ABSTRACT OF THE THESIS OF

Kristen L. N. Fields for the degree of Master of Science in Forest Science presented on November 11, 2003.
Title: Impact of Armillaria and Annosus Root Diseases On Stand and Canopy Structure, Species Diversity, and Down Woody Material in a Central Oregon Mixed-Conifer Forest

Abstract approved:

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White and grand fir are both valuable components of the mixed-conifer stand structure managed for late-successional reserves in central Oregon. However, they are often short-lived species because of high susceptibility to root diseases, defoliating insects, bark beetles, and wildfire. This study focuses on the effects of root diseases caused by *Heterobasidion annosum* and *Armillaria* spp. on the stand and canopy structure, understory forb and shrub species diversity, and fuel loadings (coarse woody material) in high elevation late-successional reserves 10 years after a severe western spruce budworm outbreak.

The study is based on plots established in the late 1990s for the White Fir Administrative Study on the Sisters Ranger District, Deschutes National Forest. Field data were collected during the summer of 2001 on 25 quarter-ha plots with varying levels of root disease. Data analyses were done using regression techniques.

There was a significant positive relationship between the amount of root disease (as measured by infected basal area) and the mortality of white fir and

Douglas-fir. The large (>60 cm dbh) Douglas-fir and ponderosa pine component significantly increased in density in highly infected areas. There was a strong positive relationship between fuel loadings (coarse woody material) and the amount of root disease. There was a significant negative relationship between amounts of canopy cover and root disease; however, this was varied by canopy layer. The level of root disease was positively correlated with elevation. This could potentially arise because of the increase in available moisture and a subsequent increase in white fir with increased elevation. An elevation gradient also directly influenced the shrub and herbaceous species composition. However, the relationship between understory species composition and root disease was not clear.

Two species of Armillaria, *A. ostoyae* and *A. gallica*, were found in the study area. This is the first documentation of *A. gallica* in central Oregon and is also the first documentation of this species on white fir in Oregon.

Impact of Armillaria and Annosus Root Diseases On Stand and Canopy Structure, Species Diversity, and Down Woody Material in a Central Oregon Mixed-Conifer Forest

By
Kristen L.N. Fields

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APPROVED:

Major Professor, representing Forest Science

Head of the Department of Forest Science

Dean of the Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University. My signature below authorizes release of my thesis to any reader upon request.

Kristen L.N. Fields, Author

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INTRODUCTION

Maintaining and managing mixed-conifer stands in Late Successional Reserve (LSR) habitats in central Oregon requires an understanding of the role of white and grand fir and their disturbance agents in these complex ecosystems. LSRs are designated areas on federal lands that are mandated under the Northwest Forest Plan to be managed for old-growth habitat (USDA For. Serv. 1994), which in this case are favorable sites for the northern spotted owl (*Strix occidentalis caurina*). White and grand fir are integral species for achieving the desired canopy cover and structure required for late successional species habitat. However, accomplishing long-term management objectives that require maintaining white or grand fir as an integral component of the stand can be difficult.

While they are climax species in many forest types, true firs (*Abies* spp.) in central Oregon, are a relatively short-lived genus in comparison to the early seral species such as ponderosa pine and western larch. True firs are prone to attack by several different insects and diseases, including annosus (*Heterobasidion annosum* (Fr.)Bref.) and Armillaria (*Armillaria ostoyae* (Romagn.) Herink (*A. mellea sensu lato*)) root diseases, western spruce budworm, (*Choristoneura occidentalis* Freeman), and fir engraver beetle (*Scolytus ventralis* LeConte). Thus, management actions designed to improve forest health conditions must focus on the interaction of

annosus and Armillaria root diseases with other insects and diseases and their impact on creating current and future stand conditions.

Management practices, drought, and several species of insects and diseases can adversely affect the health and longevity of white and grand fir. White and grand fir have most of their fine roots located in the upper soil horizons, which make them more susceptible to damage from heavy equipment and lethal fire temperatures (Vogt and Grier 1981, Filip and Schmitt 1990, Petaisto et al. 1999). The root pathogenic fungus *A. ostoyae* can kill trees, especially true firs that have suffered from drought, overstocking, defoliation, and injuries, particularly to roots (Wargo and Shaw 1985, Hadfield et al. 1986, Shaw and Kile 1991). Tree species with non-resinous wood, such as white fir, are very susceptible to decay, especially caused by *H. annosum*, following mechanical injury (Aho et al. 1987, Filip et al. 1995b). In addition, many stands of white fir are infested with white fir dwarf mistletoe (*Arceuthobium abietinum* ex. Munz f. sp. *concoloris*), which causes branch mortality, tree growth loss, and predisposition to fir engraver beetles (Filip and Goheen 1982, Filip 1994).

These mixed-conifer stands have developed from centuries of root disease, wildfire, and other natural disturbances. However, since Euro-American colonization, these stands have been altered. Over the past century, selective harvesting of ponderosa pine and Douglas-fir and fire suppression activities have created conditions favorable to white and grand fir regeneration in central and eastern Oregon (Wickman 1992, Agee 1994). The increase in the amount of true

firs has led to the potential increase in occurrence and expression of root and stem diseases (Hagle and Schmitz 1993, Filip et al. 1995b, Ferguson et al. 2003) and has contributed to the overall decline in the health of mixed-conifer stands (Filip 1994). Because of the high susceptibility of white and grand fir to insects and diseases and past fire exclusion practices (Filip 1994), LSRs in central Oregon are at risk of losing their existing desired stand structures.

The primary root pathogens affecting LSRs in central Oregon are Armillaria and annosus root diseases. Both diseases occur worldwide and affect many different coniferous and deciduous hosts. In central Oregon, *Abies concolor* (Gord. &Glend.) Lindl. Ex Hildebr. (white fir), *Abies grandis* (Dougl. ex D. Don) Lindl. (grand fir), *Abies lasiocarpa* (Hook) Nutt. (subalpine fir), *Pseudotsuga menziesii* (Mirb.) Franco (Douglas-fir), *Picea engelmannii* Parry ex Engelm.(Engelmann spruce), *Pinus ponderosa* Dougl. ex Laws. (ponderosa pine), *Pinus contorta* Dougl. ex. Loud. (lodgepole pine), *Calocedrus decurrens* [Torr.] Florin (incense-cedar), and *Larix occidentalis* Nutt.(western larch) have varying levels of susceptibility.

Further research is needed to understand the impact of these diseases on mixed-conifer stands and to improve the ability to maintain 20-30% true fir in mixed-conifer stands to meet long-term management objectives in central Oregon (Petaisto et al. 1999). This research needs to start with an understanding of how insects and diseases impact the mixed-conifer stand structure and fire risk. Over a decade of attack by fir engraver beetle and Douglas-fir bark beetle (*Dendroctonus pseudotsugae* Hopkins), defoliation by western spruce budworm and Douglas-fir

tussock moth (*Orgyia pseudotsugata* McDunnough), growth loss and subsequent mortality from white fir and Douglas-fir dwarf mistletoe (*Arceuthobium douglasii* Engelm.), and root and stem decay by Armillaria and annosus root diseases have resulted in as much as 50% tree mortality in some mixed-conifer stands on the Deschutes National Forest (Eglitis 1991, 1992; Cochran 1998; Petaisto et al. 1999). With this mortality comes an increased fire risk in LSRs. Understanding the relationships of these insects and diseases to the current and future stand structure is essential for land managers in developing management plans and silvicultural prescriptions.

Root diseases have the potential to change the stand structure, understory vegetation, fuel loadings, and woody material in the mixed-conifer stands of central Oregon. My research focused on these changes in stands containing *A. grandis* and *A. concolor*. Since these two species often hybridize in central Oregon (Foiles et al. 1990), they are not differentiated in this study and will be considered as white fir. Specific objectives include determining the effects of Armillaria and annosus root diseases on stand and canopy structure, species diversity, and woody material 10 years after a severe western spruce budworm outbreak in LSRs on high elevation sites of the Sisters Ranger District, Deschutes National Forest (Fig. 1.1).

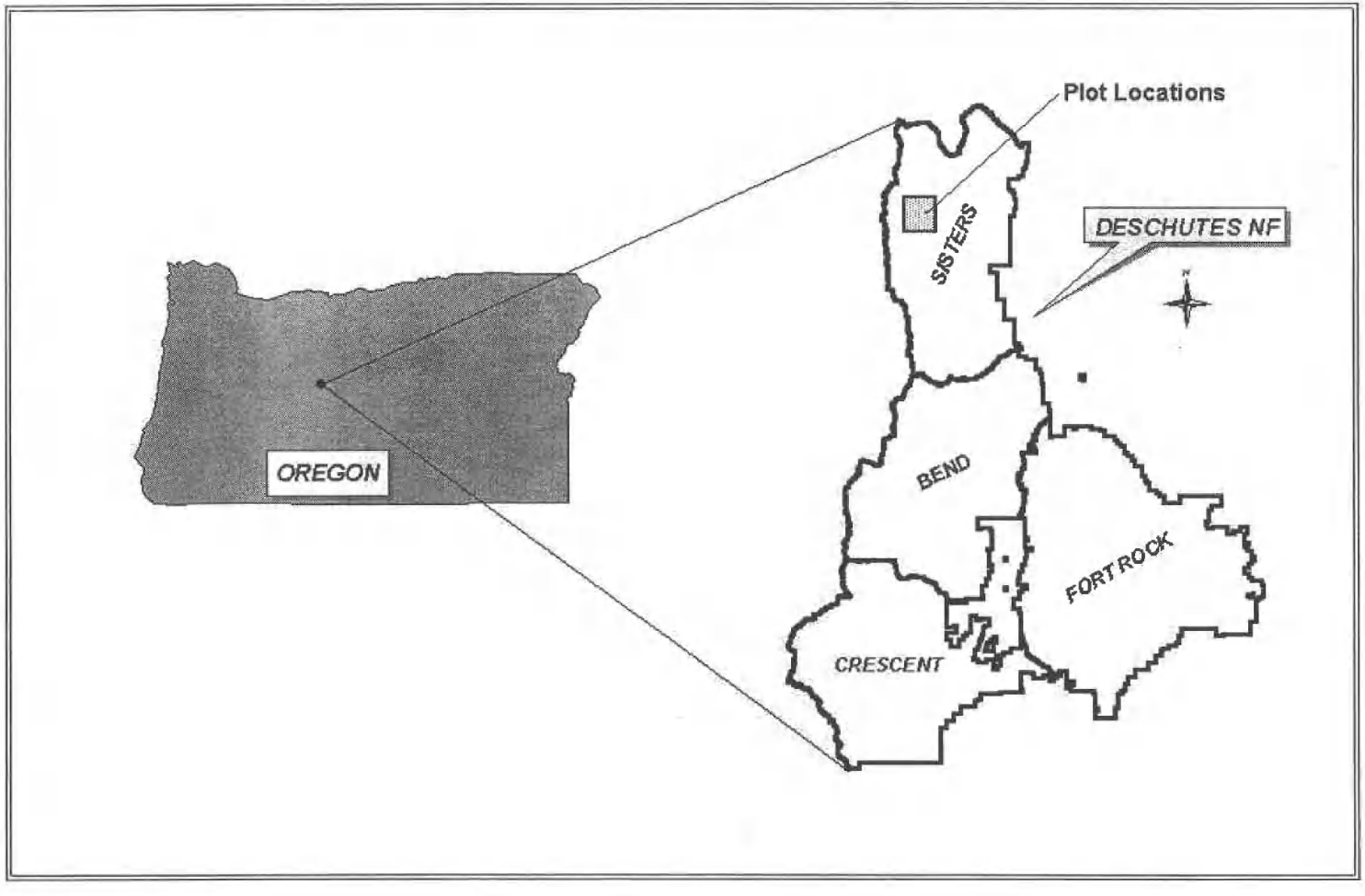

Fig. 1.1: Sisters Ranger District Location Map

Literature review

Ecology of mixed-conifer stands of central Oregon: white and grand fir

The ranges of white and grand fir overlap on the east side of the Cascade crest in central Oregon where they hybridize freely (Hall 1981, Hopkins 1981, Foiles et al. 1990). Franklin and Dyrness (1988) describe this area as the grand fir zone in the *A. grandis-A. concolor* complex. This creates a unique transition zone between the white fir plant associations of the southern Oregon Cascades and the grand fir associations of the northern Cascades. With little difference between the two associations (Mike Simpson, Area Ecologist, Deschutes National Forest, 2003 pers. comm.) in these areas, it has historically been at the discretion of the researcher to define areas as white or grand fir climax (see Cochran 1998).

The mixed-conifer stands of central Oregon primarily comprise Douglas-fir, western larch, ponderosa pine, and lodgepole pine, with white fir and grand fir as climax species (Hall 1981, Hopkins 1981, Foiles et al. 1990, Laacke 1990). Fire suppression activities have favored the climax white and grand fir communities in stands that historically were dominated by Douglas-fir and ponderosa pine (Wickman 1992, Agee 1994). These Douglas-fir and ponderosa pine stands were historically maintained by low- to moderate-intensity wildfires (Agee 1993, 1994). However, fire suppression and selective harvesting have favored the establishment of late seral true fir species.

White fir is an aggressive shade-tolerant tree species and historically occurred as a co-dominant and/or intermediate at lower elevations (Laacke 1990,

Agee 1994). Frequent natural fires maintained this historical structure (Laacke 1990, Agee 1994). Today, white and grand fir typically dominate these mixed-conifer forests. There are a few sites in central Oregon where grand fir and white fir grow in pure stands; however; they are more commonly part of the mixed-conifer forests (Hopkins 1981, Foiles et al. 1990, Laacke 1990).

White fir in central Oregon is found on both wet and xeric sites. The wet white fir sites historically experienced longer fire return intervals than the xeric sites (Agee 1994). These wet white fir sites are differentiated from the xeric sites by the presence of either starflower (*Trientalis latifolia* Hook.) or Queen's cup (*Clintonia uniflora* [Menzies ex J.A. & J.H. Schultes] Kunth) in their understories.

Elevation influences east of the Cascade crest

The forests of the eastern Cascades change in species composition and community structure with changes in altitude and distance from the crest (Cobb et al. 1993). Elevation on the east side of the Cascades is a surrogate for the amount of available moisture. The east side of the Cascade Range experiences a rain shadow with approximately 165 cm of annual precipitation at 1310 m elevation and 46 cm at 975 m (approximately spanning the elevation range of this study) (West 1969). In other areas, elevation also is an excellent predictor of the general pattern of forest habitat types (Riegel et al. 1990). Mean annual precipitation has been used for hazard-risk rating white fir stands for mortality primarily from fir engraver on the Modoc National Forest (see Cochran 1998). In central Oregon this hazard-risk rating corresponds with the elevation gradient. This system rates white fir in stands

at lower elevations as being at high risk and stands at high elevations as being at low risk.

Biology of Armillaria and annosus root diseases

The genus *Armillaria* occurs worldwide and is known for its occurrence on stressed trees (Wargo and Shaw 1985, Filip 1988, Shaw and Kile 1991). In the Pacific Northwest *A. ostoyae* infects several conifer species, and susceptibility is usually related to the site conditions. West of the Cascade crest, *A. ostoyae* is typically an opportunistic saprophytic fungus that often attacks weakened trees (Hadfield et al. 1986). On the east side, however, damage from *A. ostoyae* is common and can continue throughout the life of a stand (Hadfield et al. 1986) as an aggressive tree-killing pathogen (Wargo and Shaw 1985, Shaw and Kile 1991). This species has been known to survive in stumps and root systems for up to 50 or more years (Roth et al. 1980). Infection of adjacent trees occurs by root-to-root contact and by rhizomorphs. Rhizomorphs are fungal structures that grow from established food bases and infect adjacent trees. Armillaria produces mushrooms in the fall; however, spread by spores is thought to be rare in dryer climates (Wargo and Shaw 1985, Shaw and Kile 1991, Ferguson et al. 2003). Armillaria root disease progression and severity vary by geographic area, by site, and by host (Goheen and Otrosina 1998).

Heterobasidion annosum has slightly different characteristics. There are two intersterility groups, s-type and p-type, found in central Oregon (Sullivan et al. 2001). The s-type infects true firs, Douglas-fir, and several other conifers. The p-

type infects mainly ponderosa pine, larch, and incense-cedar (Woodward et al. 1998). In this region, white fir and grand fir are highly susceptible and readily killed by *H. annosum* (Hadfield et al. 1986). While pines are not susceptible to the s-type, true firs can be susceptible to the p-type (Allen et al. 1996, Woodward et al. 1998). In Oregon and Washington, Douglas-fir (mostly var. *menziesii*) is listed as being slightly susceptible and rarely damaged (Hadfield et al. 1986). However, in the Rocky Mountains, Douglas-fir (var. *glauca*) is highly susceptible and readily killed by *H. annosum* (Goheen and Otrosina 1998).

H. annosum is spread by both root-to-root contact and wind-blown spores. Wind-blown basidiospores from the fruiting bodies can infect freshly cut stumps and wounded boles of trees. When the spore lands on a stump or wound it germinates and forms mycelia that colonize the wood. Once the fungus colonizes the stump it spreads through the root system and may infect adjacent trees by mycelial contact along the infected roots. The larger stumps may be viable inoculum sources to infect surrounding trees for as long as 50 years (Woodward et al. 1998).

Identification and diagnosis of Armillaria and annosus root diseases

Armillaria root disease is easily recognized in the field. It is identified by white mycelial fans present in or under the bark in the cambial zone on the roots or root collar of infected trees. Black rhizomorph structures may be found emerging from the root surfaces. The fungus produces honey-colored mushrooms near the base of infected trees in the fall. However, the production of sporocarps is

infrequent and not easily used as an identification tool. Individual tree symptoms include crown thinning or changing color from green to yellow or red. There is also a possibility of a stress crop of cones on trees that are slowly weakened over time. In some instances there is resin flow from the base of the tree. In white fir this flow may resemble water-soaked bark. Affected trees are often found in groups or patches on the east side of the Cascades. The incipient decay is yellow to brown in color and has a water-soaked appearance (Allen et al. 1996), then the wood becomes very stringy in the later stages of decay.

Annosus root and butt rot is more difficult to identify. The fruiting bodies are perennial, leathery, and vary in shape and size. They are difficult to find and are only present in the advanced stages of decay. These conks are usually located around the root collar of standing dead trees, inside hollow stumps, or on the underside of windthrown trees. The incipient decay of annosus root disease is yellow- or red-to-brown. The advanced stages of decay are white, and the wood is a spongy, stringy mass that may have black flecks running with the grain of the wood (Allen et al. 1996). The wood may delaminate with small, elongated pits on one side of the laminated sheet (Sullivan 1997).

Impacts of root disease on forest stands

Armillaria and annosus root diseases and their biology, interactions with other major pathogens and insects, impacts on volume loss, and control have been extensively studied. Research has focused on many different aspects of both root diseases, from the genetic structure of the different species to the effects of

harvesting (Shaw and Kile 1991, Woodward et al. 1998). Tree mortality from root diseases causes significant loss in timber volume in the Pacific Northwest (Childs and Shea 1967, Filip and Goheen 1984, Filip and Schmitt 1990, Cochran 1998). However, there are information gaps in our understanding about how these root diseases impact the stand and canopy structure and understory vegetation. Impacts of fungal root pathogens on the community structure and development are only starting to be understood (Holah et al. 1993, 1997). We can hypothesize that damage caused by these fungi leads to an increase in fuel loadings and fire risk (Filip 1994); however, this has not been proven.

In response to the abundance of true fir species in current stands, management of these species is increasing (Hubert 1955, Filip and Schmitt 1990, Filip 1994). This interest in management of true firs creates a need for more information on the associated insects and diseases that impact *Abies* species.

Filip and Goheen (1984) examined 14 stands (2,750 ha) of white and grand fir in the Pacific Northwest for three root pathogens, *A. ostoyae*, *H. annosum*, and *Phellinus weirii* (Murrill) R. L. Gilbertson, and their related mortality. They found that losses from root disease varied from 4-55% of the trees, with 19% occurring on their Cache Mountain site and 14% on the Warm Springs Indian Reservation. Basal area losses ranged from 8 to 39% with 21 and 17% at the Cache Mountain and Warm Springs sites, respectively. Volume losses were between 7-33% with 22% losses at Cache Mountain and 18% on the Warm Springs Indian Reservation. All

three root pathogens were found on the Cache Mountain site, while *P. weirii* was the primary pathogen on the Warm Springs site.

Filip and Goheen (1982) studied the effects of the major root pathogens (*A. ostoyae, H. annosum, P. weirii,* and *Leptographium wageneri* (Kendrick) M. J. Wingfield) on stocking and volume of conifers. In central Oregon they found that one or more of the major root pathogens had killed 21.6% of the merchantable wood volume during the previous 20 years.

Changing management practices and the associated increases in true fir densities, commonly produce increases in disease expression as well as changes in stem disease causal agents. The Indian paint fungus, *Echinodontium tinctorium* (Ellis & Everh.) Ellis & Everh., is one of the primary causes of stem decay in true firs and has been found to cause up to 80% of the decay in old-growth stands of grand fir in the Blue Mountains (Aho et al. 1987). However, Filip et al. (1992) found that, where stands were shifting from old-growth grand fir to young regeneration, an associated shift from Indian paint fungus to *H. annosum* as the major cause of stem decay was occurring. With our changing management practices comes a change in the role, impact, and expression of these root diseases.

Predisposition to *A. ostoyae* and *H. annosum*

There are several management practices and site conditions that predispose mixed-conifer stands to *H. annosum* and *A. ostoyae*. It is hypothesized that mixed-conifer forests are more severely damaged by Armillaria and annosus root diseases after harvesting and in intensively managed stands (Filip 1977, Filip and Goheen

1982, Filip and Goheen 1984, McDonald et al. 1987a, Filip 1990, Morrison and Mallett 1996). The creation of stumps by extensive logging of conifer forests has favored the establishment of *H. annosum* in stands where it may have originally been rare (Garbelotto et al. 1999). In areas where large-diameter, live-infected trees have been harvested, a greater amount of *A. ostoyae* inoculum may result because the fungus can rapidly colonize the stumps and root systems (Filip 1977, Filip and Goheen 1982, Morrison and Mallett 1996). Harvesting of trees affected by laminated root disease has been found to leave larger amounts of inoculum in the soil than in areas where previously trees would have been up rooted; thus, reducing the amount of inoculum (Thies and Sturrock 1995). This can also occur in Armillaria- and annosus-infected stands. In these areas, historically, the diseased tree may have been up rooted, causing removal of the stump and many of the larger supporting roots and reducing the amount of inoculum on the site. Silvicultural prescriptions that leave true firs in the overstory have a high risk of causing annosus stem and root decay if bole wounding occurs during harvesting activities. Resulting mortality from annosus root disease in residual trees is possible if *H. annosum* infects stumps or wounds and spreads through root contact.

Fire suppression activities have led to an abundance of susceptible hosts within mixed-conifer stands (Filip 1994). Increases in stand densities and a shift to climax species such as true firs leads to a decrease in individual tree vigor. These changes can increase the effects and expression of annosus and Armillaria root diseases by leaving susceptible hosts and increasing stand density, possibly allowing

for root pathogens to spread more rapidly though stands (Hagle and Schmitz 1993). The theory that changes in the stand structure and species composition have increased the area colonized by Armillaria root disease is subject to some debate (Ferguson et al. 2003).

Changing management practices have increased the number of entries into stands as we move away from even-age management systems. As the number of entries into a stand increases, the incidence of annosus root disease increases (Korhonen and Stenlid 1998). However, others (Schmitt et al. 1994) have found that it may be more a function of time since last entry than the number of entries into a stand.

Environmental conditions

The severity of Armillaria root disease has been found to be largely dependent on environmental conditions such as soil properties (Mallett and Maynard 1998) and site conditions (McDonald et al. 1987a, 1987b). Soil properties such as pH level, nutrient composition and level, texture, and moisture content have been found to positively or negatively influence the expression or incidence of mortality due to Armillaria root disease (Mallett and Maynard 1998). The influence and importance of these different soil properties has varied greatly among studies and tree species. Mallett and Maynard (1998) concluded that conifer stands on coarse-textured soils, such as very sandy soils, were at an increased risk for mortality caused by Armillaria root disease. Their study found that sand content was the most important factor in lodgepole pine mortality from Armillaria root

disease. They also found that stand characteristics such as density, height, age, and elevation had no influence on the incidence of Armillaria. Shields and Hobbs (1979) found that mortality from Armillaria root disease in Douglas-fir stands was correlated with low soil nitrogen and pH, and in grand fir stands it was correlated with low soil calcium and phosphorous and high soil potassium in Idaho.

Site conditions also play a large role in determining if Armillaria is present and causing mortality in stands. In the northern Rocky Mountains, McDonald et al. (1987a) demonstrated that the distribution of *Armillaria* spp. was related to habitat type. McDonald et al. (1987a) concluded that the pathogenic behavior of Armillaria is largely dependent on habitat type and stand development history. They found that the incidence of pathogenic Armillaria was higher in habitat types with lower productivity in the northern Rocky Mountains. They then assumed that the pathogenic observations in their study were solely caused by *A. ostoyae*, which suggested that there is variation in the pathogenicity of *A. ostoyae* and that this is closely linked to site productivity, host adaptation, or host stress.

Silvicultural methods to reduce impacts of root diseases

Host species resistance

Conifers have varying levels of susceptibility to both *H. annosum* and *A. ostoyae*. In central Oregon, white fir are generally considered highly susceptible to both diseases, while Douglas-fir is moderately susceptible to *A. ostoyae* and is not found to be a host to *H. annosum*. Ponderosa pine is considered moderately

susceptible, but this can be site dependent. Western larch is considered resistant, and incense-cedar is generally thought of as rarely infected or immune (Hadfield et al. 1986, Morrison and Mallett 1996, Robinson and Morrison 2001). Morrison and Mallett (1996) consider all conifers in British Columbia to be moderately to highly susceptible to Armillaria root disease until the age of 12-15. Robinson and Morrison (2001) compared lesion formation and host response of 6-8- and 18-19-year-old Douglas-fir and western larch from infection by *A. ostoyae*. They found the young trees of both species to be very susceptible. Older trees were more resistant with the host forming necrophylactic periderm (Mullick and Jensen 1973) in advance of the fungal infection. In the older trees western larch formed necrophylactic periderm more often than Douglas-fir; and was therefore considered to be more resistant. However, in a shade house study with inoculated seedlings, Omdal et al. (1995) found that host susceptibility among 7 conifers varied greatly over the length of their 30-month study.

The knowledge of tree susceptibility can aid in reducing the impacts of mortality. Planting resistant species in root disease pockets is one management tool (Filip and Schmitt 1990, Shaw and Kile 1991, Morrison and Mallett 1996, Woodward et al. 1998, Sullivan et al. 2001). When sites are to be planted after harvesting, planting resistant or less susceptible species will reduce the mortality rates of the regeneration. Species such as western larch and western white pine (*Pinus monticola* Dougl.ex D. Don), in the absence of white pine blister rust (*Cronartium ribicola* J.C. Fisch.), can help to increase stand diversity since both are

more resistant to the s-type of *H. annosum* and to *A. ostoyae*. Ponderosa pine and Douglas-fir are also recommended for planting on some sites; however, their susceptibility is site-dependent. In general, silvicultural practices should favor early seral species.

Precommercial and commercial thinning

Precommercial thinning in stands has been studied in relation to root diseases and stem decays. Precommercial thinning has been shown to reduce damage to residual trees from defoliating insects, stem decays and root diseases (Filip et al. 1992, Filip 1994, Filip et al. 1995b, Filip et al. 1999). Increasing individual tree vigor through precommercial thinning in central and eastern Oregon generally does not leave stumps large enough for annosus to develop (Smith 1970, Filip and Schmitt 1990). Filip and Goheen (1995) stated that mortality caused by annosus root disease in precommercially thinned stands of Douglas-fir or true fir needs further investigation.

Precommercial and commercial thinning has had both beneficial and detrimental effects on increasing survival of residual trees with Armillaria root disease in several studies. Thinning, while not impacting mortality rates between thinned and unthinned stands (Filip and Goheen 1995), has been shown to increase individual tree vigor. Filip et al. (1995b) found that wounded white and grand fir trees in thinned stands react differently than wounded trees in unthinned stands. Thinning tended to increase tree vigor and diameter growth for several years after infection. They also found that the amount of stem decay in grand fir was lower

after wounding in thinned stands as compared to unthinned stands. Therefore, they concluded that management of white and grand fir stands should include thinning practices. However, wounding in these stands should be minimized. Filip and Goheen (1995) found that thinned Douglas-fir, hemlock, and true fir stands on the west side of the Cascades exhibited no significant differences from unthinned stands in mortality caused by Armillaria root disease. However, they did find that tree radial growth 10 years later was significantly increased in thinned stands. Rosso and Hansen (1998) found the opposite, where precommercially thinned plots experienced more mortality from *Armillaria ostoyae* than unthinned plots in the western Cascades. In a 30-year report on thinning in Armillaria-infected ponderosa pine in central Oregon, Filip et al. (1999) found that mortality from Armillaria in unthinned stands was significantly more than in thinned stands. Schmitt et al. (1984) found large amounts of mortality from annosus root disease (15-23% of the true firs were dead) in stands on the Ochoco and Freemont National Forests that had been entered for harvest. This was substantially higher than the incidence of mortality in stands that had not been entered (2%).

