

Tomato Disease Guide



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A PRACTICAL GUIDE FOR SEEDSMEN,
GROWERS AND AGRICULTURAL ADVISORS

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Preface

This guide provides general descriptions and pictures of the more common tomato diseases and disorders worldwide. For each disease and disorder, the reader will find the common name, the cause, where it occurs, symptoms, conditions necessary for development and control measures.

The photographs were chosen to illustrate characteristic symptoms of each disease and disorder. It is important to note, however, that the variety grown, cultural practices, environmental conditions, and the pathogen population all influence the appearance and severity of a disease or disorder and, thus, the control measures.

The primary audience for this guide includes tomato producers and those who service these tomato producers. This service group would include agricultural advisors, private consultants, farm managers, agronomists and representatives of food processors, chemical companies and seed companies. We hope this book can be used in the field as a quick guide to information about some common tomato diseases and their control. However, it should be noted that positive diagnosis of tomato problems by using only this book is not recommended, nor encouraged, and this guide should not be substituted for the professional opinion of a producer, grower, agronomist, pathologist or similar professional dealing with this specific crop. Even the most experienced plant pathologist uses both laboratory and greenhouse techniques to confirm suspicions from the field. Moreover, this guide by no means covers every tomato disease. Rather, an attempt has been made to present those diseases which are prevalent worldwide. The insect pests discussed here are those that are involved in the transmission of viruses. Cloudy spot caused by various stinkbugs is described only because of the unusual nature of the fruit symptoms.

A glossary of words used in the text can be found at the end of the book, along with a list of references for further disease information.

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



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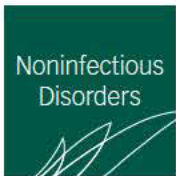
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Bacterial Diseases

INFECTIOUS DISEASES

Bacterial Canker

Causal Agent:

Clavibacter michiganensis subsp. *michiganensis*

Distribution:

Worldwide

Symptoms:

The first symptom is the downward turning and wilting of the lower leaves of the plant. Leaves may exhibit unilateral wilting and light colored streaks may extend up and down the outside of the leaf midrib, petiole and stem. These streaks may break open to form cankers. Infected leaves and petioles characteristically remain attached to the stem. Internally, the stems show light brown or yellow vascular discoloration, which progresses to reddish brown, and often the pith turns yellow, becomes mealy and hollow. A yellow bacterial ooze can be squeezed from the cut end of an infected stem. Fruit infection occurs as small, white lesions, which develop into brown, scabby lesions. These are surrounded by white halos, giving the lesions a birds-eye appearance. Typically, the vascular tissue extending from the stem scar into the fruit will have a yellow-brown discoloration, and cavities may develop in the pith. These fruit symptoms are common in the greenhouse.

Conditions for Disease Development:

Infection generally occurs through wounds in the plant tissue, however, it can also occur through the leaf stomata or roots. The bacterium can survive for up to five years in the soil and infected plant debris. It can also survive on weeds, volunteer tomato plants and seed. Secondary spread occurs from splashing water, contaminated equipment and tools used in pruning, clipping and transplanting operations. Moderate (18–24°C, 65–75°F) temperatures and greater than 80% relative humidity favor disease development. Optimum moisture conditions for plant growth, low light intensity and high nutrient concentrations, especially nitrogen, also enhance disease development. Symptoms tend to be more severe in sandy soils than in organic soils.

Control:

The use of clean seed and transplants, as well as sterilization of the transplanting mix, flats and all equipment used in the transplanting operations can reduce losses from this disease. Transplants should not be topped or mowed since secondary spread can occur rapidly this way. A rotation to a non-host crop for at least three years can reduce losses from bacterial canker.



Unilateral wilting of the leaf.



Vascular discoloration of the stem.



Canker development on the stem.



Canker development on the petiole.



"Bird's eye" lesion on green fruit.

Bacterial Speck

Causal Agent:

Pseudomonas syringae pv. *tomato*

Two races (0 and 1) have been reported.

Distribution:

Worldwide

Symptoms:

This bacterium can attack the leaves, stems, petioles and flowers of the plant. Leaf symptoms appear as dark brown to black spots often surrounded by a yellow halo. Black lesions with yellow borders can also occur on leaf margins where guttation droplets collect. Large areas of leaf tissue are killed as these lesions coalesce. Oval to elongated black lesions occur on stems and petioles. Generally, fruit lesions remain small (1 mm), speck-like and are superficial, however, they can be larger and sunken. On immature fruit they are surrounded by a green halo.

Conditions for Disease Development:

Disease development is favored by cool (13–25°C, 55–77°F), rainy weather or sprinkler irrigation. Usually one day of leaf wetness is required for the disease to develop. Bacterial speck often occurs in arid areas where sprinkler irrigation is used, but seldom occurs under drip or furrow irrigation in those areas. The organism can survive on the roots or leaves of many crops and weeds. Seed can be infected, however seed transmission is usually of minor importance.

Control:

The use of resistant varieties is the most effective way to control this disease. Copper sprays applied early can reduce disease incidence. Sprinkler irrigation will generally increase disease incidence where the bacteria are present, and therefore, furrow or drip irrigation should be used when possible.



Foliar symptoms in the field.



Leaf lesions with chlorotic halos.



Leaf margin necrotic lesions.



Expanding petiole lesion.



Typical small lesions "specks" on green fruit.



Large lesions on red fruit.

Bacterial Spot

Causal Agent:

Xanthomonas euvesicatoria, *X. vesicatoria*,
X. perforans, *X. gardneri*

Five races have been reported.

Distribution:

Worldwide

Symptoms:

Symptoms can appear on all above ground parts of the plant. The first symptoms observed on the leaves are dark, water-soaked, circular spots that are less than 3 mm in diameter. These spots become angular and the surface may appear greasy with a translucent center and a black margin. The centers of these lesions soon dry and crack, and a yellow halo may surround the lesion. Lesions tend to be more numerous on the young foliage. During periods of high moisture (heavy rain, fog or dew) leaves will take on a blighted appearance rather than the typical leaf spots. Fruit infection begins as small, black, raised specks, which may be surrounded by a white halo that has a greasy appearance. These lesions can enlarge to 4–5 mm (0.25 inch) in diameter and become brown, slightly raised and scabby in appearance. They can also have raised margins and be sunken in the center.

Conditions for Disease Development:

The bacterium can survive in crop debris, on volunteer plants, weeds and seed. This disease spreads rapidly through seed beds and fields by sprinkler irrigation and wind-driven rains. Infection generally occurs through wounds, such as those made by insects, wind-driven sand and rain, and by high pressure spraying. Warm (24–30°C, 75–86°F) temperatures with sprinkler irrigation or heavy rains favor disease development.

Control:

The use of disease-free seed and transplants is important for the early control of bacterial spot. Copper sprays can provide moderate levels of protection. When bacterial spot is present, avoid the use of overhead irrigation. Rotation to non-host crops and controlling weeds and volunteer plants are good preventive measures. Good sanitation practices, which include cleaning equipment used in diseased fields and plowing under all plant debris immediately after harvest, can help reduce losses from this disease.



Foliar symptoms in the field.



Leaf lesions with black margins.



Small lesions on green fruit.



Small and large lesions on red fruit.

Bacterial Wilt (syn: Southern Bacterial Wilt)

Causal Agent:

Ralstonia solanacearum

Five races have been reported.

Distribution:

Worldwide (subtropical and tropical areas)

Symptoms:

Symptoms begin as drooping of the lower leaves, followed soon after by wilting of the entire plant. No foliar yellowing is associated with this wilt disease. When the stem is cut a slimy, gray exudate oozes from the cut end. A longitudinal section of the stem reveals a yellow to light brown vascular discoloration which later turns darker brown and/or hollow as the disease progresses. A quick aid to diagnosis is to place a freshly cut stem piece in water. A white, milky stream of bacteria will ooze from the cut stem.

Conditions for Disease Development:

This bacterium has a host range of greater than 200 plant species, which it can infect and survive on. It can also survive in the soil where it infects 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 the bacterium. It can be spread in irrigation water, in soil on cultivation equipment and in diseased transplants. Warm (29–35°C, 84–95°F) weather and high soil moisture levels favor the development of this disease.

Control:

The use of disease-free transplants, soil fumigation, weed control and crop rotation can all reduce the incidence of this disease. Grafting onto resistant rootstocks or using tolerant varieties can be effective in minimizing losses from bacterial wilt.



Wilting symptoms in the field.



Cut stem showing pith discoloration.



Testing for bacterial streaming from a cut stem.



Bacterial streaming from a cut stem.

Pith Necrosis

Causal Agent:

Pseudomonas corrugata
P. mediterranea

Distribution:

Worldwide

Symptoms:

Foliar symptoms begin as yellowing and wilting of the younger leaves in the upper portions of the plant. As the disease progresses, dark brown to black lesions form on the stems and the entire plant may wilt or die if infection is severe. A longitudinal section of the affected stems may reveal a dark brown discoloration of the pith and vascular system. Also, the stem may contain hollow or segmented cavities. Profuse adventitious root development can occur along the stem where the pith is affected.

Conditions for Disease Development:

The disease has been associated with cool night temperatures, high humidity and excessive nitrogen fertilization. The onset of symptoms often occurs when the fruit reach the mature green stage, especially if the plants are too succulent.

Control:

Avoid excessive applications of nitrogen fertilizers and other practices that could contribute to increased plant succulence. The disease can be spread by pruning and mechanical wounding so these practices should be avoided as much as possible, especially when the foliage is wet. Disinfecting pruning implements can help reduce the rate of spread of the disease.



Wilting symptoms in the field.



Stem symptoms showing black lesions.



Stem longitudinal-section showing collapsing, necrotic pith and fruit necrosis.



Stem longitudinal-section showing pith collapse.

Syringae Leaf Spot

Causal Agent:

Pseudomonas syringae pv. *syringae*

Distribution:

Worldwide

Symptoms:

Symptoms on leaves may vary from brown spots that lack a halo, to dark brown or black spots with bright yellow halos that look very similar to those of bacterial speck. The leaf spots from syringae leaf spot can be larger than those of bacterial speck. However, it is necessary to isolate the bacterium and conduct laboratory tests to determine which pathogen is involved.

Conditions for Disease Development:

The bacterium is known to survive on both host and non-host plants in a non-parasitic state, and can spread from these plants when cool and wet environmental conditions favor disease development. Wounding is required for infection and the pathogen may invade lesions already caused by another disease. The causal bacterium is a weak pathogen, and the overall disease development may be less than that caused by bacterial speck.

Control:

Control may be obtained with copper sprays, but with this disease, spraying is usually not necessary since economic damage is not common. If disease occurs, it should be determined whether the symptoms are caused by syringae leaf spot and not by another bacterial disease which might require more stringent control.



Foliar lesions with chlorosis.



Leaf lesions without chlorosis.

Fungal Diseases

INFECTIOUS DISEASES

Alternaria Stem Canker

Causal Agent:

Alternaria alternata f. sp. *lycopersici*

Distribution:

USA (California)

Symptoms:

Symptoms may occur on all above ground parts of the plant. Dark brown cankers with concentric rings form on stems and are often associated with wound sites. These cankers may enlarge, eventually girdling the stem and killing the plant. A brown, dry rot develops and brown streaks can extend into the pith above and below the cankers. A toxin produced by the fungus growing in the stem canker moves into the upper part of the plant, killing the interveinal leaf tissue. As the disease progresses, curling of leaf margins and eventual death of the leaf occurs. Fruit symptoms initially appear as small, gray flecks which later enlarge and become dark and sunken with the characteristic concentric rings. Symptoms may not be present on mature green fruit, however, they can develop rapidly in transit.

Conditions for Disease Development:

The fungus can survive in the soil and crop debris for more than a year. Rain, overhead irrigation and dews favor development of this disease, and its conidia are easily spread by the wind. Stem wounds created by pruning allow entry of the fungus, however, infection can also occur without wounds.

Control:

It is difficult to control this disease with fungicides so resistant varieties should be used.



Typical black stem lesions.



Close up of the stem canker.



Toxin induced leaf interveinal chlorosis and necrosis.



Dark sunken fruit lesions.

