TOMATO DISEASE

FIELD GUIDE

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PREFACE

This guide provides descriptions and photographs of the more common tomato diseases and disorders worldwide. For each disease and disorder the reader will find the common name, causal agent, distribution, symptoms, conditions for disease development and control measures. We have also included a section on common vectors of tomato viruses. New to this guide are several bacterial, virus and viroid descriptions as well as several tomato disorders. The photographs illustrate characteristic symptoms of the diseases and disorders included in this guide. It is important to note, however, that many factors can influence the appearance and severity of symptoms. Many of the photographs are new to this guide. We are grateful to the many academic and private industry individuals who contributed photographs for this guide.

The primary audience for this guide includes tomato crop producers, agricultural advisors, private consultants, farm managers, agronomists, food processors, and members of the chemical and vegetable seed industries. This guide should be used as a reference for information about common diseases and disorders as well as their control. However, diagnosis of these diseases and disorders using only this guide is not recommended nor 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 tomato crops. Even the most experienced plant pathologist relies upon laboratory and greenhouse techniques to confirm a plant disease and/or disease disorder diagnosis. Moreover, this guide is by no means inclusive of every tomato disease. Rather, we present those diseases that are currently most prevalent worldwide in protected culture and open field productions.

Always read and follow label directions for any herbicide, fungicide, insecticide or any other chemical used for treatment or control.

A glossary of words used in the text can be found at the end of this guide along with a list of references for additional information on the diseases and disorders addressed within this publication.

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BACTERIAL CANKER

Leaf firing from secondary spread of *Clavibacter michiganensis* subsp. *michiganensis.*

Severe outbreak of bacterial canker inside a plastic tunnel.

CAUSAL AGENT

Clavibacter michiganensis subsp. *michiganensis*

DISTRIBUTION

Worldwide

SYMPTOMS

Symptoms may appear on cotyledons or young leaves as small cream-colored spots, expanding to 1-2 mm in diameter, but this is not common. Young plants may wilt and die, depending on the initial level of infection. As infected plants mature and set fruit, wilting can occur on one side of a leaflet or to leaflets on one side of a leaf. This symptom is known as unilateral wilting and is very characteristic of bacterial canker. Plants may initially wilt temporarily during the warmest part of the day and then recover under cooler conditions. Leaves may exhibit greenish-gray regions of collapsed tissue, a symptom more commonly observed in protected culture. Secondary infection manifests as necrosis beginning at the margins of leaves and spreading inward, known as "firing." Adventitious roots may develop. As the disease progresses, yellow to brown cankers occasionally form longitudinally on stems and petioles, hence the name of the disease. Yellow bacterial exudate can sometimes be squeezed from the cut end of an infected stem. Longitudinal sectioning of the stem reveals yellow to reddish-brown to dark-brown vascular discoloration; pith tissue may later deteriorate. Vascular tissue extending from the peduncle into the fruit can have a yellow-brown discoloration, particularly in protected culture. Secondary fruit symptoms in the field begin as small, white spots which expand and become light-brown and raised in the center, surrounded by a white halo. These are referred to as "bird's-eye" spots and can reach 3-6 mm in diameter. In protected culture, fruit can exhibit a netted appearance.

CONDITIONS FOR DISEASE DEVELOPMENT

Infection generally occurs through wounds in plant tissue, but can also occur through stomata, hydathodes or roots. This bacterium has been shown to survive for up to five years associated with plant debris and can also survive on weeds, volunteer tomato plants and seed. Secondary spread occurs from splashing water, contaminated equipment, tools and activities in which plants are handled (e.g., grafting, pruning, cultivation, harvesting). Moderate temperatures (18-24°C) and greater than 80% relative humidity favor disease development. Soil moisture conditions optimal for plant growth, as well as low light intensity and high nutrient levels (especially nitrogen), also enhance disease development. Symptoms tend to be more severe in sandy soils.

CONTROL

Sow only seed that has been tested and certified free of *Clavibacter michiganensis* subsp. *michiganensis*. In seedling production, use only steam-sterilized growing media and hard plastic trays, which can be cleaned and sanitized more effectively than Styrofoam. Inspect seedlings regularly for bacterial canker symptoms.

Avoid planting tomatoes in fields where *C. michiganensis* subsp. *michiganensis* has been detected within the past five years or where tomatoes or other solanaceous crops (e.g., pepper or eggplant) have been grown within the past three years. Use new stakes and twine. Sanitize equipment between fields. Overhead irrigation should be avoided; drip irrigation is preferred. Avoid activity in the field when plants are wet. At the end of the season, crop debris should be thoroughly plowed under.

In protected culture, implement a comprehensive sanitation program for equipment, tools and personnel. Manage the greenhouse environment to avoid dew formation on plants. Remove pruning debris. Do not interplant; instead, thoroughly clean and disinfect production facilities between crops. Do not reuse growing media.

BACTERIAL CANKER

Marginal chlorosis and necrosis of a leaflet.

Interveinal leaflet chlorosis and necrosis on a greenhouse-grown plant.

Interveinal leaflet necrosis on a greenhouse-grown plant.

BACTERIAL CANKER

Atypical marginal chlorosis and necrosis of leaflet.

Unilateral wilting.

Bacterial exudate from stem. (Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

Stem canker on processing tomato. (Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

Initiation of adventitious roots.

Internal stem necrosis.

BACTERIAL CANKER

Stem cross-section showing internal decay and fruit with “bird’s-eye” spots.

“Bird’s-eye” spots on green fruit.

“Bird’s-eye” spots on red fruit.

Longitudinal cross-section of immature green fruit showing vascular discoloration.

Longitudinal cross-section of ripe red fruit showing vascular discoloration.

BACTERIAL SPECK

Dark-brown lesions with chlorotic halos.

CAUSAL AGENT

Pseudomonas syringae pv. *tomato* (Two races, 0 and 1, have been identified.)

DISTRIBUTION

Race 0 - Worldwide
Race 1- Bulgaria, Canada, Italy, Serbia and United States

SYMPTOMS

This bacterium can attack the leaves, stems, petioles, flowers and fruit. Foliar symptoms appear as dark-brown to black spots, often surrounded by a yellow halo. These lesions can be circular to angular in shape and may have a greasy appearance. Lesions can also form on leaf margins where guttation droplets collect. Large areas of leaf tissue become necrotic as lesions coalesce. Oval to elongated black lesions may occur on stems and petioles. Generally, fruit lesions are superficial and remain small (1 mm) and speck-like, but occasionally can be larger and sunken.

CONDITIONS FOR DISEASE DEVELOPMENT

Disease development is favored by cool to moderate temperatures (13-25°C) and rainfall or sprinkler irrigation. Usually one day of continuous leaf wetness is required for disease to develop. Transplant operations provide a conducive environment for bacterial speck due to high plant densities and overhead watering. Under these conditions, symptoms may express in the transplant operation or remain undetected until after transplanting, where rain or overhead irrigation promote disease development and spread. This bacterium can survive on many crops and weeds. While an outbreak can originate from infested seed, seed transmission is usually of minor importance.

CONTROL

Plant race 0-resistant varieties that carry the *Pto* gene to effectively control bacterial speck where race 0 is present. There is currently no race 1 resistance deployed in commercial germplasm. Transplant operations should implement a comprehensive sanitation and bactericide spray program. Copper sprays applied early as protectants can reduce disease incidence and severity in transplant operations and in the field. Avoid sprinkler irrigation; furrow or drip irrigation should be used when possible. Control weeds and volunteer tomatoes in and around fields and incorporate debris after harvest.

Leaflet with typical speck lesions.

Bacterial speck on processing tomato. (Courtesy of Eugene Miyao University of California Division of Agriculture and Natural Resources)

BACTERIAL SPECK

Circular, concave lesions on abaxial leaflet surface. (Courtesy of Enrico Biondi, Department of Agricultural Sciences, University of Bologna)

Coalescing petiole and leaflet lesions.

Lesions on green fruit.

Lesions on ripe red fruit.

BACTERIAL SPOT

Foliar spots and chlorosis.

Spots on upper and lower leaflet surfaces.

CAUSAL AGENTS

Xanthomonas euvesicatoria (synonym = *Xanthomonas campestris* pv. *vesicatoria* race 1)

Xanthomonas vesicatoria (synonym = *Xanthomonas campestris* pv. *vesicatoria* race 2)

Xanthomonas perforans (synonym = *Xanthomonas campestris* pv. *vesicatoria* races 3 & 4)

Xanthomonas gardneri

DISTRIBUTION

Worldwide

SYMPTOMS

Foliar symptoms appear as dark, water-soaked, circular spots less than 3 millimeters in diameter. These spots become angular and the surfaces may appear greasy, with translucent centers and black margins. The centers of these lesions soon dry and crack, and yellow halos may surround the lesions. During periods of high moisture (heavy rain, fog or dew) leaves will turn chlorotic and may eventually become blighted. Disease can develop on all above-ground parts of the plant, with lesions tending to be more numerous on young foliage. Fruit infection begins as small, black, raised spots which may be surrounded by a white, greasy-appearing halo. Fruit lesions typically enlarge to four to five millimeters in diameter and can be dark-brown with raised margins and sunken, tan centers or tan with raised margins and sunken, dark-brown centers. Fruit lesions often appear scab-like or corky.

CONDITIONS FOR DISEASE DEVELOPMENT

These bacteria can survive in crop debris, on volunteer plants, weeds and seed. Overhead irrigation can result in rapid spread of this disease in transplant operations. In the field, infection generally occurs through wounds, such as those made by insects, wind-driven sand and rain, and by high-pressure spraying. Warm temperatures (24-30°C) combined with heavy rains or sprinkler irrigation favor disease development.

CONTROL

Sow only seed that has been tested and certified free of these bacteria and ensure that transplants are disease-free. Copper sprays can provide moderate levels of protection, although copper-resistant strains have become more common. Avoid overhead irrigation. Rotate to non-host crops and control weeds and volunteer plants. Good sanitation practices, including cleaning of equipment and plowing under all plant debris immediately after harvest, can reduce losses from this disease. It is valuable to know which race of bacterial spot predominates in an area, as resistant tomato varieties may be available.

BACTERIAL SPOT

Necrotic spots with surrounding chlorosis. (Courtesy of Maja Ignatov, Institute of Field and Vegetable Crops)

Necrotic spots coalescing on abaxial surface of leaflet.

Rachis lesions.

BACTERIAL SPOT

Rachis lesions.

Calyx lesions.

Healthy fruit (left); infected fruit (right).

BACTERIAL SPOT

Lesions on green fruit. (Courtesy of Maja Ignatov, Institute of Field and Vegetable Crops)

Lesions on ripe red fruit.

BACTERIAL STEM ROT

Brown bacterial stem rot lesion.

Bacterial stem rot affecting main stem, side branch and petioles.

CAUSAL AGENTS

Pectobacterium carotovorum subsp. *carotovorum*

Pectobacterium carotovorum subsp. *brasiliensis*

DISTRIBUTION

Pectobacterium carotovorum subsp. *carotovorum:* Worldwide

Pectobacterium carotovorum subsp. *brasiliensis:* Brazil, China, Columbia, Israel, The Netherlands, Poland, South Africa, South Korea, USA (Florida)

SYMPTOMS

Bacterial stem rot manifests as wilting in plants with mature green fruit ready for harvest. Cross section cuts through the stem usually reveal that pith tissue has rotted away and is no longer present. Instead, the stem is hollow. The surface of the stem is slimy from the soft rot bacteria that destroyed the pith. Bacterial stem rot can also manifest as dark-brown to black lesions on stem surfaces. These lesions can quickly elongate along stems. Later, the epidermis and vascular tissues slough off to reveal a hollow stem.

CONDITIONS FOR DISEASE DEVELOPMENT

Pectobacterium carotovorum is a ubiquitous bacterium that has a wide host range among vegetables. *Pectobacterium carotovorum* can survive for long periods in soil, plant debris and water. Both field-grown and greenhouse-grown tomatoes can develop bacterial stem rot. Tomatoes grown on trellises or in protected culture are managed very differently than processing tomatoes or fresh-market tomatoes that are not trellised or staked. The cultivation of trellis-grown tomatoes or of tomatoes grown in protected culture requires activities (e.g., pruning, tying and staking plants) that can create wounds that allow *P. carotovorum* to infect and spread within the crop. Dispersal of *P. carotovorum* occurs via splashing rain, irrigation water, tools, equipment, workers and insects. In protected culture facilities, *P. carotovorum* was shown to survive under roof gutters where it was splashed onto plants via contaminated water. Growing conditions in protected culture that favor bacterial stem rot include high relative humidity and free moisture on plants.

CONTROL

Implement a comprehensive sanitation program for workers, tools, equipment and facilities for both greenhouse and field-grown tomatoes. Avoid working with plants when foliage is wet, and remove infected plants, pruning debris and crop residues. In protected culture operations, provide adequate air circulation to reduce relative humidity.

BACTERIAL STEM ROT

Cross-section through a diseased stem. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Brown bacterial stem rot lesion. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

Fruit infected with *Pectobacterium carotovorum* subsp. *carotovorum.* (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Internal soft rot of pith and vascular tissues. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

BACTERIAL WILT

Yield reduction can approach 100% when disease pressure is high.

CAUSAL AGENTS

Ralstonia pseudosolanacearum (synonym = *Ralstonia solanacearum* phylotype I and III strains)

Ralstonia solanacearum (synonym = *Ralstonia solanacearum* phylotype II strains)

Ralstonia syzygii subsp. *indonesiensis* (synonym = *Ralstonia solanacearum* phylotype IV strains from tomato)

DISTRIBUTION

Ralstonia pseudosolanacearum: Africa, Asia

Ralstonia solanacearum: Caribbean, Central America, North America

Ralstonia syzygii subsp. *indonesiensis:* Indonesia

SYMPTOMS

Onset of disease begins with drooping of lower leaves followed soon after by wilting of entire plants. No foliar yellowing is associated with this wilt disease. A longitudinal section of stem reveals yellow to light-brown vascular discoloration which may later turn dark brown or hollow the stem completely as disease progresses. A quick and simple diagnostic method for these *Ralstonia* species is to place a freshly cut stem in a glass of water. A white, milky stream of bacteria will ooze from the stem.

CONDITIONS FOR DISEASE DEVELOPMENT

The *Ralstonia solanacearum* species complex has a host range of more than 200 plant species that it can infect and survive within. These pathogens can also survive in soil where they infect roots through natural wounds caused by the formation of secondary roots, or through wounds caused by transplanting, cultivation practices or nematode feeding. Chewing insects may also transmit these *Ralstonia* species. These bacteria can be spread by rain water or surface irrigation, in soil on cultivation equipment and via diseased transplants. Warm (29-35°C) or hot (36-40°C) weather and high soil moisture favor development of bacterial wilt.

CONTROL

When possible, avoid land with a history of bacterial wilt. Ensure transplants are disease-free and grow hybrid varieties with resistance to bacterial wilt or graft onto resistant rootstocks. Transplant onto raised beds, manage soil-moisture content including drainage of water away from roots, and control weeds that may serve as asymptomatic hosts of these bacteria to reduce incidence and severity of bacterial wilt. Soil fumigation or solarization, and cover-cropping or crop rotation to a non-host species can reduce incidence of bacterial wilt in some situations.

Fresh market tomato field with high incidence of bacterial wilt.

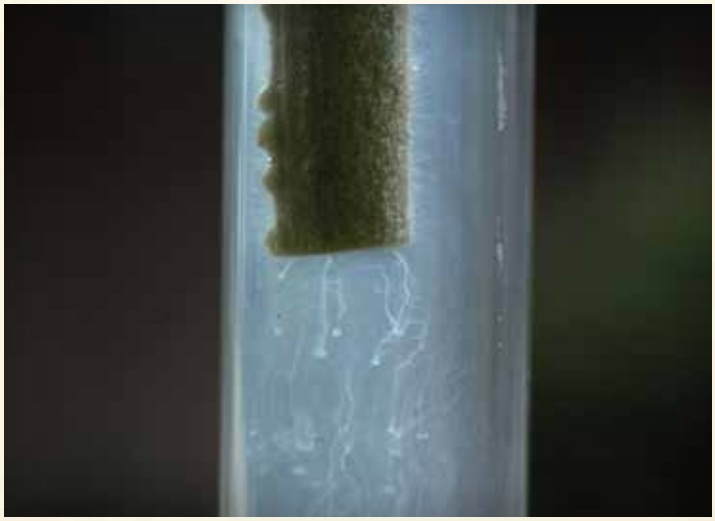
BACTERIAL WILT

Young plant wilting from infection by *Ralstonia pseudosolanacearum.*

Vascular discoloration of stem caused by *Ralstonia solanacearum.*

Cross-section of stem showing discoloration and pith decay.

Vascular discoloration of excised stem pieces.



Ralstonia solanacearum cells streaming from stem piece placed in water.

BACTERIAL
PITH NECROSIS

Chlorotic, wilting leaves and stem lesions.

CAUSAL AGENTS

Pseudomonas cichorii

Pseudomonas corrugata

Pseudomonas fluorescens

Pseudomonas mediterranea

Pseudomonas viridiflava

DISTRIBUTION

Worldwide

SYMPTOMS

Foliar symptoms begin as yellowing and wilting of younger leaves in the upper portions of the plant. As the disease advances, dark-brown or black lesions form on stems, which can cause wilting and may lead to plant death if the lower stem is affected. A longitudinal section of a stem lesion may reveal dark-brown discoloration of pith and vascular tissues. Stems may contain hollow or segmented cavities. Adventitious roots often develop along stems where pith tissue is affected.

CONDITIONS FOR DISEASE DEVELOPMENT

Though pith necrosis occurs in field-grown tomatoes, it is of much greater importance in protected culture. Disease outbreaks have been associated with cool night and warm day temperatures, high humidity and excessive nitrogen fertilization. The onset of symptoms often occurs when fruit reach the mature green stage, especially if plants are too vegetative. These bacteria survive variably as pathogens on alternate hosts, in tomato debris in soil and as epiphytes on host and non-host species. Spread of these bacteria occurs via splashing rain and irrigation water, on contaminated pruning tools and equipment, and on workers' hands during harvest.

CONTROL

Implement a comprehensive hygiene program for workers and sanitize pruning tools and other equipment to minimize disease spread. Avoid working with plants when they are wet as workers can easily spread bacteria to adjacent plants. In protected culture productions, avoid practices that favor increased vegetative growth such as excess nitrogen and high humidity.

Longitudinal section of stem lesion reveals dark brown discoloration. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

BACTERIAL
PITH NECROSIS

Adventitious roots developing from stem lesion. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Longitudinal section of stem lesion reveals discolored vascular and pith tissues. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Longitudinal section showing pith necrosis and hollowing.

PSYLLID YELLOWS AND VEIN-GREENING

Psyllid yellows on foliage. (Courtesy of Judy Brown, University of Arizona)

CAUSAL AGENT OF PSYLLID YELLOWS

Bactericera cockerelli (tomato psyllid) (synonym = *Paratrioza cockerelli)*

CAUSAL AGENT OF VEIN-GREENING

'*Candidatus* Liberibacter solanacearum'
(synonym = '*Candidatus* Liberibacter psyllarurous')

VECTOR DISTRIBUTION

Central America, New Zealand, North America

PATHOGEN DISTRIBUTION

Central America, Europe, New Zealand, North America

SYMPTOMS

Psyllid yellows are induced by toxins excreted when tomato psyllids feed on plant tissue. Interveinal foliar chlorosis is the primary symptom associated with psyllid yellows. Yellowing of leaves appears first on older leaves but may also be expressed in younger leaves if the psyllid population is not controlled. Foliar chlorosis may be paired with vein-clearing and thickening, curled leaves, older leaves with a leathery texture, younger leaves with a purple hue, and stunting. Symptom severity can be reduced by controlling the psyllid vector.

Vein-greening occurs when tomato psyllids transmit the obligate bacterial pathogen, '*Candidatus* Liberibacter solanacearum.' Vein-greening symptoms may also be accompanied by typical symptoms of psyllid yellows. Flower and fruit production may be affected, with an increase in flower production but an overall decrease in the number of flowers that set fruit. Of the fruit that are produced, most are discolored, deformed, smaller than normal, and have a reduced sugar content. As the infection progresses, symptoms may escalate to foliar necrosis, widespread wilting, and death of the plant.

CONDITIONS FOR DISEASE DEVELOPMENT

The tomato psyllid grows and reproduces at warm temperatures (24-26°C is optimal). An increased psyllid population can be correlated to increased symptoms of psyllid yellows. If the psyllids are transmitting '*Ca.* L. solanacearum,' it can be expected that symptoms of vein-greening will also be more prevalent at warm temperatures due to increased proliferation of the vector.

CONTROL

The primary control measure for psyllid yellows and vein-greening is management of the psyllid vector. Monitor the presence of the vector using yellow sticky traps to ensure the timely application of insecticides. Insecticides that are active against psyllids at all life stages are the most effective in eliminating the vector and psyllid yellows symptoms. Predatory insects may also be utilized as a source of biological control for the psyllid vector, although their effectiveness may be limited. Predators of psyllids include a parasitic wasp (*Tamarixia triozae*), lady beetles, lacewings and damsel bugs.

Eliminating the psyllid vector following transmission of the pathogen, '*Ca.* L. solanacearum', will not alleviate symptoms of vein-greening; however, insecticide treatments will help minimize spread from infected to healthy plants. Once tomato plants are infected with the bacterium they cannot be cured. In some greenhouse environments, severity of vein-greening symptoms can be lessened with application of micronutrient solutions. Other cultural practices, including crop rotation and roguing of alternate hosts and solanaceous weeds, can help minimize available vector habitat.

PSYLLID YELLOWS AND VEIN-GREENING

Psyllid yellows on new growth. (Courtesy of Judy Brown, University of Arizona)

Vein-greening of foliage. (Courtesy of Judy Brown, University of Arizona)

Tomato psyllid, *Bactericera cockerelli* (synonym = *Paratrioza cockerelli*). (Courtesy of Whitney Cranshaw, Colorado State University, Bugwood.org)

BACTERIAL
SYRINGAE LEAF SPOT

Leaf lesions without chlorosis.

CAUSAL AGENT
Pseudomonas syringae pv. *syringae*

DISTRIBUTION
Worldwide

SYMPTOMS
Symptoms caused by *Pseudomonas syringae* pv. *syringae* can be easily confused with symptoms caused by *P. syringae* pv. *tomato*. Symptoms on leaves may vary from brown lesions that lack a chlorotic halo to dark-brown or black lesions with surrounding bright-yellow halos. Syringae leaf spot lesions can be larger than those of bacterial speck; however, it is necessary to isolate the causal agent in pure culture and conduct laboratory tests to determine etiology.

CONDITIONS FOR DISEASE DEVELOPMENT
This bacterium survives on both host and non-host plants as an epiphyte and can spread from these plants when cool, wet environmental conditions favor disease development. Although economic damage from this disease is often limited, infection at the seedling stage can be severe under ideal environmental conditions. Wounding is often associated with infection although this bacterium can also enter via natural openings in leaves (e.g., stomata, hydathodes). *Pseudomonas syringae* pv. *syringae* is a weak, opportunistic pathogen on tomato, and overall disease development may be less than that caused by *P. syringae* pv. *tomato.*

CONTROL
Control may be obtained with copper-based spray applications, though this is usually not necessary since economic damage from *P. syringae* pv. *syringae* is not common. Nurseries should implement a comprehensive hygiene program and apply bactericides as protectants rather than in response to symptoms developing on seedlings. Avoid low temperature and high humidity conditions in nurseries. Inspect seedlings for symptoms before transplanting to avoid introducing disease into the field. Abstain from using overhead irrigation whenever possible. If a bacterial disease is suspected, samples should be submitted to a reputable laboratory to determine if symptoms are caused by *P. syringae* pv. *syringae* and not by another bacterial pathogen that might require more stringent control.

Foliar lesions with chlorosis.

TOMATO ROOT MAT

Root proliferation in a grow cube.

Root proliferation in several grow cubes.

