The interaction between competition in interior Douglas-fir plantations and disease caused by *Armillaria ostoyae* in British Columbia

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Abstract

Interior Douglas-fir trees in plantations were assessed for size differences related to the level of diseased neighbours infected with *Armillaria ostoyae*. The four Douglas-fir stands studied ranged from 25- to 34-years-old, and represented the oldest accessible planted stands in the Interior Cedar Hemlock (ICH) ecosystem in British Columbia. Twenty-three to 25, 10-m radius plots were established in each stand. The spatial coordinates, total height, and diameter at breast height of all live and dead trees in the plots were recorded. Subject trees whose competitors were contained in the 10-m radius plots were also identified. Trees were pulled out of the soil using a mechanical excavator and the root systems were surveyed for evidence of infection by *A. ostoyae*. Stem disks were taken from each tree at 1.3 m above the ground for a determination of basal area. Increasing proportion of diseased trees in the plots resulted in less total plot basal area,,but did not affect the mean basal area or height. Individual subject tree basal area was negatively related to the level of disease in surrounding competitors, opposite to expectations; however, diseased subject trees had reduced height and basal area compared to disease-free subject trees. Increasing competition reduced both the height and basal area of the trees, while regular distribution of all trees increased both total and mean plot basal area but not height. Disease incidence at the plot level and in individual subject trees was mainly affected by the neighbourhood conditions in which it grew, and was also related to disease intensity in the tree root systems. Although disease may alter resource partitioning among trees, the utilization of these resources is mostly limited by the increasing disease incidence as the stands age, the higher probability of larger trees being diseased with time, the occurrence of dead trees in clumps, and the high probability that dead trees will eventually infect live neighbours. The widespread belowground incidence of *A. ostoyae* in the ICH, its rapid colonization of stumps, and its wide host range can reduce site potential in managed stands.

Keywords: Root disease, conifer, disease impacts and control, compensatory growth, spatial epidemiology, disease incidence, stand density.

1 Introduction

Information concerning impact of pests in managed and unmanaged areas is used to allocate resources to control pests, to determine treatment thresholds, and to calculate the economic impact and risk of pests for industrial, ecological, and social concerns. Losses due to pests are usually determined as a damage function relating percent loss to the level of pest damage (Walker, 1983). The damage function is critical for determining thresholds for control and for calculating economic impacts. One factor that can affect the shape of the damage function is the ability of surviving plants to compensate for losses by increased growth. Compensation growth can occur as regrowth of damaged individuals such as after browsing, but more commonly with insects and disease this occurs at the neighbourhood level due to changes in competitive interference.

Competition is usually defined as the reciprocal negative interactions between two organisms, but can also include apparent competition where two species interact through a shared enemy or where different species interact through multiple plant species (Connell, 2003). Competition can exert its effect in a population through changes in resource availability or the ability to tolerate changes in resources. Disease can affect plant competition by changing the way resources are partitioned among individuals. Disease-free plants may have competitive advantage if resources become available because of their infected or dead neighbours. The effect of disease on site productivity may not represent a simple loss of growth in diseased plants if healthy neighbours can compensate for some losses.

Compensation growth has been recognized in agricultural settings for some time (Kirkpatrick and Blodgett, 1943; de Wit, 1960). More recently studies have reported that disease-free annual plants were able to compensate for yield losses in diseased neighbours (Adams and Lapwood,1983; Friess and Maillet, 1996; Mihail et al., 1998), but plants weakened by infection are not always able to compensate for the reduced growth of infected neighbours (Adams and Lapwood, 1983). In mixed stands of Douglas-fir [*Pseudotsuga menziesii* var. glauca (Beissn.) Franco] and lodgepole pine (*Pinus contorta* Dougl. ex Loud. var. latifolia Engelm.), growth of Douglas-fir increased after the pine was attacked by insects (Heath and Alfaro, 1990). Similarly, growth efficiency of disease-free trees in stands affected by Phellinus root disease increased compared to a stand with no disease (Oren et al., 1985). Disease-free trees in stands with Annosus root disease had no additional growth associated with diseased neighbours (Bendz-Hellgren and Stenlid, 1997).

Armillaria ostoyae (Romagn.) Herink causes Armillaria root disease of conifers in western North America (Wargo and Shaw, 1985; Kile et al., 1991). The fungus is widespread in the northern hemisphere, and can infect most tree species (Kile et

al., 1991). In the southern interior of British Columbia (BC), Armillaria root disease is found in most biogeoclimatic zones (Braumandl and Curran, 1992; Lloyd et al., 1990), but is most problematic in the Interior Cedar Hemlock (ICH) biogeoclimatic zone. The ICH zone is second only to the Coastal Western Hemlock zone in productivity within BC and Canada and has the highest diversity of tree species within BC (Meidinger and Pojar, 1991). The incidence of infection increases slowly over time and causes mortality and understocked openings, with a peak in mortality around stand age 15-20 (Morrison and Pellow, 1994). Since the fungus spreads slowly via root to root contact and rhizomorphs in the soil, only some trees are infected at any one time (Morrison et al., 2000) and only a smaller percentage of these actually die from an initial infection (Cruickshank et al., 1997; Morrison et al., 2000). By stand age 30, this results in a mix of dead trees, living diseased trees, and disease-free trees in stands (Morrison et al., 2000). Inoculum is long-lived, and may survive for decades in stumps (Roth et al., 1980).

Many interior Douglas-fir plantations have been established over the last 40 years in the southern interior of BC where the impacts of the disease are not well known. Losses in growth and mortality may be overestimated if disease-free trees can compensate for some of the production loss in diseased neighbours. Infected mature interior Douglas-fir trees suffer mortality and reduced growth after infection (Bloomberg and Morrison, 1989), but disease-free trees may be released from competitive interference by dead and diseased neighbours. The current study addresses the hypothesis that disease in interior Douglas-fir can alter intraspecific tree interactions. This was tested in individual trees (subject trees) and in groups of disease-free trees growing in neighbourhoods with varying disease incidence.

2 Materials and Methods

2.1) Sites, plot locations, and tree measurements

Four, 25- to 34-year-old Douglas-fir plantations in south-central BC (in the ICH biogeoclimatic zone) were identified for sampling. Separated by at least 50 km, these sites were located at Chuck Creek (CC) near Clearwater, East Barriere (EB) near Barriere, Kingfisher (KF) near Enderby, and Kuskanax (KX) near Nakusp (Table 1). Site selection was limited to areas with access roads that would accept a lowbed trailer carrying a 20-ton excavator, and to areas that would permit excavator travel on site. Three sites (CC, EB, and KF) were previously clearcut and then planted with interior Douglas-fir, the other site (KX) was planted after a wildfire. For each site 10-m radius plots (0.03 ha) were randomly distributed throughout the site on either side of the main access road, except where excavator travel would not permit and the plots had to be moved locally. In almost all plots, EB had slightly greater proportion of other tree species (20%) mainly as naturally regenerated spruce (*Picea* spp.) and aspen (*Populus* spp.) species (Table 1). The chosen sites had low to moderate disease incidence for this ecosystem based on a walkthrough survey.

In each plot, all dead and living trees were tagged and the diameter at 1.3 m (breast height) and total height were recorded by species. The horizontal distance from the plot center to the center of each tree was measured with a tape measure, and the azimuth between the north line and the tree in question was measured using a survey transit. Douglas-fir subject trees (median 4 per plot) were identified so that all of their competitor trees were located within the 10-m radius plots. Competitor trees were trees of any species with height exceeding a projected 60^{o} angle from the horizontal and originating at the base of the subject tree (Biging and Dobbertin, 1992); the competitor trees associated with a particular subject tree comprise that subject tree's neighbourhood.

All trees in every plot were pulled out of the soil in the late fall using a 20-ton Link Belt excavator with a clamshell bucket attachment to minimize the breakage of roots, stems and branches. Pulled trees were gently laid on the ground and left in the plot over winter. After the soil thawed the following spring, the soil was removed from the roots of all trees.. Lesions caused by *A. ostoyae* were identified on all roots and confirmed by observing mycelial fans in the bark or cambium (of the lesions) or as mycelial fan impressions in or under the bark of older lesions. The proportion of diseased primary roots (> 15 mm diameter arising from collar) was recorded for each tree except at site EB.

