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Ten-year growth and mortality in young Douglas-fir stands experiencing a range in Swiss needle cast severity

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34 **Abstract**

35

36 Swiss needle cast, a foliar disease caused by the Ascomycete *Phaeocryptopus gaeumannii*, continues
37 to afflict Douglas-fir in north coastal Oregon. Permanent plots were installed in 1998 to assess growth
38 impacts and monitor disease severity. Gross periodic annual increment was measured for three 2-yr
39 growth periods and one 4-yr growth period, and ranged from 0.37 to 31.74 m³/ha/yr. Foliage retention,
40 defined as the average number of annual needle age classes held by a tree, was also estimated as an index
41 of disease severity. Assuming negligible losses in stands with maximum needle retention (approximately
42 3.9 years), growth losses in net periodic annual increment reached slightly over 50% in stands with the
43 lowest needle retention (approximately 1 year). Mixed-effects regression models supported a consistent
44 relationship between foliage retention and both gross and net periodic annual increment among the four
45 growth periods. Periodic annual mortality ranged from 0 to 19.12 m³/ha/yr, but was not significantly
46 influenced by Swiss needle cast as measured by average foliage retention. Minimum and maximum
47 foliage retention have fluctuated annually from 1998 to 2008 on the permanent plots, but growth losses at
48 a given level of foliage retention appear to have remained stable. Estimated growth losses are similar to
49 those reported for comparable levels of defoliation by other agents.

50 Keywords: Foliage loss, foliar fungi, growth impact, needle longevity, stand dynamics.

Introduction

Swiss needle cast (SNC) is a foliar disease of Douglas-fir (*Pseudotsuga menziesii*) caused by the Ascomycete *Phaeocryptopus gaeumannii* (Hansen et al. 2000). This pathogen causes premature loss of older foliage, resulting in needle longevity of only one year in the most severe cases, relative to a maximum of approximately four years in unaffected plantations of similar age and geographic location (Hansen et al. 2000; Maguire et al. 2002). Annual growth losses under severe SNC have been shown to average approximately 23% of stem volume increment and reach 50% under the lowest levels of foliage retention (Maguire et al. 2002). Although the majority of growth loss under low needle retention is imposed by reduction in tree and stand foliage mass, some growth loss also accrues from inhibition of gas exchange on surviving foliage due to stomatal occlusion by fungal fruiting bodies, or pseudothecia (Manter et al. 2000).

Numerous agents cause direct loss of foliage in coniferous trees, including various classes of herbivores (Pook et al. 1998) and foliar pathogens (Kurkela et al. 2009). Defoliating insects have been shown to cause growth reductions, with the amount of growth loss dependent on the degree and duration of defoliation (Williams 1967; Alfaro and Shepherd 1991). Some insects progressively consume foliage of the youngest age classes, e.g., *Choristoneura fumiferana* on *Abies balsamea* (Piene et al. 2003), while others cause premature loss of older foliage, e.g., the aphid *Essigella californica* on *Pinus radiata* (Hopmans et al. 2008) and the aphid *Elatobium abietinum* on *Picea sitchensis* (Straw et al. 2005). Because photosynthetic efficiency declines with needle age (Ethier et al. 2006), insects like *Choristoneura fumiferana* should have a more deleterious effect on growth for a given amount of foliage loss than agents causing premature loss of older foliage. Artificial defoliation studies generally support this expectation, particularly for 1-yr-old and/or 2-yr-old versus older needles (e.g., Linzon 1958; Kulman 1965). *Dothistroma* blight on radiata pine does not seem to impact tree growth until 20-25% of the foliage is affected (Gibson et al. 1964), probably because the disease starts on the oldest and most shaded foliage, with subsequent spread to younger and more photosynthetically efficient foliage (Gibson 1972, 1974). Given its similar progression from older to younger foliage, Swiss needle cast might also be

expected to cause a relatively low growth impact at low levels of foliage loss, followed by an exponentially increasing growth impact as foliage loss proceeds to younger foliage; however, superimposed on this pattern of foliage loss is an advance wave of physiological disruption as a precursor to premature abscission (Manter et al. 2000).

In an effort to gauge the degree of growth loss from recent intensification of SNC in north coastal Oregon, a retrospective study was implemented by the Swiss Needle Cast Cooperative (SNCC; www.cof.orst.edu/coops/sncc/) at Oregon State University in 1997 (Maguire et al. 2002). In this phase, trees were destructively sampled to demonstrate that growth losses over the previous several years were proportional to current foliage retention. In the following year, a network of permanent plots was established in the same young Douglas-fir plantations to monitor tree growth and disease symptoms concurrently. Because stands that have expressed severe SNC for multiple years have experienced substantial cumulative growth loss that could potentially impose a general decline in physiological condition, it is uncertain whether the quantitative relationship between foliage retention and either growth loss or mortality may have shifted over time. Therefore, 10 years of permanent plot data were analyzed to meet the following objectives: 1) to verify the relationship between foliage retention and Douglas-fir stem volume growth estimated from the retrospective study; 2) to quantify current growth losses attributable to SNC by combining permanent plot data from all growth periods over the last 10 years; 3) to test the null hypothesis that the relationship between foliage retention and stem volume growth loss has remained stable over successive growth periods; and 4) to test the null hypothesis that Douglas-fir mortality rate is not affected by SNC severity.

Methods

The target population for the SNCC Growth Impact Study was the collection of 10- to 30-yr-old Douglas-fir plantations in north coastal Oregon. Symptoms of SNC had been intensifying throughout the early 1990s in this region and the disease was increasingly recognized as a potential impediment to growing Douglas-fir near the Pacific Coast. Concern was also growing that some of these plantations

would not survive to commercial size, or that they would grow so slowly as to be economically non-viable.

Field Work

A list of 4,504 10- to 30-yr-old Douglas-fir stands was first compiled in 1996, with geographic bounds defined by Astoria to the north (N46°11',W123°50'), Newport to the south (N44°38', W124°04'), the Pacific Coast to the west (W124°05'), and the crest of the Oregon Coast Ranges to the east (W123°30'). A set of 76 stands was randomly selected from this list and permanent plots were established in the late winter/early spring of 1998. Plots were square, 0.08 ha in area (28.45 x 28.45 m), and centered on the fifth point of an ODF (Oregon Department of Forestry) transect established in spring 1997 (retrospective plots reported by Maguire et al. (2002) were centered on the third point). On each measurement plot, all trees with diameter at breast height (dbh, nearest 0.1 cm) greater than 4 cm were tagged at a height of 1.37 m, and at least 40 Douglas-fir were measured for total height (nearest 0.01 m) and height to crown base (nearest 0.01 m) at time of plot establishment. Trees with dbh<4 cm were measured for dbh but were not tagged. On several plots with dense natural regeneration of hardwoods or other conifers, these smaller trees were measured only on a 0.02-ha circular plot in the center of the 0.08-ha plot. After two, four, six, and ten growing seasons, all trees were remeasured for dbh, and all undamaged trees from the original height subsample were remeasured for total height and height to crown base. During the 10-year study period some plots were inadvertently thinned or otherwise disturbed by management activities, so were excluded from further remeasurement. These losses resulted in 284 observations rather than the 304 that would be expected if all 76 plots had been observed for all four growth periods. Douglas-fir trees averaged 20.6 cm in dbh at the start of any given growth period, but ranged from 0.1 to 59.3 cm (Table 1). Some plots contained a significant amount of western hemlock (*Tsuga heterophylla*), as well as various broadleaved species, most commonly cascara (*Rhamnus purshiana*), red alder (*Alnus rubra*), and red elderberry (*Sambucus racemosa*) (Tables 1 and 2). Other conifers that occurred less frequently included Sitka spruce (*Picea sitchensis*), western redcedar (*Thuja*

plicata), noble fir (*Abies procera*), and grand fir (*Abies grandis*). Other hardwood species included bitter cherry (*Prunus emarginata*) and bigleaf maple (*Acer macrophyllum*).

Ten dominant or codominant Douglas-fir on each plot were also scored for SNC at time of plot establishment in 1998, and just prior to bud break in years 1999-2004. A tree was scored for SNC by dividing the crown vertically into thirds and visually estimating the average number of years (nearest 0.1 year) that foliage was retained in each crown third. Plot ratings were computed as the average of all crown thirds from all ten trees.