SYNONYM = "CRAZY ROOTS"

CAUSAL AGENT

Agrobacterium radiobacter biovar 1

DISTRIBUTION

Europe, Japan, New Zealand, North America, Russia

SYMPTOMS

The primary symptom is dense proliferation of roots against the inside surface of artificial substrate cubes and slabs. The swelling of the substrate cube is quite noticeable. Roots of affected plants lack branching and are thicker than healthy tomato roots. Affected plants produce additional vegetative growth, which can lead to poor fruit set and smaller fruit. Affected plants may also be more sensitive to other pathogens (e.g., *Pythium* spp.).

CONDITIONS FOR DISEASE DEVELOPMENT

Strains of *Agrobacterium radiobacter* biovar 1 can harbor a root-inducing (Ri) plasmid, pRi. Infection of tomato by *A. radiobacter* occurs through root wounds. Root mat symptoms develop following insertion of T-DNA of pRi into tomato root cells. Once present in a propagation house or greenhouse, *A. radiobacter* is very difficult to eradicate despite aggressive hygiene programs and use of new substrate each crop cycle due to its ability to survive as biofilms on surfaces of production facilities. *Agrobacterium radiobacter* has been found colonizing the rhizosphere of several weed species which grow in and around protected culture facilities. Moreover, *A. radiobacter* can survive in soils for many years (>15 years). Sources of infection have been attributed to contaminated water silos, drain water, irrigation lines and drip emitters.

CONTROL

There is no registered chemical treatment that can be applied to tomato plants during cultivation. Implement a strict hygiene program to limit the persistence of *A. radiobacter* within nursery and production houses to keep tomato root mat under control. Thoroughly clean and sanitize production facilities between tomato crops. Control weeds in areas surrounding nurseries and production facilities. Treat recirculated water systems with UV light. Never re-use artificial substrate (e.g., rock wool, coco peat). Suppress *A. radiobacter* by lowering irrigation water to pH 5.0. Many horticultural disinfectants have activity against *A. radiobacter.* Products with a sodium hypochlorite (5 ppm) or hydrogen peroxide (15 ppm) base are especially effective.

Root proliferation in a coco peat slab.

Root proliferation is primarily on the outer surface of the grow slab.

De Ruiter™ Seminis®

FUNGAL AND OOMYCETE DISEASES

ALTERNARIA STEM CANKER

ANTHRACNOSE

BUCKEYE FRUIT AND ROOT ROT

CERCOSPORA LEAF MOLD

CORKY ROOT ROT

DAMPING-OFF

DIDYMELLA STEM ROT

EARLY BLIGHT

FRUIT ROTS

FUSARIUM CROWN AND ROOT ROT

FUSARIUM FOOT ROT

FUSARIUM WILT

GRAY LEAF SPOT

GRAY MOLD

LATE BLIGHT

LEAF MOLD

PHOMA ROT

POWDERY MILDEW

SEPTORIA LEAF SPOT

SOUTHERN BLIGHT

TARGET SPOT

VERTICILLIUM WILT

WHITE MOLD

ALTERNARIA STEM CANKER

Toxin-induced interveinal chlorosis and necrosis.

CAUSAL AGENT

Alternaria alternata f. sp. *lycopersici*

DISTRIBUTION

USA (California)

SYMPTOMS

Symptoms may occur on all above-ground parts of tomato plants. Dark-brown cankers that are often associated with wound sites form on stems. These cankers develop concentric rings as they enlarge. Dry, brown stem rots develop with brown streaks extending into pith tissue above and below cankers. A toxin produced by *Alternaria alternata* f. sp. *lycopersici* moves from the stem cankers into upper parts of affected plants, killing interveinal leaf tissue. As this disease progresses, curling of leaf margins and then leaf senescence occurs. Often, stem cankers eventually girdle stems, which leads to plant death. Fruit symptoms initially appear as small, gray flecks which later enlarge and become dark and sunken with characteristic concentric rings. Symptoms may not be present on mature green fruit; however, symptoms can develop rapidly with ripening in transit.

CONDITIONS FOR DISEASE DEVELOPMENT

Alternaria alternata f. sp. *lycopersici* can survive in soil and crop debris for more than one year. Stem wounds created by pruning allow entry of this fungus into plants; however, infection also occurs without wounds. Rain, overhead irrigation and dews favor development of Alternaria stem canker. Conidia that sporulate from cankers are easily spread by wind to initiate infection on additional plants.

CONTROL

Use of resistant varieties effectively controls this disease. Fungicides have not shown good efficacy against *A. alternata* f. sp. *lycopersici.*

Black-bordered stem lesions.

ALTERNARIA STEM CANKER

Stem canker with longitudinal cracking. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Stem lesion with cracking. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Sunken fruit lesions with black borders.

ANTHRACNOSE

CAUSAL AGENTS

Colletotrichum coccodes

Colletotrichum dematium

Colletotrichum gloeosporioides

DISTRIBUTION

Worldwide

SYMPTOMS

Infection of fruit, stems, leaves and roots may occur, with fruit and root infections being most serious. Although green fruit are readily infected, symptoms do not appear until fruit ripen. Initial lesions are sunken, circular and develop concentric rings as they expand. Lesion centers turn tan where many dark microsclerotia develop within lesions. During wet weather, numerous conidia form in slimy, salmon-colored and gelatin-like masses on lesion surfaces. Infected roots develop brown lesions with microsclerotia developing on root surfaces. *Colletotrichum coccodes* is most commonly associated with fruit symptoms and is the only *Colletotrichum* species associated with black dot root rot. Root infection is commonly associated with another common soilborne disease of tomato called corky root rot, which is caused by *Pyrenochaeta lycopersici.* Leaf infection is rarely a problem and is characterized by small, brown, circular lesions surrounded by yellow halos.

CONDITIONS FOR DISEASE DEVELOPMENT

These fungi are generally considered weak pathogens; however, they have a wide host range (more than 65 plant species) and can survive in soil on decaying plant tissue for several years. Free moisture and temperatures between 10 and 30°C favor host infection. The longer fruit surfaces remain wet from rainfall or overhead irrigation, the greater the disease severity. Conidia and microsclerotia infect host tissue directly in contact with infested soil or they are disseminated to host tissue by splashing rain and overhead irrigation. These fungi penetrate host tissue directly or enter through wounds. Root infection generally occurs when fungal inoculum levels are high and plants are nutritionally stressed due to unfavorable growing conditions or infection from another pathogen, especially *P. lycopersici.*

CONTROL

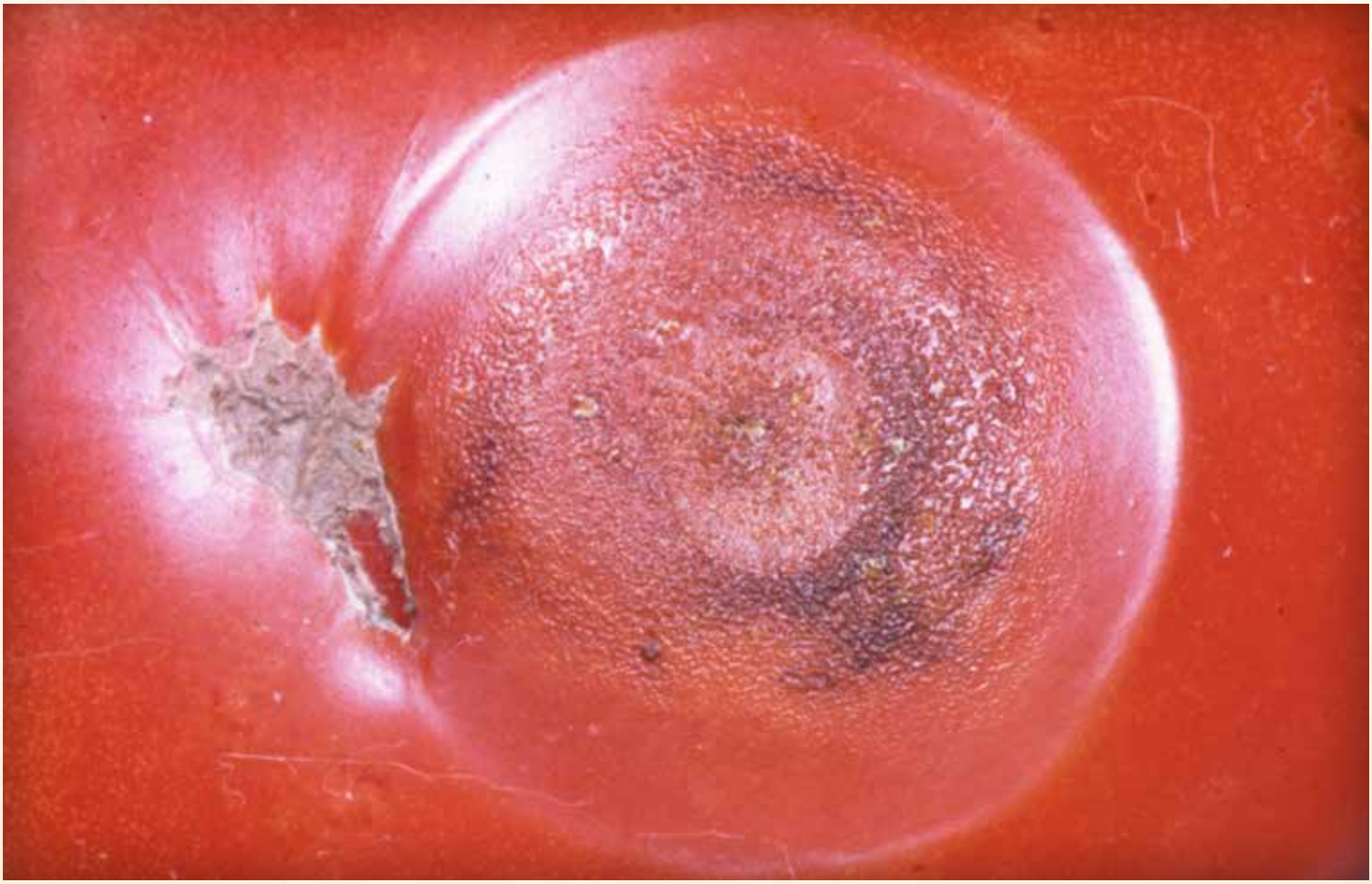
Initiate a fungicide spray program at the first green fruit stage and continue until harvest to help control this disease. To further reduce losses from these fungi choose fields that drain well, avoid root injury during cultivation, remove weeds and infected debris from fields and apply a broad spectrum fumigant in the offseason. Rotate to non-host crops for two to three years to prevent buildup of inoculum in soil.

Sunken, circular fruit lesions.

ANTHRACNOSE

Sunken, circular lesions on fruit. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Black microsclerotia on the surface of an infected root.



Fruit lesion with concentric rings developing. (Courtesy of Clemson University – U.S. Department of Agriculture Cooperative Extension Slide Series, Bugwood.org)

BUCKEYE FRUIT AND ROOT ROT

Phytophthora capsici-infected plants in a greenhouse. (Courtesy of Flavia Ruiz, Erieview Inc.)

Low area in field affected by Buckeye fruit and root rot.

CAUSAL AGENTS

Phytophthora nicotianae var. *parasitica*

Phytophthora capsici

Phytophthora drechsleri

DISTRIBUTION

Worldwide

SYMPTOMS

The *Phytophthora* species that cause buckeye fruit and root rot can infect all parts of tomato plants. They can cause seedling damping-off, root and crown rot, foliar blight and fruit rot. Root rot symptoms include large, brown, sunken, and water-soaked lesions on secondary roots and the tap root that can extend above the soil line onto the stem. As disease progresses, smaller roots collapse and decay. A longitudinal section through the tap root reveals a chocolate-brown discoloration of the vascular system that extends a short distance beyond the lesion. Severely infected plants eventually wilt and die. Infected leaves initially develop water-soaked, irregular-shaped lesions that collapse quickly and dry. Stem lesions can develop at any level on stems but are typically found near the soil line. Stem lesions are dark-green and water-soaked at first, and eventually become dry and brown. As stem lesions expand they can completely girdle stems, causing pith tissue to turn brown and collapse. Fruit symptoms start as grayish-brown, water-soaked lesions that expand rapidly, forming brown concentric rings that resemble a buckeye nut—hence the name. Brown discoloration can extend into fruit centers with young green fruit becoming mummified, while mature fruit quickly rot from invasion by secondary organisms.

CONDITIONS FOR DISEASE DEVELOPMENT

These *Phytophthora* species have a relatively wide host range and can survive in soil and infested plant debris for at least two years. They can be spread by irrigation water and farm equipment. Initial infection is favored by moderate soil moisture levels and temperatures (20°C). Excessive irrigation or rain, in combination with heavy or compacted soils, favors further disease development.

CONTROL

Use fungicides in combination with cultural practices to manage buckeye fruit and root rot. Plant on raised beds to promote water drainage away from plant roots, avoid compacting field soil and fields with poorly drained soil, and irrigate for shorter lengths of time to avoid extended periods of soil saturation. Mulch or stake plants to raise fruit off soil. Follow a three-year rotation to non-host crops.

BUCKEYE FRUIT AND ROOT ROT

Stem and root lesions showing vascular discoloration.

Root lesion with vascular discoloration.

Lower stem lesion caused by *Phytophthora capsici* on a greenhouse-grown plant. (Courtesy of Flavia Ruiz, Erieview Inc.)

Buckeye fruit rot symptoms on fresh market tomato.

Brown concentric rings typical of buckeye fruit rot.

CERCOSPORA LEAF MOLD

SYNONYM = BLACK LEAF MOLD

CAUSAL AGENT

Pseudocercospora fuligena (synonym = *Cercospora fuligena*)

DISTRIBUTION

Worldwide

SYMPTOMS

The first symptom to appear is a yellowish discoloration on upper leaf surfaces that later expands to form brownish lesions surrounded by yellow halos. When humidity is high, gray to blackish-gray conidia develop on lower leaf surfaces. This disease is sometimes referred to as "black leaf mold" because of this dark fungal sporulation. When Cercospora leaf mold is severe, lesions coalesce resulting in collapse of leaf tissue. Due to similarity of symptoms, Cercospora leaf mold can be confused with leaf mold caused by *Passalora fulva.*

CONDITIONS FOR DISEASE DEVELOPMENT

Pseudocercospora fuligena can survive on infested plant debris and on alternate solanaceous hosts like black nightshade. High humidity and warm (27°C) temperatures favor disease development and sporulation. Wind, splashing water from overhead irrigation and rain, as well as workers' clothing, tools and cultivation equipment, readily disseminate conidia of this pathogen.

CONTROL

Use a calendar-based protectant fungicide spray program and resistant varieties to reduce losses from Cercospora leaf mold. Prune and provide adequate plant spacing to encourage air movement within plant canopies, and mulch and furrow or drip irrigate to reduce spread of this pathogen from splashing water. Turn under or remove all plant debris and rotate to non-host crops to lower field inoculum levels.

Extensive brown-tan foliar lesions.

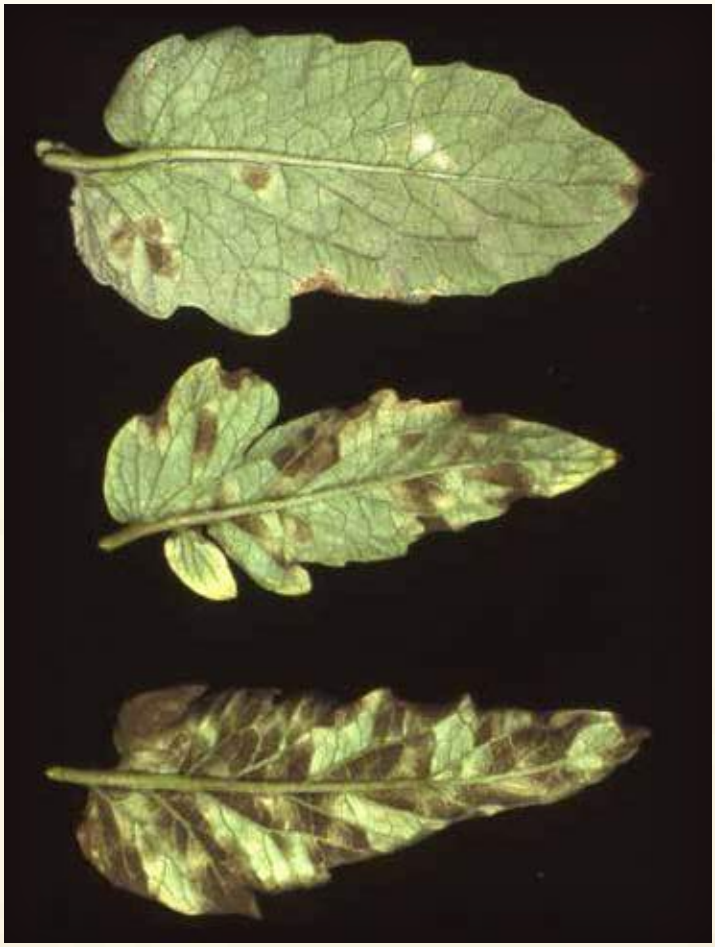
Chlorotic lesions on upper leaf surface. (Courtesy of Dan Egel, SW Purdue Agricultural Program)

CERCOSPORA LEAF MOLD

Lesions can form without causing chlorosis. (Courtesy of Zelalem Mersha, Lincoln University Cooperative Extension)

Sporulating lesion on leaf undersurface. (Courtesy of Zelalem Mersha, Lincoln University Cooperative Extension)

Blackish-gray sporulation on the lower leaf surface.



Light (top) to heavy (bottom) leaf sporulation.

CORKY ROOT ROT

Coalesced root lesions on root.

CAUSAL AGENT

Pyrenochaeta lycopersici

DISTRIBUTION

Worldwide

SYMPTOMS

Infected plants may be stunted and generally lack vigor. As disease progresses, plants may show diurnal wilting and develop leaf chlorosis and premature defoliation. First symptoms on small roots are smooth, elliptical, light-brown lesions. This stage of the disease is frequently called brown root rot. As disease progresses, primary and secondary roots are infected and develop large brown lesions that swell and crack longitudinally, giving them a corky appearance. Feeder roots may be decayed completely and no longer present. The tap root and stem base eventually turn brown and rot.

CONDITIONS FOR DISEASE DEVELOPMENT

This fungus is pathogenic on several agricultural crops, and its microsclerotia can survive in soil and tomato root debris for several years. Disease is most severe under cool (15-20°C) soil temperatures and relatively high soil moisture; however, strains from warmer climates were found to be pathogenic at soil temperatures between 26 and 30°C. Spread of *Pyrenochaeta lycopersici* occurs with cultivation and on contaminated farm equipment.

CONTROL

Soil fumigation is the most effective means of controlling this disease. Grafting onto corky root rot-resistant rootstocks has been used effectively for greenhouse tomato production.

Extensive lesion development on infected roots.

CORKY ROOT ROT

Root lesion with superficial cracking.

Swelling and cracking of an infected root.

DAMPING-OFF

Damping-off caused by *Phytophthora* sp.

Seedling wilting from infection by *Phytophthora capsici.*
(Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

CAUSAL AGENTS

Pythium species

Phytophthora species

Rhizoctonia solani (teleomorph: *Thanatephorus cucumeris*)

Botrytis cinerea

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 plants wilt and collapse.

***Pythium* species and *Phytophthora* species:** Symptoms of pre-emergence damping-off include dark-brown to black water-soaked lesions that rapidly spread over the entire seedling. Post-emergence damping-off is characterized by brown water-soaked lesions that first appear on the roots and spread up the hypocotyl above the soil line, eventually girdling the hypocotyl and causing the seedling to wilt and die.

Rhizoctonia solani: Pre-emergence damping-off is characterized by tan to reddish-brown lesions developing on the hypocotyl and by death of the growing tip. Symptoms of post-emergence damping-off include reddish-brown to black lesions developing on roots and the hypocotyl at or below the soil line. Later the hypocotyl constricts and the infected seedling quickly wilts and dies. *Rhizoctonia solani* can also infect older plants; however, as plants mature they become more tolerant to infection, and lesions are usually restricted to cortical tissue.

Botrytis cinerea: Post-emergence damping-off can occur in the nursery as cotyledons senesce. It is characterized by beige to brown discoloration of the hypocotyl. If not properly controlled early, presence of *Botrytis cinerea* can lead to additional problems on all parts of the tomato plant during the vegetative stage.

CONDITIONS FOR DISEASE DEVELOPMENT

These fungi are capable of surviving for long periods of time in soil and may persist in plant debris or on roots of weeds. Damping-off tends to be most severe under conditions of high soil moisture, overcrowding, compaction, poor ventilation and cool, damp, cloudy weather. In greenhouses, damping-off can be problematic when soil is not pasteurized completely or when seedling trays are inadequately sanitized. Water splashing can move infested soil from diseased to healthy seedlings. The water source can be a potential cause for introducing these damping-off pathogens into a protected or open-field environment.

CONTROL

Improving drainage and moisture regulation to prevent soil saturation reduces damping-off. Fungicidal soil drenches and seed treatments are available to help manage damping-off. Rotation to cereal crops and soil fumigation or solarization may reduce damping-off in the field. Use pasteurized soil mixes in nurseries.

DAMPING-OFF

Girdling of hypocotyl caused by *Pythium* sp.

Damping-off caused by *Pythium* sp.

Girdling of epicotyl caused by *Pythium* sp.

Girdling of stem caused by *Pythium* sp.

DIDYMELLA STEM ROT

Stem canker with numerous black pycnidia. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

CAUSAL AGENT

Didymella lycopersici (anamorph: *Phoma lycopersici)*

DISTRIBUTION

Africa, Australia, Canada, Europe, Great Britain, India, New Zealand

SYMPTOMS

Infection usually occurs on stems at or above the soil line; however, all foliar parts of tomato plants can be affected. Dark-brown, sunken lesions form on the stem at the base of the plant. These lesions eventually expand and girdle the stem, which results in yellowing and wilting of older leaves. As wilting progresses, the plant may eventually die. Numerous black pycnidia, which is the asexual fruiting structure of this fungus, frequently form in stem lesions. Splashing water spreads conidia formed within pycnidia to stems, leaves and fruit, resulting in additional infections and disease spread. Leaf infection begins as small spots which develop into brown lesions with concentric rings. Pycnidia may develop in the center of these lesions with leaves eventually taking on a shot-hole appearance or dying. Fruit infection typically occurs at the calyx end and appears as a water-soaked lesion that progresses rapidly to form a sunken black lesion with concentric rings.

CONDITIONS FOR DISEASE DEVELOPMENT

Didymella lycopersici can survive in soil, in infected plant debris and seed, as well as on nightshade and other solanaceous hosts. Didymella stem rot occurs over a wide range of conditions; however, a temperature of 20°C accompanied by splashing water from rain or overhead irrigation is optimal for disease development and spread. Plants become more susceptible as they mature, and nitrogen and phosphorus deficiency can exacerbate disease severity.

CONTROL

Fungicide spray programs can be effective if properly applied in a timely manner. Practice good sanitation including removal of all infected plant debris and alternate hosts. A three-year crop rotation between tomato crops can reduce losses from this disease. Avoid overhead irrigation and provide adequate ventilation when growing plants in a greenhouse.

DIDYMELLA STEM ROT

Didymella stem rot often starts at the stem base. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

EARLY BLIGHT

Mature plant with severe infestation of early blight.

CAUSAL AGENTS

Alternaria tomatophila

Alternaria solani

DISTRIBUTION

Worldwide

SYMPTOMS

Symptoms may develop on leaves, stems and fruit and typically appear first on older leaves as irregular, dark-brown, necrotic lesions. These lesions expand as disease progresses and they eventually develop concentric, black rings, which give early blight lesions a target-board appearance. A chlorotic area often surrounds leaf lesions. If there are numerous lesions on a leaf, then the entire leaf will turn yellow and senesce. Complete defoliation of plants can occur when conditions are favorable for disease development. Lesions may appear as dark-brown, elongated, sunken areas on stems and petioles. Lesion development at the soil line can result in collar rot that may girdle stems. Fruit lesions often occur at the calyx end and are dark, leathery and sunken.