Anthracnose

Causal Agent:

Colletotrichum coccodes, *C. dematium*,
C. gloeosporioides and other species.
(teleomorph: *Glomerella cingulata*)

Distribution:

Worldwide

Symptoms:

Infection can occur on the fruits, stems, leaves and roots, with the fruit and root infections being the most serious. Although the fruit is readily infected when it is green, symptoms do not appear until it ripens. The initial lesions are sunken and circular and become depressed with concentric ring markings as they expand. The lesion centers become tan and many dark specks (microsclerotia) develop. In wet weather numerous conidia are produced in a slimy, pink, gelatin-like mass on the lesion surface. Infected roots develop brown lesions with microsclerotia developing on the root surface. This symptom has given rise to the common name for this disease, black dot root rot. Root infection is commonly associated with corky root caused by *Pyrenochaeta lycopersici*. Leaf infection is rarely a problem and is characterized by small brown circular lesions surrounded by a yellow halo.

Conditions for Disease Development:

This fungus is generally considered a weak pathogen, however, it has a wide host range (68 species) and can survive in the soil on decaying plant material for several years. Free moisture and temperatures between 10–30°C (50–86°F) favor host infection. Fungal conidia and microsclerotia can infect host tissue directly in contact with infested soil or they can be disseminated to host tissue by splashing rain and overhead irrigation. They then penetrate the tissue directly or enter through wounds. Root infection generally occurs when the fungal inoculum is high and the plants are nutritionally stressed due to unfavorable growing conditions or infection from another pathogen, especially *Pyrenochaeta lycopersici*.

Control:

A fungicide spray program initiated at the first green fruit stage and continued to harvest can be used to control this disease. Crop rotation to a non-host can prevent the buildup of the fungus in the soil and reduce losses from this disease. Broad-spectrum fumigants and avoiding root injury can also reduce losses from black dot root rot.



Sunken circular fruit lesions.



Sunken circular fruit lesions.



"Black dot root rot", note the microsclerotia on the roots.

Buckeye Fruit and Root Rot (syn: Phytophthora Root Rot)

Causal Agent:

Phytophthora nicotianae var. *parasitica*
P. capsici
P. dreschleri

Distribution:

Worldwide

Symptoms:

These fungi can infect all parts of the plant. They can cause a damping-off of seedlings, a root and crown rot, a foliar blight and a fruit rot. The symptoms caused by root rot are water-soaked brown lesions on the secondary roots and the tap root that can extend above the soil-line onto the stem. As the disease progresses the smaller roots collapse and decay, and large brown, sunken lesions develop on the larger secondary roots and the tap root. 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 quickly collapse and dry. Stem lesions can develop at any level on the stem but are typically found near the soil-line. The lesions are first dark green and water-soaked, and eventually turn dry and brown. As the lesions expand they can completely girdle the stem with the pith becoming brown and collapsing. The fruit symptoms start as grayish-brown water-soaked lesions that can expand rapidly, forming brown concentric rings that resemble a buckeye nut, hence the name. The brown discoloration can extend into the fruit center with the young green fruit becoming mummified, while the mature fruit quickly rots from invasion by secondary organisms.

Conditions for Disease Development:

These fungi have a relatively wide host range and can survive in the soil and infested plant debris for at least two years. They can be spread through irrigation run-off and on farm equipment. Initial infection is favored by moderate soil moisture levels and warm (20°C, 68°F) temperatures. Excessive irrigation or rain, in combination with heavy or compacted soils, favors further disease development.

Control:

Fungicides can help reduce losses from this disease. In addition, cultural practices that can help reduce losses include using a three-year rotation to non-host crops, improving soil drainage, avoiding soil compaction, using raised beds to improve drainage and using shorter irrigation times to avoid extended periods of soil saturation.



Wilting and dead plants in the field.



Stem lesion.



Root vascular discoloration.



Root vascular discoloration and collapse of the smaller roots.



Buckeye fruit rot showing the brown concentric rings.



Seedling damping-off in the field.

Cercospora Leaf Mold

Causal Agent:

Pseudocercospora fuligena
(syn: *Cercospora fuligena*)

Distribution:

Africa, China, India, Japan, Malaysia, Mexico, Philippines and USA

Symptoms:

The first symptom is a yellowish discoloration on the upper leaf surface that later expands to form a brownish lesion surrounded by a yellow halo. When the humidity is high, gray to blackish-gray fungal sporulation occurs on the lower leaf surface. This disease is sometimes referred to as "black leaf mold" because of this dark fungal sporulation. When the disease is severe the lesions will coalesce, resulting in the collapse of the leaf tissues. Due to the similarity of the symptoms, this disease has been confused with Leaf Mold caused by *Fulvia fulva*.

Conditions for Disease Development:

This fungus can survive on infested plant debris and alternative hosts like black nightshade. High humidity and warm (27°C, 81°F) 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 the fungal spores.

Control:

The use of fungicides and resistant varieties greatly reduce losses from this disease. Cultural practices like turning under plant debris, as well as pruning and spacing plants to provide adequate air movement, also help reduce losses.



Extensive sporulation on the foliage.



Gray sporulation on the lower leaf surface.



Blackish-gray sporulation on the lower leaf surface.



Light (right) to heavy (left) leaf sporulation.

Corky Root Rot

Causal Agent:

Pyrenochaeta lycopersici

Distribution:

Canada, Europe, New Zealand and USA

Symptoms:

Infected plants may be stunted and generally lack vigor. As the disease progresses the plants may show diurnal wilting and premature defoliation. The first symptoms on the roots are small elliptical light brown areas on the thin roots. This stage of the disease is frequently called brown root rot. As the disease progresses the larger roots are infected and develop extensive brown lesions that are somewhat swollen and cracked along their length, giving them a corky appearance. By this stage the smaller roots can be completely rotted away, and 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 the soil and plant debris for several years. Disease is most severe under cool (15–20°C, 59–68°F) soil temperatures and relatively high soil moisture, however, strains from warmer climates were found to be pathogenic at soil temperatures between 26–30°C (79–86°F). Spread of the fungus probably occurs on contaminated farming implements.

Control:

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



Extensive root lesion development.



Swelling and cracking along the length of the lesion.

Damping-Off

Causal Agent:

Pythium species and *Phytophthora* species
Rhizoctonia solani (teleomorph: *Thanatephorus cucumeris*)

Distribution:

Worldwide

Symptoms:

Seeds may rot before germinating and seedlings may decay before emergence (preemergence damping-off), giving the appearance of poor germination. After emergence (postemergence damping-off) the seedlings develop lesions at the base of the stem, and the tissue becomes soft, constricted, and the plants wilt and fall over.

Pythium species and *Phytophthora* species —

Preemergence damping-off commonly occurs with these fungi and typical symptoms are a soft mushy rot and dark brown to black water-soaked lesions that rapidly spread over the entire seedling. Postemergence damping-off is characterized by dark colored water-soaked lesions that start on the roots and spread up the stem to above the soil-line. The lesions continue to expand above the soil-line, eventually girdling the stem, causing the plant to wilt and die.

Rhizoctonia solani — Preemergence damping-off is characterized by tan to reddish-brown lesions developing on the seedling and by the death of the growing tip. Postemergence damping-off symptoms include reddish brown to black lesions developing on the roots and the lower stems at or below the soil-line. The stem becomes constricted and infected seedlings quickly wilt and die. Older plants can become infected, however, as the plant matures it becomes more tolerant to infection and the lesions are usually restricted to the cortical zone.

Conditions for Disease Development:

These fungi generally survive for long periods 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 more common when improperly pasteurized soil or previously used seedling trays are used for planting. Water splash can move infested soil from diseased to healthy plants and spread this disease.

Control:

Crop rotation with cereal crops and soil fumigation or solarization may help reduce damping-off in fields. Improving soil drainage by using raised beds and regulating soil moisture by avoiding excessive irrigation help to reduce disease. In greenhouses, good sanitation, including using sterilized planting trays and proper soil pasteurization, helps reduce damping-off. Some fungicide seed treatments or soil drenches can help prevent serious damping-off.



Phytophthora damping-off.



Pythium damping-off, note constricted stem.



Pythium damping-off.

Didymella Stem Rot

Causal Agent:

Didymella lycopersici
(anamorph: *Phoma lycopersici*)

Distribution:

Denmark, Morocco, New Zealand, Romania, Russia
and United Kingdom

Symptoms:

Infection usually occurs on the stem at or above the soil-line, however, all foliar parts of the plant can be affected. Dark brown, sunken lesions form at the base of the plant and eventually expand to girdle the stem, resulting in yellowing and wilting of the older leaves. As the wilting progresses, the plant may eventually die. Numerous black specks (pycnidia), which are the fruiting structures of the fungus, frequently form in the darkened stem lesions. Splashing water spreads the fungal spores from the pycnidia to the fruit, leaves and stems resulting in additional infections and disease spread. Fruit infection typically occurs at the calyx end and starts as a water-soaked lesion that progresses rapidly into a sunken black lesion with concentric rings. Leaf infection begins as small spots, which develop into brown lesions with concentric rings. Pycnidia may develop in the center of these lesions with the leaf eventually taking on a shot-hole appearance, or dying.

Conditions for Disease Development:

The fungus can survive in the soil, in infected plant debris and seed, as well as on nightshade and other related hosts. *Didymella* stem rot occurs over a wide range of conditions, however, 20°C (60°F) accompanied by splashing water from rain or overhead irrigation is optimum for disease development and spread. Plants become more susceptible as they mature, and deficiency of soil nitrogen and phosphorus can contribute to disease severity.

Control:

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



Seedling black stem canker.



Large black stem canker.

Early Blight

Causal Agent:

Alternaria solani

Distribution:

Worldwide

Symptoms:

Symptoms may occur as leaf, stem or fruit lesions. Typically, they appear first on older leaves as irregular, dark brown, necrotic areas. These lesions expand as the disease progresses and they eventually develop concentric, black rings, which give them a target-board appearance. A yellow chlorotic area often surrounds the leaf lesions, and if there are numerous lesions the whole leaf turns yellow and quickly dries up. Complete defoliation of the plant can occur when conditions are favorable for disease development. Lesions may appear as dark brown, elongated, sunken areas on the stem and petiole. Lesion development at the soil-line can result in a collar rot that often girdles the stem. Fruit lesions often occur at the calyx end and are dark, leathery and sunken with the characteristic target-board appearance.

Conditions for Disease Development:

The fungus generally survives from season to season on decayed plant material in the soil. Volunteer tomatoes, potatoes and other solanaceous weeds can also serve as inoculum sources. Infection and fungal spore production occur during periods of warm (24–29°C, 75–84°F) and rainy or humid weather. The fungal spores are then disseminated by the wind and rain. This disease can spread rapidly when favorable conditions persist. It can also be serious in arid climates if there are frequent dew periods, or if sprinkler irrigation is used.

Control:

A fungicide spray program combined with a blight forecasting system is generally the most effective means of controlling early blight.



Stem canker showing concentric rings.



Seedling foliar lesions.



Mature plant foliar lesions.



Leaf lesion showing concentric rings.



Soil-line stem canker causing collar rot.



Fruit lesion development on the calyx end.

Fruit Rots

Distribution:

Worldwide

Symptoms:

Bacterial Soft Rot — Causal Agent: *Erwinia carotovora* subsp. *carotovora*. The first symptom is a light to dark colored, sunken lesion. As the rot progresses the lesion expands, causing a slimy rot, and bacteria may ooze through cracks in the epidermis.

Black Mold Rot — Causal Agent: *Alternaria alternata* and *Stemphylium* species. 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 the lesion surface.

Pythium Fruit Rot — Causal Agent: *Pythium* species. Lesions start as water-soaked spots on both green and ripe fruit. The lesions quickly expand, engulfing the entire fruit, giving it a water balloon appearance. When the epidermis is ruptured, the fruit quickly collapses. When humidity is high, a white cottony growth will cover the lesion surface.

Rhizoctonia Fruit Rot — Causal Agent: *Rhizoctonia solani*. Begins as a firm rot, which rapidly becomes a watery soft rot. It usually occurs on ripe fruit, which are in contact with the soil, and is characterized by rings that form in the infected area. A brown fungus growth frequently occurs over the surface of the infected fruit.

Rhizopus Rot — Causal Agent: *Rhizopus stolonifer*. Lesions develop rapidly and have a puffy, water-soaked appearance. A white fungal growth interspersed with tiny black spheres (fungal spores masses) on raised stalks may spread over the surface of the affected area. *Rhizopus* rot tends to have a fermented odor compared to the putrid odor of bacterial soft rot and sour rot.

