damping-Off. Rotation to cereal crops and soil fumigation or solarisation may help reduce Damping-Off in the field. Use pasteurised soil mixes in nurseries.

Capsicum (left) and eggplant (right) showing rot.

Girdled hypocotyl leads to seedling death.

Post-emergence Damping-Off in the field.

ALTERNARIA ROT

Casual Agent

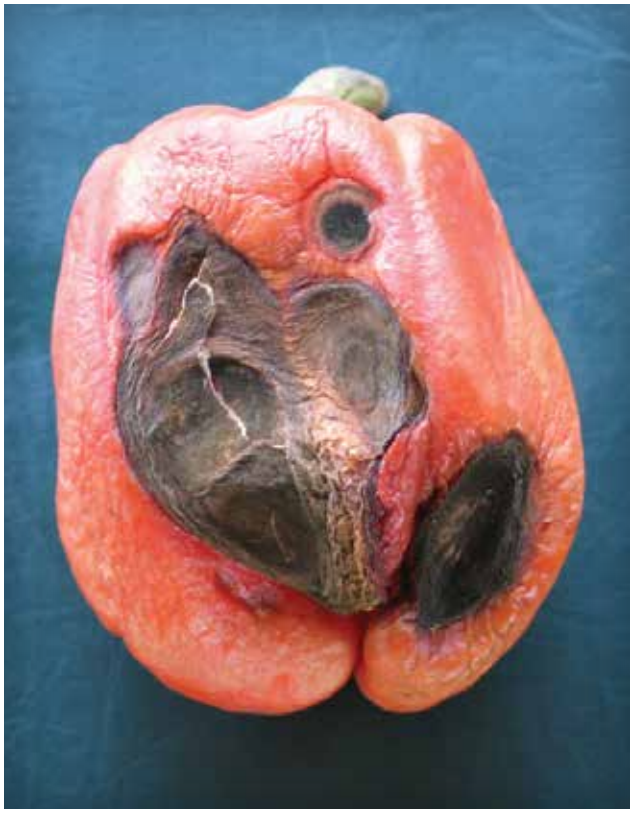
Alternaria alternata

Distribution

Worldwide

Symptoms

Symptoms first appear as water-soaked, grey lesions that collapse and darken. Lesions turn velvety as spores are produced. Infection generally occurs at growth cracks, injuries or at the blossom-end of fruit. Internal colonisation of capsicum fruit without external signs of infection can occur when flowers are infected.



Alternaria fruit rot.

Velvety sporulation of *A. alternata*.

ANTHRACNOSE FRUIT ROT (RIPE FRUIT ROT)

Casual Agent

Colletotrichum truncatum (ex Colletotrichum capsici), C. gloeosporioides, C. coccodes, C. acutatum.

Distribution

Worldwide

Symptoms

These fungi may infect the epidermis of immature fruit and remain latent until harvest. Symptoms usually develop on ripe fruit, giving this disease its common name: “Ripe Fruit Rot.” Fruit lesions first appear as small, indefinite, slightly sunken, water-soaked spots that may enlarge rapidly and coalesce. Later, fruiting bodies form in concentric circles to cover the surface of lesions. The lesions appear tan or brown and are covered with salmon to orange gelatinous spores. If the fruit rot extends to the seed cavity, it may infest and infect the seed.

BOTRYTIS FRUIT ROT (GREY MOULD ROT)

Casual Agent

Botrytis cinerea

Distribution

Worldwide

Symptoms

Initial infection occurs when fruit are in direct contact with the soil. The fungus also colonises dying flowers and fruit through the stem end, growth cracks and wounds. *Botrytis* also infects cold-injured fruit. Soft rot may develop and consume the fruit entirely. Affected areas are grey to olive green, slightly sunken and have distinct margins. The epidermis peels away easily from lesions to reveal softened, watery underlying tissue. Under humid conditions, grey-brown mycelia develop on the surface, and grape-like clusters of spores can be seen with a hand lens. *Botrytis* can also infect plant tissue; this is further described in the Grey Mould section.

Anthracnose fruit rot.

Botrytis fruit rot.

INTERNAL FRUIT ROT

Causal Agent

Fusarium lactis, F. oxysporum, F. proliferatum

Distribution

Worldwide

Symptoms

Symptoms of internal fruit rot can be hard to observe and vary during the season. Early in the season the most visible symptom is peduncle rot, where the peduncle become necrotic, blocking the fruit from further development. Later in the season the fungus can be found inside the fruits as visible fungal tissue or rotten black spots; outside, black soft spots can be observed. Most problematic is that the symptoms only become visible a few days after harvest.

Conditions for Disease Development

Infection occurs during the flowering stage where spores grow via the stigma into the fruit. After infection, the fungus remains latent until maturation. Of the three fungi that are associated with internal fruit rot, F. lactis is the most common one. The fungus can survive on crop debris, including aborted flowers or fruits. Dry conditions favour spread of the disease.

Control

The fungus remains viable on crop debris and aborted flowers, releasing spores that are spread via air. Spores can remain on horizontal surfaces and spread again by air movement. Removal of crop debris and spray of horizontal surfaces with clean water will reduce the amount of spores.

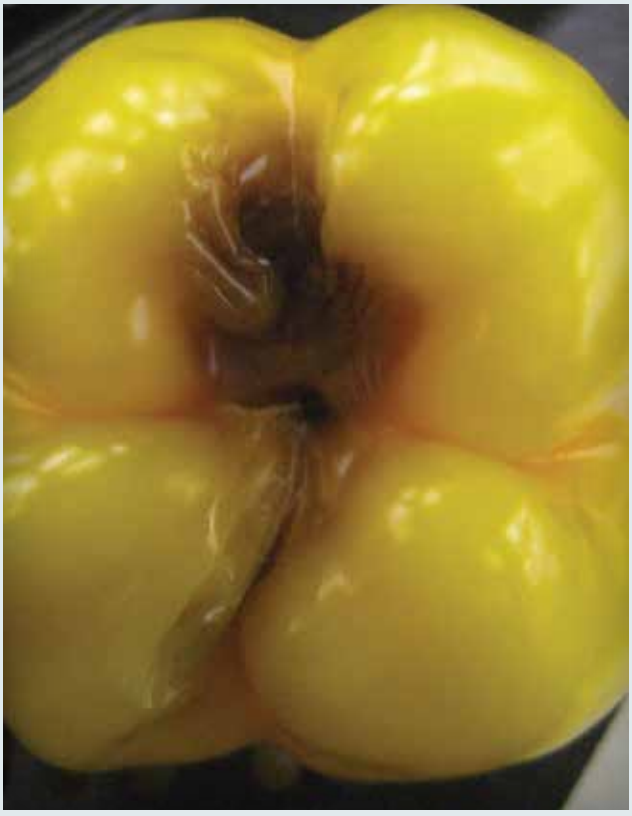
Peduncle rot.

Peduncle rot.

Internal rot.

Internal rot.

Black spots.



Black spots.

PHYTOPHTHORA FRUIT ROT

Causal Agent

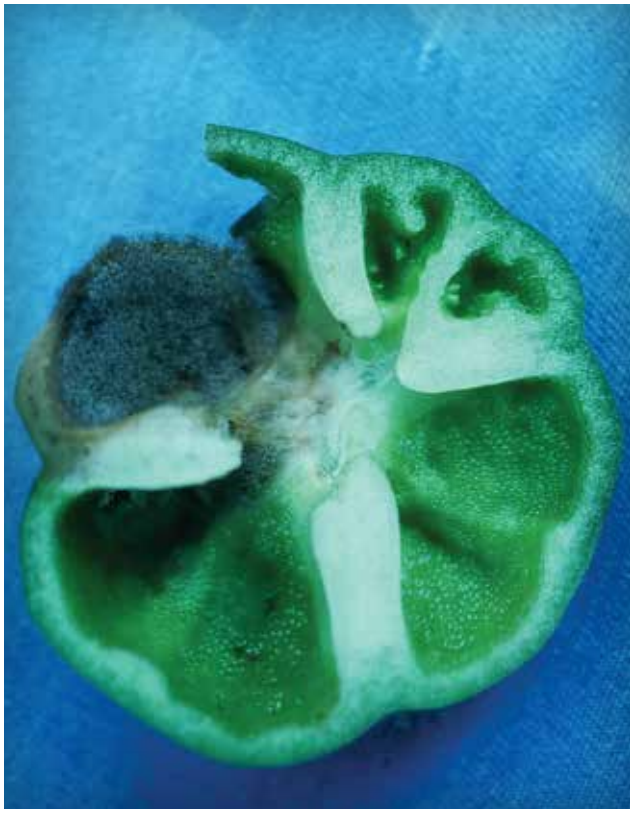
Phytophthora capsici, P. nicotianae var. *parasitica*

Distribution

Worldwide

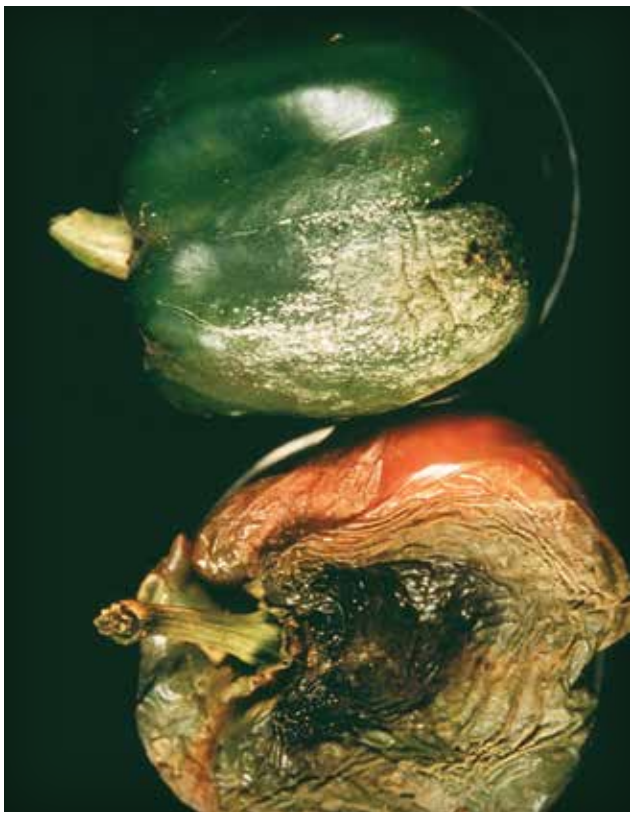
Symptoms

Phytophthora rot occurs when fruit are in contact with the soil or mycelia grows through the peduncle into the fruit. Infected fruit tissue is water-soaked and dark-green at first; later, white mycelium and sporangia develop on the surface of the affected area and, within several days, consume the entire fruit. In contrast to infected tomato fruit, no concentric rings develop. Fruit affected by these fungi dry rapidly and shrivel, but do not drop.



Botrytis cinerea sporulating internally.

Phytophthora fruit rot.



Shriveled fruit infected by a *Phytophthora* species.

RHIZOPUS ROT

Causal Agent

Rhizopus stolonifer

Distribution

Worldwide

Symptoms

Contamination and wounding of fruit during the packing process is the primary means of infection. Symptoms first appear as soft, water-soaked lesions that are not discoloured. Lesions develop from wounds, the stem end or inner walls, and quickly enlarge to engulf the entire fruit. When the epidermis ruptures, liquefied tissue is released. Under high humidity, profuse, coarse mycelia cover lesions. Later, white sporangia develop that turn black as they mature, giving a capsicumy, speckled appearance to the mycelia. In storage, these fungi penetrate directly from nests of infected fruit into adjacent healthy fruit..

Conditions for Disease Development

Rain splashes overwintering spores from soil and crop debris onto developing fruit. Symptom development is generally favoured by high humidity. Botrytis fruit rot occurs during periods of cool, wet weather. The remaining four fruit rots occur during warm, wet weather.

Control

Fruit injury during harvest and packing should be avoided. Improved sanitation in the field and in the packing house is effective at helping to reduce losses due to fruit rots. All harvest equipment, the packing line and packing boxes should be sanitised daily. Dump tank water and packing-line washers should maintain a minimum available chlorine concentration of 150 ppm at pH 6.0-7.5. Culling infected and injured fruit during packing help reduce losses due to post-harvest decays. Wet surfaces should be dried promptly before packing, and fruit should be cooled quickly to 10° C.