CONDITIONS FOR DISEASE DEVELOPMENT

Alternaria tomatophila and *A. solani* generally survive from season to season on plant debris in the soil. Volunteer tomatoes, potatoes and solanaceous weeds can also serve as inoculum sources. Infection and sporulation occur during periods of warm (24-29°C), humid or rainy weather. Conidia are disseminated from sporulating lesions by wind and rain. Early blight spreads rapidly when favorable conditions persist. This disease can also be serious in arid climates when dew periods are frequent or when the crop is sprinkler-irrigated.

CONTROL

A fungicide spray program combined with an early blight forecasting system is the most effective means of controlling this disease. Use field sanitation techniques such as crop rotation and weed control, and turn under or remove debris from previous crops to reduce disease severity.

Circular, coalescing early blight lesions. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Early blight lesions surrounded by chlorotic halos.

EARLY BLIGHT

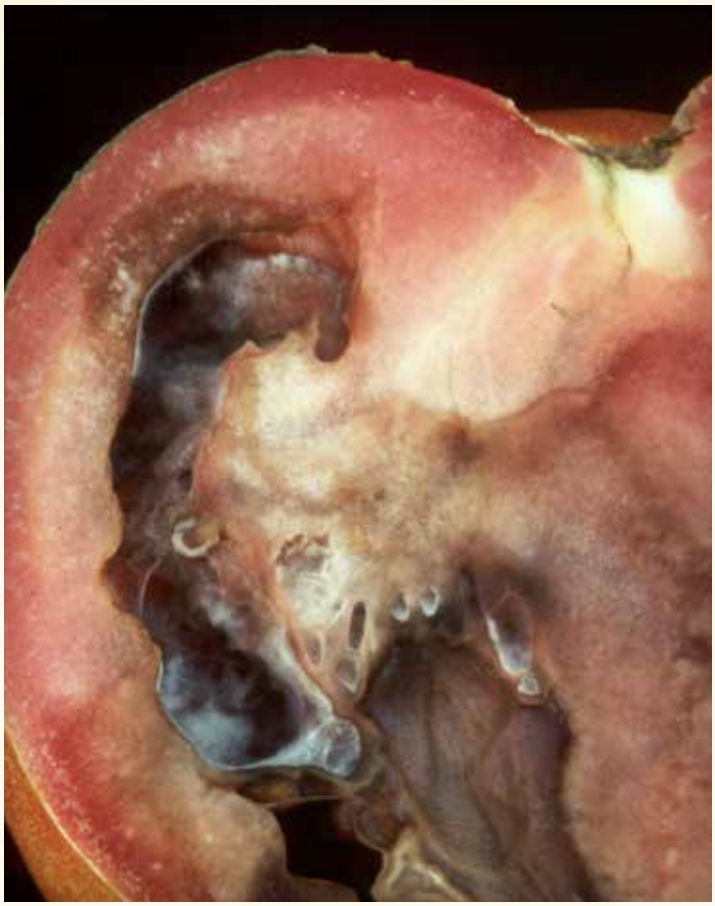
Circular and angular lesions with chlorotic halos.

Elliptical stem lesions caused by *Alternaria solani.* (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Stem lesions with target-board appearance. (Courtesy of Rebecca Melanson, Mississippi State University Extension, Bugwood.org)

Dark, sunken lesion on the calyx end of a fruit.

FRUIT ROTS



Internal fruit infection from *Alternaria alternata.*

CAUSAL AGENTS

Alternaria alternata, Botrytis cinerea, Dickeya chrysanthemi, Geotrichum candidum, G. penicillatum, Pectobacterium carotovorum, Pleospora lycopersici, Pythium aphanidermatum, P. ultimum, Rhizoctonia solani, Rhizopus stolonifer, Stemphylium botryosum, S. consortiale

DISTRIBUTION

Worldwide

CONDITIONS FOR DISEASE DEVELOPMENT

Both bacteria and fungi can enter through natural openings like stem scars or through growth cracks and wounds caused by insect feeding and mechanical damage. In general, warm (25-30°C) temperatures and high humidity provide adequate conditions for bacteria and fungi to infect fruit. However, gray mold rot can be problematic under cool to moderate (18-23°C) temperatures and wet conditions.

CONTROL

Cultural methods which keep fruit from contacting the soil help prevent many of the fruit rots. Improve air movement in the field by orienting rows in the direction of prevailing winds, and use proper row and plant spacing to shorten the length of time that fruit remain wet. Schedule sprinkler irrigation to minimize the length of time that free water remains on fruit. Fungicides may offer some protection against some fruit rots. Tomato varieties which produce firm fruit and those that are resistant to fruit cracking may escape infection by some fruit rots.

Avoid fruit injury during harvest and packing. Field and packinghouse sanitation is effective at reducing losses due to fruit rots. Sanitize all harvest equipment, the packing line and packing boxes daily. Maintain a minimum available chlorine concentration of 150 ppm at pH 6.0-7.5 in dump tank and packing line wash water. Cull infected and injured fruit during packing. Dry wet fruit surfaces before packing and cool fruit quickly to 10ºC.

Alternaria alternata-infected fruit.

FUNGAL AND OOMYCETE
FRUIT ROTS

DISEASE	ORGANISM	SYMPTOMS
Bacterial soft rot	*Pectobacterium carotovorum, Dickeya chrysanthemi*	A light- to dark-colored, sunken lesion is usually the first symptom to develop. As the lesion expands, it takes on a soft rot appearance. Bacteria may ooze from cracks in the fruit epidermis.
Black mold rot	*Alternaria alternata, Pleospora lycopersici, Stemphylium botryosum, S. consortiale*	Symptoms can range from superficial flecking to brown-black, dry, sunken lesions which may extend into fruit cavities. V-shaped lesions often develop on the stem scar and with adequate humidity, a dense layer of black fungal conidia develops on lesion surfaces.
Gray mold rot	*Botrytis cinerea*	Lesions begin as gray-green to gray-brown, water-soaked spots. They usually form on the calyx end of fruit and quickly expand to form gray-brown sporulating lesions which later develop into a watery rot.
Pythium fruit rot	*Pythium aphanidermatum, P. ultimum*	Lesions begin as water-soaked spots on both green and ripe fruit. Lesions quickly expand, engulfing entire fruit and giving fruit a water balloon appearance. When humidity is high, a white cottony growth covers lesion surfaces. When the epidermis is ruptured, fruit quickly collapse.
Rhizoctonia fruit rot	*Rhizoctonia solani*	Lesions begin as a firm rot which later becomes soft. Rhizoctonia fruit rot usually develops on ripe fruit that contact the soil. It is often characterized by rings that form in the infected area. A brown fungal growth frequently develops on the surface of infected fruit.
Rhizopus rot	*Rhizopus stolonifer*	Lesions develop rapidly and have a puffy, water-soaked appearance. White mycelium interspersed with fungal spore masses borne on raised stalks form on the surface of lesions. Rhizopus rot tends to have a fermented odor compared to the putrid odor of bacterial soft rot and sour rot.
Sour rot	*Geotrichum candidum, G. penicillatum*	Sour rot can develop on mature green fruit as well as ripe fruit. This rot usually begins at the stem scar and may extend in sectors down the side of fruit. A dull white fungal growth often develops from cracks in the skin. Fruit often remain firm until the rot is advanced and then a sour odor develops. Under favorable conditions infection can proceed rapidly on ripe fruit.

Fruit with water-soaked lesion caused by a *Pythium* sp.

Fruit infected with *Rhizoctonia solani.*

FRUIT ROTS

Fruit with lesions caused by *Rhizoctonia solani.*

Lesions with rings caused by *Rhizoctonia solani* infection.

FRUIT ROTS

Rhizopus stolonifer sporulating from infected fruit.

Sour rot caused by *Geotrichum candidum.* (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Geotrichum candidum-infected fruit. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

FUSARIUM CROWN AND ROOT ROT

Chlorosis and wilting of foliage. (Courtesy of Flavia Ruiz, Erieview Acres, Inc.)

Chlorosis and wilting of foliage. (Courtesy of Flavia Ruiz, Erieview Acres, Inc.)

CAUSAL AGENT

Fusarium oxysporum f. sp. *radicis-lycopersici*

DISTRIBUTION

Worldwide

SYMPTOMS

Typically, first symptoms are observed when plants are at the mature-green fruit stage. Symptoms include a yellowing of the oldest leaves that gradually progresses to the youngest leaves. When Fusarium crown and root rot is severe, plants may wilt quickly and die; however, diurnal wilting during hot, sunny days is more common. As disease progresses the entire root system of affected plants turns brown and the tap root often rots away. Chocolate-brown lesions develop at or near the soil line and extend into the vascular system. This brown vascular discoloration typically does not extend more than 25 cm above the soil line, which helps to distinguish this disease from Fusarium wilt. When humidity is adequate, fungal sporulation may be observed on exposed lesions.

CONDITIONS FOR DISEASE DEVELOPMENT

Fusarium oxysporum f. sp. *radicis-lycopersici* can survive in the soil, on infected plants and on the roots of alternative hosts (e.g., eggplant, pepper and several legume crops) for several years. Fungal conidia can be spread in soil by farm machinery, in irrigation water and through the air. Infection takes place through feeder roots and wounds caused by secondary root formation. Disease development is favored by cool (20°C) soil temperatures. In protected environments, infection is often observed at the graft union of the scion and rootstock or at sites where pruning has occurred.

CONTROL

The use of varieties and rootstocks resistant to Fusarium crown and root rot is the most effective way to control this disease. Implement a thorough hygiene program for workers, tools and equipment to mitigate disease severity and reduce the spread of this pathogen in protected culture. Practices such as inter-planting and year-round production can have a negative effect on controlling *F. oxysporum* f. sp. *radicis-lycopersici* as growers are unable to properly sanitize their facilities. Soil fumigation does not appear to be an effective control measure due to rapid colonization of sterilized soil by the fungus. Biologicals (e.g., *Trichoderma*) applied to artificial substrates in protected environments may have some impact on reducing the spread of this pathogen.

FUSARIUM CROWN AND ROOT ROT

Stem lesion at graft union. (Courtesy of Gillian Ferguson, Ontario Ministry of Agriculture, Food and Rural Affairs, Ontario, Canada)

Crown and root infection.

FUSARIUM CROWN AND ROOT ROT

Scion with lesion.

Vascular discoloration of crown and secondary root decay.

FUSARIUM CROWN AND ROOT ROT

Vascular discoloration.

Cross-section through crown showing vascular discoloration and pith decay.

FUSARIUM FOOT ROT

Leaflet with interveinal chlorosis and necrosis. (Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

CAUSAL AGENT

Fusarium solani f. sp. *eumartii* (teleomorph: *Haematonectria haematococca*)

DISTRIBUTION

Australia, India, Israel, Ivory Coast, Turkey, USA

SYMPTOMS

Fusarium foot rot manifests on mature plants as leaf interveinal chlorosis and necrosis. In severe cases, leaves turn brown and collapse, and the entire plant may die. Reddish, dark-brown lesions form on the taproot and main lateral roots up to 30 cm below the soil line. Internal vascular discoloration may extend two to ten centimeters beyond lesions.

CONDITIONS FOR DISEASE DEVELOPMENT

Fusarium solani f. sp. *eumartii* can survive in soil for two to three years. Cooler temperatures favor disease development, even though this fungus can grow well at a soil temperature of 27°C. Infection occurs through root wounds. Recently, both *F. solani* f. sp. *eumartii* and *F. oxysporum* f. sp. *radicis-lycopersici* have become important diseases in protected culture.

CONTROL

Use fungicides, fumigate or solarize soil, and rotate to a non-host crop for four years to reduce losses from Fusarium foot rot. Today, there are no resistance claims among tomato varieties and rootstocks against *F. solani* f. sp. *eumartii.*

Crown lesion with vascular discoloration.

FUSARIUM FOOT ROT

Stem, crown and taproot vascular discoloration.

Taproot vascular discoloration.

Lesion on taproot.

FUSARIUM WILT

Yellowing of oldest leaves.

CAUSAL AGENT

Fusarium oxysporum f. sp. *lycopersici*
Three races have been described.

DISTRIBUTION

Worldwide

SYMPTOMS

Infected seedlings are stunted and the oldest leaves and cotyledons turn yellow and wilt. Severely infected seedlings frequently die. On older plants symptoms begin with a yellowing of the oldest leaves at fruit set. Symptoms are often characterized by a yellowing on only one side of a leaf or branch. Later entire branches turn yellow producing a "yellow flag" appearance in the field. Affected leaves wilt and die, though they remain attached to the stem. Affected plants are often stunted and may display diurnal wilting during sunny days. A characteristic red-brown discoloration of vascular tissue develops in affected plants and can be seen if the main stem is cut longitudinally or when a branch is snapped off the main stem. Vascular discoloration may extend throughout the main stem and side branches or even further.

CONDITIONS FOR DISEASE DEVELOPMENT

Fusarium oxysporum f. sp. *lycopersici* can survive in soil for several years and is spread by farm machinery during cultivation. This fungus can also be spread in infected plant debris and in irrigation water. Infection takes place through root wounds caused by cultivation, secondary root formation and nematode feeding. Disease develops quickly when soil temperatures are warm (28°C). Fusarium wilt is enhanced by high levels of micronutrients, and phosphorous and ammonia nitrogen.

CONTROL

Today three races of *F. oxysporum* f. sp. *lycopersici* are recognized and they are designated as race 1, 2 and 3 in the United States and as race 0, 1 and 2 in Europe. The use of resistant varieties is the most effective way to control Fusarium wilt. Thoroughly disinfect equipment before moving from infested to clean fields.

Field infected with *Fusarium oxysporum* f. sp. *lycopersici* race 3 (= R-2 in Europe).

Fusarium-resistant (left) and susceptible (right) varieties.

FUSARIUM WILT

One-sided yellowing and wilting.

Vascular discoloration.

Vascular discoloration.

Discolored vascular bundles at leaf axil.

GRAY LEAF SPOT

Seedling infected in nursery at the cotyledon stage.

CAUSAL AGENTS

Stemphylium botryosum f. sp. *lycopersici*

Stemphylium lycopersici

Stemphylium solani

DISTRIBUTION

Worldwide

SYMPTOMS

Leaf lesions appear as small, brownish-black specks. These lesions develop into grayish-brown, glazed, angular lesions approximately three millimeters in diameter. They are often surrounded by chlorosis. The lesions eventually dry and tissue in the center of the lesions cracks. When numerous lesions develop on leaves, entire leaves turn chlorotic and drop, which may lead to defoliation. Tomato fruit and stems are not affected by these fungi.

CONDITIONS FOR DISEASE DEVELOPMENT

These fungi can survive in soil and on plant debris from one year to the next. In addition, volunteer tomato plants, as well as other solanaceous crops and weeds, can serve as inoculum sources. Infected transplants may also be an important inoculum source. Conidia sporulate from infected leaves and are spread by wind and splashing water. Warm and humid or wet weather are favorable for disease development. Gray leaf spot can also develop in arid regions when dew periods are long or if the crop is sprinkler irrigated.

CONTROL

Widespread use of resistant varieties has reduced the importance of this disease. Use fungicides to control gray leaf spot if the variety being grown is susceptible to this disease. Remove plant debris and provide adequate ventilation in seedling beds.

Lesions with dark borders and tan centers. (Courtesy of Clemson University USDA Cooperative Extension slide series, Bugwood.org)

GRAY LEAF SPOT

Lesions on abaxial leaflet surface.

Tan to brown lesions with yellow halos.

Petiole with small, circular lesions.

GRAY MOLD

Necrotic v-shaped lesion with sporulation.

CAUSAL AGENT

Botrytis cinerea (teleomorph: *Botryotinia fuckeliana*)

DISTRIBUTION

Worldwide

SYMPTOMS

Botrytis cinerea can infect all above ground parts of tomato plants and typically enters through wounds. On stems, initial infections appear as elliptical, water-soaked lesions. These lesions develop under high humidity into a gray, moldy growth, which can girdle and kill plants. Stem lesions often show concentric banding. Leaf infections usually begin at points of injury and develop into v-shaped lesions that are covered with gray fungal sporulation. *Botrytis cinerea* commonly infects the calyx end of fruit where it can spread rapidly, forming gray-brown sporulating lesions that later develop into a watery rot. Ghost spot, an unusual fruit symptom, is characterized by small, white to pale-yellow or green rings that develop on green or red fruit. Ghost spot rings result when *B. cinerea* infects fruit but further disease development is halted when fruit are exposed to direct sunlight and high temperatures. Ghost spots reduce market quality.

CONDITIONS FOR DISEASE DEVELOPMENT

Botrytis cinerea has a wide host range. It is an efficient saprophyte and can survive in soil and infected plant debris for long periods in the form of sclerotia. It is considered a weak parasite and typically infects plant tissues through wounds. When humidity is adequate, gray masses of fungal spores are produced, which are readily wind-disseminated. Overcast, cool and humid conditions are required for disease development. Close plant spacing and poor ventilation can lead to severe gray mold problems.

CONTROL

Reduce losses from this disease with an appropriate fungicide spray program. Provide adequate ventilation to plants by pruning and subsequently applying fungicide to pruning wounds. Implement a strong sanitation program that includes timely removal of pruning debris from greenhouse and field-grown fresh market tomato production systems.

Stem lesions.

Water-soaked fruit with sporulation from anthers that are still attached at the blossom end.

GRAY MOLD

Fruit, calyx and pedicel with gray-brown sporulation.

Sporulation on calyx and fruit.

Fruit cluster infected with *Botrytis cinerea.* Note ghost spot on the ripening fruit and sporulation on an infected petiole.

Ghost spot rings.

LATE BLIGHT

Whitish-gray sporulation on abaxial leaf surface.

CAUSAL AGENT

Phytophthora infestans

DISTRIBUTION

Worldwide

SYMPTOMS

The first symptom of late blight is a bending down of petioles of infected leaves. Leaf and stem lesions manifest as large, irregular, greenish, water-soaked patches. These patches enlarge and turn brown and paper-like. During wet weather, *Phytophthora infestans* will grow and sporulate from lesions on abaxial leaf surfaces. Rapid blighting of foliage may occur during moist, warm periods. Entire fields can develop extensive foliar and fruit damage. Fruit lesions manifest as large, firm, irregular, brownish-green blotches. Surfaces of fruit lesions are rough and greasy in appearance.

CONDITIONS FOR DISEASE DEVELOPMENT

Phytophthora infestans can survive on volunteer and home garden tomatoes and potatoes, in potato cull piles and on solanaceous weeds, from which it produces sporangia that can be carried long distances by storms. Cool, wet weather favors development of this disease. Under these conditions disease progresses rapidly and can destroy a mature tomato field in a few days. Tomatoes grown in protected culture environments (greenhouse, high tunnel) are also subject to late blight infection. Disease development may progress even more rapidly in protected culture compared to the open field. *Phytophthora infestans* requires only high humidity to infect, whereas many other diseases additionally require leaf wetness for infection to occur.

CONTROL

Implement a late blight forecasting system in conjunction with an effective spray program to control late blight. Avoid planting on land previously cropped to potatoes or near a potato field because *P. infestans* is also a pathogen of potato. In protected culture, maintaining lower humidity will discourage infection and disease development.

Blighted foliage.

Expanding lesion on abaxial leaf surface.

LATE BLIGHT

Stem and petiole lesions.

Stem lesions.

Phytopthora infestans infection on a greenhouse tomato.

White sporulation on infected fruit. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Rough, brown fruit lesions.

Cross-sections of infected red and green fruit.

LEAF MOLD

***Passalora fulva*-infected foliage.** (Courtesy of Rebecca A. Melanson, Mississippi State University Extension, Bugwood.org)

CAUSAL AGENT

Passalora fulva (synonym = *Fulvia fulva; Cladosporium fulvum*)

Five races have been described.

DISTRIBUTION

Worldwide

SYMPTOMS

Passalora fulva usually infects leaves; however, stems, blossoms, and fruit may also be infected. Symptoms first appear as light-green to yellowish areas on the adaxial surface of older leaves. This coincides with development of masses of olive-green conidia on the abaxial surface of leaves. As disease progresses, lower leaves turn yellow and drop. A black, leathery rot may develop on the calyx end of infected fruit. Although this disease occurs in the field, it is mainly a problem in greenhouses where it spreads rapidly under favorable conditions.

CONDITIONS FOR DISEASE DEVELOPMENT

Passalora fulva is an efficient saprophyte and can survive as conidia and sclerotia in soil and plant debris for at least one year. Conidia are readily dispersed by wind and rain. Dissemination can also occur on workers' clothing and equipment. High (90%) relative humidity and moderate (24°C) temperatures are optimal for disease development; however, disease can occur between 10 and 32°C. Leaf mold will not develop if relative humidity is less than 85%.

CONTROL

Implement a fungicide spray program. In greenhouses, provide adequate air movement and heating to reduce relative humidity to less than 85%. Grow resistant varieties when possible; however, the extreme diversity of this fungus often makes it difficult to grow a variety resistant to all races of *P. fulva* present in a region.

Chlorosis on upper leaf surface and sporulation on lower leaf surface.

LEAF MOLD

Sporulation on upper leaf surface.

Sporulation on lower leaf surface.

Sporulating lesion.

PHOMA ROT

Necrotic lesions with and without chlorosis. (Courtesy of Don Ferrin, Louisiana State University Agricultural Center, Bugwood.org)

CAUSAL AGENT

Phoma destructiva var. *destructiva*

DISTRIBUTION

Africa, Bolivia, Great Britain, India, Italy, The Netherlands, Pacific Islands, Russia, USA

SYMPTOMS

Phoma rot can affect all above-ground parts of tomato plants. On leaves, this disease manifests as numerous small, dark-brown to black spots. Later, the spots may develop concentric rings as they enlarge. Older leaves are often infected first; however, all leaves are susceptible and defoliation can result when disease is severe. The leaf spot caused by *Phoma destructiva* var. *destructiva* looks like that caused by *Alternaria tomatophila,* except that the leaf spot caused by *P. destructiva* var. *destructiva* contains numerous minute, black fruiting bodies (pycnidia). Elongated, dark-brown lesions with concentric rings develop on stems. Fruit lesions usually develop at the calyx end and appear as small, sunken lesions that later develop into sunken, black, leathery lesions with numerous pycnidia in the centers of the lesions. Both green and ripe fruit can be infected.

CONDITIONS FOR DISEASE DEVELOPMENT

Phoma destructiva var. *destructiva* survives from one season to the next in infected tomato and pepper debris. Injuries to plants — such as pruning wounds and other mechanical damage, insect feeding, and stem or fruit cracks — provide a means of entry for *P. destructiva* var. *destructiva.* During periods of high moisture from rain, fog or dew, or overhead irrigation, masses of conidia exude from pycnidia. Conidia are readily spread by rain, overhead irrigation and on workers' clothing and equipment. Long distance spread occurs with seed and movement of transplant seedlings. Low soil nitrogen and phosphorus levels may contribute to plant susceptibility.

CONTROL

Couple a calendar-based fungicide spray program with good sanitation practices to reduce losses from Phoma rot. Maintain good soil fertility, practice long crop rotations and control solanaceous weeds in and around fields cropped to tomato. Only harvest fruit after free moisture from rain or dew has dried from fruit surfaces and avoid injuring fruit during harvest. Do not pack fruit when wet.

Necrotic lesions.

PHOMA ROT

Necrotic stem and leaf lesions.

POWDERY MILDEW

Leveillula taurica: **leaf chlorosis and sporulation.**

Leveillula taurica: **leaf chlorosis and sporulation.**

CAUSAL AGENTS

Leveillula taurica (anamorph: *Oidiopsis sicula*)

Oidium neolycopersici

Oidium lycopersici

DISTRIBUTION

Leveillula taurica: Worldwide

Oidium neolycopersici: Worldwide

Oidium lycopersici: Australia, USA (California)

SYMPTOMS

Leveillula taurica: Initial symptoms manifest as light-green to bright-yellow lesions on upper leaf surfaces. Eventually light, powdery fungal sporulation forms on lower leaf surfaces. Under ideal conditions, white, powdery masses of conidia develop on both leaf surfaces. As disease progresses, lesions turn necrotic; if disease is severe, entire leaves will die. Affected plants may defoliate, resulting in reduced yields and small, sunburned fruit.

