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# Interactions of Insects, Woodpeckers, and Hypoxylon Canker on Aspen

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Cover: *An illustration of the ecological participants in hypoxylon canker on aspen.*

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# Interactions of Insects, Woodpeckers, and Hypoxylon Canker On Aspen

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Trembling aspen (*Populus tremuloides*) is the most important pulpwood species in the Lake States, accounting for 47 percent of the total roundwood production in 1996 (Piva 1997). Aspen is damaged by many insect pests and diseases, but, hypoxylon canker (fig. 1) is the major cause of aspen mortality in the Lake States (Anderson 1964). Conditions that predispose aspen to infection by the causal agent *Entoleuca mammata* (=*Hypoxylon mammatum*) are not completely understood. Hypoxylon canker of aspen is the result of a complex web of interactions among an extremely variable host and pathogen, a number of wounding agents, and various environmental variables (Anderson and Martin 1981, Manion and Griffin 1986, Ostry and Anderson 1990). Since the first research paper was published on hypoxylon canker (Povah 1924), research has provided insights that partially explain the infection process (French and Oshima 1959), the development of the canker on individual trees (Valentine *et al.* 1976), and the distribution of the disease in nature (Anderson 1964, Froyd and French 1967, French and Hart 1978, Copony and Barnes 1974). However, many factors in this host-pathogen system remain unknown, limiting our ability to minimize the damage caused by this disease.

Figure 1.—*Aspen stems killed by hypoxylon canker. Stem breakage at the canker is common as the fungus decays cellulose and weakens infected stems.*

Although there have been reports of aspen infected by *E. mammata* without evidence of wounds (Manion 1975, Chapela 1989, Anderson and French 1972), several studies have implicated insect wounds as infection sites. Our research suggests that *E. mammata* requires a specific type of wound to infect trees. The wound must be deep into the xylem tissue so that the phytotoxic compounds (French and Oshima 1959, Hubbes 1962) in the green layer of the bark cannot inhibit the fungus. After infection in the xylem, the fungus produces a toxin that allows it to expand into the bark tissue (Schipper 1978). A wound into the xylem tissue favors the pathogen that produces cellulose-degrading enzymes. Many diseased tree stems eventually break because the fungus has decayed the wood behind the canker.

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Several investigators have found hypoxylon cankers that originated in galls made by the poplar-gall saperda (*Saperda inornata*) in Michigan (Graham *et al.* 1963, Nord and Knight 1972), Minnesota (Anderson *et al.* 1979), and New York (Manion 1975). Larvae of *S. inornata* are winter food for the downy woodpecker (*Picoides pubescens*). When extracting larvae, the woodpeckers make large wounds in the galls that are frequently not closed by callus growth and thus the likelihood of infection by *E. mammata* is increased (Ostry *et al.* 1982).

Oviposition wounds made by the poplar borer (*Saperda calcarata*) (Gruenhagen 1945, Graham and Harrison 1954, Anderson and Martin 1981), the periodical cicada (*Magicicada septendecim*) and dogday cicada (*Tibicen linnei*) (Ostry and Anderson 1983), the aspen branch borer (*Oberea schaumii*) (Nord and Knight 1972, Bier 1940), and the aspen treehopper (*Telamona tremulata*) (Ostry and Anderson 1986) have also been associated with infection of aspen by *E. mammata*. The wounds made by these insects are deep into xylem tissue of branches or the main stems of small trees.

In this paper, we summarize the history of hypoxylon canker incidence in two aspen plantations: Minnesota (1968-1995) and Wisconsin (1971-1995). The objective of the study was to examine hypoxylon canker in terms of host resistance, spatial and temporal distribution, and the role of wounds in the infection process.

## MATERIALS AND METHODS

Two plantations of aspen derived from controlled crosses were established near Rosemount, Minnesota, and Langlade, Wisconsin. Parent trees were selected throughout the range of aspen in Minnesota from clones arbitrarily rated as resistant and susceptible to hypoxylon canker based on the prevalence of cankers among ramets of the clone when collections were made. The study design was described in an earlier paper (Anderson *et al.* 1979). The Minnesota plantation was established in 1968 and included 526 trees from 38 crosses. The Wisconsin plantation was established in 1971 with 290 trees from the same 38 crosses.

