Survival and yield of Douglas-fir in the Cedar-Hemlock ecosystem of the southern interior of British Columbia.

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Abstract

Extended tree lifespans require investments against decay, herbivory, wind and fire. Tree root diseases are global pathogens impacting growth and survival over a large range of tree species. The commercially important conifer Douglas-fir highlights conditions affecting its survival and growth in nine planted and four natural study sites in British Columbia. Planted Douglas-fir ranged from 20-40 years, and natural stands from 63-110 years. Average survival for all tree species in planted stands was 86% after 30 years, with 80% mortality from *Armillaria ostoyae.* Average survival of Douglas-fir in natural stands was 60% at age 90 with 86% mortality from *A. ostoyae*. Mortality began in planted stands about tree ages 6-10, and in most natural stands about tree ages 55-65. Stand age, site index, proportion of Douglas-fir, precipitation, and temperature, but not stand density, accounted for most of the hazard. Percent dead trees were correlated with percent plot basal area losses at 1 to 1 ratio in both stand types. Larger planted Douglas-fir trees had greater hazard sooner up to age 30 than smaller trees. Rapid early tree growth likely caused extensive root contact with fungal stump inoculum. Older and larger trees were affected by interacting climate limitations and disease status.

1) Introduction

An organism's life history is the sequence of events related to survival and reproduction throughout life until death representing a complex process changing in time and space (Franklin et al. 1987). Organism size (height, diameter etc.), growth rate, productivity, biotic and abiotic stress tolerance (e.g. pathogen or drought, respectively) through time are common life history traits. Mortality is an important forest trait because of the long-term implications it has on stand composition, structure, and productivity or sustainability (Harcombe 1987). Mortality may represent a stand limiting factor as individuals removed from a population may not be replaced quickly for long-lived species such as trees.

One of the most common tree life history traits studied is size related mortality within and among species populations. These include high mortality with smaller size (Luo and Chen 2011, Yang et al 2003, van Mantgem et al. 2009), higher morality in both size extremes (Chen et al. 2008, Groom et al. 2012, Temesgen and Mitchell 2005), or less commonly form a mortality peak at mid-range sizes at certain time periods (Dietz and Moorcroft 2011). Mortality can also be greater in areas with greater net primary productivity (Stephenson and van Mantgem 2005). Explanation for these patterns cover a range of hypotheses that include that the trees succumb through: competitive size interference (Yang et al 2003); drought and or high temperature limitations (Allen et al. 2010, van Mantgem and Stephenson 2007, van Mantgem et al. 2009); increasing

respiration costs per leaf area in larger trees (Oliver and Larson 1990, Yang et al 2003). Other factors may interact through a combination of competition, lower defense, or greater biotic agent activity (Lorimer et al. 2001, Stephenson and van Mantgem 2005). Biotic agents can be especially damaging when combined with abiotic host stressors that exceed a physiological threshold level for some time period especially repeatedly (Schoeneweiss 1981, Woodall et al 2005). Most of these factors may ultimately link mortality hazard with factors affecting photosynthesis and or respiration in some fashion (Temesgen and Mitchell 2005, Waring 1987).

Some factors affecting growth of interior Douglas-fir growth are low precipitation and high temperatures (Chen et al. 2010). These in turn are affected by elevation where moisture is limiting in lower areas and temperature limiting at high elevations (Lo et al 2010). Temperature may cause tradeoff between growth and cold hardiness requirements (Darychuk et al. 2012); however, precipitation can also limit growth at Douglas-fir's northern range more than cold temperatures (Griesbauer and Green 2010). High growth rate and earlier bud burst in interior Douglas-fir allows growth before high temperature and low moisture become limiting (St. Clair et al. 2005). Temperatures up to 35C do not appear to limit photosynthesis directly (Sorensen and Ferrell 1973), but potential leaf area expansion may be more growth limiting (Brix 1967).

Pathogens are the only biotic agents that attack all forest successional stages of Douglas-fir (Franklin et al. 1987). Armillaria root disease has been implicated as one of the most common diseases interacting with tree stress (Wargo 1996). The disease is caused by a multi-species fungus with large host and global geographic range in natural forest (Kile et al. 1991) and planted stands including agricultural crops (Hood et al. 1991). Mortality occurs when the fungus spreads to and girdles the cambium of the tree root collar, which may require multiple infections events over many years (Morrison 2011). Factors predisposing trees to infection or mortality to root disease include low light, high and low temperature, high and low moisture, nutrient imbalance, and pollution (Wargo and Harrington 1991). However, many of these factors were identified through observational studies only.

Summer drought has been implicated with mortality due to Armillaria root disease in British Columbia (BC) (Buckland 1953) and may act as a predisposing stress (Desprez-Loustau et al 2006). Drought and competition were predisposing factors for Abies species to Heterobasidion root disease, and drought may be more important than temperature (Linares et al. 2010). Conclusions from BC forest health surveys concerning root diseases state that it is difficult to assign site risk to root diseases (Hodge et al 1994). Since then, ecosystem management and some silvicultural practices can be used to assign risk between larger areas (Morrison et al 2000, 2001), but prediction of risk within an ecosystem is still poor. Geographic location is important in predicting damage by Armillaria root disease, being more common in southern BC and north western United States, and the disease appears to be more severely damaging with increasing continentality without soil moisture extremes (Cruickshank et al 1997, Williams and Marsden 1982, Wargo and Shaw 1985). Differences in disease expression with

geographic location are probably not due to a difference in fungal virulence (Morrison and Pellow 2002).

The study objectives were to sample live and dead trees in 20-110 year-old stands, to associate the time of death and with stand and climate factors, and relate them to Douglas-fir life history and their interaction with biotic factors. Mixed model survival analysis and linear regression accounted for trees that were correlated in time and in space within sample location hierarchies.

2) Methods

2.1 Stand measures

2.1.1 Ecosystem description

The biogeoclimatic ecosystem classification system is based on soils, climate, and indicator plants as described by Braumandl and Curran (1992) and zones are named after the dominant climax plant species. Study sites were located in the Interior Cedar Hemlock (ICH) zone. The ICH is isolated from the maritime influence by a large range of coastal mountains that maintain a strong continental climate with moist summers, cold wet winters, and a snow pack that reduces some of the summer moisture deficits. Tables 1 and 2 give stand description information for the study sites. Common trees in this ecosystem are balsam fir [*Abies lasciocarpa* (Hook.) Nutt.]; Douglas-fir [*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco]; lodgepole pine (pine) [*Pinus contorta* Dougl. *Et* Loud. Var. *latifolia* Engelm. *Ex* S. Wats.]; western larch [*Larix occidentalis* Nutt.]; western redcedar [*Thuja plicata* Donn *ex* D. Don]; paper birch [*Betula papyrifera* Marsh]; and trembling aspen [*Populus tremuloides* Michx.]. These are referred to as balsam, Douglas-fir, pine, larch, redcedar, birch, and aspen hereafter.

2.1.2 Planted stands

2.1.2.1 Retrospective sampling

Site selection was limited to access roads that would accept a lowbed trailer carrying a 20-ton excavator and to excavator travel on site. Six sites were previously clearcut (CC, EB, HL, KF, MM, and NB) and then planted with interior Douglas-fir, the other site (KX) was planted with Douglas-fir after a wildfire. Although records were not kept on planting density, 1200-1600 stems/ha was a common target for these areas. For each site, 10-m radius plots (0.03 ha) were randomly distributed throughout the site within 100 m of either side of the main access road, except where excavator travel would not permit and the plots had to be moved locally. A total of 22-25 10-m radius plots were established on the sites. For all stands, the latitude, longitude, and elevation were recorded for the center of each site.

All trees in every plot were then pulled out of the soil in the late fall using a 20-ton Link Belt excavator with a clamshell bucket attachment to minimize the breakage of roots, stems and branches. Pulled trees were gently laid on the ground and left in the plot to overwinter. After the soil thawed the following spring, the soil was removed from the roots of all trees. Lesions caused by *A. ostoyae* were identified on all roots and confirmed by observing mycelial fans in the bark or cambium (of the lesions) or as mycelial fan impressions in or on the bark of older lesions. Trees were classified as dead, healthy or infected and the cause of the infection or death was noted.

Diameter breast height (1.3 m) and tree height were taken for all trees (dead and alive). A stem cross-section was taken at 1.3 m on trees at sites CC, KF, and KX to test for size effects on hazard. Stem disks were also taken from the base of all the dead trees. Stem disks were air dried, sanded, and the area bounded by the rings at age 15 was determined for all trees on a digitizing tablet. Dead trees were dated through ring counting at the basal disk (soil line).