Oidium neolycopersici **and** ***O. lycopersici:*** Disease first appears as small, circular areas of whitish fungal growth with sporulation occurring mainly on upper leaf surfaces. As sporulating lesions enlarge, underlying leaf tissue turns yellow, eventually becoming brown and shriveled. Sporulation that typically occurs on upper leaf surfaces distinguishes *Oidium* from *Leveillula,* which typically sporulates on lower leaf surfaces. When infection is severe, masses of powdery conidia will cover entire leaf surfaces, as well as petioles and calyces; however, fruit remain uninfected. *Oidium neolycopersici* has been reported in field-grown tomato but it is primarily an issue for protected culture production.

CONDITIONS FOR DISEASE DEVELOPMENT

Leveillula taurica: This fungus has a wide host range on which it can survive and spread to tomatoes. *Leveillula taurica* conidia can travel long distances in air currents and can germinate under low (52-75%) relative humidity. Disease development is favored by warm (>27ºC) temperatures; however, *L. taurica* conidia can germinate between 10 and 32ºC.

Oidium neolycopersici **and** ***O. lycopersici:*** Optimum conditions for disease development include low light intensity and temperatures between 20 and 27ºC accompanied by high (85-95%) relative humidity. However, as with *L. taurica,* infection can occur at low (50%) relative humidity.

CONTROL

Several fungicides, including sulfur, can be effective in controlling this disease if good coverage is obtained and they are applied in a timely manner. Inorganic bicarbonate slates and cupric hydroxide have also been effective in controlling powdery mildews.

POWDERY MILDEW

Leveillula taurica: chlorotic lesions on upper leaf surfaces and white sporulation on lower leaf surface.

Oidium neolycopersici: sporulation on upper leaf surface.

Oidium neolycopersici: leaves of susceptible (left) and intermediate-resistant (right) plants.

Oidium neolycopersici: sporulation on petioles. (Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

SEPTORIA LEAF SPOT

Lesions with brown borders and gray centers. (Courtesy of Tom Zitter, Cornell University)

CAUSAL AGENT

Septoria lycopersici

DISTRIBUTION

Worldwide

SYMPTOMS

Symptoms first appear as small, dark, water-soaked spots on older leaves. Later, the spots enlarge to form circular lesions about five millimeters in diameter. These lesions have black or brown borders with gray centers that are peppered with small, black pycnidia. Lesions on stems, petioles and calyces tend to be elongated with pycnidia developing in centers of lesions. When Septoria leaf spot is severe, lesions coalesce, leaves collapse and eventually plants defoliate.

CONDITIONS FOR DISEASE DEVELOPMENT

Septoria lycopersici can survive on debris from previous crops as well as on several weed hosts including nightshade, horse nettle, jimson weed and ground cherry. Extended periods of high (100%) relative humidity and temperatures between 20 and 25°C favor infection and disease development. Numerous conidia are exuded from pycnidia when humidity is high. Conidia can then be spread by wind and splashing water from rain or overhead irrigation, and on workers' clothing and tools, on cultivation equipment, and by insects.

CONTROL

Establish a fungicide spray program in conjunction with cultural practices that lower inoculum potential such as turning under plant debris or plant debris removal. Rotate to a non-host crop for three years to reduce losses from Septoria leaf spot.

Necrotic lesions with surrounding chlorosis.

SEPTORIA LEAF SPOT

Individual and coalescing necrotic lesions.

Stem and leaf lesions.

Pycnidia in center of stem lesions.

SOUTHERN BLIGHT

White mycelium and sclerotia on lower stem.

CAUSAL AGENT

Sclerotium rolfsii (teleomorph: *Athelia rolfsii*)

DISTRIBUTION

Worldwide

SYMPTOMS

This fungus can cause damping-off, crown and root rot, and fruit rot. The first symptom on seedlings is a dark-brown lesion at or below the soil line. Stem tissue is invaded completely, causing seedlings to quickly damp-off and die. On older plants, developing lesions girdle stems, causing plants to wilt without a change in foliage color. These lesions may continue expanding to cause root rot below the soil line and may also extend several centimeters above it. If moisture is adequate, white mycelium grows over lesion surfaces and tan sclerotia (one to two millimeters in diameter) are readily formed. Severely infected plants may eventually die. Fruit that contact *Sclerotium rolfsii* are invaded quickly, resulting in sunken, yellowish lesions with ruptured epidermises. White mycelium grows from fruit lesions and sclerotia form on lesion surfaces.

CONDITIONS FOR DISEASE DEVELOPMENT

Sclerotium rolfsii is an efficient saprophyte and can survive in soil and plant debris for several years. This fungus can be spread in surface water and by the movement of infested soil on cultivation equipment. High (30-35°C) temperatures and soil moisture levels favor disease development.

CONTROL

Regulate soil moisture levels and deep-plow plant residues to reduce losses from southern blight. Implement a sanitation program that includes removal or burning of all infected plants. Apply fungicides, fumigate soil and rotate from tomato to non-host crops like corn, grain sorghum and wheat for three years to reduce losses from southern blight.

Sclerotia on artificial media.

SOUTHERN BLIGHT

White mycelium and sclerotia on stem. (Courtesy of Clemson University – U.S. Department of Agriculture Cooperative Extension Slide Series, Bugwood.org)

TARGET SPOT

Initial leaf lesions.

CAUSAL AGENTS

Corynespora cassiicola

DISTRIBUTION

Worldwide

SYMPTOMS

All above-ground parts of tomato plants can be infected by *Corynespora cassiicola.* Symptoms begin on leaves as tiny lesions that rapidly enlarge and develop into light-brown lesions with distinct yellow halos. Often, lesions grow together causing infected leaf tissue to collapse. Symptoms on stems also begin as small lesions that rapidly enlarge and elongate, and may eventually girdle stems, resulting in collapse of foliage above where stems were girdled. When disease is severe, numerous leaf and stem lesions form on plants causing extensive tissue collapse and eventually plant death. Infection of immature fruit begins as minute, dark-brown, sunken spots that enlarge as the disease progresses. Large, brown, circular lesions with cracked centers develop on mature fruit. Fungal sporulation commonly occurs from these lesions.

CONDITIONS FOR DISEASE DEVELOPMENT

Corynespora cassiicola has a broad host range on which it survives. Infection occurs when temperatures are between 16 and 32°C during relatively long periods (>16 hours) of high moisture. Conidia, which form abundantly on surfaces of infected tissues, are spread by air movement and rainfall.

CONTROL

Initiate a fungicide spray program prior to the onset of symptoms to help reduce losses from target spot.

Coalescing lesions surrounded by chlorosis.

TARGET SPOT

Coalescing lesions.

Large, brown-black, circular, sporulating lesions. (Courtesy of Yuan-Min Shen, Taichung District Agricultural Research and Extension Station, Bugwood.org)

VERTICILLIUM WILT

Leaflets with chlorosis and necrosis. (Courtesy of Gerald Holmes, California Polytechnic State University at San Luis Obispo, Bugwood.org)

CAUSAL AGENTS

Verticillium albo-atrum

Verticillium dahliae

DISTRIBUTION

Worldwide

SYMPTOMS

Wilting of older leaves begins at leaflet margins which progress to yellow then brown v-shaped lesions. Older leaves eventually turn yellow and later, necrotic. Affected plants are stunted, do not respond to fertilizer or water, and display diurnal wilting on sunny days. When the base of the main stem is cut at the crown, a light-tan discoloration of vascular and pith tissues can be seen. This discoloration does not usually extend up the main stem to any extent, but can be present near the shoot tips when disease is severe.

CONDITIONS FOR DISEASE DEVELOPMENT

Verticillium albo-atrum and *V. dahliae* have extremely wide host ranges and can survive in plant debris as mycelia and microsclerotia for several years. Disease development is favored by moderate (21-25°C) temperatures. These fungi enter plants through root wounds caused by cultivation, secondary root formation and nematode feeding.

CONTROL

Grow resistant varieties to reduce losses from Verticillium wilt. Soil fumigation and solarization have been shown to reduce disease incidence.

Wilting begins at leaflet margins. (Courtesy of Flavia Ruiz, Erieview, Inc.)

Leaflet chlorosis and necrosis. (Courtesy of Gerald Holmes, California Polytechnic State University at San Luis Obispo, Bugwood.org)

VERTICILLIUM WILT

Leaflet with v-shaped lesions. (Courtesy of Gerald Holmes, California Polytechnic State University at San Luis Obispo, Bugwood.org)

Stem cross-sections showing diseased (left) and healthy (right) vascular tissue.

WHITE MOLD

Stem lesion.

Stem lesion with white mycelium.

CAUSAL AGENTS

Sclerotinia sclerotiorum

Sclerotinia minor

DISTRIBUTION

Worldwide

SYMPTOMS

Sclerotinia sclerotiorum and *S. minor* can infect leaves, stems, petioles and occasionally fruit. Initially, water-soaked lesions develop on stems, eventually causing a softening of infected areas. Typically, white mycelium grows on stem lesions. A progressive soft decay of external tissues is followed by an internal hollowing of pith tissue. Large areas of affected stems eventually die and become dry and tan-gray. Irregularly shaped black pebble-like bodies called sclerotia develop on surfaces of and inside stems and are diagnostic for this disease. Infected fruit develop grayish lesions that quickly turn into a watery rot with white mycelium and sclerotia developing on lesion surfaces.

CONDITIONS FOR DISEASE DEVELOPMENT

These fungi have a wide host range and can survive as sclerotia in soil and in infested plant debris from one season to the next. Sclerotia are the main source of inoculum for this disease. Conditions of prolonged moisture from high humidity, frequent rains, dews and fog, and cool temperatures (16-21°C) favor disease development. The most important means of long-distance spread is airborne ascospores that erupt from sclerotia. Moving contaminated soil and fertilizing with manure from animals fed infected plant debris are two common ways of short distance spread of sclerotia and mycelium. Irrigation water may also spread these fungi within fields and from field to field.

CONTROL

Avoid fields where white mold losses have occurred previously. Plant in well-drained soil, use wide row spacing and water very 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 sterilized. Establish a crop rotation with non-host crops such as corn, small grains and grasses. Fumigate or steam-sterilize soil to reduce soilborne inoculum, and apply fungicides to further reduce losses from white mold.

Crown lesion with white mycelium on surface.

WHITE MOLD

Large, black sclerotia within a hollowed stem.

Sclerotium on calyx and pedicel.

White mycelium and gray sclerotia on infected fruit.

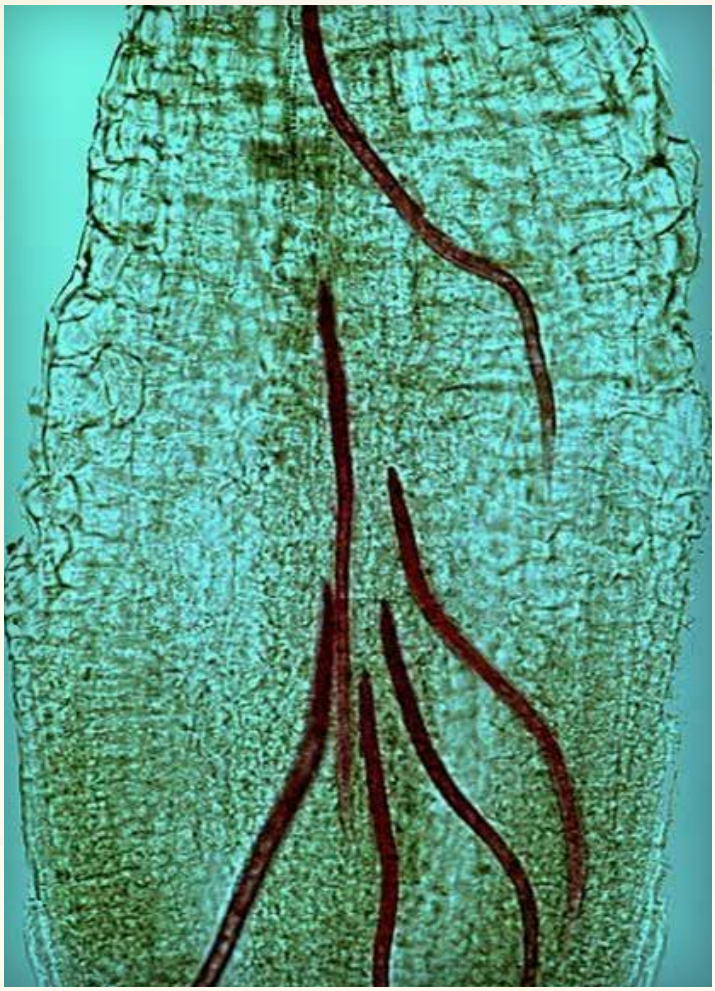
De Ruiter™ Seminis®

NEMATODE DISEASES

ROOT-KNOT NEMATODES

ADDITIONAL NEMATODE DISEASES

NEMATODE
ROOT-KNOT



Second-stage juveniles of root-knot nematode, *Meloidogyne* sp., penetrating tomato root tip. (Courtesy of Jonathan D. Eisenback, Virginia Polytechnic Institute and State University, Bugwood.org)

Extensive root-galling. (Courtesy of Gerald Holmes, California Polytechnic State University at San Luis Obispo, Bugwood.org)

CAUSAL AGENTS

Meloidogyne hapla (Northern root-knot nematode)
Meloidogyne incognita (Southern root-knot nematode)
Meloidogyne arenaria (Peanut root-knot nematode)
Meloidogyne javanica (Javanese root-knot nematode)
Meloidogyne chitwoodi (Root-knot nematode)
Meloidogyne enterolobbii (Root-knot nematode)

DISTRIBUTION

Worldwide

SYMPTOMS

The first above-ground symptoms are stunting, wilting and a general off-color appearance of affected plants. Other symptoms include chlorosis and characteristic symptoms of nutrient deficiency (e.g., abaxial leaf surfaces turn purple, which can indicate phosphorous deficiency). When diseased plants are pulled-up, irregular swellings of the roots, referred to as galls or knots, are easily observed. Southern root-knot nematode galls tend to be larger and more irregular in shape and size compared to those caused by the northern root-knot nematode, which produces smaller, discrete and more uniform galls with lateral roots developing adjacent to the galls.

CONDITIONS FOR DISEASE DEVELOPMENT

Root-knot nematodes have a very wide host range that encompasses many agricultural crops as well as weeds on which they can grow and survive. Disease is more severe in areas where there is a long growing season with a mild or no winter season. Although these nematodes can cause disease in many soil types, root damage is most serious on light, sandy soils. Warm (27°C) soil temperatures favor disease development by *M. arenaria, M. javanica,* and *M. incognita* whereas cool (16-20°C) soil temperatures favor disease development by *M. hapla. Meloidogyne incognita* is by far the most widely distributed root-knot nematode species worldwide.

CONTROL

The use of resistant varieties is the most effective way to control *Meloidogyne* species. The most common resistance gene used in tomato is designated *Mi.* The *Mi* gene confers resistance against *M. arenaria, M. incognita* and *M. javanica* but it is not effective against *M. hapla* and some of the other *Meloidogyne* species, which highlights the importance of correct identification of the *Meloidogyne* species present in a region or field. Tomato varieties carrying the *Mi* gene should be managed with cultural practices that reduce nematode populations because continued use of resistant varieties alone will result in selection of nematodes that overcome resistance conferred by the *Mi* gene. *Mi* resistance can be broken when soil temperatures reach 27-33°C for prolonged periods of time. Additional root-knot control measures include crop rotation with cover crops (e.g., sun hemp, *Crotalaria juncea;* hairy indigo, *Indigofera hirsuta*) that are poor hosts of root-knot nematodes and which return high amounts of nitrogen and organic matter to soil. Crop rotation with small grains may help in some tomato-growing regions where nematodes are problematic. Crop rotation to *Brassica* species in combination with soil solarization has shown some effectiveness in controlling root-knot nematodes. Soil pasteurization and fumigation as well as transplanting disease-free seedlings can also help reduce losses from root-knot nematodes.

NEMATODE
ROOT-KNOT

Root-knot nematode-infected plants on non-fumigated ground (left) and healthy plants on fumigated ground (right).
(Courtesy of Joe Nunez, University of California Division of Agriculture and Natural Resources)

Root-galling on plant removed from soil. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Meloidogyne enterolobbii root infection. (Courtesy of Jeffrey W. Lotz, Florida Department of Agriculture and Consumer Services, Bugwood.org)

ADDITIONAL NEMATODES

Potato cyst nematode: brown to whitish-gray cysts attached to roots.
(Courtesy of Dominque Blancard, French National Institute for Agricultural Research)

Reniform nematode: infected root. (Courtesy of Jonathan D. Eisenback, Virginia Polytechnic Institute and State University, Bugwood.org)

CAUSAL AGENTS

Belonolaimus species (Sting nematode)
Globodera species (Potato cyst nematode)
Pratylenchus species (Root lesion nematode)
Rotylenchus species (Reniform nematode)

DISTRIBUTION

Worldwide

SYMPTOMS

***Belonolaimus* species (Sting nematode):** Roots of affected plants are short and stubby with dark lesions at root tips. The entire root system is reduced in size compared to a healthy root system. When damage to root tips is severe, new roots may form above *Belonolaimus*-infected roots, which gives a highly branched appearance to the root system. Foliage of affected plants may turn yellow or red, indicating a nutrient deficiency or a physiological response to nematode feeding.

***Globodera* species (Potato cyst nematode):** The host range of the potato cyst nematode is relatively small (potato, tomato and eggplant). Reddish-brown necrotic lesions with brown to whitish-gray cysts, which are egg-containing female nematodes, develop on roots. Later, roots of affected plants may decay from root rot. *Globodera*-infected plants may display uneven growth and be tall and sparse with decreased fruit production. Lower leaves eventually turn a pale yellow.

***Pratylenchus* species (Root lesion nematode):** Root infections do not usually cause economic damage. However, lesions that develop on roots can often be invaded by soilborne fungal and bacterial pathogens. Infected, asymptomatic plants can serve as a reservoir, allowing nematodes to reproduce and increase populations.

***Rotylenchus* species (Reniform nematode):** Above-ground symptoms on tomato include stunting and defoliation. *Rotylenchus*-infected roots may be discolored and necrotic, and develop areas of decay. Affected plants yield less compared to healthy plants due to impaired root systems. Plant death can occur if nematode populations are high.

CONDITIONS FOR DISEASE DEVELOPMENT

These nematodes are commonly associated with high sand-content soils and thrive under warm, irrigated conditions. These nematodes all can survive on several weed species which can act as asymptomatic reservoirs of nematode populations.

CONTROL

Proper identification of the nematode species present in a field and its population is critical for determining effective management strategies. Decrease nematode populations with nematicides and weed-free fallow periods. Grow cover crops like sun hemp (*Crotalaria juncea*) and hairy indigo (*Indigofera hirsuta*) that are poor nematode hosts but which return high amounts of nitrogen and organic matter to soils. Implement cultural practices that promote moisture and nutrient availability throughout the crop cycle.

De Ruiter™ Seminis®

PARASITIC
PLANTS

BROOMRAPE

DODDER

BROOMRAPE

Broomrape growing on roots.

Broomrape flowering at base of plant.

CAUSAL AGENT

Orobanche species

DISTRIBUTION

Asia, Central America, Europe, India, Middle East, North Africa, Pakistan, USA

SYMPTOMS

The first noticeable sign of broomrape is appearance of whitish-yellow shoots at the base of infected tomato plants. When soil is removed, broomrape roots are found to be attached to roots of affected plants. Later these yellow snapdragon-like parasitic plants produce flowers, and as broomrape plants mature they turn brown and their seed capsules release tiny, black seeds.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Broomrape seeds may lie dormant in soil for more than 20 years. Root exudates from host plants stimulate germination and young broomrape seedlings attach their roots to roots of host plants from which they extract nutrients to grow and reproduce. After flowering, very small seeds are produced that can be distributed within and between fields as soil is cultivated, on cultivation equipment and in irrigation water. Conditions favorable for tomato plant growth also favor broomrape growth and parasitism.

CONTROL

Fumigation and crop rotation can reduce losses from *Orobanche* species when used in conjunction with good sanitation practices that include removing and destroying all affected tomato plants along with parasitic broomrape plants.

DODDER

CAUSAL AGENT
Cuscuta species

DISTRIBUTION
Worldwide

SYMPTOMS
More than one hundred 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 solely on the host plant for its nutrition. Infected plants will appear weak and discolored. 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 contacts 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 ten years. Seeds usually germinate in late winter and spring in cold climates; however, germination can continue through the summer. Environmental conditions that favor tomato growth are also beneficial to dodder.

CONTROL
Immediately remove or burn dodder, along with infested plants upon detection. Apply contact herbicides to control localized infestations. If an infestation is widespread, apply pre-emergence herbicides, deep-plow crop debris, and rotate to grasses.

Dodder scattered throughout processing tomato field.

Extensive dodder infestation in processing tomato field.

Dodder and bindweed infestation in processing tomato field.

De Ruiter™ Seminis®

PHYTOPLASMA

DISEASE

TOMATO BIG BUD

TOMATO BIG BUD

Phytoplasma-infected plant with reduced leaf size and interveinal chlorosis. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

Phytoplasma-infected plant with enlarged flower buds, shortened internodes, and chlorotic leaves. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

CAUSAL AGENTS

Tomato big bud group of phytoplasmas including:

Candidatus Phytoplasma asteris (aster yellows taxon)

Candidatus Phytoplasma aurantifolia (faba bean phyllody taxon)

Candidatus Phytoplasma trifoli (clover proliferation taxon)

Several additional taxa have been found to be present in tomato plants symptomatic for tomato big bud.

VECTORS

Orosius argentatus (Common brown leafhopper)

Macrosteles quadrilineatus (Aster leafhopper)

Neoaliturus tenellus (Beet leafhopper) (synonym = *Circulifer tenellus*)

DISTRIBUTION

Australia, Brazil, China, India, Israel, Italy, Japan, Jordan, Russia, South Africa, USA

SYMPTOMS

Phytoplasmas, formerly known as mycoplasma-like organisms, are pleomorphic, obligate bacteria that lack cell walls. They live and move within plants through phloem vessels. Several phytoplasma diseases have been associated with tomato under various names (stolber, tomato big bud, tomato yellows). The primary symptom of tomato big bud, and the one for which it is named, is enlargement and abnormal development of flower buds. Sepals may not separate as flowers open, and buds are swollen and green. Other symptoms include a thickening of stems, proliferation of small side shoots and aerial root initials. Shortening of the internodes, an erect growth habit and development of an overall yellowish appearance may also occur. Fruit may be small and deformed.

CONDITIONS FOR SYMPTOM DEVELOPMENT

This phytoplasma can survive in crops such as hot pepper, lettuce, eggplant and potato, as well as weeds such as dock, lambsquarter, nightshade, sowthistle and jimson weed, and is readily transmitted to tomato by the common brown leafhopper. Transmission occurs when leafhoppers carrying the phytoplasma migrate to tomato and feed.

CONTROL

Eradicate solanaceous weeds that harbor the vector and spray insecticides to 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.

TOMATO BIG BUD

Enlarged flower buds on phytoplasma-infected plant.

Phytoplasma-infected plant with enlarged flower buds.

Deformed fruit.

De Ruiter™ Seminis®

VIRAL DISEASES

ALFALFA MOSAIC

CRINIVIRUS DISEASES

CUCUMBER MOSAIC

GEMINIVIRUS (*BEGOMOVIRUS*) DISEASES

GEMINIVIRUS (*CURTOVIRUS*) DISEASE

ILARVIRUS DISEASES

PELARGONIUM ZONATE SPOT

PEPINO MOSAIC

POTYVIRUS DISEASES

TOBAMOVIRUS DISEASES

TOMATO BUSHY STUNT

TOMATO DOUBLE-VIRUS STREAK

TORRADOVIRUS DISEASES

TOSPOVIRUS DISEASES

COMMON VECTORS OF TOMATO VIRUSES

ALFALFA MOSAIC

Extensive yellowing of foliage.