Wound prevention

Knowledge of decay incidence after wounding is crucial to reducing the amount of annosus infections after harvesting. Most federal agencies are moving away from the traditional clearcut and shelterwood regeneration systems towards structure, species, and density management. Many of the management objectives are requiring that more true fir species be left in the overstory. Thus, there is

increased potential for wounding. Filip et al. (1992) found that stem decay in true firs following wounding is extremely rapid. They recommended that wound prevention guidelines, such as those in Filip and Schmitt (1990), be used during harvest operations. Sullivan et al. (2001) found that 72% of the wounded trees and 83% of the unwounded trees had decay caused by *H. annosum* in one previously thinned noble fir stand on the Warm Springs Indian Reservation, Oregon. They found that 94% of the wounded trees and 80% of the unwounded trees on another study site had decay.

Stump treatments as management options

Treating stumps to prevent infection by *H. annosum* or to reduce the amount of inoculum can be a practical management tool. The application of boron-containing products (e.g. borax, Sporax, TimBor) to freshly cut stumps to reduce infection by *H. annosum* has been widely used and studied. Boron prevents infection by *H. annosum* in treated stumps (Smith 1970). Filip and Schmitt (1990) recommend that boron be applied to stumps > 30.5 cm in diameter. Stumps < 30.5 cm in diameter need not be treated because they quickly dry out and are not considered potential sources for infection and subsequent spread of *H. annosum* (Smith 1970, Filip and Schmitt 1990). By preventing infection of stumps and the associated spread by root contact, managers may decrease mortality rates of regeneration. However, recent studies in eastern Oregon have found that even with high infection levels of *H. annosum* in stumps 10 to 20 years after harvesting occurred, mortality rates of surrounding regeneration were very low (0.7-1.4%)

(Filip et al. 2000). Mortality rates of true firs after harvesting have yet to be studied in central Oregon. Treatment of stumps with boron in stands where a large portion of the residual overstory is composed of true fir is still recommended to prevent infection and to reduce loss of true fir (Filip et al. 2000).

Methods to reduce the existing inoculum levels have also been studied. The removal of Armillaria- or annosus-infected stumps is a viable option from a biological standpoint and is recommended in high-valued areas (Roth et al. 1980, Roth et al. 2000). However, the associated labor costs make stump removal impracticable in most cases (Roth et al. 2000). Filip and Roth (1977) and Thies and Sturrock (1995) fumigated stumps infected with *A. ostoyae* and *P. weirii*, respectively, and proved it to be a successful fungal eradication tool, although the treatment has not been implemented for actual management purposes.

Root diseases, defoliating insects, and bark beetle interactions

Mortality caused by *H. annosum* and *A. ostoyae* is common east of the Cascade crest. Infection by these two fungal pathogens is a predisposing factor to insect attack and windthrow. More specifically, these root pathogens have been found to increase the susceptibility of white and grand fir to fir engraver beetles (Lane and Goheen 1979, Scharpf 1993). Hertert et al. (1975) found that the numbers of attacks by fir engraver (under endemic population levels) on grand fir in northern Idaho were substantially less on trees with less than 80% decay (the threshold for their definition of "extensive decay"). They found that 87% of the true

fir trees attacked by bark beetles had root systems that were extensively decayed. They also found that bark beetles did not differentiate among root pathogens. Lane and Goheen (1979) found similar results in eastern Oregon and Washington: 85.6% of the true fir trees that were infested with bark beetles were also infected with root pathogens. They also found that most of these trees were colonized by the root pathogens before being attacked by bark beetles.

Defoliated trees are also subject to attack by fir engravers. Wright et al. (1984) found that during a Douglas-fir tussock moth outbreak in the Blue Mountains of eastern Oregon and Washington, trees attacked by fir engravers averaged 83% defoliation. They also found that 50% of the beetle-infested trees were defoliated more than 95%. They sampled six top-killed grand fir within the study area and found that five of the six had been attacked by fir engravers. Top-killed trees had higher densities of bark beetles and higher beetle emergence rates than the killed trees. Wright et al. (1979) found that sugar concentration of the inner bark decreased in the first year and starch concentration decreased in the second year after defoliation of grand fir by the Douglas-fir tussock moth in the Blue Mountains. This lowered monoterpene concentration and increased susceptibility to fir engraver attack.

Defoliation of hardwoods and conifers by insects is known to predispose trees to infection and subsequent mortality from *Armillaria mellea* (*sensu lato*). This interaction has been consistently documented and observed in forest settings around the world (Wargo and Harrington 1991). Colonization by *Armillaria* spp.

after defoliation of oak species by the European gypsy moth (*Lymantria dispar* L.), is common in the eastern United States, and a similar relationship with defoliating insects has been found in oaks in Europe (Wargo and Harrington 1991). Hadfield et al. (1986) noted that mortality in conifers from the Armillaria root pathogens increases in the years after defoliation by western spruce budworm and Douglas-fir tussock moth. Wright et al. (1984) examined the incidence of root disease in defoliated and bark beetle-attacked stands in the Blue Mountains. They found that 54% of the trees that were extensively defoliated >90% had root decay. They also found that all trees infested with fir engraver and with <90% defoliation had root pathogen symptoms. Wargo and Harrington (1991) cited other examples of increased Armillaria infection after insect defoliation, one by eastern spruce budworm (*Choristoneura fumiferana* Clemens) in Canada and one by larch case bearer (*Coleophora laricella* Hubner) in Idaho. Raske and Sutton (1986) found that Armillaria root disease increased from 30-85% when defoliation by spruce budworm exceeded 80% in black spruce (*Picea mariana* (Mill.) B.S.P.).

Conversely, Parks (1994) and Parks et al. (1994) used grand fir seedlings in a greenhouse study to explore these relationships with drought stress. She found that seedlings that had been defoliated by western spruce budworm (both drought stressed and non-drought stressed) were less likely to have successful infection and subsequent mortality by *Armillaria ostoyae.* She concluded that defoliated seedlings had fewer carbohydrates in the roots to support successful infection by *A. ostoyae.* This study suggested that the cumulative effects of drought stress, defoliation, and

infection by *A. ostoyae* may not occur as frequently as thought; however, it is difficult to extrapolate these results to mature trees.

Objectives

The overall objective of my study is to determine the effect of root diseases caused by *Armillaria* spp. and *H. annosum* on stand structure, understory species diversity, and fuel loadings 10 years after a severe western spruce budworm outbreak in Late Successional Reserves on the Sisters Ranger District, Deschutes National Forest. This is accomplished through the following sub-objectives:

1) To determine the relationships between increasing root disease (infected basal area) and the size-class structure of white fir, Douglas-fir, and ponderosa pine at the stand level. My study seeks to determine and define how stand structure changes with increasing levels of root disease by exploring the following question: Does increasing infection by *Armillaria* spp. and *H. annosum* significantly change size-class structure and mortality levels of white fir, Douglas-fir, and ponderosa pine at the stand level?

2) To determine the impact of root diseases on canopy cover and stratification. The impact of root disease on the degree of canopy stratification and the mean foliage height of the forest canopy will be mediated through the mean plot-level live crown ratio (LCR).

3) To determine if understory species richness and total understory cover increase with increasing root disease.

4) To determine if the fuel loading of down wood increases with increasing root disease.

5) To determine the species of *Armillaria* occurring across the study site.

Methods

Study area

The study sites are located on the Sisters Ranger District, Deschutes National Forest and managed by the USDA Forest Service as Late Successional Reserves (Petaisto et al. 1999). The study sites are located north and northeast of Suttle Lake in T13, R8E, Sec 2, 3, 10, 11, 12, 14, 15, and 22; and R9E, Sec 7, 18, and 30 (Fig. 1.2). The study areas ranged from 975-1,400 m in elevation. The study site is in the *A. grandis* zone (*A. grandis- A. concolor* complex) (Franklin and Dyrness 1998) and in the plant association CWC2-11, mixed-conifer/ snowbrush-chinkapin/ brackenfern (Volland 1976) as classified by Petaisto et al. (1999). The soil textures on my study sites are of two types: The southern sites (TS2, 5, 6, 7, and 15 in Fig 1.2) have a deep layer of Blue Lake ash which is coarse and primarily cinder and is approximately 1500 years old; The northern sites have Sand Mountain ash which is a fine ash (sandy loam) and approximately 4000 years old (T. Craigg soil scientist, USDA Forest Service, 2003 pers. comm.). Zero-50% of the area has light to heavy soil compaction as determined by Petaisto et al. (1999) following the methodology of Howes et al. (1983) from multiple entries into the stands and mechanical piling of the slash.

White fir composition across the study area is, on average, 61.4% of the trees > 12.7 cm (5 in) dbh (diameter at 1.4 m above ground) (Petaisto et al. 1999). Other tree species in the study area include Douglas-fir, ponderosa pine, lodgepole pine, western white pine, western larch, incense cedar, subalpine fir, and Engelmann spruce. The mean tree dbh ranged from 38-46 cm (15-18 in).

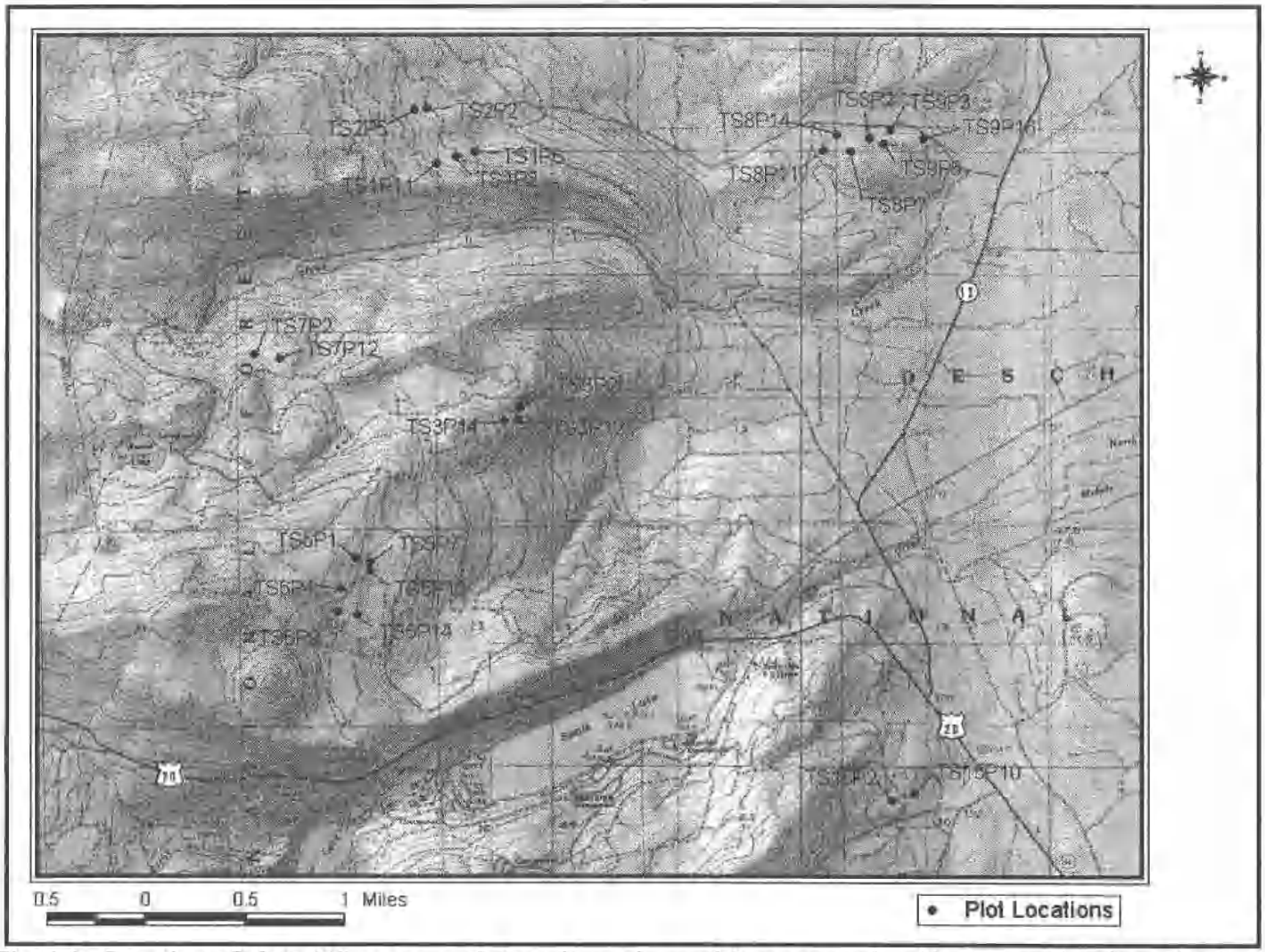

Fig. 1.2: Location of plots. TS corresponds to unit number and P corresponds with plot number from Petaisto et al. 1999.

Pre-existing data

Details of the site and stand characteristics, tree mortality, causal pests, and soil compaction appear in a preliminary report (Petaisto et al. 1999). The White Fir Administrative Study established 167 plots in 12 timber sale units in 1998 and 1999. Preliminary stand data included: dominant plant species (by canopy cover), elevation, aspect, slope (%), site index (ft/50yr), percentage of area with mortality (ocular estimate), crown closure (%), stand structure, and topographic position.

Permanent plots for Petaisto et al. (1999) were established using a 20 or 40 (ft^2) BAF prism for the variable-size plot, which included all trees ≥ 12.7 cm dbh, and a fixed-plot radius of 3.6 m or 0.00405 ha for trees < 12.7 cm dbh. Live and dead tree data were collected. Live tree data included all trees taller than 1.4 m above ground, tree species, dbh, live crown ratio, dwarf mistletoe rating, and tree wounds. Dead tree data included tree species, dbh, years since death, and cause of death. Years since death were categorized as: 0-5 years dead, 6-10 years dead, and 10+ years dead. This was determined by the presence or absence of bark at the root collar (Parks et al. 1997). All stumps >12.7 cm (5 in.) in diameter and dead trees taller than 15.2 cm (6.0 in.) were measured in the fixed plot. Measurements included diameter inside bark for stumps and the dbh of standing trees. The results of the pre-harvest data are summarized in Petaisto et al. (1999).

Petaisto et al. (1999) found high mortality levels of white fir in the higher elevation plots. Mortality agents included Armillaria and annosus root diseases and bark beetles. The amount of mortality due directly to western spruce budworm was

not quantified or noted. The higher elevation white fir also had higher amounts of mistletoe. Annosus root disease was found affecting the white fir across the treatment units.

White fir is susceptible to both Armillaria and annosus root diseases. It is also common for both *Armillaria* spp. and *H. annosum* to occur on the same tree. In a nearby site, Filip and Goheen (1982) found that 17% of dead, felled grand fir had both pathogens.

In central Oregon *H. annosum* has two strains; live ponderosa pine is only susceptible to the p-type (Hadfield et al. 1986) and was not found to be affected by annosus root disease in this study or by Petaisto et al. (1999) in these study plots. However, ponderosa pine stumps can act as inoculum foci for the s-type of *H. annosum*, which in turn can cause mortality of true firs from annosus root disease. *H. annosum* was not included in the infected BA for Douglas-fir because it is rarely found to cause mortality in Oregon (Hadfield et al. 1986).

Site history

The primary disturbances affecting stand composition over the past century have been harvesting, fir engraver beetle, western pine beetle (*Dendroctonus brevicomis* LeConte), Douglas-fir bark beetle, western spruce budworm and Douglas-fir tussock moth defoliation, root diseases caused by *Armillaria* spp. and *H. annosum*, and fire suppression. Before European settlement and fire suppression, it is hypothesized that the fire cycle for mixed-conifer stands in central Oregon was about 30 years (McNeil and Zobel 1980, Atzet and Wheeler 1982, Agee 1991).

However, the Sisters Ranger District has estimated a fire return interval slightly more than this (35 yrs) by interpolating known fire frequencies from the Metolius Basin and the Mt. Jefferson Wilderness (Brian Tandy, Silviculturist, Sisters Ranger District, 2001 pers. comm.). The grand fir zone in the Mt. Jefferson Wilderness has an estimated average fire return interval of 53 years for the last 470 years (Simon 1991). The stands within the study area experienced high mortality rates of 10-50% of the basal area after a complex of root diseases and bark beetles interacted with a western spruce budworm epidemic in the late 1980s to early 1990s (Eglitis 1991, 1992; Cochran 1998; Petaisto et al. 1999).

All of the study stands have been harvested at least once in the past century. In the 1940s and 50s the stands were salvage harvested to remove the larger trees after western pine beetle or Douglas-fir bark beetle attacked, as was evident from the presence of large ponderosa pine and/or Douglas-fir stumps in and around the study plots.

History of defoliation

All stands within the study area experienced severe defoliation from a western spruce budworm outbreak. The defoliation was first detected by aerial survey in 1985 and lasted for 8 years with the peak of the defoliation occurring in 1991 and 1992 (Eglitis 1991, 1992) and the population subsequently crashing in 1993. In 1991 three monitoring units were developed on the Sisters Ranger District. My study area is located in the Jack Creek monitoring unit. In 1991 the Jack Creek area averaged 47.3 (+/- 15.0) western spruce budworm larvae per tree and 0.66 (+/-

0.67) Douglas-fir tussock moth larvae per 1000 in^2 of foliage. These data were collected from lower crown beating. Based on these data, moderate to heavy budworm defoliation in 1992 was predicted (Eglitis 1991).

In 1992 the Jack Creek area had 29.15 (+/- 2.95) western spruce budworm larvae per tree from lower crown beating sampling methods, and 41.2 (+/- 4.3) larvae per pheromone trap. These results again predicted moderate to heavy budworm defoliation in 1993 (Eglitis 1992). However, the defoliation ended in 1993 as the population collapsed. The 8 years of defoliation resulted in some mortality of host species in some stands. In 1992, 72% of the host trees/acre in the Jack Creek area were live without any dieback of the crown, 2 % were live with a bare top (1-10% of the crown), 5% were live with a bare top (>10% of the crown), and 2% of the host trees/acre were dead. In the Suttle Lake monitoring unit (approximately 5 km south of the White Fir Administrative Study units 3, 4, 5, 6, and 7 [Petasio et al. 1999]), forest health survey results indicated that mortality levels were around 35% on some plots with much of the mortality attributed to root diseases (Eglitis 1992).

Eglitis (1992) also noted that in the Jack Creek monitoring unit there was "a high incidence of Armillaria root disease on the Douglas-fir and white fir especially at the higher elevations". The lower elevation areas of defoliation in the monitoring unit had a larger pine component, and the host trees of western spruce budworm were generally younger. Therefore it was predicted that mortality levels would be low in these areas.

Cochran (1998) had white fir levels-of-growing-stock study plots in the Jack Canyon area of Sisters Ranger District. All of the growing stock levels (20, 30, 40, and 50% of normal density) experienced high amounts of mortality (ranging from 4.4 to 22.7 m^2 of the basal area) after defoliation. Cochran noted that all of the dead trees at his Sisters study site showed evidence of Armillaria root disease. He attributed the mortality to the combination of root disease and defoliation by western spruce budworm.

Plot infection level and inoculum index

My study used permanent plots established by Petaisto et al. (1999). In each unit they randomly selected a starting point and then systematically established a grid of plots across the unit. Each unit had from 11-18 plots (depending on the size of the unit) with 3-5 chains (66 ft or 20.1 m per chain) between plots. From their data I calculated inoculum indices (II) (Thies 1986, Filip et al. 1995a) of *H. annosum* and *Armillaria* spp. in 90 plots. Inoculum indexing was used to determine the plot infection level. Armillaria indexing has been done in past studies (Filip et al. 1995a) and is based on live or dead trees or stumps using the equation (Thies 1986):

$$II = D^3 \times C / 1000$$

where:

II = inoculum index for each tree
D = diameter inside bark at 15.2 cm (6 in) above ground for stumps or DBH for trees.
C = years dead: 0-5 years = 1.0, 6-10 years = 0.9, and > 10 years = 0.5

This equation provided a value for each stump or tree. This was calculated for every tree, live or dead, or stump that Petaisto et al. (1999) noted as showing signs or symptoms of one or more root pathogen. I determined inoculum indexes for the variable plots. Therefore, all tree II values were summed to calculate the plot II. Plot II is a unit-free number and is used as an indicator of the present volume of inoculum. In most cases this has been used to predict the future impact of the disease on the stand (Thies 1986, Filip et al. 1995a).

For my purposes, it was more desirable to determine past plot II values, since they affected the current stand condition. Therefore, the equation was manipulated to determine past plot II by giving more weight to trees that had died earlier and thus showing a greater impact on the current stand characteristics. In this case, C was manipulated so that 0-5 years = 0.5, 6-10 years = 0.9 and > 10 years = 1.0. This estimated past stand inoculum levels that may have created the current stand conditions.

Plot layout

The plots were divided into low, medium, and high levels of root disease by their plot II. Ten plots per infection level were chosen using a random number table. This was done to stratify the plots across levels of inoculum. I established one 0.25-ha square permanent plot centered on each of the 30 randomly chosen permanent plots established by Petaisto et al. (1999). For ease of measurement, each 0.25 ha plot was divided into 16 subplots (Fig. 1.3). All live and dead trees >15.0 cm dbh were measured on the large plot (0.25 ha). Within each 0.25-ha plot,

I subsampled trees of 5-15 cm dbh on five medium-size plots (4 m radial circular plot) and trees less than 5 cm dbh on 10 small plots (1 m^2 square plots) (Table 1.1, Fig. 1.3).

Table 1.1: Plot size and data collection characteristics.

Plot Size	Data Collection within plot	% sample	# of plots per 0.25 ha plot
0.25 ha	Trees > 15 cm DBH	100%	1
	Coarse woody material > 15 cm large end diameter		
50 m^2 (4 m radial plot)	Trees 5- 15 cm DBH	10.05%	5
	Coarse woody material 5-15 cm large end diameter		
	Shrubs - ocular estimate of % cover		
1.0 m^2	Trees < 5 cm DBH	0.8%	10
	Small woody material 1-5 cm large end		
	Herbs- all species % cover		

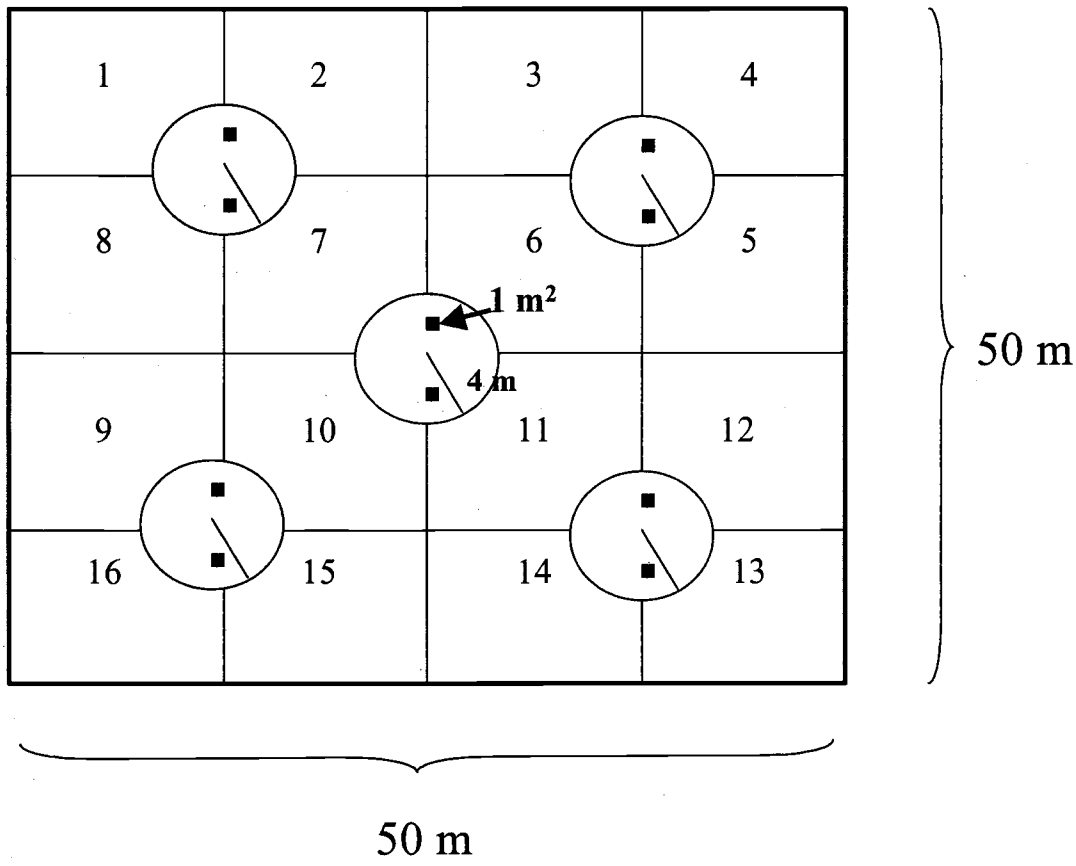

Fig. 1.3: Plot layout

Data collection

In the summer of 2001, data were collected for twenty-five 0.25 ha plots (five of the original plots were not sampled because of time constraints). Data included tree, canopy, and understory variables within each plot. In the 0.25 ha plot, live-tree variables included tree species, dbh, live crown ratio (LCR), tree height, crown base height (to a packed crown), presence of epicormic branching, presence of top-kill, and dwarf mistletoe rating. For dead trees, data included tree species, dbh, years dead, and mortality factors specifically focusing on Armillaria and annosus root diseases, Douglas-fir bark beetles, and fir engraver beetles. Insect data were collected because of the interactions of insect attack on trees with root disease and recent insect outbreaks.

All dead trees were checked at or below the root collar for presence of root pathogens. Only live trees showing symptoms of root disease, or trees on the edge of infection centers were checked for root pathogens. They were examined at the root collar just below the bark for the presence of mycelial fans. If mycelial fans of Armillaria were not present, then adjacent stumps or windthrown trees were examined for signs of annosus decay. The plot II and the infected BA were calculated for the entire plot following data collection in 2001. The infected BA was determined for *H. annosum* and *Armillaria* spp. for each plot. This was calculated by summing the BA of all live and dead infected trees on the plot.

Many of the stands were infected with white fir dwarf mistletoe that could potentially become a confounding variable when analyzing the canopy attributes.

Therefore, the presence of dwarf mistletoe and the dwarf mistletoe rating were quantified using Hawksworth's dwarf mistletoe rating system (Hawksworth 1977), on a scale of 0-6 where 0 indicates the absence of mistletoe and 6 indicates severe infections by mistletoe.

The size-class structure was divided into four diameter-size classes: tall-emergent = >60 cm dbh, emergent = 30-59.9 cm dbh, dominant = 15-29.9 cm dbh, and tall-intermediate = 5-14.9 cm dbh (Godfree et al. 2002).

Summary of plot variability

There was significant variation in some attributes among the plots (for example: diameter distribution, trees/ha, species composition, canopy closure, and overall stand structure). Some of the variables were standardized to account for this variation. For the following analyses, the response variables were the ratios of live or dead trees/ha to the total number of trees/ha. This was done to account for the variability of stand density and species composition among plots. Stump basal area (m^2/ha) was used in each of the following models as an indication of past harvesting activities.

Data storage

The data for this study will be stored and accessible from two locations: one with the U.S. Forest Service Pest Trend-Impact System (PTIPS) data in Fort Collins, Colorado. This database is maintained by the Forest Service Forest Health Technology Enterprise Team (FHTET) and is accessible to the public. The other

location will be with Oregon State University and the Forest Science Data Bank (FSDB). The data will be available through either location by February 2004.

ROOT DISEASE IMPACTS ON STAND SPECIES COMPOSITION AND STAND STRUCTURE

Literature review

Stand structure and root pathogens

The impacts of root diseases and their associated insect complexes have been studied and understood in terms of volume loss. However, as federal management practices move away from timber volume production, they have shifted towards managing for other objectives such as wildlife and forest health. Therefore, the information gaps on just how these root pathogens impact overall stand structure need to be addressed so we can successfully manage for different objectives. Interactions of root diseases, bark beetles, and defoliating insects have been researched in the past. However, the understanding of their impacts on stand structure after insect-defoliator epidemics has only been hypothesized.

Root disease pathogens have the capability of changing the stand structure (Kile et al. 1991, Goheen and Otrosina 1998). DeBell et al. (1997) recognized the capability of root pathogens to create the desired stand structures of small openings and to favor trees of diverse species and sizes. In western Oregon it has been found that *Phellinus weirii* can alter stand structure by killing susceptible species, thus creating gaps and allowing the stand to enter a different successional trajectory (Holah et al. 1997, Hansen and Goheen 2000). Hansen and Goheen (2000) stated that *P. weirii* changes the structure, composition, and canopy layering of infected stands. Holah et al. (1997) found that inside *P. weirii* infection centers in the

Cascade Mountains there were more late-successional species and that these species had higher basal areas inside infection centers compared to adjacent stands. However, this did not hold true for coastal sites in Oregon, where openings were rapidly colonized by shrubs that potentially out-competed or created a site unfavorable for late-successional species establishment. Thus, the way in which the successional trajectory is changed depends on the present successional status of the affected species.

On the east side of the Cascades, two more root pathogens, *H. annosum* and *A. ostoyae*, alter stand structure in ponderosa pine and mixed-conifer forests (Filip and Goheen 1982, 1984). The impacts of these root pathogens have been enhanced by management activities such as fire suppression and harvesting (Filip and Goheen 1994, Hansen and Goheen 2000, Ferguson et al. 2003).