2.1.2.2 Permanent sample plots

Plots to monitor the spatial and temporal development of Armillaria root disease were established in Douglas-fir plantations near Sugar Lake (SL) and Nakusp (NK) in the ICH. The Sugar Lake site was planted in 1967 following logging of the 350-year-old cedar-hemlock stand. The site was slash burned prior to planting of Douglas-fir. The Nakusp site was planted in 1972 after wildfire burned the 80-year-old Douglas-fir stand. In 1984 at Sugar Lake and 1986 at Nakusp, a 200 m x 100 m (2 ha) plot was established, where stocking and species composition were uniform; the plot was divided into eight 50 m x 50 m blocks. Each block was carefully searched for dead trees and mycelial fans confirmed death by the fungus. These plots were surveyed every two years and the year of death and cause was noted.

2.1.3 Natural stands

Douglas-fir leading stands that had no signs of previous cutting in the study area were located. Two 10 x 100 m survey strips across slope were established and all dead and living trees were recorded. The survey strips were located by placing a 100 m base line nearest the access road across slope. A perpendicular line was established to this into the site at one end of the base line. Random starting points were picked along this perpendicular line to established the starting points for the for the two survey lines which ran parallel to the base line. The survey lines were established with a separation of at least 50 m but not more than 200 m. The cause of death was determined for all dead trees. All trees within the lines were classified into species, alive, dead, infected, or other causes. Older trees that had died from *A. ostoyae* could be determined from impressions of mycelial fans in the bark which remain intact for many years.

Diameter at 1.3 m was taken for all trees and a basal disk (soil line) section of the dead Douglas-fir stems were taken. Disks that were in poor condition were wrapped in cellophane tape first before cutting in order that they remain intact for transport. Disks

were air dried and then sanded. Disks that would not accept sanding were immersed in hot paraffin for several minutes and then left to harden. The paraffin strengthened the wood matrix so it could be cut on a band saw. Two disk radii were located according to Chapman's method (Chapman and Meyer 1949). A sharp scalpel prepared the rings which were measured on a dendrochronometer along each radius (digital positiometer manufactured by L. Kutschenreiter, Austria) whose precision was ±0.01 mm.

A living tree ring chronology was built from one 5 mm core on 5-10 dominant and co-dominant Douglas-fir trees in each survey line. The rings for dead trees were cross dated using living trees with the program COFECHA. The year of death could be determined against the living chronology. Chronologies for each tree were also visually inspected by plotting the ring widths to confirm the COFECHA results. The Douglas-fir trees examined in these sites were fast growing species on highly productive sites. Rings were clearly identifiable on most samples, except for some rotted disks, and missing rings in most of the chronology were rare. Some rings at the very outer edge may have been missed as reduction in increment or growth on one side of the tree occurs a few years before death and where advanced decay also occurred. Ring counts along both disk radii generally agreed, but the largest ring count was taken as the year of death on the same disk where they did not agree.

2.1.4 Site index

For planted stands, the five tallest Douglas-fir trees per 10 m radius plot were used to calculate site index at 50 years (BC Ministry of Forests, Lands, and Natural Resource Operations 2015). Site index for natural stands was based on a 10 m radius plot centered on each survey line for Douglas-fir trees in which all of the Douglas-fir trees were measured for age, height and diameter at 1.3 m. Site index (SI) was estimated using Site Tools program (BC Ministry of Forests, Lands, and Natural Resource Operations 2015). The SI indices for each line or plot were averaged within stands.

2.2 Climate data

Annual climate data for a geographical location (latitude, longitude and elevation) was determined using the ClimateBC program. For climate descriptions considered see Table 3, or Wang et al. (2012).

2.3 Statistical analyses

2.3.1 Survival

Hazard h is a conditional failure rate defined as the probability that a tree dies during the interval t to t +1 given that it has survived till time t. Time is counted forward yearly for each site location. The survival function with respect to time is obtained from the hazard function: S(t) = probability of being alive at the end of time t = [1-h(1)] x [1-h(2)] x …..x [1-h(t)]. The survival curves based on this function are displayed in the figures. The hazard is derived directly from the model coefficients b for each term where hazard (h) = exp(b). Odds ratios reported are the ratios of the odds of an event occurring in one

group relative to another group, or the odds of an event occurring over a unit increase of a continuous variable, while holding other model variables constant. An odds ratio of greater than one means that hazard was greater in that group compared to the reference group, or indicates the change in odds with one unit increase in that predictor variable. Odds ratios less than one means lower hazard in that group compared to the reference group, or lower hazard with one unit increase of a continuous variable.

The hazard statistical models took the form of:

$$h(t) = \exp(\beta_0 + \beta_1 \text{time} + \beta_2 \text{time}^2 + \beta_3 \text{climate}) \quad [1]$$
$$h(t) = \exp(\beta_0 + \beta_1 \text{time} + \beta_2 \text{time}^2 + \beta_3 \text{stand}) \quad [2]$$

where: $h(t)$ is the hazard, a binary response (dead=1 alive=0) modelling dead; β_0 is the overall mean, β_1 and β_2 describe the fixed effect parameters associated with time and time^2 (quadratic time), β_3 describe the fixed effect time related covariate for climate. Eq. 2 is the same except that stand variables (e.g. site index, density etc.) are inserted for climate, and stand variables have constant values over time measured at the sampling date.

All climate and stand variables used in Eq. 1and 2 had significant effect on the models. A random effect for site and sample plot location was added to the best 10 of these models, and the effect on site variance was noted. These random terms account for the hierarchical sample structure. Stand and climate variables are site related and should describe the site conditions similar to a random site location variable since they compete for similar variation; hence, the variance component for site location should be reduced with the addition of the stand and climate fixed effects. The final model was built using combinations of these variables that were not highly correlated, that had significant coefficient values (i.e. $p<0.05$ that $\beta <> 0$), and that reduced the random site location variance. A random term for the effect of time with site location was tested in the final model in Eq. 3 and 4, but was dropped because it was not significant.

For the juvenile sites the final model took form:

$$h(t)_{ijk} = \exp(\beta_0 + \beta_1 \text{time}_i + \beta_2 \text{time}^2_i + \beta_3 \text{DDL0}_{ij} + \beta_4 \text{DDL0}_{ij*} \text{time}_i + \beta_5 \text{DDL0}_{ij*} \text{time}^2_i + \beta_6 \text{MAR}_{ij} + \beta_7 + \text{MAR}_{ij*} \text{time}_i + \beta_7 + \text{MAR}_{ij*} \text{time}_i^2 + a_1 \text{site}_j + b_1 \text{plot}_{k(j)}) \quad [3]$$

where $h(t)$is the hazard at time i in site j and plot k for fixed effect conditions β_1 to β_7 and random effects a_1 and b_1; β_0 is the overall mean; β_1 and β_2 describe quadratic time; β_3 describe the effect of the degree days less than zero for time i and site j, and β_4 and β_5 describe its interaction with quadratic time i; β_6 describe the effect of mean annual radiation at time i and site j, and β_7 describe it interaction with time i; a_1 is the intercept for site location j; b_1 is the intercept for plot k within site location j.

Final model for natural stands was:

$$h(t)_{ijk} = \exp(\beta_0 + \beta_1 \text{time}_i + \beta_2 \text{time}^2_i + \beta_3 \text{SHM}_{ij} + \beta_4 \text{SHM}_{ij*}\text{time}_i + \beta_4 \text{PFIR}_{jk} + \beta_5 \text{PFIR}_{jk*}\text{time}_i + \beta_6 \text{SI}_j + a_1 \text{site}_j + b_1 \text{line}_{k(j)}) \quad [4]$$

which is the same for Eq. 3 except that β_3 is the effect of summer heat moisture on site *j*, and β_5 is its interaction with time *i*; β_4 is the proportion of Douglas-fir in transect line k for site *j*, and β_5 is its interaction with time; β_6 is the effect of site index for site *j*; a_1 is the random intercept for site *j*; b_1 is the random intercept for transect line *k* on site *j*.

To test for the effect of tree diameter inside bark at age 15 on future mortality of juvenile Douglas-fir in site locations CC, KF, and KX where data was available for all trees in the stands, the model form was:

$$h(t)_{ijk} = \exp(\beta_0 + \beta_1 \text{time}_i + \beta_2 \text{time}_i^2 + \beta_3 \text{dbh15}_{l(jk)} + \beta_4 \text{dbh15}^2_{l(jk)} + \beta_5 \text{time}_i * \text{dbh15}_{l(jk)} + a_1 \text{site}_j + b_1 \text{plot}_{k(j)}) \quad [5]$$

where all variables are as described in Eq. 3 except dbh15 is the diameter inside bark at age 15 for tree *l* nested in site *j* and plot *k*.

Kaplan-Meir (KM) survival estimates were generated using a non-parametric program (SAS proc LIFETEST) similar to a life table and not the result of a model fit. KM estimates were also used to assess model fits by comparing them against the results obtained by the fitted hazards for the mixed model (SAS proc GLIMMIX) shown in the figures. The GLIMMIX procedure fits random effects using best linear unbiased predictors (BLUPs) that modify the hazard coefficients for each hierarchy, in this case site location and sample plots within sites, and these were used to determine differences between site and plot locations.

