



CUCURBIT DISEASE

FIELD GUIDE

A DISEASE REFERENCE GUIDE FOR CUCUMBER, MELON, SQUASH AND WATERMELON




Seminis


De Ruiter



CUCURBIT DISEASE

FIELD GUIDE

PREFACE

This guide provides descriptions and pictures of the more commonly found diseases and disorders of cucurbits worldwide. For each disease and disorder, the reader will find the common name, causal agent, distribution, symptoms, conditions for disease development and control measures. Among the virus disease descriptions, a vector for each disease has been identified. The photographs illustrate characteristic symptoms of the diseases and disorders included in this guide. It is important to note, however, that many factors can influence the appearance and severity of symptoms.

The primary audience for this guide includes cucurbit crop producers, agricultural advisors, farm managers, agronomists, private consultants, food processors, and members from the chemical and seed industries. This guide should be used as a reference for information about common diseases and disorders as well as their control. However, diagnosis of these diseases and disorders using only this guide is not recommended nor encouraged, and it is not intended to be substituted for the professional opinion of a producer, grower, agronomist, plant pathologist or other professionals involved in the production of cucurbit crops. Even the most experienced plant pathologist relies upon laboratory and greenhouse techniques to confirm a plant disease and/or disease disorder diagnosis. Moreover, this guide is by no means inclusive of every cucurbit disease. Rather, we present those diseases that are currently most prevalent worldwide under protected culture and open field productions. With one exception, insect pests were not included in this publication.

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

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

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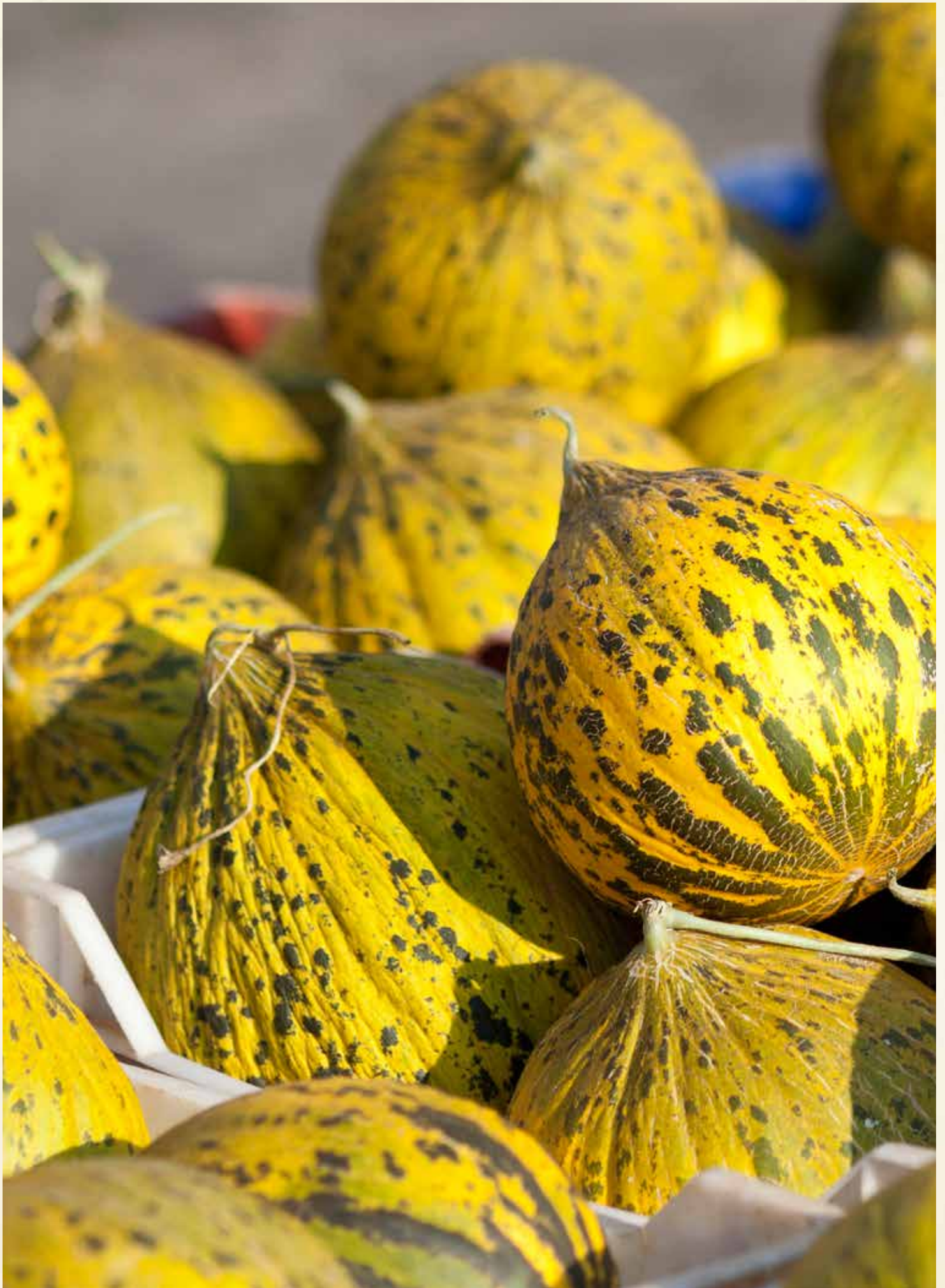
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BACTERIAL

DISEASES

ANGULAR LEAF SPOT

BACTERIAL FRUIT BLOTCH

BACTERIAL FRUIT ROTS

BACTERIAL LEAF SPOT OF CUCURBITS

BACTERIAL WILT

BACTERIAL ANGULAR LEAF SPOT



Water-soaked lesions on the abaxial surface of a cucumber leaf.

CAUSAL AGENT:

Pseudomonas syringae pv. *lachrymans*

DISTRIBUTION:

Worldwide

SYMPTOMS:

This disease can occur on most cucurbits but is of greatest importance on cucumbers. Foliar symptoms initially appear as small, water-soaked areas on the underside of the leaf, which develop an angular appearance due to restriction by the small leaf veins. Under humid conditions, a milky exudate may appear from the water-soaked areas on the lower leaf surface. As this exudate dries, a white crust is left behind. Leaf spots turn brown and may develop yellow haloes. The centers of the spots may eventually disintegrate, giving leaves a tattered appearance. Infection on stems, petioles and fruit first appears as water-soaked spots, which may also produce the milky exudate under humid conditions and corresponding white crust upon drying. Infection of young fruit may result in deformation at maturity. Secondary soft rots often develop on infected fruit.

CONDITIONS FOR DISEASE DEVELOPMENT:

This disease can originate with infested seed, infected transplants, or in the field from infested crop residue or infected volunteer plants. Infection occurs through stomata, hydathodes and wounds. On sandy soils, wind-blown sand can be particularly conducive to infection by abrasion of plant tissues. Humid conditions favor disease development. The bacterium can be spread from plant to plant by splashing water, insects, farm equipment and workers. Moisture on the leaves is especially conducive to spread by equipment and when workers contact plants.

CONTROL:

Rotate out of cucurbits for at least two years. Avoid overhead irrigation and entry into the field when foliage is wet. Copper-based sprays may help limit spread. Resistant varieties of cucumber are available.



Cucumber leaves: left—old, dry and necrotic leaf spots of angular leaf spot; right—young leaf spots surrounded by a yellow halo.

BACTERIAL ANGULAR LEAF SPOT



Tattered leaves characteristic of angular leaf spot on cucumber.



Necrotic lesions on foliage and fruit of Blue Hubbard squash. (Courtesy of Thomas A. Zitter)



Pickling cucumber infected with *Pseudomonas syringae* pv. *lachrymans*.

BACTERIAL BACTERIAL FRUIT BLOTCH

CAUSAL AGENT:

Acidovorax citrulli (synonym = *Acidovorax avenae* subsp. *citrulli*)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Watermelon: The disease may first appear in the nursery on cotyledons as irregularly shaped water-soaked tissue which progresses to brown-black lesions. On young expanding true leaves, small discrete brown lesions may develop along leaf veins. Additional symptoms on seedlings may include chlorosis, pin-point lesions, veinal or interveinal necrosis and damping-off. In the field, lesions that develop along leaf veins eventually dry and may turn reddish-brown to black. Watermelon fruit symptoms first appear as dark, gray-green, water-soaked lesions or blotches on rind surfaces not in contact with the soil. Blotches that develop on fruit tissue in contact with soil are most often associated with fungal infection. As the disease progresses, infected areas on the fruit rinds may rupture or crack.



Water-soaked lesion on a watermelon cotyledon.



Water-soaked lesions on squash cotyledons.



Tan veinal necrosis on a melon leaf. (Courtesy of Ron Walcott)

Atypical bacterial fruit blotch symptoms have been observed on fruits of watermelon grown for edible seed in dry, cool climates. Lesions initially appear on the epidermis as small, pinpoint-sized necrotic spots. As lesions enlarge, brownish-black, star-shaped cracks form in the centers. While light green chlorotic halos may surround lesions, water-soaking is typically not observed. Beneath the external lesions, flesh of the fruit often disintegrates into dry, firm rotten cavities. At advanced stages, fruit may become misshapen and deformed (see images on page 12).

Melon: Cotyledon and leaf lesions on melon are tan-brown in color. Necrosis usually develops sooner and is more prevalent in melon compared to watermelon. Symptoms vary with fruit type. Lesions on smooth-skinned fruit can range from pinpoint spots to small raised or sunken circular areas. Net formation may be disrupted and water-soaking may occur around sunken lesions. While lesions do not necessarily expand externally on the rind, lesions initiating from the fruit surface often expand internally to a conical shape. Secondary fruit rot may develop from internal lesions. Additional fruit symptoms for all melon types may include epidermal cracks and scab-like lesions.

Squash/Pumpkin: Symptoms on cotyledons range from water-soaking to dry necrotic lesions. Damping-off of seedlings may also occur. Pumpkin foliar symptoms may include extensive chlorosis as well as elongated tan lesions along the leaf veins. Shot-holing of leaves is also commonly observed. Fruit symptoms on pumpkin are similar to those found on melon and include water-soaked areas, cracks in the rind and internal rotting of the fruit.

CONDITIONS FOR DISEASE DEVELOPMENT:

Acidovorax citrulli is a seed-borne and seed-transmitted pathogen. Contaminated seed or infected transplants are often the primary source of inoculum leading to outbreaks. Volunteer plants and wild cucurbits species such as citron can also serve as inoculum sources. *Acidovorax citrulli* does not survive for long periods in soil in the absence of host tissue. Infection and disease development are favored by high relative humidity, heavy dew formation or rainfall, combined with warm temperatures. The bacterium is spread by splashing rain, irrigation water, people and equipment. Fruit may be infected through stomata early in development. Infection occurs before formation of the waxy layer in watermelon fruit. Hence, unwounded mature fruit are not considered to be susceptible to infection, although abrasions and other wounds may allow entry of the pathogen leading to fruit infection. *Acidovorax citrulli* is not known to move systemically within the plant. Foliar symptoms can often be mistaken for symptoms caused by other cucurbit pathogens (e.g., *Didymella bryoniae*).

CONTROL:

Use seed that has tested negative for the presence of *Acidovorax citrulli* using a validated seed health testing method. Incorporate crop residues to accelerate breakdown of debris and rogue volunteer seedlings. Rotate out of cucurbits for a minimum of three years and implement a sanitation program for cultivation equipment and field crews. Applications of copper-based products to transplants and throughout the growing season can help minimize disease outbreaks and spread.

BACTERIAL BACTERIAL FRUIT BLOTCH



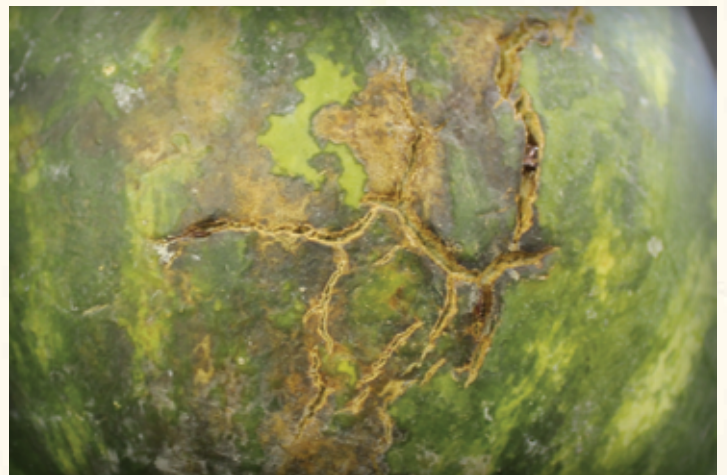
Watermelon leaf collected from the field with typical foliar lesions of bacterial fruit blotch. (Courtesy of Kathryn Everts)



Foliar leaf symptoms on squash. (Courtesy of Parm Randhawa)



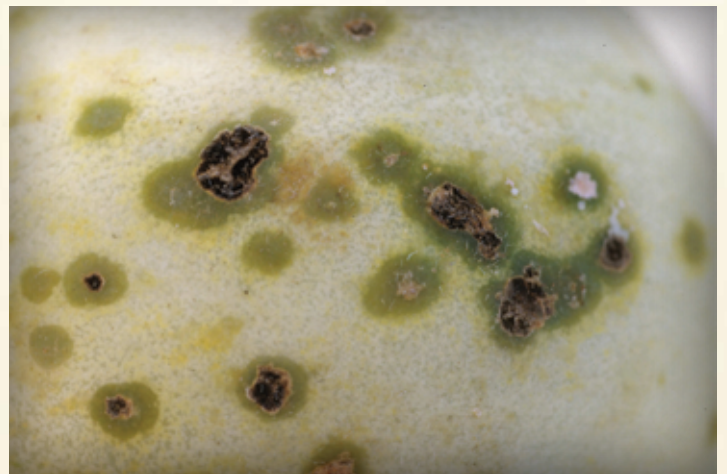
Severe leaf symptoms on melon. (Courtesy of California Department of Food and Agriculture)



Cracking of watermelon rind caused by *Acidovorax citrulli*. (Courtesy of Kathryn Everts)



Epidermal cracking, atypical without water-soaking.

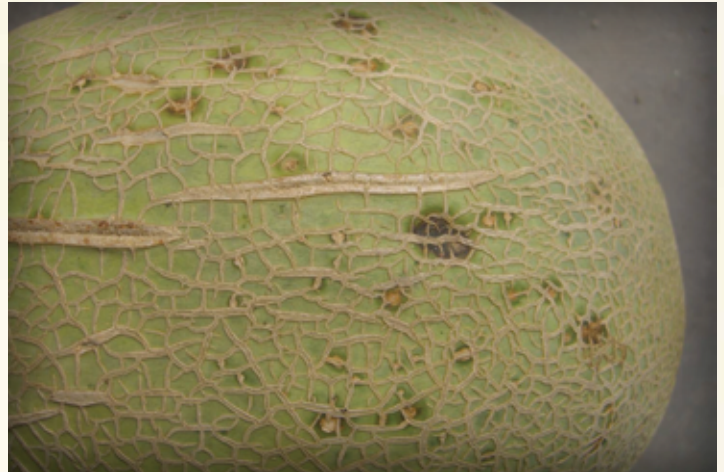


Honeydew melon fruit infection. (Courtesy of Tom Isakeit)

BACTERIAL BACTERIAL FRUIT BLOTCH



Internal and external fruit symptoms on Hami melon. (Courtesy of Laixin Luo)



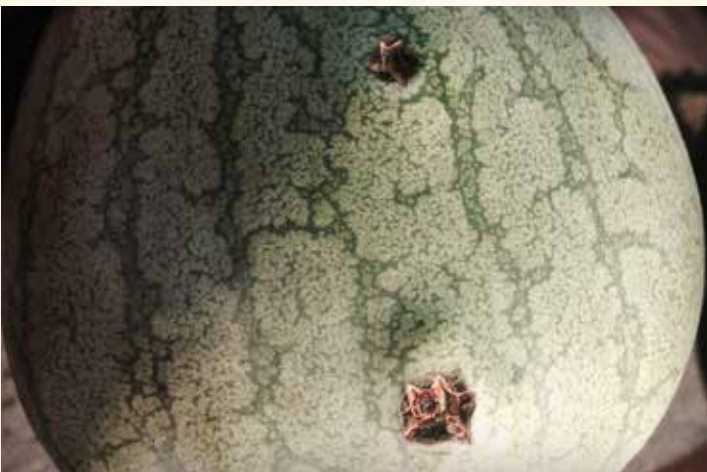
Fruit blotch lesions on a netted melon. (Courtesy of Hu Baishi)



“Classic” bacterial fruit blotch symptoms on a Charleston Gray-type watermelon.



External fruit symptoms on edible watermelon seed fruit. (Courtesy of Ron Walcott)



External fruit symptoms on edible watermelon seed fruit. (Courtesy of Ron Walcott)



Internal fruit symptoms on edible watermelon seed fruit. (Courtesy of Ron Walcott)

BACTERIAL BACTERIAL FRUIT ROTS

CAUSAL AGENTS:

Soft Rot: *Pectobacterium carotovorum* subsp. *carotovorum* (synonym = *Erwinia carotovora* subsp. *carotovora*), *Pseudomonas* spp. and several other bacteria.

Brown Spot: *Pantoea ananas* (synonym = *Erwinia ananas*)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Soft rot manifests as a water-soaked area of the fruit, developing very quickly to complete softening and tissue collapse.

Brown spot has been reported on cantaloupe and honeydew melon types. Lesions are typically smooth, firm and yellow-brown in color. Lesions may extend one to two millimeters into the epidermis, sometimes entering the cavity of the fruit. Symptoms are less conspicuous on netted types.

CONDITIONS FOR DISEASE DEVELOPMENT:

Soft rot occurs most commonly under hot and wet or humid conditions. Other diseases or disorders (e.g. angular leaf spot, anthracnose, blossom-end rot) can predispose fruit to soft rot bacteria. Wounds created during harvest or packing can also be sites for soft rot to develop.

Brown spot develops under similar conditions as bacterial soft rot.

CONTROL:

Avoid bruising, puncturing and other mechanical damage to fruit during harvest and packing. The use of chlorinated fruit dips or sprays in packing houses has been shown to lower incidence of soft rot. Store fruits at an appropriate temperature and relative humidity to prevent condensation on fruit surfaces.



An external wound on a honeydew melon (above), allowing *Pectobacterium carotovorum* subsp. *carotovorum* into the seed cavity where soft rot has developed (below).



Bacterial soft rot caused by *Pectobacterium carotovorum* subsp. *carotovorum*.



Bacterial brown spot of melon caused by *Pantoea ananas*.

BACTERIAL

BACTERIAL LEAF SPOT OF CUCURBITS



Close-up of necrotic leaf spots on pumpkin. (Courtesy of Margaret T. McGrath)

CAUSAL AGENT:

Xanthomonas cucurbitae

DISTRIBUTION:

Worldwide

SYMPTOMS:

Symptoms initially appear on the underside of the leaf as water-soaked lesions, which are mostly angular in shape but may be somewhat rounded. Leaf veins do not appear to define the lesion shape in all cases. Yellowish spots form on the upper surface of the leaf. These spots eventually turn brown or become translucent, retaining a distinct yellow halo. Foliar symptoms may resemble those of angular leaf spot (*Pseudomonas syringae* pv. *lachrymans*). Leaf spots caused by *Xanthomonas cucurbitae* are initially smaller than those caused by the angular leaf spot pathogen, but may resemble angular leaf spot more as they coalesce. The appearance and size of lesions on fruit can vary with rind maturity and the amount of moisture present. Symptoms generally initiate as small, rounded, slightly depressed lesions with tan centers surrounded by dark haloes (somewhat scab-like in appearance). Lesions may become sunken as they progress, resulting in cracking of the rind and fruit rot in the field or in storage.

CONDITIONS FOR DISEASE DEVELOPMENT:

Xanthomonas cucurbitae is known to be associated with seed and can overwinter in crop debris. Infection is favored by high temperatures [25–30°C (77–86°F)] and high relative humidity. Occurrence is common following heavy rains, dew or overhead irrigation.

CONTROL:

Avoid overhead irrigation and entry into the field when foliage is wet. Copper-based sprays applied prior to infection may help limit spread. Destroy infected crop debris by either plowing into the soil or burning. Rotate out of cucurbits for at least two years.



Necrotic lesions on pumpkin caused by *Xanthomonas cucurbitae*. (Courtesy of Margaret T. McGrath)

BACTERIAL BACTERIAL LEAF SPOT OF CUCURBITS



Fruit lesions on cucumber caused by *Xanthomonas cucurbitae*.



Variation in the severity of *Xanthomonas cucurbitae* fruit lesions on pumpkin. (Left: courtesy of Margaret T. McGrath; right: courtesy of Gerald Holmes)

BACTERIAL BACTERIAL WILT



Severe wilting of a cucumber due to *Erwinia tracheiphila* infection.
(Courtesy of Thomas A. Zitter)



Pumpkin runner exhibiting wilting.

