Forest Ecology and Management 480 (2021) 118654

Contents lists available at ScienceDirect

Forest Ecology and Management

journal homepage: www.elsevier.com/locate/foreco

Impacts of thinning treatments on dynamics of needle disease caused by *Elytroderma deformans* (Weir) Darker and interactions with bark beetle-attacks in the northern Rocky Mountains

Ekaterina Smirnova [a,*], Patrick Bennett [b], Joel Egan [b], Leonid Kalachev [c], John Goodburn [d], I. Blakey Lockman [e], Cheri Hartless [f]

[a] Department of Biostatistics, Virginia Commonwealth University, Box 980032, Richmond, VA 23298, United States
[b] USDA Forest Service, Northern and Intermountain Regions, State and Private Forestry, Forest Health Protection, 26 Fort Missoula Rd., Missoula, MT, 59804, United States
[c] Department of Mathematical Sciences, University of Montana, 32 Campus Dr, Missoula, MT 59812, United States
[d] W.A. Franke College of Forestry & Conservation, University of Montana, 32 Campus Dr, Missoula, MT 59812, United States
[e] USDA Forest Service, Pacific Northwest Region, 1220 SW 3rd Ave, Portland, OR 97204, United States
[f] USDA Forest Service, Northern Region, Bitterroot National Forest, 88 Main St., Stevensville, MT 59870, United States

ARTICLE INFO

Keywords:
Ponderosa pine
Pre-commercial thinning
Elytroderma needle disease
Mountain pine beetle
Montana
Elytroderma crown infections

ABSTRACT

The fungus *Elytroderma deformans* causes a serious needle disease of pines in western North America and is considered the most important needle disease of ponderosa pine in Montana. While important, there has been limited evaluation of pre-commercial thinning or other treatments on disease incidence. Our study assessed the efficacy of various silvicultural treatments to reduce impacts from Elytroderma disease within monitoring plots that were established in 2004 and surveyed until 2014. Treatments were randomly assigned through incomplete block design to: (1) thin to 3.7 × 3.7 m spacing; (2) thin to 5.5 × 5.5 m spacing; (3) thin to 3.7 × 3.7 m spacing plus pruning; and (4) control with no thinning or pruning. For assessment, a statistic was developed to represent the average change in Elytroderma presence that occurred from 2006 to 2014 for each plot. This statistic was utilized as the response variable in a model that controlled for initial Elytroderma occurrence within plots to test for treatment effects. Overall, Elytroderma intensified across all plots during the study period and treatments were not effective at preventing new infections. Plots that received the 3.7 m × 3.7 m residual spacing exhibited slightly reduced progression of disease incidence relative to the other treatments. A mountain pine beetle (*Dendroctonus ponderosae* Hopkins, MPB) eruption occurred during this study. Tree-level MPB and Elytroderma interactions were assessed and no evidence was found to indicate trees infected with Elytroderma had a different likelihood of being attacked by MPB relative to uninfected trees. However, the trees located in plots with high levels of initial Elytroderma disease presence had greater chances of having tree mortality in general, caused by MPB or other factors. MPB did not attack any trees in plots that had the 5.5 × 5.5 m spaced treatment, even where adjacent plots were attacked. Results of this study are presented to inform management of young ponderosa pine forests on impacts of the silvicultural techniques tested on post-treatment Elytroderma dynamics and interactions with MPB-attack.

1. Introduction

Silvicultural treatments are intended to address dynamic interactions of stand structural development with increased interspecific competition and disturbance pressures from biotic and abiotic agents (Oliver and Ryker, 1990). Pre-commercial thinning (PCT) is a widely used management activity that is often applied in lower montane ponderosa pine (*Pinus ponderosa* var. *ponderosa* Dougl. ex Laws.) forests (Ferguson et al., 2011). This is done for two main reasons: (1) to enhance growing space and alter forest structure to reduce competition; and (2) to promote individual tree growth and vigor while providing resistance to potential disturbances (Oliver and Larson 1996). While resource

* Corresponding author.
E-mail address: ekaterina.smirnova@vcuhealth.org (E. Smirnova).

https://doi.org/10.1016/j.foreco.2020.118654
Received 3 August 2020; Received in revised form 16 September 2020; Accepted 23 September 2020

objectives are often site-specific, managers use research findings to design treatments to resist impacts from high-severity wildland fire and drought (Graham et al., 1999; Fettig et al., 2019), to reduce susceptibility to bark beetle-attack (Olsen et al. 1996, Fettig et al. 2007, Egan et al. 2010), and to reduce infection rates and potential spread of disease-causing pathogens and parasitic plants (i.e. sanitation) (Oester et al., 2018).

Decades of fire exclusion have resulted in substantial changes to stand composition and structure in western coniferous forests leading to increases in stand density and greater susceptibility to disturbances such as pathogens and insects (Gruell et al., 1982; Byler and Hagle, 2000; Parker et al., 2006; Crotteau and Keyes, 2020). Modifications to stand structure to mitigate potential fire behavior typically aim to reduce ladder fuels and canopy bulk density (Graham et al., 1999; Agee and Skinner, 2005; Peterson et al., 2005). These fire-oriented objectives are often implemented in conjunction with forest thinnings to remove some trees while retaining others and increasing inter-tree spacing. Thinning treatments have the added benefits of altering microsite conditions to reduce bark beetle habitat suitability and promoting residual tree vigor and defensive capacity (Olsen et al., 1996; Fettig et al., 2007; Egan et al., 2016). Furthermore, management strategies often alter stand composition in a manner aimed to promote sanitation through selective removal of infected hosts and disease-causing agents such as dwarf mistletoe (caused by *Arceuthobium* spp.), western gall rust (caused by *Endocronartium harknessii*), white pine blister rust (caused by *Cronartium ribicola*), and Elytroderma needle disease (caused by *Elytroderma deformans* (Weir) Darker) (Oester et al., 2018).

Without treatments, even-aged ponderosa pine stands go through a self-thinning process of stand-development in which competition-induced mortality eventually reduces stand density (Oliver and Larson, 1996). This is often facilitated by *Dendroctonus spp*. beetle-attacks, disease-causing agents, and/or suppression-related mortality. This mortality can contribute to spatially clumped fuel loadings and tree mortality which may not lead to stand structural attributes resistant to subsequent disturbance (Oliver, 1995; Egan et al., 2016). Various studies have reported benefits of thinning to enhance tree vigor and modify bark beetle microsite suitability to effectively change stand structure to reduce post-treatment disturbance susceptibility (Fettig et al., 2007; Egan et al., 2010). However, few studies to-date have investigated the effects of pre-commercial thinning on the dynamics of needle disease or interactions with mountain pine beetles (*Dendroctonus ponderosae* Hopkins, MPB).

The fungus *E. deformans*, first described by Weir (1916), is one of the most damaging needle diseases of ponderosa pine in western North America. Its disturbance ecology can be influenced by multiple spatial factors, including tree spacing and microclimate effects (Childs, 1968; Scharpf 1990). Successful infection occurs in mid- to late-summer when mature fruiting bodies (hysterothecia) of *E. deformans* are present and producing viable spores, young susceptible foliage of the pine host is present, and moist conditions persist due to fog, humidity, or precipitation (Childs et al., 1971; Scharpf, 1990). The following spring, infected needles turn red and new hysterothecia are produced (Childs et al., 1971). After spore release, infected needles are shed throughout fall and winter. The fungus grows from the needles into the inner bark of infected branches (Scharpf, 1990) and may also penetrate the main stem in younger trees, which often exhibit characteristic dark resinous lesions in the internal tissues (Childs, 1968; Childs et al., 1971). This vegetative growth inside of the stems allows the fungus to spread within a tree even when conditions are unfavorable for spore dispersal and infection (Childs et al., 1971). Infected branches often develop into dense "witches' brooms" which may survive and grow for many years (Childs et al., 1971; Lockman and Hartless, 2008; Scharpf, 1990).