2.3.2 Productivity-Basal area for juvenile and natural stands

Basal area models took the form of:

$$Y_{ijk} = \beta_0 + \beta_1 \text{NTREES}_{ij} + \beta 2 \text{PDEAD}_{ji} + a_1 \text{site}_i + b_1 \text{plot}_{j(i))} + \varepsilon_{ij} \quad [6]$$

where *Yij* is the plot basal area (m^2/ha) of all living tree species in plot *j* on site *i* given the fixed effects β_1 and β_2 and random effects a_1 and b_1 ; NTREES is the number of living plus dead trees of all species; PDEAD is the percent of all dead trees; b_1 is the intercept for plot location *j* within site *i*; ε_{ij} is the residual error. For natural stands, transect line is divided into four equal parts within each line forming 5 x 25 m plots. Random site intercept was also not significant for either natural or planted stands but was left in the model. There was no significant interaction between fixed effect terms, and none of the fixed terms could also be included as random effects within at the site or plot hierarchies. A second order polynomial term was added for PDEAD to check for nonlinearity but this did not significantly improve the model in either stand type. Analyses were completed using the SAS proc MIXED procedure.

Results

3) Hazard and survival

3.1 Planted stands
3.1.1 Mortality hazard and cause

Of the 13,295 trees of all species surveyed in the nine planted stands, 19% had died by the final sampling date. *A. ostoyae* was responsible for 80% of this mortality, the largest single cause, and 24% of living trees had aboveground symptoms of *A. ostoyae*. Douglas-fir comprised 91% of all species since it was the target planted species on all sites, and 19% of these trees were dead with site SL having the highest mortality at 38%. Overall mortality for Abies sp. was 11% (100% from *A. ostoyae*), aspen sp. 4% (50%), western red cedar 1% (100%), paper birch 15% (60%), western hemlock 12% (90%), western larch 17% (100%), lodgepole pine 5% (100%), western white pine 23% (36%), willow sp. 8% (56%). The proportion of living trees with aboveground signs of *A. ostoyae* were Abies 41%, aspen 10%, western red cedar 20%, paper birch 10%, western hemlock 27%, western larch 20%, lodgepole pine 33%, western white pine, 22%, and willow sp. 23%. The cause of death in all tree species by site other than by Armillaria species was less than 1% except NK at 4% and SL at 8% mainly due to snow and stem breakage after age 30.

Generally, hazard began as early as age 6 (Fig. 1, NK site) and increased till about age 30 before declining. Averaged hazard rate over all sites peaks between age 13-15, and then declines by age 30 (Fig. 1 all sites). Annual hazard (Fig. 1) ranged between 0.002 and 0.02 (0.007 average) over all sites after age 14. Annual hazard was the highest on site SL at 0.08 at age 35 due to combined effects of Armillaria root disease, winter wind, and wet snow (Fig. 1).

The quadratic polynomial model for continuous time fitted the average hazard trend (Eq. 3) showing increase in hazard till about age 30. Annual climate variables allowed hazard deviation from the average, all with some significant effect on hazard after the effect of stand age was accounted for. Most of the best models were affected by temperature (Table 4). Degree days less than 0C (DDL0) proved to the best model when random effects were also considered, and was negatively correlated to hazard (lower mortality in colder years) (Fig. 1). Plot density (st/ha) was also tested for model inclusion but had no effect on the model and was not considered further. None of the precipitation variables tested ranked in the list of five best fitting models except the precipitation as snow, but which was also correlated with temperature. A second climate variable in addition to DDL0 was investigated for model inclusion, but since most of the best model terms were related to temperature, they were highly correlated to DDL0 and not considered. The addition of mean annual solar radiation (MAR) was not correlated with DDL0 and improved the model further (Eq. 3, Fig. 1). Both DDL0 and MAR interacted with time

and indicated that the effect of these terms was small at young ages but began to explain more of the annual variation as stands aged (Fig.1). DDL0 had the greatest effect on hazard compared to MAR. Combined, these hazard model terms give rise to the survival curves for each site (Fig. 2). DDL0 and MAR conditions for up to five years previous were applied to the current year's hazard, but the best fit occurred for the current year climate.

SI was not available for one site (SL) so comparison of models for all sites was not possible. SI was added to the model in Eq. 3 which lowered site and plot location variance indicating that SI explained most of the variation in hazard between sites, and also explained more annual variation as the stands aged. The study data indicated that colder sites did have lower SI but not highly correlated (Spearman's r=-0.28). With SI present in the model, the effect DDL0 was constant over time, but the effect of MAR was not significant; however, this finding is also based on one fewer site than the previous model.

The random intercepts for site or plot hierarchies (BLUPs) describe hazard differences associated with factors that are constant over time and differ from the fixed effects model. The variance associated with the site intercept was greatest at 0.36 compared to variation at the plot level at 0.18 (Table 5). For example, site HL had the greatest range in plot level odds ratio from 0.6 to 2.7 times as much hazard as the average (average odds=1), and only plot 5 was significantly different from the average; whereas, site BLUPs ranged from 0.4 to 3.2 times the average. Site SL only had plots with significantly greater ($p>0.05$) hazard than average occurring adjacent to each other. Site KF had two plots, and NK one plot significantly greater or lesser than the site average. None of the other site locations had plots differing from the average of all plots for that site, indicating that hazard within site between plots was similar. Random plot and site locations varying with time did not improve the model. Random site location odds ratio decreased (lower hazard) in order for site SL-3.2, NK-1.4, NB-1.3, HL-1.2, MM-0.9, KF-0.8, EB-0.8, CC-0.6, and KX-0.4. Sites NK and SL were different than the other sites in that they showed greater hazard between tree ages 9-14 than the average for all sites possibly because these sites were repeatedly observed, while on the retrospectively sampled sites, some of the very small trees that had died may have been difficult to locate later.

3.1.2 Diameter inside bark at breast height by age 15

For three sites CC, KF and KX, inside bark diameters were available at breast height (1.3 m) at age 15 for all trees, and this was used to relate initial size before infection to hazard in future time periods (Eq. 5) ($p<0.0001$, Fig. 3). All trees at age 15 in this analysis are not infected. Diameter showed a positive correlation with hazard ($p<0.0001$) but that this effect also interacted with time ($p=0.001$). The largest trees had the greatest hazard which also and peaked and declined sooner than the smallest trees. On average between ages 15 and 30, the odds of dying were 4.9 times greater for each cm increase in diameter inside bark from the average (4.8-5.8 cm). By age 30 no size related hazard differences were

evident. There were no significant effects of adding random site or plots intercepts, nor did these hierarchies differ over time. This indicated that the effect of tree diameter on hazard in sites and plots was similar.

3.1.3 Mortality impact

Plot basal area for the four older juvenile sites CC, EB, KF and KX were used to study the effect of tree mortality on plot yield (Eq. 6). Plot basal area was reduced linearly ($p=0.005$) with an increase in the proportion of dead trees per plot (average dead 6), and the number of dead and live trees per plot ($p=0.001$, average number 38) (Table 6). Using average predictor values in the model but varying the proportion of dead trees, the model predicts that a change over the range of dead trees in the plots (0 to 25%) reduced the plot basal area by 27% (Fig. 4, 18 to 13 m^2/ha) or almost 1 to 1 (percent plot basal area to percent dead trees). There were no significant differences between sites or for plots within sites. There was no interaction between site location and proportion of dead trees on hazard which showed the effect of dead trees on basal area was similar for each site location.

3.2 Natural Douglas-fir stands

3.2.1 Mortality and cause

Of the 1142 total tree species assessed on all the natural sites, 37% of trees were dead and 86% of these were dead from *A. ostoyae*. Of the living trees, 30% of these were infected with *A. ostoyae*. The most numerous species, Douglas-fir, comprised 49% of all tree species, with 54% dead by sampling and 91% due to *A. ostoyae*. The proportion of Douglas-fir that had died from other causes mostly occurred at sites RS (5%) and WL (3%) mainly because of wind, stem breakage, and suppression, with wind probably interacting with root disease due to damaged roots on many of these trees. Overall mortality for western hemlock was 28% (73% *A. ostoyae*); 37% paper birch (96%); 56% trembling aspen (100%); 68% western larch (17%); and 1.5% western red cedar (75%). The percent of living trees with aboveground signs of *A. ostoyae* were western red cedar 56%, Douglas-fir 33%, and western hemlock 15% and the rest less than 5% total.

