1979

Hypoxylon canker of Aspen

R.L. Anderson

G.W. Anderson

Arthur L. Schipper, Jr

Forest Insect & Disease Leaflet 6

U.S. Department of Agriculture Forest Service

Hypoxylon Canker of Aspen

Ralph L. Anderson[1] and Gerald W. Anderson[2]
Revised by Arthur L. Schipper, Jr.[3]

[1]Plant Pathologist, North Central Forest Experiment Station, St. Paul, Minn., retired.
[2]Assistant Director, Northeastern Forest Experiment Station, Durham, N.H.
[3]Principal Plant Pathologist, North Central Forest Experiment Station, St. Paul, now with the WO staff.

Hypoxylon canker, caused by the fungus *Hypoxylon mammatum* (Wahl.) Mill. (formerly *H. pruinatum* (Klot.) Cke.), is one of the most important killing diseases of aspen in eastern North America. In Michigan, Minnesota, and Wisconsin, the total impact of Hypoxylon canker has been estimated to be 30 percent of the annual net growth of aspen; in 1972, trees worth more than $4 million were lost to the disease.

Range

The disease is present throughout the range of aspen in North America, except Alaska. It occurs in the Eastern United States and adjacent Canada west into British Columbia and south through the Rocky Mountains into Arizona. The disease has also been found on European aspen in Russia, Czechoslovakia, and France.

Hosts

The fungus is primarily a pathogen of quaking aspen, *Populus tremuloides* Michx. Infection levels in that species average 12 percent, but susceptibility of aspen to Hypoxylon canker varies by clone. Clones have been found where 100 percent of the trees were infected; other clones have been found with less than 1 percent of the trees infected. Bigtooth aspen, *P. grandidentata* Michx., is occasionally infected. Infection of balsam poplar, *P. balsamifera* L., is rare. European aspen (*P. tremula* L.), Chinese aspen (*P. adenopoda* Maxim.), and Bolleana poplar (*P. alba* var. *pyramidalis*) are additional hosts for the fungus. Hybrids of white poplars (section *Leuce*), where *P. tremuloides* is one of the parents, are also hosts of the fungus.

Symptoms

Young cankers first appear on aspen bark as slightly sunken, yellowish-orange areas with irregular margins (fig. 1). As infection progresses, the outer bark is raised in blisterlike patches and sloughs off, exposing the blackened crumbling cortex (fig. 2). Old cankers, which may be several feet long, are rough and blackened at the center and yellowish-orange at the newly invaded margins (fig. 3).

Figure 1 - *A young canker of* Hypoxy1on mammatum *on quaking aspen. Note the irregular margin and the yellowish-orange color of the canker.*

Figure 2 - *Blistered bark on a* H. mammatum canker *that is just over 1 year old. Beneath the blisters, which are loosened periderm, are the conidial pillars.*

Although callus may develop occasionally at the margin of a canker, the fungus usually invades new tissue so rapidly that callus has no time to form. Many trees infected on the lower bole are girdled and killed within 5 years. An infection on the upper bole may cause only part of the crown to die, after which a lower branch may become the growing tip of the tree, but the entire tree may then die from suppression. Some trees are so weakened by decay in the cankered zone that wind breaks the stems before girdling is complete (fig. 4).

Figure 4 - *Quaking aspen tree broken during a windstorm at the point of Hypoxylon canker infection.*

Figure 3 - *An older* H. mammatum *canker with an orange margin and darker central area that contains the conidial pillars and sexual stroma.*

The most reliable field symptom for identifying young Hypoxylon cankers is the laminated or mottled black and yellowish-white cortex, which can be exposed by cutting into young cankers or cutting near the margins of older cankers. Removing the bark exposes white mycelial fans in the cambial zone (fig. 5).

Figure 5 - *Mycelial fan beneath the bark on a quaking aspen tree infected with Hypoxylon canker.*

Description and Life History of the Fungus

How the Hypoxylon canker fungus infects aspen is not definitely known. Most cankers originate in the immediate area of a dead branch stub or break in the bark. Recent evidence suggests strongly that insects, especially gall insects such as *Saperda inornata* and the periodical cicada (*Magicicada septendecim* L.), are involved, but whether these insects actually inoculate aspen or merely provide wounds for infection courts has not been proven.

Both ascospores and conidia of the fungus have been tried as inoculum without reproducible success, although infection by mycelium is easy to obtain when the mycelium is placed in a wound through the

bark. Some reports have also suggested that other factors such as bark moisture levels and chemical or physical characteristics of the bark may be related to infectability of the host.

