Hypoxylon canker of aspen

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Photo G. Laflamme

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Cover:
On young trees in poorly stocked stands, the canker may be located at any height on the trunk. The cankered trunk often breaks before the tree is killed by the disease.

Interior design:
Lucil Lepage

Hypoxylon canker is common throughout the range of trembling aspen across Canada although its observed incidence in Newfoundland, the northern Rockies, and Alaska is lower. In the central northern and northeastern United States, it was detected more than 50 years ago.

In southern parts of the North American trembling aspen range, it has been estimated that 1 to 2 % of forest trees die annually from the hypoxylon canker, and that the canker reduces the net annual growth of remaining trees by 30 %. In poorly stocked and severely affected stands, annual tree mortality due to the canker may exceed growth.

In 1981 in Quebec, annual canker losses in the trembling and largetooth aspen were estimated at 1 260 000 m^3, representing 4 and 3 % of the merchantable volume for the two Populus species. In many aspen stands, foci with some 5 % of living stems affected can be observed.

Trees invaded and killed by the fungus rapidly lose their commercial value since the underlying wood is degraded by decay appearing a few years after the start of the infection.

CAUSAL AGENTS AND HOSTS

The pathogen that causes hypoxylon canker is an Ascomycetes, *Hypoxylon mammatum* (Wahl.) J.H. Miller, previously known as *H. pruinatum* (Klotz) Cke. The fungus was initially described in 1833, but the disease it causes was not detected until 1924 and the pathogenicity of *H. mammatum* was demonstrated only around 1940.

The principal host for hypoxylon canker is trembling aspen (*Populus tremuloides* Michx.), but largetooth aspen (*P. grandidentata* Michx.), European aspen (*P. tremula* L.), white poplar (*P. alba* L.), and possibly balsam poplar (*P. balsamifera* L.), may also be affected.

CONDUCIVE CONDITIONS

Many studies on environmental factors governing the disease have been published to date, but very few general concepts have emerged from the results. In

urban areas, the canker is rarely found on isolated trees. However, many small stands around urban developments are severely affected.

Dominant, vigorous trees and suppressed, weak trees are equally susceptible to infection. Although, in forests, no clear relationship between site index and frequency and severity of hypoxylon canker has yet been determined, the percentage of trees affected and the tree mortality in poor stands or near clearings in large stands may be two to three times greater than those in well-stocked stands. Some studies report a higher incidence of canker in pure trembling aspen stands, while others report the opposite.

The number of new infections varies from year to year but appears to increase in years of drought. Some forms of artificial inoculation have also been more successful under dry conditions. *Hypoxylon mammatum* penetrates the bark either through a wound, with or without the aid of insects, or directly through young, intact branches. Old wounds often seem more prone to infection. On small trees less than 10 cm in diameter, the fungus may invade the trunk at any level above the ground. Frequently, a canker on a branch less than 30 cm from the trunk will reach the trunk itself.

Bark age appears to be a determining factor for pathogen infection. Thus, a number of authors postulate that new invasions by the fungus are often localized in the upper parts of older trees, where bark is tender. Many attempts at inoculation through wounds at the base of the main

Photo A. Carpentier

Figure 1.
Rough, blackish surface of the bark following the eruption of conidial masses (asexual spores). The dark masses are easily identifiable upon lifting the outer layer of bark.

trunk have failed, probably because there are biochemical factors as well as injury involved in the initial infection.

SYMPTOMS AND DISEASE CYCLE

At the start of the infection, the bark takes on a yellowish-orange to reddish-brown coloration and begins to deform. The margin of the deformed area is irregular, often seeping brownish sap. All bark cells except those of the periderm are invaded. Subsequently, the cankered bark becomes rough and mottled grey and black (Figure 1), contrasting with the rest of the trunk, which has a smooth, greenish-grey surface.

H. mammatum toxin invades healthy tissues with such speed that a callus does not have time to form properly. If the canker invades far enough to affect most of the trunk's circumference, the part of the crown above the canker bears smaller leaves that change color earlier in the autumn.

A few months after the start of infection, masses of white or dark green mycelium form under the periderm near the canker margin in the bark and the cambium. Between 3 and 14 months later, the mycelium causes the periderm to lift and slough off, and a powdery mass of asexual spores forms on the mycelium. Asexual spores can be produced anew every year, between spring and the month of August, particularly on newly infected bark. In the autumn of the second or third year of infection, the beginnings of fruiting bodies formed in the perfect stage (Figure 2) become visible at the center (the oldest part) of the canker, which may by then be up to 1 m long. The crusted, grey or black fruiting bodies are a few millimetres across and contain perithecia, which, over two or three

Photo D. Lachance

Figure 2.
Greyish fruiting bodies, several millimetres in diameter, which repeatedly release ascospores for some three years after they form.

years, release ascospores (sexual spores) (Figure 3) each time they are moistened by rain or snow, even at temperatures slightly below 0°C. It is believed that ascospores, which generally require 24 to 48 hours in a moisture-laden atmosphere to germinate, are the primary means of transmission. Ascospore production can continue for several years after the host dies.