Cross-sectional stem disks were cut using a chainsaw from all trees at 1.3 m above the soil line and at the soil line (0 m) for all dead trees. The age of the dead trees was determined by counting annual growth rings on the 0 m disks. Very small trees with advanced decomposition may not have been included in the sample. Total tree height from the soil line to the apex was measured with a tape measure after the trees were pulled out of the soil. The disks for all trees were air dried, sanded, and the cross-sectional areas of all trees at age 10 (representing disease-free growth) and sampling age were determined by digitizing the areas according to the methods of Cruickshank (2002). For a smaller number of random subject trees, basal area growth was determined on the stem disks in 5-year increments starting from age 10.

A competition index (CI) was calculated for each subject tree based on distance-diameter methods outlined in Biging and Dobbertin (1992) according to:

$$CI=\Sigma \left[\text{diameter of competitor tree} / \left(\left(\text{diameter of subject tree}\right) \left(\text{the distance between the subject and competitor}\right)\right)\right]$$

The median competitor radius of influence (the distance to the furthest competitor) was 6.6 m (range 4-8 m) for all sites.

A tree aggregation index (AI) was calculated for each plot using nearest neighbour methods according to Krebs (1999):

$AI = r_{A/} r_c$

where r_A = the mean distance to the nearest neighbour in the plot, and r_C = the expected distance to the nearest neighbour corrected for plot edge bias. The r_C coefficient is defined as:

$r_C = r_E + [(0.051 + (0.041/(n)^{-2})) (L/n)]$

where r_E= 2 (density of trees in the study plot)$^{-2}$, L= the length of the boundary of the study area, and n= the numbers of trees in the study area.

The proportion of diseased trees in the 10-m radius plots and in the competitor neighbourhoods surrounding a subject tree was determined by the number of dead and live belowground-diseased trees divided by the total number of trees for the plot and neighbourhood respectively.

2.2 Statistical analyses

2.2.1 Models

Statistical models were identified before analysis that included a measure of competition either using plot density or the competition index, the initial conditions at tree age 10, an index of aggregation and the proportion of disease neighbours in the plot or in the competitor neighbourhood where applicable. Subject tree models also made possible the comparison of disease and disease-free subject tree growth. Scatter plots were generated for all combinations of dependent and independent variables and Pearson correlation coefficients were calculated for each pair. Non-linear relationships between dependent and independent variables were fitted with Table Curve (SPSS version 4) before analysis.

Regression models for fixed effects were first tested using SAS Proc GLM (Ver. 9.1.3); residuals were checked for homoscedasticity and normality and plotted against the overall model predictions and all independent variables. Within the GLM procedure, multicollinearity was assessed using tolerance (Glantz and Slinker, 2001) and using scatter plots. Second-order interactions were considered, but included in the models only if they were significant and did not interfere with model assumptions. For subject tree analyses, after fitting with GLM, the models were fitted using Proc Mixed and for binary responses (disease incidence) with Proc GLIMMIX. For subject trees, an additional hierarchy was identified and accounted for by adding a random plot within a site variable assumed to be independent and $N(0,\sigma^2)$ distributed for all plots. Least-squares means for categorical variables were compared using the Bonferroni method at α=0.05.

Basal area of the plot or subject trees at age 10 (the initial growth condition) was used in the basal area models as a measure of tree size before disease occurs because disease incidence is very low at this age (Morrison et al., 1988). The initial size is important because growth becomes increasingly confounded with disease (Morrison and Pellow, 1994), and disease occurs more frequently on larger trees (Bloomberg and Morrison, 1989; Morrison et al., 2000).

2.2.2 10-m radius plots

2.2.2.1 Plot basal area, mean plot basal area, and mean plot tree height

Regression models for total plot basal area were tested in the 10-m radius plots using proc GLM:

$$Y_{ij} = \mu + \alpha_i + \tau_i + \delta_i + \phi_i + \lambda_j + \varepsilon_{ij}$$

where: Y_{ij} is the plot basal area difference between age 10 and the sampling age given its initial size BA10 in plot *i,* and growing conditions τ, δ, ϕ, and λ in plot *i* on site *j* ; μ is a measure of overall basal area difference; α is a measure of initial plot basal area at age 10 in plot *i*; τ is the proportion of diseased trees in plot *i*; δ is the tree aggregation index in plot *i*; ϕ is the number of trees in plot *i*; λ is the categorical effect of site *j*; and ε_{ij} is the residual error.

The model is essentially the same for mean plot basal area except that BA $1\tilde{0}$ is the initial mean plot basal area at age 10 and the response is mean plot basal area. Mean plot tree height is modelled in a similar way except that no initial height conditions at age 10 were collected, and the response is mean plot height. A log transformation was necessary for mean plot height to normalize the residuals.

2.2.2.2 The proportion of diseased trees

The regression model using Proc GLM for the proportion of diseased trees per 10-m radius plot was:

$$Y_{ij} = \mu + \alpha_i + \tau_i + \delta_i + \phi_j + \varepsilon_{ij}$$

where: Y_{ij} is the proportion of diseased trees growing in plot *i* under conditions α, τ, δ, and ϕ on site *j*; μ is the overall proportion of diseased trees per plot; α is the mean plot basal area at age 10 in plot *i;* τ is the tree aggregation index in plot *i;* δ is the number of trees in plot *i;* ϕ is the categorical effect of site *j;* and ε_{ij} is the residual error.

2.2.3 Subject trees

2.2.3.1 Subject tree basal area

Subject trees were analysed using Proc Mixed in a similar manner as for plot analyses, but included the disease status of the subject tree, and a competition

index in place of the number of trees. Since there was more than one subject tree in most plots, a hierarchy of plots within sites was identified and accounted for with a random plot parameter. This model was fitted by the method of restricted maximum likelihood according to:

$$Y_{ijk} = \mu + \alpha_i + \tau_i + \phi_i + \delta_i + \lambda_i + \beta_j + e_{k(j)} + \varepsilon_{ijk(j)}$$

where: Y_{ijk} is the basal area difference between age 10 and the sampling age for subject tree *i* given the growing conditions α, τ, ϕ, λ, and β on site *j*, with random effect of plot *k* nested in site *j*; μ is the overall basal area difference; α is the basal area at age 10 of subject tree *i;* τ is the effect of disease status (diseased or not) of subject tree *i;* ϕ is the proportion of diseased competitors for subject tree *i;* δ is the competition index of subject tree *i;* λ is the competitor aggregation index for subject tree *i;* β is the categorical effect of site *j;* e is the random effect of plot *k* within site *j;* and $\varepsilon_{ijk(j)}$ is the residual error. A log transformation of the subject tree basal area was necessary to normalize the residuals.

2.2.3.2 Recent mean annual basal area increment

The average annual basal area growth for approximately the most recent 5-year period was determined. The different stand ages resulted in different numbers of years for the most recent time period because the stem disk areas were determined every 5 years. For site EB the recent period was between tree age 20 and 25 years, for site KF between age 25 and 30 years, for site KX between age 25 and 32 years, and for site CC between age 30 and 34 years. The average of each of the recent periods was used as the response in the regression models. The model independent variables were identical to the model in 2.2.3.1 and fitted in a similar manner.