Only Douglas-fir mortality was analyzed to determine the hazard in natural stands. It was possible to date Douglas-fir trees that had died up to approximately 45 years previously. The proportion of Douglas-fir trees that were too rotten to transport from the site were CN (8%), RS (15%), VB (6%), WL (5%); these were assumed to have died prior to the trees in this study because of their advanced decay. Some Douglas-fir samples transported to the lab had advanced decay which eliminated some of the key rings needed to match chronologies: CN (5% of sample), RS (10%), VB (4%), and WL (8%). These also likely died within the early time frame of the study trees due to the advanced decay.

The hazard began in three stands about age 55, except on the RS site where it began much earlier at age 36 (Fig. 5). These hazard patterns give rise to the survival curves for

each site which highlight these patterns (Fig. 6). Over all stands, Douglas-fir hazard increased ($p<0.0001$) but also slowed with time ($p=0.0002$) (Fig. 6) indicating that all sites shared common variation with time (summarized Table 7). The best hazard climate predictor SHM was positively associated hazard ($p=0.0091$) on all sites, and which increased with time ($p=0.03$, SHM x time interaction). The model gave best fit with hazard when the SHM three years prior to the year of tree death was used. SHM was best related mean summer moisture having higher values with lower moisture (Spearman`s $r=-0.97$). The proportion of Douglas-fir in the stand was associated with greater hazard ($p=0.0008$) but this also slowed with time ($p=0.0029$, PFIR x time interaction). PFIR was best related with precipitation as snow (Spearman`s $r=-0.35$). Greater site index (SI) was associated with increased hazard regardless of time ($p=0.0341$), and was negatively related to the extreme minimum and maximum temperatures (Spearman's $r=-0.93$). The addition of a random site or plot intercept or fixed effect of tree density (alive plus dead stems/ha) had no effect on the model.

3.2.2 Mortality impact

Plot basal area of all tree species was linearly reduced with an increase in the proportion of all dead tree species per plot ($p=0.002$, mean dead=38%) and increased with the number of all trees per plot ($p=0.001$, mean number/ha=36) (summarized Table 8, Fig. 7). Over all sites, the percent of dead trees in the plots ranged 50% (from 20% to 70%), which the model predicts results in a 56% reduction of basal area (about 1:1 percent mortality to percent basal area change), or a change from 46 to 20 m^2/ha at mean plot density. There were no plots with zero mortality from root disease; therefore, the yield estimate was not reflective of the disease-free condition. There were no statistical differences in average yield between site locations, but there were differences in yield between plots within sites. The largest positive plot intercept BLUP on the Wilson site showed better than average yield for a given the level of mortality. The plot had lower percent dead trees, but Western redcedar trees contained more than half the basal area alone being the largest diameter of any trees in the plot.

The effect of tree size on hazard in the natural stands could not be modelled because there was incomplete data on the growth of the living trees over time. Despite this, average dead tree diameter actually exceeded this of the living trees for at least two site locations (Table 2).

4 Discussion

4.1 Hazard related to temporal and spatial factors

Time of death up to 45 years prior to the sampling date was determined using tree ring chronology. Similar methods for conifers in temperate forests showed accurate dating up to 50 years previously (Dynesius and Jonsson 1991). A small percentage of trees in this study were either too rotten to sample or had damaged key indicator rings, and probably died before any of the trees in this study. Increasing decay was correlated with time since death in another study (Daniels et al 1997). The exact year of death may also vary in

trees associated with Armillaria root disease because they often have very small outer rings (Cherubini et al. 2002), or trees may fail to produce complete rings (Mast and Veblen 1994).

At any one time, there are a limited number of trees with heavily infected root systems likely to succumb to an additional limiting climate event. Mortality associated with limiting climate events would therefore also reflect the available number of predisposed trees at that time. A second limiting climate event occurring later may not have the same effect if the pool of heavily infected trees was exhausted previously. Even when climate interacts with disease, fungal spread in larger trees may take a few additional years to reach and girdle the root collar, while smaller infected trees probably die the same year. Older natural stands with larger trees did show that mortality peaked on average about 3 years after a limiting climatic event, but this pattern was not detected in the planted stands. Bigler et al (2007) also noted that tree morality was delayed after limiting climate events assumed to be biotic causes.

Averaged over all planted stands, hazard increased till about age 30, and then declined, and the timing and amount of hazard varied by site location. The hazard patterns vary because of the number of infections on a tree, their distance to the root collar, the size of the inoculum and the fungal spread rate (Morrison 2011). Soil physical conditions and climate also likely interact with all these variables. The observed plantation disease epidemiology results from a slow transfer of *A. ostoyae* from primary inoculum (stump) to healthy tree roots which begins about age 5 (Morrison et al 2014) and is maximized around tree age 15 belowground (Morrison 2011). Small trees infected near the root collar would likely die soon after contact with the fungus. For larger trees and trees with infections occurring more distally, fungal spread would be mainly within root systems and intensifying root system damage over longer time periods. The inoculum in stumps and that building in dead tree roots and living trees with girdled roots over time were reflected in the mortality peak between ages 20-30. After age 30, slowing mortality may be partly due to increasing Douglas-fir resistance to root disease that slows fungal spread (Robinson and Morrison 2001, Morrison 2011) or that some of the original stump inoculum is no longer infective.

The averaged hazard rate in planted stands after age 14 was 0.7% annually. This agreed with species selection trials for planted Douglas-fir in its southern range with similar temperature and moisture ranges, but not that of the northern range trials in wetter and cooler conditions than the ones discussed here (Vyse et al. 2013). Annual mortality in western inland US juvenile Douglas-fir was 2-3% (Hagle 2010), which is about 3 to four times greater than in BC except for the worst site. The US sites are located more southerly and likely are both warmer and dryer than those in BC. If these climate conditions move northward and become more prevalent in BC, similar patterns may be expected.

On the four older natural sites, Douglas-fir hazard also increased after stand age 55-65 but slowed with time. Hazard increased on one site 20 years earlier than this, but anecdotal observations indicate this is not common. With the caveat that early mortality

may not be represented properly in the natural stands, there appears to be a lag in root disease mortality after age 30 till about age 55-65. Douglas-fir in similar natural ecosystems of the US killed by Armillaria root rot was maximized in the six or seventh decade (Watt 1960). This process began about age 40-45 (Haig et al 1941, Hagle 2010) causing stand replacement by the end of the century. Mortality rates for mature Douglas-fir in the western US ranged from about 2-3% annually (Hagle 2010), similar to the rates in the current study after age 60. Temesgen and Mitchell (2005) note that Douglas-fir mortality in the southern BC interior was about 1.5% annually, lower than reported in this study or in the US; however, plots with disease were excluded from that study.

There was significant site-level hazard variation that resulted in the different survival curve trajectories, but both random study hierarchies (site and plot location) were constant with time. Possible factors associated with the hierarchies could be soil properties, or slowly changing fungal inoculum, fungal inoculum potential, or fungal competition differences within hierarchies, but less likely to be factors that can change rapidly year to year such as climate. Site level variation might be affected by larger trees that increase contact with inoculum more frequently and sooner (Cruickshank et al 2011). There is an absence of field studies documenting factors that affect stump colonization or substrate utilization by Armillaria sp., but competition for stump substrate has been shown to occur for some fungi (Pearce and Malajczuk 1990) and by root feeding beetles and weevils (personal observations). Soil nutrient status can affect rhizomorph biomass through fungal uptake and translocation of nutrients (Morrison 1975); subsequently, this also would result in greater fungal to host contact and mortality on fertile sites. Fertile sites would also be expected to have greater site index which is discussed below.

4.2 Hazard affected by life history

Life history traits related to tree size, growth, or disease susceptibility were significant factors in this study. Growth of interior Douglas-fir was affected by stand-level factors as much or more than climate (Lo et al 2010). Growth and survival are closely related in many tree species (Bigler et al 2004, Das et al 2007) because growth reflects the balance of photosynthesizing versus respiring tissue (Oliver and Larson 1990). In the natural stands, the addition of fixed effects PFIR and SI were largely responsible for reduction in random site location hazard variance. Increased hazard associated with PFIR is consistent with Douglas-fir's high susceptibility ranking (Morrison et al 1991), and greater density of disease susceptible species probably increased hazard. Alternatively, Douglas-fir is a fast growing tree species which may increase its contact with inoculum sooner. The effect of PFIR decreased with time probably due to the increasing distance between living disease susceptible trees species like Douglas-fir as they die.

SI was positively related to hazard in seven planted stands where data existed, and in the four older natural stands. SI showed increasing effect with time in the planted sites and explained much of the variation in hazard between site locations. SI for planted Douglas-fir was positively correlated with greater moisture and temperature in BC (Nigh et al 2004). A positive association between SI and root disease mortality has not been noted in

other studies. Stands of coastal Douglas-fir are generally more productive with greater SI and lower root disease than interior stands (Morrison et al 1991), and the results apply to interior ecosystems. Interior stands with high SI would promote greater root contact between the Douglas-fir roots and increase contact with fungal inoculum. An increase in mortality with SI and has been noted for Norway spruce (Eid and Tuhus 2001), ponderosa pine (Uzoh and Mori 2012), and mixed conifer stands (Hann and Hanus 2001) which was traditionally explained by increased competition at earlier ages. However, many of the annual rings from dead trees in the natural stands showed that early growth was initially superior compared to live trees, but this was reversed some years before death. Likewise, this pattern was noted in other dendrochronological studies of trees infected with Armillaria root disease (Bloomberg and Morrison 1989, Hrib et al 1983).

