20 Hypoxylon Canker

Michael E. Ostry*
USDA Forest Service, Northern Research Station,
St Paul, Minnesota, USA

20.1 Pathogen, Significance and Distribution

Entoleuca mammata (Wahlenb.) J.D. Rogers & Y.M. Ju (syn. *Hypoxylon mammatum* (Wahlenb.) P. Karst.) causes the most damaging canker disease of quaking aspen (*Populus tremuloides* Michx.) – Hypoxylon canker – in many areas of North America. A study by Anderson (1964) in the Lake States (Michigan, Minnesota and Wisconsin) estimated that Hypoxylon canker killed 1–2% of the aspen volume each year, which is equivalent to 31% of the net annual growth. The estimated yearly volume loss in Ontario (Canada) was 2 million m^3 (Pitt *et al.*, 2001). Bigtooth aspen (*P. grandidentata* Michx.) is occasionally infected but the disease is not nearly as damaging to this species as it is to quaking aspen.

Quaking and bigtooth aspens are the most abundant commercially and ecologically important species in the region. Aspens are critical to numerous wildlife species for food and habitat, and contribute substantially to many local and regional economies. It was estimated in 1971 that tree mortality attributed to Hypoxylon canker in the Lake States was equal to a loss of 4.4 million US$ a year at harvest (Marty, 1972). Since that time, the value of aspen for pulp and paper and solid wood products has significantly increased, with a corresponding increase in its utilization by the forest industry.

Hypoxylon canker is widely distributed in the north-eastern and Lake States regions of the USA but less common in the western USA (Hinds, 1985) and, for reasons unknown, it is absent from Alaska (Hinds and Laurent, 1978). The disease is widely distributed in Canada (Bier, 1940). In Europe, it has been reported affecting European aspen (*P. tremula* L.) from Finland, Germany and Sweden (Miller, 1961), and from France (Pinon, 1979). Other hosts in Europe include white poplar (*P. alba* L.), black cottonwood (*P. balsamifera* L. subsp. *trichocarpa* (Torr. & A. Gray ex Hook.) Brayshaw) and hybrid aspen (*P. tremula* × *P. tremuloides*) (Kasanen *et al.*, 2004). The fungus has also been collected in Italy and Switzerland (Kasanen *et al.*, 2004).

Various hybrid *Populus* spp. are occasionally damaged by *E. mammata*. Hypoxylon cankers were found on plantation-grown trees of *P. nigra* L. var. *betulifolia* × *P. nigra* 'Volga', *P. nigra* var. *betulifolia* × *P. balsamifera* subsp. *trichocarpa*, *P. maximowiczii* A. Henry × (*P.* ×*berolinensis* C. Koch), *P. balsamifera* subsp. *balsamifera* × (*P.* ×*berolinensis*), and *P. deltoides* Bartram ex Marsh. × *P. nigra* 'Incrassata' (Ostry and McNabb, 1986). Experimental evidence

* E-mail: mostry@fs.fed.us

for confirming reports of the saprophytic or pathogenic relationships of *E. mammata* on several hardwood (broadleaved) species other than *Populus* have largely been inconclusive (Ostry and Anderson, 2009). Its pathogenicity on aspen, along with several key morphological features, were evidence that led Rogers and Ju (1996) to remove the aspen fungus from the genus *Hypoxylon* and place it in the genus *Entoleuca*.

The incidence and impact of Hypoxylon canker is greatest in the first 20 years of a developing aspen stand. Stem cankers on trees of this age are generally low on the stem, resulting in the death of affected trees; this contrasts with the upper stem cankers that develop in older trees and generally are not lethal if the trees develop new leaders (Anderson and Martin, 1981; Ostry and Anderson, 2009). In addition to trees in native forests, aspens in plantations and ornamental landscape plantings are vulnerable to damage by Hypoxylon canker.

20.2 Diagnosis

Cankers can develop anywhere on the branches and stems of aspens of all ages. Symptoms of infection by *E. mammata* are quite variable depending on the stage of disease development. Young cankers first become visible as slightly sunken, yellowish orange areas with irregular margins. Later, the outermost bark (periderm) within the canker becomes blistered, eventually cracking open, and exposing a powdery grey mat of fungal tissue, conidial pillars and conidia. Conidia are single celled, hyaline, fusiform to ellipsoid and range from 5.5–8.0 × 1.5–4.0 μm in size.

As patches of bark flake off, cankers become rough and black in the centre while the bark at the advancing margins of the enlarging cankers becomes yellowish orange. In the oldest areas of canker tissue, perithecial stromata develop in the crumbling cortex. The patches of hard, cushion-like stromata are white when young and turn grey to black as they age. Ascospores are single celled, dark brown, elongate ellipsoid and range from 9.0–12.0 × 20.0–33.0 μm in size.

Callus occasionally develops at the margins of cankers but usually the fungus invades new tissues and the canker expands too rapidly for callus to develop, resulting in characteristic diffuse cankers that can eventually girdle and kill affected trees. Variation in callus production and wound closure may explain differences in canker resistance and susceptibility among aspen clones. Cutting into cankers exposes white or grey mycelial fans that usually extend beyond the visible canker margin, and the diagnostic mottled black and yellowish cream colour of the sapwood.

'Flags', i.e. dead leaves remaining on branches girdled by a canker, are a common disease symptom in the crowns of affected trees, and are especially visible during the dormant season. Wood decay by *E. mammata* (Merrill *et al.*, 1964) often results in branch and stem breakage at cankers and is also a damage characteristic of the disease (Plate 27).

20.3 Infection Biology

Even after many years of research, questions remain concerning some aspects of the infection biology of *E. mammata*, but the consensus of investigators is that ascospores infect wounded xylem (Ostry and Anderson, 2009). Hubbes (1964) described *E. mammata* as a sapwood parasite that required a wound into the wood or dead bark to avoid toxic compounds in the living bark that inhibit growth of the fungus (Hubbes, 1962, 1969).

Toxins produced by *E. mammata* are thought to be involved in pathogenesis but their specific role still remains unclear (Griffin and Manion, 1985; Bélanger *et al.*, 1989, 1990; Mottet *et al.*, 1991; Pinon and Manion, 1991; Kruger and Manion, 1993a,b). Hubbes (1964) provided the first evidence that diffusible substances produced by *E. mammata* inhibited the toxic bark effects and prevented callus formation at wound sites on aspen. It was postulated that the toxin was important in the initial process of infection and disease development (Schipper, 1975). Cell-free culture extracts of *E. mammata* caused aspen bark necrosis and collapse, and inhibited wound callus formation (Schipper, 1978).