2.2.3.3 Proportion of diseased subject trees

Categorical data analysis of disease incidence in subject trees was done using logistic regression with Proc GLIMMIX and fitted by residual pseudo-likelihood estimation according to:

$$P_{ijk} = \frac{1}{1 + \exp^{(\mu + \alpha_i + \tau_i + \phi_i + \delta_j + e_{k(j)})}}$$

where: P_{ijk} is the probability that subject tree *i* is diseased at the final sampling age given growing under conditions α, τ, ϕ, and δ on site *j* and plot k within site *j* and ; μ is the overall infection, α is the basal area of the subject tree *i* at age 10; τ is the competition index of subject tree *i;* ϕ is the aggregation index of competitors for subject tree *i;* δ is the categorical effect of site *j;* and e is the random effect of plot *k* within site *j.*

3 Results

3.1 10-m radius plots

3.1.1 Plot basal area

The initial plot basal area at age 10, a measure of plot growth before disease occurred, was related to the plot basal area at sampling age ($p=0.0001$ Table 2) as a square root function. Plots with large initial basal area values tended to also have large values by sampling age, but were increasingly reaching an upper growth limit. The proportion of diseased trees was negatively associated with the plot basal area ($p=0.01$, Table 2). This was a linear relationship that showed decreased plot basal area with increased proportion of diseased trees (Figure 1). Using average values for the independent variables, the model predicts an increase in the proportion of diseased trees from 0 to 0.8 caused a 13-18% reduction in plot basal area depending on the site.

More trees in the plots corresponded to more plot basal area ($p=0.02$, Table 2) by a power function (0.58); in effect, increasing the number of trees yielded diminishing returns in plot basal area. The number of trees in the plots ranged from approximately 500 to 2300 stems per ha (19-72 / plot, Table 1).

The effect of tree aggregation on plot basal area was first investigated by analyzing three categories of trees. An aggregation index within plot was calculated for dead trees alone, for dead and living diseased trees, and for all trees (dead plus alive) and compared for effects on plot basal area (Figure 2). The distribution of dead trees alone and dead plus infected trees was not related to the plot basal area (Figure 2). Aggregation of dead trees was more clumped but had some overlap with current living diseased and disease-free trees. The dead and living diseased trees, representing the current inoculum, was largely randomly distributed (Figure 2). Considering all trees, a more regular distribution resulted in increased plot basal area (Figure 2). The aggregation index of all trees was used in the multiple regression models. Regular tree distribution increased the basal area ($p=0.04$, Table 2) resulting in greater basal area per plot. Plot basal area differed at the four study sites ($p<0.0001$, Table 2) with sites KX and CC having the highest plot basal area (LS means).

3.1.2 Mean plot basal area

The square root of the mean plot basal area at age 10 explained some of the variation in the mean plot basal area at sampling age ($p=0.004$, Table 3), and plots with initial large values were reaching an upper growth limit by sampling age. The proportion of diseased trees (dead plus alive) in the plots did not affect the mean plot basal area ($p=0.87$, Table 3). More regular distribution of trees in the plots corresponded to more mean plot basal area of the trees ($p=0.04$, Table 3). More trees per plot corresponded to a lower mean plot basal area ($p<0.0001$) by a power function (0.58) relating to diminishing effects at higher values. Site

differences occurred ($p<0.0001$, Table 3). Sites KX and CC had significantly greater mean plot basal area than others (LS means).

3.1.3 Mean plot height

Mean plot tree height in the 10-m radius plots was modeled as a function of the same variables used for plot basal area except that the height at age 10 was not collected and therefore not included in the model. Mean plot height was negatively but not significantly related to the proportion of diseased trees (dead plus alive) in the plots ($p=0.13$, Table 4), and was positively but not significantly related to regular tree distribution ($p=0.22$, Table 4). The number of trees was negatively related to mean plot height ($p<0.0001$, Table 4) as a power function (0.58) also relating to diminishing effect at higher values. Mean plot height differed significantly at the four study sites ($p<0.0001$, Table 4) with site KX having plots with greater mean height than the other sites (LS means).

3.1.4 Proportion of diseased trees

The mean plot basal area at age 10 was not related to the proportion of diseased trees ($p=0.46$ Table 5), but the number of trees (live plus dead) was negatively related to the proportion of diseased trees ($p=0.007$, Table 5) as a power function (0.58) showing that disease incidence decreased but slowed at higher tree densities. Tree aggregation of all plot trees had no effect on the proportion of diseased trees ($p=0.73$, Table 5). There were differences in the proportion of diseased trees by site ($p<0.04$ Table 5); site KX had significantly more disease than site CC (LS means). The proportion of diseased trees in the plot was also positively related to infection intensity of the diseased trees (mean percent diseased roots) within plots (Spearman's $r=0.34$ $p=0.003$). Unfortunately data on infection intensity of roots systems was not collected for site EB.

3.2 Subject trees

3.2.1 Subject tree basal area

The subject tree basal area was positively related to the basal area at age 10 ($p<0.0001$, Table 6) by a power function (0.3). Subject trees with initial large basal area values tended to remain large but reached an upper growth limit by sampling age. Diseased subject trees had significantly less basal area ($p=0.003$, Table 6) (8% or 10 cm^2, LS means) relative to disease-free subject trees. The proportion of diseased competitors (dead plus alive) was negatively correlated with basal area ($p=0.03$, Table 6). The model predicted 15% basal area loss when the proportion of diseased competitors increased from 0 to 0.8. Increasing subject tree competition reduced subject tree basal area (Figure 3, and $p<0.0001$, Table 6). The competition index is sensitive to the number of trees,

the size of competitors relative to the size of the subject tree, and the distance between the subject and competitor. The aggregation of competitor trees was not significantly related to subject tree basal area (p=0.86, Table 6). The subject tree basal area varied among sites (p=0.0001, Table 6); sites KX and CC had almost identical basal area, and they had significantly more basal area (LS means) than other sites.

3.2.2 Subject tree mean annual basal area increment for the most recent 5-year period

Mean annual basal area increment for the current five years was related to basal area growth at age 10 ($p<0.0001$, Table 7) as a power function (0.3). Subject tree disease status (diseased or not) was a more important variable in this model, and diseased trees were associated with about 24 % reduction (LS means) in mean annual basal area increment. The proportion of diseased competitors and the aggregation index were not important (p=0.14 and p=0.55 respectively, Table 7) but subject tree competition reduced subject tree mean basal area increment ($p<0.0001$, Table 7).

3.2.3 Subject tree height

Diseased subject trees were shorter than disease-free trees (p=0.002, Table 8) by about 4% (53 cm, LS means). The proportion of diseased competitors was negatively but not significantly related to subject tree height (p=0.08 Table 8). Increased competition on the subject tree was correlated with reduced height ($p<0.0001$, Table 8) as a power function (0.58). Regular competitor aggregation was positively, but not significantly, associated with subject tree height (p=0.10, Table 8). There were significant differences in height among sites ($p<0.0001$, Table 8); site KX had the tallest trees (LS means) and 12% more height than the next tallest site (KF).

3.2.4) Subject tree disease incidence

The basal area of the subject trees at age 10 prior to disease and the competitor index measure were not related to the probability of subject tree disease ($p>0.29$, Table 9). As the incidence of diseased neighbours increases the largest trees are more frequently diseased than smaller trees (Figure 4), but disease status is better explained by the competition index. The competition index was negatively correlated with the incidence of subject tree disease (p=0.004, Table 9). The incidence of subject tree disease varied among sites ($p<0.002$, Table 9; Table 1), with site CC having the lowest disease incidence in plots.

4 Discussion

4.1 Tree size and tree density

4.1.1 Plot basal area in 10-m radius plots

Evidence for compensatory growth in agricultural crops has been detected by the shape of their yield-to-damage curves. A non-linear relationship between crop yield and pest damage (convex up) is given as evidence for compensatory growth (Hughes, 1988 and 1990; James, 1974; Walker, 1983). Low damage initially has little effect on yield if the remaining plants can compensate; however, yield is increasingly reduced as more of the population becomes affected. An initial lag in impacts is assumed to result from the removal of some competitive interference of the remaining plants. We found a negative linear relationship between the incidence of dead and infected trees and plot basal area associated with substantial losses; compensatory growth, if it does occur, was not a large factor. However, the shape of the damage curve could easily be affected by factors other than compensatory growth, and in isolation this method does not allow us to draw definite conclusions, especially for forest conditions.

