Allometries of coarse tree, stem, and crown measures in Douglas-fir are altered by Armillaria root disease

M.G. Cruickshank and C.N. Filipescu

Abstract: We used allometric relationships to quantify Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) stem and crown adaptation to Armillaria root disease (caused by *Armillaria ostoyae* (Romagn.) Herink). At four sites, we measured height, diameter, height to live crown, crown width and length, sapwood area at base of live crown, and infection duration for healthy and infected Douglas-fir trees. Diseased trees were on average smaller than healthy trees for all measured variables, but there were also proportional changes between tree parts suggesting allocation shifts to disease. Infected trees were shorter in relation to stem diameter compared with healthy trees by 4% on average. Crown diameter was positively related to stem diameter (0.24 $m \cdot cm^{-1}$) but not to disease or competition. Diseased tree crown lengths were on average 0.5 m shorter for a given crown diameter than healthy trees—akin to response to light competition except this also occurred in the upper canopy. Prolonged infection reduced crown length probably through shedding of lower branches and by reducing stem apical growth, possibly related to changed hydraulic architecture or light requirements. Crown surface area was related to stem sapwood area (0.81 $m^2 \cdot cm^{-2}$) but unaffected by disease or competition. We discuss how shifting allocation could reveal important implications for life strategies involving whole tree adaptations to disease and tree to tree interactions, and for wood quality and forest inventory.

Key words: Armillaria, allometry, disease resistance/tolerance, Douglas-fir, tree crown.

Résumé : Les auteurs ont utilisé les relations allométriques pour quantifier l'adaptation de la tige et de la couronne du sapin Douglas (*Pseudotsuga menziesii* (Mirb.) Franco) à la pourriture par l'armillaire (provoquée par *Armillaria ostoyeae* (Romagn.) Herink). Ils ont mesuré, sur quatre sites, la hauteur, le diamètre, la hauteur jusqu'à la couronne vivante, la largeur et la longueur de la couronne, l'aire de l'aubier à la base de couronne vivante et la durée de l'infection, chez des sapins Douglas sains et infectés. Les arbres malades sont généralement plus petits que les arbres sains pour toutes les variables mesurées, mais on observe également des modifications proportionnelles entre les parties de l'arbre, suggérant un déplacement de l'allocation vers la partie malade. Les arbres infectés sont plus courts en relation avec le diamètre de la tige comparativement aux arbres sains, de 4 % en moyenne. On observe une corrélation positive entre le diamètre de la couronne (0,24 $m \cdot cm^{-1}$) mais non pas avec la maladie ou la compétition. Les longueurs des couronnes chez les arbres malades sont en moyenne plus courtes de 0,5 m, pour un diamètre de la couronne donné, que chez les arbres sains—proche de la réaction à la compétition lumineuse, sauf que ceci survient également dans la canopée supérieure. Une infection prolongée réduit la longueur de la couronne probablement par l'ombrage des branches inférieures et par la réduction de la croissance apicale de la tige, possiblement en lien avec un changement de l'architecture hydraulique ou des besoins en lumière. L'aire de la surface de la couronne est reliée avec l'aire de l'aubier (0,81 $m^2 \cdot cm^{-2}$), mais non affectée par la maladie ou la compétition. Les auteurs discutent comment le déplacement de l'allocation pourrait révéler d'importantes implications pour les stratégies vitales faisant intervenir des adaptations de l'ensemble de l'arbre à la maladie et aux interactions d'arbre en arbre, ainsi que la qualité du bois et l'inventaire forestier.

Mots-clés : Armillaria, allométrie, résistance/tolérance à la maladie, sapin Douglas, couronne de l'arbre.

[Traduit par la Rédaction]

Introduction

Allometry quantifies the changes in dimensions of an organism's parts relative to changes in overall organism size (Huxley and Teissier 1936). For trees, relationships among height, crown, stem, and roots are highly correlated because of structural and physiological requirements among different plant parts (Bartelink 1996). Phenotypic plasticity allows trees to alter different plant parts in response to environmental stress, and this can affect allometric relationships because changes in physiology of one part can affect a more distant tissue (Kozlowski 1969). Allometry has a wide range of uses in ecology and forestry, such as determining tree height from easily obtained measures like diameter. From a practical point

Received 6 January 2012. Accepted 31 March 2012. Published at www.nrcresearchpress.com/cjb on xx July 2012.

M.G. Cruickshank and C.N. Filipescu. Natural Resources Canada, Canadian Forest Service, Canadian Wood Fibre Centre, Pacific Forestry Centre, 506 W. Burnside Road, Victoria, BC V8Z 1M5, Canada.

Corresponding author: M.G. Cruickshank (e-mail: mcruicks@nrcan.gc.ca).

of view, quantifying changes in tree diameter and height in relation to crown area is important for disease simulation and impact models (Bloomberg 1988) and models of timber yield and value (Mitchell 1988). Allometry has only rarely been used to examine the potential of whole plant adaptation to disease and a variety of coinciding stresses (Sadras et al. 2000; Weiskittel 2003). No studies exist examining how root disease affects allometry of conifers.

Allometric studies of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) relating to stand density, shade, and disease suggest a requirement to balance resource capture with respiratory, growth, and physical/structural restrictions faced by the tree. Stem diameter at breast height (dbh) is positively but nonlinearly related to height, linearly to crown radius, and positively related to needle area and crown length mainly for structural reasons; further, needle biomass to branch biomass is greater in the upper crown where light is the strongest because of position (Bartelink 1996). Tree height to diameter ratio is reduced at lower stand density mainly owing to wind and snow stability (Wilson and Oliver 2000), and increased with lower light levels mainly owing to slower diameter growth (Chen 1997). Shading favors crown lateral growth over terminal growth (Chen et al. 1996). Allometry is also affected by shade through lower crown length to tree height ratio and the number of branches per whorl, which are thought to be adjustments to increase light capture and reduce respiratory costs (Williams et al. 1999). Trees that partition more to crown diameter versus length may have greater drought tolerances owing to lower crown volumes and transpiration; however, this may be less advantageous in competitive highly productive dense stands (St Clair et al. 2005). Specific leaf area (SLA, the ratio of projected leaf area to leaf dry mass) describes the light capture relative to the biomass invested in the leaf, and this increases towards the crown base where shade is greater (Marshall and Monserud 2003). The disease Swiss Needle Cast (caused by *Phaeocryptopus gäumannii)* causes smaller leaf length and width with higher SLA, fewer branch laterals on primary branches, higher branch length to diameter, shorter crown length to width, and greater height to live crown; consequently, an increase of the relative proportion of foliage in the upper crown may reduce maintenance respiration and structural allocation costs while maximizing resource capture (Weiskittel 2003). Longer crown length to tree height was associated with superior volume growth (disease tolerant) in trees infected with root disease, but this was limited to smaller tree size when first infected (Cruickshank et al. 2011), possibly suggesting carbon or hydraulic limitation in larger trees.

