Does it Make the Cut? Evaluating the Efficacy of Pruning to Mitigate White Pine Blister Rust Infection in Sugar Pine

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ABSTRACT

White pine blister rust, a potentially lethal disease caused by the invasive pathogen *Cronartium ribicola*, threatens the survival of sugar pine populations in California's Sierra Nevada range, and common mitigation measures possess substantial drawbacks. Pruning has emerged as a promising tactic for reducing infection-caused mortality, but the relative impacts of single-stage ("first-lift") and multiple-stage ("second-lift") treatments have not been widely studied. My project investigated the effects of first- and second-lift pruning on the development of blister rust. I assessed previously studied pruned and unpruned sugar pines at Blodgett Forest Research Station to quantify mortality, infection rate, and infection severity. I also examined the ramifications of pruning treatments on relative volumetric growth. Annual mortality for the 2006-2016 period was greatest for first-lift trees (1.95%), followed by second-lift trees (1.29%) and unpruned trees (1.12%). Infection rates were not significantly different between treatment groups ($P > .05$), and groups exhibited similar distributions of severity ratings. A logistic regression model indicated that 2016 live-crown ratio was not a significant variable in predicting infection presence. Mean relative volume growth did not vary significantly ($P > .05$) with treatment group or infection status, implying that pruning neither inhibited nor improved growth. These long-term results indicate that pruning may not be a promising approach for white pine blister rust abatement, but further study is needed to account for the influence of climate and other important factors in determining the spread of blister rust infection.

KEYWORDS

silviculture, forest pathology, invasive species, mortality, growth and yield

INTRODUCTION

Among the thousands of exotic species introduced to North America, invasive pathogens pose one of the greatest risks to the health of the continent's forests (Lovett 2006). Pathogens are responsible for major changes in forest structure, nutrient cycling, food webs, and tree species composition, occasionally decimating entire tree species (Loo 2009). Sugar pine (*Pinus lambertiana*), one of the most prominent conifer species in the western United States, is currently threatened by *Cronartium ribicola*, an invasive, fungal pathogen that afflicts five-needled pine species (collectively known as white pines) with the lethal disease white pine blister rust (Liebhold et al. 1995). Sugar pine is prized for its durable wood and its provision of food for many wildlife species (O'Hara et al. 2010); its growing stock volume accounts for an estimated 2 percent of live biomass in the 7.98 million acres of mixed-conifer forest in California (FIA 2016).

In light of sugar pine's substantial economic and ecological value, it is concerning that up to 95 percent of original sugar pine and western white pine stands have been damaged or killed by blister rust (Liebhold et al. 1995), and annual sugar pine timber production has declined by more than half over the past few decades (Kinloch 1984; Morgan et al. 2012). Blister rust-related mortality of sugar pine alters the development of mixed-conifer stands, shifting species composition, increasing fire hazard, and hindering the establishment of young sugar pines in the understory (Waring and O'Hara 2009). Added physiological stress from blister rust cankers may also indirectly increase mortality of white pines by intensifying outbreaks of bark beetles, which kill trees through feeding and fungus introduction (Bockino and Tinker 2012). Economic research has highlighted a willingness to pay for management of *C. ribicola* on the scale of billions of dollars (Meldrum et al. 2013). Unfortunately, existing management options are limited, and implementation carries significant drawbacks.

Since the accidental introduction of blister rust to North America in 1906 via pine seedlings imported from Europe, its impacts on white pines have spurred several notably flawed efforts to control the spread of *C. ribicola* (Maloy 1997). For the fungus to complete its life cycle, it must alternate between five-needled pines and plants in the *Ribes* genus (currants and gooseberries) over a period of at least four years (Liebhold et al. 1995). Therefore, aggressive eradication of *Ribes* spp. has been the dominant approach to mitigating the pathogen's influence on pines. Blister rust control efforts relying on quarantines and the manual or chemical destruction of *Ribes* plants lasted

from 1909 to 1967 and spanned across 20 million acres of federal, state, and private lands in more than 30 states (Maloy 1997). Despite the $150 million invested in labor-intensive control programs, efforts were regarded as largely futile, as very few appreciable differences in infection rate were detected between stands never eradicated and stands receiving up to eight rounds of eradication (Kinloch 1984; Maloy 1997). Interest has since shifted toward alternative blister rust control treatments. Forest restoration plans dealing with blister rust have heavily emphasized burning and thinning treatments, but these techniques can exacerbate the frequency of *Ribes* spp. by increasing light availability near the forest floor (Maloney et al. 2008). Breeding programs aimed at encouraging genetic resistance in sugar pine have demonstrated promise and feasibility (Kinloch and Dulitz 1990); however, they may diminish the genetic diversity of sugar pine stands (O'Hara et al. 2010) and lead to the evolution of races of *C. ribicola* able to overcome resistance in sugar pine (Kinloch and Comstock 1981). Considering the shortcomings of popular treatment options, there is a palpable need for a strategy that can preserve existing genetic diversity and restore forest health without increasing blister rust hazards.

Artificial pruning, or the removal of tree branches, has exhibited considerable value for ameliorating blister rust infection in white pines. Pruning is already well known for reducing the prevalence of defects in wood, but it may possess the extra benefit of treating blister rust while preserving genetic diversity (O'Hara et al. 2010). In general, white pines tend to experience most blister rust infections within 2.5 meters of the ground surface (Hayes and Stein 1967, Hunt 1982), so the removal of lower branches may be effective at suppressing blister rust. Research on eastern white pine suggests that pathological pruning, the removal of all branches up to a certain height, is far more successful at reducing infection rates than removing only visibly infected branches because the latter procedure often overlooks latent infections (Lehrer 1982). A first round of pruning, referred to as a first "lift," can be useful in reducing infection with minimal effects on tree growth, but some also opt for a second lift later on, and it is uncertain whether this process is worth the extra effort since cost increases exponentially above operator height (O'Hara et al. 2010). In addition to knowledge about how to prune for blister rust management, there are some suggestions about when and where to prune to maximize the probability of success. Younger trees, flat slopes, lower frequency of *Ribes* spp., and a drier climate may all boost efficacy (Hunt 1982, Schnepf and Schwandt 2006). The majority of existing literature on pruning to abate blister rust

deals with single-stage pruning in species other than sugar pine, but more research is needed to quantify the long-term results of multiple-stage pruning on infection in sugar pine.

The overarching goal of my study was to test the effects of first- and second-lift pruning treatments on blister rust-related mortality of sugar pine trees. My work builds upon a study that spanned nearly seven growing seasons and examined the impacts of a first-lift pruning treatment on tree growth and blister rust infection in initially uninfected trees. Within this framework, I aimed to address three sub-objectives: (1) evaluate the effects of first- and second-lift pruning treatments on infection rate and severity, (2) compare mortality rates between unpruned, first-lift pruned, and second-lift pruned trees, and (3) quantify the impacts of pruning treatments on tree volume accumulation rates. I analyzed existing data in conjunction with newly collected measurements to assess trends in infection, mortality, and tree growth. I hypothesized that infection incidence, mortality, and infection severity would be significantly reduced with both forms of pruning. I expected volumetric growth to occur at a similar rate in unpruned and first-lift pruned trees, but I predicted a moderate decrease in growth rate for trees receiving a second round of pruning, due to a reduction in photosynthetic capacity (O'Hara et al. 2010).

