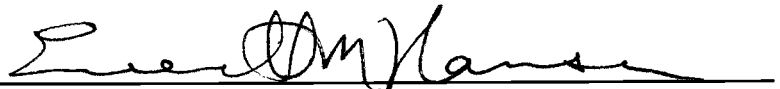


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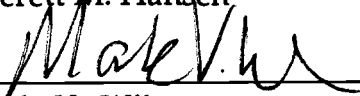
Jennifer C. Holah for the degree of Master of Science in
Botany and Plant Pathology presented on September 19, 1991.

Title: Effects of *Phellinus weirii* on Plant Community Composition and Succession of Mature and Old-growth Douglas-fir Forests.

Abstract approved: _____



Everett M. Hansen



Mark V. Wilson

Disease is often overlooked as a natural disturbance agent in plant communities. This study examines what effects, if any, a disease-mediated disturbance has on the plant community as a whole in old-growth and mature forests of western Oregon.

Phellinus weirii (Murrill) Gilbertson (Family: Hymenochaetaceae) is a native root-rotting pathogen that has co-existed with its conifer hosts for thousands of years. *P. weirii* can infect the roots of nearly all conifers, infection eventually leading to mortality for Douglas-fir, mountain hemlock, and true fir species. As the pathogen grows slowly via root contacts and grafts of conifers, areas of mortality are left in its wake, areas commonly called infection centers.

Herb, shrub, and tree species presence and percent abundance were noted inside and outside six infection centers located throughout the

Cascade and Coast ranges. Douglas-fir is the major species experiencing mortality due to the disease in this region. The vegetation inside infected areas was compared to that found immediately adjacent to infection centers.

A Multi-Response Permutation Procedure (MRPP), a non-parametric multivariate analysis of variance technique, was used to test for significant differences in the composition as a whole between the two areas of each site. An ordination technique, Detrended Correspondence Analysis (DCA), was used to examine if the effect the disease had on the plant community was a major factor underlying composition patterns. Common herb, shrub, and tree species, excluding Douglas-fir, were examined separately for significant differences in cover between the infected and non-infected areas for each site. Differences in the abundance of late-successional species and their regeneration between the two areas were tested in order to assess possible past and future impacts the disease had on succession in these forests.

All six infection centers had significantly different overall species composition compared to the composition of the adjacent non-infected areas. The effect of the disease on the forest composition was a major agent influencing community composition patterns for the six areas.

The responses of herb, shrub, and tree populations to disease presence was species-specific and varied across sites, responses varying especially between Cascade and Coast range sites for some species.

Effects of the disease on overall plant diversity appear to be dependent

on site characteristics. Though generalizations are often made about disease as a diversifying agent in communities, vascular plant diversity was not significantly enhanced by P. weirii, with the exception of one site.

In terms of successional impacts, the succession rates may be accelerated in Cascade forests because the growth of late-successional species is promoted within infection centers.

Effects of Phellinus weirii on Plant Community
Composition and Succession of Mature and Old-growth
Douglas-fir Forests

by

Jennifer C. Holah

A THESIS

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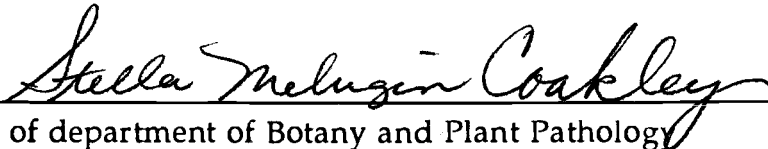
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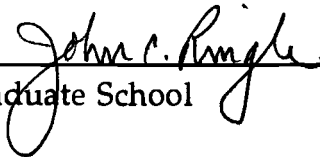
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The thesis is dedicated to Andrew, for his patience, and to my mom, who always said I could.

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EFFECTS OF Phellinus weirii ON PLANT COMMUNITY COMPOSITION AND SUCCESSION OF MATURE AND OLD-GROWTH DOUGLAS-FIR FORESTS

CHAPTER I. INTRODUCTION

Background Information on Phellinus weirii

James Weir collected the first samples of Phellinus weirii (Murrill) Gilbertson (Class: Basidiomycete; Family: Hymenochaetaceae) in 1912 from western red cedar in northern Idaho. The fungus was described and named Fomitiporia weirii, or Poria weirii, by Murrill (1914). Gilbertson (1974) later placed it in the genus Phellinus.

The fungus is heterothallic and lacks clamp connections (Hansen, 1979a). Basidiocarps of Phellinus weirii are brown, resupinate, perennial or annual, and are found on the underside of fallen infected trunks. Basidiospores are hyaline, ovoid, smooth, and their size ranges between 4.5 - 6 x 3.5 - 4.5 μ m. Abundant setal hyphae in the mycelium and the laminated yellowish decay it causes are characteristic of the fungus (Gilbertson & Ryvarden, 1987).

Two strains of the fungus are commonly recognized in the western United States: one causing a butt rot of western red cedar that occurs primarily in the Rocky Mountains, and the other, the strain dealt with here, causing a root rot of a diversity of conifers in the Pacific Northwest (Mounce et al., 1940). Angwin (1989) has demonstrated the genetic isolation

of these two strains, indicating that they are distinct biological species. A third Asian strain was also found to be genetically distinct from the two strains, or species, in the U.S (Angwin, 1989).

The incipient decay is reddish brown to brown appearing as an irregular shape or as a crescent-shape in a cross-section of the heartwood. Roots of dead trees are often broken transversely near the trunk base. Symptoms of infected standing trees include ragged crowns, poor color, thin foliage, decreased terminal and lateral growth, stress crops of cones, and a greater tendency to be attacked by bark beetles (Childs, 1970; Hadfield & Johnson, 1977). It has been estimated that the fungus is present within 11 meters from the last visibly infected tree encountered (Hadfield & Johnson, 1977).

Though the Pacific Northwest strain of P. weirii can infect nearly all kinds of conifer roots, tolerance to decay varies widely among tree species. The tree species in which mortality due to the fungus is considered inevitable are mountain hemlock (Tsuga mertensiana), Douglas-fir (Pseudotsuga menziesii), and true fir species. Larch, Shasta red fir, and Engelmann spruce are intermediately susceptible. Western white pine and lodgepole pine are tolerant of infection, and ponderosa pine is considered resistant (Filip & Schmitt, 1978). P. weirii has never been reported on roots of hardwood species.

Economic Importance of Phellinus weirii

Because Douglas-fir is of great economic importance to the Pacific Northwest, Phellinus weirii research is focused primarily on this species, though its effects on true firs can be devastating (Filip & Goheen, 1984). The disease was believed to be rare or virtually absent from old-growth Douglas-fir stands and was perceived as an economic threat only in the younger stands (Childs, 1960). It was predicted decades ago that, as management of second-growth Douglas-fir became more intensive, so would the effects of this fungus (Buckland *et al.*, 1952). Today it is the major disease problem of Douglas-fir and local surveys indicate that P. weirii may affect 5 to 12% of Douglas-fir forests in western Oregon (Gedney, 1981; Goheen & Hansen, unpubl.). Over 80% of the second growth Douglas-fir stands in the Vancouver Forest Region are believed to be infected by P. weirii (Bloomberg & Reynolds, 1985).

Spread of Phellinus weirii

Spread of P. weirii is believed to be solely vegetative through root grafts or root contacts (Wallis & Reynolds, 1965). Inoculum is long-lived and may remain viable in stumps for up to 50 years (Hansen, 1979b). The fungus produces basidiospores and there has been some indication that spores may play a role in the establishment of new infection centers (Dickman, 1984), but there is no definitive evidence to prove their importance.

Infection centers have a clumpy spatial distribution across a landscape (Childs, 1970), and centers are visible by air (Martin & Williams, 1986), particularly in the mountain hemlock areas (Nelson & Hartman, 1975).

In mountain hemlock forests, *P. weirii* is estimated to spread at a rate between 34 - 43 cm/year (Cook, 1982; Nelson & Hartman, 1975), whereas spread rates in Douglas-fir forests have been estimated to be less than 30 cm/year on the average (Childs, 1970). However, uneven advances seem to be the norm in most infection centers (Childs, 1970; Bloomberg, 1990).

Old growth stands of Douglas-fir may superficially appear to have a slower rate of disease spread as compared to young stands. Disease spread may not appear as rapid due to a coalescing of smaller infection centers in older stands or the fact that larger trees take longer to be killed by the fungus (Hansen, pers. comm.). Bloomberg and Reynolds (1985) have proposed that a "disease stability" may be reached in older stands. Larger trees may be more tolerant of infection and may benefit from decreased competition with other dominant Douglas-fir inside infection centers. Also, there is a trend of decreased infection in areas that have a higher component of more resistant tree species, such as western hemlock (Bloomberg & Beale, 1985), or greater tree diversity in general (McCauley & Cook, 1980). Areas devoid of trees, or containing trees infected by other agents, such as *Armillaria* sp., are most important in arresting fungal advance (Bloomberg, 1990).

Unlike what has been found for other root-rotting pathogens, it has been difficult to determine what environmental factors, if any, play a role

in the spread of P. weirii (see Bloomberg, 1990). Soil moisture seems to be one of the more important variables associated with the fungus. Areas with either very dry or very wet soils have been found to contain the least disease, whereas those with soils on the dry side, usually those rich in nutrients as well, seem to be associated with more disease (Bloomberg & Beale, 1985; Williams & Marsden, 1982). Steptomyces sp. resident in forest soils may play a role in the suppression of P. weirii (Hutchins & Rose, 1984).

Effect of Phellinus weirii on Plant Communities

The effects of Phellinus weirii on a community level have been examined in the mountain hemlock forests in the Waldo Lake area of the high Cascades in western Oregon. Root rot infection centers are large, often hundreds of meters across, and are readably visible by air as 'doughnut-holes' across the landscape (Nelson & Hartman, 1975). The large area of pure stands of mountain hemlock are unique to this area, and may explain why the disease is particularly severe here (Hansen, pers. observ.).

Copsey (1985) found successional pathways of communities affected by the disease were altered, and that these changes persist for hundreds of years, perhaps indefinitely. Early succession after fungal attack results in a "superficially similar community" to that resulting from fire (Dickman & Cook, 1989). Both types of disturbance cause a decrease in canopy cover and an increase in the pine populations. However, different species assemblages are involved with the disease-induced disturbance and compositional

differences with adjacent non-infected forest persist inside infection centers (Copsey, 1985; Dickman & Cook, 1989).

As Phellinus weirii advances, virtually every large mountain hemlock is eliminated (Cook, 1982). Though mountain hemlock is a major regeneration species inside infection centers, there is a higher hemlock sapling density outside the infected areas, and hemlocks inside centers never reach the size or age that is attained in non-infected areas (Cook, 1982; McCauley & Cook, 1980). The large trees that are encountered most frequently in infected areas are white pines and noble firs (Copsey, 1985). Though Abies amabilis is susceptible to P. weirii, infection centers are one of the few places where this species can attain canopy status with mountain hemlock (Cook et al., 1989). A steady-state climax in which mountain hemlock dominance is shared with other species is governed principally by the presence of P. weirii (Cook, 1982).

McCauley and Cook (1980) make the analogy between P. weirii and a "predator" in the mountain hemlock studies as it opens the system to species that otherwise would not be as prevalent. Tree species diversity was higher at the center of infection centers as compared to the non-infected old-growth mountain hemlock stands in areas where A. amabilis was not common. Because there are many oscillations of re-infection inside the centers however, diversity could vary greatly (Cook et al., 1989). The increased diversity found inside infection centers may slow the rate of fungal advance, though it is difficult to sort out which is cause and which is effect (McCauley & Cook, 1980).

Despite the increase in less susceptible and resistant species inside infection centers, P. weirii does persist and will continue to re-infect regenerating mountain hemlock (Cook, 1982). A hypothesis posed for why the disease has not engulfed the entire forest is a "quasi-equilibrium" control of P. weirii by fire (Dickman & Cook, 1989). Fire may reduce P. weirii infection areas by favoring the regeneration of those species least susceptible to infection, pines. P. weirii infestation may enhance the probability of fire because it increases the fuel load (Dickman, 1984). The possibility has also been raised that P. weirii will eventually spread to such an extent that mountain hemlock will lose its present dominance (Dickman & Cook, 1989).

Little work has been done examining the effects of P. weirii on natural Douglas-fir communities. This may be because the communities are more complex, multi-layered in both composition and structure, and the effect of the disease is not as visually obvious. Childs (1970) examined the response of western hemlock to P. weirii incidence in the Coast range. He found that hemlock release to canopy status was more likely in those areas with high P. weirii incidence. Most of his data illustrating this fact was unfortunately lost.

Research intents

Up to this point, however, there has not been a definitive study exploring if P. weirii affects Douglas-fir communities as it does the mountain hemlock forests.

Using the work that has been done in the mountain hemlock communities as a starting point, this study examines whether or not the presence of the disease affects community composition in Douglas-fir forests at all, as its effects are not immediately obvious in this area as compared to the mountain hemlock area. This study also examines whether or not the disease enhances diversity as it does in the mountain hemlock areas, and if it redirects successional pathways.

My study differs from previous ones in that the total vascular plant community is taken into account, instead of the focus being primarily on the tree species. No previous studies have examined the indirect effects that this root-rotting pathogen may have on the herb and shrub understories of forest communities. Changes in the understory composition could have ramifications on the regeneration of canopy species. Also, it would be interesting to know how far the effects of this pathogen extend in old-growth forests, a highly stratified community. Very little is currently known concerning the effects of species-specific pathogens on a community level in general.

Study areas

The areas chosen were all located in the lower Cascade and Coast ranges of western Oregon. All are part of the *Tsuga Heterophylla* Zone as defined by Franklin and Dyrness (1984). All stands but the Rooster Rock site were unlogged old-growth, having Douglas-fir as the major canopy species. The

Rooster Rock stand had naturally regenerated after fire and was approximately 100 years old. See Figure I - 1 for the location of the six study areas.

Although an area may possess more than one infection center, the study took place in only one center per site. Examination of non-infected communities took place immediately adjacent to the infection center at each site. Refer to Table I - 1 for the plant association type of each site as defined by Dyrness et al. (1974) and Table I - 2 for a general description of each site.

Infection centers were characterized by the death of six or more Douglas-fir, usually in various stages of decay. Identification of the fungus as the mortality agent of the fallen trees was confirmed with the presence of setal hyphae in the laminated rot, which are readably visible with a hand lens.

TABLE I - 1: Classification of plant communities at each site based on association types defined by Dyrness et al. (1974).

SITE	PLANT ASSOCIATION TYPE
HJA 1	<i>Pseudotsuga menziesii</i> / <i>Acer circinatum</i> / <i>Berberis nervosa</i>
HJA 2	<i>Tsuga heterophylla</i> / <i>Polystichum munitum</i> - <i>Oxalis oregana</i>
HJA 3	<i>Pseudotsuga menziesii</i> / <i>Acer circinatum</i> / <i>Gaultheria shallon</i>
Rooster Rock	<i>Tsuga heterophylla</i> / <i>Polystichum munitum</i>
Mary's Peak	<i>Pseudotsuga menziesii</i> / <i>Acer circinatum</i> / <i>Gaultheria shallon</i>
Aalsea	<i>Tsuga heterophylla</i> / <i>Acer circinatum</i> / <i>Polystichum munitum</i>

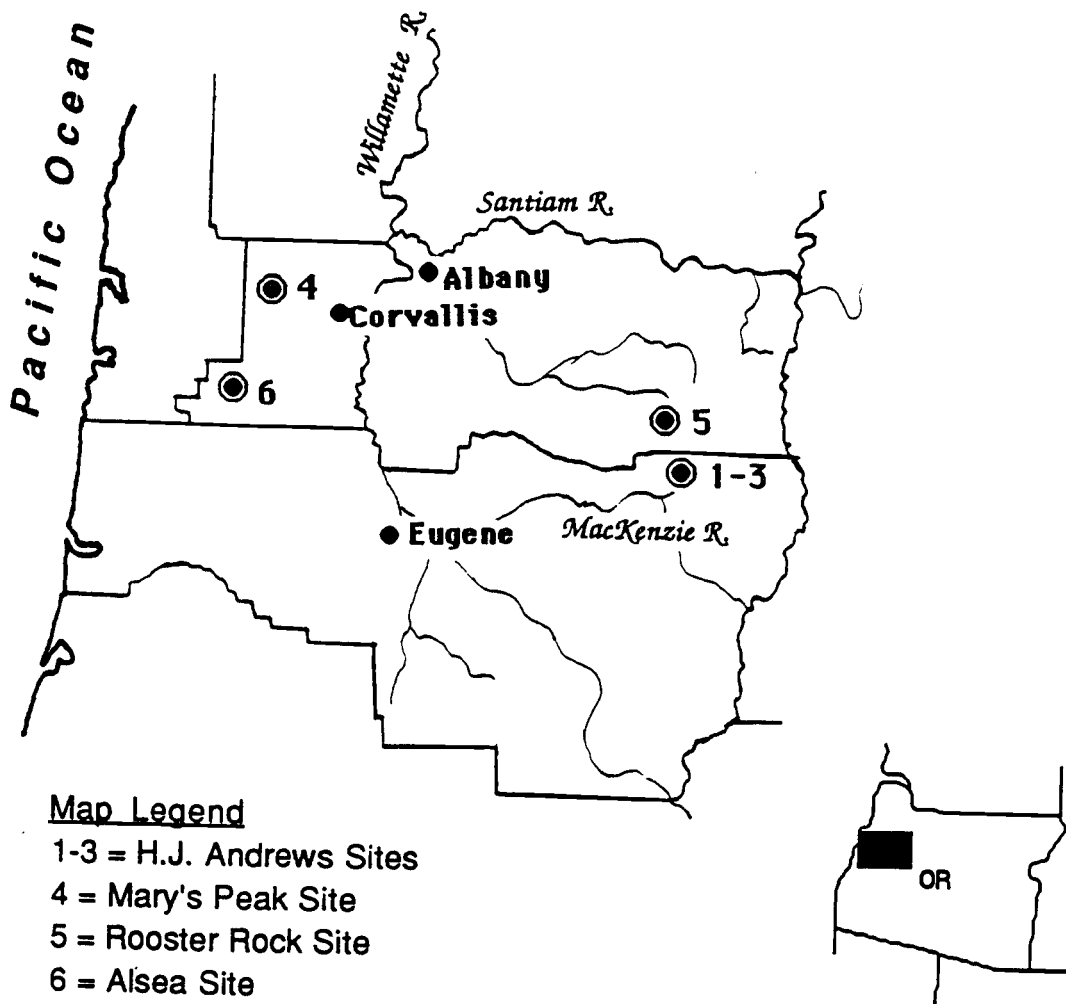


Figure I - 1. Location of the six study areas in western Oregon.

Table I - 2: General characteristics of each site.

Site	Elevation	Approximate infection center diameter	Average Douglas-fir basal area (m ² /ha)		Slope aspect	Stand type
			inside	outside		
HJA 1 (199 plots)	1036 m	150 m	40	61	south	old-growth
HJA 2 (40 plots)	512 m	50 m	2	89	south-east	old-growth
HJA 3 (40 plots)	914 m	40 m	35	93	north-west	old-growth
Rooster (39 plots)	610 m	20 m	29	69	south	mature
Mary's Peak (40 plots)	457 m	25 m	3	39	east	old-growth
Aalsea (40 plots)	340 m	40 m	22	85	west	old-growth

Site criteria and sampling methods are described in more detail in the following chapter.

CHAPTER II. EFFECTS OF PHELLINUS WEIRII ON FOREST COMPOSITION

Abstract

Phellinus weirii (Family: Hymenochaetaceae) is a fungal pathogen that causes extensive rot in the roots and bole of Pseudotsuga menziesii (Douglas-fir) and true firs, eventually leading to tree mortality. The native pathogen spreads slowly via root grafts and root contacts of conifers, leaving behind areas of Douglas-fir mortality commonly called infection centers. This study examines whether or not the slow, systematic removal of the Douglas-fir overstory by P. weirii changes the community composition of old-growth and mature forests and, if it does have a significant effect, to what degree P. weirii influences the composition. What effects, if any, the disease has on individual populations and on vascular plant diversity in general are also addressed. Possible mechanisms for such changes are explored as well.

The herb, shrub, and tree strata were randomly sampled within and adjacent to six P. weirii infection centers located across the low elevation Cascade and Coast ranges of western Oregon.

A non-parametric multivariate analysis of variance test, Multi-Response Permutation Procedure (MRPP), revealed significant ($p \leq 0.05$) differences in species composition between plots inside the infection center and outside the infection center for all sites. Detrended Correspondence Analysis (DCA) showed that the distance of vegetation to the infection center edge was either the major, or a major, factor underlying the forest community's structure.

Of the eight species most common to all sites, Acer circinatum, Tsuga heterophylla, and Berberis nervosa had significantly different ($p \leq .05$) percent cover values inside and outside infection centers for two or more sites. Percent cover of Polystichum minutum exhibited similar distribution trends across all sites, though the data was statistically significant ($p \leq .05$) at only one location. Changes in cover due to disease differed between Cascade and Coast range sites for T. heterophylla and A. circinatum. The average cover of all herbaceous species per plot was higher inside infection centers as compared to outside for all locations, though statistically significant at only two sites.

Changes in the herb and shrub species inside infection centers resulting from the gradual removal of the Douglas-fir overstory by disease could not be attributed to changes in canopy cover. Canopy cover was not significantly different ($p > .05$) between infected and non-infected areas of all sites, with the exception of the mature stand site.

Plant diversity was lower inside infection centers at most old-growth Cascade sites, whereas the mature Cascade site and one of the Coast Range sites had significantly ($p \leq .05$) greater diversity inside infection centers.

The results suggest that P. weirii acting as a long-term disturbance agent in the study areas significantly altered the forest community composition, and that the effects are species-specific and site dependent.

Introduction

Disturbance has gained notoriety as an important factor determining plant community structure. A community's composition (Denslow, 1985; White, 1979), diversity (Canham & Loucks, 1984; Connell, 1989; Denslow 1980, 1985; Kimmerer & Allen, 1982; Loucks, 1970; Miller, 1982), and successional pathway (Menges & Loucks, 1984; Pickett & White, 1985), generally may be influenced by disturbance. Attention has shifted from the importance of large scale 'allogenic' disturbances in forested ecosystems to the importance of smaller scale 'autogenic' disturbances, such as single treefall gaps, in determining community structure (Oliver & Stephens, 1977; Pickett & White, 1985; Runkle, 1985; Runkle & Yetter, 1987; Spies & Franklin, 1989).

Species composition can, in part, be a function of the disturbance regimes for a particular area (Ehrenfeld, 1980; White, 1979). Few studies recognize the importance of disease or the full spectrum of biotic agents that may play a role in disturbance, especially in forested communities (Worrall & Harrington, 1988; Menges & Loucks, 1984). Plant disease has traditionally been viewed as rare in natural systems, as compared to agricultural systems, and, if present, as a sign that the system is 'out of balance' (Dinoor & Eshed, 1984; Harlan, 1976; Harper, 1977). However, fungal diseases can be as severe in natural populations as in crop situations (Kranz, 1990). Disease has been implicated as an important factor in limiting host population distributions (Alexander, 1984; Burdon & Shattock, 1980; Rochow, 1970), especially with cases of

introduced pathogens (Smith, 1986), in influencing host population fitness (Alexander, 1988; Burdon & Shattock, 1980), and in changing the competitive interactions between populations (Alexander, 1988; Burdon & Chilvers, 1974). Disease is used to explain how plant populations can co-exist in what appears to be shared niches (Alexander, 1988; Burdon & Chilvers, 1974). The presence of disease in patchy communities may increase the competitive effect of non-infected plants (Burdon & Shattock, 1980) or resistant plants of the same species (Finckh & Mundt, unpubl.).

These effects on a population level can have impacts on a community level, although our understanding of the role of pathogens in plant communities is comparatively poor (Burdon, 1982). As an example, the high diversity of eucalypt species reaching canopy may in part be attributed to past high levels of pathogen attack on the more abundant eucalypt species seedlings (Burdon & Chilvers, 1974). Impacts at the community level may be different depending on the nature of the disease and the type of community affected. The devastating chestnut blight in eastern forests was correlated with an increase of hemlock gaining overstory status (Oliver & Stephens, 1977), but overall no one tree species has yet to dominate the canopy after chestnut removal (McCormick & Platt, 1980; Woods & Shank, 1959). However, the plant communities in the Jarrah forest of Australia have vastly different composition compared to their composition prior to infection by Phytophthora cinnamomi (Newhook & Podger, 1972).