Although most commonly encountered as a stem canker, the fungus can infect twigs and branches throughout the crowns of trees. Advanced stem cankers are often centred on a branch stub. Cankers commonly originate on branches and, if they are close by, eventually grow into the main stems of affected trees. Dead wood is not infected by *E. mammata* and cankers do not expand far into dead wood. Cankers have been found originating at the base of young, unwounded branches (Manion, 1975). The absence of the green layer above branch axils, which, if present, consists of cortex and part of the secondary phloem containing host defence compounds, may also provide an entry court for the fungus (French and Oshima, 1959).

The ideal wound for infection of aspen by *E. mammata* was described by Bagga and Smalley (1969). Their hypothesis was that bark-boring insects could cause deep wounds and tunnels with high relative humidity into which the fungus could penetrate and colonize trees. Because *E. mammata* utilizes cellulose, cellobiose and glucose in the xylem, but not sucrose in the phloem (Bagga, 1968; Schipper and Anderson, 1971), wounds deep into the xylem not only provide access to the sugars it requires for growth, but the pathogen also avoids the toxins in the bark. The fungus forms sheets of hyphae that decay the woody xylem, obtaining nutrients that allow it to grow into the phloem and eventually producing enzymes that detoxify the fungistatic compounds in the green bark layer (Ostry and Anderson, 1998).

Several species of insects make oviposition wounds on aspen that have been demonstrated to be frequent sites for infection (Anderson *et al.*, 1979; Ostry and Anderson, 1983, 1986). The habits of these insects and of downy woodpeckers (*Picoides pubescens* (Linnaeus)) foraging on the insect larvae partially explain the distribution of Hypoxylon canker within affected trees and aspen stands (Ostry *et al.*, 1982; Ostry and Anderson, 1998). On average, there is a latent period of 26 months from infection by ascospores through galls resulting from oviposition by the poplar gall saperda (*Saperda inornata* Say) until symptom expression (Ostry and Anderson, 1995), which has contributed to the past difficulty in determining the origin of cankers (Plate 28).

Approximately a year after symptoms develop, hyphal pegs form and break through the bark periderm revealing the grey mat of fungal tissue and exposing the conidia. Conidia are not infectious, nor are they involved in the spread of the fungus in the field. Rather, they are thought to function as spermatia for the production of ascospores that develop in the perithecial stromata that replace the conidial mats 1–2 years later (Griffin *et al.*, 1992).

20.4 Epidemiology

There have been many studies of Hypoxylon canker conducted in natural stands and many of the results have been contradictory. The wide geographical range of aspen, with many site and environmental variables, its clonal habit and variation in disease susceptibility, and differences in the ages of trees under study have confounded interpretations of the research results. In addition, care must be exercised in reading these reports because it is not always clear whether the authors were including branch cankers as well as stem cankers in their analyses. Furthermore, variation in disease resistance among aspen clones expressed as rapid stem death, in contrast to tolerance in which diseased stems can support cankers for longer periods of time, has complicated studies of the disease. More importantly, it is not always evident whether the results are expressed in terms of the prevalence (ratio of living infected trees to total living trees at a given time) or incidence (ratio of new infected trees to the initial total of trees during a given time interval).

Factors contributing to moisture stress were found to increase canker susceptibility (Bagga and Smalley, 1974). Moisture stress, through its effect on increasing host amino acids favourable for the growth of *E. mammata* (Griffin *et al.*, 1986; Bélanger *et al.*, 1990), and in reducing levels of bark compounds inhibitory to the growth of *E. mammata* (Kruger and Manion, 1994), has been suggested as a major factor in increasing tree susceptibility and

canker elongation. Moisture stress has also been shown to influence infection by ascospores (Bier and Rowat, 1962b).

Ascospores are liberated from perithecia during wet weather throughout the year, including during the winter months in Minnesota at temperatures as low as –4°C (Wood and French, 1965a), although infection of trees during those months has not been demonstrated. In the field, branch galls resulting from oviposition by the poplar gall saperda on aspens inoculated with ascospores resulted in canker development, thus providing evidence of the critical role of insect wounds and ascospores in the biology of the disease (Ostry and Anderson, 1995).

Detailed study of the increase in canker incidence in an aspen plantation (Manion and Blume, 1975) and in naturally occurring clones (Falk *et al.*, 1989), and genetic studies of the population structure of *E. mammata* within plantations (Griffin *et al.*, 1992; Ostry and Anderson, 2009) have revealed that the spread of the fungus and increase in disease incidence were random and that each canker in the populations studied was genetically different. It was concluded that cankers result from infection by single ascospores and that somatic incompatibility keeps individual canker isolates genetically isolated, thereby preventing pathogenic races from developing. Evidence for outcrossing in populations of *E. mammata* was obtained in experimental pairings of single ascospore-derived strains from the same perithecium (Bier, 1940; Sharland and Rayner, 1989).

Yearly fluctuations in the prevalence of Hypoxylon canker (i.e. 'wave years') reported by Schmiege and Anderson (1960) were suggested to be the result of cankers that developed under favourable conditions for infection in the galleries made by various insects (Nord and Knight, 1972).

A conceptual model incorporating various pre-infection and post-infection factors in the Hypoxylon canker disease cycle was developed to illustrate how the host, pathogen and environment interact within individual trees and within stands. Pre-infection factors, such as early wound responses, defence compounds and resistance to woodboring insects are difficult to select for but may correlate with natural canker prevalence better than post-infection factors, such as rate of canker elongation, callus formation or branch death (Ostry and Anderson, 2009).

20.5 Management Strategies and Tactics

A large body of literature on Hypoxylon canker produced by numerous researchers has added to our understanding of this disease. However, it is difficult to provide examples of direct, operational control efforts or changes in management directed at minimizing its impact as a result of this research. More often, the loss in volume and quality resulting from a high incidence of white trunk rot caused by *Phellinus tremulae* (Bondartsev) Bondartsev & P.N. Borisov is the major factor influencing harvest age over much of the range of aspen, imposing what has been referred to as the 'pathological rotation' age of aspen.

Aspen has several unique traits that complicate silviculture and favour single-entry, even-aged management (Mowrer, 1988). Among these traits are the clonal growth of aspen, its root sucker regeneration, its extreme shade intolerance, the thin bark that makes it vulnerable to damage by many biotic and abiotic factors and its ability to highly self-regulate its stand density over time.

In the past, the large areas of aspen in the Lake States that established and developed on disturbed sites after the harvest of pine (*Pinus* spp.) and following the many ensuing slash fires have been managed extensively, applying no specific silvicultural practices other than the stands being predominantly regenerated using clear-cutting (clear-felling). For the most part, dependable, natural regeneration was obtained by this method, and stands were allowed to develop until the next felling cycle.