Sour Rot — Causal Agent: *Geotrichum candidum*. This rot may occur on mature green as well as ripe tomatoes. The rot usually begins at the stem scar and may extend in sectors down the side of the fruit. A dull white fungal growth may occur where the epidermis cracks. The fruit often remains firm until the rot is advanced, and then a sour odor develops. Under favorable conditions infection can proceed rapidly on ripe fruit.

Conditions for Disease Development:

Both bacteria and fungi will enter through natural openings like the stem scars or through growth cracks and wounds caused by insect feeding and mechanical damage. Generally, warm temperatures and high humidity provide adequate conditions for bacteria and fungi to infect the fruit.

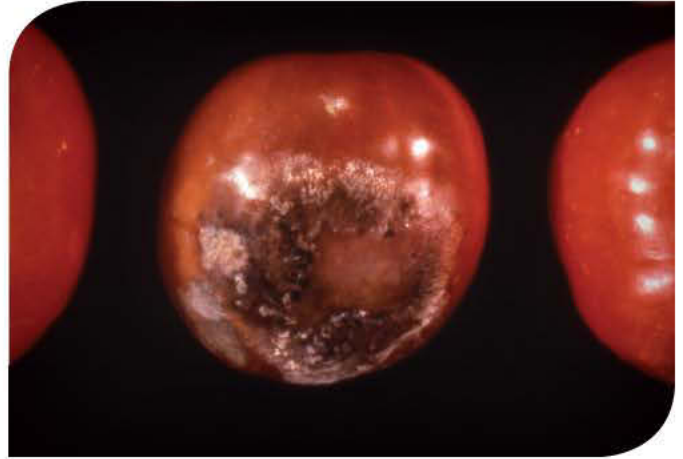
Control:

Avoid fruit injury, especially at harvest. Cultural methods, which keep fruit from coming in contact with the soil, help prevent many of the fruit rots. Improve air movement in the field by orienting planting rows in the direction of prevailing winds, and use proper row and plant spacing to shorten the length of time that the fruit remains wet. Schedule sprinkler irrigation to minimize the length of time that free water remains on the fruit. Fungicides may offer some protection against some fruit rots. Firm fruited, crack-resistant varieties may escape infection by some fruit rots.

Fruit Rots (continued)



Black Mold Rot.



Rhizoctonia Fruit Rot.



Rhizoctonia Fruit Rot, note concentric rings.



Rhizopus Rot.



Pythium Fruit Rot.



Sour Rot.

Fusarium Crown and Root Rot

Causal Agent:

Fusarium oxysporum f. sp. *radicis-lycopersici*

Distribution:

Worldwide

Symptoms:

Typically, the first symptoms are observed when the plant is at the mature green fruit stage. These include a yellowing of the oldest leaves that gradually progresses to the youngest leaves. When the disease is severe the plants may quickly wilt and die, however, a diurnal wilting during hot sunny days is more common. As the disease progresses the entire root system 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 (10 in.) above the soil-line, which helps to distinguish this disease from Fusarium wilt. When the humidity is adequate, fungal sporulation may be observed on the exposed lesions.

Conditions for Disease Development:

The fungus can survive in the soil, on infected plants and on the roots of alternative hosts (eggplant, pepper and a number of legume crops) for several years. Fungal conidia can be spread in soil, on 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, 68°F) soil temperatures.

Control:

The use of varieties resistant to Fusarium crown and root rot is generally the most effective way to control this disease. Soil steaming in combination with fungicide drenches may also reduce losses. Fumigation does not appear to be an effective control measure due to rapid colonization of the sterilized soil by *Fusarium oxysporum* f. sp. *radicis-lycopersici*.



Wilting and dead plants in the field.



Internal vascular discoloration.



External crown rot lesion.

Fusarium Foot Rot

Causal Agent:

Fusarium solani
(teleomorph: *Nectria haematococca*)

Distribution:

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

Symptoms:

Fusarium foot rot symptoms are similar to those of Buckeye root rot, and first appear on mature plants as interveinal chlorosis and necrosis of the leaves. 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 (12 in.) below the soil-line. Internal vascular discoloration may extend 2–10 cm (1–4 in.) beyond the lesions.

Conditions for Disease Development:

This fungus can survive in the soil for 2–3 years. Cooler temperatures favor disease development, even though the fungus can grow well at a soil temperature of 27°C (81°F). Infection can occur through wounds in the plant roots.

Control:

The use of fungicides, soil fumigation, soil solarization or a four-year rotation to a non-host crop may help reduce losses from this disease.



Foliar interveinal chlorosis and necrosis.



Root lesion on the tap root.



Root internal vascular discoloration.

Fusarium Wilt

Causal Agent:

Fusarium oxysporum f. sp. *lycopersici*

Three races (1, 2 and 3) have been reported.

Distribution:

Worldwide

Symptoms:

Infected seedlings are stunted and their older leaves and cotyledons turn yellow and wilt. Severely infected seedlings frequently die. On larger plants the symptoms begin with a yellowing of the older leaves. Entire branches turn yellow, producing a "yellow flag" appearance in the field. Symptoms are often characterized by a yellowing on only one side of a leaf or branch. The affected leaves wilt and die, though they remain attached to the stem. The plants will show diurnal wilting during sunny days and their growth is often stunted. When the stem is cut diagonally, or when branches are snapped off the main stem, a characteristic red-brown discoloration of the vascular tissue is evident. This discoloration may extend far up the plant.

Conditions for Disease Development:

The fungus can survive in the soil for several years and can be spread in soil, on farm machinery, infected plant debris and irrigation water. Infection takes place through root wounds caused by cultivation, secondary root formation and nematode feeding, and the disease develops quickly when soil temperatures are warm (28°C, 82°F). Fusarium wilt is enhanced by high levels of micronutrients, phosphorous and ammonia nitrogen.

Control:

The use of resistant varieties is generally the most effective way to control this disease.



Resistant (left) and susceptible (right) varieties.



Foliar chlorosis and flagging.



Stem internal vascular discoloration.



Seedling wilting and chlorosis.

Gray Leaf Spot

Causal Agent:

Stemphylium solani

S. lycopersici (syn: *S. floridanum*)

S. botryosum f. sp. *lycopersici*

Distribution:

Worldwide

Symptoms:

Leaf lesions initially appear as small, brownish-black specks. These lesions develop into grayish-brown, glazed angular lesions approximately 3 mm (0.13 in.) in diameter and are often surrounded by a yellow area. Eventually they dry up and develop cracks in their centers. If numerous lesions develop, yellowing of the leaf occurs, followed by leaf drop and, eventually, defoliation of the plant. The fruit and stems are not affected by this fungus.

Conditions for Disease Development:

The fungus can survive in the soil and on plant debris from one year to the next. In addition, volunteer tomato plants, as well as other solanaceous crops and weeds, serve as inoculum sources. Infected transplants are also thought to be an important inoculum source. The fungus spores are spread from the surface of infected tissues by wind and splashing water. Warm, humid or wet weather is favorable for disease development. The disease can also be a problem in arid areas when there are long dew periods, or if sprinkler irrigation is used.

Control:

The widespread use of resistant varieties has reduced the importance of this disease. Fungicides should be used for disease control when susceptible varieties are grown.



Foliar necrotic and chlorotic lesions.



Leaf necrotic and chlorotic lesions.



Grayish-brown angular lesions with yellow halos.

Gray Mold

Causal Agent:

Botrytis cinerea
(teleomorph: *Botryotinia fuckeliana*)

Distribution:

Worldwide

Symptoms:

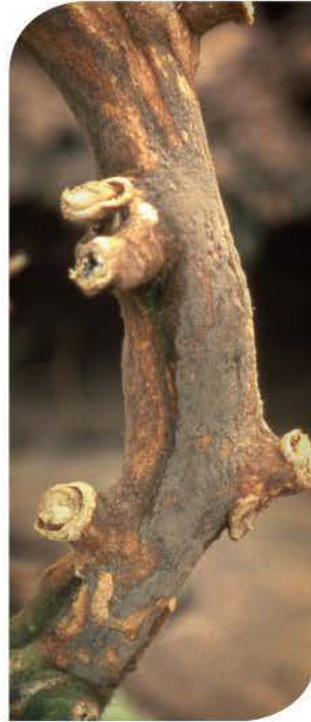
This fungus can infect all above ground parts of the plant and typically enters through wounds. On the stem, initial infection appears as elliptical, water-soaked lesions. These lesions develop under high humidity into a gray, moldy growth, which can girdle and kill the plant. Stem lesions often show concentric banding. Leaf infection usually begins at a point of injury and develops into a V-shaped lesion, which is covered with gray fungal sporulation. This fungus commonly infects the calyx end of the fruit where it can spread rapidly, forming gray-brown sporulating lesions that later develop into a watery rot. Ghost spot, an unusual fruit symptom often observed, is characterized by small white to pale yellow or green rings developing on green or red fruit. These result when the fungus infects the fruit but further disease development is halted when the fruit is exposure to direct sunlight and high temperatures. Ghost spot does not develop further, but spots reduce market quality.

Conditions for Disease Development:

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

Control:

A good fungicide spray program and providing adequate ventilation to the plants by pruning and subsequently applying a fungicide to the pruning wounds can help reduce losses from this disease.



Gray sporulation on the stem lesion.



Gray sporulation on the petiole lesion.



Gray sporulation on the fruit calyx end.



Typical V-shaped leaf lesion.



"Ghost spot" whitish circular rings.

Late Blight

Causal Agent:

Phytophthora infestans

Four races have been reported.

Distribution:

Worldwide

Symptoms:

The first symptom of the disease is a bending down of the petiole of infected leaves. Leaf and stem lesions are large, irregular, greenish, water-soaked patches. These patches enlarge and turn brown and paper-like. During wet weather, the underside of the leaf may have a white sporulating fungal growth. A rapid blighting of the entire foliage may occur during moist, warm periods. Entire fields can have extensive foliar and fruit damage. Fruit lesions are firm, large, irregular, brownish-green blotches. The surface of the fruit lesion has a greasy, rough appearance.

Conditions for Disease Development:

This fungus can survive on volunteer and home garden potatoes and tomatoes, in potato cull piles and on solanaceous weeds. Spores of the fungus can be carried long distances by storms. Cool, wet weather favors the development of this disease. Under these conditions, the disease progresses rapidly and can completely destroy a mature tomato field in a few days.

Control:

A fungicide spray program combined with a blight forecasting system is generally the most effective means of controlling late blight. Avoid planting on land previously cropped to potatoes or close to a potato field because this disease is frequently associated with potatoes.



Burnt foliar appearance in the field.



Fluffy white sporulation on leaf.



Large necrotic stem lesions.



Necrotic expanding leaf lesion.



Rough brown fruit lesions.

Leaf Mold

Causal Agent:

Fulvia fulva

(syn: *Cladosporium fulvum*)

Many physiological races have been reported.

Distribution:

Worldwide

Symptoms:

The first symptoms appear as light green to yellowish areas on the upper surface of the older leaves. This coincides with the development of masses of olive-green fungal conidia on the lower leaf surface. As the disease progresses, the lower leaves turn yellow and drop off. The fungus typically occurs on leaves, but the stems, blossoms and fruit may also become infected. Infected fruit develop a black leathery rot on the calyx end. Although this disease occurs in the field, it is mainly a problem in greenhouses where it can spread rapidly under favorable conditions.

Conditions for Disease Development:

This fungus is an efficient saprophyte and can survive as conidia and sclerotia in the soil and plant debris for at least one year. The conidia are readily dispersed by wind and rain. Dissemination can also occur on workers' clothing and equipment. High (90%) relative humidity and warm (24°C, 75°F) temperatures are optimal for disease development. However, disease can occur between 10–32°C (50–90°F). Leaf mold will not develop if the relative humidity is less than 85%.

Control:

A good fungicide spray program, as well as providing adequate air movement and heating to reduce the relative humidity to less than 85%, can be effective in reducing losses from this disease. Resistant varieties should be used when possible, however, the extreme diversity of the fungus often makes this difficult.



Typical chlorosis (upper) and sporulation (lower) on the leaf surfaces.



Fungal sporulation on the leaf lower surface.