Foliage loss in the fall and winter often causes crowns to appear thin and may impact tree vigor (Childs et al., 1971; Scharpf, 1990). Direct mortality from Elytroderma has been documented in trees of all ages in areas with long-term severe infections (Childs et al., 1971; Lockman and Hartless, 2008). Moderately infected trees may also be more susceptible to biotic disturbance a bark beetles and root disease compared to uninfected trees (Childs, 1968; Scharpf and Bega, 1981; Lockman and Hartless, 2008). Trees with severe *E. deformans* infections become less attractive to bark beetles over time (Childs et al., 1971), potentially due to reduced nutritional phloem quality.

Severe Elytroderma symptoms often occur in densely stocked stands that exhibit low vigor due to inter-tree competition or other stressors (Childs et al., 1971; Scharpf and Bega, 1981). Trees located in draws, meadows, and other areas where fog persists and moist air settles are also more likely to become infected, as the intensification and spread of the disease is dependent upon moisture availability (i.e. high humidity and/or spring rainfall) (Childs et al., 1971; Scharpf and Bega, 1981; Scharpf, 1990). Maintaining proper spacing in young stands may be critical to reduce impacts from *E. deformans* (Lockman and Hartless, 2008), as this would maintain good airflow to reduce humidity and leaf wetness. However, thinning in stands with preexisting infections may exacerbate the disease impacts in the short term (Childs et al., 1971; Lockman and Hartless, 2008). The best management practices for ponderosa pine stands in areas prone to *E. deformans* are thinning stands early and maintaining good inter-tree spacing (Lockman and Hartless, 2008). However, little information exists regarding ideal spacing levels and whether pruning is effective in preventing new infections.

Pruning was hypothesized to be beneficial for treating stands with Elytroderma due to epidemiological similarities shared with white pine blister rust caused by *C. ribicola*. Like *E. deformans*, *C. ribicola* infects pine trees through the needles and penetrates branches and stems via vegetative spread, and thus pruning not only functions in sanitation (i.e., removing infected branches), but also prevents the progression of branch cankers to lethal bole cankers (Schwandt et al., 2010; Zambino, 2010). While there is no conclusive research indicating that pruning is effective for managing Elytroderma, it has been recommended in the past (Lockman and Hartless, 2008). Most *E. deformans* infections occur in the lower crowns due to higher humidity, thus pruning of the lower branches could help control the disease. Pruning could alleviate these conditions of high humidity in the lower crown while also reducing future inoculum sources and the potential for vegetative spread to branches higher in the canopy.

Thinning has also been investigated as a potential control method for white pine blister rust. Results from multi-year monitoring plots indicate, however, that this treatment may exacerbate disease in western white pine stands due to the removal of spore dispersal barriers and/or the promotion of habitat for the alternate host (*Ribes* spp.) to increase in abundance and amplify spore loads produced (Schwandt et al., 2010; Zambino 2010). Similarly, Childs (1968) suggested that large openings in stands with Elytroderma may exacerbate disease because these openings create conditions that are more favorable for spore dispersal. While the impacts of density reduction treatments in stands impacted by Elytroderma are largely unknown, empirical evidence has consistently reported yellow pine stands with reduced forest density have greater resistance to successful MPB attack relative to dense forested areas (Fettig et al., 2007; Egan et al., 2010). However, various post-treatment inter-tree spacings and residual density targets have been proposed to-date and warrant further study.

Our study was established in young, pre-commercial-sized ponderosa pine forest located in an area that had a history of Elytroderma infections. The goal of this work was to evaluate effects of various thinning and pruning treatments on the local intensification of the disease ten years post-treatment (Lockman and Hartless, 2008). Prior to completion of the study, a MPB outbreak occurred which enabled evaluation of an additional insect and disease interaction factor. Specific objectives of this study were to: (1) test alternative thinning densities, with and without pruning, in terms of potential effects on disease intensification; (2) evaluate potential interactions between Elytroderma and MPB; and (3) provide managers with pre-commercial treatment specifications to promote resistance to multiple disturbances in young ponderosa pine

stands.

2. Materials and methods

2.1. Description of stands

The study area overlooks Lake Como in the Darby Ranger District of the Bitterroot National Forest, approximately 60 miles south of Missoula, Montana. Twelve ponderosa pine stands with average age of 20 years were scheduled for pre-commercial thinning and were available for long term monitoring. The stands generally faced east, with aspects ranging from northeast to southeast and elevations ranging from 1400 m (4600 ft) to 1707 m (5600 ft).

2.2. Treatments and study design

In 2004, 24 plots were established to monitor the effects of thinning and pruning on ponderosa pine stands affected by Elytroderma needle disease (Lockman and Hartless, 2008). Originally, 12 stands were selected for pre-commercial thinning within the study area on the Darby Ranger District of Bitterroot National Forest, and two plots were randomly selected within each stand according to the incomplete block design. The majority of plot-pairs belonging to the same stands were characterized by approximately the same elevations, slopes at plot centers and aspects at plot centers (Fig. 1A). Four treatments were assigned at random to the plots (six plots per treatment; approximately 50 trees per plot). The treatments were: (1) thin to 3.7 m × 3.7 m (12 × 12 feet) spacing ($PCT_{3.7m}$); (2) thin to 5.5 m × 5.5 m (18 × 18 feet) spacing ($PCT_{5.5m}$); (3) thin to 3.7 m × 3.7 m (12 × 12 feet) spacing plus pruning ($PCT_{3.7m}$-Prune); and (4) control with no thinning or pruning (Control). The azimuth and distance from a corner of each stand to the approximate center of the stands was determined and permanently marked with a piece of rebar installed for plot center. The plots were thinned using standard silvicultural practices that are aimed at retaining trees with the most desirable features (i.e., straight bole, conical crown, etc.). Thus, the thinning treatments likely removed trees that had damage from Elytroderma or other diseases. For the stands that were pruned in addition to the thinning treatment ($PCT_{3.7m}$-Prune), the maximum height of branches to be pruned was the lesser of 8 feet or ½ of the total height of the tree.

2.3. Pre-treatment data

Pre-treatment data were collected in July 2004 after disease symptoms had fully developed. The boundary of a 0.135 ha (1/3 acre) circular plot was flagged (radius equal to approximately 18.3 m (60 feet)), using the rebar as the center of the plot. Fifty potential crop trees were located by walking around this plot starting from the north and moving clockwise, spacing approximately 3.7 m (12 feet) between each tree, and keeping within the flagged margins. If fifty potential crop trees could not be located within the flagged margins of the plot, then the margin was expanded until fifty trees were located. These trees were permanently tagged at approximately 1.37 m (4.5 feet) facing plot center in the control plots, and temporarily flagged in the plots scheduled for treatment. Data recorded for each tree included the following: diameter at breast height (dbh); total tree height; crown ratio; crown class; lowest live crown height; any damage and severity of damage; and Elytroderma infections. Each crown was rated for systemic infections (indicated by the presence of witches' brooms) using the Hawksworth rating system for dwarf mistletoe infections (Hawksworth, 1977). The live crown was divided into thirds: lower, middle, and upper. Each third received a rating of 0–2, where 0 = no systemic infections; 1 = ½ or less of total branches have systemic infections; 2 = more than ½ of the total branches have systemic infections. The overall tree-level rating was the sum of the values for the three sections and ranged between 0 and 6 with larger values corresponding to more severe infection.

Changes in Elytroderma infection ratings were systematically evaluated across measurement years to identify a measure of the disease. Some practical challenges with the ratings described above were that: (1) the ratings are subject to inter-observer bias, and thus might vary from one year to another since different crews rated Elytroderma symptoms; (2) the distribution of ratings is highly skewed towards lower values, with only a few trees assigned ratings greater than 4. Thus, for further analyses, Elytroderma severity estimated from the rating system were converted to binary Elytroderma presence or absence; namely if a tree's Elytroderma rating was >0, Elytroderma was classified as present and otherwise as absent.