As root disease pockets expand, trees on the edges die and create canopy openings for pockets of regeneration (Filip and Schmitt 1990). These disease centers often contain trees that are in several stages of deterioration (Hadfield et al. 1986), with infected trees being prone to windthrow. On the east side of the Cascades, late-successional species such as white fir commonly regenerate in these openings.

Godfree (2000a, 2000b) and Godfree et al. (2002) assessed the impacts of pathogens on stand structure in central Oregon on the Crescent Ranger District, Deschutes National Forest, with lodgepole pine dwarf mistletoe (*Arceuthobium americanum* Nutt. Ex Engelm.) and its host, *Pinus contorta*. They quantified the

role of lodgepole pine dwarf mistletoe on changes in stand structure, individual tree crown structure, and understory vegetation. They researched dwarf mistletoe's potential role in increasing fire risk by maintaining a lower crown through infected branches. These studies and their associated methodologies are precursors to my research.

Godfree (2000a, 2000b) found that lodgepole pine stands infected with *A. americanum* exhibited stand characteristics similar to the reverse-J diameter-class distribution: a high number of trees in the smaller size classes and relatively few dominant trees. Dwarf mistletoe in pure stands of lodgepole pine "reduced the overall degree of canopy stratification, the number of canopy layers, and the mean canopy foliage height." Godfree (2000a, 2000b) and Godfree et al. (2002) found that in heavily infected stands the mean LCR increased linearly with mean plot-level dwarf mistletoe ratings. Infected stands had shorter trees with individual tree crown bases closer to the ground. Godfree (2000b) found no relationship between mean plot infection level of dwarf mistletoe and the richness and diversity of the understory forb and shrub species.

Objective and hypotheses

The objective of this part of my study was to determine the relationship between increasing root disease (infected BA) and size-class structure of white fir, Douglas-fir and ponderosa pine at the stand level. My study seeks to determine and define how stand structure changes with increasing levels of root disease by exploring the question: Does increasing infection by *Armillaria* spp. and *H.*

annosum significantly change the size-class structure and mortality levels of white fir, Douglas-fir, and ponderosa pine at the stand level?

Null and alternative Hypotheses:

1) $\mathbf{H_O}$: Percent of mortality is independent of infection level, and mortality rate does not differ by species.

$\mathbf{H_A}$: Percent of mortality is dependent on infection level, and mortality rate differs by species.

2) $\mathbf{H_O}$: Percent mortality does not differ by diameter class among differing levels of infection.

$\mathbf{H_A}$: Percent mortality differs by diameter class among differing levels of infection.

3) $\mathbf{H_O}$: Percent of white fir (by tph) in the tall-intermediate and dominant size classes does not differ by infection level and is $\leq 60\%$

$\mathbf{H_A}$: Percent of white fir (by tph) in the tall-intermediate and dominant size classes does not differ by infection level and is $\geq 60\%$.

4) $\mathbf{H_O}$: Percent of Douglas-fir and ponderosa pine (by tph) in the emergent (30-59.9 cm dbh) and tall-emergent (60.0+ cm dbh) size classes does not differ by infection level and is $\leq 60\%$.

$\mathbf{H_A}$: Percent of Douglas-fir and ponderosa pine (by tph) in the emergent and tall-emergent size classes differs by infection level and is $\geq 60\%$.

Statistical analysis

Multiple regression techniques were used to test the hypotheses. A full model was developed to predict the percentage of mortality in each plot. Backward selection (Ramsey and Schafer 1997) was then used to determine the reduced model and the best predictors for the percent mortality by species. The full regression model included the BA of infected trees (m^2/ha), slope (%), aspect (NW, N, NW, or E), elevation (m), total BA of live and dead standing trees (m^2/ha), and the BA of stumps (m^2/ha) as the explanatory variables to predict the ratio of dead trees to the total trees/ha by diameter class. Infected BA was used to describe the amount of inoculum of *Armillaria* spp. and/or *H. annosum*. The pathogen species included in the infected BA was dependent on the tree species of the dependent variable. The total BA of live and dead standing trees was used as an indicator of competition over time. However, it is underestimated, because windthrown trees were not included.

The explanatory variables were checked for correlation before they were included in the model (Table 2.1). Pearson's correlation coefficient was used as a measure of collinearity (George and Mallery 2001). All possible explanatory variables for objectives 1, 2, 3, and 4 (from chapter 1) were included in the analysis unless they had a significant Pearson's correlation coefficient with other variables. For further understanding of these relationships, I used scatter plots to visually examine the trends.

A backward stepwise procedure was used to determine which of the variables had the best fit. In this procedure, variables were excluded from the

analysis one at a time. The variable with the highest p-value was removed from the model first, and then the model was rerun and the next variable was dropped. Variables were excluded from the analysis if they had an α level ≥ 0.05. At each step in the procedure, the residuals were checked for constant variance and any potential outliers.

Results were reported using the adjusted-R^2 to penalize for including unnecessary explanatory variables (Ramsey and Schafer 1997, George and Mallery 2001). This was appropriate since explanatory variables were only excluded at the $\alpha \geq 0.05$ level. All statistical analyses were performed using SPSS 10.0 unless otherwise noted.

Tree mortality--Hypothesis 1

Due to its correlation with many of the variables, elevation was excluded from any regression equations (see Table 2.1). The full model was as follows:

$$\arcsin\sqrt{\rho} = \beta_0 + \beta_1 BA_{INF} + \beta_2 ASP + \beta_3 BA + \beta_4 BA_{STUMP} + \varepsilon$$

where:

$Y = \arcsin\sqrt{\rho}$
ρ = ratio of mortality (BA of mortality/BA total live and dead)
BA_{INF}= BA of annosus- and/or Armillaria-infected trees (m^2/ha)
ASP = aspect of the plot (NW=1, N=2, NE=3, E=4)
BA = total BA of standing live and dead trees (m^2/ha)
BA_{STUMP} = BA of stumps (m^2/ha)
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1,β_2, β_3, and β_4 are parameters to be estimated from the data

Only *Armillaria* spp.-infected BA was included in the full model for Douglas-fir and ponderosa pine. For white fir, infected BA for the two pathogens was combined and represented by the variable "infected BA".

Live-to-dead tree ratios and infection levels--Hypothesis 2.

In order to determine the relationship between the amount of infection from *Armillaria* spp. and *H. annosum* and the current stand structure, linear regression was used to predict the percentage of dead trees in each diameter class. A full model was first developed that included all independent variables that could potentially influence the stand structure. These variables were aspect, total standing BA (as an indication of competition), stump BA (to assess past harvesting activities), and infected BA (including both root disease pathogens). The dependent variable, ratio of dead trees (standing) to total standing trees in each diameter class (tall-intermediate, dominant, emergent, and tall-emergent), was used to assess impacts within a certain diameter class. A backward selection criterion was used to determine the final model from the full model presented below. Variables were excluded if they had an α level > 0.5. The full model was: $\arcsin\sqrt{\rho} = \beta_0 + \beta_1 BA_{INF} + \beta_2 ASP + \beta_3 BA + \beta_4 BA_{STUMP} + \varepsilon$

where:

$Y = \arcsin\sqrt{\rho}$
ρ = the ratio of dead trees to all trees (by diameter class)
BA_{INF} = BA of infected trees (m^2/ha)
ASP = plot aspect (NW=1, N=2, NE=3, E=4)
BA = the total BA of standing live and dead trees (m^2/ha)
BA_{STUMP}= BA of stumps (m^2/ha)
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1,β_2, β_3, and β_4 are parameters to be estimated from the data

Species dominance by diameter class--Hypotheses 3 and 4

This hypothesis was explored using MANOVA (multiple analysis of variance) and by descriptive statistics. Two models were developed to test the hypotheses. The arcsine square-root transformation was used on all of the response variables to meet regression assumptions of constant variance. For Hypothesis 3, the proportions of white fir in the tall-intermediate and dominant diameter classes were regressed against infected basal area. For Hypothesis 4 the percent composition of Douglas-fir and ponderosa pine in the same diameter classes were added together. Then the proportions of Douglas-fir and ponderosa pine in the emergent and tall-emergent diameter classes were regressed against infected BA (only Armillaria infected BA). Analyses were performed using SAS 8.1 and the proc GLM procedure. The models were as follows:

H_O 3: $\text{arcsine}\sqrt{P_{WF}} = \beta_0 + \beta_1 BA_{INF}$ for both tall-intermediate and dominant diameter classes.

H_O 4: $\text{arcsine}\sqrt{P_{DF+PP}} = \beta_0 + \beta_1 BA_{INF}$ for both emergent and tall-emergent diameter classes.

Results

Correlation

There were strong correlations among multiple explanatory variables (Table 2.1). Elevation was correlated with aspect, cover, infected BA, live BA, and dead BA. Dead BA had a strong positive correlation with elevation. There was no correlation between elevation and total BA (BA of live and dead standing trees), an indication of stand density before the western spruce budworm defoliation. This may indicate that the mortality agents (root pathogens, bark beetles, and spruce budworm defoliation) had a greater impact at higher elevations. In a separate correlation matrix, white fir tph (total and dead) have significant Pearson correlation coefficients ($p= 0.039$ and 0.45 respectively). This reinforces the correlation of infected BA and elevation (Table 2.1). There was a corresponding increase in the infected BA with an increase in elevation. The correlation of aspect with elevation is due to the original harvest unit layout by Petaisto et al. (1999), where the higher elevation units tended to have an east-facing aspect.

The plots ranged in elevation from 975 to 1402 m and occurred around three different elevation levels (1000, 1200, and 1400 m) (Fig. 2.1). The infected BA had a wide range of values at each elevation level. However, there was an overall increase in infected BA with increasing elevation. All of the significant correlations with elevation were similar to that shown in Fig. 2.1. Elevation was significantly correlated with dead basal area, live basal area, infected basal area, and canopy cover.

Table 2.1: Pearson's correlation coefficients for explanatory variables. P-values are two tailed, N=25. Shaded values indicate significant correlations.

		Aspect	Slope	Elevation	Stump BA	Dead BA	Live BA	Total BA	Infected BA	Canopy Cover
Aspect	Correlation	1.000	0.118	0.534	-0.194	0.286	-0.171	-0.208	0.297	-0.148
	P-value		0.573	0.006	0.352	0.165	0.414	0.318	0.149	0.481
Slope	Correlation	0.118	1.000	0.150	-0.030	0.418	0.087	0.050	0.446	-0.283
	P-value	0.573		0.474	0.888	0.037	0.679	0.813	0.026	0.171
Elevation	Correlation	0.534	0.150	1.000	-0.014	0.613	-0.568	-0.093	0.493	-0.584
	P-value	0.006	0.474		0.947	0.009	0.003	0.657	0.012	0.002
Stump BA	Correlation	-0.194	-0.030	-0.014	1.000	-0.298	-0.407	-0.104	-0.272	-0.386
	P-value	0.352	0.888	0.947		0.148	0.044	0.621	0.188	0.057
Dead BA	Correlation	0.286	0.418	0.613	-0.298	1.000	-0.106	0.080	0.966	-0.231
	P-value	0.165	0.037	0.009	0.148		0.615	0.703	0.000	0.267
Live BA	Correlation	-0.171	0.087	-0.568	-0.407	-0.106	1.000	-0.174	-0.102	0.670
	P-value	0.414	0.679	0.003	0.044	0.615		0.406	0.627	0.000
Total BA	Correlation	-0.208	0.050	-0.093	-0.104	0.080	-0.174	1.000	0.066	-0.074
	P-value	0.318	0.813	0.657	0.621	0.703	0.406		0.754	0.727
Infected BA	Correlation	0.297	0.446	0.493	-0.272	0.966	-0.102	0.066	1.000	-0.274
	P-value	0.149	0.026	0.012	0.188	0.000	0.627	0.754		0.185
Canopy Cover	Correlation	-0.148	-0.283	-0.584	-0.386	-0.231	0.670	-0.074	-0.274	1.000
	P-value	0.481	0.171	0.002	0.057	0.267	0.000	0.727	0.185	

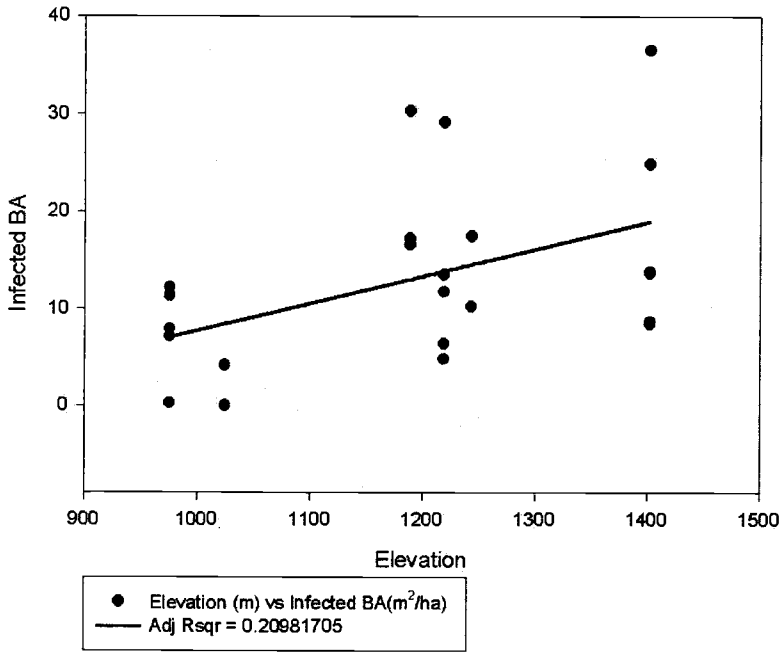

Fig. 2.1: Infected BA values at different elevations.

Tree mortality--Hypothesis 1

The null hypothesis one was rejected: with increasing infection levels (BA_{INF}), the percentage of mortality of each tree species (white fir and Douglas-fir) increased. This relationship was not rejected for ponderosa pine.

White fir mortality

The standardized residual data were skewed (long-tailed to the left), and an arcsine square root transformation of the dependent variable, ratio of mortality, was used to account for the assumption of constant variance. Plot 12 was determined to be an outlier since it had a Cook's distance of 1.66, studentized residual of –3.36, and leverage of 0.27 (Ramsey and Schafer 1997); hence, it was not used in further analysis for predicting mortality of Douglas-fir and white fir. Compared to other

plots plot 12 was an outlier in that it had a structure more typical of a root disease center with large ponderosa pine in the overstory and a blanket of white fir in the understory. This was a typical of the plots sampled in this study.

The null hypothesis was rejected. White fir mortality was primarily described by infected BA and secondarily by stump BA. Both of the explanatory variables had positive relationships with the amount of white fir mortality. The final model had an adjusted-R^2 of 0.729. The positive relationship between stump BA and white fir mortality is most likely because stumps act as inoculum foci for both root pathogens. For *H. annosum* this potential is magnified because spores are capable of infecting freshly cut stumps and subsequently spreading by root-to-root contact.

Douglas-fir mortality

The null hypothesis was rejected for Douglas-fir, and the final model only included infected BA as an explanatory variable (Table 2.2). The final model had an adjusted-R^2 of 0.255 ($p = 0.007$). Douglas-fir mortality had a weak positive relationship with *Armillaria* spp.-infected BA. For every 1 m^2/ha increase in infected BA there was an associated increase in Douglas-fir mortality of 2.17% (95% CI of 0.072 to 3.62%).

Ponderosa pine mortality

Ponderosa pine mortality was not significantly related to any of the predictor variables; therefore, the null hypothesis was not rejected (Table 2.2). There was a

heavy snowstorm in the winter of 1999-2000 that caused top breakage in the small-diameter ponderosa pine at the lower elevations. This could be the cause of the mortality in the plots with small-diameter (5-30 cm dbh) ponderosa pine. Specifically, plot 19 had an extensive amount of snapped-out ponderosa pine.

Table 2.2: Linear regression results for mortality by species. Numbers in bold represent significant results.

Tree Species	Root Pathogens	Transformation	Outlier variables	Adjusted R^2	Degrees of Freedom	F-Value	P-Value	Significant Coefficients	Beta of Coefficients	Standard Error	Significance of coefficients
White Fir	*Armillaria* spp. and *H. annosum*	Arcsin√p	Plot 12	**0.751**	2,21	31.92	**0.000**	Constant	-0.111	0.91	0.238
								Infected BA*	0.02678	0.003	**0.000**
								Stump BA	0.01326	0.004	**0.001**
Douglas-Fir	*Armillaria* spp.	None	Plot 12	**0.255**	1,22	8.868	**0.007**	Constant	0.19214	0.098	0.063
								Armillaria spp. Infected BA	0.02172	0.007	**0.007**
Ponderosa Pine	*Armillaria* spp.	None	None	0.064	2,22	2.654	0.117	Constant	-0.14429	0.223	0.523
								Total BA	0.00758	0.001	0.117

* Infected BA includes trees infected with either *H. annosum* and/or *Armillaria* spp.

Percentage of mortality by diameter class and infection levels--Hypothesis 2

The results of hypothesis 2 were dependent on the diameter class and species (Table 2.3). Increasing infection levels (BA_{INF}) significantly decreased the ratio of dead trees to total standing trees in the dominant, emergent, and tall-emergent diameter classes for white fir. Increasing infection levels (BA_{INF}) did not significantly reduce the ratio of dead to total standing trees in the tall-intermediate class for white fir or all other species.

Tall-intermediate diameter class

With white fir excluded, in the tall-intermediate diameter class (5-14.9 cm dbh) none of the variables in the full model successfully predicted the ratio of dead trees/ha to the total number of trees/ha in that diameter class (Table 2.3), and the null hypothesis was not rejected ($p= 0.31$). For this analysis, 11 plots were excluded because there were no standing live or dead trees in this diameter class.

I assessed only white fir because it is generally more susceptible to root disease. In this case, 3 plots were excluded from the analysis because they lacked any live or dead white fir 5-14.9 cm dbh. As in the analysis for all other species, none of the variables in the full model significantly predicted the ratio of tall-intermediate live white fir to the total number of white fir in the tall-intermediate diameter class, and the null hypothesis was not rejected ($p= 0.19$) (Table 2.3). Fig. 2.2 shows the lack of dependence of the percentage of dead white fir in the tall-intermediate diameter class on infected BA.

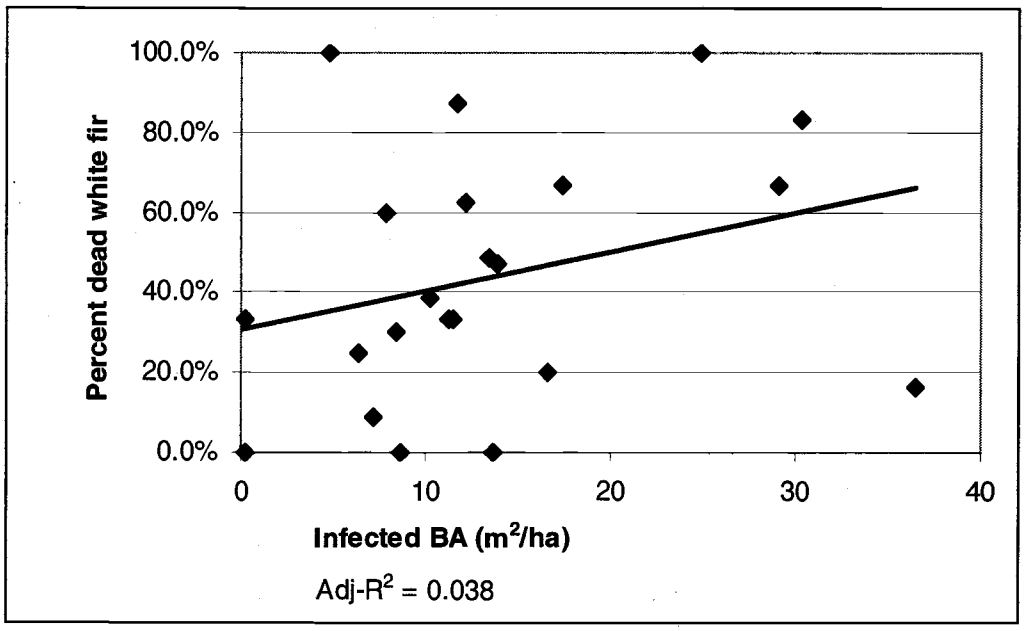

Fig. 2.2: Tall-intermediate diameter class percentage dead of white fir predicted by infected BA.

Dominant diameter class

Infected BA did not significantly predict the ratio of dead trees when all tree species except white fir in the dominant diameter class (15-30 cm dbh) were grouped together and the null hypothesis was not rejected ($p < 0.08$, Table 2.3).

Dead white fir in the dominant diameter class showed a positive relationship with infected BA (Table 2.3) and the null hypothesis was rejected. The total BA was also a significant explanatory variable. If stump BA and total BA were held constant, then a 1 m^2/ha increase of infected BA was associated with a increase of dead white fir by 2.72% (95% CI 1.89 to 3.55%). Total BA had a negative

relationship with the ratio of dead white fir. Increases in the total BA of live and dead trees were associated with decreases in mortality of white fir 15-30 cm dbh.

Emergent diameter class

The null hypothesis was rejected in the emergent diameter class (30-59.9 cm dbh) (Table 2.3) when all tree species except white fir were included in the model. The percent dead trees/ha in the emergent diameter class had a positive relationship with infected BA. The final model only included infected BA ($p < 0.002$, Table 2.3). With an 1 m^2/ha increase in infected BA, then the dead trees/ha in the emergent diameter class increases by 1.9% (95% CI 0.87 to 2.93%).

The dead white fir emergent diameter class model yielded similar results with infected BA and the total BA as explanatory variables (Table 2.3), and the null hypothesis was rejected. The model had an adjusted-R^2 of 0.79. Holding the total BA constant, there was a 3.03% increase (95% CI 2.41 to 3.65%) in the dead white fir with an increase of 1 m^2/ha of infected BA.

Tall-emergent diameter class

The null hypothesis was not rejected for all tree species in the tall-emergent diameter class (>60 cm dbh) (white fir excluded) (Table 2.3). Four cases were excluded from this analysis because there were no live or dead trees ≥ 60 cm dbh that were not white fir.

Further analysis was done for the dead white fir in the tall-emergent diameter class. Plots 20, 22, 24, and 25 (TS9P6 and P9, TS15P2 and P10 in fig. 1.2)

were excluded from analysis because they lacked any white fir trees ≥ 60 cm dbh. The final model for the dead white fir only included infected BA as a variable (adjusted-R^2= 0.412), and the null hypothesis was rejected (p=0.001). The ratio of dead white fir in the emergent diameter class increased by 2.3% (95% CI 1.0 to 3.5%) with a 1 m^2/ha increase of infected BA.

Table 2.3: Linear regression results for stand structure when the dependent variable was the ratio of the dead TPH/ total TPH in that diameter class and species of interest. Significant values are in bold.

Diameter size class	DBH range	Dependant Variable	Excluded Cases	Adjusted-R^2	Degrees of Freedom	F-value	P-Value	Significant Coefficients	Beta of coefficients	Standard Error	Significance of coefficients
Tall-intermediate	5-14.9 cm	All tree species-	1,2,3,4,6,7,8,1 0,11,16,17,21,	0.012	1,9	1.124	0.317	Constant	-0.0513	0.342	0.8842
		Except white fir	22,23					Stump BA	0.0163	0.015	0.3166
		White fir	8, 24 , 25	0.038	1,20	1.838	0.190	Constant	0.3060	0.117	0.0165
								Infected BA*	0.0097	0.007	0.1903
Dominant	15-29.9 cm	All tree species-	None	0.133	2,22	2.836	0.080	Constant	-0.3593	0.363	0.3336
		Except white fir						Stump BA	0.0153	0.008	0.0844
								Total BA**	0.0121	0.005	0.0339
		White Fir	**None**	**0.728**	**3,21**	**22.399**	**0.000**	**Constant**	**0.3614**	**0.171**	**0.0464**
								Stump BA	**0.0069**	**0.004**	**0.0792**
								Total BA**	**-0.0071**	**0.003**	**0.0381**
								Infected BA*	**0.0272**	**0.004**	**0.0000**
Emergent	30-59.9 cm	**All tree species-**	**None**	**0.323**	**1,23**	**12.474**	**0.002**	**Constant**	**0.2428**	**0.081**	**0.0064**
		Except white fir						**Infected BA***	**0.0190**	**0.005**	**0.0018**
		White fir	**None**	**0.79**	**2,22**	**46.246**	**0.000**	**Constant**	**0.4296**	**0.096**	**0.0002**
								Total BA**	**-0.0091**	**0.003**	**0.0018**
								Infected BA*	**0.0303**	**0.003**	**0.0000**
Tall-emergent	60+ cm	All tree species-	5,16,18,19,22, 23	0.027	1,17	1.493	0.238	Constant	0.4939	0.249	0.0641
		Except white fir						Total BA**	-0.0063	0.005	0.2384
		White fir	**20, 22, 24, 25**	**0.412**	**1,19**	**15.011**	**0.001**	**Constant**	**-0.0443**	**0.100**	**0.6613**
								Infected BA*	**0.0229**	**0.006**	**0.0010**

*Infected BA included trees infected with either *Armillaria* spp. or *H. annosum*.

**Total BA included BA of all live and dead standing trees.

Species dominance by diameter class--Hypotheses 3 and 4

The null hypothesis 3 was not rejected: at higher infection levels white fir did not dominate the tall-intermediate and dominant diameter classes (Table 2.4). Instead, in the tall-intermediate diameter class (5-14.9 cm dbh) live-white fir dominated (> 60% of trees/ha) across all infection levels (Fig. 2.4). Douglas-fir and ponderosa pine were present at the lower infection levels (<15 m^2/ha). Across the study plots the dominant diameter class (15-29.9 cm dbh) was composed primarily of white fir (>60% of the trees/ha) (Fig. 2.4). Douglas-fir and ponderosa pine were consistently present at lower infection levels (<5 m^2/ha). In the high infection level plots, Douglas-fir and ponderosa pine composed less than 5% of the trees/ha in the tall-intermediate and dominant diameter classes.

The null hypothesis 4 was not rejected for the emergent and tall-emergent diameter classes (Table 2.4). The emergent diameter class (30-59.9 cm dbh) still retained white fir at > 60% of the trees/ha on most of the plots regardless of the infected BA. Douglas-fir and ponderosa pine dominated the trees/ha on only three of 10 plots at lower infected basal areas (<10.0 m^2/ha). The tall-emergent diameter class (60+ cm dbh) had a different distribution of species compared to the other three diameter classes (Fig. 2.5). In four cases Douglas-fir composed >60% of the trees/ha; ponderosa pine did so in three plots; white fir in seven plots; and for the remainder of the plots, trees/ha was split between at least two of the tree species.

Table 2.4: Results for species composition by diameter class.

Dependent variable	Df	F-value	p-value	R-square	Estimate-BA_{INF}	SE BA_{INF}
arcsine√Tall-Intermediate white fir	1,19	0.19	0.6642	0.0101	0.00472	0.01
arcsine√Dominant white fir	1,23	0.92	0.3467	0.0386	-0.00774	0.0081
arcsine√Emergent Douglas-fir + ponderosa pine	1,23	0.58	0.4541	0.0246	0.00595	0.0078
arcsine√Tall-Emergent Douglas-fir + ponderosa pine	1,22	0.86	0.3636	0.0377	-0.0118	0.012

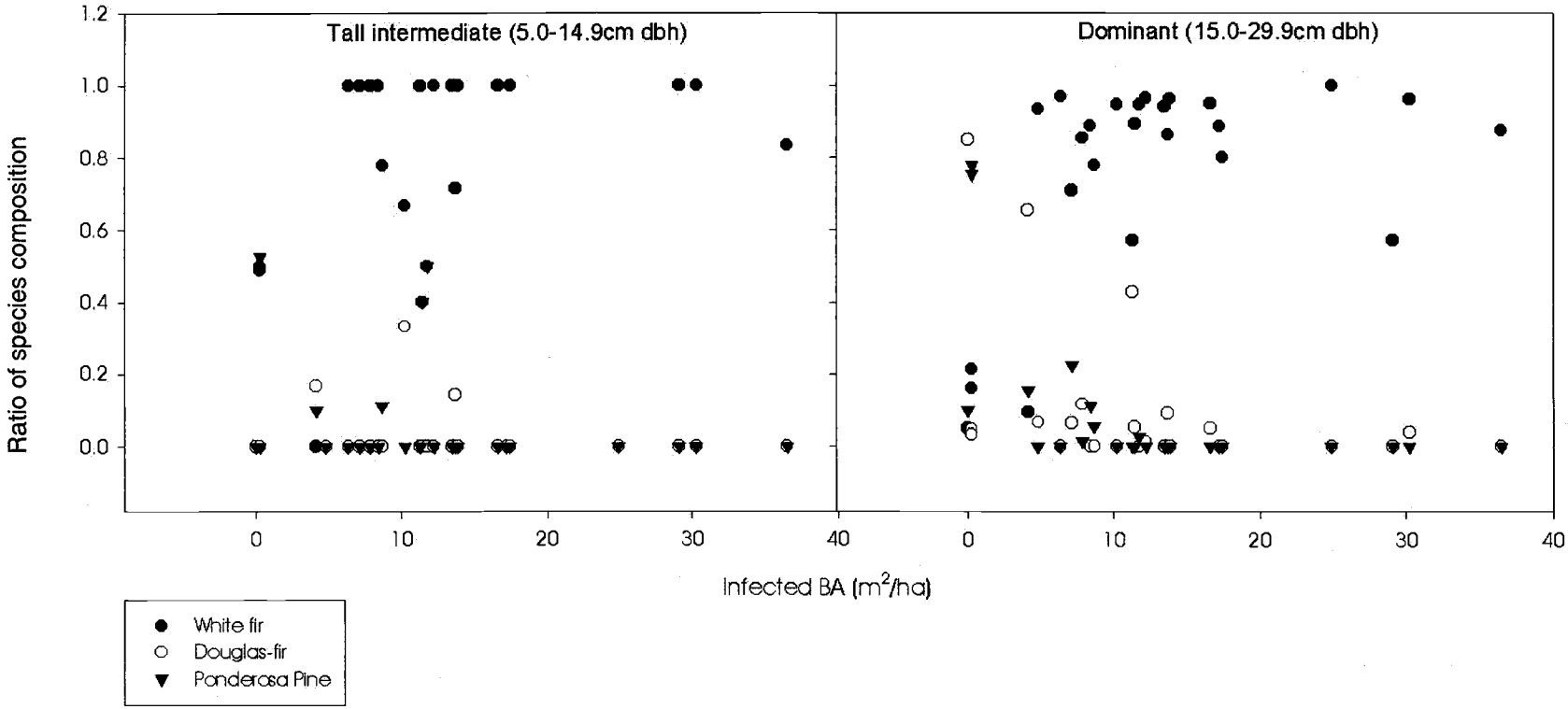

Fig. 2.4: Ratio of species composition for the tall-intermediate and dominant diameter classes.