Phoma Rot

Causal Agent:

Phoma destructiva

Distribution:

India, Italy, Pacific Islands, Russia, United Kingdom and USA

Symptoms:

The disease can affect all above-ground parts of the plant. On leaves, it causes numerous, small dark brown to black spots, which may develop concentric rings as they enlarge. Older leaves can be infected first, however, all leaves are susceptible and defoliation can result when disease is severe. The leaf spot looks very similar to that caused by early blight except that the *Phoma* lesions contain numerous minute black fungal fruiting bodies (pycnidia). Dark brown lesions with concentric rings form on the stems, and both the green and ripe fruit can be infected. Fruit lesions usually develop at the calyx end as small sunken lesions that later develop into sunken, black, leathery lesions with numerous pycnidia in the center.

Conditions for Disease Development:

The fungus can survive from one season to the next in the soil, in infected plant debris and on pepper and closely related weeds. Injury to the plant such as pruning, insect feeding, mechanical damage or cracking provide openings for invasion by the fungus. When the temperature (20°C, 68°F) and humidity are optimum, masses of fungal conidia are exuded from the pycnidia. These are readily spread by rain, overhead irrigation and on workers' clothing and equipment. Low soil nitrogen and phosphorus levels may contribute to plant susceptibility.

Control:

A regular fungicide program coupled with good sanitation practices can help reduce losses from this disease. Avoiding fruit injury at the time of harvest, and picking dry fruit to minimize the spread of the disease in packing containers, can also help reduce losses. Maintaining good soil fertility, using long crop rotations and removing all related weeds, such as nightshade, from the field can help reduce losses as well.



Necrotic leaf lesions.



Necrotic leaf and stem lesions.

Powdery Mildew (*Leveillula*)

Causal Agent:

Leveillula taurica
(anamorph: *Oidiopsis sicula*)

Distribution:

Worldwide

Symptoms:

The first symptoms include a light green to bright yellow lesion developing on the upper leaf surface. Eventually, a light powdery fungal sporulation forms on the lower leaf surface. Under ideal conditions, white powdery masses of conidia will develop on both surfaces of the leaf. As the disease progresses the lesions become necrotic, and if it is severe the whole leaf dies. Affected plants may be defoliated, resulting in reduced yields, smaller fruit sizes and sunburned fruit.

Conditions for Disease Development:

This fungus has a wide host range on which it can survive and spread to tomatoes. Powdery mildew conidia can travel long distances on air currents and are able to germinate under low (52–75%) relative humidity. Disease development is favored by warm (27°C, 80°F) temperatures, however, the fungal conidia can germinate between 10–32°C (50–90°F).

Control:

A good fungicide spray program can help reduce losses from this disease.



Foliar chlorosis and necrosis.



Chlorotic lesions on the upper leaf surface; white sporulation on the lower leaf surface.



Close up of fungal sporulation.

Powdery Mildew (*Oidium*)

Causal Agent:

Oidium neolycopersicum

Distribution:

Australia, Canada, Europe, Japan, UK and USA

Symptoms:

The disease first appears as small circular areas of whitish fungal growth with sporulation occurring mainly on the upper leaf surface. As the sporulating lesions enlarge, the underlying leaf tissue turns yellow, eventually becoming brown and shriveled. Sporulation typically occurs on the upper leaf surface, distinguishing *Oidium* from *Leveillula*, which typically sporulates on the lower leaf surface. When infection is severe, masses of powdery fungal sporulation will cover the entire leaf surface as well as the petiole, stem and calyx, however, the fruit remains uninfected. Although this fungus has been reported in the field, it is mainly a problem in greenhouses where yield reductions result from defoliation.

Conditions for Disease Development:

This fungus has a relatively wide host range on which it can survive, and the fungal conidia are readily disseminated for long distances by air movement. Optimum conditions for disease development include low intensity light and temperatures between 20–27°C (68–81°F) accompanied by high (85–95%) relative humidity. However, infection can occur at a lower (50%) relative humidity.

Control:

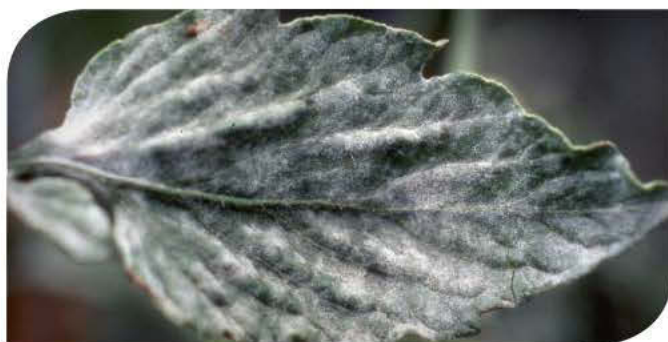
Several fungicides, including sulfur, can be effective in controlling this disease if good foliage coverage is obtained and they are applied in a timely manner.



White sporulation on the foliage.



Sporulation, tissue collapse and necrosis.



Fungal sporulation on the upper leaf surface.

Septoria Leaf Spot

Causal Agent:

Septoria lycopersici

At least two races have been reported.

Distribution:

Worldwide

Symptoms:

Symptoms first appear as small, dark, water-soaked lesions on the older leaves. These enlarge to form circular lesions about 5 mm in diameter with black or brown borders and gray centers peppered with small black fungal sporulating structures (pycnidia). Lesions on the stem, petiole and calyx tend to be more elongated, with pycnidia developing in the center. When disease is severe the lesions coalesce, resulting in the leaves collapsing and eventual defoliation of the plant.

Conditions for Disease Development:

This fungus can survive on debris from previous crops, as well as on several weeds including: nightshade, horse nettle, jimson weed and ground cherry. Extended periods of high (100%) relative humidity and temperatures between 20–25°C (68–77°F) favor infection and disease development. Numerous fungal conidia are exuded from the pycnidia when the humidity is high. They can then be spread by wind and splashing water from rain or overhead irrigation, on workers' clothing and tools, on cultivation equipment and by insects.

Control:

A good fungicide spray program in conjunction with cultural practices, such as removing or turning under all plant debris and a three-year crop rotation, can help reduce losses from this disease.



Stem and leaf lesions.



Necrotic and chlorotic leaf lesions.



Stem lesions with black margins.



Pycnidia in the lesion center.



Necrotic leaf lesions.

Southern Blight

Causal Agent:

Sclerotium rolfsii
(teleomorph: *Athelia rolfsii*)

Distribution:

Worldwide

Symptoms:

This fungus can cause a 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 the plants to quickly damp-off and die. On older plants the lesion girdles the stems, causing the plants to wilt without a change in foliage color. Severely infected plants may eventually die. The lesion continues to expand, causing a root rot below the soil-line and extending several centimeters above the soil-line. If moisture is adequate, a whitish fungal growth covers the lesion surface and tan sclerotia 1–2 mm (0.06 in.) in diameter are readily produced. Fruit coming into contact with the fungus are quickly invaded, resulting in sunken, yellowish lesions with a ruptured epidermis. A white fungal growth and sclerotia are formed on the lesion surface.

Conditions for Disease Development:

This fungus is an efficient saprophyte and can survive in the soil and plant debris for several years. It can be spread in surface water and by the movement of infested soil on cultivation equipment. High temperatures (30–35°C, 86–95°F) and high moisture levels favor disease development.

Control:

Cultural practices, such as careful regulation of soil moisture and deep plowing of residue, and a good sanitation program, which includes removal and burning of all infected plants, can help reduce losses from this disease. Also, fungicides, fumigation and a three-year rotation with crops such as corn and sorghum can help reduce losses from this disease.



Sclerotia development on stem and soil.



White mycelium and sclerotia on stem.



Close up of sclerotia.

Target Spot

Causal Agent:

Corynespora cassiicola

Distribution:

Europe, India, Nigeria, the Caribbean and USA

Symptoms:

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

Conditions for Disease Development:

This fungus has a broad host range on which it can survive. Infection occurs readily during periods of mild temperatures between 16–32°C (61–90°F) and high moisture. Fungal spores, which often form abundantly on the surface of infected tissues, are spread by air movement and rainfall.

Control:

A good fungicide spray program initiated prior to the onset of symptoms can help reduce losses from this disease.



Initial leaf lesions.



Expanding leaf lesions.



Brown fruit lesions with cracking centers.



Stem lesions and leaf collapse.



Close up of sporulation on a lesion.

Verticillium Wilt

Causal Agent:

Verticillium albo-atrum
Verticillium dahliae

Two races (1 and 2) have been reported.

Distribution:

Worldwide

Symptoms:

Wilting of older leaves begins at the leaflet margin and progresses into a yellow-then-brown V-shaped pattern. Older leaves eventually turn yellow and dry up. Diseased plants are stunted, do not respond to fertilizer or water and will show diurnal wilting on sunny days. When the base of the main stem is cut, a light tan discoloration can be seen in the vascular system and across the pith at the crown. This discoloration usually does not extend far up the plant. However, under severe disease pressure, it can be found nearer the shoot tips.

Conditions for Disease Development:

The fungus has an extremely wide host range and can survive in the soil and plant debris as microsclerotia for several years. Disease development is favored by cooler temperatures (21–25°C, 70–77°F). The fungus can enter the plant through root wounds caused by cultivation, secondary root formation and nematode feeding.

Control:

The use of resistant varieties is generally the best way to reduce losses from this disease. Soil fumigation and solar sterilization have been shown to reduce disease incidence.



Wilting plants in the field.



Foliar yellowing and necrosis.



Typical V-shaped leaf lesions.



Typical tan vascular discoloration.

White Mold (syn: Timber Rot or Sclerotinia Stem Rot)

Causal Agent:

Sclerotinia sclerotiorum
Sclerotinia minor

Distribution:

Worldwide

Symptoms:

This fungus can infect the leaves, stems, petioles and occasionally the fruit. Initially, water-soaked lesions develop on the stem, eventually causing a softening of the infected area. Typically, a white fungal growth develops on the lesions. A progressive soft decay of external tissues is followed by an internal hollowing of the pith. Large areas of the stem eventually die, becoming dry and tan-gray. Irregular shaped black pebble-like bodies called sclerotia can be found on the surface and inside the stems, and are diagnostic for this disease. Infected fruit develop grayish lesions that quickly turn into a watery rot with white fungal growth and sclerotia developing on the lesion surface.

Conditions for Disease Development:

This fungus has a wide host range and it can survive as sclerotia in the soil and 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 moderate temperatures between 16–21°C (61–70°F), favor disease development.

Control:

Fumigation or steam sterilization of the soil, and the timely application of fungicides, can help reduce losses from this disease. Also, good cultural practices such as sanitation, crop rotation and providing good drainage and ventilation will help reduce losses. Avoid planting in fields that have previously had this disease when possible.



White fungal growth on the stem lesion.



Large black sclerotia in the stem.



Fungal growth and black sclerotia on the fruit.

Nematode Diseases

INFECTIOUS DISEASES

Northern Root-Knot Nematode (syn: Root-Knot or Root Gall)

Causal Agent:

Meloidogyne hapla

Distribution:

Worldwide

Symptoms:

The first above-ground symptom is a general lack of vigor by the plants. Eventually the foliage becomes chlorotic with a progressive dying of the older leaves, and the plants are stunted. When diseased plants are pulled up, irregular swellings, called galls or knots, can easily be seen on the roots. These galls tend to be small and uniform in size compared to those caused by *Meloidogyne incognita*, which tend to be large and compound. When this disease is severe, the entire root system will be covered with small galls and have a branched appearance as a result of secondary root formation adjacent to the developing galls.

Conditions for Disease Development:

This nematode has a very wide host range, including many vegetable crops and weeds on which it can grow and overwinter. It may be introduced into fields or greenhouses on infected transplants, in infested soil on cultivation equipment or through irrigation water. Once present in a field or greenhouse, it can spread from its alternate hosts to tomato through typical cultivation practices. Although this nematode can cause disease in many soil types, root damage is most serious on lighter, sandy soils. Moderate (16–20°C, 61–68°F) soil temperatures favor nematode reproduction and disease development.

Control:

No commercial varieties are currently resistant to *Meloidogyne hapla* and therefore cultural and chemical controls should be used to control this disease. Soil fumigation or pasteurization should be used when feasible. Also, the use of certified disease-free transplants and rotating to a non-host crop can help reduce losses.



Discrete root galls visible on the root system.



Close up of secondary root development above a root gall.