Annual tree growth may not be completely reflective of the length of the growing season because high growth rate in the early part of the season can offset lower growth later (Gould et al 2012) before moisture or temperature limitations occur (St. Clair et al. 2005). In other words, sites with high SI can growth rapidly early in the season, and then have increasing water potential from low summer precipitation. Trees on dry sites are associated with increased damage from Armillaria root disease (Cruickshank et al 1997). Consequently, it is possible that trees on sites with higher SI and that also have dry late summer conditions would maximize contact with fungal inoculum and then be under physiological constraint.

It is difficult to separate tree size hazard from disease hazard because larger trees could one or all of greater physiological constraints, greater tradeoff of growth and defense, or more frequent infections. Temesgen and Mitchell (2005) showed that larger interior Douglas-fir in southern BC had increased mortality over that of mid-sized trees that could not be clearly explained by competition. Pathogen and insect activity was implicated with greater conifer mortality than expected in larger sized trees (Smith et al. 2005) suggesting that larger trees or more productive areas are more conducive to pest attack or that they have lower defense (Stephenson et al. 2011). More productive stands in the BC interior sites do have greater disease pressure because larger trees contact inoculum more frequently (Morrison et al 2000, 2001). It might also mean that larger trees operate near physiological constraints and are least able to handle some types of additional stresses (Ryan et al 2006, Woodruff et al 2008).

To support that size related morality is due to inoculum contact, planted trees with larger diameter at age 15 were both more frequently killed and occurring sooner than small trees up to age 30 only. Diameter at age 15 was used as a measure of tree size because planted trees of this age were largely disease free. Since largest planted trees have greater probability of fungal infection (Morrison 2000), appear to die more frequently and sooner, and have the least competition, the study results are not suggestive of a tradeoff between defense and growth or that they operate closer to physiological constraints, at least in the younger planted stands. Stumps from the original harvest are mostly gone 25 years later which more closely fits the hazard pattern in this study.

There was no evidence directly linking plot density to hazard at any age, possibly because site productivity is a more important measure than density alone. Incidence of Armillaria root disease mortality in Douglas-fir was found to be lowest in high density natural plots (3000-4000 stems/ha) (Rosso and Hansen 1998). Higher crop plant densities for a given age should promote higher fungal transmission rates (Burdon and Chilvers 1982) resulting in greater mortality in susceptible species. Among the current study sites, density did not range greatly (about 600-1500 st/ha), but might show more effect with greater extremes. In younger stands tree growth becomes less affected by stand density and more affected by disease status with time (Cruickshank et al 2009, 2011). Tree mortality in the Western United States was related to temperature and moisture constraints not to density (van Mantgem and Stephenson 2007, van Mantgem et al 2009). Density also did not affect the presence of dead trees in coastal Douglas-fir stands infected with Armillaria root disease (Pielou and Foster 1962). Competition alone was unable to explain the spatial pattern of large and small tree mortality that may possibly be better explained by disturbance agents (Lutz et al 2012). Where biotic disturbance agents are common, these agents could be important determinants of stand structure. How tree density interacts with root disease is still poorly studied.

4.3 Hazard related to climate

4.3.1 Planted stands

Temperature was a greater limiting factor than precipitation on hazard in the planted stands in this study. The study site ecosystems have dry summers but severe moisture limiting events are infrequent. After controlling for time, stand attributes, and sample hierarchical structure, hazard in planted stands decreased with DDL0 and increased with MAR and became greater with time in nine study locations. DDL0 had the greatest effect on hazard. A second model additionally controlling for SI in eight of these locations agreed with the previous model in that colder sites had lower hazard, but that MAR was not now significant. The range of Douglas-fir includes colder continental sites (Burns and Honkala 1990) which suggest that cold is not affecting tree survival by itself. Soil temperatures less than 10 C can inhibit the fungus through slow rhizomorph growth (Rishbeth 1978) which is the chief form of fungal spread. Further, there is a tradeoff between cold hardiness and growth in interior Douglas-fir (Darychuk et al 2012, St. Clair et al 2005) meaning that trees on colder sites also have earlier dormancy resulting in reduced root growth at the same time. This probably interacts with lower rhizomorph growth resulting in lower contact between the host and pathogen.

A. ostoyae is limited at the northern range of Douglas-fir (Morrison et al. 1985) probably because the soils are too cold and wet to allow adequate root and rhizomorph growth, or lower temperatures might affect fungal inoculum potential or longevity itself. Inoculum potential is the energy available to the pathogen for infection at the site of contact with the host (Garrett 1960). Inoculum potential also includes the rate of substrate utilization and energy transfer along the rhizomorph network to the site of infection. Cold and wet conditions are not conducive to Douglas-fir wood decay (Progar et al. 2000) suggesting that substrate utilization is more difficult with cold and therefore would lower inoculum

potential. Cold conditions also directly lower rhizomorph growth, and wet conditions with saturated soils reduce oxygen content and fungal respiration (Rishbeth 1978). Direct freezing damage to fungal inoculum viability has not been studied, and freezing might have some effect on stump inoculum especially in underdeveloped canopies.

4.3.2 Natural stands

In natural stands, hazard was positively correlated with SHM and became stronger with time as the trees aged and became larger. Lower mean summer precipitation for a given mean summer temperature was responsible for most of the variation in SHM values. Increasing tree size and non-lethal infection of root systems over time probably interact with SHM to affect tree growth and survival. Tree growth was explained by a heat moisture index in interior Douglas-fir (Chen et al 2010), and low summer precipitation was a good predictor of Douglas-fir growth alone (Darychuk et al 2012). Higher temperatures alone did not affect survival in interior Douglas-fir on the current study sites, but lower summer rainfall without a corresponding drop in temperature could create higher tree water potentials. Under high water potential, cavitation may be a limiting factor (Cochard and Delzon 2013) which could be more frequent in taller or older trees (Ryan et al 2006). Longer droughts can also promote carbon starvation and possibly reduce resistance to pests (McDowell et al 2008, McDowell et al 2011). Carbon starvation might also occur by stress impaired transport or metabolism mechanisms in the trees that have intact but unusable carbon pools (Sala et al 2010); consequently, this might allow the fungus to utilize carbon in roots locally for infection and colonization that the tree is unable to utilize. Armillaria species can also alter root carbohydrate source into forms more suitable for fungal degradation of host defense compounds (Wargo and Shaw 1985). Larger trees with more damaged root systems exposed to severe climatic events likely have both carbon limitation and greater defense cost (Oliva et al 2014) as well as reduced hydraulic limitation directly (Baker 1994, Joseph et al 1998), all of which can kill the tree or promote fungal spread within root systems. Changes in the frequency of droughts could be especially important in the currently wetter parts of the Douglas-fir range due to lower host resistance in the tree population in these areas (Cruickshank et al 2010).

4.4 Stand yield and its relation to mortality

The ratio of percent tree mortality to plot basal area yield was approximately 1 to 1 (percent basal area to percent dead trees) in both stand types. There was no evidence that this effect differed between sites, or that the effect was nonlinear. For a given tree species, yield is affected by tree density and site productivity, and basal area is disproportionately represented in larger stems. Stand biomass in older natural stands in California was not affected by increasing drought mortality mainly because the smallest trees were dying (van Mantgem and Stephenson 2007). The expected situation for stand self-thinning competition is that tree biomass increases while density decreases; in other words, surviving trees grow fast enough to replace biomass lost to mortality (Peet and Christensen 1987); however, loss of large trees can cause yield to be negative over time

(Hoshizaki et al 2004). The current study results suggest that growth does not keep pace with mortality likely because of the combined effects of some mid to larger sized trees dying and growth reduction in living trees with infected roots. A few current study sample plots had greater basal area yield for a given percentage of dead trees than the average of all plots on that site. The most extreme plots occurred in natural stands with the majority of the plot basal area contained in western redcedar trees. In a similar ecosystem, redcedar was termed disease tolerant meaning disease in this conifer species had almost no disease impact on yield; furthermore, these trees were replacing basal area lost to Douglas-fir mortality from root disease (Morrison et al 2014).