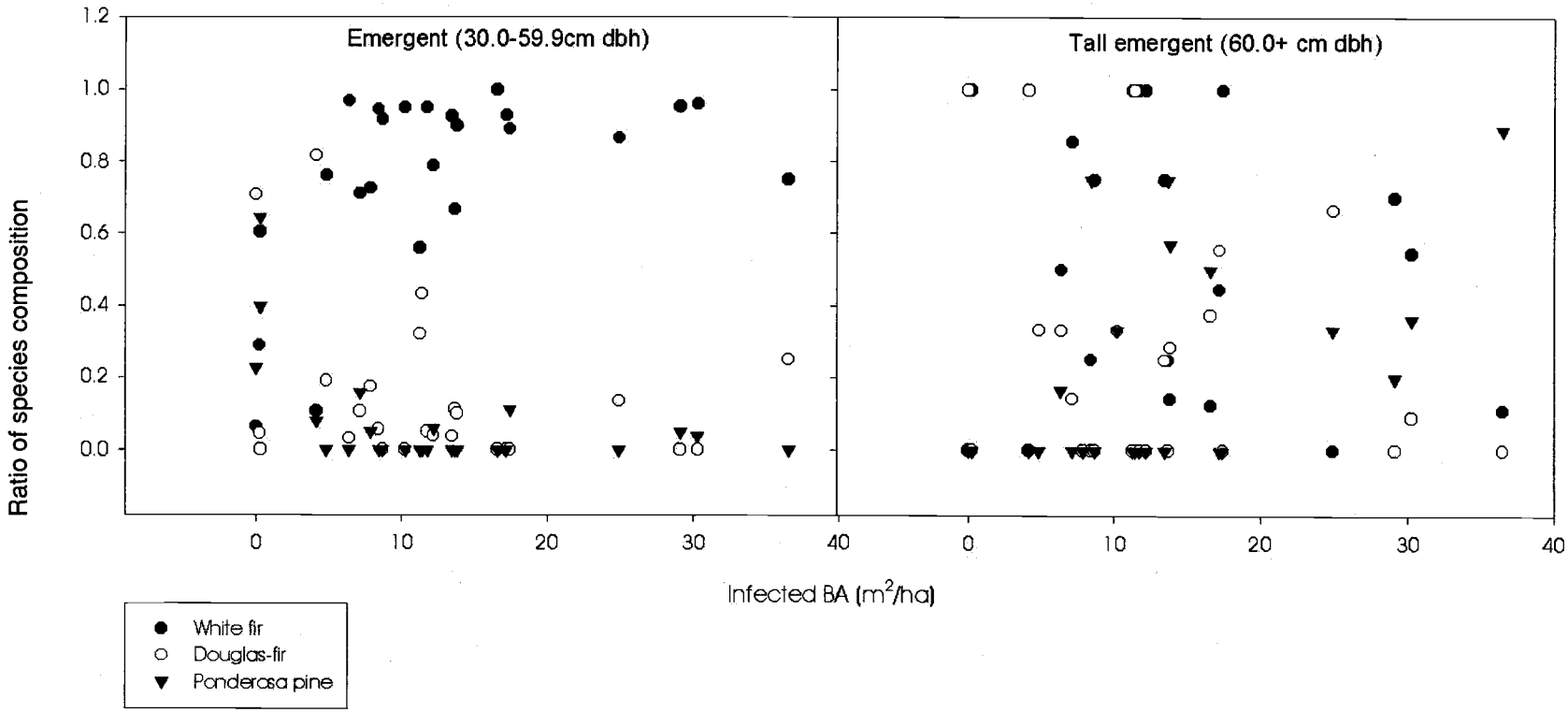

Fig. 2.5: Ratio of species composition for the emergent and tall-emergent diameter classes.

Discussion

Stand structure and species composition varied greatly among plots and were largely dependent on infected BA. White fir and Douglas-fir mortality increased with an increase in the amount of root disease present (as defined by infected BA). The amount of mortality also varied by diameter class. The larger diameter classes had significant increases in the percentage of dead trees/ha, and this was primarily dependent on the infected BA. Surprisingly, white fir in this study was still a component of the tall-emergent (>60 cm dbh) diameter class regardless of infected BA. The percentage of tree species composition was not dependent on the infected BA, and was fairly consistent when infection levels were >5 m^2.

The mortality by species results indicated that it was primarily white fir that was being killed by root diseases in this study area. Armillaria root disease was also a factor in the mortality of Douglas-fir; however, this was a weak linear relationship. Ponderosa pine mortality was poorly correlated with the amount of root disease. The mortality results support other research indicating that Douglas-fir and ponderosa pine are both hosts to *Armillaria* spp.; however, they can be fairly weak hosts, and their susceptibility is site dependent (McDonald et al. 1987a). Results indicate that, in mixed-conifer stands in central Oregon, white or grand fir is highly susceptible and readily killed, Douglas-fir is moderately to highly susceptible, and ponderosa pine is moderately susceptible to resistant.

The spruce budworm outbreak in the late 1980s and early 1990s might have also contributed to the mortality in Douglas-fir and white fir. However, Sisters

Ranger District surveys found that, at the time of the outbreak, the large majority of dead trees surveyed around Suttle Lake (located approximately 5 km south of this study area) had one or more root pathogens present (Eglitis 1991, 1992; Scott Beyer and Helen Maffei, Deschutes National Forest, 2003 pers. Comm.). Therefore, a combination of the causal agents, insect defoliation, root disease, and bark beetles most likely contributed to the mortality. Western spruce budworm could have created a weakened host, thus increasing susceptibility to root disease and bark beetles. This relationship has been well documented. Hadfield et al. (1986) noted that increased mortality from *A. ostoyae* could be found in the years following defoliation outbreaks. The defoliation could have also given the fungus an opportunity to rapidly colonize already infected trees. On these sites, trees recently killed by root disease were found in all plots approximately 10 years after the defoliation ended.

Understanding which tree size classes the root disease is impacting and the possible impacts on the current and desired stand structure are also important. The Northwest Forest Plan mandates that federal lands provide, retain, and manage for stand conditions that retain large-diameter trees in Late Successional Reserves. However, most of these stands have one or more root pathogens on the east side of the Cascade crest. These pathogens can adversely change the outcome of the desired stand structure, especially if white fir is to be retained as a part of the large tree structure and provide the desired canopy cover.

As shown in the second hypothesis, the percentage of dead trees was different for each diameter class. With larger amounts of infected BA, it was found that the ratio of live trees in the dominant (15-29.9 cm dbh) and emergent (30-59.9 cm dbh) diameter classes decreased significantly. White fir in the tall-emergent diameter class (>60 cm dbh) was greatly impacted by *Armillaria* spp. and *H. annosum*. The other tree species in this diameter class had a weak relationship with the root pathogens. These results indicated that, in areas with high levels of root disease, retaining large white fir to meet habitat requirements may not be possible.

In the tall-intermediate diameter class (5-14.9 cm dbh), however, infected BA did not significantly impact the ratio of dead trees to the total trees/ha. This indicated that *Armillaria* spp. and *H. annosum* may not be as significant in causing mortality of small-diameter trees as compared to the larger diameter classes. The spruce budworm defoliation may have contributed more to mortality of white fir in the tall-intermediate diameter class. Defoliation by western spruce budworm causes little mortality except in the small-suppressed understory trees (Brookes et al. 1985). However, even this is a small effect. Ferguson (1988) found that only 3% of regeneration sample trees died in a study in Idaho. Ferguson noted that small live crown ratios or high defoliation levels increased the probability of smaller tree mortality.

Most dead trees in the tall-intermediate diameter-class were on the ground; therefore, using just standing live and dead trees may not be an accurate way to explore the relationship between root diseases and mortality of the tall-intermediate

diameter class. The timing of data collection for this study was approximately 10 years after the western spruce budworm epidemic ended. Therefore, the interaction of this defoliation and root diseases would have resulted in the smaller suppressed trees dying approximately 10 years ago; at the time of sampling they would be on the ground and not readily counted.

The lack of significance of Armillaria- and annosus-infected basal areas in the small-diameter class may contradict some of the assumptions that ponderosa pine, Douglas-fir, and western larch are more susceptible to Armillaria root disease under the age of 15 and that resistance increases after 15 years in coastal and interior British Columbia (Morrison and Mallett 1996).

Stump basal area and total basal area were significant predictors in several of the equations. Large Douglas-fir and ponderosa pine stumps were found in or around all plots, as evidence of salvage harvesting. Stump BA was a significant variable in predicting the percentage of live trees in the dominant diameter class. With an increase in stump BA there was a corresponding increase in the percentage of dead trees/ha. There are a few possible explanations: 1) stumps act as inoculum foci for both root disease pathogens (Morrison and Mallett 1996), which could have increased the amount of root disease in the stand over time and contributed to the increased amount of mortality; or 2) Salvage harvesting created small canopy openings for white fir establishment, or released the understory white fir and created stand conditions more susceptible to root disease, and thus increased the current mortality; or 3) This is a residual effect of stand density reduction caused by

salvage. Most likely it is some combination of these explanations that created this condition.

Total BA of live and dead trees was significant in several models. Total BA had a negative relationship with the emergent dead white fir. There was lower absolute mortality of white fir with an increase in total BA of live and dead trees. The same relationship was true in the emergent diameter class. In several stands (e.g. plot 23, TS9P16 in fig. 1.2) there were high stand densities (55.8 m^2 BA), and although there was a moderate amount (12.19 m^2) of infected BA, there was still a very dense uninfected stand (41.6 m^2 live BA) within the 0.25 ha plot.

Species composition had no obvious relationship with the amount of root disease. This was not surprising since the majority of the high-elevation stands had a high percentage white fir in all diameter classes except in the tall-emergent diameter class. However, the lack of significance does make it difficult to discern changes in successional pathways with differences in root disease infection levels.

Elevation was an underlying factor in all of the variables. Personal observation showed that the Douglas-fir and ponderosa pine component decreased with an increase in elevation except in the tall-emergent diameter class. Higher elevation stands may be further along the successional pathway with little opportunity for Douglas-fir and ponderosa pine establishment in the understory. Mortality from root pathogens also seemed to be higher at higher elevations, as was also noted by Eglitis (1991, 1992). Mortality could be attributed to the presence of more root disease. However, *Armillaria* spp. and *H. annosum* were found in or

adjacent to all of the low elevation plots. With these plots distributed across 400 m in elevation there were more ponderosa pine and Douglas-fir in the tall-intermediate, dominant, and tall-emergent diameter classes at the lower elevations. However, at lower elevations I found the most aggressive Armillaria and annosus root disease centers in plots 20, 22, 23, and 24 (TS9P3, P6, and P16, TS15P2 in fig. 1.2). Plot 24 (TS15P2 in fig. 1.2), which was dominated by Douglas-fir at 1024 m in elevation, had a live infected Douglas-fir at plot center with several dead white fir and Douglas-fir around it. Plot 23 (TS9P16), which was dominated by white fir and located at 975 m in elevation, had two distinct root disease centers; one was recent blow down with extensively decayed roots from *H. annosum*. The other center, found in 2002 after the majority of the data were collected, had several white fir in the dominant and emergent diameter classes that had resinosus at the root collar, fading crowns, and mycelial fans at the root collar. *A. ostoyae* was successfully isolated from one of these trees. This suggested that the spruce budworm outbreak could have accelerated mortality at higher elevations because there was a substantial amount of root disease that provided a weakened host for the spruce budworm and/or fir engraver beetles. Spruce budworm defoliation at lower elevations was still just as severe (Eglitis 1991, 1992). However, because of either host abundance, differences in site conditions, or less root disease, the trees were able to recover and only sustained top-kill in some locations. Observations during the budworm outbreak suggested that most of the affected area sustained minimal mortality, while

areas with the highest mortality were unpredictable (Andris Eglitis, USDA Forest Service, Entomologist, pers. comm. 2002).

The stands in this study site developed under slightly different conditions (See appendix 1). This was determined by summing all of the basal area of live and dead trees and stumps > 60 cm for each plot (m^2/ha). This indicates that some of the relationships explored in this analysis may be attributable to the differences in initial stand conditions instead of as a result of differences in root disease.

The stand structure in most of the plots did not have the appearance of a typical root disease center, with older mortality and regeneration in the center and recently killed trees around the edges (Filip 1977). Instead, mortality from root disease in most plots had a diffuse pattern and lacked a distinct edge. Plot 12 (TS6P9 in fig. 1.2) was an outlier for both Douglas-fir and white fir mortality. While it significantly influenced the regression equation, it had characteristics of a typical root disease center. In the plot were 600 trees/ha of white fir and subalpine fir in the tall-intermediate diameter class that were clumped together. The plot had few trees (32 and 16 trees/ha, respectively) in the dominant and emergent diameter classes and 32 ponderosa pine trees/ha in the tall-emergent diameter class. This plot had a high amount of true fir natural regeneration in the root disease center and larger trees on the center edges with ponderosa pine retained in the overstory. Recently killed white fir saplings were adjacent to the plot. They had distinct mycelial fans under the bark, indicating that they were killed by root disease. The overall picture in plot 12 was that root diseases were pushing back the successional

trajectory and allowing for retention of the older ponderosa pine. The idea of root diseases pushing back the successional trajectory has also been noted by Dickman and Cook (1989) in *P. weirii* centers of the high-elevation Cascades where *P. weirii* slowly removes the late successional species *Tsuga mertensiana*, allowing for the seral species *P. contorta* to regenerate in the openings and establish on the site.

Root pathogens are selectively killing the late seral *Abies* species in the stands of central and eastern Oregon. However, only those species susceptible to root disease are regenerating in the stands, thus continuing the root disease cycle. This is typical of east-side mixed-conifer stands as noted by Goheen and Hansen (1993), because the early seral ponderosa pine and western larch require bare mineral soil to regenerate.

Combining the results from this study and the observations of Eglitis (1991, 1992), Filip (1994), and Cochran (1998) shows that mortality from Armillaria and western spruce budworm has been ongoing for at least 10 years with white fir in all size classes. It is predicted that this mortality will continue. Recent mortality from root diseases was noticed in all of the plots in the two years (2002 and 2003) after the data were collected. This paints a picture of continuing mortality through the life of the stand, and a small steady amount of disturbance occurring in the study area every year. This is not an uncommon observation in areas with root disease. Rosso and Hansen (1998) found that mortality in Douglas-fir in a stand on the west side of the Cascades had continued for several years. Their observations showed that it would continue throughout the life of the stand.

Although some plots were excluded from the analyses because they lacked live or dead trees in certain diameter classes, their absence also has implications for the present and future stand structure. Plots 8, 24, and 25 (TS3P14, TS15P2 and P10 in fig. 1.2) lacked any white fir in the tall-intermediate diameter class. These plots all had low levels of infected BA (0-1 m^2/ha), were dense, and lacked seedling and saplings in the understory. Plots 24 and 25 (TS15P2 and P10 in fig. 1.2) were primarily Douglas-fir with smaller ponderosa pine and white fir in the understory. Since there was little disturbance in these plots, openings were not created to promote white fir regeneration. These plots are currently in an early seral state with white fir being a very small component of the stand structure within the plots. There was an abundance of white fir adjacent to plots 24 and 25, much of which was killed in 2001 from drought stress, Armillaria and annosus root disease, and subsequent beetle attack. These trees all showed signs of resinosis at the root collar, indicating that they were colonized by a pathogenic form of Armillaria before they died (McDonald et al. 1987a).

The 0.25 ha plots used in this study were heterogeneous across any one plot. In many plots there were large openings in one section with a clump of dominant and emergent white fir in another area. Annosus and Armillaria root diseases were usually diffuse throughout the plot. There were recently killed seedlings and saplings with mycelial fans under the bark below the root collar near several plots. These seedlings and saplings are worth noting, because they indicate that *A. ostoyae* is in the stand and is aggressively killing trees.

The data showed that the diameter distributions were not a reverse J-shaped curve as Godfree (2000b) found. Instead, plots such as 1 (TS1P11 in fig. 1.2) (29 m^2/ha infected BA) had 1) a bimodal distribution with 80 trees/ha of primarily white fir in the tall-intermediate diameter class, 2) few trees in the dominant (28 trees/ha) diameter class, 3) a surprisingly larger number (84 trees/ha) in the emergent class, and 4) 40 trees/ha of ponderosa pine and white fir in the tall-emergent diameter class. Other plots such as plot 6 (TS3P2 in fig. 1.2) (30 m^2/ha infected BA) had a bell-shaped curve for diameter with a distribution of 40, 104, 104, and 44 trees/ha in the tall-intermediate, dominant, emergent, and tall-emergent diameter classes, respectively. Four plots, 9, 10, 13, and 21, (TS5P1 and P5, TS6P14 and P6 in fig 1.2) (all had between 10 and 13 m^2/ha infected BA) showed the characteristic reverse J-shaped curve (for example plot 10 with 360, 108, 40, and 28 trees/ha in the tall-intermediate, dominant, emergent, and tall-emergent diameter classes, respectively).

All 25 plots examined in this analysis were very different from each other in species composition, the amount of mortality, stand density, the root pathogens present, the amount of inoculum present, and successional stage. Observations from this and other studies (Holah et al. 1997) show that all of these variables affect the future development and pattern of disturbance of the stand. Further research is needed to understand how to successfully manage these stands to maintain or achieve the desired habitat needed for late successional species.

ROOT DISEASE IMPACTS ON CANOPY COVER AND CANOPY STRATIFICATION

Literature review

Canopy stratification and root pathogens

Canopy stratification, the horizontal and vertical distribution of tree crowns in a forest (Fiala 2003), is altered by root disease pathogens that selectively kill host species (Thies and Sturrock 1995, Holah et al 1997, Hansen and Goheen 2000). Different species of root pathogens, sometimes acting with bark beetles, have been found to create gaps in the forest canopy, thus changing the canopy cover and stand structure (Thies and Sturrock 1995, Beatty et al. 1995, Hansen and Goheen 2000). Selectively killing susceptible tree species leaves non-host species to fill in gaps in the canopy and become part of the overstory. These gaps also provide openings in the forest canopy for shrub and tree regeneration.

These pathogens have the potential to directly influence the amount of canopy cover and, over time, can influence the type of canopy cover by changing species composition and stratification. Holah et al. (1993) found that laminated root rot centers in the Cascade Range generally had higher (although not statistically significant in all cases) canopy cover of the non-host species *Tsuga heterophylla* (Raf.) Sarg., *Thuja plicata* Donn ex D. Don, and *Taxus brevifolia* Nutt. when compared to the adjacent uninfected stand. Douglas-fir, the primary host, however, had less tree cover in all infection centers as compared to the adjacent uninfected stand.

Gaps in the forest canopy can be created by several different agents and are highly variable in size and structure. The disturbances that create gaps in the forest canopy play important roles in changing ecosystem function at a stand level (Beatty et al. 1995). Lundquist (2000) studied the different causes of canopy gaps and their associated sizes in ponderosa pine forests of South Dakota, and found that Armillaria root disease was the primary agent directly affecting canopy gap size. These gaps create ecological niches necessary to flora and fauna. Within canopy gaps there is usually more coarse woody material such as stumps, downed logs, and snags that are required by cavity nesting birds and other fauna (Beatty et al. 1995).

Gaps in the forest canopy create openings for a change in successional pathway. In many cases the type of gap created can influence the direction of change in succession. Krasny and DiGregorio (2001) hypothesize that "periodic disease and insect outbreaks could create more severe disturbances needed to maintain canopy diversity in forests." They cite several indigenous and introduced insects and diseases and their associated changes in succession in eastern forests. In their study they found that forests with beech bark disease and gypsy moth increased the area in openings from 19.7 to 31.6% in a six-year period. Parish et al. (1999) studied the stand development patterns in an old-growth subalpine forest. They found that gaps created in the canopy by mortality from spruce bark beetle (*Dendroctonus rufipennis* [Kirby]) create small openings (<10 m) for Englemann spruce and subalpine fir regeneration. This disturbance agent has continually been shaping the forest for 300 years.

Retaining or achieving a certain amount of canopy cover has guided short- and long-term management goals. For example, the Record of Decision for the Northwest Forest Plan has specific guidelines for east-side forests. It states that "salvage may only occur in areas where disturbance has reduced canopy closure to less than 40 percent, because stands with more closure are likely to provide some value for species associated with these forests" (USDA For. Serv. 1994). This magic 40% has guided managers to manage for 40% canopy cover, a condition that may not be within the natural range of variability in eastside mixed-conifer stands (Petaisto et al. 1999).

Root diseases can also influence the type of canopy cover and canopy structure. Mallett and Volney (1999) studied the effects of Armillaria root disease on tree growth in lodgepole pine stands in the Upper Boreal Cordilleran Ecoregion in Canada. They found that infected trees had significantly greater crown lengths than the uninfected trees at one of their study sites. They found that on average (although not statistically significant), trees inside the infection centers had smaller dbh's, were shorter, and had lower crown base heights.

The type of canopy structure can influence the stand's susceptibility to crown fire. The stand crown base height directly influences the potential for surface fires to climb into tree crowns (Keyes and O'Hara 2002). Keyes and O'Hara (2002) recommended silvicultural prescriptions that modify the stand crown base height to prevent crown fire development.

Objective and hypotheses

This study focuses on the impact of root diseases on canopy cover and canopy stratification. I hypothesized that:

1) $\mathbf{H_O}$: There is no effect of large white fir, Douglas-fir, and ponderosa pine on the dominant diameter class tree density. This has no relationship with infection level.

$\mathbf{H_A}$: The increasing presence of large live white fir, Douglas-fir and ponderosa pine (>30 cm dbh) influences canopy structure by reducing the density of live trees in the dominant (15-29.9 cm) diameter class. This changes as infected BA increases.

2) $\mathbf{H_O}$: Percent canopy cover is not related to infection level.

$\mathbf{H_A}$: The percentage of tree canopy cover decreases with increasing infection levels. This is based on total canopy and canopy cover within each of three layers (which are independent of tree diameter).

3) $\mathbf{H_O}$: The average LCR of trees in the tall-intermediate, dominant, and emergent diameter classes is not related to infection level.

$\mathbf{H_A}$: The average LCR of trees in the tall-intermediate, dominant, and emergent diameter classes decreases (is lower) with increasing infection levels, and this relationship is dependent on tree species.

4) $\mathbf{H_O}$: The average height of trees in the tall-intermediate, dominant, and emergent diameter classes is not related to infection level.

$\mathbf{H_A}$: The average height of trees in the tall-intermediate, dominant, and emergent diameter classes decreases (is lower) with increasing infection levels, and this relationship is dependent on tree species.

5) $\mathbf{H_O}$: The average height to the bottom of the live crown of the tall-intermediate, dominant, and emergent diameter classes is not related to infection level.

$\mathbf{H_A}$: The average height to the bottom of the live crown of the tall-intermediate, dominant, and emergent diameter classes decreases (is lower) with increasing infection levels, and this relationship is dependent on tree species.

Methods

The canopy cover data were collected in the summer of 2002 using the line-intercept method (Canfield 1941, O'Brien 1989). The line-intercept method is a ground-based measurement that is generally considered more accurate than ocular estimates of intercepts from photos (O'Brien 1989) or the predictive models in Forest Vegetation Simulator (FVS) (Fiala 2003). Percent canopy cover in each of three layers by species was calculated using the line-intercept method. The horizontal canopy layers in a stand were classified as upper, middle, and lower. In the field, each tree was assigned to a canopy layer (Fig. 3.1). These layers differed by at least 5 m in height. Actual heights varied among plots, and the canopy layers were relative to the stand conditions. In a few cases, the middle canopy layer was absent. Canopy cover was sampled on four 25-m long horizontal transects originating at the plot center and radiating out to the NW, NE, SW, and SE (Fig.

3.2). For every tree species within a canopy layer > 1.4 m tall, crown boundaries were vertically projected onto transects. The distance along a transect line that the crown intercepted was recorded. The proportion of transects that were intercepted by the tree crowns was the ground-estimated canopy cover. The total cover, individual species cover, and layer cover were calculated for each plot.

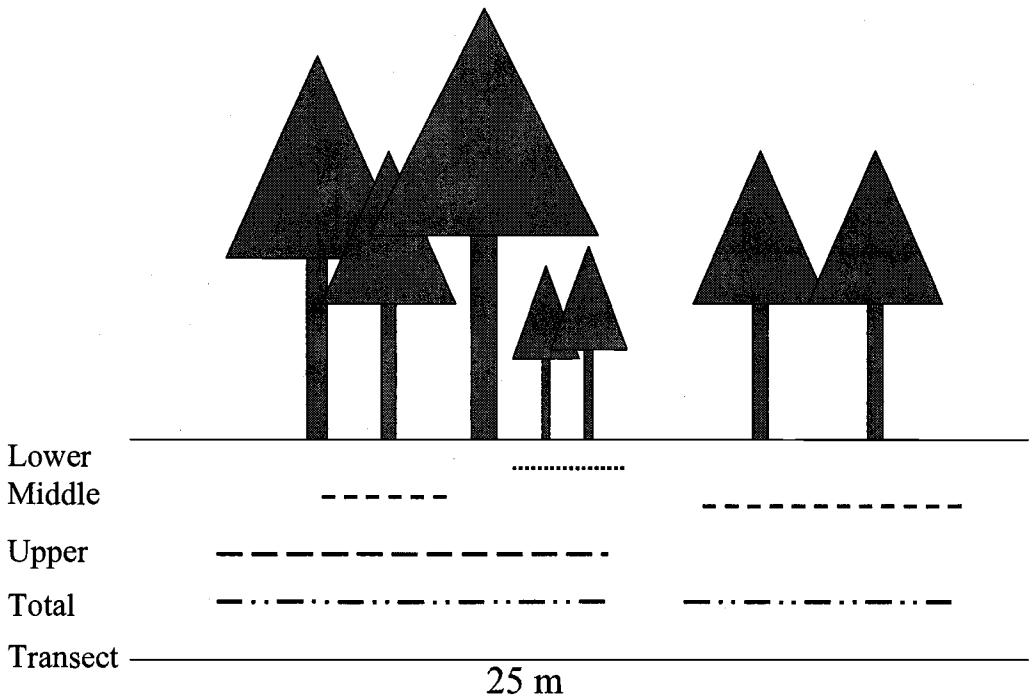

Fig. 3.1: Diagram of line-intercept method of collecting tree canopy cover by layer and by species.

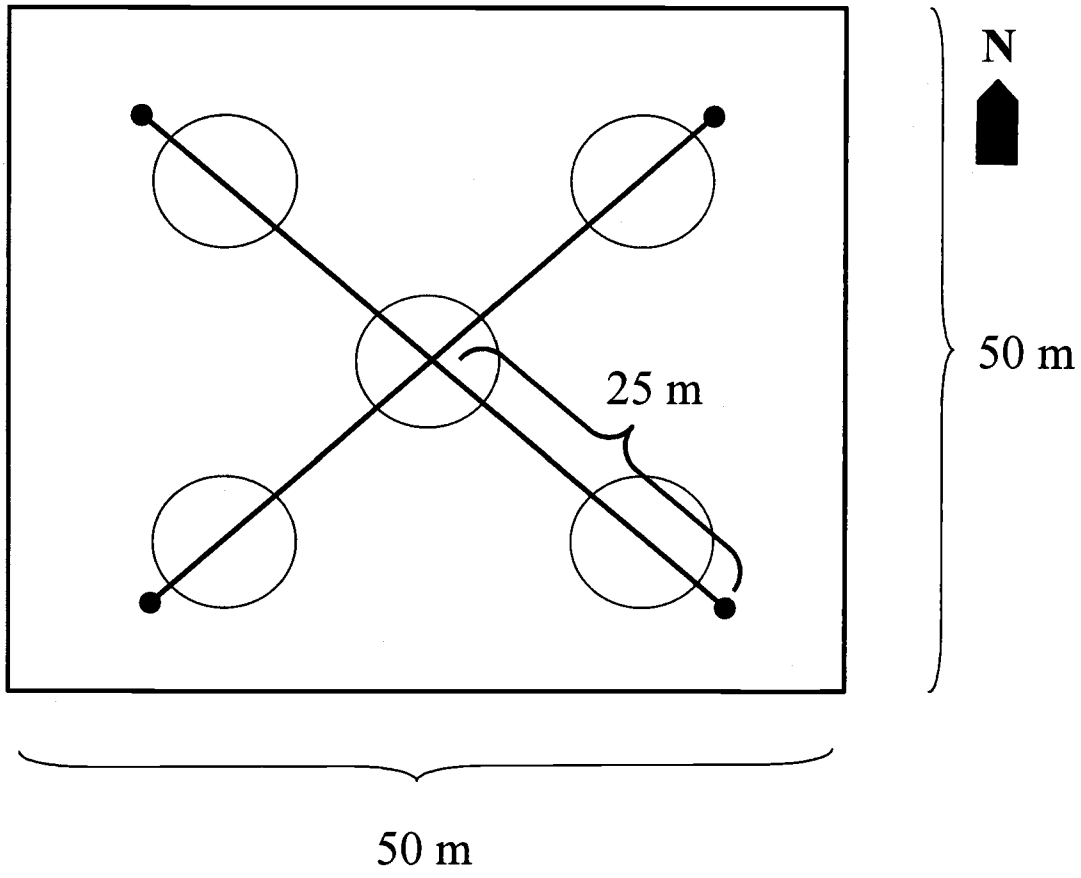

Fig. 3.2: Diagram of the plot layout for collecting canopy cover data.

