Site, plot, and individual tree yield reduction of interior Douglas-fir associated with non-lethal infection by Armillaria root disease in southern British Columbia

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Keywords: forest resiliency, forest productivity, root disease, *Armillaria solidipes*, growth reduction, Douglas-fir.

Abstract

Root pathogens are one of the principle factors affecting forest productivity in many forests but few estimates of impacts are available. Non-lethal root infections associated with Armillaria root disease were studied to determine their effect on stem volume yield in seven planted Douglas-fir stands and a naturally regenerated stand in British Columbia's southern interior. Volume reductions per tree ranged from 0–30 dm^3 (0%–27%) depending on the tree age and disease duration. Yield reduction reached 27 m^3/ha and averaged 15 m^3/ha for the three oldest planted sites by age 30 (7%–15%), but was lower at the naturally regenerated site. Yield reduction at the site level correlated best with the number of diseased trees and an unknown site factor. Sites with slow juvenile growth had the least yield reduction owing to their lower incidence of disease over time. Yield was less affected by the proportion of diseased primary roots per tree than by the cumulative time since infection. A few of the diseased trees maintained growth after infection so that volume was similar to disease-free trees; interestingly, these trees were smaller than average to begin with. Overall, trees suffer accumulating growth reduction with time without recovery. Root disease prevents full expression of site potential, but minimizing disease impact in respect with other forest management goals is also discussed.

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1. Introduction

Non-lethal yield reductions occur in many plants after attack by pests. This is thought to result from: 1) direct loss of tissue (Kozlowski 1969); 2) xylem damage restricting water and nutrient uptake (Kozlowski 1969) or reduced phloem transport (Froelich et al. 1977) or both, or 3) induced defenses (Franceschi et al. 2005) that divert resources from growth. All these factors may vary based on the duration and intensity of attack. The most studied effect is reduced growth from direct herbivory, after which growth eventually recovers; reduction is usually related to the amount of damage and its duration but may be delayed after first attack for several years (Alfaro et al. 1982). Less frequently studied is reduced growth due to chronic infection, where growth may slow immediately and indefinitely because agents such as root disease persist on the host, requiring continuous response (Hrib et al. 1983).

Armillaria root disease is a common disease of both forest and agricultural crops. More than 30 *Armillaria* species exist worldwide (Watling et al. 1991). In general, losses attributed to Armillaria root disease in forests are most severe in drier Mediterranean or continental climates (Kile et al. 1991). But in the moist regions of the north temperate and boreal coniferous forests *Armillaria solidipes* Peck (formerly *A. ostoyae*) causes lethal primary root disease and reductions in timber yield (Kile et al. 1991). The fungus is common to most forested ecoregions across Canada. In British Columbia (B.C.), the fungus occurs in the southern regions of the province, but causes the greatest problems in the southern interior. The fungus is a virulent primary pathogen with a broad host range including trees and some herbs and shrubs; consequently, mortality, reduced growth, and windthrow are associated with the disease. In Douglas-fir, *A. solidipes* girdles roots, which can kill the tree, or, most commonly, causes patch infections contained to one side of the root (Robinson and Morrison 2001), but it rarely causes butt rot. Few infected trees show aboveground disease symptoms (Morrison et al. 2000) and only a small percentage of trees die from an initial infection.

In Douglas-fir plantations mortality caused by *A. solidipes* begins about age five and its incidence increases slowly to about one percent per annum (Morrison 2009). Work by Cruickshank (unpublished results) in fire-origin, 80- to 100-year-old seral stands dominated by Douglas-fir suggests that mortality also occurs throughout a rotation, but with increasing in frequency after age 80. Following harvest of similar sites, evidence of colonization by *A. solidipes* was found in 60%–90% of stumps (Morrison et al. 2000, 2001; Woods 1994). The percentage of Douglas-fir infected in plantations also increases slowly, reaching 20%-50% in stands aged 15–20 years in many habitats (Morrison 2009). Disease epidemiology may be altered in managed stands by leaving stumps infected by *A. solidipes*, by planting, or by altering species mixtures; hence the timing and magnitude of impacts could differ from those in natural stands. In B.C., Bloomberg and Morrison (1989) estimated *A. solidipes*' impact on growth of 80- to 100-year-old naturally occurring Douglas-fir, but impacts were not scaled to the stand level. No information exists for managed stands. One problem with determining stand-level impacts is that individual tree growth may differ from stand growth if trees interact, especially since disease could reduce competitive interference between neighboring trees. Fortunately, Armillaria root disease is not likely to alter resource partitioning among Douglas-fir trees, meaning that healthy trees cannot easily take advantage of mortality or reduced growth of their infected neighbors (Cruickshank et al. 2009).

The current study investigates the effect of non-lethal Armillaria root disease on volume yield of interior Douglas-fir in plantations and in a naturally regenerated immature stand. Volume yield was used to determine the magnitude of disease impacts, their timing, and their relationship to disease intensity and duration. The implications and methodology would be useful in other locations where the fungus occurs. The study attempted to solve the problem of determining exactly when the infection occurred and how much infection was present by: a complete excavation of the root systems of all trees, determining the infection date of every root lesion on selected trees (Cruickshank et al. 2006), reducing measurement bias by digitizing stem disk areas (Cruickshank 2002), and using hierarchical mixed model longitudinal analysis. The results form the yield reduction component of disease risk from non-lethal infection estimated by the incidence of infection and impacts (consequences) over time.

2. Materials and methods

2.1 Ecosystem and disease epidemiology

The study area covers Douglas-fir plantations within the Interior Cedar Hemlock (ICH) biogeoclimatic zone (BEC) (Braumandl and Curran 1992). In Canada, the ICH zone ranks second in productivity after the Coastal Western Hemlock zone and has the highest diversity of tree species (Meidinger and Pojar 1991). *Armillaria solidipes* occurs in the Interior Douglas-fir (IDF), montane spruce (MS), Engelmann spruce subalpine fir (ESSF) and cedar hemlock (ICH) BEC zones (Morrison et al. 1991); incidence and damage are highest in the ICH. The interior variety of Douglas-fir [*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco] is frequently a dominant species in mature seral stands in the IDF, ICH, and MS. However, interior Douglas-fir is susceptible to killing by *A. solidipes* in this area (Cleary et al. 2008, Morrison et al. 1991) and is a preferred species for regeneration of harvested sites (Braumandl and Curran 1992). The ICH in the southern interior has about 71 000 ha of interior Douglas-fir plantations (M. Cleary, personal communication, 2009) in addition to naturally occurring stands.

2.2 Sites, plot locations, and tree measurements

Seven 20- to 34-year-old Douglas-fir plantations in the ICH BEC zone in southern BC were identified for sampling. These sites were at Chuck Creek (CC) and MacMurphy near Clearwater, East Barriere (EB) and North Barriere (NB) near Barriere, Kingfisher (KF) and Hidden Lake (HL) 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. The sites are representative of stands in their districts. All sites but one were previously clearcut and planted with interior Douglas-fir; the other site (KX) was planted after a wildfire. For each site, 23 to 27 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. Also in the ICH near Rosebery, 10 8-m radius plots were established in a 55-year-old stand comprised of 55% Douglas-fir with smaller percentages of red cedar, hemlock, spruce, larch, birch, and aspen. The stand had regenerated naturally after wildfire.

In each plot in the plantations and at Rosebery, all dead and living trees were tagged and diameter at breast height (DBH), 1.3 m, was recorded. All trees in the plots were pulled out of the soil in late fall using a 20-ton Link Belt excavator with a clamshell bucket attachment with minimum breakage of roots, stems, and branches. Pulled trees were gently laid on the ground and left over winter. After the soil thawed the following spring, soil was removed by hand from the roots of all trees in the plots. Lesions were attributed to *A. solidipes* by their mycelial fans in the bark or cambium. For each tree, the proportion of diseased primary roots (>15 mm arising from the root collar) and height from the soil line to the apex were recorded. For sites CC, EB, KF, KX, and some trees at RB, the height to live crown was also measured.

In the plantations, Douglas-fir trees in each plot were identified for full stem analysis (median 5 to 8 per plot, Table 1). This was achieved by stratified random sampling in a matrix consisting of two size classes above and below the median DBH, and in four infection classes 0%, 1%–33%, 34%–66%, and greater than 66% of the primary roots infected. An attempt to fill the sampling matrix was made for all plots, but at least two trees were sampled in each plot. At Rosebery, all diseased and disease-free Douglas-firs without stem defects such as crooks and forks were selected for stem analysis. In plantations and at Rosebery, cross-sectional stem disks were cut using a chainsaw from all trees at 0, 0.3, 1.3, and then every 2 m till 5 cm diameter. Annual tree height increment was measured between branch whorls from at least age 10 until the sampling age.

Stumps from all diseased trees selected for stem analysis were transported to Victoria, B.C., and dissected. Each identified root lesion was located spatially in the horizontal plane. Diseased root cross-sections (2.5 cm thick) were cut immediately proximal to the lesion (towards root collar) and every 20 cm along the root measured from the root collar. Root and stem disks were air dried and sanded; the cross-sectional areas for stem disks of all trees and at age 10 and every 5 years and the sampling age were determined by digitizing the areas (Cruickshank 2002). Stem volume was calculated for each time period using a frustum of a cone between disks, a cone from the top disk to the apex, and then by summing the sections. Traumatic resin canals invoked by fungus were traced along the root disks into the root collar section and lower stem disk to date each infection (Cruickshank et al. 2006).

A non-spatial competition index was calculated for each sampled tree using the sum of the plot's basal area of trees that are larger (BAL) than the target tree (Wykoff et al. 1982).

