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forest ecology

Survival, and Growth Response of Douglas-Fir Trees to Increasing Levels of Bole, Root, and Crown Damage

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We applied a range of bole and root damage treatments to young Douglas-fir (*Pseudotsuga menziesii* [Mirb.] Franco) trees. Significant natural damage occurred to tree crowns over the course of the 10-year study allowing for an analysis of how damage severity to tree boles, tree roots, and tree tops impacts growth and cumulative survival of trees. Tree bole damage severity, measured by percent bole circumference removed, exponentially reduced cumulative survival across the study period, whereas damage to tree roots had no significant impact on survival. Bole damage had different impacts on survival depending on the relative size of trees that were damaged. At higher levels of bole damage, trees with larger relative diameters had accelerated mortality rates compared with trees with smaller relative diameters. Tree crown height loss averaged at the treatment within stand level reduced the impacts of bole damage on tree cumulative survival response. Increasing bole, root, and tree crown damage severity led to reductions in estimated maximum diameter and height. Bole damage and damage to tree crowns hastened the decline of diameter growth rates and delayed the decline of height growth rates.

Keywords: Douglas-fir growth, Douglas-fir survival, bole damage, crown damage, root damage

Damage to coniferous tree boles and roots can reduce the economic value, height and diameter growth, and survival of trees. Tree damage comes in many forms and from many vectors. Management activities related to tree removal can cause damage to tree boles and roots of the residual stand (Vasiliauskas 2001). Animals can damage tree boles (Kanaskie et al. 2001, Miller et al. 2007), roots (Witmer et al. 1998), and tree crowns (Stricklan et al. 1995). Weather events can cause stem breakage in the upper portions of tree crowns (Pukkala et al. 2016) reducing tree photosynthetic potential via the loss of leaf area. Tree recovery from damage requires the use of photosynthates that could have been used for other purposes had the damage not occurred.

Tree damage that reduces photosynthetic capacity or reallocates photosynthates to wound closure could reduce tree growth and survival (Waring 1987). Damage to tree boles has been shown to reduce both diameter and height growth rates (Hanus et al. 1999, Hann and Hanus 2002a, 2002b), and lead to reduced survival (Ganio and Progar 2017). Damage to tree roots is associated with reductions in height and diameter growth in conifers and can expose roots to decay fungi (Vasiliauskas 2001, Harrington and Thies 2007), potentially leading to early tree mortality. Damage to tree crowns directly reduces photosynthetic capacity and, if severe enough, can lead to early tree mortality (Ryall and Smith 2005).

The presence or absence of damage has been used to investigate mean differences in tree size and mortality (Isomäki and Kallio 1974, Vasiliauskas 2001). Broad categories of tree damage have been integrated into tree diameter and height growth rate models (Hann and Hanus 2002a, 2002b). Analyzing the relationship between broad categories of damage and tree growth or survival can be useful in estimating an average tree response to damage. However, there have been no studies on how trees respond to increasing levels of damage severity across a gradient of tree characteristics (diameter, height, height to live crown, etc.) over time. Determining the nature of tree response to

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damage across gradients of tree characteristics may yield important insights.

Douglas fir (*Pseudotsuga menziesii* [Mirb.] Franco) is an ecologically important species (Franklin and Dyrness 1973). As for other conifers, the response of Douglas-fir trees to increasing levels of damage severity is not well understood. Currently no models exist that incorporate tree survival or growth response to damage severity across gradients of tree characteristics. In order to examine the nature of tree diameter, height, and survival responses to increasing levels of damage severity, we incorporated percent damage severity measurements based on bole circumference, root cross-sectional area, and cumulative tree crown height loss into models that were designed to predict cumulative survival, tree diameter, and tree height over time. This approach was used to answer the following questions:

1. How does damage to tree boles, roots, and crowns influence cumulative survival over time, and how does this relationship vary with competition, and relative tree size?
2. How does percent damage to tree boles, roots, and crowns influence tree diameter and height growth over time, and how do these relationships vary across gradients of relative tree size, tree crown metrics, and competition indexes?

Methods

Stand, Plot, and Tree Selection

Six stands were selected to represent common young Douglas-fir plantation stand structures. The stands were located in the Capitol State Forest near Olympia, WA. Stands were even-aged and planted between 1983 and 1986. Site preparation consisted of either broadcast burns or vegetation control via herbicides. Stands were located around a central latitude of 46.8949° and a longitude of –123.125°. All stands had received a pre-commercial thinning treatment 5–14 years prior to plot establishment in the Spring of 2006, with grid pattern target spacings of 3.18–4.14 m. All stands were on Olympic series soils classified as fine, mixed, active, mesic Xeric Palehumults soils. This soil series consists of very deep, well-drained soils formed in residuum and colluvium weathered from basic igneous rock. Stand average slopes ranged from 5 to 40 percent with elevation ranging from 130 to 340 m.

In the spring of 2006, one plot was established in each stand and was designed to contain at least 240 Douglas-fir trees greater than 10 cm in diameter at breast height (dbh). Each plot was established within a 24 × 24 row area in the stands described above. Plot areas were as uniform as possible, dominated by Douglas-fir with no obvious signs of past damage, and minimal indication of root rot pockets. Plots were also located at least 64 m from streams and had at least a 32 m visual barrier from nearby roads to reduce the potential for vandalism. Plots ranged in size from 0.58 to 0.85 hectares. Each plot had a 20 m buffer around it to avoid any potential edge effects near stand boundaries. Initial mean tree characteristics for the stands were: diameter at 1.3 m (dbh) ranging between 14 and 28 cm, total tree height (ht) ranging between 12.4 and 20.0 m, and height to base of crown (hbc) ranging between 2.7 and 10.6 m. Reineke's (1933) stand density index (SDI) for the stands ranged from 126 to 300, and the number of trees per hectare ranged from 633 to 995.

Prior to the growing season of 2006, all trees 1.3 m or taller were measured for dbh, species were recorded, and any damage was noted as light (small healed over wound, or small fork at top) or heavy (broken top, unhealed wound or large old wound). These trees were then tagged and labeled with a unique number. Where 1.3 m along the bole corresponded to a branch whorl, the dbh measurement was offset above the whorl by 10 cm, and the diameter recorded along with height to that diameter. Between 87 and 93 trees with no damage or light damage were assigned to each treatment category (averaging 90 trees per treatment). A stratified random sampling algorithm, with stratification performed across dbh measurements, was used to make sure that treatments were applied to similar sizes within each stand and to make sure that treatments covered the range of tree sizes within the stands. These treatment trees were then measured for ht and hbc. Diameter measurements were to the nearest 1 mm using a metric d-tape, and height measurements were to the nearest 0.1 m using a height pole.