Statistical analysis

Trees/ha in the dominant diameter class--Hypothesis 1

Hypothesis One was tested by comparing the slopes and intercepts of the dominant, emergent, and tall-emergent diameter classes. Visual inspection of the data revealed that a natural log transformation of the response variable (trees/ha) was needed for further data analysis. TPH in the tall-intermediate diameter class was not used in this analysis because infected BA did not significantly predict the ratio of live trees (see Table 2.3). The full model included an interaction term to determine if the diameter classes had the same slopes. The interaction term was omitted if it was insignificant at an α level > 0.5. Where the interaction term was omitted, the model was then rerun to test for differences in the intercepts. The model was as follows: $\ln TPH = \beta_0 + \beta_1 BA_{INF} + \beta_2 D_{EM} + \beta_3 D_{TEM} + \beta_4 BA_{INF} * D_{DOM} + \beta_5 BA_{INF} * D_{EM} + \varepsilon$

where:

BA_{INF} = BA of annosus and/or Armillaria infected trees (m^2/ha)
D_{DOM} = Indicator for diameter class 1=dominant 0=other
D_{EM} = Indicator for diameter class 1= emergent, 0=other
$BA_{INF} * D_{DOM}$ = The interaction of infected BA and the indicator for the dominant diameter class
$BA_{INF} * D_{EM}$= The interaction of infected BA and diameter class and the indicator for the emergent diameter class
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1, β_2, β_3, β_4, and β_5 are parameters to be estimated from the data

Once the interaction term was found to be significant, the model was then rerun to compare the dominant and emergent diameter classes and then the dominant and tall-emergent diameter classes.

Canopy cover--Hypothesis 2

Line-intercept canopy data were regressed against the following independent variables: aspect, live BA (or live trees/ha), infected BA, and stump BA. Variables that were correlated were not included in the model (see Table 2.1). The variable live trees/ha was used to predict canopy cover for the lower canopy layer instead of the live BA, because although smaller trees are underrepresented in BA measurements they were an important part of predicting the cover in the smaller layers. A backward selection method was used to determine the variables that best predicted canopy cover. Variables were excluded if they had an α level >0.1. All analysis were performed using SPSS 10.0. The full model was as follows:

$$\rho = \beta_0 + \beta_1 BA_{INF} + \beta_2 ASP + \beta_3 BA_{LIVE} + \beta_4 BA_{STUMPS} + \varepsilon_i$$

where:

$Y=\arcsin\sqrt{\rho}$
ρ = percent canopy cover
BA_{INF} = BA of annosus and/or Armillaria infected trees (m^2/ha)
ASP = aspect of the plot (NW=1, N=2, NE=3, or E=4)
BA_{LIVE} = live BA (m^2/ha)
BA_{STUMPS} = BA of stumps (m^2/ha)
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1, β_2, β_3, and β_4 are parameters to be estimated from the data

Height, live crown ratio, and crown base--Hypotheses 3-5

The initial regression models for height, live crown ratio, and crown base heights were similar to the canopy regression models, where the average height, LCR, and crown base by species and diameter class were regressed separately against total BA, infected BA, and aspect. A backward selection criterion was used to find the best model. Independent variables were excluded from the model if they had an α level ≥ 0.1. All analysis were performed using SPSS 10.0. The full model was as follows: $AVG = \beta_0 + \beta_1 BA_{INF} + \beta_2 ASP + \beta_3 BA_{TOTAL} + \varepsilon_i$

where:

AVG = average LCR, crown base height or canopy height within each diameter class.
BA_{INF} = BA of annosus and/or Armillaria infected trees (m^2/ha)
ASP = aspect of the plot (NW=1, N=2, NE=3, or E=4)
BA_{TOTAL} = Total BA
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1,β_2, and β_3 are parameters to be estimated from the data

Results

Trees/ha by diameter class--Hypothesis 1

The interaction term of infected BA and the indicator for the tall-emergent diameter class was significant ($F = 12.20$, $p = <0.0008$). Therefore, the slope of the tall-emergent diameter class was not equal to the slopes of the dominant and emergent diameter classes and the null hypothesis was rejected. The interaction term of infected BA and the indicator the dominant diameter class was not significant indicating that the dominant and emergent diameter classes had the same slopes. Results for the dominant and emergent diameter classes indicated that they

had the same slopes, but slightly different intercepts (Table 3.1, Fig. 3.3). The dominant diameter class had 1.064 (back transformed) times more live trees/ha than the emergent diameter class. This decreased by 34.8 trees/ha with an increase in infected BA (10-20 m^2/ha). An interaction between the tall-emergent diameter class and the dominant diameter class was discovered and unexpected. The results indicate that, the number of trees/ha (primarily Douglas-fir and ponderosa pine) increases in the tall-emergent class (>60 cm dbh) with an increasing infected BA (Fig. 3.3).

Table 3.1: Comparison of regression lines for hypothesis 1.

Interaction term	F-value	P-value
Infected BA * Indicator for tall-emergent	12.20	0.0008
Infected BA* Indicator for dominant	0.03	0.8672

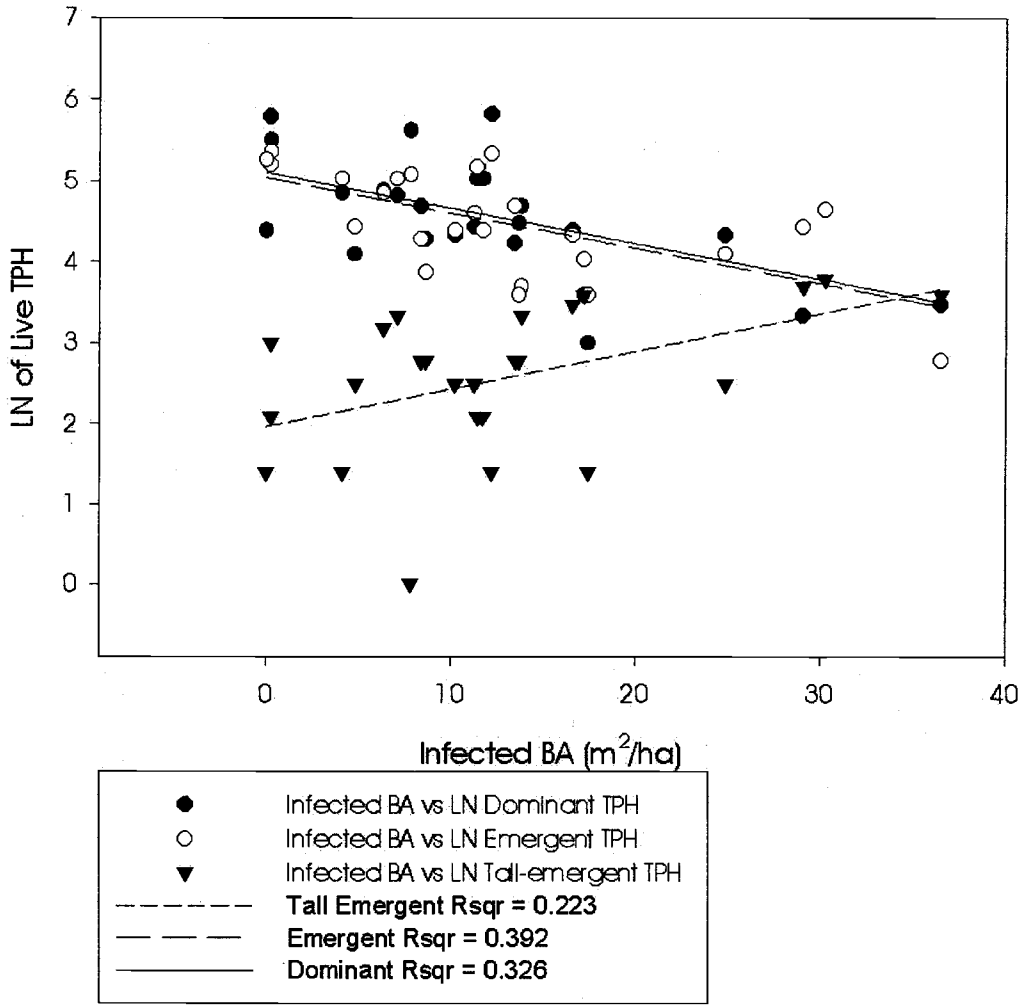

Fig. 3.3: The ln trees/ha by diameter class as they change with infected BA.

Further investigation into the proportion of white fir in each diameter class showed that the tall-intermediate, emergent, and dominant diameter classes are composed primarily (> 50%) of white fir across all infected BA's (Fig. 3.4). The proportion of white fir in the tall-emergent diameter class showed no relationship

with infected BA. However, there were proportionally more white fir in the dominant and emergent diameter classes with an increase in infected BA.

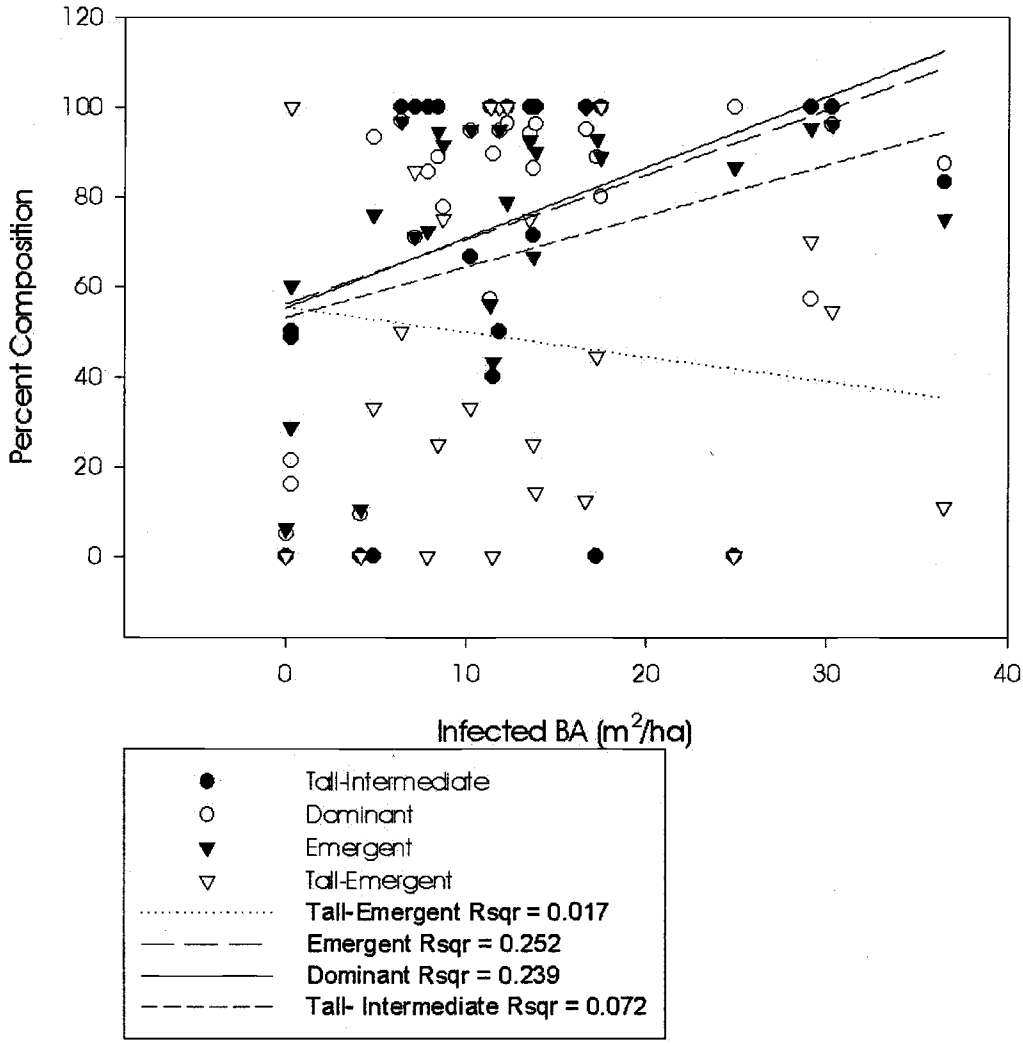

Fig. 3.4: Percentage of live white fir compared to other tree species as white fir changes with increasing infected BA by diameter class.

Canopy cover--Hypothesis 2

Total live BA best predicted the total canopy cover (Table 3.2). Infected BA had no significant relationship with total canopy cover or cover in the upper canopy

layer and the null hypothesis was not rejected. The live BA and infected BA significantly predicted the amount of canopy cover for the middle canopy layer, and the null hypothesis was rejected. Holding the live BA constant, the amount of canopy cover for the middle layer decreased by 0.57% (95% CI 0.07 to 1.07%) with an associated increase of 1 m^2/ha of infected BA. The live trees/ha and infected BA predicted cover for the lower canopy layer (Table 3.2), and the null hypothesis was rejected. However, this relationship was inverse of that in the middle layer (Fig. 3.3). An increase of 1 m^2/ha of infected BA increased the lower layer canopy cover by 0.59% (95% CI 0.18 to 1.01%) when live trees/ha were held constant.

Table 3.2: Regression results for canopy cover by layer.

Canopy Cover	Excluded Cases	Adjusted-R^2	Degrees of Freedom	F-value	P-value	Coefficient	Beta	Standard Error	P-value
Total	None	0.425	1,23	18.729	**0.000**	Constant	16.1212	8.0284	0.0565
						Live BA	1.1583	0.2676	**0.0002**
Upper	3	0.369	2,21	7.736	**0.003**	Constant	2.1918	6.8537	0.7523
						Infected BA	-0.3111	0.1804	0.0993
						Live BA	0.6501	0.2011	**0.0040**
Middle	None	0.304	2,22	6.245	**0.007**	Constant	5.9686	8.7613	0.5028
						Infected BA	-0.5697	0.2404	**0.0270**
						Live BA	0.6247	0.2640	**0.0272**
Lower	None	0.542	2,22	15.217	**0.000**	Constant	-4.2552	5.3919	0.4384
						Infected BA	0.5920	0.1995	**0.0071**
						Live TPH	0.0470	0.0094	**0.0001**

Height, live crown ratio, and crown base--Hypotheses 3-5

Live Crown Ratio--Hypothesis 3

The average LCR of trees in the tall-intermediate and dominant diameter classes significantly increased (longer live crowns) with increasing infection levels. This relationship was supported for ponderosa pine that were 5-29.9 cm dbh. The tall-intermediate and dominant ponderosa pine had greater live crown ratios with an increase in infected BA (Table 3.3 Fig. 3.5 and 3.6). The null hypothesis was not rejected for all Douglas-fir, white fir, and ponderosa pine > 30 cm dbh. The null hypothesis was not rejected for species other than ponderosa pine < 29.9 cm dbh.

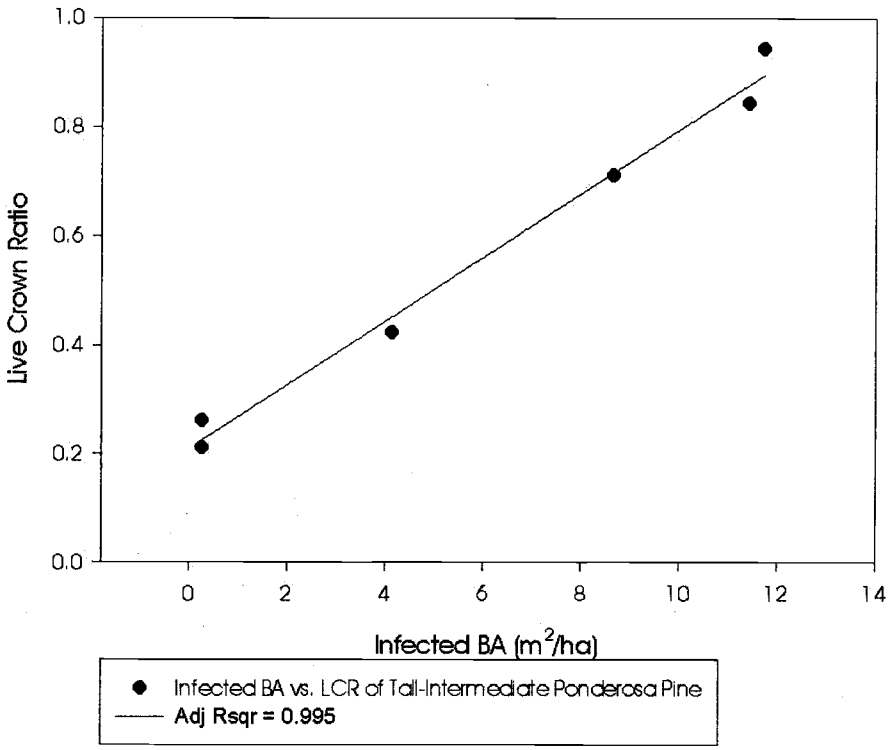

Fig. 3.5: Tall-intermediate (5-14.9 cm dbh) ponderosa pine LCR predicted by infected BA. Note that infected BA is only projected to 14 m^2. Ponderosa pine in this diameter class were not in plots with higher infection levels.

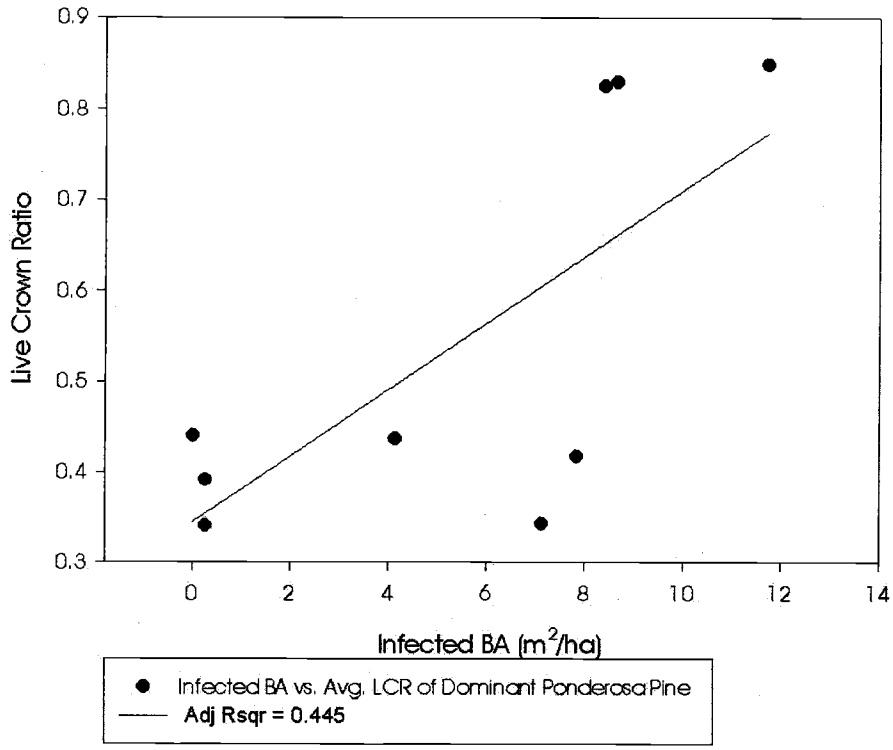

Fig. 3.6: Dominant ponderosa pine (15-29.9 cm dbh) LCR predicted by infected BA. Note that infected BA is only projected to 14 m^2. Ponderosa pine in this diameter class were not in plots with higher infection levels.

Table 3.3: Final regression models for average live crown ratio by diameter class and species. Numbers in bold indicate significance at the 0.05 α level. Abbreviations are as follows: PSME is Douglas-fir; PIPO is ponderosa pine; and ABCO is white fir.

Diameter class	SPECIES	Number of plots present	Adjusted R2	Degrees of Freedom	F-value	P-value	Significant Coefficients	Beta of Coefficients	Standard Error	Significance of Coefficients
Tall-intermediate 5-14.9	PSME	2	0.000	0,3			Constant	0.789	0.072	0.002
	PIPO	6	0.955	1,4	107.250	**0.000**	Constant	0.288	0.038	**0.002**
							Infected BA	0.051	0.005	**0.000**
	ABCO	21	0.000	0,20	*	*	Constant	0.676	0.060	0.000
Dominant 15-29.9	PSME	13	0.166	1,11	3.389	0.093	Constant	0.416	0.061	0.000
							Aspect	0.038	0.021	0.093
	PIPO	9	0.445	1,7	7.404	**0.030**	Constant	0.345	0.091	0.007
							Infected BA	0.036	0.013	**0.030**
	ABCO	25	0.000	0,24	*	*	Constant	0.543	0.026	0.000
Emergent 30-59.9	PSME	19	0.000	0,18	*	*	Constant	0.585	0.026	0.000
	PIPO	8	0.000	0,7	*	*	Constant	0.457	0.040	0.000
	ABCO	24	0.000	0,23	*	*	Constant	0.550	0.020	0.000
Tall-emergent >60	PSME	11	0	0,10	*	*	Constant	0.588	0.034	0.000
	PIPO	11	0.596	1,9	15.769	**0.003**	Constant	0.123	0.091	0.209
							Aspect	0.097	0.025	**0.003**
	ABCO	21	0	0,20	*	*	Constant	0.616	0.027	0.000

*F-values and p-values are not given because no explanatory variables were significant at the α 0.1 level.

Height--Hypothesis 4

The null hypothesis 4, the average height of trees is not related to infection level, was rejected for Douglas-fir 15-59.9 cm dbh and for ponderosa pine 5-14.9 cm dbh. In most cases (white fir >5 cm dbh, ponderosa pine >15 cm dbh, and Douglas-fir 5-14.9 and >60 cm dbh), the explanatory variable, infected BA, did not significantly predict the average height (Table 3.4) and the null hypothesis was not rejected. There was a weak, non-statistically significant relationship between tall-emergent white fir ($p= 0.052$) and infected BA. The dominant and emergent Douglas-fir had a weak statistically significant relationship ($p= 0.015$ and 0.24 respectively) with infected BA. The tall-intermediate ponderosa pine had a weak statistically significant ($p= 0.018$) relationship with infected BA.

Table 3.4: Final regression models for average height by diameter class and species. Numbers in bold indicate significance at the 0.05 α level. Abbreviations are as follows: PSME is Douglas-fir; PIPO is ponderosa pine; and ABCO is white fir.

Diameter class	SPECIES	Number of plots present	Adjusted R^2	Degrees of Freedom	F-value	P-value	Significant Coefficients	Beta of Coefficients	Standard Error	Significance of Coefficients
Tall-intermediate 5-14.9	PSME	4	0	0,3	*	*	Constant	7.5132	4.0141	0.158
	PIPO	6	0.739	1,4	15.161	**0.018**	Constant	14.775	2.117	0.002
							Infected BA	-1.063	0.273	**0.018**
	ABCO	21	0.294	2,18	*	*	Constant	6.198	0.685	0.000
Dominant 15-29.9	**PSME**	13	0.412	2,10	5.200	**0.028**	Constant	14.947	2.634	0.000
							Aspect	2.080	0.927	0.049
							Infected BA	-0.440	0.150	**0.015**
	PIPO	9	0.272	1,7	3.992	0.086	Constant	22.150	3.122	0.000
							Infected BA	-0.920	0.460	0.086
	ABCO	25	0.088	1,23	3.303	0.082	Constant	65.390	9.805	0.095
							Infected BA	-11.503	1.954	0.107
Emergent 30-59.9	**PSME**	19	0.222	1,17	6.14	**0.024**	Constant	28.901	2.1264	0.000
							Infected BA	-0.3821	0.1542	**0.024**
	PIPO	8	0.000	1,7	*	*	Constant	25.607	2.348	0.000
	ABCO	24	0.000	0,23	*	*	Constant	24.147	0.538	0.000
Tall-emergent >60	PSME	11	0.27	1,9	4.701	0.058	Constant	32.0995	3.0375	0.000
							Infected BA	0.4106	0.1894	0.058
	PIPO	11	0.000	0,10	*	*	Constant	37.460	2.117	0.000
	ABCO	21	0.142	1,19	4.305	0.052	Constant	30.644	2.022	0.000
							Infected BA	0.259	0.125	0.052

*F-values and p-values are not given because no explanatory variables were significant at the α 0.1 level.

Crown base--Hypothesis 5

The null hypothesis 5, the average height to the bottom of the live crown is not related to infection level, was rejected for ponderosa pine 5-14.9 cm dbh ($p= 0.004$). Douglas-fir 15-29.9 cm dbh had a negative, non-statistically significant relationship with infected BA ($p= 0.07$). Infected BA had a weak, non-statistically significant relationship with the average crown base heights of Douglas-fir in the dominant diameter class. The average crown base results were similar to the live crown ratio results, where the tall-intermediate ponderosa pine and dominant Douglas-fir crown bases were closer to the ground with higher infected basal areas (Table 3.5). The null hypothesis was not rejected for all other species and diameter classes.

Table 3.5: Final regression models for average height to crown base by diameter class and species. Numbers in bold indicate significance at the 0.05 α level. Abbreviations are as follows: PSME is Douglas-fir; PIPO is ponderosa pine; and ABCO is white fir.

Diameter class	Species	Number of plots present	Adjusted-R^2	Degrees of Freedom	F-value	P-value	Significant Coefficients	Beta of Coefficients	Standard Error	Significance of Coefficients
Tall-intermediate	PSME	4	0.000	0,3	*	*	Constant	2.953	2.033	0.242
	PIPO	6	0.877	1,4	36.741	**0.004**	Constant	10.457	1.164	0.001
							Infected BA	-0.909	0.150	**0.004**
	ABCO	21	0.000	0,20	*	*	Constant	3.044	0.488	0.000
Dominant	**PSME**	13	0.233	2,10	2.820	**0.017**	Constant	5.268	1.769	**0.014**
							Aspect	1.146	0.623	0.095
							Infected BA	-0.204	0.101	0.070
	PIPO	9	0.337	1,7	5.075	0.059	Constant	13.675	2.592	0.001
							Infected BA	-0.861	0.382	0.059
	ABCO	25	0.000	0,24	*	*	Constant	6.558	0.578	0.000
Emergent	PSME	19	0.000	0,18	*	*	Constant	12.183	1.130	0.000
	PIPO	8	0.000	0,7	*	*	Constant	12.922	1.453	0.000
	ABCO	24	0.220	1,22	7.506	**0.012**	Constant	6.880	1.988	0.002
							Aspect	1.712	0.625	**0.012**
Tall-emergent	PSME	11	0.000	0,10	*	*	Constant	13.888	1.404	0.000
	PIPO	11	0.448	1,9	9.125	**0.014**	Constant	38.829	6.435	0.000
							Aspect	-5.229	1.731	0.014
	ABCO	19	0.000	0,20	*	*	Constant	12.364	0.998	0.000

*F-values and p-values are not given because no explanatory variables were significant at the α 0.1 level

Discussion

Root disease changes canopy cover and structure; however, these relationships differ for each canopy layer, species, and diameter class. Total canopy cover showed no significant relationship with the amount of root disease, but each canopy layer responded differently to an increase in the amount of root disease.

Infected BA did not change the total canopy cover or canopy cover in the upper layer. This could be because there was not a strong relationship between the live BA and infected BA (Table 2.1). Infected BA significantly predicted the mortality in each diameter class (see Chapter II). However, it did not have any correlation with live BA, which was significantly correlated with the ground-based canopy cover estimates (Table 2.1).

The middle and lower canopy layers both displayed significant relationships with infected basal area. The middle canopy layer showed the expected relationship, where cover decreased with an associated increase in infected basal area. There were high levels of mortality of white fir in this canopy layer in plots with higher levels of root disease. I expected the canopy cover of the lowest layer to decrease with the interaction of budworm and root disease. However, the opposite was true; a positive relationship was found. Areas where there was more root disease had increases in the lower canopy-cover layer. Chapter II focused on the percentage of white fir retained in each diameter class. The results indicated that infected BA in the tall-intermediate diameter class (5-14.9 cm dbh) did not influence the percentage of live trees/ha. Therefore, it would seem that the results

presented here for the lower canopy layer contradict results presented in chapter two. This may indicate a difference between the percentage of live trees and the absolute number of live trees. However, the canopy cover measurements used in this analysis were independent of tree diameter class because of the methodology, and they included all trees taller than 1.4 m, whereas the analysis in chapter two only included trees ≥ 5.0 cm dbh. Therefore, the ground-based canopy estimates of the lower layer may more accurately account for the trees >1.4 m tall but < 5.0 cm dbh.