Conclusions

Armillaria root disease was the principle cause of Douglas-fir hazard in all sites in the study, and a sizable number of living trees had disease signs or symptoms. Hazard in young stands increased with time but slowed by age 35. Hazard was reduced in years with greater accumulation of cold temperatures which probably reduced inoculum potential or reduced contact between inoculum and the host. However, a negative effect of cold on fungal stump inoculum directly could not be ruled out. In older and younger stands, SI was positively related to hazard, which was likely a direct result of the greater contact with fungal inoculum associated with large trees. In older natural stands hazard was positively related to SHM but this slowed with time. Lower summer rainfall was mainly responsible for higher SHM hazard which possibly increased the hazard due to cavitation, vascular occlusion, or carbon starvation or a combination of these in diseased trees. Mortality in this study impacted negatively on basal area yield, meaning that growth of living trees was not able recover basal area lost to dead trees.

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Table 1. Descritive statistics for planted stands

Attribute	Chuck Creek (CC)	East Barriere (EB)	Hidden Lake (HL)	Kingfisher (KF)	Kuskanax (KX)	MacMurphy (MM)	North Barriere (NB)	Nakusp (NK)	Sugar Lake (SL)
Lat./Long. Elevation (m)	51.6N 119.7W 690	51.3N 119.8W 700	50.5N 118.9W 650	50.7N 118.8W 420	50.3N 117.8W 950	51.7N 119.5W 980	51.2N 119.8W 670	50.3N 117.7W 850	50.4N 118.6W 600
Site age at final sampling	34	25	24	30	32	24	20	41	38
Number of plots (ha)	25 (0.03)	23(0.03)	24(0.03)	25(0.03)	25(0.03)	23(0.03)	27(0.03)	8(0.25)	8(0.25)
Proportion dead trees all species	0.07	0.05	0.08	0.08	0.04	0.05	0.05	0.17	0.40
Site index age 50 (m)	26.8	28.2	30.5	29.2	29.2	27.2	26.5	-	-
Mean DBH (cm),[alive, dead], (alive max.,min.)	[15.9,10.9] (34.0,6.4)	[12.2,7.5] (24.4,3.4)	[12.0,8.8] (23.4,5.1)	[15.1,10.6] (27.9,6.2)	[16.5,10.6] (29.6,6.0)	[12.1,6.5] (24.6,1.4)	[9.4,6.2] (19.6,2.6)	[17.5,8.1] (33.0,3.1)	[11.6,7.3] (22.2,2.4)
alive plus dead stems/ha (max.,min.)	1056 (48,19)	1405 (72,31)	1310 (28,53)	1258 (55,26)	1246 (53,21)	1573 (58,38)	1356 (62,34)	1074 (51,24)	821 (51,24)
Proportion Douglas-fir[1]	0.90 (1.00,0.55)	0.81 (0.97,0.69)	0.93 (1.00,0.75)	0.86 (1.00,0.69)	0.95 (1.00,0.78)	0.94 (1.00,0.87)	0.87 (1.00,0.62)	0.98 (0.99,0.79)	0.95 (0.99,0.79)

Table 2. Descritive statistics for natural stands

Attribute	Connor Creek (CN)	Russell (RS)	Vavenby (VB)	Wilson Creek (WL)
Lat./Long./Elevation (m)	49.4N 117.5W 950	51.3N 119.9W 720	51.6N 119.8W 740	50.1N 117.4W 550
Site age at sampling	110	63	93	95
Number of plots (ha)	2(0.1)	2(0.1)	2(0.1)	2(0.1)
Mean DBH Douglas-fir (cm) alive, dead (alive-max.,min.)	39.0,25.2 (73.0,11.3)	21.4,15.7 (47.5,10.9)	30.7,27.2 (50.6,10.7)	23.0,16.6 (67.0,10.2)
Site index age 50 (m)	24	26	22	19
Mean DBH all trees (cm) alive, dead	22.1, 26.8	20.0, 15.2	18.7, 22.0	19.9, 15.1
Proportion of Douglas-fir on site (dead plus alive)	0.29	0.76	0.51	0.44
Proportion Douglas-fir alive at sampling age (all species alive)	0.22 (0.65)	0.42 (0.48)	0.47 (0.59)	0.59 (0.70)
Proportion of all trees with aboveground symptoms of *A. ostoyae*	0.39	0.17	0.19	0.32
Stems/ha alive all species (alive plus dead)	855 (1300)	620 (1280)	580 (975)	1520 (2155)
Proportion of basal area alive Douglas-fir	0.40	0.59	0.54	0.74

Table 3. List of abbrevations used in the study.

Variable	Description
AHM	annual heat:moisture index (MAT+10)/(MAP/1000))
DDG5	degree-days above 5 (°C)
DDL0	degree-days below 0 (°C)
DDL18	degree-days below 18 (°C)
eFFP	julian date on which FFP ends
EMT	extreme min. temp. 30 years (°C)
EXT	extreme max. temp. 30 years (°C)
KM	Kaplan Meier hazard
MAR	Mean annual radiation (MJ $m^{-2}day^{-1}$)
MAP	mean annual precipitation (mm)
MAT	mean annual temp.(°C)
MCMT	mean coldest month temp.(°C)
MSP	mean annual May to Sept. precipitation (mm)
MWMT	mean warmest month temp.(°C)
NFFD	number of frost-free days
PAS	precipitation as snow (mm)
PFIR	proportion of Douglas-fir stems
SHM	summer heat:moisture index ((MWMT)/(MSP/1000))
SI	site index age 50 (m)
Study sites	CN= Connor Creek; EB= East Barriere; HL= Hidden lake; KF=Kingfisher; KX=Kuskanax; MM=MacMurphy; NB=North Barriere; NK=Nakusp; RS= Russell; SL= Sugar Lake; VB= Vavenby; WL=Wilson Creek

Table 4. Goodnes of fit statistics for model Eq. 1 and showing the top ten best fixed effect parameters.

Planted sites[1]	**-2 log likelihood**	**AIC**
None	28519.3	28525.3
DDL18	28170.3	28180.3
DDL0	28183.0	28193.0
MAT	28209.2	28219.2
PAS	28215.9	28225.9
NFFD	28256.4	28266.4
EXT	28325.3	28335.3
AHM	28331.9	28341.9
MCMT	28337.1	28347.1
MAP	28345.5	28355.5
DDG5	28352.1	28362.1
Natural sites		
None	2362.4	2368.4
PFIR	2222.6	2232.6
SI	2244.2	2254.2
EMT	2332.2	2342.2
PAS	2334.5	2344.5
EXT	2341.8	2351.8
eFFP	2346.2	2356.2
SHM	2349.8	2359.8
DDL18	2351.0	2361.0
MSP	2351.4	2361.4
MAT	2352.2	2362.2

[1]SI does not show in the list for planted sites since it is based on one less site than this list.

Table 5. Maximum likelihood analysis of variance table for the effects of climate and stand variables on hazard rate in planted sites.

Effect	**Estimate**	**Std. Error**	**p value**
Fixed effects			
1) main effects			
a) Intercept	-5.0183	0.9466	<0.0001
b) Stand age	-0.1877	0.0842	0.0257
c) Stand age^2	0.0058	0.0018	0.0011
d) DDL0	-0.0048	0.0009	<0.0001
e) MAR	-0.0952	0.0541	0.0785
2) interactions			
b*d	0.00058	0.00009	<0.0001
b*e	0.01225	0.00493	0.0129
c*d	-0.00002	1.9×10^{-6}	<0.0001
c*e	-0.00021	0.00011	0.0450
Random effects		**Variance**	
Site location intercept		0.3635	
Plot location intercept		0.1766	
χ^2/D.F.	0.81		

Table 6. Maximum likelihood analysis of variance table for the effect of the percent of dead trees in sample plots and the number of trees on the total plot basal area in planted sites (m^2/ha).

Effect	**Estimate**	**Std. error**	**p value**
Fixed effects			
Intercept	13.7664	1.8138	<0.0001
Percent dead trees	-0.1568	0.0537	0.0052
Number of trees	0.1247	0.0368	0.0012
Random effects		**Variance**	
Site location intercept		3.2816	
Plot within site intercept		5.1973	
Residual		0.8504	
Akaike's information criteria		365.6	

Table 7. Maximum likelihood analysis of variance table for the effects of climate and stand variables on hazard rate in natural sites.

Effect value	**Estimate**	**Std. Error**	**p**
Fixed effects			
1) main effects			
a) Intercept	-22.1543	2.6909	<0.0001
b) Stand age	0.2798	0.0679	0.0002
c) Stand age^2/100	-0.0012	0.0003	0.0008
d) SHM	-0.0284	0.0109	0.0091
e) PFIR	0.1391	0.0287	0.0323
f) SI	0.1392	0.0388	0.0341
2) interaction			
b*d	0.00034	0.00016	0.0323
b*e	-0.00162	0.00043	0.0029
Random effects		**Variance**	
Site location intercept		0	
Plot location intercept		0.0044	
χ^2/D.F.	0.78		

Table 8. Analysis of variance table for the effect of the percent dead trees in plots and the number of alive plus dead trees on the total plot basal area in natural sites (m^2/ha).