In one of the few studies done on the causal nature of disturbance

regimes across a broad landscape, root rot in combination with wind throw was the primary mortality factor causing forest gaps in high elevation spruce-fir forest of New Hampshire (Worrall and Harrington, 1988). Most of the work on the role of native diseases in natural populations, however, has dealt with herbaceous perennial species, not with tree species (see Burdon & Shattock, 1980), and with air-borne fungal pathogens (Sewall, 1981).

Disease as a natural disturbance agent affecting community structure has been examined extensively in the *Tsuga mertensiana* (mountain hemlock) forests of the high Cascades in Oregon. *Phellinus weirii*, a native fungal root rot pathogen, is the major determinant of forest community structure in areas where the pathogen is, or was, present (Copsey, 1985). Differences in forest community development inside and outside of infected areas are directly related to species-specific responses to infection by *P. weirii* and may persist for hundreds of years (Cook, 1982; Copsey, 1985). Some of the observed differences include changes in successional pathways of infected compared to non-infected areas, higher species diversity within infection centers (Cook et al, 1989; Copsey, 1985; Dickman & Cook, 1989; McCauley & Cook, 1980), and changes in the microbial flora (Burket, 1989) and soil nutrient status (Matson & Boone, 1984).

Pseudotsuga menziesii (Douglas fir) is another tree species highly susceptible to *P. weirii* infection where infection inevitably leads to mortality. The pathogen is quite common in Douglas-fir forests; an estimated 5 to 12 % of Douglas-fir plantations are believed to be infected by *P. weirii* (Gedney,

1981; Goheen & Hansen, unpubl.). Currently, we do not know if the disease effects these forests as it does the mountain hemlock old-growth. Due to the greater structural and compositional complexity of old growth Douglas-fir forests (Franklin & Dyrness, 1984) compared to mountain hemlock communities, the effects of the pathogen have perhaps not been as obvious in these areas.

The purpose of the following research was to:

- determine whether or not the presence of Phellinus weirii in the old-growth Douglas fir communities of the Cascade and Coast ranges of Oregon has an effect on forest composition.
- assess the importance of this effect, if present, as an underlying factor determining community patterns using ordination techniques.
- determine the effect disease has on the herb, shrub, and tree components of the community, information which is absent in prior P. weirii studies and few gap studies of forested areas in general (Spies et al., 1990).
- examine if the presence of disease at each site enhances species diversity.
- test the hypothesis that changes in canopy cover is the mechanism responsible for changes in the understory inside infection centers.

Understory development usually is not considered important in canopy development, but it has been shown that herbaceous and shrub species can form stable associations over time that may outcompete tree reproduction, or delay it to such an extent that overstory composition is affected (Ehrenfeld, 1980). It would also be useful to know how far the gradual overstory removal

of Douglas fir in the areas examined extends in the multiple strata levels of old-growth forest, and what types of species are particularly impacted.

Because laminated root rot is selectively removing an overstory dominant tree species at a steady rate in the study areas, the hypothesis was posed that the disease was a diversification agent of the community.

Disturbance agents in general can enhance species diversity by lowering the dominance of certain species, thus freeing up resources for others and increasing the environmental heterogeneity of a community (Connell, 1978; Levin & Paine, 1974; Denslow, 1985). Pathogens, especially species-specific pathogens, are ideal candidates for increasing species diversity (Harper, 1977), through both of these mechanisms.

Augsburger's (1983, 1984) work on the role of pathogens in tropical tree seedling establishment suggests that the high floristic diversity found in the tropics may be in part attributed to host-specific pathogens. It has also been suggested that pathogen pressure in a community may create, or maintain, the genetic diversity of host populations (Alexander, 1987; Burdon *et al.*, 1989). Forest regeneration after *P. weirii* infestation had a higher tree species diversity compared to regeneration after fire in mountain hemlock forests of the high Cascades (Copsey, 1985). In areas where *Abies* sp. were uncommon, tree diversity was higher in infection centers as compared to non-infected areas (Cook *et al.*, 1989; McCauley & Cook, 1980).

Because *Phellinus weirii* is decreasing Douglas-fir's dominance in these forests, and the disease was found to be a diversifying agent previously in the mountain hemlock studies, the hypothesis was made that plant diversity

would be higher in infection centers.

Materials and Methods

Study sites. Six sites of Phellinus weirii infection ('infection centers') were chosen in the Cascade Mountains and Coast Ranges of western Oregon. An infection center was chosen as a study site if it fulfilled the following criteria: 1) the major canopy tree species in the area was Pseudotsuga menziesii, 2) the forest qualified as "old-growth", i.e. the canopy tree species were 200 years or older, 3) the area had never been logged, 4) the infection center consisted of the death of at least six P. menziesii, 5) P. weirii was the major disturbance agent presently active at the site, and 6) infection centers were derived from single clones of the fungus. Three of the six sites were located in the H. J. Andrews Experimental Forest near Blue River, OR, hereafter called sites HJA 1, 2, 3, at elevations of 1036, 512, and 914 meters respectively. Two sites were located in Siuslaw National Forest in the Coast Range; one in the Mary's Peak watershed near Corvallis, OR, the other in the Deadwood Creek area near Alsea, OR, with elevations of 457 and 340 meters respectively. The last site was located at the Rooster Rock area of the Menagerie Wilderness in the Cascade Mountains near Cascadia, OR, with an elevation of approximately 610 meters.

The five criteria were met at all six study sites chosen except that the Rooster Rock site was a mature stand (approximately 100 years old) naturally

regenerated after fire, and two or three large western red cedars at the HJA 2 site were logged probably in the 1950's (A. W. McKee, pers. comm.).

Fungal isolates were collected from various locations within the infected area from sites where the leading edge of infection appeared to be the possible result of several small centers converging. The isolates were cross-plated and tested for vegetative compatibility (Childs, 1963). Data were collected from infection centers originating from single fungal clones only.

Sampling strategy. At each site, the infection center edge was defined as the area half-way between the last Douglas-fir killed by *P. weirii* and the first symptomless Douglas fir encountered. The edge was marked with flagging at regular intervals. With the exception of site HJA 1, 40 random vegetation samples were taken from each site; 20 random samples from the outside of the marked infection center edge (non-infected forest) and 20 random samples from inside the marked edge (infected forest). Site HJA 1 consisted of a large infection center approximately 150 meters in diameter. This site was more intensively sampled than the other sites, with data collected from 226 plots. Twenty-seven of these plots were eliminated because the plots were located in or near riparian areas, on rocks, or in an area that had recently experienced a ground fire.

The random samples were found by generating random directions and distances with a random number table in the field. A compass bearing from 1 to 360 was chosen, and distances, ranging from 6 to 15 meters, were measured with a meter tape. At each random sample point, distance to the flagged edge

was measured. Distances were negative for plots inside the infection center, and positive for plots outside the center. This measurement was called the Plot Location Assignment (PLA) for that sample.

Data collection. Data on the herb, shrub, and tree species present at each plot were taken. The percent cover and species of each vascular plant present in a 1m x 1m area was noted. Any shrub or tree species present below a 1 m height over the plot was noted. A 6m radius (a 4m radius at the Mary's Peak site) from the center of each sample point was measured and the diameter at breast height (dbh) of all live tree and shrub species with diameters greater than 1 cm were recorded.

At each plot percent canopy cover was measured with a spherical convex densiometer (Lemmon, 1956). The relative slope, the percent cover of fine and coarse (>5 cm dbh) woody debris, and the cover of rocky talus, if present, were all recorded for each random plot as well.

Data reductions. The diameter data was converted into basal area data. The data were then standardized to be compatible with the data taken for the 1m x 1m plots. This was done by dividing the basal area of a species per hectare by the greatest possible basal area found per hectare for species of that group, according to which level in the canopy they were usually found. The greatest basal area out of all sites for the dominant canopy tree, P. menziesii, was 352 m²/ha. The replacement species, Tsuga heterophylla, Thuja plicata, Taxus brevifolia, and Acer macrophyllum, each did not have basal areas exceeding 143 m²/ha. The last group consisted of species that would never

obtain canopy status; these shrubby species included Rhododendron macrophyllum, Acer circinatum, Corylus cornuta, and Holodiscus discolor. The shrub species never exceeded 8 m²/ha for any sample taken.

Plots for each site were categorized in two ways in terms of their relationship to the infection center. The first categorization labelled plots as either inside or outside the infection center. The second categorization labelled plots as either outside, inside, or on the edge of the infection center. The definition of 'edge' for each site varied depending on the size of the infection center. In most cases the 'edge area' consisted of an area 6m wide on either side of the flagged edge.

The only direct effect of the pathogen on the communities sampled was the removal of the Douglas-fir canopy. Because the purpose of this study was not to assess this known direct effect of the disease, but if and how the removal of the dominant canopy species by P. weirii affected other components of the forest community, the Douglas-fir data were not included in the species x plots data matrices.

Statistical analyses. A Multi-Response Permutation Procedure (MRPP) was used in order to determine if the presence of the disease had a statistically significant effect on the community (McCune, 1987; Zimmerman et al. 1985). MRPP is similar to a multivariate analysis of variance but with relaxed requirements on the data structure. The test statistic, delta, is based on the within group average of pairwise distance measures, groups defined a priori, of data points in euclidean space.

Ordination techniques were used to determine the strength of this effect,

if significant, for each site. Ordination techniques in general are excellent for exploring patterns in large, confusing data sets, as they can boil down highly multivariate information to a few axes that may then be interpreted with regards to an environmental factor of interest. Detrended Correspondance Analysis (DCA, Hill & Gauch, 1980) was chosen as distortion of entities in ordination space is minimal and this method is good for exploring large data sets with no known environmental gradients underlying vegetation patterns. DCA ordination scores of the vegetation data for each site were correlated using Spearman's Rank Correlation coefficient against the PLA's for that site. A plot's PLA, or location relative to the leading edge of the infection, was a criterion defined solely by disease presence. Disease presence was interpreted to be the major, or a major, factor underlying community composition patterns if the PLA's were significantly correlated with the main ordination axes.

Mann-Whitney Rank tests (Conover, 1980) were used to examine which populations had significantly different ($p \leq .05$) cover values between infected and non-infected areas of each site. Because most species were absent or uncommon at several sites, eight of the most common species that occurred across all sites were chosen.

To explore the mechanisms by which disease was influencing vegetation, Mann-Whitney tests were also used to determine if changes in total canopy cover, and cover of individual canopy species were significantly related to PLA.

For each plot of each site, diversity of the herb, shrub and tree species present (with the exception of Douglas-fir) was calculated using Hill's Diversity Index (Hill, 1973): $e^{H'}$, where H' is Shannon's diversity index. An unpaired t-test was used to determine if diversity was significantly different between infected and non-infected areas.

Results and Discussion

The cover and frequency of all vascular plants that occurred on two or more sites are catalogued in Tables II - 1a and 1b.

Does the disease have an effect on the forest community? The composition of infected areas was compared to the plant community found in adjacent non-infected areas for each site in order to address this question, utilizing a multivariate, non-parametric analysis of variance test, called Multi-Response Permutation Procedure (MRPP). The composition of the forest community inside the infection center was significantly different from that found outside the infection center at all sites using MRPP analysis ($p \leq 0.05$). These results indicate that the presence of *P. weirii* has an effect on forest community composition, as the community data used in this analysis did not include changes in the cover for Douglas-fir, the only species directly affected by the pathogen.

The significant results of the MRPP analysis were derived from a simple two category scheme for each site: a plot was considered either inside or outside the infection center. It was conceivable however, that the leading edge of the infection center had a composition unique to either the center area

Table II - 1a: Average basal area of common trees and shrub species per hectare (%) for each site. Frequency of each species is in parentheses.

	<u>HJA 1</u> (199 plots)	<u>HJA 2</u> (40 plots)	<u>HJA 3</u> (40 plots)	<u>Mary's Peak</u> (40 plots)	<u>Rooster Rock</u> (39 plots)	<u>Alsea</u> (40 plots)
<i>Acer circinatum</i>	9 (103)	3 (27)	16 (37)	10 (31)	2 (17)	10 (19)
<i>Holodiscus discolor</i>	trace ¹ (2)	-----	-----	4 (25)	-----	-----
<i>Pseudotsuga menziesii</i>	13 (77)	13 (14)	18 (17)	6 (31)	14 (36)	15 (30)
<i>Rhododendron macrophyllum</i>	trace (1)*	1 (17)	5 (8)*	-----	2 (20)	-----
<i>Taxus brevifolia</i>	1 (16)	0.3 (19)	7 (36)	-----	-----	-----
<i>Thuja plicata</i>	trace (2)	2 (19)	8 (22)	-----	-----	-----
<i>Tsuga heterophylla</i>	21 (188)	19 (39)	5 (34)	2 (22)	-----	8 (34)

¹ = % basal area < 0.1/ha (%).

* = cover/m² (%) data.

Table II - 1b: Average cover (%)/m² for herb and shrub species that occurred at two or more sites. Frequency of each species is in parentheses.

	<u>HJA 1</u> (199 plots)	<u>HJA 2</u> (40 plots)	<u>HJA 3</u> (40 plots)	<u>Mary's Peak</u> (40 plots)	<u>Rooster Rock</u> (39 plots)	<u>Aisea</u> (40 plots)
<i>Achlys triphylla</i>	trace ¹ (3)	trace (2)	-----	-----	0.3 (3)	-----
<i>Adenocaulon bicolor</i>	-----	-----	-----	0.1 (1)	0.3 (5)	-----
<i>Anemone oregana</i>	0.1 (12)	-----	0.1 (2)	-----	0.6 (16)	-----
<i>Berberis nervosa</i>	11 (128)	3 (14)	6 (24)	15 (37)	18 (21)	2 (7)
<i>Castanopsis chrysophylla</i>	-----	-----	0.2 (1)	0.1 (2)	-----	-----
<i>Chimaphila menziesii</i>	trace (2)	-----	-----	-----	trace (1)	-----
<i>Cornus canadensis</i>	0.2 (5)	-----	3 (23)	-----	-----	-----
<i>Coptis laciniata</i>	8 (133)	3 (24)	-----	-----	-----	-----
<i>Gallium triflorum</i>	-----	0.1 (3)	-----	trace (3)	1 (18)	trace (1)
<i>Gaultheria shallon</i>	-----	1 (6)	8 (21)	15 (23)	6 (12)	10 (31)
<i>Goodyera oblongifolia</i>	0.1 (11)	trace (1)	0.4 (8)	-----	-----	-----
<i>Linnaea borealis</i>	1 (53)	4 (32)	8 (31)	0.1 (2)	0.1 (1)	-----
<i>Oxalis oregana</i>	-----	13 (29)	-----	-----	-----	0.8 (6)
<i>Pachystima myrsinites</i>	trace (2)	-----	0.1 (1)	-----	-----	-----
<i>Polystichum munitum</i>	0.8 (18)	15 (22)	2 (6)	6 (17)	13 (14)	13 (18)
<i>Rosa gymnocarpa</i>	0.1 (4)	-----	-----	0.6 (6)	-----	0.1 (1)
<i>Rubus sp.</i> ²	0.6 (57)	0.8 (12)	1 (13)	-----	0.5 (11)	1 (15)
<i>Symphoricarpos mollis</i>	0.2 (1)	-----	-----	0.1 (1)	-----	-----
<i>Tiarella unifoliata</i>	0.3 (30)	0.6 (15)	-----	-----	-----	-----
<i>Trientalis latifolia</i>	trace (4)	0.1 (2)	trace (2)	trace (2)	0.3 (7)	trace (1)
<i>Trillium ovatum</i>	trace (5)	0.1 (2)	trace (1)	trace (1)	-----	0.1 (4)
<i>Vaccinium membranaceum</i>	trace (4)	-----	-----	trace (1)	-----	-----
<i>Vaccinium parvifolium</i>	0.1 (12)	1 (7)	0.1 (2)	-----	0.2 (2)	1 (7)
<i>Vancouveria hexandra</i>	0.1 (6)	0.2 (4)	-----	-----	0.1 (2)	-----
<i>Viola orbiculata</i>	1 (74)	0.4 (18)	0.1 (1)	-----	3 (26)	-----

¹ = % cover < 0.1%/m², ² = either *R. nivalis* or *R. ursinus*.

of the infection or non-infected outside areas, as this was the area where the most recent mortality had occurred. In order to determine whether or not the area near the leading edge of infection was significantly different from the other areas, MRPP was used with the community data divided into three categories: a plot for each site was either in the center of the infected area ('center'), in an adjacent non-infected area removed from the edge ('non-infected'), or on the edge of the infection center ('edge').

The edge area was arbitrarily defined for each site based on that site's size and ranged between 5 and 8 meters on either side of the flagged infection edge for all sites. For every site significant differences between all combinations of the three areas were tested for using MRPP (Table II - 2).

Table II - 2: Probability values that the composition of one area of a study site is different from another in the three category scheme using the Multi-Response Permutation Procedure (MRPP).

	Center vs. Edge	Non-infected vs. Edge	Center vs. Non-infected
HJA 1	.102	.008*	.014*
HJA 2	.520	.266	.001*
HJA 3	.049*	.359	.018*
Mary's Peak	.245	.215	.001*
Rooster Rock	.459	.108	.014*
Aalsea	.854	.131	.012*

* = significant at $p \leq .05$ level.

Results indicate that the edge area does not have a composition that is unique to either the center or non-infected outside areas for four out of the six

sites ($p > .05$). The edge area's composition was significantly different from the inside of the infection center for HJA 3 ($p = .049$), and was significantly different from the outside area in HJA 1 ($p = .008$). Several infected Douglas-fir trees had died within the past two years in the edge areas of HJA 1 and HJA 3. Because of differences in microhabitat that are associated with very recent treefalls, the edge areas here differed from the surrounding areas. For all sites, the inside and the outside areas in the three category grouping were significantly different ($p < .02$).

As was found in the mountain hemlock community in the high Cascades, the presence of Phellinus weirii does influence community composition.

To what degree does Phellinus weirii influence community composition? Though the MRPP test yielded significant results for all six sites, this test cannot assess the strength of the disease effect on the community composition. Because ordination techniques reveal vegetation patterns based on their major underlying environmental influences, this technique was used to examine if the presence of the root rot was a major agent responsible for the composition patterns in the community.

Ordination scores for the first two DCA axes for each site were correlated with the PLA's for that site. If PLA was found to be significantly correlated with the major DCA axes, then the presence of the disease was likely to be the primary, or secondary, factor determining community composition.

Ordination scores for the first DCA axis for each site were significantly

correlated ($p < .02$) with the PLA's for four out of the six sites (Table II - 3).

The first DCA axis of HJA 3 was significantly correlated ($p = .005$) with the site's basal area of Douglas-fir/ha (%) for each plot. Because a plot's PLA was defined solely by the presence of the infection, whereas a plot's basal area of Douglas-fir was not dependent on the presence or absence of disease, the PLA correlations with the ordinations were considered the more robust test of whether or not disease was a major agent responsible for pattern in the community structure. However, because the presence of live Douglas-fir inside an infection center, as was especially the case at HJA 3, would produce conditions similar to those found in adjacent non-infected forest, a plot's distance to the defined edge could be meaningless. Thus correlations between Douglas-fir basal area and ordinations were examined as well.

The first DCA axis of the Mary's Peak site was not correlated with either the site's PLAs or the Douglas-fir cover data. However, the second axis ordination scores for this site were significantly correlated ($p < .003$) with the PLAs (Table II - 3).

Because other unexamined environmental factors such as water relations, or soil nutrient gradients, may have been playing an important role in community patterns, the conclusion cannot be drawn that disease presence was the primary factor underlying the forest structure. However, *P. weirii* can be a major determinant of community patterns. In order to test if the presence of the disease is the major agent influencing composition, the other influential variables would need to be measured and directly compared

against the effects due to disease.

Table II - 3: Spearman rank correlation coefficients and their associated probability values for the Plot Location Assignments (PLAs) and the first two DCA ordination axes for each site.

	PLA vs. DCA Axis 1	PLA vs. DCA Axis 2
HJA 1	-.1722 (.0154)*	-.0655 (.3494)
HJA 2	-.5321 (.0009)*	.3837 (.0166)*
HJA 3	.0040 (.9801)	-.0423 (.7914)
Mary's Peak	.2403 (.1386)	.4963 (.0022)*
Rooster Rock	-.5882 (.0003)*	-.0945 (.5601)
Alsea	-.3676 (.0217)*	.1049 (.5124)

* = significant at the $p \leq .05$ level.

Which plant populations are affected by P. weirii presence? Eight species from the tree, shrub, and herb strata that occurred most commonly across all sites were chosen for this analysis.

The tree species examined were Tsuga heterophylla and Thuja plicata. Pseudotsuga menziesii, the canopy species for all six sites, had significantly different cover values ($p \leq .05$) for all sites except HJA 3 ($p=.149$). This last site was different because of large Douglas-fir present directly inside the infection that were either more resistant to the root rot, were infected but still symptomless, or had been isolated in some way such that their roots were not exposed to the fungus.

Western hemlock (Tsuga heterophylla) and western red cedar (Thuja

plicata), both late successional species in these forests, generally had higher percent basal area on plots located inside the infection center (Table II - 4).

The exception to this again was HJA 3. This site had significantly higher hemlock basal area outside the infection center. Hemlock in this area was not as common as in the other old-growth Cascade sites, and was not the major replacement canopy tree at the site. Pacific yew (Taxus brevifolia) and western red cedar both had a higher average basal area (10.1 and 13.7% basal area/ha respectively for inside areas) than hemlock at this site. The unusually high cover of yew may have been due to a suppression of hemlock dominance in the area by two other native diseases at the site: a mistletoe (Arceuthobium tsugense) stunting seedlings and young trees, and white heart rot of hemlock (Phellinus hartigii) killing mature canopy trees (Boyce, 1961).

The Coast range sites had no significant differences in hemlock cover inside and outside P. weirii infection centers. Hemlock, the only replacement tree at these sites, generally had a lower relative basal area than what was found in the old-growth Cascade sites. Coastal forests tend to develop dense shrub communities, especially with disturbance (Franklin & Dyrness, 1984). Increased competition with shrubs in infection centers may have hindered hemlock establishment and growth in the Coast sites examined, as compared to establishment in the Cascade sites.

The mature site, Rooster Rock, did not have any potential canopy tree species present to replace P. menziesii.

Western red cedar had greater basal area inside infected areas for the two

Table II - 4: Average basal area (b.a.) (%) for *Tsuga heterophylla* (TSHE) and *Thuja plicata* (THPL) inside and outside *Phellinus weirii* infection centers, and the associated Mann-Whitney test statistic and probability values for these differences. Only sites in which the tree species occurred are listed.

	<u>HJA 1</u>	<u>HJA 2</u>	<u>HJA 3</u>	<u>Mary's Peak</u>	<u>Aisea</u>
<u>Average b.a. TSHE/ha (%)</u>					
inside outside	22.7 18.8	26.0 11.1	1.2 9.0	1.7 2.7	8.4 7.8
±s.e.	±2.1 ±1.6	±5.2 ±1.7	±0.6 ±1.8	±0.6 ± 0.7	±2.5 ±1.7
(sample size)	(n=73) (n=126)	(n=20) (n=20)	(n=20) (n=20)	(n=20) (n=20)	(n=20) (n=20)
T (test statistic)	2.702	2.813	17.081	3.046	.459
(p-value)	(p=.100)	(p=.094)	(p=.000)	(p=.081)	(p=.498)
<u>Average b.a. THPL/ha (%)</u>					
inside outside	-----	4.7 0.2	13.7 3.0	-----	-----
±s.e.	-----	±2.2 ±0.2	±6.6 ±1.7	-----	-----
(sample size)	-----	(n=20) (n=20)	(n=20) (n=20)	-----	-----
T (test statistic)	-----	15.779	4.389	-----	-----
(p-value)	-----	(p=.000)	(p=.122)	-----	-----

sites in which it was present, significantly higher at HJA 2 (Table II - 4).

The shrub species examined were Acer circinatum, Berberis nervosa, and Gaultheria shallon. For all of the Cascade sites, the average percent cover of vine maple was lower inside infection centers, but significantly so ($p \leq .05$) at only two sites, HJA 1 and HJA 2 (Table II - 5). The Coast range sites had a lower percent cover of vine maple on the outside of infection centers, significantly ($p \leq .05$) lower at the Alsea site and nearly significant at the Mary's Peak site ($p=.077$) (Table II - 5). The different responses of vine maple to disease presence at these two types of sites may be because replacement trees, such as hemlock with dense canopies, were not as common at the Coast sites examined. The reduced competition for the light made available by P. menziesii removal at the Coast sites may promote an increase in vine maple, whereas replacement canopy species eventually suppress vine maple inside the infection centers at old-growth Cascade sites.

