1993

Effects of Pathogens and Bark Beetles on Forests

D J. Goheen

E M. Hansen

Follow this and additional works at: https://digitalcommons.usu.edu/barkbeetles

Part of the Ecology and Evolutionary Biology Commons, Entomology Commons, Forest Biology Commons, Forest Management Commons, and the Wood Science and Pulp, Paper Technology Commons

Recommended Citation
9.1 INTRODUCTION

Pathogenic fungi and bark beetles are important components of most coniferous forest ecosystems. Their relationships to each other and to the trees that dominate these plant communities have been moderated by millions of years of evolution into a dynamic equilibrium. Both pathogenic fungi and bark beetles have successful life history strategies for exploiting scattered stressed hosts and, under natural conditions, neither threatens the long-term productivity of the forests on which they depend.

Pathogenic fungi and herbivorous and saprophagous insects play important roles as consumers and decomposers in the energy flow and nutrient cycles of the forest. Humans are also important components of most forest ecosystems, but in evolutionary terms, they have arrived on the scene very recently. Changes in the forest environment resulting from human activity have, in many cases, increased populations and activity of root disease fungi and bark beetles. Humans also brought the concept of economic value to the forest. From this perspective, root pathogens and bark beetles are among the most destructive of the many threats to forest productivity.

This chapter addresses the varied roles that root pathogens and bark beetles play in western coniferous forests as (1) regulators of ecological structure and processes, (2) arbiters of management success and (3) agents of significant economic loss. Pathologists, entomologists, and forest managers often speak of the “impact” of fungal and insect “pests” on forest values. This terminology carries connotations of death and destruction that reflect only part of the role that these organisms play in the forest. The death of a tree may represent the loss of many cubic meters of timber, but at the same time, may improve soil fertility and grow-
ing conditions for surrounding trees and increase the non-economic diversity value of the forest by promoting non-host vegetation and creating new habitat for cavity-nesting birds and mammals. In most situations, it is impossible to compute a net "impact" because of the mixture of economic and non-economic values and ecological processes involved. We have chosen to avoid this problem by referring to the "effects" of root pathogens and bark beetles on various values and processes. Our goal, in part, is to provide a compilation of both economic and ecological effects of these organisms.

9.2 EFFECTS OF BARK BEETLE–PATHOGEN INTERACTIONS

The ultimate effect of pathogens and bark beetles is tree mortality. The effect of tree death on forests depends on the management context in which the effect is judged (Leuschner and Berck, 1985). The economic and ecological consequences of tree mortality are often quite different in forests where tree harvest is a significant objective compared to forests managed for other values. Both management scenarios are important in North America today, and a full accounting of the effects of pathogens and insects on forests must consider both.

The root disease fungi and bark beetles in North American forests are diverse groups both taxonomically and behaviorally (see Chapter 2, 3 and 6). It should not be surprising that their effects on forests are at least as varied. The forests themselves are dramatically different, ranging from vast natural and planted monocultures to the most diverse assemblages of conifers in the world. The consequences of infection by the root pathogen *Heterobasidion annosum*, for example, are very different in coastal *Tsuga heterophylla* forests from those in interior *Abies* stands. The result of infection in *Tsuga* forests is butt rot with only a slightly increased chance of tree breakage, while in *Abies* forests, trees often are killed in gradually expanding infection centers. Insects appear to play no significant role when *T. heterophylla* is infected, but *Scolytus ventralis* regularly attacks and hastens the death of *H. annosum*-infected *Abies*. In recognition of these limitations, we have focused our review on specific organisms acting in specific ecological situations.

Five major and widespread interactions are developed in detail: (1) *Phellinus weirii* and *Dendroctonus pseudotsugae* on *Pseudotsuga menziesii* west of the Cascade Mountains in the Pacific Northwest, (2) *Leptographium wageneri* var. *pseudotsugae* and associated beetles on *P. menziesii*, (3) *L. wageneri* var. *ponderosum* and associated beetles on *Pinus ponderosa*, (4) several species of root pathogens and *Dendroctonus ponderosae* on *P. contorta*, and (5) several species of root pathogens and *Scolytus ventralis* on *A. concolor* and *A. grandis*. Interactions among *Dendroctonus frontalis* and root pathogens in *Pinus* forests in the southern US and interactions among several root-feeding beetles and root pathogens in *P. resinosa* forests in the north central states also will be described.

We will explore the nature and extent of the relationships between these root pathogens and their bark beetle associates, summarize available information on the economic losses to timber production that accrue as a result of their activity, and summarize their effects on forest community structure, dynamics, and ecosystem processes.

9.2.1 Interactions in *Pseudotsuga* forests: *Phellinus* and *Dendroctonus*

9.2.1.1 Nature of relationship

In western Oregon and Washington, USA, and British Columbia, Canada, endemic populations of *D. pseudotsugae* primarily infest scattered windthrown, injured, and diseased
Effects of Pathogens and Bark Beetles on Forests

Pseudotsuga menziesii (Furniss and Carolin, 1977). Phellinus weirii is a major cause of root disease in Pacific Northwest forests, and D. pseudotsugae is especially likely to be found attacking trees infected by this fungus (Wright and Lauterback, 1958; Hadfield, 1985; Hadfield et al., 1986). Rudinsky (1966) demonstrated that D. pseudotsugae is able to detect differences between volatile oleoresin terpenes in healthy and physiologically stressed Pseudotsuga menziesii and is thus able to locate low-vigor hosts over substantial distances (see Chapter 6). Dendroctonus pseudotsugae outbreaks do develop periodically, and numerous healthy trees are infested. Usually, such outbreaks follow major windthrow events (Furniss and Carolin, 1977).

Beetle-brood production is optimal in previously healthy, windthrown P. menziesii, especially if the fallen material is shaded. The large populations of beetles emerging from substantial numbers of windthrown P. menziesii may attack and kill nearby standing trees (Wright and Lauterbach, 1958; Furniss and Carolin, 1977). Outbreaks collapse rapidly, because brood success is poor in such vigorous hosts. Large populations cannot be sustained unless there are additional fallen trees. Phellinus weirii plays an important role in the dynamics of D. pseudotsugae by providing stressed hosts that maintain low-level beetle populations between disturbance events that can trigger outbreaks.