Damage Treatments

Sixteen damage treatments were applied to trees selected using the process described earlier. Treatments were coded by type of damage, damage intensity, and season, e.g., R25-B40-1S had a target root damage of 25 percent, and bole damage to 40 percent of the circumference along 0.9 m of the bole, both applied during spring. The treatments consisted of: control in which nothing was done; B20-1S, B40-1S, B60-1S, B80-1S, B90-1S, B100-1S in which 20 percent, 40 percent, etc. of the bole circumference was removed along 0.9 m of the bole in the spring; B40-1F, identical to the B40-1S treatment but applied in the fall; B40-2, B80-2, identical to the respective 0.9-m treatments, but damage was applied along 1.8 m of the bole instead of 0.9 m; and R25, R50, R25-B40-1S, R50-B40-1S. In addition to these treatments, there was a prune treatment in which branches were pruned up to 2 m, and a soil treatment in which the soil was removed around the roots and replaced. The damage treatments can be divided into three broad treatment groups for ease of comparison, 0.9-m bole treatments (called 1 m hereafter), 1.8-m bole treatments (called 2 m hereafter), and root-damage treatments. Each treatment group has a control,

Management and Policy Implications

Damage to young Douglas-fir stands can be a concern for forest management when the goal is to produce valuable timber products. Our study captured tree response to damage across a wide range of damage severities. On average, bole damage severity levels less than 60 percent resulted in little additional mortality when damage was contained to the lower 1 m of the bole. Longer bole wounds resulted in reductions in tree survival, and trees with larger relative diameters showed a greater sensitivity to bole damage than smaller, more suppressed trees. Trees with lower relative heights had lower baseline survival rates than trees with higher relative heights. Although average mortality is relatively low at bole damage levels of 60–80 percent, mortality is occurring in more valuable trees and therefore may be a matter of concern to forest managers. Root damage of less than 40 percent of the cross-sectional root area is unlikely to impact survival but may lead to minor reductions in growth. Damage to tree crowns will reduce growth, but may also reduce the sensitivity of trees to bole damage. Trees that survive damage are likely to show little reduction in growth within 10 years after the damage occurred.

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with the control serving that purpose for the 1-m group, prune for the 2-m group, and soil for the root group. These additional controls were to test if tree response was impacted by pruning or soil movement in addition to the damage applied.

Bole Damage

In the 1-m and 2-m treatment groups, wound width was determined as a percentage of bole circumference based on the treatment damage intensity (20 percent, 40 percent, etc.) at the midpoint of the wound. Bark was removed from the bole at the same width along the entire length of the wound (0.9 m or 1.8 m), starting at 15 cm above the groundline. After bark was removed, the underlying tissue was scarified to a depth of approximately 0.3 cm (height of saw tooth) using a hand saw to mimic the roughening typical of bole wounds. Bole damage as a percentage of circumference removed (d_b) was used as a primary fixed effect in modeling to determine the nature of tree-survival response to relative bole-damage intensity.

Root Damage

Root damage (d_r) treatments had soil removed around the base of the tree to expose roots. First, lateral roots near the stem were measured until the sum of cross-sectional root area was approximately equal to 25 percent or 50 percent of the total estimated root area. Once roots on a given tree were selected, the small roots (on small trees, first lateral roots were small) were severed with clippers, and large roots were girdled using a pocket chainsaw. Root area was assumed to be directly proportional to the cross-sectional area of the bole at ground level based on previous research on southern pines (Carlson and Harrington 1987). Later work by Gould and Harrington (2008) determined that the relationship between bole cross-sectional area at ground level and root area was nonlinear. This nonlinear relationship was used to recalculate the achieved damage severity based on the original target level of the treatment groups. The recalculated values revealed that the 25 percent root damage group had root damage that ranged from 15 percent to 23 percent, whereas the 50 percent root damage group had damage that ranged from 31 percent to 45 percent.

Crown Damage

Tree crowns experienced loss of crown height (top breakage) throughout the study, with the majority of damage occurring after an ice storm that took place prior to measurements taken in year 6 of the study. Crown-height loss was determined to occur when negative differences in in height measurements between measurement years were detected. No height losses were greater than the crown length in the previous years measurement, so the damage was assumed to be confined to the crown. Crown damage impacted a significant number of study trees with a range of average percentage crown height losses observed between treatments (Table 1). Because the extent and intensity of crown damage varied substantially among individual trees, this natural damage created a new study opportunity to integrate damage from boles, roots, and tree crowns. This was part of the impetus behind our choice of modeling approaches as nonlinear mixed-effects models that can easily incorporate the types of damage the trees experienced within the study stands, and deal with unbalanced datasets caused by trees dropping out of the analysis because of mortality in the case of the growth models. This approach also allowed for incorporation of covariates related to tree characteristics into the parameters of biologically interpretable models.

Each of the damage and control treatments was replicated 15 times within each plot following the stratified random sampling described above. All treatments were applied in the spring of 2006 with the exception of the fall treatment in the 1-m group, which was applied in the fall of 2006. Study tree dbh, ht, and hbc were remeasured prior to, or at the beginning of, the growing season in 2007, 2008, 2010, 2012, 2014, and 2016. A summary of tree characteristics by treatment group is provided in Table 1.

Survival Modeling

To determine whether bole damage had different impacts on trees with different relative sizes, trees were divided into stand level relative height quartiles and plotted over time by damage intensity. There appeared to be a change in slopes for larger trees beginning at

Table 1. Summary of tree characteristics by treatment group.

Treatment	dbh	ht	hbc	Circ. removed	Root damage	Trees with crown damage	Crown ht loss	Treatment group
	(cm)	…….. (m) ……..		…….. % ……..				
Control	20.7 (6.8)	15.4 (3.6)	6.5 (3.5)	0	0	31.1	3.2	0.9 m
B20-1S	20.7 (6.8)	15.6 (3.5)	6.5 (3.4)	20	0	23.3	1.9	
B40-1F	21.0 (6.9)	15.8 (3.6)	6.4 (3.6)	40	0	27.7	2.8	
B40-1S	20.8 (6.8)	15.8 (3.4)	6.8 (3.5)	40	0	29.2	3.1	
B60-1S	20.7 (6.8)	15.8 (3.6)	6.7 (3.5)	60	0	35.5	4.8	
B80-1S	20.7 (6.7)	15.6 (3.7)	6.6 (3.6)	80	0	26.9	2.3	
B90-1S	20.7 (6.9)	15.6 (3.6)	6.5 (3.5)	90	0	22.0	4.3	
B100-1S	20.8 (6.9)	15.9 (3.6)	6.8 (3.5)	100	0	3.3	6.4	
Prune	20.9 (6.9)	15.9 (3.8)	6.4 (3.5)	0	0	31.1	3.5	1.8 m
B40-2S	20.8 (6.8)	15.9 (3.4)	6.6 (3.5)	40	0	30.0	3.2	
B80-2S	21.0 (6.9)	15.7 (3.5)	6.5 (3.5)	80	0	23.0	2.1	
Soil	21.0 (6.9)	15.7 (3.4)	6.4 (3.4)	0	0	31.1	5.1	Root
R25	20.9 (6.8)	15.7 (3.6)	6.5 (3.2)	0	19.1	28.9	5.5	
R50	20.9 (6.9)	15.5 (3.6)	6.2 (3.3)	0	36.7	24.4	3.5	
R25-B40-1S	20.9 (6.9)	15.7 (3.7)	6.3 (3.3)	40	18.8	25.5	2.5	
R50-B40-1S	20.9 (6.9)	15.8 (3.3)	6.7 (3.4)	40	37.5	30.0	5.1	

Mean values for bole diameter at 1.3 m from the ground (dbh), tree height (ht), and height to the base of branch node with at least three live branches (hbc) are followed by standard deviations in parentheses. The mean percentage of initial trees in treatment with observed crown damage and the mean percentage crown ht length lost of crown damage trees by treatment are shown. Percentage root damage refers to the mean estimate of achieved damage based on recalculations from target damage. The sample size for the trees was 1,440.

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the 60-1S treatment level, so the data were divided into high-intensity (60–90 percent bole damage) and low-intensity (20–40 percent damage) groups. Each group was then fit to a mixed-effects linear regression with live tree count as the response variable, quartile (q), the interaction between time after treatment applied and q ($t \times q$) as fixed-effects, with random-effects at the stand-treatment (treatment groups within a stand) nested within stand level. Welch's t-test (Welch 1947) was used to test for significant differences between the parameters of the two regressions.