With the exception of the Mary's Peak site, all sites had a higher average percent cover of Berberis nervosa outside infection centers, significantly higher ($p \leq .05$) at two sites (Table II - 5). Gaultheria shallon exhibited a more variable pattern with cover significantly higher ($p \leq .05$) outside the infection center at HJA 2, and lower at the Mary's Peak site (Table II - 5).

Differences in total shrub cover inside and outside diseased areas across all sites was not significant except at Mary's Peak, where shrub cover was significantly higher inside the infection center ($p=.015$) and at the Rooster Rock site where cover was significantly lower inside the infection center

Table II - 5: A comparison of the average cover/m² (%) for Acer circinatum (ACCI), Berberis nervosa (BENE), and Gaultheria shallon (GASH) inside and outside Phellinus weirii infection centers using the Mann-Whitney tests and the associated probability values for these differences. Dashed lines indicate the species was very rare or not present at that site.

	<u>HJA 1</u>	<u>HJA 2</u>	<u>HJA 3</u>	<u>Mary's Peak</u>	<u>Rooster Rock</u>	<u>Alsea</u>
<u>Aver. cover ACCI/m²(%)</u>						
inside outside	6.1 10.8	0.7 5.1	15.7 16.5	14.6 5.3	1.2 3.1	17.1 3.0
± s.e.	±1.3 ±1.4	±0.3 ±1.2	±3.5 ±3.3	±3.2 ±1.3	±0.8 ±1.1	±4.8 ±1.4
(sample size)	(n=73) (n=126)	(n=20) (n=20)	(n=20) (n=20)	(n=20) (n=20)	(n=19) (n=20)	(n=20) (n=20)
T (test statistic)	4.267	11.940	.036	3.127	3.071	6.775
(p-value)	(p=.039)	(p=.001)	(p=.850)	(p=.077)	(p=.080)	(p=.009)
<u>Aver. cover BENE/m²(%)</u>						
inside outside	8.3 12.4	1.7 4.5	4.3 8.1	16.6 13.5	2.7 34.1	0.5 3.3
± s.e.	±1.7 ±1.7	±0.7 ±1.8	±1.4 ±2.2	±2.8 ±3.9	±1.1 ±8.2	±0.5 ±1.4
(sample size - see above)						
T (test statistic)	1.818	1.738	2.076	1.692	11.087	4.174
(p-value)	(p=.178)	(p=.187)	(p=.150)	(p=.193)	(p=.001)	(p=.041)
<u>Aver. cover GASH/m²(%)</u>						
inside outside	-----	0 1.7	11.5 4.5	22.8 8.0	5.6 6.6	7.4 12.2
± s.e.	-----	±0 ±1.7	±4.4 ±1.5	±7.3 ±4.9	±3.5 ±3.1	±2.3 ±4.2
(sample size - see above)						
T (test statistic)	-----	8.177	.066	9.000	.427	.053
(p-value)	-----	(p=.004)	(p=.800)	(p=.003)	(p=.514)	(p=.817)

($p=.009$). Overall, the three shrub species had variable responses to disease presence. A. circinatum was probably the most sensitive to disease presence, Berberis nervosa exhibited a general trend across sites, and Gaultheria shallon seemed little influenced by P. weirii presence.

The herbs that were most common in the sites examined were Polystichum munitum, Linnaea borealis, and Coptis laciniata. Each of these herb species had significantly higher cover inside infection centers at only one of the six sites ($p \leq .05$). A statistically non-significant trend of higher average percent cover inside infected areas for the the three species was exhibited across most sites (Table II - 6).

The total percent cover of all the herb species across all sites was generally higher inside infection centers (Fig. II - 1), significantly higher ($p=.000$) at only at the Rooster Rock site. Because the edge areas of HJA 1 and HJA 3 had been determined earlier to be 'unique' areas, all comparisons were made utilizing the three category scheme, described previously, as well as the two category scheme reported thus far. Herb cover at HJA 1 showed significant differences in the three category sampling scheme in which the edge area is distinct from the area removed from the infection center ($p < .001$). In this case herb cover was much lower (8%/plot) in the edge area as compared to either outside or inside areas (13% and 17%/plot respectively.)

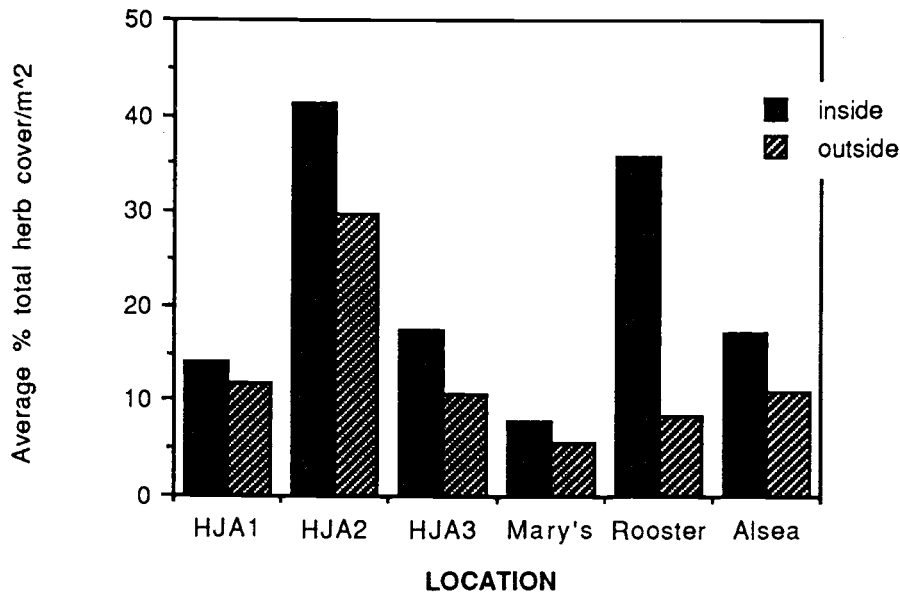
In general, the response of plant populations to the gradual overstory removal of Douglas-fir by P. weirii is species specific and often depends on site

Table II - 6: A comparison of the average cover/m² (%) for Coptis laciniata (COLA), Linnaea borealis (LIBO), and Polystichum munitum (POMU) inside and outside Phellinus weirii infection centers using the Mann-Whitney tests and the associated probability values for these differences. Dashed lines indicate the species was either very rare or not present at that site.

	<u>HJA 1</u>	<u>HJA 2</u>	<u>HJA 3</u>	<u>Mary's Peak</u>	<u>Rooster Rock</u>	<u>Alsea</u>
<u>Aver. cover COLA/m²(%)</u>						
inside outside	8.9 8.0	1.2 4.2	-----	-----	-----	-----
± s.e.	±1.6 ±1.2	±0.5 ±1.3	-----	-----	-----	-----
(sample size)	(n=73) (n=126)	(n=20) (n=20)	-----	-----	-----	-----
T (test statistic)	.043	6.454	-----	-----	-----	-----
(p-value)	(p=.836)	(p=.011)	-----	-----	-----	-----
<u>Aver. cover LIBO/m²(%)</u>						
inside outside	1.5 0.7	2.0 5.1	10.5 6.3	-----	0.7 0.1	-----
± s.e.	±0.3 ±0.2	±0.4 ±2.0	±5.2 ±1.7	-----	±0.4 ±0.1	-----
(sample size)	see above	see above	(n=20) (n=20)	-----	(n=19) (n=20)	-----
T (test statistic)	20.208	.023	.042	-----	2.396	-----
(p-value)	(p=.000)	(p=.880)	(p=.838)	-----	(p=.122)	-----
<u>Aver. cover POMU/m²(%)</u>						
inside outside	1.0 0.6	24.8 4.6	2.9 1.4	6.4 5.0	-----	15.6 10.3
± s.e.	±0.4 ±0.3	±6.1 ±2.8	±2.1 ±1.0	±2.7 ±2.6	-----	±5.4 ±5.5
(sample size)	see above	see above	see above	(n=20) (n=20)	-----	(n=20) (n=20)
T (test statistic)	1.575	11.596	0.758	0.588	-----	1.818
(p-value)	(p=.209)	(p=.001)	(p=.383)	(p=.443)	-----	(p=.178)

characteristics, as was the case for vine maple.

Figure II - 1: Average % cover of all herbaceous species inside and outside Phellinus weirii infection centers.



Does the diversity of plant species differ between infected and non-infected areas? The old-growth Cascade sites, HJA 2 and 3, exhibited a trend toward decreased species diversity in the infected forest (Table II - 7), with diversity significantly lower at HJA 2 ($p=.039$), and nearly significant at HJA 3 ($p=.097$). The mature stand at Rooster Rock and the Mary's Peak site both had significantly higher diversity in the infected area of the forest ($p < .005$). HJA 1 and the Alsea site had no significant differences in vascular plant diversity inside and outside infection centers ($p > .25$).

Table II - 7: Hill's diversity index inside and outside infection centers for all sites. Shown also are the results of an unpaired t-test used to test if the diversity between these two areas is significantly different from random.

	Hill's Diversity Index (species)		F test statistic (p-value)
	inside	outside	
HJA 1	3.15	2.90	F=1.288 (p=.258)
HJA 2	3.46	4.51	F=4.574 (p=.039)*
HJA 3	3.75	4.47	F=2.895 (p=.097)
Mary's Peak	3.89	2.74	F=9.682 (p=.004)*
Rooster Rock	4.10	2.43	F=12.823 (p=.001)*
Ailea	2.63	2.55	F= .084 (p=.777)

* = significant at $p \leq .05$ level.

These results exemplify how the population responses are site-dependent. Similar species occurred across all sites and yet diversity is enhanced at one center in the Coast range and in the mature stand, and is decreased in two of the old-growth Cascade sites. Late successional species were not common in the Coast range stands, thus understory growth increased. Late-successional species increased with disease presence in the old-growth Cascade sites, decreasing understory diversity.

Though the analogy has been made between host-specific diseases and predators that act to diversify community structure (Harper, 1977), this does not seem to be the case for *P. weirii* in the Cascade sites. Diversity here did not significantly increase with disease presence. Processes affecting species diversity perhaps should be examined in terms of the interactions between specific disturbance regimes and specific species life histories before such

generalizations are made.

What are the possible mechanisms by which disease presence induces species compositional changes? The only direct effect Phellinus weirii has on the forest community is the selective mortality of Douglas-fir. However, the results presented here show that this effect has far-reaching consequences throughout the plant community. The initial hypothesis was that a change in canopy cover induced the compositional changes. Canopy cover was expected to be higher inside of infection centers because canopy of the late successional species that would replace P. menziesii would have denser, lower canopies (Grier & Logan, 1977). However, only the Rooster Rock site had significant differences in canopy cover ($p=.019$), and this difference was slight, with 76% cover inside the infection center and 81% outside. This mature stand did not have any potential replacement canopy tree species present, such as hemlock or cedar, thus canopy cover was not expected to be higher inside this infection center.

An infection center is composed of a somewhat orderly series of single, sometimes multiple, tree-gaps that have occurred over a long period of time in one location. Of gap studies done in old-growth forests of the Pacific Northwest, single tree-gaps have been found to have very little effect on the light regime of the understory. This is because of the high ratio of canopy height to gap diameter (Canham et al., 1990) and the high degree of crown overlap (15-30%) of dominant Douglas-fir with the subdominant canopy species (Spies et al., 1990). Only the simultaneous death of 5 to 10 trees, rare in

P. weirii infection centers, would create a gap large enough for high light intensities at ground level (Spies et al., 1990). Insignificant canopy results may also be because densimeters tend to over-estimate percent cover in canopies that have many gaps (Bunnell & Vales, 1990).

Pure canopies of late successional species such as hemlock and cedar do have less direct and diffuse radiation that reaches the forest floor over the growing season as compared to a pure Douglas-fir canopy (Stewart, 1988). A decreased understory diversity and development of herbs, shrubs, and regenerating tree species found under hemlock canopies have been attributed to a poor lighting regime associated with hemlock canopies (Stewart, 1986; 1988).

There are alternate hypotheses however. Because tree species vary in root uptake and exudate, the nutrients and microbial communities beneath different canopy species can vary significantly (Collins & Good, 1986; Turner & Franz, 1985). In old-growth hemlock-cedar forests of northern Idaho, there is strong microsite differentiation between cedar and hemlock (Turner & Franz, 1985). That understory cover was less diverse under hemlock as compared to cedar was attributed to lower pH, fewer nitrifying bacteria, and less available calcium (Turner & Franz, 1986), rather than differences in the light regime.

If the quantity or quality of light, and/or soil changes beneath different canopies was what elicited the change in the understory of infection centers, infection centers should possess a different canopy composition than uninfected areas.

All of the old-growth Cascade sites examined do have at least one replacement canopy species that has significantly higher basal area inside infection centers. HJA 1, a site which was determined previously to have an edge area composition unique to the non-infected forest, had significantly more hemlock in the center (20.5%) and on the edge (25.3%) of the infection center as compared to outside (16.7 %) ($T=10.717$, $p=.005$). HJA 2 had significantly more western red cedar and nearly significantly more hemlock inside infection centers as well (Table II - 4). Cedar and yew had higher basal area inside the infection center at HJA 3, yew basal area significantly higher inside ($T=7.471$, $p=.006$). All sites had significantly higher Douglas-fir relative basal area outside infection centers ($p \leq .05$). The exception is HJA 3 ($p=.149$) which contained three large symptomless Douglas-fir inside the infection center.

The mature stand at Rooster Rock did not have any hemlock or red cedar of significance present, but changes in composition at this site can be explained by the significant differences in canopy cover.

The Coast range sites, Mary's Peak and Alsea, did not have significant changes in hemlock basal area (Table II - 4), the only late successional species found at these sites. Western hemlock had a lower average cover at these sites as compared to the old-growth Cascade sites (Table II - 4). Though the elimination of Douglas-fir from the canopy favors hemlock replacement, the growth of shrub species seem to be favored more at these sites. The major shrub species at these sites: Acer circinatum, Holodiscus discolor, and Corylus

cornuta, all had significantly higher basal area/ha (%) ($p < .01$, Mann-Whitney tests), with the exception of vine maple basal area at the Mary's Peak site which was nearly significantly inside ($T=3.127$, $p=.077$).

Shrub species in old-growth Coast range forests generally form dense covers, especially in response to disturbance (Franklin & Dyrness, 1984). Because of hemlock's limited regeneration niche, on nurse logs and not on the soil itself (Christy & Mack, 1984), the thick shrub cover inside infection centers at the Coast sites may present a formidable barrier to its re-establishment.

Conclusions

This study has shown that the long-term presence of laminated root rot in old-growth and mature Douglas-fir stands of the Pacific Northwest can significantly impact community composition. The important point is not so much what the specific species changes are, as these differ from site to site, but that it is possible for a disease that affects one species to have a cascading effect throughout various populations in these multi-strata forests.

Results indicate that species response is highly species-specific and dependent on site characteristics, thus making it difficult for generalizations to be made. Some species, such as vine maple, were quite sensitive to the disturbance induced by the disease; cover increased with disease in the Coast sites and generally decreased inside infection centers of the old-growth Cascade sites. Other species, such as salal (Gaultheria shallon), generally did

not exhibit cover differences in response to disease.

Responses of herbs in particular to canopy gaps has been found before to be species-specific (Collins & Pickett, 1988), often associated with increased site availability due to tip-up mounds, or some sort of soil disturbance (Beatty, 1984) which are common in infection centers. Response to P. weirii disturbance was largely a reorganization of populations already present on the site, rather than the establishment of new species. This 'reorganization' response has been found before in openings caused by gypsy moths in eastern mature oak forests (Ehrenfeld, 1980).

The life histories of fast colonizing or strongly competitive species may reflect a natural disturbance regime for a particular community (Miller, 1982). However, the question of whether species have discrete regeneration niches to particular disturbance regimes remains largely unanswered (Pickett & White, 1985). It is interesting to note that species such as hemlock and cedar, which are generally favored by the disturbance generated by P. weirii at the Cascade sites, do have life history characteristics amenable to this disturbance regime; both are tolerant of infection, can survive long periods of suppression, and regenerate on decaying wood. Before examining species' responses to disturbance, taking into consideration the nature of the disturbance itself and how it fits into community processes will provide a stronger foundation for any study.

On a community level, disturbance frequency (Denslow, 1985; Runkle & Yetter, 1987), size (Miller, 1982), and severity (Runkle, 1985) can all play important roles in determining the types of species able to grow or establish

after disturbance. As a disturbance agent, Phellinus weirii is unique compared to the types of disturbance induced by fire, windthrow, or other root and butt diseases present in the old-growth Douglas-fir forests. The frequency of encountering this type of disturbance is high and because it expands at a known rate, an average of 30 - 40 cm/ year (Childs, 1970; Cook, 1982; Nelson & Hartman, 1975), its distribution in an area is predictable through time.

The intensity of disturbance by P. weirii is generally low since the slow spread of the fungus across root contacts leads to individual tree deaths scattered over time. Damage can be more intense if combined with winter storms, which can topple trees at varying stages of decay (pers. observ.). The size of areas affected by this root rot may be quite large. Eight-hundred year old fungal clones spanning hundreds of meters are not uncommon in the Mountain Hemlock zone (Copsey, 1985). Thus, P. weirii, and possibly other root rot diseases, possess unusual combinations of disturbance size, intensity, and frequency.

Because it is unlike other disturbance agents, the composition changes associated with P. weirii can be expected to be quite different on spatial and temporal scales from other types of disturbances the community may experience. The non-competitive, selective mortality associated with this disturbance may also play an important role in the maintenance of species equilibria in communities (see Petraitis et al., 1989).

On an ecosystem level, it would be interesting to know if the presence of the disease is able to produce a steady-state shifting mosaic, as is suggested in the mountain hemlock ecosystem (Copsey, 1985). A steady-state shifting mosaic is composed of a patchwork of disturbances that vary on spatial and

temporal scales across a landscape but which together constitute an equilibrium (Bormann & Likens, 1979). Characteristic disturbances that would shape such shifting mosaics in steady state systems would be small and frequent in an otherwise homogeneous area, and their frequency would be regulated in part by the community affected (Pickett & White, 1985).

An examination into disturbances that would create such mosaics may prove fruitful if natural disease agents such as P. weirii are examined. This type of disturbance is small, frequent, predictable, and it does affect community compositional structure. There is also a feedback mechanism implicated in the root rot's frequency. Dickman and Cook (1989) state that in the Mountain Hemlock zone P. weirii frequency is in part regulated by the fire frequency, which is determined in part by the compositional structure that has been altered by the root rot. Also, the enhanced tree species diversity created by the presence of the disease in the high Cascades may act as a feedback mechanism slowing reinfestation (MacCauley & Cook, 1980). Whether such mechanisms are present in the Douglas fir forests is unknown. Studies on the distribution of root rot pockets across landscapes (pockets are visible in aerial photographs (Martin & Williams, 1986)) over long time periods would be useful to our understanding of possible impacts of disease on an ecosystem level.

CHAPTER III. IMPACTS OF PHELLINUS WEIRII ON SUCCESSION**Abstract**

This study examines the effects that a native root-rotting pathogen has on succession in old-growth and mature Douglas-fir forests of the Pacific Northwest. Phellinus weirii (Murrill) Gilbrt. (Class: Basidiomycete, Family: Hymenochaetaceae) is a fungal pathogen that causes a white rot in the roots and boles of many conifers, a rot that eventually leads to mortality in Douglas-fir, mountain hemlock, and true firs. The pathogen spreads slowly via root-grafts or root contacts, and single fungal clones may affect areas of a hectare or more. Affected areas are commonly called infection centers.

The disease was determined previously to have a significant impact on the composition of six infection centers located in the lower Cascade Mountains and Coast Ranges of western Oregon (see Chapter 2). Douglas-fir was the sole species directly affected by P. weirii.

To assess current impacts of the disease on succession, the basal area of late-successional tree species and common shrubs were compared inside and outside infection centers. To determine the future successional impacts within infection centers, the abundance of regenerating tree species was examined with respect to disease presence. The initial hypothesis was that infected areas would appear to have their successional clock 'pushed forward' as disease eliminated Douglas-fir, a seral dominant, favoring late-successional species.

Results suggest that disease presence promoted late successional species to canopy status in the three old-growth Cascade sites. This was not the case for the Coast Range sites; shrub growth was strongly favored inside infection centers, whereas the growth of late successional species seemed relatively unaffected. Late successional species were rare at the Cascade mature stand.

Future successional impacts caused by P. weirii are likely to be minimal, as tree seedling establishment inside and outside infection centers was not significantly different for any species at any site. The presence of western hemlock appears to be a more important factor determining tree seedling establishment and survival, since seedling abundance and hemlock basal area were negatively correlated across most sites.

Introduction

Many forest tree species, especially late-successional species, are dependent on small scale disturbances, such as canopy tree gaps, in order to attain canopy status (Barden, 1989; Canham, 1989; Spies et al., 1990; Wilson, 1990; Woods, 1984). Though few studies examine the etiology of gaps in forest canopies, naturally occurring diseases may play an important role in their initiation (Menges & Loucks, 1984; Worrall & Harrington, 1988).

Phellinus weirii (Murrill) Gilbrt., a native root-rotting pathogen of many coniferous hosts, was found in earlier studies to redirect successional pathways in old-growth mountain hemlock forests of the high Cascades in western Oregon (Cook et al., 1989; Copsey, 1985). Infection centers in these forests appear similar to communities present after a disturbance by fire (Dickman & Cook, 1989). However, the increased heterogeneity of sizes and

ages of tree species associated with these disturbances persists in P. weirii infected areas and infection centers contain some tree species not usually found after fire. The successional pathways of infected and non-infected communities do not converge even after 400 years (Copsey, 1985).

The tree species most susceptible to the root rot in the high Cascade area is mountain hemlock, a late-successional species. Douglas-fir (Pseudotsuga menziesii), an early successional tree species, is also highly susceptible to P. weirii root rot and is found throughout the western montane regions of North America. Douglas-fir reaches its peak abundance in the Tsuga heterophylla Zone, the most extensive vegetation zone in Oregon and Washington, extending from southern British Columbia, through the Olympic Peninsula, to the Cascade and Coast Ranges of western Oregon (Franklin & Dyrness, 1984). This study examines whether or not the presence of P. weirii affects successional pathways in this zone, an area where Douglas-fir is the major seral tree species.

Because P. weirii was directly affecting a major seral dominant in this zone, western hemlock (Tsuga heterophylla) and western red cedar (Thuja plicata), the late successional or "climax" species, would be favored. Both of these late-successional species are shade-tolerant and often present during early succession (Barbour & Billings, 1988). Their survival and growth are highly dependent on canopy openings (Spies et al., 1990). The presence of the root rot would provide regular openings of the Douglas-fir canopy, as well as providing the woody substrate that western hemlock prefers to establish on (Christy & Mack, 1984). Douglas-fir has low regenerating capacity in these

forests without very large openings (Spies et al., 1990) and young trees would eventually become infected with P. weirii if regeneration did take place.

Thus, though disturbance is often viewed as an interrupter of some pre-ordained successional path (Levin & Paine, 1974; Pickett & White, 1985), I hypothesized that the succession should be accelerated with disease present in the Tsuga heterophylla zone. Greater basal area and regeneration of late-successional species was expected inside the infected area, as compared to the adjacent non-infected forest.

Materials and Methods

See the previous chapter for site locations, characteristics, and sampling strategy.

Data collection. At each random plot, the diameter at breast height (dbh) of all tree species was measured within a 6m radius (4m at the Mary's Peak site) of the plot center. The number of tree seedlings were recorded within this area, a seedling being defined as any tree with a dbh < 1cm.

Statistical analyses. The basal area (m^2/ha) for each late successional tree species and common shrub species and tree seedling abundance were compared inside and outside of each sites' infection center using Mann-Whitney tests, a non-parametric one-way analysis of variance by rank (Conover, 1980).

Results and Discussion

Impacts of Phellinus weirii on current succession. The three old-growth Cascade sites, HJA 1, HJA 2 and HJA 3, exhibited a trend of higher basal area of late successional species inside infection centers (Table III - 1). At the Coast Range sites, Mary's Peak and the Alsea site, western hemlock, the only late-successional present in the sample plots, did not differ inside and outside then infection center. Hemlock was the only late-successional species present in the plots of the mature stand at Rooster Rock, but occurred too infrequently to statistically test for differences inside and outside the infection center. Acer macrophyllum (big leaf maple) was a more common tree species found in the sample plots at Rooster Rock and its abundance was not significantly affected by the disease (Mann-Whitney test, $p=0.67$).