2.3 Statistical models

Two separate mixed-effects regression models to predict volume were fitted, one to data from the 7 planted sites, and the other to data from the naturally regenerated (Rosebery) site. Regression models were fitted using SAS Proc Mixed (Ver. 9.1.3) for tree volume at 5-year observation intervals (tree age) starting at age 15 for planted trees and age 20 for the naturally regenerated site. Models identified prior to analysis included a measure of competition at the final observational period using BAL, the tree volume at age 10 for planted stands and at age 15 for the natural stand, the proportion of diseased primary roots, and the accumulated time since infection. The proportion of diseased roots and the time since infection varied with tree age, while the other variables were time invariant. Non-linear relationships between the dependent and

independent variables were fitted with Table Curve (SPSS version 4) before analysis. The nonlinear relationships determined between independent variables and the response were: (tree age)$^{-1}$, ln (volume tree age 10 or 15), (competition index)$^{1.5}$, (number of years tree was infected)$^{0.5}$ for both models. The independent variables were then scaled between 0 and 10 for analysis.

The response for both models used a natural log transformation to normalize the residuals. Residuals were plotted against the dependent and independent variables. Graphical model fits of volume presented in the figures were generated using Sigma Plot 10.0. The variance-covariance residual matrix used in the models was determined using graphical and statistical methods, and the inclusion of model parameters for the two models was based on likelihood ratio tests and information criteria (Littell et al. 2006). Three levels of hierarchy were identified in the data: the site, plot, and individual (tree) level. Therefore the random coefficients (i.e., intercepts and slopes) in the models used these hierarchies as subjects, and the variance-covariance matrix for these random coefficients was assumed unstructured (i.e., covariance among random coefficients was accommodated) (Littell et al. 2006). The best linear unbiased predictors (BLUPs) describing the coefficients for each subject of the random effects were examined for significant differences from the population average at each hierarchy.

Tree volume at age 10 for planted stands and at age 15 for the naturally regenerated stand was used in the models as a measure of healthy growth before infection . Disease incidence is very low at this age (Morrison 2009) and all trees were disease-free according to the root and root collar lesion dates. The Rosebery site with naturally regenerated trees initially grew more slowly, so that many trees took longer to reach 1.3 m; as a result, the onset of infection was delayed. Tree size is important because growth becomes increasingly confounded with disease over time (Bloomberg and Morrison 1989), and disease is more frequent in larger trees (Bloomberg and Morrison 1989; Morrison et al. 2000). As a model covariate, volume at tree age 10 (age 15 Rosebery) accounts for size differences between trees before infection occurs in the sample trees and stand.

The model used to determine volume growth reduction for planted sites due to disease took the form of:

$$[1]\quad \begin{aligned} y_{ijkl} &= \beta_0 + \beta_1 \mathrm{vol\,age\,10}_{ijk} + \beta_2 \mathrm{BAL}_{ijk} + \beta_3 \mathrm{age}_{ijkl} + \beta_4 \mathrm{yrs\,infected}_{ijkl} + \beta_5 \mathrm{prop\,infected}_{ijkl} \\ &\quad + a_{1i} + a_{2i}\mathrm{age}_{il} + b_{1(i)j} + b_{2(i)j}\mathrm{age}_{ijl} + c_{1(ij)k}\,\mathrm{yrs\,infected}_{ijkl} + \varepsilon_{ijkl} \end{aligned}$$

where: Y_{ijkl} is the log of the volume difference (ln[volume at age *l* - volume at age 10]) of tree *k* at age *l* (starting at tree age 15) on site *i* in plot *j* given the fixed growing conditions β_1 to β_5 and random conditions a_1, a_2, b_1, b_2, and c_1; β_0 is the overall mean, β_1 is the continuous fixed effect of the volume of tree *k* at age 10 on site *i* in plot *j*; β_2 is the continuous fixed effect of competition index (BAL) for tree *k* at the final sampling period on site *i* in plot *j*; β_3 is the continuous fixed effect of tree age *l* of tree *k* on site *i* in plot *j*; β_4 is the continuous fixed effect of the number of years tree *k* has been infected at age *l* on site *i* in plot *j*; β_5 is the continuous fixed effect of the proportion of infected primary roots on tree *k* on site *i* in plot *j* at age *l*; a_{1i} is the random intercept for site location *i*; a_{2i} is the random effect of site *i* at tree age *l*; $b_{1(i)j}$ is the random intercept for plot *j* in site *i*; $b_{2(i)j}$ is the random effect of plot *j* in site *i* at tree age *l*; $c_{1(ij)k}$ is the random

effect for the number of years that tree *k* has been infected on site *i* in plot *j* at age *l*; and ε_{ijkl} is the residual error.

The model for the naturally regenerated site (Rosebery) took the form:

[2] $$y_{jkl} = \beta_0 + \beta_1 \text{vol age 15}_{jk} + \beta_2 \text{BAL}_{jk} + \beta_3 \text{age}_{jkl} + \beta_4 \text{yrs infected}_{jkl} + b_{1j} + b_{2j}\text{age}_{jl} + c_{1(j)k}\text{yrs infected}_{jkl} + c_{2(j)k}\text{age}_{jkl} + \varepsilon_{jkl}$$

where: Y_{jkl} is the log of the volume difference (ln[volume at age *l* - volume at age 15]) of tree *k* at age *l* (starting at tree age 20) in plot *j* given the fixed growing conditions β_0 to β_4 and random conditions b_1, b_2, c_1, and c_2. Coefficients for equation 2 are the same as described for equation 1 except that there is no site term and c_2 is the random effect of tree *k* in plot *j* at age *l.*

A temporal autocorrelation structure was assumed for the residuals or both models since repeated observations were made on the same tree. This structure implies that the correlation between observations on a tree measured at times t_l and t_{l+m} is $\rho^{|t_l - t_{l+m}|}$, where $|\rho| < 1$. The residual correlation among different trees was assumed to be zero.

2.4 Yield reduction estimates at the stand level

To calculate volume yield reduction, averaged values of the fixed effects predictor variables (average tree) for each time period were input into the statistical model. To predict disease-free (i.e., potential) volume yield, the number of years since infection and the proportion of infected primary roots were kept at zero. The yield reduction is the difference between the predicted diseased and disease-free trees or stands.

In order to check that the sub-sampling of the plot trees was representative at the site level (all trees in plots) we completely sampled all trees at the Kuskanax site by taking a stem cross-sectional disk at soil line (0 m) and one at 1.3 m. Although we did not have the volume at age 10 for every tree we compared a closely related measure, the basal area, and found that our sub-sample of the plots was very close to all the trees in plots at age 10. The estimate for BAL was within 3% between the two sample populations. We also estimated the proportion of diseased primary roots for every tree at the sampling age; consequently, we determined that the mean for this variable was overestimate in the sub-sample by 50%. For the average number of years that trees had been infected in any time period, we examined the 0 m disks for traumatic resin canals and used this to estimate when the tree had become infected (Cruickshank et al. 2006). The average number of years infected was found to be underestimated in the sub-sample by 2 years below tree age 25, the same at age 25, and overestimated by 2-3 years above age 25. The new averaged predictor variables were input in the model equation 1 to compare between the sub-sample (188 trees) and the plot level population (916 trees). We found that differences in proportionate losses between the two methods was lower by 1% below age 15 and overestimated by 2% above age 25 for the sub-sample. The differences were so small that the sub-sample values were used. For the naturally regenerated site, the sub-sample was the same as the plot level sample since it was a complete sample of all the Douglas-fir plot trees.

For all stands, the number of sampled diseased trees and the total number of diseased trees in the plots at the sampling age were known. At the sampling age, a ratio of the

number of sampled diseased trees to the number of total diseased trees was calculated for each site. This ratio, assumed constant through time, was used to scale the known number of sampled diseased trees at each tree age to the stand level. For each tree age, the predicted yield reduction for an average sampled tree was multiplied by the number of diseased trees in the stand to calculate the total yield reduction (m^3/ha).

1. Results

1.1 Disease in planted Douglas-fir

The model's fixed effects represent population averages, summarized in Table 2. The model response, measured as tree volume between age 11 and the sampling age, increased with tree age as expected (p<0.0001, negative correlation with inverse of age). Tree volume at age 10 was the most important fixed effect (p<0.0001), accounting for the size difference between trees before infection occurs in the stand or sampled trees. Neighbour tree competition reduced the yield (p<0.0001). Stem volume was reduced with time since infection (p<0.0001). Yield reduction occurred rapidly after infection, then slowed, but none of the trees recovered to previous growth levels. The proportion of infected primary roots is a second measure of disease intensity in the root system that often, but not always, increases with time. The root lesions can range from a necrotic patch on one side of a root (most common) to part of or the entire root being girdled by the fungus. Increased proportion of infected roots had a significant negative linear effect on tree volume (p=0.0022), but had the least effect on the model.

Variance components are summarized in Table 2. Random variation in yield at the site, plot, and individual tree levels was greatest at the site intercept level ($\hat{\text{var}}(a_1) = 0.82$, p=0.04; Figure 1), and variation in tree age (slope) among sites was lower than for site intercept ($\hat{\text{var}}(a_2) = 0.02$, p=0.04; Figure 1). Variation in plot intercept ($\hat{\text{var}}(b_1) = 0.2$ p<0.0001) and tree age among plots were lower ($\hat{\text{var}}(b_2) = 0.004$, p<0.0001) than at the site level, but there still significant plot differences within each site (e.g. Figure 2). Covariance for site intercepts and tree age among sites ($\hat{\text{cov}}(a_1, a_2)$= -0.13, p=0.09) and for plot intercepts and tree age among plots ($\hat{\text{cov}}(b_1, b_2)$= -0.03, p<0.0001) were negatively correlated.

At the individual tree level, a random effect the number of years since the first infection ($\hat{\text{var}}(c_1)$= 0.005, Table 2, p<0.0001) explained further significant variation in addition to the fixed effect (β_4). No other random effects improved the model fit (Table 3).