Detailed observations of plantation trees were made periodically through 1995. Data collected included canker position and origin, canker expansion, type of wounding agent, tree response to wounding and fungal colonization, date of sporulation, and time needed for cankers to girdle and kill infected trees.

## RESULTS

### Minnesota Plantation

Oviposition wounds made by the poplar-gall Saperda on branches ranging from 1 to 2 cm in diameter (fig. 2) first occurred in 1972 and the resulting galls had caused serious damage to trees by 1976 (fig. 3). The insect population remained high for 8 years and then collapsed in 1984-1986 presumably due to record cold winter temperatures in 1983 and 1985.

A total of 1,660 hypoxylon cankers were recorded for detailed study from 1968 through 1995 (table 1). Eighty-five percent (1,409) of these cankers originated in *S. inornata* galls.

Figure 2.—*Oviposition wounds made by the poplar-gall Saperda*, Saperda inornata.

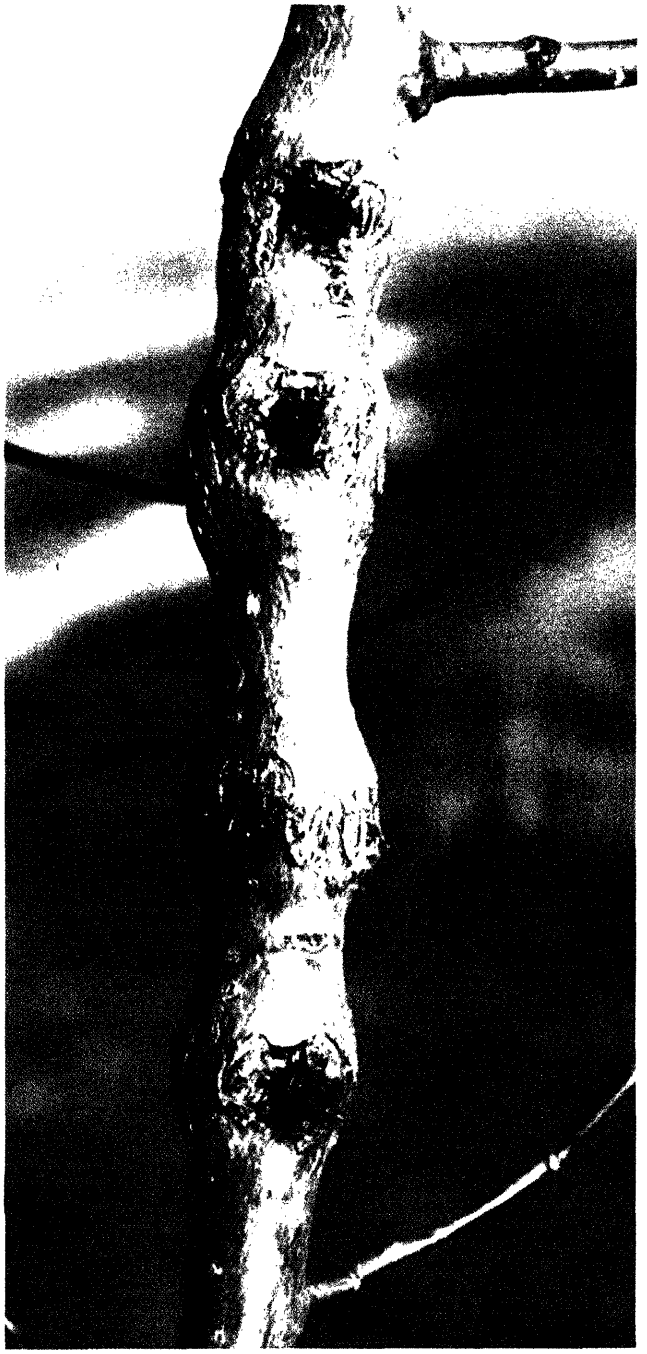

Figure 3.—*Galls of the poplar-gall Saperda.*

Of the galls colonized by *E. mammata*, over 80 percent had probing and extraction wounds made by downy woodpeckers (fig. 4).