Western red cedar (Thuja plicata) had a higher basal area inside infection centers at both sites where it occurs, significantly higher ($p \leq 0.05$) at HJA 2 (Table III - 1).

Pacific yew (Taxus brevifolia) had a significantly higher basal area inside the infection center at HJA 3 and significantly lower basal area at HJA 2 (Table III - 1). An explanation for these conflicting results may lie in the variation of hemlock cover at the two sites. In addition to the elimination of Douglas-fir by P. weirii at HJA 3, hemlock was also being suppressed in this area by mistletoe (Arceuthobium tsugense), as well as a native heart rot, Phellinus hartigii (see Boyce, 1961). However, hemlock was the most common species at HJA 2 and its growth appeared to be favored with the

Table III - 1: A comparison of the average basal area (m^2/ha) of Tsuga heterophylla (TSHE), Thuja plicata (THPL), and Taxus brevifolia (TABR) inside and outside Phellinus weirii infection centers using the Mann-Whitney tests and the associated probability values for these differences. The Rooster Rock site is not included because of the paucity of late successional species at that site. Dashed lines indicate the species was absent or rare at that site. Total basal area results are for late-successional tree species present only.

Table III - 1.

	HJA 1	HJA 2	HJA 3	Mary's Peak	Alsea
Average b.a. TSHE (m²/ha)					
inside outside	32.5 26.8	37.2 15.8	1.8 12.9	2.5 3.8	12.1 11.2
± s.e.	±3.0 ±0.9	±7.5 ±2.5	±0.9 ±2.6	±0.9 ±0.9	±3.5 ±2.5
(sample size)	(n=73) (n=126)	(n=20) (n=20)	(n=20) (n=20)	(n=20) (n=20)	(n=20) (n=20)
T (test statistic)	2.70	2.81	12.85	3.05	0.46
(p-value)	(p=0.10)	(p=0.09)	(p=0.00)	(p=0.08)	(p=0.50)
Average b.a. THPL (m²/ha)					
inside outside	-----	6.7 0.3	19.8 4.2	-----	-----
± s.e.	-----	±3.2 ±0.3	±9.5 ±2.4	-----	-----
(sample size - see above)					
T (test statistic)	-----	15.78	1.47	-----	-----
(p-value)	-----	(p=.00)	(p=.48)	-----	-----
Average b.a. TABR (m²/ha)					
inside outside	-----	0.1 0.7	14.6 6.3	-----	-----
± s.e.	-----	±.05 ±0.2	±2.2 ±1.8	-----	-----
(sample size - see above)					
T (test statistic)	-----	6.167	7.47	-----	-----
(p-value)	-----	(p=.013)	(p=.01)	-----	-----
Total tree b.a. (m²/ha)					
inside outside	32.5 26.8	44.1 16.8	36.1 23.5	2.5 3.8	12.1 11.2
± s.e.	±3.0 2.3	±6.8 ±2.5	±9.0 ±3.9	±0.9 ±0.9	±3.5 ±2.5
(sample size - see above)					
T (test statistic)	2.70	8.30	0.35	3.05	0.46
(p-value)	(p=0.10)	(p=0.00)	(p=0.55)	(p=0.08)	(p=0.50)

removal of Douglas-fir by P. weirii. Hemlock with its dense canopy (Grier & Logan, 1977) may be inhibiting yew growth inside the infection center at HJA 2, whereas yew growth is stimulated at HJA 3 with hemlock suppression in the vicinity of the infection center.

Why the hemlock at the Coast Range sites did not respond as it did at the Cascade sites is not exactly clear. Shrub growth increased dramatically with respect to P. weirii presence for both of the Coast sites, whereas shrub cover was usually higher outside infection centers at the Cascade sites (Table III - 2). Because this type of disturbance appears to strongly favor the growth of shrubs in the Coast Range, the establishment and growth of hemlock may be inhibited inside infection centers at these sites. The lower elevation and milder climate at the Coast sites compared to sites in the Cascades are possible explanations for the differing shrub responses.

It is interesting that Phellinus weirii has different impacts in different environments. In the mountain hemlock areas of the high Cascades, P. weirii presence induces a composition resembling the early stages of succession (in areas where Abies amabilis is absent), a composition indicative of later succession sequences is associated with P. weirii presence in the Tsuga heterophylla zone of the lower Cascades, and little overall effect seems to take place in the Coast sites examined. The varying P. weirii responses in the Douglas-fir and the mountain hemlock old-growth are probably due to a difference in host species. In one case a seral dominant is eliminated, while in the other a 'climax' species is being eliminated. Differences in response between the Cascade and Coast sites may be due to

Table III - 2: A comparison of the average basal area (m^2/ha) of Acer circinatum (ACCI), Corylus cornuta (COCO), Holodiscus discolor (HODI), and Rhododendron macrophyllum (RHMA) inside and outside Phellinus weirii infection centers using the Mann-Whitney tests and the associated probability values for these differences. Dashed lines indicate that the species was rare or not present for that site. Total shrub cover results are for arborescent species only.

Table III - 2.

	HJA 1	HJA 2	HJA 3	Rooster Rock	Mary's Peak	Alsea
<u>Aver. b. a. ACCI (m²/ha)</u>						
inside outside	0.5 0.9	0.1 0.4	1.3 1.3	0.1 0.2	1.2 0.4	1.4 0.2
± s.e.	±0.1 ±0.1	±0.02 ±1.0	±0.3 ±0.3	±0.1 ±0.1	±0.3 ±0.1	±0.4 ±0.1
(sample size)	(n=73) (n=126)	(n=20) (n=20)	(n=20) (n=20)	(n=19) (n=20)	(n=20) (n=20)	(n=20) (n=20)
T (test statistic)	4.27	11.94	0.36	3.07	3.13	6.78
(p-value)	(p=.007)	(p=.001)	(p=.085)	(p=.080)	(p=.077)	(p=.009)
<u>Aver. b.a. COCO (m²/ha)</u>						
inside outside	-----	-----	-----	-----	0.7 0.01	-----
± s.e.	-----	-----	-----	-----	±0.2 ±0.003	-----
T (test statistic)	-----	-----	-----	-----	11.67	-----
(p-value)	-----	-----	-----	-----	(p=.001)	-----
<u>Aver. b.a. HODL (m²/ha)</u>						
inside outside	-----	-----	-----	-----	0.5 0.1	-----
± s.e.	-----	-----	-----	-----	±0.09 ±0.05	-----
T (test statistic)	-----	-----	-----	-----	20.15	-----
(p-value)	-----	-----	-----	-----	(p=.000)	-----
<u>Aver. b.a. RHMA (m²/ha)</u>						
inside outside	-----	0.1 0.1	-----	0.21 0.20	-----	-----
±s.e.	-----	±0.04 ±0.04	-----	±0.1 ±0.1	-----	-----
T (test statistic)	-----	5.97	-----	0.34	-----	-----
(p-value)	-----	(p=.015)	-----	(p=0.560)	-----	-----
<u>Aver. b.a. total shrubs (m²/ha)</u>						
inside outside	0.5 0.9	0.1 0.5	1.26 1.32	0.3 0.4	2.4 0.5	1.4 0.2
± s.e.	±0.1 ±0.1	±0.04 ±0.1	±0.3 ±0.3	±0.1 ±0.1	±0.4 ±0.1	±0.4 ±0.1
T (test statistic)	4.27	8.92	0.36	0.41	16.25	6.78
(p-value)	(p=.007)	(p=.003)	(p=.085)	(p=.524)	(p=.000)	(p=.009)

environmental factors.

The presence of the disease at the Cascade old-growth sites appears to support the hypothesis that the rate of succession is enhanced at these sites because *P. weirii* removal of Douglas-fir canopy has promoted the growth of the late successional subcanopy to canopy status. That the growth of these species is increased with the removal of Douglas-fir is not surprising, since canopy gaps have been implicated in playing important roles in late-successional species dominance and successional trends (Spies et al., 1990). However, the changes in species dominance are not taking place in isolated, single treefall incidents, but across large areas over long periods of time. In terms of understanding successional changes in old-growth systems generally, this disturbance agent needs to be recognized for the Cascade forests. *P. weirii* is common, occurs throughout the *Tsuga heterophylla* zone, and is present for long periods of time in any one location. Because the disease is not a stochastic disturbance agent and its presence can be readily recognized with some training, modelling the effects of this disturbance agent across a landscape is possible. Such an approach could be useful in answering the question whether or not shifting steady-state mosaics exist (see Bormann & Likens, 1979).

Impacts of Phellinus weirii on future successional patterns. Seedling abundance was not significantly different inside and outside of infection centers for any of the late successional species at any site ($p > .05$) (Table III - 3). With the exception of hemlock at a few sites, seedlings were generally infrequent. With the exception of a few scattered seedlings at a few sites,

Table III - 3: Number of seedlings found inside and outside Phellinus weirii infection centers are shown for each site accompanied by the results of Mann-Whitney tests used to statistically test for differences in seedling abundance between the two areas.

Table III - 3.

	HJA 1	HJA	HJA 3	Rooster Rock	Mary's Peak	Alsea
# of seedlings of:						
<i>Tsuga heterophylla</i>						
inside outside	82 135	68 46	32 27	1 3	0 7	20 13
sample size	(n=126) (n=73)	(n=20) (n=20)	(n=20) (n=20)	(n=19) (n=20)	(n=20) (n=20)	(n=20) (n=20)
T (test statistic)	0.19	0.02	1.67	0.33	2.05	2.68
(p-value)	(p=.666)	(p=.899)	(p=.200)	(p=.563)	(p=.152)	(p=.100)
<i>Thuja plicata</i>						
inside outside	-----	12 2	14 5	-----	-----	-----
T (test statistic)	-----	2.78	0.80	-----	-----	-----
(p-value)	-----	(p=.095)	(p=.372)	-----	-----	-----
<i>Taxus brevifolia</i>						
inside outside	1 8	-----	20 3	-----	-----	-----
T (test statistic)	NA ¹	-----	3.05	-----	-----	-----
(p-value)	-----	-----	(p=.081)	-----	-----	-----
<i>Pseudotsuga menziesii</i>						
inside outside	-----	1 1	-----	0 1	4 0	-----
T (test statistic)	-----	NA	-----	NA	NA	-----
¹ = species occurred too infrequently to test statistically.						

Douglas-fir was not regenerating.

Earlier studies done in similar Douglas-fir stands show hemlock regeneration to be greatest under canopy openings (Spies et al., 1990). Though infection centers are a collection of Douglas-fir canopy openings that have taken place over hundreds of years, the canopy cover between infected and non-infected forests appears similar at any single point in time. None of the sites examined, with the exception of the mature stand, had significant differences in canopy cover inside and outside infection centers (Mann-Whitney tests, $p > .05$).

Hemlock regeneration was negatively correlated with hemlock basal area for three out of the four sites where hemlock regeneration was present in the sample plots (Fig. III - 1). For only one of these sites, HJA 1 (Fig. III - 1A) was this relationship was significantly negative (Spearman's Rank Correlations, $r_s = -.27$, $p = .000$).

One hypothesis that may explain this relationship is that hemlock's dense canopy, which tends to harbor depauperate plant assemblages generally (Stewart 1988), is shading out its own regeneration. A comparison of hemlock basal area and canopy cover for the five sites in which hemlock is common suggests this may be the case (Fig. III - 2). The canopy cover for all sites was positively related to hemlock basal area, significantly related at HJA 1 (Fig. III - 2A) and the Alsea site (Fig. III - 2E). A trend of this type was not observed for the other late successional species present.

Thus the regeneration of late-successional species may be more strongly influenced by quantity of light than by disease presence directly, a variable in

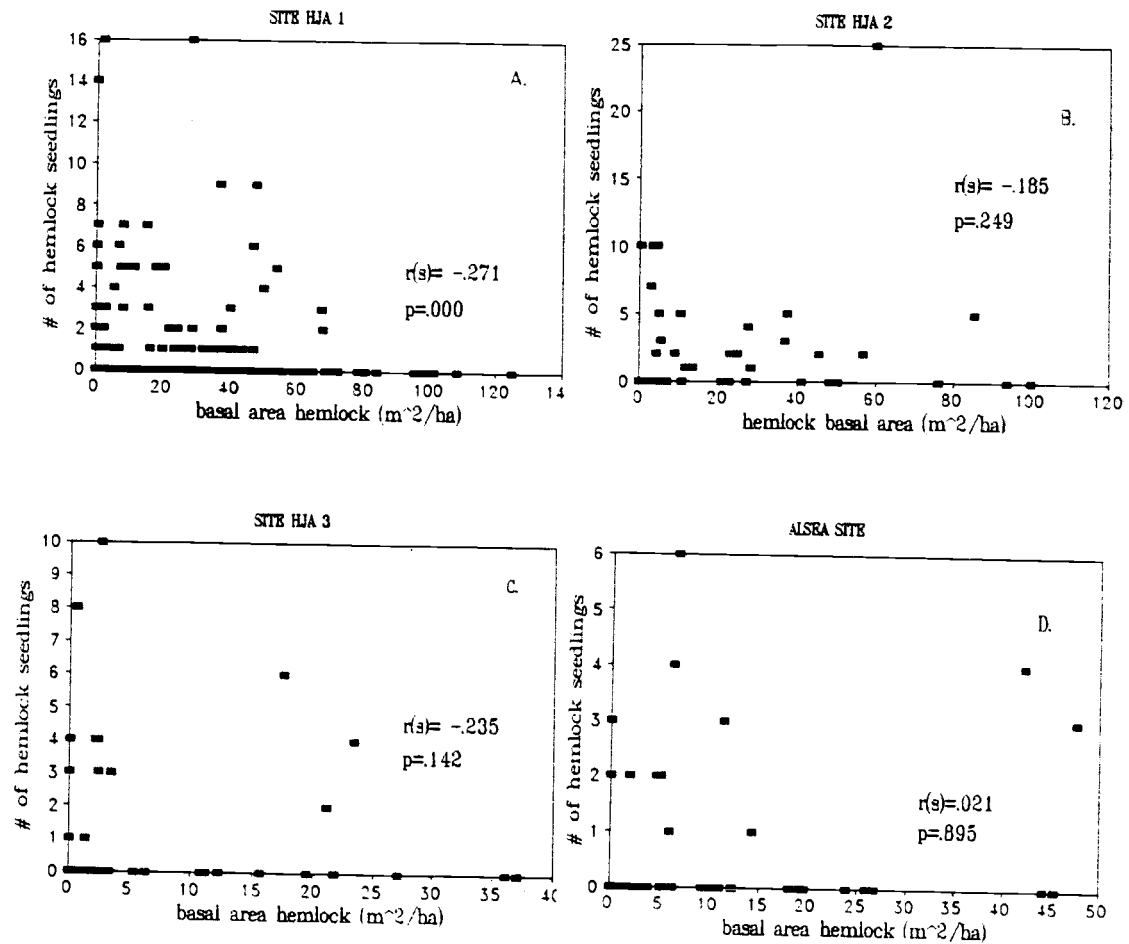


Figure III - 1: Regeneration of western hemlock in relation to the amount of hemlock basal area found for each sample in HJA 1 (A), HJA 2 (B), HJA 3 (C), and the Alsea site (D). Spearman's correlation coefficients and the associated probability values for the relationships are given for each.

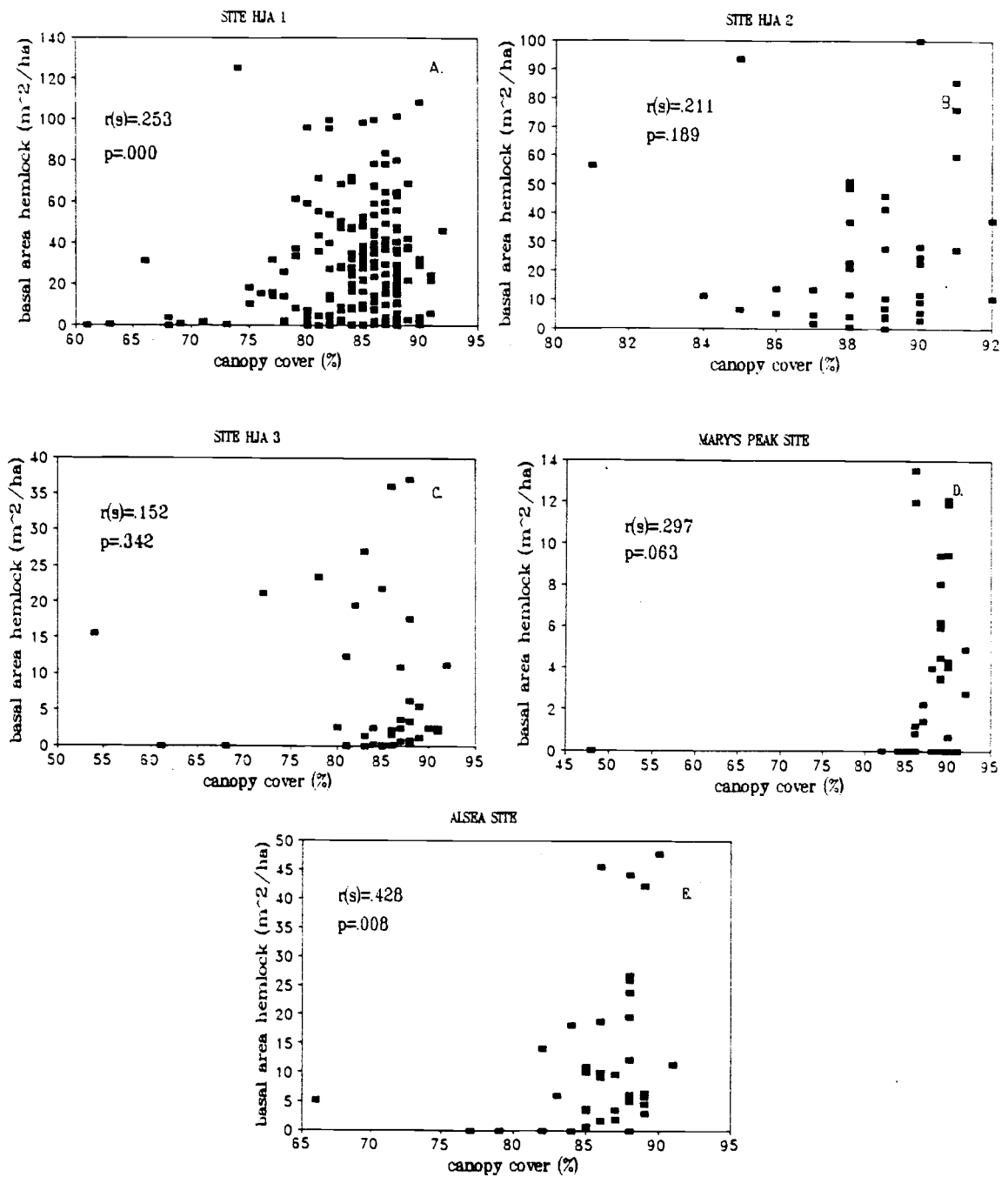


Figure III - 2: The basal area of western hemlock (m²/ha) found on each plot for HJA 1 (A), HJA 2 (B), HJA 3 (C), the Mary's Peak site (D), and the Alsea site (E) plotted against the canopy cover (%) found for that plot. Spearman's correlation coefficients and the associated probability values for the relationships are given for each.

influenced by hemlock presence. Regeneration of canopy species in similar Douglas-fir old-growth forests was found primarily in light gaps (Spies et al., 1990). Disease presence does, however, seem to influence the growth of the late-successional species once established in the Cascade old-growth forests.

CHAPTER IV. CONCLUDING REMARKS

Research Implications

Results of this research show that compositional response to disease presence depends both on the species involved and site characteristics of the infection center. The influence of disease on species diversity and succession also depends on site characteristics. The specifics of what changes in the community due to the presence of Phellinus weirii are interesting, but this was not the principal purpose of my thesis. That it is possible for the long-term presence of a native disease in an area to change species composition, diversity, and successional fate is the most important conclusion made from this project.

Though disease is paid lip-service in the ecological literature as being an important forest disturbance agent, it hasn't gotten the sort of attention it deserves. Some pathologists believe that a tree never simply dies; some biotic agent either kills it, or weakens it so that the tree is more susceptible to mechanical damage. The opposite is also true; a tree damaged mechanically may become more susceptible to disease. The view of forest gaps and tree mortality as unpredictable across a landscape may not always be sound if disturbances are commonly biotic in nature. Though the initial distribution of infection centers across a landscape may be random, subsequent spread of the disease through the forest is not random. In general, mortality due to root rots, beetle-vectored diseases, and diseases with steep spore dispersal gradients are probably not stochastic, though this

will depend on one's spatial scale. Damage by insects is also often closely associated with tree disease (see Goheen & Hansen, unpubl.) as is mortality due to windthrow (Hansen, pers. observ.). Thus small forest disturbances that on first appearance seem to be random and abiotically caused may have been wholly or partially biotically caused and their occurrence may have been predictable given some knowledge of the biotic interactions involved.

This view of disturbance caused by biotic agents has important ramifications if one is using disturbance to help predict forest composition changes through time. Incorporating disturbance into forest composition models can be used to explore future, as well as past, successional patterns, to predict timber volume over time for management practices, and to test hypotheses on how varying disturbance characteristics affect composition through time. If biotic influences, such as root disease, play a role in the forested ecosystem, the size, frequency, and intensity of the disturbance variable(s) could be realistically estimated. Models would then gain in their accuracy, and thus predicting power.

Today there is renewed interest in old-growth forests of the Pacific Northwest because it is an endangered ecosystem and so little is known about the factors that are important to the system's maintenance. When attempting to make long-term predictions on the fate of old-growth Douglas-fir forest composition, *P. weirii* should be taken into account. Herb, shrub, and tree composition, successional direction, and vascular plant diversity can all be influenced, as shown in this study, by its presence.

Perhaps the examination of other biotic disturbance agents that have co-existed with the forest for millenia may affect the forest similarly, and that these forces together influence of vegetation patterns on a variety of scales.

Future studies

In the preceding studies, vegetation inside an infection center was compared to that found immediately outside the center. The edge area was combined in these two categories because in nearly all cases it did not differ from either the center area of the infection center or the non-infected forest outside the center. The most significant differences in composition were between the center area of an infection and the non-infected adjacent forest for all sites. One could analyze the data set using these two categories, the same categories used in the preceding analyses but with the edge plots removed. Analyzing the data set in this manner would probably help to pinpoint the specific changes the plant community experienced with disease presence. However, one needs to be able to justify eliminating an area that is part of the community being examined.

The long-term observation of the infection centers would also be interesting to examine in order to know if changes in populations and successional patterns due to disease change with infection center enlargement. That is, are there qualitative differences in the composition and successional direction based on the age of an infection center? Unfortunately the time scale for a direct study of the question would not be practical, but it might be possible to locate centers of varying sizes across a

very similar environments, size being a very rough indication of the age of a center.

In terms of management of the disease, it would be interesting to know if the response of plantations to *P. weirii* are similar to those found in naturally regenerated mature forests and old-growth. Different responses to an endogenous disturbance agent may tell us whether the resilience of managed and natural ecosystems to disturbance differs.

Thesis improvements

Though the presence of disease was shown in this study to affect old-growth community dynamics, the reasons for such effects are still unclear. Changes in the canopy cover due to the removal of Douglas-fir by *P. weirii* was the only environmental factor examined in this study that could have led to the community responses. Because canopy cover was not quantitatively different inside and outside infection centers, other factors responsible for triggering changes in populations should have been examined. Good candidates that could have been looked at are the quality of light or the quantity of light received over time for plots inside and outside infection centers, as well as differences in the soil microbial and nutrient status with changing canopy species.

Some other improvements that could be made on this thesis are having larger sample sizes for each infection site, and having more infection centers to sample from. By comparing population responses to disease over a wider variety of vegetation types and stand ages, one could also find out what community attributes make a community more, or less,

sensitive to disease presence.

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APPENDICES

Appendices 1-6: The following appendices are the data sets collected at each site and used in the analyses. The first matrix of each appendix is a species x plot matrix, the second is the accompanying environmental matrix. In the species matrices, all numbers represent the cover (%) of that species per m² plot except for those species with a "BA" suffix. The values given for these species are transformed basal areas for the whole plot. For more information on how these transformed values were derived, see Materials and Methods of Chapter II.