In British Columbia, Douglas-fir is susceptible to at least three common root diseases: Armillaria, Phellinus, and Annosus root diseases (Morrison 1979; Morrison et al. 1991; Thies and Sturrock 1995), and at least one of these in many other areas of the world. In its interior range in British Columbia, Douglas-fir is moderately shade tolerant (Burns and Honkala 1990) and is also highly susceptible to Armillaria root disease. *Armillaria ostoyae* (Romagn.) Herink spreads slowly to new roots from multiple infection sources by root contacts, across small gaps between roots via rhizomorphs, but rarely by spores, and inoculum survives for many years on site. The host range of *A. ostoyae* covers many tree species; as a result, the fungus occurs circumpolar in the northern hemisphere with more than 30 additional species of this fungus on other forested continents. Given enough inoculum the fungus can kill trees, but more commonly infected trees survive for decades often with ongoing root infection events (Cruickshank et al. 2011). Signs and symptoms of this disease are difficult to detect aboveground as a result. The pattern of fungal infection and spread creates a spatial dynamic where healthy and diseased trees occur as neighbours and where infected trees can be outgrown by healthy neighbours because disease reduces height, diameter, and volume growth (Cruickshank et al. 2009, 2011). Infected trees are faced not only with impaired root systems and activation of costly host defense, but also with other important constraints such as competition for light and maintaining water balance, all of which affect photosynthesis and growth. The amount of light trees intercept is an important determinant of tree growth (Wright et al. 1998). Tree crowns might be linked to how trees respond to disease in terms of allocation to growth, defense (Cruickshank et al. 2011), and resource capture.

We investigated the effect root disease has on coarse tree crown and stem allometry in Douglas-fir trees. The objectives were to examine if diseases alters allocation between tree parts within and between the stem and crown.

Materials and methods

Study area

This study was conducted in Douglas-fir plantations within the Interior Cedar Hemlock (ICH) biogeoclimatic zone (Braumandl and Curran 1992) of British Columbia. Forests of the ICH zone are amongst the most productive forests in Canada and have the highest diversity of tree species (Meidinger and Pojar 1991). The ICH zone has warm dry summers and cold wet winters, and much of the winter precipitation falling as snow.

Armillaria ostoyae occurs throughout many biogeoclimatic zones in British Columbia, including the Interior Douglas-fir, montane spruce, Engelmann spruce–subalpine fir, and ICH (Morrison et al. 1991), but disease incidence and the damage it causes are highest in the ICH zone, where we conducted our study. Under natural conditions, the Interior variety of Douglas-fir (*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco) is frequently one of the dominant tree species from early to mid-seral succesional stages of stand development and is commonly infected by *A. ostoyae*. Interior Douglas-fir is moderately shade tolerant and adapts to a wide range of soil moisture conditions (Burns and Honkala 1990).

Plot locations and tree measurements

Four 24- to 34-year-old Douglas-fir plantations in the ICH zone were sampled: Chuck Creek (CC) near Clearwater, East Barriere (EB) near Barriere, Kingfisher (KF) near Enderby, and Kuskanax (KX) near Nakusp (Table 1). We selected plantations 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 plantations chosen are representative of other forest plantations in the region. All plantations but one were clearcut and reforested with Interior Douglas-fir; the other plantation (KX) was reforested after a wildfire. Within each plantation, 23–25 10 m radius plots

Table 1. Summary of study site data.

Attribute	Chuck Creek (CC)	East Barriere (EB)	Kingfisher (KF)	Kuskanax (KX)
Latitude/longitude	51.6°N, 119.7°W	51.3°N, 119.8°W	50.7°N, 118.8°W	50.2°N, 117.8°W
Elevation (m)	690	700	420	480
Site index (m at age 50)	26.8	28.2	29.2	29.2
Tree age at sampling	34	25	30	32
No. of plots (plot radius in m)	25 (10)	23 (10)	25 (10)	25 (10)
Basal area ($m^2 \cdot ha^{-1}$)	22.1	17.4	23.0	27.5
Mean dbh (cm) (min., max.)	16.8 (7.8, 30.5)	13.8 (6.5, 24.4)	15.1 (6.8, 26.0)	16.2 (6.9, 28.2)
Mean height (m) (min., max.)	13.1 (6.2, 18.7)	9.8 (5.6, 13.0)	13.0 (5.9, 18.2)	14.4 (6.4, 19.9)
Mean crown length (m) (min., max.)	9.6 (3.3, 15.1)	8.7 (4.5, 12.5)	8.9 (3.3, 14.9)	8.5 (1.5, 15.9)
Mean crown diameter (m) (min., max.)	4.08 (1.4, 6.25)	3.28 (1.6, 5.05)	3.5 (2.1, 6.2)	3.4 (1.8, 5.4)
Mean number of stems/plot (min., max.)	33 (19, 48)	44 (31, 72)	39 (26, 55)	39 (21, 53)
Mean height to live crown (m) (min., max.)	3.6 (1.9, 6.7)	1.1 (0.1, 4.7)	4.1 (1.4, 8.5)	5.8 (2.3, 9.0)
Mean Douglas-fir sampled/plot (min., max.)	6 (2, 6)	8 (4, 14)	8 (4, 12)	7 (5, 11)
Proportion of dead trees/plot (min., max.)	0.04 (0.00, 0.25)	0.04 (0.00, 0.13)	0.06 (0.00, 0.24)	0.03 (0.00, 0.09)
Belowground proportion of live Douglas-fir with *A. ostoyae*/plot (min., max.)	0.30 (0.00, 0.76)	0.33 (0.15, 0.49)	0.52 (0.13, 0.90)	0.59 (0.27, 0.88)
Proportion of Douglas-fir in plots/plot (min., max.)	0.89 (0.07, 1.00)	0.80 (0.69, 0.97)	0.86 (0.69, 1.00)	0.94 (0.78, 1.00)
Mean proportion of Douglas-fir primary roots infected (min., max.)	0.31 (0.06, 1.00)	0.39 (0.07, 1.00)	0.30 (0.07, 1.00)	0.31 (0.07, 1.00)
Mean years Douglas-fir was infected (min., max.)	11 (2, 20)	7 (1, 15)	13 (2, 23)	15 (1, 25)

(0.03 ha) were randomly distributed on either side of the main access road to the plantation.

In each plot, all dead and living trees were tagged, and tree diameter at 1.3 m (dbh), as well as crown radius (m) at cardinal points, was measured. Crown area or diameter was based on the average radius of the cardinal measures. 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. There was minimum breakage of roots, stems, and branches. Pulled trees were gently laid on the ground and left over winter. Tree height (m) and height to lowest live branch (m) were measured after the trees were pulled. After the soil thawed the following spring, soil was removed by hand from the roots of all trees in the plots. Lesions attributed to *A. ostoyae* were identified based on mycelial fans in the bark or cambium of the roots. For all trees, the proportion of diseased primary roots (>15 mm arising from the root collar) and height from the soil line to the lowest live branch and the tree apex were recorded.

Several Douglas-fir trees in each plot were identified for additional sampling to determine length of infection (median 5–8 trees per plot, Table 1). This was achieved by stratified random sampling from two dbh 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 (718 trees total). An attempt to fill the sampling matrix was made for all plots, but at least two trees were sampled in each plot. A disk at the base of the live tree crown, defined as the lowest live branch, was taken from about 25 randomly selected trees per site (97 trees total) to determine sapwood area. Sapwood area was marked by visual inspection of the disk and the area was determined by digital integration.

Stumps from the random subsample of diseased trees were transported to Victoria, British Columbia, and dissected to determine how long each tree had been infected. Each root lesion was located spatially in the horizontal plane, and 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 disks were air dried and sanded, and traumatic resin canals invoked by fungus were traced along the root disks into the root collar section and lower stem to date each infection (Cruickshank et al. 2006). Dead roots and root collar infections were traced back into the living root collar tissue to determine their age.