2.4. Post-treatment data

Thinning and pruning of the stands were accomplished with a

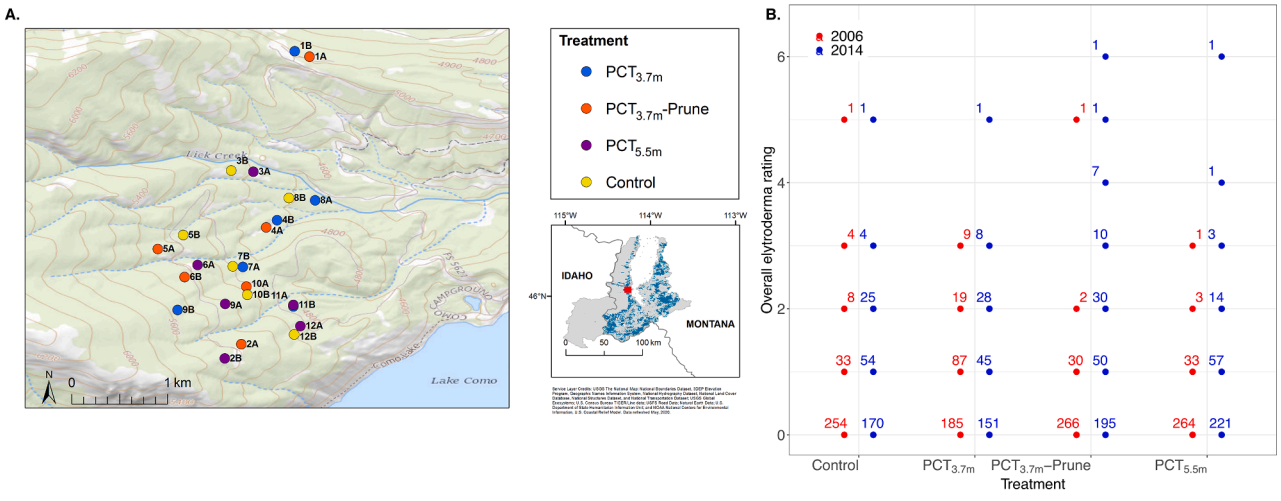

Fig. 1. (A) Geographic locations of ponderosa pine plots near Lake Como in the Bitterroot National Forest in western Montana. Plot colors indicate the treatment applied. 12 = 3.7 m × 3.7 m (12 ft. × 12 ft.) residual spacing, 12P = 3.7 m × 3.7 m (12 ft. × 12 ft.) residual spacing plus pruning, 18 = 5.5 m × 5.5 m (18 ft. × 18 ft.) residual spacing, C = control (no thinning). Inset map shows the location of the study area in red, the Bitterroot National Forest boundary in gray, and the cumulative mountain pine beetle (*Dendroctonus ponderosae*) (MPB) impacts from 1999 to 2015, as assessed via aerial detection survey, in blue. (B) Distribution of tree-level elytroderma disease ratings in 2006 (red) and 2014 (blue) for each treatment group. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

contract crew and were completed by September 2004, at which time post-treatment data were collected. The control plots did not receive a treatment, and thus did not need data collected again in 2004. For the treatment plots, the margin of a 20 m radius circular plot (0.135 ha) was again flagged, using the rebar as the center of the plot. Fifty crop trees were located by walking around this plot starting from the north and moving clockwise and keeping within the flagged margins. If fifty trees could not be located within the flagged margins of the plot, then the margin was expanded until fifty trees were located. These trees were permanently tagged at DBH facing plot center. Data recorded for each tree were: DBH; total tree height; crown ratio; lowest live crown height; and any damage and severity of damage. Crown-class was determined to have no value after treatment, so it was not included in the post-treatment data collection. Incidence of Elytroderma, which is estimated as the fraction of trees infected (the number of infected trees divided by the total number of trees) within each stand/plot combination, was not evaluated at this measurement.

2.5. Remeasurement

The first remeasurement was not done until July 2006, to allow for a complete infection cycle of *E. deformans* to occur after stand treatment. Trees were monitored for their status (healthy, Elytroderma infected, dead) every year from 2006 to 2014. Some of the tree characteristics (such as diameter at breast height, etc.) were measured in 2004 (before treatments were applied), and then again in 2009 and in 2014. The goal of the study was to establish if the treatments influenced the incidence of Elytroderma between 2006 and 2014.

3. Statistical analysis

3.1. Model description

Modeling assessed whether the rate of change in Elytroderma incidence from 2006 to 2014 was influenced by treatments. All trees that died by 2014 were removed from analysis to avoid potential confounding due to the trees that did not have exposure for a full 10-year time period that could potentially contract Elytroderma infection. Specific information regarding the cause of mortality was not recorded for individual trees except for those that died from MPB. Thus it is not known whether the mortality of these trees was directly related to Elytroderma infection or was caused by some other agent. An initial infection incidence baseline was estimated as the fraction of trees infected by Elytroderma within each assigned stand in the 2006 treatment application year. To evaluate the treatment effects, the change in the observed infection incidence was evaluated for each tree by examining whether Elytroderma was: (1) present in 2006 and absent in 2014; (2) present in 2006 and present in 2014; (3) absent in 2006 and absent in 2014; and (4) absent in 2006 and present in 2014. Since the initial (2006) and the final (2014) observed infection incidences were both binary presence/absence values, these four cases represented all possible changes in the observed infection incidence.

Finally, to test for differences in treatment effects, we assigned the following numerical score values to the change in the observed infection incidence for the k^{th} tree given i^{th} treatment within the j^{th} stand,

$$y_{ijk} = \begin{cases} -1 \text{ if infection improved (present in 2006 and absent in 2014);} \\ 0 \text{ if infection did not change (present in 2006 and 2014 or absent in 2006 and 2014);} \\ 1 \text{ if infection worsened (absent in 2006 and present in 2014).} \end{cases}$$

These values were averaged to represent the aggregate, treatment and stand-level response variable that represented change in Elytroderma incidence through the study duration. The significance test to assess treatment effect on this response was performed using incomplete block design ANOVA model $y_{ij} = \tau_i + \beta_j + \epsilon_{ij}$, where τ_i is the i^{th} treatment effect, β_j is the j^{th} stand effect, and ϵ_{ij} are the normally distributed error terms. Statistical significance of the differences in the mean change between initial and final disease incidence among the treatment groups, the simultaneous 95% Tukey Honest Significant Difference (HSD) confidence intervals for differences in treatment effects ($PCT_{3.7m}$, $PCT_{3.7m}$-Prune, $PCT_{5.5m}$, Control) were constructed using the fitted ANOVA model via function TukeyHSD in the multcomp package (Hothorn et al., 2008) in R (R Core Team, 2019).

These model results had potential for spatial bias; thus, we assessed for spatial autocorrelation of model residuals. First, tree-level residual terms were averaged for each plot. Then the planar Earth approximation was used to generate a proximity matrix of inverse weights, where plots in proximity to each other had higher weights. Spatial autocorrelation was tested using Moran.I() function in R package ape (Paradis and Schliep, 2018).

3.2. Dead trees data analysis model

To clarify the effects of various treatments on Elytroderma incidence, the dead trees were eliminated from the models described in Section 3.1. However, the data collected on the dead trees observed in each of the plots (excluded from the statistical analysis of treatment effects) allowed us to investigate: (1) the relationship between the cause of death (MPB vs. other causes) and the Elytroderma infection status; and (2) the effect of the geographic location of each stand on the number of dead trees. The two-way contingency tables summarizing the number of healthy and Elytroderma-infected trees that were killed by MPB versus other causes were first constructed for the trees that died in all stands and then in stands stratified by low (ranging from 0 to 0.4) and moderate (ranging from 0.4 to 0.6) 2006 Elytroderma incidence rates. The nonparametric Fisher's exact test was used to test the null hypothesis of no nonrandom association between two categorical variables versus the alternative of a nonrandom association between the variables at 5% significance level.