Rhizopus fruit rot.

Causal Agent

Capsicum: *Fusarium oxysporum f. sp. capsici*
Eggplant: *Fusarium oxysporum f. sp. melongenae*

Distribution

Capsicum: Argentina, Asia, Italy, Mexico and USA
Eggplant: Worldwide

Symptoms

Symptoms first appear as a slight yellowing of foliage and wilting of upper leaves. As wilting progresses, leaves may turn dull-green to brown and remain attached to the plant. When the stem and roots are cut diagonally, reddish-brown streaks are visible in the vascular tissues.

Conditions for Disease Development

The fungus survives in the soil for several years and is spread by farm equipment, irrigation water and infected plant debris. Warm soil temperatures (33° C) and high soil moisture generally favour rapid disease development.

Control

Plant on raised beds to help promote soil water drainage away from roots. Thoroughly disinfect equipment before moving from infested to clean fields. Soil fumigation, solarisation and crop rotation to non-hosts help reduce disease incidence.

Initial symptoms on capsicum include yellowing of foliage.

32 / FUSARIUM WILT

Affected capsicum plants wilt and die.

Leaves remain attached to wilted capsicum plant.

Fusarium Wilt symptoms on eggplant.

Vascular tissues with reddish-brown streaks.

Causal Agent

*Stemphylium solani, S. lycopersici (*synonym: *S. floridanum)*

Distribution

Worldwide

Symptoms

Small spots develop on capsicum leaves, petioles, stems, peduncles and calyxes. Although mature plants can be infected, young seedlings are most susceptible. Infection begins as small red to brown spots that later expand into lesions with white to grey centres and red to brown margins. When numerous lesions develop, leaves turn yellow and drop. Grey leaf spot does not affect fruit.

Conditions for Disease Development

These fungi survive in soil and on plant debris from one year to the next. In addition, volunteer capsicum and tomato and solanaceous weeds can serve as sources of inoculum. Fungal spores are spread from the surface of infected tissues by wind and splashing water. Warm and humid or wet weather generally favour disease development. The disease also can be a problem in arid climates when dew periods are long.

Control

Remove plant debris, provide adequate ventilation for seedling beds and treat with fungicides to help reduce losses from this disease.

Lesions with light centres and dark margins.

Developing lesions.

Numerous lesions with developing leaf chlorosis.

Causal Agent

Botrytis cinerea

Distribution

Worldwide

Symptoms

This fungus typically causes damping-off or tip dieback in young seedlings. However, it can infect through wounds in all above-ground parts of mature plants. On stems, initial infection appears as elliptical, water-soaked lesions that later expand, and can girdle and kill the plant. Leaf infections usually begin at points of injury and develop into V-shaped lesions. Under high humidity, stem and leaf lesions can be covered by sporulating grey mycelia. Fruit infection begins as water-soaked spots that increase rapidly in size to form grey-brown sporulating lesions.

Conditions for Disease Development

This fungus has a wide host range. It is an efficient saprophyte and can survive as sclerotia in soil and infected plant debris for long periods. Botrytis cinerea is considered a weak parasite and typically infects plant tissues through wounds. Overcast, cool, humid weather is required for disease development. Under these conditions, grey masses of fungal spores are produced and are readily wind-disseminated. Close spacing and poor ventilation in greenhouses can lead to severe grey mould problems.

Control

Prune plants to promote adequate ventilation, and apply fungicides to the pruning wounds to help reduce losses from this disease. Carefully manage irrigation and air circulation to avoid long periods of high relative humidity in greenhouses.

Typical V-shaped lesion.

Lateral tip dieback.

Girdling of stem and sporulation.

Causal Agent

Alternaria spp., Septoria melongenae, Cercospora spp.

Distribution

Worldwide

Symptoms

First visible symptoms of foliar infection are expanding necrotic spots with yellow to dark-brown margins. Infection usually starts on lower leaves and moves up the plant as the disease progresses. Leaf spots caused by *Stemphylium melongenae* later break apart, giving the appearance of "shot holes." Leaf spots caused by *Alternaria* spp. are irregular in shape with concentric rings that enlarge, and can cover, the leaf blade. Early infections by *Alternaria* spp. may cause a seedling dieback known as "collar rot." *Cercospora* spp. can also cause small, irregular leaf spots that later are covered with grey sporulation. Fruit lesions start as necrotic spots and develop into sunken, scab-like lesions that extend into the flesh of the fruit, turning it hard and brown. Defoliation caused by extensive infection exposes fruit to direct sunlight, resulting in sunscald.

Conditions for Disease Development

Disease development is generally dependent on high relative humidity. These fungi overwinter in or on infested debris and organic matter in the soil. Splashing irrigation water to the foliage facilitates spread of the disease. Wounding predisposes fruit to infection when these fungi are present.

Control

Use field sanitation techniques such as crop rotation, weed control and removal of debris from previous crops to help reduce disease severity. Use mulching and furrow irrigation to reduce splashing and excess leaf wetness. Use good quality seeds.

Leaf spot of eggplant.

Causal Agent

Phomopsis vexans

Distribution

Worldwide, in tropical and subtropical areas

Symptoms

This fungus attacks seedlings soon after emergence. Dark-brown lesions develop on the stem above the soil line. Eventually, a dry rot or canker girdles the stem and the seedling collapses and dies. When older plants are infected, circular or irregular grey to brown lesions develop on lower leaves and stems. Lesions enlarge and coalesce, causing complete yellowing of foliage and severe defoliation. Cankers on stems can cause wilting and death of the upper plant. Fruit lesions start as soft, light-brown, sunken oval areas. Later, fruit lesions deepen, enlarge and coalesce to develop a soft, spongy rot. In dry weather, fruit may shrink and mummify. A diagnostic characteristic is the minute black fruiting bodies (pycnidia) that develop in a circular pattern in the centre of mature lesions. Pycnidia are the inoculum source for later infections.

Conditions for Disease Development

This fungus can survive in plant debris or in mummified fruit in the soil. Infection may occur when rain or overhead irrigation splash inoculum to foliage and stems. Seed produced on plants grown in affected fields can be infested with fungal spores and may initiate disease on seedlings. Phomopsis blight is generally favoured by hot, wet weather.

Control

Sow high-quality seed to help produce pathogen-free transplants. Remove and destroy all infected plant material, and establish a crop rotation to break the disease cycle. Mulch and furrow irrigate to help reduce splashing of water and soil. A regular schedule of protectant fungicide sprays may reduce damage in areas where the disease is known to occur.

Pycnidia form on mature lesions.

Dark lesions on stem.

Progressive fruit rot.

Pycnidia form in concentric rings.

Causal Agent

Leveillula taurica (anamorph: *Odiopsis sicula)* on capsicum and eggplant, and *Golovinomyces cichoracearum (*synonym: *Erysiphe cichoracearum)* *(*anamorph: *Oidium cichoracearum)* on eggplant

Distribution

Worldwide *(L. taurica);* Asia *(G. cichoracearum)*

Symptoms

Leveillula taurica: During initial stages of infection, light-green to bright-yellow blotches appear on upper surfaces of leaves. These areas later turn necrotic. Infected leaves curl upward, and a powdery, white growth is visible on the underside of leaves. When lesions are numerous, they often coalesce, resulting in general chlorosis and leaf drop. The disease progresses from older to younger leaves. Fruits on affected plants are overexposed to sunlight and may develop sunscald.

Golovinomyces cichoracearum: Initially, small circular to irregular, whitish, powdery areas appear on upper and lower leaf surfaces. Infected areas can expand to cover leaves, petioles and stem tissues. Older leaves are affected first, and later, the disease progresses up towards new growth. Affected leaves eventually turn yellow and necrotic.

Conditions for Disease Development

These fungi have a wide host range. Airborne conidia from previous crops or weeds can be carried long distances by wind and act as initial sources of inoculum. Higher temperature, alternating humidity and low light conditions generally favour disease development.

Control

Apply protectant fungicides before an epidemic or immediately after the first symptoms are observed. Provide for air circulation around plants and light penetration through the canopy. Excessive fertilisation has been reported to increase the severity of powdery mildew epidemics.

Necrotic lesions develop in later stages of disease.

Close-up of sporulation.

Sporulating lesion on eggplant stem.

Initial symptoms of infection on capsicum leaves.

Profuse sporulation on undersurface of capsicum leaves.

Sporulation on eggplant cotyledons.

Initial symptoms on chilli plants.

Causal Agent

*Sclerotium rolfsii (*teleomorph: *Athelia rolfsii)*

Distribution

Worldwide

Symptoms

This fungus infects emerging seedlings below or at the soil level and causes damping-off. Under favourable environmental conditions, *Sclerotium rolfsii* is able to infect any part of the plant. The first symptoms of disease in mature plants are dark-brown lesions on the stem at or just beneath the soil line. First foliar symptoms are progressive yellowing and wilting. Later, the fungus produces fan-like webs of whitish mycelium around the rotted stem. Small brown sclerotia form within the mycelial mass. As the disease progresses, infected plants wilt and die.

Conditions for Disease Development

Sclerotium rolfsii has a wide host range. The fungus overwinters as mycelium or sclerotia in and on infected plant debris. Sclerotia can survive in soil for many years. Rainfall or irrigation following a period of drought generally stimulates germination of sclerotia and initiates the infection process. High humidity and warm temperatures generally favour rapid fungal growth and disease development. A soil pH between 3 and 5 is best for fungal growth. At a soil pH of 7 or above, germination of sclerotia is inhibited. Sclerotia spread short and long distances in infected transplants, plant debris, soil, surface water, and on farm equipment and poor quality seed.

Control

Rotate with grasses and deep-plow to bury sclerotia to help reduce soil inoculum level. Grow plants in raised beds to promote soil drainage. In small-scale plantings, rogue infected plants when symptoms are first visible to reduce disease spread. Soil fungicides and biological control using *Trichoderma* spp. and *Gliocladium virens* offer some protection.

Irregular distribution in the field.

Yellowing and wilting of foliage.

Mycelia and sclerotia at the base of an infected capsicum plant.

Causal Agent

Verticillium dahliae, V. albo-atrum, Verticillium spp.

Distribution

Worldwide

Symptoms

These soilborne pathogens cause a wilt by blocking the vascular system of the plant. Although seedlings can be infected, symptoms usually are not observed until plants are older. In eggplant, symptoms of Verticillium Wilt infection progress slowly. A characteristic symptom of infection is a V-shaped lesion that develops on older leaf tips that later expands to cover the leaf. Infected capsicums are stunted and lower leaves are slightly chlorotic. As the disease progresses in capsicums and eggplant, stunting and chlorosis become severe with diurnal wilting. Wilting can be asymmetric, with sections of the plant remaining turgid. Permanent wilt and plant death follow. Dissecting through the crown of affected plants reveals dark-brown vascular discolouration, which can extend into the pith and up into the stem and branches. Fruit that form are small and deformed with internal discolouration.

Conditions for Disease Development

These fungi have an extremely wide host range and can survive in soil and plant debris for several years. Temperatures between 21° and 25° C generally favour disease development. *Verticillium* enters plants through root wounds caused by cultivation, secondary root formation and nematode feeding. Symptomatic plants may be few and restricted to one area, or occur throughout an entire field or greenhouse. Disease development is favoured in heavy clay soils.

Control

Soil fumigation, solarisation and crop rotation to nonhosts help reduce disease incidence. Although no existing commercial varieties have resistance, grafting onto resistant rootstocks is practiced in some countries.

Yellowing of eggplant foliage.

Wilt and V-shaped leaf lesions on eggplant.

Discolouration extends into stems and branches.

Vascular discolouration of eggplant stem.

Exposed stem tissues of infected capsicum (left) and healthy capsicums (right).

Causal Agent

Sclerotinia sclerotiorum

Distribution

Worldwide

Symptoms

The first symptoms of white mould are dark-green, water-soaked lesions that develop on foliage, stems and fruit. Occasionally, the host may exhibit dry lesions on the stalk, stem or branches with a well-defined border between healthy and diseased tissues. Stem infections frequently girdle the stem at the soil line, causing plants to wilt and die. Petiole or bud infections proceed downward in the plant rapidly. Fruit infected directly from the soil surface or through the peduncle rot quickly and turn into a watery mass. In advanced stages, white, cottony mycelium blankets affected tissue, and sclerotia form on the surface. Sclerotia also may form within the stem pith and fruit cavities, becoming black and hard as they mature.