Changes in canopy structure were also dependent on species and size class. The canopy structure changed with an increase in infected basal area. In areas with higher levels of infection, there were more tall-emergent trees/ha retained in the overstory. This corresponded with a decrease in the number of trees/ha in the dominant and emergent diameter classes. Figure 3.4 demonstrated that there were proportionally more white fir in the dominant and emergent diameter classes at higher infection levels, possibly because the lack of fire-created disturbances increased the amount of white fir in these two diameter classes and decreased the regeneration of the early seral species. There may also be more inoculum in these stands that has created small-scale disturbances over time to increase the regeneration of late seral species. This increase in the proportion of white fir corresponds with a decrease in the number of live trees/ha. More mortality is occurring in these plots because of the abundance of susceptible hosts, thus decreasing the live trees/ha.

As infected BA increased, there were fewer live trees/ha in the dominant and emergent diameter classes and an increase in trees/ha in the tall-emergent diameter class. Mortality of white fir in areas with high levels of root disease decreases stand density, thus decreasing competition, which may allow for an increased survival of tall-emergent Douglas-fir and ponderosa pine. For example, plot 12 (TS6P9 in fig. 1.2) had 38 m^2/ha of infected BA and had 32-ponderosa pine/ha that were >60 cm dbh. This creates a favorable picture, at least for the short term, in maintaining tall-emergent trees in stands where root disease is present. However, at the higher infection levels the smaller diameter classes had a large component of white fir with less than 20% ponderosa pine and Douglas-fir. There are few trees besides white fir present to replace the tall-emergent trees (Fig. 3.4). This changes the canopy cover type and canopy cover stratification, which has the potential to be detrimental in reaching management objectives of maintaining and managing for stand structures that support late-successional species.

The live crown ratio and crown base results show that ponderosa pine retains longer crowns in areas with higher infected BA's. This is possibly due to the mortality of the white fir, which in turn releases the ponderosa pine and allows for longer live crowns. The results of ponderosa pine LCR in the smaller diameter classes implies that these tree species are healthier in areas where the white fir was killed by root disease pathogens. Lower live crowns could be due to the release of the ponderosa pine. Ponderosa pine was only found in plots with 0-14 m^2 of

infected BA. Therefore, the response of LCR of tall-emergent ponderosa pine in areas with higher levels of root disease is not known.

There was not a strong linear relationship between height to live crown base and the amount of root disease. Therefore, ladder fuels and dead lower crown branches should not increase the chance of a crown fire developing when compared to stands without root disease (Keyes and O'Hara 2002).

These stands are very dynamic systems with several factors contributing to the differences in canopy cover and structure. In this case it is not easy to predict differences in the horizontal and vertical distribution of trees. Significant relationships with infected basal area as an explanatory variable still had relatively small adjusted-R^2 values. Further research is needed to validate and define the relationships discovered in this study. This study had a relatively small sample size, and is limited to mixed-conifer stands in central Oregon. Since the level of susceptibility of different hosts is largely dependent on site conditions, these relationships need to be explored in areas other than central Oregon mixed-conifer forests.

ROOT DISEASE IMPACTS ON UNDERSTORY FORB AND SHRUB SPECIES COMPOSITION AND COVER.

Literature review

Changes in understory species

As a result of changes in canopy cover and canopy stratification, there is a potential for change in understory species richness and diversity. The impacts of disturbances on plant community composition and diversity has been hypothesized and tested in several ecosystem types under several different disturbance regimes. The intermediate disturbance hypothesis (IDH) (Connell 1978, Huston 1979) states that diversity is highest on sites that have experienced intermediate frequency of disturbance that prevents competitive exclusion and is lower on sites that have experienced very high or very low disturbance frequencies. IDH was developed, studied, and supported in marine and tropical systems. IDH has since been tested and studied on several scales, in a variety of ecosystems, on a variety of species groups, with mixed results. Vujnovic et al. (2002) found results that supported IDH where total species diversity was highest at intermediate levels of disturbance (cattle grazing) in grasslands of central Alberta. Others have found that moss and lichen species diversity may be the highest at some intermediate level of disturbance (Vujnovic et al. 2002). Schwilk et al. (1997) tested the hypothesis in fire adapted shrub lands in South Africa were they found that the opposite was true, and sites with intermediate fire frequencies had the lowest species richness. Beckage and Stout (2000) found no relationship among understory species richness, diversity,

stem abundance, and the number of burns in a pine savanna in Florida. Little has been done with fungal pathogen related disturbance and the IDH hypothesis.

Hansen (1999) addressed the changes in diversity that natural and non-native pathogens present in native and disturbed forest ecosystems. Native root pathogens have slow-acting indirect effects on forest diversity and species richness (Hansen 1999). In high elevation mountain hemlock (*Tsuga mertensiana* (Bong.) Carr) forests, gaps created by *Phellinus weirii* have been found to increase tree species diversity (McCauley and Cook 1980).

Holah et al. (1993) found that the selective removal of Douglas-fir by *P. weirii* in western Oregon stands indirectly changed the community composition. They also found that changes in the abundance and cover of understory shrub species were largely species-specific as well as site dependent. These changes also varied between sites sampled in the Coast and Cascade Ranges of Oregon. Holah et al. (1993) found an increase in herbaceous cover within disease centers across all study sites as compared to adjacent uninfected areas. They noted that the community response to the disturbance was "largely a reorganization of populations already present on the site, rather than the establishment of new species." They found that diversity of plant species in infected areas in the Cascades decreased as compared to adjacent uninfected areas, whereas the opposite relationship existed on sites in the Coast Range.

Godfree (2000) found that there was an increase in the understory species cover in lodgepole pine stands infected with dwarf mistletoe. However, the forb

and shrub species richness and diversity were not significantly different in stands with dwarf mistletoe as opposed to stands without dwarf mistletoe.

Objectives and hypotheses

The objective of my study was to determine if understory species richness and total understory cover increase with increasing root infection levels Root infection level and abiotic variables including slope, aspect, and elevation would directly influence the tree size class factors in the structural models (Chapters 2 and 3). Differences in size classes would alter overstory canopy cover and, in turn, impact the richness and total cover of the understory. With an increasing infected BA, the stands will contain less dominant white fir, have lower canopy cover, and these stands will have greater understory forb and shrub cover, and greater species diversity.

Hypotheses:

1) $\mathbf{H_O}$: The understory forb and shrub cover is not related to canopy cover.

$\mathbf{H_A}$: The understory forb and shrub cover will increase with decreasing canopy cover.

2) $\mathbf{H_O}$: The understory forb and shrub cover is not related to infection levels.

$\mathbf{H_A}$: The understory forb and shrub cover will increase with increasing infected BA.

3) $\mathbf{H_O}$: Species richness is not related to canopy cover.

$\mathbf{H_A}$: Species richness will increase with decreasing canopy cover.

4) $\mathbf{H_O}$: Species richness is not related to infection level.

H_A: Species richness will increase with increasing infected BA.

Methods

Forb and shrub data collection

I collected data on the understory shrub and herbaceous cover and their species composition. Since Petaisto et al. (1999) collected data on the understory species composition as an ocular assessment on the stand level, these data were recollected using two smaller plots. The 1 m^2 plots were used to determine the impact of canopy cover on the diversity and cover of understory species (Fig. 1.3). In each 1 m^2 plot, all understory plant species were identified (grasses and sedges were grouped together), and an ocular estimate of percent cover in the plot was noted. Estimates of cover were grouped as 1, 3, 5, 10, 20, 30, 40, 50, 60, 70, 80, 90, 100, and 100+%. Since plant species can occur in several strata, it is possible to obtain values over 100%; however, this was rare on these plots. Plant species that were not identified were classified as "other". Since ocular estimates are biased, I and at least one other person discussed the percent cover to maintain consistency among plots. In most cases, the 1 m^2 plots were divided into quarters to reduce bias.

I then classified all of the species into one of four categories (open stands, open to filtered light, filtered light, and filtered to closed stands). These categories described the stand and light conditions where the plant species were usually found. This was done using several references (Parish et al. 1996, Hopkins and Rawlings 1985, Johnson 1993) and with the help of Signe A. Hurd, a Deschutes National

Forest botanist. All species identified in this study were cross-referenced with the Deschutes National Forest plant list to verify accurate identification.

The line-intercept transects used to estimate canopy cover (see previous chapter) were placed so that each 1 m^2 plot was within 2 m of a transect. Therefore, the canopy cover estimates were indicators of the canopy cover directly over the 1 m^2 plots.

Statistical analysis

Initial data were explored using descriptive statistics to look for any trends in species cover and diversity. Further analysis was done using multiple regression techniques to explore the relationships between the canopy cover, infected BA, and the response of the shrub, forb, and grass cover. The models for analyzing the forb and shrub cover and richness are based on the results found in Chapters II and III, and the full models for forb, shrub, or grass percent cover were regressed against aspect, percent overstory canopy cover, and infected BA. Variables included in the full model were first checked for correlation. Elevation and live BA were significantly correlated with the total canopy cover (Table 2.1); therefore, only total canopy cover was included in the model. Results from objective two (see Chapter III) indicated that infected BA does not significantly change the total canopy cover; therefore, both infected BA and total canopy cover were included as explanatory variables in the full model.

A similar analysis was done for individual species. For species that had more than three observations on separate plots, a regression model determined

whether the percent cover of the forb species increased or decreased and if this relationship was statistically significant. Since several species only had a few observations (out of 25 possible) only three variables, percent canopy cover, aspect, and infected BA, were included in the full model. A backward selection method was used to determine the reduced model that best predicted forb cover. If a forb species was missing from a plot (having zero cover), that plot was not included in the analysis. Zeros were not included in the model because of the uncertainty as to whether species were absent because of the site conditions (stand structure, recent disturbance, aspect, total overstory cover etc.) or because of a lack of available seed source.

In order to address the species richness hypothesis, the total number of forb, shrub, and grass species present on each site (grasses were grouped together) was regressed against aspect, percent canopy cover, and infected BA. For this study, species richness is defined by the number of shrub, forb, and grass species.

Results

Species cover--Hypotheses 1 and 2

Initial data results are listed in Table 1. *Pteridium aquilinum* var. *pube* L. Kuhn (bracken fern), *Trientalis latifolia* Hook (starflower), *Chimaphila umbellata* (L.) Bart. (Prince's pine), and *Hieracium* spp. (hawkweed) were found in the majority of the plots and across all canopy cover and infection levels. Unfortunately, there were no obvious trends in forb cover or frequency by species associated with the overstory canopy cover. Most forb species were found under all

canopy cover estimates. For example, *Pyrola* spp. (wintergreen) and *Linnaea borealis* L. (twin flower), which are generally found in closed stands (Parish et al. 1996), occurred more commonly in plots with lower canopy cover.

Table 4.1: Plant species percent cover and frequency found in the study plots. Only species that occurred on two or more 0.25 ha plots are listed. Frequencies are listed in the parentheses and are based on the number of $1m^2$ plots they occurred on (0-10). Grasses includes both sedges and grasses. Plots are arranged from lowest to highest canopy cover.

Plot Number	14	15	2	9	10	5	4	13	1	8	11	3	7	22	23	6	16	21	12	24	18	20	17	19	25
Open stands																									
Achillea sp.																					2.5(2)			0.7(3)	
Cirsium vulgare	1.2(6)	1.7(5)		1.8(6)	1.6(4)	0.7(2)		0.7(3)		1.3(2)	1.6(5)	1.2(6)						2.2(7)	1.6(3)			1(1)			
Cynoglossum occidentale	0.1(1)					0.5(1)																	0.5(1)		
Epilobium angustifolium	0.3(1)	0.3(1)		2.3(2)		0.3(1)			0.5(1)				0.1(1)				1.9(5)					0.3(3)	1.4(3)		
Heiracium sp.	1.3(5)	1.6(6)	1.8(4)	1.5(5)	3.2(9)	1.3(9)	3.8(6)	1.1(5)	0.9(7)	1.2(8)	0.9(7)	1.4(8)	2.1(6)	0.7(3)	0.7(3)	5(9)	2.6(4)	1(6)	1.6(6)	0.5(5)	1.2(6)	2.4(8)	1.5(10)	0.8(4)	0.2(2)
Phacelia hastata				0.3(1)		0.5(1)													0.2(2)						
Phacelia sericea	2.1(4)										0.7(3)	0.1(1)													
Pteridium aquilinum	17.5(5)	2(1)		2.6(3)	3(2)	8(3)	0.2(1)	5.8(5)	9(3)	2.5(2)	18.6(9)	6.5(4)		0.3(1)	4.5(3)		18.5(7)	16.6(8)	15.5(7)	3.5(4)			4.3(3)		2.5(2)
Rubus ursinus	0.4(3)	6.5(5)	1.1(3)			6.1(7)	0.6(2)	2.5(3)	5.8(4)	1.1(2)		7.1(9)								0.8(2)					4.5(9)
Vicia sp.																						1(2)		0.2(2)	
Open to filtered light																									
Anaphalis margaritacea								0.3(1)										1.1(2)							
Arctostaphylos uva-ursi			0.1(1)			0.8(4)						0.1(1)													
Aster sp.																		1.1(3)	0.1(1)						
Fragaria sp.	0.7(3)	0.9(3)	0.2(2)			0.6(4)	0.6(3)	0.1(1)	1.3(2)	0.8(6)	1.3(2)	3.2(5)	0.1(1)	0.1(1)			1.2(4)	3.8(5)			1.4(3)			0.6(2)	
Galium sp.*	0.7(5)	0.9(5)	0.8(4)		0.4(2)	0.6(4)	0.6)3)	0.4(4)	2.5(8)	1.3(4)	0.3(3)	1.3(5)	0.6(4)					0.5(3)	1(6)						
Lactuca muralis	0.5(1)		1.4(6)						19.6(8)				0.1(1)									0.4(2)			
Lupinus sp.				1(4)	0.1(1)					0.2(2)								0.4(2)							
*Pedicularius racemosa**		0.1(1)			1.9(4)			1(2)			1.6(5)														
*Smilacina racemosa**		1.3(5)		2.8(7)	2.7(4)	2.1(4)		0.6(2)	0.5(3)	0.6(4)	0.6(4)	0.2(2)	0.6(6)			0.6(2)		4.8(7)	1.2(3)			4(2)			
Taraxacum officinale										0.2(2)							1(1)					1(1)			

Table 4.1 continued:

Plot Number	14	15	2	9	10	5	4	13	1	8	11	3	7	22	23	6	16	21	12	24	18	20	17	19	25
Filtered light stands																									
Anemone sp.	1.5(5)	3.2(6)		0.2(2)	0.8(4)			5.6(5)			0.9(5)							0.9(3)	0.5(1)						
Asarum caudatum	0.1(1)											0.3(3)													
*Campanula scouleri**	0.3(1)	0.4(2)		1(3)	1.4(4)						1.2(4)	0.5(2)						1.4(8)	0.8(3)						2.2(6)
*Clintonia uniflora**		4.6(6)		0.9(3)	5.6(7)	1.6(3)		8.1(8)		0.1(1)	1.3(3)							1.4(3)	3.7(7)						
Osmorhiza sp.*	0.1(1)	0.4(2)		0.2(2)	0.9(5)	0.1(1)		0.4(2)	0.3(3)	0.8(4)	0.1(1)	0.6(6)	1.2(3)	0.1(1)				0.2(2)	1.2(3)						
Stellaria sp.										0.7(3)		1.2(8)													
*Trientalis latifolia**	0.9(5)	1.7(7)	0.6(4)	0.2(2)	0.8(2)	0.6(6)	0.2(2)	1.7(7)	0.8(4)	1.2(8)	1.7(7)	2.9(8)	1.3(5)	0.5(1)	1.3(2)	0.7(3)	4.2(5)	1.4(6)	0.3(3)	1.3(5)		0.1(1)	1.4(4)	0.2(2)	0.7(5)
Filtered to closed stands																									
*Chimaphila umbellata**	0.5(3)	1.7(4)		0.1(1)	6.3(7)	1.4(6)	1(1)	6.4(7)	0.2(2)	1.3(2)	3.5(5)	1.6(3)	10.6(8)	3.7(5)	0.1(1)	0.6(4)	7(3)	1.8(3)	10.6(8)	12.3(7)	0.1(1)	1(1)	1(4)	7.6(4)	2.8(3)
*Linnaea borealis**	4(2)	8(3)				4.2(3)		1(1)	0.5(1)	0.1(1)		5.1(2)													
*Pyrola picta**					0.4(2)	0.1(1)			0.1(1)		0.1(1)		0.1(1)			0.1(1)		0.1(1)							
Pyrola sp.*		0.1(1)		0.1(1)	1.4(4)			0.8(4)										0.2(2)							
Unclassified species																									
Grasses*	7.6(9)	13.5(9)	19.7(10)	7.5(8)	9.9(9)	22.6(10)	13.6(7)	3.5(8)	13.5(8)	7.5(8)	8.1(9)	24.1(1)	19(8)	1.4(3)	3.7(3)	9.9(8)	3.5(7)	5.9(7)	18.2(7)		10.1(7)	16.3(7)	13.8(7)	2.2(5)	4.6(5)
Other	1.5(7)	0.5(3)	1.2(4)	1(4)		0.3(3)	1.8(5)	0.4(3)	1.5(4)	1.5(7)	2.7(6)	0.4(4)	0.8(6)			0.7(5)	0.1(1)	0.3(3)	1.3(2)			0.5(1)	0.2(2)	0.2(2)	0.1(1)
Canopy Cover (%)	18	26	31	34	34	36	38	41	43	46	49	50	51	54	54	54	58	58	59	60	63	64	68	74	75
Live BA (m^2/ha)	8.02	23.74	23.96	18.56	26.22	21.04	31.54	18.99	34.62	29.29	24.24	18.87	32.37	31.29	41.62	42.8	25.13	29.68	25.18	29.85	39.56	31.21	38.9	42.84	30.77
Infected BA (m^2/ha)	17.42	10.22	13.45	8.67	13.81	11.75	6.36	13.67	29.1	17.2	24.91	4.83	16.59	7.85	12.19	30.28	11.28	8.41	36.51	4.13	0.26	11.42	7.13	0.25	0

Note: Frequencies are listed in the parenthesesand are based on the number of 1m2 plots they occurred on (0-10). The species listed occurred in at least two large plots.

*Grasses includes both sedges and grasses.

The null hypothesis 1 was rejected; that is, grass cover decreased with an increase in overstory cover. The null hypothesis 1 was not rejected for shrub or forb cover. The null hypothesis 2 was rejected; forb cover had a significant positive response to an increase in infected BA. Forb cover had a weak ($p= 0.036$, adj-R^2 0.142) positive relationship with the infected BA (Table 4.2). Null hypothesis 2 was not rejected for shrub and grass cover. For both the grass and shrub models, none of the explanatory variables significantly predicted the amount of cover.

For most of the forb species none of the independent variables predicted the percent cover. However, there were a few exceptions (Table 4.2). The cover of *Lactuca muralis* (L.) Fresen (Wall lettuce), *Pteridium aquilinum*, and *Pyrola* spp. all significantly increased with an increasing infected BA. *P. aquilinum* increased with infected BA and was expected due to its occurrence on disturbed sites (Parish et al. 1996, Johnson 1993). The same relationship occurred with *L. muralis*, an introduced, annual or biennial herb that grows in moist forest openings and edges, and roadsides (Parish et al. 1996). However, the increase in *Pyrola* spp. is unexpected because they usually occur in filtered or closed stands (Parish et al. 1996). *Campanula scouleri* Hook. decreased in cover with an increase in infected BA (Table 4.2).

Table 4.2: Final model results of backward regression model selection for percent cover and species richness.

Forb Species	Adjusted R^2	F-statistic	Degrees of Freedom	P-value	Coefficient	Beta	Standard Error	P-value
Grass cover	0.035	1.876	1,23	0.184	Constant	16.823	4.766	0.002
					Overstory cover	-0.127	0.092	0.184
Forb cover	0.142	4.97	1,23	**0.036**	Constant	14.343	4.284	0.003
					Infected BA	0.609	0.273	**0.036**
Shrub cover	0.088	3.325	1,23	0.081	Constant	-3.345	8.530	0.699
					Aspect	4.832	2.650	0.081
Campanula scouleri	0.757	16.63	2,7	**0.002**	Constant	-0.654	0.333	0.090
Scouler's harebell					Aspect	0.590	0.103	**0.001**
					Infected BA	-0.024	0.010	**0.043**
Galium spp.	0.346	4.436	2,11	**0.039**	Constant	1.353	0.517	0.024
Bedstraw					Aspect	-0.439	0.148	**0.013**
					Overstory cover	0.023	0.013	0.098
Lactuca muralis	0.815	18.663	1,3	**0.023**	Constant	-15.841	4.963	0.050
Wall lettuce					Infected BA	1.150	0.266	**0.023**
Pteridium aquilinum	0.232	6.446	1,17	**0.021**	Constant	2.540	2.329	0.291
Bracken fern					Infected BA	0.379	0.149	**0.021**
Pyrola spp.	0.719	11.249	1,3	**0.044**	Constant	-1.593	0.645	0.090
Wintergreen					Infected BA	0.193	0.058	**0.044**
Trientalis latifolia	0.079	2.981	1,22	0.098	Constant	1.949	0.543	0.002
Star flower					Aspect	-0.286	0.166	0.098
Species Richness	0.311	11.833	1,23	**0.002**	Constant	28.421	4.040	0.000
					Overstory Cover	-0.270	0.078	**0.002**

Species richness--Hypothesis 3 and 4

Null hypothesis 3 was rejected; species richness increases with a decrease in canopy cover (Table 4.2). Hypothesis 4 was not rejected; infected BA had no significant relationship with species richness.

Discussion

Changes in the understory species composition in areas with root disease, like all systems, are dynamic and difficult to predict. The presence of more root

disease or mortality in a stand has several direct impacts on the stand structure and canopy cover. This in turn has indirect impacts on the understory forb, shrub, and grass species cover, composition, and frequency. These impacts are difficult to discern, as they are based on individual forb, shrub, and grass species' life histories.

Forb species presence, like tree species, is a function of seed source, seed dispersal, site conditions, stand structure, and stand disturbance history. In these plots, the presence of some species, such as *Pyrola* spp., in sites with low canopy cover might reside in the fact they were on site before high levels of disturbance.

It was surprising that the chinquapin (*Chrysolepis chrysophylla* (Dougl. ex Hook.) Hjelmqvist) and snowbrush (*Ceanothus velutinus* Dougl. ex Hook. var. *velutinus*) cover had no significant relationship with canopy cover or infected basal area in this study. In central Oregon these shrub species tend to colonize sites where there is disturbance. Sites with clearcuts, fires, and road banks are easily colonized by snowbrush. In some of the undisturbed plots (such as plots 13, 24, and 25, or TS6P14, TS15P2, and P10 in fig. 1.2), chinquapin was a large component of the understory. Snowbrush covered half of plot 9 (TS5P1 in fig. 1.2) with large white fir on the plot edges and white and Douglas-fir regeneration in the plot center.

It is difficult to link these results with the intermediate disturbance hypothesis since all plots experienced severe defoliation ten years ago. However, studies in these systems have the potential to test the IDH.

Predicting the change in diversity due to disturbance agents is largely unpredictable, as is shown by these results and other studies (Holah et al. 1993,

Hansen 1999). This stresses the importance of avoiding generalizations about the diseases increasing or decreasing diversity. Instead, these generalizations should focus specifically on the stand type, stand structure, successional pathway, disease and its disturbance regime, and individual species' life history (Holah et al. 1993).

ARMILLARIA AND ANNOSUS IMPACTS ON FUEL LOADINGS AND DOWN COARSE WOODY MATERIAL

Literature review

Fire and root pathogens

Fire suppression has increased stand density in many mixed-conifer stands. Agee (1994) believes that some of the most prominent effects of fire suppression have occurred in the white fir, Douglas-fir, and grand fir series. In these stands fire suppression has favored a stand structure with few old trees and a large percentage of young cohorts usually less than 40 cm dbh. These stand structures are more susceptible to defoliating insects (Wickman 1992), pathogens, and high intensity wildfires because of the loss of the pine and larch overstory and the creation of fuel ladders (Agee 1994).

Fire suppression activities are often blamed for the increase in host abundance and subsequent mortality from *Armillaria ostoyae*. Some believe that this has increased the area colonized by the fungus in the western United States (Hagle and Schmitz 1993). However, this change in species composition and stand structure may only result in a greater expression of the fungus in susceptible species and not an increase in the distribution of the fungus (Ferguson et al. 2003). Studies show that the rapid rate of establishment of new *A. ostoyae* genets needed to significantly change the area the fungus has colonized is rare (Adams 1974, Shaw and Roth 1976, Ferguson et al. 2003).

Ferguson et al. (2000, 2003) found a single *A. ostoyae* genet occupying 965 ha. They estimated the genet to have persisted 1900-8650 years. This age was

based on the area the genet occupied and its estimated rate of spread. A genet occupying an area this large and persisting this long has many impacts on the stand structure and has strong implications for future management activities. Even using the younger age, the single genet has withstood several drastic changes in species composition and structure, and several wildfires of varying intensities. Fires in these forests were historically of low- to moderate-severity with occasional severe fires (Agee 1994).

Dickman and Cook (1989) mapped fire occurrence, fire size, and *Phellinus weirii* populations in a high-elevation mountain hemlock and lodgepole pine forest in west-central Oregon. By dating the clones and fires, they hypothesized that fungal populations might impact the initiation and spread of a fire by creating fuel loadings and fuel ladders into tree crowns. They also concluded that the *P. weirii* clones had survived at least one catastrophic wildfire, if not several.

Although the amount of mortality caused by Armillaria and annosus root diseases has been quantified in Oregon (Filip 1977, Filip and Goheen 1984), the topic of higher fuel loadings (down woody material) as a result of root disease has not been quantified. However, it has been noted to increase the accumulation of down coarse woody material (Hansen and Lewis 1997). Beatty et al. (1995) identified amount of coarse woody material to be an indicator of forest health. Their preliminary results did not indicate that there was any relationship between the amount of coarse woody material and the presence of root pathogens.

The effects of prescribed fire on fungal populations are being studied, yet more research is needed (Thies 1990). Prescribed and natural fires have very few direct impacts on the amount of root disease in a stand. Direct impacts on *H. annosum* may include the destruction of fruiting bodies on the base of stumps and infected trees (Thies 1990). Filip and Yang-Erve (1997) studied the impact of prescribed burning on the viability of *A. ostoyae* in eastern Oregon mixed-conifer stands. They found that fall prescribed burning significantly reduced the recovery of *A. ostoyae* immediately following burning down to 8 cm in the soil. At 30 cm soil depth, there was no difference between burned and unburned plots. Spring burning showed no effect on reducing the recovery of the pathogen.

There were indirect effects of burning on *Armillaria ostoyae* populations noted by Reaves et al. (1990). They evaluated fire effects in a ponderosa pine forest in central Oregon on the soil population of *Trichoderma* spp. They found that the ash layer that accumulated after prescribed burning changed the soil properties by increasing the concentration of cations in the soil. They noted that these cations have a negative effect on the *in vitro* growth and development of *A. ostoyae*. They also found that leachates from the ash layer that accumulate after a prescribed fire had a positive effect on *Trichoderma* spp., which then reduced the growth of rhizomorph formation by *A. ostoyae*.

Other indirect effects of prescribed or natural fire may include a shift to earlier seral species including western larch and ponderosa pine that are more resistant to the major root disease pathogens in the west (Thies 1990).

Objective and hypotheses

The objective of this study was to determine if fuel loading (down wood) increases with increasing root disease (infected BA).

Hypotheses:

1) **H_O:** Total fuel loadings are not related to infection level.

H_A: Total fuel loadings will increase with increasing infection levels.

2) **H_O**: Decayed and non-decayed 1000-hr fuels are not related to infection level.

H_A: Decayed and non-decayed 1000-hour fuels will increase with increasing infection levels.

3) **H_O**: Average fuel height above ground is not related to infection level.

H_A: Average fuel height above ground will increase with increasing infection levels.

4) **H_O**: Average duff depth is not related to infection level.

H_A: Average duff depth will increase with increasing infection levels.

5) **H_O**: The smaller fuels (1-, 10-, and 100-hour fuels) are not related to increasing infection level.

H_A: Smaller fuels (1-, 10-, and 100-hour fuels) will increase with increasing infection levels.

6) **H_O**: The number of windthrown trees (trees uprooted or broken at the root collar) > 5.0 cm in diameter at the root collar is not related to infection level.

H_A: There is an increasing number of windthrown trees (trees uprooted or broken at the root collar) > 5.0 cm in diameter at the root collar with increasing infection levels.

Methods

Data collection

For the larger woody material, I did a 100% sample of the 0.25 ha plots. All down wood > 15 cm diameter at the large end was measured for large- and small-end diameters (nearest cm) and length (nearest 10 cm). Damaging agents and structural decay class as described by Parks et al. (1997) were also noted. Parks et al. (1997) describe three structural classes for logs based on the amount of bark attached, the amount of branches, and the decay condition of the wood. Structural class 1 logs are recently fallen trees that still have bark and branches intact and have very little decay present. The structural class 2 logs have lost some of the branches and the bark is loose with some decay of the wood. The structural class 3 logs are decayed and lack limbs and branches. In the smaller 4 m radial plots, woody material from 5-15 cm diameter was collected using the same techniques as above. On the 1 m^2 plots, all woody material 1-3 cm diameter was collected. For all material > 5.0cm at the large end the damaging agents annosus, Armillaria, fir engraver, and/or Douglas-fir bark beetles were noted.