Southern Root-Knot Nematode (syn: Root-Knot or Root Gall)

Causal Agent:

Meloidogyne incognita, *M. javanica*, *M. arenaria*
At least four races have been reported.

Distribution:

Worldwide

Symptoms:

The first above-ground symptoms are stunting, wilting and general off-colored appearance of the affected plants. The undersides of the leaves develop a purple discoloration, which resemble symptoms caused by phosphate deficiency. When diseased plants are pulled up, irregular swellings of the roots, referred to as galls or knots, can easily be seen. These galls tend to be larger and more compound than those caused by *Meloidogyne hapla*, which produces smaller, discrete galls with lateral roots developing adjacent to them.

Conditions for Disease Development:

These 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 mild winters. Although these nematodes can cause disease in many soil types, root damage is most serious on lighter, sandier soils. Warmer (27°C, 81°F) soil temperatures favor disease development. *Meloidogyne incognita* is by far the most widely distributed of the three species that cause this disease.

Control:

The use of resistant varieties is generally the most effective way of controlling this disease. These varieties should be used in combination with cultural practices that reduce nematode populations because the continued use of resistant varieties alone may result in the breakdown of this resistance. Also, soil pasteurization, fumigation and using disease-free transplants can help reduce losses from this nematode.



Resistant (left) and susceptible (right) variety.



Large, compound galls on the roots.



Large galls on a seedling root system.



Large, compound galls on the roots.

Parasitic Plants

INFECTIOUS DISEASES

Broomrape

Causal Agent:

Orobanche species

Distribution:

Asia, Central America, Europe, India, Mediterranean region, Middle East, Pakistan and USA

Symptoms:

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

Conditions for Disease Development:

Broomrape seeds may lie dormant in the soil for more than 20 years. The root exudates from host plants stimulate germination and the young seedling attaches its roots to the roots of its host, from which it extracts nutrients to grow and reproduce. After flowering, very small seeds are produced that can be distributed on cultivation equipment and in irrigation water, both between and within fields. Conditions favorable for tomato plant growth also favor broomrape growth and parasitism.

Control:

Fumigation and crop rotation, in conjunction with good sanitation practices such as removing and destroying any affected plants along with the broomrape, can help reduce losses from this parasite.



Broomrape growing on tomato roots.



Broomrape flowering.

Dodder

Causal Agent:

Cuscuta species

Distribution:

Worldwide

Symptoms:

Dodder is a whitish-yellow parasitic plant that lacks chlorophyll, leaves and roots, and therefore must derive its nutrients from other plants. It first becomes noticeable as a yellow thread-like vine that winds around the stem and branches of the infected plant. Later, it can spread down the row, covering other plants with a mass of vines and giving a yellowish color to the affected area of the field. Dodder produces small, inconspicuous flowers (often white), which mature and produce thousands of small seeds. When the seed germinates, the emerging vine twists in a counter-clockwise fashion until it comes into contact with its host. Attachment to the plant is by sucker-like projections (haustoria), through which the dodder extracts nutrients from its host plant.

Conditions for Disease Development:

Dodder has a very wide host range, including many weed and crop species, and its seed can survive in the soil for up to 10 years. Dodder seeds can be spread on cultivation equipment and in irrigation water between and within fields. Conditions favorable for tomato plant growth also favor dodder growth and parasitism.

Control:

Removal and destruction of dodder along with infected plants reduces subsequent spread by seed. Herbicides, either contact or pre-plant, along with crop rotation can help reduce losses from dodder parasitism.



Extensive dodder infestation in the field.



Dodder spreading on tomato plants.



Dodder flower and seed capsules.

Phytoplasma Disease

INFECTIOUS DISEASES

Big Bud

Causal Agent:

Phytoplasma

Vector:

Brown Leafhopper (*Orosius argentatus*)

Distribution:

Australia, Brazil, India, Israel, Russia and USA

Symptoms:

The primary symptom of this disease, and the one for which it is named, is the enlargement and abnormal development of the flower buds. Sepals may not separate as the flower opens, and the buds will be 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 the development of an overall yellowish appearance of the plant may also occur. Fruit may be small and deformed.

Conditions for Disease Development:

This phytoplasma can survive in crops such as hot pepper, lettuce, eggplant and potato, as well as weeds such as dock, lamb's-quarter, nightshade, sowthistle and jimsonweed. It is readily transmitted to tomato by the common brown leafhopper. Transmission occurs when leafhoppers carrying the phytoplasma migrate to tomato and feed.

Control:

Big Bud is generally only of occasional importance. Removing host weeds adjacent to tomato plants and controlling the leafhopper vectors with an insecticide program are usually sufficient.



Enlarged swollen flower buds.



Close up of enlarged flower bud.



Fruit symptoms showing deformations.

Viral Diseases

INFECTIOUS DISEASES

Alfalfa Mosaic

Causal Agent:

Alfalfa mosaic virus (AMV)

Vector:

Aphids (*Aphis gossypii*, *Myzus persicae* and others)

Distribution:

Worldwide

Symptoms:

Typically, yellow and purple areas develop in the young leaves, resulting in the foliage developing an overall yellow bronzing. Infected plants stop growing and leaves 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 this disease. Roots often show this same red-brown phloem discoloration. Depending on the age of the plant at the time of infection, developing fruit show various degrees of external and internal brown spotting and distortion. Generally, the disease incidence is greater the closer tomatoes are to alfalfa fields and gradually diminishes with increasing distance from the source.

Conditions for Disease Development:

This virus 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 the plant. 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 is a possible means of avoiding this disease. Spraying for aphids will generally not eliminate primary infection of tomatoes.



Extensive yellowing of the foliage.



Leaf interveinal yellowing and necrosis.



Brown discoloration of the phloem.



Stem discoloration and fruit symptoms.



Dark-brown lesions on the fruit.

Chino del Tomate

Causal Agent:

Chino del tomate virus (CdTV)

Vector:

Whitefly (*Bemesia tabaci*)

Distribution:

Northern Mexico and Southeastern USA

Symptoms:

Chino (curling) and rolling of leaves, interveinal yellowing of newly infected leaves and purpling of older leaves are all characteristic symptoms of this disease. Plants may be severely stunted and distorted if infected at an early stage, and dramatically reduced fruit set may result.

Conditions for Disease Development:

This virus has a relatively wide host range that includes beans, peppers and tomatoes, as well as malvaceous and solanaceous weeds. Peak transmission of the disease occurs when whitefly populations are high. Whiteflies can acquire the virus from nearby infected plants and transmit it to healthy tomatoes, sometimes resulting in up to 100% of the plants becoming infected. Mechanical transmission of the disease has not been demonstrated.

Control:

Previous efforts to control whitefly populations with non-systemic insecticides have generally been unsuccessful, due to the difficulty in getting good coverage of the lower leaves where the insects congregate. However, the more recently developed systemic insecticides have demonstrated dramatic effects in controlling whiteflies. Cultural practices, such as the adoption of crop-free periods where possible and roguing infected plants, should be incorporated into a control strategy to help reduce the likelihood of the whiteflies developing resistance to insecticides.



Interveinal chlorosis of the leaves.



Close up of foliar symptoms

Cucumber Mosaic

Causal Agent:

Cucumber mosaic virus (CMV)

Several strains have been reported.

Vector:

Aphids (*Aphis gossypii*, *Myzus persicae* and many others)

Distribution:

Worldwide

Symptoms:

Symptoms of this disease vary greatly, depending on the virus strains involved. Often, the virus causes stunting of the plant, giving it a bushy appearance. Leaf symptoms may vary from a mild, green mottling to a chlorosis or severe necrosis, or a severe "shoestring" symptom in which the leaf blades are greatly reduced with only the central rib of the leaflet remaining. Shoestring symptoms are similar to the severe symptoms observed with ToMV infection. However, with the 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 alternative hosts survive year round as well as in greenhouses, where once introduced it can readily be spread from plant to plant by aphids. CMV can also be mechanically transmitted. However, because it is an unstable virus, the chances of transmission by greenhouse workers and their tools is much less than with a virus like Tomato mosaic virus (ToMV).

Control:

In greenhouse crops, controlling the aphids can greatly help reduce the incidence of this disease. The elimination of weeds and ornamental plants that harbor the virus, as well as roguing of infected plants, can help reduce the spread of this virus and disease incidence.



Shoestringing leaf symptoms.



Yellowing mosaic leaf symptoms.



Severe shoestringing leaf symptoms.



Extensive necrotic stem lesions.



Extensive necrotic stem lesions.



Internal necrosis of the fruit.

Curly Top

Causal Agent:

Curly top virus (CTV)

syn: *beet curly top virus* (BCTV)

Many strains have been reported.

Vector:

Beet Leafhopper (*Circulifer tenellus* and *C. opacipennis*)

Distribution:

Canada, Mediterranean Region, Mexico and USA

Symptoms:

Typically, infected plants are erect and stunted in appearance, and severely infected seedlings may die. Leaves thicken with their margins rolling upward as the petioles curve downward. Later, the leaves turn a dull yellow with an accompanying purpling of their veins. Very few fruit are produced, and those that set before infection ripen prematurely. Fruit affected by the disease are dull, small and wrinkled with a dried out 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 the beet leafhopper. 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 a tomato field. Other common hosts for this virus are watermelon, cantaloupe, squash, pepper, spinach and beans.

Control:

Spraying insecticides on weeds to control leafhoppers, as well as avoiding beet fields and range land has helped reduce losses from this disease. Double-row planting of processing tomatoes has been used successfully in areas where CTV is present, though it is not known whether this practice actually discourages leafhoppers from visiting plants or whether it allows more individuals to escape infection. Spraying tomato fields for leafhoppers will generally not control curly top.



Leaf curling and vein purpling of the foliage.



Leaf curling and vein purpling of the foliage.

Tobacco Etch

Causal Agent:

Tobacco etch virus (TEV)

Vector:

Aphids (*Myzus persicae* and others)

Distribution:

North and South America

Symptoms:

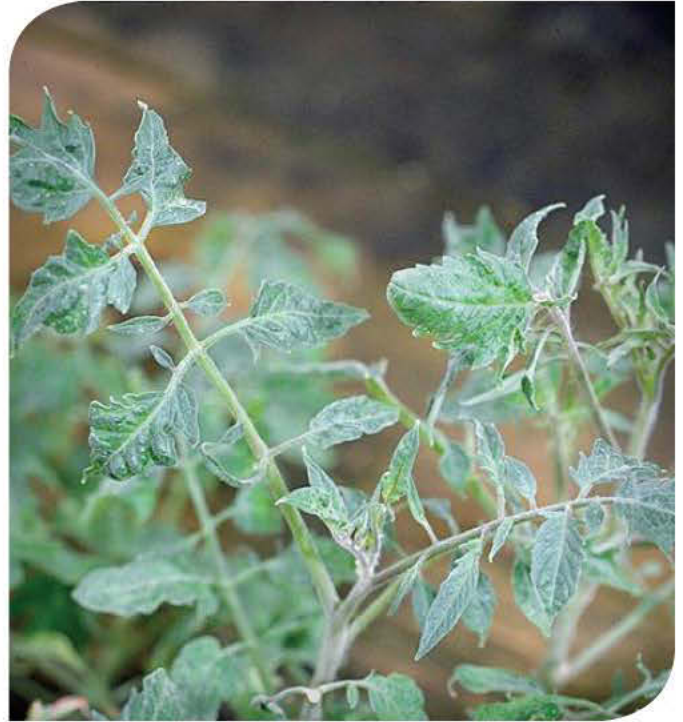
Disease symptoms have been reported on tomato that range from a mild mottling and slight distortion of the foliage to a severe mottling and crinkling of the foliage. Fruit are often reduced in size, mottled and misshapen. Usually, the earlier the plant is infected, the greater the effects on plant stunting and yield.

Conditions for Disease Development:

Many species of weeds can act as a host for the virus, and it can be transmitted by at least 10 species of aphids. TEV is transmitted in a non-persistent manner by virus-carrying aphids moving into tomato fields from nearby solanaceous weeds, peppers and other tomato fields that are already infected. Secondary infection may be caused by aphids, or the virus may be mechanically transmitted through staking, pruning or handling of infected plants.

Control:

Removing weed hosts that may harbor the virus is important. Avoid locating tomato productions near pepper crops because peppers can be a major source of the virus. Reducing the spread of the disease by controlling the aphid vector population is very difficult and generally not practical.