Table 1.—*Summary of hypoxylon canker on aspen 1968-1995, Rosemount, MN*

| Canker origins | Number |
|---|---|
| Total hypoxylon cankers examined | 1,660 |
| Total hypoxylon cankers associated with *Saperda inornata* galls | 1,409 |
| Cankers associated with *S. inornata* galls on branches | 1,290 |
| Cankers associated with *S. inornata* galls on branches that expanded into the main stem | 151 |
| Cankers associated with *S. inornata* galls on the main stem | 119 |
| Cankers associated with *Telemona tremulata* oviposition wounds | 84 |
| Main stem cankers associated with small, unwounded branches | 41 |
| Cankers associated with pruning wounds | 43 |
| Cankers associated with wounds of unknown origin | 82 |

Of all the cankers observed, 1,290 (91 percent) began in galls on branches (fig. 5). Of these, 151 (12 percent) expanded into the main stem (fig. 6), moving an average of 17 cm (0.6-120.0). The average time needed for a branch canker to expand into the main stem was 21 months, and once in the main stem the time needed to girdle and kill the pole-size trees averaged 52 months (fig. 7). As of 1995, a total of 323 trees (61 percent) were killed by hypoxylon canker. Cankers on small branches did not progress after a branch died. Decreased bark moisture and invasion by competing saprophytic fungi commonly prevented *E. mammata* from moving into the main stem.

There were 119 hypoxylon cankers that originated in Saperda galls on the main stem of small trees (fig. 8). These cankers eventually girdled and killed all of the infected trees. An additional 41 stem cankers originated at the base of small branches with no visible injuries associated with them, similar to those reported by Manion (1975), and 84 branch cankers originated in oviposition wounds made by the aspen treehopper on branches approximately 0.5 cm in diameter (figs. 9 and 10).

Figure 4.—*Wounds made by a woodpecker in extracting larvae from a gall.*

Figure 5.—*Aspen branch infected by* E. mammata *through a Saperda gall.*

Figure 6.—*A hypoxylon canker in the main stem of an aspen which originated on the branch at a gall.*

Figure 7.—*Same tree as in figure 6 that has been nearly girdled by the fungus. Note signs of woodpecker probing at the canker margins.*

Figure 8.—*A small tree infected by* E. mammata *through a gall directly on the main stem.*

Figure 9.—*Branch canker originating in a treehopper oviposition wound. Note the dead leaves of the branch referred to as "flags" that are common in the upper crowns of affected trees.*

Cankers also developed in wounds other than those caused by insects. In 1988, 59 cankers were found associated with 1,450 pruning wounds made in 1985. All of these cankers were associated with wounds resulting from cuts made flush with the main stem that removed the branch collar. Cankers did not develop on 106 pruning wounds that did not injure the branch collar. In addition, 46 cankers originated in wounds of unknown origin that exposed xylem tissue. In June 1995, a total of 779 wounds on stems and branches caused by hail were tagged on 63 trees. When these wounds were examined in November 1997, no cankers had developed at these sites.

Figure 10.—*Branch canker associated with a treehopper wound that is producing pegs and conidia under the blistered bark.*

We noted striking differences among the progeny of the various crosses in two traits: branch diameter and wound callus production (fig. 11), which influenced disease severity after infection by *E. mammata*. Average branch diameter varied considerably among trees of different crosses. Progeny with large diameter branches tended to have more total stem cankers as well as more stem cankers that originated farther out on branches than progeny with smaller diameter branches. Small diameter branches that became infected were rapidly girdled and killed, preventing the fungus from expanding into the main stem. Prolific callus production was characteristic of progeny with a low incidence of canker, or trees that limited canker expansion, resulting in less tree mortality.