Cumulative tree survival can be effectively modeled using probability distribution functions (Buford and Hafley 1985). A modification of the Weibull cumulative probability model (Weibull 1961) of the form shown in Equation 1 was fit to cumulative survival proportion data at the stand-treatment level. Cumulative survival proportion was determined as the total number of trees alive within a given stand-treatment grouping at the beginning of a time period divided by the total number of trees in a stand-treatment grouping at the beginning of the study. This two-parameter model fit the data better than a two-parameter Gompertz distribution (Gompertz 1825) when treatment ID was used as a categorical fixed effect in both parameters.

$$y_{ij} = e^{-(p-\beta)\times t^{(p+\delta+\varphi_{ij})}} \tag{1}$$

where y_{ij} is equal to the proportion of trees surviving t years after treatment of stand-treatment i, within stand j, e is Euler's constant, p is the damage-severity-response model, and β and δ are fixed-effect parameter covariate matrixes (a $1 \times m$ matrix containing parameter estimates multiplied by corresponding covariates for a given number [m] of fixed-effects) containing best-fit covariates and their associated parameter estimates. φ_{ij} is a random parameter estimate for stand-treatment i, nested within stand j. $(\beta + p)$ controls the inflection point of the survival curve with consecutively lower values of $(\beta + p)$ resulting in greater levels of cumulative survival for a given time period, all else being equal. $(p + \delta + \varphi_{ij})$ controls the rate of decline in cumulative survival with higher values related to more rapid declines in survival curves.

To determine whether treatments had a significant impact on the parameters of the survival function, treatment categorical variables were assigned as fixed effects to each parameter, creating a parameter covariate matrix (a $1 \times n$ matrix containing parameter estimates multiplied by corresponding covariates for a given number [n] of covariates). These parameter covariate matrixes were used to fit Equation 1 to the data, with the damage-severity-response function set to 0. Random effects at the stand treatment nested within stand level were tested in each parameter of the model, and the final random-effects location was selected based on the lowest Bayesian information criterion (BIC) (Schwarz 1978) of the overall model. This led to random effects assigned to δ at the stand-treatment nested within stand level as shown in Equation 1. To determine the best model form for the damage-response function, linear, one- and two-parameter exponential, and power functions were tested using percentage bole damage as the primary covariate. Each damage-response model form was substituted in place of p in Equation 1, and then the entire model was fit to the cumulative survival proportion data using maximum log-likelihood regression methods so that meaningful comparisons between nested models and models with different functional forms could be made. Independent damage-response functional forms were tested in association with parameter covariate matrices β and δ, but the best fits were found when the same functional form was used in relation to both the β and δ parameter covariate matrices. The best damage-response functional form was determined using the lowest BIC value of the fitted models. Additional covariates that represented average stand-treatment level metrics were tested by adding them to the individual model parameters of Equation 1, including parameters of the best-fit damage-response function. The covariate testing process involved adding different covariates to each of the model parameters using the nonlinear mixed effects (NLME) modeling approaches described in Pinheiro and Bates (2000). A covariate was kept in the model when (1) it significantly increased the log-likelihood value of the model using log-likelihood ratio testing, and (2) it had an associated parameter estimate significantly different from zero. NLME modeling was performed using the NLME package (Pinheiro et al. 2007) in the R-statistical platform (R Development Core Team 2015). The final form of the NLME model was refit using reduced maximum log-likelihood methods, because this process results in unbiased parameter estimates. Individual tree metrics for the covariate data tested for this process were averaged at the treatment within stand level for consistency with the response data. These tree groups are refered to as stand-treatment groups in the remainder of the paper.

Growth Modeling

A Weibull function (Weibull 1961), Chapman–Richards function (Richards 1959), and Gompertz function (Gompertz 1825) were fit to the diameter and height data because these functions are suitable for modeling size measurements over time. Initial examination of the model fits revealed that the models had significantly lower BIC values when initial tree size measurements (dbh_i or ht_i) were added to the base functions named above, with time since treatment applied used instead of tree age. In each of the three models, there are parameters associated with an asymptote (α), an inflection point at which the curve changes from increasing to decreasing growth rates (β), and a growth rate for the modeled size variable (δ). Random effects at the tree-within-stand level were tested in each parameter of the models, and the best model was selected based on the lowest BIC. This approach screens for the model form that best fits the average of the data when accounting for stand- and tree-level variation.

Data used for diameter growth modeling were a subset of all data excluding observations on or after the observance of tree mortality. Data used for tree-height growth modeling were a subset of the diameter growth data that also excluded all observations on or after the observance of height damage. Initial size when treatment was applied for each tree was added to the growth function yielding a model that tracks growth after initial bole damage. Using the model fitting process described above, random-effects parameters were assigned at the individual tree nested within stand level in the α and δ parameter covariate matrixes and assigned to the α parameter covariate matrix in the height growth model as shown in Equations 2 and 3.

Additional covariates were tested in the parameters of the height and diameter models to account for factors that might alter growth and either moderate or enhance the effects of damage severity. New covariates were kept in the models if they met the following two

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criteria: the new covariate significantly increased the log-likelihood of the model, and it had an associated parameter estimate significantly different from 0. The significance of the increase in log-likelihood was determined through log-likelihood ratio tests. All NLME models were fit using the NLME package in R (Pinheiro et al. 2007), with parameter estimates derived from maximum log-likelihood methods during the model fitting process, and reduced maximum log-likelihood methods for parameter estimation of the final model. Maximum log-likelihood estimation allows for comparison of models with different covariates, whereas reduced maximum log-likelihood methods allow for less biased parameter estimates in the final model. The final forms selected for diameter and height modeling are shown in Equations 2 and 3.

$$\mathrm{dbh}_{ij} = (\alpha + \theta_{ij}) \times (1 - \mathrm{e}^{-\frac{t}{\beta}})^{(\delta + \vartheta_{ij})} + \mathrm{dbh}_{ij0} \tag{2}$$

$$\mathrm{ht}_{ij} = (\alpha + \theta_{ij}) \times (1 - \mathrm{e}^{-\frac{t}{\beta}}) + \mathrm{ht}_{ij0} \tag{3}$$

Equations 2 and 3 are forms of a shifted Chapman–Richards growth curve. The only difference between the two equations is that the rate parameter is set to 1 for the ht growth model. The subscripts *ij* after dbh and ht refer to measurements of tree *i* within stand *j*. The $(\alpha + \theta_{ij})$ component controls the upper asymptote, with larger values leading to greater estimated maximum heights or diameters. dbh_{ij0} and ht_{ij0} are initial dbh and ht measurements. The β component controls the inflection point of the curves, with larger values leading to a curve shifted further to the right of the origin. θ_{ij} and ϑ_{ij} represent nested random effects at the tree within the stand level associated with the specified parameter covariate matrixes.

Results

Survival

Cumulative survival at the end of the study ranged from 0 percent in the B100-1S treatment to 99 percent in the B40-1F treatment (Table 2—B100-1S not shown). Survival declined over time for all treatments, with high survival in year 1 and greater declines in survival with increasing damage severity over time (Figure 1, Supplemental Figure 1). Differences between the treatment trend lines become negligible at low levels of damage severity. In general, the survival curves begin to separate more above 60 percent bole damage, and with increased damage lengths with the B40-2S group demonstrating lower survival than the B40-1S group. This relationship between survival and damage severity shows a nonlinear survival response to bole damage. This is also evident in the cumulative survival data shown in Table 2, where there is a noticeable drop in mean survival beginning above 60 percent bole damage.