METHODS

Study site

Located in the central Sierra Nevada at 38°52' N, 120°40' W (Figure 1), Blodgett Forest consists of over 1200 hectares of high-site mixed-conifer forest, oak forest, and brushland at elevations ranging from 1200 to 1500 meters (University of California Division of Agriculture and Natural Resources 2016). The forest is divided into roughly 90 management units called "compartments" (Figure 2), with each compartment subject to a particular combination of management activities. My study included trees from seven mixed conifer-dominated compartments examined by O'Hara et al. (2010) in a previous study of the effects of artificial pruning on blister rust in sugar pine; these compartments were harvested at different times and subsequently planted with a mixture of conifers which included both blister rust-resistant and nonresistant sugar pine. Each compartment received a combination of herbicide and mechanical treatments to reduce inter-tree competition and suppress non-tree vegetation (Table 1). All

compartments showed evidence of active blister rust infection prior to their selection for study in 2000 (O'Hara et al. 2010).

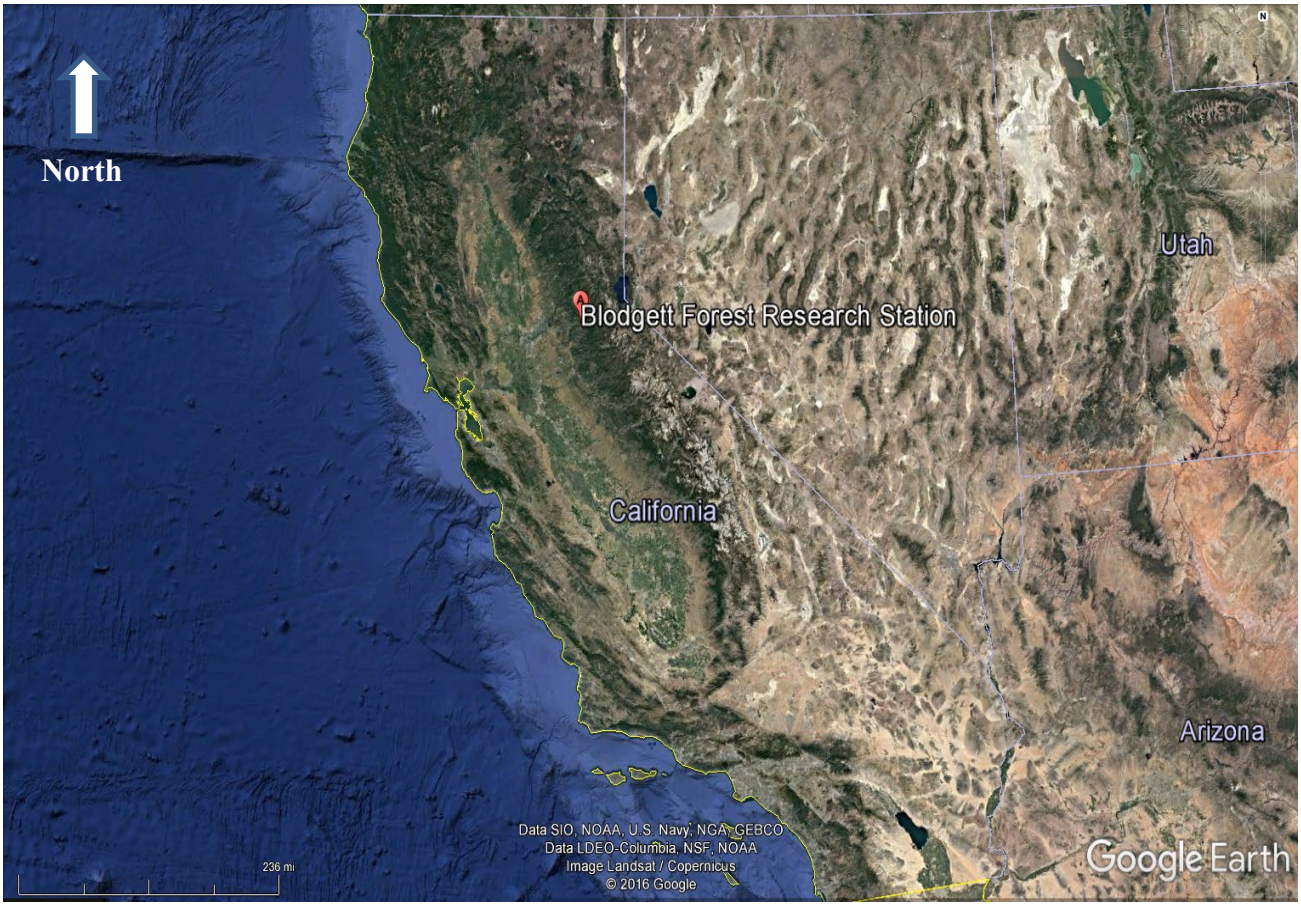

Figure 1. Location of Blodgett Forest Research Station. Map data: Google, Landsat / Copernicus