4. Results

4.1. Description of the data

Tree characteristics by each treatment and plot for the initial pre-treatment (2004) and final (2014) measurements are summarized in Table 1. For most plots, average tree diameter and height were similar across treatments and increased over the study duration. While most plots had no trees killed by MPB, plots with treatments $PCT_{3.7m}$ and Control appeared to be more susceptible to MPB mortality.

The center of each plot and treatments applied to it are shown in Fig. 1A. The stands are represented by numbers (from 1 to 12) and corresponding plots are identified with letters A and B following the stand number. Different colors were used to designate the randomly assigned treatments ($PCT_{3.7m}$, $PCT_{3.7m}$-Prune, $PCT_{5.5m}$, Control). The paired plots corresponding to the same stands were in close proximity to one another, suggesting that stand effects associated with geographic location need to be analyzed along with the assigned treatment effects. The Elytroderma ratings for the stands corresponding to different treatments are shown in Fig. 1B. These data illustrate the previously mentioned observation that the ratings for all stands associated with treatments are highly skewed towards the lower ratings, thus justifying the use of binary Elytroderma rating system to solely indicate disease presence or absence. The initial infection incidence within each assigned stand in 2006 is shown in Table 2. These data indicate that some stands had high initial Elytroderma infection incidence, indicating an initial pre-treatment study design effect.

The data collected for various plots from 2006 through 2014 is shown in Fig. 2. For each plot the total number of healthy, Elytroderma-infected, and dead trees are shown. The initial number of infected trees differed for various plots with a stand effect evident (i.e. for majority of stands, plots within the same stand had similar numbers of initial Elytroderma infection). It is important to note that treatments assigned to

Table 1
Mean ± S.E.M. tree attributes by treatment and plot replicate for initial pre-treatment (2004) and final (2014) measurements.

Treatment	Plot ID	2004 Diameter (cm)	2014 Diameter (cm)	2004 Height (m)	2014 Height (m)	Trees Killed by MPB (2004–2014) (number (%))
$PCT_{3.7m}$	11A	15.1 ± 0.6	13.5 ± 0.9	9.1 ± 0.3	10.8 ± 0.6	36(72)
$PCT_{3.7m}$	1B	9.0 ± 0.5	17.1 ± 0.6	4.3 ± 0.2	9.3 ± 0.3	0(0)
$PCT_{3.7m}$	4B	7.6 ± 0.5	11.0 ± 0.6	4.7 ± 0.3	7.4 ± 0.3	0(0)
$PCT_{3.7m}$	7A	13.3 ± 0.5	17.5 ± 0.7	7.1 ± 0.2	10.2 ± 0.4	11(22)
$PCT_{3.7m}$	8A	10.4 ± 0.6	14.0 ± 0.7	5.6 ± 0.2	9.2 ± 0.4	7(14)
$PCT_{3.7m}$	9B	14.3 ± 0.6	19.2 ± 0.8	7.6 ± 0.3	11.6 ± 0.5	6(12)
$PCT_{5.5m}$	11B	11.1 ± 0.5	15.7 ± 0.7	6.5 ± 0.2	9.9 ± 0.4	0(0)
$PCT_{5.5m}$	12A	12.4 ± 04	17.7 ± 0.6	7.5 ± 0.2	10.9 ± 0.4	0(0)
$PCT_{5.5m}$	2B	12.6 ± 0.5	19.4 ± 0.6	6.3 ± 0.2	10.7 ± 0.3	0(0)
$PCT_{5.5m}$	3A	8.7 ± 0.6	13.5 ± 0.8	4.8 ± 0.3	7.8 ± 0.5	0(0)
$PCT_{5.5m}$	6A	10.0 ± 0.4	14.5 ± 0.5	5.2 ± 0.2	8.2 ± 0.3	0(0)
$PCT_{5.5m}$	9A	8.1 ± 0.5	12.0 ± 0.7	4.7 ± 0.3	7.7 ± 0.4	0(0)
$PCT_{3.7m}$-Prune	10A	10.6 ± 0.5	15.1 ± 0.6	6.0 ± 0.3	9.6 ± 0.4	0(0)
$PCT_{3.7m}$-Prune	1A	10.4 ± 0.3	17.2 ± 0.4	5.0 ± 0.1	10.1 ± 0.2	1(2)
$PCT_{3.7m}$-Prune	2A	9.2 ± 0.5	14.0 ± 0.7	5.1 ± 0.2	8.2 ± 0.3	0(0)
$PCT_{3.7m}$-Prune	4A	7.4 ± 0.6	10.8 ± 0.8	4.0 ± 0.2	6.6 ± 0.4	0(0)
$PCT_{3.7m}$-Prune	5A	9.1 ± 0.5	13.1 ± 0.7	5.1 ± 0.2	7.7 ± 0.4	0(0)
$PCT_{3.7m}$-Prune	6B	9.6 ± 0.4	13.9 ± 0.5	5.9 ± 0.2	9.7 ± 0.4	0(0)
Control	10B	11.3 ± 0.6	14.3 ± 0.7	6.5 ± 0.3	9.1 ± 0.4	4(8)
Control	12B	11.3 ± 0.5	10.4 ± 0.6	6.8 ± 0.3	7.5 ± 0.4	26(52)
Control	3B	7.2 ± 0.4	10.9 ± 0.5	4.0 ± 0.2	7.2 ± 0.3	0(0)
Control	5B	6.8 ± 0.3	9.4 ± 0.5	4.2 ± 0.2	6.6 ± 0.3	0(0)
Control	7B	9.8 ± 0.6	11.4 ± 0.7	5.9 ± 0.3	8.3 ± 0.5	6(12)
Control	8B	11.1 ± 0.5	15.4 ± 0.7	5.8 ± 0.3	10.4 ± 0.4	1(2)

Table 2
Initial 2006 Elytroderma infection incidence by stand.

	Stand											
	1	2	3	4	5	6	7	8	9	10	11	12
Number of infected trees	2	1	22	25	10	22	39	37	0	5	25	1
Total number of trees	100	98	101	99	99	101	76	84	93	93	62	72
Observed initial disease incidence	0.020	0.010	0.218	0.253	0.101	0.218	0.513	0.440	0.000	0.054	0.404	0.014

