

# Cucurbitaceae

Gourd family

THE CUCURBITACEAE (gourd family) includes a wide variety of vegetable crops that are commercially grown primarily for their edible fruits and sometimes immature blossoms. Cultivated cucurbits are classified into two groups, the Cucurbitaceae and Sicyoideae. Notable commercial species include the following: cucumber (*Cucumis sativus*); muskmelon and other melons (*Cucumis melo*); cantaloupe (*C. melo* var. *cantalupensis*); watermelon (*Citrullus lanatus*); summer and winter squashes, gourds, or courgette (*Cucurbita pepo*); vegetable marrow (*C. pepo* var. *medullosa*); pumpkin (*Cucurbita maxima* and *C. pepo*); Chinese winter melon (*Benincasa hispida*); bitter melon (*Momordica cochinchinensis*). It is believed that many of these species originated from tropical regions in Africa, the Americas, and Asia.

## *Acidovorax avenae* subsp. *citrulli*

### BACTERIAL FRUIT BLOTCH

#### Introduction and significance

Compared to most cucurbit diseases, bacterial fruit blotch is a recently described problem, being first observed and characterized in 1988–1989. Watermelon is the primary host. However, honeydew and musk melons, cantaloupe, pumpkin, citron, and squash are also susceptible.

#### Symptoms and diagnostic features

Symptoms on leaves are not particularly striking and may resemble other diseases. Leaf spots are small, water-soaked to brown, irregularly shaped, and with angular edges. Such spots may sometimes be surrounded by chlorotic borders (264, 265). The pathogen is seedborne, resulting in seedling infections where the cotyledons develop irregularly shaped water-soaked lesions (266). Lesions can expand and run along the cotyledon midrib and become brown to red-brown and necrotic. Hypocotyls on young seedlings can develop lesions and subsequently collapse and die.

The most important impact of this disease is on watermelon fruits. The upper portion develops small (less than 1 cm) irregularly shaped lesions that rapidly expand into large blotches that can cover most or all of the fruit (267). Early in development, blotches appear either water-soaked, dull gray-green, or dark green in color. In time the older, central area of lesions can turn brown to red-brown and necrotic, with the epidermis cracking and an amber-colored exudate oozing out of

**264** Watermelon leaf showing leaf spots caused by *Acidovorax avenae* subsp. *citrulli*.



**265** Watermelon leaf showing necrotic lesion caused by *Acidovorax avenae* subsp. *citrulli*.



**266** Watermelon cotyledons infected with the bacterial fruit blotch pathogen.

the central blotch area. Blotches are shallow infections that usually do not penetrate into the watermelon flesh. Secondary decay organisms that invade fruit blotch lesions are mostly responsible for fruit breakdown, rot, and collapse. Triploid (seedless) cultivars of watermelon may be less susceptible.

#### Causal agent

Bacterial fruit blotch is caused by *Acidovorax avenae* subsp. *citrulli* (formerly named *Pseudomonas pseudoalcaligenes* subsp. *citrulli*). This bacterium is a Gram-negative rod having a single polar flagellum, is nonfluorescent in culture, and forms white colonies. When inoculated into tobacco leaves, this pathogen generally causes a hypersensitive reaction. However, some strains are not able to cause the tobacco hypersensitivity reaction, and infect watermelon seedlings but not fruit.

#### Disease cycle

The pathogen is seedborne in many cucurbit species and can therefore be present when seedlings germinate in the field or in transplant greenhouses. Warm temperatures and high humidity favor disease development. Splashing water from rain and sprinkler irrigation spread the bacteria from plant to plant. Environmental conditions inside transplant greenhouses are particularly favorable for this disease. The bacterium infects immature fruit through stomata, and young fruit apparently are most susceptible to infection. It appears that the bacterium can infect fruit pulp and seeds if it enters through blossoms early in fruit development. The pathogen survives in infested crop residues and fruit rinds, and on diseased volunteer and weed cucurbits.



**267** Fruit lesions of bacterial fruit blotch on watermelon.

Apparently this disease does not readily spread in storage from diseased to healthy watermelon fruit if proper postharvest conditions are maintained.

#### Control

Use seed that does not have significant levels of the pathogen. Seed treatments such as bleach, other bactericides, and fermentation plus seed-drying procedures may be helpful in cleaning up infested seed. Remove cucurbit weeds and volunteer reservoir hosts. Complete resistance is not yet available for watermelon. Rotate out of cucurbit crops for at least 3 years. Reduce or eliminate the use of sprinkler irrigation. For transplant greenhouses, practice good sanitation, reduce humidity levels, reduce sprinkler irrigation, and remove symptomatic plants and surrounding trays. Accept and plant transplants that do not show symptoms of the disease. Copper sprays provide some control.