CAUSAL AGENT:

Erwinia tracheiphila

VECTORS:

Acalymma vittatum (striped cucumber beetle)

Diabrotica undecimpunctata howardi (spotted cucumber beetle)

DISTRIBUTION:

North America, Asia, Africa, Europe

SYMPTOMS:

This disease is severe on cucumber and melon, but is less damaging to squash and watermelon. Symptoms begin with wilting, which may be confined to individual runners or may involve the entire plant. Plants may wilt at any growth stage, but wilting is often most severe during periods of rapid growth. Affected leaves display marginal chlorosis and necrosis. In time, the entire plant becomes necrotic and dies. A diagnostic procedure for identifying this disease in the field is to make a cut through a symptomatic stem, rejoin the ends and slowly pull the pieces apart. In infected plants, bacteria from the vascular tissue will cohere as filamentous strands between the two pieces.

CONDITIONS FOR DISEASE DEVELOPMENT:

Erwinia tracheiphila is transmitted by cucumber beetles. Environmental conditions have little effect on incidence and spread of the disease, but can influence symptom expression. The bacterium is short-lived in dried plant debris, and does not typically survive in crop debris from one season to the next. Weeds and volunteer cucurbits serve as alternate hosts and facilitate survival between crops.

CONTROL:

Control cucumber beetles which vector *Erwinia tracheiphila* to help control this disease. Eliminate all weeds and volunteer cucurbits. Remove and destroy infected plants as soon as they are identified. Rotate out of cucurbits for 2-3 years.



Filamentous strands of bacteria between two cut stem pieces is a diagnostic for bacterial wilt. (Courtesy of Gerald Holmes)

BACTERIAL BACTERIAL WILT



Squash plant collapse due to bacterial wilt infection. (Courtesy of Howard F. Schwartz)



The striped cucumber beetle (left) (Courtesy of Whitney Cranshaw) and the spotted cucumber beetle (right) are vectors of *Erwinia tracheiphila*.



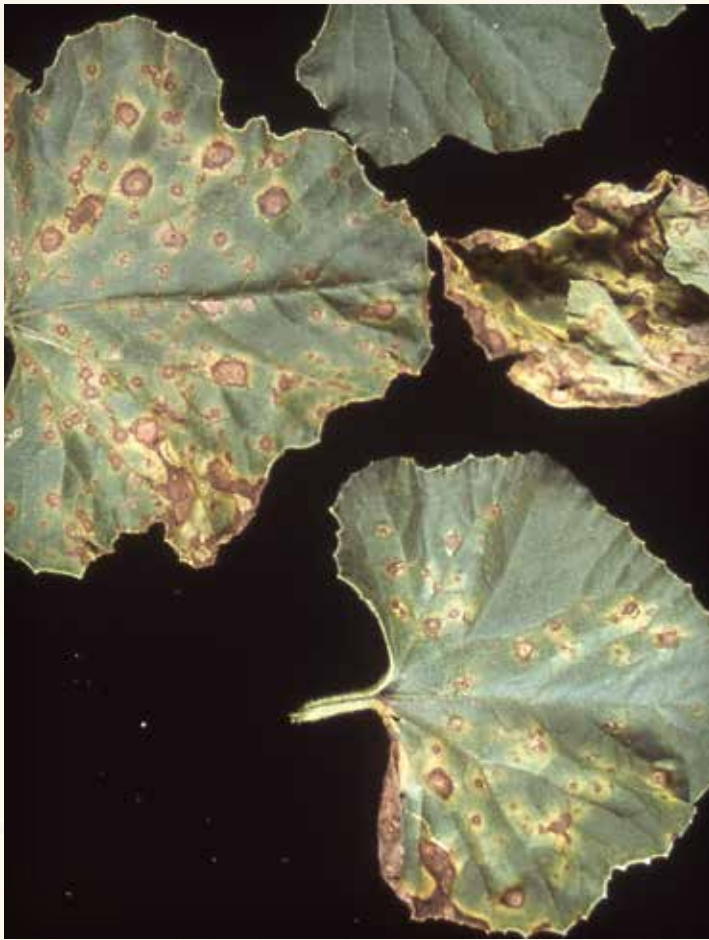


FUNGAL DISEASES

- ALTERNARIA LEAF BLIGHT
- ANTHRACNOSE
- BLACK ROOT OF CUCURBITS
- CERCOSPORA LEAF SPOT
- CHARCOAL ROT
- DAMPING-OFF
- DOWNY MILDEW
- FUNGAL FRUIT ROTS
- FUSARIUM CROWN AND FOOT ROT OF SQUASH
- FUSARIUM ROOT AND STEM ROT OF CUCUMBERS
- FUSARIUM WILT
- GUMMY STEM BLIGHT
- MONOSPORASCUS ROOT ROT AND VINE DECLINE
- PHYTOPHTHORA CROWN AND ROOT ROT
- PLECTOSPORIUM BLIGHT
- POWDERY MILDEW
- SCAB
- SCLEROTINIA STEM ROT
- SOUTHERN BLIGHT
- TARGET LEAF SPOT
- VERTICILLIUM WILT

FUNGAL

ALTERNARIA LEAF BLIGHT



Yellow halos surrounding leaf spots on melon leaves.

CAUSAL AGENT:

Alternaria cucumerina

DISTRIBUTION:

Worldwide

SYMPTOMS:

Alternaria leaf blight is a common disease on cantaloupe and of less importance on cucumber, watermelon and squash. Symptoms first appear on the upper leaf surface as small, circular, tan spots with white centers. These spots enlarge, turn light brown and form a slight depression. Small leaf veins within the spots darken, resulting in a netted appearance. As the spots enlarge on muskmelon and watermelon, concentric rings develop that are visible only on the upper leaf surface, giving the spot a target-like appearance. These circular spots can eventually affect the entire leaf. Defoliation may occur, resulting in sunburn damage to the fruit and may lead to a decrease in fruit soluble solids. Severely affected plants also are more susceptible to heat and wind damage. Infected fruit develop circular, brown sunken lesions. Fruit lesions may develop a dark olive to black-colored powdery mat on the fruit surface. Undetected fruit infection at harvest can result in later losses in transit or storage.

CONDITIONS FOR DISEASE DEVELOPMENT:

Alternaria cucumerina survives in crop debris or on weeds and other cucurbit hosts. Disease spread can occur with rain, irrigation, wind, cultivation, equipment and field workers. This disease is favored by warm temperatures and moisture from dew, rain or overhead irrigation. Infection can be initiated with two to eight hours of leaf wetness, but as the hours of leaf wetness increase infection level increases. The frequency of rain and the length of dew periods play a greater role in disease development than the volume of rain that falls.

CONTROL:

Implement a preventative fungicide spray program. Employ other cultural control measures such as crop rotation (two years out of cucurbits), avoid overhead irrigation, thoroughly incorporate crop debris following harvest and implement a hygiene program for personnel and equipment. For some crops (e.g., cucumber), resistant varieties are available.



Leaf spots with concentric rings. (Courtesy of Paul Bachi)

FUNGAL ALTERNARIA LEAF BLIGHT



Pronounced yellow halos surrounding lesions of *Alternaria cucumerina* on melon. (Courtesy of Anthony Keinath)



Over time, leaf spot lesions on melon coalesce leading to the blighting of foliage. (Courtesy of Kenneth Seebold Jr.)

FUNGAL ANTHRACNOSE



Lesions with cracked centers on a cucumber leaf, characteristic of anthracnose. (Courtesy of Anthony Keinath)



Foliar symptoms on melon. (Courtesy of Dominique Blancard)

CAUSAL AGENT:

Colletotrichum orbiculare

DISTRIBUTION:

Worldwide

SYMPTOMS:

This disease is most commonly found on cucumber, melon and watermelon. Symptoms on leaves begin as water-soaked spots which typically become yellowish in appearance on cucumber and melon or dark brown to black on watermelon. These spots eventually turn brown and may expand over the leaf surface. Foliar lesions are not restricted by leaf veins and often have cracked centers. Infected petioles and stems may develop shallow, elongated, tan lesions on melon but the lesions are less obvious on cucumber. Stem lesions on melon can girdle the stem and cause plant wilting. Infected fruit develop circular, sunken, blackish lesions where tiny fruiting bodies (acervuli) may develop. Under humid conditions, the fruiting bodies produce conidia which give the lesions a pinkish-salmon color, which is very characteristic of this disease. When pedicels of young fruit become infected, the fruit may shrivel and abort.

CONDITIONS FOR DISEASE DEVELOPMENT:

Colletotrichum orbiculare can be associated with seed and infected crop debris. Spread of this fungus can occur by splashing rain, overhead irrigation, insects, field workers and equipment. Disease development is favored by warm, humid weather. Optimum temperature for disease development is 24°C (75°F). Late infection of the crop may result in fruit becoming unmarketable during storage, shipment or display.

CONTROL:

Implement a comprehensive preventative fungicide spray program. Employ other cultural control measures, such as crop rotation (two years out of cucurbits), avoid overhead irrigation, thoroughly incorporate crop debris following harvest and implement a hygiene program for personnel and equipment. Use resistant varieties when available.



Foliar symptoms on watermelon. (Courtesy of Gerald Holmes)

FUNGAL ANTHRACNOSE



Close-up of a fruit lesion on melon covered by tan-orange conidia. (Courtesy of Paul Bachi)



Post-harvest fruit symptoms on slicing cucumber. (Courtesy of Charles Averre)



Fruit infection on a Charentais melon. (Courtesy of Dominique Blancard)

FUNGAL

BLACK ROOT OF CUCURBITS



Cucumber plants exhibiting wilting due to *Phomopsis sclerotioides* infection.

CAUSAL AGENTS:

Phomopsis sclerotioides

DISTRIBUTION:

Asia, Europe and Canada

SYMPTOMS:

Black root of cucurbit is an important soil-borne pathogen that attacks cucumber, although melon and bottle gourd are also susceptible. Young plants are stunted and wilted. Roots are underdeveloped and rotted, exhibiting a blackened appearance due to the formation of pseudosclerotia. Leaf senescence increases on affected plants, resulting in appreciable yield losses. Foliar symptoms can appear similar to symptoms caused by vascular wilt fungi (e.g., *Fusarium*, *Verticillium*).

CONDITIONS FOR DISEASE DEVELOPMENT:

Infection is favored by temperatures below 20°C (68°F). However, as temperatures become warmer and/or water requirements increase, disease progression also increases. *Phomopsis sclerotioides* survival in soil is believed to be by means of pseudostromata and pseudosclerotia. The potential for infection becomes greater in fields where cucurbits have been grown year after year.

CONTROL:

Crop rotation has not been shown to be an effective control measure for black root of cucurbits due to the longevity of pseudosclerotia in soil. Soil fumigation and/or steam sterilization can help reduce fungal populations in soil, but grafting onto a squash rootstock and/or moving production out of soil and into an artificial substrate offer the best options for control.



Pseudosclerotia on melon roots. (Courtesy of Dominique Blancard)



Root lesions on melon. (Courtesy of Dominique Blancard)

FUNGAL BLACK ROOT OF CUCURBITS



Melon roots decaying due to *Phomopsis sclerotioides* infection. (Courtesy of Dominique Blancard)



Charentais melon plant collapse caused by *Phomopsis sclerotioides*. (Courtesy of Dominique Blancard).



Wilting and plant death of greenhouse-grown cucumber.

FUNGAL

CERCOSPORA LEAF SPOT



Circular to irregularly shaped lesions on melon leaves. (Courtesy of Gerald Holmes)

CAUSAL AGENT:

Cercospora citrullina

DISTRIBUTION:

Worldwide

SYMPTOMS:

Cercospora leaf spot occurs on all cucurbits but is most common on watermelon, cantaloupe, and cucumber. This disease is usually found only on the foliage, but if the environment is suitable, symptoms may also occur on petioles and stems. The fungus is not known to infect fruit. On watermelon, leaf spots manifest on young leaves as small grey or white spots with black margins. Larger leaf spots which are circular to irregularly circular develop on other cucurbits. The centers of these leaf spots are tan to light brown becoming transparent and brittle with time. Lesions with surrounding chlorotic halos may coalesce and turn leaves yellow. Although defoliation from the disease may reduce fruit size and quality, serious economic losses are rare.

CONDITIONS FOR DISEASE DEVELOPMENT:

Conidia of *Cercospora citrullina* become airborne and may be carried great distances on moist winds. Infection requires free moisture and is favored by temperatures of 26–32°C (80–90°F). *Cercospora citrullina* survives on crop debris, volunteers and cucurbit weeds.

CONTROL:

Incorporate cucurbit debris into the soil to hasten its breakdown and /or remove pruning debris entirely from the field. Rotate out of cucurbits for two to three years and establish a fungicide spray program to help control this disease.



Necrotic lesions with light brown centers caused by *Cercospora citrullina* infection on melon leaves. (Courtesy of Gerald Holmes)

FUNGAL CERCOSPORA LEAF SPOT



Cercospora citrullina lesions on a watermelon leaf. (Courtesy of Tom Isakeit)



Melon infected with *Cercospora citrullina*. (Courtesy of Gerald Holmes)

FUNGAL CHARCOAL ROT



Microsclerotia on a mature melon stem. (Courtesy of Dominique Blancard)

CAUSAL AGENT:

Macrophomina phaseolina

DISTRIBUTION:

Worldwide

SYMPTOMS:

This soil-borne fungus can attack roots, stems or fruit in contact with the soil. On seedlings, black, sunken cankers may appear on hypocotyls at the time of emergence. These cankers may develop a concentric ring pattern, stunt affected plants and cause wilt. When older plants are attacked, runners and crown leaves may turn yellow and die. Typically, a water-soaked lesion will occur at the soil level and extend several centimeters up the stem. Brown, water-soaked lesions are also symptomatic of fruit infection. Amber-colored droplets of exudate may form within the affected area. Eventually, the lesion dries up, turns light tan and microsclerotia form.

CONDITIONS FOR DISEASE DEVELOPMENT:

Macrophomina phaseolina is seed-borne and can be seed-transmitted. Infection and disease development are favored by high temperatures. High soil salinity, drought stress and heavy fruit load can predispose plants to infection. Microsclerotia in infected host tissue and in soil are the primary propagules and survival structures. Microsclerotia reside in the top 0–20 cm of soil and are able to survive from 2–15 years, depending on environmental conditions.

CONTROL:

Manage irrigation to avoid drought stress. If soil salinity is high, leach to reduce salt buildup. Drip irrigation may result in higher soil salinity compared to furrow irrigation if salinity of the irrigation water is moderate to high. Destroy or deep-plow all plant debris at the end of the season. A three-year rotation out of cucurbits to a non-host species may be beneficial. However, this strategy is not as effective at controlling *Macrophomina phaseolina* as it is with other pathogens due to its wide host range and the longevity of the microsclerotia.



Yellowing of melon leaves due to charcoal rot infection. (Courtesy of Tom Isakeit)

FUNGAL CHARCOAL ROT



Dark amber droplets exuded from a lesion near the crown of a melon plant.
(Courtesy of Dominique Blancard)



Charcoal rot on melon fruit.



External (left) and internal (right) charcoal rot symptoms on melon. (Courtesy of Gerald Holmes)

FUNGAL DAMPING-OFF



Pythium sp. infection of a melon seedling in a nursery tray. (Courtesy of Dan Egel)



A watermelon seedling infected by *Pythium* sp. (Courtesy of Gerald Holmes)

CAUSAL AGENTS:

Pythium spp., *Rhizoctonia solani*, *Acremonium* spp., *Fusarium equiseti*, and other fungi.

DISTRIBUTION:

Worldwide

SYMPTOMS:

Pre-emergence damping-off: Seeds may rot before germinating or seedlings may die prior to emergence.

Post-emergence damping-off: Young seedlings develop a rot at the crown; later, the tissue becomes soft and constricted and the plants wilt and fall over.

***Pythium* spp.:** Seedlings turn dull green and cotyledons droop. Water-soaked lesions develop on the hypocotyls at the soil line and seedlings wilt and collapse. Seedlings may also rot in the soil before emergence.

***Rhizoctonia solani*:** This fungus can infect seed, preventing germination. Symptoms on young seedlings are similar to those caused by *Pythium* species. On older seedlings, a depressed tan to reddish-brown dry lesion may be observed on the hypocotyl.

***Acremonium* spp.:** Symptoms develop 7–10 days after seedlings emerge. Infection begins where the seed coat remains attached to the hypocotyl. This area turns a light yellow-brown color. Within two to three days a dry red-brown rot develops, which may lead to seedling death. Surviving seedlings remain stunted.

***Fusarium equiseti*:** A dry, reddish-brown rot occurs on the hypocotyl. The fungus causes both pre- and post-emergence damping-off.

***Thielaviopsis basicola*:** Lesions begin as grey to reddish areas that almost immediately turn coal black in color. In wet soil, a frosty coating may cover parts of the black lesion.

CONDITIONS FOR DISEASE DEVELOPMENT:

Damping-off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather. In addition, *Acremonium* root rot is favored by deep planting. *Fusarium equiseti* attacks melons which have been seeded into cool, moist soil which later crusts around or over the hypocotyls. Seedlings are most susceptible to damping-off prior to emergence or within the first week after emergence. In greenhouses, incompletely pasteurized soil is common source of damping-off fungi and overwatering commonly exacerbates damping-off.

CONTROL:

Open Field: In addition to the greenhouse measures described above, avoid soil compaction, prepare high beds to obtain better drainage and avoid long irrigation periods. *Acremonium* root rot can also be minimized by shallow planting in dry soil followed by irrigation.

Protected Culture: Ensure that substrate/soil consists of components which favor drainage and aeration. Use a reputable substrate/soil supplier. Implement sanitation measures for supplies and equipment. Manage irrigation practices to avoid long periods of high soil moisture. Use high quality seed to help reduce damping-off. Fungicidal soil drenches and seed treatments are available that help manage damping-off. The use of a biological control agent (e.g., *Trichoderma harzianum*) has been shown to be effective in controlling damping-off pathogens in various cucurbits.

FUNGAL DAMPING-OFF



Watermelon seedlings infected with *Thielaviopsis basicola*. (Courtesy of Gerald Holmes)



Rhizoctonia sp. infection of watermelon seedlings.

FUNGAL DOWNY MILDEW



Lesions becoming chlorotic on the adaxial side of a cucumber leaf.



Chlorotic and developing necrotic lesions assuming an angular appearance.

CAUSAL AGENT:

Pseudoperonospora cubensis

DISTRIBUTION:

Worldwide

SYMPTOMS:

Symptoms initially appear as small chlorotic lesions on older leaves, later appearing on the younger leaves. The margins of these lesions are generally irregular on most cucurbit species. However, on cucumber, lesion margins are defined by the leaf veins which give an angular appearance to the lesions. When leaf surfaces remain wet for extended periods, water-soaked lesions develop on the undersides of leaves. These lesions can appear similar to those caused by *Pseudomonas syringae* pv. *lachrymans*. In humid environments, sporangia form on the underside of leaves, giving the appearance of a whitish-gray to purple fine downy growth. Lesions eventually coalesce and become necrotic, but may continue to expand until the entire leaf dies. Severe infection results in defoliation, stunting of plants and poor fruit development.

CONDITIONS FOR DISEASE DEVELOPMENT:

Survival of *Pseudoperonospora cubensis* between growing seasons is dependent on living cucurbit hosts. Sporangia may be transmitted considerable distances between fields by wind. Within fields sporangia are spread by air currents, splashing water, workers and/or equipment. Fog, dew and frequent rain favor disease development, which can be rapid when temperatures are moderate to warm. High temperatures [$>35^{\circ}\text{C}$ ($>95^{\circ}\text{F}$)] are not favorable for disease development, but disease development may progress if night temperatures are cool [$15\text{--}20^{\circ}\text{C}$ ($59\text{--}68^{\circ}\text{F}$)].

CONTROL:

Provide adequate spacing between plants to reduce canopy density. Grow varieties with genetic resistance to *Pseudoperonospora cubensis*. Implement a preventative fungicide spray program. Regional disease forecasting models have been used successfully to predict onset of symptoms and to time spray applications for effective control of *Pseudoperonospora cubensis*.



Necrotic foliar lesions on melon. (Courtesy of Gerald Holmes)

FUNGAL DOWNY MILDEW



Downy mildew infecting foliage of a grey zucchini plant.



Water-soaked and necrotic lesions on the abaxial side of a cucumber leaf.



Downy mildew sporulation on cucumber can differ in appearance under protected culture setting (left) as opposed to open field infection (right).

FUNGAL

FUNGAL FRUIT ROTS



Botrytis cinerea infection on cucumber fruit.



Botrytis cinerea infection on melon fruit.