Dendroctonus pseudotsugae may enhance long-term survival of P. weirii by attacking and killing some declining Pseudotsuga menziesii in root disease centers before the trees are windthrown. Beetle-killed trees are less prone to be windthrown than live trees because they lack foliage to catch the wind. The fungus survives in dead roots that remain in the soil. When trees are windthrown and infected roots are pulled out of the ground, most, if not all, inoculum is prevented from spreading.

The fungus survives for up to 50 years in infected stumps and snags, and infects susceptible trees that are regenerated on the site as growing roots contact infected material (Buckland and Wallis, 1956; Hansen, 1979; Tkacz and Hansen, 1982). The pathogen spreads little, if at all, by windborne spores or any means other than mycelial growth on or within roots. It usually takes 12–15 years for contacts to develop between roots of stumps and adjacent saplings and for the fungus to grow along the roots and up to the root collars of the young trees. Significant amounts of mortality in the new stand generally appear at this age, initially involving scattered trees adjacent to the old stumps. Disease centers develop as the pathogen subsequently grows from tree to tree across roots at a rate of about 30 cm year⁻¹ (Nelson and Hartman, 1975; McCauley and Cook, 1980).

9.2.1.2 Extent of relationship

Though many investigators have noted the common association between Phellinus weirii and D. pseudotsugae, few have attempted to quantify the degree of relationship. Goheen and Schmitt (unpublished data) surveyed on the ground a randomly selected sample of aerially detected, endemic D. pseudotsugae infestations in western Oregon (e.g. Fig. 9.1). They found that 77% of the areas where trees had been infested by bark beetles were infected with P. weirii. The remaining areas with infested trees were associated with undiseased, windthrown trees on steep slopes. Goheen et al. (unpublished data) found that over 90% of dead P. weirii-infected Pseudotsuga menziesii 20 cm or greater diameter at breast height (DBH) exhibited evidence of D. pseudotsugae galleries in a 7290 hectare area surveyed in northwest Oregon. Both standing dead and windthrown diseased P. menziesii exhibited evidence of infestation. However, beetle larval gallery development appeared to be more extensive on the windthrown trees. Pseudotsuga menziesii smaller than 20 cm DBH frequently
showed evidence of infestation by other bark beetles, primarily *Scolytus unispinosis* and *Pseudohylesinus nebulosus*.

**9.2.1.3 Timber losses**

*Phellinus weirii* causes annual losses estimated at 900 000 m$^3$ of wood in Oregon and Washington forests (Childs and Shea, 1967) and 1 050 000 m$^3$ in British Columbia (Wallis, 1967). Much of this loss is the result of *Pseudotsuga menziesii* mortality that also involves *D. pseudotsugae* infestation. Timber loss might better be attributed to the root disease–bark beetle association (Fig. 9.1). These estimates do not result from actual surveys but rather are based on projections that at least 5% of the area occupied by the *P. menziesii* forest type is affected, and losses within infected areas approach 50% over a rotation. No regional root disease surveys have been done, but the few local surveys suggest that perhaps even larger proportions of this forest type may be affected (Table 9.1). Within *Phellinus weirii* centers, reduction in *Pseudotsuga menziesii* volume has been measured at 10% (Bloomburg and Reynolds, 1985) to 55% (Goheen *et al.*, unpublished data).

*Phellinus weirii* also increases the chances of windthrow during storms. Root-rotted trees obviously are prone to windthrow, but healthy trees exposed to high winds at the edge of root rot openings also are more vulnerable. Windthrow resulting from winter storms, in February 1990, on Mary’s Peak in the Oregon Coast Range was almost exclusively related to root rot or to recent clear-cut margins, even in thinned stands (Hansen, unpublished data). Losses attributed to *D. pseudotsugae*, alone, during outbreaks have not been well documented but can be considerable. In outbreaks associated with windthrow, one green tree commonly is infested for every four windthrown trees (D. Bridgewater, USDA Forest Serv., unpublished data). Wright and Lauterbach (1958) reported that 2.8 billion cubic meters of timber were windthrown in major storms during the winters of 1949–1950 and 1951 in
Effects of Pathogens and Bark Beetles on Forests

Table 9.1. Local area surveys for incidence of *Phellinus weirii* in *Pseudotsuga menziesii* forests in western Oregon, USA

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Total area surveys</th>
<th>% of area affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goheen (1979)</td>
<td>Mapleton Ranger District (50,000 ha)</td>
<td>4.7</td>
</tr>
<tr>
<td>Lawson et al. (1983)</td>
<td>Black Rock Forest (195 ha)</td>
<td>4.9</td>
</tr>
<tr>
<td>Kanaskie (1985)</td>
<td>Columbia and Clatsop Co. (40,000 ha)</td>
<td>5.3</td>
</tr>
<tr>
<td>Goheen and Goheen (unpublished)</td>
<td>Alsea Ranger District (38,000 ha)</td>
<td>16.0</td>
</tr>
<tr>
<td>Goheen et al. (unpublished)</td>
<td>Scappoose Block (7290 ha)</td>
<td>10.9</td>
</tr>
<tr>
<td>Kastner and Kral (unpublished)</td>
<td>Tillamook Resource Area (30,000 ha)</td>
<td>7.4</td>
</tr>
<tr>
<td>Hansen (1978)</td>
<td>Sweet Home Ranger District (3840 ha)</td>
<td>11.0</td>
</tr>
</tbody>
</table>

western Oregon and Washington. By 1953, when populations collapsed, *D. pseudotsugae* had killed an additional 0.8 billion cubic meters of standing *Pseudotsuga menziesii* in the vicinity of the blowdown.

9.2.1.4 Ecological effects

Most of the forest area west of the Cascade Mountains in Oregon, Washington, and British Columbia is dominated by the coastal variety of Douglas-fir (*P. menziesii* var. *menziesii*). This tree develops in relatively pure stands following major disturbances such as fires, wind storms, bark beetle outbreaks, and clearcut harvest. *Pseudotsuga menziesii* grows rapidly, and by about age 30 often forms a closed canopy. It continues to dominate the site until the next major disturbance, sometimes for 500 years or more. On all but the driest sites, however, *T. heterophylla* is considered the climax species. This is the "Tsuga heterophylla zone" of Franklin and Dyrness (1969). While *T. heterophylla* can establish itself and grow in the shade beneath a *P. menziesii* canopy, it will not assume dominance until the canopy is opened, through death of *P. menziesii*. *Phellinus weirii* and associated bark beetles are the principal agents of mortality in the *Pseudotsuga menziesii* forest, allowing establishment of the *T. heterophylla* climax (Holah, 1991).