Conditions for Disease Development

This fungus has a wide host range and survives from one season to the next as sclerotia in soil and in plant debris. White mould is most common in temperate regions but is also known to occur in hot, dry areas. Dew, fog and frequent rain generally favour disease development. The most important means of long-distance spread are airborne ascospores that erupt from sclerotia. Moving contaminated soil and fertilising with manure from animals fed infected plant debris are two common ways of short-distance spread of sclerotia or mycelium. Irrigation water may also spread the fungus from field to field.

Control

Plant in well-drained soil, use wide row spacing and water deeply, early in the day. Remove all plant debris from previous crops. Manure and plant mulches suspected to come from infected locations should not be used unless sterilised. Establish a crop rotation with non-host crops such as corn, small grains and grasses. Soil fumigation can be effective at helping to reduce soilborne inoculum.

Mycelia and sclerotia on stem surfaces.

Infections may girdle stem, causing wilt.

Fruit infected through peduncle.

Sclerotia within a stem.

INSECT PESTS
& INSECTS AS VECTORS

APHIDS

BEETLES

EGGPLANT FRUIT AND SHOOT BORER

LEAFHOPPERS

RED SPIDER MITE

THRIPS

WHITEFLIES

APHIDS

Causal Agent

Myzus persicae, Macrosiphum euphorbiae

Distribution

Worldwide

Aphids are small, pear-shaped, gregarious insects. In warm climates or protected heated environments, they produce live offspring without mating. Aphids can travel from leaf to leaf and plant to plant as wingless nymphs and as winged or wingless adults. They can also travel for miles when carried by wind. Aphids usually invade fields as winged adults. Once established, aphids can be found on the growing points and on the undersides of newer leaves. Aphids can cause significant damage to capsicum and eggplant, causing spotting and chlorosis of leaves, leaf curling and distortion and abscission of flowers. The fungi that cause sooty mould can grow on the sugary honeydew excreted by aphids, reducing fruit quality. Two common aphid pests of capsicum and eggplant are the potato aphid (*Macrosiphum euphorbiae*), which is large (3mm) and coloured pink or green, and the green peach aphid (*Myzus persicae*), which is smaller (1.5mm) and coloured light to dark green. The number and diversity of viruses vectored by aphids far exceed those moved around by other vectors. Aphids are known to transmit viruses in both a persistent and non-persistent manner.

Cucumber mosaic virus, Potyviruses like *Tobacco etch virus* and Alfalfa mosaic virus are examples of non-persistent transmitted viruses. In these cases, the virus can be directly acquired by aphids and transmitted within seconds. Viruses persistently transmitted by aphids include *Potato leaf roll virus* and *Beet western yellows virus*. With persistent transmission, the virus transmission starts a few days after acquisition of the virus.

Nymphs of the green peach aphid.

Winged adult of the green peach aphid.

Aphis on eggplant.

Sooty mould colonising honeydew excreted by aphids or whiteflies.

BEETLES

Causal Agent

Epilachna borealis, Diabrotica undecimpunctata

Distribution

Asia, North U.S.

The Epilachna beetle is an eggplant pest in Asia. Adults are red to brown with black spots; larvae are brown with spines. Adults and larvae feed on leaves and new growth, leaving behind skeletonised areas of leaf tissue. Generally, larvae cause more damage to the crop than adults. The Diabrotica beetle is found throughout North America. Feeding damages foliage in capsicum seedlings.

Diabotrica beetle damage on capsicum.

EGGPLANT FRUIT AND SHOOT BORER

Causal Agent

Leucinodes orbonalis

Distribution

South and Southeast Asia

The eggplant fruit and shoot borer (*Leucinodes orbonalis*) is a serious eggplant pest in South and Southeast Asia. Adult moths fly in from adjacent fields or infested debris and deposit eggs on new leaves. Larvae emerge from eggs and travel a short distance to bore into new shoots or fruits. The first symptoms of infestation are freshly wilted shoots. Larvae feeding on fruit render them unmarketable. Yield losses can approach 100 percent. The best way to manage infestations is through good crop sanitation, use of pheromones to trap male moths and judicious use of insecticides to protect natural predators. Effective sanitation includes cutting, removing and destroying damaged shoots until the final harvest. Crop residues should be uprooted and destroyed to remove eggs and larvae.

The eggplant fruit and shoot borer.

Entry sites of the eggplant fruit and shoot borer.

Wilting caused by the eggplant fruit and shoot borer.

LEAFHOPPERS

Causal Agent

*Circulifer tenellus (*synonym = *Neoaliturus tenellus), Hishimonus phycitis*

Distribution

Worldwide

Leafhoppers are found in warm, dry regions worldwide. They are wedge-shaped, can be up to 3 mm long and are green to greenish-yellow to brown in colour. Leafhoppers have a very wide host range, including numerous weeds and vegetables. Leafhoppers feed on phloem, leaving pale, circular spots or peppery specks on leaves. Adult females make hatch cuts across leaf veins and stems to insert eggs. Their life cycle can be completed in 40 to 45 days under favourable environmental conditions. Nymphs are similar to adults in appearance, except they lack fully developed wings. The beet leafhopper, Circulifer tenellus, transmits *Beet curly top virus* to capsicums. The virus is acquired and transmitted persistently by immature and adult stages of leafhoppers. Once acquired, the virus can be transmitted throughout adult life, but it is not passed to the next generation via the egg. The cotton leafhopper or jassid, *Hishimonus phycitis*, is an eggplant pest in Asia. Leafhoppers feed on the undersides of leaves causing small, yellow patches. In severe attacks, interveinal yellowing and necrotic areas that resemble nutrient deficiencies develop. Infestation also causes a reduction in yield. Leafhoppers may transmit a phytoplasma that causes little leaf disease.

The cotton leafhopper or jassid.

Leaf symptoms caused by jassid feeding on eggplant.

Jassid feeding damage on eggplant.

RED SPIDER MITE

Causal Agent

Tetranychus urtucae, Polyphagotarsonemus latus

Distribution

Worldwide

Fine webbing is visible on and under eggplant and capsicum leaves in mite-infested fields. The two-spotted spider mite (*Tetranychus urtucae*) feeds on the underside of capsicum and eggplant leaves, causing pale, stippled spots and bronzing in infested foliage where the broad mite (*Polyphagotarsonemus latus*) is particularly problematic in greenhouse capsicum crops.

Mites feed on new growth, flowers, fruit and the undersides of leaves. Infested leaves are elongated, curled, deformed and small. Broad mite feeding in flowers and fruit causes abortion and scarring of fruit. Mites are more destructive under dry conditions. Elimination of this pest before flowering and fruit set is important. After fruit formation, mites can feed and lay eggs under the calyx, making them difficult to eradicate. Miticide applications and/or washing infested plants with water or insecticidal soaps can reduce damage.

Distorted leaves caused by mites on chilli.

Russeting caused by mites on eggplant.

Russeting caused by mites on eggplant.

THRIPS

Causal Agent

Frankliniella occidentalis, Thrips tabaci, Frankliniella schultzii, Thrips parvispinus and *Thrips palmi*

Distribution

Worldwide

Thrips are the oldest pests persistent worldwide. The western flower thrips (*Frankliniella occidentalis*) are native to the western USA but have been introduced into many regions worldwide. *Thrips tabaci* and *Frankliniella schultzii* are also present worldwide in capsicums. predominantly. An addition to the list of damaging thrips is known as the tobacco thrips, invasive thrips, or southeast Asian thrips, *Thrips parvispinus*. This species has been present mostly around Southeast Asia to Australia the past two decades, but it is now reported in countries in Europe, Asia, Africa and North America.

Generally, thrips reproduce without mating. The larvae are relatively inactive, but the tiny adults (< 0.5 mm) are winged and mobile. Adults live up to 20 days, and populations can increase quickly. Thrips feed on new leaves and developing flowers, causing misshapen, twisted and cupped leaves and browning of lower leaf surfaces. The feeding by larvae causes scarring and discolouration in developing fruits. Yield losses can be severe, though the thrips themselves are difficult to see in plants. Thrips can be seen by shaking flowers and new foliage over a white sheet of paper.

Besides the direct damage caused by thrips, they vector virus diseases such as tospoviruses (*Tomato spotted wilt virus* and *Peanut bud necrosis virus*), *Chilli leaf curl virus, Tobacco streak virus*, and *Capsicum chlorosis virus* in capsicums. Thrips larvae acquire tospoviruses after short feeding periods and transmit these viruses primarily as adults. Occasionally, transmission occurs by nymphs. These viruses are not passed to the next generation, although there is some evidence to support replication in the vector. However, there is no documented evidence to suggest that invasive tobacco thrips transmit viruses or viral diseases.

Male and female thrips on capsicum flower.

Thrips on capsicum flower.

Leaf distortion from thrips feeding.

The western flower thrips nymph.

The western flower thrips adult.

Thrips damage on chilli fruit.

Severe leaf distortion from thrips feeding.

WHITEFLIES

Causal Agent

Trialeurodes vaporariorum, Bemisia tabaci

Distribution

Worldwide

The greenhouse whitefly (*Trialeurodes vaporariorum*) and sweet potato whitefly or silverleaf whitefly (*Bemisia tabaci*; synonym = *B. argentifolii*) are serious insect pests worldwide. *B. tabaci* represents a complex of over 35 cryptic species (e.g., Middle East Asia Minor 1 [MEAM1], formerly termed B biotype and Mediterranean [MED], formerly termed Q biotype) and has emerged as an invasive pest that spreads viruses worldwide. Over 500 plant species including weeds, vegetables, ornamentals and agronomic crops are attacked. Immature and adult whiteflies colonise the undersides of leaves. The larval stages are sedentary, whereas the tiny (1 mm) adults fly short distances from leaf to leaf or plant to plant or are carried for miles by the wind. Once established, populations build up rapidly due to a life cycle of 20 days or less in dry, warm climates. Rain and cold weather reduce whitefly populations. Plant damage is similar to that caused by aphids.

Whiteflies feed on phloem and produce sugary honeydew on leaves and fruit. Sooty mould fungi colonize the honeydew, reducing fruit quality and yield. Whitefly infestations can also slow plant growth and cause stunting and defoliation. In capsicum and eggplant, *B. tabaci* and *B. argentifolii* are important vectors of geminiviruses, such as *Pepper golden mosaic virus, Sinaloa tomato leaf curl virus, Pepper hausteco yellow vein virus*, Tomato yellow mosaic virus and other geminiviruses found worldwide. These viruses are all carried persistently, generally throughout the adult life of the vector. There is no documented evidence to suggest that these viruses are passed to the offspring of infected adult whiteflies.

Larval (top left) and adult (right) stages of the silverleaf whitefly.

Leaf distortion caused by whitefly feeding.

Whiteflies on leaf.

Whitefly on leaf.

NEMATODE DISEASES

ROOT-KNOT

Causal Agent

Meloidogyne incognita, M. javanica, M. arenaria, M. enterolobii

Distribution

Worldwide *except M. enterolobii* (Mexico and Southern U.S.)

Symptoms

Plants infected by root-knot nematodes are generally less vigorous than healthy plants. In foliage, symptoms of nutrient deficiency and diurnal wilting are visible due to reduced function of the root system. Diagnosis can be confirmed by carefully digging up the roots and observing the presence of bead-like galls. Galls are irregular enlargements of root tissue induced by nematode feeding and tend to be larger when multiple infections occur. Galls caused by different species of root-knot nematode may be similar in appearance.