More recently, there has been an interest in applying alternative silvicultural systems for aspen management (Ruark, 1990; Zasada *et al.*, 2001; Gradowski *et al.*, 2010). These systems of intensive management may make it necessary to develop and apply strategies such as genetic improvement and clone

manipulation to minimize the impact of Hypoxylon canker and other damaging agents.

20.5.1 Avoidance

The prevalence of Hypoxylon canker and mortality of infected trees have been subjects of study by many investigators who have attempted to correlate site and stand factors with disease incidence and severity. A greater prevalence of Hypoxylon canker on poorer sites has been reported (Lorenz and Christensen, 1937; Gruenhaugen, 1945). Study of the cause of aspen clone deterioration in the Lake States revealed that under similar environmental conditions, genetic differences among clones determined clone longevity, and that stocking differences among clones were attributed to variation in insect damage and to thinning by Hypoxylon canker (Shields and Bockheim, 1981).

Perala (1984) reported significantly less injury by both wood-boring insects and Hypoxylon canker on an excellent aspen site (50 years old, site index 27 m) compared with a good site (50 years old, site index 23 m). However, in an earlier study, a large number of aspen field plots established across the range of aspen in the Lake States to examine the prevalence of Hypoxylon canker as affected by stand and site characteristics yielded ambiguous results (Anderson, 1964). There was no correlation between canker prevalence and site index. The only consistent relationships were that canker prevalence fluctuated widely from year to year and prevalence was less in older stands. The author suggested that climate, microclimate and weather were most likely more important in the occurrence of canker than soil and other characteristics affecting aspen growth and vigour. Looking back on this study though, it was done before the research that demonstrated the importance of wounds made by various insects, which, in turn, influence the incidence of the disease in aspen stands (Anderson *et al.*, 1979; Ostry and Anderson, 1998).

Inoculations of greenhouse-grown aspen plants revealed that factors related to host moisture stress increased susceptibility to infection and canker development (Bagga and Smalley, 1974). A survey of natural aspen stands in central New York for the prevalence of canker revealed that several physical and chemical factors contributing to the availability and retention of moisture were negatively correlated with canker prevalence (Bruck and Manion, 1980). Several interacting soil and host variables were used to develop a prediction model for evaluating sites for the risk of Hypoxylon canker.

One of the most frequent observations on the incidence of Hypoxylon canker cited is its relationship with stand density. Greater disease has been reported among trees along stand edges and in low-density stands by many investigators (Ostry and Anderson, 2009). Based on these reports, aspen management recommendations often include statements of regenerating and maintaining high stocking densities and a closed canopy to avoid excessive losses to Hypoxylon canker (Perala, 1977).

20.5.2 Exclusion

Hypoxylon canker can be found throughout most of the range of aspen in North America except Alaska. Because historically it was assumed that the disease occurred widely, long-distance dispersal and geographic spread of the pathogen was not studied in detail and no explanation for its absence in Alaska has been provided.

Molecular analysis and historical evidence suggests that *E. mammata* was introduced to Europe several centuries ago (Kasanen *et al.*, 2004). The latent period in this disease that was previously mentioned (Ostry and Anderson, 2009) means that the pathogen could inadvertently be moved to unaffected areas on infected, asymptomatic plants. Therefore, reasonable care should be taken not to establish plantations or landscape plantings with infected stock.

20.5.3 Eradication

The removal of infected trees during thinning to reduce inoculum within aspen stands has

been suggested (Bier, 1940). However, the removal of infected trees from a stand isolated from other infected aspens failed to eliminate ascospores that were thought to have originated on overlooked cankers and carried into the stand by wind (Froyd and French, 1967). Felling infected trees did not significantly reduce spore inoculum because ascospores continued to be ejected from cankers on these trees and could be collected on spore traps for 2 years after felling (Froyd and French, 1967). The authors concluded that eradication was not an efficient measure of control.

As previously noted, population studies of *E. mammata* in plantations (Griffin *et al.*, 1992; Ostry and Anderson, 2009) demonstrated that the presence of a fungal isolate from an infected tree does not increase the risk of infection of adjacent trees. The availability of suitable wounds, rather than the presence of inoculum, plays a major role in the random occurrences of the disease within stands. Thus, eradication of *E. mammata* inoculum is not a strategy for managing healthy aspen.

20.5.4 Protection

There have been only a few research studies published on the use of chemical protectants against Hypoxylon canker. Cycloheximide was found to inhibit mycelial growth of *E. mammata in vitro*, and cankers did not develop on inoculated, sterilized black cottonwood cuttings dipped in actdione unless the cuttings were water stressed (Bier, 1962). In addition, it was found that the antibiotic treatment was injurious to the developing shoots and roots of the treated cuttings.

Saprophytic fungi on well-watered greenwood cuttings of black cottonwood prevented canker development when inoculated with *E. mammata*, but were not inhibitory when the cuttings were water stressed (Bier and Rowat, 1962a, 1963a). The authors proposed that the antagonistic activity of the facultative parasites would provide an opportunity for biological control using natural bark extracts. Some authors (Bier and Rowat, 1963b; Bier, 1965) have suggested that the susceptibility or resistance of living bark of quaking aspen to *E. mammata* may be related to the presence of microorganisms, which may either be directly antagonistic to *E. mammata* (as just noted) or act via the inactivation of bark pyrocatechol, which is fungistatic towards *E. mammata* (Hubbes, 1962).

Naturally occurring bacteria and fungi on or in *E. mammata* stromata and aspen bark were found to be inhibitory to ascospore germination (Wood and French, 1965b). The authors suggested that at certain times conditions within infection courts might differentially favour the germination of ascospores over development of these naturally occurring microorganisms.

Fertilization by treatments with NPK (nitrogen + phosphorus + potassium) did not result in significant reduction in the incidence of Hypoxylon canker on inoculated aspen in a field test in Michigan (Teachman *et al.*, 1980). The authors postulated that lack of host moisture stress and clonal variation in host susceptibility to infection may have been among the factors confounding the effects of fertilization on canker development.

20.5.5 Resistance

Recognition of the wide variation in many traits among natural aspen clones, including damage by insect pests and pathogens, has provided optimism for the selection and breeding for Hypoxylon canker resistance. In a study in Michigan, Copony and Barnes (1974) found that the number of aspen stems within various clones affected by Hypoxylon canker ranged from 9% to 90%. They reported that 55% to 80% of the variation in the disease among clones was genetic. Discussing the relationship between stand density and canker incidence, the authors suggested that genetic control of variation in suckering ability may be one mechanism involved, with clones producing dense suckers experiencing less damage by Hypoxylon canker than clones producing fewer suckers.