In typical *P. menziesii* stands, *Phellinus weirii* and endemic populations of *D. pseudotsugae* act together, preferentially killing *Pseudotsuga menziesii* and creating gradually expanding openings in the canopy where non-host tree species and shrubs are favored. Only conifers are susceptible to *Phellinus weirii*, and *Pseudotsuga menziesii* is the most susceptible of those commonly encountered in the *Tsuga heterophylla* zone. *Tsuga heterophylla*, *Pinus* spp., and *Thuja plicata* may be infected but are seldom killed. The fungus is usually confined as a butt rot in these species. *Dendroctonus pseudotsugae* attacks only *Pseudotsuga menziesii*.

Succession is either advanced or reset to more seral stages by *Phellinus weirii*, depending on the proximity of the infection center to seed sources or vegetative propagules of climax or seral species. *Tsuga heterophylla*, *Picea sitchensis*, *Thuja plicata*, *Calocedrus decurrens*, *Taxus brevifolia* and *Pinus monticola*, in addition to such hardwoods as *Alnus rubra*, *Acer macrophyllum*, and *A. circinatus* commonly proliferate in disease centers (Fig. 9.1). The effects of the *Phellinus weirii*/*D. pseudotsugae* complex on community composition and structure change with stand age.

Plant community development after catastrophic disturbance such as wildfire or clearcut harvesting is affected by *P. weirii* almost from the beginning. Woody shrubs and hardwood trees, established in root disease openings in the previous stand, sprout back and may quickly dominate the former infection center. The rodent *Aplodontia rufa* also may be especially
abundant in old infection centers after disturbance. In mature stands, their colonies are largely confined to areas with herbaceous vegetation, such as root disease centers. The combination of early shrub competition and animal damage may delay the establishment of conifers on the site. Susceptible conifers growing through the seral vegetation are increasingly likely to contact old \textit{P. weirii}-infected roots.

Understory vegetation generally is shaded out as the conifer canopy closes. Root disease centers, in contrast, represent gradually expanding islands of light in the dark forest. Mining of dead \textit{Pseudotsuga menziesii} by \textit{D. pseudotsugae} accelerates decomposition and nutrient turnover from wood (Edmonds and Eglitis, 1989; Schowalter et al., 1992). Nitrogen availability is increased as killed trees decompose (Waring et al., 1987). Temperature and humidity fluctuate more widely. Herbaceous plant diversity is higher than in the closed-canopy forest. There are corresponding differences in populations of soil microbes (Hutchins and Rose, 1984) and herbivorous animals. The regular accretion of snags and fallen trees provides habitat for a different assemblage of bird, small mammal, and amphibian species.

In most plant associations of the \textit{Tsuga heterophylla} zone, \textit{Pseudotsuga menziesii} cannot regenerate successfully in brush-filled root disease centers, but the disease-tolerant \textit{T. heterophylla} and \textit{Thuja plicata} can. As a consequence, infection centers may assume quite different compositions as old-growth stands and as young forests. Vegetational response to root pathogen infection has been examined in an old-growth forest of the central Oregon Cascades (Holah, 1991). In these stands, \textit{P. menziesii} killed by \textit{Phellinus weirii} and \textit{D. pseudotsugae} is replaced by \textit{T. heterophylla} as the pathogen slowly spreads through the forest. The resulting \textit{T. heterophylla} climax, in turn, forms a denser canopy than the \textit{Pseudotsuga} canopy, with the result that there may be less understory vegetation in the old infection centers than outside the areas of infection.

On particularly dry sites (\textit{Pseudotsuga/Holodiscus} association; Franklin and Dyrness, 1969), stands are more open, and \textit{P. menziesii} is the climax species. Accumulating evidence suggests that \textit{Phellinus weirii} is both more common and more extensive on these sites where \textit{Pseudotsuga menziesii} regenerates successfully in its own shade (Bloomberg and Beale, 1985; W. Kastner, unpublished data).

### 9.2.2 Interactions in \textit{Pseudotsuga} forests: \textit{Leptographium} and bark beetles

#### 9.2.2.1 Nature of relationship

\textit{Leptographium wageneri} var. \textit{pseudotsugae} affects \textit{P. menziesii} in California, Oregon, Washington, Idaho, Montana, and British Columbia, but it is most widely distributed and severe in 10- to 30-year-old plantations and young natural stands on disturbed sites in northwest California and southwest Oregon (Fig. 9.2). Two species of curculionids, \textit{Pissodes fasciatus} and \textit{Steremnius carinatus}, and one scolytid, \textit{Hylastes nigrinus}, have been implicated as vectors of \textit{L. wageneri} var. \textit{pseudotsugae} (Witcosky and Hansen, 1985; Harrington et al., 1985; Witcosky et al., 1986a, b). \textit{Pissodes fasciatus}, \textit{S. carinatus}, and \textit{H. nigrinus} prefer or are most successful in roots of injured, stressed, or dying \textit{P. menziesii} (Blackman, 1941; Chamberlain, 1958; Zethner-Moller and Rudinsky, 1967; Furniss and Carolin, 1977), and \textit{H. nigrinus} has been shown to be attracted to low molecular weight volatiles produced by stressed hosts (Rudinsky and Zethner-Moller, 1967), including \textit{L. wageneri}-infected roots (Witcosky et al., 1987).

Unlike most root pathogens, \textit{L. wageneri} does not cause a root decay but rather a vascular wilt-type disease. The fungus grows in the xylem tracheids and physically blocks water conduction by the host. Hosts decline rapidly and are infested commonly by stem-attacking
bark beetles in addition to the root-feeding insects. Large trees (20 cm DBH or greater) may be attacked by *D. pseudotsugae*, while smaller trees usually are infested by *Scolytus unispinosis*, *Pseudohylesinus nebulosis*, *Dryocoetes autographus*, buprestids, and cerambycids (Goheen and Hansen, 1978).

