An experimental comparison of stand management approaches to sudden oak death: prevention vs restoration

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Abstract

Many coastal forests stretching from central California to southwest Oregon are threatened or have been impacted by the invasive forest pathogen *Phytophthora ramorum*, cause of sudden oak death. We analyzed a set of stand-level forest treatments aimed at preventing or mitigating disease impacts on stand composition, biomass, and fuels, using a before-after-control-intervention experiment with a re-evaluation after five years. We compared the effects of restorative management for invaded stands and preventative treatments for uninvaded forests with two stand-level experiments. The restorative treatments contrasted two approaches to mastication, hand-crew thinning, and thinning with pile burning with untreated controls replicated at three distinct sites (N=30) while the preventative treatments were limited to hand-crew thinning (N=10) conducted at a single site. Half of the restoration treatments had basal sprouts removed two- and four-years after treatment. All treatments significantly reduced stand density and increased average tree size without significantly decreasing total basal area both immediately and five years after treatments. Preventative treatments did not reduce the basal area or density of timber species not susceptible to *P. ramorum*, suggesting relative dominance of these species increased in accordance with host removal. Follow-up basal sprout removal in the restoration experiment appears to maintain treatment benefits to average tree size and may be associated with small decreases in stand density five years after initial treatment. Our study demonstrates that for at least five years, a range of common stand management practices can improve forest conditions threatened or impacted by sudden oak death.

Key words: treatments, management, sudden oak death, Phytophthora

Introduction

Emerging infectious forest diseases, often caused by the introduction of nonnative pathogens into naive and highly susceptible host populations, can generate severe population- to ecosystem-level impacts, including shifts in community composition, changes in biogeochemical cycling, and local extinction of host species (Sturrock et al. 2011, Ghelardini et al. 2016, Simler-Williamson et al. 2019). As novel agents of tree mortality and landscape-scale disturbance, these nonnative pathogens have prompted an urgent need for practical stand-management strategies that not only reduce the epidemiological potential of forest outbreaks, but also mitigate the higher-order ecological impacts associated with emerging disease including sustaining carbon storage and improving firesafe conditions.

Phytophthora ramorum, the cause of sudden oak death, has become a damaging invasive pathogen in parts of coastal California and southwestern coastal Oregon forests since its introduction circa 1990 (Rizzo et al. 2005, Hansen et al. 2008, Cobb et al. 2020). The pathogen can cause acute local tree mortality which increases dead ground fuels, reduces carbon sequestration, and challenges biodiversity conservation in the evergreen forests of coastal California, which include those dominated by redwood (*Sequoia sempervirens*) and several endemic broad leafed species (Kuljian and Varner 2010, Metz et al. 2012, Cobb et al. 2013a). However, pathogen-driven mortality is limited to tanoak (*Notholithocarpus densiflorus*) and several oak species in the red-oak clade, predominantly coast live oak (*Quercus agrifolia*). In California, sudden oak death results in mortality of aboveground tissues and most disease-killed trees resprout prolifically, a common adaptation in forests shaped by fire, which results in high density stands dominated by small stems which remain at risk of infection (Cobb et al. 2012b, 2017a, Simler-Williamson et al. 2021). Recent estimates suggest over 50 million trees have been killed as of 2019 by *P. ramorum* in an area spanning central-coastal California to Southwestern

Oregon (Cobb et al. 2020). This mortality initiates directional changes in forest composition that have the potential to increase pathogen persistence within stands and accelerate spread within the landscape (Meentemeyer et al. 2008b, Cobb et al. 2010).

Live and dead fuel accumulation is a key management concern in this region due to the threat of wildfire. Sudden oak death-related tree mortality increases dead fuels in the canopy and on the forest floor in addition to substantial increases in the number of small diameter live stems which originate as basal sprouts (Valachovic et al. 2011, Cobb et al. 2012b, 2017a). Accumulation of disease-related fuels has been shown to increase fire severity and fire-driven ecosystem impacts under some conditions (Metz et al. 2011, 2013, Cobb et al. 2012a, 2016), potentially magnifying other fire management challenges in coastal California associated with climate change (Westerling and Bryant 2008, Williams et al. 2019). Without active disease management, impacts including loss of larger trees with corresponding shifts to smaller average tree sizes and fuel accumulation are likely in many susceptible stands (Cobb et al. 2012b, Metz et al. 2012, Forrestel et al. 2015). Overlap of wildfire and disease impacts could even lead to conversion of forest to chaparral in some conditions (Wurzburger and Miniat 2014, Cobb et al. 2017b, Simler-Williamson et al. 2021). *Phytophthora ramorum* is expected to spread further into at risk, but currently uninvaded forests, therefore management strategies need to consider the costs and benefits of disease prevention alongside those for restoration of heavily-impacted forests (Meentemeyer et al. 2004, Valachovic et al. 2017, Hansen et al. 2019).