Statistical Analysis

Missing total heights and heights to crown base for Douglas-fir trees were estimated as a function of dbh by fitting regression models specific to each plot and growth period. Cubic stem volume of each Douglas-fir was estimated with equations previously developed for second-growth Douglas-fir (Bruce and DeMars 1974). Plot-level periodic annual increment (PAI) of Douglas-fir trees was first computed as gross increment, i.e., net change in standing live stem volume plus mortality. Douglas-fir ingrowth was zero for the vast majority of plots and trivial for the rest. The basic statistical model described gross PAI at the plot level as a function of initial Douglas-fir basal area, initial breast height age, site quality, foliage retention, and basal area of competing conifer and hardwood species:

$$[1] \quad \ln[gPAI] = \beta_{10} + \beta_{11}\ln(BA_{DF}) + \beta_{12}\ln(AGE_{BH}) + \beta_{13}\ln(SI) + \\ \beta_{14}BA_{OC} + \beta_{15}BA_{HARD} + \beta_{16}\ln(FR-0.5) + \varepsilon_1$$

where $gPAI$ = gross periodic annual stem volume increment of Douglas-fir trees ($m^3ha^{-1}yr^{-1}$)

BA_{DF} = Initial Douglas-fir basal area (m^2ha^{-1})

AGE_{BH} = Initial age at breast height (years)

SI = Bruce's (1981) site index (m at 50 years)

BA_{OC} = Initial basal area in other conifers (m^2ha^{-1})

BA_{HARD} = Initial basal area in broadleaved species (m^2ha^{-1})

FR = Foliage retention (years)

ε_1 = Random error term with $\varepsilon_1 \sim N(0, \sigma_1^2)$

β_{1k} = Parameters estimated from the data

This basic model form resulted from considering the response and predictor variables as both untransformed and log-transformed variables to address expected and demonstrated non-linear effects. A range of constants (0.1, 0.2, . . . 0.9) was also considered for translating the response along the foliage retention axis to represent better the observed growth as foliage retention approached its minimum value. Plot PAIs were measured repeatedly over time, and tree growth fluctuated with annual climatic conditions; therefore, the error structure of the model was potentially more complex than indicated by the distribution of ε_i in Model [1]. Mixed-effects models were fitted to the data under several different assumptions using PROC MIXED in SAS version 9.2 (Littell et al. 2006). These assumptions included a random plot effect, a random growth period effect, both random plot and random growth period effects, and alternative error covariance structures that recognized repeated observations within a plot. Model comparisons were based on Akaike's Information Criterion (AIC), which is a function of the maximum likelihood and number of parameters from the fitted model (Burnham and Anderson 2002). Normality and homoscedasticity of residuals were verified for the final models by standard graphical analysis. This same model was fitted to data representing net periodic annual increment (nPAI) and similar comparisons were made among alternative random effects and error structures. Variables were considered significant at $\alpha=0.05$, but those with p-values between 0.05 and 0.10 were identified as marginally significant. Final models were required to have only significant variables.

To meet the first objective of verifying growth losses estimated from the retrospective phase of the growth impact study, plot-level data from 1997 and an indicator variable were added to model [1] for both gross and net increment. Interactions between the retrospective indicator and predictors were included, and the model was reduced by backward elimination of non-significant variables ($\alpha=0.05$).

SNC generally does not seem to cause direct mortality of Douglas-fir trees, but this general observation had not been rigorously tested in the subject population. To perform this test with the permanent plot data, mortality was assessed in two phases. In the first phase, occurrence of Douglas-fir mortality on a given plot during any given growth period was regarded as a binary response, with the

“event” defined as mortality in at least one year within the period. This analysis of mortality differed from more conventional analysis of individual-tree mortality because plot-level mortality could have occurred repeatedly in all years of a given growth period, or in only one of the years. The following binomial regression model (McCullagh and Nelder 1989) was fitted to the data from the Growth Impact Study to test whether the occurrence of Douglas-fir mortality was influenced by intensity of SNC:

$$[2] \eta = \ln[\mu/(1-\mu)] = \beta_{20} + \beta_{21}f(BA_{DF}) + \beta_{22} f(BA_{OC}) + \beta_{23}f(BA_{HARD}) + \beta_{24} f(AGE_{BH}) + \beta_{25}f(SI) + \beta_{26} f(FR) + \beta_{27} f(GP)$$

where μ = probability of observing Douglas-fir mortality on the plot during the growth period

GP = Growth period length (2 or 4 years)

f = Identity function (untransformed variable) or natural logarithm

β_{2k} = Parameters estimated from the data

and BA_{DF} , AGE_{BH} , BA_{OC} , BA_{HARD} , SI , and FR are defined above.

Growth period length was included to accommodate the two- and four-year growth periods and to address the lack of further information about the number of years that the plot experienced mortality within the growth period. To address covariance among repeated observations within a plot, random plot and growth period effects and alternative error structures were explored using PROC GLIMMIX in SAS version 9.2. All forms of predictor variables (untransformed and logarithmically-transformed) were initially included in a full model which was reduced by backward elimination of non-significant variables ($\alpha=0.05$). Alternative model formulations were compared by AIC.

In the second phase of assessing SNC effects on mortality, cubic volume of Douglas-fir mortality was regarded as conditional on occurrence of mortality in a given plot (i.e., only those plots experiencing mortality were included). The statistical model took the following form:

$$[3] \ln[PAM] = \beta_{30} + \beta_{31}\ln(BA_{DF}) + \beta_{32}\ln(AGE_{BH}) + \beta_{33}\ln(SI) + \beta_{34}BA_{OC} + \beta_{35}BA_{HARD} + \beta_{36}\ln(FR-0.5) + \epsilon_3$$

where PAM = Conditional periodic annual mortality of Douglas-fir trees ($m^3ha^{-1}yr^{-1}$)

ε_3 = Random error term with $\varepsilon_3 \sim N(0, \sigma_3^2)$

β_{3i} = Parameters estimated from the data

and BA_{DF} , AGE_{BH} , BA_{OC} , BA_{HARD} , SI , and FR are defined above.

As with gross and net PAI, the model was fitted with various combinations of random plot and growth period effects and specific covariance structures to account for repeated observations within a plot. All models were fitted with PROC MIXED in SAS version 9.2 (Littell et al. 2006), alternative models were compared by AIC, and normality and homoscedasticity of residuals were verified by standard graphical analysis.

Results

Trends in gross PAI over four growth periods showed strong plot effects attributable primarily to differences in inherent site quality and secondarily to differences in growing stock (Fig. 1). Periodic variation in gross PAI for a given plot reflects a combination of age effects, annual weather fluctuations, episodic mortality, and some residual measurement error. Over the range of plantation ages sampled in 1998 (10- to 30-yr-old), PAI generally depicted the expected trend of increasing growth potential with increasing age and the accompanying increase in site occupancy (combination of leaf area index and cambial surface area). Gross PAI ranged from 0.37 to 31.74 m³/ha/yr (Table 2).

The importance of accounting for covariance among repeated observations of individual plots was underscored by the significant improvement (reduction) in AIC achieved by all models that assumed any error structure beyond independence of observations (Table 3). Likewise, the correctly specified error structures demonstrated how some variables that were incorrectly identified as significant under the assumption of independence were in fact not significant when autocorrelation was accounted for. Random plot effects did improve the fit of the models, presumably by addressing, at least in part, covariance among repeated observations within a plot; however, direct estimation of the variance-covariance matrix was more effective. In the selected model, the variance-covariance matrix was block diagonal, consistent with independence of individual plots and non-zero covariance of repeated observations within plots. In one simple form, the non-zero diagonal blocks are a Toeplitz or diagonal-

constant matrix that assumes equal variance among plots and years, and equal covariance among observations separated by the same time interval. However, the variation applied in the final PAI model was a heterogeneous Toeplitz covariance structure (Littell et al. 2006), which assumed constant variance among plots for a given growth period but unequal variance among growth periods (diagonals $\sigma_1^2 - \sigma_4^2$). Covariances among repeated observations within a plot were constrained to be the product of the two growth period standard deviations and a constant ρ (off-diagonals $\rho\sigma_1\sigma_2, \dots, \rho\sigma_3\sigma_4$). The effect of site index was non-significant under this error structure ($p=0.49$), but would have been incorrectly inferred as very significant under the assumption of independent observations ($p<0.0001$). All other variables were strongly significant after dropping site index (all $p<0.007$), resulting in the following final model for gross PAI of Douglas-fir (standard errors for parameter estimates in parentheses):

$$\begin{aligned}
 [4] \quad \ln[\text{gPAI}] = & 1.6597 + 0.9245 \cdot \ln(\text{BA}_{\text{DF}}) - 0.6727 \cdot \ln(\text{AGE}_{\text{BH}}) \\
 & (0.1360) \quad (0.03672) \quad (0.06985) \\
 & - 0.00600 \cdot \text{BA}_{\text{OC}} - 0.01928 \cdot \text{BA}_{\text{HARD}} + 0.3589 \cdot \ln(\text{FR}-0.5) \\
 & (0.002202) \quad (0.007093) \quad (0.05052)
 \end{aligned}$$

Random growth period effects added very little to any of the alternative models. The average loss in gross PAI implied by the final model was 18.7%, with a maximum loss of 49.1%. The average losses in gross PAI implied by the same model fitted separately to each growth period ranged from 17.4% to 25.6%, with maxima ranging from 36.6% to 58.2% (Table 4). Pairwise tests among foliage retention parameter estimates for specific growth periods failed to reject the null hypothesis that the relationship between foliage retention and gross increment remained consistent over the 10-yr study period.