### Extent of relationship

*Pissodes fasciatus*, *Steremnius carinatus*, and *H. nigrinus* commonly breed in root systems of *L. wageneri*-infected *Pseudotsuga menziesii*, and several generations of beetles may develop before diseased trees die (Harrington et al., 1985; Witcosky and Hansen, 1985). *Leptographium wageneri* sporulates in beetle galleries. Conidiospores are borne in sticky slime droplets and adhere to the bodies of beetles as they emerge. Adult beetles disperse and locate new hosts in spring. *Hylastes nigrinus* and *Pissoides fasciatus* can fly considerable distances. *Steremnius carinatus* is flightless but can walk up to 100 m in 7 weeks, based on mark–recapture data (J. Witcosky, unpublished data). All three species burrow through soil to roots and engage in maturation feeding, sometimes visiting and wounding roots of several trees before constructing galleries. *Leptographium wageneri* requires wounds penetrating to the xylem for infection to occur. Witcosky et al. (1986b) isolated *L. wageneri* from 2.3% of *H. nigrinus*, 0.5% of *S. carinatus*, and 0.5% of *P. fasciatus* in a sample of beetles emerging from diseased *Pseudotsuga menziesii*. The vector relationship was confirmed by Harrington et al. (1985) and Witcosky et al. (1986b) when it was demonstrated that both artificially and naturally infested beetles introduced *L. wageneri* into living *Pseudotsuga menziesii* seedlings. Successful infection was associated with 1–5% of seedlings caged with naturally infested beetles, suggesting that the vectors are highly efficient at inoculating this fungus. This is borne out by the large numbers and widespread distribution of new *L. wageneri* centers observed in most years in areas where site and stand conditions are favorable for the disease.
Occurrence of *L. wageneri* in *P. menziesii* is strongly associated with a history of site disturbance. Disease centers are most likely to be found along roads, in areas where drainage patterns have been changed and especially on or near old tractor trails and landings (used to drag and stack harvested timber for transport), where soils have been compacted or topsoil removed (Goheen and Hansen, 1978; Hansen, 1978; Lawson and Cobb, 1986; Hansen et al., 1988; Morrison and Hunt, 1988). Occurrence of infection centers in areas where low tree vigor has resulted from adverse site conditions undoubtedly reflects vector preference for stressed hosts.

*Leptographium wageneri* also is much more likely to be found in *P. menziesii* plantations that have been precommercially thinned than in unthinned plantations (Harrington et al., 1983; Witcosky et al., 1986a; Hansen et al., 1988). Insect vector activity increases greatly in thinned stands, especially if green slash is created or still present early in the year when insect dispersal occurs. Witcosky et al. (1986a) found particularly dramatic insect population increases in plantations thinned after August and before June. They also found considerable evidence of new *L. wageneri* infections associated with vector attacks in *P. menziesii* crop-trees and stumps.

The effect of stem-attacking beetles on *L. wageneri*-infected *P. menziesii* has received less attention than the vector relationship. Nevertheless, it appears that these insects play an important part in administering the *coup de grace* to disease-weakened trees. In a sample of 231 recently dead *L. wageneri*-infected *P. menziesii* in western Oregon and Washington, Goheen and Hansen (1978) found that 52% were infested by stem-attacking beetles. Twenty-four percent of the trees exhibited galleries of *Pseudohylesinus nebulosus*. Other beetles were also present, including *Scolytus unispinosis* (14%), buprestids (14%), cerambycids (4%), *Dryocoetes autographus* (3%), and *Dendroctonus pseudotsugae* (1%). Typical of *L. wageneri*-affected trees in the region, this sample was composed mainly of trees smaller than 20 cm DBH. *Dendroctonus pseudotsugae* is more abundant when larger trees are affected.

In southwestern Oregon where *L. wageneri* var. *pseudotsugae* is of considerable concern to forest managers, 10- to 25-year-old *Pseudotsuga menziesii* plantations on about 400 000 hectares were surveyed in three separate evaluations (Goheen et al., unpublished data; Hessburg et al., USDA Forest Serv., unpublished data; Table 9.2). The surveys indicated that *L. wageneri* was common and widely distributed throughout *P. menziesii* plantations in the areas evaluated, but levels of associated mortality were quite variable. On the Siskiyou National Forest and the Medford District, 90% or more of the affected plantations exhibited low levels of mortality, averaging less than 2% of the trees. However, in the Tioga Resource Area, half of all units examined showed more than 2% mortality, and 10% suffered more than 10% mortality. Some plantations in this area exhibited extremely severe losses, as much as 50% of all *P. menziesii* (Fig. 9.2). Losses were especially severe considering that this mortality represented crop trees after precommercial thinning. Greater damage in the

Table 9.2. Frequency of black stain root disease (BSRD) in southwest Oregon, USA

<table>
<thead>
<tr>
<th>Survey area</th>
<th>Number</th>
<th>Percent with BSRD</th>
<th>Percent of trees infected in infected plantations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;0.1%</td>
<td>0.1-2.0%</td>
</tr>
<tr>
<td>Siskiyou N.F.</td>
<td>100</td>
<td>25</td>
<td>45</td>
</tr>
<tr>
<td>Tioga R.A.</td>
<td>100</td>
<td>29</td>
<td>40</td>
</tr>
<tr>
<td>Medford Dist.</td>
<td>500</td>
<td>19</td>
<td>93</td>
</tr>
</tbody>
</table>
Tioga Resource Area, compared to the other areas surveyed, probably resulted from greater site disturbance associated with previous tractor logging.

In southwest Oregon, *L. wageneri* appears in plantations at age 10–15, spreads at a rate of 0.4–0.9 m year⁻¹ in radially expanding infection centers, and slows dramatically at age 30–40 (Hansen and Goheen, 1988a). The disease seldom is observed affecting older trees. In Mendocino County, California, however, high levels of infection have been detected in 60- to 80-year-old trees (Jackman and Hunt, 1975; Lawson and Cobb, 1986).