Regarding survival within treatments by height quartiles, the overall mean survival does not represent survival in all size categories (Figure 2, Supplemental Figure 2). The bottom quartile corresponds to trees that fall into the lowest 25 percent of tree heights at the beginning of the study, the second quartile corresponds to trees that fall into the next 25 percent of tree heights, and so on. The trends shown in Figure 2 indicate that smaller trees are more likely to decline in number than larger trees when bole damage severity is lower than 60 percent. In the 60–90 percent damage groups, larger declines in the higher quartile trees related to increasing damage severity are noticeable, compared with the lowest quartile. Supplemental Figure 2 shows survival by quartile for all treatments. These visual trends are validated by the significant differences found between parameter estimates of the mixed-effects linear models (Supplemental Table 1), demonstrating that the slope of the smallest quartile experienced less change than the slopes of all larger quartiles.

Table 2. Summary of treatment metrics in year 10.

Treatment	dbh	ht	hbc	Cumulative survival
	(cm)	…….. (m)……..		
Control	27.8 (4.5)	24.1 (2.9)	13.6 (2.8)	0.94 (0.04)
B20-1S	27.8 (4.3)	24.3 (3.2)	13.8 (3.0)	0.92 (0.04)
B40-1F	27.9 (4.6)	24.0 (2.7)	13.9 (2.8)	0.99 (0.02)
B40-1S	27.4 (4.3)	24.1 (2.4)	13.9 (2.8)	0.93 (0.07)
B40-2S	–	24.4 (2.8)	13.8 (2.8)	0.87 (0.08)
B60-1S	28.0 (4.6)	23.6 (2.6)	13.6 (2.8)	0.89 (0.04)
B80-1S	28.3 (4.6)	23.6 (3.1)	13.6 (3.0)	0.83 (0.03)
B80-2S	–	22.9 (3.3)	13.5 (3.0)	0.84 (0.10)
B90-1S	26.9 (6.0)	22.4 (3.8)	13.4 (3.2)	0.68 (0.10)
Prune	28.1 (4.9)	24.6 (3.2)	13.9 (2.9)	0.89 (0.06)
R25	28.1 (4.7)	24.5 (2.9)	13.8 (2.9)	0.92 (0.06)
R25-B40-1S	28.0 (5.1)	24.0 (3.3)	13.7 (2.9)	0.93 (0.06)
R50	27.6 (5.2)	23.8 (3.2)	13.7 (3.0)	0.88 (0.06)
R50-B40-1S	28.0 (5.0)	23.9 (2.9)	13.8 (3.0)	0.91 (0.06)
SOIL	28.0 (4.8)	24.4 (3.0)	13.8 (2.9)	0.89 (0.06)

Mean values followed by standard deviation in parentheses are shown for treatments. dbh is the diameter at 1.3 m, ht is the total height of the tree, hbc is the height to the base of the live crown, and cumulative survival is the proportion of trees that survived to the end of the study period divided by the number of trees in the treatment at the time the treatment was applied.

Survival Model

The best-fit damage-response functional form was an exponential model of the form shown in Equation 4. The model functional forms are compared in Table 3. Equation 4 predicts 1 when bole damage equals 0, and e^{γ} when bole damage equals 1. The best-fit functional form before fitting additional covariates (Model 8) performed better than a comparable model with treatments as categorical variables (Model 7). Given that treatment accounts for variation between treatment groups in addition to damage level, it is somewhat surprising that the damage-response model fits the data better than a model that is fit to each treatment. Model 7 estimated significantly lower survival rates for the B80-1S, B90-1S, B100-1S, B40-2S, and B80-2S treatments than the control treatment, with no significant differences for any other category. The final best-fit parameters and covariates for Model 9 are listed in Table 4. These results suggest a complex relationship between tree survival and bole damage that is best captured by nonlinear models.

$$p = \mathrm{e}^{\gamma \times d_{\mathrm{b}}^{\omega}} \tag{4}$$

Predicted cumulative survival curves (solid lines) were generally within the 95 percent confidence intervals (mean ± 1.96 × SE) of the data (shaded region) (Supplemental Figure 1) indicating that the model is tracking the trends in the data well given the stochastic nature of tree mortality. Model predictions were derived using the covariate parameter matrix estimates shown in Table 4, within Equation 1.

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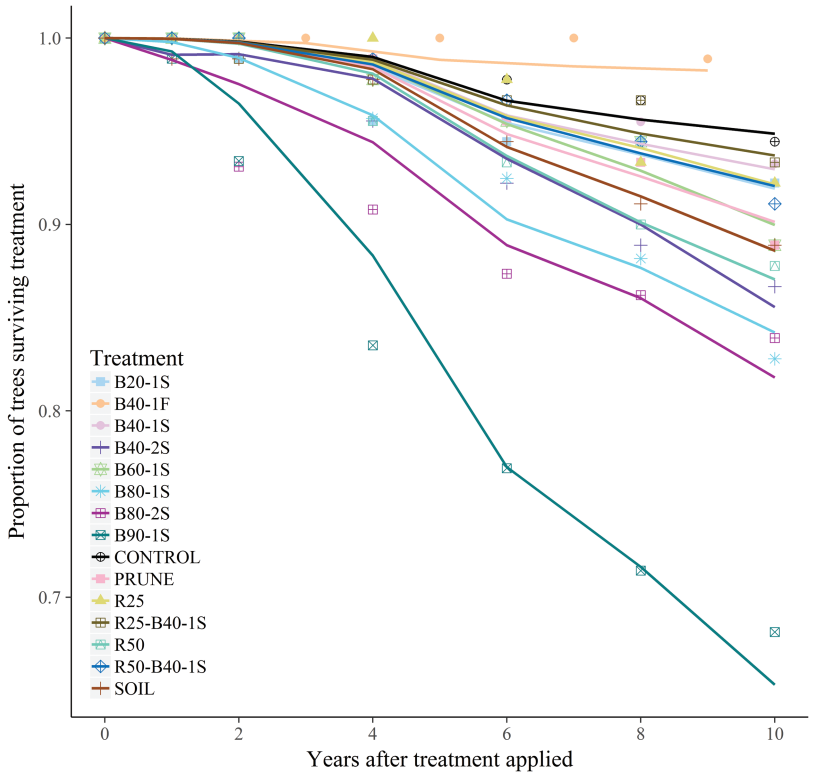

Figure 1. Survival curves for the treatment means of the observed data over time. The lines show the mean predicted survival by treatment, and the points show the mean observed survival by treatment. Treatment ID is signified by color and point shape, both of which are indicated in the legend of each panel in Figure 1. The B100-1S treatment was removed to allow for a zoomed in view of the remaining treatments. All treatments are shown in Supplemental Figure 1.

The positive correlation between the relative height (rht) covariate and the inflection point (β) (Table 4) indicates that groups with larger rht values had higher baseline cumulative survival proportions over the study period than groups with lower rht values. Taller trees would be expected to be less impacted by competition than smaller more suppressed trees leading to a higher baseline survival in the absence of damage.

Positive correlations between covariates and the growth-rate parameter (δ) indicate a more rapid decline in survival over time. The positive intercept value for this parameter means that survival rates should be expected to continue to decline over time.