4.1.2 Mean plot basal area and height in 10-m radius plots

Compensatory growth has also been detected as an increase in the size of the remaining plants as damage increases, especially in disease-free plants (Alexander and Mihail, 2000; Bendz-Hellgren and Stenlid, 1997; Gilbert, 2002; Mihail et al., 1998). It is assumed that disease reduces the competitive interference between plants resulting in increased growth, but we found no effect. Mean plot basal area or height should have been greater if compensation growth occurred or smaller if growth reduction occurred. Any effects were probably masked by taking the mean values of plot measures. Greater tree density correlated with decreased mean plot basal area and height; this agrees with previous observations in a range of different conifers (Sjolte-Jørgensen, 1967). Differences in tree density in the study plots were probably the result of differences in initial planting, planting survival, and some natural regeneration.

4.2 Subject tree basal area and height

At the subject tree level (disease-free and diseased), basal area decreased with increasing level of disease in neighbours, but this trend was not found in height. This was opposite to the expected results for basal area if compensatory growth is assumed. Tree growth should be greater when the trees are surrounded by more infected or dead neighbours (Bendz-Hellgren and Stenlid, 1997). There were no significant interactions between disease status in subject trees and disease in competitors on subject tree basal area growth, meaning that both disease and disease-free subject trees were affected similarly by the infection intensity of their neighbours. Diseased trees had less basal area and height compared to disease-free subject trees for a given level of competition. We interpret this to mean that the disease prevented individual trees from reaching

their full growth potential without affecting the growth of neighbouring trees. Disease reduced the height of the subject trees less than on basal area (4% and 8% respectively) probably because disease represents a new sink (defence) that competes with radial growth to a greater degree. Height growth is less sensitive than basal area growth to competing sinks because the apical meristem of the leading shoot is thought to be a stronger sink than the cambium (Lanner 1985). Growth impacts will continue to accumulate in diseased trees as they age; growth reduction in diseased trees has been shown to accumulate for time periods of up to 50 years in Douglas-fir trees that survive infection, with no indication of recovery (Bloomberg and Morrison, 1989). Since most of the disease expression is currently on living trees, the diseased live trees apparently still compete effectively for resources in these monoculture stands.

During the most recent 5-year growth period, the average annual basal area of subject trees was not related to disease in competitor trees. Changes to tree growth resulting from disease should be greater in the most recent time period when more of the trees have become diseased; however, this would be a weaker indicator of overall impacts. The competition index was significantly correlated with reduced growth, but it also had lower effect than the other basal area models. This occurred mainly because of an increase in the effect of disease status (disease-free or diseased) for the subject trees, which is better reflected in more recent time periods. Knowing when the subject tree became diseased could further improve the relationship between growth and disease. If growth is increasingly reduced in diseased trees in the future, they should become less dependent on stand density. If the effect of disease is to reduce basal area and height, then the interaction of competition and disease would put diseased trees at a long-term disadvantage.

4.3) Tree aggregation, disease incidence, and primary and secondary inoculum

Regular distribution of diseased plants creates the best conditions for neighbours to take advantage of any reduction in competitive interference resulting from disease. Diseased plants occurring in clumps will tend to have diseased neighbours more often than disease-free neighbours; as a result of this, the likelihood of compensation growth is minimized and losses maximized (Alexander and Holt, 1998; Hughes, 1996; James, 1974). Consequently, we expected clumped distribution of diseased trees in 10-m radius plots would reduce plot yields or tree size more than for diseased trees that were regularly spaced; however, in this study, the distribution of all trees (dead plus alive) and not diseased or dead trees was related to basal area or height at the plot level. This suggests that the initial distribution of the trees and not the distribution of dead or diseased trees affected stand growth.

Individual subject trees (disease-free and diseased) had lower basal area with increasing incidence of disease in neighbours, opposite to what would be expected with compensatory growth. There was also a positive relationship between mean percent diseased roots per tree and plot disease incidence; therefore, higher disease incidence is also related to greater disease intensity on the roots of diseased trees. Growth reduction in Douglas-fir is positively associated with diseased root intensity (Bloomberg and Morrison, 1989). The negative relationship between the proportion of diseased competitors and subject tree basal area probably is caused by a slightly greater proportion of larger trees being diseased in plots with greater incidence, which then suffer more growth reduction because of the higher percentage of diseased roots. This effect was only detected at the individual tree level.

Diseased trees in 10-m radius plots (dead and alive) were mostly randomly distributed but a few plots had clumped or regular distributions. The original distribution of primary inoculum in our study was probably largely random because the current diseased trees also occur randomly in most cases. Most current infections are likely due to contact with primary inoculum. Primary inoculum appears to be limited in these stands as evidenced by the low proportion of diseased trees in the stands. The incidence of root disease in plants is expected to increase with plant density when inoculum is not limiting, but the opposite occurs when inoculum is limited (Burdon and Chilvers, 1982). We found the latter to be true in this study, suggesting that primary inoculum is limited in its contact with surrounding trees, and that fungal spread from secondary inoculum must also be low at present. If secondary spread was not limited, then increasing tree density in plots of the size used in this study should also increase the proportion of diseased and dead trees.

Infection foci for root diseases develop when roots of trees contact the nearest primary inoculum in root systems of stumps (Morrison and Pellow, 1994); the stands in question are sufficiently mature for this to occur. Primary inoculum is contained in the stumps left after harvest but secondary inoculum is contained in dead roots on living trees, and to a much higher extent, in dead tree roots and root collars. If primary and especially secondary inoculum is not limited, then spread should increase in dense stands because root density is the best predictor of root contacts (Reynolds and Bloomberg, 1982). Increasing tree density might reduce access to the limited primary inoculum for a time because more trees would tend to have a healthy tree as a neighbour rather than an infected stump. In effect, trees surrounding stumps would block access to stump inoculum for some time. If secondary inoculum spread was not limited, then greater mortality would also be needed to allow the fungus to spread to the opposite side of the tree from where it became infected and contact new trees; however, the amount of mortality and clumping in dead trees was still low in our study plots.

We expected that the dead trees would have a more clumped distribution because diseased trees are clustered around a local inoculum source (stumps and dead neighbours), and this can affect the probability of disease in adjacent neighbours. Other Canadian studies have shown that dead trees killed by *A. ostoyae* tend to occur in clumps (Bruhn et al., 1996), that clumps are regularly distributed in the landscape, and that a plot size of 15 m^2 was large enough to capture the clumps in a nearby ecosystem (van der Kamp, 1995). We do not expect the situation as exists currently in these stands to continue indefinitely.

Increasing probability of infection for individual trees has traditionally been attributed to tree size (Bloomberg and Morrison, 1989; Morrison et al. 2000, and 2001; Hrib et al., 1983; Rosso and Hansen, 1998; van der Kamp, 1992 and 1995) probably due to larger root systems with increasing chance of contacting primary and secondary inoculum clusters (Reynolds and Bloomberg, 1982). The incidence of diseased subject trees also suggests that the neighbourhood of a tree is a more important factor affecting disease because the competition index was the main predictor of disease incidence. The competition index is sensitive to size differences between the subject and its neighbours and to the number of neighbours. These results complement those at the plot level in that more competitors might block access to limited primary inoculum, but also that the size of the neighbour might also affect access. Competitors larger than their neighbours may initially intercept the bulk of primary inoculum because of their size, and then limit the growth of adjacent trees towards the primary inoculum for a time. These results suggest that disease-free trees increasingly represent an underestimate of potential stand growth as the stands age and more trees become diseased; further, compensatory growth would become less likely in older stands as a greater proportion of the largest trees became diseased.

Tree distribution did not affect disease incidence probably because of the uniform tree distribution of the plantations. Disease was expected to be more prevalent in aggregated areas because of increased transmission due to higher root density and secondary inoculum transfer. One other Canadian study also found that disease incidence caused by *A. ostoyae* in plantation Douglas-fir was not affected by live tree aggregation (Pielou and Foster, 1962).

