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Nectria Canker of Hardwoods

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Nectria canker is the most common canker of hardwood trees and one of the most serious diseases of our hardwood forests. Though the disease kills few trees, it has serious impact on the quantity and quality of lumber produced.

Nectria canker has been known in the United States since 1900, but was of little concern to forest pathologists until the beginning of intensive stand-improvement activities in the 1930's forced its recognition as a significant disease.

Several species of Nectria cause cankers. The most widespread and damaging is *Nectria galligena* Bres. Of lesser importance are *N. magnoliae* on the magnoliae and yellow-poplar, *N. coccinea* on sugar maple, and *N. coccinea* var. *faginata* on American beech—in association with a scale insect. *N. mammoidea* and *N. cinnabarina* may be serious in local situations.

Hosts and Distribution

No North American hardwood can be considered immune to *Nectria galligena*. This includes ornamental shrubs and fruit and shade trees, as well as forest trees. Tree species frequently attacked are red and sugar maples; black walnut; sweet, yellow, and paper birches; American beech; and largetooth aspen. White oak may be heavily infected in certain localities. Scarlet and black oaks are often cankered. But hickory, ash, and elm are relatively free from the disease.

The fungus-causing cankers on one host species (except yellow-poplar, magnolia, and sassafras) may readily infect other tree species. The degree of cankering seems to be related more to variations in tree vigor than to host preferences.

Nectria canker occurs throughout the Temperate Zones of the world, and is particularly common in northern Europe and North America. Distribution appears to be influenced by local climatic factors. Heavy buildup of Nectria canker is favored at high altitudes by exposed slopes with shallow and infertile soils, and at lower altitudes by cold pockets with poorly drained soils. Diseased trees on good sites tend to heal their Nectria cankers, but on poorer sites the rate of canker enlargement can surpass the healing rate.

Damage

Although Nectria often does not kill, a canker may eventually girdle a tree and cause death. Species of birch and black walnut are particularly liable to death through girdling.

The greatest loss results from the large number of defective trees that live on for years, occupying growing space in the stand, which otherwise could be producing fast-growing merchantable trees. Distorted form (fig. 1) and stunted growth result

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Figure 1.—Nectria cankers on sweet birch. Most such damage results from infection incurred early in the life of the tree.

from cankering, and such trees have little current value and small chance of becoming productive. However, some actively growing young trees are able to heal over superficial Nectria cankers and obtain fair form and size.

Incidence of cankering and re-

sultant damage are greater where heavy snow and ice storms are common. In such areas cankers may occur on 60 to 70 percent of the trees in young hardwood stands. Cankered trees may easily be broken by wind or ice storms.

Since the majority of infections arise on trees between 2 and 20 years old, it is usually on the lowest 10-foot part of trees that the oldest, largest, and most damaging cankers are found. The loss from cull depends largely on the number and location of these cankers and the intended use for the logs.

Though decay organisms sometimes enter through Nectria cankers and further lower the value of infected trees, the wood behind them is typically sound. There is even some evidence that under certain conditions Nectria infection may prevent establishment of decay fungi.

Symptoms

Well-developed Nectria cankers are the most distinctive and easily recognized of cankers. Yet in early stages they are extremely difficult and often impossible to find. The first visible symptom is usually a dark red or blackened, water-soaked appearance of the bark adjacent to the opening through which infection occurred. Later a depression and further discoloration of the bark at the canker's center occur, soon followed by fissuring of bark at the margins (fig. 2).

The characteristic target appearance of an older canker (fig. 3) results from a continued alternation between killing of bark tissues by the fungus in the autumn and winter months and growth of host callus material in spring and early summer. Cankers may extend vertically

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Figure 2.—A young Nectria canker, showing development of cracks and bark depression. This stage occurs prior to formation of callus by the host.

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Figure 3.—Target or callus-roll type of canker on sugar maple, typical of older Nectria cankers on many host species.

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Figure 4.—Sugar maple with a closed type of canker. Many cankers are not so evident as this and are difficult or even impossible to find.

for several feet, but laterally their size is limited by the diameter of the host tree.

The cankers are usually open (fig. 3), but dead bark may remain in place to produce a covered canker (fig. 4). In general, some surface disturbance or irregularity in the bark usually exists to give a clue to the presence of the canker beneath. Sometimes the only indication is a bulge in the stem; then there is no way, short of cutting into the tree, to identify the trouble.

Although the red fruiting bodies of the Nectria fungus may develop on either open or closed cankers, their small size makes them of little use in scouting for the disease.