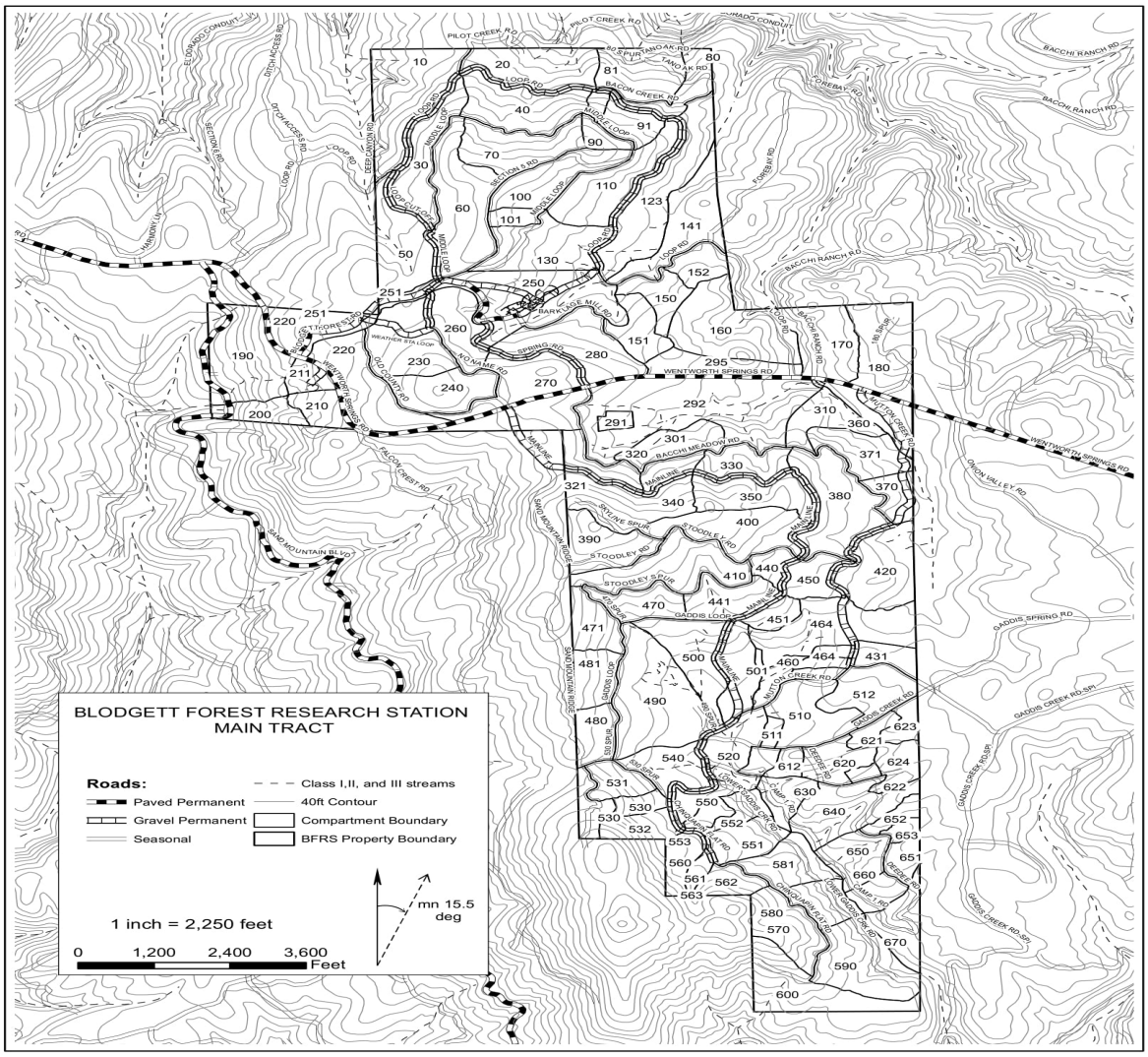

Figure 2. Map of Blodgett Forest Research Station Compartments. Study trees are located in Compartments 141, 190, 280, 330, 400, 480, and 501.

Table 1. Treatment history of study compartments. Adapted from O'Hara et al. 2010.

Study Compartment	Age of Stand in 2016	Regeneration Harvest Method	Postgeneration Treatments (Year)
141	26	Clear-cut	Herbicide (1995), pre-commercial thinning (1998), mastication (2005)
190	27	Group selection	Herbicide (1997), pre-commercial thinning
280	36	Shelterwood	Pre-commercial thinning (1995), herbicide (1984, 1989)

330	25	Clear-cut	Pre-commercial thinning (1998), herbicide (1999), mastication (2003)
400	25	Clear-cut	Pre-commercial thinning (1998), herbicide (1999), mastication (2003)
480	29	Clear-cut	Pre-commercial thinning (1998), herbicide (1991)
501	28	Group selection	Pre-commercial thinning (1998), herbicide (1992, 1997)

Study trees and previous pruning treatments

My study included 75 unpruned, 28 first-lift pruned, and 41 second-lift pruned sugar pine trees. These sugar pines were alive when last measured in 2006; they collectively represent 144 of the 214 trees at Blodgett originally chosen for study by O'Hara et al. (2010). The study trees are a mixture of single-gene resistant and nonresistant sugar pine trees. Trees were initially selected to be free of stem defects and signs of white pine blister rust. A pruning status (either pruned or unpruned) was originally assigned to alternate trees using a random starting point within each compartment, and trees were each tagged with a unique number (using even numbers for unpruned trees and odd numbers for pruned trees). First-lift pruned trees had their branches cut with loppers as close to the stem as possible and were pruned to an approximate live-crown ratio of 50% or no greater than 8 feet in height; needle fascicles below the pruning height were also removed. A subset of originally pruned trees received a second round of pruning, or a second lift, in 2007, having their branches removed to a height of roughly 12 feet or to a remaining live-crown ratio of no less than 50%. I visited these trees for data collection from October of 2016 to March of 2017. Hereafter, I will refer to data I collected as 2016 data, since my period of data collection followed the 2016 growing season.

Data collection

Status and pruning treatment type

Using Global Positioning System (GPS) points and physical tree location maps provided by Blodgett Forest Research Station, I navigated to each tree to check its status (alive or dead) and

record its pruning treatment type. I considered a tree to be alive if any green foliage was present on its branches. To determine whether each tree was pruned or unpruned, I visually inspected its bole and consulted past data sheets for pruning status information. For each pruned tree, I estimated the height to the lowest branch to determine whether the tree had been first-lift or second-lift pruned. If the lowest branch occurred at approximately 8 feet, I considered the tree first-lift pruned, but if the lowest branch occurred noticeably higher than 8 feet, I considered the tree second-lift pruned. To increase the visibility of study trees, I retagged trees where necessary and tied flagging tape to the branches of unpruned trees or around the stems of pruned trees.

Infection presence and severity

To quantify the magnitude of blister rust infection, I assigned an infection severity rating to each tree. I used a slightly modified version of the rating system implemented by Bockino and Tinker (2012) to measure blister rust severity in whitebark pine, because this system accounts for stress from infection in both the crown and the bole. The rating system assigns separate scores of 0-2 to the crown and the bole based on the prevalence of blister rust cankers. For the crown, a score of 0 indicates that no branches were infected, a score of 1 indicates that at least one branch and no more than a third of all branches were infected, and a score of 2 indicates that greater than one third of all branches were infected. For the bole, a score of 0 means that no cankers were present, a score of 1 means that one to three cankers was present, and a score of 2 reflects that four or more cankers were present. I confirmed the presence of cankers with evidence of branch flagging, swelling, resin, thin or dead bark, and/or orange *C. ribicola* spores (Hoff 1992). In addition to a severity rating, I also assigned each tree a binary variable indicating the presence or absence of blister rust infection, with a value of 0 indicating absence and a score of 1 indicating presence.

Tree volumetric growth

To track the volumetric growth of each study tree, I collected height and diameter measurements to compare with previously collected height and diameter data (O'Hara et al. 2010). I measured each tree's DBH, or diameter at breast height (i.e., the diameter at 1.37 meters above

the ground on the uphill side), in centimeters with a 30-meter Spencer Logger's Tape. I used a Vertex IV ultrasound hypsometer to measure in meters the height to the top of each tree and the height to the base of its live crown. I then converted DBH values to inches and height values to feet. From these measurements, I calculated current stem volume with the Pacific Northwest Forest Inventory and Analysis equations for estimating sugar pine volume (MacLean and Berger 1976):

$$\mathbf{TMP_{DBH}} = \mathbf{DBH}$$

$$\textbf{If DBH} < 6.0 \textbf{ inches then } \mathbf{TMP_{DBH}} = 6.0 \textbf{ inches and BA} = 6^2 \text{ x } 0.005454154$$

$$\mathbf{CF4} = 0.358550 - 0.488134 \text{ x } \left(\frac{1}{\text{TMP}_{\text{DBH}}}\right)$$

