Bacterial Fruit Blotch
Acidovorax avenae subsp. *citrulli*

Hosts: Watermelon (*Citrullus vulgaris*), cucumber (*Cucumis sativus*), pumpkin (*Cucurbita pepo*), squash (*Cucurbita* spp.), melon (*Cucumis melo*), gourd (*Lagenaria* spp.), white-flowered gourd (*Lagenaria leucantha*), citronmelon (*Citrullus lanatus*), cantaloupe (*Cucumis melo* var. *cantalupensis*), and a wide range of cucurbitaceous hosts, including weeds such as wild bur (*Sicyos angulatus*) and gherkin (*Cucumis anguria*).

Disease common name: Bacterial fruit blotch (BFB).

Pathogen: *Acidovorax citrulli*; syn.: *Acidovorax avenae* subsp. *citrulli*, *Pseudomonas avenae* subsp. *citrulli*, and *Pseudomonas pseudoalcaligenes* subsp. *citrulli*.

Disease Cycle

Inoculum: Infested seed, infected transplants, and many cucurbit hosts are sources of inoculum. Infected transplants are the most important source, although volunteer watermelon seedlings also may carry the bacterium. The bacterium can be a surface or internal contaminant of seed harvested from infected watermelon fruit.

Transmission: The bacterium can spread throughout a field by wind-driven rain, aerosols, or mechanical means.

Infection: BFB has a wide range of cucurbit hosts; fruit and foliage are susceptible to infection at different stages of growth. Bacteria from infected transplants can cause infections on susceptible 2- to 3-week-old watermelon fruit. The naturally occurring waxy layer that develops on maturing watermelon hinders bacterial invasion, but when the fruit rind is injured, the protective waxy layer is breached and infection can occur. Disease development is favored by warm, wet weather, such as exists in the southeastern United States during the spring watermelon season (May–June) and during the fall watermelon season (August–September). The disease develops quickly under these conditions and may spread from a few primary inoculum foci to all plants within the field.

Symptoms and signs: The images (Figs. 1–10) show the array of symptoms caused by BFB. Initial symptoms on infected transplants are dark, water-soaked areas on the underside of cotyledons and true leaves. Brown necrotic lesions may appear on the foliage as the disease progresses. Young seedlings may develop lesions on the hypocotyl, causing eventual collapse and death of the seedling. Disease symptoms on foliage can progress throughout the growing season and may not be particularly obvious or may be confused with other diseases. Leaf lesions are generally light to reddish brown and often spread along the midrib of the infected leaf. These leaf lesions act as inoculum reservoirs. Defoliation is not usually a factor with BFB infections. Symptoms on susceptible fruit begin as small, water-soaked areas (a few millimeters in diameter) and rapidly expand into larger lesions with irregular margins. The entire surface of the fruit, except the ground lesions, may become covered with dark green lesions within a few days. Older fruit lesions become necrotic and may crack, exposing the interior of the rind and the flesh of the melon. Whitish foam may exude from the cracks. Eventually, the infected fruit rots.

Survival: The bacterium survives on seed and on its many hosts.

Disease Management

Preventative measures are needed to reduce the likelihood of BFB outbreaks. Introduction of the pathogen into production fields should be avoided by planting disease-free seed, as

determined by an accepted seed health assay, and by using transplants from seedling producers with no history of BFB. Transplant growers should conduct routine inspections and remove seedlings with suspicious symptoms. If feasible, all seedlings from transplant houses with BFB outbreaks should be discarded. Alternatively, symptomless seedlings proximal to symptomatic seedlings should be eliminated to limit the risk of disease development in the field. Personnel working in transplant houses should minimize the handling of seedlings. After contact with plants, hands, benches, containers, and tools should be decontaminated, and discarded plant material should be destroyed. No work should be performed in a wet, contaminated field. Copper-based fungicides can reduce the incidence of BFB if preventative treatments are initiated prior to fruit set. In field operations, watermelon culls and plant debris should be buried. New watermelon production areas should be located as far as possible from previously farmed watermelon fields. Volunteer watermelon seedlings and wild cucurbit weeds should be destroyed to remove inoculum sources and potential alternate hosts. Resistance to BFB is a desirable option for management. It has been suggested that watermelons with light green rinds (e.g., 'Charleston Gray') are the most susceptible to infection. Light and dark green striped fruits (e.g., 'Crimson Sweet') are more resistant than the light-skinned varieties, and the solid dark green varieties (e.g., 'Sugar Baby') are the most resistant to BFB. Nevertheless, there currently is no commercial cultivar with a reliable resistance.

References

Leahy, R. Bacterial fruit blotch of watermelon *Acidovorax avenae* subsp. *citrulli*. Florida Department of Agriculture and Consumer Services, Division of Plant Industry, Pest Alert. www.doacs.state.fl.us/pi/enpp/pathology/fruit-blotch.html.

Walcott, R., Langston, D., Gitaitis, R., Hopkins, D., Kucharek, T., and Egel, D. 2001. Guidelines for managing bacterial fruit blotch disease. Georgia Extension Vegetable Team, University of Georgia, College of Agricultural and Environmental Sciences. www.tifton.uga.edu/veg/Alerts/managing%20fruit%20blotch.pdf.

Zitter, T. A., Hopkins, D. L., and Thomas, C. E., eds. 1996. Compendium of Cucurbit Diseases. American Phytopathological Society, St. Paul, MN.

Figure 1. Severe infection with copious white foam on watermelon fruit. (Courtesy R. Gitaitis)

Figure 2. Severely rotted watermelon fruit. (Courtesy R. Gitaitis)

Figure 3. Rind infection of citronmelon. (Courtesy R. Gitaitis)

Figure 4. Leaf infection of citronmelon seedlings. (Courtesy R. Gitaitis)

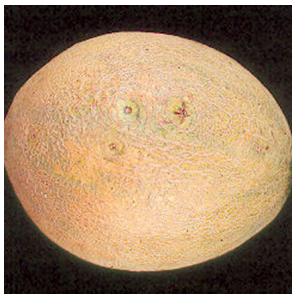

Figure 5. Discolored blotches on surface of cantaloupe. (Courtesy R. Gitaitis)

Figure 6. Disease path from rind to internal rot of cantaloupe. (Courtesy R. Gitaitis)

Figure 7. Cantaloupe leaf lesions. (Courtesy R. Gitaitis)

Figure 8. Pumpkin fruit lesion. (Courtesy R. Gitaitis)

Figure 9. Severe internal rot of pumpkin. (Courtesy R. Gitaitis)

Figure 10. Pumpkin leaf lesions. (Courtesy R. Gitaitis)