Effect	**Estimate**	**Standard error**	**p value**
Fixed effects			
Intercept	26.9566	11.0502	0.0343
Percent dead trees	-0.4992	0.1527	0.0028
Number of trees	0.8408	0.2026	0.0095
Random effects		**Variance**	
Site location intercept		1.75	
Plot within site intercept		22.63	
Residual		120.37	
Akaike's information criteria		251.4	

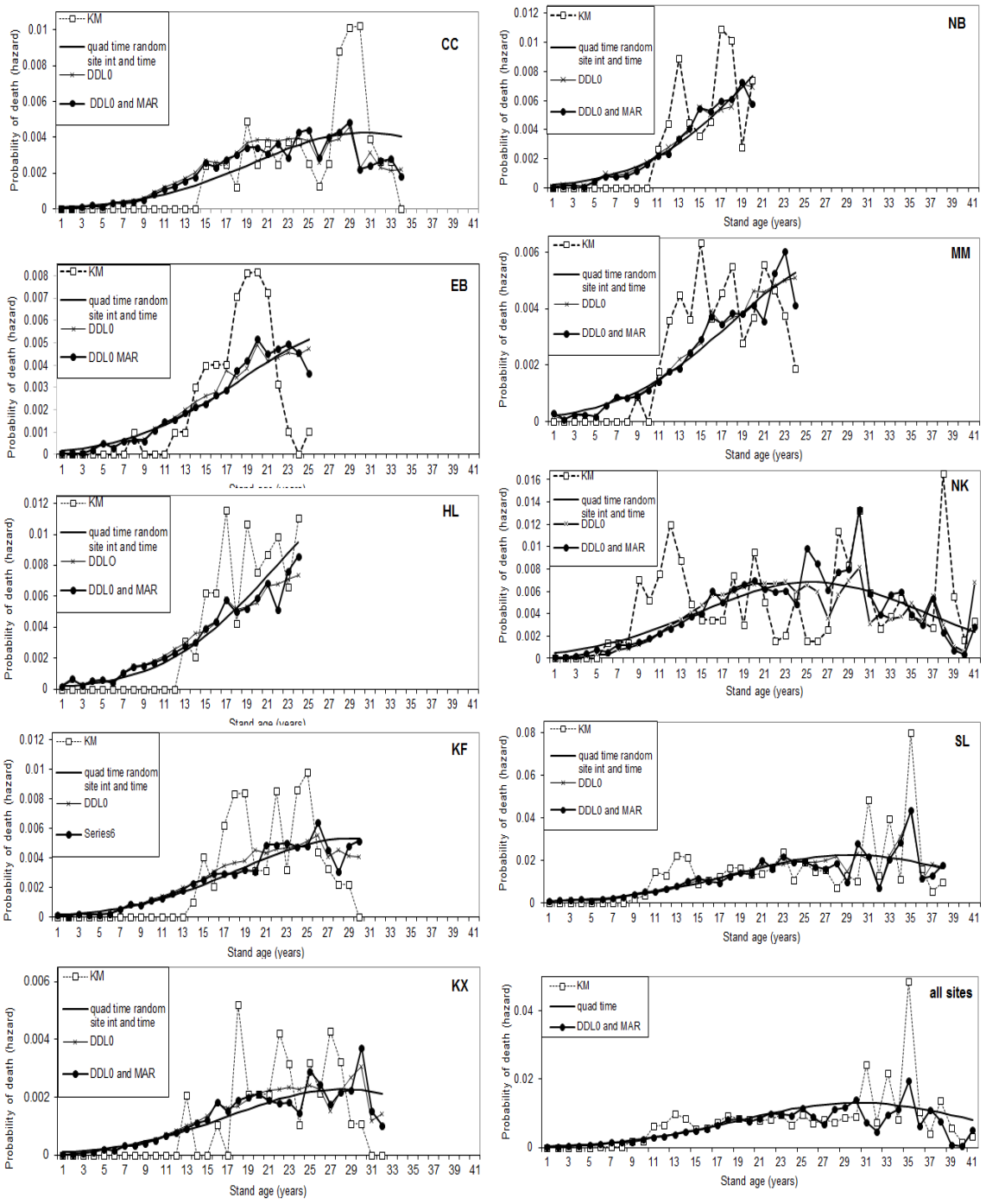

Figure 1. Hazard rates for nine planted Douglas-fir sites and the average for all sites. KM means Kaplan-Meier hazard estimates, quad time means a quadratic time hazard model with random site effects, DDL0 means this term was added to the quad time model, and the DDL0 and MAR means these terms were added to the quad time model for hazard.

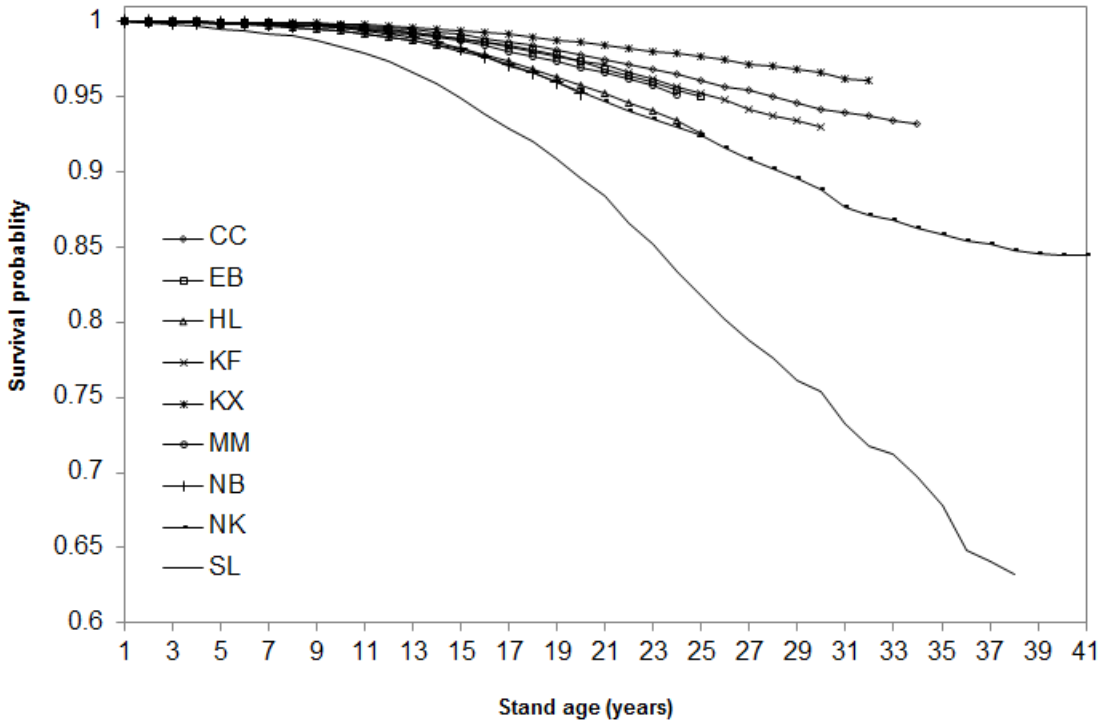

Figure 2. Survival proablity for each of the planted Douglas-fir sites.

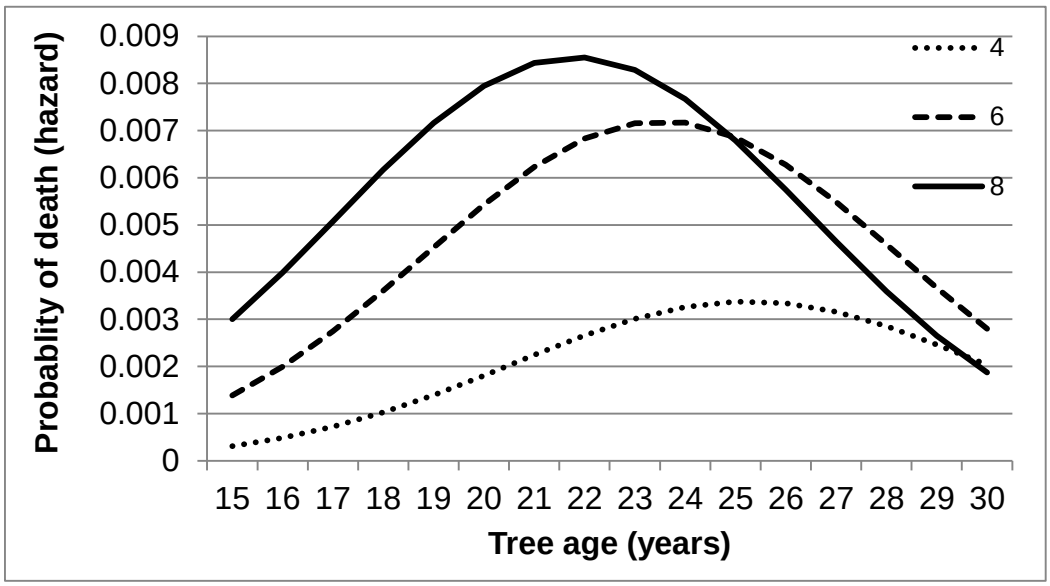

Figure 3. Future hazard of planted Douglas-fir was afected by inside bark diameter (1.3 m). Hazard curves are shown for age 15 model prediction of 4, 6, and 8, cm diameter stems.