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### *Erwinia tracheiphila*

## BACTERIAL WILT

### Introduction and significance

Bacterial wilt is a serious disease of cucurbits such as cucumbers and muskmelons, while it is less of a concern on various other cucurbit crops. The disease is more important in North America than in Europe.

### Symptoms and diagnostic features

Initial symptoms consist of the wilting of a few to several leaves along a stem. As disease develops, the foliage will exhibit extensive wilting. The wilting can occur on individual runners or stems, or throughout an entire plant's foliage. Wilting foliage takes on a dark green color, but later can become chlorotic and then necrotic. In advanced stages of the disease the entire plant will collapse and die (268). Sticky strands of bacterial ooze can be observed when the cut ends of symptomatic stems are slowly drawn apart (269). Squash fruit can be infected and show small, irregular water-soaked areas on the fruit surface.

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268 Collapsing oriental melon vines caused by bacterial wilt.



269 Bacterial strands extending from cut ends of melon stem infected with bacterial wilt.

### Causal agent

The cause of bacterial wilt is the Gram-negative bacterium *Erwinia tracheiphila*, which is classified in the *Erwinia amylovora* taxonomic group. This pathogen differs from most *Erwinia* species in that *E. tracheiphila* does not liquefy gelatin, shows weak action in milk, does not reduce nitrates, and does not metabolize inorganic nitrogen sources. This pathogen is host specific to the Cucurbitaceae. *Erwinia tracheiphila* is mechanically transmitted by a number of insects, primarily the striped cucumber beetle (*Acalymma vittatum*) and the spotted cucumber beetle (*Diabrotica undecimpunctata*). Insects that are in contact with and feed on diseased plant tissue become contaminated with the pathogen. These insects move to other plants and inoculate them when their infested mouthparts feed on the plants. The bacteria enter the xylem via these feeding wounds and become systemic within the plant. Wilting occurs when the multiplying bacteria and resulting resins plug the vascular tissue of the host plant. This disease develops most rapidly in young, succulent cucurbit plants.

### Disease cycle

The complete epidemiology of bacterial wilt has not yet been determined. The pathogen is apparently not seedborne, nor does it survive in soil beyond 2 or 3 months. In contrast to earlier studies, it now appears that the pathogen does not persist within the insect vector's intestinal tract. The initial inoculum of bacterial wilt, therefore, is not known. Infected weed or volunteer cucurbit hosts may be the source of inoculum from which insects become infested. The pathogen may survive in dried plant tissues for a limited time.

### Control

Plant resistant cultivars as they become available. Control the vector insects by applying systemic insecticides to the soil or contact insecticides to host plants.

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*Pseudomonas syringae* pv. *lachrymans*

## ANGULAR LEAF SPOT

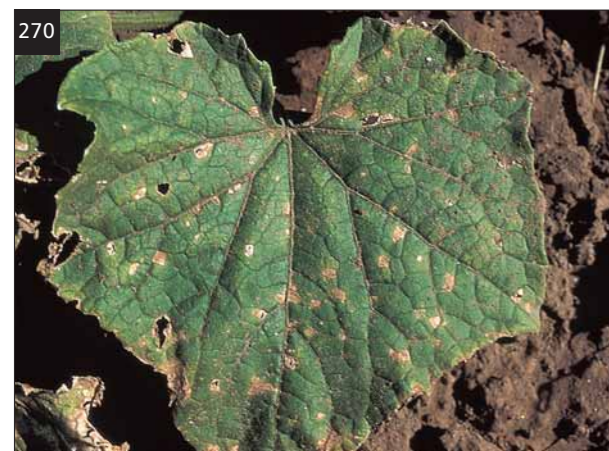
## Introduction and significance

Angular leaf spot occurs on cucurbits throughout the world and is particularly serious in more humid, warmer regions. The disease can be severe on cucumber grown outside in fields, but is less commonly found on cucurbits grown inside greenhouses.

## Symptoms and diagnostic features

Initial symptoms occur on leaves and consist of small, irregularly shaped, water-soaked to gray-colored lesions. As these lesions expand, they become angular in shape as the lesion edges become delimited by leaf veins (270). On some cultivars the lesions can be surrounded by chlorotic borders. As lesions age they turn tan to gray and become dry. The dried tissue often tears and falls out, resulting in a 'shot hole' or tattered appearance (271). If conditions are wet or humid, bacterial exudates can ooze onto lesion surfaces and dry into a white residue. Lesions and bacterial ooze can also develop on petioles and stems.

Early symptoms of fruit infections consist of typically oval to circular lesions that are small (1 to 5 mm in diameter) and water-soaked. The water-soaked appearance can later turn into a brown discoloration of fruit tissues. Exudates sometimes collect on these fruit spots. Lesions can later develop into deep internal rots; such infections, along with the activity of secondary decay organisms, result in unmarketable fruit.