CAUSAL AGENTS:

Alternaria alternata f.sp. *cucurbitae*, *Botrytis cinerea*, *Choanephora cucurbitarum*, *Fusarium* spp., *Lasiodiplodia theobromae*, *Myrothecium roridum*, *Penicillium digitatum*, *Phomopsis cucurbitae*, *Phytophthora* spp., *Pythium* spp., *Rhizoctonia solani*, *Rhizopus stolonifer*, *Trichothecium roseum* and other fungi.

DISTRIBUTION:

Worldwide

SYMPTOMS:

Symptoms vary depending on environmental conditions and the fungal fruit pathogen(s) present.

CONDITIONS FOR DISEASE DEVELOPMENT:

Fruit-rotting fungi enter fruit when the fruit contact damp soil. Wounds and attached blossoms are also points of entry for fruit-rotting fungi. Fruit are more likely to be infected when relative humidity is high or if free moisture is present on fruit surfaces. Chilling damage also makes fruit more susceptible to infection by fruit-rotting fungi.

CONTROL:

Crop management practices that prevent fruit contact with the soil surface, reduce fruit injury especially during harvest and packing, and reduce post-harvest free moisture on fruit surfaces will help decrease fruit rot incidence. In some cases post-harvest application of fungicides may help reduce fruit rot losses. Post-harvest treatment of fruit with hot water and fungicides show limited success in controlling latent infections.



Choanephora cucurbitarum infection on summer squash fruit. (Courtesy of Gerald Holmes)

FUNGAL

FUNGAL FRUIT ROTS

DISEASE	ORGANISM	CROP SPECIES	SYMPTOMS
Alternaria fruit rot	<i>Alternaria alternata</i> f. sp. <i>cucurbitae</i>	Melon	Small, dark, shallow, circular lesions that can occur anywhere on the fruit surface. The lesions can extend into the fruit flesh. In some cases, light-gray to black mycelium will grow abundantly over the melon surface.
Belly rot	<i>Rhizoctonia solani</i>	Cucumber, Cantaloupe	Symptoms occur where the fruit epidermis was in contact with the soil surface. Water-soaked areas of decay turn from tan to dark brown. Small cracks in the epidermis may occur in rotted areas.
Blue mold rot	<i>Penicillium digitatum</i>	Cantaloupe	The blossom end is colonized by bluish furry-appearing fungal growth with a white border.
Choanephora rot	<i>Choanephora cucurbitarum</i>	Squash	The fungus infects the blossoms first. Fluffy, white mycelial growth colonizes the fruit surface. Purple-black fruiting bodies then develop from the mycelium. The fruit turns soft and watery.
Crater rot	<i>Myrothecium roridum</i>	Melon	Dark, sunken lesions that can be deep or shallow and range in diameter from 2–50 mm develop. Large lesions may penetrate the seed cavity. Rotted fruit tissue may appear 'watery' but will have no pronounced odor.
Cottony leak	<i>Pythium aphanidermatum</i> , <i>P. debaryanum</i> , <i>P. ultimum</i> and other species	Cucumber, Squash, Watermelon	A soft, watery rot progresses from the blossom end of the fruit. Rotted areas are covered with white cottony growth.
Fusarium rot	<i>Fusarium roseum</i> and other species	Cantaloupe	Ripe fruit are usually affected. Tan-colored spots with white mycelial growth beneath the epidermis. The spots can be easily teased apart from surrounding tissue.
Gray mold	<i>Botrytis cinerea</i>	Cucumber	The blossom end of the fruit is colonized by furry gray fungal growth.
Lasioidiplodia fruit rot = Diplodia stem-end rot	<i>Lasioidiplodia theobromae</i> (synonym = <i>Diplodia natalensis</i>)	Cantaloupe, Watermelon	The stem end of the fruit shrivels and turns brown. The fruit first appears water-soaked and later darkens and shrivels.
Phomopsis fruit rot	<i>Phomopsis cucurbitae</i>	Cucumber, Melon, Watermelon	Soft, circular, slightly sunken water-soaked lesions 1–5 cm in diameter develop. Affected tissue is easily delineated from healthy tissue and the texture is spongy, not watery.
Phytophthora rot	<i>Phytophthora capsici</i> and other species	Cantaloupe, Cucumber, Watermelon	Soft, sunken spots form on fruit surfaces. Under conditions of high humidity, white mycelium may colonize the fruit surface.
Pink mold rot	<i>Trichothecium roseum</i>	Cantaloupe	The blossom end of fruit is colonized by furry, pink mycelial growth.
Rhizopus soft rot	<i>Rhizopus stolonifer</i>	Cantaloupe, Cucumber, Squash, Watermelon	Large, water-soaked spots with defined margins develop on fruit. Later the water-soaked spots become soft and sunken. Gray fungal mycelium may grow in the seed cavity.

FUNGAL FUNGAL FRUIT ROTS



Fusarium sp. infection on melon fruit exhibiting mycelial growth. (Courtesy of Bill Copes)



Internal symptoms of *Fusarium* sp. in melon fruit.



Melon fruit on the left is infected with a *Fusarium* sp. (Courtesy of Bill Copes)

FUNGAL FUNGAL FRUIT ROTS



Rhizoctonia solani fruit infection of cucumber fruit.



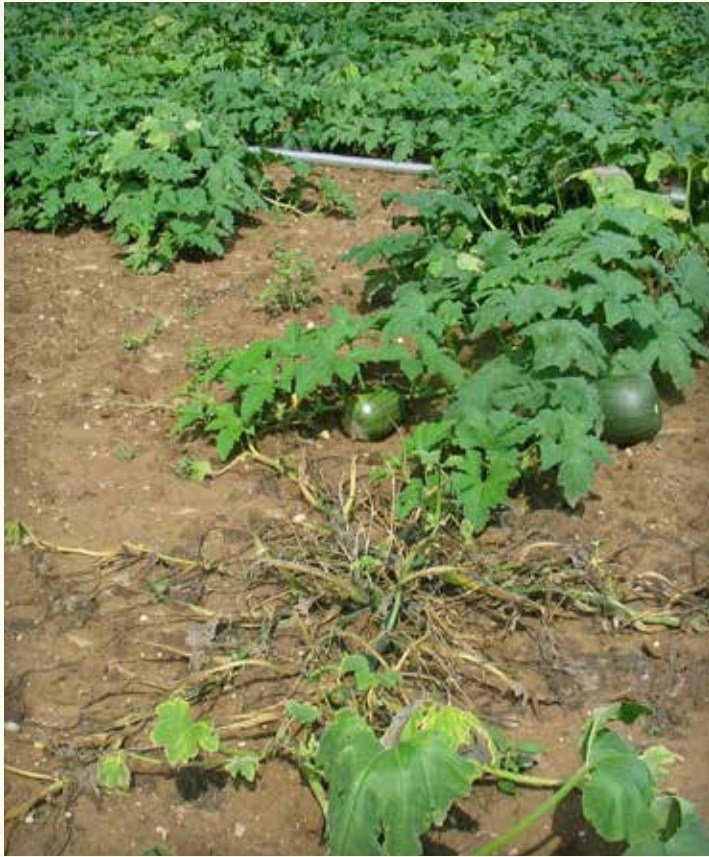
Pythium sp. fruit infection on cucumber fruit.



Pythium sp. fruit infection on squash. (Courtesy of Gerald Holmes)

FUNGAL

FUSARIUM CROWN AND FOOT ROT OF SQUASH



Total plant collapse of pumpkin due to *Fusarium solani* f. sp. *cucurbitae*.
(Courtesy of Margaret T. McGrath)



Wilting of pumpkin runners due to infection by *Fusarium solani* f. sp. *cucurbitae*.

CAUSAL AGENT:

Fusarium solani f. sp. *cucurbitae*

DISTRIBUTION:

Africa, Asia, Europe, and North America

SYMPTOMS:

The disease is most serious on squash and pumpkin, although it may also be found on watermelon, cantaloupe and cucumber. *Fusarium solani* f. sp. *cucurbitae* race 1 attacks roots, stems and fruit, but *Fusarium solani* f. sp. *cucurbitae* race 2 attacks only fruit. Symptoms are similar to Fusarium wilt and include stunting of the plant, and mid-season wilting of the entire plant. However, with Fusarium crown and foot rot there is a distinctive dark brown necrotic rot of the crown and upper portion of the tap root. This decay extends around the stem and girdles the plant. The affected area turns soft and mushy. During humid weather, white mycelial growth may be found on the affected area. The roots of the plant can also become infected but the pathogen primarily is limited to the crown and fruit of the plant. When fruit are attacked a firm dry rot develops. The decayed areas are circular and may develop a concentric ring pattern.

CONDITIONS FOR DISEASE DEVELOPMENT:

The fungus can survive in soil although not for long periods of time. Early infection may result in seedling damping-off. *Fusarium solani* f. sp. *cucurbitae* can also infect fruit which are in contact with infested soil. Severity of fruit rot is dependent on soil moisture and inoculum density.

CONTROL:

Plant fungicide-treated seed to reduce disease initiated from infected seed. The fungus outside of a host is not long-lived in the soil, therefore a three-to-four-year rotation out of cucurbits is usually adequate to control the disease.



Crown area of a pumpkin plant showing severe *Fusarium solani* f. sp. *cucurbitae* infection.
(Courtesy of Margaret T. McGrath)

FUNGAL FUSARIUM CROWN AND FOOT ROT OF SQUASH



Heavy sporulation of *Fusarium solani* f. sp. *cucurbitae* on a pumpkin fruit.
(Courtesy of Margaret T. McGrath)



Two corky-like lesions of *Fusarium solani* f. sp. *cucurbitae* (race 1) invading the flesh of a pumpkin fruit. (Courtesy of Hillary Mehl)



Fusarium solani f. sp. *cucurbitae* (race 2) sporulation on an ornamental gourd.



Circular firm, dry lesions characteristic of *Fusarium solani* f. sp. *cucurbitae* infection on the underside of a pumpkin fruit. (Courtesy of Hillary Mehl)

FUNGAL

FUSARIUM ROOT AND STEM ROT OF CUCUMBER



Cucumber plant wilting due to *Fusarium oxysporum* f. sp. *radicis-cucumerinum* infection.

CAUSAL AGENT:

Fusarium oxysporum f. sp. *radicis-cucumerinum*

DISTRIBUTION:

Worldwide

SYMPTOMS:

Cucumber and muskmelon (e.g., cantaloupe, honeydew) are very susceptible to *Fusarium oxysporum* f. sp. *radicis-cucumerinum*. In cucumber, initial symptoms manifest six to eight weeks after sowing as pale yellow lesions at the stem base. These lesions may expand and spread to cause a root and stem rot. As the disease progresses, stems are colonized by the fungus leading to breakdown of cortical tissues. In severely affected plants, pinkish-orange masses of macroconidia and microconidia and/or a cottony-like mycelial growth may be observed on the outside of the stem. Plants bearing high fruit loads may eventually turn brown and die, especially when grown under high temperatures.

CONDITIONS FOR DISEASE DEVELOPMENT:

This fungus can survive for several years as thick-walled chlamydozoospores in soil or embedded in plant debris. Dispersal of *Fusarium oxysporum* f. sp. *radicis-cucumerinum* within and between fields occurs in crop debris, wind-blown soil, rain and irrigation water, and field crews and equipment. Infection commonly occurs at the root tips or through roots wounded during transplant and cultivation.

In protected culture, *Fusarium oxysporum* f. sp. *radicis-cucumerinum* can colonize artificial growing media (e.g., blocks and/or slabs). Spread of this fungus occurs through root-to-root contact or by aerial dispersal of macroconidia and microconidia. Pruning creates wounds which are ideal entry sites for airborne macroconidia and microconidia. Disease development is favored by cool air temperatures and soil temperatures ranging from 17–20°C (63–68°F).

CONTROL:

Two key measures aid control of *Fusarium oxysporum* f. sp. *radicis-cucumerinum*: crop rotation and a strict sanitation program for facilities, equipment, tools and people. Early implementation of a strict sanitation program is critical to preventing this disease in protected culture, as infection occurs most often within the first four weeks of a crop cycle. Employ routine scouting and immediately remove all weak and unhealthy transplants. Following harvest, dispose of all crop debris and materials used during production, and all artificial media that contained infected plants. Infected plant debris may be incinerated, buried or taken to a landfill. Sanitize greenhouses and or other growing structures with a recommended disinfectant. If growing media must be reused, it should be steam sterilized, although starting with new substrate is preferable. Resistance to *Fusarium oxysporum* f. sp. *radicis-cucumerinum* has been identified in rootstocks of *Cucurbita* spp. (e.g., *C. ficifolia*, *C. moschata*). However, there are no known sources of resistance in cucumber (*Cucumis sativus*). Currently there are no registered fungicides targeting this *Fusarium* species. The biological control agent *Clonostachys rosea* f. sp. *catenulata* has been shown to suppress *Fusarium oxysporum* f. sp. *radicis-cucumerinum* in cucumber grown hydroponically in an artificial substrate.

FUNGAL FUSARIUM ROOT AND STEM ROT OF CUCUMBER



Stem rot infection on cucumber grown in an artificial substrate.



Stem rot infection near the soil line.



Severe infection leading to plant death.

FUNGAL FUSARIUM WILT



Cucumber plant wilting due to *Fusarium oxysporum* f. sp. *cucumerinum* infection.



Yellowing of leaves on a melon seedling infected with *Fusarium oxysporum* f. sp. *melonis*.

CAUSAL AGENTS:

Fusarium oxysporum f. sp. *cucumerinum* (cucumber)

Fusarium oxysporum f. sp. *melonis* (cantaloupe)

Fusarium oxysporum f. sp. *niveum* (watermelon)

DISTRIBUTION:

Worldwide

SYMPTOMS:

These fungi can infect host species at any stage of plant development. Pre-emergence rot and damping-off can occur when soil temperatures are cool [18–20°C (64–68°F)]. However, infection of older plants is most common. Wilted leaves may exhibit chlorosis and later, interveinal necrosis. Initially one or more runners wilt and later the entire plant wilts. In some cases, sudden collapse occurs without any chlorosis of the foliage. Mature infected plants with high fruit loads are prone to plant collapse. White mycelial growth may be visible on the stem at the base of infected plants. Vascular discoloration can be seen in both the roots and stems. In advanced stages, the roots begin to decompose, which may lead to plant death.

CONDITIONS FOR DISEASE DEVELOPMENT:

Fusarium wilt is favored by warm soil temperatures. Wilting of the lower leaves occurs at warm temperatures [25–28°C (77–82°F)] and the leaves recover under cooler temperatures. The fungus survives as chlamydozoospores in the soil and in plant debris. The chlamydozoospores are readily disseminated in soil and in debris during cultivation of fields, in irrigation water, by wind-blown soil and workers.

CONTROL:

For all cucurbit species, grow resistant varieties when available.

Open Field: Graft onto resistant rootstock (e.g., *Cucurbita ficifolia*). Soil solarization has been shown to be beneficial in light to moderately infested fields. Maintaining a soil pH of 6.5 and use of a NO₃ nitrogen source can help reduce disease incidence and severity. Implement an equipment and worker sanitation program to control movement of the pathogen between fields. Crop rotation out of cucurbits for a minimum of five years may provide control in some cases, although is not always an effective strategy due to long-term persistence of chlamydozoospores in many soils.

Protected Culture: Implement a strict sanitation program for workers, tools, equipment and structures. Produce crops in artificial substrate and do not reuse substrate. Graft crop species onto a resistant rootstock (e.g., *Cucurbita maxima*, *Cucurbita ficifolia*). Collect and remove pruning debris from site or destroy by burning or burying. Remove infected plants upon recognition. Crop production during cooler months may reduce disease incidence and severity since *Fusarium* wilt is not favored at lower temperatures.

FUNGAL FUSARIUM WILT



A melon field showing plant collapse due to Fusarium wilt.



Wilting of runners on a watermelon plant. (Courtesy of Tom Isakeit)



Vascular discoloration characteristic of *Fusarium* sp. infection. (Courtesy of Dan Egel)



Cross section of a stem demonstrating vascular browning and mycelial growth. (Courtesy of Tom Isakeit).

FUNGAL

GUMMY STEM BLIGHT



Seedling infection. (Courtesy of Dan Egel)



Foliar blight on melons.

CAUSAL AGENT:

Didymella bryoniae (anamorph: *Phoma cucurbitacearum*)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Didymella bryoniae is a foliar pathogen that can infect all cucurbit species. Young seedlings can quickly damp-off following infection. On older plants, leaf symptoms appear as circular, dark tan to black spots surrounded by a yellow halo. Over time these lesions dry, crack and fall out, which is often called “shot-holing.” Infection at the leaf margin begins as a wilt and progresses toward the center, resulting in leaf blight. Infected stems may develop cankers which produce a characteristic red or brown, gummy exudate. Severely infected stems may be girdled, resulting in vine death. Tiny black fruiting bodies (pycnidia or pseudothecia) may develop within the infected leaf or stem tissue. Fruit symptoms can range from small, water-soaked, oval or circular spots to large necrotic panels. Black fruiting bodies may develop within the lesions. Infection can lead to softening at the blossom end which may be brown or green. When the pedicel is infected, fruit abortion may occur.

CONDITIONS FOR DISEASE DEVELOPMENT:

Open Field: The fungus overwinters on infected cucurbit crop debris and cucurbit volunteers. This pathogen can also be seed-borne. Wounds caused by pruning, insects or field work can be important entry points for the fungus. The disease is most serious in open field production during periods of moderate temperatures and wet weather. The optimum temperature range for infection is 20–25°C (68–77°F).

Protected Culture: Cool night temperatures and high humidity favor disease development. Infection of open flowers can lead to serious fruit quality problems, making fruit unmarketable.

CONTROL:

Avoid overhead irrigation. Rotate out of cucurbits for a minimum of three years to non-susceptible hosts in order to break the disease cycle. Control weeds and remove wild cucurbits from potential field locations. Implement a preventative fungicide spray program. Use fungicide-treated seed. In addition to the above measures, soil sterilization and a strict sanitation program for equipment and personnel should be implemented in protected culture.



Necrotic *Didymella bryoniae* lesions coalescing on melon leaf.

FUNGAL GUMMY STEM BLIGHT



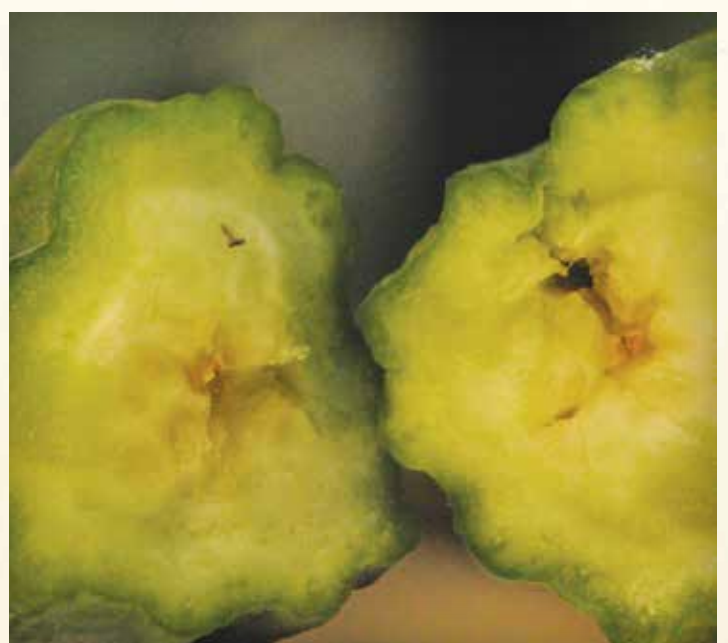
Brown, water-soaked stem section.



Fruiting bodies (pycnidia) forming near the crown area on melon. (Courtesy of Dan Egel)



Black rot of cucumber caused by *Didymella bryoniae*. (Courtesy of John R. Hartman)



Fruit infection on a greenhouse grown cucumber. (Courtesy of Gillian Ferguson)

MONOSPORASCUS ROOT ROT AND VINE DECLINE



Melon root system showing multiple lesions. (Courtesy of Gerald Holmes)

CAUSAL AGENT:

Monosporascus cannonballus

DISTRIBUTION:

Worldwide

SYMPTOMS:

Initial symptoms include stunting and poor growth of plants. However, this may go undetected if an entire field is uniformly affected. The older crown leaves begin to turn chlorotic, wilt and collapse within weeks of harvest. Within five to ten days of the first foliar symptoms, most of the canopy may be killed. Tan to reddish-brown lesions form on the roots. Root infection leads to a loss of feeder roots. Eventually the root system may become necrotic, resulting in plant death. Large, black perithecia form on dead roots and are often visible. Fruit of diseased plants are smaller or cracked and may abscise from the pedicle before ripening and have reduced sugar content. Fruit may also become sunburned due to lack of foliage. Stem lesions are generally lacking and above ground symptoms may be confused with other vine declines.