To evaluate the impact of competition, a nonspatial competition index was calculated for each sampled tree using the sum of the plot basal area of trees that are larger (BAL $m^2 \cdot ha^{-1}$) than the target tree (Wykoff et al. 1982).

Statistical models

Nonlinear mixed models testing the relationship between dbh, height, crown, competition, and disease were run using the nlme package in R (Ver. 2.12.0), and linear models were run using SAS Proc Mixed (Ver. 9.2). Models were assessed using likelihood ratio tests and Akaike's information criteria (AIC). Lower values indicate better fit. The solutions for the random effects were output as best linear unbiased predictors (BLUPs). Residuals were checked for normality and homoscedasticity against predicted and independent variables.

dbh models

The effect of disease on dbh and its relationship to tree height, crown area, and competition were first tested using fixed effects. The best-fitting model describing diameter (summarized in Table 2) was a function of:

$$[1] \qquad \mathrm{dbh} = (\beta_0 + \beta_1 \mathrm{BAL})(\mathrm{HT} - 1.3)^{\beta_2} \beta_3^{(\mathrm{HT}-1.3)} \mathrm{CA}^{\beta_4}$$

where dbh is the diameter at εbreast height, HT is the height of the tree, CA is the crown area of the tree, and BAL is the basal area of larger trees in the plot.

We then added the random effect of plot j nested within site location k, and location k variables, to the model in eq. 1 to get eq. 2. A difference of 17.4 between the –2 log likelihoods of eqs. 2 and 3 indicated that infection duration

Table 2. Models considered for testing the effects of tree height, competition, and crown variables on tree diameter (dbh).

Model	Residual (cm) SE	AIC
$\text{dbh} = (\beta_0 + \beta_1\text{BAL})(\text{HT} - 1.3)^{\beta_2}\beta_3^{(\text{HT}-1.3)}\text{CA}^{\beta_4}$	1.585	2694.9
$\text{dbh} = (\beta_0 + \beta_1\text{BAL})(\text{HT} - 1.3)^{\beta_2}\beta_3^{(\text{HT}-1.3)}\text{CL}^{\beta_4}$	1.931	2977.4
$\text{dbh} = (\beta_0 + \beta_1\text{BAL})(\text{HT} - 1.3)^{\beta_2}\beta_3^{(\text{HT}-1.3)}\text{CSA}^{\beta_4}$	1.712	2804.6
$\text{dbh} = (\beta_0 + \beta_1\text{BAL})(\text{HT} - 1.3)^{\beta_2}\beta_3^{(\text{HT}-1.3)}\text{CV}^{\beta_4}$	1.648	2750.4
$\text{dbh}_{ijk} = (\beta_0 + \beta_1\text{BAL}_{ijk} + \beta_5\text{INF}_{ijk})(\text{HT}_{ijk} - 1.3)^{\beta_2}\beta_3^{(\text{HT}_{ijk}-1.3)}\text{CA}_{ijk}^{\beta_4}$	1.582	2693.1
$\text{dbh}_{ijk} = (\beta_0 + \beta_1\text{BAL}_{ijk} + \beta_5\text{PINF}_{ijk})(\text{HT}_{ijk} - 1.3)^{\beta_2}\beta_3^{(\text{HT}_{ijk}-1.3)}\text{CA}_{ijk}^{\beta_4}$	1.578	2689.0
$\text{dbh}_{ijk} = (\beta_0 + \beta_1\text{BAL}_{ijk} + \beta_5\text{YRI}_{ijk})(\text{HT}_{ijk} - 1.3)^{\beta_2}\beta_3^{(\text{HT}_{ijk}-1.3)}\text{CA}_{ijk}^{\beta_4}$	1.576	2690.0

Note: BAL, competition index ($m^2 \cdot ha^{-1}$); HT, tree height (m); CA, crown area (m^2); CL, crown length (m); CSA, crown surface area (m^2); CV, crown volume (m^3); PINF, proportion of primary roots infected; YRI, number of years since first infection. INF is indicator variable 0 if infected, 1 if healthy.

(YRI) had a significant improvement ($p < 0.0001$) on the final model.

$$[2]\quad \text{dbh}_{ijk} = (\beta_0 + \beta_1\text{BAL}_{ijk})(\text{HT}_{ijk} - 1.3)^{(\beta_2 + b_{2jk} + b_{2k})}\beta_3^{(\text{HT}_{ijk}-1.3)}\text{CA}_{ijk}^{\beta_4} + \varepsilon_{ijk}$$

$$[3]\quad \text{dbh}_{ijk} = (\beta_0 + \beta_1\text{BAL}_{ijk} + \beta_5\text{YRI}_{ijk})(\text{HT}_{ijk} - 1.3)^{(\beta_2 + b_{2jk} + b_{2k})}\beta_3^{(\text{HT}_{ijk}-1.3)}\text{CA}_{ijk}^{\beta_4} + \varepsilon_{ijk}$$

where YRI_{ijk} is the number of years since infection for tree i in plot j in location k, BAL_{ijk} is the competition index for tree i in plot j in location k, HT_{ijk} is the total height (m) of tree i in plot j in location k, CA_{ijk} is the crown area (m^2) of tree i in plot j in location k, b_{2k} is the random effect parameter for location k, b_{2jk} is the random effect parameter for plot j in location k, and ε_{ijk} is the random error.

Crown and sapwood models

The crown diameter and the height to lowest live branch was modeled according to:

$$[4]\quad \text{Y}_{ijk} = \beta_0 + \beta_1\text{BAL}_{ijk} + \beta_2\text{YRI}_{ijk} + \beta_3\text{HT}_{ijk} + b_1\text{location}_k + b_2\text{plot}_{jk} + \varepsilon_{ijk}$$

where the model terms are the same as in eq. 3, except that Y_{ijk} is the crown diameter or height to live crown (m) for tree i in plot j nested in site location k.

The relationship between crown length, competition, tree height, crown diameter, and disease was modeled according to:

$$[5]\quad \text{Y}_{ijk} = \beta_0 + \beta_1\text{BAL}_{ijk} + \beta_2\text{YRI}_{ijk} + \beta_3\text{HT}_{ijk} + \beta_4\text{Crown Dia}_{ijk} + b_1\text{location}_k + b_2\text{plot}_{jk} + \varepsilon_{ijk}$$

where all variables are the same as for eq. 3, except that Y_{ijk} is the crown length (m) and Crown Dia_{ijk} is the diameter of the crown (m) for tree i in plot j nested in site location k.

Crown surface area was calculated based on a circular cone and was modeled as:

$$[6]\quad \text{Y}_{ik} = \beta_0 + \beta_1\text{sapwood}_{ik} + \beta_2\text{HT}_{ik} + b_1\text{location}_k + \varepsilon_{ik}$$

where Y_{ik} is the surface area of the crown (m^2) and sapwood $_{ik}$ is the area of sapwood at the base of the live crown (cm^2), HT is the tree height (m), and location is the random effect of site location for tree i in location k.