Brown discoloration of the phloem.

CAUSAL AGENT

Alfalfa mosaic virus (AMV)

VECTORS

Aphis gossypii (melon or cotton aphid)

Myzus persicae (green peach aphid)

Additional aphid species

DISTRIBUTION

Worldwide

SYMPTOMS

Typically, first symptoms appear as yellowing or bronzing of young leaflets as well as necrosis of leaflet veins. Severely affected plants stop growing and leaflets curl downward. The main stem has a dark-brown discoloration of the phloem at the soil line which can be seen by lightly scraping the epidermis from the stem. This discoloration may extend into the upper shoot tips. Irregular, brown streaking in the stem pith is characteristic of alfalfa mosaic. Roots often show this same red-brown discoloration. Depending on the age of the plant at the time of infection, fruit may develop external and internal brown spots and may be distorted. Generally, disease incidence is greater the closer tomatoes are to alfalfa fields and gradually diminishes with increasing distance from the source.

CONDITIONS FOR DISEASE DEVELOPMENT

AMV has a wide host range on which it can survive and is typically found in older alfalfa fields or permanent pastures. Aphids carry AMV in a non-persistent manner and transmission occurs during feeding or probing of plants. During hay-mowing, winged aphids carry the virus from alfalfa to nearby tomato fields. Little or no secondary spread occurs within a field.

CONTROL

Locating fields away or upwind from alfalfa are possible means of avoiding alfalfa mosaic. Spraying for aphids will not eliminate primary infection of tomatoes.

ALFALFA MOSAIC

Dark-brown lesions on fruit.

Distorted fruit with brown necrotic lesions.

Irregular, brown streaking of pith tissue and fruit with necrotic lesions.

CRINIVIRUS DISEASES

TICV: interveinal chlorosis.

TICV: severe interveinal chlorosis.

TICV: interveinal bleaching.

CAUSAL AGENTS

Tomato chlorosis virus (ToCV)

Tomato infectious chlorosis virus (TICV)

VECTORS

TICV: *Trialeurodes vaporariorum* (greenhouse whitefly)

ToCV: *Bemisia tabaci* (sweet potato or silverleaf whitefly)

Trialeurodes abutilonea (banded winged whitefly)

Trialeurodes vaporariorum (greenhouse whitefly)

DISTRIBUTION

TICV: Asia, Europe, Middle East, North America, Tunisia

ToCV: Worldwide

SYMPTOMS

Symptoms that develop in TICV- and ToCV-affected plants can be mistaken for magnesium or nitrogen deficiency or even pesticide phytotoxicity. First symptoms express approximately three to four weeks after initial infection and include development of interveinal chlorotic blotches on older leaves while the leaf veins remain green. As symptoms progress to younger leaves, the chlorotic blotches turn bleached-white or necrotic depending on the variety, and affected leaf tissues become brittle. Fruit color may be impacted. Severe losses due to poor fruit set have been reported.

CONDITIONS FOR DISEASE DEVELOPMENT

Several species of whitefly transmit these two viruses in a semi-persistent manner in protected culture. These viruses are not seedborne nor can they be transmitted mechanically. Once acquired by the whitefly, the virus remains infectious in the insect's body for a few days. Virus spread only occurs when virus-carrying whiteflies move from infected to healthy plants. Early infection can result in severe yield loss in certain varieties.

CONTROL

Management of these viruses can be very challenging due to high populations of vectors, high vector efficiencies transmitting the viruses and the wide host range of vectors and viruses. Chemical and cultural practices have been the primary methods used to control these viruses. Although insecticides can reduce whitefly populations, this method of control was shown to be inefficient because whiteflies still transmit TICV and ToCV before dying from exposure to an insecticide. Also, in the time it takes for symptoms of infection to express (three to four weeks), the viruses can be spread extensively before symptoms are observed. Use insect exclusion screening (50 mesh/297 micron) on all protected-culture structures to minimize entrance of whiteflies into greenhouses. Obtain all transplants from a whitefly-free nursery. Remove all plant material from greenhouses after crop completion and implement a plant-free period to ensure the absence of whiteflies in greenhouses.

CRINIVIRUS DISEASES

TICV: symptoms can be confused with magnesium deficiency.

ToCV: leaf chlorosis.

ToCV: leaf chlorosis with purpling. (Courtesy of Rafael Fernández-Muñoz, IHSM La Mayora, UMA-CSIC)

ToCV: interveinal chlorosis. (Courtesy of Moshe Lapidot, Institute of Plant Sciences, Volcani Center)

ToCV: symtomatic plant (left); asymptomatic plant (right).
(Courtesy of Rafael Fernández-Muñoz, IHSM La Mayora, UMA-CSIC)

CUCUMBER MOSAIC

Tomato field infected with cucumber mosaic virus. (Courtesy of Ed Sikora, Auburn University, Bugwood.org)

Leaf chlorosis and mosaic.

Chlorotic mosaic.

CAUSAL AGENT

Cucumber mosaic virus (CMV)

Several strains have been reported.

VECTOR

Aphis gossypii (melon or cotton aphid)

Myzus persicae (green peach aphid)

Additional aphid species

DISTRIBUTION

Worldwide

SYMPTOMS

Symptoms of this disease vary greatly depending on the virus strain. Often CMV stunts tomato plants to give affected plants a bushy appearance. Leaf symptoms may vary from mild, green mottling to chlorosis or severe necrosis. Some CMV strains cause a severe "shoestring" symptom in which leaflet blades are tapered or even greatly reduced with only the midrib of the leaflet remaining. "Shoestring" symptoms can be confused with the severe symptoms observed with ToMV infection; however, with the CMV-caused shoestring symptoms, the blade of the leaflet is usually more suppressed. Fruit are reduced in size and often misshapen.

CONDITIONS FOR DISEASE DEVELOPMENT

This virus has a wide host range (800 species) from which it can be acquired by aphids and transmitted in a non-persistent manner. CMV is mainly a problem where infected alternate hosts survive all year long as well as in greenhouses where, once introduced, it can be spread readily from plant to plant by aphids. CMV can also be mechanically transmitted; however, because the virus particle is unstable, the likelihood of transmission by greenhouse workers and their tools is much less than with a virus like ToMV.

CONTROL

In greenhouse crops controlling aphids can greatly reduce the incidence of this disease. In field-grown tomato, eliminate weeds and ornamental plants that harbor the virus. Rogue infected tomato plants to reduce spread of CMV.

CUCUMBER MOSAIC

Leaf crumpling with chlorotic and necrotic lesions.

Leaflet tapering.

Leaflets with severe "shoestring" appearance. (Courtesy of Ed Sikora, Auburn University, Bugwood.org)

Leaflets with mild "shoestring" appearance.

Extensive necrotic stem lesions.

Fruit with internal necrosis.

GEMINIVIRUS (*BEGOMOVIRUS*) DISEASES

CdTV: chlorotic, leathery leaflets.

ToMHaV: yellow mosaic symptoms.

CAUSAL AGENTS (partial listing of *Begomoviruses*)		DISTRIBUTION
Ageratum enation virus	AEV	India
Ageratum yellow vein virus	AYVV	Asia
Ageratum yellow vein Hualien virus	AYVHuV	China, Taiwan
Chino del tomate virus	CdTV	Mexico, USA
Croton yellow vein mosaic virus	CYVMV	India
Euphorbia mosaic virus	EuMV	Central America, Venezuela
Papaya leaf curl China virus	PaLCuCNV	China
Pepper golden mosaic virus	PGMV	North America
Pepper huasteco virus	PHV	Mexico, USA
Pepper huasteco yellow vein virus	PHYVV	Central America
Pepper leaf curl Bangladesh virus	PLCBDV	India
Pepper yellow leaf curl Indonesia virus	PepYLCIV	Indonesia
Potato yellow mosaic Panama virus	PYMPV	Central America (Panama)
Pepper yellow vein Mali virus	PeYVMLV	Central and West Africa
Rhynchosia golden mosaic Yucatan virus	RhGMYuV	Ecuador
Serrano golden mosaic virus	SGMV	Mexico, USA
Sida golden mosaic virus	SiGMV	USA (Florida)
Sinaloa tomato leaf curl virus	STLCV	Mexico, Nicaragua
Texas pepper virus	TPV	Mexico, USA
Tobacco leaf curl Yunnan virus	TbLCYNV	China
Tomato chlorotic mottle virus	ToCMoV	Brazil
Tomato curly stunt virus	ToCSV	South Africa
Tomato dwarf leaf virus	ToDLV	Argentina
Tomato golden mosaic virus	TGMV	Brazil
Tomato golden mottle virus	ToGMoV	Mexico
Tomato golden vein virus	TGVV	Brazil
Tomato leaf crumple virus	ToLCrV	Nicaragua
Tomato leaf curl virus	ToLCV	Asia, Australia
Tomato leaf curl Anjouan virus	ToLCAnjV	Indian Ocean Islands
Tomato leaf curl Arusha virus	ToLCAV	Africa
Tomato leaf curl Bangalore virus	ToLCBV	India
Tomato leaf curl Bangladesh virus	ToLCBDV	Asia
Tomato leaf curl Cebu virus	ToLCCeV	Philippines
Tomato leaf curl Cameroon virus	ToLCCMV	Central and West Africa
Tomato leaf curl China virus	ToLCCNV	China
Tomato leaf curl Comoros virus	ToLCKMV	Indian Ocean Islands

GEMINIVIRUS (*BEGOMOVIRUS*) DISEASES

CAUSAL AGENTS (partial listing of *Begomoviruses*)		DISTRIBUTION
Tomato leaf curl Diana virus	ToLCDiV	Madagascar
Tomato leaf curl Ghana virus	ToLCGHV	West Africa
Tomato leaf curl Gandhinagar virus	ToLCGNV	India
Tomato leaf curl Guangdong virus	ToLCGuV	China
Tomato leaf curl Guangxi virus	ToLCGxV	China
Tomato leaf curl Gujarat virus	ToLCGV	India
Tomato leaf curl Hainan virus	ToLCHnV	China
Tomato leaf curl Hanoi virus	ToLCHanV	Vietnam
Tomato leaf curl Hsinchu virus	ToLCHsV	Taiwan
Tomato leaf curl Iran virus	ToLCIV	Iran
Tomato leaf curl Java virus	ToLCJV	Indonesia
Tomato leaf curl Joydebpur virus	ToLCJoV	Bangladesh, India
Tomato leaf curl Karnataka virus	ToLCKV	India
Tomato leaf curl Kerala virus	ToLCKeV	India
Tomato leaf curl Kumasi virus	ToLCKuV	India
Tomato leaf curl Laos virus	ToLCLV	Southeast Asia
Tomato leaf curl Madagascar virus	ToLCMadV	Madagascar
Tomato leaf curl Malaysia virus	ToLCMYV	Malaysia
Tomato leaf curl Mali virus	ToLCMLV	West Africa
Tomato leaf curl Mindanao virus	ToLCMiV	Philippines
Tomato leaf curl Moheli virus	ToLCMoV	Philippines
Tomato leaf curl Namakely virus	ToLCNaV	Madagascar
Tomato leaf curl New Delhi virus	ToLCNDV	Asia, Europe
Tomato leaf curl Nigeria virus	ToLCNGV	West Africa
Tomato leaf curl Oman virus	ToLCOV	Southern Arabia
Tomato leaf curl Palampur virus	ToLCPaV	India
Tomato leaf curl Patna virus	ToLCPatV	India
Tomato leaf curl Philippines virus	ToLCPV	Philippines
Tomato leaf curl Pune virus	ToLCPuV	India
Tomato leaf curl Rajasthan virus	ToLCRaV	India
Tomato leaf curl Seychelles virus	ToLCSCV	Indian Ocean Islands
Tomato leaf curl Sinaloa virus	ToLCSiV	Central America, Mexico
Tomato leaf curl Sri Lanka virus	ToLCLKV	Indian Ocean Islands
Tomato leaf curl Sudan virus	ToLCSDV	Africa
Tomato leaf curl Sulawesi virus	ToLCSuV	Indonesia
Tomato leaf curl Taiwan virus	ToLCTWV	China, Taiwan

ToMoV: vein-yellowing.

ToSLCV: shortening of terminal internodes and leaflet puckering.

GEMINIVIRUS (*BEGOMOVIRUS*) DISEASES

TYLCV: stunting caused by young crop infection.

TYLCV: interveinal chlorosis and shortening of internodes on greenhouse-grown tomato. (Courtesy of Don Ferrin, Louisiana State University Agricultural Center, Bugwood.org)

TYLCV: interveinal chlorosis and cupping of leaflets. Note whitefly infestation. (Courtesy of Bill Wintermantel, U.S. Department of Agriculture, Agricultural Research Service)

CAUSAL AGENTS (partial listing of *Begomoviruses*)		DISTRIBUTION
Tomato leaf curl Toliara virus	ToLCToV	Madagascar
Tomato leaf curl Uganda virus	ToLCUV	Africa
Tomato leaf curl Vietnam virus	ToLCVV	Vietnam
Tomato leaf deformation virus	ToLDeV	Ecuador, Peru
Tomato leaf distortion virus	ToLDV	Brazil
Tomato mild mosaic virus	ToMMV	Brazil
Tomato mild yellow leaf curl Aragua virus	ToMYLCV	Venezuela
Tomato mosaic Havana virus	ToMHaV	Caribbean, Central America, Mexico
Tomato mottle virus	ToMoV	Nicaragua, Puerto Rico, USA
Tomato mottle leaf curl virus	ToMLCV	Brazil
Tomato mottle Taino virus	ToMoTaV	Central America, Cuba
Tomato mottle wrinkle virus	ToMoWV	South America
Tomato rugose mosaic virus	ToRMV	Brazil, Central America
Tomato rugose yellow leaf curl virus	TRYLCV	South America
Tomato severe leaf curl virus	ToSLCV	Central America, Mexico
Tomato severe rugose virus	ToSRV	Brazil
Tomato yellow leaf curl virus	TYLCV	Worldwide
Tomato yellow leaf curl Axarguia virus	TYLCAxV	Spain
Tomato yellow leaf curl China virus	TYLCCNV	China
Tomato yellow leaf curl Indonesia virus	TYLCIDV	Indonesia
Tomato yellow leaf curl Kanchanaburi virus	TYLCKaV	Thailand
Tomato yellow leaf curl Malaga virus	TYLCMaV	Spain
Tomato yellow leaf curl Mali virus	TYLCMLV	West Africa
Tomato yellow leaf curl Sardinia virus	TYLCSV	Italy
Tomato yellow leaf curl Thailand virus	TYLCTHV	Asia
Tomato yellow leaf curl Yunnan virus	TYLCYnV	China
Tomato yellow leaf distortion virus	ToYLDV	Central America
Tomato yellow margin leaf curl virus	ToYMLCV	Venezuela
Tomato yellow mottle virus	ToYMoV	Costa Rica
Tomato yellow mosaic virus	TYMV	Venezuela
Tomato yellow spot virus	TYSV	Brazil
Tomato yellow vein streak virus	ToYVSV	Brazil

GEMINIVIRUS (*BEGOMOVIRUS*) DISEASES

VECTORS

Bemisia tabaci (synonym = *Bemisia argentifolii*; common names = sweet potato or silverleaf whitefly)

VECTOR DISTRIBUTION

Worldwide

SYMPTOMS

Plants infected when young can be bushy and stunted with shortened internodes and erect branches. Leaflets are often small, chlorotic, cupped upward and have a leathery texture. Other characteristic begomovirus symptoms include leaf rolling, interveinal chlorosis of new leaves and purpling of older leaves. Severely affected plants generally do not set fruit. Plants infected later tend to develop less severe symptoms, but also include leaf chlorosis, cupping, flower abortion and failure to set fruit. Fruit that have set before infection often ripen normally, but fruit number can be dramatically reduced.

CONDITIONS FOR DISEASE DEVELOPMENT

Begomoviruses have a relatively wide host range which includes cereals, legumes, vegetables, fiber crops (cotton) and weeds. Begomoviruses are transmitted in a circulative persistent manner by whiteflies in both temperate and tropical regions of the world. Approximately 15-30 minutes of whitefly feeding can result in transmission of the virus. Mechanical transmission of begomoviruses has only been observed in a few strains. Severe outbreaks are associated with large populations of whiteflies. Older abandoned tomato fields can remain an important source of the virus. Although resistant varieties are available and produce acceptable yields, they can still act as a reservoir to infect newly planted susceptible varieties.

CONTROL

The use of resistant varieties can greatly reduce losses from begomoviruses. Host-free periods have been shown to be an effective measure for controlling the whitefly vector. Insecticide spray programs have been largely ineffective. Control weeds, incorporate crop debris immediately after harvest and avoid planting near infected solanum crops. In protected culture, the use of a 50+ mesh/297-micron screen is recommended to exclude whiteflies and aphids. The use of fans at the entrances along with a comprehensive hygiene program can aid in minimizing insect entry.

TYLCV: interveinal chlorosis and cupping of leaflets.

ToYVSV: pronounced vein-yellowing.

ToLCNDV: pronounced yellowing with leaf curling.

VIRAL
GEMINIVIRUS (*CURTOVIRUS*) DISEASE

Stunted plant with leaf curling.

CAUSAL AGENT
Beet curly top virus (BCTV)

VECTORS
Circulifer tenellus (beet leafhopper) (synonym = *Neoaliturus tenellus)*

Circulifer opacipennis

DISTRIBUTION
Mediterranean region, North America

SYMPTOMS
Typically, infected plants are erect and stunted; severely infected seedlings may die. Leaves thicken with their margins rolling upward as petioles curl downward. Later, leaves turn a dull-yellow with an accompanying purpling of leaflet veins. Very few fruit are produced and those that set before infection ripen prematurely. Fruit affected by this disease are dull, small and wrinkled with a dried appearance.

CONDITIONS FOR DISEASE DEVELOPMENT
This virus has a wide host range of 300 species from which it can be transmitted in a persistent manner only by beet leafhoppers. Sugar beet is a common host for both the virus and leafhoppers. Virus-carrying leafhoppers can be moved by wind into adjacent tomato fields and can also migrate from their overwintering weedy hosts to tomato fields in the spring. Patterns of infection in the field indicate a "raining" of virus-carrying leafhoppers. Little or no secondary spread occurs within tomato fields. Other common hosts for this virus are watermelon, cantaloupe, squash, pepper, spinach and beans.

CONTROL
Spray insecticides on weeds to control leafhoppers and avoid growing on land near beet fields and range land to reduce losses from beet curly top. Spraying tomato fields for leafhoppers will not control beet curly top. In areas where BCTV is present, transplant double rows of processing tomatoes to allow more individual plants to escape infection.

Severe beet curly top virus field infection. (Courtesy of Brenna Aegerter, University of California Division of Agriculture and Natural Resources)

GEMINIVIRUS (*CURTOVIRUS*) DISEASE

Severely stunted plant with necrotic foliage.

Prematurely ripe, red fruit. (Courtesy of Eugene Miyao, University of California Division of Agriculture and Natural Resources)

Curled leaflets with purple veins. (Courtesy of Brenna Aergerter, University of California Division of Agriculture and Natural Resources)

ILARVIRUS DISEASES

PMoV: leaflet and stem lesions. (Courtesy of Testi Valentino, Consorzio Fitosanitario Provinciale di Parma)

PMoV: leaflet with chlorotic rings and scarred, misshapen fruit. (Courtesy of Testi Valentino, Consorzio Fitosanitario Provinciale di Parma)

ToNSV: foliar necrosis. (Courtesy of Ozgur Bautman, University of Florida, Department of Plant Pathology, Southwest Florida Research and Education Center)

CAUSAL AGENTS

Parietaria mottle virus (PMoV)

Tobacco streak virus (TSV)

Tomato necrotic spot virus (ToNSV)

Tomato necrotic streak virus (TomNSV)

VECTOR

Mechanically transmitted

DISTRIBUTION

PMoV: France, Greece, Italy

TSV: Worldwide

ToNSV: USA (California)

TomNSV: USA (Florida)

SYMPTOMS

PMoV symptoms include mosaic or mottling of leaves, developing on to apical necrosis and dieback. Fruit develop chlorotic rings, which later become necrotic leading to fruit deformation. ToNSV and TomNSV symptoms are almost identical to one another and can be easily confused with the ilarvirus type species, Tobacco streak virus. Foliar symptoms of ToNSV and TomNSV infection include necrotic spots that tend to initiate at the base of leaflets and develop progressively outward. Infected upper foliage tends to twist and curl, and dieback of petioles and stems often occurs in severely infected plants.

CONDITIONS FOR DISEASE DEVELOPMENT

Little is known about the biology of ilarviruses because inoculum sources remain unknown. The host range for these viruses is not completely understood but it is known that PMoV infects two weed species: *Parietaria pensylvanica* and *P. officinalis*. Low levels of ToNSV infection have been found in pepper, onion and *Solanum nigrum* (black nightshade). Thrips and pollen are suspected to be involved in Tomato necrotic streak virus transmission, similar to other ilarviruses including Tomato necrotic spot virus and Tobacco streak virus. Mechanical transmission of PMoV and ToNSV also occurs quite readily.

CONTROL

Control of ilarviruses is challenging because the epidemiology of the diseases they cause is not well understood. Implement an IPM program including weed and insect control to manage ilarviruses.

ILARVIRUS DISEASES

TomNSV: leaflet necrosis. (Courtesy of Scott Adkins, U.S. Department of Agriculture, Agricultural Research Service)

ToNSV: leaf necrosis.

ToNSV: stem and leaf necrosis on an inoculated plant.

TomNSV: necrotic rings on fruit. (Courtesy of Scott Adkins, U.S. Department of Agriculture, Agricultural Research Service)

PELARGONIUM ZONATE SPOT

Leaflets with line patterns. (Courtesy of Marisol Luis, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza)

CAUSAL AGENT

Pelargonium zonate spot virus (PZSV)

VECTOR

Mechanically transmitted

DISTRIBUTION

France, Israel, Italy, Spain, USA (California)

SYMPTOMS

PZSV belongs to the family Bromoviridae (genus *Anulavirus*) and was originally recovered from *Pelargonium zonale,* but in the 1980s PZSV was found to be the causal agent of a disease in tomato. On tomato, symptoms of PZSV on leaves, stem and fruits are characterized by line patterns, and chlorotic and necrotic rings, together with plant stunting, leaf malformation and poor fruit set. Moreover, infected tomato plants exhibit stem necrosis, which may often result in plant death. PZSV symptoms are highly dependent on environmental conditions such as light intensity, temperature, and host species and are often weak under greenhouse conditions.

CONDITIONS FOR DISEASE DEVELOPMENT

In addition to tomato, natural infections of PZSV have been observed in artichoke (*Cynara cardunculus*), capsicum (*Capsicum annuum*), kiwifruit (*Actinidia deliciosa*), *Chrysanthemum coronarium,* and weeds (e.g., *Capsella bursa-pastoris, Diplotaxis erucoides, Picris echoides, Sonchus oleaceus*). Host range was essentially similar between different PZSV isolates (*Cucumis sativus*, *Solanum melongena*, *Datura stramonium*, *Nicotiana benthamiana*, *N. glutinosa* and *N. tabacum*) with the major differences being (i) isolates from Israel, France and California infect *N. benthamiana* whereas the Italian isolate does not, (ii) isolates from Israel, Spain and France all infect *D. stramonium* whereas the Italian isolate does not and (iii) reactions on cucumber plants to PZSV were not clear. PZSV can also be transmitted by grafting. Recent studies showed that PZSV is also transmitted vertically via both infected tomato seeds (virus not on seed surface) and pollen. It has been suggested that thrips could transport infected pollen grains and contribute to disease spread.