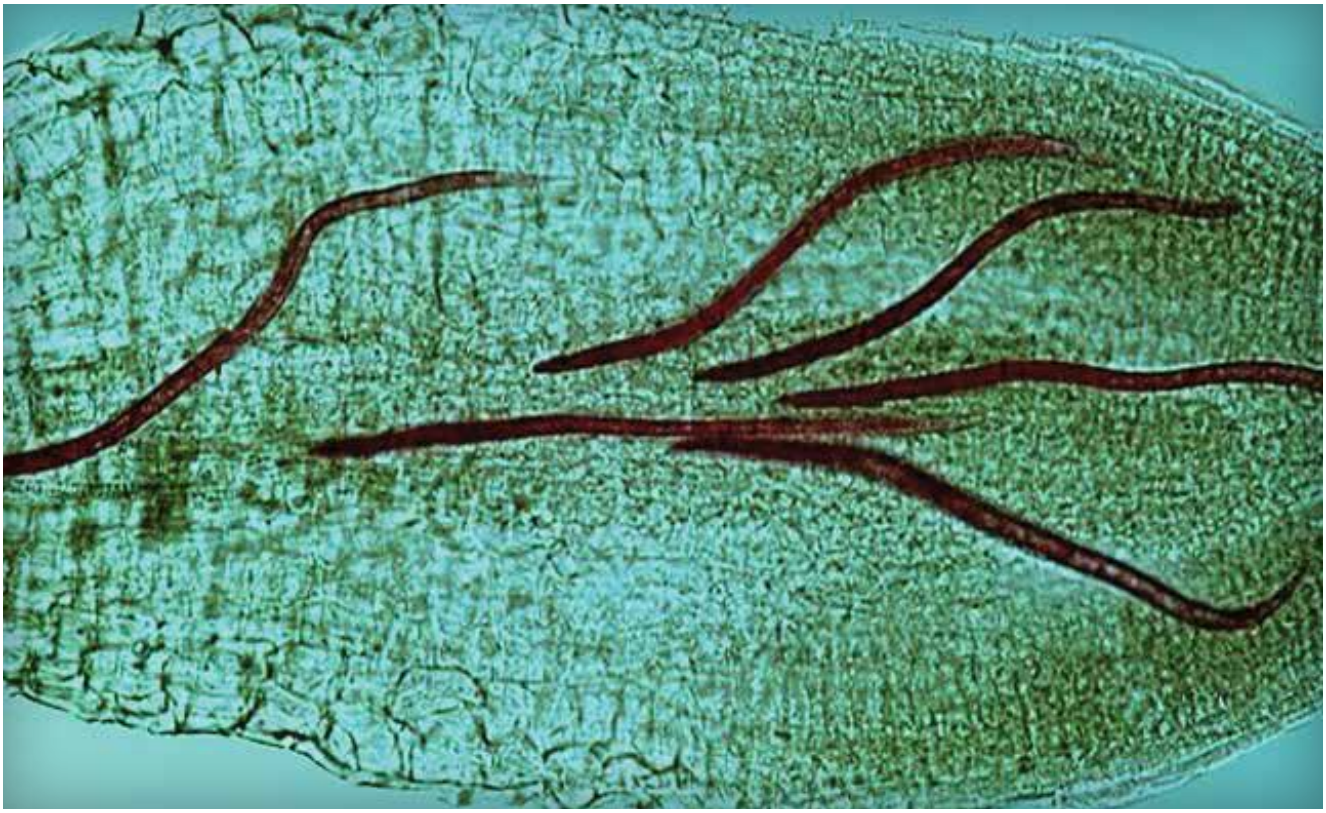
Conditions for Disease Development

The host range of these three nematode species is very wide and includes many agricultural crops and weeds. Disease is most severe in warm areas with long growing seasons. In general, lighter, sandy soils favour nematode infection and result in more severe damage to roots.

Control

Fumigate infested soil and rotate to non-host grass crops to help reduce nematode populations. Plant root knot nematode-resistant varieties.

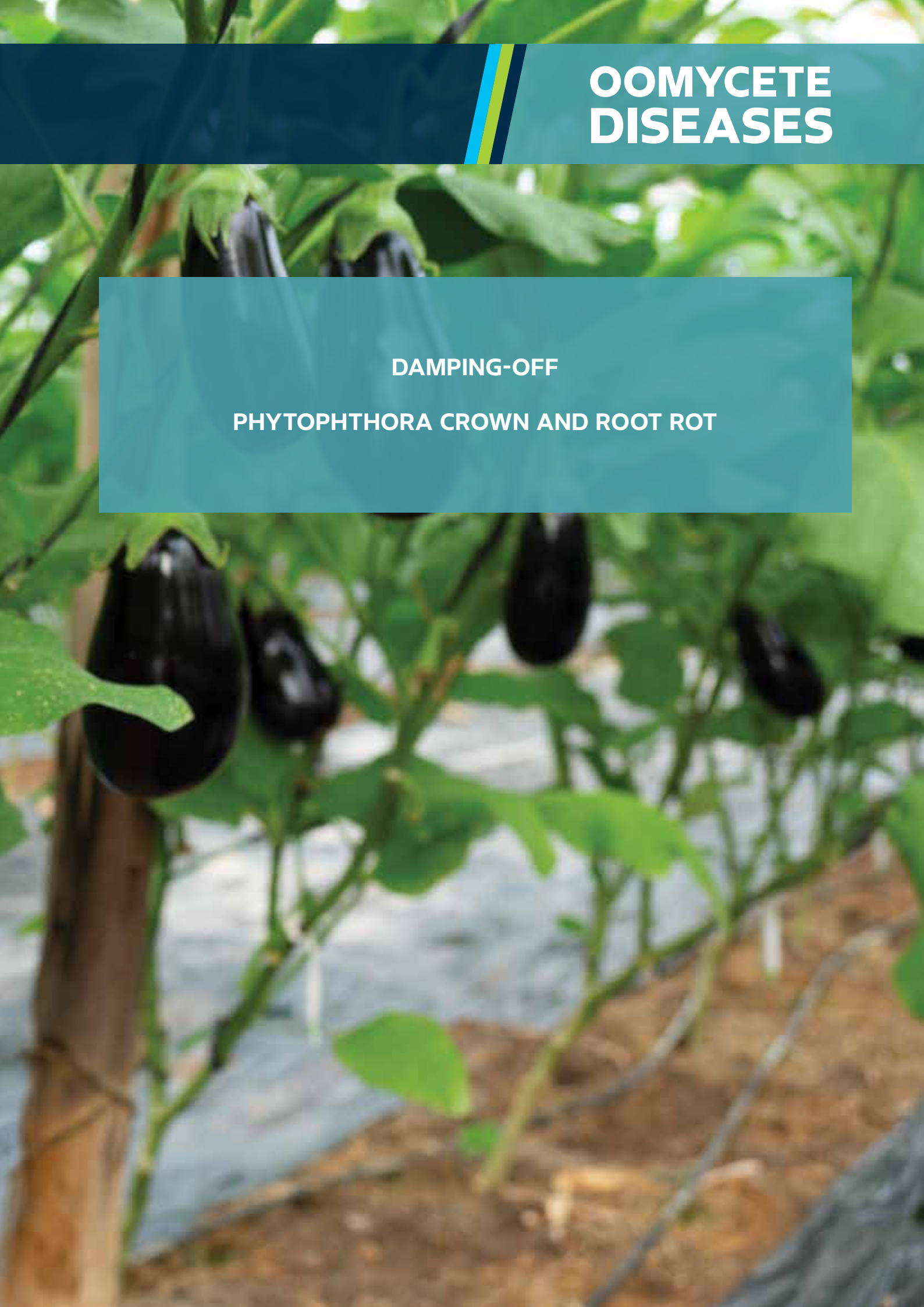
Root-knot nematode galls in eggplant.



Second-stage juveniles of root-knot nematode, Meloidogyne sp., penetrating roots.

Root-knot nematode galls in capsicum.

OOMYCETE DISEASES



DAMPING-OFF

PHYTOPHTHORA CROWN AND ROOT ROT

Causal Agent

Phytophthora capsici

Distribution

Worldwide

Symptoms

Pre-emergence Damping-Off: Seeds may rot before germinating or seedlings may die prior to emergence.

Post-emergence Damping-Off: Young seedlings develop a rot at the crown. Later, the tissue becomes soft and constricted, and the plants wilt and fall over.

Pythium spp.: This fungus is the most common cause of pre-emergence Damping-Off. Typical symptoms include dark-brown to black, water-soaked lesions that rapidly spread over the entire seedling. Brown, water-soaked lesions that start on the roots and later extend up the hypocotyl characterise post-emergence Damping-Off. Eventually, the lesions girdle the hypocotyl, causing seedlings to wilt and die. The root cortex becomes macerated and easily sloughs off.

Conditions for Symptom Development

These fungi have a wide host range and can survive for long periods in soil, plant debris and weeds. Damping-Off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather. Water-splashing moves infested soil from diseased to healthy plants.

Control

Improving drainage and moisture regulation to help prevent soil saturation reduces Damping-Off. Fungicidal soil drenches and seed treatments are available that help manage Damping-Off. Rotation to cereal crops and soil fumigation or solarisation may help reduce Damping-Off in the field. Use pasteurised soil mixes in nurseries.

Capsicum (left) and eggplant (right) showing rot.

Girdled hypocotyl leads to seedling death.

Post-emergence Damping-Off in the field.

Causal Agent

Phytophthora capsici

Distribution

Worldwide

Symptoms

All plant parts can be infected. Root infection typically results in a rapid wilting of the plant. Infected roots turn dark-brown and the outer cortex sloughs off easily. Crown infections start as dark-green, water-soaked lesions that turn dark-brown as they expand. A cross-section through the infected crown reveals dark-brown discolouration that may extend throughout the cortex. Leaf symptoms start as small irregular, water-soaked lesions. As lesions expand, infected tissue dries and turns tan. Sporulation may be observed on leaf surfaces under conditions ideal for fungal growth. Infected stems may be girdled and later dieback. Fruit lesions initially appear dark-green and water-soaked. As infection expands, fruit shrivel, but typically do not drop. Fungal spores and mycelium may be observed on fruit surfaces.

Conditions for Symptom Development

Rainfall, wet soils and poor drainage generally favour disease development and spread. In climates where irrigation is used, extended periods of soil wetness also favour the root and crown rot, stage of Phytophthora Blight. Disease spread usually follows the direction of surface water run-off. In climates with heavy rainfall, foliar blight, as well as root and crown rot occur. Splashing rain and wind spread disease from plant to plant. Spread can also occur when soil is carried on equipment and footwear. *Phytophthora capsici* can survive in the soil for years in tropical, subtropical and temperate climates.

Control

Use fungicide sprays in combination with cultural practices such as water management and crop rotation to manage Phytophthora Blight. Plant on raised beds to help promote water drainage away from plant roots.

Leaf symptoms on capsicum.

Localised area of wilting and plant death in a field.

Wilt symptoms on capsicum.

Leaf blight and stem dieback on capsicum.

Black stem lesions on capsicum.

68 / PHYTOPHTHORA CROWN AND ROOT ROT

Root and crown rot on eggplant.

Blight and wilt on eggplant.

Sporulation on eggplant fruit.

PARASITIC PLANTS

DODDER

Causal Agent

Cuscuta spp.

Distribution

Worldwide

Symptoms

More than 100 species of *Cuscuta* occur worldwide. Dodder is an annual parasitic plant that can be identified by slender, white, yellow or red, leafless strands that twine around the host plant. Dodder has no chlorophyll and depends on the host plant for its nutrition. As a result, infected plants appear weak and discoloured. Growth and yield can be significantly reduced. Under heavy dodder infestations, small host plants may die. As the season progresses, dodder grows down a row to cover plants with a mass of vines. Heavily infested fields appear yellow.

Conditions for Symptom Development

Generally, dodder has a very wide host range and is adapted to a wide range of environments. After germination, the seedling depends on nutrients stored for its survival. If a suitable host is not found within a few days, it will die. Once a seedling makes contact with a host, it forms sucker-like projections (haustoria) that penetrate plant tissues. Dodder produces small, inconspicuous flowers (often white) that mature and produce two to four yellow to black seeds. Irrigation water and cultivation equipment are common modes of long distance dispersal. Dodder seeds are small and can remain viable in the soil for up to 10 years. Seeds usually germinate in late winter and spring in cold climates, however, germination can continue through the summer. Environmental conditions that favour capsicum and eggplant growth are also beneficial to dodder.