The proportion of diseased trees was positively related to the proportion of trees that died in the plots, therefore they should not be considered as separate variables. There may have been a few small trees that were killed by the fungus at an early age and excluded from the dead tree inventory; however, we were able to recover trees that had died up to 22 years prior to sampling. It is not clear how tree size affected disease mortality at any site, and this is difficult to assess since trees die at different times. Mortality was not high in most plots, but, more importantly, disease-free trees adjacent to dead trees are unlikely to remain disease free for any length of time (Kellas et al., 1987, Morrison et al. 2000, Shaw 1980), and they would be unable to fully utilize the increased space. In stands with established root contacts, the fungus colonizes the dead tree root

system within a few years, and then transfers to neighbouring roots mainly by root contact a short time later (Cruickshank et al., 1997), so infection of neighbours is likely to occur a few years after tree death.

4.4) Early mortality, pathogen virulence and host range

Substantial early mortality may increase the chances of compensatory growth. In one study of a *Pythium* species, high primary inoculum levels caused rapid mortality of legume seedlings, and the increased growth of the surviving plants partly compensated for mortality losses (Alexander and Mihail, 2000). Another study of legumes showed that compensatory growth did not occur when inoculum levels and early mortality were low, but only when plant density was high and there was extensive early mortality (Mihail et al., 1998). Population level compensation is more likely when plant damage is limited to early life-cycle stages before the onset of competition (Crawley, 1983). This assumes that disease affects the stand mainly by primary inoculum and that any secondary inoculum subsides and does not cause large patches of under-stocked areas, but this is not the case for forested stands in the ICH. Further, and more importantly for tree root pathogens, the secondary inoculum created by early mortality is probably too small to remain viable for long, thus reducing the threat of secondary spread. Gaps created by mortality later in the life cycle should affect stands more for two reasons: the trees have been competing with each other for some time and have less time to recover the lost growth before harvest, and larger dead trees represent a much a more substantial inoculum source that will remain viable longer after the roots are colonized.

The type of pathogen, its virulence, its dispersive capabilities, and its host range all probably affect the timing and quantity of mortality and the incidence of disease. Norway spruce trees infected with *Heterobasidion annosum* and mainly causing butt rot and reduced growth did not affect the growth of neighbours; in this case, competition directly reduced both diameter and height of the trees (Bendz-Hellgren and Stenlid, 1997). On the other hand, a more virulent root pathogen like *P. weirii* caused enough mortality in young stands to promote growth efficiency in the remaining disease-free Douglas-fir trees as measured by sapwood area (Oren et al., 1985). In BC, the virulence of *A. ostoyae* in Douglas-fir is probably intermediate between these two root pathogens, but *A. ostoyae* differs in its broad host range and ability to spread between roots via rhizomorphs in addition to root contacts. *Armillaria ostoyae* does not commonly cause substantial mortality in BC before stand age 15 because of the low probability of contact before 10 years of age (Morrison et al., 1988; Morrison and Pellow, 1994). Observations from this study indicate that the bulk of infections were caused by rhizomorphs.

4.5 Implications for forest management

The results relating stand density and disease incidence should be interpreted with caution because the stands in question appear to have limited secondary spread at present. This could change if the number of root lesions on the infected trees intensifies causing a flush of mortality, or it could be different in stands with higher primary inoculum density or inoculum potential than these stands. This secondary inoculum (dead trees and roots) would probably spread mainly by root contacts that have higher inoculum potential than rhizomorphs. Infection of the planted trees in stands with high initial inoculum density resulted from both primary and secondary inoculum, but mostly from primary inoculum in stands with less dense initial inoculum (Lung-Escarmant and Guyon, 2004). For stands with higher initial inoculum density than those studied here, this would imply that greater tree density might also allow greater fungal spread especially if it results in mortality. If disease intensity (% diseased roots) is related to disease incidence as suggested in this study, then there will probably be mortality in the future given the number of trees infected in some plots. The stand age at which this might occur is not clear, but in most stands, disease incidence increased with stand age.

The interaction between tree size at a given age and mortality is not well understood, with some studies claiming that tree size has no effect on mortality (Rosso and Hansen, 1998), that disease progresses faster in smaller trees (van der Kamp, 1995) or that faster growing (larger) trees tend to callus more frequently (Cruickshank et al., 1997). It is also not clear how host resistance interacts with tree density (Burdon and Chilvers, 1982), an interaction which could lead to a rapid increase in secondary transfer and impact. More work needs to be done to understand how stand density affects disease and stand yields in the long term. One point that is becoming increasingly clear is that forestry activities that reduce stand density likely increase the proportion of diseased trees (belowground), and interior Douglas-fir stands will probably not be able to fully utilize the site potential compared to stands with little disease. This will affect the economics of the treatment where disease is prevalent, especially when new inoculum is created by stumps and mortality results. Further work needs to be done to determine the effect of disease along the bole and in the crown, to be able to link the effect to infection intensity or infection date, and to calculate economic impacts and disease risk.

When inoculum was initially randomly distributed, increased clumping of crop plants tended to reduce root disease spread from secondary inoculum between clumps, especially when the clump area was kept low (Burdon and Chilvers, 1976); further, reduced secondary spread occurred because disease arrived at clumps mainly from primary and not secondary inoculum. Clumping may limit the extent of pest damage by increasing distance between clumps, but not occurrence (Brown and Bolker, 2004). Planting clumps of susceptible species

within a regular pattern of resistant species, where density is controlled within clumps, may reduce impacts of secondary inoculum. A mixture of resistant and susceptible conifer species could also slow the creation and spread of secondary inoculum. Host tolerance to a pathogen is the ability to sustain damage while avoiding serious crop loss or injury (Agrios, 1988). Mixtures of resistant or susceptible and tolerant species that have similar growth characteristics may still allow secondary spread, but they might also create conditions where compensatory growth becomes more likely. Resistant or susceptible species would both be impacted by the disease, but the tolerant species could potentially compensate for some of the losses in their infected neighbours. One caveat is that this may create an asymmetrical relationship between the two host species mediated by disease (Alexander and Holt, 1998); in other words, growth characteristics and the competitive abilities of different species would need to be matched carefully in the presence of disease. Mixtures of tree species with similar growth characteristics (that increase the likelihood of compensatory growth), where the most susceptible species is planted in small density-controlled clumps might be the most cost-effective way to limit disease impacts, especially if facilitative interaction occurs between the tree species.

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References

Adams, M.J., Lapwood, D.H. 1983. The effect of *Erwinia carotovora* subsp. *atroseptica* (blackleg) on potato plants. II. Compensatory growth. Annals of Applied Biology, 103: 77-85.

Agrios, G.N. 1988. Plant pathology. Academic Press. San Diego, CA.

Alexander, H.M. and Holt, R.D. 1998. The interaction between plant competition and disease. Perspectives in Plant Ecology, Evolution and Systematics 1/2: 206-220.

Alexander, H.M., Mihail, J.D. 2000. Seedling disease in an annual legume: consequences for seedling mortality, plant size, and population seed production. Oecologia, 122: 346-353.

Bendz-Hellgren, M., Stenlid, J. 1997. Decreased volume growth of *Picea abies* in response to *Heterobasidion annosum* infection. Can. J. For. Res., 27: 1519-1524.

Biging, G.S., Dobbertin, M. 1992. A comparison of distance-dependent competition measures for height and basal area growth of individual conifer trees. For. Sci., 38: 695-620.

Bloomberg, W.J., Morrison, D.J. 1989. Relationship of growth reduction in Douglas-fir to infection by Armillaria root disease in southeastern British Columbia. Phytopathology, 79: 482-487.

Braumandl, T.F., Curran, M.D. 1992. A field guide to site identification and interpretation for the Nelson forest region. Research Branch, British Columbia Ministry of Forests, Victoria. Land Management Handbook 20.

Brown, D.H., Bolker, B.M. 2004. The effects of disease dispersal and host clustering on the epidemic threshold. Bulletin of Mathematical Biology, 66: 341-371.

Bruhn, J.N., Mihail, J.D., Meyer, T.R. 1996. Using spatial and temporal patterns of Armillaria root disease to formulate management recommendations for Ontario's black spruce (*Picea mariana*) seed orchards. Can. J. For. Res., 26: 298-305.

Burdon, J.J., Chilvers, G.A. 1976. The effect of clumped planting patterns on epidemics of Damping-off disease in cress seedlings. Oecologia, 23: 17-29.