CONTROL

For the highly contagious, mechanically transmitted PZSV, control depends largely on a thorough sanitation program for workers, plant debris, tools, equipment and facilities because resistant cultivars are not yet available. The use of non-fat dry milk (3.5% protein) or a 20% bleach solution is effective for sanitizing tools for other mechanically transmitted viruses. Clearly, pollen and seed transmission of PZSV is of major concern for the tomato industry. PZSV transmission rate from seed to seedling was 11-29%, regardless of the seed-disinfection treatment. Thus, even low rates of viral seed transmission can facilitate the introduction of PZSV into new geographical areas. It is important to eradicate symptomatic plants and produce tomato hybrids in tomato production areas that are free of PZSV.

PELARGONIUM ZONATE SPOT

Expanding symptomatic leaflet. (Courtesy of Craig Sandlin, Syngenta)

Leaf and stem symptoms reproduced by mechanical inoculation. (Courtesy of Fernando Escriu, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza)

Chlorotic concentric rings on stem. (Courtesy of Craig Sandlin, Syngenta)

Necrotic zonate pattern on stem. (Courtesy of Marisol Luis, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza)

PEPINO MOSAIC

Yellow leaf spotting. (Courtesy of Kai-Shu Ling, U.S. Department of Agriculture, Agricultural Research Service)

Interveinal mosaic.

CAUSAL AGENT

Pepino mosaic virus (PepMV)

VECTOR

Mechanically transmitted

DISTRIBUTION

Worldwide

SYMPTOMS

Initially, this virus was confused with another *Potexvirus, potato virus X* (PVX), due to similar symptoms observed. PepMV symptoms are influenced by variety, age of plant and environment. Generally, symptoms are most easily recognized near the plant apex. The upper portions of the plant may become less dense and vigorous, developing a grayish cast. Affected foliage may bend downward and curl toward the stem. Emerging leaves may be distorted and needle-like. One or many small yellow leaf spots can also develop throughout the plant. These spots may enlarge while retaining their distinct margins. The entire plant may appear to turn yellow as disease progresses. If blistering on the leaves occurs, it appears slightly darker than the surrounding tissue. Mottling of varying degrees of severity can occur on ripening and mature fruit. Symptoms can initially remain restricted to a single tomato cluster, but eventually more clusters will show this abnormal coloring or marbling of the fruit.

CONDITIONS FOR DISEASE DEVELOPMENT

PepMV has a relatively narrow host range which does not include pepper. It is highly contagious via mechanical transmission. Disease expression is enhanced under low light conditions and during periods of slow plant growth. Under more favorable growing conditions infected plants can remain symptomless or the severity of visible symptoms may decrease. Regardless of the appearance of plants, PepMV cannot be eradicated from plants once they are infected. PepMV is easily transmitted by contact between healthy plants and contaminated hands, clothing, tools, equipment and trays. Bumblebees may possibly transmit PepMV indirectly during flower visits. PepMV can remain infective in plant debris that are not properly decomposed but the virus does not remain infective in soil. Irrigation water can be a source of PepMV in a protected culture environment if not properly treated with ultraviolet light.

CONTROL

Plant resistance is currently not an option in tomato. The best management to minimize infection by this disease is to implement a strict sanitation and hygiene program for plant debris, workers, tools, equipment and facilities. The use of non-fat dry milk (3.5% protein) or bleach (20%) solution effectively prevents transmission from tools, and non-fat dry milk is a safe and effective treatment for workers' hands. Many seed and fruit production companies implement policies that do not allow workers to bring fresh solanum fruit to their facilities because the fruit could be contaminated with PepMV and other pathogens. The virus can be seed-transmitted, albeit at very low rates, but all seed producers and commercial growers should start with clean seed.

In recent years, many tomato-growing companies across Europe are implementing a cross protection strategy against PepMV. The basis of this strategy is also known as an acquired immunity phenomenon, where a mild virus isolate/strain can protect plants against economic damage caused by a severe challenge strain/isolate of the same virus.

PEPINO MOSAIC

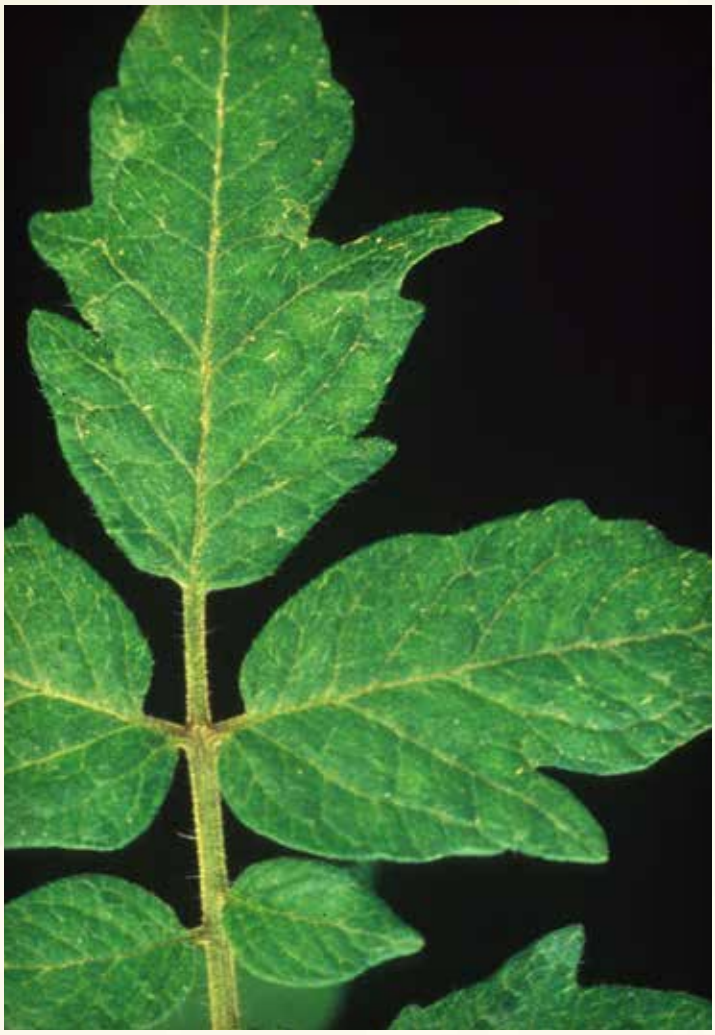
Necrotic stem lesions.

Necrotic, wrinkled fruit cluster.

Leaflet blistering and distortion.

Irregular fruit ripening. (Courtesy of Kai-Shu Ling, U.S. Department of Agriculture, Agricultural Research Service)

POTYVIRUS DISEASES



PVY: mild mottling of leaf after mechanical inoculation. (Courtesy of Tom Zitter, Cornell University)

CAUSAL AGENTS

Potato virus Y (PVY)

Tobacco etch virus (TEV)

VECTORS

Myzus persicae (green peach aphid)

Additional aphid species

DISTRIBUTION

Worldwide

SYMPTOMS

Disease symptoms reported on tomato range from a mild mottling and slight distortion of leaves to a severe mottling and crinkling of leaves. Overall, plants infected with potyviruses tend to have a bushy appearance and be lighter in color than healthy plants. Fruit are often reduced in size, and may be mottled and misshapen. Internally, fruit may develop necrosis which is commonly associated with irregular ripening. In general, the earlier a plant is infected, the greater the negative effect on growth and yield.

CONDITIONS FOR DISEASE DEVELOPMENT

Many species of weeds are hosts of potyviruses, which can be transmitted by at least ten species of aphids. Both PVY and TEV are transmitted in a non-persistent manner by virus-carrying aphids that move into tomato fields from nearby solanaceous weeds, peppers and other tomato fields that are already infected. Virus spread is often rapid and localized. Secondary infection may occur with aphids, or these viruses may be mechanically transmitted through staking, pruning and handling of infected plants. It is common to find plants infected with more than one potyvirus. CMV has also been associated with potyvirus infections.

CONTROL

Remove weed hosts which may harbor potyviruses. Avoid locating tomato productions near pepper crops because peppers can be a major source of potyviruses. Reducing the spread of the disease by controlling aphid population is very difficult and generally not practical. The use of silver plastic mulches may delay infection by potyviruses by repelling aphids that transmit these viruses during early plant development. The effectiveness of silver mulches decreases when more than 60% of the mulch is covered by foliage.

PVY: leaf mottling.

PVY: mild mottling and blistering of an artificially inoculated leaf.
(Courtesy of Tom Zitter, Cornell University)

POTYVIRUS DISEASES

TEV: stunted plant next to healthy plant. (Courtesy of Tom Zitter, Cornell University)

TEV: leaf distortion. (Courtesy of Tom Zitter, Cornell University)

TEV: mottling and leaf distortion.

TEV: mottled fruit. (Courtesy of Tom Zitter, Cornell University)

TOBAMOVIRUS DISEASES

"Fernleaf" symptoms.

Chlorotic mottling and blistering of leaflet.

Leaf distortion and chlorotic mottling.

CAUSAL AGENTS

Tobacco mild green mosaic virus (TMGMV)

Tobacco mosaic virus (TMV)

Tomato brown rugose fruit virus (TBRFV)

Tomato mosaic virus (ToMV)

Tomato mottle mosaic virus (ToMMV)

VECTOR

Mechanically transmitted

DISTRIBUTION

ToMV, TMV, TMGMV: Worldwide

ToMMV: Brazil, China, Iran, Mexico, USA

TBRFV: Israel, Jordan, Kuwait, Saudi Arabia

SYMPTOMS

Foliar mosaic is a characteristic symptom of tobamovirus infection. Leaf symptoms can also include chlorotic mottling, upward leaf rolling, malformation, reduced leaf size and necrosis. Symptom development depends on virus strain, cultivar, age at infection, temperature and light. Early infection often stunts plants. Under cool conditions, emerging leaves may exhibit a shoestring or "fernleaf" appearance. During high temperatures and under high light conditions, foliar symptoms may be masked, especially in field production. Fruit can occasionally ripen unevenly and exhibit internal browning of the fruit wall, known as brown wall. Brown wall typically occurs on the first two fruit clusters several days prior to foliar symptoms. Some varieties with heterozygous resistance to ToMV will show necrotic streaks or spots on stems, petioles, fruit and foliage. ToMV is the most prevalent tobamovirus infecting tomato.

CONDITIONS FOR DISEASE DEVELOPMENT

Tobamoviruses that infect tomato have a wide host range that includes many agricultural crops and weeds, all of which can serve as inoculum sources. These viruses are readily transmitted mechanically by workers, tools and equipment during plant handling and are considered some of the most stable and infectious viruses known. Infested debris from a previous crop can be a source of inoculum in subsequent crops. Chewing insects can spread these viruses but are not considered a major source of infection. Tobamoviruses can be seedborne in tomato but infection and spread most likely occur when seedlings are thinned in the nursery or transplanted.

CONTROL

Planting resistant varieties is generally the best control strategy, although strains capable of overcoming resistance have been reported and new tobamoviruses (e.g., TBRFV) can also overcome deployed resistance genes. Avoid fields where the previous crop was infected. Implement a comprehensive crop inspection and hygiene program, including tool and equipment sanitation, worker hand-washing and protective clothing. In protected culture, use non-fat dry milk (3.5% protein) and chemical disinfectants during plant handling, pruning and leaf removal. Use hard plastic trays, which can be cleaned and sanitized more effectively than Styrofoam. Do not reuse growing media.

TOBAMOVIRUS DISEASES

Leaflet distortion with mottling and blistering.

ToMMV: leaf mosaic. (Courtesy of Kai-Shu Ling, U.S. Department of Agriculture, Agricultural Research Service)

Uneven ripening.

Uneven ripening and necrotic lesions.

Mild mosaic caused by TBRFV. (Courtesy of David Levy, Hazera Seeds, Ltd.)

TBRFV: uneven ripening and necrotic lesions. (Courtesy of David Levy, Hazera Seeds Ltd.)

TOMATO BUSHY STUNT

Leaf necrosis developing on an inoculated plant. (Courtesy of Marisol Luis, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza).

CAUSAL AGENT

Tomato bushy stunt virus (TBSV)

VECTOR

Mechanically transmitted

DISTRIBUTION

Argentina, Canada, France, Germany, Great Britain, Italy, Mexico, Morocco, Portugal, Spain, Tunisia, USA (California)

SYMPTOMS

Initial symptoms may vary from irregular light banding patterns on leaves to necrotic lesions or rings. Leaves soon turn from green to pale-yellow, sometimes with interspersed green patches. Eventually affected leaves may drop from the plant. New leaves frequently are twisted and have necrotic tips. An abundance of side shoots develops, giving affected plants a bushy, stunted appearance. As disease progresses, lower leaves turn chlorotic and purple. Infected seedlings that receive too much fertilizer may develop lesions on the epicotyl at the soil line, resulting in girdling and seedling death. Fruit symptoms can vary from chlorotic blotches to rings or line patterns.

CONDITIONS FOR DISEASE DEVELOPMENT

TBSV is a very stable virus with a very diverse natural host range. This virus is soilborne and readily transmissible in water. A natural vector of TBSV is currently unknown but it is believed that TBSV infects plants through wounds in the roots. TBSV has been found in river water so it is possible that it is spread in irrigation water.

CONTROL

Avoid planting in soils known to contain TBSV. This virus is difficult to control once it is present in a soil.

Fruit necrosis on an inoculated plant. (Courtesy of Marisol Luis, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza)

TOMATO BUSHY STUNT

Discoloration and deformation of fruit. (Courtesy of Marisol Luis, Instituto Universitario de Investigación Mixto Agroalimentario de Aragón, Universidad de Zaragoza)

Discoloration and necrotic rings on fruit. (Courtesy of Mary Ann Hanson, Virginia Polytechnic Institute and State University, Bugwood.org)

TOMATO DOUBLE-VIRUS STREAK

Necrotic spots on leaflets and necrotic streaks on petiole.

SYNONYM = DOUBLE STREAK

CAUSAL AGENTS

Tomato mosaic virus (ToMV)

Potato virus X (PVX)

VECTOR

Mechanically transmitted

DISTRIBUTION

Worldwide

SYMPTOMS

Symptoms occur on young leaves as small, brown spots and on petioles and stems as narrow dark-brown streaks. Lesions may coalesce to form large, dead areas and leaves that curl downward. Lesions on the fruit are small and only skin deep but may run together, forming large lesions. These lesions give a greasy appearance to the fruit which eventually may become rough and misshapen.

CONDITIONS FOR DISEASE DEVELOPMENT

ToMV and PVX are individually easily mechanically transmitted by greenhouse workers, their tools and on cultivation equipment; however, for this disease to occur both viruses must be present in the same plant. If young plants already infected with ToMV become infected with PVX, double streak occurs. Symptom severity is affected by virus strain, plant age and day length.

CONTROL

Grow ToMV-resistant varieties, rogue infected plants and avoid handling potatoes prior to working with tomatoes to reduce losses from double streak. Also, do not plant in fields that were planted to potatoes the previous season, and sterilize tools immediately after handling infected plants to reduce spread and disease incidence.

Necrotic lesions on fruit.

TORRADOVIRUS DISEASES

ToANV: severe apical necrosis. (Courtesy of Craig Sandlin, Syngenta)

ToANV: leaf necrosis. (Courtesy of Craig Sandlin, Syngenta)

CAUSAL AGENTS

Tomato apex necrosis virus (ToANV)
[also known as *Tomato marchitez virus* (ToMarV)]
Tomato chocolate spot virus (ToCSV)
Tomato necrotic dwarf virus (ToNDV)
Tomato torrado virus (ToTV)

VECTORS

Bemisia tabaci (synonym = *B. argentifolii;* common names = sweet potato or silverleaf whitefly)
Trialeurodes abutilonea (banded winged whitefly)
Trialeurodes vaporariorum (greenhouse whitefly)

DISTRIBUTION

ToANV (ToMarV): Mexico
ToCSV: Guatemala
ToTV: Australia, Central America, Europe, South Africa
ToNDV: USA (California)

SYMPTOMS

Necrotic lesions initially appear at the base of leaflets and later expand to cover petioles, leaves and stems. The shoot apex may eventually become necrotic, resulting in dieback. Other symptoms may include mild leaf mottling and stunting of the plant. Foliar symptoms caused by Torradoviruses can be easily confused with symptoms caused by tomato spotted wilt virus. Fruit may develop a necrotic, corky ring pattern and be deformed.

CONDITIONS FOR DISEASE DEVELOPMENT

Torradovirus host range appears to be limited to Solanaceae. ToTV has been shown to infect several weed species, sometimes asymptomatically. In addition to whitefly transmission, torradoviruses can be mechanically transmitted at a very low rate. There is no evidence that these viruses are seed-transmitted.

CONTROL

Resistance to ToANV (ToMarV) exists in indeterminate tomato types whereas no resistance is known in determinate (e.g., processing) tomato varieties, which appear to be extremely susceptible. Ensure transplants are free of disease. A host-free period of two to three months will reduce whitefly populations leading to a decrease in virus incidence. Implement an insecticide program, weed control and a comprehensive sanitation program. In protected culture (e.g., transplant operations, commercial production) facilities utilize a screen size of 50-mesh (297-microns) to reduce whitefly entry.

TORRADOVIRUS DISEASES

ToANV: necrotic, corky ring pattern on fruit.

ToMarV: necrosis developing at the base of leaflets.

ToMarV: agroclone-inoculated plant exhibiting leaflet necrosis. (Courtesy of Bryce Falk and Inmaculada Ferriol Safont, UC-Davis)

ToMarV: agroclone-inoculated plant exhibiting leaflet necrosis. (Courtesy of Bryce Falk and Inmaculada Ferriol Safont, UC-Davis)

TORRADOVIRUS DISEASES

ToTV: necrotic scarring of fruit.

ToTV: necrotic scarring of fruit cluster.

ToNDV: necrosis developing on leaflets. (Courtesy of Bill Wintermantel, U.S. Department of Agriculture, Agricultural Research Service)

TOSPOVIRUS DISEASES

GBNV: necrotic ringspots on leaflets and petioles. (Courtesy of Rakesh Kumar, Indian Agricultural Research Institute)

GBNV: Note uneven ripening and ringspots.

CAUSAL AGENTS		DISTRIBUTION
Capsicum chlorosis virus	CaCV	Asia, Australia
Groundnut bud necrosis virus (synonym = *Peanut bud necrosis virus*)	GBNV (PBNV)	Asia
Groundnut ringspot virus	GRSV	Asia, USA (Florida)
Impatiens necrotic spot virus	INSV	Worldwide
Tomato chlorotic spot virus	ToCSV	Asia, USA (Florida)
Tomato necrotic ringspot virus	TNRV	Asia
Tomato spotted wilt virus	TSWV	Worldwide

VECTORS

Frankliniella occidentalis (western flower thrips)

Thrips tabaci (onion thrips)

Several additional thrips species

SYMPTOMS

Symptoms tend to be more severe on plants infected when young. Symptom expression can also vary with variety. Plants infected when young may exhibit stunting and inward cupping of leaves that develop a bronze cast followed by dark spots. Other characteristic tospovirus symptoms include mottling, chlorotic and necrotic leaf spots, which often lead to chlorosis and necrosis of entire leaves. Concentric ring spots are also a characteristic tospovirus symptom. Tospovirus symptoms can manifest as necrosis of growing tips, leading to bud necrosis. On fruit, symptoms can manifest as concentric rings that become necrotic.

CONDITIONS FOR DISEASE DEVELOPMENT

TSWV has a wide host range, whereas the host range of most other tospovirus species is more limited. Tospoviruses are transmitted in a persistent manner by several thrips species. Larvae acquire the virus after short feeding periods, and adult thrips transmit these viruses for the duration of their lives. The presence of infected weeds or mature plants in adjacent fields can pose a threat to newly planted crops. 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. In protected culture (transplant operations, commercial production) facilities utilize a minimum screen size of 72-mesh (192-microns) and implement a comprehensive hygiene program to minimize tospovirus infection by excluding thrips. 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. Biological control agents have also been successfully implemented in protected culture. Avoid planting downwind of ornamental or older crops, which can serve as reservoirs for thrips and tospovirus. Tospovirus resistance is currently available for TSWV in tomato.

TOSPOVIRUS DISEASES

GRSV: necrotic ringspots on young fruit.

ToCSV: necrotic ringspots on young, misshapen fruit. (Courtesy of Scot Adkins, U.S. Department of Agriculture, Agricultural Research Service)

INSV: chlorosis and uneven ripening. (Courtesy of Mary Ann Hanson, Virginia Polytechnic Institute and State University, Bugwood.org)

TNRV: chlorotic and necrotic ringspots on leaflet.

TOSPOVIRUS DISEASES

TNRV: chlorotic and necrotic ringspots on leaf.

TNRV: chlorotic ringspots on fruit.

TSWV: necrotic spots and leaf necrosis.

TOSPOVIRUS DISEASES

TSWV: discolored fruit.

TSWV: uneven ripening and ringspot formation.

COMMON VECTORS OF TOMATO VIRUSES

Myzus persicae: green peach aphid nymphs.

Myzus persicae: green peach aphid winged adult.

Aphids, whiteflies, thrips and leafhoppers are among the more common vectors of viruses that cause severe damage in tomato. The mode of transmission can be unique to each virus and vector combination. Transmission ranges from non-persistent or passive transport of viral particles on external mouth parts to the more complex internal virus-vector relationships characteristic of persistent movement. In non-persistent movement, virus particles are acquired during feeding or probing of infected plants and carried on mouth parts to the next feeding site. Virus acquisition takes place within seconds and transmissibility lasts several hours to a day. Longer acquisition and inoculation periods characterize persistent transmission. The virus is acquired during feeding, but before transmission can occur, the particles must move through the digestive system and into the salivary glands. New infections occur during feeding when viruliferous vectors inject viable virus particles with saliva into the plant cells or vascular tissues of healthy plants. In general, once they become infective, persistent vectors can transmit viruses for the duration of their adult lives.

Aphids: Aphids are small, pear-shaped, gregarious insects. They can lay eggs and, in warm climates, 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. Aphids usually invade fields as winged adults. Once established, aphids can be found on the growing points and on the underside of newer leaves.

Aphids can cause significant damage to tomato, causing spotting and chlorosis of leaves, leaf curling and distortion and abscission of flowers. The fungi that cause a sooty mold can grow on the sugary honeydew excreted by aphids, reducing fruit quality. Two common aphid pests of tomato are the potato aphid (*Macrosiphum euphorbiae*), which is large (3 mm) and colored pink or green, and the green peach aphid (*Myzus persicae*), which is smaller (1.5 mm) and colored light to dark green.

The number and diversity of viruses vectored by aphids far exceed those moved around by other vectors. Aphids transmit viruses in both a persistent and non-persistent manner. There is no evidence to suggest the viruses are carried to the next generation via eggs. The viruses carried passively by aphids to tomato can be acquired and transmitted within seconds and include Cucumber mosaic virus, Tobacco etch virus, and Alfalfa mosaic virus. Viruses persistently transmitted by aphids include Potato leaf roll virus and Beet western yellows virus, neither of which is a major problem in tomato.

Whiteflies: 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), Mediterranean (MED: formerly termed Q biotype)] and has emerged as an invasive pest to spread viruses worldwide. Over 500 plant species including weeds, vegetable, ornamental and agronomic crops are attacked. Immature and adult whiteflies colonize the underside 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 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. Whiteflies feed on phloem and produce sugary honeydew on leaves and fruit. Sooty mold fungi colonize the honeydew, reducing fruit quality and yield. Whitefly infestations can also slow plant growth and cause stunting and defoliation.

COMMON VECTORS OF TOMATO VIRUSES

The viruses transmitted by whiteflies are very important in tropical and subtropical regions, but are not confined to these areas. In tomato, *B. tabaci* is an important vector of begomoviruses such as Tomato yellow leaf curl, Tomato mottle virus, and the numerous begomoviruses found throughout Mexico, and Central and South America. *T. vaporariorum* was shown to transmit Tomato infectious chlorosis virus, a new disease in California tomato production regions. These viruses are carried persistently or semi-persistently. There is no evidence to suggest that these viruses are passed to the offspring of infected adult whiteflies.