Associated with the random effect for the years since infection ($\hat{\text{var}}(c_1)$) were 40 diseased trees from all sites and many plots with significant BLUPs showing these trees had varying effects of disease on yield over time. Figure 3 shows these effects for two trees on the Kuskanax site plot 22. Diseased tree 1293 had significantly lower yield with tree age (p=0.06) and diseased tree 1296 had greater yield with tree age (p=0.003) than the average. Both trees had about 50% of their roots infected at about the same time and rate, and were equal in all other fixed effect variables. The apparent lack of fit for the model on tree 1296 (Figure 3) is due to the mixed model shrinkage towards the population mean. The shrinkage depends on the amount of variation among trees

compared to within trees, and is affected by the number of observations per tree (Fitzmaurice et al. 2004).

Further investigation into these 40 unusual trees revealed that all of them were diseased and that 12 of these trees had better-than-average yield. Comparing these 12 trees to the other 28 trees with lower yields revealed about the same percent infected primary roots (59% to 58% respectively, p=0.88); similar percent contained (callused) lesions (56% to 62% respectively, p=0.2); similar mean time infected (12.9 to 10.9 years, p=0.18); were shorter at age 10 before infection (134 to 220 cm respectively, p=0.001) but were taller by sampling age (987 to 878 cm respectively, p=0.13); lower volume at age 10 (0.25 to 1.33 dm^3, p=0.01 respectively), but similar volume by sampling age (36.51 to 36.44 dm^3, p=0.06 respectively); had larger crown length to total tree height ratio (CLR, 0.71 to 0.59 respectively, p=0.02). For disease-free trees the average CLR was 0.74 and average volume was 1.01 dm^3 at age 10 and 98.69 dm^3 at sample age. For all infected trees the average CLR was .70, and volume was 1.02 dm^3 at age 10 and 67.64 at sample age. Crown data was available for only 26 of the 40 of the unusual trees indentified in the analysis covering the oldest sites CC, EB, KF, and KX.

Yield reduction per tree was estimated as the difference in predicted volume growth between an 'average' diseased tree and a disease-free tree at age 32 (sampling age). An 'average' diseased tree was determined from averaged values for predictor variables. The model predicts a reduction in volume of about 41 dm^3 per tree (33%) in this case. An average tree with all its roots infected at age 11, compared to the same tree with 10% of it roots infected at age 11 (and then remaining static over time), experienced only a 3% greater yield reduction. The effect of disease on yield varied among planted sites, with sites HL and KX having the greatest average reduction per tree at age 25 and site CC the lowest yield reduction (Figure 4). On a proportional basis per tree, site NB had the highest overall rate of yield reduction, followed by sites HL and MM, and with CC having the lowest (Figure 5). By sampling age, the older sites CC, KX, and KF had the highest average yield reduction per tree (Figure 4). The largest yield reduction for an average tree over a 5-year period was 15 dm^3 at the KX site between ages 25 and 30. Proportionally, the average cumulative proportionate yield reduction across all sites between tree ages 11-15 and then every 5 years till age 30 was 4, 12, 17, 23% respectively; the highest yield reduction per tree was 27% at site KX by sampling age (Figure 5).

On a volume-per-area basis by age 25, two sites KX and KF had the highest actual yield reduction/ha, mainly due to more diseased trees/ha by age 20 and by sampling age (Figure 6, Table 1). Site HL had the highest m^3/ha reduction (Figure 6) by age 20, mainly due to its greater average loss per tree (Figure 4) and high number of diseased trees by age 20 (Table 1); however, by age 25 the greater number of diseased trees/ha on KX and KF sites accounted for their greater yield reductions (Figure 6, Table 1). Yield reduction/ha for the CC site remained low until age 25 due to its very slow initial growth (Figure 6) and consequent low number of diseased trees (Table 1). Proportionate yield reduction at age 30 ranged from about 7% to 15% and increasing with age (Figure 7). For the seven planted stands, diseased trees/ha at age 20 correlated positively to the stand's average tree volume for that age (r=0.96).

1.2 Disease in naturally regenerated Douglas-fir

The fixed effect model for the Rosebery site (RB) resembled the model used for plantations. One difference was that the proportion of infected primary roots did not improve model fit, as it did for the planted sites, and was dropped (likelihood ratio test). Tree volume increased with tree age (p<0.0001 Table 4). Tree volume at age 15 was an important factor (p<0.0001 Table 4) accounting for variation in size differences between trees while disease in the stand was minimal. Increasing competition measure (BAL) reduced the tree volume (p<0.0001 Table 4). Tree infection duration was negatively related to tree volume (p<0.0001 Table 4), as it was with the planted stands.

At the plot level, random variation in volume yield was greatest for trees among plot intercepts (age 20) than for tree age among plots (slope) ($\hat{\text{var}}(b_1) = 0.10$, p=0.02 and $\hat{\text{var}}(b_2) = 0.004$, p=0.03, Table 4). This indicates that trees in plots yield differently to start but have similar growth trajectory over time (Figure 8). Plot intercept and slopes were negatively correlated ($\hat{\text{cov}}(b_1, b_2)$= -0.02, Table 4). Unlike the planted stands, the model was improved by adding a separate random at the tree level for changes in volume with age (slope) ($\hat{\text{var}}(c_2)$= 0.001, p<0.0001, Table 3); in turn, this was negatively correlated with tree years infected ($\hat{\text{cov}}(c_1, c_2)$= -0.001, p<0.0001, Table 4).

Like planted stands, a random tree level effect for the number of years infected at each time period was needed ($\hat{\text{var}}(c_1) = 0.002$, p<0.0001, Table 4 and 5) also indentifying 13 diseased trees that had significant BLUPS (p≤0.05) with better or worse yield than average. For example, three diseased trees from plot ten varied in volume growth (Figure 9). Tree 384 had significantly less volume (p=0.002) and trees 6361 and 6513 both had significantly greater volume (p<0.032) with tree age (slope) than average. Tree 384 had been infected for the least time (age 36) when its growth was sharply reduced (Figure 9). The 5 diseased trees showing significantly better-than-average growth were less tall at age 10 (before infection) than the 8 slower growing trees (110 to 154 cm respectively, p=0.06), but taller by sampling age (1613 to 1347 cm respectively, p=0.008), with similar height to the disease-free trees (1633 cm); less volume at age 10 (0.09 to 0.30 dm^3 respectively, p=0.08) but more volume by sampling age (138.49 to 83.09 dm^3 respectively, p=0.12); infected for a greater number of years than for those with worse-than-average growth (32.8 and 22.3 respectively, p=0.01), opposite that expected. Only 6 of the 13 trees unusual trees had measured crown data, but like the planted trees, the diseased trees with better growth also had higher CLR (0.43 to 0.33). The CRL's were not compared because of the few observations.

The model predicted an average tree infected at age 16 would be about 18% smaller in volume than a disease-free tree by age 55. The difference in growth between diseased and disease-free trees accumulates over time (Figure 4, RB) so that by age 55 an average diseased tree is 16 dm^3 (11%) smaller than the average disease-free tree. Annual 5-year average yield reduction per tree was highest at age 50–55 at about 5 dm^3 or 3%. On an area basis per hectare, yield reduction accumulates similarly to the planted stands, affected largely by the number of diseased trees over time and their growth. The RB site had the smallest trees at age 20 and fewest diseased trees stands (Table 1), and similar to the CC site accounting for the density of Douglas-fir. As a result, the cumulative actual and proportional yield reduction never exceeds that of the

planted stands at similar ages. The natural stand's yield reduction is still substantially lower than for any planted stand, even considering its Douglas-fir density is about half that of the planted stands (Figure 6, Table 1). The mean volume of the naturally regenerated Douglas-fir is 1.36 times greater (Table 1) than then next oldest site CC but is also 20 years older. In the natural stand, the proportion of infected roots is slightly higher than the planted stands, the incidence of diseased trees is greater, but the proportion of dead trees is the lowest among all sites (Table 1). Consequently the number of trees in the plots on an area basis is higher in the natural stands (RB plots are smaller).

3.0 Discussion

The outcome of contact between a host and a fungal pathogen is determined by the effect of the ecosystem on disease, the amount of fungal inoculum, pathogen virulence, and host resistance. Diseased trees can succumb and die, or live as long as disease-free trees if they can control or cope with infection. This study suggests that the chronic belowground lesions that remain largely unseen, and rarely accounted for, considerably decrease the potential yield of individual trees and stands. This factor adds to the more normally accepted impacts of stem decay, mortality, and predisposition to wind and pests. Belowground levels of infection from root diseases appear to be quite high in many areas of Canada, reaching nearly 100% by stand age 100, and especially following harvest because of stumps which become colonized by the fungi (Morrison et al. 2000, 2001; Whitney 1989).

As early as 1874 (Hartig 1874), it was shown that asymptomatic trees can be infected below ground, reducing height and radial increment. Since the growth of healthy trees is used as a benchmark in measuring yield reduction (expected growth), then including asymptomatic but infected trees in this category underestimates impact. Inspection, preferably after removal of the whole root system, is necessary to gauge tree health and thus provide accurate expected growth. This study suggests that yield reduction is best explained by the length of time the tree had been infected belowground. Among the study trees, disease reduced yield soon after infection and accumulated for up to 40 years with no signs of recovery. A study of older Douglas-fir accounted for yield reduction starting at tree age 50 and lasting at least 50 years without evidence of recovery (Bloomberg and Morrison 1989), suggesting that the reduction in planted trees will also continue.

The reduction in increment is correlated to time since infection and occurs immediately following infection. This may indicate a shift in resource allocation from stem growth to active defense; however, this cannot rule out changes in other factors such as foliar efficiency or foliar area which were not measured. Infected trees do react to infection partly through reduced height growth (Cruickshank et al. 2009) and since apical meristems are known to be strong sinks, this at least suggests a change sink priority or fewer resources available for all sinks. We interpret the lower importance of infection intensity (percent infected roots) on yield to mean that xylem transport or root loss are not major immediate problems for the tree growth; however, girdling of entire roots may affect tree survival during dryer years or wind stability. Several studies have found that yield reduction in conifers from root disease cannot be correlated to infection intensity (Bloomberg and Hall 1986; Froelich et al. 1977; Shaw and Toes 1977; Thies 1983), but others have found the opposite (Bloomberg and Morrison 1989; Bradford et al. 1978) or mixed results (Kimberley et al. 2002). The differences between studies probably occur

due to the difficulty in determining the infection date or its exclusion from analysis. In this study, the problem was solved by determining the date of each infection.