In parts of western North America other than southwest Oregon and northwest California, *L. wageneri* on *P. menziesii* generally is much less widely distributed and damaging (Goheen and Hansen, 1978; Byler et al., 1983; Morrison and Hunt, 1988). When found, *L. wageneri* usually affects small numbers of young trees in plantations or scattered older *P. menziesii* that have been weakened by other root diseases, adverse site factors, or defoliation by *Choristoneura occidentalis* (Goheen and Hansen, personal observation).

### 9.2.2.3 Ecological effects

*L. wageneri* usually occurs on *P. menziesii* in the early stages of forest succession and is strongly associated with soil disturbance, usually the result of human activity. It is most common in relatively pure young *P. menziesii* stands that have been planted on sites where the former stand was harvested by clearcutting. It also is found in naturally regenerated *P. menziesii* on sites where drastic disturbance such as road building or fire has destroyed the previous forest.

Three host specific variants of *L. wageneri* (Harrington and Cobb, 1984, 1986; Chapter 3) are known. *Leptographium wageneri* var. *pseudotsugae* affects only *P. menziesii* in nature, although there is crossover to other species in artificial inoculations. Non-host tree species, shrubs, and herbaceous plants increase in frequency and cover as *L. wageneri* kills *P. menziesii* in disease centers. Although the insect vectors usually introduce *L. wageneri* into stressed or injured *P. menziesii*, the pathogen subsequently spreads to adjacent trees via root contact, regardless of vigor. *Pseudotsuga menziesii* is not eliminated from the infection centers since some individuals escape or tolerate infection (Hansen and Goheen, 1988a; Morrison and Hunt, 1988).

In southwest Oregon, the typical *L. wageneri* infection center contains a much higher proportion of *Tsuga heterophylla*, *Thuja plicata*, *Calocedrus decurrens*, *Pinus monticola* and/or *P. lambertiana* than nearby unaffected portions of the same stand. However, this depends on seed availability. In northwestern California, *Sequoia sempervirens* also is favored by death of *Pseudotsuga menziesii*. Hardwood trees and shrub species increase dramatically. Herbaceous plant species richness is higher in the diseased areas, and many wildlife species appear to prefer the habitat in disease centers to that in healthy *P. menziesii* stands, especially as canopies close and light becomes limiting (Goheen, personal observation). Because disease spread often diminishes after stands reach ages of 30–40 years, the disease-caused openings gradually fill with shade-tolerant tree species.

### 9.2.3 Interactions in *Pinus ponderosa* forests

#### 9.2.3.1 Nature of relationship

*Leptographium wageneri* var. *ponderosum* occurs at scattered locations throughout western North America, causing a vascular wilt disease of hard pines (Wagener and Mielke, 1961; Smith, 1967; Filip and Goheen 1982). The disease is especially common and damaging on
P. ponderosa in northeastern California and the central Sierra Nevada. The causal fungus is believed to be insect vectored, probably by the root-feeding bark beetle *Hylastes macer* (Goheen and Cobb, 1978). The biology of *H. macer* is imperfectly known, though the insect has been shown to attack the roots of weakened pines, appears to be attracted to wounds, and is most active when soils are moist (Blackman, 1941; Chamberlain, 1958; Bright and Stark, 1973).

*Leptographium wageneri* on *P. ponderosa* has been demonstrated to play a highly significant role as a predisposing agent for stem-infesting bark beetles (Wagener and Mielke, 1961; Cobb et al., 1973; Goheen and Cobb, 1980; Cobb, 1988b). Results of some studies indicate that several species of tree-attacking bark beetles actually are attracted to *L. wageneri*-infected trees (Goheen et al., 1985; Witosky et al., 1987). Other beetles may land on trees at random and simply have a better chance of successfully infesting diseased trees due to their weakened condition (Wood, 1972; Moeck et al., 1981; Owen, 1985; Chapter 6).

### 9.2.3.2 Extent of relationship

An insect vector for *L. wageneri* var. *ponderosum* had been suspected for some time, based on (1) the often widely separated distribution of *L. wageneri* infection centers in *P. ponderosa* stands, (2) the apparent association of the disease with weakened and injured trees in early stages of infection center development, and (3) the morphology of the causal fungus, which is particularly well adapted for insect dispersal. Evidence to support this hypothesis was provided when both the perfect and imperfect states of the fungus were found in galleries of *H. macer*, and excavation studies showed a close association between *H. macer* galleries and discrete areas of xylem staining by *L. wageneri* far down the roots of some newly infected pines (Goheen and Cobb, 1978). These findings were made in 1975, a year with an unusually moist spring. *Hylastes macer* galleries with *L. wageneri* fruiting were not found in subsequent attempts during drier years.

Cobb et al. (1982) studied sequential aerial photographs of *P. ponderosa* stands on the Georgetown Divide in the Sierra Nevada of California, where *L. wageneri* is common and widely distributed, and found that the average rate of generation of new infection centers was quite low, only one per 1000 ha per year. Although insect vectoring likely does occur, efficiency of disease transmission apparently is not as great in the *P. ponderosa* system as in the *Pseudotsuga menziesii* system. Furthermore, vector success may be dependent on uncommonly high moisture conditions. Both *H. macer* and *L. wageneri* var. *ponderosum* are known to be favored by high soil moisture (Blackman, 1941; Goheen et al., 1978).

In areas where *L. wageneri* var. *ponderosum* occurs, the primary (tree-killing) bark beetles of *P. ponderosa*, *Dendroctonus brevicomis*, and *D. ponderosae*, are found much more commonly infesting diseased than healthy trees (Cobb et al., 1973). Cobb (1988a) showed substantially reduced oleoresin yield and oleoresin exudation pressure in severely diseased trees, suggesting that these trees might be more susceptible to successful colonization by stem-attacking bark beetles than are uninfected trees. In the central Sierra Nevada, Goheen and Cobb (1980) monitored a sample of 256 pines with predetermined levels of *L. wageneri* var. *ponderosum* infection for 4 years. They found that 73% of the severely diseased trees (those with 50% or more of the root collar stained) were infested by *D. brevicomis* and *D. ponderosae* during the study, while 29% of the moderately diseased trees (those with 1-50% of the root collar stained) and only 2% of the apparently healthy trees were infested. Several other species of beetles, including *D. valens*, *Ips* spp., *Spondylis upiformis*, and *Melanophila* spp., also more commonly constructed galleries on *L. wageneri*-infected trees.
9.2.3.3 Timber losses

Leptographium wageneri var. ponderosum and associated bark beetles cause rapidly expanding mortality centers in *P. ponderosa* stands of any age, although 60- to 100-year-old sawtimber size trees are affected most commonly (Fig. 9.3). Once established in a suitable stand, the pathogen spreads to surrounding hosts at a rate of 1 m year⁻¹ across closely associated root systems (Cobb et al., 1982). Losses due to the disease can be locally great but have not been documented often. Byler et al. (1979) found that *L. wageneri* occurred on 65 of 11 560 ha on the Georgetown Divide, California. The disease and associated bark beetles had killed trees containing 110 000 m³ of wood.