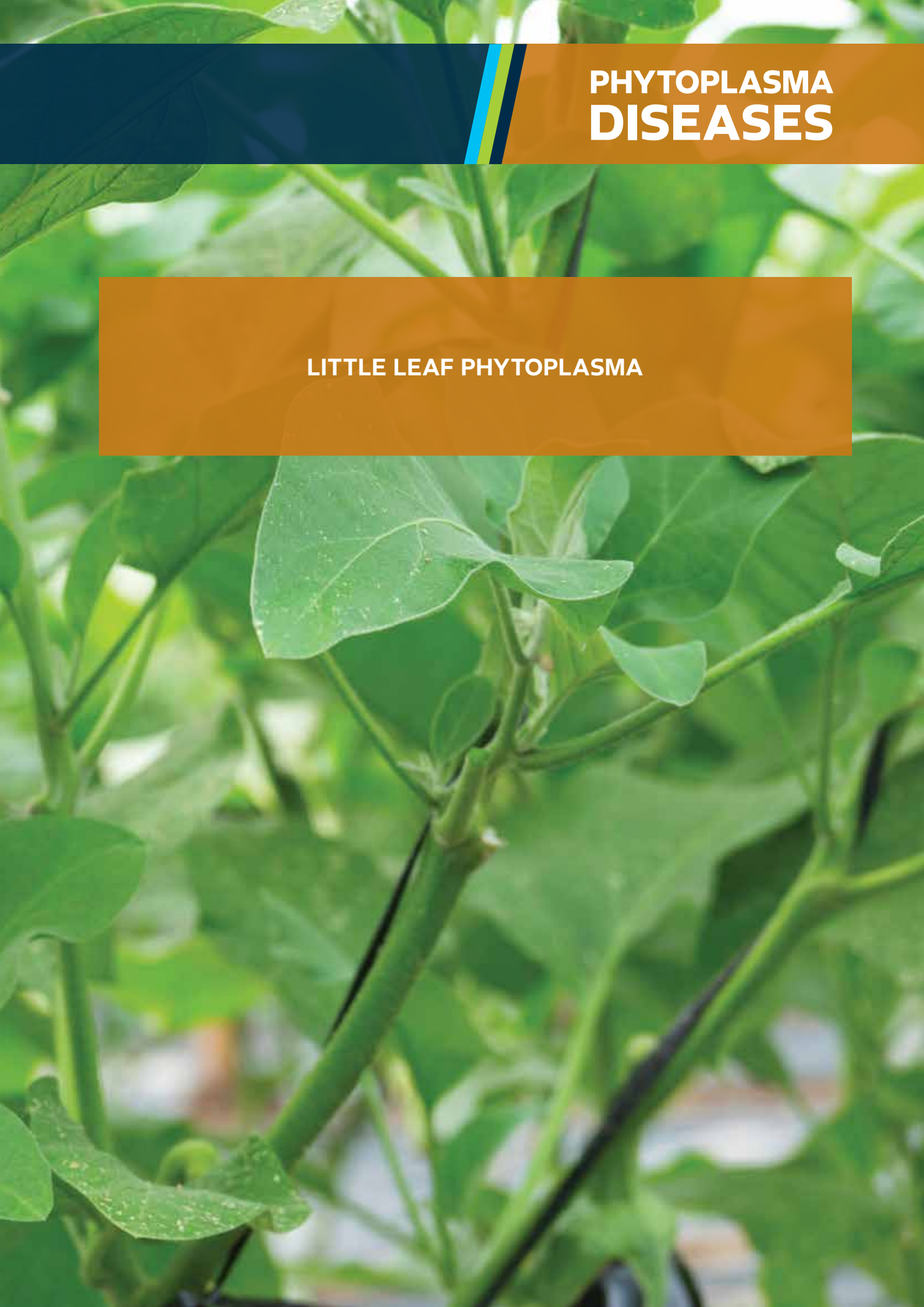
Control

Immediately remove or burn dodder, along with infested plants upon detection. Contact herbicides can be used to help control localised infestations. If an infestation is widespread, apply pre-emergence herbicides, deep-plow crop debris and rotate to grasses. Dodder seeds are often difficult to distinguish from host crops so close inspection of seed lots is suggested.

Yellow strands of dodder covering capsicum plants.

Dodder infestation in the field.

Flowers produce seeds that may remain viable many years.



PHYTOPLASMA DISEASES

LITTLE LEAF PHYTOPLASMA

Causal Agent

Little leaf phytoplasma

Vectors

The cotton leafhopper or jassid *(Hishimonus phycitis)*

Distribution

India, Bangladesh

Symptoms

Infected plants produce tiny, pale-green leaves on very short petioles. Stem internodes are short, and plants are stunted and bushy due to stimulation of axillary buds. Excessive root branching is common. Flowers formed after infection are leaf-like and sterile. Yield losses can approach 100 percent.

Conditions for Symptom Development

Little leaf disease is widespread in India, where overlapping crop cycles and weeds ensure high populations of leafhoppers and provide reservoirs for the phytoplasma. Eggplants can be infected at any stage of growth when phytoplasma-carrying leafhoppers are present. Grafting also can spread the disease.

Control

Eradicate solanaceous weeds that harbor the vector, and spray insecticides to help reduce leafhopper populations to control the spread of this disease. Rogue symptomatic plants as soon as they are detected to reduce secondary spread of this disease.

Infected plants develop shortened internodes and distorted leaves.

Plants remain stunted and bushy.

Late infections only affect new growth.

VIRAL DISEASES

ALFALFA MOSAIC

BEET CURLY TOP

CHILLI VEINAL MOTTLE

CUCUMBER MOSAIC

GEMINIVIRUSES

POTATO X

POTYVIRUS: PEPPER MOTTLE

POTYVIRUS: PEPPER YELLOW MOSAIC

POTYVIRUS: POTATO Y

POTYVIRUS: TOBACCO ETCH

TOBAMOVIRUSES

TOSPOVIRUSES

Causal Agent

Alfalfa mosaic virus (AMV)

Vectors

Many species of aphids

Distribution

Worldwide

Symptoms

A distinctive bright-yellow mosaic develops on leaves. Often fruits are mottled and distorted.

Conditions for Disease Development

AMV has a wide host range among dicotyledonous plants. Infections generally occur when capsicums are grown near alfalfa fields. The virus is transmitted by many aphid species in a non-persistent manner. Viruliferous aphids spread the disease rapidly within a field for very short periods of time.

Control

To best manage AMV, avoid planting capsicums near alfalfa fields. There is no effective chemical control of this disease because aphids immediately transmit the virus by feeding on infected plants. This virus can be transmitted mechanically and by seed. There is currently no resistance in commercial capsicums cultivars.

80 / ALFALFA MOSAIC

Distorted fruit and mosaic symptoms in leaves.

Bright-yellow mosaic on foliage.

Mosaic symptoms in leaves.

Causal Agent

Beet curly top virus (BCTV)
[synomym: *Curly top virus* (CTV)]

Vectors

The beet leafhopper (Circulifer tenellus)

Distribution

Worldwide, in arid and semi-arid regions

Symptoms

When seedlings are infected, leaves turn yellow, twist and curl upward, and thicken to become stiff and crisp. Petioles may curl downward. Fruit set is reduced. Fruit appear dull and wrinkled, and tend to ripen prematurely. This virus is not mechanically transmitted.

Conditions for Disease Development

This virus has a wide host range, affecting more than 300 species. Common hosts are tomatoes, beets, capsicums, squash, beans, cucurbits, spinach, potatoes, cabbage and alfalfa. The beet leafhopper transmits BCTV in a persistent manner. Warm temperatures and dense leafhopper populations are conducive to the spread of BCTV. Viruliferous leafhoppers migrate seasonally and can be moved long distances by wind.

Control

Transplant virus-free seedlings. Rogue infected plants to help avoid transmission in the field. Control weeds near capsicum fields to reduce vector and virus reservoirs. Transplant early or late to escape leafhopper infestations, and increase plant density to compensate for losses due to BCTV. Insecticides are generally not effective in controlling Beet curly top.

82 / BEET CURLY TOP

Thickened leaves curl upward.

Foliage and fruit symptoms in the field.

Affected plants have shortened internodes.

Causal Agent

Chilli veinal mottle virus (ChiVMV)

Vector

Many species of aphids

Distribution

Asia

Symptoms

Typically, leaves of infected plants develop a mottle or mosaic with dark-green vein banding. Plants infected when young usually are stunted, with dark-green streaks on stems. Most flowers drop before fruit set. Affected fruit may be mottled and deformed. Symptom severity is dependent upon the variety, infecting strain and age of the host at the time of infection. Eggplant can be infected, but remains asymptomatic.

Conditions for Disease Development

Capsicums, tobacco, tomatoes and weeds such as *Physalis* spp. are hosts of ChiVMV. Tropical climates support the continuous presence of ChiVMV and its vectors. ChiVMV is transmitted by several species of aphids in a non-persistent manner and can also be transmitted mechanically through pruning and grafting. There is no evidence of seed transmission.

Control

Use resistant varieties and virus-free transplants. Manage aphid populations using reflective mulches, stylet oil sprays and insecticides. Controlling the aphid vector population with chemical treatment is very difficult and generally provides limited control. In mature plants, it is difficult to achieve complete insecticide coverage of leaves to effectively eradicate all aphids.

Close-up of mosaic and vein banding.

Chlorotic mosaic with dark-green vein banding.

Dark-green streaks on stems.

Causal Agent

Cucumber mosaic virus (CMV)

Vectors

Many species of aphids

Distribution

Worldwide

Symptoms

Symptoms can vary greatly depending upon the affected variety, age of the plant at the time of infection and the strain of the virus. Leaves may become narrow, distorted and mottled. In capsicum, defoliation may occur when mature plants are infected. Tip dieback and leaf discolouration in an "oak-leaf" pattern may develop. Infected plants are usually stunted and the fruits distorted with occasional concentric rings. Infection of young plants results in unmarketable fruit and severe yield losses.

Conditions for Disease Development

The host range of CMV includes as many as 800 plant species. The virus is acquired by aphids and is transmitted from plant to plant in a non-persistent manner. CMV often remains in infected alternate hosts near agricultural areas and is transmitted to capsicums and eggplant when environmental conditions support disease development. This virus is also mechanically transmitted.

Control

Control aphids, and rogue-infected plants to reduce the incidence of CMV in greenhouse crops. In field-grown capsicums and eggplant, eliminate adjacent weeds and ornamentals, and use reflective mulches to deter aphids and a combination of stylet oil and insecticide sprays to reduce losses caused by this virus. Controlling the aphid vector population with chemical treatment is very difficult and generally provides limited control. In mature plants, it is difficult to achieve complete insecticide coverage of leaves to effectively eradicate all aphids. Resistant varieties are available in capsicum.

Distorted and mottled fruit.

Stunted plants with distorted foliage.

Leaf mottling and distortion.

Vein banding and mottling in eggplant.

Characteristic “oak-leaf” pattern.

Causal Agent

Pepper Golden Mosaic Complex: *Pepper huasteco yellow vein virus (PHYVV)* [synonym: *Pepper huasteco virus* (PHV)]. *Sinaloa tomato leaf curl virus* (STLCV), *Chilli Leaf Curl Virus (ChiLCV), Pepper Yellow Leaf Curl Thailand Virus* (PYLCThV), *Pepper Golden Mosaic Virus* (PepGMV) and additional uncharacterized begomoviruses)

Vector

Sweet potato whitefly *(Bemisia tabaci)*, Silverleaf whitefly *(B. argentifolii)*

Distribution

Worldwide

Symptoms

Symptoms vary depending on the variety infected, the viruses present, environmental conditions and age of plant at the time of infection. Early symptoms may include yellow vein-etching and clearing, and distortion of young leaves. As infection progresses, symptoms of chlorosis, mosaic and mottling develop, and distortion is more prominent. Infected plants are stunted, and fruit are small, discoloured and distorted. This family of viruses can cause significant yield reductions in capsicums and eggplant.

Conditions for Disease Development

The pepper golden mosaic complex is comprised of closely related viruses that infect in many combinations. Pepper huasteco yellow vein and Sinaloa tomato leaf curl are serious diseases of capsicum grown in Mexico, where large areas of chillis are grown and whiteflies are established. Geminiviruses are spread from host to host by whiteflies in a persistent manner and are not mechanically transmitted. These viruses also infect tomatoes, which are a preferred whitefly host. Consequently, tomatoes can be a source of vector and virus to nearby capsicum fields.