CONDITIONS FOR DISEASE DEVELOPMENT:

Infection by *Monosporascus cannonballus* is believed to occur early in the season; however, tissue colonization is encouraged as the soil temperature increases. This rise in the soil temperature encourages perithecia formation in the roots. Ascospores are the long-term survival structures of the fungus. Disease spread is by movement of infested soil or infected plant material.

CONTROL:

Management of *Monosporascus cannonballus* has proven to be difficult due to its heat tolerance and thick-walled resting structures. Avoid planting melons and watermelons in known infested fields. Also, avoid excessive irrigation, which may only delay plants collapse. Allowing infested roots to dry out in the field followed by fumigating soon after harvest has shown to be beneficial. The use of rootstocks has been beneficial in watermelon, although additional work is needed for melon. Chemigation through drip irrigation lines has also been shown to be effective.

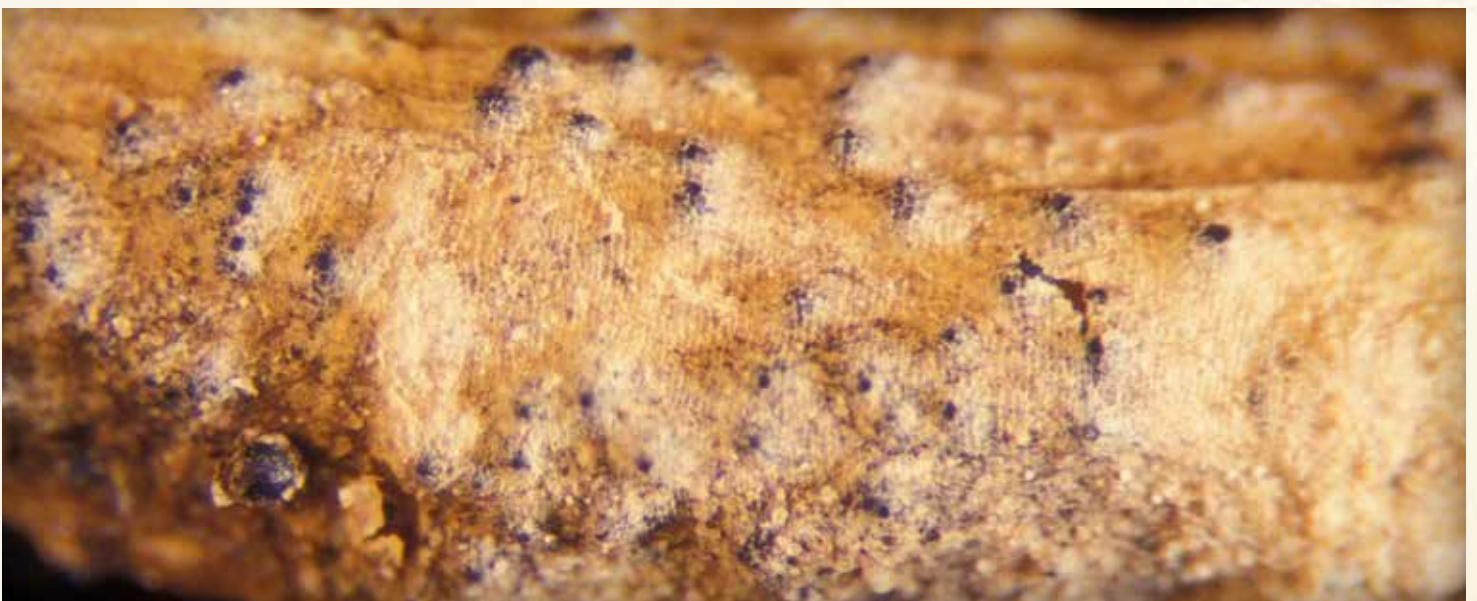


Plant collapse and death in the field due to *Monosporascus cannonballus*. (Courtesy of Gerald Holmes)

FUNGAL MONOSPORASCUS ROOT ROT AND VINE DECLINE



Perithecia on melon roots. (Courtesy of Gerald Holmes)



Perithecia embedded in melon root tissue. (Courtesy of Gerald Holmes)

FUNGAL

PHYTOPHTHORA CROWN AND ROOT ROT



Squash fruit infection.



Crown area of summer squash plant showing *Phytophthora capsici* infection.
(Courtesy of Gerald Holmes)

CAUSAL AGENTS:

Phytophthora capsici

Phytophthora spp.

DISTRIBUTION:

Worldwide

SYMPTOMS:

Phytophthora capsici and other *Phytophthora* spp. can cause a range of symptoms in cucurbits from damping-off in young seedlings to crown and root rot, leaf spots, foliar blights and pre- and post-harvest fruit rots in mature plants. Symptoms of Phytophthora crown and root rot often manifest quickly, with plant death occurring within a few days of symptom onset. Often, affected plants exhibit a sudden, permanent wilt. Stems can collapse while the foliage of wilted plants remains green. Water-soaking develops in the roots, crown and lower stem near the soil line. Tissues become soft, turning from a healthy white to tan-to dark brown. In advanced stages, the lateral roots slough off and eventually the entire root system may be destroyed.

CONDITIONS FOR DISEASE DEVELOPMENT:

These *Phytophthora* spp. survive from one season to the next in infected cucurbit tissue or in tissue of one of its other hosts (e.g., eggplant, pepper and tomato). This organism is able to disperse itself as zoospores that are released from sporangia into surface and irrigation water, and also as sporangia that are released into the air. Over-irrigation, heavy rainfall and poor drainage favor Phytophthora crown and root rot. High temperatures in mid- and late-season further stress already weakened plants and the disease can progress rapidly. Phytophthora crown and root rot incidence is greatest in low lying areas of fields where soil remains saturated for extended periods. Increasing the frequency and/or duration of irrigation will increase the incidence of this disease. Favorable conditions for this pathogen include soil temperatures above 18°C (65°F) and prolonged wet periods with air temperatures between 24–29°C (75–85°F).

CONTROL:

No single method is available to provide adequate control of Phytophthora crown and root rot. Cultural practices shown to reduce infection include irrigation management (e.g., drip irrigation) and high plant beds to improve drainage. When possible, avoid crop rotation with pepper and, to a lesser degree, other solanum species (e.g., eggplant, tomato). Additional practices to manage the disease include pathogen exclusion by following good sanitation and cultural practices. Fungicide sprays and soil drenches have also been shown to be effective.

FUNGAL PHYTOPHTHORA CROWN AND ROOT ROT



Watermelon fruit infection.



Pumpkin fruit infected with *Phytophthora capsici*. (Courtesy of Tom Isakeit)

FUNGAL

PLECTOSPORIUM BLIGHT



Foliar infection is confined to the veins. (Courtesy of Thomas A. Zitter)

CAUSAL AGENT:

Plectosporium tabacinum (synonym = *Microdochium tabacinum*)

DISTRIBUTION:

United States, Europe and Asia

SYMPTOMS:

Plectosporium tabacinum has a host range that includes cucurbits, peanut, snap bean, soybean and sunflower. It remains unclear whether these alternate hosts play a role in the epidemiology of *Plectosporium* blight of cucurbits, as it appears that some host specialization by isolates occurs. In the United States, pumpkin and squash are the most commonly affected cucurbit crops. *Plectosporium tabacinum* can infect all parts of the plant and often causes significant crop losses. Lesions on stems, leaf veins, petioles and peduncles are often sunken, spindle- or diamond-shaped and tan to white in appearance. Initially, stem lesions are small but can quickly enlarge and coalesce over the entire stem, turning it white. Leaf infections are confined to leaf veins and do not spread to interveinal leaf tissue. Infected petioles and peduncles can quickly turn dry and brittle, resulting in death of attached leaves or flowers. When a plant is severely infected, complete defoliation and plant death may occur. Infected fruit develop small, circular, tan to white raised lesions. Fruit lesions are usually constricted, but can expand to form a corky, necrotic panel. Fruit lesions often serve as entryways for secondary soft-rot organisms that cause various fruit rots.

CONDITIONS FOR DISEASE DEVELOPMENT:

The disease cycle of *Plectosporium tabacinum* is not well understood. Disease outbreaks have been associated with high humidity and temperatures between 25–32°C (75–90°F). The pathogen has been reported to survive in crop residue in the soil for up to three years. Conidia can be splash-dispersed by rain and overhead irrigation or they can be carried by wind.

CONTROL:

Rotate out of cucurbits for three years. Increase air circulation within fields by reducing plant density, orient fields with prevailing winds, and avoid locations prone to high humidity. Implementation of drip irrigation along with a preventative fungicide spray program can help minimize or prevent outbreaks of *Plectosporium* blight.



Diamond-shaped lesions on petiole. (Courtesy of Mary Ann Hansen)

FUNGAL PLECTOSPORIUM BLIGHT



Plectosporium blight of pumpkin crown and petiole tissues. Note that foliage is mostly unaffected. (Courtesy of Kenneth Seebold Jr.)



Peduncle and stem infection on pumpkin. (Courtesy of Thomas A. Zitter)

FUNGAL POWDERY MILDEW



Powdery mildew on melon. (Courtesy of Laixin Luo)



Powdery mildew on watermelon.

CAUSAL AGENTS:

Golovinomyces cichoracearum (synonym = *Erysiphe cichoracearum*)

Podosphaera xanthii (synonym = *Sphaerotheca fuliginea*)

DISTRIBUTION:

Worldwide

SYMPTOMS:

All cucurbits are susceptible to powdery mildew. Symptoms appear as pale yellow spots on stems, petioles, and leaves. Infection may occur on the upper and/or lower leaf surface. As the spots enlarge, conidia are produced from affected tissue and the spots take on a powdery appearance. Infected leaves gradually turn yellow and may become brown and papery. In general, cucurbit fruits are not directly attacked by powdery mildew fungi. However, due to the loss of plant foliage, fruit may be exposed to direct sunlight, resulting in sunburn and reduced fruit quality. Fruit infection, although rare, can occur on watermelon and cucumber.

CONDITIONS FOR DISEASE DEVELOPMENT:

The fungi that cause powdery mildew are obligate parasites, meaning that they require a living host to survive. These fungi commonly overwinter on weeds and are carried for long distances by air currents. Infection can take place without free moisture on the plant surface, although high humidity (50–90% RH) is necessary. Colonization, sporulation, and dispersal of conidia are favored by dry conditions. Disease development is favored by vigorous plant growth, moderate temperatures, low light and dew formation. Infection can occur between 10–32°C (50–90°F) with an optimum temperature for infection between 20–27°C (68–80°F).

CONTROL:

Grow resistant varieties when available. Implement a preventative fungicide spray program to delay infection and help lower disease incidence. In protected culture, avoid practices which promote lush growth. Weed control and good sanitation practices can also aid in controlling powdery mildew.



Powdery mildew on squash.

FUNGAL POWDERY MILDEW



Powdery mildew on pumpkin.



Powdery mildew on cucumber.



Powdery mildew infecting the crown of a squash plant. (Courtesy of Gerald Holmes)

FUNGAL SCAB



Cucumber fruit infection.

CAUSAL AGENT:

Cladosporium cucumerinum

DISTRIBUTION:

Worldwide

SYMPTOMS:

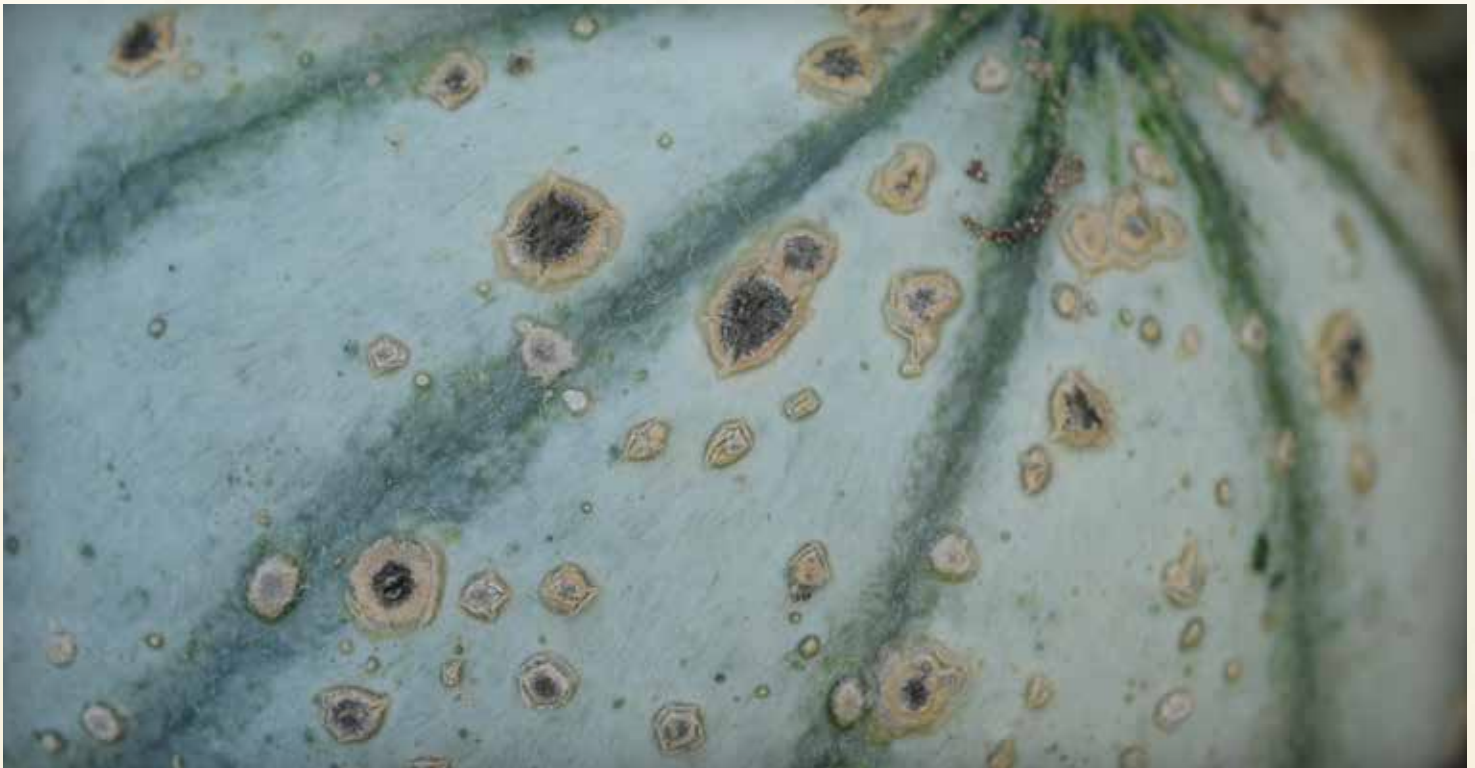
Scab is most common on cucumber, but can also affect cantaloupe, pumpkin and squash. All exposed portions of the plant and fruit can be infected. Leaf symptoms appear as circular to angular, brownish, water-soaked spots with yellow margins. Infected plants may have shortened internodes which can give the appearance of virus infection. A gray to olive-colored sporulation can develop on infected tissue. Infection appears on young fruit as water-soaked spots which develop into crater-like depressions as the fruit mature. The crater-like depressions develop an irregular, scab-like appearance as fruit age. Fruit lesions are commonly shallow and spongy. Often a gummy brown substance appears on the scabby surface. Under humid conditions, a mass of fungal spores (conidia) may develop on fruit.

CONDITIONS FOR DISEASE DEVELOPMENT:

Cladosporium cucumerinum survives in infected plant material. Conidia can be dispersed by wind, insects, farming equipment, and workers. The disease develops rapidly under cool [21–24°C (70–75°F)], moist conditions. Higher temperatures inhibit disease development.

CONTROL:

Grow scab-resistant cucumber varieties. Implement a preventative fungicide spray program. Control volunteers and cucurbit weed hosts, which can serve as sources of inoculum.

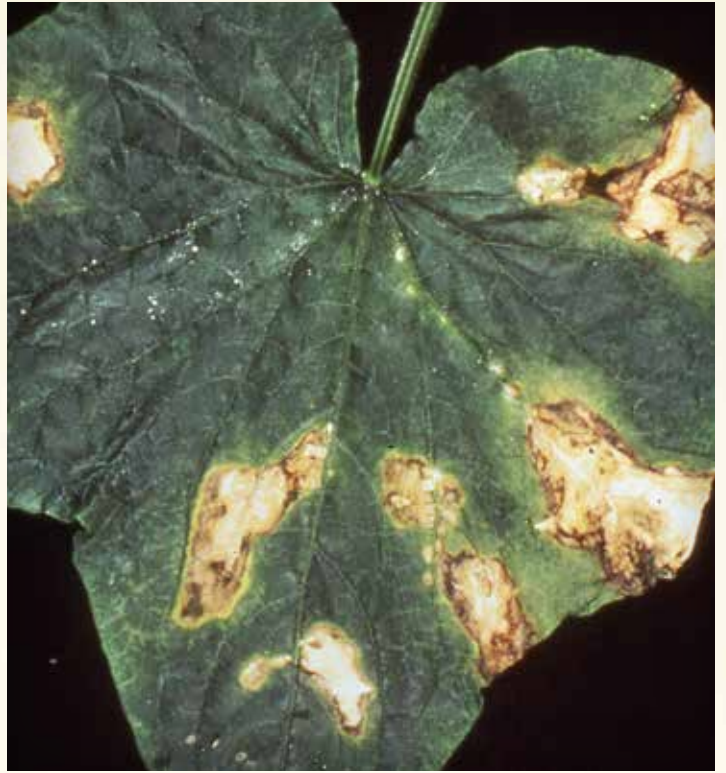


Melon fruit infection. (Courtesy of Dominique Blancard)

FUNGAL SCAB



Yellow squash infected with *Cladosporium cucumerinum*. (Courtesy of Clemson University - USDA Cooperative Extension Slide Series)



Foliar lesions of *Cladosporium cucumerinum* on a cucumber leaf.



A melon plant showing infection by *Cladosporium cucumerinum* on a runner and leaves. (Courtesy of Dominique Blancard)

FUNGAL

SCLEROTINIA STEM ROT



Sclerotinia sclerotiorum stem of squash.

CAUSAL AGENT:

Sclerotinia sclerotiorum

DISTRIBUTION:

Worldwide

SYMPTOMS:

All cucurbits are susceptible to sclerotinia stem rot. This fungus is able to infect stems at the soil level, as well as leaves and fruit above ground. The first sign of disease is a white, cottony growth on infected tissue. As the disease progresses, the plant gradually turns yellow and may die. When an infected stem is cut open, white mycelium may be seen in the pith along with large (6–12 mm) black sclerotia. Upon infection, fruit may be colonized by the white mycelium of *Sclerotinia sclerotiorum* and quickly become soft and watery.

CONDITIONS FOR DISEASE DEVELOPMENT:

The fungus can survive in soil for many years as sclerotia. These overwintering structures can infect plants by producing mycelium and forming apothecia, which release ascospores. The disease develops under cool to moderate temperatures and humid conditions. Prolonged periods of leaf wetness (12–24 hours) are optimal for disease development. The fungus has an extremely wide host range of over 500 species of plants.

CONTROL:

Implement good cultural practices such as a three- to five-year crop rotation with non-hosts (corn, wheat, and sorghum), sanitation and deep plowing after a crop to help reduce this disease. In addition, careful irrigation management may minimize disease occurrence. Soil application of a biological control agent (i.e., *Coniothyrium minitans*) has been shown to reduce populations of viable sclerotia in the top two centimeters of soil. Application of fungicides has been shown to help control *Sclerotinia* stem rot. Soil fumigation is usually an effective method of control in greenhouses.

FUNGAL SCLEROTINIA STEM ROT



Melon fruit infected with *Sclerotinia sclerotiorum*. (Courtesy of Dominique Blancard)



Black sclerotia of *Sclerotinia sclerotiorum*. (Courtesy of Dominique Blancard)

FUNGAL

SOUTHERN BLIGHT



Melon stem infected with *Sclerotium rolfsii*. Note the small spherical tan-to-brown sclerotia forming on and within the white mycelia. (Courtesy of Tom Isakeit)

CAUSAL AGENT:

Sclerotium rolfsii (teleomorph: *Athelia rolfsii*)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Southern blight is commonly observed on cantaloupe, squash and watermelon. The first symptom of the disease is a mid-day wilting of the plant. Leaves turn yellow and within a few days the plant collapses leading to plant death. The rapid collapse of the plant is due to girdling of the stem at the soil surface, and the entire root system is often completely rotted. The fungus develops a white mycelium, which may be fan shaped, over the surface of the stem. Light brown bodies (sclerotia) may be observed embedded within the white mycelium. These sclerotia turn dark brown with age. The fungus also infects fruit in contact with infested soil, developing sunken, yellow spots which decay and collapse. Large amounts of white mycelium and sclerotia can form on the fruit as it decays.

CONDITIONS FOR DISEASE DEVELOPMENT:

Sclerotia, which are the overwintering structure, allow *Sclerotium rolfsii* to survive for many years in the soil. Sclerotia are spread by movement of soil or by surface water. The disease is favored by high temperatures [27–32°C (80–90°F)] and high soil moisture.