9.2.3.4 Ecological effects

In nature, *L. wageneri* var. *ponderosum* is specific to the hard pines (Harrington and Cobb, 1984, 1986). It rarely affects other tree species in *P. ponderosa* infection centers although on occasion, *P. contorta*, *P. monticola*, and *P. lambertiana* growing in association with *P.

Fig. 9.3. Mortality caused by *Leptographium wageneri* var. *ponderosum* and associated *Dendroctonus brevicomis* and *D. ponderosae* in a 60-year-old *Pinus ponderosa* stand in the central Sierra Nevada of California. *Abies concolor* and *Calocedrus decurrens* are replacing *P. ponderosa* as the latter die.
ponderosa are damaged. Generally, *L. wageneri* var. *ponderosum* promotes other conifer species, as well as hardwood trees and shrubs.

*Leptographium wageneri* and associated bark beetles tend to affect *P. ponderosa* in pure, heavily stocked stands. In the Central Sierra Nevada of California, *L. wageneri* occurs in 60- to 80-year-old essentially pure pine stands having basal areas of >50 m² ha⁻¹ (Byler et al., 1979). Such patches of *P. ponderosa* are scattered within a mixed-conifer forest composed of *P. lambertiana, Pseudotsuga menziesii, Calocedrus decurrens, Abies concolor,* and *Quercus kelloggii,* as well as *Pinus ponderosa.* The pure *P. ponderosa* stands exist in areas where logging disturbance at the turn of the century was especially great. Many of the sites were burned with high intensity fires or had soil removed during skidding of logs to transport sites and railroad operations. *Leptographium wageneri* and associated bark beetles speed up succession in such locations by killing most of the *P. ponderosa.* Late successional (shade-tolerant) tree species, especially *C. decurrens* and *A. concolor,* which are immune to the disease, regenerate readily in the openings (Fig. 9.3).

In Oregon, as well, *L. wageneri* and associated bark beetles often are found in dense pine stands on poor or disturbed sites. However, when old-growth pines are affected, other species may not respond as readily, particularly on the drier sites of the "*Pinus ponderosa zone*" (Franklin and Dyrness, 1969). Rather, infection centers remain as gaps in the conifer canopy that progressively enlarge. *Leptographium wageneri* and beetles kill trees as they stand, often with extensive resinous in the butt and roots. These snags are attractive to cavity nesting birds and may stand longer than snags created by other agents.

### 9.2.4 Interactions in *Pinus contorta* forests

#### 9.2.4.1 Nature of relationship

In interior western North America, *D. ponderosae* is the primary bark beetle species killing *P. contorta.* Epidemic beetle populations periodically infest *P. contorta* over extensive areas. Large diameter (16 cm DBH or greater), mature (80 years old or more) *P. contorta* in dense stands are the most likely to be infested (Cole and Amman, 1973; Safranyik et al., 1974; Burnell, 1977). The bark beetles appear to be most successful on trees that are under stress from competition (Berryman, 1978; Mitchell et al., 1983). Apparent associations between *D. ponderosae* and the root pathogens, *Phaeolus schweinitzii, Leptographium wageneri,* and *Armillaria* sp., also have been reported by Geiszler et al. (1980), Hunt and Morrison (1986), and Tkacz and Schmitz (1986), respectively. Root diseases may play a role in providing suitable host material that maintains endemic *D. ponderosae* populations (Cobb et al., 1973). They also may trigger population increases at the beginning of outbreaks. Some evidence indicates that endemic *D. ponderosae* populations are preferentially attracted to root-diseased trees (Gara et al., 1984).

#### 9.2.4.2 Extent of relationship

Hunt and Morrison (1986) reported that in surveys of *P. contorta* stands severely infected by *L. wageneri* var. *ponderosum* in the Kootenay Region of British Columbia, Canada, *D. ponderosae* infestations were observed infrequently. However, when bark beetle infestations were encountered, they almost always were associated with *L. wageneri* infection. Other bark beetles, especially *Ips laitidens* and *I. mexicanus,* also were found attacking *L. wageneri*-infected trees. In *P. contorta* stands with endemic *D. ponderosae* populations in
Utah, Tkacz and Schmitz (1986) found *D. ponderosae* infesting 90% of a sample of pines with *Armillaria* sp. infections. Only 14% of the trees without detectable Armillaria root disease were infested. In central Oregon, Geiszler et al. (1980) found that 59% of a sample of *D. ponderosae*-killed *P. contorta* were infected by *Phaeolus schweinitzii*, while no infection was found in a sample of uninfested live trees from the same area.

### 9.2.4.3 Timber losses

Timber losses due to *D. ponderosae* outbreaks in *P. contorta* have been enormous. When populations of this aggressive bark beetle reach large size, they are capable of killing apparently healthy trees (see Chapter 6). It is estimated that *D. ponderosae* has killed trees containing about 12,000,000 m$^3$ of wood annually since 1895 (Wood, 1963). In 1970 alone, *D. ponderosae* killed trees containing 13,400,000 m$^3$ of wood in the Rocky Mountain states (Cole and Amman, 1980). Losses in *P. contorta* stands in eastern Oregon have been correspondingly great. Root disease is not directly involved in causing these large timber losses. Rather, the pathogens probably enhance long-term survival of *D. ponderosae* populations when stand conditions are suboptimal for the insects. Where they occur, root diseases certainly predispose *P. contorta* to beetle infestation.