Random site and plot level variation in the intercept and slope of the growth curve was greater at the site level, indicating that trees were more similar within plots than between sites. Competing brush in early stages of stand regeneration is suspected of delaying rapid juvenile growth in at least one planted stand (CC), but there is still some unexplained site factor interacting with disease. The naturally regenerated site RB had additional random variation at the individual tree level that affected yield with increasing tree age. It is not clear what causes this tree level variation at this site, but structure and composition in this stand was more heterogeneous due to the natural regeneration.

Further random variation in yield at the individual tree level associated with infection duration was found in 40 planted and 13 naturally regenerated diseased trees (53 total). Models could not accommodate a random intercept for individual trees indicating that growth was not significantly different between individual trees initially (after accounting for fixed effects); however, yield trajectory with tree age (slope) was different for these 53 trees for a given time since infection. We are not certain of the source of this variation, except that it describes trees with better or worse-than-average yield after infection. The diseased trees having better-than-average yield by sampling age were actually shorter at age 10 than diseased trees with less-than-average yield. Additionally by sampling age, the higher yield trees had a ratio of crown length to height similar to disease-free trees, which the lower yield trees did not have or were not able to maintain. Crown length to height ratio in interior Douglas-fir is reduced with increasing height and competition (Temesgen et al. 2005). There still may be a small number of individual trees that able to maintain high crown ratios following an increase in social status while coping with disease. Greater crown length relative to tree height might allow these diseased trees to keep growing similarly to disease-free trees. We assume that these trees had greater crown to height ratio before infection or that it developed soon after infection. We are also not sure why some trees show significantly lower yield than average but they had lower crown to height ratio, but it is possible they could not provide photosynthetic capability for an additional sink. Crown data was only available for 32 of the 53 unusual trees, and more study is needed to confirm these results.

Although we do not know how much inoculum the trees were exposed to, we assume that the level should correlate well to the percentage of diseased roots (dead and living) which was accounted for. Variation in fungal virulence should also be somewhat related to the percent infected roots. The percent infected roots had low importance in yield reduction. Further, we also expect that if virulent isolates occur, it its effect on yield would have a more clumped distribution within a site or affect certain sites more frequently, which did not occur.

We detected no unusual yields for individual disease-free trees like that of the 40 planted and 13 naturally regenerated diseased trees. The few diseased trees that were able to match yield volumes and crown length ratios with diseased-free trees suggests that this ability might be related to disease tolerance. The ability to maintain yield after infection similar to disease-free plants does appear to be like the pathological phenomenon described as “tolerance” (Agrios 1988). Tolerance is often detected by measuring plant yield under varying levels of pest damage, such as was done in this study, but is usually done with genetically related individuals (Strauss and Agrawal 1999). Although this study was not designed to test tolerance, future studies should

consider testing for tolerance in genetic trials which measure at least crown length, total tree height, volume, and disease duration preferably over time.

Volume and proportional yield reduction continue to increase with age. Cumulative proportional yield reduction for individual planted trees was as high as 27% by sampling age and still increasing. At the naturally regenerated site, reductions were as high as 11% per tree over 55 years and increasing with age, but lower than at the planted sites probably because the disease-free trees were initially growing slowly. Mixed stands might also alter disease epidemiology, because not all tree species die or allow entire roots to be girdled easily after infection, which limits inoculum and fungal spread between trees. Average annual percent volume increment reduction over 10 years in Douglas-fir with Phellinus root disease was approximately the same as the greatest estimate in this study (Table 6). Reductions over a 5-year period for Annosus root disease in spruce or pine stands of similar age were higher than those reported the current study for Douglas-fir (Table 6). In the naturally regenerated stand, 5-year average proportional reduction estimates by age 55 compare with four undisturbed 100-year-old Douglas-fir stands with Armillaria root disease (Table 6) at 3%; but the total losses were much higher in the undisturbed stands, ranging from 26%–57% per tree, probably because they had been diseased longer. One hundred-year-old spruce infected with Tomentosus root disease (Table 6) had substantially lower average 5-year annual volume reductions than the reduction estimates for *A. solidipes* in the naturally regenerated stand, but were still significant. Estimated yield reduction in 98 to 147-year-old pine (Table 6) was approximately double that of our estimates and higher than other studies for any root disease. This pine study calculated the potential growth after infection from its pre-infected growth rate; however, potential growth is likely overestimated by using past growth projected into the future.

The number of diseased trees over time was one of the largest factors affecting yield reduction at the stand level in this study. Several studies have shown that trees with root disease tend to faster growth (larger) before infection and probably touch the inoculum sooner than other trees do (Bloomberg and Morrison 1989; Hrib et al. 1983; Lewis 1997; Morrison et al. 2000). If faster growing trees have greater probability of infection, then root disease affects the largest trees in a population more strongly. Over time, the remaining disease-free trees would represent an underestimate of site potential and therefore of yield reduction. The proportion of diseased trees in the planted stands was still low enough that the availability of larger disease-free trees was not a problem. However, at the naturally regenerated site, disease-free tree yield (site potential) might be underestimated at later time periods because most large trees were infected.

Non-lethal yield reduction at the site level is rarely calculated but is needed to estimate disease impacts for biomass predictions and economic analyses. In spaced radiata pine in New Zealand, non-lethal yield reduction at the site level was estimated at 6%–13% (5.5 to 11 m^3/ha) at 28 years (Mackenzie 1987), close to the current study. For the naturally regenerated stand, losses at age 30 were much lower at approximately 0.24 m^3/ha even considering only 55% of the stand was Douglas-fir. Yield reductions in New Zealand pine were estimated to be 4% at age 12 (Kimberley et al. 2002). New Zealand Armillaria species appear to infect trees sooner than *A. solidipes* does in Canada, which accounts for the greater reduction at age 12 than in the current study, but only temporary.

4.0 Conclusions and implications for forest management

Forest productivity is defined as the plant biomass (usually trees) produced per area per unit of time. Long-term productivity is the ability of the land to sustain multiple interventions or rotations. The potential productivity of the land is fixed by climate, topography, and edaphic conditions that are expressed through the genetically constrained growth of trees (Powers 1989). Root diseases interfere with the genetic potential of plants which limit full expression of site potential. This serious effect is not easily detectable aboveground; the disease incidence and severity can be increased by leaving stumps (Morrison et al. 2001); the inoculum of this fungus in these stumps is long-lived (Roth et al. 1980). The yield reduction from non-lethal infections suggests the site potential will not be fully reached even in stands with disease but without mortality. By virtue of its wide host range, Armillaria root disease can interfere with yields—especially in managed stands with stumps and altered stand composition and succession.

It is difficult to compare productivity between the naturally regenerated and planted stands especially in stands of different ages; however, any stand would be less productive with disease. Average Douglas-fir tree volume in planted stands is greater than the natural stand but tree density is also lower. It is not certain that planted tree density or growth rates will be maintained into the future. The planted stands are mostly Douglas-fir which is a susceptible species to this disease, and in some cases, more than half of the planted trees are already infected by about age 30. The natural stand has higher disease intensity and incidence, but still has lower mortality and more dense stands despite stand age being about 2-3 times greater. Western red cedar is a component in the naturally regenerated stand, and the only known conifer species to be disease tolerant, which probably acts to slow inoculum build-up and spread. At the very least, the natural stand with its mix of species would likely be at lower risk to other types of disturbances.

We found that cumulative yield reductions were as high as 23 m^3/ha by age 30 (average 15 m^3/ha for the three older sites), which already significantly affect the economics of stands. We expect that the yield reductions will continue to accumulate as more trees in the stand become infected. Furthermore, since *Armillaria* species can infect most tree species (Hood et al. 1991), non-lethal yield reduction might be even more relevant in other tree species that resist mortality. As long as the infected trees live, yield reduction will be a limiting stand factor; however, it is expected that mortality losses will increase in the planted stands with time. If root disease negatively affects stem taper and wood recovery as it does with butt rot (Kallio and Tamminen 1974), or affects the value of timber product quality (Cruickshank 2010), then these would also need consideration. Finally, root disease affects root biomass proportionally to stem biomass (Bloomberg and Hall 1986) which together could alter carbon sequestration.

This study's results indicate that keeping planted conifer growth rates low in the initial years minimizes the disease spread and lowers the number of infected trees and yield reduction over time. The planted stand with the lowest yield reduction (CC) appeared to have had competing brush, probably also occurring in the naturally regenerated stand. Some competing vegetation in the early years would keep part of the site free of living conifer roots while stump inoculum degrades: in essence, a type of crop rotation. Planting conifers with subsequent high juvenile growth rate probably shortens the time till the tree becomes infected. Delaying contact with the fungus may allow fungal inoculum to degrade. Disease-free trees provide better yields, but a possibility exists

that some of the trees with slower initial growth may actually perform better longer-term with disease. If host tolerance exists, then breeding tolerant stock may reduce the risk of non-lethal yield reduction and mortality, but its occurrence and its relationship to host resistance remains to be determined. Alternatively, removing the stumps reduces root disease but also adds a financial cost, or planting mixed species may add little cost but at a lower level of disease control (Morrison et al. 1988). Root disease treatment strategies should be viewed as a potential opportunity to improve the site, or at the very least, to preserve site productivity and stand stability.