$$\text{If CF4} < 0.3 \text{ then CF4} = 0.3$$

$$\text{If CF4} > 0.4 \text{ then CF4} = 0.4$$

$$\mathbf{CV4} = 0.005454154 \text{ x } \text{TMP}_{\text{DBH}}^2 \text{ x HT x CF4}$$

$$\mathbf{TARIF} = \frac{\text{CV4 x } 0.912733}{\text{BA} - 0.087266}$$

$$\text{If TMP}_{\text{DBH}} > 6.0 \text{ then}$$

$$\mathbf{CVTS} = \text{CV4 x} \frac{\left(1.033 \text{ x } \left(1.0 + 1.382937 \text{ x } exp\left(-4.015292 \text{ x } \left(\frac{\text{DBH}}{10}\right)\right)\right)\right) \text{x } (\text{BA} + 0.087266) - 0.174533}{\text{BA} - 0.087266}$$

$$\mathbf{CVT} = \frac{\text{TARIF x } \left(0.9679 - 0.1051 \text{ x } 0.5523^{\text{DBH}-1.5}\right) \text{x } \left(\left(1.033 \text{ x } 1.0 + 1.382937 \text{ x } \exp\left(-4.015292 \text{ x } \left(\frac{\text{DBH}}{10}\right)\right)\right)\right) \text{x } (\text{BA} + 0.087266) - 0.174533}{0.912733}$$

$$\text{If TMP}_{\text{DBH}} = 6.0 \text{ then}$$

$$\mathbf{SMALL_{TARIF}} = 0.5 \text{ x } (6.0 - \text{DBH})^2 + (1.0 + 0.063 \text{ x } (6.0 - \text{DBH})^2 \text{ x TARIF})$$

$$\text{If SMALL}_{\text{TARIF}} \leq 0 \text{ then SMALL}_{\text{TARIF}} = 0.01$$

$$\mathbf{CVTS} = \text{SMALL}_{\text{TARIF}} \text{ x } \left(\left(\left(1.033 \text{ x } \left(1.0 + 1.382937 \text{ x } exp\left(-4.015292 \text{ x } \left(\frac{\text{DBH}}{10.0}\right)\right)\right)\right) \text{x } (\text{BA} + 0.087266) - 0.174533\right)\right)$$

$$\mathbf{CVT} = \frac{\text{TARIF x } \left(0.9679 - 0.1051 \text{ x } 0.5523^{\text{DBH}-1.5}\right) \text{x } \left(\left(1.033 \text{ x } 1.0 + 1.382937 \text{ x } \exp\left(-4.015292 \text{ x } \left(\frac{\text{DBH}}{10}\right)\right)\right)\right) \text{x } (\text{BA} + 0.087266) - 0.174533}{0.912733}$$

Where:

- DBH (inches) = DBH (cm) converted to inches (DBH/2.54)
- HT (feet) = HT (m) converted to feet (HT/0.3048)
- BA = basal area (square feet/acre): BA = 0.005454154 × DBH in inches
- CVTS = cubic foot volume, including top and stump
- TARIF = TARIF number equation (see Brackett 1977)
- CVT = cubic-foot volume above stump
- CV4 = cubic-foot volume above stump, 4-inch top

I also used these procedures to estimate tree stem volume in 2006, when DBH and height measurements were last collected. To compare the change in volume between trees while accounting for differences in initial tree size, I calculated the difference between the 2016 and 2006 above-stump cubic-foot tree volume estimates (i.e., CVT values) and normalized this difference by the 2006 volume estimate to provide a unitless measure of relative volumetric growth:

$$\textbf{Relative Volumetric Growth} = \frac{2016\ \text{CVT} - 2006\ \text{CVT}}{2006\ \text{CVT}}$$

Data Analysis

Pruning and blister rust infection rate

I analyzed the difference in infection rates between treatment groups with a chi-squared test of independence. For this test, I arranged my data in a 2-by-3 contingency table, with each row corresponding to a treatment category (unpruned, first-lift pruned, or second-lift pruned) and each column corresponding to an infection status (uninfected or infected). I summarized the data as the number of trees infected for each treatment category.

In addition to the chi-square test, I used logistic regression to attempt to predict the probability of tree infection in 2016 from a tree's 2016 live crown ratio (LCR), which I calculated as total tree height in feet minus height to the live crown base in feet, all divided by total tree

height. I used the statistical significance of the coefficients in the resulting model to determine whether 2016 LCR was a significant predictor of 2016 infection status.

Pruning and blister rust infection severity

I conducted a multivariate ordinal logistic regression in R (R Core Development Team 2014) to predict severity score as a function of pruning status and average compartment basal area, a measure of stand density. I defined the latter variable as the average cross-sectional stem area (in square feet) per acre of ground area across a given compartment. I calculated average basal area values (in $ft.^2$ per acre) using Blodgett Forest Research Station's database of inventory data, incorporating DBH data collected between 2006 and 2017 from all trees with DBH > 4.5" in all permanent, tenth-acre inventory plots in each compartment. Stand density varies widely between compartments due to differences in management prescriptions, and stand basal area may exert a strong influence on the competitive abilities of individual trees (Eitzel et al. 2015). Therefore, I designated average compartment basal area as a covariate in this analysis to control for differences in density between different compartments. I did not conduct post-hoc tests to examine the direction of any basal area-related effects because it was primarily intended to reduce variation in my comparisons.

Pruning and mortality

I approximated the degree of mortality experienced by each group and by all study trees with finite annual mortality rates, which provide estimates of annual mortality assuming a constant rate of mortality over a study period (Sheil et al. 1995). I calculated each annual mortality rate as follows:

$$m = 1 - \left[1 - \frac{N_0 - N_1}{N_0}\right]^{\frac{1}{t}}$$

Where:

- m = finite annual mortality rate
- N_0 = number of live trees at start of study period
- N_1 = number of live stems at end of study period

- t = duration of study period (years)

Data collection from the start of the study period occurred in late 2006, and data collection from the end of the study period spanned from late 2016 to early 2017, so I used a duration of 10 years to simplify my calculations.

To attach a measure of uncertainty to my estimates, I constructed 95% confidence intervals for each finite annual mortality rate. I generated these confidence intervals in R by running 999 Monte Carlo simulations of mortality outcomes from a binomial distribution (with the possible outcome of each individual tree-survival trial in a simulation being either 0 for live or 1 for dead) for each treatment group. I examined the overlap of confidence intervals between groups to assess the statistical significance of any differences in my estimates.

Pruning and Volumetric Growth

To analyze the differences in mean 2006-2016 relative volumetric growth between unpruned trees, first-lift pruned trees, and second-lift pruned trees, I conducted an Analysis of Covariance (ANCOVA), selecting pruning lift (unpruned, first lift, or second lift) and infection status (infected or uninfected) as my independent variables of interest and relative volumetric growth as my dependent variable of interest. As with my analysis of infection severity, I controlled for variations in stand density by designating the average compartment basal area ($ft.^2$ per acre) as a covariate in my analysis. I omitted dead trees from my relative volumetric growth analysis because each of these trees died (and therefore ceased growing) at an unknown point in the study period.