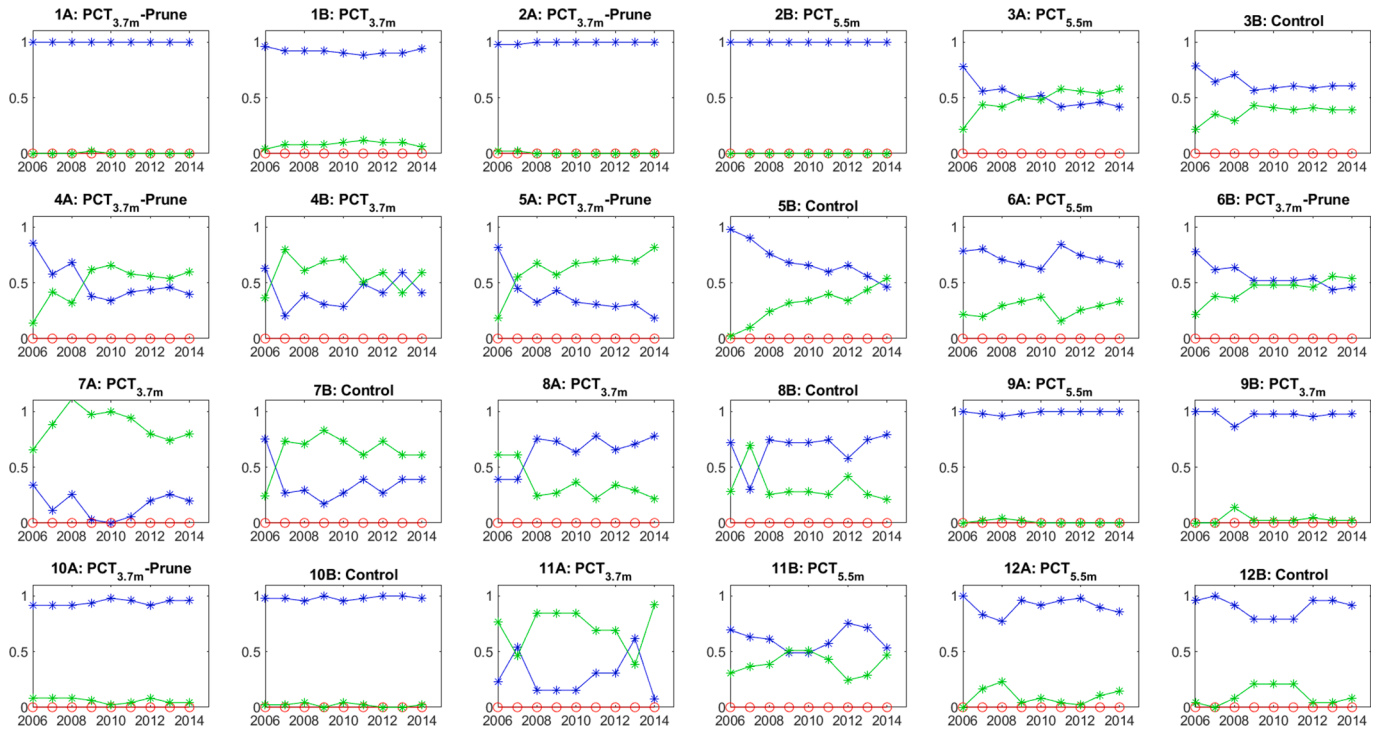

Fig. 2. Total number of the trees that are healthy (blue stars), infected with ELY (green stars), and dead (red circles) plotted for the years from 2006 to 2014. The graphs are shown for the pairs of blocks located in close proximity of each other. The plots are identified by numbers (1 through 12) and letters (A, B) shown on the top of each graph; the treatment types are indicated in the brackets. Horizontal axis represents years when the measurements were takes; vertical axis - the number of trees. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

each plot were expected to produce an observable effect over duration of the study, however at the initial year of the observations (2006) the treatments have been applied but have not yet had a chance to produce any effect. Thus, in 2006 only the stand effect was taken into account while in 2014 both stand and treatment effects were important.

The plot-level disease incidence in 2006 and 2014 were compared for the initial (Fig. 3A) and final (Fig. 3B) observed infection incidence. The treatments did not yet have any effect in 2006, and therefore Fig. 3A shows only the disease status of the plots (i.e., bubble sizes indicate plot-level incidence; the treatment status indicated by color for each plot is presented just for reference). As treatments were expected to have had time to exert their influences by 2014, Fig. 3B shows the treatments applied to each stand as well as the observed plot-level incidence. Fig. 3C shows the locations and numbers of dead trees, which died for any reason over the course of the study. The number of trees killed by MPB and the locations of corresponding plots and treatments are indicated in Fig. 3D. Importantly, no trees were killed by MPB on the plots with treatment $PCT_{5.5m}$.

4.2. Testing treatment effects

Histograms of the proportions of trees allow for a comparison of the changes in observed Elytroderma incidence between 2006 and 2014 by treatment group (Fig. 4). The color of each bar corresponds to the observed change in Elytroderma status from 2006 to 2014. The negative values of the change in the observed infection incidence ($y_{ijk} = -1$) correspond to the trees with Elytroderma present in 2006 and absent in 2014 (green category), and thus indicate that there was an improvement in Elytroderma condition. The positive values of the change in the observed infection incidence ($y_{ijk} = 1$) correspond to the trees with Elytroderma absent in 2006 and present in 2014 (red category). Zero values of the change in the observed infection incidence ($y_{ijk} = 0$) correspond to the trees with Elytroderma either present (light blue category) or absent (dark blue category) in both 2006 and 2014, and thus indicate that there was no change in the disease condition. By this logic, an effective treatment would result in more trees present in the green category (Elytroderma in 2006 and no Elytroderma in 2014) and fewer trees present in the red category (no Elytroderma in 2006 and Elytroderma in 2014). This is the case for treatment $PCT_{3.7m}$ which, compared to all other treatments, has the largest number (n = 28) and proportion (prop = 0.120) of trees in the green category together with the smallest number (n = 32) and proportion (prop = 0.137) of trees in the red category. Overall, these data represent large numbers of trees with worsening in the disease condition (red category). Specifically, there are 61 (prop = 0.240), 32 (prop = 0.137), 74 (prop = 0.252) and 49 (prop = 0.165) trees in the red category in the Control, $PCT_{3.7m}$, $PCT_{3.7m}$-Prune, $PCT_{5.5m}$, treatments, respectively. This suggests that treatment $PCT_{3.7m}$ had slightly lower increases in Elytroderma incidence over the course of the study relative to other treatments.

The Moran's I test for the spatial autocorrelation between model residuals (averaged by plot) revealed that the spatial autocorrelation was not significant at 0.05 significance level (p-value = 0.054). Table 3

