

Bacterial Canker of Tomato

Clavibacter michiganensis subsp. *michiganensis*

Hosts: Tomato (*Solanum lycopersicum*); pepper (*Capsicum annuum*), and solanaceous weeds; several nightshades, including perennial nightshade (*Solanum douglasii*), black nightshade (*Solanum nigrum*), and cutleaf nightshade (*Solanum triflorum*) are naturally infected.

Disease common name: Bacterial canker.

Pathogen: *Clavibacter michiganensis* subsp. *michiganensis*; syn.: *Corynebacterium michiganense*.

Disease Cycle

Inoculum: Bacteria are found in contaminated seed, undecomposed plant debris, weed hosts, and volunteer tomatoes and peppers and on contaminated wooden stakes and pruning implements.

Transmission: The pathogen is spread by seed, water, contaminated farm implements, and workers.

Infection: Bacteria from contaminated seed infect the growing hypocotyls and epicotyls as seedlings emerge. They then invade the stem cortical tissues and xylem elements, moving up the plant, successively colonizing leaf traces and causing water stress and necrosis at leaf margins. Invasion of peduncles eventually results in infected fruit. Colonization of cortical cells by bacteria causes browning of stem tissues, and rapid multiplication results in the formation of cavities, ruptured epidermis, and stem cankers. Secondary infections occur readily as bacteria gain entrance through wounds made during pruning operations. They also enter through broken trichomes as plants rub against stakes.

Symptoms and signs: Characteristic symptoms of diseased young plants are poor growth and temporary wilting of branches. The plants exhibit signs of water stress. Necrosis of leaf margins is an early symptom of leaf infection (Fig. 1). This may be followed by yellowing and shriveling of lower leaves with curved and distorted growth. Characteristically, there is downward turning of lower leaflets and upward curling of leaflet edges, but symptoms may not develop until plant maturity. There are two types of symptoms on mature plants depending on whether infection becomes systemic by entering the vascular system or is secondary by bacteria infecting leaves, stems, and fruit. With systemic infection, the leaflets curl, yellow, wilt, turn brown, and die, a condition known as firing (Fig. 2). Such symptoms vary and there may be interveinal necrosis (Fig. 2) or gray areas around leaf veins (Fig. 3). With secondary infection, leaf margins are commonly infected and lesions are brown to almost black. Necroses and cankers on stems are common. Stems may display internal discolored streaks (Figs. 4 and 5) and external discolored streaks and lesions. The pith eventually becomes discolored and mealy. Blossoms and calyxes also can be infected (Figs. 6–8). Tomato fruit may be small and deformed if infected when young (Fig. 9). Lesions on fruit develop as raised, yellowish brown to black spots surrounded by a creamy white halo, referred to as bird's eye (Figs. 9–11).

Survival: Bacteria overwinter in undecomposed plant debris in soil and may survive for several years. However, seed is the principal means of survival and viable bacteria have been recovered from stored seed 4 years after harvest. Bacteria also have survived up to 10 months on wooden stakes.

Disease Management

The use of pathogen-free seed and transplants is the most important control method. Detection of the pathogen in seed is essential to restricting new disease introductions during international trade of hybrid tomato seed. Seed may be disinfested with diluted hydrochloric acid, calcium hypochlorite, several other chlorine compounds, or hot water. Bleach is generally used at 0.5–1% concentrations. Since the pathogen is specific to tomato and pepper, crop rotation with nonsusceptible hosts is important. Contaminated fields must be completely turned under and not planted to tomato or pepper for several growing seasons. Cultural practices include adequate separation of plantings to avoid disease spread from mature plants to the more susceptible young seedlings. Benches and equipment must be disinfested and kept clean. Copper sprays and antibiotics have minimal effect on disease control. Biological control with bacteriophages has reduced disease in greenhouse operations. Resistant plants are not available.

References

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- Gleason, M. L., Braun, E. J., Carlton, W. M., and Peterson, R. H. 1991. Survival and dissemination of *Clavibacter michiganensis* subsp. *michiganensis* in tomatoes. *Phytopathology* 81:1519-1523.



Figure 1. Early symptoms of necrosis of leaf margins. (Courtesy S. Miller)



Figure 2. Firing and interveinal necrosis of leaves. (Courtesy D. Ingram)

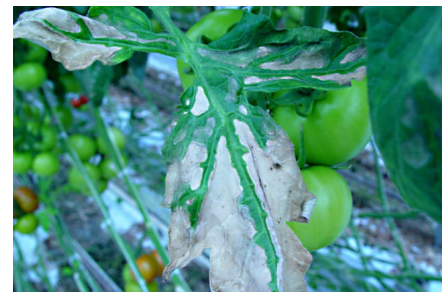


Figure 3. Gray necrosis around leaf veins. (Courtesy D. Ingram)



Figure 4. Brownish streaks in vascular tissues. (Courtesy S. Miller)



Figure 5. Advanced stage of internal infection seen as reddish brown discoloration of vascular tissue. Healthy stem is at the top. (Courtesy D. Ingram)



Figure 6. Lesions on stems and sepals. (Courtesy S. Miller)



Figure 7. Severe infection of calyx and internal infection of stems. (Courtesy J. Young)



Figure 8. Infection of blossom and calyx. (Courtesy D. Ingram)



Figure 9. Green tomatoes with bird's-eye spots with raised brownish centers surrounded by white halos. (Courtesy J. Young)

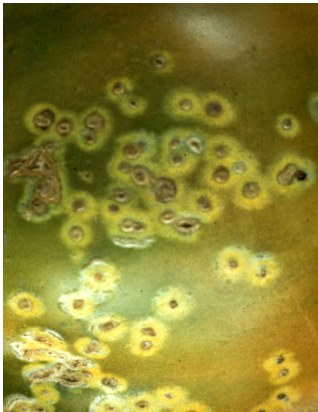


Figure 10. Green tomato with close-up view of bird's-eye spots with brownish raised centers and yellow halos. (Courtesy A. Hayward)



Figure 11. Ripe fruit with light brown lesions with white halos. The small, black, raised lesions are caused by bacterial speck (*Pseudomonas syringae* pv. *tomato*). (Courtesy M. Schroth)