RESULTS

Pruning and infection rate

I did not observe a clear reduction in infection rate with the application of first-lift or second-lift pruning. A chi-square test of independence on a 2-by-3 contingency table containing

counts of live trees, organized by treatment group and infection status (Table 2), revealed statistically non-significant ($P = 0.786$) deviations from expected counts in each category.

Table 2. 2-by-3 contingency table for chi-square analysis. Percentages of trees within each pruning lift category are followed by tree counts in parentheses. Table only includes study trees that were not infected in 2006 in order to exclude pre-existing infections. $X^2(2) = 0.48157$, $P = 0.786$.

Pruning Lift	Infected	Uninfected	Total
First Lift	47.37% (9)	52.63% (10)	**100.00% (19)**
Second Lift	57.14% (16)	42.86% (12)	**100.00% (28)**
Unpruned	55.56% (20)	44.44% (16)	**100.00% (36)**
Total	**45**	**38**	**83**

A logistic regression model determined that live crown ratio was not a statistically significant factor ($P = 0.831$) in predicting infection rates. The model, which included all study trees that showed no visible signs of infection in 2006 (even trees that were dead as of 2016), aimed to predict the probability of tree infection solely as a function of 2016 live crown ratio.

Pruning and infection severity

I observed fairly uniform proportion distributions of severity scores across groups (Figure 3). Unpruned, first-lift pruned, and second-lift pruned trees showed similar proportions of trees across infection severity categories, except for the absence of second-lift pruned trees in the category corresponding to a rating of 3. A multivariate ordinal regression model to predict infection severity based on pruning lift while controlling for average compartment basal area showed that pruning lift was not a statistically significant predictor of infection severity ($P = .29$). Average compartment basal area, the covariate in the model, demonstrated statistical significance

in predicting infection severity (P = .0144). Overall, the regression resulted in a failure to reject at the P = .05 level the null hypothesis that infection severity distribution changes with pruning status.

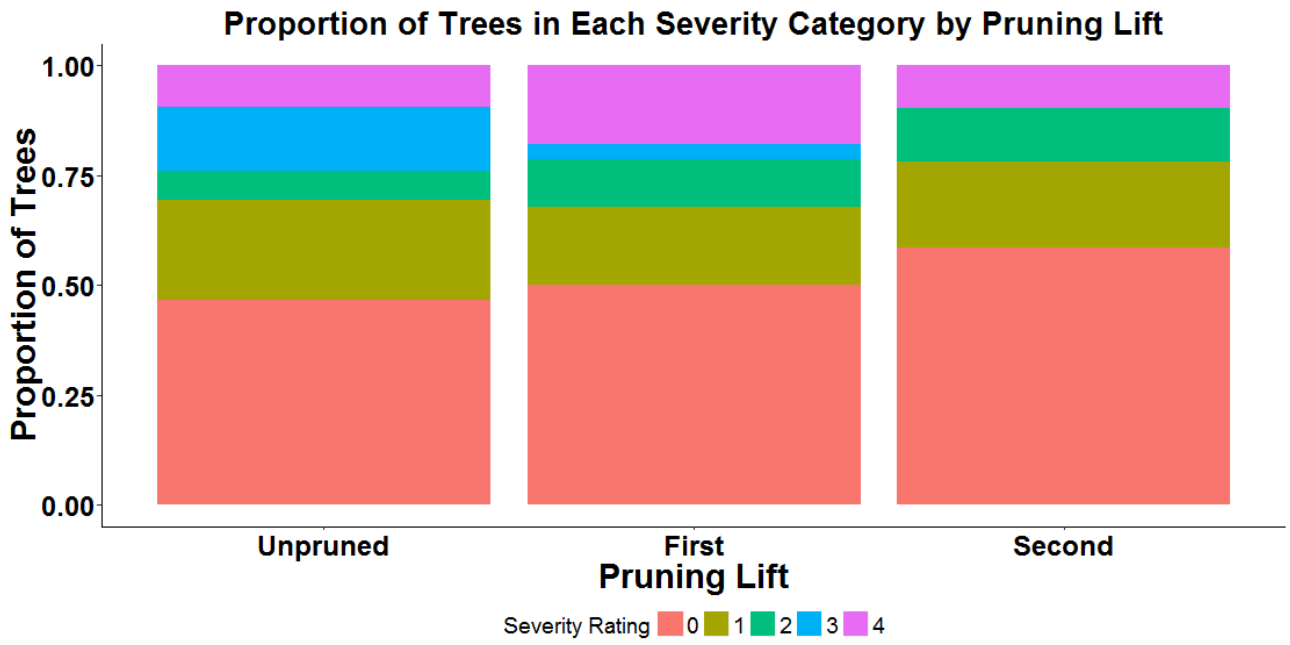

Figure 3. Proportion of trees in each severity category, grouped by pruning lift (Unpruned, First Lift, and Second Lift). 0 = no infection, 1 = light infection, 2 = moderate infection, 3 = heavy infection, and 4 = lethal infection. Distributions are similar across treatment groups, and pruning lift was not a statistically significant predictor of infection severity (P = .29)

Pruning and mortality

Finite annual mortality rates for the 2006-2016 period differed by pruning lift (Table 3). Out of 144 study trees, 18 died and 126 survived through 2016, translating to 12.5% mortality over the duration of the study period. The overall finite annual mortality rate was 1.326%. First-lift pruned trees exhibited the greatest finite annual mortality rate (m = 1.948%) with almost double the annual percent mortality of unpruned trees (m = 1.122%). Second-lift pruned trees displayed a finite annual rate between those of the other two groups (m = 1.292%) and slightly lower than the overall rate. However, the 95% confidence intervals constructed for group estimates of finite annual mortality overlap heavily (Figure 4), suggesting that differences in the estimated rates are not statistically significant.

Table 3. Finite annual mortality rates by pruning lift for the 2006-2016 study period. N_0 = number of live trees at beginning of period, N_1 = number of live trees at end of period, and m = finite annual mortality rate.

Pruning Lift	N_0	N_1	m	95% CI for m
Unpruned	75	67	1.122%	[0.407, 2.045]
First Lift	28	24	1.948%	[0.363, 3.803]
Second Lift	41	36	1.292%	[0.247, 2.448]
Overall	**144**	**126**	**1.326%**	**[0.791, 1.972]**