Other investigators question whether canker incidence in the field is an accurate measure of resistance to natural infection (Manion and Griffin, 1992). Results from the

inoculation of branches on 100 aspen clones were not correlated with the natural prevalence of canker (French and Hart, 1978). In another study, canker expansion on inoculated stems of three of five clones was not correlated with the natural prevalence of cankers on those clones, although results from the inoculation of grafted plants of the five clones in the greenhouse were (Enebak *et al.*, 1999).

Aspen breeding in the Lake States was initiated in 1955, supported in part by the forest industry (Li *et al.*, 1993a). Large family differences in Hypoxylon canker incidence were found in a 30 year old aspen field trial of 25 full-sib families (Li *et al.*, 1993b). Families that self-pruned well had a lower incidence of Hypoxylon canker than other families. The authors suggested that trees that self-pruned had fewer branches on which cankers could develop and grow into the main stem. Interspecies crosses, however, have not been as successful in aspen breeding because of the high susceptibility of aspens to damage by several diseases (as well as to Hypoxylon canker) (Ostry and Anderson, 2009).

Screening aspen for resistance to Hypoxylon canker has been conducted by inoculating seedlings (Enebak *et al.*, 1996), branches (French and Manion, 1975; Valentine *et al.*, 1976; French and Hart, 1978; Griffin *et al.*, 1984) and main stems (Enebak *et al.*, 1999). In these tests, mycelium of *E. mammata* was introduced through mechanical wounds and canker expansion or the production of canker-inhibiting callus were measured. The results have been variable and open to multiple interpretations. Results from the inoculation of seedlings of two full-sib families with a 30 year canker history in the field were correlated, but highly dependent on seedling age, with the more lignified 9 month old seedlings being better predictors than the 3 month old seedlings (Enebak *et al.*, 1996). Chemical and morphological barriers to *E. mammata* in two aspen genotypes with different levels of field resistance varied by age of tissue and correlated with canker history (Enebak *et al.*, 1997).

A study of the early wound responses of greenhouse-grown aspen genotypes resistant and susceptible to *E. mammata* demonstrated differences in the development of an intact and localized lignified barrier zone and wound callus with phenolic substances that correlated with canker resistance (Bucciarelli *et al.*, 1998, 1999). The levels of defence-related genes were found to be associated with aspen genotype, wounding and time after wounding, and were roughly associated with Hypoxylon canker resistance (Thamarus and Furnier, 1998).

No differences in natural canker incidence were found among trees in a plantation of nearly 600 aspen progeny from controlled crosses of parents that were either both diseased, or only one diseased or both healthy after 20 years (Ostry and Anderson, 2009). Canker incidence among parent trees propagated via root cuttings from diseased and disease-free trees was also similar, illustrating the difficulty of selecting parent trees based on canker prevalence in the field (Manion and Griffin, 1992).

The establishment of stands of improved aspen will require the recognition of not only the genetic component of the disease cycle but also the spatial and environmental factors influencing the disease. One approach that may minimize the impact of Hypoxylon canker in the first 15–20 years of a plantation of widely spaced aspen would be to establish the plantation with resistant progeny selected from long-term tests of clones. After coppicing, spatial resistance (McNabb *et al.*, 1982) resulting from dense sucker reproduction will then further reduce disease incidence.

20.5.6 Therapy

With the exception of pruning infected branches before the canker reaches the main stem (Ostry and Anderson, 1979), or excising the fungus in the early stages of the disease on high-value trees (Hinds and Krebill, 1975), treatment for the direct control of Hypoxylon canker is not available. There have been a few published reports on the investigation of the use of antibiotics to control the canker, but this line of research produced little success.

Treatment of cankers on aspens in Colorado with cycloheximide was not effective in inhibiting the disease (Hinds and Peterson, 1966). The application of cycloheximide in fuel

oil directly on to stem cankers arrested canker enlargement, and treated trees survived longer than non-treated trees during a small-scale field test in New York (Brown and Silverborg, 1970). Although the authors stated that their results were limited and inconclusive, they did suggest that direct control of Hypoxylon canker may be useful on high-value trees.

In Minnesota, three formulations of phytoactin and two of semicarbazone in an oil emulsion were aerially applied by helicopter to trees with cankers during the summer (Anderson, 1971). The treatments were not effective in controlling the cankers or increasing the survival of infected trees.

20.5.7 Integrated disease management

As no direct control measures are available, the concept of integrated pest management and silvicultural strategies to manage aspen have been suggested to minimize the impact of Hypoxylon canker (Ostry, 1986). Results from numerous studies and surveys (Schreiner, 1925; Day and Strong, 1959; Copony and Barnes, 1974; Bruck and Manion, 1980; Brandt *et al.*, 2003) have indicated that the disease is less severe in well-stocked stands, so managers have been encouraged to obtain and retain optimum stand stocking. It should be pointed out, however, that there is evidence that infected stems die more rapidly when shaded (Anderson and Anderson, 1968; Anderson, 1972), and therefore infected stems in denser stands may be removed (i.e. die) more quickly than in more open stands. Surveys would thus give the impression that these stands had a lower prevalence of canker than the less stocked stands in which infected stems survive for longer periods of time.

Spatial resistance (McNabb *et al.*, 1982), that is, the interactions of environmental and biotic factors with tree density, can influence disease incidence and this relationship has been proposed as influencing the incidence and severity of Hypoxylon canker in aspen stands. For example, persistent branches on the lower stems of aspen in low-density stands may provide entry courts such as insect oviposition wounds (Ostry and Anderson, 1998) that account for a greater incidence of canker than among trees in dense stands that promote self-pruning (Ostry and Anderson, 1979).

The practice of thinning aspen in general (Perala, 1991; Penner *et al.*, 2001) and, particularly, its effect on the occurrence of Hypoxylon canker (Pitt *et al.*, 2001; Ostry *et al.*, 2004) has been controversial owing to the economic aspects, variation in site, clone genetics, an incomplete understanding of the biology of the disease and contradictory results from research plots. Anderson (1964) reported on two thinning studies: one with significant effects and the other with less certain results. An increase in Hypoxylon canker in thinned plots was reported early in a study in Minnesota (Anderson and Anderson, 1968), but further analysis after 47 years revealed that canker incidence was affected to greater extent by clonal differences in canker susceptibility than to the thinning treatment (Ostry *et al.*, 2004). It has been suggested that forest managers consider factors that affect aspen regeneration densities and rotation length when thinning is planned (Bates *et al.*, 1989). Cleland *et al.* (2001) suggested that the thinning of aspen stands should be delayed until the lower branches have naturally self-pruned.