270 Leaf lesions of angular leaf spot on cucumber.

## Causal agent

The pathogen is the aerobic, Gram-negative bacterium *Pseudomonas syringae* pv. *lachrymans*, strains of which are host specific to cucurbit plants. When cultured on Kings medium B, this organism produces a diffusible pigment that fluoresces blue under ultraviolet light.

Other *Pseudomonas* pathogens affect the cucurbit group of plants, though in many reports it is not clear exactly which species and pathovars are involved. Recently a *Pseudomonas* disease has been reported on cantaloupe in Europe. The pathogen is a pathovar of *P. syringae* and causes necrotic leaf spots with water-soaked margins, stem and petiole cankers, and sunken fruit lesions. Fruit infection can cause immature fruit to drop and more mature fruit to develop dry rot cavities. The bacterium is also pathogenic to sugar beet and therefore may be *P. syringae* pv. *aptata*. Another bacterium, *P. syringae* pv. *syringae*, is reported to also cause disease on cantaloupe and squash in the USA.

## Disease cycle

This pathogen is internally seedborne, which results in infection of the seedling cotyledons upon germination. Bacteria are splashed from plant to plant by splashing water from rain and sprinkler irrigation. Insects and physical contact from passing equipment and harvesters can also spread the pathogen. Warm, humid conditions favor disease development. Optimum temperatures for disease development are 24–28° C, though the pathogen can survive at higher (36° C) temperatures. The pathogen can also survive in the soil on infested plant residues for perhaps up to 2 years.



271 Advanced symptoms of angular leaf spot on cucumber.

## Control

Use seed that does not have significant levels of the pathogen. Seed treatments for this disease usually consist of hot water that has been acidified, or heated zinc or manganese sulfate solutions. Rotate crops so that seed and fruit production plantings are placed in fields that have not had cucurbits for 2 years. Do not use overhead sprinkler irrigation. Conduct production and harvesting procedures only when the foliage is dry. Use resistant cultivars if they are available. The application of copper sprays may also be useful. Do not over fertilize the crop with high nitrogen fertilizers, as excessive nitrogen may increase disease severity, especially on younger leaves.

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*Serratia marcescens*

## CUCURBIT YELLOW VINE DISEASE

## Introduction and significance

A relatively newly discovered disease, cucurbit yellow vine disease (CYVD) was first seen on squash and pumpkin plantings in the USA (Oklahoma, Texas) in 1988. The disease has since been documented in other states in the southeastern USA as well as in Massachusetts. Levels of damage vary, but CYVD can cause extensive crop loss to cantaloupe, pumpkin, squash, and watermelon.

## Symptoms and diagnostic features

Initial symptoms usually occur 10 to 15 days before fruit maturity. Foliage rapidly turns a lime green, then bright yellow. Plants show stunted growth and eventual decline. Older leaves can be blighted or burned in appearance, and younger leaves on vine tips can curl and not properly expand. A characteristic symptom is the gold to honey-brown-colored phloem in the main roots and crowns of diseased plants. In advanced stages of CYVD, the root systems will rot; however, this is thought to be due to secondary decay organisms in the soil. In Oklahoma, watermelon and cantaloupe fields that are planted before 15 June show the greatest disease severity.

In some cases, at the time of flowering and fruit set, plants will not turn chlorotic but instead will wilt and collapse in a day or two. Fruit appear normal in shape, but in the case of watermelon the fruit lose their green color and become unmarketable. Because this bacterium is vectored by an insect, symptoms often appear in aggregated small patches or along the edges of fields; such patterns are related to the movement of the insect.

## Causal agent

The pathogen is the aerobic, rod shaped, Gram-negative bacterium *Serratia marcescens* that inhabits the phloem tissue of cucurbit hosts. The pathogen can be isolated by grinding phloem tissue in buffer and then streaking the resulting suspension onto potato dextrose agar, nutrient agar, or purple agar. The bacterium can be maintained on nutrient agar. This bacterium is vectored by the squash bug (*Anasa tristis*).

*Serratia marcescens* is interesting in that strains of this bacterium are found in a number of diverse environments. On plants, *S. marcescens* is a non-pathogenic endophyte in cotton and rice, a crown rot pathogen of alfalfa and sainfoin (*Onobrychis vicifolia*), and now an important cucurbit pathogen. Other strains cause disease in insects, are opportunistic pathogens on humans, and inhabit water and soil ecosystems. Unlike many other *S. marcescens* strains, cucurbit strains do not produce the red prodigiosin pigment. While genetic analysis shows that the cucurbit strains are *S. marcescens*, other experiments show that the cucurbit strains form a distinct group and differ from other non-CYVD strains in biochemical and physiological aspects.

The host range of this cucurbit pathogen has not yet been determined. Researchers found, however, that the *S. marcescens* endophyte strains from rice were not pathogenic to cucurbits.