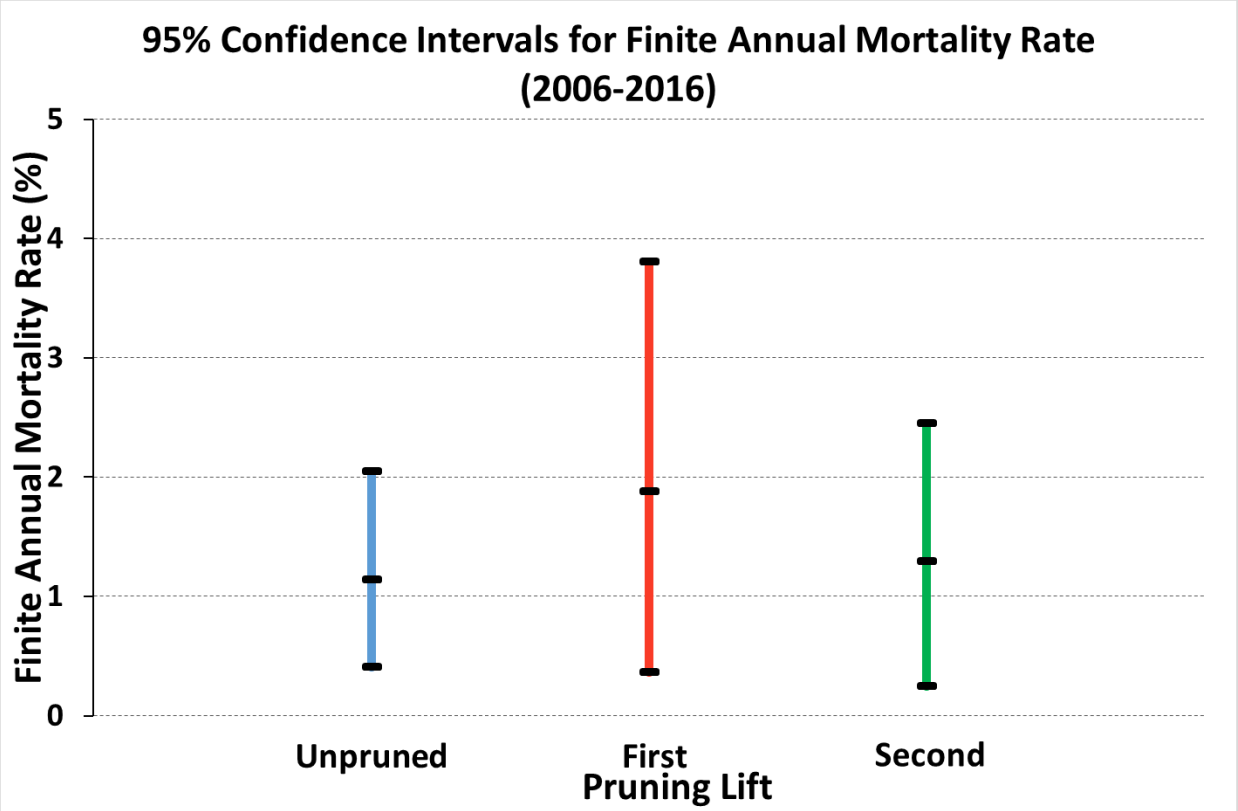

Figure 4. 95% confidence intervals for estimated finite annual mortality rates of treatment groups. Each mean estimate of m is contained within the confidence intervals of both other groups, suggesting a failure to reject at the *P* = .05 level the null hypothesis that finite annual mortality rate differs between treatment groups.

Pruning and volumetric growth

Relative volumetric growth from 2006 to 2016 averaged 3.51 for all live trees. The mean increment for first-lift pruned trees was 3.10 and the mean increment for second-lift trees was 3.35. These values were only slightly lower than the mean increment of 3.71 for all unpruned trees. The mean increment for all uninfected trees (3.55) was slightly greater than the mean increment of all infected trees (3.44). Table 4 summarizes relative volumetric growth values for all combinations of pruning lift and infection status.

Table 4. Per-tree stem volume and relative volumetric growth estimates (mean ± standard dev.).

Pruning Lift, Infection Status	Relative Volumetric Growth (mean ± s.d.)	2006 CVT in cubic feet (mean ± s.d.)	2016 CVT in cubic feet (mean ± s.d.)
Unpruned, Uninfected	3.82 ± 1.53	2.31 ± 0.79	11.30 ± 5.83
First Lift, Uninfected	3.26 ± 1.53	2.01 ± 0.48	8.42 ± 3.04
Second Lift, Uninfected	3.36 ± 1.76	2.75 ± 1.39	11.69 ± 6.66
Unpruned, Infected	3.63 ± 1.83	2.71 ± 1.28	12.79 ± 7.75
First Lift, Infected	2.84 ± 1.91	2.04 ± 0.79	7.87 ± 4.98
Second Lift, Infected	3.36 ± 1.60	2.27 ± 0.39	9.98 ± 4.14

An ANCOVA test for differences in mean relative growth increment for all live trees failed to show statistically significant discrepancies based on pruning lift (P = .243) or infection status (P = 0.569) and found no statistically significant interaction between infection status and pruning lift (P = 0.976). Average compartment basal area, which I designated as a covariate, had a statistically significant effect on mean relative volumetric growth, suggesting an influence of stand density on tree growth during the study period. Relative volume growth distributions were fairly similar across pruning lifts (Figure 5).

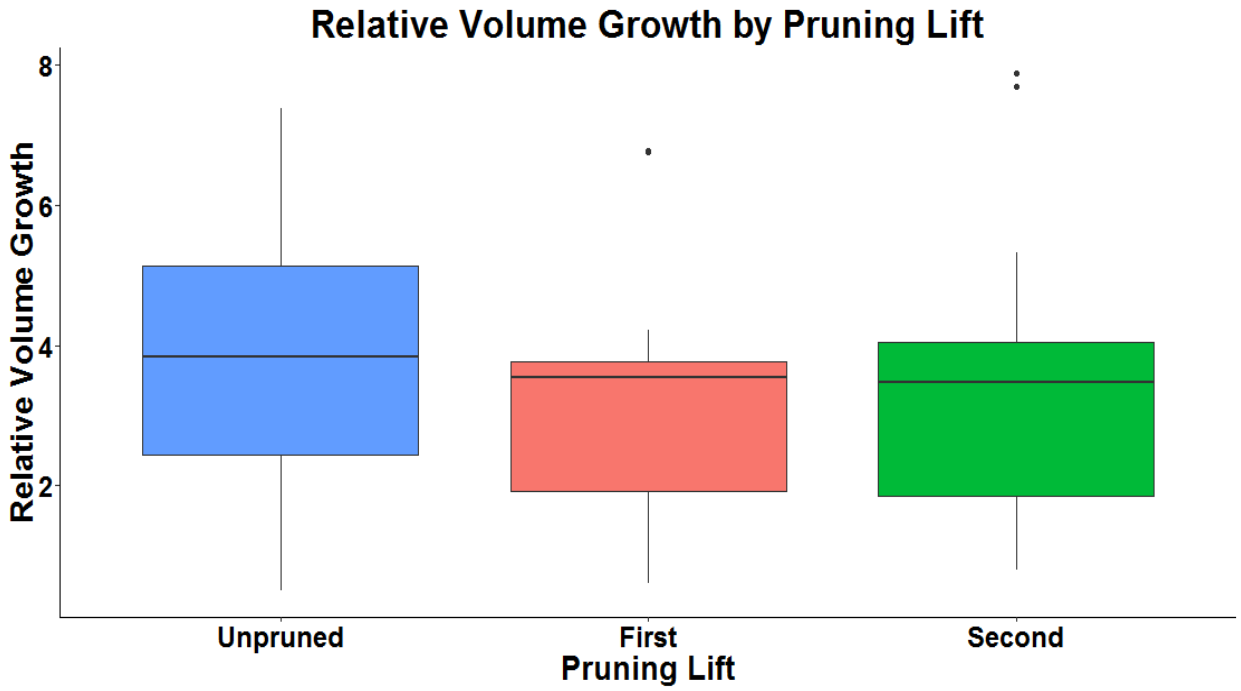

Figure 5. Relative volumetric growth of infected trees by pruning lift.