In Ontario, precommercial thinning resulted in an elevated incidence (based on the initial trees in the stand) of Hypoxylon canker on living trees but no significant differences in the prevalence (based on the number of surviving trees) of canker among the living trees (Pitt *et al.*, 2001). The authors concluded that even though thinning as a management tool could be used without concern for occurrence of the canker, thinning and tree mortality attributed to Hypoxylon canker and other causes may increase the risk of achieving less than optimum stocking for maximum fibre yield at the end of the rotation, and these losses should be factored into determining the desired final densities. Partial felling in aspen resulted in damage by sun scald, logging wounds invaded by canker fungi and infestations by wood-boring insects, all of which weakened the residual trees (Walters *et al.*, 1982).

Clonal differences in the prevalence of Hypoxylon canker, as well as in growth and yield, have been the basis for the suggestion of

clone management in aspen stands in which superior clones are favoured over disease-susceptible clones (Perala, 1977). By not harvesting inferior, disease-susceptible clones, the root suckers from the harvested superior clones are allowed to expand and predominate on the site. It has been recommended that if 15–25% of the aspens in a stand is affected by Hypoxylon canker, the stand should be harvested early (Schipper and Anderson, 1976). If more than 25% of the aspens are affected the site should be converted to other species, assuming that clone genetics is responsible for the high disease incidence.

Whether there is a site relationship to canker incidence is uncertain. However, managing aspen on the best sites will increase yields and avoid losses caused by insect pests and other pathogens that are more severe on lower quality sites. Studies have shown that aspen that had a history of defoliation by the forest tent caterpillar (*Malacosoma disstria* Hübner) had an increased susceptibility to wood-boring insects and Hypoxylon canker (Churchill *et al.*, 1964; Anderson and Martin, 1981).

Based on the importance of insect wounds as infection sites, aspen stands should be monitored for insect outbreaks that can increase the risk of Hypoxylon canker. Depending on the current age and stocking of affected stands, it may be desirable to harvest early with the objective of obtaining uniform, full stocking in the regenerating stand. It has been suggested that shearing (i.e. clear-cutting using specialized equipment) young understocked (non-commercial) aspen stands, so that they resprout from the remaining root systems, can be used to regenerate these stands to full stocking and restore their productivity (Perala, 1983). Delaying the harvesting of an aspen stand may be warranted to avoid exposing a young, regenerating stand to known current or expected outbreaks of defoliating insects, such as the forest tent caterpillar, and to subsequent increases of stem-boring insects (Churchill *et al.*, 1964) or to emergences of the periodic cicada (*Magicicada septendecim* Linnaeus); these can all increase the risk of aspen stands to damage by Hypoxylon canker (Ostry and Anderson, 1983).

Hypoxylon canker is just one of the many potentially damaging agents of aspen. Managers need to consider the many values of both 'healthy' and diseased or infested aspen stems for fibre production, wildlife habitat, soil stabilization, aesthetics, etc., throughout its range on various sites. How associated insect pests and pathogens interact with aspen on different sites and under different environmental conditions will determine the vulnerability and ultimate impact to individual trees, clones and stands. Managers will need to determine in many cases whether practical strategies to minimize potential damage are available, warranted or even desirable.

References

Anderson, G.W. (1971) Aerially applied antibiotics fail to control Hypoxylon canker on aspen. *Plant Disease Reporter* 55, 650.

Anderson, G.W. (1972) Development of *Hypoxylon mammatum* cankers on artificially shaded aspen stems. *Forest Science* 18, 316–318.

Anderson, G.W. and Anderson, R.L. (1968) Relationship between density of quaking aspen and incidence of Hypoxylon canker. *Forest Science* 14, 107–112.

Anderson, G.W. and Martin, M.P. (1981) Factors related to incidence of Hypoxylon cankers in aspen and survival of cankered trees. *Forest Science* 27, 461–476.

Anderson, N.A., Ostry, M.E. and Anderson, G.W. (1979) Insect wounds as infection sites for *Hypoxylon mammatum* on trembling aspen. *Phytopathology* 69, 476–479.

Anderson, R.L. (1964) Hypoxylon canker impact on aspen. *Phytopathology* 54, 253–257.

Bagga, D.K. (1968) Physiology of *Hypoxylon pruinatum* and its pathogenesis on quaking aspen. PhD thesis, University of Wisconsin, Madison, Wisconsin.

Bagga, D.K. and Smalley, E.B. (1969) Factors affecting canker development on *Populus tremuloides* artificially inoculated with *Hypoxylon pruinatum*. *Canadian Journal of Botany* 47, 907–914.

Bagga, D.K. and Smalley, E.B. (1974) The development of Hypoxylon canker of *Populus tremuloides*: role of interacting environmental factors. *Phytopathology* 64, 658–662.

Bates, P.C., Blinn, C.R., Alm, A.A. and Perala, D.A. (1989) Aspen stand development following harvest in the Lake States region. *Northern Journal of Applied Forestry* 6, 178–183.

Bélanger, R.R., Falk, S.P., Manion, P.D. and Griffin, D.H. (1989) Tissue culture and leaf spot bioassays as variables in regression models explaining *Hypoxylon mammatum* incidence on *Populus tremuloides* clones in the field. *Phytopathology* 79, 318–321.

Bélanger, R.R., Manion, P.D. and Griffin, D.H. (1990) Amino acid content of water-stressed plantlets of *Populus tremuloides* clones in relation to clonal susceptibility to *Hypoxylon mammatum in vitro*. *Canadian Journal of Botany* 68, 26–29.

Bier, J.E. (1940) *Studies in Forest Pathology. III. Hypoxylon Canker of Poplar*. Publication 691/Technical Bulletin 27, Canadian Department of Agriculture, Ottawa, Ontario, Canada.

Bier, J.E. (1962) Acti-dione and natural bark extracts in the control of Hypoxylon canker of poplar. *Forestry Chronicle* 38, 363–365.

Bier, J.E. (1965) Some inoculum factors in pathogenicity studies of *Hypoxylon pruinatum* (Klotzsche) Cke. on *Populus tremuloides* Michx. *Canadian Journal of Botany* 43, 877–883.

Bier, J.E. and Rowat, M.H. (1962a) The relation of bark moisture to the development of canker diseases caused by native, facultative parasites. VII. Some effects of the saprophytes on the bark of poplar and willow on the incidence of Hypoxylon canker. *Canadian Journal of Botany* 40, 61–69.