Acknowledgements
The authors gratefully acknowledge the participation in this project of many individuals, government agencies, forestry companies, and forestry contractors: technicians, students, terms and casuals for cleaning stumps, collecting and processing data, including, Jody Huxter, Mike Leslie, Jeanine Bond, Carol Cutforth, Katherine Duthie, Dominique Lejour, Holly Williams, Dion Manastyrski, Michelle Cleary, Leslie Munro, Thane Isert, Kevin Pellow, Leslie Glover, Victoria Levesque, Holly Douglas, Kerry O'Connor, Frank Courage, Garrett Field, Jeremy Field, Karen Brelsford, Karen Coe, and Robert Paul; for in-kind support and advice concerning logistics, site location, and funding raising, the Southern Interior Growth and Yield coop, Steve Stearns-Smith, Atco Lumber Ltd., Riverside Forest Products Ltd., West Fraser Timber Ltd., Weldwood of Canada Ltd., Slocan Forest Products Ltd., Adams Lake Indian Band, Pope & Talbot Ltd., the Kamloops, Arrow, Cariboo-Chilcotin and Boundary Timber Supply Area groups, the Cariboo-Chilcotin Land Use Plan Timber enhancement Strategy Committee, the BC Ministry of Forests and Range District Offices for Arrow-Boundary, Kamloops, Kootenay Lake, Okanagan-Shuswap and Headwaters districts, Ian Cameron, Jim Goudie and Ken Mitchell; for contractor expertise in a particular area, Ramsoft Systems Ltd., J.S. Thrower & Associates, Saunders and Associates, Mibar Logging Ltd., Terra Silviculture, Mountain Man Contracting, and Stearns-Smith & Associates; for project funding, the BC Forest Science Program, Forest Investment Account and Forestry Innovation and Investment, the BC Ministry of Forests and Range Southern Interior Region and Research Branch, Pope and Talbot Ltd., International Forest Products Ltd., Adams Lake Lumber, Tolko Industries, Ltd., Kootenay Lake Timber Supply Area group, Kalesnikoff Lumber Co. Ltd., and Natural Resources Canada Canadian Forest Service; and for comments and advice on the draft manuscript, Art Groot, Charles (Terry) Shaw, Peter Ott and Krista Zala.

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Table 1. Site, plot, and tree characteristics for the seven planted sites and the naturally regenerated site Rosebery.

Attribute	Chuck Creek (CC)	East Barriere (EB)	Hidden Lake (HL)	Kingfisher (KF)	Kuskanax (KX)	MacMurphy (MM)	North Barriere (NB)	Rosebery (RB)
Lat/Long	51.6N 119.8W	51.3N 119.7W	50.5N 119.0W	50.7N 118.7W	50.2N 117.7W	51.7N 120.4W	51.2N 120.1W	50.0N 117.4W
Tree age at sampling	34	25	24	30	32	24	20	55 (mode)
# of plots (radius- m)	25 (10)	23 (10)	24 (10)	25 (10)	25 (10)	23 (10)	27 (10)	10 (8)
Basal area (m^2/ha)	0.26	0.18	0.16	0.23	0.28	0.18	0.09	32.36
Median DBH (cm) (max, min per plot)	15.2 (17.8, 9.7)	12.2 (13.6, 8.1)	11.7 (13.2, 9.9)	14.6 (16.2, 10.7)	16.2 (17.3, 12.0)	11.9 (13.2, 10.4)	9.4 (10.6, 8.2)	13.5 (27.5, 2.6)
Median number of stems/plot (max, min)	33 (48, 19)	44 (72, 31)	40.5 (28, 53)	39 (55, 26)	39 (53, 21)	49 (58, 38)	41 (62, 34)	39.5 (51, 24)
Median Douglas-fir sampled/plot (max, min)	6 (6, 2)	8 (14, 4)	6 (9, 4)	8 (12, 4)	7 (11, 5)	6 (10, 4)	5 (10, 3)	13.5 (23, 9)
Proportion of dead trees (max, min/plot)	0.04 (0.25, 0.00)	0.04 (0.13, 0.00)	0.08 (0.28, 0.00)	0.06 (0.24, 0.00)	0.03 (0.09, 0.00)	0.05 (0.15, 0.02)	0.05 (0.17, 0.00)	0 0
Belowground incidence of live Douglas-fir with *A. solidipes* (max, min/plot)	0.30 (0.76, 0.00)	0.33 (0.49, 0.15)	0.34 (0.76, 0.08)	0.52 (0.90, 0.13)	0.59 (0.88, 0.27)	0.22 (0.44, 0.08)	0.23 (0.46, 0.02)	0.72 (0.85, 0.44)
Proportion of Douglas-fir (max, min/plot)	0.89 (1.00, 0.07)	0.80 (0.97, 0.69)	0.93 (1.00, 0.75)	0.86 (1.00, 0.69)	0.94 (1.00, 0.78)	0.94 (1.00, 0.87)	0.88 (1.00, 0.62)	0.55 (0.75, 0.31)
Mean Douglas-fir tree volume dm^3 at age 20 and final	15, 106	27, 52	32, 45	32, 83	34, 105	26, 40	23	13, 144
# of live diseased Douglas-fir/ha age 20	92	230	339	334	434	287	270	48
Mean proportion of Douglas-fir primary roots infected (max, min)	0.31 (1, 0.06)	0.39 (1, 0.07)	0.27 (1, 0.07)	0.30 (1, 0.07)	0.31 (1, 0.07)	0.32 (1, 0.08)	0.30 (1. 0.05)	0.40 (1, 0.06)
Mean years Douglas-fir was infected (max, min)	10.8 (20, 2)	7.3 (15, 1)	9.7 (15, 2)	12.5 (21, 2)	14.5 (23, 1)	8.5 (15, 2)	6.3 (10, 2)	20.3 (41, 2)

Table 2. Restricted Maximum Likelihood estimates of fixed and random parameters of equation 1, predicting the log of volume of planted trees between age 10 and sampling age in seven Douglas-fir plantations.

Type III fixed effects	**[1]Num. D.F**	**[1]Den. D.F**	**F value**	**P (type III)**	**Coefficient**
1) ln (volume age 10) (β_1)	1	4147	2603.28	<0.0001	0.549
2) (Competition BAL)$^{1.5}$ (β_2)	1	4147	531.99	<0.0001	-0.119
3) (Tree age)$^{-1}$ (β_3)	1	4147	234.68	<0.0001	-0.869
4) (Years infected)$^{0.5}$ (β_4)	1	4147	174.79	<0.0001	-0.074
5) Proportion of diseased primary roots (β_5)	1	4147	9.38	0.0022	-0.006

Variance Components	**Variance**	**Std. error**	**P value (approximate)**
1) Intercept among sites (var a_1)	0.8221	0.4801	0.0434
2) (Tree age)$^{-1}$ among sites (var a_2)	0.0223	0.0130	0.0429
3) (Tree age)$^{-1}$ among site x intercept (cov a_1,a_2)	-0.1341	0.0876	0.0876
4) Intercept among plots (var b_1)	0.1971	0.0268	<0.0001
5) (Tree age)$^{-1}$ among plots (var b_2)	0.0043	0.0006	<0.0001
6) Intercept among plots x (tree age)$^{-1}$ among plots (cov b_1,b_2)	-0.0273	0.0038	<0.0001
7) (Years infected)$^{0.5}$ among trees (var c_1)	0.0047	0.0005	<0.0001
8) Correlation (ρ) among repeated observations	0.9656	0.0017	<0.0001
9) Residual	0.06808	0.0028	<0.0001

[1] Numerator (Num) or denominator (Den) degrees of freedom

Table 3 – Comparison of random effect models using Restricted Maximum Likelihood and fixed effect model terms in equation 1 for the seven planted stands. Model 7 was chosen.

Model number	Model description	-2 log likelihood	Akaike's info. criteria
1	fixed effects only	811.3	815.3
2	fixed effects + random intercept among sites + random intercept among plots + random intercept among trees	594.8	602.8
3	fixed effects + random intercept among sites + random intercept among plots	594.8	602.8
4	model 3 + random age coefficient among sites + random age coefficient among plots + random age coefficient among trees	-1957.7	-1941.7
5	model 3 + random age coefficient among sites + random age coefficient among plots	-1957.7	-1941.7
6	model 5 + random years infected among trees + random percent infected roots among trees	-2133.1	-2115.1
7	model 5 + random years infected among trees	-2133.1	-2115.1

Table 4. Restricted Maximum Likelihood estimates for the effects of fixed and random parameters of equation 2, predicting the log of the volume of naturally regenerated Douglas-fir trees between age 15 and sampling age at the Rosebery site.

Type III fixed effects	**[1]Num. D.F**	**[1]Den. D.F**	**F value**	**P (type III)**	**Coefficient**
1) ln volume age 15 (β_1)	1	1085	60.26	<0.0001	0.174
2) (Competition)$^{1.5}$ (β_2)	1	1085	170.52	<0.0001	-0.223
3) (Tree age)$^{-1}$ (β_3)	1	1085	2073.77	<0.0001	-0.921
4) (Years infected)$^{0.5}$ (β_4)	1	1085	45.20	<0.0001	-0.032

Variance Components	**Variance**	**Std. error**	**P value**	**(approximate)**
1) Intercept among plots (var b_1)	0.1042	0.05159	0.0217	
2) (Tree age)$^{-1}$ among plots (var b_2)	0.0037	0.0019	0.0263	
3) Intercept among plots x (tree age)$^{-1}$ among plots (cov b_1,b_2)	-0.0162	0.0090	0.0730	
5) (Years infected)$^{0.5}$ among trees (var c_1)	0.0019	0.0006	<0.0001	
6) (Tree age)$^{-1}$ (var c_2)	0.0013	0.0003	<0.0001	
7) (Years infected)$^{0.5}$ among trees x (tree age)$^{-1}$ (cov c_1,c_2)	-0.0012	0.0003	<0.0001	
8) Correlation (ρ) among repeated observations	0.9890	0.0018	<0.0001	
9) Residual	0.04265	0.0064	<0.0001	

[1] Numerator (Num) or denominator (Den) degrees of freedom

Table 5 – Comparison between of random effect models using Restricted Maximum Likelihood and fixed effect model terms in equation 2 for the naturally regenerated site Rosebery. Model 6 was chosen.