Control

Overlapping cropping systems make control of these viruses and their vectors very difficult. Exclude whiteflies from nurseries using protective netting or screenhouses to grow virus-free transplants. Apply systemic insecticides early to reduce whitefly populations and limit the spread of these viruses. In mature plants, it is difficult to achieve complete insecticide coverage of leaves to effectively eradicate all whiteflies. Even low whitefly populations efficiently transmit geminiviruses. Destroy residual crops after harvest and maintain a host-free period to help manage whiteflies and geminiviruses.

Mosaic caused by the pepper golden mosaic complex.

Rough and distorted fruit.

Severe distortion of capsicum leaves.

Twisted and distorted leaves.

PVH-infected fruit (left) and healthy fruit (right).

Developing mottle in eggplant.

Chlorosis and mottling of foliage.

90 / GEMINIVIRUSES

Pepper geminivirus on chilli *(Pepper Yellow Leaf Curl Thailand Virus).*

Pepper geminivirus on chilli in field *(Chilli Leaf Curl Virus).*

Bright leaf discolouration in eggplant.

Pepper geminivirus on chilli in field *(Pepper Yellow Leaf Curl Thailand Virus)*.

Pepper geminivirus on chilli in field *(Chilli Leaf Curl Virus)*.
Whiteflies present on several leaves.

Pepper geminivirus on capsicum in field *(Pepper Golden Mosaic Virus)*.

Causal Agent
Potato virus X (PVX)

Vector
Mechanically transmitted with no known insect vectors

Distribution
Worldwide

Symptoms
Foliar symptoms of infection include necrotic spots, distortion and ringspots. Mosaic symptoms may also develop in eggplant. Affected leaves are small and may eventually drop. Plants appear stunted and bushy. Fruit that develop are small and distorted, and yield can be reduced.

Conditions for Disease Development
Potatoes, tomatoes and Brassica spp. are common hosts of this virus, and often serve as sources of infection from adjacent fields or volunteer plants. Introduction and spread of PVX is largely due to movement of personnel and equipment. Cultural practices such as transplanting, grafting, staking and pruning, as well as plant-to-plant contact, contribute to the spread of this disease.

Control
Problems in capsicums are infrequent. Roguing infected plants, and sanitation of equipment and tools can help reduce the spread of this virus in capsicums and eggplant. Do not follow a field of potatoes with capsicums or eggplant.

92 / POTATO X

Vein necrosis.

Necrosis and distortion of foliage.

Causal Agent

Pepper mottle virus (PepMoV)

Vector

Many species of aphids

Distribution

Southern United States, California, Mexico and Central America

Symptoms

Symptoms vary depending on the capsicum variety infected, isolate of the virus present, age of plants when infected and environmental conditions. Affected field plants may develop a systemic mottle, distortion, and may be stunted. Infected greenhouse plants first develop vein-clearing followed by chlorotic mottle in newer leaves. Fruit may be distorted, mottled and small. Field plants often are infected with more than one virus. Multiple infections usually result in symptoms different from those caused by PepMoV alone and may appear more severe.

Conditions for Disease Development

Like TEV, PVY and CMV, PepMoV can be transmitted by many species of aphids. PepMoV is transmitted on the mouthparts of aphids in a non-persistent manner. Weeds, such as Datura, and other solanaceous plants can harbor the virus and vector. Aphids may spread infection within a crop, or the virus may be transmitted mechanically through cultural practices such as staking, pruning or handling of infected plants.

Control

Remove crop residues and weeds that serve as a reservoir for both virus and vector. Use reflective mulches to deter aphids, and combine the use of stylet oils and insecticide sprays to reduce losses in young plants. Controlling the aphid vector population with chemical treatment is very difficult and generally provides limited control. In mature plants, it is difficult to achieve complete insecticide coverage of leaves to effectively eradicate all aphids. Resistant varieties are available in both capsicums and chillis.

94 / POTYVIRUS: PEPPER MOTTLE

Typical mottle symptoms in leaves.

Mottling and distortion of foliage.

Deformed and mottled fruit.

Causal Agent

Pepper yellow mosaic virus (PepYMV)
[synonym: *Potyvirus Y - montemor* (PVYm)]

Vector

Many species of aphids

Distribution

Brazil

Symptoms

Typical symptoms of Pepper yellow mosaic include vein banding, blistering and a bright-yellow mosaic. Leaves also may be distorted and develop epinasty. Generally, plants are stunted. Fruit develop a mosaic and may be distorted.

Conditions for Disease Development

Aphids transmit this virus in a non-persistent manner. The widespread use of Potyvirus Y resistant cultivars in commercial vegetable production in the 1970s may have contributed to emergence of PepYMV as a serious capsicum pathogen in Brazil. Pepper yellow mosaic also affects tomato.

Control

Plant PepYMV-resistant capsicum varieties. Use of insecticides to control the disease is generally not efficient due to the short times for acquisition and transmission of this virus by aphids. Cultural practices that may delay infection include use of reflective mulches to deter aphids and weed control to help remove virus and vector reservoirs.

96 / POTYVIRUS: PEPPER YELLOW MOSAIC

Mosaic and distortion of foliage.

Foliar symptoms of *Pepper Yellow Mosaic Virus*.

Close-up of affected leaf.

Causal Agent

Potato virus Y (PVY)
[synonym: *Brinjal mosaic virus* (BMV)]

Vectors

Many species of aphids

Distribution

Worldwide

Symptoms

Symptoms are variable in appearance and severity depending on the variety infected, the isolate of the virus present, age of the plant at the time of infection and environmental conditions. Leaf symptoms include mottling, vein-banding and clearing, and distortion. Plants may be stunted with necrosis of the stems and apical buds. Infected eggplants may show concentric rings on the foliage. Affected fruits may be distorted, discoloured and small. Field plants are usually infected by more than one virus. Multiple infections result in symptoms more severe in appearance and more complex than those caused by PVY alone.

Conditions for Disease Development

Many weed species serve as alternate hosts for this virus. Like *Tobacco etch virus, Cucumber mosaic virus* and *Pepper mottle virus,* PVY can be transmitted by many species of aphids. All four viruses are transmitted in a non-persistent manner on the mouth parts of aphids moving into fields from nearby host plants (including capsicums, tomatoes, eggplant, tobacco, potatoes and solancaceous weeds). Aphids can cause secondary spread of infection within a crop, or the virus may be transmitted mechanically through staking, pruning or handling infected plants.

Control

Remove crop residues and weeds that can help harbor the virus, and rogue symptomatic plants to help reduce disease spread. Use reflective mulches to deter aphids and stylet oils or insecticides to reduce losses in young plants. Chemical control of the aphid populations is very difficult and provides limited control. In mature plants, it is difficult to achieve complete insecticide coverage of leaves to effectively eradicate all aphids. Resistant varieties are commercially available in both capsicum and chilli. However, new strains may emerge that overcome existing resistance genes.

Mosaic symptoms on eggplant.

Vein banding and distortion on eggplant.

Mottling and distortion on capsicum.

Mosaic and distortion on eggplant.

Necrotic rings on eggplant leaves.

Necrotic streaks on eggplant stem.

Causal Agent

Tobacco etch virus (TEV)

Vector

Many species of aphids

Distribution

North and South America

Symptoms

The appearance and severity of symptoms may vary with the capsicum variety infected, the isolate of virus present, age of the plant at the time of infection and environmental conditions. Plant and leaf symptoms include mottling, distortion and stunting. Unlike capsicums, infected chilli plants can develop severe wilting. Affected fruits may be distorted with chlorotic streaks or mosaic patterns. In the field, plants are usually infected by more than one virus. Multiple infections result in symptoms more severe and complex than those caused by TEV alone.

Conditions for Disease Development

Many weed species serve as alternate hosts for this virus. Like *Tobacco etch virus, Cucumber mosaic virus* and *Pepper mottle virus,* TEV is transmitted by aphids in a non-persistent manner as aphids move into capsicum fields from nearby host plants (capsicums, tomatoes, tobacco and weeds). Secondary spread occurs as aphids move from plant to plant. The virus may also be transmitted mechanically through activities such as staking, pruning or handling infected plants.

Control

Remove crop residues and weeds that can harbor the virus. In young plants, use reflective mulches to deter aphids and stylet oil and insecticide sprays to help reduce losses. However, controlling the aphid vector population with chemical treatments provides limited control. As plants increase in size, it is difficult to achieve complete insecticide coverage of leaves to effectively kill all aphids. Moreover, aphids can acquire and transmit the virus after very short feeding periods. Resistant varieties are commercially available in both capsicum and chillis. However, commercial resistance is not effective against all isolates of this virus.

100 / POTYVIRUS: TOBACCO ETCH

"Fernleaf" symptoms.

Leaf mottling and distortion.

Resistant (left) and susceptible (right) capsicum plants.

Causal Agent

Tomato brown rugose fruit virus (ToBRFV)

Vector

Mechanically transmitted with no known insect vectors

Distribution

Worldwide

Symptoms

Symptoms of infection by TMV and ToMV in capsicums and eggplant can vary greatly with the strain of virus, temperature, light intensity, day length, age of the plant when infected and cultivar. Foliar symptoms include chlorotic mosaic, distortion and, at times, systemic necrosis and defoliation. Plants infected as seedlings can be stunted and are generally chlorotic. Infected plants produce disfigured fruit that are usually small with distinct chlorotic and/or necrotic areas. Foliar symptoms of PMMoV in capsicums are also variable but are generally mild. Plants infected as seedlings remain stunted. Leaves develop a subtle mosaic, can be crinkled, and remain small. Symptoms may first appear on fruit. Fruit can be mottled and necrotic, are usually small and distorted, and have a rough or wrinkled appearance.

Conditions for Disease Development

Tobamoviruses are mechanically transmitted by contact between any infected surface and plant tissue to an uninfected plant. Because Tobamoviruses are very stable, extensive spread can occur through handling, tools, trays, pots, stakes, twine and clothing, as well as pollination, pruning and other cultural practices. Infected transplants, seed and debris can also act as sources of inoculum.

TMV and ToMV have very wide host ranges and can infect over 200 plant species, including varieties of capsicums, tomatoes, eggplant and tobacco. PMMoV is specific to all species of capsicums but does not infect tomatoes, tobacco or eggplant. Tobamoviruses can remain viable for several years in plant debris but generally lose their ability to infect as debris decomposes. However, these viruses can live on surfaces for equally as long outside of a host if left unsanitised.

The seed industry uses multiple techniques to test for presence of the virus and secure clean seeds for customers. Certified clean seed has been inspected and tested for presence of the virus with stringent testing thresholds, and customers should feel comfortable using these seed materials.

Yellow mosaic in capsicum.

Mosaic symptoms in eggplant.

Control

Enforce strict sanitation practices in all steps of the cropping period. Disinfect soil and renew artificial growth media, sanitise all equipment and greenhouses using appropriate sanitising agents, and use seed tested and treated for tobamoviruses.

During the season, restrict visitors' access to the crop, wash hands and equipment with a soap solution before entering a greenhouse or moving between plants or rows of plants. There are reports of successful prevention of Tobamovirus spread by coating hands, plants and equipment with a solution of rehydrated powdered nonfat milk of an appropriate protein content.

When infected plants are found, remove symptomatic and adjacent plants in a plastic bag and rotate to non-solanaceous crops to manage the disease. Resistance to different races of Tobamoviruses is widely available, and use of resistant varieties in greenhouse production is highly recommended. New races of virus that can overcome known resistances have been reported; however, these have not been established and therefore are not relevant for current growing.

Yellowing and mottling in capsicums.

Chlorotic mottle in eggplant.

Rough surfaces of infected fruit.

Fruit blotches.

Deformed and mottled capsicum fruit.

104 / TOBAMOVIRUSES

TOMATO BROWN RUGOSE FRUIT VIRUS

Causal Agent

Tomato brown rugose fruit virus (ToBRFV)

Vector

Mechanically transmitted

Distribution

Israel, Jordan, Kuwait, Saudi Arabia, Parts of Europe, Mexico, Australia.