Sapwood area was modeled as:

$$[7]\quad \text{Y}_{ik} = \beta_0 + \beta_1\text{BAL}_{ik} + \beta_2\text{CSA}_{ik} + \beta_3\text{BAL} \cdot \text{CSA}_{ik} + b_1\text{location}_k + \varepsilon_{ik}$$

where Y_{ik} is the sapwood area at the base of the live crown (cm^2), BAL is the competition index, CSA (m^2) is the surface area of the crown (m^2), and location is the random effect for site location for tree i in location k.

To control for heteroscedasticity in eqs. 6 and 7, the Power of X model of within subject variation (Littell et al. 2006) was used:

$$\text{Var}[\varepsilon_i] = \sigma^2\exp(x_i \cdot \gamma)$$

where ε_i is the residual variance, σ^2 is the estimated variance, and γ is an estimated dispersion factor.

Results

Tree diameter models

The significant explanatory variables in predicting tree dbh were the competition index (BAL), tree height, and crown area (summarized in Table 3). Using average values for explanatory variables, the model predicts that infected trees were 4% larger in dbh (9% basal area) after 25 years of infection. In other words, when stem diameter and crown area are accounted for, infected trees are shorter. Stem diameter in relation to tree height is affected by both crown area and disease (Fig. 1). We determined that a categorical predictor for disease and no disease was less effective than a continuous effect for either of the proportion of infected roots or the time the tree had been infected (Table 2). We chose the time the tree had been infected as a predictor, and it is correlated to the proportion of infected roots (Spearman $r = 0.82$). The years of infection had proved to be a better overall predictor in another study (Cruickshank et al. 2011) and in crown models in the next section of this study. Random variation was greatest between plots within site location rather than between locations, but this variation was not large.

The average ratio between height and diameter for the lowest 25th percentile of BAL (low competition) was 0.70 and 0.72 for infected trees and healthy trees, respectively. The average ratio between height and diameter for the highest 25th percentile of BAL (high competition) was 1.00 and 1.20 for infected trees and healthy trees, respectively. The average of

Table 3. Maximum likelihood model coefficients for parameters predicting tree dbh from competition, height, crown area, and the number of years the tree had been infected.

Variable—eq. 3	Estimate	SE	*P*
β_0 Intercept	2.212	0.264	<0.0001
β_1 BAL	–0.034	0.004	<0.0001
β_2 Tree height	0.977	0.086	<0.0001
β_3 Tree height	0.949	0.007	<0.0001
β_4 Crown area	0.170	0.010	<0.0001
β_5 Years infected	0.003	0.0009	<0.0001
b_2 Location (var.)	2.89×10^{-4}		
b_2 Plot within location (var.)	8.41×10^{-4}		
Residual	1.225		
AIC	2413.8		
BIC	2454.9		
–2 Log likelihood	–1197.9		

actual data for dbh and height for healthy trees were 16.7 cm (±5.2 SD) and 13.2 m (±3.1), respectively, and 14.7 cm (±3.9) and 12.2 m (±2.7) for infected trees, respectively.

Crown and sapwood models

The crown diameter model (intercept = 1.44 m) was best fit using only the fixed effect of stem diameter (coefficient = 0.14 cm) and the random effects from eq. 4 rather than including the duration of infection, competition, and (or) tree height (Fig. 2). The vertical distance to lower live crown (eq. 4) was increased by greater duration of infection, competition, and tree height (not shown); consequently, using average values of the predictor variables in the model, live crown is 3.77 m, which is 0.41 m higher for a tree infected for 25 years compared with height of crown for a healthy tree (3.36 m). Crown length (eq. 5) was positively correlated with crown diameter and tree height, but became increasingly smaller with greater number of years the tree was infected and with greater levels of competition (Table 4). The crown length model predicts a tree with average crown diameter, height, and competition to be approximately 0.46 m shorter than a healthy tree (9.81 m). Crown length as a proportion of crown diameter (Fig. 3) and crown length as a proportion of tree height (Fig. 4) are both reduced with increasing time since first infection.

Crown surface area was positively related to sapwood area at base of live crown and to tree height, but not to BAL or the length of time the tree had been infected; consequently, these last two terms were dropped from the model (eq. 6; Table 5). Actual data for crown surface area averaged 46 m^2 for infected trees and 63 m^2 for healthy trees, and the average ratio of crown surface area to sapwood area for all trees was 0.81 $m^2 \cdot cm^{-2}$. Crown surface area relative to tree height covered a range of values, but it was always related to the sapwood area at the base of live crown (Fig. 5).

Sapwood area at the base of the live crown (eq. 7) was negatively related to the level of competition and positively to the crown surface area, and crown surface area and competition also interacted with each other negatively on crown surface area (not shown). The average of the actual data for sapwood area versus crown surface area was 1.36 $cm^2 \cdot m^{-2}$.

Random effects and model fits

For all models, except the dbh model, random variation was largest between site locations compared with between plots within site locations, but there were no clear trends associated with the magnitude or sign of the BLUP coefficients. Sites CC and KX were nearly consistent in that they had the most extreme site BLUPS, and the sign of BLUPs shifted depending on the model but were always the opposite sign to the other site. The only exception was site EB associated with a larger site BLUP for crown length compared with the other sites. Sites CC and KX contained the largest average trees and were the oldest sites (Table 1).

Model fits for final models and for other important models that were tested are given in Table 6. The simplest model was taken as the best model unless more complex models were justified by a likelihood ratio test.