Five to 14 months after infection, asexual spores are produced under the blistered bark. These gray, powdery spores appear annually, from spring until early August, on small bristlelike conidial pillars under the blistered outer bark of the younger portions of the canker (fig. 6).

Figure 6 - *Removal of blistered bark reveals conidial pillars.*

About 3 years after infection, sexual spores are produced in fruiting bodies called perithecia on the older portions of the canker. The fruiting bodies are small crustlike growths ranging from a few to several millimeters in diameter (fig. 7). When young, they are covered with a grayish bloom, which soon disappears. As the fruiting bodies age, they turn black and appear carbonaceous. When the fruiting bodies get wet, the spores are forcibly discharged.

Sporulation can continue for several years after the host tree dies.

Figure 7 - *Sexual stroma (perithecia) of* H. mammatum. *These are formed in*

portions of Hypoxylon cankers that are between 2 and 3 years old.

Prevalence of the disease varies from one geographical area to another for reasons not known at present. For example, Hypoxylon canker is a major problem of aspen in the Lake States, but only a minor problem of aspen in the Rocky Mountains. It also varies within a single area, with high levels of infection occurring in occasional "wave years." Susceptibility of aspen to Hypoxylon is apparently greater on poor sites. Poor stocking, stand openings, and edges in aspen stands seem to create conditions favorable to the fungus. Cankers cause only about half as many stem losses in well-stocked stands as in poorly stocked ones, and about two to three times as many trees are infected near exposed stand edges as in well-stocked parts of the same stand.

Susceptibility of aspen to Hypoxylon canker does not seem to change as trees age. The disease does the most damage to juvenile trees because they are often cankered low on the bole where cankers usually are lethal. Large trees are attacked on the upper bole; the top of the tree is killed and a new leader often takes over. Appreciable losses from the disease can occur in older stands. Vigorous dominant trees and weak suppressed trees are both susceptible to infection.

Control

No direct control measures for Hypoxylon canker are known. The general distribution and prevalence of the fungus, and the lack of knowledge about how the disease is disseminated and how infection occurs, indicate that sanitation measures would be costly and of questionable value. The few attempts made at sanitation to eliminate the disease from a stand have been unsuccessful. While Hypoxylon canker cannot be prevented, certain silvicultural techniques can minimize its impact. All aspen stands should be checked routinely for the amount of disease present. If 15 to 25 percent of the aspen trees are infected with Hypoxylon canker, the stand should be harvested early and the site treated to encourage good aspen reproduction. If more than 25 percent of the trees are infected, the stand should be harvested as soon as the trees are large enough, and then the site should be converted to other species (susceptibility varies by clone and very susceptible clones should not be perpetuated). Lightly infected stands can be managed on rotations longer than 40 years.

Because thinning has been shown to favor Hypoxylon infection in some stands, thinning of aspen stands cannot be recommended as a management tool unless care is taken. If thinning is done, only fully stocked aspen stands with very low levels of the disease should be treated, and all infected trees should be removed, because they will die within 3 to 5 years anyway.

In established stands, there is little opportunity to eliminate exposed stand edges or to increase stocking to prevent infection. In future stands, however, losses would be reduced by obtaining full stocking. A uniform well-stocked stand is a general management objective for all forest types. Being able to reduce losses caused by Hypoxylon canker and *Phellinus tremulae* (Bond.) Bond & Boriss heart rot (also greater in poorly stocked stands) should be an added incentive for achieving this objective.

References

Anderson, G.W., and A.L. Schipper, Jr. 1975. Variation among isolates of H~poxyton mammatum. Eur. J. For. Pathol. 4:216-224.

Anderson, N.A., M.E. Ostry, and G.W. Anderson. 1976. Hypoxylon canker of aspen associated with *Saperda inornata* galls. U.S. Dep. Agric. For. Serv., Res. Note NC- 214, 3 p. U.S. Dep. Agric. For. Serv., North Cent. For. Exp. Stn., St. Paul, Minn.

Anderson, R.L. 1964. Hypoxylon canker impact on aspen. Phytopathology 54:253-257.

Berbee, J.G., and J.D. Rogers. 1964. Life cycle and host range of *Hypoxy1on pruinatum* and its pathogenesis on poplars. Phytopathology M:257-261.

Bier, J.E. 1940. Studies in forest pathology, III. Hypoxylon canker of poplar. Can. Dep. Agric. Tech. Bull. 27, 40 p.

Graham, S.A., and R.P. Harrison. 1954. Insect attacks and Hypoxylon infections in aspen. J. For. 52:741-743.

Revised October 1979
Formatted for the Internet September 1997

Return to the Forest and Tree Health Publications