Similar results were obtained for the model describing net PAI (Table 4). The lowest AIC was associated with the model assuming a heterogeneous Toeplitz covariance structure. As was the case for gross PAI, site index was not significant under this error structure ($p=0.76$), but would have been incorrectly identified as very significant under the assumption of independent observations ($p=0.0002$). All other variables were significant after dropping site index (all $p \leq 0.021$), resulting in the following final model for net PAI of Douglas-fir (standard errors for parameter estimates in parentheses):

$$\begin{aligned}
[5] \quad \ln[nPAI] = & 1.7551 + 0.9420 \cdot \ln(BA_{DF}) - 0.7403 \cdot \ln(AGE_{BH}) \\
& (0.1545) \quad (0.04257) \quad (0.08125) \\
& - 0.00621 \cdot BA_{OC} - 0.02633 \cdot BA_{HARD} + 0.3859 \cdot \ln(FR-0.5) \\
& (0.002673) \quad (0.008611) \quad (0.06153)
\end{aligned}$$

Random growth period effects again provided no significant improvement in any of the alternative models. The average loss in net PAI implied by this model was 20.0%, with a maximum loss of 51.6%. The average losses in net PAI implied by the same model fitted separately to each growth period ranged from 18.3% to 20.3%, with maxima ranging from 36.2% to 58.6% (Table 4). As with gross increment, pairwise tests among foliage retention parameter estimates for specific growth periods failed to reject the null hypothesis that the relationship between foliage retention and net increment remained consistent over the 10-yr study period. Curves depicting loss in net PAI for individual growth periods yielded a set of very similar curves that fell within the confidence envelope for model [5], assuming average levels of Douglas-fir basal area, broadleaved basal area, other conifer basal area, and breast height age (Fig. 3). The 95% confidence intervals for the parameter estimates associated with FR in equations [4] and [5] (0.36 ± 0.11 and 0.30 ± 0.12 , respectively) did not include 1.0, indicating that the relationship between periodic annual increment and foliage retention was significantly nonlinear.

Re-analysis of models [4] and [5] with the retrospective indicator variable and its interaction with other covariates failed to detect any differences in the relationship between plot-level increment and foliage retention in the retrospective and permanent plot phases of the growth impact study.

The selected binomial model for predicting probability of Douglas-fir mortality incorporated a simple Toeplitz covariance structure (Table 5). As described above, this structure assumed equal variance among plots and growth periods; however, covariances among repeated observations within plots were assumed to be non-zero and equal for any set of growth periods separated by the same time interval (diagonal-constant). The greater flexibility provided by the heterogeneous Toeplitz structure offered little gain in the binomial mortality model (Table 5). Foliage retention had no significant effect on the

probability that a plot would experience Douglas-fir mortality during any given growth period ($p=0.94$), leading to the following final model (standard errors of parameter estimates shown in parentheses):

$$\begin{aligned}
 [6] \quad \ln[p/(1-p)] = & -3.0091 + 0.5882 \cdot \ln(\text{BA}_{\text{DF}}) + 0.2069 \cdot \ln(\text{BA}_{\text{OC}}) \\
 & (0.8776) \quad (0.2896) \qquad (0.08831) \\
 & + 0.6168 \cdot \ln(\text{BA}_{\text{HARD}}) + 0.6231 \cdot \text{GP} \\
 & (0.1305) \qquad (0.1588)
 \end{aligned}$$

where p is the predicted probability of observing Douglas-fir mortality within the growth period, and all other variables are defined above. As would be expected, the probability of Douglas-fir mortality increased with increasing basal area in all three stand components, i.e., Douglas-fir, other conifers, and broadleaved species. The influence of broadleaved basal area was particularly strong, with a very rapid increase in the probability of mortality with even low amounts of hardwood basal area (Fig. 4). Not surprisingly, the probability of mortality was higher for the 4-yr growth period.

As was the case for the binomial mortality model, the simple Toeplitz covariance structure was sufficient for accounting for repeated observations in the conditional model for volume of Douglas-fir mortality. Mortality volume was not influenced by foliage retention under this structure ($p=0.49$). The following final model for Douglas-fir mortality retained only initial Douglas-fir basal area, and the competitive effects depicted by basal area of broadleaved species and western hemlock (standard errors of parameter estimates shown in parentheses):

$$\begin{aligned}
 [7] \quad \ln[\text{PAM}] = & -5.2712 + 1.3323 \cdot \ln(\text{BA}_{\text{DF}}) + 0.1332 \cdot \text{BA}_{\text{HARD}} + 0.04507 \cdot \text{BA}_{\text{WH}} \\
 & (0.6252) \quad (0.1928) \qquad (0.03208) \qquad (0.01649)
 \end{aligned}$$

Periodic annual Douglas-fir mortality increased as initial basal area of Douglas-fir, broadleaved species, and western hemlock increased. As with probability of mortality, foliage retention was not a significant predictor of conditional Douglas-fir mortality. For a given amount of initial basal area, broadleaved species had a relatively strong effect on mortality (Fig. 5), probably because bitter cherry, bigleaf maple and red alder are all capable of overtopping Douglas fir due to more rapid juvenile height growth.

Discussion

Periodic annual stem volume increment

In the retrospective phase of the growth impact study, stem volume increment of Douglas-fir during the 1996 growing season was estimated by destructively sampling 0.02-ha fixed area plots in the spring of 1997 (Maguire et al. 2002). The 1996 annual increment was regressed on standard plot-level covariates backdated to their initial conditions, with the exception that foliage retention was available only for the spring of 1997 (time of destructive sampling). In contrast, volume increment was regressed on initial foliage retention for the growth periods in the permanent plot analysis. Likewise, mortality was unknown for the 1-yr retrospective growth period, so the 1996 annual increment was limited to survivor growth. Despite these unavoidable inconsistencies between the retrospective and permanent plot phases of the growth impact study and, hence, the possibility of differences in the estimated relationship between foliage retention and volume increment, the retrospective estimates of growth losses for a given level of foliage retention did not differ significantly from those of the permanent plot phase. Likewise, the fundamental relationship between foliage retention and growth loss remained consistent over successive growth periods comprising the 10-yr permanent plot phase of the growth impact study. In short, all evidence failed to reject the null hypothesis of stability in the relationship between foliage retention and growth loss. Although by convention this result does not prove the null hypothesis, no gradual decline in growth vigor at a given level of foliage retention could be detected in these permanent plot data.

The increase in growth loss with declining foliage retention exhibited some curvature, but lacked evidence of any obvious threshold of foliage retention at which growth loss suddenly appeared or worsened. Instead, the model described a relatively steady decline in growth with decreasing foliage retention, a trend that would be expected with significant physiological disruption of gas exchange in younger foliage, in advance of premature abscission (Manter et al. 2000).

To our knowledge no other links between foliage retention and growth loss have been quantified in other regions of the world where Douglas-fir is grown. Douglas-fir in New Zealand experienced a growth decline attributed to *Phaeocryptopus gaeumannii* starting in 1963, and comparison of pre-1963 to

post-1963 growth suggested a growth loss of 26-40%; however, foliage retention was not reported (Manley 1985). Cameron et al. (1978) observed that infected trees in Hanmer Forest in New Zealand generally retained 20-30% less foliage than uninfected trees, with 7-yr-old infected trees at Rotorua holding 95%, 43%, 8%, and 0% of 1-, 2-, 3-, and 4-yr-old needles, respectively. In a more comprehensive analysis using 312 permanent sample plots on both North and South Islands, Kimberley et al. (2011) compared growth rates for the years before SNC detection (prior to 1959) to growth for the years following detection. Average volume growth loss was estimated to be 32%, but was greater on the North Island (35%) than on the South Island (23%). These estimates were similar to the average loss of 27% derived from the relationship between foliage retention and growth loss in the retrospective phase of the north coastal Oregon study (Maguire et al. 2002), but were higher than the average loss of 20% in net increment reported above for the permanent plot phase.