In 2002 a typical fuels survey was done using methods from Brown (1974). Fuels were divided into four size classes (1-, 10-, 100-, and 1000-hour fuels) and collected along predetermined sections of the 15 m transect (Table 5.1). Four 15 m

transects were laid out, one each in the NW, NE, SW, and SE directions, with the endpoint being the plot center (Fig. 5.1). Fuel height, duff depth, and litter depth were taken at 0, 3, and 6 m on each transect. The information for each transect was summed to determine mton/ha (1 metric tonne = 1000 kg or 2200 lb) of fuel in each size class. The 1-, 10-, 100-, and 1000-hr size classes were used because they are standard moisture time lag classes used by managers and researchers (Agee 1993).

Table 5.1: Fuel measurement methods.

Fuel category	Fuel diameter	Length of transect	Method of measurement
1-hour	0-0.64 cm	2 m	Count
10-hour	0.65-2.54 cm	3 m	Count
100-hour	2.55-7.62 cm	5 m	Count
1000-hour	7.63+ cm	15 m	Diameter and decayed or non-decayed

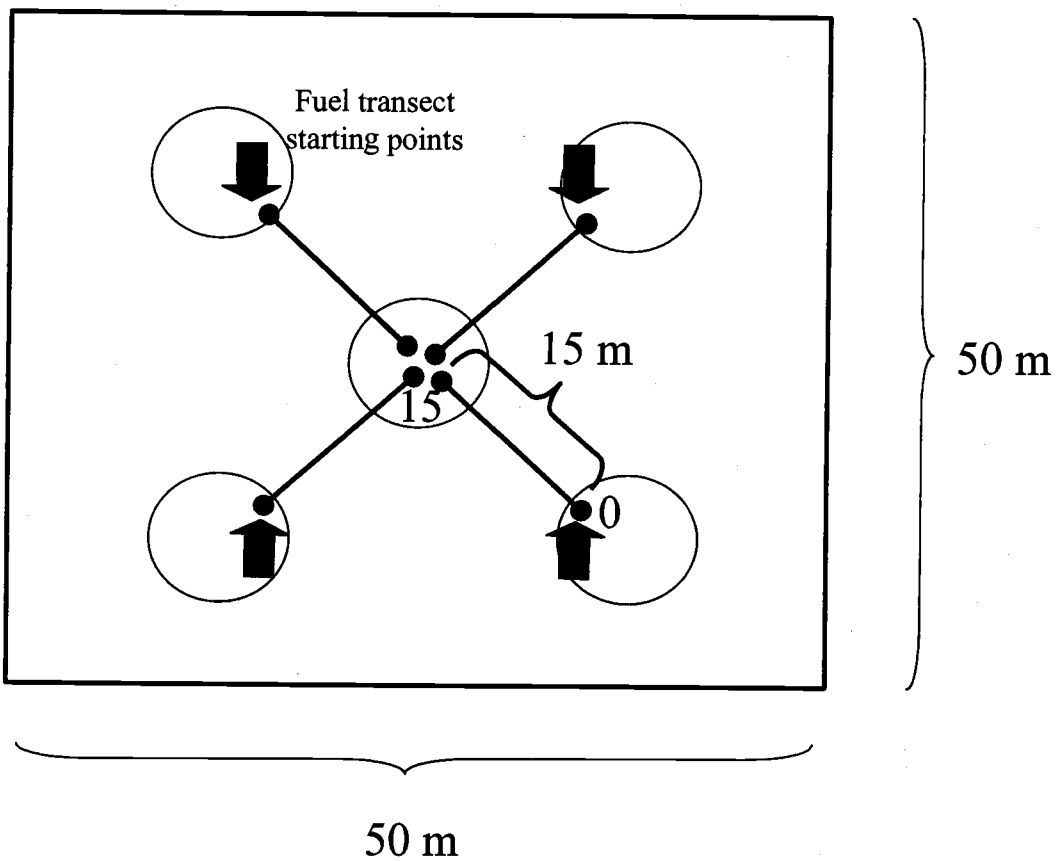

Fig. 5.1: Fuel transect layout within the 0.25 ha plot.

Statistical analysis

Fuel loadings

A full model was first developed that included variables that might influence the fuel loadings, including aspect, the number of live and dead standing trees/ha, and the infected BA. These variables were included in the full model after being checked for collinearity (see Table 2.1). This full model was used as a baseline for analyzing each diameter class. A backward selection criterion was used to determine the final model. Variables were excluded at $\alpha > 0.1$. The full model was as follows: $\text{mton/ha} = \beta_0 + \beta_1 BA_{INF} + \beta_2 ASP + \beta_3 TPH + \varepsilon$.

where:

BA_{INF} = BA of annosus- and Armillaria-infected trees (m^2/ha)
ASP = aspect of the plot (NW=1, N=2, NE=3, E=4)
TPH = the number of live and dead standing trees/ha
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1,β_2, and β_3 are parameters to be estimated from the data

Wind-thrown trees

The presence of either root disease pathogen was noted when found, and the number of windthrown trees is noted in Table 5.3. Unfortunately, windthrown trees that did not have any root pathogens were only counted as pieces of down woody material and were not noted as windthrown trees. Therefore, no conclusions can be made as to the percentage of windthrown trees/ha with root diseases and discussion is restricted to the frequency of windthrown trees.

The percentage of the total basal area that was windthrown with one or more root pathogens was used to test hypothesis 6. The percent of basal area windthrown was regressed against infected basal area. A quadratic term (infected BA^2) was used to fit the model. This was then compared with the relationship of percent live basal area. The model was as follows: $\rho = \beta_0 + \beta_1 BA_{INF} + \beta_2 BA_{INF}^2 + \varepsilon$

Where:

$Y = \arcsin\sqrt{\rho}$
ρ = percent of BA windthrown with one or more root pathogen
β_0 = line intercept
$\beta_1 BA_{INF}$ = Basal area infected with annosus or Armillaria
$\beta_2 BA_{INF}^2$ = quadratic term
$\varepsilon \approx N(0, \sigma^2)$
β_0, β_1, and β_2 are parameters to be estimated from the data

Results and discussion

Fuel loadings

The null hypothesis one was rejected. There was a significant positive relationship with infected BA. The final model for the mton/ha of fuel only included infected BA (p-value < 0.001, adjusted-R^2 of 0.677). With an increase in infected BA by 1 m^2/ha, there is an associated increase of 5.258 mton/ha (95% CI 3.749 to 6.765 mton) of fuel.

Sound and decayed 1000-hr fuels responded very differently from each other. The sound 1000-hr fuels significantly increased with an increasing amount of *H. annosum* or *Armillaria* spp. infected BA (p-value < 0.0001, adjusted-R^2 of 0.477). With a 1 m^2 increase in infected BA there is an associated 1.09 mton/ha (back transformed) increase in 1000-hr sound fuels (95% CI –1.02 to 3.2 mton/ha). However, the decayed fuels had no significant relationship with any of the predictor variables (p-value of 0.129, adjusted-R^2 of 0.061). The model results are presented in Table 5.2. Therefore null hypothesis 2 was only rejected for non-decayed 1000-hr fuels.

For the 100-hr fuels there was a significant linear relationship with infected BA (Table 5.2), and the null hypothesis (hypothesis 5) was rejected. With an increase of 1 m^2/ha of infected BA there is an increase of 0.27 mton/ha of the 100-hr fuels (95% CI 0.167 to 0.393 mton/ha). The one- and 10-hour fuels lacked a significant linear relationship to infected BA, and the null hypothesis was not rejected for these fuel classes.

Average fuel height above ground was significantly predicted by infected BA and the number of standing trees/ha (p-value 0.001, adjusted-R^2 of 0.42), and the null hypothesis was rejected. Holding the total trees/ha constant, with an increase of 1 m^2/ha infected BA the fuel height increases by 0.19 cm (95% CI of –0.024 to 0.356 cm). The duff and litter depth final models did not include the independent variable infected BA (Table 5.2), and the null hypothesis was not rejected.

Table 5.2: Final model results for fuel data. Significant models are shaded.

Fuel type	Excluded Plots	Transformation used	Adjusted R^2	F-value	Degrees of Freedom	P-value	Coefficients	Beta	Standard Error	p-value
Total Fuel (mton/ha)	None	None	0.677	26.152	1,22	**0.000**	Constant	49.398	14.762	0.582
							Infected BA	5.258	0.727	**0.000**
							Slope	-2.676	0.852	**0.005**
Fuel height (cm)	None	None	0.42	9.707	2,22	**0.001**	Constant	-1.92	2.22	0.396
							Infected BA	0.19	0.08	**0.030**
							TOTTPH	0.01	0.00	**0.004**
Duff depth (cm)	None	None	0.054	2.359	1,23	0.138	Constant	1.53	0.28	0.000
							Infected BA	-0.03	0.02	0.138
Litter depth (cm)	None	None	0.059	2.509	1,23	0.127	Constant	0.99	0.40	0.021
							Total TPH	0.00	0.00	0.127
1000hr decayed (mton/ha)	Number 12	None	0.061	2.488	1,22	0.129	Constant	12.52	8.03	0.133
							Aspect	3.98	2.52	0.129
1000hr sound (mton/ha)	None	Natural Log of Response	0.477	22.900	1,23	**0.000**	Constant	1.51	.288	0.000
							Infected BA	0.09	0.02	**0.000**
100hr (mton/ha)	None	None	0.537	28.869	1,23	**0.000**	Constant	3.96	0.79	0.000
							Infected BA	0.27	0.05	**0.000**
10hr (mton/ha)	None	None	-0.011	0.728	1,23	0.398	Constant	6.09	0.78	0.000
							Infected BA	0.04	0.05	0.398
1hr (mton/ha)	None	None	-0.038	0.122	1,23	0.741	Constant	1.37	0.21	0.000
							Infected BA	0.004	0.03	0.741

Wind-thrown trees

Table 5.4 shows that in plots with less than 5 m^2/ha of infected BA, there are few or no windthrown trees with either root pathogen. However, on plots with more than 5 m^2/ha of infected BA there was a high number of down trees/ha with one or both root pathogens.

Fig. 5.2 shows that the percentage of basal area that is windthrown increases as infected BA increases. However, at the higher infected BA levels(> 20 m^2), this relationship then decreases. The null hypothesis was rejected (adj.-R^2 = 0.301, F = 6.178, p-value = 0.007).

The reason for the decrease in the percent basal area of windthrown trees at higher infected BA was unknown. Therefore, the percentage of live basal area was examined. Fig. 5.3 shows that as infected BA increases, there is a significant dramatic decrease in the percentage of live BA (see Chapter II). The decrease in percentage basal area windthrown at higher infection levels is possible because of a significant decrease in live basal area at higher infection levels. Therefore, there is less available material to be windthrown.

Table 5.3: Model results for percent windthrown BA.

Coefficient	β	SE β	Sig.
Constant	0.004275	0.019058	0.8246
Infected BA	0.00919	0.002648	0.0022
Infected BA^2	-0.004275	0.0000742	0.0022

Table 5.4: The number of windthrown trees/ha with *Armillaria* spp. and/or *H. annosum* listed by tree species.

Plot	Infected BA		*Armillaria* spp.			*H. annosum*	*Armillaria* and *H. annosum*	Total windthrown TPH w/ root disease
	Armillaria spp.	*H. annosum*	White fir*	Ponderosa pine	Douglas-fir	White fir*	White fir	
TS15P10	0.00	0.00	0/0	0	4	0	0	4
TS8P14	0.25	0.00	0/0	0	0	4	16	20
TS8P11	0.26	0.00	0/0	0	0	0	0	0
TS15P2	4.06	0.07	0/0	0	0	0	0	0
TS1P6	4.56	0.27	4/0	0	0	0	0	4
TS8P7	5.02	2.11	20/40	0	0	32	0	92
TS9P3	5.86	5.56	12/40	12	4	12/80	0	160
TS2P2	6.36	0.00	8/0	0	0	8	0	16
TS9P6	6.88	0.98	40/120	4	12	8	4	188
TS6P4	8.41	0.00	8/120	0	0	0	0	128
TS5P1	8.67	0.00	20/0	0	0	0	0	20
TS7P12	9.88	0.34	28/120	0	0	0	0	148
TS9P16	10.41	1.79	56/400	12	0	44/40	4	556
TS8P2	11.28	0.00	84/0	4	8	0	0	96
TS2P5	11.29	0.46	8/40	0	0	8	0	56
TS1P2	12.97	0.49	24/0	0	0	0	0	24
TS5P7	13.45	0.36	52/400	0	0	0	0	452
TS6P14	13.67	0.00	20/0	0	4	0	0	24
TS3P14	15.06	2.14	14/0	0	0	4	0	18
TS3P12	15.17	1.41	28/40	0	0	0	0	68
TS7P2	17.42	0.00	28/40	0	12	0	0	80
TS5P13	23.66	1.25	32/120	0	0	12	0	164
TS1P11	29.10	0.00	16/0	0	0	0	0	16
TS3P2	29.15	1.14	28/120	4	0	0	0	152
TS6P9	36.51	0.00	20/60	0	0	0	0	80

*Numbers on the left are windthrown trees/ha with diameters ≥ 15 cm at the root collar. The numbers on the right are windthrown trees 5-15 cm in diameter at the root collar and were measured on the 4 m radial plots.

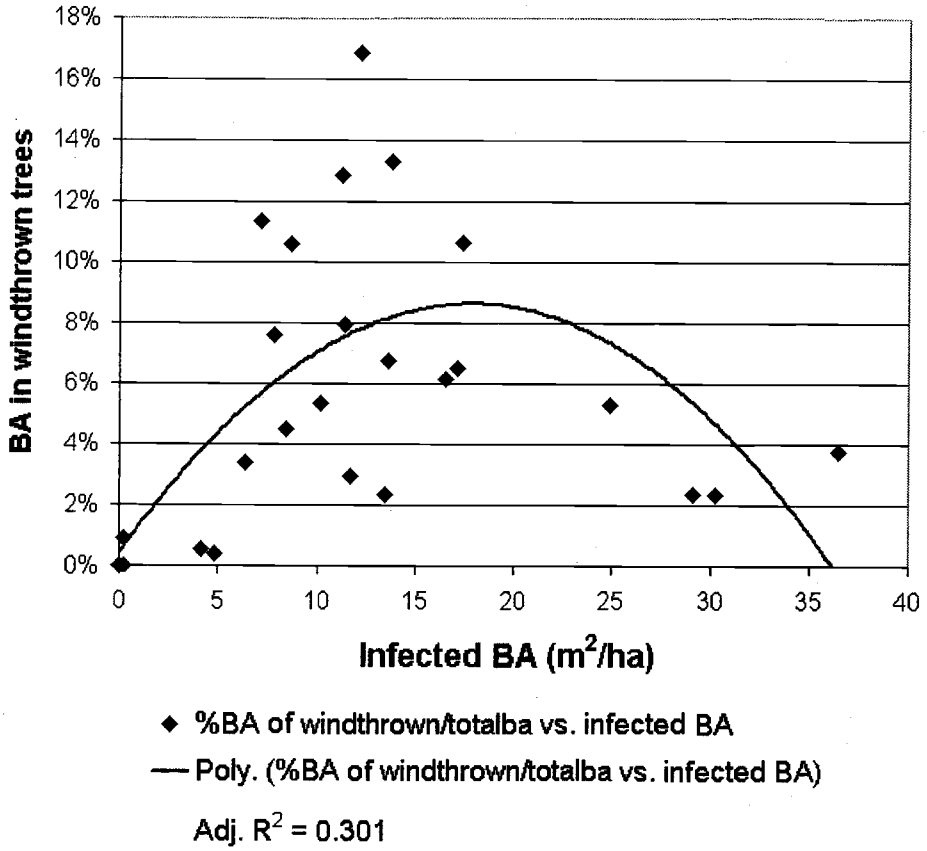

Fig. 5.2: Percent basal area of wind-thrown trees/ha > 5 cm at the root collar with one or more root pathogens found on the roots.

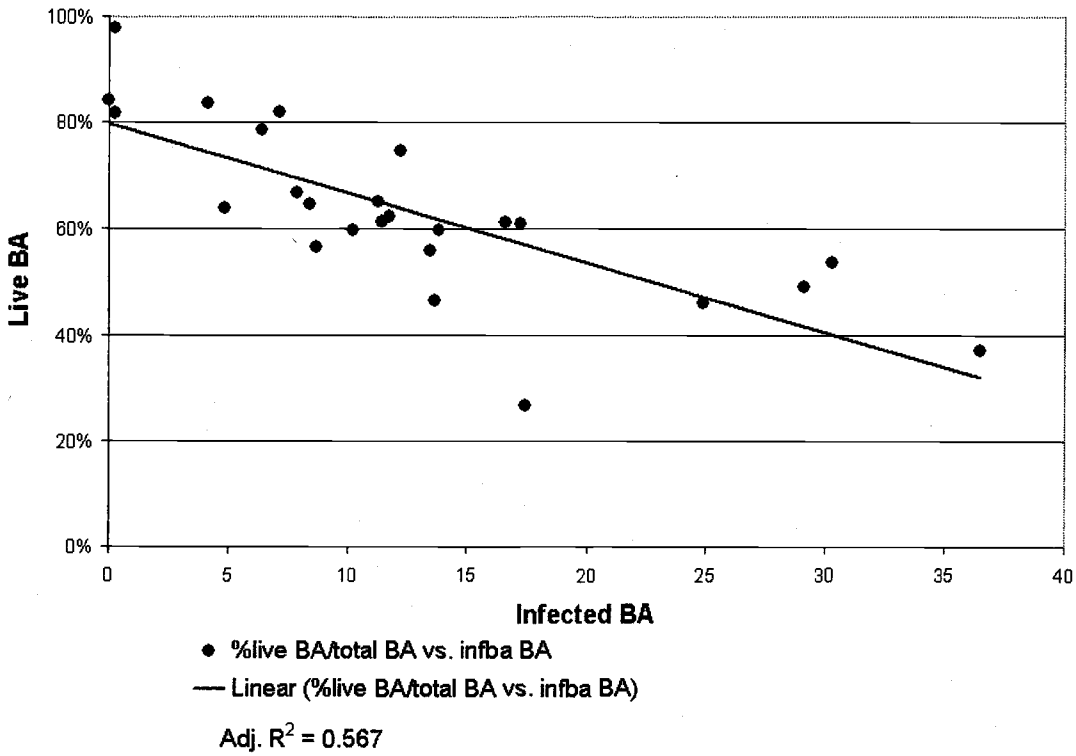

Fig. 5.3: Percent live BA as it relates to infected BA.

Discussion

Results from this analysis support the hypothesis that in areas with higher levels of root disease there is an associated increase in fuel loadings. However, this relationship is not statistically significant for all fuel sizes. The larger material (100- and 1000-hr fuels) shows a very strong relationship with the amount of root disease in a stand. As might be expected, the fine fuels (1- and 10-hr, composed of fine branches) are not directly influenced by the amount of root disease. The 1000-hr decayed fuels showed no relationship with infected BA. This may be because in some areas there were several (70+ cm) large decayed Douglas-fir logs that were left from salvage harvesting operations in the mid-1900s.

The results of objective two and the crown base height also impact the possibility of a ground fire ascending into the tree crowns and becoming a crown fire. The tall-intermediate ponderosa pine had lower crown bases with an increase in infected BA. These lower crowns could potentially lead to a fire getting into the crowns. However, there are several other site factors that would influence the conditions for a crown fire, such as stand density, stratification, slope, aspect, and crown bulk density (Keyes and O'Hara 2002).

The percentage of basal area that was windthrown with one or more root pathogens found on the roots had a non-linear relationship with infected basal area. It is possible that this non-linear relationship is due to a significant decrease in the amount of material available to be windthrown. This is the case in plot 12 where the majority of the stand was small (<30 cm dbh) white fir which is not likely to be windthrown, and 32 ponderosa pine/ha in the tall-emergent diameter class which are more resistant to root diseases (especially in this study) and less likely to be wind thrown due to root disease.

The past few decades have seen increasing concern over forest susceptibility to catastrophic wildfires in the western United States. This study shows that in areas with higher levels of root disease there is a direct increase in fuel loadings. In turn this may cause a higher probability for intense high severity wildfire.

ARMILLARIA SPECIES IN CENTRAL OREGON

Literature review

Historically the different species in the genus *Armillaria* were classified together as *A. mellea* (Vahl: Fr.) Kummer (=*Armillariella mellea* (Vahl: Fr.) Karst.) (Wargo and Shaw 1985). Anderson and Ullrich (1979) demonstrated that *A. mellea* in North America was actually 10 distinct biological species. These were then defined as the 10 North American Biological Species (NABS) (Table 6.1), and for many years, nomenclature was not yet developed for the majority of them. Today, nine *Armillaria* species are found in North America, and one North American biological species, NABS X, which is taxonomically undescribed and reproductively isolated (Banik et al. 1996, Baumgartner and Rizzo 2001). Another biological species (NABS XI) has also been found in Washington (Banik et al. 1996). There is some evidence that NABS XI is partially compatible with the European species *A. cepistipes* (Shaw and Kile 1991, Banik et al. 1996).

To date five species, *A. ostoyae*, *A. gallica* (formerly *A. bulbosa*), *A. mellea*, *A. sinapina*, and NABS X have been found in Oregon (Reaves and Mc Williams 1991, Baumgartner and Rizzo 2001, Filip and Ganio 2004). *A. ostoyae* has been documented in British Columbia and other parts of the western US, where it is often assumed to be the pathogen when there are serious root disease problems in conifers (Gregory et al. 1991). Ferguson et al. (2003) isolated *A. ostoyae* and NABS X from conifers in northeastern Oregon. NABS X has also been reported in the Rocky

Mountains and British Columbia (Morrison et al. 1985, Baumgartner and Rizzo 2001).

The range of *A. gallica* begins in Baja California, Mexico and is fairly continuous up through British Columbia (Morrison et al. 1985, Baumgartner and Rizzo 2001). It has been documented on white fir in California (Reaves and McWilliams 1991) and on hardwoods in central Washington (Shaw 1983). *A. gallica* is known as a weak pathogen of conifers and hardwoods (Morrison et al. 1985). McDonald et al. (1988) noted that *A. gallica* is a "prolific producer of rhizomorphs".

Several other species of *Armillaria* have been found in the western United States and Canada. In a study to determine the species of *Armillaria* in California, *A. mellea, A. gallica, A. nabsnona*, and NABS X were found in the northern half of the state (Baumgartner and Rizzo 2001). The collections from California were in the southern Cascades. Other *Armillaria* species have been found in western North America. Banik et al. (1996) identified *A. sinapina* in their study on the Olympic Peninsula, and it was the second most abundant *Armillaria* species collected. A. *nabsnona* has a wide range from Alaska into British Columbia and the Pacific Northwest and south to northern California (Morrison et al. 1985, Baumgartner and Rizzo 2001). It has even been found in Idaho (Volk et al. 1996). *A. mellea* is found on both the east and west coasts of North America. However, on the west coast the range is discontinuous, and it is usually found in coastal habitat in Oregon, California, and Washington (Baumgartner and Rizzo 2001). Other species of

Armillaria may occur in Oregon; however, a more thorough study of the species distribution needs to be conducted (Filip and Ganio 2004).

Table 6.1: *Armillaria* species of North America. All of the other species occur on other continents (Baumgartner and Rizzo 2001).

Species	Location	Source
A. ostoyae (NABS I)	Washington Oregon British Columbia and Ontario New Hampshire	Ferguson et al. 2003 White et al. 1998 Morrison et al. 1985 Kim et al. 2000
A. mellea (Vahl:FR) P. Kumm.(NABS IV)	California (throughout) Northeastern United States	Baumgartner and Rizzo 2001 Kim et al.2000
A. tabescens (Scop.) Emel		Southeastern United States, west to Texas and Oklahoma,
A. calvescens Berube & Dessur.*	Eastern North America New Hampshire Michigan and Quebec Alberta	 Rizzo and Harrington 1993 Kim et al. 2000 Mallett and Maynard 1998
A.gemina Berube & Dessur.*	Eastern North America New Hampshire New York and West Virginia	 Rizzo and Harrington 1993 Kim et al. 2000
A. sinapina Berube & Dessureault (NABS V)	Washington-Olympic peninsula British Columbia, Alberta SW Oregon Michigan	Banik et al. 1996 Morrison et al. 1985, White et al. 1998, Mallet and Maynard 1998 Baumgartner and Rizzo 2001 Kim et al. 2000
A. gallica Marxmuller & Romagnesi (NABS VII)	California Washington British Columbia Hawaii Michigan and Wisconsin	Reaves and McWilliams 1991, Baumgartner and Rizzo 2001 Shaw 1983 Morrison et al. 1985 White et al. 1998 Baumgartner and Rizzo 2001 Kim et al. 2000
A. nabsnona (NABS IX)	British Columbia Northwest Coast of California Hawaii Washington Idaho and Alaska	Morrison et al. 1985, Volk et al. 1996, White et al. 1998 Volk et al. 1996, Baumgartner and Rizzo 2001 Volk et al. 1996 Kim et al. 2000
NABS X	Oregon- Blue Mountains Northern California British Columbia Idaho	Ferguson et al. 2003 Baumgartner and Rizzo 2001 Morrison et al. 1985 Kim et al. 2000
NABS XI (partially compatible with European *A. cepistipes* Velenovsky)	Washington Olympic Peninsula British Columbia	Banik et al. 1996 Morison et al. 1995, Kim et al. 2000

*Indicates it is believed to be unique to North America. All of the other species occur on other continents in the Northern Hemisphere (Baumgartner and Rizzo 2001).

Objective and hypothesis

The objective of this study was to determine the species of *Armillaria* occurring across the study site. I hypothesized that there were other *Armillaria* species besides *A. ostoyae* in the study stands.

Methods

Samples of *Armillaria* spp. were collected immediately after snowmelt in April, May, and again in October, 2002 from each of the study plots. Samples were collected from both recently killed trees and trees that had been dead for more than five years. All trees were located in or within 10 m of a 0.25 ha plot. The samples were refrigerated at 5° C until wood chips were removed for isolation. About 1-2 mm long wood chips were extracted from the center of each sample and placed on an Armillaria-selective media (30 g malt extract, 20 g dextrose, 5 g peptone, 19 g agar, 1-liter dH_2O [Ferguson et al. 2003] and 4 mg Benlate, and 200 mg Streptomycin [E. Hansen, Forest Pathologist, Oregon State University, 2002 pers. comm.]). Cultures were then stored in the dark at room temperature for at least two weeks, after which cultures that resembled *Armillaria* spp. were removed (1-2 mm in size) and placed on the same selective media minus the Benlate and Streptomycin.

DNA (Deoxyribonucleic acid) was extracted and a PCR (polymerase chain reaction) assay was performed to obtain RFLP's (restriction fragment length polymorphisms) following protocols used in Harrington and Wingfield (1995) and White et al. (1998). Harrington and Wingfield (1995) developed a one-day

procedure to identify 11 different species of *Armillaria* isolates. White et al. (1998) used an extended, more traditional procedure to identify cultures using PCR analysis.

A small piece of mycelium was collected from the edge of 8 *Armillaria* isolates and then subjected to the following procedures to obtain RFLP's. DNA was extracted by first bead beating the samples with 3.5 mm glass beads at 4200 rpm for 30 seconds to break up the mycelium. Then a DNA extraction buffer of 1.5 M NACL, 100 mM Tris-HCl pH 8, 20 mM EDTA pH 8, 0.1% PVPP, and 2% CTAB was added to the sample. Samples were then incubated at 65° C for two hours. After spinning for 3 minutes, one ml of the supernatant was removed and placed in a new tube. One ml of chloroform (24 chloroform: 1 isoamyl alcohol) was then added to the tube, and the sample was shaken for 5 min. and spun for 5 min. The top 0.5 ml was removed, and 0.5 ml of isopropanol was added, and the sample was incubated at room temperature for one hour. It was then spun for 10 minutes, and the liquid was poured off, which left a white pellet. Then 0.5 ml of ice-cold 70% etoh was added and the sample was left for 5 minutes. This was spun for 5 minutes and the liquid was poured off. The tube was left to air dry with the white pellet still inside. Then 100 µl of 0.5 M TE was added, and the tube was left overnight in the refrigerator.

The intergenic spacer (IGS region) of nuclear ribosomal DNA was then amplified with a polymerase chain reaction assay, using 0-1 and LR12R primers. The program was as follows: 1) 95 C for 95 sec; 2) 60 C for 40 sec; 3) 72 C for 2

minutes; 4) 90 C for 30 sec; 5) 60 C for 40 sec repeated 34 times; and 6) 72 C for 10 minutes.

The restriction enzyme, Alu 1, was added and then allowed to proceed for 1-16 hours at 37° C (Harrington and Wingfield 1995). Following the digestion, the DNA fragments were electrophoresed in agarose gels in a TBE buffer system at 200 V for 30-40 minutes. The gels were stained with ethidium bromide and visualized under UV light. The fragment bands from the eight isolates formed two distinct banding patterns. Representative isolates (L1 and L3 from fig. 6.1) were chosen from the two groups and submitted to the Central Services Lab at OSU for sequencing. These sequences were then blasted against sequences in the GenBank database for identification.

Results

The RFLP results indicated that there are at least two species (*A. ostoyae* and *A. gallica*) found in the study, and the null hypothesis was rejected. This was determined from amplification of the IGS region. Seven of 8 the isolates were identified as the same species (*A. ostoyae*), and the other isolate was identified as *A. gallica*. The one identified as *A. gallica* was located near another plot that had *A. ostoyae*, and both plots were located within the same timber sale unit.