Phytophthora ramorum is a generalist pathogen with a broad host range of well over 100 native species within California and Oregon forests (Rizzo et al. 2005, Metz et al. 2011, LeBoldus et al. 2022). However, four general host classes drive epidemiological dynamics and subsequent disease management strategies (Figure 1). These host classes are best typified by

California bay laurel (*Umbellularia californica*), tanoak, coast live oak (as well as several other less common or less vulnerable oaks), and a much broader group of trees including redwood and Douglas fir (*Pseudotsuga menziesii*). In California, California bay laurel is the most prevalent and prolific supporter of sporulation. This common overstory tree in coastal forests is a major contributor to pathogen spread and mortality at stand-to-landscape levels (Davidson et al. 2008, Cobb et al. 2010, Cunniffe et al. 2016); however, the resulting transmission originates from minor leaf lesions which do not impact its growth or cause mortality (DiLeo et al. 2009). Tanoak also supports significant levels of sporulation on leaf and twig infections which contribute to regional spread and mortality, but this species also develops lethal bole cankers and represents the majority of mortality caused by the disease at the regional scale (Davidson et al. 2008, Metz et al. 2012, Cobb et al. 2020). Coast live oak, Douglas fir, and redwood are susceptible to infection, but sporulation is not supported or is epidemiologically trivial for each species (Figure 1; Davidson et al. 2005, 2011, Rosenthal et al. 2021). However, unlike redwood and Douglas fir, coast live oak also develops lethal bole cankers, which have killed nine million additional trees across the invaded region (Brown and Allen-Diaz 2009, Metz et al. 2012, Cobb et al. 2020). With the exception of Douglas fir, all of these dominant species are capable of resprouting following harvest, burning, or *P. ramorum*-related mortality.

Since the emergence of sudden oak death, disease management has predominantly focused on targeted thinning or removal of bay laurel and tanoak, which are designed to reduce local-to-regional pathogen pressure, decrease stand density, and limit oak or tanoak mortality (Cobb et al. 2013b, Thompson et al. 2016, Hansen et al. 2019). In forest stands, efforts have primarily focused on reducing host populations overall, often through ‘thin from below’ treatments which remove trees below a set size class (often below 25-30cm diameter at breast

height – 1.35m). In addition to inoculum reduction, in suitable areas, stand treatments have aimed to increase growth and dominance of redwood and other disease-resistant trees to create closed overstory conditions and thereby reduce dominance of more susceptible species through competitive effects (Harrington and Tappeiner 2009, Cobb et al. 2013b). In practice, thinning often employs hand-crews with piling of debris, pile burning when possible, or mastication when landform and budgets allow (Cobb et al. 2017a, Valachovic et al. 2017). Each of these three approaches is likely to have variable impacts on amounts of residual dead fuels and the often vigorous post-treatment and/or post-disease basal resprouting (Cobb et al. 2012b, Metz et al. 2012, Simler-Williamson et al. 2021). The effects of sudden oak death management aimed at reducing pathogen spread and mortality have been explored with modeling approaches however, we have few empirical companion studies of the effects and longevity of these actions (Cobb et al. 2017a, Valachovic et al. 2017, Filipe et al. 2019).

Here we experimentally evaluate stand interventions aimed at minimizing the ecological impacts of this infectious disease, comparing management outcomes from two experiments in Northern California: a case study of "preventative" treatment conducted in uninvaded but threatened forests and a "restoration" treatment in three invaded, disease-impacted stands. In the uninvaded context, preventative treatments were conceived to increase health of tanoak by avoiding pathogen invasion or reducing mortality should the pathogen become established. This goal was pursued by attempting to reduce tanoak density overall while shifting dominance to non-susceptible species, resulting in fewer but healthier tanoak (Cobb et al. 2013b, 2017a). In invaded stands, where tanoak mortality, fuel accumulation, and understory tanoak resprouting was extensive, restoration treatments aimed to reduce fire risk by removing disease-related live and dead fuels and create a new forest canopy by releasing wildfire resilient trees, especially

redwood. In both experimental contexts, we employed a common set of plot measurements which quantified changes in ground fuels, average tree size, and stand structure (density and basal area) by contrasting pre-treatment, immediate post-treatment (within days to months of treatment completion), and treatment conditions five years after initial treatment. This approach provides insights into immediate treatment effects (treatment quantification) as well as providing insight into treatment durability, here up to five years. In restoration treatments, we also compared two mastication treatments as well as hand-crew thinning with and without pile burning. We expected treatments to reduce stand density in each setting, but basal area was expected to be significantly reduced in prevention treatments where tanoak stems were large relative to the high density resprouting which dominated the restoration experiment prior to treatment. We hypothesized that restoration treatments would also reduce ground fuels where mastication was applied but did not expect the same benefit in hand-crew thinning unless follow-up pile burning was also applied.