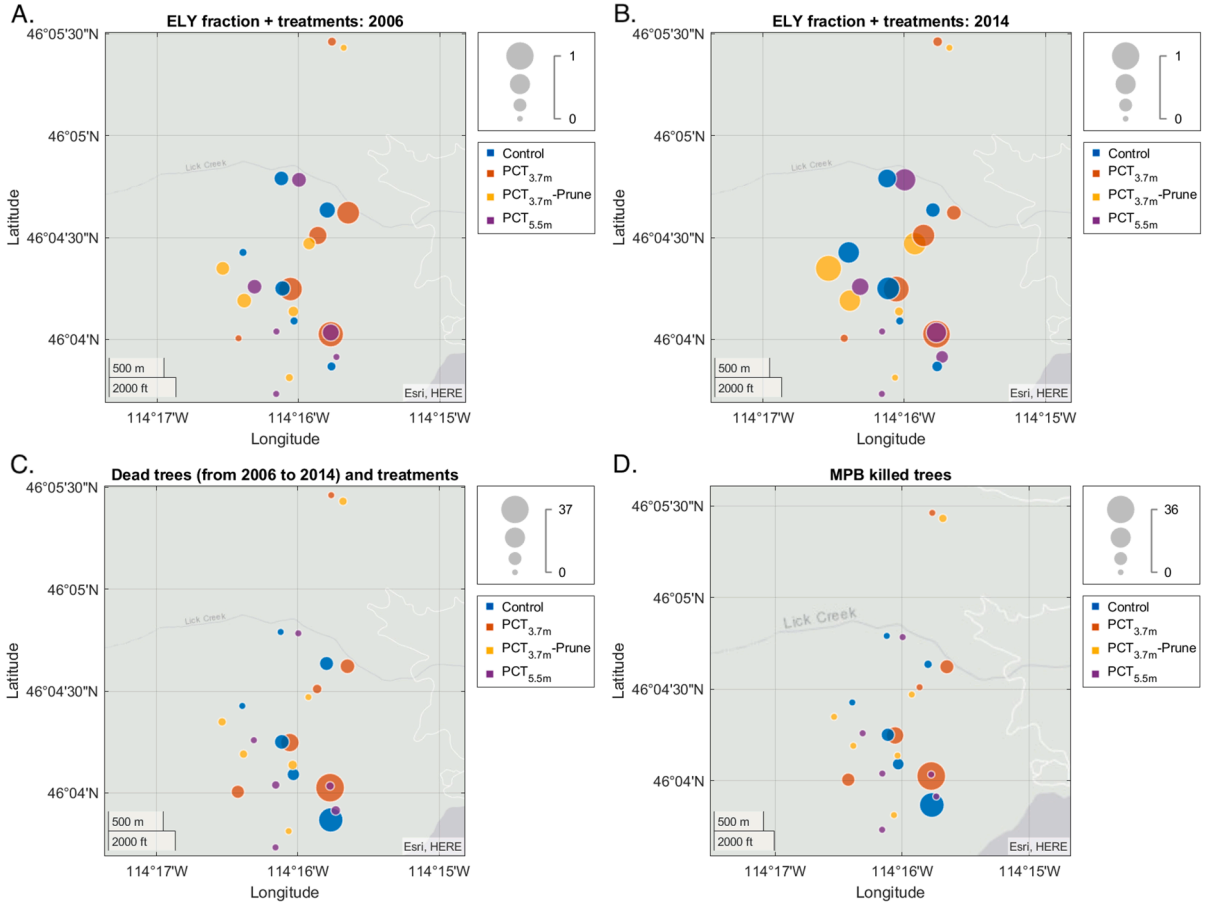

Fig. 3. (A) Geographic locations of each plot with colors representing treatments and size representing observed incidence of Elytroderma infection in 2006 (i.e., initial disease pressure measured as a fraction of threes infected). (B) Incidence of Elytroderma infection for the same plots in 2014, with colors corresponding to the treatments applied to each plot and the bubble size corresponding to fraction of trees infected. (C) Plot locations with bubble sizes representing the numbers of trees that died from any cause over the course of the study and colors representing plot treatments. (D) Plot locations with bubble sizes representing the numbers of trees killed by MPB and colors representing plot treatments.

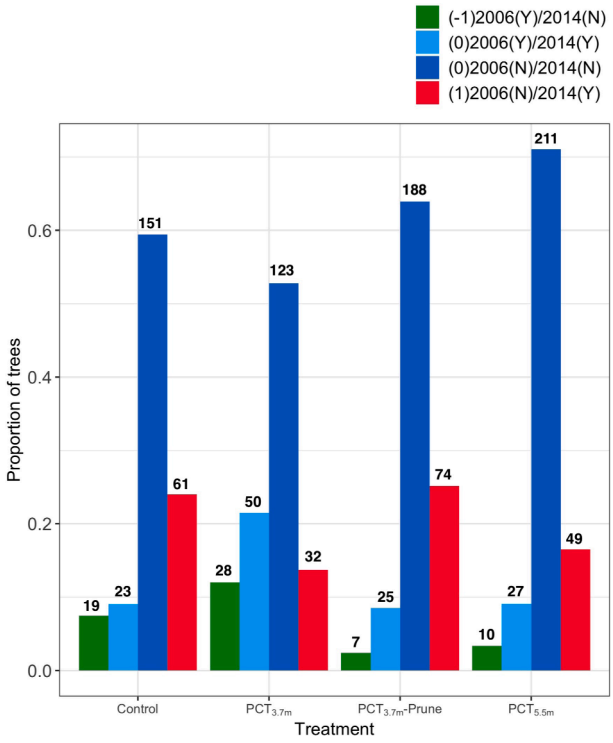

Fig. 4. Histograms showing the proportion of trees with observed elytroderma incidence change between 2006 and 2014 for control (Control), 12 × 12 feet spacing ($PCT_{3.7m}$), 12 × 12 feet spacing with pruning ($PCT_{3.7m}$-Prune) and 18 × 18 feet spacing ($PCT_{5.5m}$). Changes are colored by 4 possible outcomes: (1) infection present in 2006 and absent in 2014 (2006(Y)/2014(N); change score = −1); (2) present in 2006 and absent in 2014 (2006(Y)/2014(Y);); change score = −0); (3) absent in 2006 and absent in 2014 (2006(N)/2014(N);); change score = 0); (4) absent in 2006 and present in 2014 (2006(N)/2014(Y); change score = 1). Height of each bar corresponds to observed proportion of trees in that category and the number on top of each bar corresponds to the number of trees in that category.

shows 95% simultaneous confidence intervals for pairwise comparisons of the mean change in disease status among all treatment groups. The estimated differences for the $PCT_{3.7m}$-Prune − $PCT_{3.7m}$ (0.211, $p < 0.001$) and $PCT_{5.5m}$ − $PCT_{3.7m}$ (0.114, $p = 0.019$) comparisons are positive and significant (Table 3) indicating, on average, an increase in Elytroderma incidence in the plots that received treatment $PCT_{3.7m}$-Prune or treatment $PCT_{5.5m}$ compared to the treatment $PCT_{3.7m}$. The difference for $PCT_{3.7m}$-Prune − Control (−0.148, $p = 0.002$) (Table 3), indicates on average, a statistically significant reduction in Elytroderma incidence in the plots that received treatment $PCT_{3.7m}$ compared to control plots. On average, the plots that received the thinning treatment with 3.7 m × 3.7 m (12 ft. × 12 ft.) residual spacing had slightly lower infection incidence, as measured by the numerical scores, than plots with 3.7 m × 3.7 m (12 ft. × 12 ft.) residual spacing plus pruning, 5.5.m × 5.5 m (18ft. × 18 ft.) residual spacing, or no thinning or pruning treatment (control) (Table 3, Fig. 4). The difference for $PCT_{5.5m}$ − $PCT_{3.7m}$-Prune was negative and statistically significant (−0.097, $p = 0.043$) (Table 3), indicating slightly lower incidence of disease in plots that received treatment $PCT_{5.5m}$ compared with those that received treatment $PCT_{3.7m}$-Prune.

Table 3
Tukey simultaneous 95% confidence intervals for pairwise differences in mean Elytroderma infection score.

Treatment comparison	Estimated difference	Confidence interval	p-Value
$PCT_{3.7m}$ − Control	−0.148	(−0.253, −0.044)	0.002
$PCT_{3.7m}$-Prune − Control	0.063	(−0.036, 0.161)	0.360
$PCT_{5.5m}$ − Control	−0.034	(−0.132, 0.064)	0.809
$PCT_{3.7m}$-Prune − $PCT_{3.7m}$	0.211	(0.110, 0.312)	<0.001
$PCT_{5.5m}$ − $PCT_{3.7m}$	0.114	(0.014, 0.215)	0.019
$PCT_{5.5m}$ − $PCT_{3.7m}$-Prune	−0.097	(−0.191, −0.002)	0.043

4.3. Analyzing dead trees data

The spatial distribution of the number of dead trees (from 2006 to 2014) stratified by stand and treatment are shown in Fig. 3C. Plots containing dead trees killed by MPB together with their corresponding treatments are depicted in Fig. 3D. The total number of trees that died during the course of the study (from 2007 to 2014, considering only trees alive in 2006) was 122 pines (Table 4). These include trees killed by the MPB and those that died from other causes. In addition to the suspected cause of death in the year when the tree status changed from healthy to dead, it was also determined whether the tree status was healthy or Elytroderma-infected in the year prior to death (Table 4).

Based on the results of the nonparametric Fisher's exact test ($p = 1$), we could not reject the null hypothesis of random association between the two categorical variables, against the alternative that there is a nonrandom association between the variables at 5% significance level. These results imply that the trees that were previously healthy had similar odds of being killed by MPB as those infected by Elytroderma. The estimated odds ratio was 1.0714 with the 95% confidence interval [0.4140, 2.7725]. These results suggest that within our study area, MPB did not discriminate between healthy and infected trees.