History and Context

This rapidly spreading RNA virus is causing severe regulatory responses due to its impact on the agriculture industry and its ability to be transmitted mechanically. This disease was mistakenly thought to be caused by *Tobacco mosaic virus* (TMV) until genetic studies revealed it was an entirely new disease in 2014. This disease was first described in the Middle East/Mediterranean region at this time and has since been described in many locations worldwide since its discovery.

Symptoms

Characteristic symptoms of *Tomato brown rugose fruit virus* (ToBRFV) vary from host to host. In capsicums, leaves can become puckered, and yellow mottling on leaves can become apparent, as well as an overall stunting of the plant. The fruit can develop marks from yellow and brown dots to necrotic blotches on their surfaces. The symptoms are variable and can exhibit from very mild to severe, which can impede rapid identification of the disease in a greenhouse or field. Overall plant productivity can be impacted as well in the form of stunting via shortening of internodes and leaf narrowing and deformation. Fruit may fail to develop and can become unmarketable due to the associated symptoms.

Conditions for Disease Development

Many different crops can harbor this virus from season to season in addition to many weed species that may remain present in land adjacent to farming land. If infected materials are not disced under or greenhouses are not disinfected at the end of the season, the infected materials and equipment can serve as a source for new infections through human or insect actions. This long-term presence is possible due to the incredible stability of the viral particles within or outside of the plant cells. Any plant manipulation poses a risk to the grower — pollination, pruning, tying up, or machinery use can all

Necrotic lesions on capsicum fruit.

serve as a mechanical introduction of the virus into the plant. As it is mechanically transmitted, the infection could be due to incomplete hygiene measures like the presence of the virus on harvest crates or introduction of infected products by employees (for example in salads for lunch, etc.) or by visitors and other equipment.

The seed industry uses multiple techniques to test for presence of the virus and secure clean seeds for customers. Certified clean seed has been inspected and tested for presence of the virus with stringent testing thresholds, and customers should feel comfortable using these seed materials.

Control

Planting varieties with resistance to this disease and hygiene measures are the most effective means to combat the virus. For capsicum, resistance to PMMoV also provides resistance to ToBRFV. Cultural practices can be modified and bolstered to prevent additional spread of the disease. To sanitise equipment, growing materials and surfaces and reduce transmission incidence, apply nonfat dry milk (3.5% protein content when reconstituted) to the plants while transplanting and chemical disinfectants such as quaternary ammonia or bleach with an ideal 15 minutes of contact.

Causal Agent

Tomato spotted wilt virus (TSWV), *Peanut bud necrosis virus* (PBNV) (synonym: *Groundnut bud necrosis virus)*

Vector

Western flower thrips *(Frankliniella occidentalis)*, Tobacco thrips *(F. fusca)*, Melon thrips *(Thrips palmi)* and others

Distribution

TSWV: Worldwide; PBNV: Asia and Australia

Symptoms

These viruses are related and can be differentiated by vector, host range, serology and genetic analysis. Infection is particularly severe in seedlings that usually remain stunted and unproductive. Foliar symptoms include yellow or necrotic concentric ring patterns, and mosaic with chlorotic and necrotic spots. Necrotic streaks can develop on stems and extend to the top of the plant. In some cultivars, the apical shoot dies and leaves drop. In capsicum, reduced laminar growth can cause a shoestring appearance to leaves. Capsicum and eggplant fruit are deformed and often drop. Usually small chlorotic or discoloured spots develop that later turn necrotic. Fruit scarring and distinct concentric rings can also develop. In capsicum fruit, red, green and yellow discolouration is often present. Seeds inside affected fruit may turn black.

Conditions for Disease Development

These viruses and their vectors have a very wide host range. The presence of infected weeds or mature plants in adjacent fields can pose a threat to newly planted crops. Larvae acquire the virus after short feeding periods. Once infected, adult thrips transmit these viruses for the duration of their lives. Temperatures above 22° C accelerate the hatching of eggs, resulting in explosive spread of these vectors and viruses. Tospoviruses are not seed transmitted.

Control

Use virus-free transplants. Exclude thrips from plant nurseries. Monitor thrips populations during the growing season to determine when insecticide applications are necessary. Rogue infected plants and control weeds that serve as reservoirs for viruses and vectors in and around all crops.

Chlorotic spots on eggplant.

Concentric rings on eggplant.

Necrotic spots on capsicum foliage.

Ring spots on capsicum leaf.

Yellow concentric rings on capsicum leaf.

Deformed and discoloured chilli fruit.

Chlorotic spots on chilli fruit.

NONINFECTIOUS DISORDERS

BLOSSOM-END ROT

CHEMICAL DAMAGE

CHIMERA

CRACKING

NUTRIENT DISORDERS

SALT TOXICITY

STIP

SUNSCALD

Causal Agent

Imbalance of calcium in the fruit

Distribution

Worldwide

Symptoms

The first visible symptom of blossom-end rot is a water-soaked area near the blossom scar of the fruit. This area later develops into a tan to brown, leathery lesion. Saprophytic fungi often colonize these lesions, which gives them a grey to black, velvety appearance.

Conditions for Disease Development

Blossom-end rot is associated with insufficient calcium uptake and alternating periods of wet and dry soil. Though blossom-end rot often is associated with mature fruit, young, rapidly growing fruit are most prone to calcium deficiency. Sudden and extreme changes in water availability may induce fruit growth fluctuations that lead to blossom-end rot. Stress associated with root damage, mild drought, high soil salinity or excess nitrogen (excess ammonium) also may cause Blossom-end rot.

Control

Drip irrigate to supply an even amount of water, and apply lime to soils low in calcium. Avoid using ammonium sources of fertiliser or excess magnesium. Fertilise with calcium nitrate in areas where blossom-end rot is known to occur.

Fruit rot as a result of calcium imbalance.

Symptoms of blossom-end rot.

Saprophytic fungi often colonise lesions.

Lesions commonly develop near the blossom scar.

Colonisation of sunscald (left) and blossom-end rot (right) lesions by saprophytic fungi.

Fruit rot as a result of calcium imbalance.

Causal Agent

Herbicides and insecticides

Distribution

Worldwide

Symptoms

Symptoms of chemical damage differ depending on the dosage and plant growth stage at time of exposure. Contact herbicides, which affect only the plant tissues directly exposed, typically cause chlorotic or necrotic spots on leaves, stems and fruit, but might kill small seedlings. Systemic herbicides, which are translocated throughout the plant, cause a variety of symptoms that range from interveinal yellowing and vein necrosis to general foliar chlorosis. Necrotic spots, leaf margin necrosis, leaf twisting and cupping may also develop. Stems may be deformed, swell or crack. Fruit may be excessively large or small and irregular. Insecticide damage may show as leaf margin necrosis or necrotic lesions on the foliage.

Conditions for Disease Development

Chemicals labeled for use on capsicum or eggplant generally do not cause damage unless they are applied at excessive rates, at high temperatures, at the wrong stage of plant growth, or during unfavourable weather conditions. Some pre-plant herbicides may remain active in the soil long enough to cause injury to later plantings. Damage may occur from herbicide drift when adjacent crops or weeds have been sprayed. Usually, damage from drift is most severe at the edge of the field closest to the chemical application. Contaminated spray equipment can cause damage if the correct cleaning procedures are not followed after each chemical application.

Control

Always use herbicides and insecticides as directed by the label, and apply during appropriate weather conditions only. Always store herbicides and insecticides according to label recommendations. Avoid using herbicides and pesticides near running air intake vents for greenhouse spaces or on windy days wherein drift can move sprays large distances.

Bleaching caused by glyphosate.

2, 4-D damage on capsicum.

Mottling and distortion caused by pyrethrin.

Acephate damage on leaves.

Carbofuran phytotoxicity on capsicum.

Causal Agent

Genetic mutation

Distribution

Worldwide

Symptoms

Changes in colour or shape of leaves and fruits are associated with spontaneous genetic mutations. Typical symptoms of chimera are leaf variegation, absence of chlorophyll, filiform leaves and distorted growing points and fruit. Symptoms may be confused with those caused by viruses and herbicides.

Conditions for Disease Development

Chimeras are caused by genetic changes in the plant.

Control

Use the best quality seed available.

Genetic variegation.

Absence of chlorophyll.

Leaf and fruit distortion.

Causal Agent

Environmental, genetic

Distribution

Worldwide

Symptoms

Superficial cracks are characteristic of jalapeño peppers, but are considered problematic in other capsicum and chilli fruit types. Splitting of the epidermis occurs in fruit under stress, near maturity. Fine superficial cracks on the fruit's surface give a rough texture to the fruit, and deeper cracks are commonly colonised by secondary pathogens, which cause postharvest decay.

Conditions for Disease Development

The severity of fruit cracking is related to stress sustained by fruit during stages of rapid growth. In the field, wide differences in day and night temperatures or heavy rain or water availability promote cracking. During periods of rapid fruit expansion, high relative humidity at night in greenhouse production systems can also cause fruit cracking.

Control

Proper irrigation and nutrition management can help reduce cracking. In greenhouse operations, avoid high relative humidity and temperature fluctuations at night to reduce plant stress. Some cultivars are less susceptible to fruit cracking than others.

Surface cracks on jalapeño fruit.

Surface cracks on capsicum fruit.

Causal Agent

Insufficient or excessive nutrients

Distribution

Worldwide

Symptoms

Nitrogen (N): Plants under low N stress are smaller than normal and have an overall light-green colour, especially in the lower leaves. Fruits are small with thin walls. Excess nitrogen fertiliser can cause leaf and fruit burning, especially if applied as an ammonium formulation.

Phosphorus (P): Leaves on deficient plants are smaller than normal and dark-green. Older leaves are affected first and, in severe cases, may senesce.

Potassium (K): Symptoms of K deficiency begin on older leaves and progress to younger leaves. Foliage develops bronzing and/or burning of leaf margins and may develop chlorosis. Plants are smaller than normal and produce less fruit.

Calcium (Ca): Interveinal chlorosis and leaf margin necrosis occur at the growing points in Ca deficient plants. Later, growing points die. Leaves can be distorted. Fruit may develop blossom-end rot. Excess Ca can cause white spots below the surface of fruit. Open-pollinated capsicums may develop stip.

Magnesium (Mg): Magnesium deficient plants develop interveinal chlorosis on older leaves, which later progresses to young leaves. Interveinal tissue may become necrotic.

Sulfur (S): Older leaves of S deficient plants turn light-green and spindly.

Boron (B): When B is deficient, older leaves turn yellow and brittle, and the growing points become necrotic and die. Margins and leaf tips of mature leaves become necrotic. Fruit may also be affected and develop scattered corky areas and exposed ovaries.

Copper (Cu): Copper deficiency starts as a wilt of young leaves that later turn bluish-green and curl upwards. Severely affected plants are stunted and chlorotic.

Iron (Fe): Young leaves of Fe-deficient plants develop interveinal chlorosis, followed by a general yellowing. The leaf midrib usually remains green.

Manganese (Mn): Young leaves deficient in Mn develop interveinal chlorosis, followed by speckling or necrosis. Midribs of affected leaves remain green.

Zinc (Zn): Leaves deficient in Zn thicken and curl downward. Petioles may twist, and older leaves develop an orange-brown chlorosis.

Marginal burn due to excessive nitrogen.

Conditions for Disease Development

Nutrient deficiencies are most common in acid or alkaline soils due to immobilisation of nutrients. Low temperatures, soil compaction or excessive soil moisture may also affect nutrient availability. Nutrient disorders may also be caused by excessive or unbalanced use of fertiliser. Plant diseases that affect plant roots can induce nutrient deficiency symptoms due to reduced nutrient uptake.

Control

Conduct soil and foliar nutrient analyses regularly to verify nutritional needs, design a balanced fertiliser program and correct nutrient imbalances. Alter soil pH with the addition of lime to acid soils or sulfur and acid-forming fertilisers to alkaline soils to increase nutrient availability.

Interveinal chlorosis typical of magnesium deficiency.

Chlorotic nitrogen deficient capsicum seedlings (foreground).

Phosphorus deficient (left) and healthy (right) capsicum seedlings.

Nitrogen deficient eggplant seedlings (foreground).

Exposed ovaries typical of boron deficiency.

Phosphorus deficient (left) and healthy (right) eggplant seedlings.