CONTROL:

A good sanitation program is generally the most effective control measure. Remove and burn infected plants to prevent inoculum build-up. Deep plow crop residue to reduce inoculum levels. Rotate to non-host crops (corn and small grains) for a period of three to five years. Good water management can help reduce soil moisture, which in turn reduces the amount of germinating sclerotia in the soil. Fumigation can also provide control. Some fungicides have proved effective in controlling this disease.

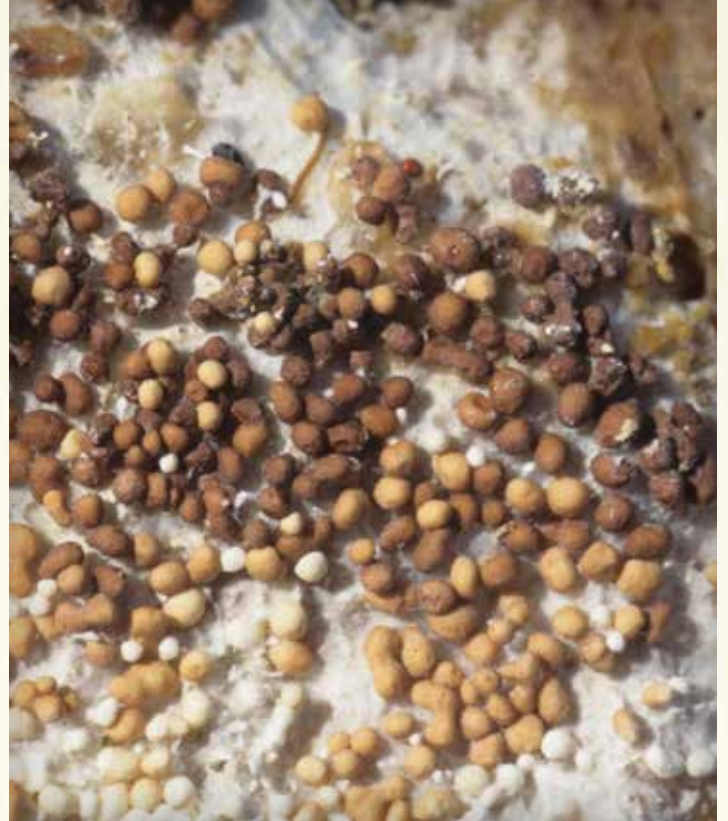


Crown of a melon plant infected by *Sclerotium rolfsii*. (Courtesy of Jason Brock)

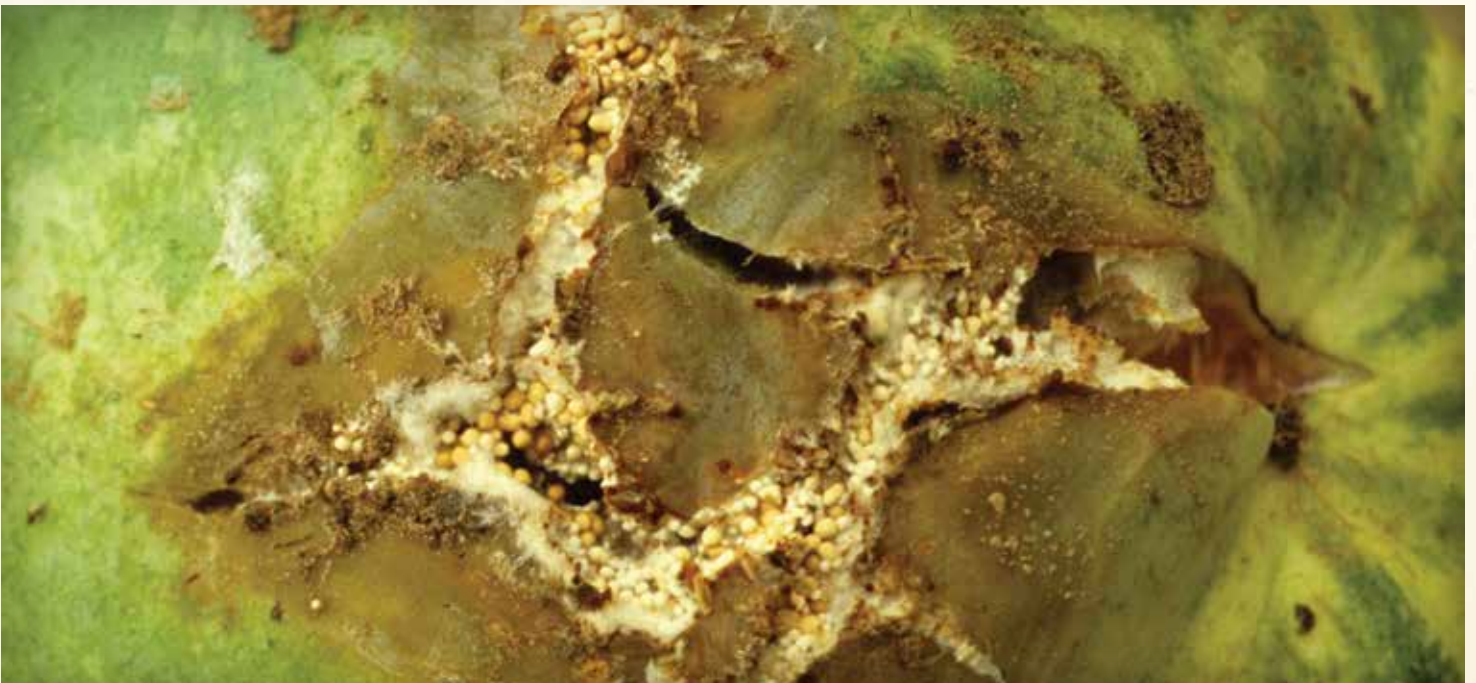
FUNGAL SOUTHERN BLIGHT



Mycelia and sclerotia forming on and beneath a melon in contact with soil.
(Courtesy of Tom Isakeit)



Light brown (younger) and dark brown (older) sclerotia on pumpkin fruit infected with *Sclerotium rolfsii*. (Courtesy of Gerald Holmes)



Watermelon fruit infection. (Courtesy of Tom Isakeit)

FUNGAL TARGET LEAF SPOT



Leaf symptoms on cucumber. Note the angular appearance lesions, which may closely resemble other cucumber foliar diseases.

CAUSAL AGENT:

Corynespora cassiicola

DISTRIBUTION:

Worldwide

SYMPTOMS:

The disease can be found on all cucurbits, although it is most common on cucumber. The first symptoms appear on older leaves as angular, yellow spots. In the open field, these spots enlarge and become circular with light brown centers and dark brown borders. Later these large spots become gray and drop out, giving a shot-hole or shredded appearance to the leaf. Under greenhouse conditions spots have light centers with rings of olive green tissue and yellow borders. Eventually, defoliation can occur. Spots on stems and petioles are more elongated, which helps to distinguish this disease from other diseases, which include anthracnose, downy mildew or angular leaf spot. Early infection at the blossom end results in darkened, shriveled fruit. Root and flower infection can also occur.

CONDITIONS FOR DISEASE DEVELOPMENT:

The fungus can exist on infected plant residue for at least two years, or on weed hosts. It is dispersed by air currents. Warm temperatures [25–35°C (77–95°F)] and long days are best for disease development, although infection takes place under humid and moderately cool temperatures [21–26°C (70–80°F)]. Also, fluctuating daily temperatures appear to favor disease development.

CONTROL:

Use resistant varieties when available. Implement a preventative fungicide spray program. In protected culture, good sanitation practices in and around structures will help avoid future infestations.



Lesions on cucumber leaf with tan-to-light brown centers and dark brown borders. (Courtesy of Dominique Blancard)

FUNGAL TARGET LEAF SPOT



Target leaf spot lesions on the underside of a cucumber leaf.



Discrete foliar lesions on cucumber leaves growing under protected culture.



Foliar lesions can rapidly coalesce under severe disease pressure resulting in complete necrosis and plant death.

FUNGAL

VERTICILLIUM WILT



Verticillium wilt symptoms on a young melon plant.



Wilting of an entire melon plant.

CAUSAL AGENTS:

Verticillium dahliae

Verticillium albo-atrum

DISTRIBUTION:

Worldwide

SYMPTOMS:

This disease affects all cucurbits. In general, symptom expression occurs at or following fruit set. Crown leaves initially wilt and take on a faded green color. As symptoms progress, leaf margins develop "V"-shaped chlorotic lesions, which eventually collapse and turn necrotic. Wilting may progress along runners, which can result in plant death. Brown discoloration of root and stem tissues at the crown of the plant is visible in longitudinal section. Symptoms may be confused with other vascular wilt pathogens (e.g., *Fusarium*).

CONDITIONS FOR DISEASE DEVELOPMENT:

These fungi have a broad host range and can survive in soil as microsclerotia for many years. Infection takes place through the roots and disease development is favored by cool soil temperatures [21–24°C (70–75°F)]. However, wilting is generally observed during warm, dry periods when plants are under stress (e.g., fruit set).

CONTROL:

Soil fumigation and solarization have been the only methods shown to effectively reduce the incidence of Verticillium wilt. Avoid infested fields. Implement good cultural practices, including proper disposal of plant debris, deep plowing and a minimum of a three-year crop rotation with non-susceptible hosts (e.g., monocots). Avoid following highly susceptible crops (e.g., cotton, potato or tomato) with cucurbits or other verticillium wilt-susceptible crops. When possible, delay planting until the soil is warm. In protected culture, grafting onto a resistant rootstock may also help provide control.

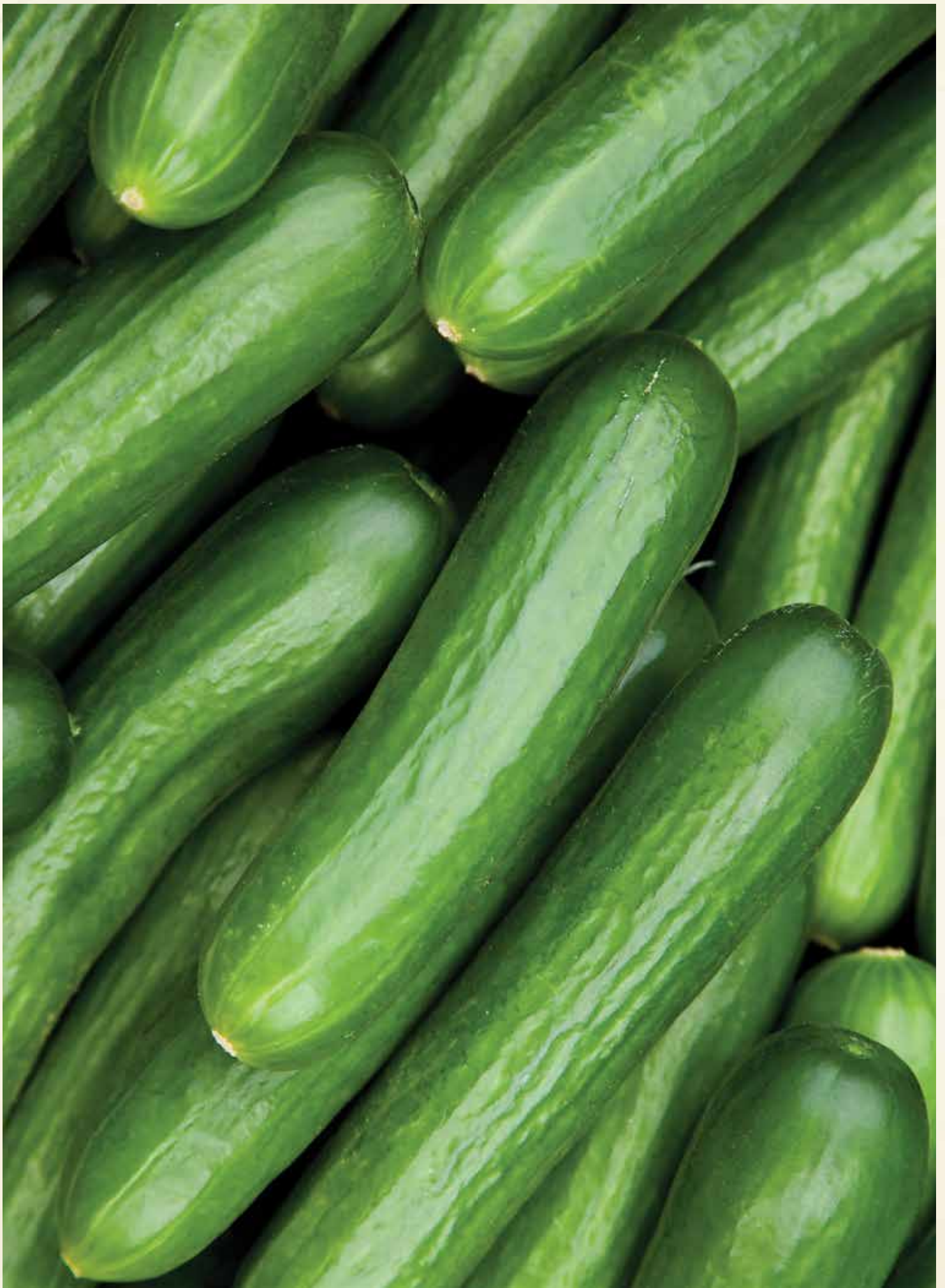
FUNGAL VERTICILLIUM WILT



Foliar symptoms and discoloration of root vascular tissue.



Light brown discoloration of vascular tissue.





VIRAL

DISEASES

BEET PSEUDO-YELLOWS
CUCUMBER MOSAIC
CUCUMBER VEIN-YELLOWING
CUCURBIT APHID-BORNE YELLOWS
CUCURBIT YELLOW STUNTING DISORDER
GEMINIVIRUSES
MELON NECROTIC SPOT
POTYVIRUSES
SQUASH MOSAIC
SQUASH VEIN-YELLOWING
TOBAMOVIRUSES
TOSPOVIRUSES

VIRAL

BEET PSEUDO-YELLOWS



Greenhouse cucumber infected with *Beet pseudo-yellows virus*.

CAUSAL AGENT:

Beet pseudo-yellows virus (BPYV)

VECTOR:

Greenhouse whitefly (*Trialeurodes vaporariorum*)

DISTRIBUTION:

Australia, France, Italy, Japan, The Netherlands, Spain and USA

SYMPTOMS:

Beet pseudo-yellows is an important disease on greenhouse cucumber and muskmelon (cantaloupe). This virus was previously known as cucumber yellows or muskmelon yellows. Symptoms first appear on older leaves as yellow spots which develop into yellow blotchy raised areas between veins, while the veins themselves remain green. These raised areas eventually coalesce to form large thickened areas, which become brittle and may disintegrate. As the disease progresses, younger leaves begin to develop symptoms, but fruit remain unaffected. Plants infected at an early stage can be stunted and may have fewer fruit. Symptoms caused by beet pseudo-yellows can easily be confused with symptoms resulting from nutritional deficiencies (e.g., magnesium), insect feeding, poor growing conditions and premature aging.

CONDITIONS FOR DISEASE DEVELOPMENT:

The greenhouse whitefly can acquire and transmit Beet pseudo-yellows virus in a semi-persistent manner. Symptoms begin to develop two to four weeks after infection. The virus is not seed-borne or mechanically transmitted. High light intensity appears to be necessary for disease development. This virus has a large host range among crops and weed species. In addition to cucumber, muskmelon (cantaloupe) and squash, BPYV also infects many ornamentals and other vegetable crops such as lettuce, endive, carrot, spinach and beet.

CONTROL:

Prevent whiteflies from entering protected culture facilities by screening openings with insect-proof netting (minimum 50–52 mesh/297 micron screen). Implement a comprehensive insecticide program, crop rotation and a host-free period. Eliminate intercropping of young and old plants to reduce inoculum levels. Remove weeds and volunteer plants in and around greenhouses. Dispose of plant debris immediately after harvest to eliminate inoculum sources.

VIRAL

BEET PSEUDO-YELLOWS



Leaf symptoms include yellow raised areas between the veins with the veins remaining green.



Open field melon infected with *Beet pseudo-yellows virus*.

VIRAL

CUCUMBER MOSAIC



Cucumber mosaic virus leaf symptoms on cucumber. Note virus symptoms progress up the plant.

CAUSAL AGENT:

Cucumber mosaic virus (CMV)

VECTOR:

Several aphid species

DISTRIBUTION:

Worldwide

SYMPTOMS:

All cucurbits are susceptible to CMV, but watermelon is rarely affected. Symptoms may vary based on host, environment and age of the plant at time of infection. Symptoms first appear on younger leaves, which curl downward and become mottled, distorted and reduced in size. Plants may become stunted with shortened internodes, resulting in a rosette-like appearance of the youngest leaves. If infection occurs after flowering, vine growth may not be reduced, but fruit may be mottled and distorted.

Cucumber: Seedlings seldom show symptoms during the first few weeks of growth, but symptoms can appear once growth becomes vigorous. Leaves become mottled and distorted with downward-curling edges. All subsequent growth is reduced, leaving the plants dwarfed. Older leaves may develop chlorotic margins, which later become necrotic. Fruit are often misshapen, mottled (yellowish-green), warty and reduced in size. Infected fruit may appear bleached due to a lack of chlorophyll production.

Melon: Melon plants may exhibit severely stunted growing tips. Even if fruit does not express distinct symptoms, overall fruit quality is often poor.

Pumpkin: Early infection often results in severe foliar mosaic. Fruit may be rendered unmarketable due to mosaic symptoms.

Squash: Early-season infection can result in severely stunted plants with deformed leaves. Petioles often exhibit a downward or bending growth pattern. Leaves may also



Leaf deformation and mosaic in squash infected with *Cucumber mosaic virus*.

VIRAL

CUCUMBER MOSAIC

be reduced in size. Fruit can become unmarketable due to pronounced rugosity of the fruit surface. In summer squash, warty raised yellow areas appear on the fruit, and are surrounded by dark green areas.

Watermelon: Foliar symptoms are often mild when compared to the other cucurbit hosts. Mild leaf crinkling with some yellowing may be observed.

CONDITIONS FOR DISEASE DEVELOPMENT:

CMV can infect both greenhouse and field-grown vegetable crops. CMV has an extensive host range (>1200 species) facilitating its survival on weeds, ornamentals, and other cultivated crops. The primary mode of transmission is by aphids in a non-persistent manner, although the virus can also be mechanically transmitted through equipment and workers. Cucumber beetles (*Diabrotica* spp.) have also been shown to transmit CMV, but aphids are the primary vector.

CONTROL:

Management of CMV through vector control (e.g., insecticides, stylet oils) has been only marginally successful. Avoid planting near older cucurbits and perennial ornamental crops, which may serve as reservoirs for the virus. Control weeds, use reflective mulches, deep plow crop residues and dispose of infected greenhouse material to manage this virus. Implement a comprehensive sanitation program for workers and equipment to minimize disease spread. Commercial varieties with resistance offer the best means to control CMV. Resistance in cucumber has been shown to be very effective on a global scale. Progress in finding resistance in other cucurbit species has not been as successful. In yellow summer squash, the presence of the precocious yellow gene has worked well against CMV infection. A few transgenic-resistant squash cultivars are grown in the USA.



Squash plant infected with *Cucumber mosaic virus*.



Infected cucumber leaves and fruit.



Grey zucchini infected with *Cucumber mosaic virus*.

VIRAL

CUCUMBER VEIN-YELLOWING



Cucumber vein-yellowing virus on cucumber.

CAUSAL AGENT:

Cucumber vein-yellowing virus (CVYV)

VECTOR:

Silverleaf whitefly (*Bemisia tabaci*)

DISTRIBUTION:

Iran, Israel, Jordan, Spain, Sudan, Turkey

SYMPTOMS:

Melon and cucumber exhibit vein yellowing, vein clearing, chlorosis, necrosis and stunting with a corresponding reduction in yield. Parthenocarpic cucumbers exhibit severe symptoms, while non-parthenocarpic cucumbers have been reported to be asymptomatic carriers of the virus. Cucumber fruit symptoms appear as a light to dark green mosaic. In watermelon, foliar symptoms are inconspicuous or not expressed, however fruit cracking has been observed. Squash may be asymptomatic or may exhibit mild vein yellowing and chlorotic mottling of leaves.

CONDITIONS FOR DISEASE DEVELOPMENT:

CVYV is transmitted in a semi-persistent manner (<six hours) by the silverleaf whitefly, *Bemisia tabaci*. Movement of infected transplants can spread the virus over long distances. Whiteflies can easily spread the virus from plant to plant. CVYV is not considered to be seed-borne nor seed-transmitted. The virus survives in cucurbit weeds and volunteers, jimsonweed, *Nicotiana* spp., sowthistle, bindweed and *Malva* spp.