### Wisconsin Plantation

Outbreaks of the periodical cicada occurred in June 1976 and June 1993. Multiple oviposition wounds on branches ranging from 1 to 4 cm in diameter were common on trees during both of these outbreaks (fig. 12). Dogday cicada were present in low numbers throughout the study period, causing occasional minor damage to tree branches within the plantation.

Figure 11.—*Canker inactivated by callus, commonly produced by trees in resistant clones.*

Figure 12.—*Oviposition wound made by cicada.*

Four hypoxylon cankers in 1976 and three in 1977 were found associated with dogday cicada oviposition wounds. In 1978, 2 years after the first outbreak of the periodical cicada, 121 cankers were found associated with cicada oviposition wounds (figs. 13 and14). Over the next 3 years, an additional 240 hypoxylon cankers originated in cicada wounds. In addition, several cankers were found associated with Saperda galls and oviposition wounds made by treehoppers.

Oviposition injuries made by cicada were on 1- and 2-year-old branch wood. The first symptoms of hypoxylon cankers in the wounds made in 1976 were apparent in the summer of 1978, and new cankers developed in these wounds through 1980. Fruitbodies (perithecia) of *E. mammata* were noted in the summer of 1980 on cankers first observed in 1978. In 1995, 2 years after the second outbreak, the number of cankers sharply increased, similar to the trend noted after the 1976 outbreak.

A total of 712 hypoxylon cankers were recorded in the plantation trees by 1995, with 482 (68 percent) originating in cicada oviposition wounds (table 2). Of all the cankers observed in this plantation, 599 originated on

Figure 13.—*A 2-year-old cicada wound with no symptoms of infection by* E. mammata.

Figure 14.—*Same wound as in figure 13 with bark removed to show the extensive colonization by the fungus.*

Table 2.—*Summary of hypoxylon canker on aspen 1971-1995, Langlade, WI*

| Canker origins | Number |
|---|---|
| Total hypoxylon cankers examined | 712 |
| Cankers associated with *Saperda inornata* galls | 119 |
| Cankers associated with cicada oviposition wounds | 482 |
| Cankers associated with *Telemona tremulata* oviposition wounds | 5 |
| Cankers associated with wounds of unknown origin | 106 |

branches, and the remainder were on the main stem when first noted. A total of 143 main stem cankers resulted from cankers that originated on branches and expanded into the main stem. Expansion of cankers from a cicada oviposition wound on a branch to the main stem averaged 10.4 cm (0.5-62.0). Of the hypoxylon cankers originating in cicada and Saperda oviposition wounds on branches, 30 and 27 percent of them, respectively, expanded into the main stem. A total of 183 (63 percent) of the trees were killed by hypoxylon canker as of 1995.

## DISCUSSION

The outbreak of Saperda and cicada in two aspen plantations gave us an opportunity to study in detail the interactions of *E. mammata* and its host over a period of nearly 30 years. During this time, several key factors influencing this disease in the Lake States were clarified.

It has been suggested for many years that *E. mammata* is a wound parasite and that various types of insect wounds on aspen were probably common entries for the fungus. Results of our study confirmed the importance of insect wounds and provided evidence that clearly showed that oviposition wounds made on aspen branches by four common insect species are frequently points of entry for the fungus. Potentially lethal stem cankers developed when the fungus successfully grew from the branch into the main stem. We previously demonstrated that ascospores (fig. 15) can infect aspen through Saperda galls (Ostry and Anderson 1995).

The distribution of hypoxylon canker has been the subject of many studies. Our results support the previously reported spatial distribution of the disease within an individual tree, within a stand, and perhaps, within the region. In addition, the temporal prevalence of the disease may be influenced by fluctuating insect populations.