Species Code Key:

ABAM=*Abies amabilis*
 ACCI=*Acer circinatum*
 ACMA=*Acer macrophyllum*
 ACRU=*Actaea rubra*
 ACTR=*Achlys triphylla*
 ADBI=*Adenocaulon bicolor*
 ANOR=*Anemone oregana*
 ASCA=*Asarum caudatum*
 BENE=*Berberis nervosa*
 BLSP=*Blechnum spicant*
 CACH=*Castanopsis chrysophylla*
 CHME=*Chimaphila menziesii*
 CHUM=*Chimaphila umbellatum*
 COCA=*Cornus canadensis*
 COCO=*Cornus occidentalis*
 COLA=*Coptis laciniata*
 GATR=*Galium triflorum*
 GASH=*Galium* sp.
 GOOB=*Goodyera oblongifolia*
 GRAM=unknown grass species
 HIAL=*Hieracium albiflorum*
 HODI=*Holodiscus discolor*
 LIBO=*Linnaea borealis*
 LILY=unknown lily species
 LUPA=*Luzula parviflora*
 OXOR=*Oxalis oregana*
 PAMY=*Pachystima myrsinites*

POMU=*Polystichum munitum*
 PTAQ=*Pteridium aquilinum*
 PYAS=*Pyrola asarifolia*
 RHMA=*Rhododendron macrophyllum*
 RHPU=*Rhamnus pursiana*
 ROGY=*Rosa gymnocarpa*
 RUNI=*Rubus nivalis*
 RUSP=*Rubus* sp.
 RUUR=*Rubus ursinis*
 SASP=saxifrage species
 SMRA=*Smiliacina racemosa*
 STST=*Streptopus streptopoides*
 SYMO=*Symphoricarpus mollis*
 TABR=*Taxus brevifolia*
 THPL=*Thuja plicata*
 TIUN=*Tiarella unifoliata*
 TISP=*Tiarella* sp.
 TRLA=*Trientalis latifolia*
 TROV=*Trillium ovatum*
 TSHE=*Tsuga heterophylla*
 UNKN=unknown species
 VAHE=*Vancouveria hexandra*
 VAME=*Vaccinium membranaceum*
 VAPA=*Vaccinium parvifolium*
 VIOR=*Viola orbiculata*
 WHMO=*Whipplea modesta*
 XETE=*Xerophyllum tenax*

Environmental variables code:

2CAT=plot location categories: 0=outside of infection center, 1=inside of infection center.
 3CAT=plot location categories:0=far outside of infection center, 1=edge area of infection center, 2=center area of infection center.
 CANOPY=canopy cover over each m² plot (%).
 COARSE=coarse woody debris (>=5cm dbh) in each m² plot (%).
 DISTAN=relative distance of each plot to edge of infection, inside plots being negative (m).
 DISTRE=DISTAN rescaled so all values positive.
 FINE=fine woody debris (<5cm dbh) found in each m² plot (%).

HERB=total herbaceous cover in each m² plot (%).

PSME=relative basal area of Douglas-fir, see Materials and Methods of Chapter 2 for full explanation of this value.

REGACC=regeneration index of *Acer circinatum* (0-3).

REGACM=regeneration index of *Acer macrophyllum* (0-3).

REGRHM=regeneration index of *Rhododendron macrophyllum* (0-3).

REGTAB=regeneration of *Taxus brevifolia* (# of seedlings).

REGTHP=regeneration of *Thuja plicata* (#of seedlings).

REGTSH=regeneration of *Tsuga heterophylla* (#of seedlings).

REGVAP=regeneration index of *Vaccinium parviflorum* (0-3).

TOTAL=HERB + SHRUB.

SHRUB=total shrub cover for each m² plot (%).

SLOPE=slope index from 0-5 for each m² plot: 0=flat, 1=flat/medium, 2=medium, 3=medium/steep, 4=steep, 5=irregular topography.

PLO167	0.	0.	0.	0.	0.	18.	0.	0.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	26.								
PLO168	0.	0.	0.	0.	0.	15.	0.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	0.	26.									
PLO169	0.	0.	0.	0.	0.	18.	0.	0.	0.	12.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	33.									
PLO170	0.	95.	0.	0.	0.	60.	0.	0.	0.	45.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	7.	1.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.									
PLO171	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	24.									
PLO172	0.	0.	0.	0.	0.	10.	0.	0.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	11.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	22.									
PLO173	0.	100.	0.	0.	2.	15.	0.	0.	0.	27.	0.	0.	0.	0.	7.	0.	0.	0.	0.	2.	0.	0.
	10.	5.	0.	2.	0.	0.	0.	0.	0.	20.	0.	0.	4.									
PLO174	0.	0.	0.	0.	0.	0.	0.	0.	0.	30.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	1.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	69.									
PLO175	0.	0.	0.	0.	0.	7.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	11.									
PLO176	0.	0.	0.	0.	0.	0.	0.	0.	0.	6.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	28.									
PLO177	0.	5.	0.	0.	0.	85.	0.	0.	0.	10.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	19.	1.	0.	0.	0.	0.	0.	0.	0.	5.	0.	0.	1.									
PLO178	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	9.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.									
PLO179	0.	0.	0.	0.	0.	0.	0.	0.	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.									
PLO180	0.	8.	0.	0.	0.	33.	0.	0.	0.	5.	1.	1.	0.	0.	0.	0.	10.	0.	7.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	15.	0.	0.	1.									
PLO181	0.	0.	0.	0.	0.	14.	0.	0.	0.	8.	0.	0.	0.	0.	13.	0.	0.	0.	0.	1.	0.	0.
	19.	1.	0.	0.	15.	0.	0.	0.	0.	2.	0.	0.	6.									
PLO182	0.	0.	0.	0.	0.	3.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.
	42.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	5.									
PLO183	0.	0.	0.	0.	0.	10.	0.	0.	0.	40.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	3.	1.	0.	0.	0.	0.	0.	0.	3.	0.	0.	49.									
PLO184	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	10.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.									
PLO185	15.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.									
PLO186	0.	30.	0.	0.	0.	25.	0.	0.	0.	13.	0.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.
	60.	0.	0.	0.	0.	0.	0.	0.	0.	7.	0.	0.	6.									
PLO187	0.	0.	0.	0.	0.	1.	0.	0.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	34.	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	0.	5.									
PLO188	0.	0.	0.	0.	0.	20.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	4.									
PLO189	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	1.	0.	0.	5.	0.	0.	0.	0.	0.	0.	0.	6.									
PLO190	0.	2.	4.	0.	0.	16.	0.	0.	0.	6.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.
	25.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.									
PLO191	0.	3.	0.	0.	0.	22.	0.	0.	0.	25.	0.	0.	0.	0.	7.	0.	0.	0.	0.	1.	0.	0.
	1.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	10.									
PLO192	0.	0.	0.	0.	0.	0.	0.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	54.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	26.									
PLO193	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	6.									
PLO194	0.	0.	0.	0.	0.	25.	0.	0.	0.	0.	0.	13.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	27.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.									
PLO195	0.	1.	0.	0.	0.	11.	0.	0.	0.	25.	0.	2.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.
	17.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.									
PLO196	0.	20.	0.	0.	0.	20.	0.	0.	0.	0.	3.	0.	0.	11.	0.	0.	0.	0.	1.	0.	0.	0.
	7.	0.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.									
PLO197	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.									
PLO198	0.	80.	0.	0.	0.	6.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	48.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.									
PLO199	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	36.									

1=ABAM, 2=ACCI, 3=ACTR, 4=ANOR, 5=ASCA, 6=BENE, 7=CHME, 8=CHUM, 9=COCA, 10=COLA, 11=GOOB, 12=L180, 13=LUPA, 14=PAMY, 15=POMU, 16=PSME, 17=PYAS, 18=RHMA, 19=ROGY, 20=RUSP, 21=SASP, 22=STST, 23=SYMU,

24=TABR, 25=ACCIBA, 26=TISP, 27=TRLA, 28=TROV, 29=TSHE, 30=UNKN, 31=VAHE, 32=VAME, 33=VAPA,
34=VIOR, 35=WHMO, 36=XETE, 37=TSHEBA

199 PLOTS

16 ATTRIBU

(F5.0,12F6.0/3F6.0)

CQQQQQCCCCQCCQ

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
0.	75.	0.	95.	9.	0.	9.	2.	0.	0.	-8.	0.	1.	2.	29.	14.
0.	85.	0.	80.	0.	1.	1.	2.	0.	0.	-13.	0.	1.	2.	24.	0.
0.	84.	0.	70.	12.	8.	20.	2.	3.	2.	-18.	0.	1.	2.	19.	0.
0.	87.	20.	0.	32.	22.	54.	3.	0.	0.	-20.	0.	1.	2.	17.	4.
0.	85.	10.	0.	19.	51.	70.	4.	0.	0.	-13.	0.	1.	2.	24.	13.
0.	87.	12.	0.	49.	22.	71.	1.	0.	0.	-20.	0.	1.	2.	17.	0.
0.	73.	20.	12.	84.	10.	94.	1.	1.	0.	-22.	0.	1.	2.	15.	0.
0.	82.	5.	0.	0.	0.	0.	1.	0.	0.	-37.	0.	1.	2.	0.	0.
0.	86.	20.	0.	0.	0.	0.	2.	0.	0.	-28.	0.	1.	2.	9.	0.
0.	83.	10.	0.	13.	4.	17.	0.	0.	3.	-31.	0.	1.	2.	6.	0.
0.	86.	30.	10.	9.	1.	10.	0.	0.	0.	-20.	0.	1.	2.	17.	0.
0.	87.	25.	5.	28.	3.	31.	2.	0.	0.	-13.	0.	1.	2.	24.	0.
0.	85.	30.	0.	21.	0.	21.	2.	0.	0.	-3.	0.	1.	1.	34.	0.
0.	83.	5.	80.	8.	6.	14.	4.	0.	0.	9.	0.	0.	0.	46.	21.
0.	86.	10.	0.	3.	1.	3.	0.	0.	0.	10.	0.	0.	0.	47.	0.
0.	87.	27.	0.	7.	0.	7.	2.	0.	2.	13.	0.	0.	0.	50.	0.
0.	86.	80.	0.	2.	0.	2.	2.	0.	0.	-5.	0.	1.	1.	32.	0.
0.	88.	20.	20.	11.	1.	12.	2.	0.	0.	-1.	0.	1.	1.	36.	0.
0.	84.	40.	10.	3.	7.	10.	2.	0.	0.	-8.	0.	1.	1.	29.	0.
0.	84.	5.	85.	0.	1.	1.	3.	0.	0.	8.	0.	0.	0.	45.	0.
0.	86.	30.	0.	28.	12.	40.	3.	3.	0.	-5.	0.	1.	1.	32.	0.
0.	84.	40.	0.	19.	6.	25.	3.	0.	0.	0.	0.	1.	1.	37.	1.
0.	85.	40.	10.	0.	5.	5.	3.	1.	0.	2.	1.	0.	1.	39.	0.
0.	83.	35.	15.	0.	2.	2.	0.	0.	0.	4.	0.	0.	1.	41.	31.
0.	82.	85.	0.	0.	7.	7.	0.	0.	0.	7.	0.	0.	1.	44.	31.
0.	81.	15.	100.	0.	9.	9.	2.	0.	0.	12.	0.	0.	0.	49.	28.
0.	86.	40.	5.	4.	25.	29.	2.	0.	0.	8.	0.	0.	0.	45.	31.
0.	82.	40.	0.	12.	75.	87.	2.	1.	3.	23.	1.	0.	0.	60.	21.
0.	76.	10.	0.	19.	75.	94.	3.	1.	0.	16.	1.	0.	0.	53.	0.
0.	77.	0.	70.	5.	19.	24.	2.	0.	0.	17.	0.	0.	0.	54.	0.
0.	82.	40.	9.	12.	15.	27.	2.	3.	0.	13.	0.	0.	0.	50.	26.
0.	84.	30.	0.	37.	0.	37.	3.	3.	0.	8.	0.	0.	0.	45.	31.
0.	80.	20.	10.	12.	47.	59.	4.	3.	0.	7.	0.	0.	1.	44.	0.
0.	80.	25.	0.	7.	3.	10.	2.	0.	0.	20.	0.	0.	0.	57.	30.
0.	82.	15.	0.	0.	0.	0.	2.	0.	0.	18.	0.	0.	0.	55.	30.
0.	79.	40.	0.	16.	24.	40.	2.	3.	0.	28.	0.	0.	0.	65.	74.
0.	77.	20.	65.	5.	5.	10.	2.	1.	0.	32.	0.	0.	0.	69.	34.
0.	71.	20.	5.	36.	19.	55.	2.	0.	0.	42.	0.	0.	0.	79.	0.
0.	79.	20.	100.	0.	0.	0.	2.	0.	0.	47.	0.	0.	0.	84.	0.
0.	74.	30.	0.	32.	13.	45.	2.	0.	0.	42.	0.	0.	0.	79.	0.
0.	78.	95.	30.	22.	1.	23.	3.	2.	1.	-8.	0.	1.	2.	29.	63.
0.	77.	30.	0.	25.	0.	25.	2.	0.	0.	-22.	0.	1.	2.	15.	0.
0.	81.	30.	0.	47.	3.	50.	2.	1.	0.	-26.	0.	1.	2.	11.	10.
0.	81.	30.	20.	1.	2.	3.	3.	0.	0.	-33.	0.	1.	2.	4.	31.
0.	82.	55.	0.	26.	6.	32.	2.	3.	0.	-34.	0.	1.	2.	3.	0.
0.	82.	10.	50.	2.	23.	25.	3.	1.	1.	-26.	0.	1.	2.	11.	0.
0.	85.	30.	0.	9.	12.	21.	3.	0.	1.	-32.	0.	1.	2.	5.	0.
0.	83.	30.	0.	38.	5.	43.	2.	0.	0.	-30.	0.	1.	2.	7.	0.
0.	84.	30.	15.	0.	0.	0.	1.	0.	0.	-29.	0.	1.	2.	8.	0.
0.	83.	30.	25.	15.	55.	70.	3.	0.	1.	-21.	0.	1.	2.	16.	41.
0.	80.	25.	30.	13.	5.	18.	3.	0.	0.	4.	0.	0.	1.	41.	0.
0.	78.	20.	60.	31.	37.	68.	3.	0.	1.	-0.	0.	1.	1.	37.	33.
0.	84.	10.	0.	12.	10.	22.	2.	0.	1.	14.	0.	0.	0.	51.	0.
0.	85.	10.	90.	0.	0.	0.	2.	0.	2.	13.	0.	0.	0.	50.	0.
0.	86.	30.	0.	13.	0.	13.	2.	0.	0.	9.	0.	0.	0.	46.	0.
0.	84.	40.	5.	1.	0.	1.	0.	0.	0.	8.	0.	0.	0.	45.	0.
0.	85.	15.	55.	1.	1.	2.	0.	0.	0.	6.	0.	0.	1.	43.	61.
0.	85.	40.	5.	12.	0.	12.	2.	0.	0.	11.	0.	0.	0.	48.	0.

0.	84.	30.	25.	1.	0.	1.	2.	0.	3.	17.	0.	0.	0.	54.	0.
0.	84.	30.	0.	25.	0.	25.	2.	0.	0.	26.	0.	0.	0.	63.	0.
0.	88.	50.	15.	1.	10.	11.	1.	0.	0.	5.	0.	0.	1.	42.	43.
0.	87.	50.	5.	0.	0.	0.	2.	2.	0.	1.	0.	0.	1.	38.	0.
0.	88.	50.	10.	25.	15.	40.	0.	0.	0.	5.	0.	0.	1.	42.	0.
0.	78.	5.	20.	9.	92.	101.	0.	3.	1.	-14.	0.	1.	2.	23.	0.
0.	89.	30.	85.	66.	0.	66.	0.	0.	0.	-20.	0.	1.	2.	17.	0.
0.	68.	20.	45.	0.	8.	8.	1.	3.	0.	-2.	0.	1.	1.	35.	0.
0.	82.	10.	90.	21.	0.	21.	5.	0.	3.	-21.	0.	1.	2.	16.	0.
0.	86.	40.	0.	26.	0.	26.	0.	0.	2.	-12.	0.	1.	2.	25.	0.
0.	85.	60.	0.	65.	3.	68.	0.	0.	0.	12.	0.	0.	0.	49.	0.
0.	84.	55.	10.	0.	9.	9.	0.	1.	0.	-6.	0.	1.	1.	31.	0.
0.	81.	70.	30.	0.	0.	0.	0.	0.	0.	2.	0.	0.	1.	39.	31.
0.	80.	40.	0.	15.	9.	24.	1.	0.	0.	-11.	0.	1.	2.	26.	0.
0.	75.	15.	10.	14.	10.	24.	2.	0.	0.	-11.	0.	1.	2.	26.	0.
0.	66.	0.	100.	0.	0.	0.	5.	0.	0.	-4.	0.	1.	1.	33.	0.
0.	68.	40.	60.	0.	0.	0.	0.	0.	0.	3.	0.	0.	1.	40.	0.
0.	68.	70.	50.	0.	0.	0.	5.	0.	0.	-5.	0.	1.	1.	32.	0.
0.	80.	20.	70.	0.	0.	0.	5.	0.	0.	4.	0.	0.	1.	41.	0.
0.	78.	100.	50.	0.	0.	0.	5.	0.	0.	2.	0.	0.	1.	39.	51.
0.	79.	50.	0.	2.	0.	2.	0.	0.	0.	9.	0.	0.	0.	46.	0.
0.	87.	50.	4.	21.	18.	39.	1.	0.	1.	11.	0.	0.	0.	48.	49.
0.	83.	55.	5.	10.	6.	16.	2.	0.	0.	2.	0.	0.	1.	39.	0.
0.	86.	90.	20.	1.	0.	1.	1.	0.	0.	6.	0.	0.	1.	43.	0.
0.	80.	20.	0.	34.	33.	67.	0.	3.	2.	17.	0.	0.	0.	54.	59.
0.	86.	0.	100.	0.	0.	0.	5.	0.	2.	8.	0.	0.	0.	45.	25.
0.	86.	95.	40.	0.	0.	0.	0.	2.	0.	3.	0.	0.	1.	40.	65.
0.	86.	95.	45.	0.	0.	0.	1.	0.	0.	-1.	0.	1.	1.	36.	100.
0.	84.	10.	50.	20.	9.	29.	0.	2.	0.	-6.	0.	1.	1.	31.	68.
0.	85.	10.	45.	1.	0.	1.	0.	2.	2.	-15.	0.	1.	2.	22.	0.
0.	85.	50.	50.	7.	0.	7.	0.	3.	0.	-11.	0.	1.	2.	26.	0.
0.	87.	55.	0.	12.	0.	12.	1.	0.	0.	4.	0.	0.	1.	41.	0.
0.	85.	25.	0.	14.	50.	64.	0.	0.	0.	2.	0.	0.	1.	39.	0.
0.	88.	20.	30.	27.	5.	31.	0.	0.	3.	-6.	0.	1.	1.	31.	0.
1.	86.	55.	0.	6.	0.	6.	0.	0.	0.	0.	0.	1.	1.	37.	0.
0.	81.	45.	15.	29.	0.	29.	0.	0.	0.	-6.	0.	1.	1.	31.	50.
0.	87.	30.	4.	18.	0.	18.	0.	0.	0.	-9.	0.	1.	2.	28.	0.
0.	69.	10.	90.	2.	21.	23.	0.	0.	0.	-15.	0.	1.	2.	22.	0.
0.	86.	40.	7.	17.	31.	48.	0.	0.	0.	-4.	0.	1.	1.	33.	0.
0.	84.	30.	7.	26.	27.	53.	0.	3.	0.	2.	0.	0.	1.	39.	22.
0.	85.	40.	7.	13.	13.	26.	1.	3.	0.	2.	0.	0.	1.	39.	0.
0.	84.	10.	50.	16.	18.	34.	1.	3.	1.	-5.	0.	1.	1.	32.	0.
0.	87.	7.	90.	9.	0.	9.	5.	2.	1.	-11.	0.	1.	2.	26.	0.
0.	87.	40.	15.	16.	10.	26.	0.	0.	0.	-22.	0.	1.	2.	15.	0.
0.	87.	5.	5.	1.	1.	2.	0.	0.	0.	-29.	0.	1.	2.	8.	0.
0.	88.	45.	0.	21.	0.	21.	0.	1.	0.	-30.	0.	1.	2.	7.	0.
0.	85.	55.	15.	0.	10.	10.	0.	1.	0.	-29.	0.	1.	2.	8.	66.
0.	83.	30.	0.	17.	56.	73.	0.	0.	0.	-20.	0.	1.	2.	17.	2.
0.	87.	50.	10.	6.	18.	24.	0.	3.	0.	-12.	0.	1.	2.	25.	2.
0.	87.	20.	0.	22.	72.	94.	2.	3.	0.	-10.	0.	1.	2.	27.	0.
0.	86.	10.	88.	0.	0.	0.	2.	0.	1.	6.	0.	0.	1.	43.	0.
2.	85.	20.	0.	10.	51.	61.	1.	0.	1.	6.	0.	0.	1.	43.	17.
2.	82.	40.	0.	1.	0.	1.	2.	0.	0.	16.	0.	0.	0.	53.	0.
0.	85.	15.	9.	50.	29.	79.	0.	0.	0.	6.	0.	0.	1.	43.	0.
0.	87.	25.	60.	0.	0.	0.	0.	1.	0.	7.	0.	0.	1.	44.	8.
0.	88.	40.	0.	31.	10.	41.	1.	1.	0.	1.	0.	0.	1.	38.	17.
0.	90.	30.	7.	0.	0.	0.	1.	0.	0.	-5.	0.	1.	1.	32.	25.
0.	86.	15.	0.	1.	1.	2.	0.	0.	2.	-2.	0.	1.	1.	35.	27.
0.	86.	20.	0.	6.	0.	6.	1.	1.	2.	-9.	0.	1.	2.	28.	21.
0.	61.	15.	0.	26.	35.	61.	1.	3.	3.	-14.	0.	1.	2.	23.	0.
0.	90.	15.	10.	19.	9.	28.	2.	2.	0.	-16.	0.	1.	2.	21.	0.
0.	88.	20.	0.	16.	6.	22.	2.	2.	0.	-18.	0.	1.	2.	19.	33.
0.	88.	30.	0.	13.	15.	28.	0.	3.	2.	-8.	0.	1.	2.	29.	0.
0.	87.	5.	0.	14.	46.	60.	1.	3.	3.	1.	0.	0.	1.	38.	0.
0.	85.	9.	0.	7.	89.	96.	1.	2.	1.	11.	0.	0.	0.	48.	0.
0.	87.	9.	0.	4.	7.	11.	2.	0.	0.	12.	0.	0.	0.	49.	4.
0.	86.	55.	5.	2.	45.	47.	1.	0.	1.	14.	0.	0.	0.	51.	0.
0.	90.	35.	7.	4.	55.	59.	2.	0.	0.	25.	0.	0.	0.	62.	36.

