

# 26. Hypoxylon Canker of Aspen

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Aspen is the most widely distributed forest tree in North America. While the tree is found in the northern States from Maine to Washington and as far south as Mexico in the West, its occurrence in the Great Plains region is sporadic, with small stands in Nebraska, North Dakota, and South Dakota.

### Hosts and Distribution

Hypoxylon canker (fig. 26-1), caused by *Hypoxylon mammatum*, is abundant east of the Rocky Mountains; it is the most important killer of aspen in the Great Lakes region. The canker is of minor importance in the tree's western range. Although the disease is present in aspen stands of the northern Great Plains, its importance is unknown.

Quaking aspen is very susceptible, bigtooth aspen is occasionally infected, and balsam poplar is rarely infected.

### Symptoms and Signs

Incipient cankers appear as slightly sunken, yellowish-

orange areas with irregular, lobed margins centered around wounds, dead branch stubs, or insect injuries and galls. As the infection progresses, the affected bark becomes mottled. A slight flow of sour-smelling brownish sap is common around the perimeter of infection. Small, blisterlike patches are formed under the outer dead bark during the second year of infection. The bark then ruptures, exposing small, bristlelike structures called hyphal pegs or pillars, upon which the spore-bearing structures are formed (fig. 26-2). The structures soon disintegrate. The diseased inner bark becomes laminated or mottled black and yellowish-white (fig. 26-3); white mycelial fans are formed near the canker margin under the bark.

Cankers are easier to identify 2 or 3 years after infec-



Figure 26-1. Canker initiation at fork of live 12-inch aspen.

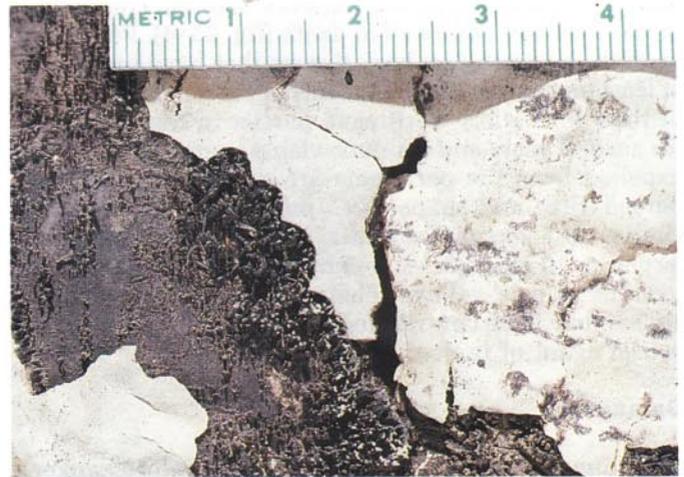


Figure 26-2. Hyphal pegs beneath blistered bark (scale in mm).



Figure 26-3. Mottled black and yellowish-white diseased inner bark.

tion, when perithecia are formed in small, crustlike stromata several mm in diameter on the dead bark (fig. 26-4). The grayish to black stromata appear to be partially covered with a white, flourlike material. The stromata are formed annually on the dead bark, and persist for several years.

Although the tree produces callus tissue at the margin of the infection, the fungus continues to invade new tissue each year. This invasion cracks the bark along the canker margins and leaves the dead bark in a slight depression. The black, dead bark in the center of older cankers begins to crack in a checkerboard fashion and sloughs off in small patches, revealing a checkering of the wood beneath (fig. 26-5). Older trunk cankers may be 3 feet or more in length before the trees are girdled and killed, usually within several years. The roughened, spotted gray and black bark of cankered trees stands out in contrast to the healthy bark of noninfected trees.

### Disease Cycle

The fungus is a wound parasite, but the means by which infection takes place is unknown. Apparently, airborne spores enter through wounds to the wood, including insect wounds. The fungus colonizes the sapwood, and then invades the bark from within by producing a toxin that causes bark collapse and necrosis.

The asexual stage of the fungus consists of conidiophores and conidiospores, which are produced on the hyphal pegs formed under the bark blisters at the end of the first or during the second growing season. The role of conidiospores in causing new infections is unknown. Between the second and third year of infection, the first immature perithecial stromata of the sexual state are formed on the dead bark. They are generally abundant on older cankers. Ascospore release requires free moisture, usually in the form of dew, rain, or snowfall, and can continue for several years.

### Damage

*H. mammatum* kills 1 to 2 percent of the standing



Figure 26-4. Perithecial stromata on dead bark (scale in mm).

aspen volume each year in the Lake States region by girdling the trees. While this canker disease is not of major importance in the Rocky Mountain region, its impact on aspen in the Great Plains is unknown.

Canker incidence varies with geographic location; the level of infection fluctuates from year-to-year. Tree vigor does not appear to influence host susceptibility. Although older stems are generally more resistant, upper bole infections, usually found on older trees, may partially kill the crown, and ultimately result in tree mortality from suppression. Trees weakened by decay behind cankers in the lower bole are susceptible to wind breakage before canker girdling is complete. Low density stands, mixed stands, thinned stands, and trees on the perimeter of stands are more susceptible to infection than fully stocked stands.

### Control

Although no direct control measures are known for Hypoxylon canker, certain silvicultural techniques can reduce canker losses. Because Hypoxylon canker is favored by stand openings and poor stocking, maintaining fully stocked stands and a closed canopy without openings should reduce volume losses.

Other silvicultural management practices advocated for aspen in the Lake States include: (1) if 15 to 20 percent of the trees are infected, harvest the stand early and treat the site to encourage good aspen reproduction; (2) if more than 25 percent are infected, harvest immediately and convert to other species (susceptible clones should not be perpetuated); and (3) lightly infected stands can be managed on rotations longer than 40 years.

### Selected References

- Anderson, R. L.; Anderson, G. W. Hypoxylon canker of aspen. Forest Pest Leaflet 6. Washington, DC: U.S. Department of Agriculture, Forest Service; 1979. 7 p.
- Schipper, A. L., Jr.; Anderson, R. L. How to identify Hypoxylon canker of aspen. St. Paul, MN: U.S. Department of Agriculture, Forest Service, North Central Forest Experiment Station; 1976. 5 p.

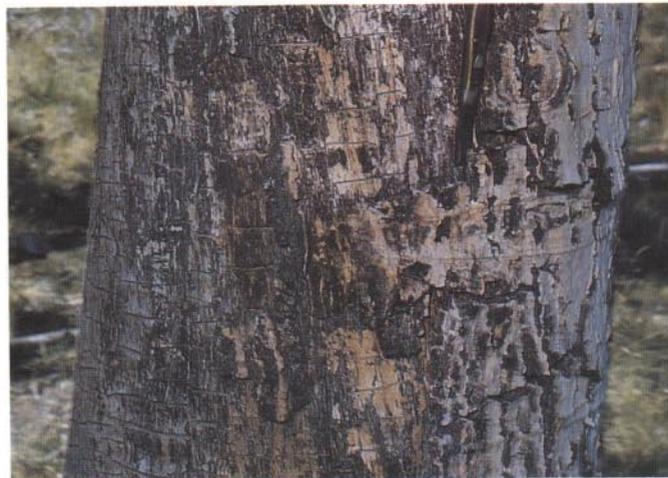


Figure 26-5. Checkering of wood beneath old canker.