Positive parameter estimates in the γ parameter covariate matrix increase the rate of decline in survival and also lead to earlier declines in survival for a given level of damage. The negative parameter estimate for cumulative percent height loss (cptl) indicates that tree groups with a higher cptl showed more gradual rates of decline beginning at a later time points than trees with a lower cptl. The positive parameter estimate for initial tree diameter relative to stand quadratic mean diameter (rdi) suggests that on average, trees with a higher rdi had higher rates of decline beginning at lower damage-severity levels than trees with a lower rdi.

Positive covariate parameter estimates in the ω parameter covariate matrix reduce the sensitivity of damage response values to bole damage severity. This positive correlation is associated with declines in cumulative survival occurring later in time, and at a more gradual rate. The sum of crown cross-sectional area above 66 percent of subject tree height (cc_{66}) and wound length (wl) were positively correlated with ω. This indicates that stand-treatment groups with higher levels of crown competition had a lower sensitivity to bole damage than trees with lower levels of crown competition. The φ parameter standard deviations at the stand and stand treatment levels suggest minor residual variation related to those grouping categories on the overall survival function.

Diameter Growth

There are noticeable downward trends in the diameter data (dashed lines) in the B100-1S and B90-1S treatments with little divergence in the remaining treatments (Supplemental Figure 3). In general, if the treatment started with a higher average diameter, it ended up with a higher average diameter relative to other treatments (Tables 1 and 2). The pattern of growth shown in the B100-1S treatment (Supplemental Figure 3) is due to a transition from a high proportion of small trees dying initially (upward trend in average diameter), followed by larger trees dying later (downward trend in diameter), indicating that the intermediate-sized trees may have survived longer. The decline in the B90-1S treatment (Supplemental Figure 3) is due to the greater proportion of large trees dying later in the study period (Figure 2) relative to other treatments. Similar slowdowns in growth are shown in the B60-1S and B80-1S treatments (Supplemental Figure 3) with those treatments also showing increased mortality occurring in larger trees later in the study period (Figure 2). The remaining treatments show little difference in growth patterns over the study period. Predictions for Supplemental Figure 3 were derived using Equation 2 with parameter estimates from Table 5. There does not appear to be a significant influence of treatment on final mean diameter by the end of the study period (Table 2); however,

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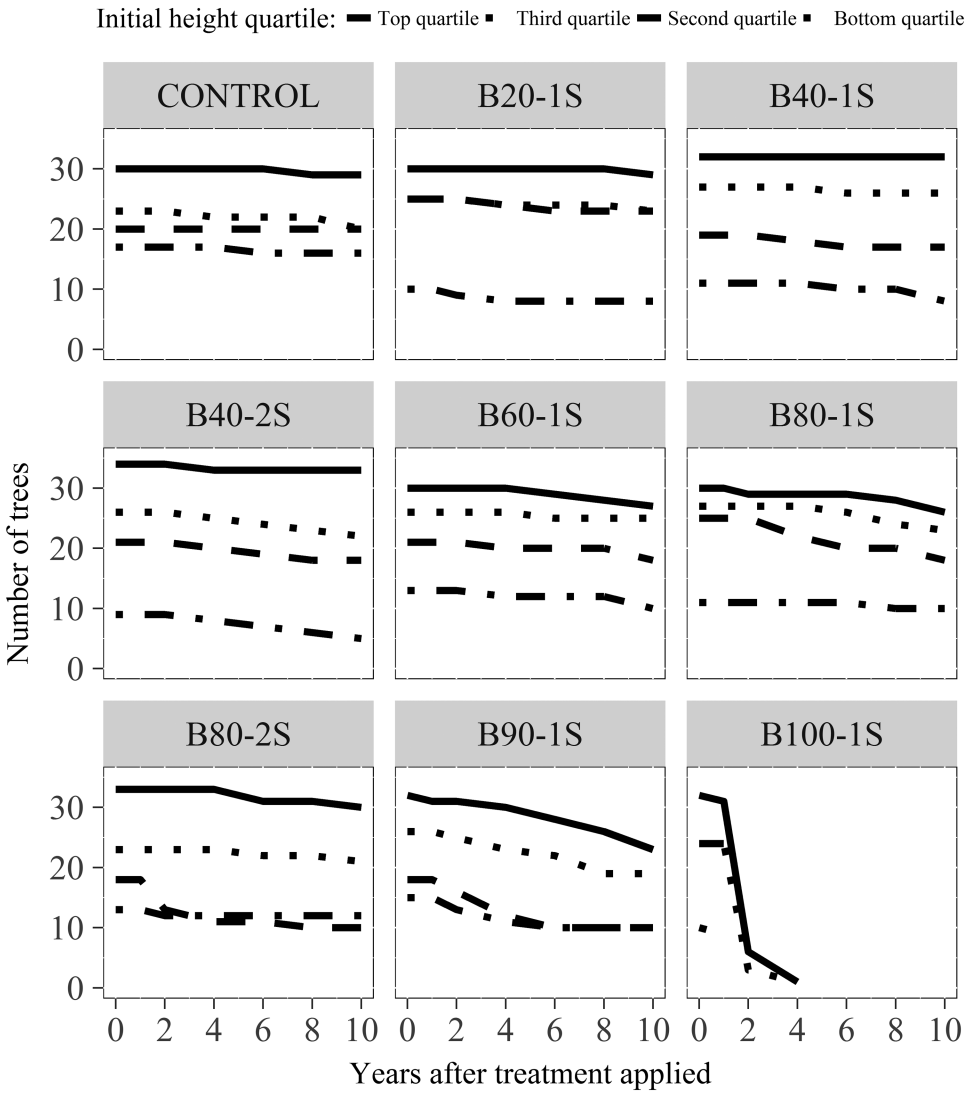

Figure 2. Number of trees in each initial height quartile for each treatment over time. Line types were assigned to the separate quartiles. The bottom quartile contains trees that are between 0 and 25 percent of tree heights, the second quartile contains trees between 25.1 and 50 percent of tree heights, the third quartile contains trees that are between 50.1 and 75 percent of tree heights, and the top quartile contains all trees between 75.1 and 100 percent of tree heights.

as shown in Figure 2, there is considerable variation in survival between height quartiles indicating that the differences in treatment mean diameters are likely driven in part by losses of different size classes within a treatment. This makes comparing mean diameter values problematic.

Diameter Growth Rates

Diameter growth rates by the end of the study were lower for bole damage severity levels above 40 percent (Supplemental Figure 4), with more noticeable drops in growth rates above 60 percent. Treatments with bole damage severity at or below 40 percent were indistinguishable from each other with the exception of the B40-1F treatment which had higher growth rates for the duration of the study period than all other treatments. There is strong agreement between predicted diameter growth rates and measured diameter growth rates indicating that the model is tracking observed changes in growth rates closely. There are transitions in relative ranking among the treatment groups, with the prune treatment in the top panel starting off with lower diameter increment, but eventually ending with higher values than the other groups in the panel. The opposite pattern is seen in the B80-1S group as it starts off with the highest diameter increment and ends lower than the B20-1S treatment, whereas the B60-1S treatment starts lower than B80-1S but ends at the same diameter increment. The B90-1S and B100-1S both start out near the middle in terms of diameter increment but end lower than all others. This ranking is in line with the results we would expect based on the model parameter estimates (Table 5). As with diameter growth, it should be noted that diameter growth rates are also influenced by the variation in the survival of different size classes within the treatments shown in Figure 2. This makes comparing mean diameter growth rate values problematic.

Diameter Growth Model

Positive parameters in the α covariate parameter matrix increase the upper asymptote of the diameter growth curve. The positive parameter values for the percent bole circumference remaining

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Table 3. Comparison of cumulative survival models with different damage-response functions.