Burdon, J.J, Chilvers, G.A. 1982. Host density and a factor in plant disease ecology. Ann. Rev. Phytopath., 20:143-66.

Connell, J.H. 2003. Apparent versus "real" competition in plants. In: Perspectives on plant competition. Eds. J. B. Grace and D. Tilman. The Blackburn Press, Caldwell, NJ, 2: 9-23.

Crawley, M.J. 1983. Herbivory: the dynamics of animal-plant interactions. University of California Press, Berkeley.

Cruickshank, M.G., 2002: Accuracy and precision of measuring cross-sectional area in stem disks of Douglas-fir infected by Armillaria root disease. Can. J. For. Res., 32: 1542-1547.

Cruickshank, M.G., Morrison, D.J., Punja, Z.K., 1997: Incidence of *Armillaria* species in precommercial thinning stumps and spread of *Armillaria ostoyae* to adjacent Douglas-fir trees. Can. J. For. Res., 27: 481-490.

de Wit, C.T. 1960. On competition. Versl Laandbouwkd Onderz, 66: 1-82.

Friess, N., Maillet, J. 1996. Influence of cucumber mosaic virus infection on the intraspecific competitive ability and fitness of purslane (*Portulaca oleracea*) New Phytol., 132: 103-111.

Gilbert, G.S. 2002. The interaction between plant competition and disease. Perspectives in Plant Ecology, Evolution and Systematics.1/2: 206-220.

Glantz, S.A., Slinker, B.K. 2001. Applied Regression and Analysis of Variance. New York: McGraw-Hill.

Heath, R., Alfaro, R.I. 1990. Growth response in Douglas-fir/lodgepole pine stand after thinning of lodgepole pine by the mountain pine beetle: A case study. J. Entomol. Soc. Brit. Columbia, 87: 16-21.

Hrib, J., Kyncl, J., Cerny, A. 1983. A tree-ring study of Norway spruce infected with the wood-destroying fungus *Armillaria mellea*. Eur. J. For. Pathol., 13: 160–165.

Hughes, G. 1988. Spatial heterogeneity in crop loss assessment models. Phytopathology, 78: 883-884.

Hughes, G. 1990. Characterizing crop responses to patchy pathogen attack. Plant Path., 39: 2-4.

Hughes, G. 1996. Incorporating spatial patterns of harmful organisms into crop loss models. Crop Protection, 15: 407-421.

James, W. C. 1974. Assessment of plant diseases and losses. Ann. Rev. Phytopath., 12: 27-48.

Kellas, J.D., Kile, G.A., Jarrett, R.G., Morgan, J.T. 1987. The occurrence and effects of *Armillaria luteobubalina* following partial cutting in mixed eucalypt stands in the Wombat Forest, Victoria. Aust. For. Res., 17: 263-276.

Kile, G.A., McDonald, G.I., Byler, J.W. 1991. Ecology and disease in natural forests. In Armillaria root disease. Edited by C.G. Shaw III and G.A. Kile. U.S. Dep. Agric. Agric. Handb. No. 691. pp. 102–121.

Kirkpatrick, H.C., Blodgett, F.M. 1943. Yield losses caused by leaf roll of potato. Am. Potato J., 20: 53-56.

Krebs, C.J. 1999. Ecological methodology 2nd ed. Addison Wesley Longman, Inc., Ontario.

Lanner, RM. 1985. On the insensitivity of height growth to spacing. For. Ecol. Manage., Vol. 13: 143-148.

Lloyd, D., Angrove, K., Hope, G., Thompson, C. 1990. A guide to site identification and interpretation for the Kamloops forest region. Research Branch, B.C. Ministry of Forests, Victoria. Land Management Handbook 23.

Lung-Escarmant, B., Guyon, D. 2004. Temporal and spatial dynamics of primary and secondary infection by *Armillaria ostoyae* in a *Pinus pinaster* plantation. Am. Phytopath., Soc. 94: 125-131.

Mihail, J.D., Alexander, H.M., Taylor, S.J. 1998. Interactions between root-infecting fungi and plant density in an annual legume, *Kummerowia stipulacea*. Journal of Ecology, 86: 739-748.

Meidinger, D., Pojar, J. 1991. Ecosystems of British Columbia. Special report series no. 6, Research Branch, Ministry of Forests, Victoria, BC.

Morrison, D.J., Wallis, G.W., Weir, L.C. 1988. Control of Armillaria and Phellinus root diseases: 20-year results from the Skimikin stump removal trial. BC-X-302, Victoria, BC. Forestry Canada, Pacific Forestry Centre, 16 pp.

Morrison, D.J., Pellow, K.W. 1994. Development of Armillaria root disease in a 25-year-old Douglas-fir plantation. *In* Proceedings of the 8th International Conference on Root and Butt Rots, Aug.9-16, 1993, Wik, Sweden, and Haikko, Finland. *Edited by* M. Johansson and J. Stenlid. Swedish University of Agricultural Sciences, Uppsala. pp. 560–571.

Morrison, D.J.; Pellow, K.W.; Norris, D.J.; Nemec, A.F.L., 2000: Visible versus actual incidence of Armillaria root disease in juvenile coniferous stands in the southern interior of British Columbia. Can. J. For. Res., 30: 405-414.

Morrison, D.J., Pellow, K.W., Nemec, A.F.L., Norris, D.J., Semenoff, P. 2001. Effects of selective cutting on the epidemiology of Armillaria root disease in the southern interior of British Columbia. Can. J. For. Res., 31: 59-70.

Oren, R., Thies, W.G., Waring, R.H. 1985. Tree vigor and stand growth of Douglas-fir as influenced by laminated root rot. Can. J. For. Res., 15: 985-988.

Pielou, E.C., Foster, R.E. 1962. A test to compare the incidence of disease in crowed and isolated conditions. Can. J. Bot., 40:1176-1179.

Rosso, P., Hansen, E. 1998. Tree vigour and the susceptibility of Douglas-fir to Armillaria root disease. Eur. J. For. Pathol., 28:43–52.

Roth. L.F., Rolph, L., Cooley, S. 1980. Identifying infected ponderosa pine stumps to reduce the cost of controlling Armillaria root rot. J. For., 78: 145-151.

Reynolds, K.M., Bloomberg, W.J. 1982. Estimating probability of intertree root contact in second-growth Douglas-fir. Can. J. For. Res., 12: 493–498.

Shaw, C.G. III. 1980. Characteristics of *Armillaria mellea* on pine root systems in expanding centers of root rot. Northwest Sci., 54: 137-145.

Sjolte-Jørgensen, J. 1967. The influence of spacing on the growth and development of coniferous plantations. Int. Rev. For. Res., 2: 43-94.

van der Kamp, B.J. 1992. Rate of spread of *Armillaria ostoyae* in the central interior of British Columbia. Can J. For. Res., 23: 1239-1241.

van der Kamp, B.J. 1995. The spatial distribution of Armillaria root disease in an uneven-aged spatially clumped Douglas-fir stand. Can. J. For. Res., 25: 1008-1016.

Wargo, P.M., Shaw III., C.G. 1985. Armillaria root rot: the puzzle is being solved. Plant Dis. 69: 826-832.

Walker, P.T. 1983. Crop losses: the need to quantify the effects of pests, diseases and weeds on agricultural production. Agric. Ecosystems Environ., 9: 119-158.

Table 1 – Site characteristics from 0.3 ha plots at the four study sites: Chuck Creek (CC), East Barriere (EB), Kingfisher (KF), and Kuskanax (KX).

Site	CC	EB	KF	KX
Tree age at sampling (y)	34	25	30	32
Number of plots	25	23	25	25
Median DBH (cm) [1]	15.2 (17.8,9.7)	12.2 (13.6,8.1)	14.6 (16.2,10.7)	16.2 (17.3,12.0)
Median number of live and dead stems [2]	33 (48,19)	44 (72,31)	39 (55,26)	39 (53,21)
Proportion of Douglas-fir [2]	0.89 (1,0.77)	0.80 (0.97,0.69)	0.86 (1,0.69)	0.94 (1,0.78)
Lat/Long	51.6N 119.8E	51.3N 119.7E	50.7N 118.7E	50.2N 117.7E
Proportion of dead trees [2]	0.04 (0.25,0)	0.04 (0.13,0)	0.06 0.24,0)	0.03 (0.09,0)
Mean tree age at death (y)	28	20	22	22
Proportion of belowground of *A. ostoyae* infected trees [2]	0.35 (0.68,0)	0.33 (0.51,0.14)	0.53 (0.87,0.19)	0.56 (0.85,0.28)

[1] Values in parentheses are maximum and minimum values.
[2] Values in parentheses are maximum and minimum values for plots.