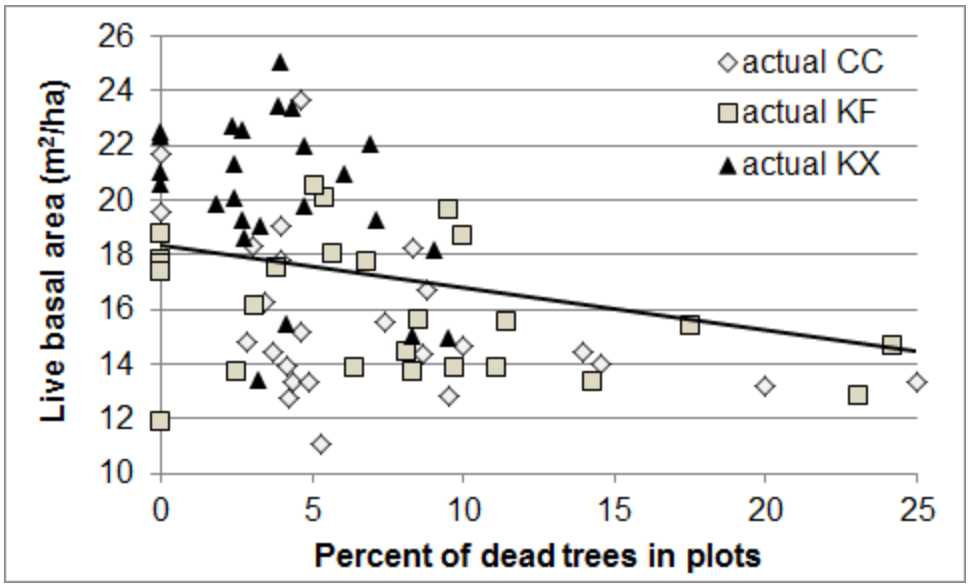

Figure 4. The effect of dead trees on basal area in four planted stands. Symbols are actual data and the solid line represents the model fit.

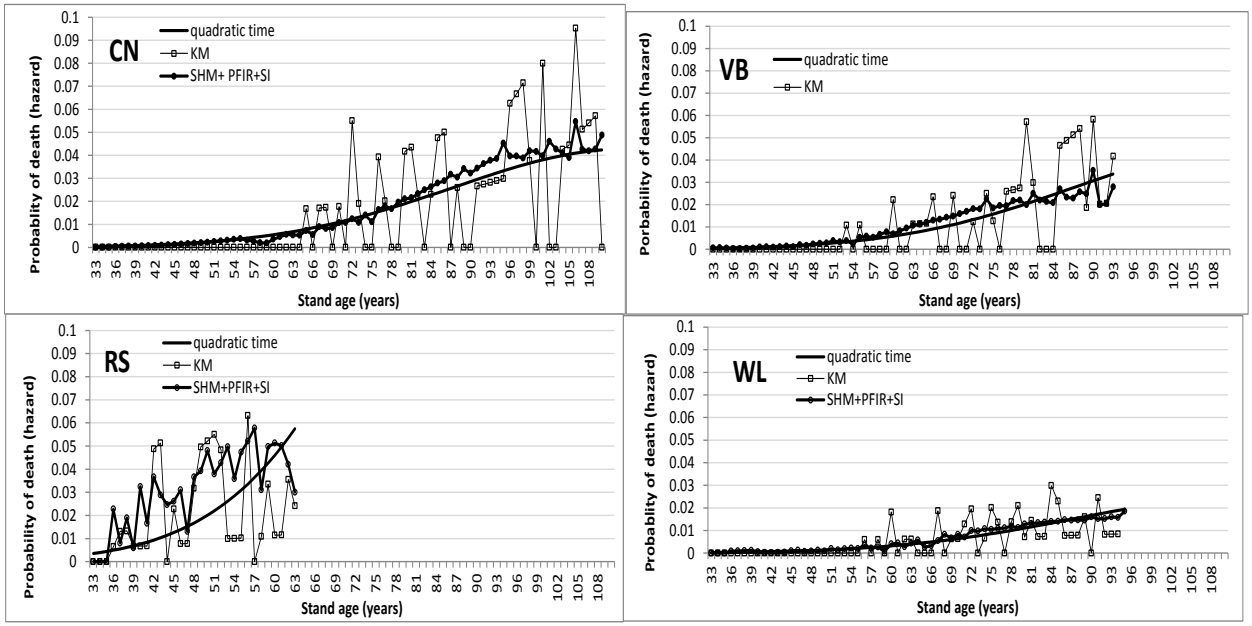

Figure 5. Hazard rates for four Douglas-fir leading natural stands. KM stands for Kaplan-Meier hazard estimates, quadratic time means a quadratic time hazard model with random site effects, SHM, PFIR and SI means these terms were added to the quad time model for hazard.

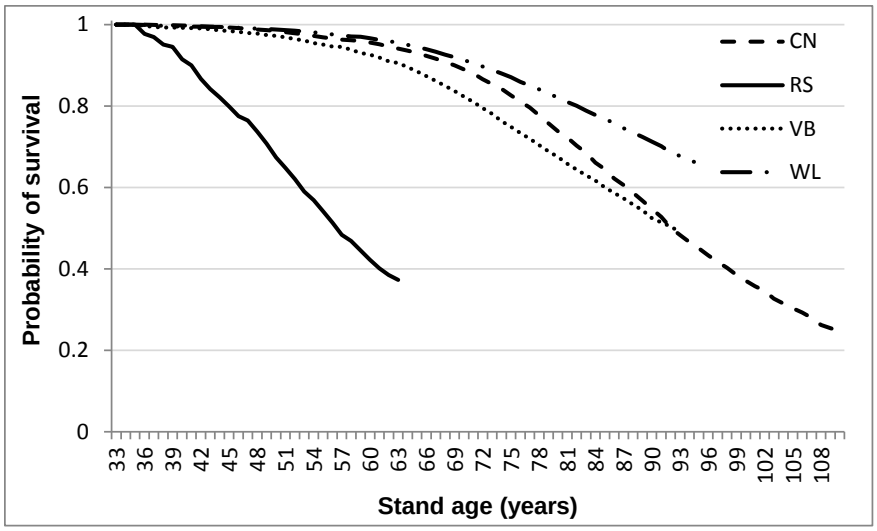

Figure 6. Survival probability for four natural Douglas-fir stands.

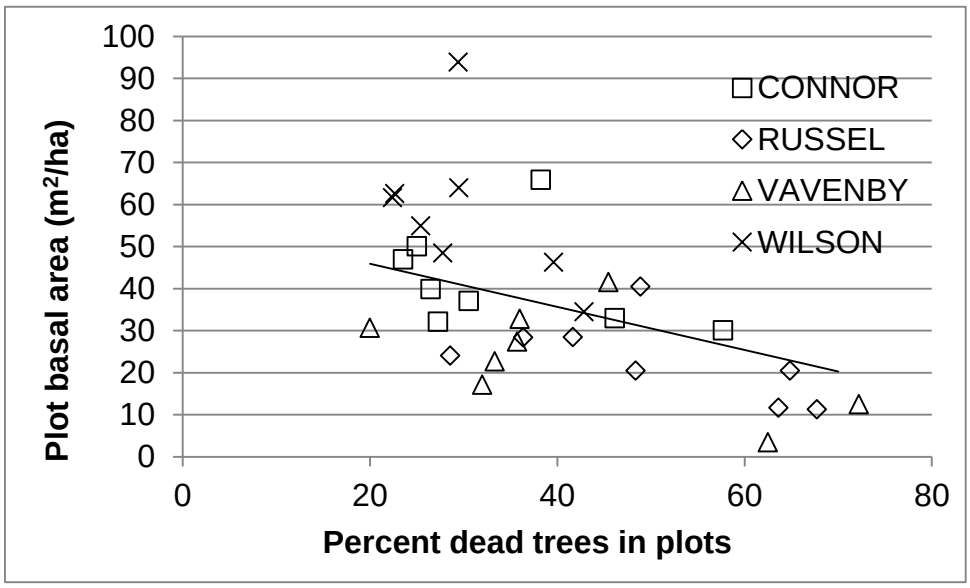

Figure 7. The relationship between percent dead trees in plots and their effect on basal area of all tree species in natural stands. Symbols represent actual data, and the solid line represents model fits combined for all sites.