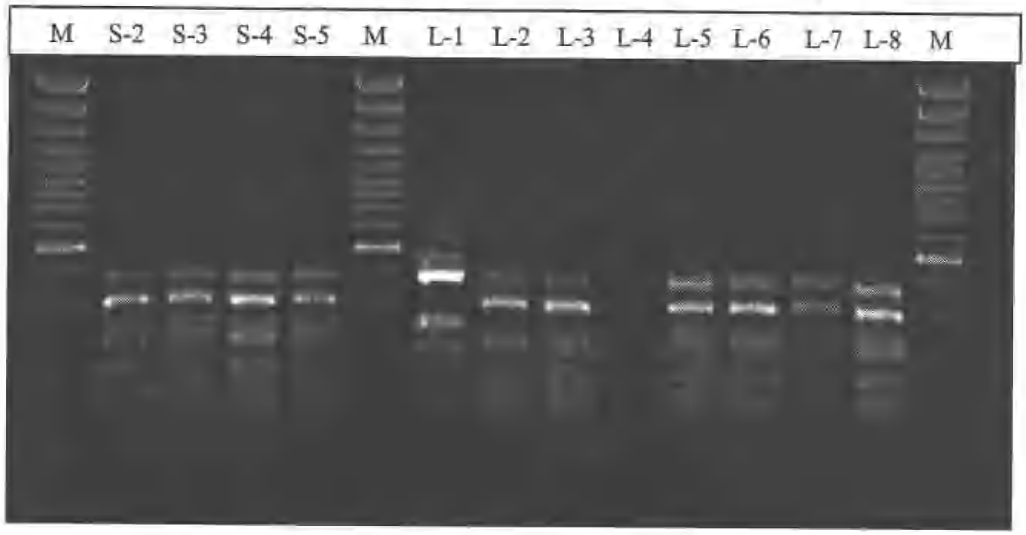

Fig. 6.1: RFLP results of ethidium bromide stained gels of Alu I digestion products. The markers (M) are 100-bp ladders. The RFLP on the left is from the shortened (S) version using methods based on Harrington and Wingfield (1995). The results on the right are from the longer extraction process (L) and yielded results for all eight isolates. L-4 is missing due to a shortage of product.

Figure 6.1 displays only L-1 (collected from TS9P3 in fig. 1.2) as having a different RFLP pattern. This isolate was sequenced by Central Services Lab at OSU. The sequence was identified as *A. gallica* when blasted against sequences in GenBank. The isolate had a difference of six base pairs from the *A. gallica* record in GenBank. This sample was taken from a white fir classified as dead for five to ten years based on the bark at the root collar. The sample was originally collected in May 2002 and was re-plated in November. It produced long slim rhizomorphs that did not branch out. The top of the isolate was dark brown and crusted with white mycelia on the corners. The white mycelium on the plate showed a wavelike pattern.

Isolate L-2 (S-2) was collected from a live infected Douglas-fir tree on plot 24 (TS15P2 in fig. 1.2) in May of 2002 and was positively identified as *A. ostoyae* by the Central Services Lab at OSU. None of the isolates collected from this plot produced rhizomorphs. I used this isolate as a template for what I expected *A. ostoyae* to resemble because it was collected from a live-infected tree with basal resinosis, indicating that it was a pathogenic species of *Armillaria* (McDonald et al. 1987a). It had white "fluffy" mycelium on the top of the sample with light brown edges. The sampled tree had a dead top as a result of the spruce budworm defoliation.

Isolate L-3 (S-3) was collected from plot 2 (TS1P2 in fig. 1.2) from a recently killed white fir that showed symptoms of resinosis at the base and fir engraver galleries on the main bole at DBH. The sample looked much like L-2 and did not produce rhizomorphs.

Isolate S-4 was collected near isolate L-1 (collected from TS9P16 in fig. 1.2) from a recently killed white fir. *H. annosum* was also isolated from the same wood sample at the root collar. The *A. ostoyae* isolate produced rhizomorphs that were thin and branched near the center of the isolate. The top of the isolate was fuzzy and covered with white mycelia.

Isolate L-5 (S-5) was also collected from plot 23 (TS9P16 in fig. 1.2) from a recently killed white fir in the same root disease pocket as L-4. The isolate did not produce rhizomorphs; however, it was covered in white fuzzy mycelia.

Isolate L-6 was collected near plot 10 (TS5P7 in fig. 1.2) from a live infected Douglas-fir. It also did not produce rhizomorphs. The isolate looked similar to L-2.

Isolate L-7 was collected from plot 8 (TS3P14 in fig. 1.2) from a white fir that had been dead five to ten years. The isolate produced rhizomorphs that were almost double in thickness compared to other samples and were extensively branching in the petri dish. The top of the sample was hard and crusty; however, it was also entirely covered with white and brown mycelia.

Isolate L-8 was collected from a white fir that had been dead 5-10 years and was near plot 8 (TS3P14 in fig. 1.2). The isolate looked similar to L-7 with thick rhizomorphs that were extensively branching. The top was also crusted but with white mycelia covering the top and a pinkish tint below.

Discussion

In western North America other Armillaria species besides *A. ostoyae* and *A. mellea* are considered weak pathogens and saprophytes. However, little is known about the distribution and impact of these species in the forests of Oregon.

The one isolation of *A. gallica* brings to question the extent of the species across the study site. Due to the frequency and abundance of rhizomorphs (McDonald et al. 1988) found on several of the plots (namely the three plots TS3P2, 12, 14 in fig. 1.2), I expect that the one isolation of *A. gallica* is just one incidence of the pathogen (McDonald et al. 1988). It was difficult to isolate *Armillaria* spp. from trees that had been dead for more than 5 years. In unit 3 the specific trees of

interest were those with a large quantity of rhizomorphs just under the bark at dbh. Most of these trees also had fruiting bodies of *Fomitopsis pinicola* (Sw.:Fr) P. Karst at the root collar, making it difficult to successfully isolate *Armillaria* spp. Therefore, the one isolation of *A. gallica* most likely underestimates its occurrence across the study site.

Because of the difficulty in identifying trees affected by *H. annosum*, annosus root disease was not easily identified as a mortality agent in this study. However, it is important to note that, on 8 of the plots where *Armillaria* spp. were isolated, *H. annosum* was also isolated (plotsTS1P11, TS2P2 and P5, TS3P14, TS8P2 and P7, TS9P6 and P16 in fig. 1.2). The objective was to isolate *Armillaria* spp. Therefore, the *H. annosum* isolations were made from wood directly under *Armillaria* mycelial fans. The common occurrence of *H. annosum* and *Armillaria* spp. from the same trees brings into question the rather low annosus infected basal areas that were measured. It also brings in to question which root disease is the causal agent of mortality and which is a secondary pathogen overcoming the tree's defense system after it is weakened by the primary root pathogen. Both of these diseases could be contributing equally, acting in conjunction with each other and contributing to the demise of the tree.

CONCLUSIONS, MANAGEMENT RECOMMENDATIONS, AND FUTURE RESEARCH

Root diseases impact the mixed-conifer forests in several direct and indirect ways. In this study, root diseases played a major role in shaping the forests of central Oregon. Contributing factors include a century of fire suppression, western spruce budworm epidemics, and decades of salvage harvesting of the large ponderosa pine, larch, and Douglas-fir. The combination of these disturbances has created a predominantly white fir forest community in the high-elevation areas on the Sisters Ranger District. These white fir forests are highly unstable, since white fir is susceptible to several different insect pests and pathogens, as is evident in this study. Root disease has caused significant changes in the stand and community structure, and has increased the fuel loadings, causing an elevated risk of a stand replacing wildfire.

The two species of *Armillaria* found in the study indicate that, in some cases, a weaker or saprophytic species of *Armillaria* might be found and associated with tree mortality. However, *A. ostoyae*, the highly pathogenic species, was isolated from all of the sites examined in this study. *A. ostoyae* has been found to kill trees for several years after a western spruce budworm or Douglas-fir tussock moth outbreak and trees that are affected by drought stress (Hadfield et al. 1986). On the east side of Cascade Range in central Oregon, the mixed-conifer forests have experienced both conditions in the 10 years previous to this study. In my study *A.*

ostoyae was associated with mortality and was part of the interaction with drought and the spruce budworm outbreak. This corresponds with the results of Wright et al. (1984) where they found severely defoliated trees generally had signs of root disease. The observations of this study indicate that mortality from root diseases can continue for up to a decade after a defoliation outbreak. For the smaller trees within the study site it is difficult to determine 10 years later if Park et al. (1994) observations held true in this study. Park et al. (1994) found that grand fir seedlings that were defoliated had less infection from *A. ostoyae.*

Elevation was strongly correlated with many of the other explanatory variables used in this study. On the east side of the Cascade Range, elevation can be used as a surrogate for the available moisture. This study spanned a 400-meter elevation gradient, which can equate to a difference of at least 100 cm of precipitation.

In general, across the study site there was more mortality at higher elevations. This also contributed to the canopy cover differences. The stand composition also switched from highly diverse, with Douglas-fir, ponderosa pine, western larch, and white fir on the lower elevation plots, to primarily white fir at the higher elevation plots with a few relic ponderosa pine or Douglas-fir in the overstory.

The amount of *Armillaria* and *P. weirri* inoculum in the soil has previously been inversely correlated with elevation gradients (Williams and Marsden 1982). Hobbs and Partridge (1979) found no correlation in the distribution of Armillaria

with elevation or stand composition. Instead, they suggest it is not related to heat or moisture regimes within their study site in Idaho. In Alberta, Mallett and Maynard (1998) found that mortality from *Armillaria* was high on sites with coarse-textured (sandy) soils and hypothesized that this may be due to limited available moisture. In this study, the opposite was found from Williams and Marsden (1982) in that there was more mortality at higher elevations where there was a greater amount of precipitation and more white fir, a highly susceptible host to Armillaria and annosus. The soils in these study sites were primarily sandy loams; thus, corresponding with the results of Mallett and Maynard (1998).

At the time of sampling the stand structure changed significantly with an increase in the amount of infected BA. Stands with higher amounts of root disease experienced higher percentages of mortality in the white fir. Douglas-fir mortality was weakly correlated with the amount of root disease. In the lower elevation plots there were Douglas-fir that had resinosis at the base with mycelial fans under the bark at the root collar. Ponderosa pine was not impacted by the amount of root disease in these plots. However, on other sites on the Deschutes National Forest it has been found to aggressively kill ponderosa pine seedlings and saplings in plantations (Adams 1974, Filip et al. 1999).

The canopy cover and canopy stratification changed in ways that were not necessarily predictable. There were more tall-emergent (>60 cm dbh) trees/ha retained in areas with higher amounts of root disease. The tall-emergent trees were

predominantly ponderosa pine and Douglas-fir. This was most likely due to the mortality of the dominant and emergent white fir trees that reduced competition.

The understory forb and shrub cover and species richness relationships with the amount of root disease were difficult to discern. This is not atypical of root disease centers and, in this study, could be confounded by the elevation and moisture gradients. The direct effects of the mortality of white fir, and in turn the indirect impacts on the understory plant diversity, varied greatly by individual forb and shrub species, as some species took advantage of the newly available resources. However, further changes in diversity of forb and shrub communities are unpredictable (Hansen 1999).

The total fuel load (coarse woody material) had a strong significant correlation with the amount of root disease. This relationship was dependent on the fuel size.

Ecological implications

The results of this study have ecological implications concerning forest disturbance and successional pathways. Root diseases in the central Oregon Cascades are a significant factor in changing the forest stand species composition and structure. In this study root diseases were present in every sampled stand and, if not found in a plot, they were directly adjacent to the plot. In most cases root diseases were diffuse throughout the stands and in every area, even with low infected basal areas, they were aggressively killing trees. Given the life cycle of

Armillaria, the root disease disturbance agent in these stands has been an ongoing factor that has constantly shaped the forest community for centuries.

Armillaria root disease has long been described as a pathogen of the site. Large genets can persist over large areas for thousands of years (Ferguson et al 2003). Other research has shown that with the slow rate of spread of root disease fungi, it is unlikely that the past 100 years of fire suppression and the subsequent increase in host density has increased the area colonized by *A. ostoyae* genets (Ferguson et al. 2003). Also, the expression of the root disease in the years following the field data collection (2002 and 2003) show that no plot was absent of one or both root pathogens; therefore, its impacts are everywhere, and the timing of disease expression may be a more important variable that should be studied.

The theory that the pattern and expression of Armillaria root disease has changed with changing management practices is supported by this study. Perhaps Armillaria root disease has a new role in the forest setting. Results from this study indicate that Armillaria root disease expresses itself as a primary associate in the complex of defoliating insects, bark beetles, annosus root disease, and drought, and that this expression has increased. In some respects this increase in root disease expression, combined with other agents, has started to play a role similar to that of fire by decreasing the density of the late-seral *Abies* species and leaving the early seral species on site. However, this disturbance leaves these stands on a different successional trajectory with white fir in the understory, high fuel loadings, and a site with unfavorable conditions to promote establishment of the fire-resistant early-

seral ponderosa pine, western larch, and even mid-seral Douglas-fir. This pattern further develops the theory of Abrams and Scott (1989), where they develop the idea of disturbance-mediated succession. Their first three steps follow that of Oliver and Larson (1996) with the stand initiation stage, stem exclusion stage, and understory reinitiation stage. After that they introduce the concept of a new stage of disturbance where all species are set back, and from this stage an accelerated succession stage develops with the late-seral species occupying the site in both the overstory and understory. On these sites this accelerated succession has developed into a predominantly white fir stand with a few relic Douglas-fir and ponderosa pine trees per hectare. Fire suppression has created the accelerated succession stage, and the complex of root diseases, drought, and defoliating insects are constantly shaping the accelerated succession stage, keeping it on site until a stand replacing wildfire resets succession.

In areas where white fir has become the dominant and co-dominant species, short-term management objectives may be met for LSR's; however, the persistence of these desired stand structures is unstable, as these sites will continue to experience drought, defoliation, root disease, and high risk of a stand replacing wildfire event. The mixed-conifer stands of central Oregon have experienced an insect-pathogen complex that continues to change stand structure and species composition.

Management implications

The results of this study have several strong management implications. 1) Root diseases may help to retain the few large diameter ponderosa pine and Douglas-fir left in the mixed-conifer forests. However, in these stands there are very few Douglas-fir and ponderosa pine on site that, over time, will replace the tall-emergent trees as they succumb to western pine beetle and Douglas-fir bark beetle. These tall-emergent Douglas-fir and ponderosa pine trees are a valuable component of the forest ecosystem. Therefore, management activities should focus on retaining these trees and reducing the potential for catastrophic wildfire. 2) Mortality of the white fir may also promote the health of small-diameter ponderosa pine; however, white fir is the primary species in the understory in all of these stands, regardless of root disease levels. Therefore, regeneration methods should focus on promoting species other than white fir. 3) Land managers should manage for species other than white fir but understand that it does need to be retained in the understory in order to meet habitat suitability requirements mandated by the Northwest Forest Plan. In many of these stands with high infected BAs, the tall-intermediate (5-14.9 cm dbh) diameter class is primarily white fir; therefore, managing for other species may require an even-aged management regime that may include planting ponderosa pine, western larch, and Douglas-fir. 4) In areas with lower amounts of root disease (in this study these were the sites with less white fir present), managing to maintain the older Douglas-fir and ponderosa pine is recommended. Silvicultural methods should also be used to remove sufficient overstory so that ponderosa pine, western larch, and Douglas-fir regeneration is successful. On many sites this may require

forfeiting short-term objectives for Late Successional Reserves to provide the long-term goals of the stand structure desired by late successional species.

With such high mortality rates in the white fir and a strong correlation between the amount of root disease and the amount of white fir mortality, there are strong implications for managing true fir in areas with root disease. There are key management practices that should continue to be carried out if true fir species are to be retained and managed on these sites. Treating stumps of host species with boron-containing chemicals should still be done in areas where annosus root disease is not already established (Filip and Schmitt 1990, Hagle and Schmitz 1993, Sullivan et al. 2001). Wound prevention guidelines should also continue to be utilized to prevent the establishment of annosus stem decay (Sullivan et al. 2001).

Future management policies concerning mixed-conifer stands in central Oregon should take into consideration the role of root diseases in creating the current and future stand structure, the amount of mortality caused by root pathogens, and these pathogens' influence on target variables such as canopy cover. This important influence is evident in this and other studies (Filip 1990).

The fires of 2003

The Booth fire started on August 19, 2003 and within a couple of days had burned through all plots within this study and the associated White fir Administrative Study (Petaisto et al. 1999). Most plots with in this study were burned at a high intensity, especially those at higher elevations and with more root

disease. Several of these plots experienced a stand replacing fire. In other lower elevation areas, moderate to high fire intensities burned through the plots.

The fire occurrence gives us a unique opportunity to follow tree survival after wildfire in areas with high levels of root disease. These plots in conjunction with the White Fir Administrative Study provide a baseline for future studies of mortality from root disease after wildfire.

Further research

Future research should focus on mortality rates of white fir and Douglas-fir from root disease in mixed-conifer stands before, during, and after defoliation. This would help to discern the role of each insect and pathogen in the complex and hopefully develop some management strategies for each agent.

The retention of the tall-emergent trees in areas with high levels of root disease should also be studied. The mechanism for retaining these large trees should be determined, and this relationship should be explored in other areas.

Mortality rates of trees 1–15cm dbh should be studied in these stands. This research brings in to question the relationship of root diseases to the amount of mortality of the tall-intermediate trees and their role in the lower canopy layer. In this area it would be beneficial for managers to know if seedlings, saplings, and pole-sized trees are less likely to be killed by root disease because of a low chance of root-to-root contact, or if this is an anomaly of this study and instead in central Oregon the same relationship exists as Morrison and Mallett (1996) and Robinson

and Morrison (2001) suggest, where all tree species are susceptible until 15 years of age in British Columbia.

The insect and disease complex in central Oregon mixed-conifer stands has dramatically impacted stands managed for late successional species. Maintenance and retention of theses desired stand structures needs further research. The risk of stand replacing wildfire is high in these stands, indicating that a complex of disturbances may hinder achievement of long-term management objectives in Late Successional Reserves.

In light of the 2003 fire season in central Oregon and the Booth fire, this study and the White Fir Administrative Study have set the stage to study the fate of fire-damaged trees and stand replacement in the face of Armillaria and annosus root diseases. Filip and Yang-Erve (1997) determined that Armillaria isolates buried > 8 cm in the soil can be recovered after prescribed burning. This fire provides the chance to follow both root pathogens on the future mortality of the stand after a wildfire.

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APPENDICES

Appendix 1: Stand Conditions

Table A1.1: Abiotic plot variables and UTM coordinates.

Timber sale unit	Plot	Elevation (m)	Slope (%)	Aspect (degrees)	Aspect	Northing	Easting
1	11	1224	25	30	NE	4923897.555	599064.101
1	2	1206	25	30	NE	4923938.941	599222.880
1	6	1177	25	20	N	4923982.011	599373.780
2	2	1197	15	100	E	4924331.542	598984.101
2	5	1209	15	100	E	4924320.929	598880.657
3	2	1207	35	140	SE	4921901.185	599744.181
3	12	1211	35	350	N	4921807.528	599722.845
3	14	1222	35	60	NE	4921800.025	599616.448
5	1	1349	15	50	NE	4920682.784	598421.800
5	7	1343	15	100	E	4920653.963	598533.050
5	13	1339	15	130	SE	4920584.852	598538.888
6	9	1338	12	140	SE	4920242.371	598284.317
6	14	1329	12	120	E	4920221.118	598434.673
7	2	1258	17	40	NE	2922348.922	597606.771
7	12	1268	17	40	NE	4922313.801	597803.424
8	2	969	12	0	N	4924082.742	602553.232
8	7	987	12	340	NW	2923980.669	602401.478
8	11	979	12	40	NE	4923995.317	602183.786
8	14	973	12	40	NE	4924122.862	602289.283
9	3	962	15	10	N	4924158.137	602720.742
6	4	1353	12	120	E	4920437.127	598298.519
9	6	979	15	340	NW	4924048.425	602670.010
9	16	964	15	30	NE	4924082.625	602981.875
15	2	1036	8	60	E	4918709.117	602740.926
15	10	1029	8	60	E	4918769.068	602916.627

Table A1.2: Basal area and TPH by plot: live, dead, and stump.

Timber sale unit	Plot	Standing BA (m^2/ha)	Standing TPH*	Dead BA (m^2/ha) (standing)	Dead TPH (standing)	Live BA (m^2/ha)	Live TPH	Stump BA (m^2/ha)	Stumps /ha
1	11	70.41	716	35.79	484	34.62	232	8.31	32
1	2	42.88	588	18.92	312	23.96	276	24.29	56
1	6	29.57	324	10.69	168	18.87	156	26.96	76
2	2	40.12	572	8.57	168	31.54	404	28.46	96
2	5	33.72	764	12.68	444	21.04	320	23.68	72
3	2	79.79	912	36.99	620	42.80	292	7.19	56
3	12	52.80	832	20.43	324	32.37	508	12.84	56
3	14	48.11	288	18.82	160	29.29	128	26.28	72
5	1	32.82	580	14.27	84	18.55	496	9.79	48
5	7	43.91	1088	17.69	548	26.22	540	15.71	60
5	13	52.70	520	28.46	372	24.24	148	17.55	36
6	9	67.94	1176	42.75	372	25.18	804	12.60	52
6	14	40.88	928	21.89	228	18.99	700	11.88	60
7	2	30.03	524	22.01	384	8.02	140	36.10	60
7	12	39.78	1192	16.04	544	23.74	648	33.47	60
8	2	38.56	572	13.43	216	25.13	356	17.51	56
8	7	47.48	448	8.58	104	38.90	344	13.75	32
8	11	40.47	688	0.91	20	39.56	668	18.74	68
8	14	52.48	1200	9.64	516	42.85	684	18.35	28
9	3	50.85	920	19.64	384	31.21	536	28.54	128
6	4	45.98	740	16.30	264	29.68	476	22.52	52
9	6	46.86	920	15.57	404	31.29	516	9.20	36
9	16	55.80	1116	14.18	448	41.62	668	18.96	56
15	2	35.74	636	5.90	112	29.85	524	18.32	212
15	10	36.59	404	5.82	124	30.77	280	15.33	152

*TPH is the number of trees per hectare.

Table A1.3: Mean stand conditions for the 25 plots used in this study.

Variable	Min	Mean	Max
Trees/ha (live and dead)			
WF	16	309.44	644
DF	4.00	75.04	336.00
PP	0	82.4	916
Stand	288	745.92	1200
Basal Area (m^2/ha)			
Trees> 60 cm dbh	0.00	11.94	38.87
Stumps > 60 cm	1.45	14.86	35.04
Stumps > 15 cm	7.19	19.05	36.10
Dead	0.91	17.44	42.75
Live	8.02	28.81	42.85
White fir	1.49	30.42	62.00
Douglas-fir	30.98	8.02	28.78
Ponderosa pine	0.00	6.31	33.29
% of Basal area			
White fir	4.08	64.72	92.00
Douglas-fir	0.46	19.09	80.53
Ponderosa pine	0.00	12.70	63.43
Live white fir*	4.85	60.28	94.60
Live Douglas-fir*	0.00	19.00	80.04
Live ponderosa pine*	0.00	16.96	66.66
Dead white fir**	0.00	69.36	100.00
Dead Douglas-fir**	0.00	16.75	81.30
Dead ponderosa pine**	0.00	10.73	94.73
Quadratic mean diameter (cm at dbh)			
Stand	20.7	29.1	46.1
White fir	18.3	27.7	41.6
Douglas-fir	17.4	42.3	81.1
Ponderosa pine	5.0	51.7	130.5

* of the total live basal area

** of the total dead basal area

Table A1.4: The mean number of trees/ha for the study plots.

Trees/ha		Min	Mean	Max	Min	Mean	Max
Live					**Dead**		
Tall-intermediate							
	White fir	0	145.76	600	0	93.12	320
	Douglas-fir	0	11.2	160	0	3.36	80
	Ponderosa pine	0	10.72	84	0	14.4	320
Dominant							
	White fir	4	83.68	324	0	99.52	224
	Douglas-fir	0	11.36	84	0	14.56	92
	Ponderosa pine	0	20.64	252	0	11.2	136
Emergent							
	White fir	12	69.76	164	0	51.84	136
	Douglas-fir	0	19.2	136	0	10.08	56
	Ponderosa pine	0	12.48	116	0	1.92	12
Tall-emergent							
	White fir	0	8.48	28	0	5.28	40
	Douglas-fir	0	3.68	20	0	1.44	16
	Ponderosa pine	0	4.96	32	0	0.64	4

Table A1.5: The live and dead average basal area/ha for the study plots.

Basal Area (m^2/ha)		Min	Mean	Max	Min	Mean	Max
Live					**Dead**		
Tall-intermediate							
	White fir	0	0.93	3.46	0	0.82	3.57
	Douglas-fir	0	0.04	0.61	0	0.04	0.87
	Ponderosa pine	0	0.11	1.27	0	0.14	3.16
Dominant							
	White fir	0.20	3.25	13.76	0	3.79	8.09
	Douglas-fir	0	0.53	4.10	0	0.58	3.49
	Ponderosa pine	0	0.81	10.26	0	0.38	4.09
Emergent							
	White fir	1.29	9.46	19.14	0	6.53	17.93
	Douglas-fir	0	2.85	19.47	0	1.22	6.36
	Ponderosa pine	0	1.37	12.64	0	0.27	1.88
Tall-emergent							
	White fir	0	3.42	12.18	0	2.22	17.88
	Douglas-fir	0	2.06	12.10	0	0.71	7.10
	Ponderosa pine	0	2.95	16.79	0	0.28	2.49

Appendix 2: Infected Basal Area

Table A2.1: Armillaria infected basal areas by plot.

Timber sale unit	Plot	Infected BA (m^2/ha)	*Armillaria* BA (m^2/ha)	*Armillaria* infected BA trees dead 1-4yr (m^2/ha)	*Armillaria* infected BA trees dead 5-10yr (m^2/ha)	*Armillaria* infected BA trees dead 10+ (m^2/ha)
1	11	29.10	29.10	4.26	15.94	6.11
1	2	13.45	12.97	2.32	5.16	5.02
1	6	4.83	4.56	3.49	0.35	0.59
2	2	6.36	6.36	2.67	2.93	0.68
2	5	11.75	11.29	4.96	4.15	2.18
3	2	30.28	29.15	12.33	12.29	2.95
3	12	16.59	15.17	3.23	9.15	2.48
3	14	17.20	15.06	8.71	3.48	2.52
5	1	8.67	8.67	0.37	1.31	6.73
5	7	13.81	13.45	5.96	6.72	0.78
5	13	24.91	23.66	3.03	12.68	7.58
6	9	36.51	36.51	1.59	15.62	17.74
6	14	13.67	13.67	2.64	9.07	1.82
7	2	17.42	17.42	0.58	15.51	1.17
7	12	10.22	9.88	2.62	5.75	1.52
8	2	11.28	11.28	6.03	3.10	0.00
8	7	7.13	5.02	0.91	0.58	2.39
8	11	0.26	0.26	0.00	0.00	0.00
8	14	0.25	0.25	0.00	0.25	0.00
9	3	11.42	5.86	2.48	0.75	0.81
6	4	8.41	8.41	0.27	5.74	2.31
9	6	7.85	6.88	3.16	2.25	1.17
9	16	12.19	10.41	5.42	3.33	0.15
15	2	4.13	4.06	0.73	2.55	0.00
15	10	0.00	0.00	0.00	0.00	0.00

Table A2.1: Annosus infected basal areas by plot.

Timber sale unit	Plot	Infected BA* (m^2/ha)	Annosus BA (m^2/ha)	Annosus infected BA trees dead 1-4yr (m^2/ha)	Annosus infected BA trees dead ha 5-10 yr (m^2/ha)	Annosus infected BA trees dead 10+ (m^2/ha)
1	11	29.10	0.00	0.00	0.00	0.00
1	2	13.45	0.49	0.00	0.49	0.00
1	6	4.83	0.27	0.00	0.00	0.00
2	2	6.36	0.00	0.00	0.00	0.00
2	5	11.75	0.46	0.00	0.32	0.00
3	2	30.28	1.14	0.63	0.30	0.21
3	12	16.59	1.41	0.39	0.64	0.38
3	14	17.20	2.14	0.00	1.18	0.96
5	1	8.67	0.00	0.00	0.00	0.00
5	7	13.81	0.36	0.00	0.11	0.24
5	13	24.91	1.25	0.08	1.17	0.00
6	9	36.51	0.00	0.00	0.00	0.00
6	14	13.67	0.00	0.00	0.00	0.00
7	2	17.42	0.00	0.00	0.00	0.00
7	12	10.22	0.34	0.11	0.23	0.00
8	2	11.28	0.00	0.00	0.00	0.00
8	7	7.13	2.11	0.78	1.05	0.27
8	11	0.26	0.00	0.00	0.00	0.00
8	14	0.25	0.00	0.00	0.00	0.00
9	3	11.42	5.56	0.82	3.76	0.38
6	4	8.41	0.00	0.00	0.00	0.00
9	6	7.85	0.98	0.00	0.85	0.00
9	16	12.19	1.79	1.59	0.20	0.00
15	2	4.13	0.07	0.07	0.00	0.00
15	10	0.00	0.00	0.00	0.00	0.00