0.	84.	40.	30.	20.	50.	70.	3.	2.	1.	33.	0.	0.	0.	70.	2.
0.	79.	20.	35.	6.	58.	64.	0.	1.	1.	8.	0.	0.	1.	45.	0.
0.	85.	5.	50.	0.	0.	0.	5.	0.	1.	2.	0.	0.	1.	39.	0.
0.	85.	12.	92.	1.	0.	1.	0.	0.	1.	0.	0.	1.	1.	37.	0.
0.	84.	20.	7.	4.	6.	10.	0.	1.	0.	-1.	0.	1.	1.	36.	0.
0.	85.	25.	25.	0.	0.	0.	0.	0.	0.	-7.	0.	1.	1.	30.	0.
0.	86.	40.	60.	1.	3.	4.	0.	1.	0.	-10.	0.	1.	2.	27.	30.
1.	86.	25.	50.	1.	9.	10.	2.	0.	3.	1.	0.	0.	1.	38.	0.
0.	82.	0.	100.	0.	0.	0.	0.	2.	2.	-1.	0.	1.	1.	36.	0.
0.	87.	25.	0.	2.	13.	15.	1.	0.	3.	-5.	0.	1.	1.	32.	16.
0.	87.	45.	0.	3.	60.	63.	1.	3.	0.	-3.	1.	1.	1.	34.	16.
0.	84.	25.	30.	3.	3.	6.	0.	1.	1.	5.	0.	0.	1.	42.	0.
0.	84.	40.	0.	4.	15.	19.	2.	2.	0.	2.	0.	0.	1.	39.	28.
0.	87.	30.	20.	0.	6.	6.	2.	2.	0.	6.	1.	0.	1.	43.	0.
1.	88.	15.	90.	0.	0.	0.	5.	0.	0.	1.	0.	0.	1.	38.	32.
0.	92.	60.	8.	0.	15.	15.	3.	0.	0.	-6.	2.	1.	1.	31.	0.
0.	89.	40.	5.	8.	2.	10.	2.	2.	0.	2.	0.	0.	1.	39.	0.
0.	88.	40.	0.	0.	0.	0.	2.	0.	0.	9.	0.	0.	0.	46.	32.
0.	87.	20.	30.	0.	15.	15.	0.	0.	0.	15.	0.	0.	0.	52.	0.
3.	91.	20.	55.	0.	26.	26.	0.	0.	0.	12.	0.	0.	0.	49.	0.
1.	84.	10.	80.	10.	4.	14.	0.	0.	0.	8.	0.	0.	0.	45.	67.
0.	81.	15.	25.	37.	4.	41.	0.	1.	1.	8.	0.	0.	0.	45.	0.
0.	84.	10.	70.	1.	0.	1.	2.	2.	0.	13.	0.	0.	0.	50.	0.
0.	90.	40.	0.	42.	7.	49.	2.	3.	0.	25.	0.	0.	0.	62.	0.
0.	88.	30.	7.	15.	7.	22.	1.	2.	0.	20.	0.	0.	0.	57.	0.
0.	91.	15.	25.	3.	13.	16.	1.	1.	1.	32.	0.	0.	0.	69.	0.
0.	89.	40.	45.	2.	10.	12.	1.	1.	0.	27.	0.	0.	0.	64.	0.
0.	89.	60.	5.	0.	5.	5.	2.	2.	0.	49.	0.	0.	0.	86.	97.
0.	63.	20.	30.	15.	72.	87.	2.	3.	3.	51.	0.	0.	0.	88.	0.
0.	87.	60.	0.	21.	8.	29.	0.	1.	0.	40.	0.	0.	0.	77.	0.
0.	84.	10.	0.	97.	90.	187.	0.	1.	0.	67.	0.	0.	0.	104.	0.
0.	88.	15.	50.	3.	7.	10.	0.	1.	0.	87.	0.	0.	0.	124.	0.
0.	88.	20.	3.	12.	13.	24.	0.	1.	0.	49.	0.	0.	0.	86.	0.
1.	89.	10.	30.	25.	1.	26.	0.	0.	2.	80.	0.	0.	0.	117.	65.
0.	88.	20.	10.	26.	1.	27.	0.	0.	0.	45.	0.	0.	0.	82.	0.
0.	80.	50.	10.	3.	28.	31.	0.	2.	1.	47.	0.	0.	0.	84.	100.
0.	88.	5.	100.	2.	0.	2.	0.	0.	0.	43.	0.	0.	0.	80.	0.
0.	88.	15.	25.	12.	16.	28.	0.	0.	2.	25.	0.	0.	0.	62.	0.
0.	89.	25.	0.	0.	0.	0.	0.	0.	0.	23.	0.	0.	0.	60.	26.
0.	88.	95.	20.	2.	2.	4.	1.	0.	0.	14.	0.	0.	0.	51.	50.
0.	87.	50.	5.	5.	18.	23.	2.	0.	3.	38.	0.	0.	0.	75.	43.
0.	88.	50.	7.	14.	15.	29.	4.	0.	0.	47.	0.	0.	0.	84.	0.
0.	88.	30.	0.	12.	19.	31.	4.	0.	0.	66.	0.	0.	0.	103.	0.
0.	90.	5.	0.	48.	155.	203.	2.	2.	3.	92.	0.	0.	0.	129.	0.
0.	88.	5.	80.	1.	1.	2.	2.	0.	0.	58.	0.	0.	0.	95.	75.
0.	90.	20.	0.	5.	10.	15.	2.	0.	1.	76.	0.	0.	0.	113.	85.
0.	91.	2.	0.	63.	117.	180.	2.	0.	2.	97.	0.	0.	0.	134.	0.
0.	82.	20.	10.	32.	0.	32.	1.	0.	0.	74.	1.	0.	0.	111.	0.
0.	85.	40.	0.	1.	7.	8.	3.	0.	0.	52.	0.	0.	0.	89.	57.
0.	86.	60.	0.	6.	0.	6.	1.	0.	0.	45.	0.	0.	0.	82.	24.
0.	86.	5.	0.	21.	60.	81.	1.	2.	1.	36.	0.	0.	0.	73.	0.
0.	86.	5.	95.	0.	0.	0.	1.	2.	2.	49.	0.	0.	0.	86.	0.
0.	83.	20.	25.	12.	0.	12.	1.	0.	0.	60.	0.	0.	0.	97.	0.
0.	80.	5.	0.	22.	58.	80.	0.	2.	0.	58.	2.	0.	0.	95.	0.
0.	85.	12.	0.	24.	15.	39.	0.	0.	3.	39.	0.	0.	0.	76.	27.
0.	83.	25.	10.	4.	6.	10.	0.	2.	3.	45.	0.	0.	0.	82.	47.
0.	84.	30.	0.	47.	10.	57.	3.	0.	0.	46.	0.	0.	0.	83.	0.
0.	82.	8.	0.	1.	0.	1.	0.	0.	1.	38.	0.	0.	0.	75.	40.
0.	82.	20.	0.	6.	0.	6.	2.	2.	0.	39.	0.	0.	0.	76.	17.
0.	85.	25.	0.	20.	58.	78.	0.	2.	2.	48.	0.	0.	0.	85.	37.
0.	86.	12.	0.	7.	1.	8.	2.	1.	3.	65.	0.	0.	0.	102.	25.
0.	88.	95.	35.	1.	2.	3.	2.	0.	1.	73.	1.	0.	0.	110.	0.
0.	85.	15.	85.	1.	2.	3.	0.	2.	3.	95.	0.	0.	0.	132.	0.
0.	86.	30.	0.	12.	19.	31.	2.	0.	1.	51.	0.	0.	0.	88.	0.
0.	88.	20.	3.	35.	26.	61.	0.	0.	3.	100.	0.	0.	0.	137.	43.
0.	89.	10.	25.	14.	0.	14.	0.	0.	0.	70.	0.	0.	0.	107.	62.
0.	83.	55.	0.	1.	0.	1.	0.	0.	0.	25.	0.	0.	0.	62.	41.
0.	80.	10.	0.	13.	25.	38.	1.	2.	3.	49.	0.	0.	0.	86.	0.

0.	86.	20.	0.	31.	13.	44.	0.	0.	0.	56.	0.	0.	0.	93.	0.
0.	86.	5.	0.	14.	41.	55.	0.	3.	1.	59.	0.	0.	0.	96.	0.
0.	86.	10.	35.	0.	0.	0.	2.	0.	0.	22.	0.	0.	0.	59.	0.
0.	88.	60.	0.	0.	86.	86.	4.	3.	2.	52.	0.	0.	0.	89.	6.
0.	85.	25.	15.	3.	0.	3.	1.	0.	0.	42.	0.	0.	0.	79.	30.

1=REGTAB, 2=CANOPY, 3=FINE, 4=COARSE, 5=HERB, 6=SHRUB, 7=TOTAL, 8=SLOPE, 9=REGACC, 10=REGTSH,
11=DISTAN, 12=REGRHM, 13=2CAT, 14=3CAT, 15=DISTRE, 16=PSME

APPENDIX 2: Data for Site HJA 2

40 PLOTS
25 SPECIES
(1A6,25F4.0)
(1A6,12F6.0/12F6.0/12F6.0/4F6.0)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
PLOT1	2.	0.	0.	0.	0.	0.	0.	2.	0.	0.	80.	0.	0.	0.	0.	30.	0.	0.	0.	0.	0.	0.	4.	0.	0.
PLOT2	0.	0.	0.	0.	0.	0.	0.	0.	0.	5.	60.	0.	0.	0.	0.	45.	0.	0.	0.	0.	2.	1.	0.	0.	2.
PLOT3	0.	0.	0.	0.	0.	0.	0.	2.	0.	35.	45.	0.	1.	0.	8.	0.	0.	0.	2.	3.	0.	0.	8.	0.	0.
PLOT4	0.	0.	0.	10.	0.	1.	0.	2.	2.	0.	0.	0.	0.	3.	0.	0.	1.	1.	0.	0.	0.	0.	3.	0.	0.
PLOT5	0.	6.	0.	3.	0.	0.	0.	1.	0.	0.	1.	9.	0.	0.	5.	0.	2.	0.	0.	0.	4.	0.	66.	0.	0.
PLOT6	0.	0.	0.	1.	0.	0.	0.	2.	0.	0.	15.	0.	0.	0.	0.	27.	3.	0.	0.	0.	0.	1.	4.	0.	0.
PLOT7	0.	0.	0.	0.	0.	0.	0.	2.	0.	40.	13.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.	82.	0.	0.
PLOT8	0.	0.	0.	0.	0.	0.	0.	0.	0.	18.	55.	0.	0.	0.	0.	4.	2.	0.	0.	0.	0.	0.	34.	0.	0.
PLOT9	0.	0.	0.	0.	0.	3.	0.	1.	0.	40.	3.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	1.	5.	0.	0.
PLOT10	0.	10.	0.	0.	0.	0.	0.	3.	0.	0.	14.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	35.	0.	0.
PLOT11	0.	4.	0.	2.	0.	0.	0.	5.	0.	1.	30.	2.	0.	3.	0.	1.	2.	0.	0.	0.	0.	0.	40.	0.	0.
PLOT12	0.	0.	0.	0.	0.	0.	0.	0.	0.	10.	85.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	9.	0.	0.
PLOT13	0.	0.	8.	3.	0.	0.	0.	1.	0.	14.	23.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	1.	60.	0.	2.
PLOT14	0.	0.	0.	0.	0.	0.	0.	3.	0.	2.	14.	3.	0.	0.	0.	8.	0.	0.	0.	0.	10.	0.	24.	0.	0.
PLOT15	0.	0.	0.	2.	0.	0.	0.	3.	0.	15.	11.	0.	0.	0.	4.	0.	0.	0.	0.	0.	15.	1.	26.	0.	0.
PLOT16	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	57.	0.	5.
PLOT17	0.	10.	0.	1.	0.	0.	0.	6.	0.	17.	0.	0.	0.	0.	0.	40.	0.	0.	0.	0.	0.	0.	26.	0.	1.
PLOT18	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	95.	1.	1.
PLOT19	0.	3.	0.	2.	0.	0.	0.	2.	0.	35.	40.	0.	0.	0.	0.	3.	1.	0.	0.	0.	0.	0.	32.	0.	2.
PLOT20	0.	0.	0.	0.	0.	0.	0.	4.	0.	1.	6.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	53.	0.	1.
PLOT21	0.	0.	0.	20.	1.	0.	0.	2.	0.	0.	0.	0.	0.	0.	4.	0.	0.	0.	1.	0.	1.	19.	0.	0.	
PLOT22	0.	1.	0.	0.	0.	0.	0.	0.	0.	2.	3.	11.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	19.	0.	0.
PLOT23	0.	12.	0.	8.	3.	0.	0.	1.	0.	30.	0.	6.	0.	2.	0.	0.	1.	0.	0.	0.	9.	1.	7.	1.	11.
PLOT24	0.	1.	0.	20.	0.	0.	0.	15.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	8.	1.	9.
PLOT25	0.	0.	0.	2.	0.	0.	0.	1.	0.	95.	16.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	8.	0.	16.	
PLOT26	0.	20.	0.	5.	0.	0.	0.	0.	0.	27.	10.	0.	0.	0.	0.	0.	0.	1.	0.	1.	0.	1.	7.	0.	2.
PLOT27	0.	0.	0.	2.	10.	0.	0.	3.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	16.	0.	2.
PLOT28	0.	0.	0.	2.	0.	0.	0.	3.	0.	19.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	53.	0.	1.
PLOT29	0.	0.	0.	1.	0.	0.	0.	20.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	15.	0.	1.
PLOT30	0.	0.	0.	4.	0.	0.	0.	35.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	1.	12.	
PLOT31	0.	0.	0.	0.	0.	0.	0.	0.	0.	12.	55.	0.	0.	0.	2.	0.	0.	1.	0.	0.	0.	1.	0.	1.	0.
PLOT32	0.	4.	0.	0.	1.	0.	0.	2.	0.	14.	0.	9.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	6.	0.	9.
PLOT33	0.	0.	0.	1.	0.	0.	0.	1.	0.	20.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	1.	17.	0.	0.	
PLOT34	1.	30.	0.	0.	3.	0.	0.	0.	0.	14.	0.	0.	0.	0.	1.	0.	1.	0.	0.	0.	0.	0.	16.	0.	2.
PLOT35	0.	0.	0.	10.	14.	1.	0.	10.	0.	14.	0.	0.	0.	0.	4.	0.	2.	0.	0.	0.	1.	13.	0.	1.	
PLOT36	0.	4.	0.	1.	2.	0.	0.	2.	0.	20.	6.	0.	0.	0.	0.	1.	0.	0.	1.	0.	0.	3.	3.	3.	
PLOT37	0.	0.	0.	1.	0.	0.	0.	1.	0.	12.	0.	1.	0.	0.	0.	1.	0.	0.	0.	0.	0.	1.	16.	1.	0.
PLOT38	0.	8.	0.	3.	0.	0.	0.	1.	0.	5.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	29.	0.	9.
PLOT39	0.	9.	0.	3.	0.	0.	1.	1.	0.	5.	0.	2.	0.	0.	1.	0.	0.	0.	0.	0.	9.	0.	10.	1.	9.
PLOT40	0.	0.	0.	1.	0.	0.	0.	4.	0.	0.	0.	5.	0.	0.	0.	9.	0.	0.	0.	0.	0.	0.	20.	1.	17.

1=ACTR, 2=BENE, 3=BLSP, 4=COLA, 5=GASH, 6=GASP, 7=GOOB, 8=LIBO, 9=LILY, 10=OXOR, 11=POMU, 12=RHMA,
13=RHPU, 14=RUNI, 15=RUUR, 16=THPL, 17=TIUN, 18=TRLA, 19=TROV, 20=VAHE, 21=VAPA, 22=VIOR, 23=TSHEBA,
24=TABRBA, 25=ACCIBA

40 PLOTS
 16 VARIABL
 (SF3.0,F5.0,F6.0,F6.0,F6.0,F6.0,F8.0,F4.0,3F4.0,F5.0)
 QQQQQQQQQQQCCCA

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
0.	3.	0.	0.	0.	84.	0.	84.	7.	0.	-18.	86.	1.	2.	0.	0.
0.	3.	2.	0.	0.	66.	2.	68.	4.	0.	-26.	88.	1.	2.	0.	11.
0.	0.	0.	0.	0.	85.	12.	97.	13.	16.	-24.	84.	1.	2.	0.	0.
0.	0.	0.	0.	0.	17.	3.	20.	4.	0.	-23.	89.	1.	2.	0.	0.
0.	0.	0.	0.	0.	7.	15.	22.	60.	55.	-18.	85.	1.	2.	0.	0.
0.	0.	0.	0.	0.	22.	0.	22.	40.	10.	-26.	85.	1.	2.	0.	0.
0.	3.	0.	2.	0.	59.	0.	59.	2.	0.	-16.	90.	1.	2.	0.	0.
0.	0.	0.	2.	1.	75.	0.	75.	8.	0.	-7.	88.	1.	1.	1.	0.
0.	0.	0.	1.	0.	49.	0.	49.	4.	0.	-8.	89.	1.	2.	0.	0.
0.	0.	0.	2.	0.	10.	18.	28.	6.	50.	-13.	88.	1.	2.	0.	0.
0.	2.	0.	2.	0.	40.	7.	47.	6.	0.	-23.	81.	1.	2.	1.	0.
0.	1.	0.	2.	0.	95.	2.	97.	9.	10.	-21.	87.	1.	2.	1.	0.
0.	3.	2.	2.	0.	42.	0.	42.	3.	45.	-17.	91.	1.	2.	1.	0.
0.	2.	2.	2.	0.	27.	10.	37.	5.	0.	-9.	90.	1.	2.	5.	0.
0.	2.	1.	2.	0.	32.	19.	51.	7.	10.	-8.	88.	1.	2.	0.	0.
2.	3.	0.	2.	0.	1.	2.	3.	0.	100.	-8.	91.	1.	2.	5.	0.
0.	3.	0.	2.	0.	24.	10.	34.	15.	45.	-17.	92.	1.	2.	0.	0.
0.	0.	1.	2.	0.	0.	0.	0.	6.	5.	-13.	90.	1.	2.	5.	0.
2.	2.	0.	0.	0.	80.	3.	83.	3.	0.	-7.	89.	1.	1.	0.	0.
2.	0.	1.	1.	0.	11.	0.	11.	11.	0.	-1.	91.	1.	1.	0.	0.
0.	3.	1.	1.	0.	23.	2.	25.	35.	0.	7.	89.	0.	1.	0.	0.
0.	0.	0.	0.	3.	5.	4.	9.	3.	90.	4.	91.	0.	1.	5.	0.
0.	3.	0.	2.	2.	41.	26.	67.	5.	0.	8.	89.	0.	1.	0.	49.
2.	1.	0.	0.	0.	35.	1.	36.	40.	5.	20.	90.	0.	0.	0.	76.
2.	1.	0.	1.	0.	115.	0.	115.	7.	0.	25.	88.	0.	0.	5.	37.
2.	3.	0.	0.	0.	42.	21.	63.	10.	0.	30.	92.	0.	0.	2.	0.
2.	0.	0.	0.	0.	9.	10.	19.	13.	16.	30.	90.	0.	0.	2.	32.
0.	2.	0.	2.	0.	24.	0.	24.	9.	0.	30.	88.	0.	0.	0.	0.
2.	0.	0.	2.	0.	26.	0.	26.	25.	5.	35.	88.	0.	0.	1.	37.
2.	0.	0.	0.	0.	40.	0.	40.	25.	7.	35.	89.	0.	0.	0.	46.
0.	0.	0.	0.	0.	68.	2.	70.	15.	5.	40.	87.	0.	0.	0.	0.
0.	2.	0.	0.	0.	17.	14.	31.	15.	30.	40.	90.	0.	0.	0.	42.
0.	2.	0.	0.	0.	24.	0.	24.	13.	45.	40.	90.	0.	0.	0.	0.
0.	0.	0.	2.	0.	16.	34.	50.	15.	0.	35.	90.	0.	0.	0.	42.
1.	3.	0.	0.	0.	38.	18.	56.	11.	5.	35.	87.	0.	0.	0.	19.
0.	3.	0.	0.	2.	30.	7.	37.	11.	0.	30.	89.	0.	0.	0.	26.
0.	2.	0.	2.	0.	16.	0.	16.	8.	60.	30.	88.	0.	0.	0.	0.
0.	0.	0.	0.	2.	10.	8.	18.	12.	5.	25.	89.	0.	0.	0.	33.
0.	1.	0.	0.	0.	10.	19.	29.	12.	0.	15.	86.	0.	0.	0.	37.
0.	1.	1.	0.	3.	5.	0.	5.	7.	30.	6.	90.	0.	1.	0.	28.

1=REGACC, 2=REGTSH, 3=REGTHP, 4=REGVAP, 5=REGRNM, 6=HERB, 7=SHRUB, 8=TOTAL,
 9=FINE, 10=COARSE, 11=DISTAN, 12=CANOPY, 13=2CAT, 14=3CAT, 15=SLOPE, 16=PSME

APPENDIX 3: Data for Site HJA 3

40 PLOTS
25 SPECIES
(1A6,2SF4.0)
(1A6,13F6.0/13F6.0/13F6.0/1F6.0)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
PLOT1	17.	0.	0.	8.	0.	0.	0.	0.	7.	0.	0.	37.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	32.
PLOT2	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	80.	0.	0.	17.	0.	0.	0.	0.	0.	0.	1.	0.	7.	27.
PLOT3	0.	0.	0.	12.	0.	4.	30.	0.	3.	0.	18.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	1.	24.	38.
PLOT4	0.	0.	0.	3.	0.	1.	21.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	6.	7.
PLOT5	0.	0.	0.	0.	0.	10.	60.	0.	3.	0.	0.	0.	0.	0.	7.	0.	0.	0.	0.	0.	0.	0.	0.	9.	1.
PLOT6	0.	0.	0.	0.	0.	4.	0.	0.	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	4.	42.
PLOT7	0.	0.	0.	0.	0.	3.	65.	0.	1.	0.	0.	0.	0.	0.	7.	0.	0.	0.	0.	0.	0.	1.	2.	16.	1.
PLOT8	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	1.	0.	0.	3.	11.	10.
PLOT9	0.	0.	0.	1.	0.	2.	5.	0.	4.	0.	1.	0.	2.	0.	0.	0.	0.	1.	0.	0.	0.	0.	7.	14.	12.
PLOT10	0.	0.	0.	3.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	8.	0.	0.	2.	0.	19.	23.
PLOT11	0.	0.	0.	0.	0.	9.	3.	1.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	15.	4.
PLOT12	0.	0.	0.	0.	0.	0.	3.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	12.	0.	13.	0.
PLOT13	0.	0.	0.	1.	0.	0.	0.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	30.	0.	0.	2.	0.	18.	7.
PLOT14	0.	0.	0.	0.	0.	7.	0.	2.	7.	0.	0.	20.	8.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	1.	27.
PLOT15	10.	0.	0.	19.	0.	2.	0.	2.	5.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	42.
PLOT16	0.	0.	0.	18.	0.	2.	21.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	74.	4.	7.
PLOT17	0.	0.	0.	0.	0.	20.	22.	0.	3.	0.	38.	0.	4.	4.	0.	0.	0.	0.	0.	0.	0.	0.	13.	11.	34.
PLOT18	0.	0.	0.	14.	0.	0.	0.	0.	70.	0.	1.	0.	10.	0.	0.	0.	1.	0.	0.	0.	0.	0.	68.	13.	0.
PLOT19	0.	0.	1.	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	11.	0.	0.	0.	0.	0.	0.	0.	0.	100.	1.	0.
PLOT20	0.	0.	1.	0.	0.	0.	0.	1.	85.	0.	0.	7.	0.	0.	6.	0.	0.	0.	0.	0.	4.	0.	0.	14.	0.
PLOT21	0.	0.	0.	0.	6.	0.	0.	0.	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	16.	0.	2.	15.
PLOT22	0.	0.	0.	4.	0.	4.	12.	0.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	25.	0.	7.	25.
PLOT23	0.	0.	0.	9.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	0.	0.	2.	0.	2.	31.
PLOT24	0.	0.	0.	9.	0.	0.	6.	0.	7.	0.	0.	0.	3.	0.	0.	0.	0.	60.	0.	0.	11.	0.	14.	19.	
PLOT25	0.	0.	0.	19.	0.	11.	22.	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	22.
PLOT26	0.	1.	0.	0.	0.	1.	10.	1.	21.	0.	0.	0.	4.	0.	0.	0.	1.	0.	0.	0.	19.	1.	2.	1.	
PLOT27	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	15.	0.	1.	0.	0.	5.	0.	0.	0.	0.	0.	4.	2.	0.	6.
PLOT28	0.	0.	0.	6.	0.	0.	4.	0.	0.	0.	0.	0.	1.	0.	0.	3.	0.	0.	0.	0.	0.	2.	0.	13.	0.
PLOT29	0.	0.	0.	1.	0.	0.	1.	0.	10.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.	0.	2.	16.	0.	33.
PLOT30	0.	0.	0.	2.	0.	2.	0.	0.	11.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	15.	0.	11.	9.
PLOT31	0.	0.	0.	21.	0.	0.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	26.	0.	18.	25.
PLOT32	0.	0.	0.	0.	0.	1.	0.	0.	2.	0.	0.	0.	0.	0.	20.	0.	0.	0.	0.	0.	0.	8.	0.	2.	39.
PLOT33	0.	0.	0.	7.	0.	3.	2.	1.	3.	0.	13.	10.	0.	4.	0.	0.	0.	0.	0.	0.	0.	9.	0.	0.	54.
PLOT34	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	10.	4.	0.
PLOT35	0.	0.	0.	0.	0.	1.	5.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	10.	0.	0.	8.	0.	0.	5.	
PLOT36	0.	0.	0.	12.	0.	5.	7.	0.	11.	0.	0.	23.	0.	0.	0.	0.	0.	0.	0.	0.	14.	0.	0.	4.	
PLOT37	8.	0.	0.	40.	0.	7.	19.	0.	2.	0.	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	25.
PLOT38	0.	0.	0.	8.	0.	6.	0.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	29.	9.	8.	
PLOT39	0.	0.	0.	16.	0.	20.	0.	0.	10.	0.	0.	12.	0.	0.	0.	0.	0.	0.	2.	0.	15.	0.	2.	4.	
PLOT40	0.	0.	0.	7.	0.	9.	0.	0.	27.	0.	0.	8.	0.	0.	0.	0.	0.	2.	0.	0.	2.	0.	0.	5.	

