Bacterial Blight of Anthurium and Other Aroids
Xanthomonas axonopodis pv. *dieffenbachiae*

Hosts: Most genera and species in the family Araceae.

Disease common name: Bacterial blight.

Pathogen: *Xanthomonas axonopodis* pv. *dieffenbachiae*; syn.: *Xanthomonas campestris* pv. *dieffenbachiae*.

Historical information: Anthuriums naturally occur as epiphytes in high-elevation tropical areas of Ecuador and Columbia. Commercial plantings of anthurium were established in Hawaii in the 1940s and were successfully grown for 40 years until 1981, when bacterial blight was first reported on the Island of Kauai. The disease reached epidemic proportions in 1985–1989, destroying the production of approximately 200 small farms in Hawaii. By 1992, the disease had been reported in most anthurium-growing areas of the world and also was reported to infect numerous other aroids. Following implementation of an integrated disease management program, production losses were eventually reduced to 5% or less, but due to the high cost of disease management, a few large farms now dominate the commercial markets. Many anthurium cultivars are hybrids of *Anthurium andraeanum* and/or *Anthurium antioquiense*, with selections based on the colorful spathe and upright spadix (Fig. 1).

Disease Cycle

Inoculum: Infected planting stocks and infected plant debris in soil are primary sources of inoculum.

Transmission: The bacteria are disseminated primarily by irrigation or rainwater splashing from diseased to healthy plants and on pruning and harvesting implements.

Infection: The pathogen enters mainly through hydathodes and wounds, occasionally through stomata, and rarely through roots.

Symptoms and signs: When bacteria invade through stomata on the undersides of leaves, water-soaked circular spots develop within 4–5 days (Fig. 2). They later become necrotic and are surrounded by a chlorotic zone (Fig. 3). The more usual mode of infection is through hydathodes and natural openings where veins end at leaf margins. At early stages, water-soaked spots are barely visible, although chlorosis is evident at leaf margins (Fig. 4). Larger water-soaked spots appear near hydathodes and zones of chlorosis surround the infected vessels (Fig. 5). Broad patches of necrotic tissues, typical of blight, extend toward the midrib and the petiole (Fig. 6). By the time infection has progressed to this stage, complete removal of the plant rather than excision of individual leaves is necessary to stop further spread in a field or nursery planting. The plant may be systemically infected long before the foliar symptoms develop, and often a systemically infected leaf exhibits only general chlorosis, characteristic of water stress or nutrient deficiency (Fig. 7). Vascular browning and discoloration of the petiole is diagnostic of bacterial infection (Fig. 8). Petioles then are easily detached from the central stalk, which has become soft, discolored, and translucent (Fig. 9). In severe infections, the central stalk blackens and decays (Fig. 10). A systemically infected plant often has several yellow, dried leaves resembling senescence (Fig. 11). Flower infection often is not observed because plants usually die long before symptoms appear. When the pathogen invades the spathe, infected areas become black and distorted (Fig. 12).

Survival: The bacterium primarily survives in leaf and stem tissues of diseased plants. It survives only for short periods (1–6 weeks) in soil in the absence of infected plant debris.

Disease Management

An integrated approach starts with the use of tissue-cultured microplants or pathogen-free cuttings as planting stocks and the avoidance of overhead irrigation. Latently infected, symptomless cuttings may harbor sufficient inoculum to initiate an epidemic. When plants begin to manifest symptoms in the field, infected plants must be immediately removed, followed by disinfection of clothing and implements and vigilant surveillance. Cultural practices, such as balanced nutrition (including avoidance of excess nitrogen), good drainage, aeration, and cultivation in cool areas (25–28°C), reduce disease development. Chemical and/or antibiotic sprays may reduce spread but are inadequate once disease has become established in a field. Copper compounds are phytotoxic to anthurium and some other aroids. *Anthurium andraeanum* varieties are more susceptible than is *Anthurium antioquiense*, and the latter is used as a source of resistance. Tolerant hybrids have been developed through both traditional breeding and bioengineering approaches. Biological control using a mixture of beneficial bacteria as foliar sprays has reduced disease by 30–45% in greenhouse tests and plastic-house operations but is not commercially available.

References

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Figure 1. Healthy anthurium with spathe and spadix. (Courtesy A. Alvarez)

Figure 2. Water-soaked spots on leaf. (Courtesy W. Nishijima)

Figure 3. Round necrotic lesions surrounded by chlorotic zones, indicative of stomatal invasion. (Courtesy A. Alvarez)

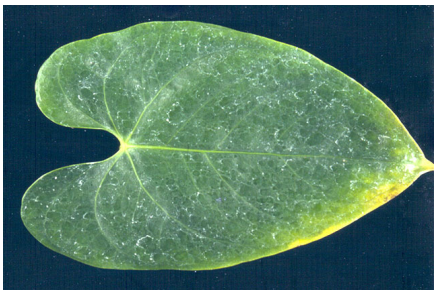

Figure 4. Marginal chlorosis at early stage of the disease. (Courtesy A. Alvarez)

Figure 5. Water-soaked spots and yellowing at leaf margin following hydathode invasion. (Courtesy W. Nishijima)

Figure 6. Broad bands of necrotic tissue extend toward the midrib and the petiole. (Courtesy W. Nishijima)

Figure 7. Chlorotic leaf exhibiting signs of water stress caused by systemic infection. (Courtesy W. Nishijima)

Figure 8. Discoloration of petiole, indicative of petiole infection. (Courtesy W. Nishijima)

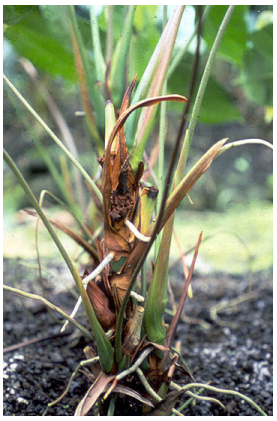

Figure 9. Blight symptoms and decay of central stalk. (Courtesy W. Nishijima)

Figure 10. Blackening and decay of central stalk. (Courtesy W. Nishijima)

Figure 11. Blighted plant with leaf resembling senescence. (Courtesy W. Nishijima)

Figure 12. Blighted plant with necrotic spathe. (Courtesy W. Nishijima)