Model no.	Functional form	AICc	BIC
1	Null	–247.8	–229.9
2	d_b^{ω}	743.3	770.2
3	$\gamma \times d_b^{\omega}$	726.6	753.5
4	$\gamma \times d_b^{\omega}$	557.3	588.7
5	$e^{d_b^{\omega}}$	–1,164.5	–1,137.6
6	$e^{\gamma \times d_b}$	–1,690.1	–1,663.2
7*	T_{id}	–1,793.2	–1,703.2
8	$e^{\gamma \times d_b^{\omega}}$	–2,051.5	–2,020.0
9†	$e^{\gamma \times d_b^{\omega}}$	–2,572.6	–2,497.0

Functional forms tested for damage response function (Equation 4) within Equation 1. AICc is the corrected AIC for models fit to cumulative survival at the stand-treatment level. The percentage of bole circumference removed is d_b, and γ and ω are fitted parameters. e is Euler's constant.

* Model 7 had treatment ID assigned to the β and δ parameter covariate matrixes shown in Equation 1, with the damage response (p) set to 0.

† Model 9, the best fit model, had additional covariates tested, and these parameter estimates are listed in Table 4.

Table 4. Parameter estimates and associated covariates of Equations 1 and 2.

Covariate matrix	Covariate	Covariate mean parameter estimate	Parameter SE or SD	Parameter *P*-value
β	int.	0.7738	0.0167	<.001
β	rht	0.2081	0.0179	.0042
δ	int.	2.3467	0.1567	<.001
γ	cptl	–2.2376	0.4929	.0351
γ	rdi	0.4302	0.0138	<.001
ω	int.	–6.5296	2.7082	<.001
ω	cc_{66}	37.5457	3.2073	<.001
ω	wl	–4.2558	1.0284	.006
φ_i	Stand-treatment	0	0.0148*	NA
φ_j	Stand	0	0.0014*	NA

int. is an intercept value for a given covariate matrix, rht is stand-treatment level average tree height compared with the stand mean height, cptl is the stand-treatment level average sum of percent tree height loss over the course of the study, wl is the treatment average length of the tree wound in meters, cc_{66} is the stand-treatment level average sum of cross-sectional crown area for competitor trees at 66 percent of subject trees height, and rdi is the stand-treatment level average initial relative diameter at the time the treatments were applied. Stand and Stand-treatment refer to the respective levels of the random parameters with means of 0.

* SD is shown instead of SE.

($1 - d_b$), and initial crown cross-sectional area multiplied by initial live crown ratio ($csa_i \times lcr_i$) covariates suggest greater potential diameters as those values increase, whereas the negative parameter values for the cptl and root damage (d_r) covariates lead to reductions in potential diameter values. This means that increases in crown and root damage lead to reductions in overall diameter growth potential, whereas trees with high live crown ratios and large crown cross-sectional areas have a higher potential diameter growth than trees with lower lcr_i and csa_i.

Table 5. Covariate parameter matrix estimates for the diameter growth model in Equation 3.

Covariate parameter matrix	Covariate	Covariate parameter estimate	Covariate parameter SE	*P*-value
α	$1 - d_b$	2.5025	0.2664	<.001
α	cptl	–9.0882	0.9421	<.001
α	d_r	–0.8904	0.3910	.0228
α	$csa_i \times lcr_i$	0.0828	0.0024	<.001
β	int.	7.2698	0.3748	<.001
β	d_{sa}	–24.2781	1.4949	<.001
β	cptl	–7.5200	0.4279	<.001
β	cl_i	0.1576	0.0355	<.001
δ	int.	1.2863	0.0284	<.001
δ	cc_{66i}	–0.0838	0.0396	.0345
δ	tt	0.0398	0.0175	.0226
θ_j	Stand	0.0000	0.6482	na
θ_i	Tree	0.0000	2.7917	na
ϑ_j	Stand	0.0000	0.0292	na
ϑ_i	Tree	0.0000	0.1649	na

$1 - d_b$ is the percentage bole circumference remaining after treatment, cptl is the cumulative percent height lost by the tree over the study period, d_r is the percentage damage severity to roots, csa_i is the initial crown cross-sectional area at the base of live crown for a tree, lcr_i is the initial live crown ratio for the tree, int. is an intercept value, d_{sa} is the average percentage tree surface area removed, cl_i is the initial crown length, tt is a value equal to 1 for the B40-1F treatment and 0 for all other treatments, and Stand and Tree refer to the level for a particular random effect, with tree nested within stand.

Positive parameters for the β covariate parameter matrix lead to later inflection points in the growth curve, resulting in later transitions to declining growth rates. The positive parameter estimate for the initial crown length (cl_i) covariate indicates longer periods of increasing growth rates for trees with longer initial crown lengths. The negative parameter estimates for bole area damage as a proportion of tree bole surface area (d_{sa}) and cptl covariates indicate that increases in these two characteristics result in earlier transitions to declining growth rates all else being equal. This suggests that damage to the bole or crown of a tree hastens the point in time at which tree growth rates decline.

Positive parameters for the δ covariates parameter matrix lead to lower predicted growth rates with increasing values of the given covariate. The negative parameter estimate for the cc_{66i} covariate indicates a more rapid rise to a final diameter in trees with greater competition than trees with less competition. The positive parameter value for the treatment timing (tt) covariate suggests that the fall treatment has a more gradual rise in growth rates than all other treatments.

The θ and ϑ parameter estimates indicate that there is significant variation at the stand and tree levels that is not fully explained by the other covariates in the model. Given the performance of the model shown in Supplemental Figure 3, no further modification of the model was deemed necessary to account for the remaining variation.

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Height Growth

As with diameter growth, there does not appear to be much difference between mean treatment values for height (Supplemental Figure 5) for treatments with 40 percent bole damage or less. Average height values based on the last recorded observations were lowest for the B100-1S treatment, followed by the B90-1S, then the B80-2S, and finally the B80-1S and B60-1S treatments. The initial heights were highest in the B40-1S and B40-1F treatments followed by the B20-1S and control treatments. There does appear to be a downward trend in average heights in year 10 as bole damage severity is increased above a bole damage level of 40 percent. This reduction in height may partially be due to the variation in survival shown by size class in Figure 2. Overall, the predicted values tracked the observed values quite well (Supplemental Figure 5), indicating that the model is tracking the observed changes in height accurately.

Height Growth Rates

In contrast to the analysis of diameter growth rate data, there are noticeable areas where the mean prediction lines for height growth rates are not overlapped by the 95 percent confidence intervals of the data (Supplemental Figure 6). The wave pattern displayed by the height growth increment data suggests a possible environmental component to height growth separate from the impact of the bole damage. The beginning values and ending values of the data and predictions are in agreement with each other. This wave pattern could have been modeled, but given the overall agreement with the height-over-time data, a further improvement in height growth rate prediction was not deemed necessary. Unlike with the diameter increment data, there are no clear transitions in relative ranking related to damage severity shown by the data (Supplemental Figure 6). Though there are periods where high damage treatments have a significantly lower height increment than treatments with lower damage, e.g., B90-1S vs. B60-1S top panel, and B80-2S vs. B40-2S (Supplemental Figure 6), overall the height increment values start to even out between treatments by the end of the study.

Height Growth Model

Bole damage (d_b), and cc_{66} were both negatively correlated with maximum estimated tree height (α) within the study period, whereas the crown cross-sectional area divided by cc_{66} ($ccsa_r$) covariate was positively correlated to maximum estimated tree heights (Table 6). This means that increasing damage severity reduces maximum height potential, whereas trees with larger cross-sectional crown areas at the base of the live crown will have a higher potential height growth.