### 9.2.4.4 Ecological effects

*Pinus contorta* is a seral species in several forest zones in western North America. It has an ability to grow on a wide range of forest sites and is also the climax species on many poorly drained or frost-prone sites. *Pinus contorta* produces both serotinus and open cones, regenerates readily, and develops very dense stands. Trees grow rapidly when young, but stands have a tendency to become stagnant at relatively early ages. In the past, periodic intense wildfires burned large areas of *P. contorta* forests and essentially regulated stand development (Brown, 1975). In the absence of fire, *D. ponderosae* also can act as a stand regulating agent by killing trees over substantial areas. The concentration of dead trees in turn increases the probability of subsequent wildfire (Leuschner and Berck, 1985). Where *P. contorta* is a climax species, trees killed by *D. ponderosae* are replaced by new *P. contorta* stands. Where *P. contorta* is seral, however, beetle-killed stands may succeed to more shade-tolerant species such as *A. lasiocarpa*, *A. grandis*, and *Picea engelmannii*.

### 9.2.5 Interactions in *Abies* forests

**9.2.5.1 Nature of relationship**

*Scolytus ventralis* is considered to be the major bark beetle species killing *Abies* spp. throughout western North America (Strubel, 1957; Furniss and Carolin, 1977). It is especially common on *A. concolor* and *A. grandis* in interior mixed-conifer stands. In addition to infesting entire trees, *S. ventralis* also commonly attacks and kills tops or portions of trees. *Scolytus ventralis* rarely infests vigorous *Abies*. Rather, it usually is found attacking injured, overmature, drought-stressed, insect-defoliated, or diseased trees (Fig. 9.4). The bark beetle may be attracted to unhealthy hosts (Ferrell, 1971). Root diseases are especially likely to be associated with *S. ventralis* infestation of *A. concolor* and *A. grandis* (Partridge and Miller, 1972; Cobb *et al.*, 1973; Miller and Partridge, 1974; Hertert *et al.*, 1975; Ferrell and Smith, 1976; Lane and Goheen, 1979; James and Goheen, 1981; Filip and Goheen, 1982; James *et*

9.2.5.2 Extent of relationship

The degree of relationship between root diseases and S. ventralis on A. concolor has been investigated by examining the root systems of large samples of S. ventralis-infested trees and determining the incidence and degree of infection by pathogenic fungi (Table 9.3). Most Abies in these samples exhibited extensive root system colonization by the pathogens. The trees undoubtedly had been infected for considerable periods of time before being infested by S. ventralis. Cobb et al. (1973) investigated a sample of A. concolor with tops killed by S. ventralis and determined that 40% of the live, beetle-damaged trees were root-diseased. It is evident that root diseases predispose A. concolor to S. ventralis infestation. Under normal circumstances, they appear to be critical to maintaining S. ventralis populations when at low densities. Many uninfected trees are infested, however, when there are other weakening fac-
Effects of Pathogens and Bark Beetles on Forests

Table 9.3. Associations between root pathogens and *Scolytus ventralis* in the western United States

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Location</th>
<th>% beetle-infested trees with disease</th>
<th>Root pathogens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miller &amp; Partridge (1974)</td>
<td>Idaho</td>
<td>98</td>
<td><em>Armillaria, Phellinus</em></td>
</tr>
<tr>
<td>Hertert et al. (1975)</td>
<td>Idaho</td>
<td>96</td>
<td><em>Armillaria, Phellinus</em></td>
</tr>
<tr>
<td>Cobb et al. (1973)</td>
<td>California</td>
<td>92</td>
<td><em>Armillaria, Heterobasidion</em></td>
</tr>
<tr>
<td>Lane and Goheen (1979)</td>
<td>Oregon, Washington</td>
<td>86</td>
<td><em>Armillaria, Phellinus, Heterobasidion</em></td>
</tr>
<tr>
<td>James and Goheen (1981)</td>
<td>Colorado</td>
<td>81</td>
<td><em>Armillaria, Heterobasidion</em></td>
</tr>
</tbody>
</table>

Tors such as major droughts or severe defoliation by insects such as *Choristoneura occidentalis* and *Orgyia pseudotsugata* (Berryman and Wright, 1978; Chapter 4).

9.2.5.3 Timber losses

Chronic mortality and growth losses due to root diseases and associated *S. ventralis* are common throughout western North America. Few large-scale damage assessments have been done, but it is believed that losses are great in a high proportion of mixed conifer stands. In surveys of several individual stands in Oregon and Washington, mortality losses of 4–55% of the trees and 7–33% of the volume have been reported (Filip and Goheen, 1984). Numerous unpublished surveys by USDA Forest Service, Forest Pest Management, Pacific Northwest Region, show similar or even higher losses in eastern Oregon and Washington stands.

Damage is particularly substantial in stands where *A. concolor* comprises 10% or more of the overstory and where one or more selective harvest entries have occurred (Schmitt et al., 1984). In surveys of 192 randomly selected stands on the Ochoco and Fremont National Forests in eastern Oregon, Schmitt et al. (1984) found that 13% of *A. concolor* in all stands had been killed by *P. weirii*, *Armillaria* sp., *H. annosum*, and *S. ventralis*. Survey plots that contained trees affected by the root disease–beetle association showed a 45% reduction in basal area compared to unaffected plots.

In the northern Rocky Mountains, surveys show that about 1% of the commercial forest land is composed of large active root disease pockets and another 13% contains numerous small scattered infection centers (James et al., 1984). Root disease centers are most common in mixed-conifer types, and the *A. grandis*–root disease–*S. ventralis* association is the cause of a substantial amount of the mortality.

9.2.5.4 Ecological effects

In this century, fire exclusion policies have favored development of late successional communities in much of the *Abies grandis* zone (Franklin and Dyrness, 1969) and comparable mixed-conifer forest types at mid-elevation in interior western North America. *Abies concolor* and *A. grandis* establish themselves readily on all but the coldest and driest sites and grow rapidly. *Abies* spp., however, are very prone to pest problems. In addition to root diseases and *S. ventralis*, they suffer significant predisposing damage from stem decays and defoliating insects, especially *Choristoneura occidentalis* and *Orgyia pseudotsugata*.