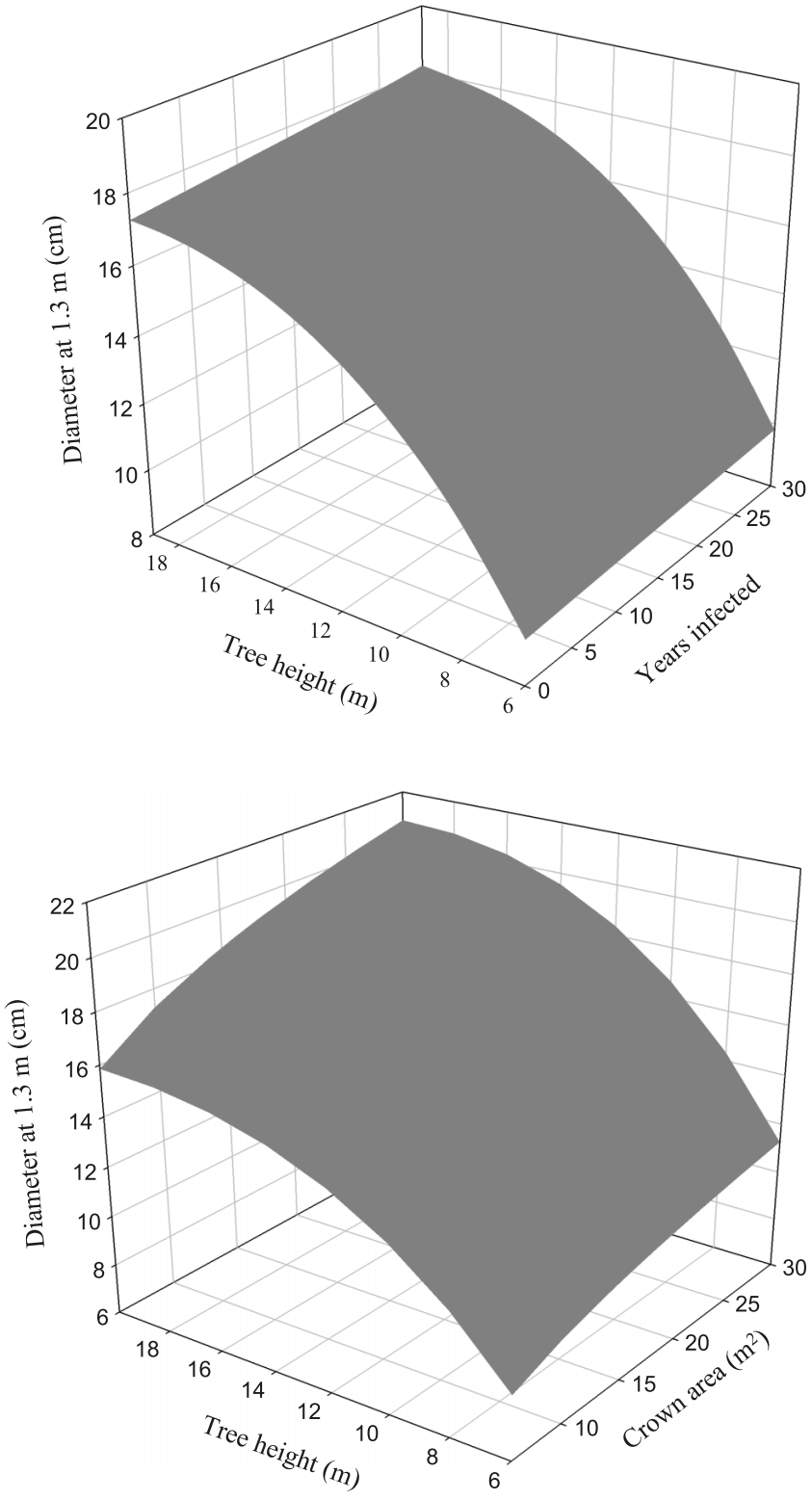
Discussion

We found that diseased trees were smaller compared with healthy trees for most of the variables measured (e.g., dbh), but had larger stem diameters than would be expected for a particular tree height, or shorter than expected heights for a given diameter. This was consistent for trees in the upper and lower sections of the canopy. Stem height and diameter are both reduced by disease (Cruickshank et al. 2009) but apparently disease, unlike competition, impacts height growth more severely in proportion to stem diameter. Similarly, crown length in diseased trees was shorter than expected for a given crown diameter compared with healthy trees. Crown length is probably reduced by disease through reduced terminal growth (Cruickshank et al. 2009) and by shedding of branches in the lower live crown. Crown diameter was strongly linked to stem diameter but not to infection duration. Interestingly, lateral root spread has been found to be correlated with crown radius in conifers (Smith 1964), suggesting that lateral crown, stem, and root growth might be linked. Crown surface area and sapwood area at the base of live crown were correlated, but both appear to be proportionally reduced in diseased trees. Sapwood area at base of live crown is a surrogate for transpiring leaf area (Maguire and Hann 1989). This does not preclude the possibility that there may be other differences such as the distribution of foliage within the crown, the years of needle retention, crown profile, or the relation between leaf area and branch size, none of which we studied. Our study trees are approaching mid-rotation, and the effects described here will continue to accrue over time so that many of the diseased trees will experience increased shading and competition for light and water.

Grime (1977) argued that plants in productive habitats compete following four general strategies: (*i*) dense canopies that exclude resources (e.g., light) from competitors; (*ii*) large lateral crown and root spread; (*iii*) highly plastic phenotypes tending to maximize vegetative growth and size; or (*iv*) high uptake of water and nutrients. Douglas-fir has many of these traits, including rapid growth and phenotypic plasticity in the face of stress agents (Burns and Honkala 1990). Reduced crown and tree height, loss of root system function through repeated attacks, and induction of host defense presents a serious long-term problem for a tree to cope with.

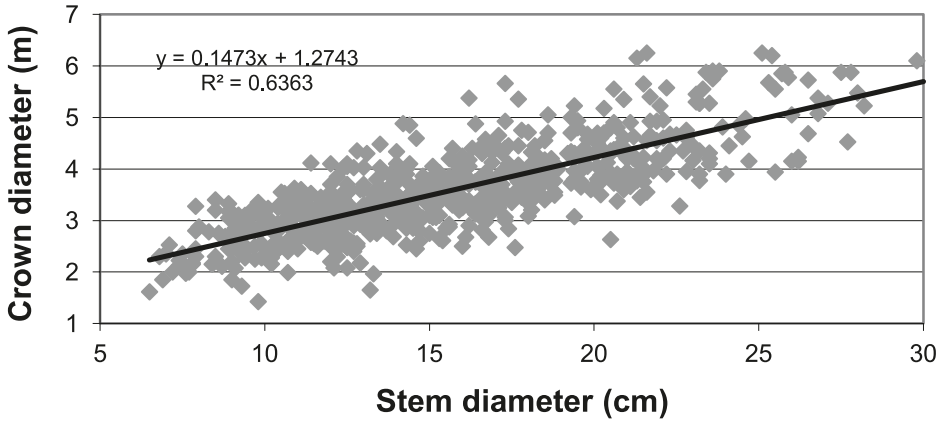
Shade produces a physiological shift in conifer crowns, where lateral growth is favored over terminal growth to allow better light capture for species capable of crown plasticity

Fig. 1. Predicted stem diameter as a function of tree height at mean competition (BAL) and infection age (top panel) and as function of crown area and tree height at mean time infected (low panel).



like Douglas-fir (Oliver and Larson 1990; Chen et al. 1996), causing reduced branch number and spacing in conifers (Stenberg et al. 1994; Van Pelt and Sillett 2008). This is similar to the effect we describe here for root disease, except that disease also affects the largest trees in a similar fashion. A vertically restricted, compact crown or canopy has less allocation to branch biomass for a given leaf area (Smith and Long 1989; Bartelink 1996). Other allometric changes affecting assimilation and allocation can include: (*i*) epicormic shoots on branches and stems to increase leaf relative to

Fig. 2. The relationship between crown diameter and stem diameter at 1.3 m.



branch diameter (Van Pelt and Sillett 2008); (*ii*) lower leaf thickness, stomatal density (related to water use efficiency and drought tolerance) per leaf surface area, or branch diameter; and (*iii*) higher specific leaf area relating allocation to leaf structure vs. surface area (Chen et al. 1996; Abrams and Kubiske 1990). Some of these effects have been reported for the disease Swiss needle cast in Douglas-fir (Weiskittel 2003).

Equal distribution of water within the canopy to prevent upper leaf stomatal closure is a serious problem facing trees especially if it leads to cavitation (Zimmermann 1978). Trees with suddenly damaged root systems combined with frequent summer drought are at increased risk; hence, hydraulic architectural requirements along the bole may also result in vertically restricted crowns. Root diseased trees probably have increased stem water potential after root attack compared with a previously healthy condition. Interestingly, higher water potential causes lower cell turgor pressure that has been proposed as a mechanism that leads to less height growth in trees (Ryan et al. 2006).