Model number	Model description	-2 log likelihood	Akaike's info. criteria
3	fixed effects only	-2021.1	-2017.1
4	fixed effects+ random intercept among plots + random intercept among trees	-2059.4	-2053.4
3	fixed effects+ random intercept among plots	-2059.4	-2053.4
4	model 3 + random age coefficient among plots + random age coefficient among trees	-2213.1	-2201.1
5	model 3 + random age coefficient among plots	-2142.6	-2132.6
6	model 4 + random years infected among trees	-2269.1	-2253.1

Table 6 - Summary of yield reduction by root disease

Author	Country	Species	Host age	[1]Pathogen	[2]Average annual volume reduction per tree (%)
Bendz -Hellgren and Stenlid 1997	Sweden	spruce	35	DRN	23 (5 yr.)
Bloomberg and Morrison 1989	Canada	Douglas-fir	80-100	DRA	2-3 (5 yr.)
Bloomberg and Reynolds 1985	Canada	Douglas-fir	40	DRL	13 (10yr.)
Bradford et al. 1978	USA	pine	not given	DRN	19 (5 yr.)
Lewis 1997	Canada	spruce	100	DRT	1-2 (5 yr.)
Mallett and Volney 1999	Canada	pine	98-147	DRA	43 (10 yr.)
Thies 1983	USA	Douglas-fir	50	DRL	13 (10 yr.)

[1]DRA= Armillaria root disease, DRL= Phellinus root disease, DRN= Annosus root disease. [2]Periodic time in brackets.

Figure Captions

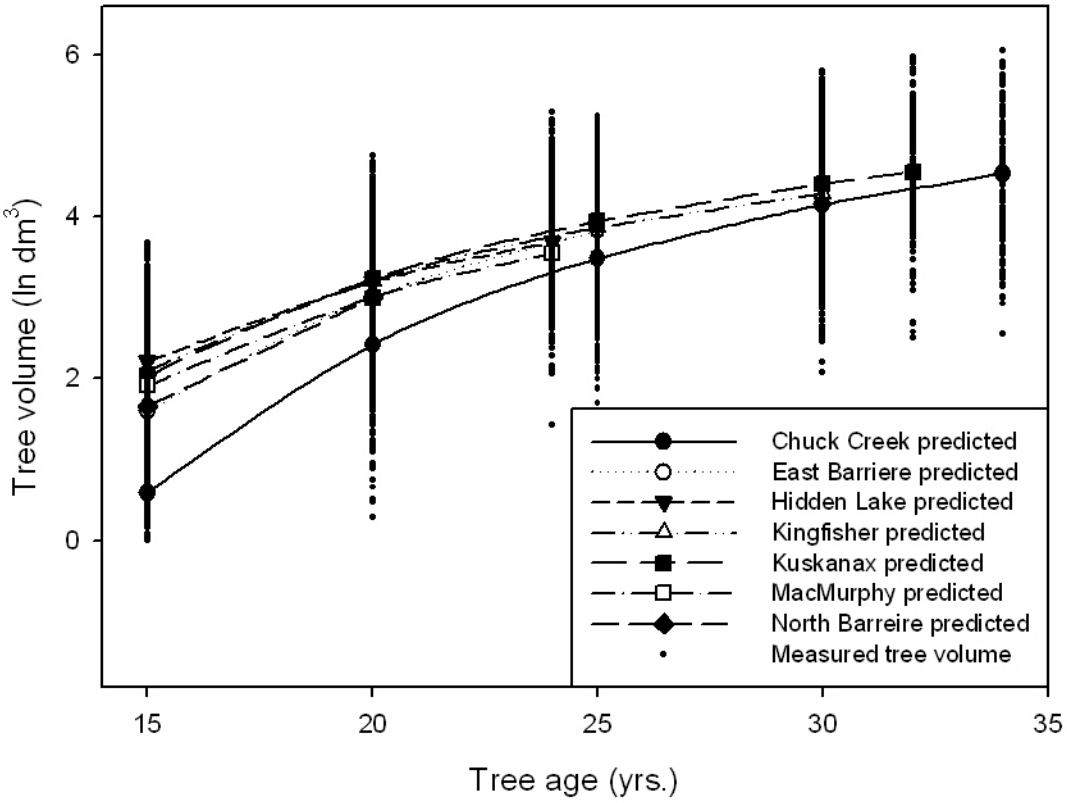

Figure 1. Predicted (equation 1) and measured tree volume versus tree age for seven planted sites.

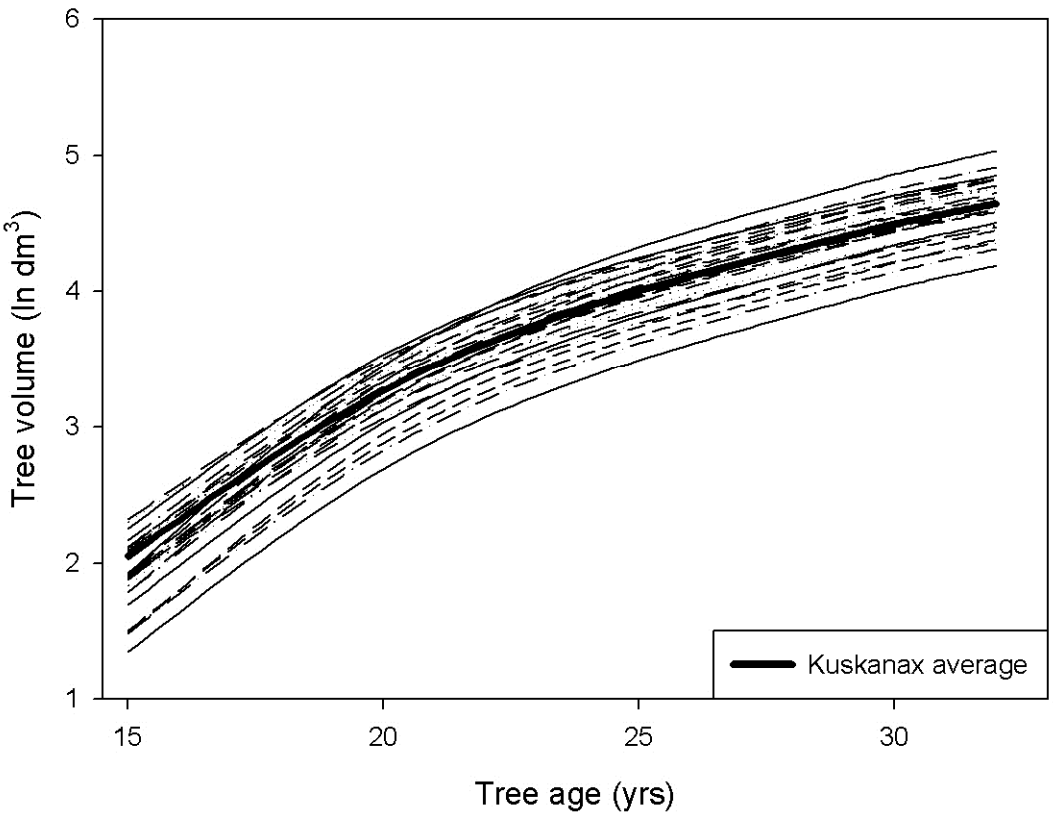

Figure 2. Predicted average tree volume versus tree age for 25 plots at the Kuskanax (KX) site and the predicted site average.

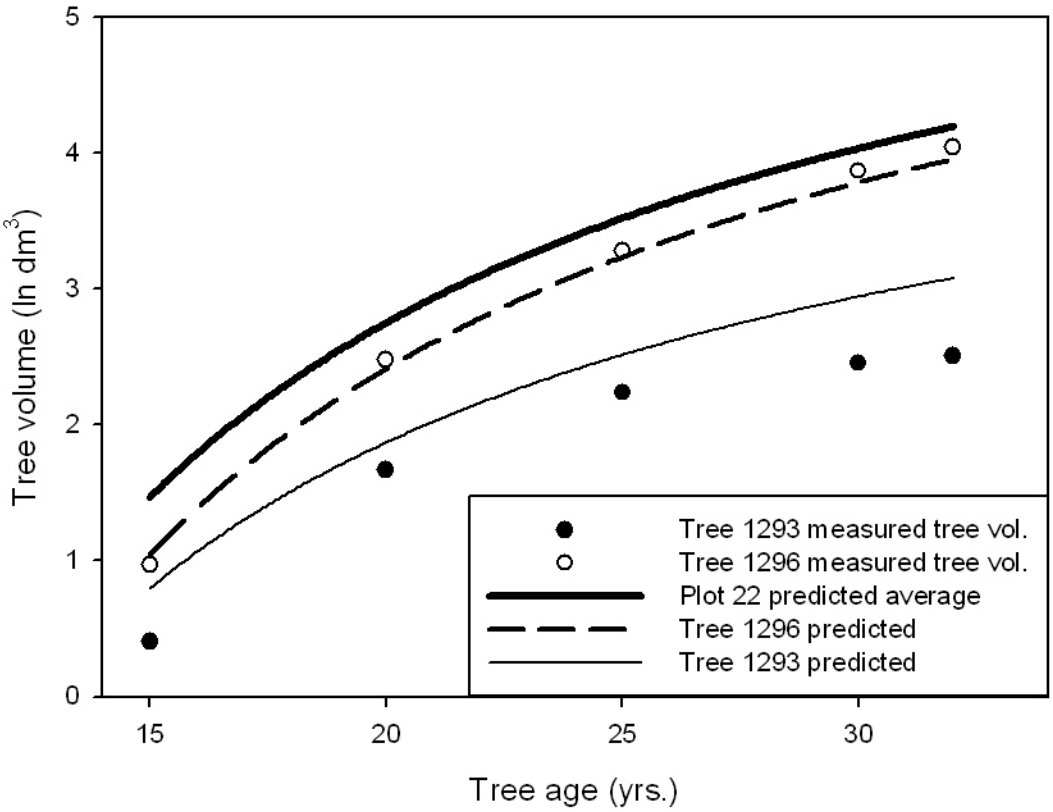

Figure 3. Predicted and actual volume versus tree age for two trees with significant BLUPs in plot 22 on the Kuskanax site. Trees 1293 and 1296 are both diseased, but tree 1296 has higher slope, and tree 1293 lower slope than average.