Douglas-fir mortality

Data from the permanent plots supported the hypothesized lack of any SNC effect on Douglas-fir mortality. Formal analysis of permanent plots in New Zealand similarly failed to detect any acceleration of mortality after the appearance of *Phaeocryptopus gaemmannii* in 1959 (Kimberley et al. 2011). As is consistent with many previous analyses and models of mortality at both the individual-tree level (e.g., Hamilton 1986) and stand level (e.g., Zhao et al. 2007), the probability of Douglas-fir mortality on the SNC permanent plots increased with stand density. In the case of Douglas-fir plantations in north coastal Oregon, increasing basal area of hardwood and other conifer species that regenerated naturally in these stands further contributed to increasing probability of mortality.

As with probability of mortality, the conditional amount of Douglas-fir mortality was driven by density of the major stand components, with the exception that other coniferous species contributed little beyond the effects of western hemlock alone. This particular aspect of stand dynamics can be best understood by considering the vertical structure of Douglas-fir plantations. Western hemlock comprised the bulk of basal area in other conifers, and the average height of this species (Table 1) supported field observations that it typically maintained a position in the main canopy. With the exception of noble fir on

only very few plots, the additional basal area in other conifers was contributed by western redcedar and Sitka spruce, both of which were much shorter on average than Douglas-fir and therefore occupied the suppressed crown class (Table 1). While these latter two species influenced the probability of Douglas-fir mortality, they did not provide sufficient competition to larger Douglas-fir to influence significantly the volume of mortality experienced by plots.

Other assessments of conditional mortality have targeted number of trees as the response variable rather than stem volume (Affleck 2006; Zhao et al. 2007); however, in all cases initial stand density was the primary driver of conditional mortality. A two-stage approach has been previously applied to stand-level mortality in loblolly pine plantations (Affleck 2006; Zhao et al. 2007). Likewise, zero-inflated Poisson, zero-inflated negative binomial, and especially hurdle models have been shown to offer a single-stage alternative for accommodating the common mixture of many plots without mortality and relatively few plots with mortality (Affleck 2006). Regardless, all approaches lead to similar inferences about the predominant effect of stand density on mortality.

Assumptions required for estimating SNC growth impact

Estimating the growth impact from Swiss needle cast requires a reference point that can be considered the potential or expected growth in absence of the disease. Four general approaches can be taken to quantify expected growth rate. The first assumes that foliage retention is controlled exclusively by *Phaeocryptopus gaeumannii* (this assumption is addressed in next section), that sampling has spanned the full range of Swiss needle cast severity, and that disease severity acts independently of other factors that may influence growth (e.g. annual and seasonal weather). Under these conditions, plots exhibiting the highest foliage retention are interpreted as disease-free. After accounting for the effects of other covariates influencing growth, the marginal effect of foliage retention in the regression models allows estimation of growth loss relative to maximum possible foliage retention. As described above, SNC growth impacts on gross and net periodic stem volume increment of Douglas-fir were estimated in this way on the SNCC growth impact plots.

A second approach is possible if the exact date of an introduction or start of an epidemic is known. In this case the growth of permanent plots prior to the known date can be compared to subsequent growth, assuming correction for other covariates influencing growth. This approach was possible in New Zealand due to the extensive network of Douglas-fir plots that was established well before the introduction of Swiss needle cast in 1959 (Manley 1985; Kimberley et al. 2011). A similar approach involved application of dendrochronological techniques to Douglas-fir and adjacent western hemlock in north coastal Oregon; in this case, the start of the Swiss needle cast epidemic was identified as the date when the radial increment of Douglas-fir diverged from the increment of adjacent western hemlock (Black et al. 2010).

A third approach relies on predictions from established models to provide the expected growth rate. Departures from expectation can then be related formally or informally to foliage retention. Swiss needle cast growth impacts at the stand level have been compared to predictions from the ORGANON growth model (Weiskittel and Maguire 2004), and impacts on individual-tree diameter and height growth have been quantified relative to the regional trends represented in ORGANON (Weiskittel and Maguire 2004; Garber et al. 2007). Similar approaches have been applied to estimate other growth losses, for example, from the western spruce budworm (Crookston 1985).

The fourth approach to estimating growth impacts requires elimination of the pathogen by fungicide in a controlled experiment (e.g., Hocking 1967; Stone et al. 2007). Growth of newly planted radiata pine in Chile protected from *Dothistroma septospora* for two years exceeded unprotected trees by 20%, implying a 17% growth loss in the first two years after planting. Relative to the most effective fungicide treatments, height growth of unprotected radiata pine seedlings was reduced 31% by *Dothistroma* blight (Hocking 1967). Aerial application of chlorothalonil to Douglas-fir stands with severe Swiss needle cast increased stem volume growth 35% over unsprayed controls during the full five-year period of application, but the increase was 60% over the final three years alone (Mainwaring et al. 2002).

Factors influencing foliage retention

A fundamental assumption behind assessment of SNC growth impact is that foliage retention or needle longevity is controlled exclusively by the presence, abundance, and/or activity of *Phaeocryptopus gaeumannii*. However, many other factors are well known to influence needle life-span between and within species (Reich et al. 1995). Patterns observed between species can be quite complex and relate to many factors like crown and needle morphology, shade tolerance, and temperature regime, and other aspects of the physical environment to which they are adapted. These interspecific patterns in leaf life-span represent genetic adaptations that ensure survival and reproductive success, so are of limited interest from the perspective of intensive silviculture, except perhaps for implications in managing forests under various climate change scenarios. Of greater interest in the present context are plastic or phenotypic responses to aspects of the growing environment that are under some degree of silvicultural control.

In general, foliage retention declines along a gradient from lower to higher net primary production, whether imposed by inherent site quality (Weidman 1939; Oleksyn et al. 2003) or fertilization and irrigation (Gower et al. 1992). Although this trend may seem counter-intuitive, it is important to recognize that lower foliage retention and shorter life-span do not necessarily imply less foliage biomass. In fact, the trend imposed by greater productivity can largely be attributed to greater foliage production, more rapid turnover, and either stable or increasing total amounts of foliage biomass. Foliated shoot length among different provenances of *Pinus ponderosa* was stable despite variation in needle retention from three years in some provenances to eight years in others (Weidman 1939). This difference was attributed to phenotypic plasticity, given that needle retention was consistently 3-4 years for all provenances when grown in a common garden. A similar study confirmed that the amount of foliage per shoot remained constant in *Pinus contorta* growing at different elevations, despite variation in needle retention from 5 to 18 years (Schoettle 1990). Results from fertilization studies are consistent with observations along productivity gradients. Based on needle litterfall rates, foliage retention was inferred to increase immediately after fertilizing *Pseudotsuga menziesii* in the southern Rocky Mountains (Gower et al. 1992), but to decrease in the long term (Gower et al. 1992). In a more detailed study of crown architecture, foliated branch length remained constant among fertilized and unfertilized *Abies grandis* and

Pseudotsuga menziesii in the northern Rocky Mountains, even though foliage longevity declined (Balster and Marshall 2000).

If foliated branch length for a given level of light availability is constant, then the pattern observed among trees with differing growth rates described above should extend to within-tree differences in branch growth. Light measurements and detailed analysis of crown architecture indicate that needle longevity is controlled by the number of annual shoots required to accumulate sufficient foliage to reduce light below the compensation point (Schoettle and Smith 1991). An increase in foliage longevity with increasing depth into the crown has in fact been broadly observed, and can be attributed to improved light conditions and greater shoot growth with increasing height in the crown (Schoettle and Smith 1991).

Biological interpretation of foliage retention

In light of the generally observed inverse relation between foliage retention and forest productivity, the positive correlation between stem volume increment of Douglas-fir and foliage retention in north central Oregon suggested that Swiss needle cast was the predominant factor controlling needle retention in this region. This conclusion is further supported by the increase in foliage retention from 1.9 to 2.8 years after fungicidal elimination of *Phaeocryptopus gaeumannii* (Mainwaring et al. 2002), suggesting that the limiting factor on foliage retention was not maximum foliated length.