Life History

Nectria spores gain entrance into trees through openings in the corky outer bark that expose the live inner bark or cambium. Entry is possible through natural openings, such as lenticels and leaf scars, or through wounds caused by any of a great number of agencies. In forest trees the most common openings are cracks at the bases of twigs and branches caused by loads of snow or ice (fig. 5). Other causes of

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Figure 5.—**Entry way for Nectria. Such cracks are caused by snow or ice during the months when spores are produced in greatest number.**

wounds susceptible to Nectria are insects, frost, hail, other fungus cankers, crotch cracks, sunscald, dead buds, and natural and artificial pruning.

If moisture and temperature conditions are favorable, spores may germinate in a fresh wound, and the fungus may become established in the tree within 3 to 4 hours. A small rootlike germ tube arises from the fungus spore, then branches out through the living bark, killing it and the cambium beneath. Surface evidence of the death of host cells and tissues may become evident within days, or such infections may remain latent for 2 or 3 years, depending upon the pathogenicity of the particular strain of fungus and the inherent susceptibility of the host tree.

As the canker develops, dying bark cells create a water-soaked area that enlarges as additional cells are killed. The bark at the center of infection may become loose and slough off to expose the wood beneath, or it may be held in place by the adjoining live bark at the periphery of the canker. Each year, while the tree is dormant, the fungus grows outward by as much as ¼ to 1½ inches.

As tree growth resumes in the spring, the advance of the Nectria fungus is delayed or stopped by the production of host callus tissue. If callus formation is vigorous and prolonged, the canker may be partially or entirely overgrown; but more commonly the fungus renews its advance in the next dormant season. After a few years, a succession of surface convolutions are produced, giving rise to the term "target canker."

In the autumn rainy season the

fungus may begin to fruit. Minute creamy-white tufts of fungus hyphae protrude through cracks or lenticels in the bark along the outer callus ridges. These hyphae give rise to asexual spores, which can cause new infections. They may be disseminated by wind or rain.

A second fruiting stage of *Nectria galligena* develops as minute, flask-shaped, red fruiting bodies, each about the size of a pinhead (fig. 6). These may occur singly or in groups. They are usually found on the dead callus tissue or, more rarely, on the exposed wood at the center of the canker. At maturity these perithecia become dark red in color and produce two-celled ascospores, which are ejected from the perithecia and dispersed by air currents to fresh wounds to start a new disease cycle.

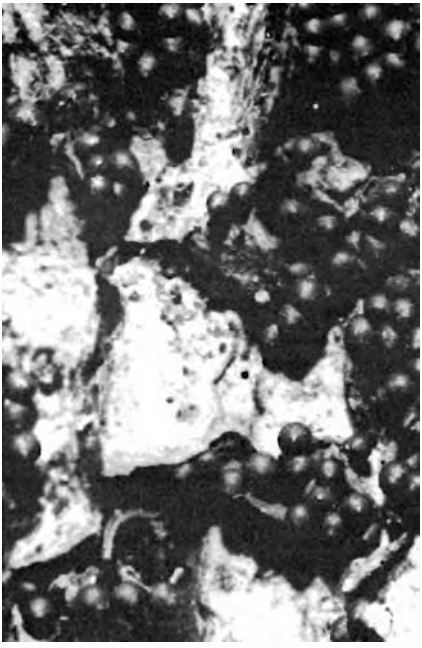

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***Figure 6.*—Perithecia of *Nectria galligena*. From these pinhead-sized bodies thousands of spores are released to be washed or blown to new host trees.**

Minimizing Losses

Part of the solution to the problem of Nectria canker rests in recognizing areas that are unsuited for growing quality timber and utilizing such areas for other purposes. On the more highly productive forest lands, intensive management—including pruning of very young stands—should further help to limit Nectria infections.

The removal of cankered trees should be worked in with thinning and general stand-improvement operations, but with no goal of eradication to prevent subsequent infection. Removal of the badly infected trees will create more growing space for the healthier ones and might assist trees with lesser infections to overgrow cankers. Any forest practice that will increase tree vigor ought to help limit Nectria activity.

It is impossible, even with trained crews, to completely eradicate Nectria-cankered trees from forest stands because of the inconspicuous and latent nature of many infections. This fact, coupled with prolific spore production and favorable conditions for infection over an extremely long period, renders control in high-hazard areas a poor goal for the forest manager. However, a sharp drop in the number of new infections occurs as a stand increases in age. With the exception of yellow birch and sweet birch, which are susceptible to stem cankering in middle age, most 20- to 25-year-old Nectria-free trees selected as final crop trees will remain free from damaging infection.

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