Allometric relationships between tree height, crown profile, stem diameter, and root systems are strongly linked by the need for mechanical compression, shear, and bending strength, and vascular function; however, there is limited information on how roots affect stem and crown growth, and even less information concerning how a root disease may impact these (Bloomberg and Hall 1986). Phellinus root disease affected Douglas-fir stem diameter or basal area more than height growth (Bloomberg and Reynolds 1985), and root or butt rot affected stem diameter more than height growth in black spruce (*Picea mariana*), but not balsam fir (*Abies balsamea*) or white spruce (*Picea glauca*) where they were both reduced equally (Whitney 1976). Although these studies did not compare height and diameter in an allometric fashion, differences in stand density alone could be an explanation for the varying results because lower density favors diameter growth over height growth (Wilson and Oliver 2000); however, this effect was controlled in the current study and the stands were originally planted at the same density. Mortality in the current study plots was low, and loss of a neighbour due to Armillaria root disease does not appear to affect growth of the remaining trees in stands of this age; this occurs for a variety of reasons outlined in Cruickshank et al. (2009), but chiefly because the infected trees still effectively compete with neighbours.

Table 4. Maximum likelihood model coefficients for parameters predicting tree crown length from competition, height, crown area, and the number of years the tree had been infected.

Variable—eq. 5	Coefficient	SE	*P*
β_0 Intercept	–1.53	0.790	<0.1022
β_1 BAL	–0.02	0.008	<0.0033
β_2 Years infected	–0.02	0.005	<0.0012
β_3 Height	0.72	0.024	<0.0001
β_4 Crown dia.	0.51	0.068	<0.0001
b_1 Location (var.)	1.76	1.465	0.1137
b_2 Plot within location (var.)	0.16	0.043	<0.0001
Residual	0.83	0.048	<0.0001
AIC	2036.7		
BIC	2034.9		
–2 Log likelihood	2030.7		

Armillaria ostoyae progressively attacks a root system over many years mainly using rhizomorphs; as a result, the intensity of root system infection within the tree generally increases over time. We found that the time since infection rather than the proportion of roots infected was the best measure of disease in the root system in this study and one other study (Cruickshank et al. 2011); however both variables are highly correlated. Time might be a slightly better predictor of allometric alteration resulting from a slowly spreading disease, because it more carefully reflects the temporal process of adjusting assimilate partitioning among respiration, structural components, and defense sinks. Moreover, trees respond to an attack in partly through systemic responses that go beyond local damage (Cruickshank et al. 2009), so measures of root system damage may not measure defense sink allocation completely accurately.

Differences in phenotypic response to disease could be an important adaptive trait relating to host defense strategies, like resistance and tolerance, because of the way it could affect light and water capture and the allocation of resources within the crown and between the stem and roots. This in turn may allow different life history strategies for tradeoffs in allocation to growth, survival, and reproduction within a population. Disease resistant plants limit fungal inoculum or the area of fungal colonization, which often comes at a cost in growth and reproduction to the plant (Mauricio et al. 1997); however, disease-tolerant plants have superior growth

Fig. 3. Crown length to crown diameter ratio is affected by the number of years a tree had been infected.

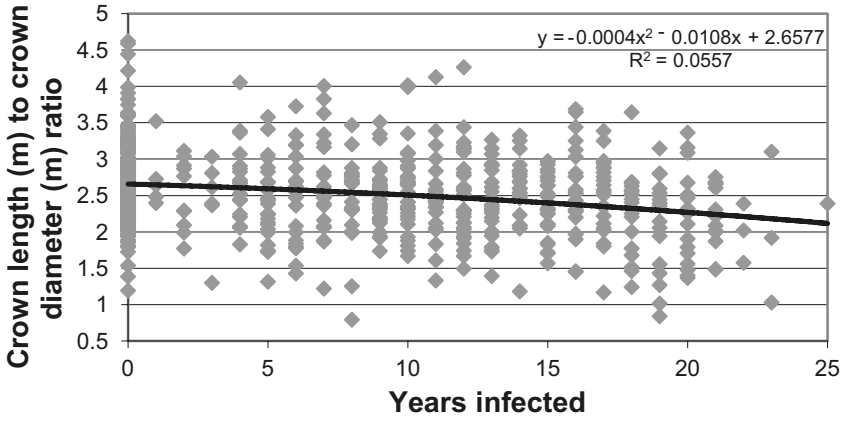
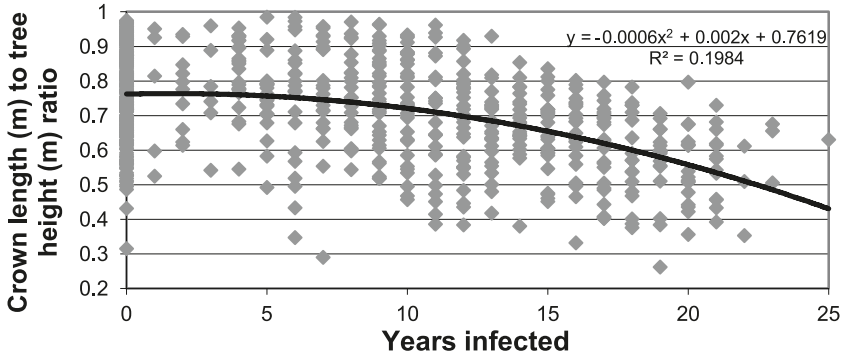


Fig. 4. Crown length to tree height ratio is related to the number of years a tree had been infected.



and reproduction for a given level of damage (Stowe et al. 2000). In a previous analysis of stem volume in the same study trees used here, punitively tolerant and resistant strategies were related to large and small crown length ratio, respectively, for a given level of damage (Cruickshank et al. 2011). We found that average crown length in proportion to tree height was reduced by the length of time infected, but there was also unexplained variation surrounding this relationship, some of which might be related to disease resistance and tolerance.

Fungal-induced resistance requires synthesis of costly compounds in roots representing a strong new sink (Entry et al. 1991); in comparison, tolerant trees could lower allocation to induced defenses to maintain resource capture and growth. Resistance could be favored in situations where damage to root systems increases risk of mortality at the cost of allocation to other parts like the crown. On the other hand, disease-tolerant trees may be able to replace lost roots more easily or be more drought tolerant, allowing the root system to support more crown. Either way, tolerant trees might be able to maintain better terminal growth or maintain crowns when diseased; in essence, tolerant trees might try to outgrow the fungus by continually adding new tissue relative to the fungal inoculum at a rate equivalent to or faster than fungal colonization. Having both strategies in the population might benefit at the stand level because the conditions that disfavor one strategy benefit the other. Only a genetic study would be capable of confirming these hypotheses, including a detailed examination of diseased tree crowns of various sizes.

Table 5. Maximum likelihood model coefficients for parameters predicting tree crown surface area (m^2) from tree height and the sapwood area at the base of the live crown.

Variable—eq. 6	Coefficient	SE	*P*
β_0 Intercept	–12.42	5.79	0.0458
β_1 Sapwood area	0.44	0.04	<0.0001
β_2 Height	2.65	0.55	<0.0001
b_1 Location (var.)	16.73	17.08	0.1637
Residual	27.88	8.12	0.0003
γ	0.015		
AIC	712.8		
BIC	711.0		
–2 Log likelihood	706.8		

In conclusion our study suggests allometry could be an important tool for studying disease in plants because it can detect proportional shifts in allocation to various plant parts. This is particularly relevant for studying host tolerance, which is poorly understood in conifers but is thought to be related to whole plant physiological adaptations relating to storage and allocation (Stowe et al. 2000). Variation in the cost of induced resistance in infected plants might also be related in the same manner. Stem form and size, somewhat related to height and diameter, are also important in Douglas-fir because they affect value of high-quality saw logs (Kellogg 1989). Disease-caused alterations in annual ring and height growth might also affect value of wood products (Cruickshank 2010). Allometry is also used in models simulating tree growth for inventory which might better approxi-

Fig. 5. Predicted crown surface area as a function of sapwood area at the base of the live crown and tree height.