Figure 15.—*Microscopic view of ascospores in the fruitbodies (perithecia) of the fungus.*

It has been reported that hypoxylon cankers are found increasingly higher on aspen stems as they mature (Bier 1940). This pattern of infection was clearly evident in both plantations in our study. Insects of each species studied oviposited on only current-season or 1- to 2-year-old branch wood. Thus, each year the majority of new oviposition wounds and infections by *E. mammata* were higher in the crowns and further away from the stem on branches of trees than in previous years. This pattern has also been observed in natural aspen clones (Falk *et al.* 1989). Sapling and pole-size aspen are more vulnerable to insect oviposition injury and infection by *E. mammata* than older aspen where cankers develop higher in the crown and are less likely to kill the entire tree. Treehoppers oviposit on small twigs, and infection through these wounds seldom results in a canker reaching the main stem.

Numerous investigators have reported that there is an inverse relationship between the incidence of hypoxylon canker and stand density (Anderson 1964, Manion and Griffin 1986). The insects we have studied are attracted to open stands. In all likelihood, the high incidence of oviposition injury by the poplar-gall Saperda in our plantations was probably influenced by the wide (3 x 3 m) spacing of the trees. In a study in northern Michigan, rarely was there more than one Saperda gall per stem in a well-stocked aspen stand (Nord *et al.* 1972). The incidence of hypoxylon canker was greater on unpruned aspen than on aspen with their lower branches removed (Ostry and Anderson 1979). Similarily, self-pruning by aspen in well-stocked stands reduces potential insect oviposition sites, and therefore may also reduce sites for infection by *E. mammata* (fig. 16).

The incidence of hypoxylon canker has been reported to fluctuate (Schmiege and Anderson 1960) and has been reported to be greater in some parts of Wisconsin and the Upper Peninsula of Michigan than in northern Minnesota (Anderson 1964). The extremely cold winters of 1983 and 1985 preceded reductions in *S. inornata* populations and, indirectly, the incidence of hypoxylon canker in the Minnesota plantation in subsequent years. *Saperda inornata* has a 1- to 3-year life cycle depending on temperature (Nord *et al.* 1972) and could indirectly affect the incidence of hypoxylon canker by influencing the number of new oviposition wounds within a stand. The impact of regional temperatures on insect populations may be a factor in the observed differences in the incidence of canker within the Lake States. In addition, outbreaks of insects such as the periodical cicada could influence canker incidence. Two years after each cicada outbreak in the Wisconsin plantation, the incidence of hypoxylon canker dramatically increased, and remained high for another 3 years.

The natural rate of infection of aspen through poplar saperda galls in Minnesota and cicada oviposition wounds in Wisconsin during this study was 1.6 and 8.1 percent, respectively. Cicada generally oviposit on larger branches and cause greater injury than Saperda, accounting for the greater likelihood of infection by *E. mammata* through these wounds. It is possible that even with a 17-year life cycle, cicadas could damage many aspen stands three times during a rotation. In the Wisconsin plantation, we observed the cicada brood emerge twice and we documented the subsequent increase in the prevalence of hypoxylon canker after each emergence.

Figure 16.--*A well-stocked young sucker stand exhibiting self-pruning of shaded branches. The dead, yellow-orange branches are often invaded by other fungi.*

Woodpeckers were frequently observed in both plantations foraging for various insect larvae around the margins of hypoxylon cankers (fig. 7) and then immediately flying to galls where they extracted Saperda larvae (fig. 4). Woodpecker damage is common on cankers and galls in natural aspen stands as well. It is likely that the birds increase the chance for infection by making additional deep wounds on branches near galls. Woodpeckers may also vector the fungus mycelium and spores as they forage on cankers and galls. We have inoculated aspen through woodpecker wounds on galls with small pieces of infected or healthy aspen bark. Cankers developed on nearly 6 percent of the branches where the infected bark was used.

Aspen in families with canker resistance exhibited the ability to respond to wounds by producing extensive callus limiting infection (figs. 17 and 18). Alternatively, trees in some families often had a high incidence of canker, but canker elongation was limited by callus and many cankers became inactive after several years (fig. 11). An aspen clone that exhibited canker resistance in the field under natural conditions and in both the Minnesota and Wisconsin plantations (Anderson and Ostry 1993) also exhibited resistance in greenhouse tests designed to examine early wound responses (Bucciarelli 1996).