Thrips: Five species of thrips are commonly found on tomato. The western flower thrips (*Frankliniella occidentalis*) is native to the western USA, but has been introduced into many regions worldwide. The flower thrips (*F. tritici*), the tobacco thrips (*F. fusca*) and the onion thrips (*Thrips tabaci*) as well as the western flower thrips mainly feed on flowers and small fruit. The greenhouse thrips (*Heliothrips haemorrhoidalis*) is found in greenhouses worldwide where it causes damage to a wide range of ornamental and vegetable plant species. On tomato, the greenhouse thrips mainly feeds on foliage. 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. Egg deposition and subsequent feeding by larvae causes discoloration, spotting and scarring in developing fruit.

Several species of thrips transmit tospoviruses. Thrips larvae acquire tospoviruses after short feeding periods and transmit these viruses primarily as adults. Occasionally transmission occurs in nymphs. These viruses are not passed to the next generation; however, there is some evidence to support replication in the vector. The western flower thrips and the onion thrips are the major vectors of Tomato spotted wilt virus. Thrips can also spread pollen-transmitted viruses while feeding and have been implicated in the spread of Tomato double virus streak.

Leafhoppers: Leafhoppers are found in warm, dry regions worldwide. They are wedge-shaped, green to greenish-yellow to brown and up to 3 mm long. Nymphs are similar in shape to adults except they lack fully developed wings. Leafhoppers have a very wide host range, including numerous weeds and vegetables. Leafhoppers feed on phloem tissues leaving pale, circular spots or peppery specks in 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 favorable environmental conditions. In California, leafhoppers overwinter in weeds, especially those in the foothills of the Sierra Nevada and Coast mountain ranges. In spring, as weeds die, leafhoppers move into adjacent tomato fields.

The beet leafhopper (*Circulifer tenellus*; synonym = *Neoaliturus tenellus*), transmits Beet curly top virus to tomato. This virus is acquired and transmitted persistently by immature and adult stages of the beet leafhopper. Once acquired, the virus can be transmitted throughout adult life but is not passed to the next generation via the egg. The California Department of Food and Agriculture conducts yearly surveys for beet leafhoppers and follows an insecticide spray program to control populations of beet leafhoppers which helps growers manage beet curly top in processing tomato. Several species of leafhopper vector tomato big bud, a disease caused by phytoplasmas.

Bemisia tabaci: last instar and cast skin. (Courtesy of Lance Osborne, University of Florida, Agricultural Research and Education Center)

Bemisia tabaci: last instar. (Courtesy of Lance Osborne, University of Florida, Agricultural Research and Education Center)

COMMON VECTORS OF TOMATO VIRUSES

Bemisia tabaci: several instar stages are present. Note wings on adult that is ready to emerge. (Courtesy of Lance Osborne, University of Florida, Agricultural Research and Education Center)

Bemisia tabaci: sweet potato whitefly.

Bemisia tabaci: late instar nymphs. (Courtesy of David Riley, University of Georgia, Bugwood.org)

Bemisia tabaci biotype B (formerly *B. argentifolii*). (Courtesy of Scott Bauer, USDA Agricultural Research Service, Bugwood.org)

COMMON VECTORS OF TOMATO VIRUSES

Trialeurodes vaporariorum: greenhouse whitefly.

Frankliniella sp: adult thrips. (Courtesy of Bruce Watt, University of Maine, Bugwood.org)

Frankliniella occidentalis: western flower thrips nymph.

Circulifer tenellus (synonym = *Neoaliturus tenellus*): beet leafhopper.
(Courtesy of A. C. Magyarosy, Bugwood.org)

VIROIDS

(POSPIVIROIDAE)

CITRUS EXOCORTIS

COLUMNEA LATENT

MEXICAN PAPITA

PEPPER CHAT FRUIT

POTATO SPINDLE TUBER

TOMATO APICAL STUNT

TOMATO CHLOROTIC DWARF

VIROIDS (POSPIVIROIDAE)

CLVd: petiole and leaflet necrosis on an inoculated plant.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

CLVd: necrosis developing on sepals of an inoculated plant.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

CAUSAL AGENTS:		DISTRIBUTION:
Citrus exocortis viroid	CEVd	Worldwide
Columnea latent viroid	CLVd	Worldwide
Mexican papita viroid (synonym = *Tomato planta macho viroid)*	MPVd (TPMVd)	Canada, Mexico
Pepper chat fruit viroid	PCFVd	Thailand, The Netherlands
Potato spindle tuber viroid	PSTVd	Worldwide
Tomato apical stunt viroid	TASVd	Ghana, Indonesia, Israel, Ivory Coast, Senegal, Tunisia
Tomato chlorotic dwarf viroid	TCDVd	Czech Republic, France, India, Mexico, Slovenia, USA (Arizona, Colorado)

SYMPTOMS

Symptoms caused by viroids can be easily confused with symptoms caused by viruses. Similar to viruses, viroid symptom expression can be impacted by plant variety, age, vigor, and environmental conditions. Symptoms manifest in tomato as chlorosis of young terminal shoots, chlorosis which can become pronounced in the form of bronzing or purpling of the leaves, stunting, leaf bunchiness, leaf epinasty, fruit distortion and color break, and necrosis of stem and leaves, which ultimately can lead to plant death. Symptomology among the viroids are not distinguishable from one another and molecular tools are necessary to provide an accurate identification of a specific viroid.

CONDITIONS FOR DISEASE DEVELOPMENT

Viroids are the smallest infective agents known to cause plant disease. They consist of single-stranded RNA and their origins remain unknown. Viroids can infect plant cells and incite disease. All tomato viroids are mechanically transmissible. Viroids are not known to be transmitted by insects, although some insects (e.g., aphids) have been implicated in the spread of viroid diseases. Several ornamental plant species (e.g., *Vinca minor, Petunia x hybrida, Cestrum* spp., *Verbena* spp., *Brugmansia* spp. and *Physalis peruviana*) have been shown to be hosts of viroids that infect tomato.

CONTROL

Breeding for resistance is often a desired trait for disease management but currently no sources of resistance have been identified. Potential sources of viroid inoculum include wild and ornamental hosts, grafted nursery plants and seed that has recently been implicated. Management of the disease relies on visual inspection followed by rogueing infected plants. The use of protective clothing and the implementation of a hygiene program for people, tools and structure is an excellent means to reduce the introduction and spread of viroids in a protected culture environment.

VIROIDS (POSPIVIROIDAE)

CLVd: pedicel necrosis and immature fruit distortion on an inoculated plant. (Courtesy of Kanungnit Reanwarakorn, Kesasart University)

CLVd: infected plant growing inside a net-cage. (Courtesy of David Levy, Hazera Seeds Ltd.)

MPVd: chlorotic leaves with necrosis.

MPVd: necrosis on foliage becomes more pronounced over time.

VIROIDS (POSPIVIROIDAE)

PCFVd: leaf and stem necrosis on an inoculated plant.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

PCFVd: severe leaf and petiole necrosis on an inoculated plant.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

PCFVd: inoculated plant with stem lesions.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

PCFVd: flower bud necrosis on an inoculated plant.
(Courtesy of Kanungnit Reanwarakorn, Kesasart University)

VIROIDS (POSPIVIROIDAE)

PSTVd: leaf bunchiness caused by shortening of internodes.
(Courtesy of Kai-Shu Ling, U.S. Department of Agriculture, Agricultural Research Service)

PSTVd: leaf bunchiness with chlorosis and necrosis.
(Courtesy of Kai-Shu Ling, U.S. Department of Agriculture, Agricultural Research Service)

TCDVd: chlorotic leaves with necrotic lesions.

TCDVd: leaf chlorosis.

ABIOTIC

DISORDERS

GENETIC DISORDERS

ENVIRONMENTAL STRESSES

INSECT DAMAGE

PLANT NUTRIENT DISORDERS

PESTICIDE INJURY

UNKNOWN ETIOLOGY

GENETIC DISORDERS

Autogenous necrosis: tan-brown necrotic lesions.

Autogenous necrosis: severe leaf necrosis.

GENETIC DISORDERS

Autogenous Necrosis

Fruit Pox

Gold Fleck

Zebra Stem

Zebra Stripe

CAUSAL AGENT

Genetic

DISTRIBUTION

Worldwide

SYMPTOMS

Autogenous Necrosis: First symptoms appear when plants are six to ten weeks old and manifest as a yellowing of upper leaf surfaces that progresses to necrosis over time. Tissue necrosis tends to be more prominent on lower leaf surfaces and typically starts at the distal end of leaflets. As lesions coalesce, leaves become necrotic and die. Typically, this disorder progresses from older foliage to younger foliage.

Fruit Pox: First symptom on green fruit is small, clear or tan, oval or slightly elongated lesions. As fruit mature these lesions enlarge and cause the fruit epidermis to rupture which gives fruit a necrotic, corky appearance.

Gold Fleck: First symptom on immature green fruit is circular, dark-green spots on fruit surfaces. As fruit mature these spots change from dark-green to light-tan and then to golden yellow on ripe red fruit.

Zebra Stem: Foliar symptoms appear as small, necrotic spots along the veins of expanding leaflets. Later, the spots coalesce and leaflets twist and die. As stem lesions develop, they coalesce to form wide, brown, necrotic bands, manifesting as a concentric ring pattern.

Zebra Stripe: Symptoms appear as a series of dark-green spots on fruit which may coalesce to give fruit a green-striped appearance. Depending on variety, these stripes may disappear as fruit ripens.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Autogenous Necrosis: This genetic disorder is caused by an incompatible reaction between a gene (*Cf-2*) for resistance to *Passalora fulva,* causal agent of leaf mold, and a tissue necrosis gene that causes necrotic spots on the foliage under certain environmental conditions.

Fruit Pox and Gold Fleck: Although these disorders commonly occur on the same fruit, they are distinct and susceptibility to both is genetically inherited. Their development is thought to be more severe when fruit are exposed to high temperatures and plants and fruit are growing rapidly. However, more work is still needed to fully understand the cause. Tomato varieties differ greatly in their susceptibility, with some varieties developing only a few lesions or gold flecks, while others develop many.

Zebra Stem: This disorder is associated with tomato varieties that are homozygous for *Pto* gene, which confers resistance to *Pseudomonas syringae* pv. *tomato* race 0. This condition is also linked to sensitivity to fenthion, an insecticide.

Zebra Stripe: Symptom expression depends on variety and temperature. High temperatures can trigger zebra stripe.

CONTROL

Grow varieties that are tolerant to these genetic disorders. Tomato hybrids that are heterozygous for *Pto* gene will not develop zebra stem.

GENETIC DISORDERS

Fruit pox: dark-green lesions on immature fruit.

Fruit pox: necrotic lesions on red fruit.

Gold fleck: dark-green spots on immature green fruit.

Gold fleck: light-tan and golden-yellow spots on ripe red fruit.

GENETIC DISORDERS

Zebra Stem: stem with mild banding pattern.

Zebra Stem: stems with concentric rings banding pattern.

GENETIC DISORDERS

Zebra Stripe: striping on mature green fruit.

Zebra Stripe: fruit with zebra stripe symptoms.

ENVIRONMENTAL STRESSES

Catface: on immature green fruit.

Catface: on ripe red fruit.

ENVIRONMENTAL STRESSES

Black Shoulder

Catface

Chilling Injury

Cracking

Edema

Gray Wall

CAUSAL AGENT

Environmental stress

DISTRIBUTION

Worldwide

SYMPTOMS

Black Shoulder: Black Shoulder is characterized by the presence of dark-grey to black spots, streaks, or irregularly sized lesions on the shoulders of fruit in the area surrounding the stem scar. Lesions may appear shriveled or sunken, indicating localized decay. The lesions and fungal growth are attributed to *Alternaria alternata,* an opportunistic pathogen infecting fruit tissue exposed by shoulder cracking.

Catface: Typical symptoms of this disorder are misshapen fruit with scarred areas and lines that often radiate from the blossom end of the fruit. Catface is often associated with cold temperatures during flowering. This disorder is especially important on large-fruited tomatoes and the severity is variety-dependent.

Chilling Injury: Symptoms include tissue browning, pitting and discoloration of skin and uneven ripening. Fruit susceptibility to fungal decay pathogens increases.

Cracking: Two types of cracking occur on fruit. Radial cracking is a splitting of the epidermis that radiates from the calyx end to the blossom end of fruit. Concentric cracking is a splitting of the epidermis in circular patterns around the calyx end of fruit. Cracking usually does not occur until fruit reach maturity.

Edema: This disorder is characterized by a blistered appearance on lower leaf surfaces resulting from ruptured guard cells.

Gray Wall: Also referred to as uneven ripening. Symptoms are first observed as flattened, blotchy, brownish-gray areas that develop on green fruit. As fruit mature these blotchy areas remain gray or turn yellow, resulting in uneven ripening. When fruit are cut open, dark-brown vascular tissue can be seen in the fruit walls.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Black Shoulder: Drastic changes in environmental conditions and mechanical injuries can lead to shoulder cracking. If mature green fruit with shoulder cracking remain in the field and are subject to cold (<10°C) temperatures, fruit rot caused by *Alternaria alternata* is likely to occur, leading to development of black shoulder symptoms. Although green fruit are more susceptible to shoulder cracking, both green and ripe fruit can develop black shoulder.

Catface: Cold (<10°C) weather during flowering and fruit set is known to enhance this disorder. Also, high soil nitrogen levels and any disturbance to the flower parts during anthesis can increase incidence of catface.

Chilling Injury: Tomatoes are highly sensitive to chilling injury when exposed to temperatures between -1 and 12.5°C. Both exposure time (e.g., two weeks at 10°C or seven days at 5°C) and fruit maturity can influence severity of damage to fruit. Green fruit are more susceptible to chilling injury than ripe fruit. Chilling injury can also occur during postharvest storage or in transit.

ENVIRONMENTAL STRESSES

Cracking: Susceptibility to cracking is related to the strength and stretching ability of the fruit epidermis. Periods of slow fruit growth followed by rapid growth resulting from large differences in day and night temperatures cause fruit cracking. Periods of low soil moisture followed by heavy rain or irrigation also cause this disorder.

Edema: This disorder develops when roots take up water faster than it can be used by the plant or transpired through the leaves. The main cause is excessively high moisture levels in soil or air. Edema typically occurs when the soil temperature is warm and the air temperature is cool. Prolonged periods of high humidity favor this disorder.

Gray Wall: Environmental factors that appear to be associated with this disorder include high nitrogen content of soil, low potassium content of soil, high soil moisture, high humidity, cool weather, low light intensity and soil compaction. In addition, certain bacteria and tobacco mosaic virus may play a role in occurrence and development of gray wall.

CONTROL

Black Shoulder: Prevent shoulder cracking of green fruit. Fresh market tomato varieties differ in response to black shoulder-conducive weather conditions; therefore, plant varieties that are less prone to shoulder cracking or that ripen evenly to reduce incidence of this disorder. Over-fertilization may increase the severity of black shoulder when environmental conditions are conducive for shoulder cracking. Avoid mechanical injuries to fruit.

Catface and Cracking: Grow tolerant varieties to reduce losses from these two disorders. Manage soil nitrogen levels and soil moisture content in field-grown tomatoes. In protected culture, manage day and night temperatures to reduce losses from catface and cracking.

Chilling Injury: In protected culture, maintain climate control systems in good working order. Ensure roof vents are working properly and are not a source of unwanted cold air. Ensure refrigeration systems in post-harvest storage facilities and long-haul trucks are properly maintained to minimize chilling injury.

Edema: In protected culture, reduce relative humidity by providing adequate ventilation and air movement. Manage ambient night temperature to prevent wide fluctuations and soil substrate moisture content.

Gray Wall: Grow gray wall-tolerant varieties.

Chilling Injury: greenhouse-produced fruit.

Chilling Injury: browning and pitting of greenhouse-produced fruit.

ENVIRONMENTAL STRESSES

Cracking: radial and concentric cracking.

Cracking: radial and concentric cracking.

Edema: callus-like growth on leaflet undersurface.

Edema: blistering on leaflet undersurface.

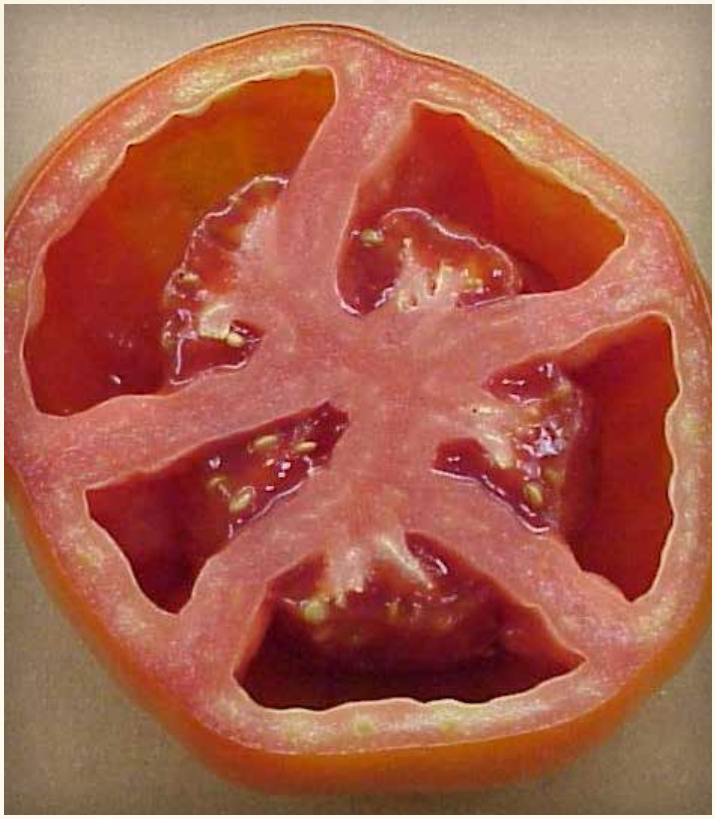
ENVIRONMENTAL STRESSES

Gray Wall: dark-brown vascular tissue in fruit walls.
(Courtesy of Ed Sikora, Auburn University, Bugwood.org)

Gray Wall: uneven ripening.
(Courtesy of Gary Vallad, University of Florida, Gulf Coast Research and Education Center)

Gray Wall: internal brown vascular tissue. (Courtesy of Gary Vallad, University of Florida, Gulf Coast Research and Education Center)

ENVIRONMENTAL STRESSES



Puffiness: incomplete locule and gel development.

Puffiness: incomplete locule and gel development.

ENVIRONMENTAL STRESSES (continued)

Little Leaf

Puffiness

Silvering

Sunscald

Yellow Shoulder

Zippering

CAUSAL AGENT

Environmental stress

DISTRIBUTION

Worldwide

SYMPTOMS

Little Leaf: This disorder manifests as interveinal chlorosis in young leaves. Later, severity of interveinal chlorosis on newly emerging leaves will increase but leaf veins will remain dark-green; leaflets will be distorted. Size of newly emerged leaves can be greatly reduced, too. Plant stunting may occur because of reduced terminal growth. Plants that have mild little leaf symptoms may still set blooms and develop fruit, but fruit are often distorted and cracked, making them unmarketable. Seed development is hindered. Plants with severe little leaf symptoms often form misshapen blooms and do not set fruit.

Puffiness: Typically, affected fruit have an angular appearance and are less dense than healthy fruit. Locule development is incomplete with very few seeds and little gel present.

Silvering: This disorder is also known as chimera or head silvering. Leaves develop gray-green to silver spots or patches and the entire leaf may become silver-like in color. Leaves can be smaller and develop blisters and stems may develop gray-green streaks. Flowers may be partially or completely sterile. Fruit may have gray-green streaks that turn pale-yellow at maturity.

Sunscald: 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 color than surrounding tissue. This area later collapses and becomes leathery. The affected area may turn black due to colonization by black mold fungi (e.g., *Alternaria alternata, Pleospora lycopersici, Stemphylium* species). Fruit near maturity are more sensitive to sunscald injury than immature fruit.

Yellow Shoulder: This disorder is characterized by the fruit shoulder (area adjacent to the stem end of fruit) not ripening completely and remaining green or yellow while the rest of the fruit ripens normally. Patches of the shoulder or the entire shoulder area may be affected. Yellow shoulder-affected areas may be associated with hardened, white fruit tissue.

Zippering: A thin, brown, necrotic scar that resembles a zipper will generally form at the stem end of a fruit and extend down the side of the fruit, sometimes all the way to the blossom scar. Sometimes more than one scar will develop on a fruit and occasionally scars do not extend to either the stem scar or blossom scar. Zipper severity can vary from thin to thick and from superficial to being so deep that the scar penetrates a fruit locule

ENVIRONMENTAL STRESSES

CONDITIONS FOR SYMPTOM DEVELOPMENT

Little Leaf: This disorder is associated with specific field conditions and specific soil microorganisms. Symptoms are often associated with warm soil, poor soil aeration (leading to high levels of water saturation), and pH in the neutral to alkaline range. Although the exact cause of this disorder is unknown, it is hypothesized that the condition of soil and the microorganisms that are supported in the rhizosphere play a role in symptom development. It is further hypothesized that the microorganisms in the rhizosphere of the plants produce and release compounds that are similar in structure to amino acids. These compounds are absorbed by the plants and the amino acid-like compounds trigger morphological responses and changes.

Puffiness: Factors that lead to poor seed set and gel formation are high and low temperature extremes, use of fruit hormones and soil moisture extremes. High soil nitrogen content may exacerbate development of this disorder.

Silvering: Although not well understood, silvering manifests when temperatures drop below 18°C. Less silvering develops in protected culture when night temperatures are managed well.

Sunscald: 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.

Yellow Shoulder: Varieties that do not ripen uniformly are more susceptible to environmental influences that contribute to the development of this disorder than varieties that ripen uniformly. If mature fruit are exposed to high temperatures or high levels of sunlight (due to insufficient foliar coverage), yellow shoulder symptoms may be more pronounced. Plants that are grown in soil with inadequate amounts of available potassium are also more susceptible to yellow shoulder.

Zippering: Zippering develops when the anthers attach to the ovary wall of a newly developing fruit. Cool temperatures and plant genetics are considered contributing factors for this disorder.

CONTROL

Little Leaf: Avoid fields that drain poorly, and monitor and optimize soil moisture to reduce incidence of little leaf. Decrease soil pH to help prevent occurrence of little leaf.

Puffiness: In protected culture, control temperature, humidity and soil substrate nutrition to reduce incidence of puffiness.

Silvering: Grow varieties that are resistant to silvering. In protected culture, avoid temperature-shocking plant apexes during ventilation of growing structures when susceptible varieties are grown.

Sunscald: Encourage abundant, healthy foliage with proper fertilization and irrigation. Grow disease-resistant varieties and follow an effective disease and pest management program to reduce losses due to sunscald. In protected culture, shade plants during summer, and prune and harvest plants carefully to minimize defoliation and fruit exposure to direct sunlight.

Yellow Shoulder: Grow varieties that ripen uniformly and manage available potassium levels in soil to reduce incidence of yellow shoulder.

Zippering: Grow varieties that are not prone to zippering. In protected culture, maintain day and night temperatures that are optimal for fruit set.

Silvering: foliage exhibiting a silver-green cast.

ENVIRONMENTAL STRESSES

Silvering: poor color development.

Sunscald: processing tomato.

Sunscald: fresh market tomato.

ENVIRONMENTAL STRESSES

Yellow shoulder. (Courtesy of Dominique Blancard, French National Institute for Agricultural Research)

Zippering: fruit with exposed locule. (Courtesy of Mathews Paret, University of Florida, North Florida Research and Education Center)

Zippering: thin, brown, necrotic scar with flower petals still attached. (Courtesy of Dan Egel, SW Purdue Agricultural Program)

CLOUDY SPOT

Yellow, irregular spots on fruit surface caused by stink bug feeding activity.

CAUSAL AGENT

Brown Stink Bug: *Euschistus servus*

Green Stink Bug: *Acrosternum servus*

Southern Green Stink Bug: *Nezara viridula*

Brown Marmorated Stink Bug: *Halyomorpha halys*

Additional Pentatomiadae species (stink bugs)

DISTRIBUTION

Worldwide

SYMPTOMS

Typically, white to yellow irregular spots develop just below the epidermis of fruit in response to stink bug feeding activity. These spots can be numerous if the level of insect feeding was high. When the fruit skin is peeled back, an area of white, glistening, spongy cells is apparent. When the fruit turns red, these areas remain light yellow. A puncture wound in the skin can generally be found in the middle of affected areas.