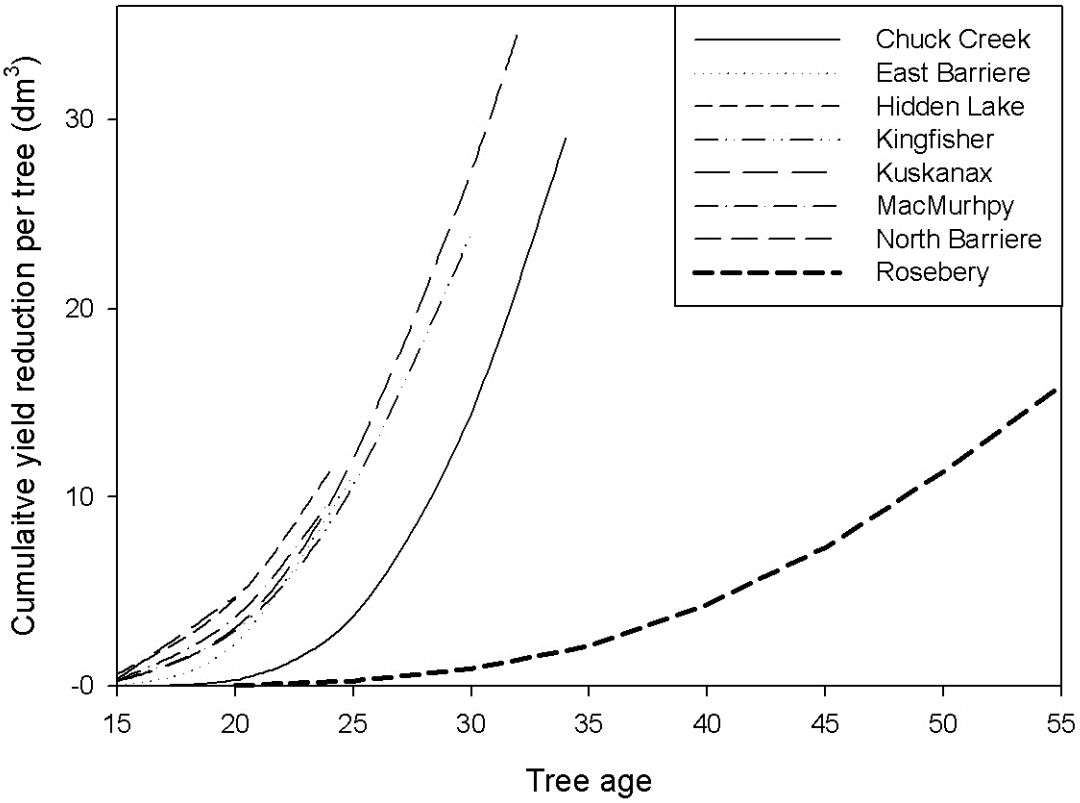

Figure 4. Predicted yield reduction versus tree age of an average diseased tree at seven planted sites and the naturally regenerated site Rosebery.

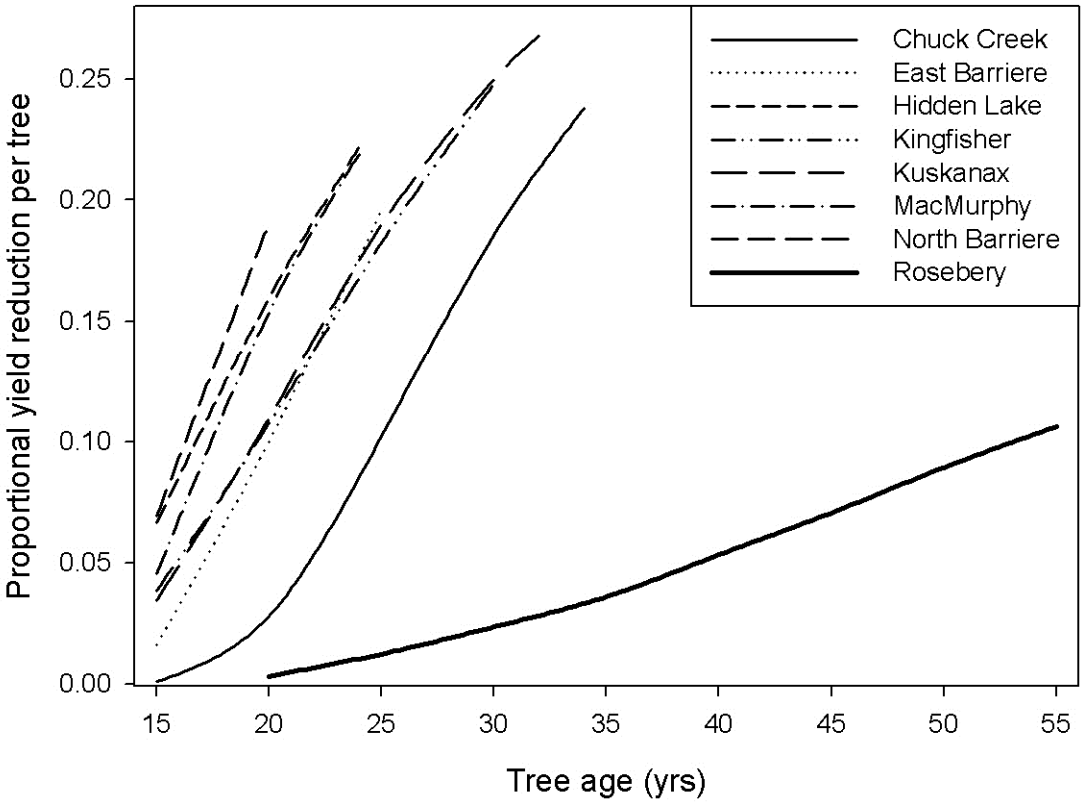

Figure 5. Predicted proportional yield reduction versus tree age for an average diseased tree relative to disease-free for seven planted sites and the naturally regenerated site Rosebery.

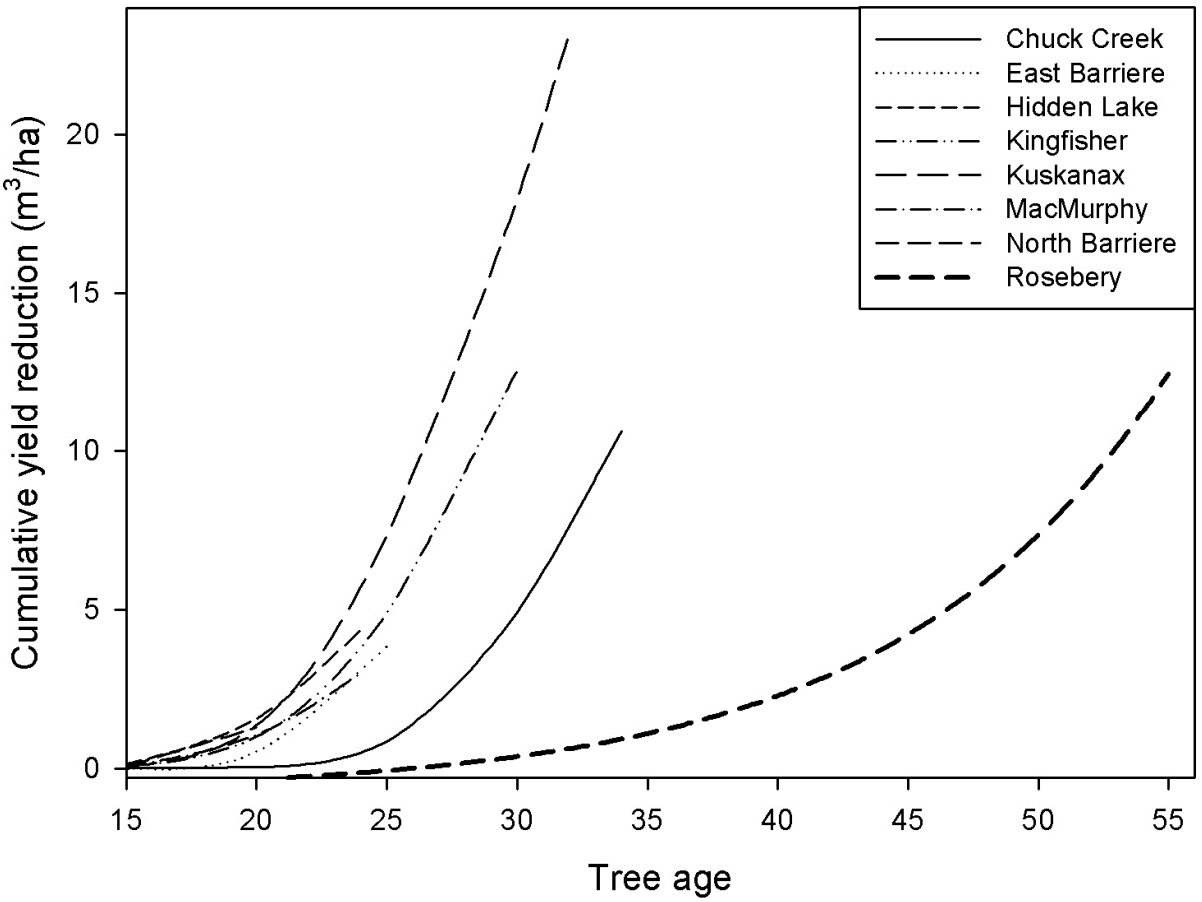

Figure 6. Predicted cumulative yield reduction per ha versus tree age for seven planted and the naturally regenerated site Rosebery.