Although the mechanisms controlling foliage retention are complex, the fact that foliage retention is correlated with total foliage amount (at least in the presence of other covariates), was indicated by the significant effect of foliage retention in statistical models of periodic annual increment. Douglas-fir trees displaying maximum needle longevity in our target population received foliage retention ratings of slightly over 3.5 years. On a tree with this foliage retention, 31%, 28%, 19%, 11%, and 11% of the total foliage mass was contributed by 1-, 2-, 3-, 4-, and ≥ 5 -yr-old needles, respectively (Weiskittel and Maguire 2006). For a tree of equal diameter, height, and crown length, a foliage retention of 1.5 yrs implied a shift in the distribution to 40%, 34%, 18%, 7%, and 1% across the same age classes; however, total foliage mass was approximately 27% less (Weiskittel and Maguire 2006). Although foliage

retention must be used with caution as a predictor of tree- or stand-level foliage mass for the reasons outlined in the previous section, foliage retention does provide a rating that can be operationally implemented in stands ≤ 40 -50 years of age, and it has consistently served as a reliable index of growth loss in Douglas-fir plantations when accompanied by appropriate covariates (Maguire et al. 2002).

Growth losses from foliar fungi

Growth impacts of defoliation by Swiss needle cast were comparable to growth impacts estimated at similar levels of defoliation by agents such as other foliar fungi and canopy insects, as well as under simulated defoliation in designed experiments. However, major differences between effects of Swiss needle cast and other agents include severity of defoliation, relative losses among age classes, duration or periodicity of defoliation, thresholds for mortality, and concomitant effects on other tree, stand, and ecosystem components. Assessing growth impacts of Swiss needle cast was relatively simple compared to other defoliators in that defoliation was never complete or episodic; rather, it fluctuated annually but was relatively constant over the decade of observation. Likewise, SNC has not yet caused significant mortality in the sampled plantations of young Douglas-fir.

Dothistroma blight is a foliar disease that has caused significant defoliation in several coniferous species and can be sufficiently severe to cause mortality (Gibson 1972). The first signs of *Dothistroma* appear in the oldest and most shaded foliage, with subsequent spread to younger foliage in more exposed parts of the crown (Gibson 1972). *Dothistroma* was first recognized as a serious disease of commercial conifers after *Pinus radiata* was introduced into eastern and central Africa. Much of the early work on *Dothistroma* growth impact was based on comparison of height and diameter growth among trees with differing levels of defoliation (Gibson 1974). Because the older and shaded foliage was attacked first, 20-30% of the foliage on *Pinus radiata* could be affected before impacts on diameter and height growth were detected (Gibson et al. 1964; Gibson 1972, 1974). Due to this pattern of disease progression, growth in older radiata pine was observed to follow a reverse sigmoid pattern as *Dothistroma* severity increased (Christensen and Gibson 1964); i.e., little detectable influence on diameter growth until 25% defoliation, 50% growth reduction at 50% defoliation, and 90% reduction at 75% defoliation. The onset of height

growth loss was much later, reaching 50% reduction at 80% defoliation. Earlier observations in Kenyan radiata pine plantations had suggested that height growth losses from *Dothistroma* reached only 20% when 75% of the tree's foliage became affected (Gibson et al. 1964). In younger trees with relatively little older foliage, the relationship between defoliation and height growth was more linear (Gibson et al. 1964). Severe levels of infection have led to tree mortality in the past, but mortality did not appear to be as dominant a factor in stand-level growth loss, relative to the more widespread effects of partial defoliation (Gibson 1974).

Lophodermium needle cast has been observed to start at the base of *Pinus sylvestris* crowns and move upward (Martinsson 1979). This progression was attributed to dispersal of spores from fruiting bodies that develop during a saprophytic phase on needles that have been cast during a parasitic phase on live needles, combined with the influence of a more humid microclimate lower in the crown. After the trees reach a minimum height, perhaps as low as two meters, the tree is less susceptible, particularly at the top. In a study of genetic resistance, trees were subjected to as many as three attacks by the fungus, but height growth losses were not detected until defoliation reached 65% (Martinsson 1979). Growth losses then increased to 24% at 80% defoliation and 50% at 96% defoliation.

Growth impacts from insect defoliation

Insects have perhaps received the greatest amount of attention as defoliators from a research perspective (e.g., Kulman 1971). MacLean (1985) provided a very comprehensive review of growth impact from the spruce budworm, including both eastern and western species. Budworm development is synchronous with bud flush, resulting in early larval instars feeding on new foliage emerging from the bud. Magnitudes of growth loss are a function of both the severity of budworm feeding in specific years and the temporal sequence of defoliation. Growth losses accrue from reduction in diameter and height growth, top kill, stem deformities in severely defoliated trees resulting from top kill, and mortality of entire trees (MacLean 1985). The net effect of these impacts were yield losses ranging from 10-81% and growth losses ranging from 19-54%, with much of the growth loss occurring after defoliation had ended (Batzer 1973). Mortality rate depends on severity and duration of defoliation, species and age range of

trees, age class distribution, species composition, spacing, site, and others (MacLean 1985). Mortality starts usually after 4 or 5 years of severe defoliation (MacLean 1980).

Douglas-fir tussock moth (*Orgyia pseudotsugata*) defoliates several conifer species, particularly *Pseudotsuga menziesii* and *Abies grandis*, by partial consumption and induced shedding of individual needles (Brookes et al. 1978). The ratio of damaged foliage to consumed foliage averages 3.2 but can run as high as 8. Outbreaks generally last for four years and damage progresses from the top of the tree downward due to larval behavior and the preference for newer foliage. Severe defoliation is typically characterized by complete loss of foliage in the upper quarter to half of the crown and damage of most current foliage in the rest of the crown. Stand-level defoliation has typically been rated as very light, light, moderate, or heavy, corresponding approximately to 10, 25, 50, and 90% average defoliation. Radial growth reductions of Douglas-fir suffering 50% defoliation or greater were estimated to average 58% in the Blue Mountains of Oregon.

Growth impacts from a variety of other insect defoliators have also been estimated. Pine sawfly defoliation of *Pinus taeda* evoked a compensatory response in growth with defoliation rates up to 30%, but 50% defoliation over a 3-yr period resulted in a cumulative loss of diameter increment of approximately 28% (Zeide and Thompson 2005). The pine butterfly, *Neophasia menapia*, feeds preferentially on older foliage of *Pinus ponderosa*, but moves to newer foliage during epidemics. During a three-year epidemic, radial growth losses averaged about 60% on surviving trees that experienced defoliation levels averaging about 75% (Cole 1966). A pandora moth (*Coloradia pandora*) outbreak in central Oregon *Pinus ponderosa* resulted in average defoliation of 45% in 1992 and a second wave in 1994 resulted in average defoliation of 67% (Cochran, 1998). These defoliation events caused a basal area growth loss of 25% in the first season after defoliation, 30% in the second season after defoliation, and 63% after the second wave of defoliation in 1994. Defoliation by the larch casebearer on two sites over a 10-yr period started at light (1-25%) to moderate (26-50%) defoliation, reached severe (76-100%) defoliation, and tapered off to moderate and light defoliation before ending (Alfaro et al. 1991). Diameter growth losses averaged approximately 29%.

Growth impacts from artificial defoliation

Most artificial defoliation experiments were designed to remove differing amounts of total foliage or specific age classes of needles. The latter experiments are more relevant to Swiss needle cast growth impact, but their interpretation is complicated by differences in season of foliage removal and unknown contributions of each age class to total foliage biomass. Linzon (1958) concluded that one-year old foliage was more important to *Pinus strobus* growth than either current or 2-yr-old foliage, but defoliation was implemented in May, well before formation of significant amounts of current foliage (Linzon 1958). In contrast, Kulman (1965) removed foliage from *Pinus resinosa* trees in mid-July and found that removal of current needles had the largest effect on shoot growth and removal of 2-yr-old needles the least effect; however, 61.7%, 18.9%, 15.6%, and 3.8% of the foliage on subject trees was current, 1-yr-old, 2-yr-old, and 3-yr-old foliage, respectively. In another experiment on *Pinus resinosa*, 0%, 33%, 66% and 99% of the foliage was removed from each age class by clipping corresponding distal portions of needles (Krause and Raffa 1996). Growth reductions from 33% removal were proportionally lower than from 66% foliage removal, a response attributed to lower nutrient concentrations in the tips of needles. Results from these and other artificial defoliation experiments suggest that relative contribution to diameter and shoot growth depend on at least three key features of the specific needle age classes: 1) its relative contribution to total foliage amount; 2) its nutrient relative concentration; and 3) its photosynthetic efficiency.