1=ACCI, 2=ACRU, 3=ANOR, 4=BENE, 5=CACH, 6=CHUM, 7=GASH, 8=GOOB, 9=LIBO, 10=PAMY, 11=POMU, 12=RHMA,
13=RUMI, 14=RUJR, 15=TABR, 16=THPL, 17=TRLA, 18=TROV, 19=TSHE, 20=VAPA, 21=VIOR, 22=TSHEBA, 23=THPLBA,
24=TABRBA, 25=ACCIBA

40 PLOTS
 15 VARIABLE
 (F5.0,4F4.0,7F6.0,F4.0,2F3.0)
 QQQQQQQQQQQCC

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
3.	0.	0.	0.	7.	62.	69.	13.	75.	-7.	88.	0.	5.	1.	2.
2.	2.	0.	0.	2.	80.	82.	40.	0.	-16.	83.	46.	0.	1.	2.
0.	0.	2.	0.	25.	42.	67.	20.	25.	-13.	84.	19.	0.	1.	2.
0.	0.	0.	0.	5.	24.	29.	12.	0.	-13.	61.	0.	0.	1.	2.
0.	0.	0.	0.	13.	60.	73.	18.	12.	-20.	68.	0.	0.	1.	2.
0.	0.	1.	1.	11.	0.	11.	12.	10.	-17.	86.	65.	0.	1.	2.
0.	0.	3.	0.	4.	65.	69.	7.	80.	-18.	91.	43.	5.	1.	2.
0.	2.	0.	3.	3.	1.	4.	3.	35.	-4.	85.	0.	0.	1.	1.
0.	2.	0.	2.	8.	8.	16.	5.	0.	-4.	85.	0.	0.	1.	1.
2.	0.	0.	0.	2.	3.	5.	5.	10.	-16.	87.	25.	0.	1.	2.
0.	3.	0.	2.	12.	3.	15.	13.	28.	-4.	84.	0.	0.	1.	1.
0.	2.	0.	0.	1.	3.	4.	9.	9.	-10.	88.	0.	0.	1.	2.
0.	0.	0.	0.	0.	5.	5.	7.	12.	-17.	86.	0.	0.	1.	2.
0.	0.	0.	0.	16.	28.	44.	5.	0.	-15.	87.	0.	0.	1.	2.
0.	0.	0.	0.	9.	31.	40.	14.	20.	-6.	89.	0.	2.	1.	2.
0.	0.	2.	0.	2.	39.	41.	30.	5.	-14.	81.	0.	1.	1.	2.
0.	1.	2.	0.	61.	30.	91.	5.	3.	-7.	86.	0.	0.	1.	2.
0.	2.	0.	0.	72.	24.	96.	3.	25.	-12.	81.	0.	0.	1.	2.
1.	2.	2.	0.	1.	7.	8.	3.	80.	-10.	85.	0.	5.	1.	2.
3.	2.	0.	0.	94.	8.	102.	4.	0.	-8.	83.	0.	0.	1.	2.
0.	0.	1.	0.	7.	6.	13.	50.	35.	6.	78.	0.	0.	0.	1.
0.	2.	0.	0.	7.	16.	23.	7.	11.	11.	86.	0.	1.	0.	0.
0.	0.	0.	0.	2.	10.	12.	16.	10.	14.	91.	47.	2.	0.	0.
0.	0.	1.	1.	7.	18.	25.	5.	10.	17.	54.	0.	0.	0.	0.
0.	0.	0.	0.	11.	41.	52.	5.	20.	26.	87.	30.	0.	0.	0.
0.	0.	0.	0.	25.	14.	39.	14.	20.	25.	83.	0.	1.	0.	0.
0.	0.	0.	2.	16.	1.	17.	8.	0.	20.	89.	0.	2.	0.	0.
0.	2.	0.	1.	0.	11.	11.	10.	0.	19.	88.	0.	0.	0.	0.
0.	0.	1.	0.	10.	2.	12.	23.	0.	6.	90.	60.	1.	0.	1.
0.	0.	0.	0.	13.	2.	15.	5.	0.	5.	85.	0.	0.	0.	1.
0.	0.	0.	0.	10.	21.	31.	6.	0.	3.	88.	0.	0.	0.	1.
0.	0.	0.	0.	3.	0.	3.	17.	23.	11.	87.	86.	2.	0.	0.
0.	0.	0.	0.	22.	21.	43.	80.	5.	11.	81.	100.	2.	0.	0.
0.	0.	0.	0.	0.	2.	2.	12.	90.	20.	88.	0.	2.	0.	0.
0.	0.	0.	0.	1.	5.	6.	15.	0.	15.	92.	47.	1.	0.	0.
3.	0.	0.	0.	16.	42.	58.	23.	20.	9.	82.	76.	0.	0.	0.
2.	0.	0.	0.	9.	79.	88.	10.	18.	12.	88.	17.	0.	0.	0.
0.	2.	0.	0.	9.	8.	17.	10.	20.	27.	81.	0.	0.	0.	0.
0.	0.	0.	0.	10.	30.	40.	8.	0.	26.	72.	35.	0.	0.	0.
0.	3.	0.	0.	36.	15.	51.	1.	80.	30.	80.	32.	0.	0.	0.

1=REGHM, 2=REGSH, 3=REGTAB, 4=REGTHP, 5=HERB, 6=SHRUB, 7=TOTAL, 8=FINE,
 9=COARSE, 10=DISTAN, 11=CANOPY, 12=PSME, 13=SLOPE, 14=2CAT, 15=3CAT

APPENDIX 4: Data for the Rooster Rock Site

39 PLOTS
24 SPECIES
(1A6,24F4.0)
(1A6,13F6.1/13F6.1/13F6.1)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
PLOT1	0.	0.	0.	1.	9.	0.	0.	9.	0.	0.	0.	4.	10.	0.	0.	0.	0.	0.	0.	0.	1.	0.	4.	2.
PLOT2	0.	0.	0.	0.	0.	0.	1.	1.	0.	0.	0.	6.	0.	95.	0.	1.	0.	0.	0.	0.	3.	0.	2.	8.
PLOT3	0.	0.	0.	0.	4.	0.	0.	6.	0.	0.	0.	60.	0.	0.	0.	4.	3.	0.	0.	0.	2.	0.	2.	2.
PLOT4	0.	0.	3.	1.	0.	0.	0.	2.	0.	0.	0.	40.	0.	0.	0.	1.	0.	0.	1.	0.	5.	0.	3.	0.
PLOT5	0.	0.	0.	3.	0.	0.	6.	1.	0.	2.	0.	80.	0.	8.	0.	0.	0.	2.	0.	0.	2.	0.	0.	9.
PLOT6	0.	0.	2.	5.	0.	0.	40.	1.	0.	0.	0.	20.	0.	0.	0.	3.	0.	0.	0.	2.	2.	0.	0.	9.
PLOT7	0.	0.	1.	0.	14.	0.	0.	0.	0.	0.	0.	0.	13.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	5.
PLOT8	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	70.	0.	0.	0.	1.	0.	1.	0.	1.	0.	3.	0.	0.	0.
PLOT9	0.	0.	0.	1.	4.	0.	0.	1.	0.	0.	0.	3.	1.	0.	1.	1.	0.	0.	0.	15.	0.	9.	3.	
PLOT10	0.	0.	3.	1.	14.	0.	0.	3.	6.	0.	0.	2.	4.	0.	0.	0.	0.	0.	0.	7.	0.	7.	0.	
PLOT11	13.	0.	0.	1.	0.	0.	3.	0.	0.	0.	0.	7.	0.	0.	0.	0.	0.	1.	0.	8.	5.	0.	0.	
PLOT12	3.	0.	0.	0.	6.	0.	4.	0.	0.	0.	0.	0.	3.	0.	0.	0.	0.	0.	0.	4.	2.	0.	1.	
PLOT13	4.	0.	0.	1.	0.	0.	0.	1.	0.	0.	0.	25.	0.	0.	0.	0.	0.	0.	0.	0.	14.	0.	0.	
PLOT14	0.	0.	0.	0.	0.	0.	55.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	1.	0.	0.	
PLOT15	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	80.	0.	0.	0.	0.	0.	6.	0.	0.	1.	0.	0.	0.
PLOT16	0.	0.	0.	1.	0.	0.	0.	3.	0.	0.	0.	2.	0.	2.	0.	0.	0.	0.	0.	1.	0.	0.	1.	
PLOT17	0.	0.	0.	0.	0.	0.	3.	1.	0.	0.	0.	18.	0.	0.	0.	0.	0.	0.	0.	6.	1.	3.	0.	
PLOT18	0.	0.	0.	2.	0.	0.	4.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	0.	5.	8.	
PLOT19	0.	0.	1.	0.	0.	0.	3.	0.	1.	0.	30.	20.	0.	0.	2.	0.	1.	0.	4.	10.	0.	0.	4.	
PLOT20	0.	0.	0.	0.	90.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
PLOT21	0.	0.	0.	1.	95.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	5.	0.
PLOT22	0.	0.	0.	1.	3.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.
PLOT23	0.	0.	0.	0.	25.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	2.	0.	0.	0.
PLOT24	0.	0.	0.	3.	90.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	6.	0.
PLOT25	0.	0.	0.	0.	100.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	8.
PLOT26	0.	11.	0.	1.	0.	0.	0.	0.	0.	0.	3.	0.	8.	0.	0.	0.	0.	0.	0.	4.	6.	3.	0.	
PLOT27	0.	0.	0.	0.	35.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.	1.	1.	0.	6.	
PLOT28	17.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	10.	19.	0.	0.	
PLOT29	0.	0.	0.	0.	10.	0.	50.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	11.	
PLOT30	0.	0.	0.	0.	23.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	5.	0.	0.	
PLOT31	0.	0.	0.	0.	0.	1.	0.	0.	1.	0.	0.	6.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	10.	
PLOT32	0.	0.	0.	0.	2.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	3.	10.	7.	0.	
PLOT33	0.	0.	0.	0.	55.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	8.	0.	
PLOT34	0.	0.	0.	0.	60.	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	5.	2.	6.	
PLOT35	0.	0.	0.	0.	60.	0.	9.	0.	0.	0.	0.	2.	0.	0.	3.	0.	0.	0.	1.	0.	0.	0.	0.	
PLOT36	0.	1.	0.	0.	14.	0.	40.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	
PLOT37	0.	0.	0.	1.	20.	0.	0.	0.	0.	0.	0.	0.	30.	0.	0.	0.	0.	0.	0.	1.	0.	2.	7.	
PLOT38	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	40.	0.	0.	0.	0.	0.	0.	0.	0.	8.	12.	0.	
PLOT39	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	55.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	6.	0.

1=ACCI, 2=ACTR, 3=ADBI, 4=ANOR, 5=BENE, 6=CHME, 7=GASH, 8=GATR, 9=GRAM, 10=HIAL, 11=LIBO, 12=POMU, 13=PTAQ, 14=RHMA, 15=RUNI, 16=RUUR, 17=SMRA, 18=TRLA, 19=VAHE, 20=VAPA, 21=VIOR, 22=ACCIBA, 23=ACHABA, 24=RHMABA

39 PLOTS
 13 VARIABLE
 (F5.0,9F6.0,2F3.0,F5.2)
 QQQQQQQQCCCA

1	2	3	4	5	6	7	8	9	10	11	12	13
0.	0.	25.	9.	34.	3.	50.	-4.	72.	2.	1.	1.	3.20
2.	0.	10.	2.	12.	15.	40.	-4.	86.	2.	1.	1.	.00
0.	0.	71.	8.	79.	20.	0.	-3.	71.	1.	1.	1.	3.20
1.	0.	51.	2.	53.	10.	20.	-7.	77.	0.	1.	2.	.00
2.	2.	90.	14.	104.	40.	10.	-7.	72.	2.	1.	2.	.00
2.	0.	30.	45.	75.	5.	20.	-5.	70.	1.	1.	1.	11.80
0.	0.	16.	14.	30.	85.	15.	-1.	80.	0.	1.	1.	17.40
0.	2.	75.	1.	76.	90.	10.	-8.	74.	0.	1.	2.	8.90
0.	0.	21.	6.	27.	15.	30.	-3.	83.	2.	1.	1.	1.80
0.	0.	26.	14.	40.	7.	20.	-5.	74.	2.	1.	2.	.00
0.	0.	20.	13.	33.	14.	30.	0.	89.	2.	1.	1.	16.70
0.	0.	7.	13.	20.	10.	25.	-5.	75.	0.	1.	2.	21.60
0.	0.	27.	4.	31.	10.	20.	0.	85.	0.	1.	1.	13.90
0.	0.	4.	55.	59.	85.	10.	0.	71.	1.	1.	1.	17.60
0.	0.	87.	0.	87.	50.	10.	-4.	76.	2.	1.	1.	15.60
0.	0.	9.	2.	11.	0.	0.	-5.	80.	5.	1.	2.	10.20
0.	0.	28.	0.	28.	7.	55.	-5.	58.	0.	1.	1.	2.30
0.	0.	16.	0.	16.	10.	50.	-4.	80.	1.	1.	1.	3.50
0.	2.	66.	6.	72.	60.	7.	-9.	73.	2.	1.	2.	8.90
0.	0.	1.	90.	91.	30.	5.	5.	72.	2.	0.	1.	15.90
0.	0.	1.	95.	96.	5.	10.	12.	82.	2.	0.	0.	9.20
0.	0.	2.	13.	15.	90.	0.	7.	80.	0.	0.	0.	31.00
0.	0.	3.	27.	30.	80.	0.	20.	84.	2.	0.	0.	24.40
0.	0.	4.	90.	94.	3.	25.	9.	81.	0.	0.	0.	16.30
0.	0.	0.	110.	110.	15.	0.	6.	80.	0.	0.	0.	29.50
1.	2.	27.	0.	27.	25.	10.	11.	81.	1.	0.	0.	21.20
2.	0.	3.	35.	38.	20.	30.	15.	79.	1.	0.	0.	16.00
2.	0.	10.	21.	31.	14.	15.	15.	84.	1.	0.	0.	17.70
2.	2.	0.	60.	60.	10.	15.	23.	78.	1.	0.	0.	27.40
0.	0.	3.	23.	26.	10.	7.	24.	75.	0.	0.	0.	22.50
2.	0.	7.	1.	8.	75.	10.	16.	76.	1.	0.	0.	16.10
0.	0.	5.	2.	7.	10.	0.	7.	84.	2.	0.	0.	24.60
0.	0.	3.	55.	58.	50.	10.	21.	77.	2.	0.	0.	4.90
0.	0.	0.	72.	72.	25.	0.	16.	84.	1.	0.	0.	21.70
0.	1.	3.	72.	75.	29.	15.	30.	79.	1.	0.	0.	25.90
0.	0.	1.	54.	55.	13.	7.	21.	82.	2.	0.	0.	20.70
0.	0.	2.	50.	52.	20.	11.	14.	88.	0.	0.	0.	11.90
0.	0.	40.	0.	40.	13.	20.	7.	84.	1.	0.	0.	8.60
0.	0.	56.	0.	56.	30.	0.	5.	84.	2.	0.	0.	27.30

1=REGHM, 2=REGACH, 3=HERB, 4=SHRUB, 5=TOTAL, 6=FINE, 7=COARSE, 8=DISTAN,
 9=CANOPY, 10=SLOPE, 11=2CAT, 12=3CAT, 13=PSME

APPENDIX 5: Data for the Mary's Peak Site

40 PLOTS
24 SPECIES
(1A6,24F6.0)
(1A6,14F6.0/14F6.0/12F6.0)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	
PLOT1	0.	0.	2.	0.	0.	5.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	68.	0.	10.	5.	2.	0.	
PLOT2	0.	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	39.	0.	0.
PLOT3	7.	0.	0.	0.	0.	60.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	36.	1.	0.	
PLOT4	0.	0.	6.	0.	0.	7.	0.	0.	0.	0.	13.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	13.	12.	4.	
PLOT5	7.	0.	18.	0.	0.	15.	0.	0.	0.	0.	13.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	30.	14.	0.	
PLOT6	3.	0.	4.	0.	0.	35.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	21.	8.	27.	
PLOT7	0.	0.	14.	0.	0.	7.	1.	0.	0.	0.	0.	7.	0.	1.	0.	0.	0.	0.	0.	0.	0.	5.	10.	20.	
PLOT8	0.	0.	7.	0.	0.	3.	0.	0.	0.	0.	10.	3.	0.	2.	0.	0.	0.	0.	0.	0.	0.	20.	10.	27.	
PLOT9	0.	0.	3.	0.	0.	9.	0.	0.	3.	0.	0.	1.	0.	0.	0.	1.	0.	0.	0.	0.	0.	39.	3.	0.	
PLOT10	0.	0.	40.	0.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	23.	19.	30.	
PLOT11	0.	0.	20.	0.	0.	10.	0.	0.	0.	0.	40.	8.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	9.	45.	
PLOT12	0.	0.	40.	0.	0.	4.	0.	0.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	4.	22.	
PLOT13	0.	2.	30.	0.	0.	95.	0.	0.	0.	0.	0.	5.	9.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	1.	
PLOT14	9.	0.	23.	0.	0.	90.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	2.	0.	
PLOT15	0.	0.	35.	0.	0.	6.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	35.	4.	0.	
PLOT16	0.	0.	10.	0.	10.	90.	0.	0.	10.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	
PLOT17	0.	0.	11.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	11.	6.	0.	
PLOT18	0.	0.	14.	0.	0.	11.	0.	0.	2.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	2.	0.	
PLOT19	0.	0.	13.	0.	0.	3.	0.	0.	0.	0.	37.	0.	5.	0.	0.	0.	0.	0.	0.	0.	0.	8.	2.	3.	7.
PLOT20	0.	0.	30.	0.	0.	0.	0.	0.	0.	0.	0.	4.	1.	0.	0.	0.	0.	0.	0.	0.	10.	11.	8.	0.	
PLOT21	0.	0.	80.	0.	0.	20.	0.	0.	0.	0.	0.	7.	13.	1.	3.	0.	0.	0.	0.	0.	0.	0.	2.	0.	
PLOT22	0.	0.	5.	0.	0.	95.	0.	0.	0.	0.	0.	0.	6.	0.	0.	0.	0.	0.	0.	0.	1.	4.	3.	0.	
PLOT23	0.	0.	10.	0.	0.	30.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	
PLOT24	3.	0.	9.	0.	0.	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	14.	0.	1.	
PLOT25	0.	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	2.	4.	0.	0.	0.	
PLOT26	0.	0.	30.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	5.	3.	0.	0.	
PLOT27	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	0.	0.	0.	
PLOT28	0.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	5.	0.	0.	0.	
PLOT29	0.	0.	15.	0.	0.	1.	0.	0.	0.	0.	20.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.	0.	
PLOT30	11.	0.	17.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.	0.	
PLOT31	0.	0.	11.	0.	0.	0.	1.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	1.	0.	0.	
PLOT32	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	0.	4.	4.	0.	0.	0.	
PLOT33	10.	0.	15.	0.	0.	0.	0.	0.	0.	9.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	11.	3.	0.	0.	
PLOT34	0.	0.	4.	0.	0.	0.	0.	0.	0.	4.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	11.	0.	0.	
PLOT35	0.	0.	10.	0.	0.	0.	0.	0.	0.	12.	0.	0.	0.	0.	0.	0.	20.	0.	0.	0.	0.	0.	0.	0.	
PLOT36	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	1.	0.	0.	
PLOT37	0.	0.	4.	0.	0.	0.	0.	0.	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	10.	7.	1.	0.	
PLOT38	0.	0.	20.	0.	0.	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	8.	21.	0.	0.	
PLOT39	0.	0.	12.	0.	0.	0.	0.	0.	0.	50.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	3.	17.	2.	0.	
PLOT40	0.	0.	10.	0.	0.	7.	0.	0.	0.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	4.	9.	12.	0.	

1=ACCI, 2=ADBI, 3=BENE, 4=CACH, 5=COCO, 6=GASH, 7=GATR, 8=GRAM, 9=HODI, 10=LIBO, 11=POMU, 12=PSME, 13=PTAQ, 14=ROGY, 15=SMRA, 16=SYMU, 17=TRLA, 18=TROV, 19=TSHE, 20=VAME, 21=TSHEBA, 22=ACCIBA, 23=HODIBA, 24=COCOBA

40 PLOTS
 12 VARIABLE
 (F5.0,8F6.0,2F3.0,F6.0)
 QQQQQQQCCCA

1	2	3	4	5	6	7	8	9	10	11	12
0.	53.	6.	59.	10.	3.	-7.	88.	2.	1.	2.	0.
0.	0.	10.	10.	65.	3.	-9.	88.	0.	1.	2.	6.
2.	35.	4.	39.	12.	0.	-2.	89.	0.	1.	1.	24.
2.	4.	27.	31.	10.	60.	-4.	89.	5.	1.	1.	0.
2.	9.	5.	14.	15.	21.	-2.	66.	1.	1.	1.	0.
2.	26.	0.	26.	9.	35.	-7.	90.	0.	1.	2.	0.
1.	18.	3.	21.	36.	3.	-7.	89.	0.	1.	2.	5.
0.	8.	0.	8.	65.	25.	-3.	85.	1.	1.	1.	16.
0.	0.	3.	3.	0.	100.	-9.	86.	5.	1.	2.	0.
2.	0.	7.	7.	25.	30.	-19.	87.	2.	1.	2.	16.
2.	92.	0.	92.	35.	3.	-17.	77.	1.	1.	2.	25.
0.	52.	6.	58.	13.	17.	-5.	82.	0.	1.	1.	0.
0.	13.	9.	22.	30.	5.	-10.	85.	1.	1.	2.	5.
0.	0.	10.	10.	13.	10.	0.	84.	5.	1.	1.	12.
0.	10.	24.	34.	7.	15.	-8.	86.	1.	1.	2.	5.
0.	1.	35.	36.	12.	55.	-6.	79.	0.	1.	2.	0.
0.	10.	34.	44.	20.	25.	-5.	88.	0.	1.	1.	0.
1.	0.	3.	3.	10.	75.	0.	82.	5.	1.	1.	12.
0.	9.	3.	12.	22.	6.	-3.	88.	0.	1.	1.	0.
2.	2.	19.	21.	7.	0.	-7.	79.	0.	1.	2.	0.
0.	0.	2.	2.	13.	0.	12.	86.	2.	0.	0.	22.
0.	0.	9.	9.	8.	0.	24.	85.	3.	0.	0.	32.
0.	0.	28.	28.	7.	0.	25.	84.	2.	0.	0.	24.
0.	0.	55.	55.	20.	30.	15.	83.	2.	0.	0.	36.
2.	0.	1.	1.	5.	60.	9.	91.	1.	0.	0.	12.
0.	0.	12.	12.	40.	17.	3.	88.	2.	0.	1.	25.
0.	80.	16.	96.	7.	16.	6.	88.	2.	0.	0.	15.
0.	83.	0.	83.	30.	7.	0.	88.	0.	0.	1.	8.
0.	0.	0.	0.	40.	10.	15.	87.	0.	0.	0.	28.
3.	13.	0.	13.	22.	0.	7.	89.	1.	0.	0.	13.
0.	9.	30.	39.	22.	22.	1.	88.	2.	0.	1.	11.
0.	8.	3.	11.	6.	25.	9.	87.	2.	0.	0.	30.
0.	9.	9.	18.	14.	0.	17.	86.	0.	0.	0.	32.
2.	0.	25.	25.	30.	10.	20.	89.	3.	0.	0.	30.
0.	0.	2.	2.	15.	80.	21.	89.	5.	0.	0.	31.
0.	14.	4.	18.	14.	12.	15.	88.	3.	0.	0.	25.
0.	0.	83.	83.	22.	22.	24.	86.	2.	0.	0.	31.
0.	0.	21.	21.	80.	40.	26.	85.	5.	0.	0.	33.
0.	0.	35.	35.	28.	0.	28.	86.	2.	0.	0.	24.
0.	5.	9.	14.	25.	35.	13.	85.	2.	0.	0.	23.