Root disease and *S. ventralis*, acting in concert, selectively kill *A. concolor* and *A. grandis*. If *Phellinus weirii* or *Armillaria* sp. are the root pathogens involved, *Pseudotsuga men-
Seral tree species, such as *Pinus* spp. and *Larix occidentalis*, are rarely damaged, and mature individuals may be favored in mixed-conifer stands with root disease (Fig. 9.4). It is uncommon, however, for *Pinus* spp. or *L. occidentalis* to establish themselves from seed in mortality centers. These species typically germinate on bare soil in open areas following fire. In the absence of fire or other catastrophic disturbance, *A. concolor* and *A. grandis* usually regenerate in disease centers, ensuring a continuous progression of regeneration, reinfection, and mortality within diseased stands. The accumulation of dead wood increases the probability of stand replacement wildfire and the opportunity for recolonization of the site by seral tree species. Disease centers may provide superior wildlife habitat because of the response of understorey vegetation to the stand opening. *Abies concolor* and *A. grandis* snags provide only short-term benefit to cavity-using birds and mammals, however, because of their rapid deterioration.

9.2.6 Interactions in eastern forests

*Dendroctonus frontalis* is the most devastating forest insect in *Pinus* forests of the southeastern U.S.; populations also are found in Arizona, Mexico and Central America (Payne, 1980). These forests occupy portions of the eastern deciduous forest biome where fire frequency was sufficient to prevent replacement of *Pinus* spp. by hardwoods (Schowalter et al., 1981a). *Dendroctonus frontalis* causes extensive mortality to *Pinus* spp., especially in dense stands composed primarily of *P. taeda* and *P. echinata* (Payne, 1980).

Endemic populations of *D. frontalis* and *Ips* spp. may be maintained in *Leptographium procerum* and *Heterobasidion annosum* infection centers in *Pinus* forests in the southern US (Hicks, 1980; Alexander et al., 1981; Skelly et al., 1981). However, *D. frontalis* is a very aggressive species. Large populations colonize all *Pinus*, regardless of condition or size, within gradually expanding infestations, much like the infection centers of root pathogens (Schowalter et al., 1981b). Schowalter et al. (1981b) reported that *D. frontalis* populations become self-sustaining after reaching threshold sizes of about 100 000 beetles by June. Smaller populations may be unable to reach sufficient attack densities to cause continued tree mortality during the hot summer.

Thinning, harvest, and salvage operations (designed to control *D. frontalis* infestations) facilitate spread of *H. annosum* (Hodges, 1969). These organisms interacting in southern forests have caused widespread and severe timber losses (Payne, 1980; Leuschner and Berck, 1985). Mortality of *Pinus* in mixed pine-hardwood forests also affects forest composition and availability of resources for other species (Leuschner and Berck, 1985).

Reduced canopy cover and transpiration may affect hydrologic processes, but effects of *D. frontalis* on water yield and quality probably are minimal (Leuschner and Berck, 1985). However, altered canopy coverage and species composition likely affect nutrient cycling (Schowalter et al., 1981a), and extensive tree mortality may fuel subsequent wildfire (Coulson et al., 1983, 1985).

Widespread decline of *Pinus resinosa* plantations has become prevalent in the Great Lakes region of eastern North America (Klepzig et al., 1991). This decline is related to interaction among *Leptographium procerum*, *L. terebrantis* and several bark beetle and root weevil vectors. The sequence of pathogen transmission, development of disease symptoms, and colonization by *Ips pini* and its fungal associate, *Ophiostoma ips*, associated with tree death (Klepzig et al., 1991) closely resembles that described above for *Leptographium*–beetle interactions in *Pseudotsuga* and *Pinus ponderosa* forests.
9.3 CONCLUSIONS

Root disease fungi and bark beetles occur together in predictable associations by host and geographic region throughout the coniferous forests of North America. The insects and fungi emphasized in this chapter are all native species acting in relationships that have evolved with the conifer forest habitat. Tree killing is an essential part of the life history strategy of these organisms.

Despite the close association between fungus and insect in most of our examples, the relationship is primarily opportunistic, not symbiotic. Both beetles and root pathogens lead independent lives, each without any requirement for the other. In most cases, the root pathogens weaken trees, which are then vulnerable to attack by bark beetles. These relationships are especially important when beetle populations are low. During outbreaks, the beetles can attack and kill apparently healthy trees. Beetles also may favor root pathogens by killing root-diseased trees before they are windthrown and thereby maintaining inoculum in the soil. Spores of *Leptographium wageneri* are transported on emerging adult root-feeding beetles to infection courts on healthy trees.

Comprehensive figures on timber loss are not available, and there is danger of significant overestimation if losses attributed to individual pathogens and beetles simply are added for a total. However, we estimate that 600 000 m$^3$ of timber volume are lost annually to root disease/bark beetle associations in western North America. This excludes the losses from bark beetles acting alone during outbreaks. There are additional significant losses resulting from death of immature trees, loss of productive land to non-commercial species, and growth loss of trees damaged but not killed.

Root diseases and bark beetles have profound impacts on forest community structure, composition, and succession. Specific effects depend on whether trees are killed and remain standing or fall over, on the pattern of mortality in the forest, on the rate of spread, and on the forest type. In general, these organisms create discontinuities in canopy structure and function, with gaps of altered light availability, temperature and humidity, evapotranspiration, and interception of precipitation.

Plants and animals respond to the altered environment resulting from tree death. Increased light on the forest floor is probably the most immediate change, but temperature and humidity fluctuation also increases. Nutrient availability increases as nutrients stored in wood are released during decomposition, which is stimulated by bark beetles and other wood borers mining the wood and inoculating it with saprophytic microorganisms. Soil fertility may be enhanced over the long term. In most situations, the diversity and cover of understory vegetation increases in mortality centers, at least until a new overstory canopy is established. However, tree mortality also may increase the probability of subsequent stand-replacement wildfire.

REFERENCES


Bloomberg, W.I. and Beale, J.D. (1985). Relationship of ecosystem to...


Lawson, T.T. and Cobb, F.W., Jr. (1986). Stand and site conditions associated with mortality by Verticicladiella wageneri in Mendocino County, California. Phytopathology 76, 1058.


Beetle–Pathogen Interactions in Conifer Forests

Edited by

T.D. SCHOWALTER AND G.M. FILIP
Oregon State University, Corvallis, USA