Conclusions

Growth loss from Swiss Needle Cast averaged approximately 50% after 75% reduction in foliage retention and approximately 25% after 50% reduction in foliage retention. Declines in foliage retention were not necessarily proportional to loss of foliage mass because foliage retention represented a complex succession of mechanisms influencing photosynthetic capacity through corresponding effects on stomatal blockage by pseudothecia, changes in light penetration and capture, translocation of foliar nutrients, and shifts in foliage distribution among age classes of differing inherent photosynthetic efficiency. Estimated growth losses from Swiss needle cast were comparable to losses from other fungal and insect defoliators

that caused similar magnitudes of foliage loss, and as well as to growth losses documented in various artificial defoliation experiments.

Acknowledgements

This project was funded by the Swiss Needle Cast Cooperative at Oregon State University. Contributors of logistical and financial support since 1998 have included Starker Forests, Stimson Lumber, Green Diamond Resource Company, USDI Bureau of Land Management, Hampton Resources, Oregon Department of Forestry, Weyerhaeuser Company, The Campbell Group, USDA Forest Service, Forest Capital Partners, Plum Creek Timber, Confederated Tribes of the Grande Ronde, Miami Corporation, Longview Fibre, Coos County Forestry Department, Confederated Tribes of the Grand Ronde, Confederated Tribes of the Siletz, Davidson Industries, Port Blakely Tree Farms, Rayonier, Rosboro Lumber, Roseburg Forest Products, and Swanson Superior Forest Products.

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Table 1. Initial tree attributes for all growth periods from the Growth Impact Study established in early 1998 by the Swiss Needle Cast Cooperative.

Species	Attribute	n	Units	Minimum	Mean (sd)	Maximum
Douglas-fir	dbh	17,638	cm	0.1	20.6 (8.9)	59.3
“	height	12,844	m	1.63	16.0 (5.4)	38.6
“	crown ratio	12,807		0.01	0.69 (0.19)	1.00
Western hemlock	dbh	5,993	cm	0.1	11.8 (9.8)	105.4
“	height	1,585	m	1.48	14.41 (5.36)	32.5
“	crown ratio	1,542		0.03	0.79 (0.15)	1.00
Sitka spruce	dbh	1,304	cm	0.1	7.8 (5.8)	46.1
Western redcedar	dbh	136	cm	0.1	6.7 (6.0)	23.4
Noble fir	dbh	202	cm	1.9	23.6 (13.6)	58.8
Grand fir	dbh	21	cm	1.4	5.0 (4.4)	13.3
Cascara	dbh	2,732	cm	0.1	4.6 (3.1)	27.4
Red alder	dbh	2,108	cm	0.1	12.4 (7.5)	43.4
Red elderberry	dbh	1,528	cm	0.1	3.5 (3.0)	17.0
Bitter cherry	dbh	448	cm	0.2	8.9 (5.4)	24.6
Bigleaf maple	dbh	103	cm	1.4	14.5 (7.5)	37.6

Table 2. Initial plot attributes and growth rates for all growth periods from the Growth Impact Study established in early 1998 by the Swiss Needle Cast Cooperative (n=284).

Variable	Units	Minimum	Mean (sd)	Maximum
Douglas-fir tree density	trees ha ⁻¹	86	602 (274)	1692
Douglas-fir basal area	m ² ha ⁻¹	0.62	21.91 (9.44)	48.35
Douglas-fir SDI*	trees ha ⁻¹	26	449 (171)	838
Douglas-fir stem volume	m ³ ha ⁻¹	0.93	150.51 (87.71)	402.18
Douglas-fir breast height age	yrs	5.9	17.5 (5.4)	34.9
Douglas-fir top height	m	4.89	20.83 (5.98)	41.93
Douglas-fir site index	m at 50 yrs	13.4	41.4 (6.6)	64.6
Basal area of other conifers	m ² ha ⁻¹	0.00	4.28 (7.32)	46.98
Basal area of broadleaved species	m ² ha ⁻¹	0.00	1.68 (2.67)	17.29
Total tree density	trees ha ⁻¹	247	1380 (933)	5705
Total plot basal area	m ² ha ⁻¹	1.71	28.01 (10.85)	65.37
Total SDI*	trees ha ⁻¹	84	633 (221)	1447
Foliage retention	years	1.01	2.38 (0.45)	3.85
Total basal area growth	m ² ha ⁻¹ yr ⁻¹	0.03	4.40 (2.05)	12.61
Douglas-fir basal area growth	m ² ha ⁻¹ yr ⁻¹	0.15	3.29 (1.80)	10.40
Douglas-fir top height growth	m yr ⁻¹	0.19	0.90 (0.24)	1.58
Douglas-fir gross volume growth	m ³ ha ⁻¹ yr ⁻¹	0.37	15.98 (6.64)	31.74
Douglas-fir net volume growth	m ³ ha ⁻¹ yr ⁻¹	0.37	15.51 (6.70)	31.68
Douglas-fir volume mortality	m ³ ha ⁻¹ yr ⁻¹	0.00	1.34 (2.68)	19.12

*SDI =stand density index = (trees ha⁻¹).(Dq/25.4)^{1.605}, where Dq=quadratic mean dbh

Table 3. Models tested for predicting gross periodic annual increment (gPAI; $m^3ha^{-1}yr^{-1}$) and net periodic annual increment (nPAI; $m^3ha^{-1}yr^{-1}$) of 76 permanent plots installed as part of the SNCC Growth Impact Study. All variables are significant ($p \leq 0.05$) except for underlined variables in bold ($p > 0.10$). (lbadf= $\ln(BA_{DF})$, lsib= $\ln(SI)$, lage= $\ln(AGE_{BH})$, baoc= BA_{OC} , bahard= BA_{HARD} , lret= $\ln(FR)$).

Response	Symbols of included variables	Error Structure	AIC
ln(gPAI)	lbadf, baoc, bahard, lage, lsib, lretm	independent	-85.8
"	lbadf, baoc, bahard, lage, lsib , lretm	random plot (compound symm)	-164.3
"	lbadf, baoc, bahard, lage, lretm	random plot (compound symm)	-164.0
"	lbadf, baoc, bahard, lage, lretm	unstructured	-173.6
"	lbadf, baoc, bahard, lage, lretm	compound symm hetero	-173.1
"	lbadf, baoc, bahard, lage, lretm	Toeplitz	-167.1
"	lbadf, baoc, bahard, lage, lretm	Toeplitz hetero	-175.9
"	lbadf, baoc, bahard, lage, lretm	AR(1)	-146.4
"	lbadf, baoc, bahard, lage, lretm	SP(POW)	-145.0
ln(nPAI)	lbadf, baoc, bahard, lage, lsib, lretm	independent	2.1
"	lbadf, baoc, bahard, lage, lsib , lretm	random plot	-77.7
"	lbadf, baoc, bahard, lage, lretm	random plot	-78.7
"	lbadf, baoc, bahard, lage, lretm	unstructured	-85.6
"	lbadf, baoc, bahard, lage, lretm	compound symm hetero	-88.8
"	lbadf, baoc, bahard, lage, lretm	Toeplitz	-79.9
"	lbadf, baoc, bahard, lage, lretm	Toeplitz hetero	-88.0
"	lbadf, baoc, bahard, lage, lretm	AR(1)	-67.0
"	lbadf, baoc, bahard, lage, lretm	SP(POW)	-65.2

Table 4. Average and minimum foliage retention and average and maximum loss in gross and net periodic annual increment of Douglas-fir (estimated from models [4] and [5] fitted separately to each growth period).

