25. Cryptosphaeria Canker of Cottonwood and Aspen

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The genus *Populus* is widespread throughout the Great Plains; five of the nine species native to the United States are found from Texas northward to North Dakota. Of the numerous pathogens known to attack poplars, *Cryptosphaeria populina* is a relatively new stem canker pathogen found on poplars in the Great Plains.

Hosts and Distribution

Although aspen is the principal host of *C. populina*, its occurrence and distribution on other poplars is becoming more apparent because of its recognition. The fungus has been found on narrowleaf cottonwood in Colorado, on Plains cottonwood in Colorado, Oklahoma, and Nebraska, on Lombardy poplar in Nebraska, and on aspen in South Dakota. Other hosts of the fungus include balsam and black poplar, and some hybrid poplars. The proper identification of this organism will no doubt extend its distribution and host range.

Symptoms and Signs

C. populina causes a stem canker that results in branch and tree mortality. Extensive discoloration and decay are attributed to the asexual stage, *Libertella* sp. The cankers, frequently associated with wounds, are long, narrow, and found on the trunk of hosts (fig. 25–1). They may be only 2 to 4 inches wide, but up to 10 feet in length, following the grain of the underlying wood. Cankers may grow up to an inch in width and many inches in length annually. Small trees usually die several years after being infected and before the trunk is girdled. Although branch infection does not always result in a prominent canker, the fungus will spread from a branch infection onto the trunk.

Cryptosphaeria populina colonizes the heartwood and sapwood, and causes extensive discoloration and decay. Various hues of gray, brown, yellow, and orange are associated with the brown-mottled decay, from which the *Libertella* stage of the pathogen is easily isolated. The fungus grows outward to the cambium and bark tissues, causing necrosis and canker formation. The infected bark becomes light brown to orange. After one or more years, the dead bark becomes black, stringy, and sootlike; however, it contains small (0.5–2.0 mm), scattered, lens-shaped, light-colored areas (fig. 25–2), and adheres tightly to the sapwood. This black, adhering dead bark with light

Figure 25–1. Elongated canker on aspen caused by *Cryptosphaeria populina*.

Figure 25–2. Lens-shaped, light-colored areas in the black, dead bark of the canker.

specks is a good diagnostic characteristic of this canker disease.

Perithecia are formed in a confined area in the dead bark that is raised on the surface in the form of a flat, broad blister that may vary from 0.3 inch in width and up to 1-foot in length (fig. 25–3). The fungus readily produces fruiting bodies on branches and smooth bark, but it is somewhat limited in penetrating and fruiting on the thick, rough bark of older trees. Light-orange fruiting bodies (acervuli) of the *Libertella* stage are occasionally found near the perimeter of cankers on aspen.

Cytospora chrysosperma frequently produces fruiting bodies along the canker perimeter and quickly colonizes the dead bark after the branch or tree dies. Because *Cytospora* fruiting bodies are very conspicuous on the dead tissue, it is often erroneously assumed to be the primary causal agent of the canker.

Disease Cycle

Branch and trunk infections become established in wounds to the bark and xylem. The sapwood is colonized by the pathogen, which causes discoloration and decay; consequently, the cankers seldom completely girdle the stem before stem death (fig. 25–4). Perithecia form in the spring on bark tissues that have been dead at least one year and mature during the summer and fall of their formation. Perithecia persist in the dead bark for several years and produce viable spores. Ascospores expelled from the perithecia are apparently wind dispersed. Size and shape of *C. populina* ascospores are similar to *Cytospora* conidia, but they are somewhat larger (8–10 by 2 μm vs. 3–5 by 1–5 μm) and vary from hyaline to pale yellow. Their similarity to *Cytospora* spores may have contributed to misidentification of the causal agent of the canker in the past.

Damage

Saplings and small trees can be killed before cankers and perithecia are produced. Trees weakened by branch and trunk decay are vulnerable to wind breakage. In a 1977 study of Colorado aspen, the canker was present on 83 percent of 30 sites examined, on 1 percent of the 2,873 live trees, and was responsible for 26 percent of the tree mortality encountered. The importance of this disease of poplars in the Plains is presently unknown.

Control

No direct control measures are known. The prevention of trunk wounds and pruning of dead, dying, or diseased branches on high-value trees should aid in reducing the incidence of disease.

Selected References

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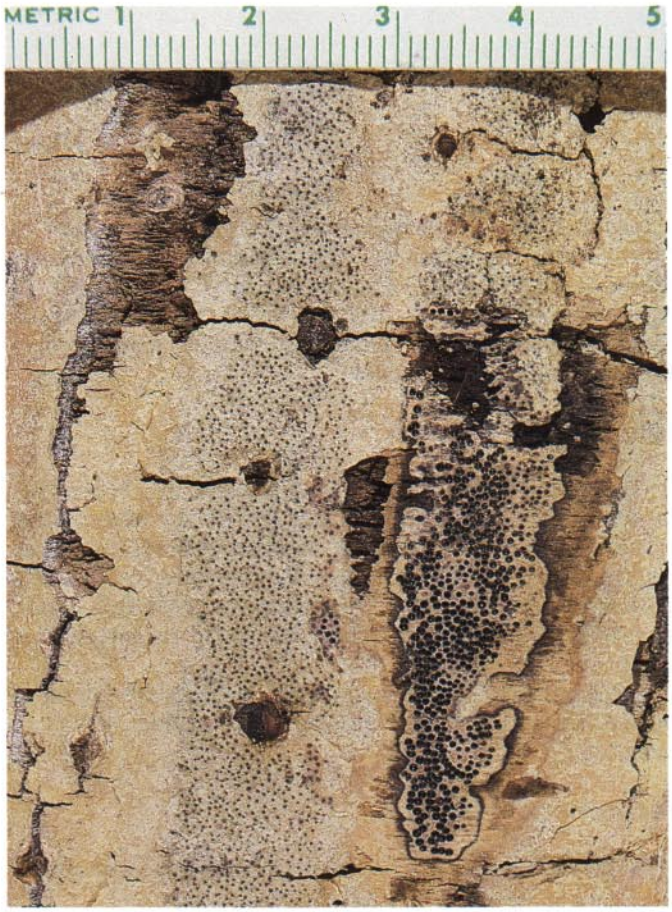

Figure 25–3. Formation of new perithecia in dead bark (left); periderm removed (right) to show perithecia formed the previous year (scale in cm).

Figure 25–4. Discoloration and brown-mottled trunk decay of aspen behind a canker (scale in cm).