1=REGTSH, 2=HERB, 3=SHRUB, 4=TOTAL, 5=FINE, 6=COARSE, 7=DISTAN,
 8=CANOPY, 9=SLOPE, 10=2CAT, 11=3CAT, 12=PSME

APPENDIX 6: Data for the Alsea Site

40 PLOTS
15 SPECIES
(1A6,15F4.0)

(1A6,13F6.0/13F6.0/13F6.0/1F6.0)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
PLOT1	0.	4.	0.	3.	50.	0.	0.	0.	0.	0.	0.	0.	2.	4.	0.
PLOT2	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	9.	24.
PLOT3	0.	2.	0.	0.	35.	0.	0.	2.	0.	0.	0.	0.	0.	30.	30.
PLOT4	0.	20.	0.	0.	0.	0.	3.	4.	0.	0.	0.	0.	0.	3.	0.
PLOT5	0.	5.	1.	3.	5.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.
PLOT6	0.	0.	0.	1.	25.	0.	0.	0.	0.	0.	0.	0.	0.	33.	0.
PLOT7	0.	1.	0.	2.	16.	0.	0.	1.	0.	0.	0.	0.	1.	4.	0.
PLOT8	0.	0.	0.	0.	8.	0.	0.	0.	0.	0.	0.	0.	0.	8.	0.
PLOT9	0.	3.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	32.	0.
PLOT10	0.	7.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	5.
PLOT11	0.	0.	0.	0.	92.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
PLOT12	0.	3.	0.	0.	52.	0.	0.	3.	0.	0.	0.	0.	0.	0.	1.
PLOT13	0.	7.	0.	13.	0.	0.	0.	2.	0.	0.	0.	0.	0.	7.	17.
PLOT14	0.	10.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	23.
PLOT15	0.	2.	0.	0.	10.	0.	0.	2.	0.	0.	0.	0.	20.	7.	38.
PLOT16	0.	33.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.	2.	0.	61.
PLOT17	0.	33.	0.	0.	10.	0.	0.	0.	0.	0.	0.	0.	1.	0.	37.
PLOT18	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	10.	14.
PLOT19	0.	3.	0.	0.	9.	0.	0.	0.	0.	0.	0.	0.	0.	18.	21.
PLOT20	0.	13.	0.	0.	0.	0.	3.	3.	0.	2.	0.	0.	0.	0.	72.
PLOT21	0.	0.	0.	0.	0.	8.	0.	2.	0.	0.	0.	0.	0.	7.	0.
PLOT22	8.	0.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	3.	0.
PLOT23	11.	16.	0.	0.	0.	0.	0.	1.	0.	0.	0.	0.	0.	13.	0.
PLOT24	10.	45.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.
PLOT25	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	8.	0.
PLOT26	0.	12.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	17.	7.
PLOT27	0.	16.	0.	0.	80.	0.	0.	0.	0.	0.	0.	0.	0.	19.	22.
PLOT28	0.	0.	0.	0.	82.	0.	0.	0.	0.	0.	1.	0.	0.	31.	0.
PLOT29	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	0.
PLOT30	0.	2.	0.	0.	10.	0.	0.	1.	0.	0.	0.	2.	0.	5.	18.
PLOT31	23.	0.	0.	0.	9.	30.	0.	7.	0.	0.	0.	0.	0.	14.	0.
PLOT32	0.	3.	0.	0.	6.	0.	0.	0.	0.	1.	1.	0.	0.	2.	11.
PLOT33	0.	9.	0.	9.	0.	0.	0.	0.	0.	0.	0.	0.	0.	1.	0.
PLOT34	0.	25.	0.	0.	0.	0.	0.	0.	0.	0.	0.	7.	0.	4.	0.
PLOT35	0.	2.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	2.	0.
PLOT36	0.	4.	0.	0.	14.	0.	0.	0.	0.	0.	0.	0.	0.	4.	0.
PLOT37	4.	75.	0.	0.	0.	0.	0.	4.	0.	0.	0.	0.	0.	13.	0.
PLOT38	0.	1.	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.	20.	2.	0.
PLOT39	10.	25.	0.	0.	0.	5.	0.	0.	0.	0.	0.	0.	0.	1.	0.
PLOT40	0.	8.	0.	0.	5.	0.	0.	1.	0.	0.	0.	0.	0.	1.	3.

1=BENE, 2=GASH, 3=GASP, 4=OXOR, 5=POMU, 6=PSME, 7=ROGY, 8=RUNI, 9=RUUR, 10=TRLA,
11=TROV, 12=TSHE, 13=VAPA, 14=TSHEBA, 15=ACCIBA

40 PLOTS
 11 ATTRIBU
 (F5.0,7F6.0,F5.0,F3.0,F6.0)
 CCCCCCCCCC

1	2	3	4	5	6	7	8	9	10	11
0.	-5.	86.	10.	30.	0.	8.	8.	1.	2.	0.
0.	-9.	92.	0.	23.	0.	12.	12.	1.	2.	0.
0.	-6.	91.	5.	9.	1.	67.	68.	1.	2.	1.
0.	-5.	88.	35.	25.	14.	13.	27.	1.	2.	1.
0.	-5.	89.	0.	8.	13.	40.	53.	1.	2.	0.
0.	-7.	89.	0.	15.	2.	42.	44.	1.	2.	0.
0.	-3.	88.	0.	20.	1.	22.	23.	1.	1.	1.
0.	-5.	90.	0.	10.	10.	12.	22.	1.	1.	1.
0.	-4.	86.	0.	14.	2.	15.	17.	1.	1.	0.
0.	-9.	86.	10.	10.	0.	45.	45.	1.	2.	0.
0.	-11.	86.	0.	8.	40.	30.	70.	1.	2.	0.
0.	-7.	92.	0.	7.	10.	44.	54.	1.	2.	0.
0.	-1.	90.	0.	10.	12.	125.	137.	1.	1.	0.
0.	-2.	82.	0.	8.	1.	122.	123.	1.	1.	10.
0.	-7.	88.	0.	7.	0.	41.	41.	1.	2.	0.
1.	-2.	48.	25.	5.	0.	122.	122.	1.	1.	0.
2.	-4.	89.	27.	15.	0.	11.	11.	1.	1.	0.
1.	-2.	88.	9.	25.	3.	25.	28.	1.	1.	0.
0.	-2.	89.	0.	5.	42.	16.	58.	1.	1.	0.
0.	-6.	90.	0.	5.	4.	31.	35.	1.	2.	0.
0.	9.	84.	0.	30.	8.	116.	124.	0.	0.	34.
1.	5.	86.	15.	80.	0.	106.	106.	0.	0.	17.
2.	12.	82.	0.	100.	0.	40.	40.	0.	0.	51.
1.	25.	88.	30.	85.	0.	19.	19.	0.	0.	31.
0.	6.	87.	30.	40.	1.	12.	13.	0.	0.	1.
2.	12.	89.	0.	20.	1.	30.	31.	0.	0.	1.
1.	15.	89.	0.	95.	0.	1.	1.	0.	0.	1.
0.	6.	89.	0.	90.	0.	4.	4.	0.	0.	1.
0.	24.	91.	0.	35.	20.	16.	36.	0.	0.	2.
0.	37.	85.	15.	45.	0.	31.	31.	0.	0.	16.
1.	36.	90.	15.	35.	2.	11.	13.	0.	0.	51.
1.	30.	89.	5.	60.	0.	1.	1.	0.	0.	2.
0.	10.	86.	7.	20.	9.	25.	34.	0.	0.	1.
0.	10.	87.	5.	80.	4.	4.	8.	0.	0.	4.
2.	3.	89.	25.	15.	12.	10.	22.	0.	1.	4.
5.	0.	89.	100.	3.	0.	0.	0.	0.	1.	0.
1.	8.	90.	40.	25.	2.	4.	6.	0.	0.	0.
0.	1.	90.	0.	85.	1.	20.	21.	0.	1.	6.
0.	12.	90.	10.	25.	50.	12.	62.	0.	0.	0.
1.	6.	90.	10.	25.	3.	18.	21.	0.	0.	0.

1=SLOPE, 2=DISTAN, 3=CANOPY, 4=COARSE, 5=FINE, 6=HERB, 7=SHRUB,
 8=TOTAL, 9=2CAT, 10=3CAT, 11=PSME

Appendix 7: Location of the six infection centers examined are illustrated in Figures A.1 - A.6. Listed below each are road directions for each site including township, range, and section numbers.

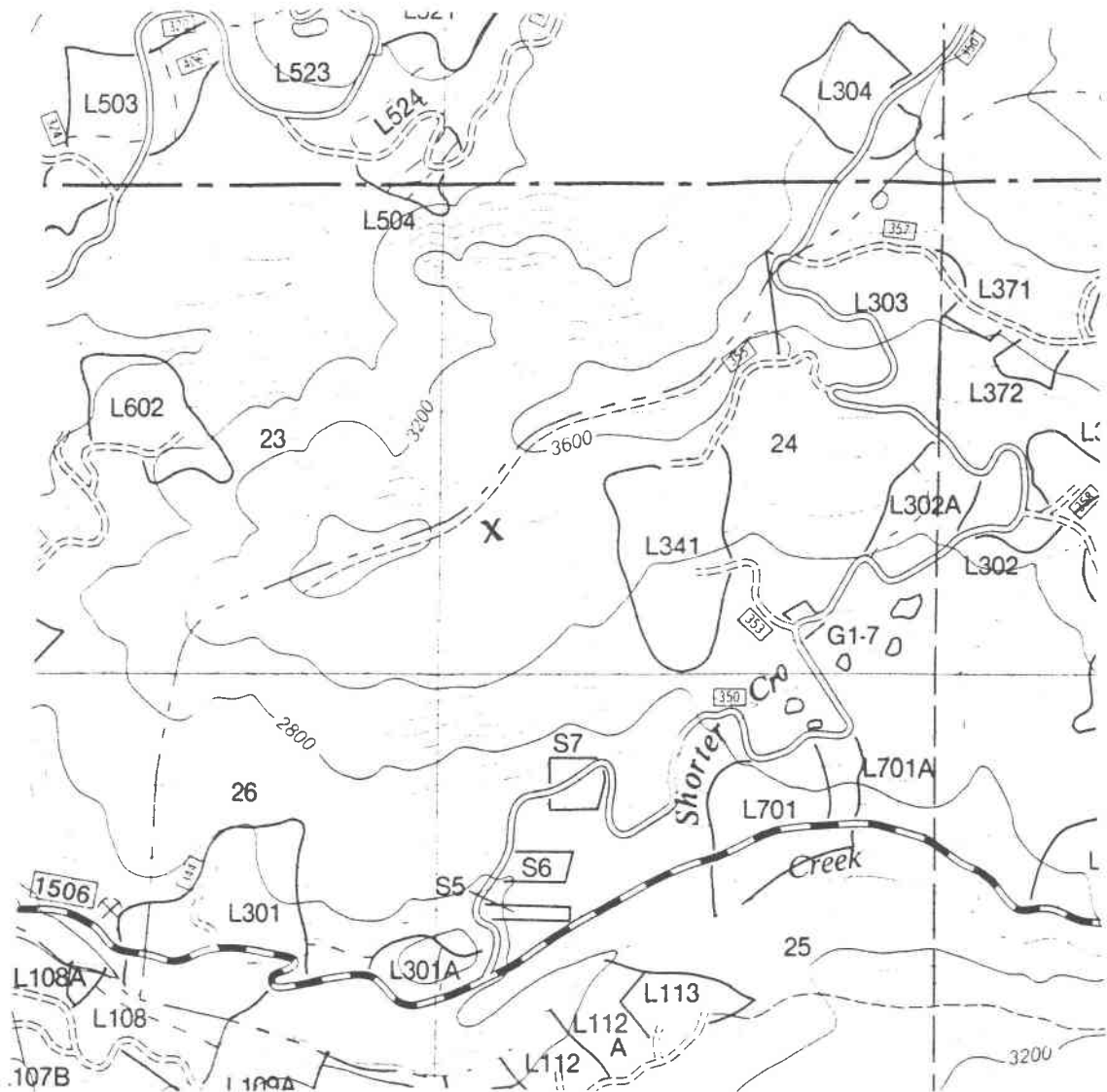


Figure A.1: HJA 1 (T. 15 S., R. 5 E., S. 24): From Eugene take 126 east to Blue River. Approximately 2 miles from Blue River take a left at the sign for the Blue River Reservoir, follow this road (15) to H.J. Andrew Experimental Forest. Turn right on road 1506 (Lookout Creek Road) until it intersects with road 350 (approximately 5.5 miles) on the left. Follow 350 for approximately 2 miles until it intersects with road 355 on the left. Just after turning on 355, a foot trail begins in the old-growth on the right-hand side. Follow this trail for approximately .75 miles to get to site.

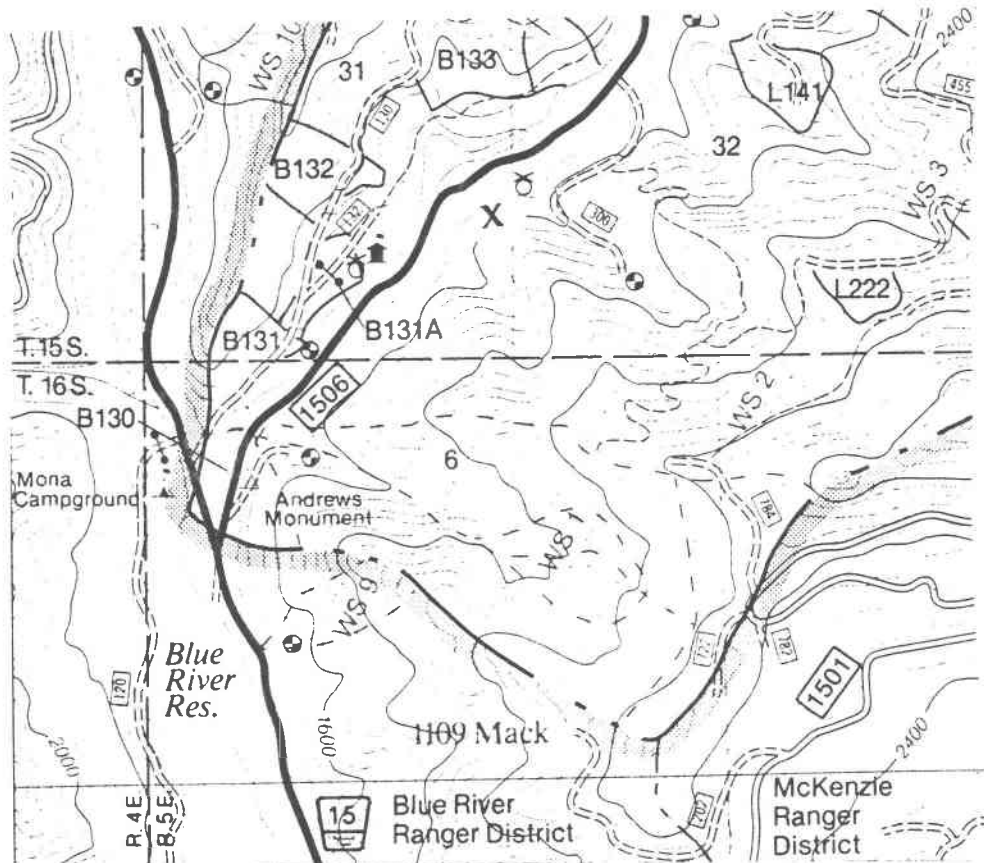


Figure A.2: HJA 2 (T. 15 S., R. 5 E., S. 32): Follow directions given above to H. J. Andrews Experimental Forest. Turn right on Road 1506 and take the second right (approximately 1 mile) onto a short gravel road. The site is located on the left-hand side of this road.

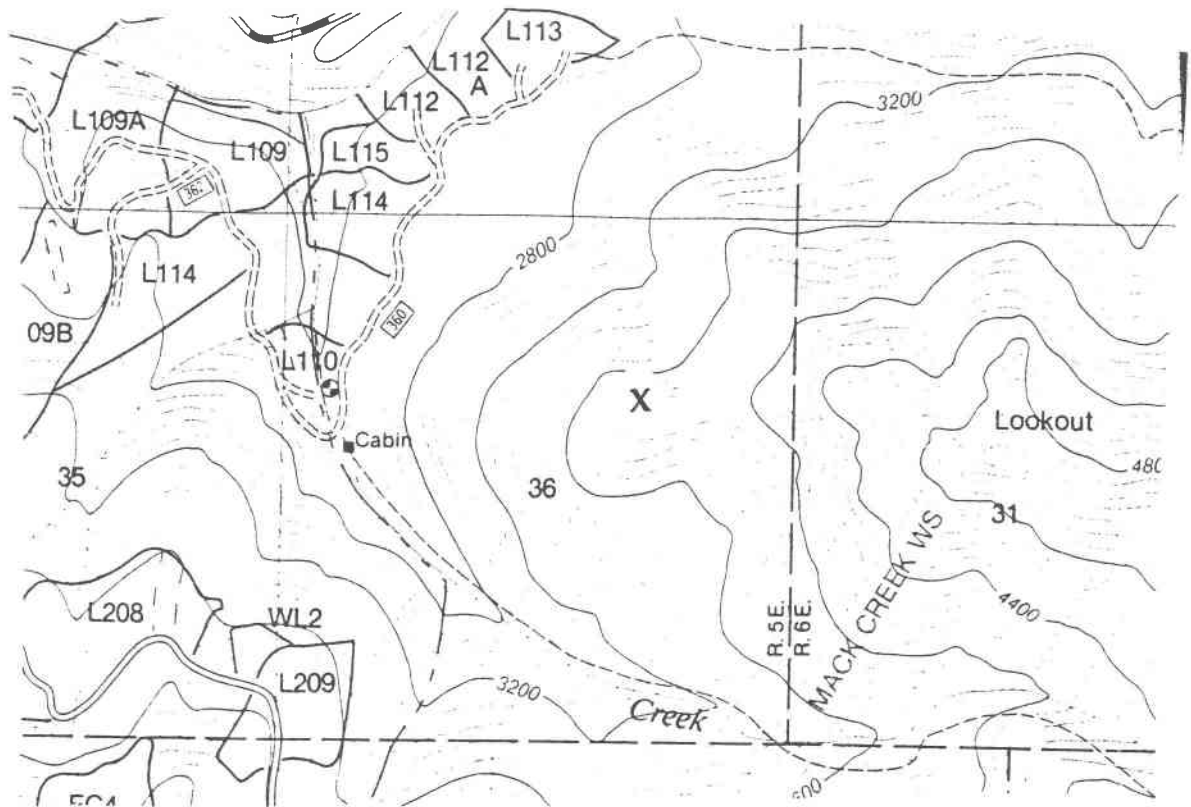


Figure A.3: HIA 3 (T. 15 S., R. 5 E., S. 36): Follow directions to H.J. Andrews Experimental Forest as given previously. Turn right on road 1506 and continue on this road until it intersects with road 360 (gated) on the right (approximately 2.5 miles). Continue on road 360 past the cabin (approximately 3 miles). To get to the site, enter the old-growth area on the right-hand side of the road just past the bend in the road near the cabin, where the terrain is nearly level. Infection center is located due east, approximately .5-.75 miles.

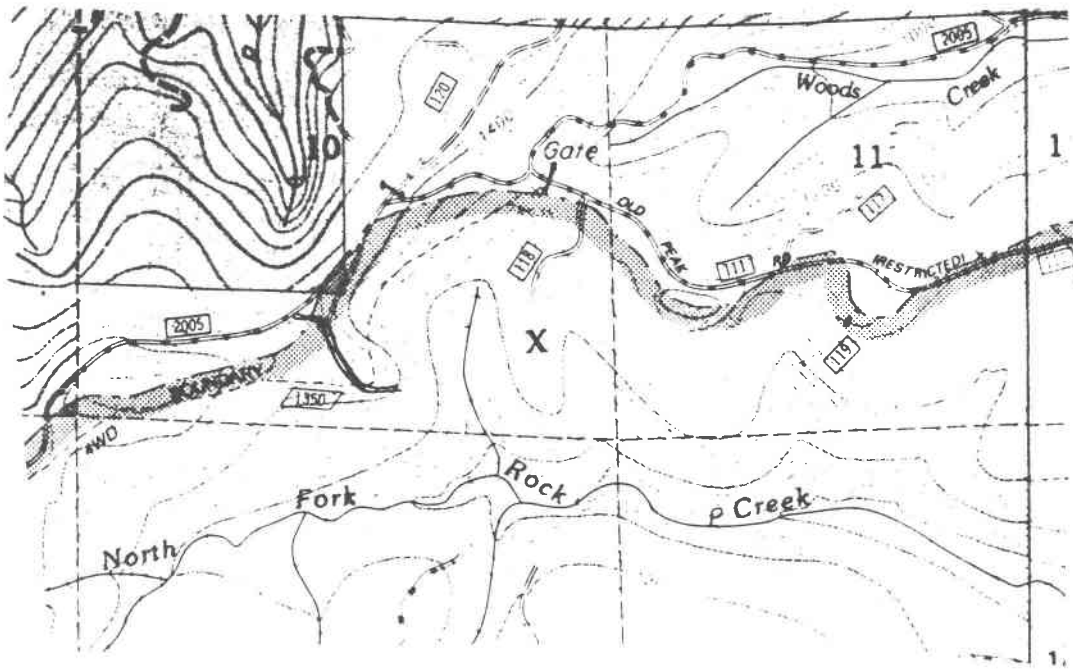


Figure A. 5: Mary's Peak site (T. 12 S., R. 7 W., S. 10): From Philomath, take highway 20 towards Newport approximately 1.6 miles, turn left onto Woods Creek Road. Follow this road until it intersects with Old Peak Road (approximately 6 miles), which is gated. From this road take the first right, road 118. The site is located in the old-growth found south of the end of road 118.

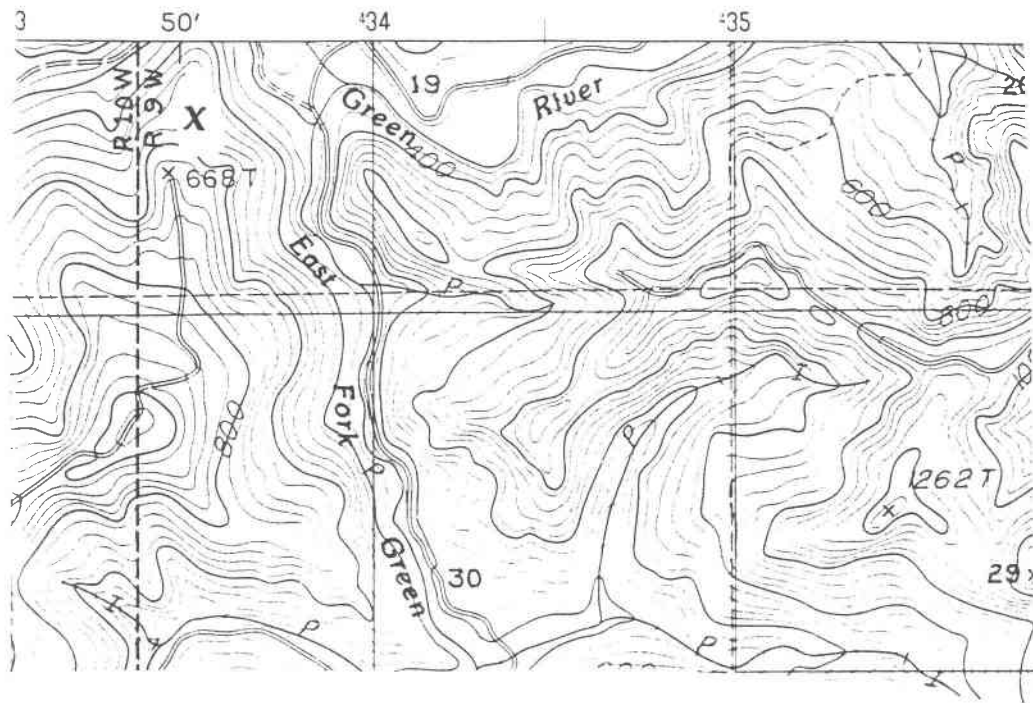


Figure A.6: Alsea site (T. 15 S., R. 8 W., S.19): From Alsea, take the road towards Alsea Falls. stay on this paved road (2015) until it intersects with road 35. Take a left on road 35 and stay on this road until pavement ends (approximately 2 miles). Infection center is located approximately .25 miles west of this point.