The positive parameter estimates for sdi_r and cl_i covariates within β indicate a later transition to declining height growth rates for trees with longer live crowns that are in stands with a higher stand density. The negative relationship with $1 - d_b$ and $1 - d_r$ suggests that trees with low levels of damage will typically transition to declining growth rates earlier. This earlier transition does not necessarily mean that total growth will be lower.

The estimates for the θ_{ij} parameters indicate that there is significant variation at the stand and tree level that is not explained by the model; however, this level of unexplained variation is acceptable for our purposes.

Table 6. Covariate parameter matrix estimates for the height growth model in Equation 4.

Covariate parameter matrix	Covariate	Covariate parameter estimate	Covariate parameter SE	*P*-value
α	int.	24.157	1.157	<.001
α	d_b	–1.433	0.439	<.001
α	cc_{66}	–14.568	0.511	<.001
α	$ccsa_r$	15.742	2.679	<.001
β	sdi_r	22.162	0.799	<.001
β	cl_i	0.206	0.043	<.001
β	$1 - d_r$	–2.821	0.383	<.001
β	$1 - d_b$	–2.216	0.482	<.001
θ_j	Stand	0.000	2.633*	na
θ_i	Tree	0.000	2.127*	na

d_b is the percentage bole circumference removed during treatment, cc_{66} is the sum of cross-sectional crown areas in m^2 of all trees in the stand at 66 percent of a subject tree's height, $ccsa_r$ is the cross-sectional crown area at the crown base of each subject tree (m^2) in the stand divided by cc_{66}. sdi_r is the relative stand density index, cl_i is the initial tree crown length, $1 - d_r$ is the percentage of target root area remaining after treatment, and $1 - d_b$ is the percentage bole circumference remaining after treatment. Stand and Tree refer to random effect estimates at those respective levels, with tree nested within stand.

Discussion

Survival

Tree mortality is a complex process that can result from high levels of competition, severe injury, infection by pathogens, or, more commonly, a combination of factors (Franklin et al. 1987). The mechanisms involved in the decline and eventual death of trees exposed to damage are not well understood, but reductions in available photosynthates for growth and respiration are likely drivers (Waring 1987). Crown competition reduces photosynthesis of a tree through shading and has been shown to be a driver of mortality (Temesgen and Mitchell 2005, Bravo-Oviedo et al. 2006). Severe injury may trigger reallocation of carbon to wound closure instead of other processes. Infection by pathogens can cause reductions in water transport to the crown, increasing the probability of mortality (Christiansen and Solheim 1990). Root damage can cause a reduction in nutrients and water transported to the crown, leading to reductions in growth and increases in mortality (Vasiliauskas 2001). These processes reduce the availability of photosynthates for growth and respiration, and, if that reduction is severe enough, could reasonably lead to tree mortality.

Survival Response to Bole Damage

The significantly better fit of the exponential model form (Model 8) than a treatment ID only model (Model 7 in Table 3) demonstrates the benefit of modeling the relationship between bole damage and tree survival with functional forms that track the shape of survival response over time. This approach captures survival trends, which is helpful in understanding *when* trees are likely to die and not just *if* they will die. Predicting the timing of mortality could be important in determining which trees should be thinned after damage and which should be left to grow. Building this type of model and incorporating competition-related covariates gives an insight into not only how much mortality to expect for a given amount of bole, or crown damage, but also how mortality might change based on relative competitive status.

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The exponential relationship between percentage bole damage and survival could be due to exposure of the xylem to the air reducing water transport within the sapwood to some unknown depth. It is possible that the relatively small response to low levels of damage is due to an increase in sap flow through the remaining sapwood area, as this mechanism has been noted in *Quercus serrata* (Chandrathilake et al. 2016). That study also showed a significantly greater response to damage above a bole damage level of 40 percent than what was predicted with a linear model of sapwood damage. This may suggest that trees can compensate for the loss of sapwood cross-sectional area up to a critical point, after which exponential declines in sap flow could occur.

Our findings suggest that future studies on tree-survival response to bole damage focus on accounting for the form of the response function rather than trying to determine a critical value of damage, above which survival is significantly lower. Knowing the functional form of the survival relationship to damage would allow forest managers to better estimate the magnitude of expected declines in survival caused by a given level of bole or crown damage.

Cavitation can be induced in otherwise functioning water-transport cells via introduction of air in surrounding cells (air seeding) (Sperry and Tyree 1988). This means that the cross-sectional area of sapwood disturbed by cavitation is likely related to the depth within the xylem that air can penetrate. This phenomenon has been noted in Douglas-fir (Kiser 2011). With the xylem exposed, the process most likely responsible for limiting the penetration of air deeper into the xylem is compartmentalization (Shigo et al. 1977). Compartmentalization may be influenced by timing of wounds and location of wounds (Armstrong et al. 1981). There is some evidence that trees wounded later in the year (closer to fall) may have larger discolored regions, indicating the potential for a more robust tree response to damage (Leben 1985, Dujesiefken et al. 2005). If this larger response to fall damage reduced the amount of air seeding, this might explain the somewhat greater survival of trees in the B40-1F treatment.

The relationship between xylem exposure to air and cavitation could also explain the negative relationship between survival and wound length found in our analysis. Douglas-fir has been observed to have a spiral water transport system within its sapwood (Vité and Rudinsky 1959). A spiral pattern of tracheids moving up the tree bole would increase the effective region of cross-sectional sapwood area exposed to the air with increasing wound length. Thus, longer wound lengths would be expected to reduce tree survival because they result in cutting off proportionally greater amounts of water flow to the tree crown. A reduction in the amount of water to the tree crown would result in a reduction in photosynthetic potential as well. This reduction in photosynthesis would result in correspondingly reduced resources for tree respiration, growth, defense, and reproduction. If available photosynthates are reduced substantially, we would expect the probability of tree mortality to increase (Waring 1987).

The reduction in transport of photosynthates to the root system through the cambium likely increases the probability of mortality. The lack of significant impact on the cumulative survival model from root cross-sectional area damage suggests that carbon transport to the root system may not be a critical driver of mortality in this study, though that may also be a function of a smaller range of root damage than bole damage and that the achieved root damage severity was less than the targeted amount. A more formal examination of nutrient flow and water flow in damaged trees would be necessary to determine the mechanisms driving their response to damage.

The changes in survival patterns shown by size class in Figure 2 with increasing levels of bole damage indicate the complexity of tree survival response to damage. The relatively high mortality in small trees at damage severity levels at or below 40 percent is to be expected because small trees are more likely to be under higher levels of competition than larger trees. What is most interesting is that as damage severity increases above 40 percent, there appears to be a transition in survival dynamics with smaller trees surviving at similar rates as they do with lower levels of damage, whereas larger trees appear to be declining at greater rates than lower levels of damage. This is an important point because land managers growing trees for timber care most about the large trees with a higher timber value surviving to harvest. Looking only at the mean survival values for treatments would suggest that few differences exist between treatments below 80 percent bole damage; however, Figure 2 clearly shows that a proportion of the mortality that is occurring in higher levels of bole damage (above 40 percent) comes from some of the larger trees. This finding is reflected in the survival model by the significant parameter estimates for covariates related to stand-treatment level average tree characteristics such as rdi. In fact, the impact of the rdi covariate in the survival model is to increase the damage response value at lower levels of damage, indicating a greater sensitivity to bole damage in the larger trees within a stand. This result makes sense if air seeding and cavitation are the primary drivers of damage-induced mortality because larger trees are more likely to have their crowns exposed to the sun and therefore more likely to have a greater negative pressure applied to the water column. Xylem cavitation can occur in trees if a high enough pressure gradient is applied to the sapwood water column (Sperry and Tyree 1988), so to some extent a greater sensitivity to wounding should be expected in the most dominant trees within a stand. This finding makes a strong argument for analyzing tree responses to damage across gradients of stand conditions and tree characteristics. After all, damage is not likely to be consistent across tree sizes, so understanding how that damage will impact trees of different sizes and within different growth environments is important for understanding survival dynamics.