CONTROL:

Planting resistant cultivars is the best means of control. Currently, resistance is only commercially available in cucumber. Implement insect exclusion (minimum 50–52 mesh/297 micron screen) to minimize whitefly infestation in greenhouse-grown crops. Seedlings should be grown in a whitefly-free environment. Use insecticides to control the vector. Rotate modes of action to prevent development of insecticide-resistant whitefly populations.



Cucumber vein-yellowing virus on melon.

VIRAL CUCUMBER VEIN-YELLOWING



Cucumber vein-yellowing virus infection in cucumber.



Irregular shaped watermelon due to *Cucumber vein-yellowing virus* infection.
(Courtesy of Moshe Lapidot)

VIRAL

CUCURBIT APHID-BORNE YELLOWS



Cucurbit aphid-borne yellows virus on pumpkin. (Courtesy of Bryce Falk)



Pumpkin leaves infected with *Cucurbit aphid-borne yellows virus*. (Courtesy of Bryce Falk)

CAUSAL AGENT:

Cucurbit aphid-borne yellows virus (CABYV)

VECTOR:

Several aphid species

DISTRIBUTION:

Worldwide

SYMPTOMS:

Early symptoms manifest as chlorotic spots on the lower leaves, progressing to interveinal chlorosis. Leaves become chlorotic, leathery and brittle, while the mid-vein and primary veins remain green. Stunting and flower abortion reduce marketable yield; however, for fruit that develop, fruit shape and quality are not affected. Prior to the development of specific detection methods, symptoms of CABYV were often attributed to nutrient deficiencies, senescence, or diseases, such as lettuce infectious yellows, cucumber yellows or cucurbit yellow stunting disorder, all of which cause similar symptoms.

CONDITIONS FOR DISEASE DEVELOPMENT:

This virus is acquired by phloem-feeding aphid vectors in a persistent manner. The cotton melon aphid, one of the vectors of CABYV, is very efficient at transmitting the virus. Cucurbits are the primary hosts of CABYV. Alternate hosts include crops such as lettuce (*Lactuca sativa*) and fodder beet (*Beta vulgaris*). Weeds are also recognized reservoir hosts of CABYV.

CONTROL:

Implement an insecticide spray program to control the aphid vectors. In open-field production, use of silver reflective plastic mulches can help to repel aphids. In protected culture, insect exclusion (minimum 50–52 mesh / 297 micron screens) can provide some control. Melons with aphid resistance usually significantly delay CABYV infection.

VIRAL CUCURBIT APHID-BORNE YELLOWS



Leaf yellowing due to *Cucurbit aphid-borne yellows virus*. (Right photo courtesy of Bill Wintermantel)



Pumpkin fruit infected with *Cucurbit aphid-borne yellows virus*.

VIRAL

CUCURBIT YELLOW STUNTING DISORDER



Symptoms initiate on older leaves an interveinal mottle.



Advanced symptoms of *Cucumber yellow stunting disorder virus* on cucumber.

CAUSAL AGENT:

Cucurbit yellow stunting disorder virus (CYSDV)

VECTOR:

Silverleaf whitefly (*Bemisia tabaci* biotypes B and Q)

DISTRIBUTION:

Worldwide

SYMPTOMS:

CYSDV was previously thought to be restricted to the Cucurbitaceae family, but it is now recognized that CYSDV also infects crop and weed species such as alfalfa, lettuce, snap bean, alkali mallow and Wright groundcherry. Symptoms initiate as interveinal mottling on older leaves, intensify with age and become systemic throughout the plant. Veins remain relatively green as the rest of the leaf turns yellow. Leaves may roll upward and become brittle. Melon and cucumber exhibit the most severe symptoms, which can be confused with nutrient deficiency or other yellowing viruses. Melon fruit does not express obvious symptoms, although sugars can be reduced dramatically.

CONDITIONS FOR DISEASE DEVELOPMENT:

CYSDV is transmitted by the silverleaf whitefly vector, *Bemisia tabaci* biotypes B and Q, which can be transported long distances via air currents. Outbreaks are often associated with high infestations of *Bemisia tabaci*. The virus is not mechanically transmitted and is not seed-borne or seed-transmitted. *Bemisia tabaci* needs to feed for at least 18 to 24 hours to transmit the virus and can remain infective for up to eight days.

CONTROL:

Insect exclusion (minimum 50–52 mesh/297 micron screens) and a preventative insecticide spray program in transplant nurseries can help to minimize whitefly infestations. Yellow adhesive traps are useful for monitoring the presence of *Bemisia tabaci*. Control weeds to eliminate potential sources of inoculum. In open-field production, early season exclusion of the vector using mesh tunnels may delay virus infection. Application of insecticides for whitefly control is not an effective method to manage virus spread in the field. Commercial varieties with CYSDV resistance are available in cucumber, but are not yet available in other cucurbits.

VIRAL

CUCURBIT YELLOW STUNTING DISORDER



Melon leaf symptoms of *Cucurbit yellow stunting disorder virus* manifest as mottling, turning to green spots (left) and advance to severe interveinal chlorosis (right). (Right photo courtesy of Judy Brown)



Melon fields infected with *Cucurbit yellow stunting disorder virus*. (Right photo courtesy of Moshe Lapidot)

VIRAL GEMINIVIRUSES



Cucurbit leaf crumple virus symptoms on melon. (Courtesy of Bob Gilbertson)



Cucurbit leaf crumple virus symptoms on watermelon. (Courtesy of Bob Gilbertson)



Foliar stunting and shortening of internodes in melon caused by *Melon chlorotic leaf curl virus*. (Courtesy of Judy Brown)

CAUSAL AGENTS:

<i>Cucurbit leaf crumple virus</i>	CuLCrV	Mexico, USA (Arizona, California, Florida, Texas)
<i>Loofa yellow mosaic virus</i>	LYMV	Vietnam
<i>Melon chlorotic leaf curl virus</i>	MCLCuV	Guatemala
<i>Pumpkin yellow vein mosaic virus</i>	PYVMV	India
<i>Squash leaf curl virus</i>	SLCV	Central America, Egypt, Mexico, Middle East, USA (Arizona, California, Florida, Texas)
<i>Squash mild leaf curl virus</i>	SMLCV	Central America, Egypt, Mexico, Middle East, USA (Arizona, California, Florida, Texas)
<i>Squash leaf curl China virus</i>	SLCCNV	China, Philippines, Vietnam
<i>Squash leaf curl Yunnan virus</i>	SLCYNV	Southern China
<i>Tomato leaf curl virus</i>	ToLCV	India, Thailand
<i>Watermelon chlorotic stunt virus</i>	WmCSV	Middle East, Sudan
<i>Watermelon curly mottle virus</i>	WCMV	India

DISTRIBUTION:

VECTOR:

Silverleaf whitefly (*Bemisia tabaci* biotypes A, B, Q)

SYMPTOMS:

Geminiviruses infect cucurbits with varying degrees of severity. Cucumber appears to be least affected by geminiviruses. Symptoms of geminivirus infection may include upward curling of leaf margins, foliar stunting, chlorosis, interveinal mottling, vein clearing and thick, distorted veins. Flowers of infected plants are small and fail to develop normally. Early-season infection results in lack of fruit set, whereas fruit set prior to infection may be reduced in size, and exhibit chlorotic blotches and deformation.



Scarring of melon fruit caused by *Melon chlorotic leaf curl virus*. (Courtesy of Judy Brown)

VIRAL GEMINIVIRUSES

CONDITIONS FOR DISEASE DEVELOPMENT:

Geminiviruses are vectored by the silverleaf whitefly, *Bemisia tabaci* (biotypes A, B, Q). The adult whitefly acquires the virus from infected plants and can transmit it to healthy plants within a few hours. Symptoms can develop within five days of virus transmission. Symptoms on cucurbits are most severe when whitefly populations are high and the crop is infected early in the season.

CONTROL:

Host-free periods have been shown to be an effective measure for controlling the whitefly vector, whereas insecticide spray programs have been largely ineffective. Cultural control methods include weed control, incorporation of infected crop debris immediately after harvest and avoidance of planting new fields near infected cucurbit fields. Plant resistance is limited in commercial cultivars.



Squash leaf curl virus aggressive strain infecting squash.



Squash leaf curl virus symptoms in greenhouse-grown squash.



Squash leaf curl virus causing extreme stunting at the apical meristem of squash.



Squash leaf curl virus-infected melon plant. (Courtesy of Moshe Lapidot)



Squash leaf curl virus symptoms on melon. (Courtesy of Moshe Lapidot)

VIRAL GEMINIVIRUSES



Tomato leaf curl virus foliar symptoms on cucumber.



Tomato leaf curl virus foliar symptoms on cucumber.



Tomato leaf curl virus foliar symptoms on cucumber.

VIRAL GEMINIVIRUSES



Symptoms of *Watermelon chlorotic stunt virus* on a young watermelon plant. (Courtesy of Moshe Lapidot)



Symptoms of *Watermelon chlorotic stunt virus* leaf symptoms on watermelon. (Courtesy of Moshe Lapidot)

VIRAL

MELON NECROTIC SPOT

CAUSAL AGENT:

Melon necrotic spot virus (MNSV)

VECTOR:

Ovipodium bornovanus

DISTRIBUTION:

Worldwide

SYMPTOMS:

This virus has a narrow host range, affecting only melon, cucumber and watermelon. Symptoms initially appear on younger leaves as chlorotic spots, which soon become necrotic. In some cultivars, necrotic lesions and streaks develop on petioles and stems, indicating systemic infection. Leaves may curl and wilt, occasionally leading to complete plant collapse. Symptom intensity may vary considerably depending on cultivar. In watermelon, MNSV manifests as local lesions only.

CONDITIONS FOR DISEASE DEVELOPMENT:

MNSV is transmitted by zoospores of the obligate fungal parasite, *Ovipodium bornovanus*. MNSV has been shown to be seed-transmitted at low rates. This virus can also be mechanically transmitted by pruning, workers and equipment. Symptoms develop mainly under cool, low light conditions. In summer, infected plants may show no symptoms.

CONTROL:

Grow resistant cultivars, incorporate plant debris, fumigate soil and rotate out of cucurbits to manage this virus. Solarization has also been effective in favorable environments. Avoid excessive irrigation to minimize spread of the fungal vector. Surfactants added to irrigation systems may reduce zoospore numbers, thus minimizing spread of the vector in soil-less media. Grafting watermelon onto MNSV-resistant rootstocks is an effective cultural practice to control MNSV.



Early onset of *Melon necrotic spot virus* in a melon leaf.

VIRAL MELON NECROTIC SPOT



Symptoms of *Melon necrotic spot virus* infection may develop from the base of the leaf (above) to the leaf margin (below).



VIRAL POTYVIRUSES



Moroccan watermelon mosaic virus symptoms in squash.



Papaya ringspot virus on squash.



Papaya ringspot virus fruit infection.

CAUSAL AGENTS:

Moroccan watermelon mosaic virus (MWMV)
Papaya ringspot virus (PRSV; formerly *Watermelon mosaic virus-1*)
Watermelon mosaic virus (WMV; formerly *Watermelon mosaic virus-2*)
Zucchini yellow mosaic virus (ZYMV)

VECTOR:

Aphis spp.

DISTRIBUTION:

PRSV, WMV, ZYMV – Worldwide
MWMV – Africa (Morocco, South Africa), France, Italy, Spain, and Portugal

SYMPTOMS:

All cucurbits are susceptible to potyviruses. Due to the similar nature of symptoms caused by the various potyviruses, it is recommended that samples be submitted to a diagnostic laboratory for proper identification. A characteristic symptom common to all cucurbit polyviruses is a narrowing of tendril-like appearance of leaves, referred to as “shoe-string.”

PRSV: Symptoms can initially appear as vein-clearing of leaves. As symptoms progress, a light to dark green mosaic develops, followed by distortion and deep leaf serration. In cucumber, leaves tend to be distorted along the margins. In melon, severe infection can cause blistering of young leaves. In squash, severely affected leaves can assume a “shoe-string” appearance. In watermelon, the growing terminals tend to stand erect and new leaves are reduced in size. Early-season infection may lead to poor fruit set while late-season infection may result in deformed, blotchy fruit. Concentric ring-spot patterns may develop on the rind of watermelon fruit.

WMV/MWMV: Symptoms manifest as chlorosis of leaf veins. As the disease progresses, leaves may develop a green mosaic and become deformed and blistered. In severe cases, leaf tissue surrounding the major veins develops a “shoe-string” appearance. Early plant infection often leads to severely distorted, discolored fruit. When virus infection occurs after fruit set, generally fruit development is normal. MWMV causes very severe mosaic and deformation of leaves and fruits in cucumber,

VIRAL POTYVIRUSES

squash and watermelon. In many melon cultivars, systemic infection manifests as necrotic leaf spots, which is often followed by complete plant collapse. MWMV is almost exclusively restricted to cucurbits, whereas WMV has the widest host range among the potyviruses.

ZYMV: Infected leaves are yellow with severe mosaic symptoms and also may exhibit blistering and “shoe-string.” Early-season infection can cause stunting of plants, uneven fruit color and fruit malformation.

CONDITIONS FOR DISEASE DEVELOPMENT:

All potyviruses are vectored in a non-persistent manner by several species of aphid. These viruses can also be mechanically transmitted by workers and equipment to a lesser extent. The host range for some of these viruses includes legumes and weeds, however, infected weeds may remain asymptomatic.

CONTROL:

Grow resistant cultivars, control aphids and weeds and avoid planting near older cucurbit fields. Reflective mulches, equipment and worker sanitation, deep plowing of crop debris and destruction of cull piles may also help control these diseases.



Watermelon mosaic virus symptoms in melon.



Watermelon mosaic virus symptoms in cucumber.



Watermelon mosaic virus symptoms in squash.



Watermelon mosaic virus infection in squash. (Courtesy of Anthony Keinath)

VIRAL POTYVIRUSES



Zucchini yellow mosaic virus symptoms in cucumber.



Zucchini yellow mosaic virus symptoms in watermelon. (Courtesy of Kai-Shu Ling)

VIRAL POTYVIRUSES



Zucchini yellow mosaic symptoms in squash.



Zucchini yellow mosaic virus infecting cucumber fruit.

VIRAL

SQUASH MOSAIC



Early symptoms on melon may result in the slight recessing of the leaf margins exposing veins giving a fringe-like appearance.



Squash mosaic virus on melon.

CAUSAL AGENT:

Squash mosaic virus (SqMV)

DISTRIBUTION:

Worldwide

VECTOR:

Striped cucumber beetle (*Acalymma* spp.)

Spotted cucumber beetle (*Diabrotica* spp.)

SYMPTOMS:

This disease is most important on cantaloupe, pumpkin and squash; however, some strains of the virus infect watermelon. Reports on commercial cucumbers have been limited, but infections are sporadically reported in breeding programs.

Melon and Squash: Green vein banding of the first or second leaf may develop in seedlings grown from squash mosaic-infected seed. Young leaves may be symptomless or may exhibit yellow spots, vein clearing and/or blistering. Leaves can be severely distorted, with marginal projections from the veins giving a fringe-like appearance to the leaf margin. Infected plants are stunted with fewer branches and fruit. Fruit may develop mild mottling to severe deformation. Netted melon types infected with SqMV may not form netting.

Cucumber: Leaves may exhibit chlorotic spots with an upward leaf curl, systemic vein-clearing or a yellow vein-banding, which may become necrotic. As the crop ages, additional symptoms on the new foliage may not be apparent and symptom expression decreases as temperatures increase, making visual identification difficult.

Watermelon: Necrotic local lesions may develop, but SqMV is generally not of economic importance on watermelon.

CONDITIONS FOR DISEASE DEVELOPMENT:

Infected seed is often the initial source of SqMV infection. The striped cucumber beetle and the spotted cucumber beetle are the primary vectors, can acquire the virus after just five minutes of feeding, and transmit the virus for 5–20 days. The virus can also be mechanically transmitted by workers and equipment. Grasshoppers may also transmit SqMV, although they are not a major vector of the virus.

CONTROL:

Use virus-free seed or transplants, control the vectors, remove cucurbit volunteers and weeds, implement sound sanitation practices and remove or deep-plow infected crop debris.

VIRAL SQUASH MOSAIC



Squash mosaic virus leaf symptoms in squash.



Green vein-banding leaf symptom on melon leaves.



Squash mosaic virus symptoms on a yellow crookneck squash.



The spotted cucumber beetle is a vector of *Squash mosaic virus*. (Courtesy of Jim Janski)



A vector of *Squash mosaic virus*, the striped cucumber beetle. (Courtesy of Whitney Crenshaw)

VIRAL

SQUASH VEIN-YELLOWING



Squash leaf exhibiting characteristic vein yellowing. (Courtesy of Scott Adkins)

CAUSAL AGENT:

Squash vein-yellowing virus (SqVYV)

DISTRIBUTION:

United States (Florida, Indiana), and Puerto Rico

VECTOR:

Silverleaf whitefly (*Bemisia tabaci*)

SYMPTOMS:

SqVYV symptoms manifest as vein-yellowing in squash and vine decline in watermelon. Watermelon symptoms appear initially as chlorotic leaves, followed by browning and collapse of the entire vine within a few weeks. Symptoms develop more rapidly as fruit matures. Affected fruit may exhibit internal rind and flesh discoloration.

CONDITIONS FOR DISEASE DEVELOPMENT:

SqVYV is transmitted in a semi-persistent manner by the silverleaf whitefly (*Bemisia tabaci*), which can transmit the virus for twenty-four hours. Host range is limited to the cucurbit family. The cucurbit weeds balsam-apple and creeping cucumber can harbor the virus asymptotically.

CONTROL:

Avoid planting near SqVYV-infected cucurbit fields, eliminate wild cucurbit weeds and immediately incorporate infected crop debris following harvest. Implement a comprehensive insecticide program, crop rotation and a host-free period. Silver plastic mulches are effective in controlling other whitefly-transmitted viruses and may help to control SqVYV.



Watermelon fruit may develop internal rind and flesh discoloration from *Squash vein-yellowing virus* infection. (Courtesy of Scott Adkins)

VIRAL

SQUASH VEIN-YELLOWING



Watermelon field exhibiting “vine decline” caused by *Squash vein-yellowing virus*. (Courtesy of Scott Adkins)

VIRAL TOBAMOVIRUSES



Cucumber green mottle mosaic virus symptoms in cucumber.



Cucumber green mosaic virus symptoms in melon. (Courtesy of Bryce Falk)

CAUSAL AGENTS:

Cucumber green mottle mosaic virus (CGMMV)

Kyuri green mottle mosaic virus (KGMMV)

Zucchini green mottle mosaic virus (ZGMMV)

VECTOR:

Mechanically transmitted with no known insect vectors.

DISTRIBUTION:

CGMMV: Australia, Canada, China, Europe, India, Iran, Israel, Japan, Korea, Lebanon, Myanmar, Pakistan, Saudi Arabia, Sri Lanka, Russia, Syria, Taiwan, Turkey, and USA (California)

ZGMMV and **KGMMV:** Korea

SYMPTOMS:

CGMMV affects bottle gourd, cucumber, squash, melon and watermelon and is particularly severe in protected culture. Early symptoms manifest as vein-clearing and crumpling of young leaves. Mature leaves may become bleached. Symptoms vary and include mild to severe leaf distortion, light and dark green mottling, yellow or silver leaf flecks and stunting. Symptoms on fruit may include spots or streaks, which may be chlorotic or silver, and fruit distortion. Symptoms may be especially severe on cucumber.

CONDITIONS FOR DISEASE DEVELOPMENT:

CGMMV is a seed-transmitted virus. Other sources of infection include contaminated crop debris and infected rootstocks. CGMMV is mechanically transmitted via equipment and workers. No insect vectors are known. Symptom expression in plants is most severe under cool, low-light conditions. High temperatures tend to enhance fruit symptom expression.

CONTROL:

Use tobamovirus-free seed and rootstocks, rogue infected plants and implement good sanitation practices to manage these diseases.



Watermelon fruit infected with Cucumber green mottle mosaic virus. Note cavities inside fruit flesh. (Courtesy of Laixin Luo)

VIRAL TOBAMOVIRUSES



Cucumber green mottle mosaic virus symptoms in watermelon.



Cucumber green mottle mosaic virus symptoms on bottle gourd.



*Cucumber fruits infected with *Cucumber green mottle mosaic virus*.*

VIRAL TOSPOVIRUSES

CAUSAL AGENTS:

<i>Groundnut bud necrosis virus</i>	GBNV	Asia, USA (Florida)
<i>Melon severe mosaic virus</i>	MeSMV	Mexico
<i>Melon yellow spot virus</i>	MYSV	Asia
<i>Tomato spotted wilt virus</i>	TSWV	Worldwide
<i>Watermelon bud necrosis virus</i>	WBNV	Asia, USA (Florida)
<i>Watermelon silver mottle virus</i>	WSMV	Asia
<i>Zucchini lethal chlorosis virus</i>	ZLCV	Brazil

DISTRIBUTION:

CONDITIONS FOR DISEASE DEVELOPMENT:

Tospoviruses are transmitted from plant to plant by several species of thrips. As with many insect-transmitted viruses, the thrips-tospovirus relationship is very specific. Only a few of the many known thrips species are able to acquire and transmit tospoviruses. Thrips can only transmit TSWV if it is acquired during the larval stage. Once the virus has been acquired, both larval and adult thrips are able to transmit the virus. Tospoviruses are not seed-borne nor seed-transmitted. The host range for many of the cucurbit tospoviruses is not completely understood. TSWV can infect over 800 plant species in more than 80 plant families. Weeds and ornamentals can serve as reservoirs for these viruses and play a role in their spread and over-wintering.