Mild mottling and distorted foliage symptoms.

Tomato Bushy Stunt

Causal Agent:

Tomato bushy stunt virus (TBSV)

Vector:

Mechanically transmitted

Distribution:

Argentina, Britain, California, Mexico, Morocco and Tunisia

Symptoms:

Initial symptoms may vary from irregular white banding patterns on the leaves to necrotic lesions or rings. Leaves soon turn from green to a pale yellow, which may have interspersed green areas. Eventually, the affected leaves may fall from the plant. New leaves frequently are twisted and have necrotic tips. An abundance of side shoots occurs, giving the plant a bushy, stunted appearance. Lower leaves become chlorotic and purple as the disease progresses. Excessive fertilizer on young plants can result in soft stems that, when infected, may develop lesions at the soil-line. The seedling may be girdled at that point, resulting in death of the plant. 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. It is soil-borne and readily transmissible in water. A natural vector of the virus is currently unknown, but it is believed that the virus infects plants through wounds in the roots. This virus 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 the virus. Once present in the soil, this virus is difficult to control.



Severe chlorosis and necrosis on infected seedlings.



Infected plants (left) showing reduced vigor and foliar chlorosis compared to noninfected (right).



Bushy stunted appearance of a mature plant.



Initial foliar symptoms showing the light banding patterns.

Tomato Double Virus Streak (syn: Double Streak)

Causal Agent:

Tomato mosaic virus (ToMV) with
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 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:

Individually, ToMV and PVX are easily transmitted mechanically 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. Severity of symptoms are affected by virus strains, plant age and day length.

Control:

The use of ToMV-resistant varieties in combination with the cultural practices of roguing infected plants and avoiding handling of potatoes prior to working with tomatoes will reduce losses from this disease. Also, not planting in fields that were planted to potatoes the previous season and sterilizing workers' tools after handling infected plants can help reduce disease incidence.



Typical leaf downward curling symptom.



Necrotic streaks on the petiole.



Necrotic lesions on the stem.



Necrotic lesions on the fruit.

Tomato Infectious Chlorosis

Causal Agent:

Tomato infectious chlorosis virus (TICV)

Vector:

Whitefly (*Trialeurodes vaporariorum*)

Distribution:

USA (California)

Symptoms:

Symptoms first appear on older leaves as interveinal yellow blotches while the veins themselves remain green. Symptoms progress to the younger leaves, and depending on the variety, the interveinal yellowing can become bleached-white or necrotic. The affected leaf tissues are brittle and can be easily crushed, and the fruit color may be affected. Severe losses due to poor fruit set have been reported in Southern California. Symptoms caused by this disease can easily be mistaken for problems caused by poor growing conditions, aging or nutrient deficiency.

Conditions for Disease Development:

First reported in 1993, TICV has been found to be transmitted in a semi-persistent manner by the greenhouse whitefly. This virus is spread in the greenhouse when virus-carrying whiteflies move from infected to healthy plants. The virus is not seed-borne, nor can it be transmitted by touching or pruning plants. Once acquired by the whitefly, the virus remains infectious in the insect's body for a few days. Plants begin to develop symptoms a few weeks after infection. Early infection can result in severe yield loss in certain varieties.

Control:

Since the greenhouse whitefly is the only vector for this virus, controlling the insect is generally the most effective control practice. A routine spray program in combination with covering openings to prevent the whitefly from entering the greenhouse should be implemented to keep the insect's population low. When the crop is finished, all plant materials should be removed from the greenhouse and a plant-free period should be implemented. Transplants should come from a whitefly-free greenhouse.



Typical interveinal chlorosis of the foliage.



Severe foliar interveinal chlorosis.



Close up of the foliar interveinal chlorosis.

Tomato Mosaic (syn: Tobacco Mosaic)

Causal Agent:

Tomato mosaic virus (ToMV)

Several strains have been reported.

Vector:

Mechanically transmitted

Distribution:

Worldwide

Symptoms:

Typical symptoms include a light and dark green mottling of the leaf tissue and stunting of the plant. Foliar symptoms can vary from a chlorotic mottling to necrosis to upward leaf rolling and stem streaking, depending on which strain of ToMV infects the plant. During cool temperatures, leaves may develop a "fernleaf" appearance where the leaf blade is greatly reduced. During high temperatures, foliar symptoms may be masked.

Occasionally the fruit will show disease symptoms, which vary from an uneven ripening to an internal browning of the fruit wall (brown wall). Brown wall typically occurs on the fruit of the first two clusters and appears several days prior to foliar symptoms. Under certain environmental conditions, some varieties with resistance (heterozygous) to ToMV will show necrotic streaks or spots on the stem, petiole, and foliage as well as on the fruit.

Conditions for Disease Development:

ToMV has a wide host range including many agricultural crops and weeds, all of which can serve as inoculum sources. It is readily transmitted by machinery or workers from infected to healthy plants during handling. Infested debris from a previous crop can lead to infection when the roots of the new tomato plants come in contact with the debris. Chewing insects can transmit the virus, but are not considered a major source of infection. Tomato seed can carry the virus, but actual infection is thought to occur when plants are thinned or transplanted.

Control:

The use of ToMV-resistant varieties is generally the best way to reduce losses from this disease. Avoid planting in soil from previous crops that were infected with ToMV. Steam sterilizing the potting soil and containers as well as all equipment after each crop can reduce disease incidence. Before handling containers or plants be sure all workers wash with soap and water. Sterilizing pruning utensils or snapping off suckers without touching the plant instead of knife pruning help reduce disease incidence. Direct seeding in the field can help reduce the spread of ToMV.



Typical leaf mosaic symptoms.



Severe leaf distortion and chlorotic mottling.



Fern leaf foliar symptoms.



Necrotic fruit lesions.

Tomato Mottle

Causal Agent:

Tomato mottle virus (ToMoV)

Vector:

Whitefly (*Bemesia argentifolii*)

Distribution:

Southeastern USA

Symptoms:

This is a new disease that is caused by a geminivirus, which was first recognized in Florida in 1989. Symptoms are more pronounced when plants are infected at an early stage. Stunting and reduced growth, upward curling and distortion of the upper and middle leaves, and chlorosis or yellowing of leaves are typical of this disease. Yield from infected plants is lower due to lack of fruit set and reduced fruit size.

Conditions for Disease Development:

Tomato Mottle Virus is transmitted by the silverleaf whitefly. The virus has a fairly narrow host range, which includes common bean, tropical soda apple and a few solanaceous weeds, but not bell pepper. The virus is easily and efficiently spread by the whitefly, and severe outbreaks of the disease can be associated with large populations of the insect. It is not easily mechanically transmitted, however, secondary spread by whiteflies is common within the crop.

Control:

Control solanaceous weeds and other alternative hosts in the vicinity of tomato fields. If possible, plant the crop during a time of the year to avoid the highest whitefly populations. Regular use of mineral oil sprays may reduce the rate of disease spread by reducing virus acquisition and infection by the whitefly vector. The use of insecticides on a regular basis may provide some control, however, whiteflies can develop resistance to pesticides if used excessively.



Yellowing mosaic leaf symptoms.



Severe yellowing foliar symptoms.



Leaf curling foliar symptoms.

Tomato Spotted Wilt

Causal Agent:

Tomato spotted wilt virus (TSWV)
Several strains have been reported.

Vector:

Thrips (*Thrips tabaci* and *Frankliniella* species)

Distribution:

Worldwide

Symptoms:

The first visible symptom is small, orangish-yellow flecks on the older leaves. Later, the leaves develop small, dark, more or less circular spots, which gives them a bronzed appearance. The growing tips of the leaves may die as the disease progresses. Dark, shiny streaks are often seen along the stem and petioles. Severely infected plants are stunted with yellowish drooping foliage, which imparts a wilting appearance to the plant. Fruit usually show characteristic green, yellow and red, slightly raised concentric rings, although some strains can cause severe necrosis.

Conditions for Disease Development:

This virus has a very wide host range from which it can be transmitted in a persistent manner by thrips. Although the virus is acquired at the larval stage, it is only spread by the adults that are wind blown to tomatoes from infected weeds or ornamental plants. Usually, there is very little spread within a tomato crop.

Control:

Elimination of thrips and host weeds through insecticide and herbicide applications is important for disease control. Resistant varieties should be used when possible.



Foliage yellowing and wilting symptoms.



Necrotic leaf lesions.



Yellowing and bronzing leaf symptoms.



Yellow chlorotic rings on the fruit.



White concentric rings on the fruit.



Deformed fruit with diffuse rings.

Tomato Yellow Leaf Curl

Causal Agent:

Tomato yellow leaf curl virus (TYLCV)

Vector:

Whitefly (*Bemisia tabaci*)

Distribution:

Worldwide where tomatoes are grown and the vector is present

Symptoms:

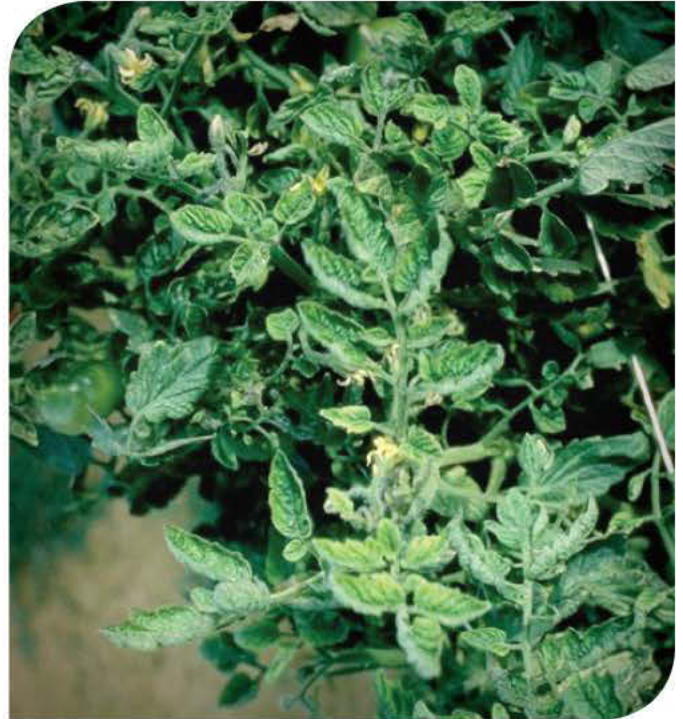
Plants infected at an early stage can be stunted, develop erect branches and have small chlorotic leaflets that cup and twist upward. Severely affected plants generally do not set fruit. Although less severe, yellowing of leaflets, leaf cupping, failure to set fruit and flower abortion can also be common when infection occurs at a later stage. Fruit that has set before the plants become infected often ripens normally.

Conditions for Disease Development:

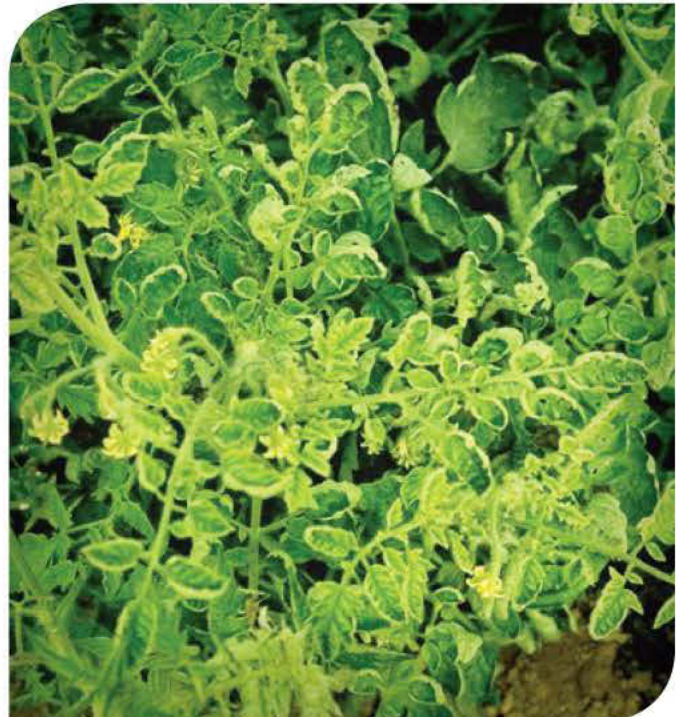
The virus is acquired from infected tomatoes or several solanaceous weeds by the larvae of the sweet potato whitefly, and is transmitted in a persistent manner by the adult whitefly into a tomato crop. Secondary spread of the virus in a field is common. The virus is not mechanically transmitted, so it is unlikely that it will be spread from infected to healthy plants by workers. Severe outbreaks of the disease are often associated with large populations of the whiteflies.