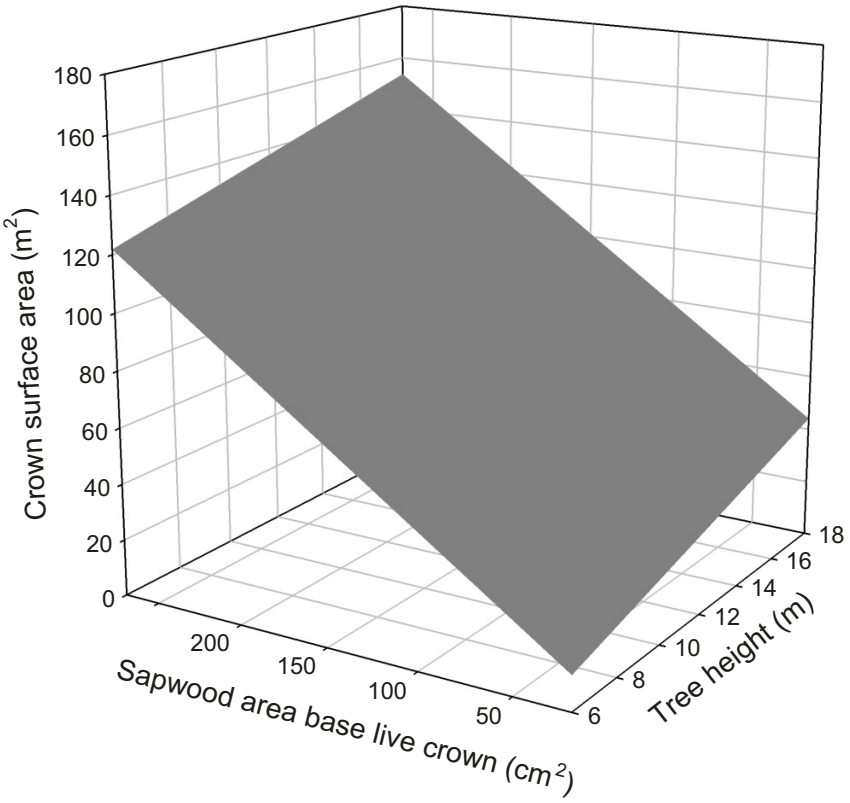


Table 6. Fit statistics for crown and sapwood models. The chosen model is first in each section.

Response variable	Explanatory varaibles	–2 Log likelihood	AIC
Crown diameter	dbh	900.1	906.1
	dbh, infection age	909.4	915.4
	dbh, BAL	896.4	902.4
	dbh, height	904.4	910.4
Height to live crown	BAL, infection age, height	8502.4	8514.4
	BAL, PINF, height	8562.2	8576.2
	BAL, infection age, height, crown length	No convergence	
Crown length	BAL, infection age, height, crown diameter	2030.7	2036.7
	BAL, PINF, height, crown diameter	2034.4	2040.4
	BAL, infection age, height, crown area	2041.0	2047.0
	BAL, infection age, height, crown diameter, dbh	2044.8	2050.8
Crown surface area	SBLC, height	706.8	712.8
	SBLC, height, BAL	707.8	713.8
	SBLC, height, infection age	703.3	709.3
	SBLC, height, PINF	709.4	715.4
	SBLC, height, dbh	706.2	712.2
Sapwood area base live crown	BAL, CSA plus interaction	791.3	797.3
	BAL, CSA, height	792.7	798.7
	BAL, CSA, infection age	797.3	803.3
	BAL, CSA, PINF	800.4	806.4
	BAL, crown length	801.2	807.2
	BAL, crown area	842.8	848.8

Note: PINF, proportion of primary roots infected; BAL, basal area larger trees; SBLC, sapwood area base live crown; CSA, crown surface area.

mate the effect of a root disease using factors that reduce annual height increment or crown expansion. Allometric relationships used in inventory to predict diameter from height would be affected by about 4% due to root disease. Future work also needs to address how disease affects annual height growth, stem taper, specific leaf area, branch to leaf allocation, root to stem and leaf allocation, and changes in wood properties.

Acknowledgements

We would like to thank Elizabeth Campbell, Kishan Sambaraju, Francesco Cortini, the Associate Editor, and two anonymous reviewers for comments on the manuscript.

References

Abrams, M.D., and Kubiske, M.E. 1990. Leaf structural characteristics of 31 hardwood and conifer tree species in central Wisconsin: influence of light regime and shade-tolerance rank. For. Ecol. Manage. **31**(4): 245–253. doi:10.1016/0378-1127(90)90072-J.

Bartelink, H.H. 1996. Allometric relationships on biomass and needle area of Douglas-fir. For. Ecol. Manage. **86**(1–3): 193–203. doi:10.1016/S0378-1127(96)03783-8.

Bloomberg, W.J. 1988. Modeling control strategies for laminated root rot on managed Douglas-fir stands. Phytopathology, **78**(4): 403–409. doi:10.1094/Phyto-78-403.

Bloomberg, W.J., and Hall, A.A. 1986. Effects of laminated root rot on relationships between stem growth and root-system size, morphology, and spatial distribution in Douglas-fir. For. Sci. **32**(1): 202–219.

Bloomberg, W.J., and Reynolds, G. 1985. Growth loss and mortality in laminated root rot infection centers in second-growth Douglas-fir on Vancouver Island. For. Sci. **31**(2): 497–508.

Braumandl, T.F., and Curran, M.D. 1992. A field guide for site identification and interpretation for the Nelson forest region. *In* Land management handbook 20. Research Branch, B.C. Ministry of Forests, Victoria, B.C.

Burns, R.M., and Honkala, B.H. 1990. Silvics of North America. Vol. 1. Conifers. Agric. Handb. 654. USDA Forest Service, Washington, D.C. pp. 527–540.

Chen, H.Y.H. 1997. Interspecific responses of planted seedlings to light availability in interior British Columbia: survival, growth, allometric patterns, and specific leaf area. Can. J. For. Res. **27**(9): 1383–1393. doi:10.1139/x97-099.

Chen, H.Y.H., Klinka, K., and Kayahara, G.J. 1996. Effects of light on growth, crown architecture, and specific leaf area for naturally established *Pinus contorta* var. *latifolia* and *Pseudotsuga menziesii* var. *glauca* saplings. Can. J. For. Res. **26**(7): 1149–1157. doi:10.1139/x26-128.

Cruickshank, M.G. 2010. Effect of Armillaria root disease on quality and value of green Douglas-fir lumber. For. Chron. **86**(2): 263–266.

Cruickshank, M.G., Lejour, D., and Morrison, D.J. 2006. Traumatic resin canals as markers of infection events in Douglas-fir roots infected with Armillaria root disease. For. Pathol. **36**(5): 372–384. doi:10.1111/j.1439-0329.2006.00469.x.