Bier, J.E. and Rowat, M.H. (1962b) The relation of bark moisture to the development of canker diseases caused by native, facultative parasites. VIII. Ascospore infection of *Hypoxylon pruinatum* (Klotzsch) Cke. *Canadian Journal of Botany* 40, 897–901.

Bier, J.E. and Rowat, M.H. (1963a) Further effects of bark saprophytes on Hypoxylon canker. *Forest Science* 9, 263–269.

Bier, J.E. and Rowat, M.H. (1963b) Some inoculum and substrate factors in the cultural inhibition of *Hypoxylon pruinatum* (Klotzsche) Cke. by pyrocatechol. *Canadian Journal of Botany* 41, 1585–1596.

Brandt, J.P., Cerezke, H.F., Mallett, K.I., Volney, W.J.A. and Weber, J.D. (2003) Factors affecting trembling aspen (*Populus tremuloides* Michx.) health in the boreal forest of Alberta, Saskatchewan, and Manitoba, Canada. *Forest Ecology and Management* 178, 287–300.

Brown, D.H. and Silverborg, S.B. (1970) Effect of cycloheximide on Hypoxylon cankers on aspen. *Plant Disease Reporter* 54, 462–465.

Bruck, R.I. and Manion, P.D. (1980) Interacting environmental factors associated with the incidence of Hypoxylon canker on trembling aspen. *Canadian Journal of Forest Research* 10, 17–24.

Bucciarelli, B., Jung, H.G., Ostry, M.E., Anderson, N.A. and Vance, C.P. (1998) Wound response characteristics as related to phenylpropanoid enzyme activity and lignin deposition in resistant and susceptible *Populus tremuloides* inoculated with *Entoleuca mammata* (*Hypoxylon mammatum*). *Canadian Journal of Botany* 76, 1282–1289.

Bucciarelli, B., Ostry, M.E., Fulcher, R.G., Anderson, N.A. and Vance, C.P. (1999) Histochemical and microspectrophotometric analyses of early wound responses of resistant and susceptible *Populus tremuloides* inoculated with *Entoleuca mammata* (=*Hypoxylon mammatum*). *Canadian Journal of Botany* 77, 548–555.

Churchill, G.B., John, H.H., Duncan, D.P. and Hodson, A.C. (1964) Long-term effects of defoliation of aspen by the forest tent caterpillar. *Ecology* 45, 630–633.

Cleland, D.T., Leefers, L.A. and Dickmann, D.I. (2001) Ecology and management of aspen: a Lake States perspective. In: Shepperd, W.D., Binkley, D., Bartos, D.L., Stohlgren, T.J. and Eskew, L.G. (eds) *Sustaining Aspen in Western Landscapes: Symposium Proceedings*. Proceedings RMRS-P-18, US Department of Agriculture, Forest Service, Rocky Mountain Research Station, Grand Junction, Colorado, pp. 81–99.

Copony, J.A. and Barnes, B.V. (1974) Clonal variation in the incidence of Hypoxylon canker on trembling aspen. *Canadian Journal of Botany* 52, 1475–1481.

Day, M.W. and Strong, F.C. (1959) A study of Hypoxylon canker on aspen. *Quarterly Bulletin of the Michigan Agricultural Experiment Station* 41, 870–877.

Enebak, S.A., Li, B. and Ostry, M.E. (1996) Seedling response of two trembling aspen (*Populus tremuloides*) families to infection by *Hypoxylon mammatum*. *European Journal of Forest Pathology* 26, 245–252.

Enebak, S.A., Bucciarelli, B., Ostry, M.E. and Li, B. (1997) Histological analyses of the host response of two aspen genotypes to wounding and inoculation with *Hypoxylon mammatum*. *European Journal of Forest Pathology* 27, 337–345.

Enebak, S.A., Ostry, M.E. and Anderson, N.A. (1999) Inoculation methods for selecting *Populus tremuloides* resistant to Hypoxylon canker. *Canadian Journal of Forest Research* 29, 1192–1196.

Falk, S.P., Griffin, D.H. and Manion, P.D. (1989) Spatial pattern analysis and sampling of Hypoxylon canker in naturally occurring clones of *Populus tremuloides*. *Plant Disease* 73, 31–33.

French, D.W. and Oshima, N. (1959) Host bark characteristics and infection by *Hypoxylon pruinatum* (Klot) Cke. *Forest Science* 5, 255–258.

French, J.R. and Hart, J.H. (1978) Variation in resistance of trembling aspen to *Hypoxylon mammatum* identified by inoculating naturally occurring clones. *Phytopathology* 68, 485–489.

French, J.R. and Manion, P.D. (1975) Variability of host and pathogen in Hypoxylon canker of aspen. *Canadian Journal of Botany* 53, 2740–2744.

Froyd, J.D. and French, D.W. (1967) Ejection and dissemination of ascospores of *Hypoxylon pruinatum*. *Canadian Journal of Botany* 45, 1507–1517.

Gradowski, T., Lieffers, V.J., Landhäusser, M., Sidders, D., Volney, J. and Spence, J.R. (2010) Regeneration of *Populus* nine years after variable retention harvest in boreal mixedwood forests. *Forest Ecology and Management* 259, 383–389.

Griffin, D.H. and Manion, P.D. (1985) Host–pathogen interactions as measured by bioassay of metabolites as produced by *Hypoxylon mammatum* with its host *Populus tremuloides*. *Phytopathology* 75, 674–678.

Griffin, D.H., Manion, P.D., Valentine, F.A. and Gustavson, L. (1984) Canker elongation, branch death, and callus formation as resistance or susceptibility responses in *Populus tremuloides* and virulence or avirulence characteristics of *Hypoxylon mammatum*. *Phytopathology* 74, 683–687.

Griffin, D.H., Quinn, K. and McMillen, B. (1986) Regulation of hyphal growth rate of *Hypoxylon mammatum* by amino acids: stimulation by proline. *Experimental Mycology* 10, 307–314.

Griffin, D.H., Quinn, K.E., Gilbert, G.S., Wang, C.J.K. and Rosemarin, S. (1992) The role of ascospores and conidia as propagules in the disease cycle of *Hypoxylon mammatum*. *Phytopathology* 82, 114–119.

Gruenhagen, R.H. (1945) *Hypoxylon pruinatum* and its pathogenesis on poplar. *Phytopathology* 35, 72–89.

Hinds, T.E. (1985) Diseases. In: DeByle, N.V. and Winokur, R.P. (eds) *Aspen: Ecology and Management in the Western United States*. General Technical Report RM-119, US Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colorado, pp. 87–106.