Causal Agent

Excessive salts

Distribution

Worldwide

Symptoms

Capsicum species are sensitive to salt toxicity. Emergence after direct seeding may be slow and patchy. Seedlings are stunted. The growing point may desiccate and collapse to cause seedling death. Older leaves develop tip burn, marginal chlorosis and scorching. In severe cases, leaves wilt and drop. Roots may appear slightly discoloured, feeder roots may be poorly developed, and root tips are usually stubby and necrotic. Symptoms of salt toxicity are similar to symptoms caused by drought or fertiliser burn. Leaf tissue analysis may be necessary to confirm a diagnosis of salt toxicity.

Conditions for Disease Development

Excess concentration of salts in soil or irrigation water usually causes toxicity. Accumulation of salts in the root zone occurs as a consequence of alternating cycles of wetting and drying that move salts to the soil surface. Concentration of soluble salts in irrigation water varies with the source, season and annual rainfall. Drought and high evapotranspiration rates contribute to the development of salt toxicity.

Control

Test the salt content of soil and irrigation water prior to planting. Irrigation water should be tested periodically throughout the growing season. When excess soil salinity is suspected, use pre-plant amendments such as gypsum to lower pH and leach salt. Prevent accumulation of salt using the correct type, amount and placement of fertiliser. Salt accumulation patterns in planting beds vary according to the water delivery system used. Plant or transplant to avoid zones of salt accumulation. In greenhouses, leach accumulated salts with excess irrigation.

Phytotoxicity caused by saline irrigation water.

Capsicum are susceptible to high soil salinity.

Salt toxicity affects growth and appearance of roots and shoots.

Causal Agent

Physiological

Distribution

Worldwide

Symptoms

Grey to black spots develop when fruit are approximately 7.5 cm in diameter. Spots occur singly or in groups. As fruit mature, the spots turn green to grey-brown to yellow, which renders the fruit unmarketable. Stip can develop in all types of capsicums but is more common on mature, red fruit harvested in the autumn.

Conditions for Disease Development

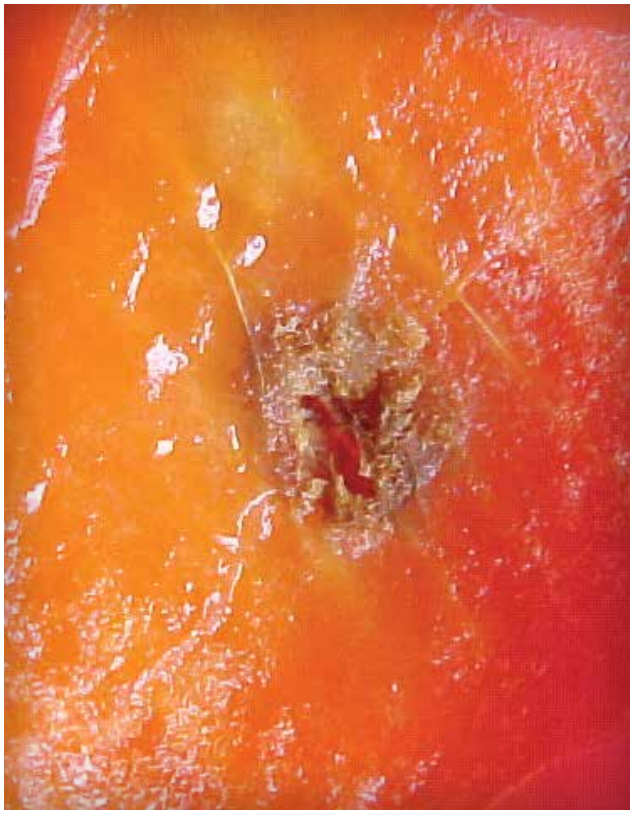
Stip is a physiological disorder believed to be caused by a calcium imbalance. It most commonly develops in older, open-pollinated varieties. Stip generally occurs during autumn months when days are short and cool. Stip is most common and severe in varieties grown on soils farmed with low calcium and/or high nitrogen and potassium fertilisation rates. Tissue analysis reveals that stip resistant varieties have higher calcium and lower nitrogen and potassium concentrations than affected varieties.

Control

Grow stip-resistant hybrids, and avoid open pollinated varieties in autumn. Calcium applications can significantly help reduce the occurrence and severity of stip.

Grey to black spots render fruit unmarketable.

Stip is more common on mature fruit harvested in autumn.



Discolouration extends below the surface.

Causal Agent

Environmental, genetic

Distribution

Worldwide

Symptoms

Sunscald occurs on the side of fruit exposed to direct sunlight. It first appears as a wrinkled area that can be soft and lighter in colour than surrounding tissue. In capsicums, this area later collapses and turns white and paper-like. The affected area often turns black due to colonisation by saprophytic fungi. Sunscald primarily affects fruit, but leaves and stems also can be injured. Fruit near maturity are more sensitive to sunscald injury than immature fruit. Symptoms are similar in appearance to those of blossom-end rot, but they are consistently associated with exposure to direct sunlight.

Conditions for Disease Development

Fruit suddenly exposed to direct sunlight due to defoliation from disease, pruning or stem breakage are most likely to develop sunscald. Sunscald occurs when internal fruit temperature increases and tissue is damaged.

Control

To prevent sunscald, the internal temperature of the fruit should not rise above 35° C. Encourage abundant, healthy foliage with proper fertilisation and irrigation. In greenhouse operations, shade plants during summer to help reduce the incidence of this disorder. Use disease-resistant varieties, and follow an effective disease and pest management program to help reduce losses due to sunscald.

Sudden direct exposure of capsicum to sunlight may cause sunscald.

Close-up of dry, papery, sunken lesion on capsicum fruit.

Tan-brown lesion on eggplant.

GLOSSARY

ANAMORPH The asexual form in the life cycle of a fungus. Asexual spores (conidia) are usually produced.

ANTHESIS The duration of life of a flower from the opening of the bud to the setting of the fruit.

ASCOSPORE Sexually derived fungal spore within a sack-like structure (ascus).

BACTERIUM (pl. bacteria) Microscopic, single-celled organism.

BLIGHT Sudden and severe necrosis of the above-ground portions of a plant.

CALYX The external green, leafy part of a flower consisting of sepals.

CANKER Localised, necrotic areas on roots or stems. Tissue may be sunken and/or cracked.

CHLOROSIS (chlorotic) The yellow or white discolouration of healthy green tissue.

COALESCE MERGING OF INDIVIDUAL LESIONS.

CONCENTRIC More than one circle in a lesion with a common centre.

CONIDIUM (pl. conidia) A fungal spore formed asexually.

COTYLEDON The first foliar structure to emerge from a seed.

DAMPING-OFF A rotting of seedlings at or below soil level.

DEBRIS Remnant plant material.

DEFOLIATION The loss of leaves.

DISTAL Located far from the point of attachment.

DIURNAL Occurring or active during the daytime.

EPICOTYL The part of the stem above the cotyledons.

EPIDERMIS The outer layer of cells occurring on plants.

FUMIGATION Sterilisation by chemical volatilisation.

FUNGICIDE A chemical used to control fungi.

FUNGUS (pl. fungi) A microscopic organism with thread-like cells that grows on living and/or dead plants.

GALL Swelling of roots, stems or leaves caused by abnormal growth of tissue.

GIRDLE The encircling of a root or stem by a pathogen that results in disruption of the phloem.

HAUSTORIUM (pl. haustoria) The penetrating feeding structure of fungi and parasitic plants.

HERBICIDE Chemical substance used to control weeds.

HOST A plant from which a parasite obtains nutrition.

HYDATHODE A leaf structure that eliminates unused salts, sugars and water from a plant through a pore at the leaf margin.

HYPERSENSITIVE A localised plant response that results in sudden death of the infected cells.

HYPOCOTYL The lower stem of a plant between the cotyledons and the roots.

IMMUNITY Not subject to attack or infection by a specified pest or pathogen.

INDICATOR A plant that produces specific symptoms to certain viruses or environmental factors and is used for their detection and identification.

INFECTION The process by which an organism attacks a plant.

INOCULUM The pathogen or its parts that can cause disease.

INSECTICIDE A substance used to control insects.

INSTAR An insect stage between molts before adulthood.

INTERMEDIATE RESISTANCE The ability of a plant variety to restrict the growth and development of the specified pest or pathogen, but may exhibit a greater range of symptoms compared to resistant varieties. Intermediate resistant plant varieties will still show less severe symptoms or damage than susceptible plant varieties when grown under similar environmental conditions and/or pest or pathogen pressure.

INTERVEINAL The area of leaf tissue bordered by veins.

LESION A well-defined, but localised, diseased area on a plant.

MICROSCLEROTIA Microscopic, dense aggregate of darkly pigmented thick-walled hyphal cells specialised for survival.

MOSAIC Variegated patterns of light and dark areas on a plant often caused by viruses.

MOTTLE Irregular light and dark areas on leaves or fruit surfaces symptomatic of viral diseases.

MYCELIUM (pl. mycelia) The mass of thin, microscopic, hair-like structures that forms the vegetative part of a fungus.

NECROTIC Dead and discoloured plant tissue.

NEMATODE Microscopic worms that can live in plants, animals, soil or water.

NYMPH Juvenile stage of an insect.

PASTEURISATION The process of partial sterilisation by heating at controlled temperatures to kill undesirable microorganisms.

PATHOGEN An organism or agent that is capable of causing disease.

PEDICEL The stalk of a flower or fruit.

PETIOLE The stalk of a leaf.

PHLOEM The food-conducting tissue of a plant.

PHYTOPLASMA A pleomorphic, obligate single-celled organism that lacks a cell wall. Formerly referred to as a Mycoplasma-like organism (MLO).

PITH Soft, spongy tissue in the centre of a plant stem.

PUSTULE A small blister-like elevation of the epidermis that forms as fungal spores develop and emerge.

PYCNIDIUM (pl. pycnidia) A spherical or flask-shaped asexual fruiting structure that gives rise to fungal conidia.

RACE A subspecific group of pathogens with distinct pathological or physiological properties.

RESERVOIR Infected plants that can serve as a source of inoculum for further infection of other plants.

RESISTANCE The ability of a plant variety to highly restrict the growth and development of a specified pest or pathogen, and/or the damage they cause when compared to susceptible plant varieties under similar environmental conditions and pest or pathogen pressure. Resistant varieties may exhibit some disease symptoms or damage under heavy pest or pathogen pressure.

ROOTSTOCK Portion of stem and associated root system onto which a bud or scion is inserted by grafting.

SAPROPHYTE An organism that lives on dead organic matter.

SATURATION Being completely filled with liquid, generally water.

SCLEROTIUM (pl. sclerotia) A compact mass of hyphae capable of surviving unfavourable environmental conditions.

SOLANACEOUS Plants in the nightshade family, including tobacco, tomatoes, potatoes, capsicums, eggplant and others.

SOLARISATION Exposure to direct sunlight to raise soil temperature to levels that kill pathogens.

SPORE A reproductive structure of fungi and some bacteria.

STOMATA A pore in a leaf surface.

STRAIN A general term referring to (a) an isolate; descendent of a pure culture of pathogen, (b) a race; one of a group of similar isolates or (c) one of a group of virus isolates that have common antigens.

SUSCEPTIBILITY The inability of a plant variety to restrict the growth and development of a specified pest or pathogen.

SYSTEMIC Spreading internally throughout a plant.

TELEOMORPH The sexual form of a fungus.

TOLERANCE The ability of a plant variety to endure abiotic stress without serious consequences for growth, appearance and yield.

TOXIN A compound produced by an organism that is injurious to plants.

TRANSLOCATION The transfer of nutrients or a virus through the plant.

TRANSPIRATION The loss of water vapor via stomata.

VARIEGATED Distinct areas, patches or spots of different colours.

VASCULAR The conductive system of a plant combining the xylem and phloem.

VECTOR An agent able to transmit a pathogen.

VEIN banding Pattern of light or dark green areas surrounding the veins often caused by viruses.

VIRULIFEROUS Containing or carrying a virus.

VIRUS A sub-microscopic obligate disease-causing agent.

WATER-SOAKED Tissue having the appearance of being soaked with water.

XYLEM The water-conducting tissue of a plant.

ZONATE Distinguished from adjacent parts by a distinctive feature (such as concentric rings).

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