Early branch death was also a trait of some progeny that increased resistance by preventing branch cankers from expanding into the main stem. Colonization of branches by saprophytic fungi and self-pruning was more evident on progeny with smaller diameter branches than on those with larger diameter branches. Canker elongation, expansion into the main stem, and subsequent tree death varied widely among progeny. These traits may be useful indicators of canker tolerance when selecting superior aspen. However, examination of aspen clones for canker prevalence at one point in time may be misleading.

Disease resistant or tolerant clones may have many diseased ramets at any given time compared to members of a clone that are girdled and killed quickly. It is important to distinguish disease prevalence (number of diseased trees based on trees present) vs. disease incidence (number of diseased trees based on all living and dead trees over time). The prevalence of hypoxylon canker can dramatically change over time among clones (Ostry and Anderson, in prep.).

Callus formation, branch death, and canker elongation have been studied in relation to host resistance and pathogen virulence (Valentine *et al.* 1976, French and Hart 1978, Griffin *et al.* 1984). *E. mammata* isolates vary in

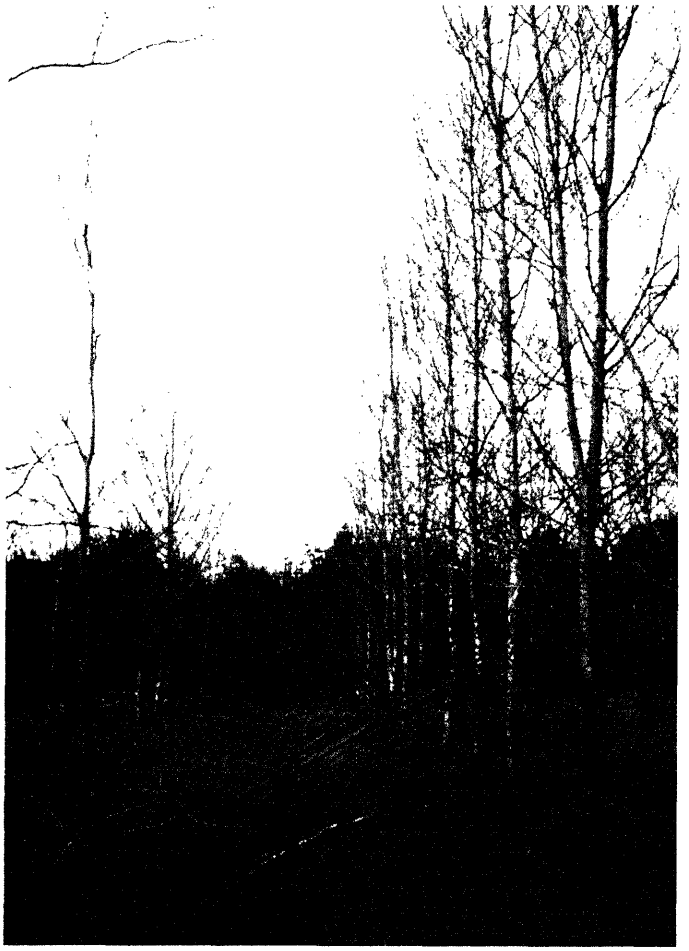

Figure 17.—*Two aspen families at the Wisconsin planting expressing susceptibility to hypoxylon canker and high mortality (left) and resistance (right).*