CONDITIONS FOR SYMPTOM DEVELOPMENT

When stink bugs feed on green fruit, they puncture the skin and secrete an enzyme that prevents development of normal color in ripening fruit. Overwintering stink bug adults become active in spring and fly into tomato fields from surrounding weeds. Edges of tomato fields nearest weedy areas are often the most affected by stink bugs. All growth stages of stink bug species can cause cloudy spot.

CONTROL

Establish a strong insect control program to reduce losses from stink bug feeding. Control weeds in areas surrounding tomato fields.

Stink bug and cloudy spot symptoms.

BLOSSOM-END ROT

CAUSAL AGENT
Calcium Deficiency

DISTRIBUTION
Worldwide

SYMPTOMS
The first visible symptom of blossom-end rot (BER) is a water-soaked area near the blossom scar of the fruit. This area later develops into a tan to brown, leathery, sunken lesion. Saprophytic fungi often colonize these lesions, which gives them a gray to black, velvety appearance. Sometimes an internal black rot of tissue in the center of fruit will develop with little or no external symptoms. Typically, immature green fruit that have begun sizing are first to develop symptoms of BER.

CONDITIONS FOR SYMPTOM DEVELOPMENT
Blossom-end rot is associated with insufficient calcium uptake and alternating periods of wet and dry soil. Though BER 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 BER. Stress associated with root damage, mild drought, high soil salinity, or excess nitrogen (excess ammonium) also may cause BER. Incidence of BER may increase when relative humidity remains high for prolonged periods due to reduced water uptake by plants.

CONTROL
Grow tolerant varieties to help reduce occurrence of BER. Drip irrigate to supply an even amount of water and apply lime to soils low in calcium. Avoid using ammonium sources of fertilizer or excess magnesium since both increase demand for calcium by plants and reduce availability of calcium in soil. Fertilize with calcium nitrate in areas where BER is known to occur. Irrigate during periods of dry weather and apply mulch to plant beds to provide more constant soil moisture to plants. Avoid saline soils and fields that are difficult to irrigate uniformly. Root injury caused by mechanical damage during cultivation or by disease can exacerbate BER and should be avoided.

Brown, leathery lesions developing on immature green fruit. Note water-soaking of fruit tissue immediately surrounding each lesion.

Sunken, necrotic lesions at blossom-end of fruit. Note colonization of necrotic tissue by a saprophytic fungus. (Courtesy of William M. Brown Jr., Bugwood.org)

Longitudinal section through center of fruit. Note that necrosis almost extends into the locule.

DEFICIENCIES and TOXICITIES

Nitrogen deficiency: light-green foliage.

Nitrogen deficiency: older leaves turn chlorotic then necrotic.

CAUSAL AGENT

Insufficient or excessive nutrients

DISTRIBUTION

Worldwide

SYMPTOMS

The following symptoms are indicative of possible plant nutrient deficiencies and toxicities; however, soil and foliar nutrient analyses should be conducted to determine nutrient content and calculate nutritional requirements.

Nitrogen (N): Plants under low nitrogen stress are smaller than normal and have an overall light-green color, especially in the lower leaves. Fruit are small with thin walls. Excess nitrogen fertilizer 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. Phosphorus toxicity is rare but when it does occur it can interfere with copper and zinc availability.

Potassium (K): Symptoms of potassium 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. Fruit disorders such as yellow shoulder, puffiness, gray wall, uneven ripening and internal whitening can result from potassium deficiency. Excess potassium is a rare condition but can disrupt uptake of other nutrients (e.g., Mg, Mn, Zn, Fe).

Calcium (Ca): Interveinal chlorosis and leaf margin necrosis occur at the growing points in calcium-deficient plants. Later, growing points die. Leaves can be distorted. Fruit may develop blossom-end rot. It is very uncommon to observe calcium toxicity.

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

Sulfur (S): Older leaves of sulfur-deficient plants turn light-green and spindly. Excess sulfur can cause extensive leaf yellowing, leading to scorched leaf edges.

Boron (B): When boron is deficient, older leaves turn yellow and brittle, and growing points become necrotic and die. Margins and leaf tips of mature leaves become necrotic. Fruit may develop scattered corky areas. Excess boron can cause yellowing of leaf tips, then leaf necrosis, moving from leaf margins inward.

Copper (Cu): Initially young leaves of copper-deficient tomato plants wilt and later turn bluish-green and curl upwards. Severely affected plants are stunted and chlorotic. Copper toxicity manifests as leaf chlorosis and necrosis, and reduced shoot growth.

Iron (Fe): Young leaves of iron-deficient tomato plants develop interveinal chlorosis followed by general yellowing. Leaf midribs usually remain green. Iron toxicity manifests as reduced growth and interveinal chlorosis.

Manganese (Mn): Young leaves deficient in manganese develop interveinal chlorosis followed by necrosis. Midribs of affected leaves remain green. Manganese toxicity manifests as brown to black lesions on older leaves but later progresses to younger leaves.

Molybdenum (Mo): Older leaves of molybdenum-deficient tomato plants develop leaf yellowing and marginal necrosis that eventually progresses to younger leaves. Molybdenum deficiency is rare in tomato. Molybdenum toxicity is also rare and only occurs after accumulation from continuous applications.

Zinc (Zn): Leaves of zinc-deficient tomato plants thicken and curl downward. Petioles may twist and older leaves develop orange-brown chlorosis. Zinc in excess is extremely toxic and can cause rapid plant death.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Nutrient deficiencies are most common in acid or alkaline soils due to immobilization 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 fertilizer. 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 fertilizer program and correct nutrient imbalances. Alter soil pH with addition of lime to acid soils or sulfur and acid-forming fertilizers to alkaline soils to increase nutrient availability..

Phosphorous deficiency. (Courtesy of Joshua Freeman, University of Florida, North Florida Research and Education Center)

Potassium deficiency: yellow shoulder. (Courtesy of Gerald Brust, University of Maryland, Central Maryland Research and Education Center)

DEFICIENCIES and TOXICITIES

Potassium deficiency: healthy tomato (top row), mild yellow shoulder (middle row) and severe yellow shoulder (bottom row). (Courtesy of Timothy Hartz, University of California)

Potassium deficiency: internal whitening of tomato. (Courtesy of Gerald Brust, University of Maryland, Central Maryland Research and Education Center)

Calcium deficiency: sidewall blossom-end rot. (Courtesy of Joshua Freeman, University of Florida, North Florida Research and Education Center)

Magnesium deficiency: interveinal chlorosis. (Courtesy of Bruce Watt, University of Maine, Bugwood.org)

DEFICIENCIES and TOXICITIES

Sulfur deficiency: plants are light-green and spindly. (Courtesy of Joshua Freeman, University of Florida, North Florida Research and Education Center)

Manganese deficiency: interveinal chlorosis. (Courtesy of Laixin Luo, China Agricultural University)

Manganese toxicity: brown, necrotic lesions.

Suspect iron deficiency: leaf yellowing. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

HERBICIDE and INSECTICIDE INJURY

2,4-D: leaf deformation and twisting.

2,4-D: vein proliferation, leaf puckering, and general distortion.
(Courtesy of Mary Ann Hanson, Virginia Polytechnic Institute and State University, Bugwood.org)

CAUSAL AGENTS

Herbicide Injury

Insecticide Injury

DISTRIBUTION

Worldwide

SYMPTOMS

Herbicide Injury: Contact herbicides, those that affect only tissues they contact, typically will cause chlorotic or necrotic spots on all parts of tomato plants. Multiple spots can result in deformation of affected tissues. Systemic herbicides, those that are translocated in tomato plants, tend to cause a variety of symptoms including stunting. These range from a general yellowing of foliage, to yellowing or necrosis of tissues in the center of the leaf, to a yellowing or necrosis of leaf veins that may expand to interveinal tissues. Systemic herbicides may also cause necrotic spots, leaf margin necrosis, twisting and upward or downward leaf cupping, as well as mild or severe deformation and swelling of stems and petioles. Root growth may be inhibited and roots may become stubby. Fruit symptoms range from development of excessively large or small, irregularly shaped fruit, to the development of flat or nippled blossom ends, or internal deformations.

2,4-D: This herbicide can cause stunting and new growth may be deformed. Leaves may curl or cup and take on a whip-like appearance. Leaves, petioles and stems may twist and become distorted.

Clomazone: This herbicide is an inhibitor of plant pigments. Leaves develop bleaching along leaf veins.

Glyphosate: Injury manifests as yellowing at the base of leaflets.

MCPA: This herbicide causes leaf and stem malformation. Stems may curl, twist, and droop whereas leaves may cup and crinkle. Fruit can also be misshapen.

Trifluralin: Lateral and secondary root development is limited. Roots that do form tend to be short and stubby.

Insecticide Injury: Insecticides typically cause leaf margin necrosis or necrotic lesions on foliage.

Acephate: Applications during high temperatures may cause curling and interveinal necrosis, especially on the distal end of leaflets.

Oxamyl: Foliar applications of this nematicide can cause leaf burn if Oxamyl is not rinsed well enough from the foliage.

Pylon: "Bleached" leaf spots and leaf burn may develop if this insecticide is applied when ambient temperature is high.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Pesticides labeled for use on tomato generally do not cause damage unless they are applied at excessive rates, at high temperatures, at the wrong stage of plant growth or during unfavorable weather conditions. Some pre-plant herbicides may remain active in the soil long enough to cause injury later when seed is sown or seedlings are transplanted. Damage may occur from herbicide drift when adjacent crops or weeds are sprayed. Usually damage from drift is most severe at the edge of the field closest to the pesticide application. Damage from pesticides may occur if spray equipment is not rinsed thoroughly after each pesticide application.

CONTROL

Use herbicides and insecticides as directed by the label and apply during appropriate weather conditions only. Store herbicides and insecticides according to label recommendations.

HERBICIDE and INSECTICIDE INJURY

Clomazone: bleaching of leaf veins. (Courtesy of Gerald Holmes, California State University, San Luis Obispo, Bugwood.org)

Glyphosate: chlorosis at the base of leaflets.

HERBICIDE and INSECTICIDE INJURY

Glyphosate: chlorosis at the base of leaflets. (Courtesy of David Langston, Virginia Tech Tidewater, Agricultural Research and Extension Center, Suffolk, VA, Bugwood.org)

MCPA: nipples formed on fruit.

Trifluralin: stem swelling.

HERBICIDE and INSECTICIDE INJURY

Acephate: curling and interveinal necrosis. (Courtesy of Bruce Watt, University of Maine, Bugwood.org)

Oxamyl: leaf margin necrosis.

Pylon: "bleached" leaf spots.

BLIND PLANT

"Blind" processing tomato plant lacking an apical meristem.

SYNONYM = BUDLESS PLANT

CAUSAL AGENTS
Unknown etiology

DISTRIBUTION
Worldwide

SYMPTOMS
Blind plants have been reported in several crops including beets, brassicas, eggplants and peppers. This disorder is characterized by loss of apical meristem function in seedlings. Affected plants exhibit stunted growth, do not form flower buds and the few leaves that form are thick and distorted. Stems may proliferate from below the apex.

CONDITIONS FOR SYMPTOM DEVELOPMENT
Blind seedlings as young as 12 days old have been observed. Several factors have been implicated with this disorder: fluctuating temperatures, low light and poor nutrition. Supplements of nitrogen and phosphorous were shown to alleviate this disorder but further studies are needed. Some tomato-growing regions (Florida, USA) have seen an increase in expression of this disorder during fall tomato production months.

CONTROL
Random occurrence and lack of evidence for what triggers this disorder makes it difficult to implement control measures. Supplements of nitrogen and phosphorous were shown to alleviate this disorder but further studies are needed. Avoid excessive irrigation to prevent nutrients from being leached from the root zone to cause nutrient stress.

"Blind" seedlings from a transplant facility.

GLOSSARY

ABAXIAL The underside of the leaf facing away from the stem.

ABIOTIC Pertaining to the absence of life, as in a disease not caused by living organisms.

ADAXIAL Surface of a leaf facing towards the stem.

AGROINFECTION A method for transfecting plants with DNA from a plant virus using the Ti plasmid from Agrobacterium.

AIRBLAST High pressure spraying in which considerable turbulence is created, often resulting in water soaking of tissue.

ALLELES One of two or more alternate forms of a gene occupying the same locus.

ALTERNATE HOST Species of host other than the principal host on which a parasite can survive.

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

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

ANTIBODY A protein produced in a warm-blooded animal which is specific to an injected foreign protein or carbohydrate.

ANTIGEN A substance that when it is introduced into the body of a warm-blooded animal stimulates the production of antibodies.

APEX Tip or end, uppermost point of a shoot or root.

ASCOSPORE Sexually derived fungal spore formed inside an ascus (sack-like structure).

ASYMPTOMATIC Lacking symptoms of a disease; pathogen may or may not be present.

AVIRULENT Non-pathogenic; lacking virulence; unable to cause disease.

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

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

CALYX (pl. calyces) The external green, leafy part of a flower consisting of sepals.

CANKER Localized, diseased areas on roots or stems where tissue shrinks and cracks open.

CAST SKIN Molted exoskeleton of an insect.

CHLOROPHYLL The green pigment used by plants in their food production process.

CHLOROSIS (adj. chlorotic) The failure of chlorophyll development caused by disease or a nutritional disturbance; the fading of green plant color to light green, yellow or white.

COALESCE Merging of individual lesions.

CONCENTRIC Different size circles having a common center.

CONIDIUM (pl. conidia) A spore produced asexually by various fungi at the tip of a specialized hypha.

COTYLEDON The seed leaf; the first foliar structure to emerge from a seed.

CULTIVAR Cultivated variety; closely related plants from a common origin within a species.

DAMPING-OFF Rotting of seeds or seedlings at or below the soil level.

DEBRIS Remnant plant material.

DEFOLIATION The loss of leaves.

DISCING To work (soil) with a disk harrow.

DISTAL Located far from the point of attachment.

DIURNAL Occurring or active during the daytime.

DNA Deoxyribonucleic acid; a molecule that carries most of the genetic instructions used in the growth, development, functioning and reproduction of all known living organisms and many viruses.

EPIDERMIS The outer layer of cells occurring on plants.

EPIDEMIOLOGY Area of plant pathology interested in understanding factors influencing the initiation and development and spread of disease.

EPINASTY Increased growth of the upper surface of a plant part, such as a leaf, resulting in a downward bending of the part.

EPIPHYTE An organism that lives non-parasitically on the leaves, stems, roots, buds, flowers or fruit of a plant.

FUMIGATION Sterilization by chemical volatilization.

FUNGICIDE A chemical used to control fungi.

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

GALL Swellings 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. Often associated with obligate parasites (e.g., downy and powdery mildews).

HERBICIDE Chemical substance used to control weeds.

HETEROZYGOUS Mixed hereditary factors; having different alleles at various loci.

GLOSSARY

HOMOZYGOUS Having paired identical genes present in the same cell; no allelic differences.

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.

HYGIENE A condition promoting sanitary practices.

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

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 (e.g., fungal spores and mycelium, bacterial cells, nematodes, virus particles, etc.) that can cause disease.

INSECTICIDE A physical or chemical agent used to kill or protect against insects; a type of pesticide.

INSTAR An insect stage between two periods of molting in the development of an insect larva before adulthood.

INTERMEDIATE RESISTANCE (adj. intermediate resistant) 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 varieties with resistance. 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.

IPM Integrated pest management; a strategy that focuses on long-term prevention of pests through a combination of techniques.

LEAF AXIL The angle between the top of a leaf and stem.

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

LOCULE A cavity within a fruit containing seeds.

MALVACEOUS Plants in the mallow family, including okra and cotton.

MICROSCLEROTIUM (pl. microsclerotia) Microscopic, dense aggregate of darkly pigmented, thick-walled hyphal cells specialized for survival.

MIDRIB The central or main vein of a leaf.

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.

NECROSIS (adj. necrotic) Tissue that dies, turning discolored.

NEMATICIDE A substance that kills or inhibits nematodes.

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

NON-PERSISTENT TRANSMISSION Type of insect transmission of a plant virus in which the virus is acquired by the insect vector after a very short acquisition feeding time and is transmitted to plants during a short transmission feeding time by the insect.

NYMPH Juvenile stage of an insect.

PASTEURIZATION The process of partial sterilization by heating at controlled temperatures to kill undesirable microorganisms.

PATHOGEN An organism or agent that can cause disease.

PEDICEL The stalk of a flower or fruit.

PERCOLATION Liquid passing through small pores.

PERSISTENT TRANSMISSION Type of insect transmission of a plant virus in which the virus is acquired by the insect vector after a long acquisition feeding time and is transmitted to plants after a long latent period in which the virus replicates and circulates through the digestive system of the insect vector and into the salivary glands. The insect vector generally remains infectious for the duration of its adult life.

PESTICIDE Any chemical or physical agent which destroys, prevents, mitigates or repels or attracts pests.

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).

PHYTOTOXIC Poisonous, injurious or lethal to plants. Usually describing a chemical.

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

PLANT NURSERY A place where plants are grown for the sake of being moved or transplanted later.

PLASMID Circular piece of double stranded DNA found in certain bacteria and fungi that carries nonessential genetic information and is self-replicating.

POLLEN Fine powdery substance, typically yellow, consisting of one-celled microspores in a seed plant.

GLOSSARY

PROTECTED CULTURE A vegetable production system that includes structures such as greenhouses, high tunnels and mini tunnels.

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

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 Plants that are infected with a disease-causing organism and can serve as a source for further infection of other plants.

RESISTANCE (adj. resistant) The ability of a plant variety to restrict the activities of a specific pathogen or insect pest and/or to restrict the symptoms and signs of a disease, when compared to susceptible varieties. Varieties with resistance may exhibit some symptoms when specified pathogen or pest pressure is severe. New and/or atypical strains of the specific pathogen or pest may overcome the resistance.

RNA Ribonucleic acid; a molecule, found in the cells of all living things and in some viruses, that helps make proteins.

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

SAPROPHYTE (adj. saprophytic) An organism that lives on dead organic matter.

SATURATION Being filled with liquid, generally water.

SCION A bud or shoot inserted by grafting.

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

SEED TRANSMISSION Movement of inoculum from an infected seed to a plant.

SEEDBORNE DISEASE Inoculum is carried on or within the seed.

SEMI-PERSISTENT TRANSMISSION Type of insect transmission of a plant virus in which the vector acquisition feeding time is short with no latent period, no virus replication occurs in the vector and the virus can be transmitted by the vector for no more than a few days.

SENESCE Growing old; aging.

SEPAL Modified leaf-like structure that is a unit of the calyx.

SOILBORNE DISEASE Inoculum is carried on or under the soil surface.

SOLANACEAE The nightshade family which includes tobacco, tomato, potato, pepper, eggplant, tomatillo and others.

SOLARIZATION Exposure to direct sunlight to raise soil temperatures to levels that kill pathogens.

SPORE A reproductive structure of fungi and some bacteria.

SPORULATE To form or produce spores.

STOMA (pl. stomata) A tiny pore in a plant leaf surrounded by a pair of guard cells that regulate its opening and closure, and serves as the site for gas exchange.

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.

STUNTING A dwarfing or hindering from attaining proper size.

SUSCEPTIBILITY (adj. susceptible) The inability of plants to restrict the activities of a specified pest or pathogen.

SYSTEMIC Spreading internally throughout a plant.

TELEOMORPH The sexual form of a fungus.

Ti PLASMID Tumor-inducing plasmid used to transfer genetic material from the plasmid to plant cells.

TOLERANCE (adj. tolerant) The ability of plants to endure a specified pest, pathogen, environmental pressure or chemical stress without serious consequences to growth, appearance or yield. A tolerant variety will sustain less damage than a susceptible variety when grown under the same conditions.

TOXIN Poison produced by an organism.

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

TRANSLUCENCE Transmitting light but diffusing it enough to cause images to be blurred.

TRANSPIRATION The loss of water vapor from the surface of leaves.

TRANSPLANT Remove or transfer a young seedling from one place to another.

VASCULAR Conductive tissue (xylem, phloem) of a plant.

VECTOR A living organism (e.g., insect, mite, bird, nematode, plant, human) able to transmit a pathogen.

VIRUS Very small, submicroscopic 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).

REFERENCES

Color Atlas of Post-Harvest Diseases and Disorders of Fruits and Vegetables, Vol. 2.: Vegetables. *1992. A. L. Snowdon. CRC Press Inc., Boca Raton, Florida.*

Compendium of Tomato Diseases and Pests, 2nd Ed. *2014. J. B. Jones, T. A. Zitter, M. T. Momol and S. A. Miller, eds. APS Press, The American Phytopathological Society, St. Paul, Minnesota.*

Compendium of Tomato Diseases. *1993. J. B. Jones, J. P. Jones, R. E. Stall and T. A. Zitter, eds. APS Press. The American Phytopathological Society, St. Paul, Minnesota.*

Crop Protection Compendium. *www.cabi.org*

Curly Top Identification Handbook. *1977. B. J. Hoyle. University of California Cooperative Extension Publication 4079.*

Diagnosis of Mineral Disorders in Plants, Vol. 2. *1983. A. Scaife and M. Turner. Her Majesty's Stationary Office, London, United Kingdom.*

Disease and Pests of Vegetable Crops in Canada. *1994. R. J. Howard, J. A. Garland and W. L. Seaman. The Canadian Phytopathological Society and the Entomological Society of Canada. M.O.M. Printing Ltd., Ottawa, Ontario, Canada.*

Genomic and Biological Characterization of Tomato necrotic streak virus, a Novel Subgroup 2 Ilarvirus Infecting Tomato in Florida. *2016. I. E. Badillo-Vargas, C. A. Baker, W. W. Turechek, G. Frantz, H. C. Mellinger, J. E. Funderburk and S. Adkins. Plant Disease 100:1046-1053.*

Glossary of Plant Pathological Terms. *1997. M. C. Shurtleff and C. W. Averre III. APS Press, The American Phytopathological Society, St. Paul, Minnesota.*

Greenhouse Tomatoes, Disease Control. *1973. R. E. Partyka and J. D. Farley. Ohio State University Cooperative Extension Service, SB-16.*

Integrated Pest Management for Tomatoes. *1982. University of California, Agricultural Sciences Publication 3274.*

Market Diseases of Tomatoes, Peppers and Eggplants. *1968. L. P. McColloch, H. T. Cook and W. R. Wright. USDA Agricultural Handbook No. 28.*

Nutritional Deficiencies and Toxicities in Crop Plants. *1993. W. F. Bennett. APS Press. The American Phytopathological Society. St. Paul, Minnesota.*

Nutritional Disorders in Glasshouse Tomatoes, Cucumbers and Lettuce. *1981. J. P. N. L. Roorda van Eysinga and K. W. Smilde, Centre for Agricultural Publishing and Documentation, Wageningen, The Netherlands.*

Physiological, Nutritional, and Other Disorders of Tomato Fruit. *2004. S. M. Olson. Document HS954, one of a series of the Horticultural Sciences Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida.*

Tomato Diseases - Identification, Biology and Control, 2nd Ed. *2012. D. Blancard, ed. Manson Publishing, Ltd., London.*

Tomato Diseases and Their Control. *1967. R. E. Webb, J. M. Good and L. L. Danielson. USDA Agriculture Handbook No. 203.*

Tomato Diseases in Florida. *1969, J. P. Jones, G. F. Weber and D. G. A. Kelbert. Florida Agricultural Experiment Station Bulletin 731.*

Tomato Diseases and Insect Pests. *1967. M. B. Linn and W. H. Luckmann. University of Illinois Cooperative Extension Circular 912.*

Vegetable Disease – A Color Handbook. *2007. S. T. Koike, P. Gladders and A. O. Paulus. Academic Press, Burlington, Massachusetts.*

Vegetable Diseases and Their Control, 2nd Ed. *1986. A. F. Sherf and A. A. MacNab. John Wiley & Sons.*

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