Table 2. Analysis of variance table of plot basal area in 10-m radius plots between age 10 and the sampling age as a function of the initial plot basal area at age 10, the proportion of diseased trees, the number of trees per plot, and the tree aggregation index at the four study sites.

Source	**DF**	**Type III MS**	**F Value**	**Pr > F**	Coefficient
Model	7	12421750.7	30.03	<.0001	
Error	90	413624.3			
R^2 =0.70					
Predictor variables					
Age 10 basal area	1	6757206.87	16.34	0.0001	+
Proportion diseased trees	1	2669272.55	6.45	0.0128	-
Tree aggregation	1	1717283.61	4.15	0.0445	+
Number of trees	1	2252883.52	5.45	0.0218	+
Site	3	11252267.98	27.20	<.0001	

Table 3. Analysis of variance table of mean plot basal area between age 10 and the sampling age in 10-m radius plots as a function of the mean plot basal area at age 10, the proportion of diseased trees, the number of trees per plot, and the tree aggregation index at the four study sites.

Source	DF	Type III MS	F Value	Pr > F	Coefficient
Model	7	4679.41	62.90	<.0001	
Error	90		74.39		
R^2=0.83					
Predictor variables					
Age 10 mean basal area	1	1085.23	14.59	0.0002	+
Proportion diseased trees	1	1.19	0.02	0.8993	+
Tree aggregation	1	317.39	4.27	0.0418	+
Number of trees	1	9733.34	130.83	<.0001	-
Site	3	1854.36	24.92	<.0001	

Table 4. Analysis of variance table of mean tree height in 10-m radius plots as the proportion of diseased trees, the number of trees per plot, and the aggregation index at the four study sites.

Source	DF	Type III MS	F Value	Pr > F	Coefficient
Model	6	0.500	137.06	<.0001	
Error	91		0.003		
R^2 =0.90					
Predictor variables					
Proportion diseased trees	1	0.005	1.41	0.2390	-
Tree aggregation	1	0.008	2.18	0.1433	+
Number of trees	1	0.065	17.84	<.0001	-
Site	3	0.556	152.42	<.0001	

Table 5. Analysis of variance table of the proportion of diseased trees in 10-m radius plots as a function of the mean tree basal area age 10, the number of trees per plot, and the tree aggregation index at the four study sites.

Source	DF	Type III MS	F	Pr > F	Coefficient
Model	6	0.3318	9.47	<.0001	
Error	91	0.0350			
R^2=0.38					
Predictor variables					
Mean basal area age 10	1	0.0187	0.53	0.4671	+
Tree aggregation	1	0.0039	0.11	0.7365	+
Number of trees	1	0.2666	7.61	0.0070	-
Site	3	0.0999	2.85	0.0417	

Table 6. Restricted maximum likelihood table for subject tree basal area between age 10 and the sampling age as a function of subject tree basal area at age 10, subject tree infection status (diseased or disease-free), the proportion of diseased competitors, subject tree competition index, and competitor aggregation index at the four study sites. Note that plot within site was treated as a random factor and that the coefficient for disease status (diseased or disease-free) is given for diseased trees.

Effects	Num. DF	Den. DF[1]	F	Pr > F	Coefficient
Fixed effect (type III)					
Age 10 basal area	1	289	93.87	<.0001	+
Disease status	1	289	8.55	0.0037	-
Proportion of diseased competitors	1	289	4.60	0.0328	-
Competition index	1	289	635.22	<.0001	-
Competitor aggregation	1	289	0.03	0.8619	-
Site	3	89	25.16	<.0001	
Variance components					
Plot within site	0.02914				
Residual	0.05308				
AIC[2]	103.5				

[1] Denominator degrees of freedom
[2] Akaike information criteria

Table 7. Restricted maximum likelihood table for recent mean annual basal area increment as a function of subject tree disease status (diseased or disease-free), the proportion of diseased competitors, subject tree competition index, and competitor aggregation index at the four study sites. Note that plot within site was treated as a random factor and that the coefficient for disease status (diseased or disease-free) is given for diseased trees.

Effects	Num. DF	Den. DF[1]	F	Pr > F	Coefficient
Fixed effect (type III)					
Age 10 basal area	1	118	6.06	0.0153	+
Disease status	1	118	27.13	<.0001	-
Proportion of diseased competitors	1	118	2.16	0.1440	-
Competition index	1	118	192.08	<.0001	-
Competitor aggregation	1	118	0.36	0.5514	-
Site	3	85	1.48	0.2260	
Variance components					
Plot within site	1.8845				
Residual	8.3385				
AIC[2]	1075.2				

[1] Denominator degrees of freedom
[2] Akaike information criteria

Table 8. Restricted maximum likelihood table for subject tree height as a function of subject tree infection status (diseased or disease-free), the proportion of diseased competitors, subject tree competition index, and competitor aggregation index at the four study sites. Note that plot within site was treated as a random factor and that the coefficient for disease status (diseased or disease-free) is given for diseased trees.

Effects	Num. DF	Den. DF[1]	F	Pr > F	Coeifficient
Fixed effect (type III)					
Disease status	1	290	9.03	0.0029	-
Proportion of diseased competitors	1	290	3.06	0.0811	-
Competition index	1	290	285.52	<.0001	-
Competitor aggregation	1	290	2.68	0.1025	+
Site	3	89	53.71	<.0001	
Variance components					
Plot within site	16467				
Residual	20799				
AIC[2]	5000.3				

[1] Denominator degrees of freedom
[2] Akaike information criteria

Table 9. Residual pseudo-maximum likelihood table for the probability of subject tree infection as a function of age 10 subject tree basal area, subject tree competition index, and competitor aggregation index at the four study sites. Note that plot within site was treated as a random factor.

Source	Num. DF	F	Pr > F	Coefficient
Fixed effects (type III)				
Age 10 basal area	1	1.25	0.2851	-
Competition index	1	7.21	0.0041	-
Competitor aggregation	1	0.26	0.3535	+
Site	3	5.40	0.002	
Variance component				
Plot within site	0.2139			
Generalized Chi-Square	χ^2/DF=0.95			

Figure 1. The 10-m radius plot basal area is negatively and linearly related to the proportion of diseased trees in the plots for the four study sites: CC=Chuck Creek, EB=East Barriere, KF=Kingfisher, KX=Kuskanax.

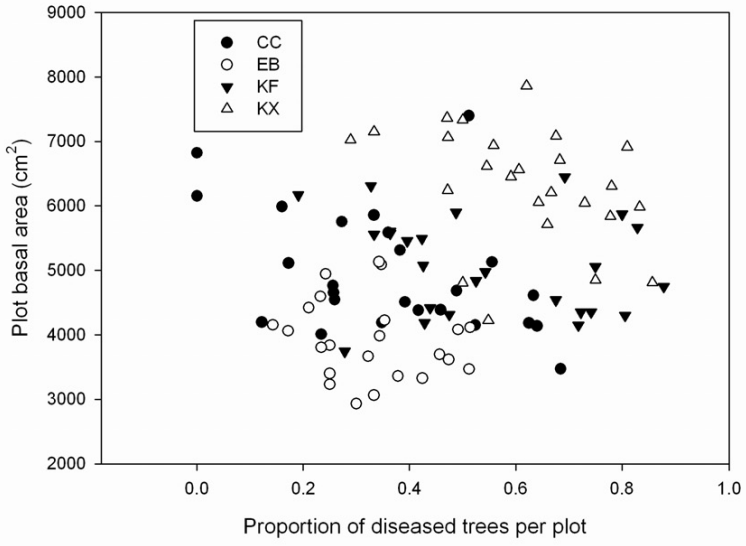

Figure 1

Figure 2. Plot basal area in 10-m radius plots is a function of the aggregation of the trees when all trees (alive and dead) are considered, but is not related to the distribution of the living diseased or dead trees alone. Higher aggregation index values indicate regular distribution, middle values indicate random distribution, while lower values indicate a clumped distribution.