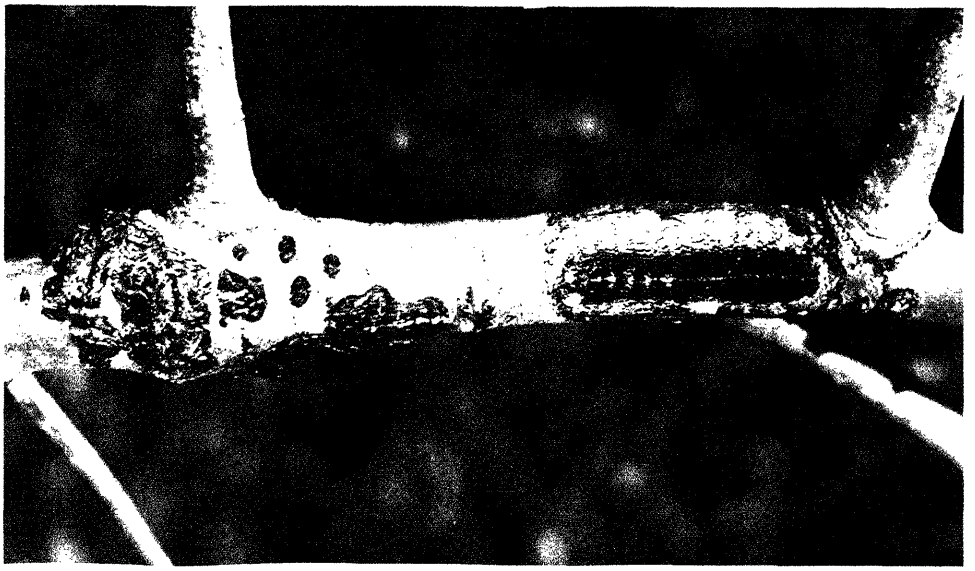

Figure 18.—*Example of an aspen expressing the ability to close oviposition wounds made by cicada (right) and Saperda (left) and probing wounds made by woodpeckers.*

virulence, making it difficult to separate host resistance from pathogen virulence. In all of the above studies, trees were wounded and inoculated with mycelium of the fungus. Any mechanism of resistance relating to early wound response, response to insect wounding, or early pathogen restriction would probably not be detected when a large amount of toxin-producing mycelium is artificially introduced into a wound.

This long-term study provided results from a system similar to that found in natural stands. Trees were exposed to local inoculum, and natural wounding agents for nearly 30 years. The long incubation period before visible symptoms developed in wounds (fig. 19), the presence of viable mycelium deep in non-symptomatic wounds (fig. 20), and the number of small, restricted cankers indicate that resistance mechanisms most likely are activated early at infection sites to limit canker development. Features of the response zone in wound-inoculated aspen have been used to distinguish resistant from susceptible genotypes (Bucciarelli 1996, Enebak *et al.* 1997).

In addition, many infected trees lived for several years after a stem canker became established, an indication that host resistance and pathogen aggressiveness are equally important once infection takes place.

Several traits that increased resistance to infection and canker elongation were identified. Traits such as early self-pruning, complete wound closure, and prolific callus production indicated high levels of canker resistance, and these characteristics could be used as selection criteria in aspen improvement programs. In addition, there may be aspen genotypes that have resistance to insect attack that would indirectly reduce disease incidence by reducing potential infection sites for the fungus.

High stand density increases self-pruning and reduces the incidence of insect oviposition injury, which also reduces the likelihood of branch cankers becoming lethal stem cankers. Silvicultural practices to regenerate and maintain stands at high stocking levels will increase self-pruning of lower branches and reduce the severity of insect damage that may