Survival Response to Tree Crown Damage

Stand-treatment level cumulative percent crown loss (cptl) appears to reduce the sensitivity of trees to bole damage (Table 4). One possible explanation for this could be that tree crown damage may reduce leaf area with high evapotranspiration rates, thereby reducing pressure on the water column of the damaged trees. The foliage at the top of the canopy is typically exposed to greater amounts of sunlight, and higher average wind speeds leading to higher evapotranspiration rates per unit leaf area (Bennett et al. 2015). Reduction in this foliage would therefore be expected to reduce the negative pressure applied to the water column, thereby reducing the potential for xylem cavitation, and increasing the probability of tree survival.

Diameter Growth Response

Damage has been shown to reduce diameter growth in Douglas-fir trees (Hann and Hanus 2002a). Our findings also demonstrate reductions in diameter growth of Douglas-fir trees related to damage. A key difference in our approach is that we were able to account

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for damage severity as a continuous variable rather than treating it as a broad damage category. In addition, we were able to test tree response to damage severity in terms of its influence on asymptotic size, inflection point of growth, and growth rate, rather than only growth rate. The quality of fit of our model is apparent by the coinciding data trend lines between the model predictions and the data shown in Supplemental Figure 3. The goodness of fit is due primarily to the fact that tree-diameter growth is very well estimated with the form of growth model used, but this also suggests that the modeled effects of damage are accurate reflections of the observed tree responses in the data. Two separate studies on Douglas-fir trees found no significant impacts on diameter growth from low levels of bole damage (Shea 1967, Kiser et al. 2017), which is in agreement with the minor differences our model would predict for low levels of damage. It should be noted that the model results are not impacted by the loss of trees from the analysis because no data were used from trees that were dead. The same cannot be said for the overall trends in the size graphs because the loss of different height classes would clearly impact the overall treatment mean.

The relationship between diameter growth and damage indicates that the asymptotic value for diameter in this study is directly proportional to bole damage as a percent of circumference, and root damage as a percentage of cross-sectional root area (Table 5). A possible cause of this could be a reduction in photosynthates to the root system via reductions in phloem cross-sectional area. Reductions in phloem cross-sectional area would be expected to reduce the potential growth of fine roots in years following damage if the remaining phloem cross-sectional area is not capable of increased flow rates. These results also suggest that unlike with tree mortality, potential tree growth does not display an exponential decline with increasing damage severity. The implications of this are that trees likely shift resources to maintain living cells at the expense of growth, thereby keeping the tree alive until resources reach a critical limit leading to mortality. The lack of difference shown between damage treatments in Table 2 is not a contradiction of this finding. The model is based on individual tree characteristics rather than broad categories and is therefore more applicable to individual tree responses rather than the categorical responses shown in Table 2.

Damage to tree crowns caused trees to transition more quickly to lower growth rates than trees with intact crowns (Table 5). This relationship is reduced with increasing live crown lengths indicating that higher initial photosynthetic capacity can partially offset the impacts of percent crown loss within Douglas-fir trees. Combined with the impacts on tree maximum size, this means that tree crown loss results in earlier reductions in growth rates and smaller final diameters than undamaged trees. The earlier transition to lower growth rates caused by increasing relative damage to bole wound area (d_{sa}) may indicate a more persistent interruption in photosynthesis related to cutting off water to the crown that is not overcome with any additional growth in sapwood that may occur after injury (Kiser 2011).

Diameter growth rates were not impacted by any type of damage; instead, competition appears to play a greater role in reducing diameter growth rates (Table 5). The combined impacts of reduced maximum size and earlier inflection points indicate that damage may have a more long-term impact on tree growth, even if average relative growth rates are not impacted. The fact that growth rates are not significantly different between damaged and undamaged trees in the study does not suggest that they will eventually reach the same size; rather, damaged trees reach their maximum size earlier than undamaged trees within the study period.

Height-Growth Response

Tree height growth is generally understood to be a higher priority for carbon allocation than diameter growth in trees experiencing competition for light (King 1990). This means that we would expect height growth to be less impacted by damage than diameter growth. The height-growth model had no significant improvement in model performance with the growth-rate parameter added (parameter δ was not included in the model), which is to be expected given the similarity of growth rates shown in Supplemental Figure 6. This suggests that height growth rate was likely driven by factors such as site index that did not differ much between treatments. There was a significant negative relationship between damage severity and both the asymptotic size and inflection point of the height-growth curve over time (Table 6).

As with diameter growth, the asymptote for height was linearly reduced with increasing levels of bole damage and crown competition. Trees that had more crown cross-sectional area than their competitors showed increased asymptotic size potential. The reduction in height potential because of bole damage could be explained by a loss of nutrient flow to the tree roots proportional to the reduction in cross-sectional phloem area caused by bole damage. Reductions in nutrient flow to the roots would likely reduce the growth of fine roots the following year, thus potentially reducing resources available for growth. A reduction in fine root growth could limit the amount of leaf area a tree could produce the following year, thereby limiting the photosynthetic potential of the tree. Less photosynthetic area could result in less photosynthate available for height growth. The influence of damage on height growth appears to be less pronounced (Supplemental Figure 5) than it is in the diameter data (Supplemental Figure 3), which would follow the concept of height growth being a higher priority for carbon allocation than diameter growth in trees experiencing competition for light (King 1990), and thus less sensitive to reductions in photosynthetic potential.

The inflection point parameter in the height-growth model (β) demonstrates a positive relationship with bole and root damage (Table 6). This is the opposite of the relationship shown in the diameter model. This indicates that height growth rate is increasing for a longer period of time than diameter growth rate at the same damage-severity level. Taken together with the reduction in asymptotic height, damaged trees have a lower maximum height potential and take longer to reach that height. Diameter growth demonstrated a different growth pattern, with reduced asymptotic diameters being reached earlier. This would imply that diameter growth rates on damaged trees may initially outpace height growth rates but that height growth rates will eventually be greater than diameter growth rates. Height–diameter ratios in these damaged trees would then be expected to increase, potentially leading to trees that are more susceptible to damage from stem breakage because of high height–diameter ratios (Wonn and O'Hara 2001). Alterations in height–diameter ratios could also impact the accuracy of diameter-based volume estimates because these models do not typically account for variable height–diameter relationships.

Conclusion

Our study found that tree survival was exponentially reduced with increasing bole damage severity over a 10-year time frame. Trees with larger relative diameters demonstrated accelerated

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mortality rates above bole damage severity levels of 60 percent. Average tree crown height loss reduced tree sensitivity to bole damage. Increasing bole, root, and tree crown damage severity led to reductions in estimated maximum diameter and height. Bole damage and damage to tree crowns hastened the decline of diameter growth rates and delayed the decline of height growth rates.

Supplementary Materials

Supplementary data are available at *Forest Science* online.

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