Hinds, T.E. and Krebill, R.G. (1975) *Wounds and Canker Diseases on Western Aspen*. Forest Pest Leaflet 152, US Department of Agriculture, Forest Service, Washington, DC.

Hinds, T.E. and Laurent, T.H. (1978) Common aspen diseases found in Alaska. *Plant Disease Reporter* 62, 972–975.

Hinds, T.E. and Peterson, R.S. (1966) Antibiotic tests on western gall rust and aspen cankers. *Plant Disease Reporter* 50, 741–744.

Hubbes, M. (1962) Inhibition of *Hypoxylon pruinatum* by pyrocatechol isolated from bark of aspen. *Science* 136, 156.

Hubbes, M. (1964) New facts on host–parasite relationships in the Hypoxylon canker of aspen. *Canadian Journal of Botany* 42, 1489–1494.

Hubbes, M. (1969) Benzoic and salicylic acids isolated from a glycoside of aspen bark and their effect on *Hypoxylon pruinatum*. *Canadian Journal of Botany* 47, 1295–1301.

Kasanen, R., Hantula, J., Ostry, M.E., Pinon, J. and Kurkela, T. (2004) North American populations of *Entoluca mammata* are genetically more variable than populations in Europe. *Mycological Research* 108, 766–774.

Kruger, B.M. and Manion, P.D. (1993a) Genetic control of *Populus tremuloides* sensitivity to metabolites of *Hypoxylon mammatum*. *Canadian Journal of Botany* 71, 1276–1279.

Kruger, B.M. and Manion, P.D. (1993b) Sensitivity of *Populus tremuloides* to toxic metabolites of *Hypoxylon mammatum* and susceptibility to *H. mammatum* infection. *Canadian Journal of Botany* 71, 1298–1303.

Kruger, B.M. and Manion, P.D. (1994) Antifungal compounds in aspen: effect of water stress. *Canadian Journal of Botany* 72, 454–460.

Li, B., Wyckoff, G.W. and Einspahr, D.W. (1993a) Hybrid aspen performance and genetic gains. *Northern Journal of Applied Forestry* 10, 117–122.

Li, B., Wyckoff, G.W. and Humenberger, E. (1993b) Early selection for quaking aspen. In: Mohn, C.A. (ed.) *Proceedings of the Second Northern Forest Genetics Association Conference, Roseville, Minnesota. July 29–30, 1993*. University of Minnesota, St Paul, Minnesota, pp. 32–46.

Lorenz, R.C. and Christensen, C.M. (1937) *A Survey of Forest Free Diseases and Their Relation to Stand Improvement in the Lake and Central States*. Bureau of Plant Industry Publication, US Department of Agriculture, Washington, DC.

Manion, P.D. (1975) Two infection sites of *Hypoxylon mammatum* in trembling aspen (*Populus tremuloides*). *Canadian Journal of Botany* 53, 2621–2624.

Manion, P.D. and Blume, M. (1975) Epidemiology of Hypoxylon canker of aspen. *Proceedings of the American Phytopathological Society* 2, 101.

Manion, P.D. and Griffin, D.H. (1992) Resistance in aspen to Hypoxylon canker. In: Blanchette, R.A. and Biggs, A.R. (eds) *Defense Mechanisms of Woody Plants Against Fungi*. Springer Verlag, New York, pp. 308–320.

Marty, R. (1972) The economic impact of Hypoxylon canker on the Lake States resource. In: *Aspen: Symposium Proceedings*. General Technical Report NC-1, US Department of Agriculture, Forest Service, North Central Forest Experiment Station, St Paul, Minnesota, pp. 21–26.

McNabb, H.S. Jr, Hall, R.B. and Ostry, M.E. (1982) Biological and physical modifications of the environment and resulting effect upon the host–parasite interactions in short-rotation tree crops. In: Heybroek, H.M., Stephan, B.R. and von Weissenberg, K. (eds) *Proceedings of the Third International Workshop on the Genetics of Host–Parasite Interactions in Forestry, Wageningen, The Netherlands, 14–21 September 1980*. Centre for Agricultural Publishing and Documentation (Pudoc), Wageningen, The Netherlands, pp. 60–71.

Merrill, W., French, D.W. and Wood, F.A. (1964) Decay of wood by species of the *Xylariaceae*. *Phytopathology* 54, 56–58.

Miller, J.H. (1961) *A Monograph of the World Species of* Hypoxylon. University of Georgia Press, Athens, Georgia.

Mottet, M.J., Dessureault, M. and Bélanger, R. (1991) Infection of *Populus* seedlings with ascospores of *Hypoxylon mammatum*: the role of toxins. *Canadian Journal of Plant Pathology* 13, 281.

Mowrer, H.T. (1988) Is managing aspen density worthwhile? In: Schmidt, W.C. (ed.) *Proceedings – Future Forests of the Mountain West: a Stand Culture Symposium, 1986 September 29–October 3, Missoula, MT*. General Technical Report INT-243, US Department of Agriculture, Forest Service, Intermountain Research Station, Ogden, Utah, pp. 201–209.

Nord, J.C. and Knight, F.B. (1972) The importance of *Saperda inornata* and *Oberea schaumii* (Coleoptera: Cerambycidae) galleries as infection courts of *Hypoxylon pruinatum* in trembling aspen, *Populus tremuloides*. *Great Lakes Entomologist* 5, 87–92.

Ostry, M.E. (1986) Aspen management minimizes disease impact. In: *Proceedings, Integrated Pest Management Symposium for Northern Forests, 1986 March 24–27, Madison, Wisconsin*. Cooperative Extension Service, University of Wisconsin, Madison, Wisconsin, pp. 191–199.

Ostry, M.E. and Anderson, G.W. (1979) Hypoxylon canker incidence on pruned and unpruned aspen. *Canadian Journal of Forest Research* 9, 290–291.

Ostry, M.E. and Anderson, N.A. (1983) Infection of trembling aspen by *Hypoxylon mammatum* through cicada oviposition wounds. *Phytopathology* 73, 1092–1096.

Ostry, M.E. and Anderson, N.A. (1986) Infection of aspen by *Hypoxylon mammatum* through treehopper wounds. *Phytopathology* 76, 957.

Ostry, M.E. and Anderson, N.A. (1995) Infection of *Populus tremuloides* by *Hypoxylon mammatum* ascospores through *Saperda inornata* galls. *Canadian Journal of Forest Research* 25, 813–816.