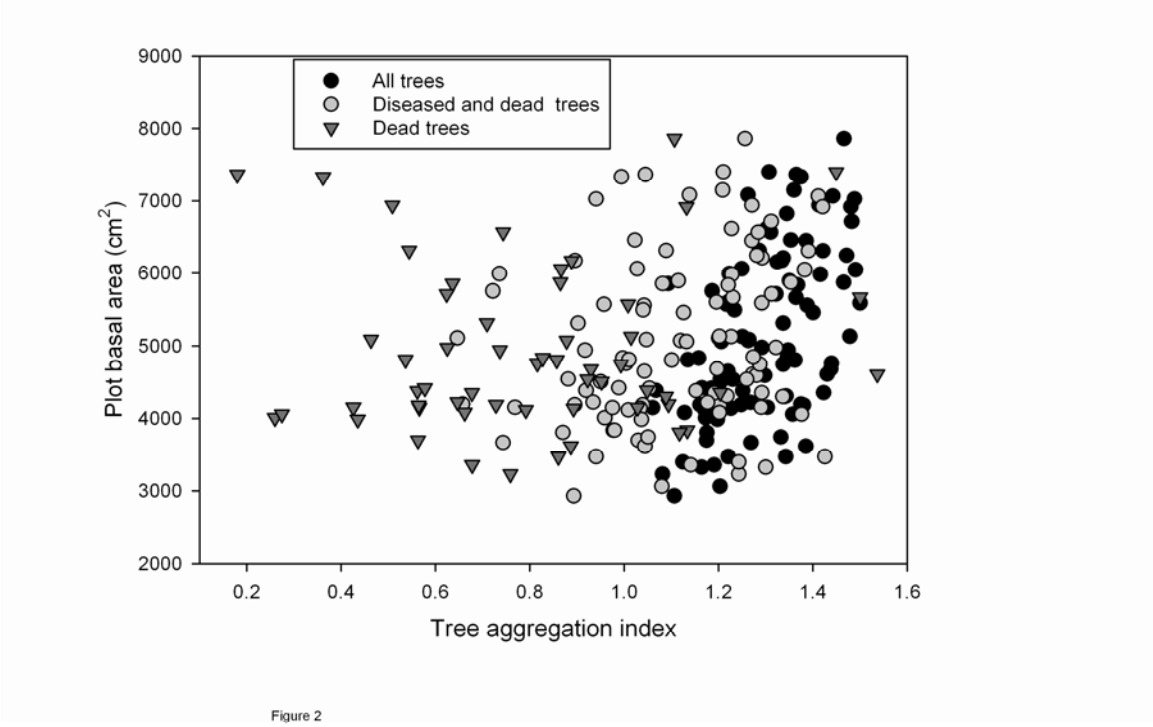

Figure 2

Figure 3. Subject tree basal area is reduced by increased competition from competitor trees as measured by the competition index.

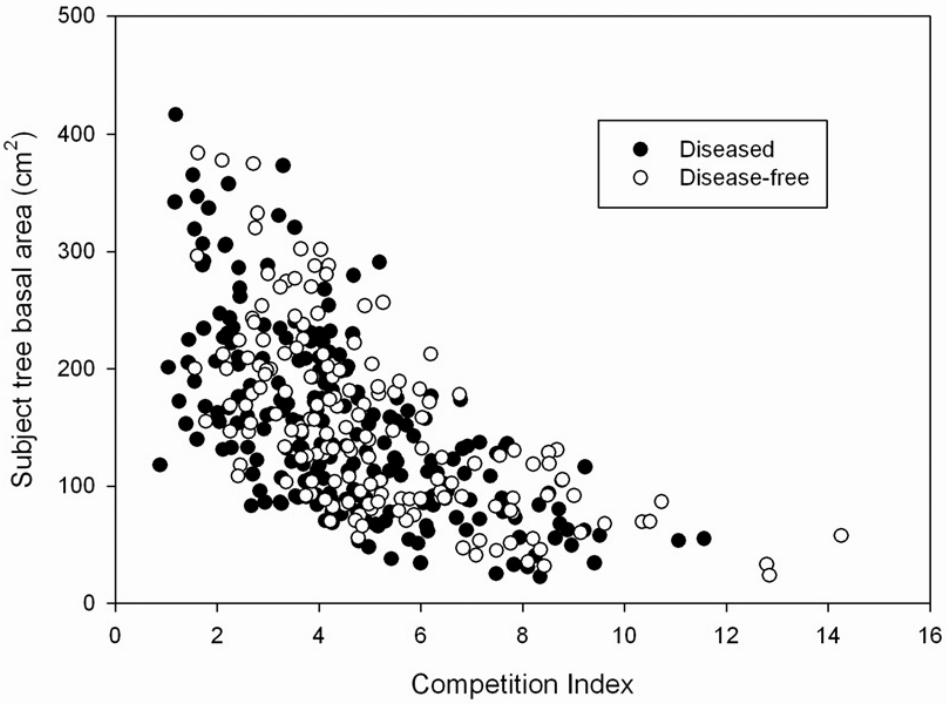

Figure 3

Figure 4. Plot 20 on site KX is used as an example of how disease intensifies in a 10-m radius plot by infecting the largest trees most often. Of the 44 trees occurring in the plot, 75% are diseased (filled circles). Circles are scaled for basal area (1:50). Of the 50% of largest trees, 68% of them were diseased.

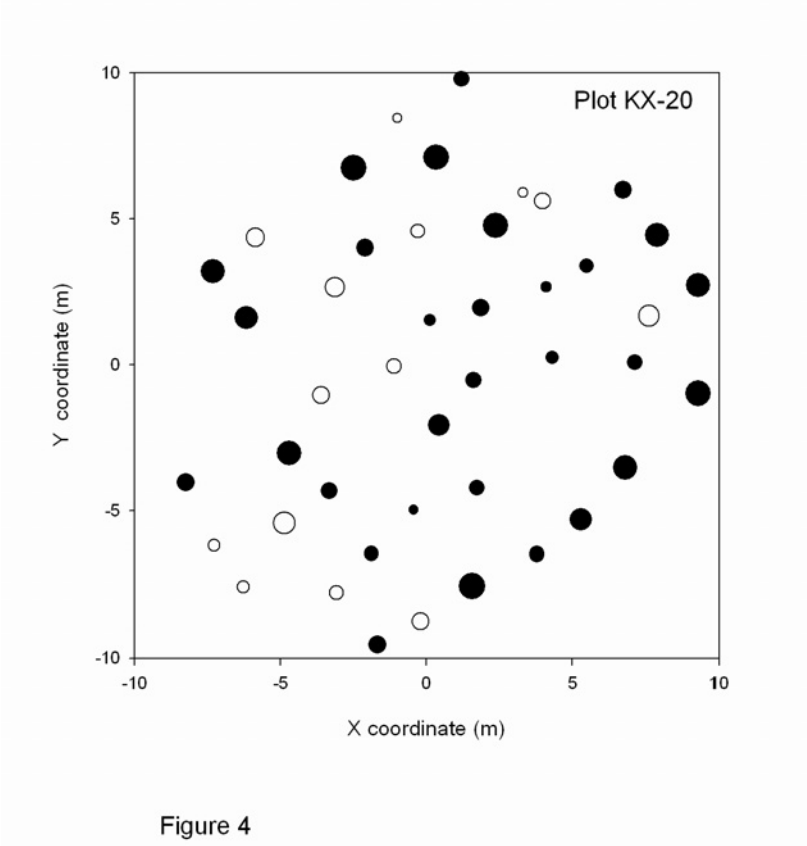

Figure 4