Cruickshank, M.G., Morrison, D.J., and Lalumière, A. 2009. The interaction between competition in interior Douglas-fir plantations and disease caused by *Armillaria ostoyae* in British Columbia. For. Ecol. Manage. **257**(2): 443–452. doi:10.1016/j.foreco.2008.09.013.

Cruickshank, M.G., Morrison, D.J., and Lalumière, A. 2011. Site, plot, and individual tree yield reduction of interior Douglas-fir associated with non-lethal infection by Armillaria root disease in southern British Columbia. For. Ecol. Manage. **261**(2): 297–307. doi:10.1016/j.foreco.2010.10.023.

Entry, J.A., Cromack, K., Jr, Kelsey, R.G., and Martin, N.E. 1991. Response of Douglas-fir to infection by *Armillaria ostoyae* after thinning or thinning plus fertilization. Phytopathology, **81**(6): 682–689. doi:10.1094/Phyto-81-682.

Grime, J.P. 1977. Evidence for the existence of three primary strategies in plants and its relevance to ecological and evolutionary theory. Am. Nat. **111**(982): 1169–1194. doi:10.1086/283244.

Huxley, J.S., and Teissier, G. 1936. Terminology of relative growth. Nature, **137**(3471): 780–781. doi:10.1038/137780b0.

Kellogg, R.M. 1989. Second growth Douglas-fir: its management and conversion for value. A report of the Douglas-fir Task Force. Forintek Canada Corp., Vancouver, B.C. Spec. Publ. No. SP-32.

Kozlowski, T.T. 1969. Tree physiology and forest pests. J. For. **67**(2): 118–123.

Littell, R.C., Milliken, G.A., Stroup, W.W., Wolfinger, R.D., and Schabenberger, O. 2006. SAS for mixed models. 2nd ed. SAS Institute Inc., Cary, N.C., USA.

Maguire, D.A., and Hann, D.W. 1989. The relationship between gross crown dimensions and sapwood area at crown base in Douglas-fir. Can. J. For. Res. **19**(5): 557–565. doi:10.1139/x89-088.

Marshall, J.D., and Monserud, R.A. 2003. Foliage height influences specific leaf area of three conifer species. Can. J. For. Res. **33**(1): 164–170. doi:10.1139/x02-158.

Mauricio, R., Rausher, M.D., and Burdick, D. 1997. Variation in the defence strategies of plants: are resistance and tolerance mutually exclusive? Ecology, **78**(5): 1301–1311. doi:10.1890/0012-9658(1997)078[1301:VITDSO]2.0.CO;2.

Meidinger, D., and Pojar, J. 1991. Ecosystems of British Columbia. Research Branch, B.C. Ministry of Forests, Victoria, B.C. Special Report Series No. 6.

Mitchell, K.J. 1988. SYLVER: modelling the impact of silviculture on yield, lumber value, and economic return. For. Chron. **64**: 127–131.

Morrison, D.J. 1979. Annosus root rot in Douglas-fir and Western Hemlock in British Columbia. Forestry Canada, Forest Insect and Disease Survey, Forest Pest Leaflet No. 15.

Morrison, D.J., Merler, H., and Norris, D. 1991. Detection, recognition and management of Armillaria and Phellinus root diseases in the southern interior of British Columbia. Canada – British Columbia Partnership Agreement on Forest Resource Development: FRDA II. British Columbia Ministry of Forests, Victoria, FRDA Rep. 179.

Oliver, C.D., and Larson, B.C. 1990. Forest stand dynamics. McGraw-Hill Inc., New York.

Ryan, M.G., Phillips, N., and Bond, B.J. 2006. The hydraulic limitation hypothesis revisited. Plant Cell Environ. **29**(3): 367–381. doi:10.1111/j.1365-3040.2005.01478.x. PMID:17080592.

Sadras, V.O., Quiroz, F., Echarte, L., Escande, A., and Pereyra, V.R. 2000. Effect of *Verticillium dahliae* on photosynthesis, leaf expansion and senescence of field-grown sunflower. Ann. Bot. (Lond.), **86**(5): 1007–1015. doi:10.1006/anbo.2000.1267.

Smith, J.H.G. 1964. Root spread can be estimated from crown width of Douglas-fir lodgepole pine and other British Columbia tree species. For. Chron. **40**: 456–473.

Smith, F.W., and Long, J.N. 1989. The Influence of canopy architecture on stemwood production and growth efficiency of *Pinus contorta* var. *latifolia*. J. Appl. Ecol. **26**(2): 681–691. doi:10.2307/2404092.

St Clair, J.B., Mandel, N.L., and Vance-Borland, K.W. 2005. Genecology of Douglas-fir in western Oregon and Washington. Ann. Bot. (Lond.), **96**(7): 1199–1214. doi:10.1093/aob/mci278. PMID:16246849.

Stenberg, P., Kuuluvainen, T., Kellomäki, S., Grace, J.C., Jokela, E.J., and Gholz, H.L. 1994. Crown structure, light interception and productivity of pine trees and stands. Ecol. Bull. **43**: 20–34.

Stowe, K.A., Marquis, R.J., Hochwender, C.G., and Simms, E.L. 2000. The evolutionary ecology of tolerance to consumer damage. Annu. Rev. Ecol. Syst. **31**(1): 565–595. doi:10.1146/annurev.ecolsys.31.1.565.

Thies, W.G., and Sturrock, R.N. 1995. Laminated root rot in Western North America. USDA For. Serv. PNW-GTR-349.

Van Pelt, R., and Sillett, S.C. 2008. Crown development of coastal

Pseudotsuga menziesii, including a conceptual model for tall conifers. Ecol. Monogr. **78**(2): 283–311. doi:10.1890/07-0158.1.

Weiskittel, A.R. 2003. Alterations in Douglas-fir crown structure, morphology, and dynamics imposed by the Swiss Needle Cast Disease in the Oregon Coast Range. M.Sc. thesis, Oregon State University, Oregon.

Whitney, R.D. 1976. Root rot of spruce and balsam fir in northwestern Ontario. I. Damage and implications for forest management. Great Lakes For. Res. Centre, Can. For. Serv., Dept. Env., Sault Ste. Marie, Rep. O-X-241.

Williams, H., Messier, C., and Kneeshaw, D.D. 1999. Effects of light availability and sapling size on the growth and crown morphology of understory Douglas-fir and lodgepole pine. Can. J. For. Res. **29**(2): 222–231. doi:10.1139/x98-189.

Wilson, J.S., and Oliver, C.D. 2000. Stability and density management in Douglas-fir plantations. Can. J. For. Res. **30**(6): 910–920. doi:10.1139/x00-027.

Wright, E.F., Coates, K.D., Canham, C.D., and Bartemucci, P. 1998. Species variability in growth response to light across climatic regions in northwestern British Columbia. Can. J. For. Res. **28**(6): 871–886. doi:10.1139/x98-055.

Wykoff, W.R., Crookston, N.L., and Stage, A.R. 1982. Users guide to the stand prognosis model. U.S. Dep. Agric. Forest Service, Intermountain Forests and Range Experimental Station, Ogden, Utah. Gen. Tech. Rep. INT-122.

Zimmermann, M.H. 1978. Hydraulic architecture of some diffuse-porous trees. Can. J. Bot. **56**(18): 2286–2295. doi:10.1139/b78-274.