Attribute		Growth period			
		1998-1999	2000-2001	2002-2003	2004-2007
Foliage retention	Average	2.32	2.34	2.51	2.41
	Minimum	1.07	1.01	1.62	1.47
Gross increment	Average growth loss	18.1%	17.8%	25.6%	17.4%
	Maximum growth loss	58.2%	53.9%	46.8%	36.6%
Net increment	Average growth loss	18.3%	18.4%	19.0%	20.3%
	Maximum growth loss	58.6%	55.2%	36.2%	41.9%

Table 5. Models tested for predicting probability of Douglas-fir mortality and conditional periodic annual mortality (PAM; $m^3ha^{-1}yr^{-1}$) of 76 permanent plots installed as part of the SNCC Growth Impact Study. Bold italicized variables are marginally significant ($0.05 < p \leq 0.10$), bold underlined variables are not significant ($p > 0.10$), and all other variables are significant ($p \leq 0.05$). (lbadf= $\ln(BA_{DF})$, lsib= $\ln(SI)$, lage= $\ln(AGE_{BH})$, baoc= BA_{OC} , bahard= BA_{HARD} , lret= $\ln(FR)$)

Response	Symbols of included variables	Error Structure	AIC
$\ln[p/(1-p)]$	lbadf, lbaoc, lbahard, lsib, per, <u>lretm</u>	random plot	1343
"	lbadf, lbaoc, lbahard, <i>lsib</i> , per	random plot	1339
"	lbadf, lbaoc, lbahard, per	random plot	1328
"	<i>lbadf</i> , lbaoc, lbahard, per	unstructured	1313
"	lbadf, lbaoc, lbahard, per	comp symm	1295
"	lbadf, lbaoc, lbahard, per	comp symm hetero	1289
"	lbadf, lbaoc, lbahard, per	Toeplitz	1288
"	lbadf, lbaoc, lbahard, per	Toeplitz hetero	1306
"	lbadf, lbaoc, lbahard, per	AR(1)	1309
"	lbadf, lbaoc, lbahard, per	SP(POW)	1310
$\ln(PAM)$	lbadf, bahard, bawh, <u>lsib</u> , <u>ret</u>	independent	434.9
"	lbadf, bahard, bawh, <u>lsib</u> , <u>ret</u>	random plot	432.5
"	lbadf, bahard, bawh, <u>ret</u>	random plot	433.7
"	lbadf, bahard, bawh	random plot	435.1
"	lbadf, bahard, bawh	unstructured	424.8
"	lbadf, bahard, bawh	comp symm hetero	434.3
"	lbadf, bahard, bawh	Toeplitz	422.3
"	lbadf, bahard, bawh	Toeplitz hetero	422.7
"	lbadf, bahard, bawh	AR(1)	427.1
"	lbadf, bahard, bawh	SP(POW)	427.7

- Figure 1.** Observed plot-level trends in gross periodic annual increment of Douglas-fir over plantation age at the start of each growth period.
- Figure 2.** Observed plot-level trends in periodic annual mortality of Douglas-fir over plantation age at the start of each growth period.
- Figure 3.** Estimated net growth loss (model [5]) for Douglas-fir cubic stem volume by initial foliage retention (index of SNC severity). Dark solid line represents the comprehensive model fitted to pooled data from three 2-yr periods and one 4-yr growth period on permanent plots in the Growth Impact Study. Dotted lines represent the 95% confidence envelope for the prediction model, and the light solid lines represent model [5] fitted separately to each of the four growth periods in this study and to the retrospective study.
- Figure 4.** Estimated effects (model [6]) of initial hardwood basal area and initial basal area in other conifers on probability of Douglas-fir mortality during the growth period (two-year growth period is assumed with initial Douglas fir basal area set at average level of $21.9 \text{ m}^2\text{ha}^{-1}$).
- Figure 5.** Estimated effects (model [7]) of initial Douglas-fir basal area and initial hardwood basal area on conditional periodic annual Douglas-fir mortality (initial western hemlock basal area set at average level of $3.5 \text{ m}^2\text{ha}^{-1}$).