#### Disease cycle

The disease epidemiology of CYVD has not been determined. There is no evidence that the pathogen is seedborne. It appears that the squash bug vector is the most important factor in pathogen survival and distribution. Overwintering populations of this insect can transmit the pathogen to new crops in the spring.

#### Control

Manage the squash bug vector by spraying insecticides, plowing down old cucurbit plantings, and removing vegetation habitats that might help the insect overwinter.

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*Acremonium cucurbitacearum*,  
*Fusarium* spp., *Phytophthora* spp., *Pythium* spp.,  
*Rhizoctonia solani*, *Rhizopycnis vagum*

### DAMPING-OFF, ROOT ROT

#### Introduction and significance

A number of soilborne pathogens cause seed and seedling diseases of cucurbits. Pathogens include species of *Acremonium*, *Fusarium*, *Phytophthora*, *Pythium*, *Rhizoctonia*, and *Rhizopycnis*. Some of these pathogens are also implicated in the vine decline complex that affects mature plants.

#### Symptoms and diagnostic features

These diseases have various phases. Cucurbit seeds can be infected prior to germination and result in seed death. Newly germinated seedlings can be infected to such a degree that plants do not emerge above the soil (pre-emergence damping-off). Finally, seedlings might emerge from the ground but become diseased after soil emergence (post-emergence damping-off) (272). Initial symptoms of post-emergence damping-off generally consist of yellow or light tan to dark brown lesions of the root and hypocotyl tissues (273). With time the hypocotyl shrivels, roots decay further, and the cotyledons and leaves wilt and collapse. Some pathogens initially attack the tissue where the seed coat is in contact with the hypocotyl after germination. Damping-off diseases often result in death of the seedling and subsequent reduction of plant stands (274). However, even if plants do not succumb to these pathogens, the surviving plant may be stunted and delayed in development.

Older, mature plants can also be attacked by these seedling pathogens. The feeder roots of mature plants will discolor and turn a water-soaked, tan to brown color. The root tissue becomes soft and can slough away. Upper taproot and lower stem tissue in contact with the soil can become first gray-green, then brown to red-brown, soft in texture, and rotted. Fruit in contact with the soil develop irregularly shaped, water-soaked to brown lesions. These fungi, along with *Verticillium dahliae*, are implicated in the vine decline disease that is caused by a complex of fungi.

#### Causal agents

*Acremonium cucurbitacearum* produces conidiophores that are simple, awl-shaped, erect monophialides measuring 13–54 µm long. Conidia collect in slimy aggregations and are usually one-celled, hyaline, oblong to cylindrical, and measure 5–7.5 x 2–4 µm. This pathogen produces chlamydospores that allow it to survive in the soil. Optimum disease development occurs at 24–27° C, though infection takes place over a broader temperature range (12–30° C).

*Fusarium equiseti* and *F. solani* can cause seedling diseases of cucurbits. However, even *Fusarium* wilt pathogens (various forma speciales of *F. oxysporum*), may sometimes cause damping-off. *Fusarium* species have branched or unbranched conidiophores that have phialides as conidiogenous cells. Phialides produce either small, one- to two-celled microconidia or larger, fusoid, curved, multicelled macroconidia. Macroconidiophores grow in clustered groups called sporodochia. Resilient chlamydospores enable *Fusarium* species to survive for long periods of time in the soil.

*Phytophthora* and *Pythium* species belong in the oomycete group of organisms. *Phytophthora drechsleri* has been reported to cause seedling damping-off. A number of *Pythium* species cause damping-off: *P. aphanidermatum*, *P. irregulare*, *P. myriotylum*, *P. ultimum*. All these pathogens survive in the soil as saprophytes and are favored by wet soil conditions. With the exception of *P. ultimum*, these organisms usually produce zoospores that swim to and infect susceptible tissues. Sexual structures (antheridia, oogonia, and oospores) are produced by all species. In addition to cucurbits, these pathogens can infect numerous other plants.

*Rhizoctonia solani* is a soilborne fungus with a very broad host range. *R. solani* has no asexual fruiting structures or spores, but produces characteristically coarse, brown, right-angle branching hyphae. The hyphae are distinctly constricted at branch points, and cross walls with dolipore septa are deposited just after the branching. Hyphal cells are multi-nucleate. Small, tan to brown loosely aggregated clumps of mycelia function as sclerotia. This fungus can survive by infecting and thriving on a great number of plant hosts, besides cucurbits, and can also persist in the soil as a saprophyte. The teleomorph stage, *Thanatephorus cucumis*, is not commonly observed.



272 Muskmelon seedlings affected by *Pythium* damping-off.

273 *Rhizoctonia* root rot on young watermelon seedlings.



274 Watermelon seedlings affected by *Pythium* damping-off.