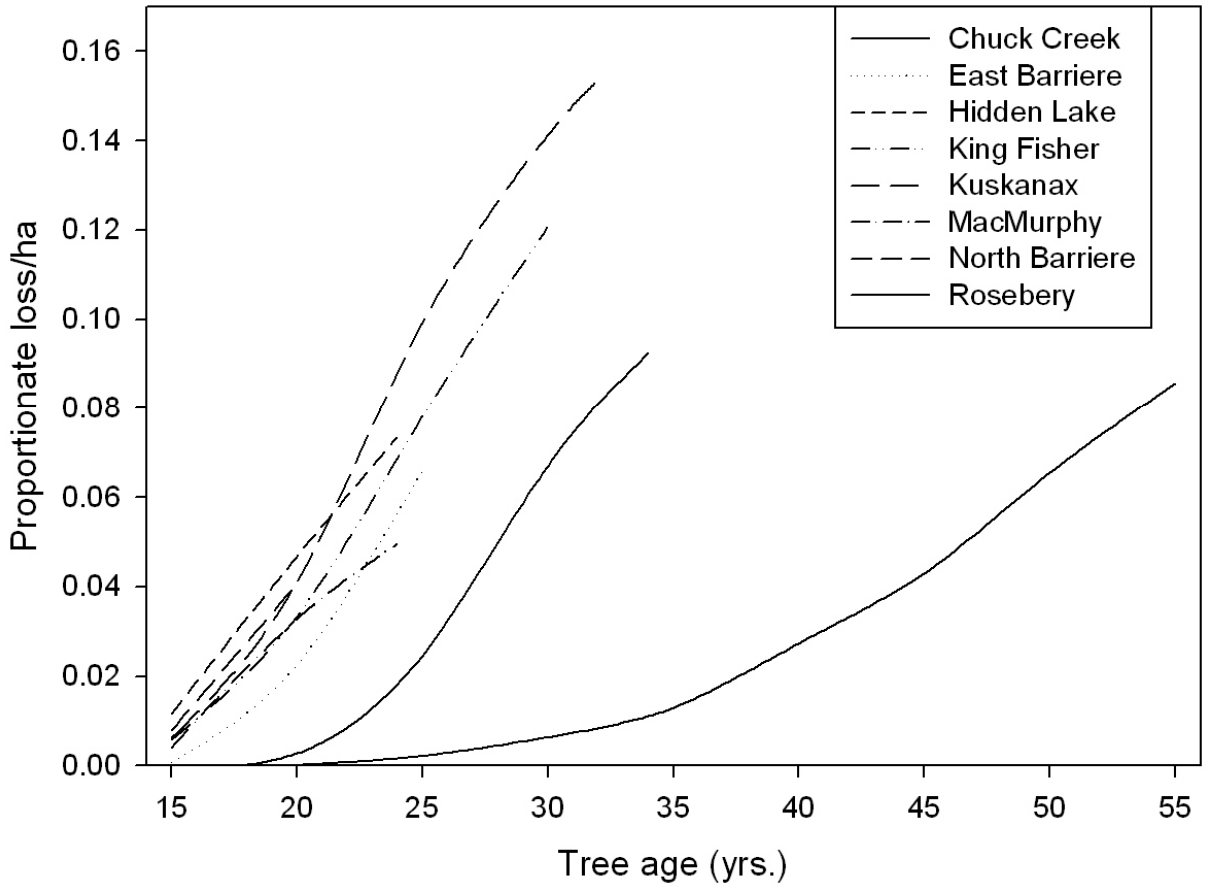

Figure 7. Predicted proportional yield reduction per ha versus tree age for seven planted sites and the naturally regenerated site Rosebery.

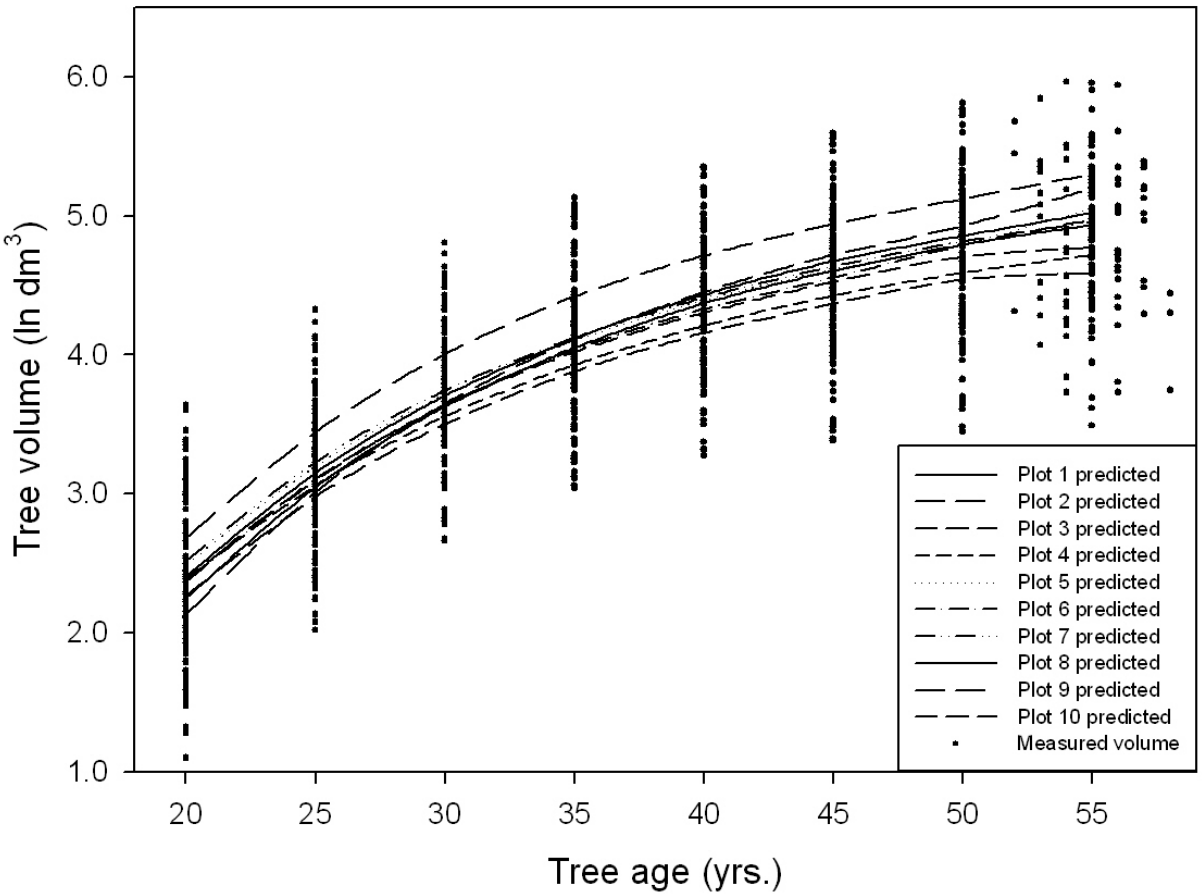

Figure 8. Predicted average and actual tree volume versus tree age for 10 plots at the naturally regenerated Rosebery site.

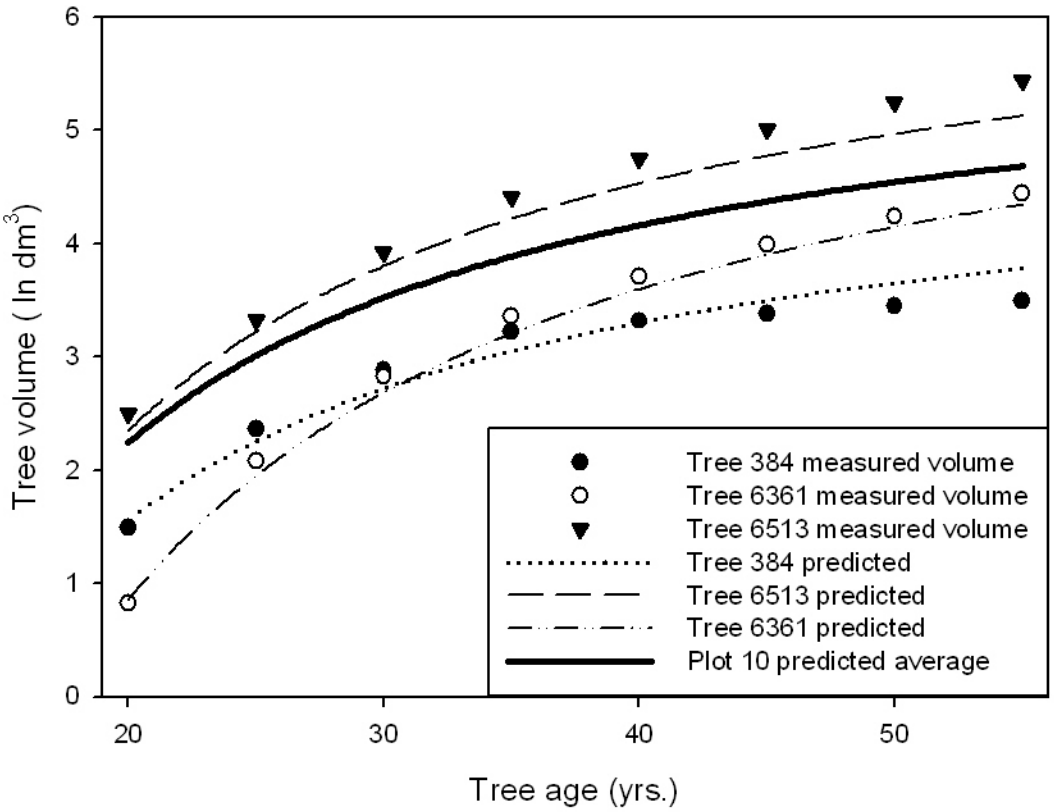

Figure 9. Predicted and measured tree volume versus tree age for three trees with significant BLUPs in plot 10 on the Rosebery site. Diseased tree 384 has lower slope and diseased trees 6361 and 6513 have higher slope than average.