A cankered tree generally dies some three to five years after the start of infec-

tion. The wood of the trunk, weakened by decay or insects tunneling under the canker, frequently breaks at the level of the canker even before the tree dies.

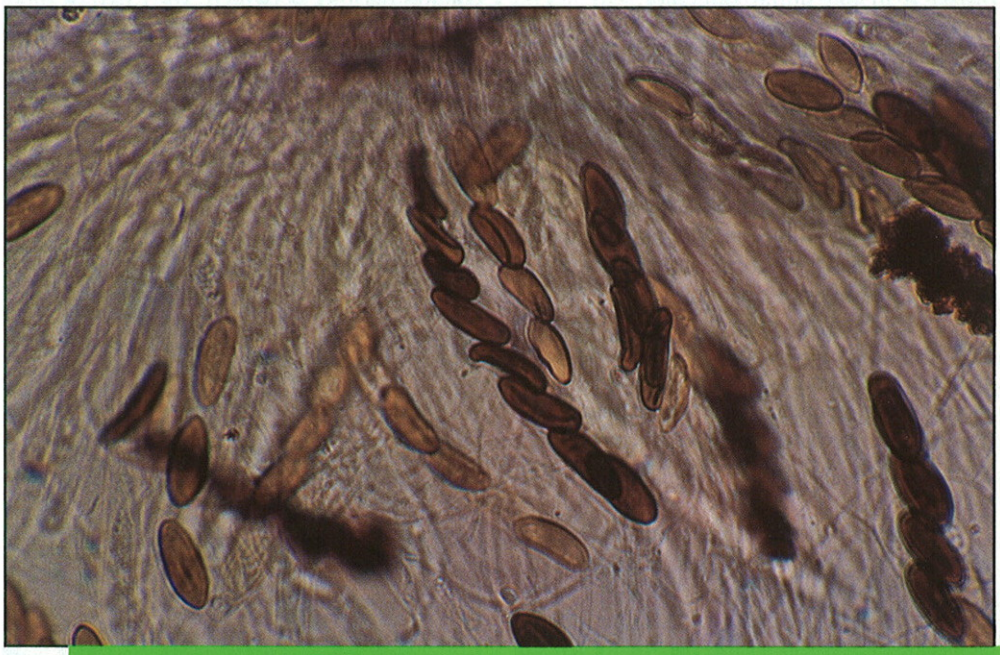

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Figure 3.
Asci are between 200 and 250 µm in length, while ascospores measure between 24 and 30 µm.

CONTROL

In the past, trembling aspen wood was not in sufficient demand to prompt intensive research into the control of the hypoxylon canker. Environmental and genetic factors relating to the progression of the disease are not yet understood well enough to warrant recommendations on the management of host stands. In general, we must try to keep pure aspen stands sufficiently well stocked. In certain areas, trembling aspen stands may consist of well-established clones developed after cutting or fire, while in other areas, stands may be of recent origin and thus less resistant to the canker. Infection rates in the two areas would probably vary, as would the means used to control the canker.

Forest sanitation measures would certainly reduce the chances of infection but could not guarantee the complete elimina-

tion of hypoxylon canker in aspen since we do not yet know how far viable ascospores can travel. Such measures should be implemented when trees are bare of leaves, making it easier to detect the canker, and should also be aimed at destroying affected parts of trees to reduce the likelihood of new infections. Note that for almost two years after infected trees are cut, cankers left on the ground can release almost as many ascospores as those on the trunks of living trees.

As part of an intensive aspen culture program, the selection and development of species and hybrids resistant to hypoxylon canker could prove to be a solution. We know that certain hybrids, including lines derived from *P. deltoides*, *P. balsamifera*, and *P. alba*, have already shown better resistance to the canker than lines crossed with *P. tremuloides*. Small-scale research on improving the genetic resistance of aspen against cankers has begun in laboratories in the northeastern United States, but many more studies are required before an intensive culture program can be developed. Chemical substances obtained from aspen bark appear to have an inhibitory effect on the germination of *H. mammatum* ascospores. Such chemicals could be considered in genetic improvement programs of the host. Moreover, we must bear in mind that, even on an improved host, *H. mammatum* is able to develop equally well in the cambium or bark, and that, as a result, wounds always constitute a significant means of entry for the fungus.

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