In stands with varied levels of Elytroderma incidence estimated in 2006, three specific stands (# 7, 8, and 11) had moderate initial observed infection incidence (range 0.4–0.6; see Table 2). Analysis of dead tree data for these stands, with nonparametric Fisher's exact test, led to the same result as described above for all the stands. That is, the trees within stands established with moderate initial infection rates that were previously healthy did not have significantly different odds of being killed by MPB than trees previously infected with Elytroderma. The estimated odds ratio is 1.9429 with the 95% confidence interval [0.571, 6.609]. We further compared the odds of a tree being dead for any reason over the time period of the observations for the three stands with moderate initial infection incidence (stands 7, 8, and 11), and the remaining low initial infection incidence stands (we have a total of nine such stands # 1– 6, 9, 10, and 12). The data are summarized in Table 5.

The results of the nonparametric Fisher's exact test ($p < 0.001$) indicated rejection of the null hypothesis of random association between two categorical variables ((1) elytroderma incidence category; and (2) tree status (dead/alive)), against the alternative that there is a

Table 4
Contingency table showing the number of trees that died over the course of the study, including tree status (healthy or infected) in the year prior to death, and whether the suspected cause of death was MPB or other cause(s).

		Suspected cause of death		
		MPB	Other cause(s)	Row total
Tree status in year prior to death	Healthy	63 (80.8%)	15 (19.2%)	78 (100%)
	Elytroderma-infected	36 (81.8%)	8 (18.2%)	44 (100%)
	Column total	99 (81.2%)	23 (18.8%)	122 (100%)

Table 5
Contingency table showing the numbers of trees alive or dead from any cause in two plot-level Elytroderma incidence categories.

		Tree status		
		Alive	Dead (from any cause(s))	Row totals
Elytroderma incidence category (plot)*	Low	856 (94.8%)	47 (5.2%)	903 (100%)
	Moderate	222 (74.8%)	75 (25.2%)	297 (100%)
	Column totals	1078 (89.8%)	122 (10.2%)	1200 (100%)

* Low: initial 2006 infection incidence between 0 and 0.4; moderate: initial infection incidence between 0.4 and 0.6.

nonrandom association between the variables at 5% significance level. Based on these test results, trees (including both healthy and infected) located on the stands with moderate initial Elytroderma infection incidence had higher odds of being dead for any reason than trees in the low initial Elytroderma infection incidence stands. The estimated odds ratio is 6.153 with the 95% confidence interval [4.152, 9.119].

5. Discussion

This study assessed the efficacy of pre-commercial thinning and pruning treatments for young ponderosa pine stands across a study area that had Elytroderma disease and subsequently was challenged by MPB. The fact that Elytroderma intensified across all plots during the study period (Fig. 1B, Fig. 4) suggests that the anticipated increases in growth and vigor of residual trees occurring post-treatment were not sufficient to prevent disease in previously asymptomatic host trees. However, plots that received treatment $PCT_{3.7m}$ did have slightly lower numbers of new infections (no Elytroderma in 2006 and Elytroderma in 2014) compared to the other groups (Fig. 4), which could be attributable to localized site conditions for treatment $PCT_{3.7m}$, including the initial spatial distribution of the disease or other unknown factors. Although there were statistically significant differences in mean Elytroderma infection scores among the treatment groups, the magnitude of the differences were quite small and the plots generally had comparable rates of increase in Elytroderma incidence over the course of the study. This indicates our results may not convey biological significance sufficient to influence management recommendations solely based on Elytroderma infections.

The majority of the trees that were infected at the beginning of the study were still infected 8 years after the treatments were applied (Fig. 4), which may reflect the fact that *E. deformans* causes systemic infections unlike most other foliage diseases. For example, in plots that received treatment $PCT_{3.7m}$, 78 trees were infected in 2006 and 50 of those trees were still infected 8 years later (Fig. 4). Regardless of whether conducive conditions for continued spore release and infection occur, vegetative spread of the fungus through the growth of mycelium in infected tissues usually causes a continual decline in crown condition (Childs 1968). There were also newly infected trees in 2014 that were not infected in the initial disease assessments in 2006 (Fig. 4), indicating that aerial transmission of the disease via spores also occurred. Large openings in stands are thought to be associated with intensification of disease since these openings facilitate aerial spore dispersal and new infections (Childs et al., 1971). This mechanism did not appear to have played a significant role in this study, as treatment $PCT_{5.5m}$ resulted in similar rates of disease incidence relative to the Control (Table 3, Fig. 4). Many trees that were rated as having some damage from Elytroderma in the 2006 assessment apparently recovered by the time the 2014 disease assessment was performed. While we are not aware of any experimental evidence to support a mechanistic biological explanation for this phenomenon, it was observed in another long-term study of Elytroderma (Scharpf and Bega, 1981). It could be that the trees that recovered over the course of our study did not have systemic infections, but rather only had localized infections in the foliage and/or branches that were lost between the 2006 and 2014 assessments. In addition to the loss of localized infections due to needle casting, it is also possible that the new growth that occurred between 2006 and 2014 remained disease-free, causing a substantial improvement in the appearance of the crowns, as suggested by Scharpf and Bega (1981).

Given that local weather and microclimate conditions appear to have a strong influence on the development of the disease, it is likely that the locality of the study plots was significant in producing the initial disease outbreak. The proximity of our study plots to Lake Como would have subjected the trees to prolonged periods of leaf wetness and high humidity during the spore dispersal period. Interestingly, other studies of the incidence and impact of Elytroderma have also taken place near bodies of water (e.g. Lake Tahoe) where condensation and fog may have occurred during the spore dispersal and infection period and may have contributed to conducive conditions for disease development (Scharpf, 1990; Scharpf and Bega, 1981).

Maintaining good tree spacing through thinning is generally considered an appropriate management strategy for foliar diseases caused by fungi, as it increases air flow and reduces foliar wetness (Oester et al., 2018). Like *E. deformans*, the initiation and progression of foliar diseases such as Swiss needle cast (SNC) of Douglas-fir caused by *Nothophaeocryptopus gaeumannii* and Dothistroma needle blight (DNB) of pines caused by *Dothistroma pini* and *D. septosporum* are strongly influenced by local weather conditions, with spore release and infection processes being strongly dependent upon temperature and moisture availability (Manter et al. 2005; Stone et al., 2008; Watt et al., 2011; Woods et al., 2005, 2016). However, trees with severe SNC had only a minimal growth response to thinning compared to uninfected trees or those with low SNC severity (Mainwaring et al., 2005), and thinning is generally not effective in reducing disease impacts (Oester et al., 2018). Although studies have shown that the increased spacing that results from thinning treatments can reduce disease incidence and mortality, and increase growth of residual trees, in some stands affected by DNB, these effects are minimal in areas where the environment is particularly conducive to severe disease (Bulman et al., 2016). It is generally accepted that thinning exacerbates root disease in mixed conifer forests where susceptible residuals or regeneration are exposed to inoculum from infected stumps (Cruickshank et al. 1997; Morrison and Mallett, 1996; Byler et al. 1990). However, there are mixed results regarding the effects of thinning in ponderosa pine stands experiencing growth loss and mortality from root disease. While some thinning studies have shown results similar to those for mixed conifer stands (Shaw et al. 1976), others have shown little or no effect from thinning in ponderosa pine stands infected with *Armillaria* (Filip et al., 1989, 2009).