Control:

The use of tolerant varieties greatly reduces losses from this disease, as does the removal of solanaceous weeds that are in the vicinity of the tomato crop. Applying mineral oil on a regular basis may help slow the rate of spread of the disease by reducing the acquisition and transmission of the virus by the whitefly. Covering plant beds with yellow plastic mulch, which attracts the whiteflies, and then spraying on a regular basis with insecticides has proven effective in some areas.



Leaf yellowing and curling.



Severe leaf yellowing and curling.

Common Vectors of Tomato Viruses

Aphids, whiteflies, thrips and leafhoppers are among the more common vectors of viruses that cause severe damage in tomatoes. The mode of transmission can be unique to each virus and vector combination, ranging from non-persistent, or passive transport of viral particles on external mouth parts, to the more complex internal virus-vector relationships of persistent movement. In non-persistent movement, virus particles are picked up during feeding or probing of infected plants and carried on mouth parts to the next feeding site. The insect acquires the virus within seconds but can only transmit it for hours or days. Long acquisition and transmission times characterize persistent movement where the virus is taken up during feeding, must move through the mid-gut lining to the hemolymph, and ultimately into the salivary glands before it can be transmitted. New infections occur when live viruses are injected along with saliva into the phloem of healthy plants. In general, once they become infected, persistent vectors can transmit viruses for the duration of their adult lives.

Aphids:

Aphids are small, pear-shaped insects that feed in groups. They produce live offspring without mating. Aphids can travel from leaf to leaf and from plant to plant as wingless nymphs, and wingless or winged adults. Adults also can travel for miles when carried by wind. Aphids usually invade fields as winged adults and, once established, can occur in large numbers on the underside of the newer leaves.

Aphids cause significant damage to tomatoes because they consume plant nutrients and their sucking feeding behavior can cause chlorosis and distortion of the leaves, abscission of blooms and plant stunting and wilting. Aphids excrete excess plant sap as a sugary honeydew, and sooty mold can grow on the honeydew that is left on fruit and foliage, thus reducing fruit quality. Two common tomato aphid pests are the potato aphid (*Macrosiphum euphorbiae*), which is large (3 mm in length) and pink or green in color, and the green peach aphid (*Myzus persicae*), which is smaller (1.5 mm) and is light to dark green.

The number and diversity of viruses vectored by the numerous genera and species of aphids far exceeds those moved around by other vectors. The ability to probe both surface leaf tissues and the deeper phloem cells without causing significant injury to the host makes aphids efficient virus vectors. Aphids transmit viruses in both a persistent and non-persistent manner. The viruses that are carried by aphids to tomatoes can be acquired and transmitted in seconds and include cucumber mosaic virus, tobacco etch virus and alfalfa mosaic virus.



Green peach aphid, nymphs (*Myzus persicae*)



Green peach aphid, winged adult (*Myzus persicae*)

Common Vectors of Tomato Viruses (continued)

Whiteflies:

The greenhouse whitefly (*Trialeurodes vaporariorum*), sweet potato whitefly (*Bemisia tabaci*) and silverleaf whitefly (*Bemisia argentifolii*) are serious insect pests worldwide. Over 500 plant species including weeds and vegetable, ornamental and agronomic crops are attacked. The undersides of leaves are colonized by all growth stages. The larval stages are sedentary, while tiny (1 mm) adult flies are mobile, moving 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. Whiteflies feed mainly from phloem tissue and cause plant damage similar to that due to aphids. Whiteflies can also cause uneven ripening and a white internal discoloration in fruit.

The viruses transmitted by whiteflies are very important in tropical and subtropical regions, but are not confined to these areas. In tomatoes, the adult sweet potato whitefly is an important vector of geminiviruses such as tomato yellow leaf curl, tomato mottle virus and the numerous Geminiviruses found throughout Mexico, and Central and South America. The adult greenhouse whitefly, *Trialeurodes vaporariorum*, transmits tomato infectious chlorosis virus, a new problem in California tomato production. Most of these viruses are carried persistently, generally throughout the adult life of the vector. There is no evidence to suggest that viruses are passed to the next generation through the egg.



The greenhouse whitefly (*Trialeurodes vaporariorum*)



The silverleaf whitefly (*Bemisia argentifolii*)



The sweet potato whitefly (*Bemisia tabaci*)

Common Vectors of Tomato Viruses (continued)

Thrips:

The western flower thrips (*Frankliniella occidentalis*) is native to the western USA, but has been introduced into many regions worldwide. The onion thrips (*Thrips tabaci*) occurs worldwide. The greenhouse thrips (*Heliothrips haemorrhoidalis*) is found in greenhouses worldwide, where it causes damage to a wide range of ornamental and vegetable plants. Generally, thrips reproduce without mating. The larvae are relatively inactive, but the adults are winged and highly mobile. Adults live up to 20 days, and populations can build up quickly. Thrips feed on the subepidermal cells of the host. The laying of eggs into small developing tomato fruit results in spotting of fruit, and the subsequent feeding by larvae causes scarring.

The onion thrips and the western flower thrips are the major vectors of the tomato spotted wilt virus (TSWV). Only the larvae can acquire the spotted wilt virus, however, it is transmitted exclusively by adults. TSWV is not passed to the next generation via the egg, but there is some evidence this virus may replicate in its vector. Thrips can also spread tobacco streak virus by carrying pollen from an infected plant to a healthy plant while feeding.



The western flower thrips, nymphs (*Frankliniella occidentalis*)



The western flower thrips, adult (*Frankliniella occidentalis*)



The greenhouse thrips, nymphs (*Heliothrips haemorrhoidalis*)



The greenhouse thrips, adult (*Heliothrips haemorrhoidalis*)

Common Vectors of Tomato Viruses (continued)

Leafhoppers:

Leafhoppers are found in warm, dry regions of North America. They are wedge-shaped, can be up to 3 mm long and are green to greenish-yellow to brown. Nymphs are similar to adults except they lack fully developed wings. Leafhoppers have a very wide host range, including numerous weeds and vegetables. They have sucking mouth parts, and 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. The life cycle can be completed in 40-45 days if environmental conditions remain favorable. In California, leafhoppers overwinter in weeds, especially those in the foothills of the Sierra Nevada and Coast ranges. In the spring, as weeds die out, leafhoppers move into adjacent tomato fields.

The beet leafhopper (*Circulifer tenellus*), vectors the curly top virus to tomato. This virus is picked up and transmitted persistently by immature and adult stages of the insect. Once acquired, the virus is carried to the next instar stage, and can be transmitted throughout the adult life. Beet curly top does not multiply in the vector, nor is it carried to the next generation via the egg. The California Department of Food and Agriculture conducts yearly surveys of beet leafhoppers and conducts a spray program for eradication of this vector to help manage curly top disease in tomatoes. The common brown leafhopper, *Orosius argentatus*, vectors big bud, a disease caused by a phytoplasma.



The beet leafhopper (*Circulifer tenellus*)

Noninfectious Disorders

A decorative horizontal band with diagonal stripes in a light beige color, located below the title.

Autogenous Necrosis, Fruit Pox, Gold Fleck

Causal Agent:

Genetic

Distribution:

Worldwide

Symptoms:

Autogenous Necrosis — The first symptom is a yellowing of the upper leaf surface that progresses to a necrosis over time. Tissue necrosis tends to be more prominent on the lower leaf surface and typically starts at the distal end of the leaflet. As the lesions coalesce, the leaves become necrotic and die. Typically, this disorder progresses from the older foliage to the younger.

Fruit Pox — The first symptom on the green fruit is small, clear or tan colored, slightly elongated or oval lesions. As the fruit matures, these lesions enlarge and cause the fruit epidermis to rupture, imparting a necrotic corky appearance.

Gold Fleck — The first symptom on immature green fruit is round, dark green spots on the surface of the fruit. As the fruit matures, these spots change in color to a light tan and then to a golden yellow on ripe fruit.

Conditions for Disease Development:

Autogenous Necrosis — This disorder is caused by an incompatible reaction between a gene for resistance to leaf mold (*Fulvia fulva*) and a tissue necrosis gene that results in 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 the fruit is exposed to high temperatures and the plant 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.

Control:

Use varieties that are tolerant to these genetic disorders.



Autogenous necrosis.



Autogenous necrosis.



Fruit pox.



Fruit pox.



Gold fleck.



Gold fleck.

Blossom-End Rot

Causal Agent:

Calcium deficiency in the distal end of the fruit

Distribution:

Worldwide

Symptoms:

The diagnostic symptom is a light tan lesion turning to a dark brown sunken area at the blossom-end of the fruit. The lesion typically enlarges and becomes more sunken and leathery, and is often accompanied by a dry rot. A black mold may grow on the surface of the lesion. Sometimes there is an internal black rot of tissue in the center of the fruit with little or no external symptoms. Normally, fruit that are about half developed are the first to show symptoms.

Conditions for Disease Development:

In general, any soil or growing condition affecting the uptake of calcium may result in this disorder. Blossom-end rot often occurs during alternating periods of high and low soil moisture as well as rapid plant growth. Other conditions that could cause blossom-end rot include excessive soil salinity and root damage.

Control:

The use of tolerant varieties may help reduce the occurrence of this disorder. Applications of lime or calcium fertilizers before planting are commonly used to reduce the incidence of blossom-end rot. Irrigation during dry weather or applications of mulch to provide a constant moisture supply to the plant can help prevent this problem. The use of excessive nitrogen, especially in the ammonium form, should be avoided since this increases the demand for calcium by the plant and reduces the availability of calcium in the soil. Fields that are difficult to irrigate uniformly or those with high salinity should be avoided. Root injury caused by mechanical damage or disease can exacerbate this condition and should be avoided.



Internal and external blossom-end necrosis.



Light brown tissue necrosis on the blossom-end.



Black sunken lesion on the blossom-end.

Catface, Cracking

Causal Agent: Environmental

Distribution: Worldwide

Symptoms:

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

Cracking — Two types of cracking occur on the fruit:
1) Radial cracking is a splitting of the epidermis that radiates from the calyx-end to the blossom-end of the fruit. 2) Concentric cracking is a splitting of the epidermis in circular patterns around the calyx-end of the fruit. Cracking usually does not occur until the fruit have reached maturity.

Conditions for Disease Development:

Catface — Abnormally cold weather during flowering is known to enhance this disorder. Also, high soil nitrogen levels and any disturbance to the flower parts during anthesis can increase catfacing.

Cracking — Susceptibility to cracking is related to the strength and stretching ability of the fruit's epidermis. Periods of slow fruit growth, followed by fast growth resulting from wide differences in day and night temperatures, and a dry period followed by heavy rain or irrigation are conducive to this disorder.

Control:

The best way to reduce losses from these disorders is to use tolerant varieties. Proper irrigation and nutritional management, and temperature management in greenhouses can also help reduce losses.



Catface.



Radial cracking.



Concentric cracking.

Chemical Damage

Causal Agent:

Herbicides and Insecticides

Distribution:

Worldwide

Symptoms:

Contact herbicides, those that affect only the tissues they contact, typically will cause chlorotic or necrotic spots on all parts of the plant. Multiple spots can result in deformation of the affected tissues. Systemic herbicides, those that are translocated in the plant, tend to cause a variety of symptoms, including stunting. These range from a general yellowing of the foliage to yellowing or necrosis of tissues in the center of the leaf, to a yellowing or necrosis of the leaf veins that may expand to the 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 the stem and petiole. Root growth may be inhibited and the roots may become stubby. Fruit symptoms range from the development of excessively large or small irregular-shaped fruit, to the development of flat or nipped blossom-ends or internal deformations. Insecticides typically cause a leaf margin necrosis or necrotic lesions on the foliage.

Conditions for Disease Development:

Generally, foliar damage occurs when the herbicides and insecticides are applied at excessive rates, at the wrong stage of plant growth or during unfavorable weather conditions. Damage often occurs from herbicide drift when crops or weeds adjacent to the tomatoes have been sprayed. Damage from drift is usually most severe at the edge of the field closest to where the herbicide was applied, with the damage decreasing with increasing distance from the source.