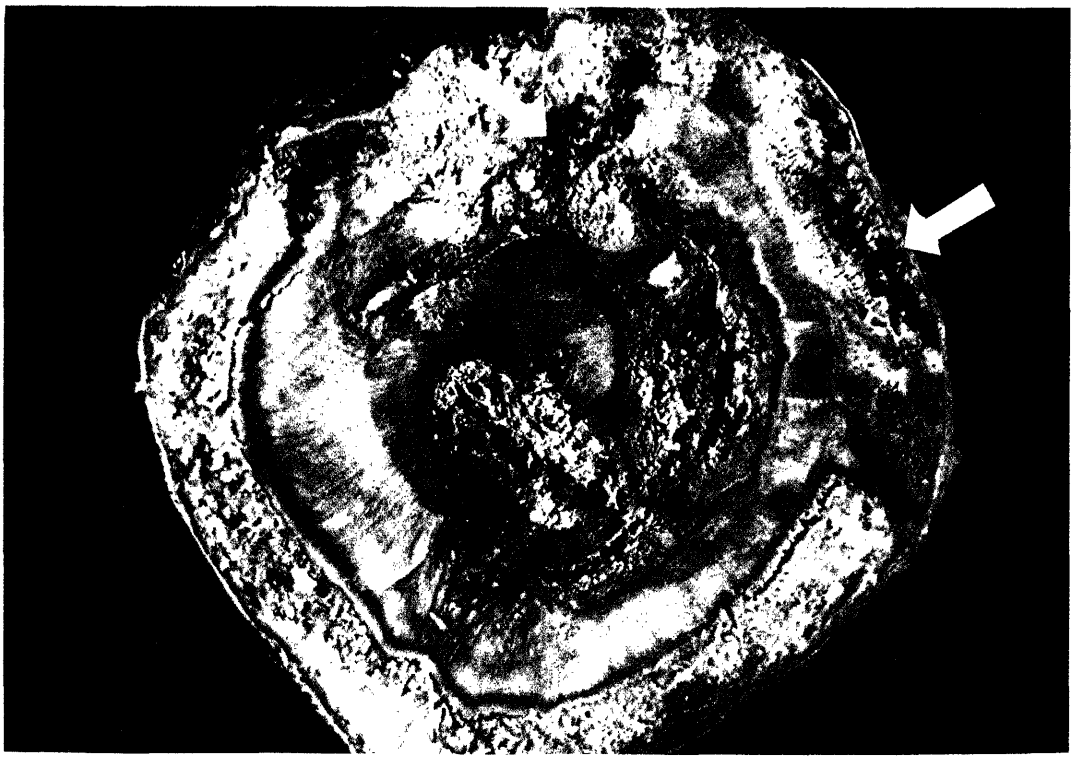

Figure 19.—*Cross-section of a Saperda gall on a branch not expressing external symptoms of infection illustrating the presence of* E. mammata (arrows). *Note the decayed xylem and phloem tissue.*

Figure 20.—*A scanning electron micrograph of the sheets of hyphae of* E. mammata *in the cambial area of an infected branch gall (1250X).*

indirectly lower the incidence of canker within stands. In establishing plantations, tree spacing should be considered because insect wounds and potential cankers originating in them can be far more damaging to young trees.

## SUMMARY

Our studies of hypoxylon canker on aspen in the Lake States suggest a sequence of events leading up to infection and disease expression involving the interactions of the host, various insects, woodpeckers, and the fungus (see cover). Ascospores of the fungus, or pieces of infected bark and wood, are deposited into insect oviposition wounds and natural openings or wounds made by woodpeckers on Saperda galls by rainsplash or foraging woodpeckers. The fungus grows by decaying the woody xylem from which it obtains nutrients, forming sheets of hyphae (fig. 20) that eventually grow into the phloem. Once in the bark, the fungus produces enzymes that detoxify the fungistatic compounds in the green bark layer, and the characteristic canker becomes visible.

Aspen has many canker resistance mechanisms, and their effectiveness in reducing infection and disease development varies widely among clones. Early responses to wounds, callus production, chemical defenses, self-pruning, and various branch characteristics such as diameter that can lead to early death when infected have been shown to vary among clones, tree stems as they mature, and during stand development. These factors can be effective in either preventing infection or reducing mortality of trees within a clone by restricting the development of potentially lethal cankers. Stand density, which has been described as a form of spatial resistance (McNabb *et al.* 1982), can be an overriding force for aspen survival. If young aspen stands are adequately stocked, then even susceptible clones will have fewer insect wounds and potential infection sites.

Numerous temporal and site factors have also been shown to influence this remarkably complex disease, contributing to the fact that even after more than 70 years of investigations, we still do not completely understand one of the most common diseases encountered in aspen throughout most of its range in North America.

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KEY WORDS: *Populus tremuloides*, *Entoleuca mammata*, disease resistance.