DISCUSSION

Pruning, infection rate, and mortality

Neither first-lift nor second-lift pruning altered the susceptibility of the study trees to white pine blister rust over a period of 16 years. The statistically non-significant differences in 2016 infection rate between treatment groups contradict the elevated infection frequency in unpruned sugar pine observed by O'Hara et al. (2010). Findings linking pruning and infection rate are mixed for other white pine species in North America. In a 9-year study on eastern white pine trees throughout Wisconsin, pruned trees had a significantly lower infection rate than unpruned trees, along with a four-fold decrease in blister rust loss with pruning (Lehrer 1982). However, in a 10-year study on the effects of pruning western white pine to reduce blister rust losses across 10 stands in British Columbia, results failed to demonstrate a significant reduction in mean infection incidence with pruning; furthermore, there was an even smaller reduction in mean numbers of both lethal and non-lethal cankers (Hunt 1998). The lack of a reduction in blister rust with pruning may be partially explained by tree age at the time of pruning. Pruning has been noted to be most

beneficial in blister rust reduction when applied to trees at a young age, as the especially susceptible lower branches of young trees comprise a greater proportion of the total crown area than in older, larger trees (Lehrer 1982). The absence of a clear association between pruning and reduced blister rust infection rates may be due to pruning trees when they are too old to receive much benefit (Hunt 1998). My study trees ranged from 9 to 20 years old at the initial time of pruning (O'Hara et al. 2010). Trees in the upper end of this age range may be older than the ideal age for combatting blister rust infection via pruning (Hunt 1998).

Another possibility is that pruning benefits for reducing infection may simply diminish over time. In some cases, trees can "outgrow" infection as their photosynthetic capacity increases and they begin to accumulate biomass at a rate exceeding the rate at which white pine blister rust damages woody tissue (Burns et al. 2008). This is especially true on productive sites where trees tend to grow quickly. Blodgett Forest is dominated by highly productive land (UCANR 2016), so many of these sugar pines may simply be outgrowing infection over time as photosynthetic capacity increases. Although the findings of O'Hara et al. (2010) suggest a clear reduction in blister rust incidence with pruning, my findings indicate that this benefit may not be present over longer periods on the order of 15 years or more. This point is further corroborated by the fact that my logistic regression model did not show live-crown ratio (LCR) to be a statistically significant predictor of infection, unlike the model developed by O'Hara et al. (2010) to predict infection based on LCR at an earlier stage of tree development.

Unpruned trees also experienced the lowest annual rate of mortality, implying that pruning did not decrease mortality as of 2016 and may have slightly increased mortality, although effects on mortality were not statistically significant. Added stress from reduced photosynthetic capacity can, in some cases, reduce the vigor of pruned trees (O'Hara et al. 2010). Because pruning did not cause a large increase in mortality, this effect may have been minimal.

While biotic factors such as volumetric growth and photosynthetic capacity may be important determinants of the efficacy of pruning for blister rust abatement, climatic variation may exert considerable influence on differences in infection and mortality rates between treatment groups. Well-established forest pathogens in the Sierra Nevada are generally expected to become more prevalent as mean temperatures increase and accelerate pathogen development (Battles 2005). Pathogens that cause stem cankers may particularly benefit from drought because canker defense mechanisms such as callusing and compartmentalization are inhibited in water-stressed

trees (Kolb et al. 2016). However, as a foliar pathogen, *C. ribicola* infects needles most efficiently under sustained periods of 100% relative humidity (Battles et al. 2005). Therefore, extended periods of drought followed by abnormally wet years may allow *C. ribicola* to spread most effectively when tree defenses are most limited, increasing the vulnerability of sugar pines to infection over time. If, regardless of pruning status, most trees are overwhelmed by climatic stresses, a general increase in infection could close the gap in infection rates between pruned and unpruned trees. A graph of monthly precipitation at Blodgett Forest Research Station from 1999 to 2016 (Figure 6) reveals more drastic and uneven fluctuations in precipitation in recent years than during the 2000-2006 study period of O'Hara et al. (2010). It is possible that the combination of 5 years of drought proceeding my data collection and extraordinarily high levels of precipitation in late 2016 and early 2017 masked the differences in infection characteristics between groups that might be noticeable under less extreme weather conditions.

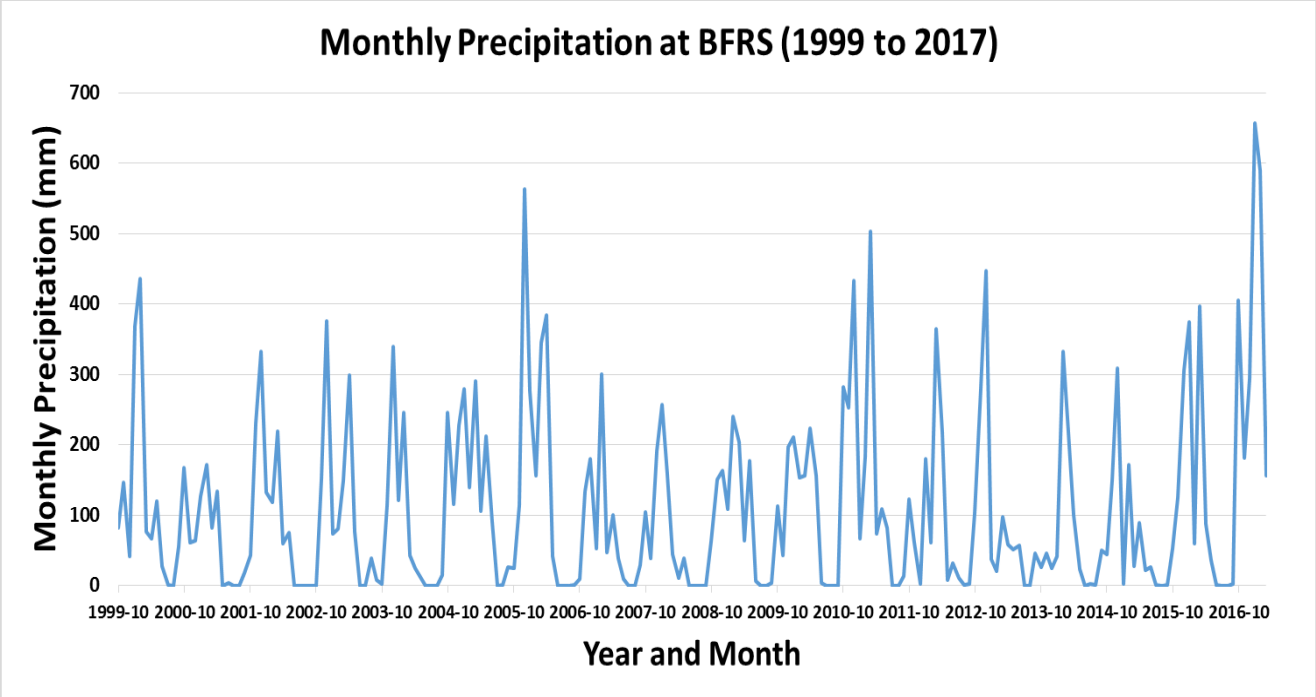

Figure 6. Monthly precipitation (in mm) at Blodgett Forest Research Station from 1999 to 2017. The beginning of each water year (starting in October of the previous calendar year) is marked on the x-axis. For example, the beginning of the 2000 water year is marked "1999-10." The 2000 to 2006 study period exhibits more temporally even fluctuations and less pronounced spikes in precipitation than recent years. In the few years preceding this study, extended periods with little precipitation, followed by large increases in precipitation, may have resulted in elevated infection rates for all groups.