VECTOR:

Several thrips species

SYMPTOMS:

Cucurbits infected with tospovirus exhibit a variety of symptoms. Leaves can show bronzing to severe systemic chlorotic spotting. Other symptoms include leaf deformation, mosaic, die-back and overall plant stunting. Fruit symptoms can range from chlorotic ring spots on young fruit to necrotic lesions on older fruit. Fruit cracking may also be observed.

CONTROL:

In protected culture and transplant nurseries, utilize mesh screening (minimum 72 mesh /192 microns) and phytosanitary measures to minimize tospovirus infection by excluding thrips. Biological control agents have also been successfully implemented in protected culture. Avoid planting near or downwind of ornamentals or older crops, which can serve as reservoirs for thrips and tospovirus. Control weeds and cucurbit volunteers. Implement a comprehensive insecticide program beginning prior to sowing or transplanting to manage thrips early larval stages and limit secondary tospovirus spread. Rotate insecticide modes of action to discourage development of insecticide-resistant thrips populations. Tospovirus resistance is currently not available in commercial cucurbit varieties.



Mosaic and leaf blistering in melon by *Melon severe mosaic virus*. (Courtesy of Bill Copes)



Leaf deformation in melon caused by *Melon severe mosaic virus*. (Courtesy of Bill Copes)



Fruit splitting of honeydew melon caused by *Melon severe mosaic virus*. (Courtesy of Bill Copes)

VIRAL TOSPOVIRUSES



Melon yellow spot virus symptoms in cucumber.



Melon yellow spot virus fruit infection.



Flower bud necrosis caused by *Watermelon bud necrosis virus*.



Flower bud necrosis and leaf chlorosis caused by *Watermelon bud necrosis virus*.
(Courtesy of Rakesh Kumar)



Watermelon fruit symptoms caused by *Watermelon bud necrosis virus*. (Courtesy of Rakesh Kumar)





NEMATODE

DISEASES

ROOT-KNOT NEMATODES
ADDITIONAL NEMATODES

NEMATODE ROOT-KNOT



Galling of summer squash root system. (Courtesy of Kenneth Seebold, Jr.)



Severe root-knot nematode galling on melon. (Courtesy of Gerald Holmes)

CAUSAL AGENT:

Meloidogyne spp.

DISTRIBUTION:

Worldwide

SYMPTOMS:

All cucurbits are susceptible. Affected plants appear stunted with poor growth. Foliage takes on a pale green to yellow appearance. Infected plants wilt during the hottest periods of the day due to reduced water uptake. Although the crop may maintain a healthy appearance throughout the growing season, yield and quality of the fruit can be greatly reduced. In heavy infections, plants will completely wilt and die as the nematode populations increase. When infected plants are removed from the soil, knobby, wart-like galls caused by the nematode can be seen singly or in clumps on the roots. Secondary infection by other soil organisms is common.

CONDITIONS FOR DISEASE DEVELOPMENT:

The disease is most severe in light, sandy soils with an optimal soil temperature of 27°C (80°F). Nematodes can survive in the soil for several years, but weed-free fallow periods can significantly reduce populations. Dispersal of nematodes may occur through contaminated irrigation water, movement of infested soil and infected plant material.

CONTROL:

Soil fumigation and weed-free fallow periods are usually the best management strategies to help control root-knot nematode. Proper identification of the nematode species and population levels are important for choosing the correct management method. Grafting onto resistant cucurbit rootstocks have been proven to be effective.



Root-knot nematode-infested cucumber field. (Courtesy of Charles Averre)

NEMATODE

ADDITIONAL NEMATODES

CAUSAL AGENTS:

Belonolaimus spp. (Sting Nematode)

Pratylenchus spp. (Root Lesion or Meadow Nematode)

Rotylenchulus spp. (Reniform nematode)

Trichodorus spp. (Stubby Root Nematode)

Paratylenchus spp. (Pin Nematode)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Sting Nematode: Infested fields often exhibit small, circular to irregularly shaped loci of stunted plants. Plants within the center of these loci begin to die as the diseased area expands. Symptoms first appear on the older leaves and foliage dies from the leaf margin inward. Young roots become brown, whereas older roots develop longitudinal brown streaks. This nematode has a wide host range and survives indefinitely on crabgrass.

Reniform Nematode: Above-ground symptoms on host plants include stunting, shedding of leaves, malformed fruit and seeds, and an impaired root system. Roots can be discolored and necrotic with areas of decay. Plant death can occur under heavy infestations.

Root Lesion Nematode: Root infections do not usually cause economic damage. However, lesions that develop on roots can often be invaded by soil-borne fungal and/or bacterial pathogens. Asymptomatic plants can serve as hosts, allowing nematodes to reproduce and build up populations.

Stubby Root and Pin Nematodes: Symptoms caused by these two nematodes include: reduced numbers of feeder roots, restricted root growth and plants which are stunted and yellow in appearance. Infected plants are seldom killed.

CONDITIONS FOR DISEASE DEVELOPMENT:

Moderate losses occur in moist sandy soils and at warm temperatures. These nematodes survive on weed hosts.

CONTROL:

Proper identification of the nematode species and population level is important for determining effective management strategies. Implement cultural practices which promote moisture and nutrient availability throughout crop cycle. Nematicides and weed-free fallow periods have been shown to lower nematode populations. Use of a non-host can aid in reducing nematode populations.



Cucumber field infected by *Rotylenchulus* spp. (Courtesy of Tom Isakeit)



Cucumber plant infected by *Rotylenchulus* spp. (Courtesy of Tom Isakeit)





PARASITIC PLANT

DODDER

PARASITIC PLANT DODDER



Cucumber plant heavily infested with dodder *Cuscuta* spp.



Dodder infesting melon petioles.

CAUSAL AGENT:

Cuscuta spp.

DISTRIBUTION:

Worldwide

SYMPTOMS:

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

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Dodder has a very wide host range and is adapted to a wide range of environments. After germination, the seedling depends on nutrients stored for its survival. If a suitable host is not found within a few days, it will die. Once a seedling makes contact with a host, it forms sucker-like projections (haustoria) that penetrate plant tissues. Dodder produces small, inconspicuous flowers (often white) that mature and produce two to four yellow to black seeds.

Irrigation water and cultivation equipment are common modes of long-distance dispersal. Dodder seeds are small and can remain viable in the soil for up to ten years. Seeds usually germinate in late winter and spring in cold climates, however, germination can continue through the summer. Environmental conditions that favor the growth of cucurbits are also beneficial to dodder.

CONTROL:

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



ABIOTIC DISORDERS

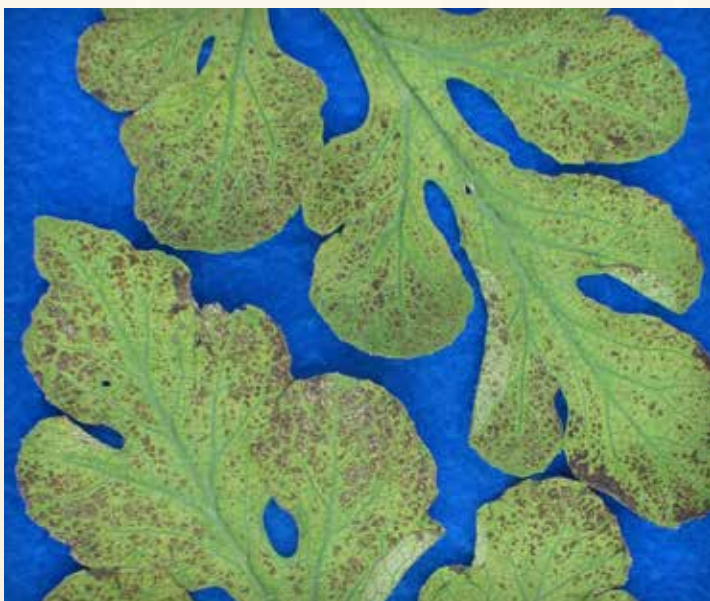
AIR POLLUTION INJURY
ENVIRONMENTAL STRESSES
NUTRIENT DEFICIENCIES
PESTICIDE INJURY
PHYSIOLOGICAL FRUIT DISORDERS
POOR POLLINATION
SALT INJURY
SQUASH SILVER LEAF
WIND AND SAND INJURY

ABIOTIC DISORDERS

AIR POLLUTION INJURY



Ozone damage on watermelon eventually develops a white to silver appearance on the upper or adaxial surface of the leaves. The abaxial side of the leaf is unaffected. (Courtesy of David Langston)



Early signs of ozone damage on watermelon leaves. (Courtesy of Margaret T McGrath)

CAUSAL AGENTS:

Ozone, sulfur dioxide, and other pollutants

DISTRIBUTION:

Worldwide

SYMPTOMS:

Symptoms vary depending on the pollutant causing injury and the host. Air pollution damage can reduce yield and affect fruit quality.

Ozone: Sensitivity to ozone among cucurbits varies. Watermelon and squash are most sensitive, pumpkin and cantaloupe are intermediate in sensitivity and cucumber is more tolerant to ozone injury compared to other cucurbits. Damage appears on the upper surface of older leaves, which develop a chlorotic, netted appearance due to loss of chlorophyll between the veins. Later, these chlorotic areas turn brown.

Sulfur Dioxide: Plants exposed to chronic, sub-lethal levels of sulfur dioxide may develop chlorosis of the margins and interveinal areas of the leaves. These chlorotic areas generally remain turgid. In cases of acute injury, the margins and interveinal areas become necrotic. New fully expanded leaves are more sensitive to acute injury than leaves that are not yet fully expanded.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Ozone is produced by the action of sunlight on the products of combustion. Most ozone is generated over large urban areas from automobile exhaust. Ozone damage may occur many miles from the original source of pollution. Ozone is absorbed passively by plants through stomata.

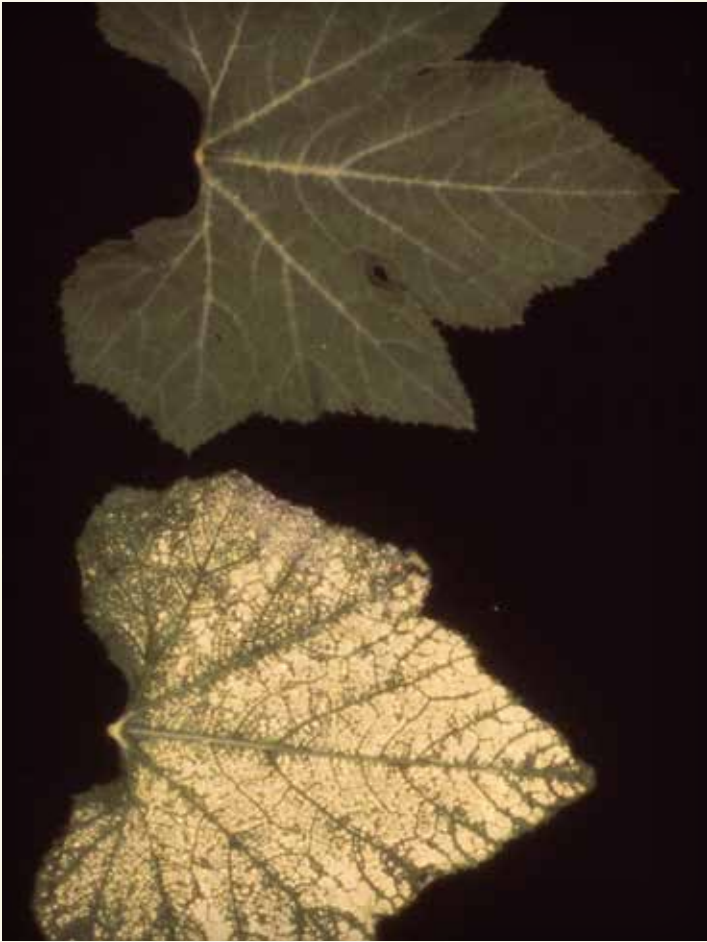
Sulfur dioxide is formed during smelting processes, when sulfuric acid is produced or during combustion of coal or oil. Likelihood of sulfur dioxide damage is greatest when temperature and humidity are high.



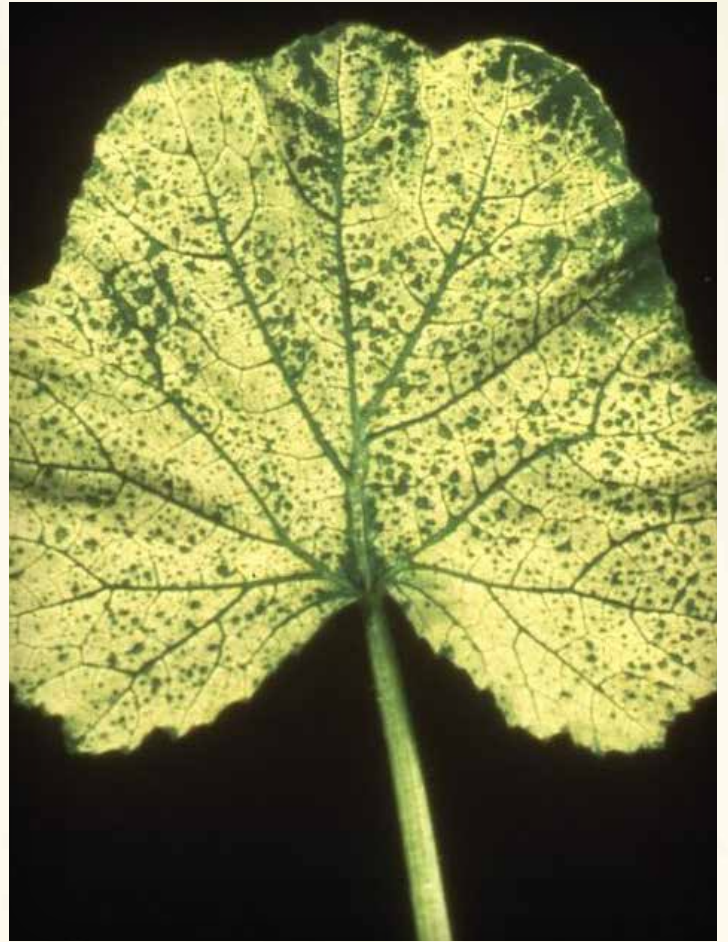
Sulfur dioxide injury on squash.

ABIOTIC DISORDERS

AIR POLLUTION INJURY



Sulfur dioxide injury on squash.



Sulfur dioxide injury on squash.



Winter squash leaves with necrotic spotting due to ozone damage. (Courtesy of Margaret T. McGrath)

ABIOTIC DISORDERS

ENVIRONMENTAL STRESSES



Cucumber cotyledons exposed to low temperatures.

CAUSAL AGENTS:

Temperature and moisture extremes

DISTRIBUTION:

Worldwide

SYMPTOMS:

Low temperatures [10–17°C (50–64°F)] can cause stunting and short, misshapen fruit in all cucurbits. Watermelon and cantaloupe are particularly sensitive to low temperatures. High temperatures may cause cucurbits to wilt temporarily and persistently high temperatures may cause marginal leaf necrosis. Drought can cause wilting or stunting of plants, and misshapen fruit. Excessive soil moisture may cause anaerobic conditions in the root zone, resulting in root injury, poor nutrient uptake by roots, leaf chlorosis, reduced plant growth and wilting.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Excessive soil moisture is often problematic in poorly drained fields, low lying areas or soils with high clay content. Drought injury is more common in light, sandy soils of low moisture-retention capacity.

CONTROL:

Grade fields to eliminate low lying areas. Deep plow and incorporate organic matter to improve drainage in clay soils. Incorporating organic matter also helps to improve moisture retention in light, sandy soils. Monitor crop water use to schedule irrigations effectively. Ensure that protected culture structures are equipped or designed to ensure the best airflow to prevent excessive temperatures or moisture.



Cucumber plant exhibiting frost injury.

ABIOTIC DISORDERS

ENVIRONMENTAL STRESSES



Honeydew melon with chilling injury: external symptoms. (Courtesy of Gerald Holmes)



Honeydew melon with chilling injury: internal symptoms. (Courtesy of Gerald Holmes)



Cucumber fruit with cold injury.

ABIOTIC DISORDERS

NUTRIENT DEFICIENCIES



Cucumber with iron (Fe) deficiency.



Cucumber with calcium (Ca) deficiency.



Cucumber with potassium (K) deficiency.

CAUSAL AGENT:

Lack of major or minor nutrient elements

DISTRIBUTION:

Worldwide

SYMPTOMS:

Nitrogen: The growth rate of nitrogen-deficient cucurbits is reduced and a general yellowing of the plant occurs, beginning at the oldest leaves. Cotyledons and older leaves die and younger leaves stop growing. Cucumber fruit are more slender and pinched at the blossom end. Cantaloupe fruit are small, light-colored and thin-skinned with small seeds.

Phosphorus: Deficient plants grow slowly, and internodes are shortened and stunted. Typically leaves show a purplish color. Phosphorous-deficient cucurbits produce poor quality flowers, and fruit- and seed-set are reduced.

Potassium: Young leaves on potassium-deficient plants are small and dull-appearing and assume a cupped appearance. Leaves develop peripheral chlorosis, which eventually becomes interveinal. Cucumber fruit are often narrow at the stem end, giving fruit a club-shaped appearance. Cantaloupe fruit develop gritty flesh and a bitter taste.

Magnesium: Near the end of the growing season, older leaves develop interveinal chlorosis, which initially appears at leaf margins and progresses inward. Eventually entire leaves become necrotic. Because magnesium deficiency develops late in the growing season, fruit yield is generally not reduced significantly.

Iron: Young leaves develop interveinal chlorosis while older leaves remain green. This is due to iron's lack of mobility in plants.

Calcium: Growth is retarded and internodes are shortened. Leaf margins stop expanding and leaves cup downward. New root growth is impaired. Blossom end rot of the fruit may occur.

Manganese: Interveinal areas of leaves become chlorotic. Deficiencies can be induced by over-liming.

Boron: Leaves become chlorotic and then necrotic, with death to the growing point. Fruit quality is decreased.

Molybdenum: Symptoms similar to nitrogen deficiency. Plants are stunted. Leaves develop marginal and interveinal chlorosis, which may lead to a scorched appearance.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Highly acid or alkaline soils often lead to major and minor element deficiencies. Excessive or unbalanced use of fertilizers may also result in some micronutrients becoming unavailable to the plant.

CONTROL:

Use a balanced fertilizer program appropriate to the soil and to the crop. Nutrient sprays can help correct many of the minor element deficiencies. Altering soil pH can often eliminate deficiency or toxicity problems.

ABIOTIC DISORDERS

NUTRIENT DEFICIENCIES



Melon with manganese (Mn) deficiency.



Melon with magnesium (Mg) deficiency. (Courtesy of Terry Jones)



Melon with molybdenum (Mo) deficiency.



Melon with molybdenum (Mo) deficiency. (Courtesy of Terry Jones)



Greenhouse-grown cucumber with Copper (Cu), Manganese (Mn) and Zinc (Zn) deficiencies.

ABIOTIC DISORDERS

PESTICIDE INJURY

CAUSAL AGENT:

2, 4-D, Atrazine, Bensulfuron-methyl, Carfentrazone-ethyl, Chlorothalonil, Clomazone, Flumioxazin, Glyphosate, Halsulfuron-methyl, MCPA, Metribuzin, Norflurazon, Oxyfluoren, Pelargonic Acid, Propanil, Sulfur, Treflan, and Triclopyr

DISTRIBUTION:

Worldwide

SYMPTOMS:

2, 4-D: Leaves become distorted and may curl downward. Stems and petioles may become flattened. Leaves often become fan-shaped and the major veins radiate from the leaf base.

Atrazine: Leaf tissue dies and plants may be stunted.

Bensulfuron-methyl: Plants develop severe stunting with reduced growth at the growing points.

Carfentrazone-ethyl: Young, expanding leaf tissue shows necrotic burn.

Chlorothalonil: Multiple applications can cause phytotoxicity on cucumber.

Clomazone: Leaves and growing tips in watermelon show a bleached appearance.

Flumioxazin: Squash leaves show yellow spotting to complete yellowing.

Glyphosate: Injury appears as a strong yellowing of newly emerged leaves and a yellowing of the center/base of older leaves. Upward curling of leaves and severe stunting may occur.

Halsulfuron-methyl: Post-emergent injury can cause yellowing and crinkling of foliage.