Pruning has also been investigated as a treatment for various foliar diseases caused by fungi. Though studies of the efficacy of pruning for the control of DNB have had mixed results, it appears that if there are any benefits, they are generally short-lived (Bulman et al., 2016). However, pruning has been effective in the management and restoration of inland western white pine (*Pinus monticola*) stands infected with the blister rust fungus (*C. ribicola*) (Schnepf and Schwandt, 2006; Schwandt et al., 2010; Zambino, 2010). The blister rust pathogen shares some key epidemiological characteristics with *E. deformans*. For example, both *C. ribicola* and *E. deformans* infect trees through needles and cause

systemic infections that spread into branches and stems. These fungal pathogens are also similar in that spore release and infection are strongly influenced by local microclimatic conditions, including moisture availability and airflow, and thus most new infections occur in the lower crowns (Scharpf, 1990; Schnepf and Schwandt, 2006; Schwandt et al., 2010; Zambino, 2010). It was hypothesized that pruning could help slow the spread of Elytroderma by increasing airflow in the lower crown, thereby alleviating humid conditions and decreasing foliar moisture, and by removing infected branches to reduce inoculum (Childs, 1968; Childs et al., 1971; Lockman and Hartless, 2008). Conversely, our results demonstrate that, when combined with thinning, the pruning treatment appeared to promote the spread and intensification of disease and led to higher Elytroderma infection scores in the $PCT_{3.7m}$-Prune relative to an equivalent thinning treatment that did not include pruning ($p < 0.001$) (Table 3, Fig. 4). Thus, pruning was not only ineffective for reducing Elytroderma incidence in the 8-years post-treatment, but appeared to exacerbate the disease. This could be due to physiological stress created by removal of substantial portion of young pine tree crowns (up to 50%) or from the creation of openings in the understory that removed barriers to spore dispersal. However, these mechanistic hypotheses related to pruning and increased rates of Elytroderma infection require additional studies to verify.

The US Forest Service Northern Region has historically had periodic outbreaks of MPB, with the most recent impacts occurring during an outbreak that lasted from 1999 to 2015 (Fig. 1A) (Lestina et al., 2019). During that period, approximately 3.65 million ha (9 million ac) of pine forest were identified as being infested via aerial detection survey (Fig. 1A), with approximately 350,000 ha (865,000 ac) of those being ponderosa pine (Lestina et al., 2019). The study plots were located in an area that had minimal MPB activity until 2010, when an outbreak began that lasted until 2014 when the study concluded. The results of this study did not indicate direct interactions between the incidence of Elytroderma and MPB attack. Trees exhibiting symptoms of Elytroderma did not have greater odds of being attacked by MPB (Table 4). Apparently, MPBs attacked trees regardless of their Elytroderma status. This result is noteworthy given previous studies that suggested trees with Elytroderma may have a greater risk of attack by pine beetles (Childs, 1968; Scharpf and Bega, 1981). The thinning treatments were not evaluated statistically for their effects on the probability of MPB attack, as (1) MPB population pressure was not evenly distributed among treatments; (2) overall MPB rates of tree mortality were low (Table 1, Fig. 3D); and (3) *a priori* considerations for MPB were not part of the original experimental design of this study. However, it is interesting to note that none of the trees in the plots with treatment $PCT_{5.5m}$, and only one tree in the plots with treatment $PCT_{3.7m}$-Prune, died from MPB even in the cases where there was immediately adjacent bark beetle population pressure and mortality within neighboring plots within a shared stand (Table 1, Fig. 3D). This may be due to the mitigating effects of the thinning and/or pruning in creating an inhospitable localized environment with non-conducive microclimate for beetle attack (Fettig et al., 2007; Egan et al., 2016; Negron et al., 2017) or may reflect random spatial variation in the distribution of MPB population pressure that allowed those plots to avoid MPB-caused mortality. While possible, the latter seems highly unlikely given that plots where MPB activity occurred were often paired and adjacent to those unaffected stands with treatment $PCT_{5.5m}$ (Fig. 3D). However, the treatment $PCT_{3.7m}$-Prune units were all paired with other treatments that experienced minimal MPB-caused mortality and these plots may not have been challenged by localized beetle pressure.

Numerous studies have documented the ecological interactions between bark beetles, pathogens, and their conifer hosts (Schowalter and Filip, 1993). For example, there is evidence to suggest that bark beetles preferentially attack trees weakened by root disease (Goheen and Hansen, 1993; Oester et al., 2018), and may select trees infected with white pine blister rust (Six and Adams, 2007; Bockino and Tinker, 2012; Dooley and Six, 2015). The severity of the damage caused by dwarf mistletoe and insects such as MPB and *Ips* spp. in ponderosa pine stands is influenced by stand density (Klutsch et al. 2014). This association is particularly relevant for interpreting the results of our study, as there appear to be stand-level characteristics that predisposed trees to invasion by pathogens and insects. While we found no evidence of direct interaction between Elytroderma and MPB at the tree-level, the insect and pathogen did co-occur within some stands suggesting that other site factors such as localized microclimate and moisture availability may influence susceptibility to both pathogen and insect.

In the present study, sites with moderate initial infection incidence had higher odds of dying for any reason compared to trees within stands that had low initial disease pressure (Table 5). It is possible that these stands contained trees that were predisposed to disease and MPB-attack due to physiological stress and/or interactions of ambient topographic, biophysical, or microclimatic conditions that provided suitable conditions for both insect and disease agents. This suggests that identifying stands with high incidence of Elytroderma may also identify forested areas predisposed to MPB attack. While treatments did not necessarily prevent or reduce the intensification of Elytroderma within trees infected at the onset of the study, treatment $PCT_{5.5m}$, and possibly $PCT_{3.7m}$-Prune, appeared to reduce post-treatment stand susceptibility to successful MPB attack (Table 1, Fig. 3D).

A goal of this study was to provide land managers with guidelines for thinning in young ponderosa pine stands to promote resistance to multiple disturbance factors. Based on our results, there is minimal evidence to suggest thinning and/or pruning can significantly reduce intensification rates of Elytroderma within forested areas that had pre-existing infections. However, the thinning treatments (without pruning) appeared to offer slight benefits. Thinning may have sanitized trees infected with Elytroderma and other disease agents and improved airflow in the stand to reduce foliar infection. Treatment $PCT_{5.5m}$ provided additional benefits that appeared to substantially reduce post-treatment stand susceptibility to MPB-attack (Table 1, Fig. 3B). The residual spacing of treatment $PCT_{5.5m}$ also may reduce the potential for severe wildland fire behavior by reducing canopy bulk density and/or reducing fuel accumulation resulting from tree mortality (Moriarty et al., 2019). Overall, our data indicates pre-commercial thinning treatments in young ponderosa pine stands to a residual 5.5 m × 5.5 m (18 ft. × 18 ft.) spacing can improve resilience to multiple disturbance types. However, it is important to consider that this study occurred over a limited spatial and temporal extent within a dry inland ponderosa pine forest type that included mostly young stands with pre-existing Elytroderma infections. Thus, these recommendations may not be appropriate to extrapolate to other forest types or stand ages.

CRediT authorship contribution statement

Ekaterina Smirnova: Conceptualization, Methodology, Software, Visualization, Writing - original draft, Writing - review & editing. **Patrick Bennett:** Conceptualization, Formal analysis, Methodology, Writing - original draft, Writing - review & editing. **Joel Egan:** Project administration, Supervision, Conceptualization, Formal analysis, Methodology, Writing - original draft, Writing - review & editing. **Leonid Kalachev:** Conceptualization, Methodology, Software, Visualization, Writing - original draft, Writing - review & editing. **John Goodburn:** Data curation, Funding acquisition, Validation, Writing - original draft, Writing - review & editing. **I. Blakey Lockman:** Data curation, Writing - original draft, Writing - review & editing. **Cheri Hartless:** Data curation, Writing - original draft, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

This project was completed through a cost-share agreement funded by the USDA Forest Service, Northern Region Forest Health Protection program and by the University of Montana, W.A. Franke College of Forestry and Conservation. Additional support was provided by the Bitterroot National Forest. The authors would like to thank the field crews that provided support for this study as well as two anonymous reviewers for their helpful comments which led to substantial improvement of the quality of manuscript.

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