Another possible explanation for the lack of a clear reduction in infection rate with pruning lies in the nature of the study design. Because my study included only surviving trees from the study by O'Hara et al. (2010), the trees I measured were, on average, likely fitter and more well-adapted to resist blister rust infection. This common problem, often called "heterogeneity's paradox," arises when studying a single cohort over a long time period, as the composition of the cohort does not remain the same in each measurement period (Vaupel et al. 1985). Unfortunately, the issue is inherent in long-term cohort studies, especially those involving elevated mortality.

Pruning and infection severity

The high degree of similarity between infection severity score distributions for the three groups indicates that pruning did not have a significant effect on the extent of infection for trees that exhibited signs of blister rust in 2016. O'Hara et al. (2010) did not examine severity, but this is an important component of infection because it may determine blister rust-caused mortality rates. In addition to direct contributions to white pine mortality, more severe blister rust infections may amplify the impacts of associated disturbance agents, including insect pests and drought. Blister rust severity has been positively correlated with the probability of attacks by bark beetles in other white pines (Bockino and Tinker 2012), so my results imply that pruning likely does not have a significant effect on reducing bark beetle attacks in sugar pine. As with infection incidence, the severity of blister rust infection and accompanying disturbances is likely linked to climatic change in the region. Increased host susceptibility due to drought stress can allow for the formation of more lethal cankers (Battles et al. 2005, Kolb et al. 2016).

Differences in infection severity may also have been obscured by the coarse resolution of the severity rating scheme I chose to implement. The rating system I used does not directly report the number of cankers on each tree, opting instead for an overall assessment of crown and bole conditions. Hunt (1998) and Kearns and Jacobi (2007) have previously implemented severity rating schemes with canker counts and locations for western white pine, and this information may increase the level of detail of severity ratings for blister rust in sugar pine, which could influence conclusions regarding the effect of pruning on infection severity.

Pruning and volumetric growth

The similar mean rates of relative volumetric growth across all treatment groups suggest that neither first-lift nor second-lift pruning substantially impacted the volume growth of sugar pine. This is consistent with the findings of O'Hara et al. (2010). Coupled with my mortality rate findings, this result indicates that tree growth was not a major cause of the mortality rate differences I observed. Infection status also did not appear to exert a statistically significant influence on relative volumetric growth rates, although mean relative growth for infected trees was slightly lower than mean relative growth for uninfected. This is expected because infection stresses trees and causes them to allocate more of their available resources to combatting damage than to expanding cambial tissue (Maloy 1997). However, the small magnitude of the mean growth rate differences between infected and uninfected trees implies that the added stress may not be a critical factor in determining the volumetric growth of sugar pines at Blodgett. Because stand density was singled out as a statistically significant factor in determining volumetric growth, further study to elucidate the direction of this effect is warranted.

Limitations and future directions

My study was impacted by a number of limitations both before and during the data-collection stage. Initially, all study trees were selected to be free of infection (O'Hara et al. 2010), and the chi-square test of independence for differences in 2016 infection rates focused only on trees that were not infected in 2006. Because I did not incorporate trees that were infected prior to the application of treatments, additional research is necessary to determine whether infected trees can be rescued from infection via pruning. During the course of this study, I established 38 fixed-radius plots including infected and uninfected young sugar pine trees and randomly assigned each a pruning status. These plots will provide an opportunity for future investigation of this topic.

Practical limitations during the study period impacted my data collection. Heavy seasonal precipitation and dangerous road conditions prevented me from collecting more data within the time allotted, so my sample is restricted to a relatively small group of sugar pines (n = 144). Consequently, my results address the efficacy of small-scale pruning operations for blister rust abatement and have limited utility in describing dynamics in larger forests. Future studies dealing

with a larger population of sugar pines could provide opportunity for more robust analysis and may generate results that are more directly applicable for more extensive mixed-conifer stands. Time constraints also precluded detailed measurements of stand density. Because I did not have enough time to directly measure stem density in the vicinity of each study tree, I inferred stand density in the immediate surroundings of each tree via average compartment basal area calculations. Because average compartment basal area was a statistically significant variable in determining blister rust infection severity and relative volumetric growth, detailed measurements of stand density could provide important information regarding its effect on blister rust infection. Existing literature presents conflicting hypotheses about the direction of the effect of stand density on blister rust infection: some authors hypothesize that a greater density of non-susceptible stems may offer a physical barrier to spore propagation (Hungerford et al. 1981, Schwandt et al. 1994), while some believe that lower stand density can lead to a decrease in infection rate due to less favorable microclimatic conditions for infection (Burns et al. 2008). Selecting study trees within fixed-radius monitoring plots across different stands may improve the accuracy of density estimates in future studies to elucidate the influence of density on infection in sugar pine stands.

This study also omitted the examination of a critical element in determining blister rust infection characteristics: the abundance of *Ribes* spp. (Maloy 1997). I initially intended to examine plot-level *Ribes* volume and its influence on the efficacy of pruning treatments for reducing blister rust infection rate and severity. However, available data from vegetation monitoring plots only recorded *Ribes* ground cover to the nearest 5%, which did not capture significant differences between compartments. A follow-up investigation using more detailed *Ribes* cover or volume data and information on distances of *Ribes* plants from study trees may allow for thorough examination of this relationship.

CONCLUSIONS

The absence of clear differences in infection presence, infection severity, and volume growth based on pruning status suggests that pruning may not be very effective in the long-term management of blister rust and associated disturbances in mixed-conifer stands containing sugar pine. The non-significant effects of treatments on volume growth imply minimal impacts of pruning on forest carbon sequestration. In using these findings to evaluate the efficacy of pruning

to mitigate blister rust in sugar pine, is important to consider the novel climatic variation experienced at Blodgett in the years directly preceding my data collection. Prolonged periods with little precipitation, followed by large spikes in precipitation, may account for the small magnitude of the observed differences in infection rate, infection severity, and tree volume growth, as trees may have been infected at a point of unusually high drought stress. If this is true, California's changing climate may further nullify the blister rust abatement potential of pruning treatments in years to come. It is also quite possible that trees may simply be outgrowing infections over time, regardless of changing climate conditions. Despite its probable inefficacy for blister rust abatement, pruning may still be an economically feasible method of improving wood quality in sugar pines (Hagle and Grasham 1988). Further studies that incorporate the influence of climatic variation and other important factors in determining infection may be necessary to gain a better idea of the true value of pruning to abate white pine blister rust in sugar pine.

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