MCPA: Fruit malformation and leaf distortion can occur. Double fruit are common.

Metribuzin: Foliar symptoms range from bronzing to bleached white necrosis of leaves.

Norflurazon: Foliage shows intense yellow vein banding. Symptoms occur more often when crops are grown in sandy soils.

Oxyfluoren: Cotyledons develop necrotic spots. Initial true leaves appear burned on squash.

Paraquat: This herbicide can create necrotic spots of relatively uniform color. The interface between the affected and healthy area of the leaf is usually distinct.

Pelargonic Acid: Damage on squash can cause leaf size reduction as well as distortion of the leaves.

Propanil: Melon expresses a necrotic burn on cotyledons and interveinal necrotic panels on leaves.

Sulfur: Symptoms include foliar burn and stunting of the plant.

Treflan: The lower portion of the stem enlarges and root growth is reduced. The plant is weak and stunted, which may lead to plant collapse.

Triclopyr: Petioles and stems in melon and squash become twisted or cracked.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Cantaloupe and squash are very sensitive to many pesticides. Sulfur, MCPA, and 2,4-D may drift from applied areas and affect unsprayed sensitive crops. Atrazine damage occurs when a sensitive cucurbit crop follows the previous season's grain crop. Treflan residues from previous crops or improper application of Treflan for current crop can lead to damage.

CONTROL:

Apply pesticide(s) in accordance with label instructions. Be aware of potential pesticide residues when planning rotations. Avoid spraying on windy days. Avoid pesticide applications when plants are under water stress.



Melon with 2,4-D injury. (Courtesy of Tom Isakeit)



Squash with bensulfuron-methyl injury. (Courtesy of Tom Lanini)



Cucumber exhibiting chlorothalonil injury. (Courtesy of Gerald Holmes)

ABIOTIC DISORDERS

PESTICIDE INJURY



Watermelon with clomazone injury. (Courtesy of Howard Harrision)



Squash with norflurazon injury. (Courtesy of Gerald Holmes)



Squash exhibiting flumioxazin injury. (Courtesy of Tom Lanini)



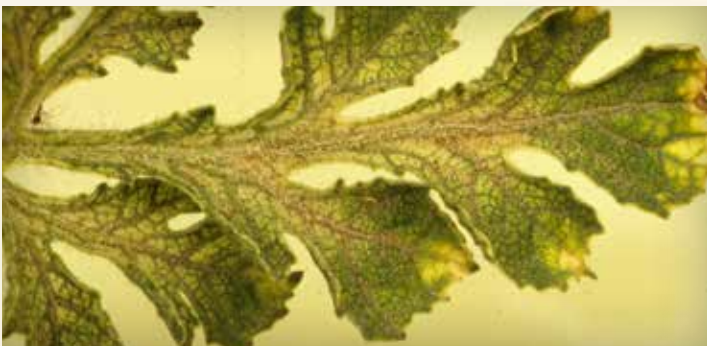
Watermelon leaves with paraquat injury. (Courtesy of Gerald Holmes)



Squash exhibiting glyphosate injury. (Courtesy of Shawn D. Askew)



Squash with pelargonic acid injury. (Courtesy of Margaret T. McGrath)



Squash with halsulfuron-methyl injury. (Courtesy of Timothy Coolong)



Melon leaf with sulfur (S) damage.

ABIOTIC DISORDERS

PHYSIOLOGICAL FRUIT DISORDERS

CAUSAL AGENT:

Adverse environmental conditions

DISTRIBUTION:

Worldwide

SYMPTOMS:

Blossom-End Rot: The blossom end of the fruit develops a dark, leathery appearance. Symptoms may progress until the entire end of the fruit turns black and rots.

Hollow Heart: Cracks in internal watermelon fruit flesh can occur due to accelerated growth in response to ideal growing conditions.

Light Belly Color: This disorder is characterized by the undersurface of cucumber fruit remaining light in color instead of turning dark green.

Measles: Symptoms are most evident on smooth-skinned melons and cucumbers. Small brown spots are scattered over the surface of the fruit. The spots are superficial and do not penetrate beyond the outer epidermal layers of the fruit. These spots also may occur on leaves and stems.

Rind Necrosis: Generally occurs in either cantaloupe or watermelon as dead, hard, dry reddish-brown to brown spots or patches of tissue in the fruit rind. Affected areas vary in size from 3mm (1/8") spots to extensive dead areas throughout the entire rind. In watermelon, symptoms are not visible from the outside and are rarely found in the flesh. In cantaloupe, dead tissue may extend into the flesh of the fruit. Circular, water-soaked depressions also develop on the cantaloupe fruit surface.

Sunscald: Papery white areas develop on fruit.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Blossom-End Rot: This disorder is associated with insufficient calcium uptake and alternating periods of wet and dry soil. Damage to the root system may also account for decreased calcium uptake and the development of blossom-end rot.

Hollow Heart: There is a genetic component to this disorder, but growing conditions can account for much of the variation observed. It appears to be associated with conditions that result in poor pollination (enough pollination to set the fruit but not enough to fertilize a high percentage of the ovules), followed by rapid fruit growing conditions (too much fertility, water and high temperatures).

Light Belly Color: Commonly occurs on fruit lying on cool, moist soil.

Measles: Associated with environmental conditions favoring guttation. The guttation droplets develop high concentrations of salts which burn the epidermis. Measles spots occur where a guttation droplet had formed.

Rind Necrosis: Not well understood. However, it is thought that environmental conditions which place stress on the plants may trigger the onset of this disorder. Susceptibility to rind necrosis varies among varieties. The disorder occurs sporadically and is thought to be associated with bacteria that may be present in fruit, but the reasons for symptom development are not understood. Drought stress also is reported to predispose melons.

Sunscald: Develops during hot summer weather when fruit are suddenly exposed to direct sunlight.

CONTROL:

Blossom-End Rot: Minimized by mulching to maintain constant soil moisture, applying calcium fertilizers and avoiding high levels of nitrogen. Drip irrigate crop to control water management.

Hollow Heart: Avoid watermelon varieties with a tendency to exhibit hollow heart. Implement best practices for irrigation and fertilization programs.

Light Belly Color: Can be partially controlled by avoiding luxuriant vine growth. Avoid excessive nitrogen.

Measles: Control measles by reducing irrigation frequency and duration as fruit approach maturity in fall-harvested crops. Irrigation reduction at the later stages of fruit development has not shown any adverse effects on fruit size and soluble solid content.

Rind Necrosis: Genetic tolerance has been identified in watermelon. Avoid drought stress in melon.

Sunscald: Minimize by maintaining strong vine growth to ensure the fruit is covered.



Watermelon blossom-end rot. (Courtesy of Gerald Holmes)

ABIOTIC DISORDERS

PHYSIOLOGICAL FRUIT DISORDERS



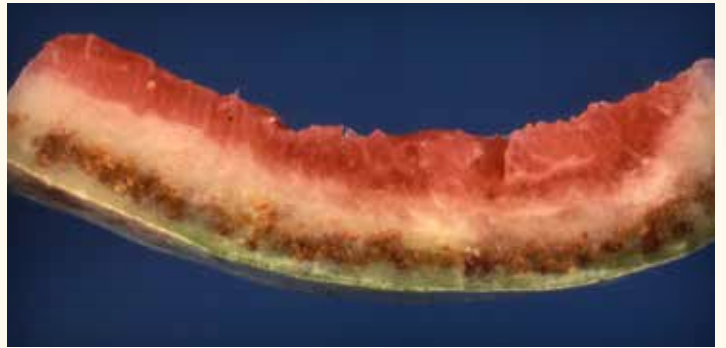
Squash blossom-end rot. (Courtesy of Gerald Holmes)



Rind necrosis on a triploid watermelon. (Courtesy of Brenda Lanini)



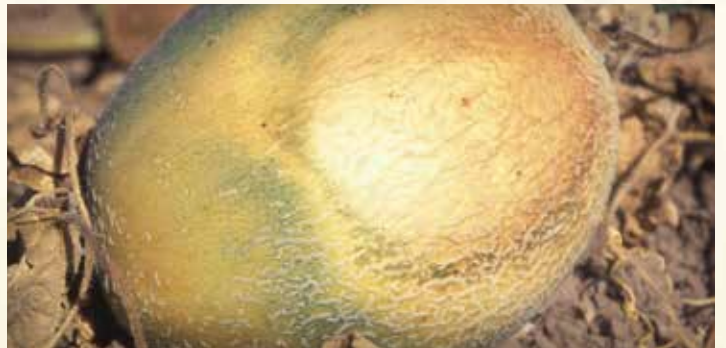
Watermelon with hollow heart.



Rind necrosis on watermelon.



Cucumber with light belly.



Melon sunscald. (Courtesy of Gerald Holmes)



Honeydew melon with measles.



Pumpkin sunscald. (Courtesy of Gerald Holmes)

ABIOTIC DISORDERS

POOR POLLINATION



Aborted squash fruit due to poor pollination.



Deformed cucumber fruit due to inadequate pollination.

CAUSAL AGENT

Insufficient or inactive pollen

DISTRIBUTION

Worldwide

SYMPTOMS

Immature fruit may turn brown starting from the blossom end, shrivel and abort. If fruit do not abort and continue to develop, they are often misshapen and/or stunted, with poor seed production.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Cucurbits require insect vectors (e.g., honeybees) for pollination. If the bee population is either low or inactive, there will not be sufficient pollen transferred from male to female flowers. Rain, hot or cold temperatures and disease can limit bee activity. Cucurbits are not successfully pollinated in cool, cloudy weather. Extremes in weather conditions are unfavorable to pollen viability.

CONTROL

Follow planting guidelines for your region. To ensure proper pollination, bee hives should be placed in or near cucurbit fields. Avoid excessive nitrogen fertilization in order to promote blossom formation and minimize vegetative growth. Avoid or minimize the use of pesticides during the pollination period.

In protected culture production, parthenocarpic varieties (i.e., cucumber and zucchini,) are best suited for these growing conditions, as fruits are set without pollination.



Insufficient pollination of a watermelon fruit.

ABIOTIC DISORDERS

SALT INJURY



Marginal yellowing of melon leaves caused by salt toxicity.

CAUSAL AGENT

Excessive soluble salts

DISTRIBUTION

Worldwide

SYMPTOMS

Cucurbits are moderately sensitive to salinity. Excess salts may damage roots resulting in stunted plants with reduced yields. Affected plants often appear darker green than normal in the early stages of salt injury. Due to salt accumulation, leaf margins eventually become white and or yellow, and then necrotic. Crops with salt injury grown in artificial substrate often tend to be more susceptible to wilting during the hottest hours of the day, even when the moisture level is sufficient.

CONDITIONS FOR SYMPTOM DEVELOPMENT

Many agricultural soils in arid climates are high in soluble salts. Additionally, irrigation water often contains excess salts. During irrigation, salts may not leach sufficiently from the root zone, resulting in salt accumulation. This problem is more severe in soils that drain poorly.

CONTROL

Measure the electrical conductivity (EC) of the soil, growing substrate and irrigation water to determine salt content. Avoid excessive fertilization. Where soil has good drainage, it may be possible to reclaim saline soil by applying sufficient water to leach salts beyond the root zone. For greenhouse-grown crops in substrate, irrigate in excess of container capacity to prevent salt buildup.



Burning of cucumber leaf margins due to salt toxicity.



Greenhouse cucumber exhibiting severe salt toxicity.

ABIOTIC DISORDERS

SQUASH SILVER LEAF



Silver leaf symptoms on a squash plant. (Courtesy of Timothy Coolong)

CAUSAL AGENT:

Silver leaf whitefly (*Bemisia tabaci* biotype B)

DISTRIBUTION:

Worldwide

SYMPTOMS:

Leaf silvering symptoms have been observed on all types of squash and are common in many zucchini varieties. Symptoms further develop interveinally until the entire upper leaf surface is distinctively silver. Silvering does not occur on the underside of the leaf. When leaf silvering is severe, fruit color is lighter than normal. Yield reductions and poor fruit quality are usually associated with leaf silvering.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Squash silver leaf is a physiological disorder induced by feeding of immature stages of *Bemisia tabaci* biotype B on squash leaves. When population density is high, adults of *B. tabaci* biotype B are capable of inducing squash silver leaf. Severity of leaf silvering in response to whitefly feeding varies among squash genotypes. The genetic controlled manifestation of silver leaf is confined to silvering along the axils of leaf veins. Genetically controlled silvering does not progress interveinally.

CONTROL:

Help control whitefly infestations with insecticides, biological control agents, plastic mulch and/or removal of whitefly-infested leaves.



Squash plant with severe symptoms of leaf silvering.

ABIOTIC DISORDERS

SQUASH SILVER LEAF



Whitefly-induced silverying found in association with *Squash leaf curl virus*.



Genetic silverying of squash.

ABIOTIC DISORDERS

WIND AND SAND INJURY



Cucumber cotyledons with wind damage. (Courtesy of Gerald Holmes)

CAUSAL AGENTS:

Wind and sand

DISTRIBUTION:

Worldwide

SYMPTOMS:

Plants wilt and become dry and brittle. Foliage can be shredded or tattered. Fruit may develop small pimple-like lesions where sand grains damage the epidermis.

CONDITIONS FOR SYMPTOM DEVELOPMENT:

Cucumbers grown on sandy soil are particularly susceptible to sand injury. Storms and high winds may cause these disorders.

CONTROL:

Wind breaks can be planted at regular intervals to reduce wind and sand injury.



Cucumber leaf with interveinal necrosis caused by wind-blown sand. (Courtesy of David Langston)



Leaf burn on melon leaves due to excessive wind and blowing sand.

GLOSSARY

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

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

ACERVULUS (pl. acervuli): A saucer-shaped asexual fruit body of a fungus bearing closely packed conidiophores and conidia.

ADAXIAL: Surface of a leaf facing towards the stem.

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

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

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

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

BUD: An immature or undeveloped structure that develops into a bodily structure.

CAMBIUM: Tissue between xylem and phloem.

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

CAUSAL ORGANISM: The organism or agent (bacterium, fungus, nematode, virus, etc.) that incites a given disease or injury.

CHEMIGATION: The application of a pesticide or a system maintenance compound through an irrigation system.

CHLAMYDOSPORE: A thick walled asexual spore produced by some fungi. Functions as an overwintering stage of a fungus.

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

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

CLEISTOTHECIUM (pl. cleistothecia): A spherical ascocarp, completely closed fruit body with no special opening to the outside.

CONCENTRIC: Different size circles having a common center.

CONIDIA: A spore produced asexually by various fungi at the tip of a specialized hypha.

CONIDIOPHORE: A simple or branched hyphal cell or group of cells bearing cells that produce conidia.

CORTEX: Tissue in root or stem between the vascular bundles and the epidermis.

CUCURBIT: A member of the family which includes cucumber, cantaloupe, watermelon, squash, pumpkin and gourd.

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

DEBRIS: Remnant plant material.

DEFOLIATION: Loss of leaves.

DISCING: To work (soil) with a disk harrow.

ELECTRICAL CONDUCTIVITY: A good indicator of the total salinity in irrigation water.

ENATION: A growth at leaf veins consisting of ridges or leaf-like formations.

FORMA SPECIALIS (f. sp.): Special form; a biotype (or group of biotypes) of a species of pathogen that differs from others in the ability to infect selected genera or species or infected plants.

FUMIGATION: Sterilization by fuming action.

FUNGICIDE: Chemical used to control fungi.

FUNGUS (pl. fungi): A microscopic organism with threadlike cells which lives on dead or living plants and animals.

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

GIRDLE: To encircle with dead tissue around a root or stem.

GUTTATION: Exudation of water and solutes from the hydathodes of plants, especially along the leaf margin. Exudation can also occur from the fruit epidermis.

HAUSTORIUM (haustoria pl.): Specialized, simple or branched structure of a fungal parasite, especially with a projection within a living host cell, for absorption of food. Often associated with obligate parasites (e.g., downy and powdery mildews)

HERBICIDE: Substance used to control weeds.

GLOSSARY

HYPHA (pl. hyphae): Microscopic tubular filament that increases in size by growth at its tip. Hyphae constitute the body of a fungus.

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

INFECTION: Process in which an organism attacks a plant.

INOCULUM: The pathogen or its parts (e.g., fungus spores, mycelium, bacterial cells, nematodes, virus particles, etc.) used for inoculating to produce disease.

INTERVEINAL CHLOROSIS: Refers to loss of normal green color of plant tissues, between veins, usually observed on leaves.

LESION: A well defined but limited disease area on a plant.

MACROCONIDIA: The larger, generally more diagnostic conidia of a fungus.

MICROCONIDIA: The smaller conidia of a fungus. A small conidia often acting as a male sex cell.

MICROSCLEROTIA: Dense aggregate of darkly pigmented, thick-walled hyphal cells specialized for survival of a fungus. Capable of germinating to produce mycelium.

MOSAIC: Pattern of light and dark areas often caused by viruses.

MOTTLE: Irregular blotches of light and dark areas.

MULCH: A protective covering, usually of organic matter such as leaves, straw, or peat, placed around plants to prevent the evaporation of moisture, the freezing of roots, and the growth of weeds.

MYCELIUM (pl. mycelia): Strands of interwoven hyphae making up a vegetative body of a true fungus.

NECROSIS (adj. necrotic): Tissue which dies, turning discolored.

NEMATOCIDE: A substance that kills or inhibits nematodes.

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

NON-PERSISTENT TRANSMISSION: Insect transmission in which the virus is acquired by the vector after a very short acquisition feeding time and is transmitted over a short window of feeding by the insect.

OBLIGATE PARASITE: A parasite that can grow and multiply in nature only on or in living tissue and that cannot be cultured on artificial medium.

PARTHENOCARPIC: Production of fruit without fertilization.

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

PATHOVAR (pv.): A type or subspecies; strain or group of strains of a bacterial species differentiated by pathogenicity on one or more hosts (species of cultivars).

PEDICEL: Small slender stalk; stalk bearing an individual flower, inflorescence or spore.

PERITHECIA: A small, flask-shaped fruiting body in ascomycetous fungi that contains the ascospores.

PERSISTENT TRANSMISSION: Refers to circulatory viruses that remain infectious and replicates within their insect or other vectors for long periods and are transmitted via salivary glands. Long acquisition feeding times as well as latent periods are associated with this virus-vector transmission.

PESTICIDE: A substance used for destroying insects or other organisms harmful to cultivated plants or to animals.

PETIOLE: The slender attachment that joins a leaf to a stem.

PHLOEM: Food-conducting tissue of a plant.

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

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

PSEUDOSCLEROTIUM (pl. Pseudosclerotia): Sclerotia-like structures; compacted mass of intermixed substrata possibly with host tissue but held together by mycelium.

PSEUDOSTROMA (pl. Pseudostromata): Interwoven hyphae combined with host cells to produce a stroma-like structure.

PSEUDOTHECIA: An ascocarp, or ascoma, is the fruiting body of an ascomycete fungus.

PYCNIDIA: A flask-shaped asexual structure containing conidia, found in certain fungi.

RACE: Specialized type of disease causing organism.

GLOSSARY

RESERVOIR: Plants which can harbor a disease causing organism.

RESISTANT: Able to withstand a disease without damage.

ROOTSTOCK: A root used as stock to which another plant is grafted.

RUGOSE: Wrinkled, roughened surface.

RUNNER: A horizontal stem that grows close to the soil surface.

SATURATION: Being completely filled with liquid, generally water.

SEMI-PERSISTENT TRANSMISSION: Refers to viruses which are associated with the stylets (mouthparts) of their arthropod vector. No virus replication occurs in the vector and the acquisition feeding time is short with no latent periods.

SCLEROTIUM (pl. sclerotia): A hardened, resting body produced by certain fungi.

SOILBORNE: Denoting a soil source or origin of pathogens; the property of a microorganism living and surviving in the soil.

SUBSTRATE: The material used in a container to grow a plant.

SPORE: A reproductive structure of fungi and some bacteria.

SPORANGIUM (pl. sporangia): A spore case of fungi whose contents are converted into an indefinite number of asexual spores (zoospores, sporangiospores).

SPORULATE: To form or produce spores.

STOMATE (pl. stomata): Pore in a leaf.

STRAIN: A specialized type of disease-causing organism.

STYLET: Feeding tube for an insect and/or nematode.

SYMPTOMS: Indication of disease by reaction of the host.

TOLERANT: Able to withstand a disease with limited damage.

TOXIN: A poison produced by an organism.

VASCULAR: Referring to the conductive system of a plant composed of the xylem and phloem.

VECTOR: An organism able to transmit a pathogen.

VIRUS: Very small sub-microscopic disease-causing agent.

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

XYLEM: Water conducting tissue of a plant.

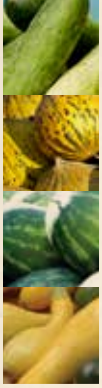
ZOOSPORE: An asexually produced fungal spore able to swim by movement of cilia or flagella.

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