Fig. 1

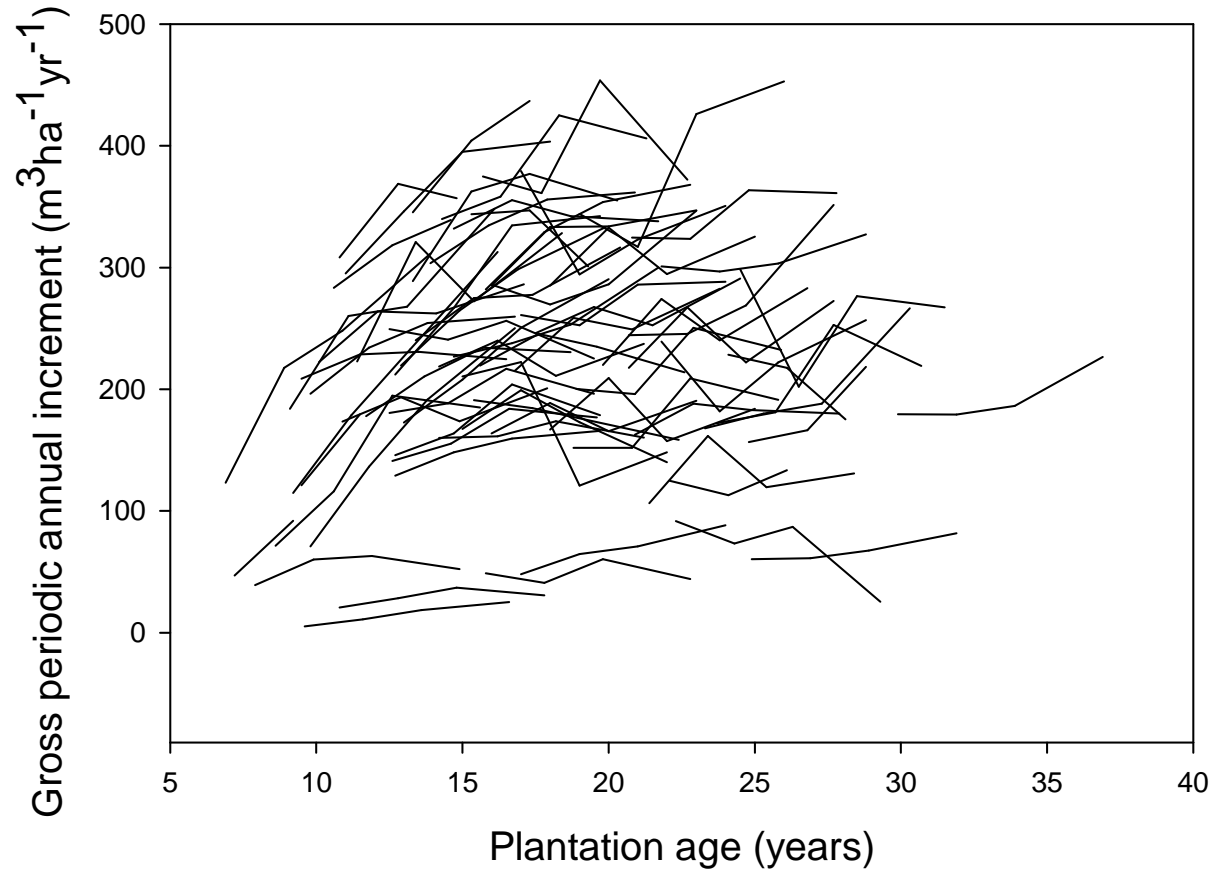


Fig. 2

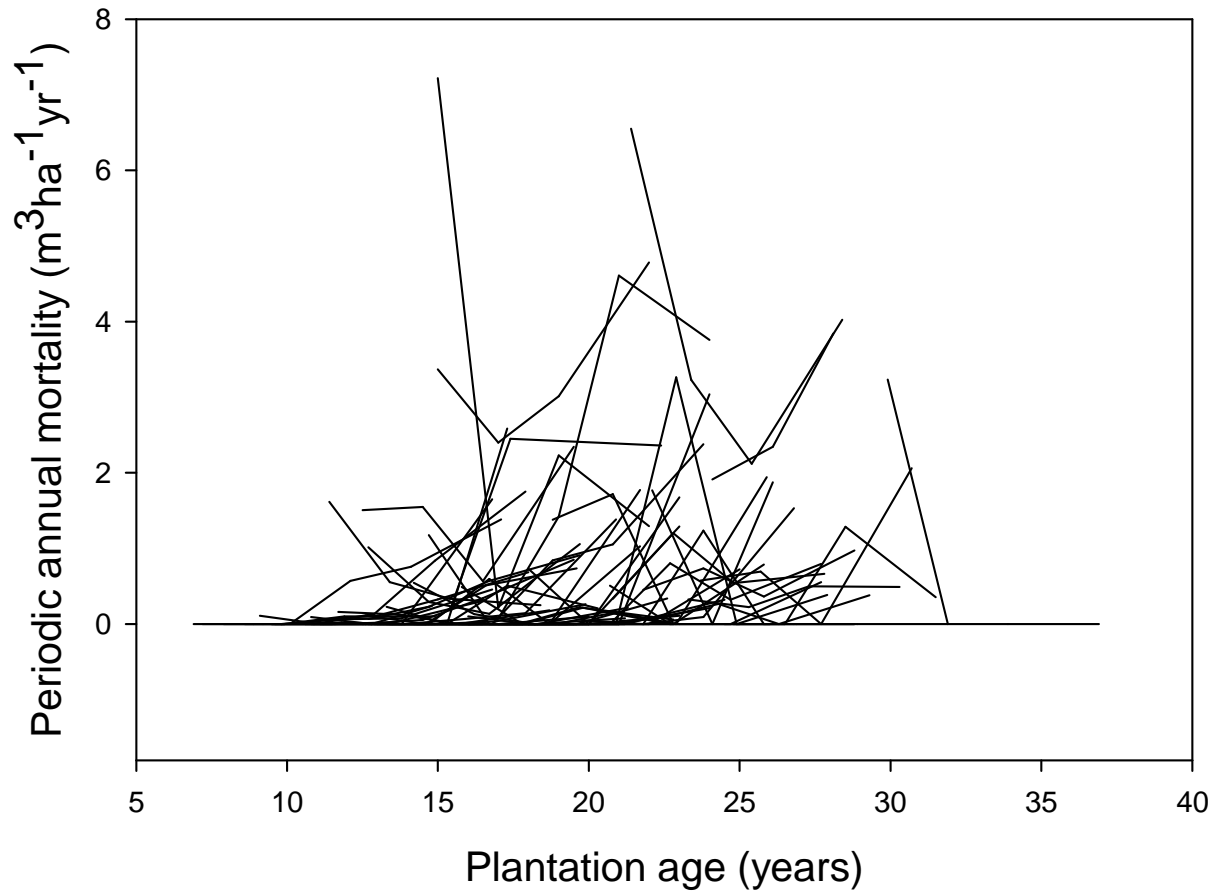


Fig. 3

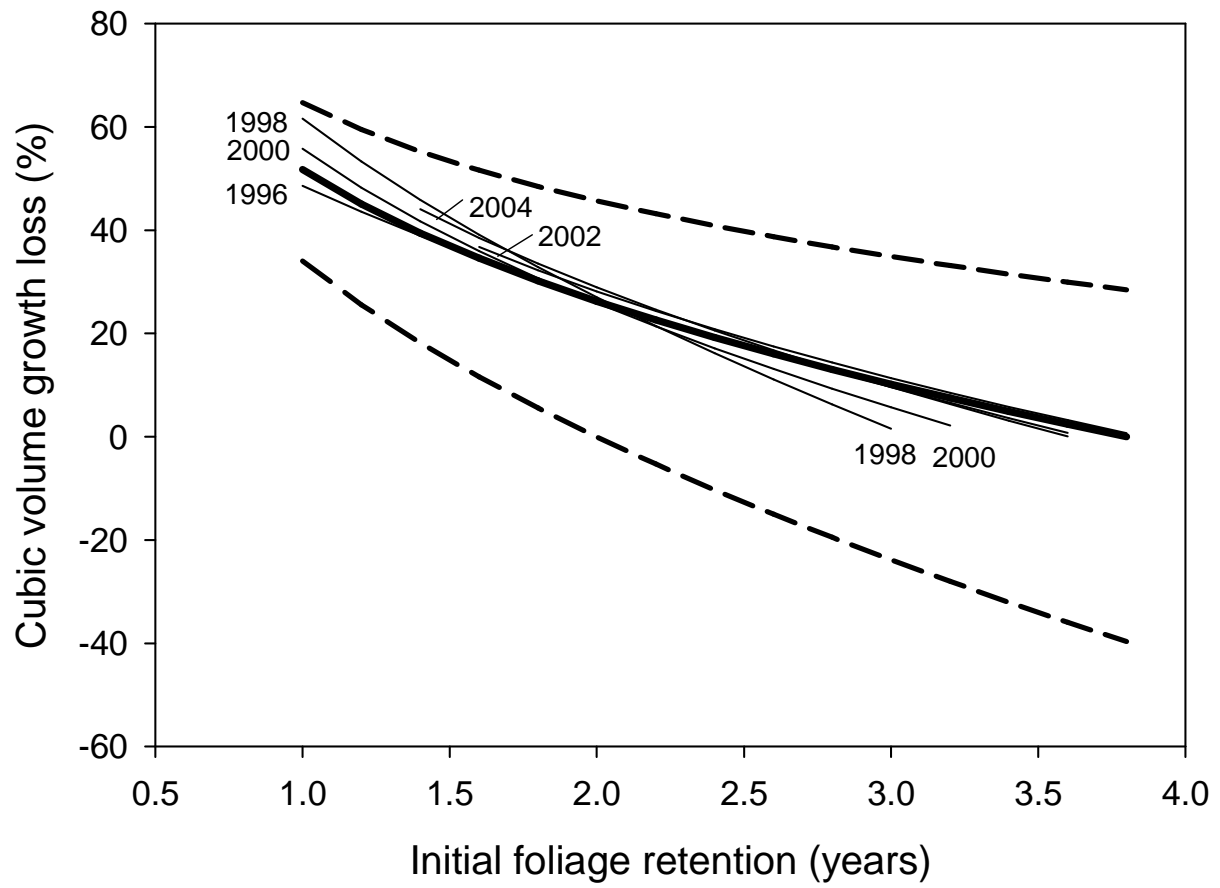


Fig. 4

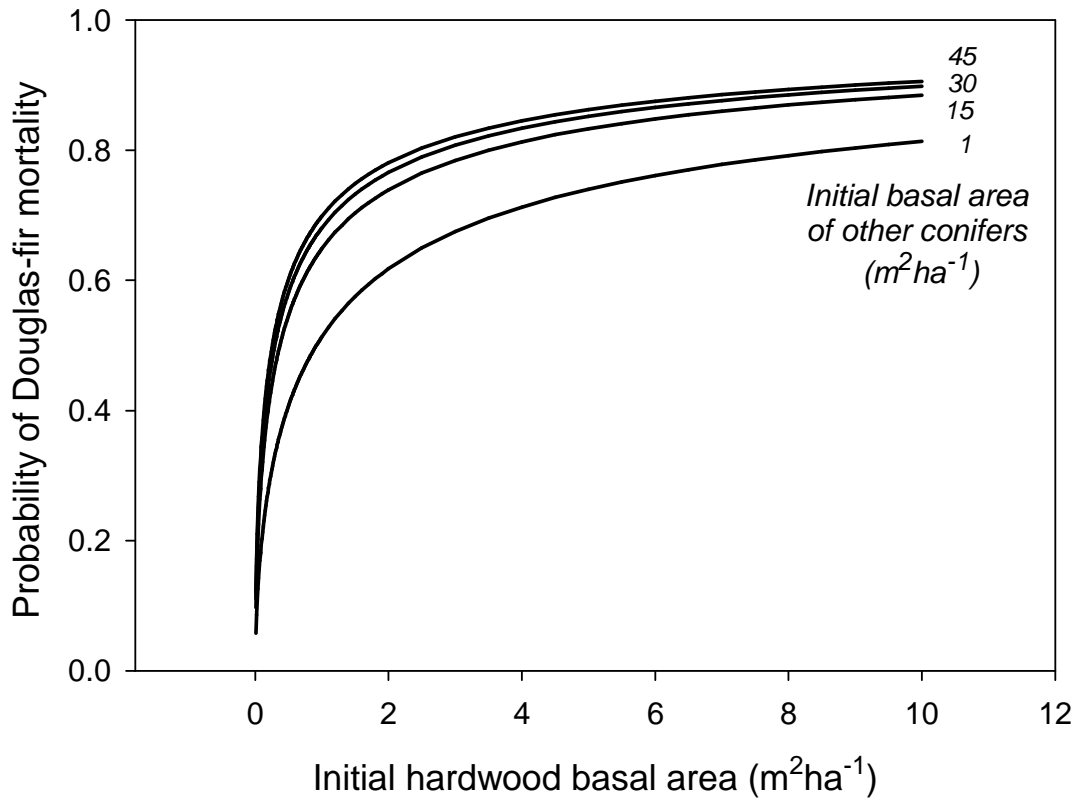


Fig. 5