Control:

Use herbicides and insecticides as directed and apply during the appropriate weather conditions.



Foliar deformation and twisting caused by 2, 4-D.



Leaf center chlorosis caused by Glyphosate.



Stem swelling caused by Trifluralin.



Fruit nipping caused by MCPA (top) compared to normal fruit (bottom).



Leaf margin necrosis caused by Oxamyl.

Cloudy Spot

Causal Agent:

Stink bug (*Pentatomids* species)

Distribution:

Worldwide

Symptoms:

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

Conditions for Disease Development:

When the stink bug feeds on the green fruit, it punctures the skin and secretes an enzyme that prevents the development of normal color in ripening fruit. The overwintering adults become active in the spring and fly into tomato fields from surrounding weeds. The edges of tomato fields nearest these weedy areas are often the most affected by the stink bug. All growth stages of the insect can cause this problem.

Control:

Generally, a good insect control program should help reduce losses from stink bug feeding.



Yellow irregular spots on the fruit surface.



Stinkbug and fruit symptoms.

Edema, Puffiness, Sunscald

Causal Agent:

Environmental

Distribution:

Worldwide

Symptoms:

Edema — This disorder is characterized by green callus-like growths on the upper and lower surfaces of the leaf. The growths may rupture as they enlarge.

Puffiness — Affected fruit have an angular appearance and are less dense than normal fruit. When the fruit is cut open, incomplete locule development is apparent, with very few seeds and little gel present.

Sunscald — A white, shiny, leathery area forms on the side of the fruit that is suddenly exposed to direct sunlight. The affected area becomes sunken, and black mold frequently develops on the affected tissue. Sunscald usually develops when the fruit are mature green.

Conditions for Disease Development:

Edema — This disorder develops when the leaf tissue becomes waterlogged, as a result of root pressure continuing to move water up the plant when transpiration is poor. This typically occurs when the soil is warm and wet and the air temperature is cool. Prolonged periods of high humidity favor this disorder.

Puffiness — Factors that lead to poor gel formation and seed set are high and low temperature extremes, the use of fruit hormones and conditions of drought or excessive water. Excessive soil fertility, especially nitrogen, may accentuate the development of this disorder.

Sunscald — Fruit that are suddenly exposed to direct sunlight, due to premature loss of foliage resulting from disease, pruning or breaking over of the plants as the fruit load increases, are the most susceptible to this disorder. Sunscald occurs when the internal fruit temperature increases dramatically, resulting in tissue damage.

Control:

Edema — Proper ventilation for greenhouse crops and maintenance of soil moisture levels can help reduce the incidence of this disorder.

Puffiness — Controlling the temperature, soil fertility and humidity in the greenhouse can help reduce the incidence of this disorder.

Sunscald — The use of wilt-resistant and foliar disease-resistant varieties as well as a good fungicide spray program can help reduce losses to sunscald. Also, losses can be reduced by pruning and harvesting plants carefully to minimize defoliation and fruit exposure to direct sunlight.



Edema on tomato leaf mid-vein.



Puffiness showing the blocky fruit shape and empty locules.



Puffiness showing poor locule, gel and seed development.



Sunscald on exposed fruit in the field.

Gray Wall (syn: Blotchy Ripening)

Causal Agent:

Undetermined etiology

Distribution:

Worldwide

Symptoms:

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

Conditions for Disease Development:

Environmental factors that appear to be associated with this disorder are high nitrogen, low potassium, high soil moisture, high humidity, temperature fluctuations, low light intensity and soil compaction. In addition, certain bacteria, fungi and/or tomato mosaic virus are thought to be involved in gray wall.

Control:

The best control for this disorder is generally the use of tolerant varieties.



Typical blotchy gray fruit walls.



Typical grayish wall discoloration.



The fruit wall has discolored tissue layer.



Cut through the fruit wall showing discolored tissue.



Note the external yellowish blotchy area and internal brown layer.

Nutrient Deficiencies

Causal Agent:

Insufficient nutrients

Distribution:

Worldwide

Symptoms:

The following symptoms are indicative of nutritional deficiencies. However, soil and foliar fertilizer analyses should be conducted to verify nutritional needs.

Nitrogen — The oldest leaves turn chlorotic and eventually senesce, while the younger leaves turn a yellowish-green. The plants may be stunted

Phosphorus — Leaves develop a dull green color and grow slowly. The underside of the leaves eventually turn a reddish-purple. Older leaves are affected first and may senesce in severe cases.

Potassium — The foliage will show a burning of the leaf margins. The older leaves may develop interveinal chlorosis while the veins remain green. The symptoms begin on the older leaves and progress to the younger ones as the disorder becomes increasingly more severe. Fruit disorders such as puffiness, graywall and ripening diseases can result from a potassium deficiency.

Calcium — Interveinal chlorosis and leaf margin necrosis occur at the growing point, which eventually dies. The fruit develop blossom-end rot.

Magnesium — Leaves develop interveinal chlorosis, starting on the older and progressing to the younger leaves. The midrib of the leaf remains green while the interveinal tissue becomes necrotic.

Sulfur — The older leaves turn light green, and the stems and petioles may turn purple and become spindly.

Boron — The older leaves turn yellow and brittle, and the growing point becomes necrotic and dies. The margins of older leaves and the leaf tips become necrotic. The fruit may also be affected and may develop scattered corky areas.

Copper — Initially, the younger leaves wilt and then may turn bluish-green and curl upwards. Severely affected plants are stunted and chlorotic.

Iron — The younger leaves develop interveinal chlorosis followed by a general yellowing. The midrib of the leaf usually remains green.

Manganese — The younger leaves develop interveinal chlorosis followed by necrosis, while the midrib of the leaf remains green.

Molybdenum — The older leaves develop a yellowing and marginal necrosis that eventually progresses to the younger leaves. This deficiency is rare in tomato.

Zinc — The leaves become thickened and curl downward. Their petioles may become twisted and the older leaves exhibit an orange-brown chlorosis.

Conditions for Disease Development:

Nutrient deficiencies are most common in acid or alkaline soils due to the immobilization of nutrients at the lower and higher soil pH's. Some soils are naturally low in specific nutrients due to their compositions. The excessive, or unbalanced, use of fertilizer may also cause some nutrients to be less available. Low temperatures, compaction or excessive soil moisture may also affect nutrient availability.

Control:

Use a balanced fertilizer program. Soil and foliar nutrient analysis can give valuable information on nutritional deficiencies. Altering soil pH and using nutrient sprays can help correct some deficiencies.

Nutrient Deficiencies (continued)



Healthy



Nitrogen (N)



Healthy

Nitrogen (N)



Phosphorus (P)



Potassium (K)



Phosphorus (P)



Potassium (K)



Magnesium (Mg)



Iron (Fe)



Magnesium (Mg)



Manganese (Mn)



Zinc (Zn)

Air Blast	High pressure spraying in which considerable turbulence is created, often resulting in watersoaking of tissue.
Anamorph	The asexual form in the life cycle of a fungus. Asexual spores (conidia) are usually produced.
Anthesis	The point at which a flower is fully open.
Antibody	A protein produced in a warm-blooded animal that 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.
Ascospore	Sexually derived fungal spore usually held with many other ascospores in a sack-like structure (ascus).
Bacterium	(pl. bacteria) Microscopic, single-celled organism.
Blight	The disease symptom in which there is sudden and severe necrosis of the above-ground portions of a plant.
Calyx	The external green, leafy part of a flower consisting of sepals.
Canker	Localized, diseased areas on roots or stems where the tissue is sunken and cracks open.
Chlorophyll	The green pigment used by plants in their food production process.
Chlorosis	(chlorotic) The yellow or white discoloration of normally green tissue.
Coalesce	To come together to form a whole.
Concentric	Different sized circles having a common center.
Conidium	(pl. conidia) A fungal spore formed asexually.
Cotyledon	The first foliar structure to emerge from a seed.
Damping-off	A rotting of seedlings at or below soil level.
Debris	Remnant plant material.
Defoliation	The loss of leaves.
Distal	Located far from the point of attachment.
Diurnal	Occurring or active during the daytime.
Epidermis	The superficial layer of cells occurring on all plant parts.
Fumigation	Sterilizing by fuming action.

Fungicide	A chemical used to control fungi.
Fungus	(pl. fungi) A microscopic organism with thread-like cells that grows on living and/or dead plants.
Gall	Swellings of roots, stems or leaves caused by abnormal growth of tissue.
Girdle	To encircle with dead tissue around a root or stem.
Herbicide	Substance used to control weeds.
Hydathode	A leaf structure that eliminates unused salts, sugars and water from a plant through a pore at the leaf margin.
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 in which an organism attacks a plant.
Inoculum	A potentially infective agent available in soil, air or liquid that could be applied to a host either naturally or artificially to elicit a response.
Insecticide	A substance used to control insects.
Instar	An insect stage between molts before adulthood.
Interveinal	The area of tissue bordered by veins.
Lesion	A well defined but limited diseased area on a plant.
Locule	A cavity within a fruit containing seeds.
Malvaceous	Plants in the mallow family, including okra and cotton.
Microsclerotia	A very small sclerotia.
Mosaic	The pattern of light and dark areas often caused by viruses.
Mottle	Irregular blotches of light and dark areas.
Mycelium	(pl. mycelia) The mass of thin, microscopic, hair-like structures that forms the vegetative part of a fungus.
Necrotic	Tissue that turns color and dies.
Nematode	Tiny worms that can live in plants, animals, soil or water.

Nymph	Juvenile stage of an insect.
Pasteurization	The process of partial sterilization by heating at controlled temperatures to kill undesirable microorganisms.
Pathogen	An agent that incites disease.
Pedicel	The stalk of a flower or fruit.
Percolation	Liquid passing through small pores.
Petiole	The stalk of a leaf.
Phloem	The food-conducting tissue of a plant.
Phytoplasma	An obligate single-celled organism, lacking a cell wall and pleomorphic. Formerly referred to as a Mycoplasma-like organism (MLO).
Pith	Soft, spongy tissue in the center of a plant stem.
Pustule	The small blister-like elevation of epidermis formed, as fungal spores develop and emerge.
Pycnidium	(pl. pycnidia) A spherical or flask-shaped asexual fruiting structure that gives rise to fungal conidia.
Race	A 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	(resistant) The ability of plants to suppress or retard the activities of a specified pest or pathogen. Also, the ability of plants to withstand a specific environmental or chemical stress.
Rootstock	A root used as stock to which another plant is grafted.
Saturation	Being completely filled with liquid, generally water.
Sclerotium	(pl. sclerotia) A hardened resting body produced by certain fungi.
Solanaceous	Plants in the nightshade family, including tobacco, tomato, potato, pepper, eggplant and others.
Solarization	Exposure to direct sunlight.
Spore	A reproductive structure of fungi and some bacteria.
Stomata	A pore in a leaf surface.

- Strain** A general term referring to (a) an isolate; descendent of a pure culture of pathogen, (b) a race; one of a group of similar isolates or (c) one of a group of virus isolates that have common antigens.
- Susceptible** The inability of plants to restrict the activities of a specified pest or pathogen. Also, the inability to withstand a specific environmental or chemical stress.
- Systemic** Spreading internally throughout a plant.
- Teleomorph** The sexual form of a fungus. Sexual spores are produced after meiosis occurs.
- Tolerance** (tolerant) The ability of plants to endure a specified pest, pathogen, environmental pressure or chemical stress. A tolerant variety will sustain less damage than a susceptible variety when grown under the same conditions.
- Toxin** A poison produced by an organism.
- Translocation** The transfer of nutrients or a virus through the plant.
- Translucent** Transmitting light but diffusing it enough to cause images to be blurred.
- Transpiration** The loss of water vapor from the surface of leaves.
- Vascular** Referring to the conductive system of a plant combining the xylem and phloem.
- Vector** An animal able to transmit a pathogen.
- Virus** Very small sub-microscopic disease-causing agent.
- Watersoaked** 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. Volume 2: Vegetables. 1992. A.L. Snowdon. CRC Press Inc. Boca Raton, Florida.

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

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.

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.

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, W. R. Wright. USDA Agricultural Handbook No. 28.

Nutritional Deficiencies and Toxicity's in Crop Plants. 1993. William 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.

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



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