Ostry, M.E. and Anderson, N.A. (1998) *Interactions of Insects, Woodpeckers, and Hypoxylon Canker on Aspen*. Research Paper NC-331, US Department of Agriculture, Forest Service, North Central Research Station, St Paul, Minnesota.

Ostry, M.E. and Anderson, N.A. (2009) Genetics and ecology of the *Entoleuca mammata–Populus* pathosystem: implications for aspen improvement and management. *Forest Ecology and Management* 257, 390–400.

Ostry, M.E. and McNabb, H.S. Jr (1986) Populus *Species and Hybrid Clones Resistant to* Melampsora, Marssonina, *and* Septoria. Research Paper NC-272, US Department of Agriculture, Forest Service, North Central Research Station, St Paul, Minnesota.

Ostry, M.E., Daniels, K. and Anderson, N.A. (1982) Downy woodpeckers – a missing link in a forest disease life cycle? *Loon* 54, 170–175.

Ostry, M.E., Anderson, N.A., Rugg, D.J. and Ward, K.T. (2004) *Long-term Response of Precommercially Thinned Aspen Clones to Hypoxylon Canker*. Research Paper NC-341, US Department of Agriculture, Forest Service, North Central Research Station, St Paul, Minnesota.

Penner, M., Robinson, C. and Woods, M. (2001) The response of good and poor aspen clones to thinning. *Forestry Chronicle* 77, 874–884.

Perala, D.A. (1977) *Manager's Handbook for Aspen in The North-Central States*. General Technical Report NC-36, US Department of Agriculture, Forest Service, North Central Forest Experiment Station, St Paul, Minnesota.

Perala, D.A. (1983) *Shearing Restores Full Productivity to Sparse Aspen Stands.* Research Note NC-296, US Department of Agriculture, Forest Service, North Central Forest Experiment Station, St Paul, Minnesota.

Perala, D.A. (1984) How endemic injuries affect early growth of aspen suckers. *Canadian Journal of Forest Research* 14, 755–762.

Perala, D.A. (1991) Déjà vu: 'does it pay to thin young aspen?' In: Haavisto, V.F., Smith, C.R. and Mason, C. (eds) *Space to Grow: Spacing and Thinning in Northern Ontario: Proceedings of a Symposium, June 18–20, 1990, Sault Ste. Marie, Ontario.* Joint Report 15, Forestry Canada/Ontario Ministry of Natural Resources, Ontario, Canada, pp. 139–144.

Pinon, J. (1979) The origin and the main characters of French isolates of *Hypoxylon mammatum* (Wahl) Miller. *European Journal of Forest Pathology* 9, 129–142.

Pinon, J. and Manion, P.D. (1991) *Hypoxylon mammatum* and its toxins – recent advances in understanding their relationships with canker disease of poplar. *European Journal of Forest Pathology* 21, 202–209.

Pitt, D., Weingartner, D. and Greifenhagen, S. (2001) Precommercial thinning of trembling aspen in northern Ontario, Part 2. Interactions with Hypoxylon canker. *Forestry Chronicle* 77, 902–910.

Rogers, J.D. and Ju, Y.-M. (1996) *Entoleuca mammata* comb. nov. for *Hypoxylon mammatum* and the genus *Entoleuca. Mycotaxon* 59, 441–448.

Ruark, G.A. (1990) Evidence for the reserve shelterwood system for managing quaking aspen. *Northern Journal of Applied Forestry* 7, 58–62.

Schipper, A.L. Jr (1975) Changes in dehydrogenase and peroxidase activities of aspen infected with *Hypoxylon mammatum. Phytopathology* 65, 440–445.

Schipper, A.L. Jr (1978) A *Hypoxylon mammatum* pathotoxin responsible for canker formation in quaking aspen. *Phytopathology* 68, 866–872.

Schipper, A.L. Jr and Anderson, G.W. (1971) Alteration of sugar translocation in aspen by *Hypoxylon mammatum. Phytopathology* 61, 366–368.

Schipper, A.L. Jr and Anderson, R.L. (1976) *How to Identify Hypoxylon Canker of Aspen.* US Department of Agriculture, Forest Service, North Central Forest Experiment Station, St Paul, Minnesota.

Schmiege, D.C. and Anderson, G.W. (1960) *The Forest Insect and Disease Situation, Lake States 1959.* Station Paper 79, US Department of Agriculture, Forest Service, Lake States Forest Experiment Station, St Paul, Minnesota.

Schreiner, E.J. (1925) Preliminary survey of Hypoxylon poplar canker in Oxford County, Maine. *Mycologia* 17, 218–220.

Sharland, P.R. and Rayner, A.D.M. (1989) Mycelial interactions in outcrossing populations of *Hypoxylon. Mycological Research* 93, 187–198.

Shields, W.J. and Bockheim, J.G. (1981) Deterioration of trembling aspen clones in the Great Lakes region. *Canadian Journal of Forest Research* 11, 530–537.

Teachman, G.E., Frederick, D.J., Perkis, W.E. and Jurgensen, M.F. (1980) Effects of fertilization on Hypoxylon canker of trembling aspen. *Plant Disease* 64, 284–286.

Thamarus, K.A. and Furnier, G.R. (1998) Temporal and genotypic variation of wound-induced gene expression in bark of *Populus tremuloides* and *P. grandidentata. Canadian Journal of Forest Research* 28, 1611–1620.

Valentine, F.A., Manion, P.D. and Moore, K.E. (1976) Genetic control of resistance to Hypoxylon infection and canker development in *Populus tremuloides.* In: *Proceedings of the Twelfth Lake States Forest Tree Improvement Conference, August 1975.* General Technical Report NC-26, US Department of Agriculture, Forest Service, North Central Forest Experiment Station, St Paul, Minnesota, pp. 132–146.

Walters, J.W., Hinds, T.E., Johnson, D.W. and Beatty, J. (1982) *Effects of Partial Cutting on Diseases, Mortality, and Regeneration of Rocky Mountain Aspen Stands.* Research Paper RM-240, US Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colorado.

Wood, F.A. and French, D.W. (1965a) Hypoxylon canker of aspen: seasonal development of cankers and ascospore ejection in winter. *Phytopathology* 55, 771–774.

Wood, F.A. and French, D.W. (1965b) Microorganisms associated with *Hypoxylon pruinatum. Mycologia* 57, 766–775.

Zasada, J.C., David, A.J., Gilmore, D.W. and Landhäusser, S.M. (2001) Ecology and silviculture of natural stands of *Populus* species. In: Dickmann, D.I., Isebrands, J.G., Eckenwalder, J.E. and Richardson, J. (eds) *Poplar Culture in North America.* NRC Press, Ottawa, Canada, pp. 119–151.