Methods

Study sites

Our preventative sudden oak death experiment was located on Lacks Creek, a Bureau of Land Management (BLM) landholding in Humboldt County, CA, dominated by second growth redwood, Douglas fir, and tanoak. This combination of epidemiological classes (Figure 1) dominates the extensive at-risk forests of Northern coastal California (Meentemeyer et al. 2008a, Václavík et al. 2010, Cobb et al. 2020). The experiment consisted of a single treatment type on a contiguous 40 ha area with relatively consistent slope (6 to 20%), aspect (Northeast), and elevation (440 to 520 m asl). All tanoak stems below 25cm were cut at the tree base, cut to ~1m

lengths, and gathered in piles. Bay laurel of any size class were also cut and piled (biomass was left on site). The pre-treatment cover of bay laurel was minimal, and thus this component of the treatment was also minimal. Although tanoak removal criteria were uniform, the treatment differed across the site in terms of total stems removed due to variation in the density and basal area of Douglas-fir and redwood prior to treatment. The treatment prescription did not allow removal of the ladder species for any size classes – that is, treatments removed hosts only and were not stand improvement treatments in a broader sense. Pre-treatment measurements were conducted in early autumn 2013, treatments were applied in late autumn 2013, treatment impacts were evaluated in spring 2014 (within 6 months of plot establishment), and a follow-up survey was conducted in summer 2019, approximately five years after treatment was applied. *Phytophthora ramorum* has been established in nearby stands since 2012 but has yet to be recovered within the treated area up to the 2019 survey.

The sudden oak death restoration treatments are located within landholdings of the Marin Municipal Water District on the slopes of Mount Tamalpais in Marin County, CA. Treatments were replicated within and between three nearby but distinct sites that vary in terms of species composition: Bolinas Ridge (10.11 ha treated; ~440 m asl, 10-22% slope, East to East Northeast aspect), adjacent to the Laurel Dell picnic area (4.05 ha treated; ~565 m asl, 12-30% slope, North aspect), and adjacent to the Peters Dam (2.02 ha treated; ~230 m asl, 15-28% slope, West to North aspect). Overall, the site overstories are dominated by redwood and/or Douglas-fir and include a significant component of California bay laurel. The initial survey showed extensive evidence of *P. ramorum* killed tanoak stems with prolific resprouting from the base of these trees. This is consistent with tanoak representing a significant component of the forest overstory prior to disease emergence circa 1995 and almost complete loss of overstory tanoak by 2014

when pre-treatment measurements were conducted. Similar stand transformation by disease has been documented in comparable coastal forests stretching ~250 km up and down the coast from these sites (Maloney et al. 2005, Ramage et al. 2011, Metz et al. 2012).

Treatments included hand-crew thinning with and without burning, two methods of mastication, and an untreated reference. The two mastication approaches applied were: a distributed mastication where mulch was deposited across the site according to the movement of machinery through the area, and an approach which combined hand crew and mastication operations to concentrate mulch in a few centralized locations on each site. By design, movement of machinery or locations of concentrated mastication within each treatment area was determined solely by the machinery operators without input from researchers.

Treatments were assigned within six blocks distributed across the three sites with three blocks in the Bolinas Ridge site, two in Laurel Dell picnic area, and one block adjacent to the Peters Dam. At each site, the five treatments were randomly assigned within a treatment block and individual treatments were applied in a minimum 0.405 ha area (1ac) with a circular 500 m^2 measurement plot embedded near the center of this area. While the study design strove for equal distribution of treatments within blocks, logistical and budgetary challenges resulted in an uneven sample size of N = 5 for the piles with no burning across three blocks, N = 5 for piles with burning across three blocks, N = 5 for centralized mastication across two blocks, and N = 9 for distributed mastication across six blocks. Piles, both burned and unburned, were introduced to the study plots as part of the treatments. Each study block included an untreated reference (N = 6). Pre-treatment measurements were conducted in summer 2014 with treatments applied within days or weeks of plot establishment; post-treatment evaluation was conducted within several weeks or months of treatment establishment. Beginning in 2017 basal sprout removal

follow-up treatment was randomly assigned and applied to half of the treated block area with stratification such that this follow up was evenly distributed across treatment types.

Field data

We used a common field survey to quantify treatment effects in both the restoration (Marin) and prevention treatments (Humboldt). For both management approaches and in all sites, we established 500 m^2 circular plots. Within each site treatment was applied across a contiguous area, thus plots were constrained to ~150 m in distance from one another and at least 50m distant from a treatment edge; 300 m distance is ideal for *P. ramorum* studies, but we could not accommodate this distance and achieve a useful level of replication. During each survey, all stems ≥1 cm diameter at breast height (1.30 m) were measured, mapped, evaluated for health and *P. ramorum* symptoms. We documented previously mapped trees (genets) as resprouting or dead but did not quantify resprouting amounts, such as number of stems per genet except when these recruited into the 1 cm dbh size class. To quantify fine dead material, we applied the Brown's fuels transect measurement during each survey using three transects emanating from the plot center (30 m total survey length; Brown et al. 1981, Valachovic et al. 2011). Each transect was spaced at 120-degree intervals with the first transect established at a randomly determined azimuth. We assumed the midpoint of diameter for each fuel class (1, 10, and 100 hours respectively) and applied an average wood density calculated from a previously published dataset of wood density (Brown et al. 1981, Cobb et al. 2012a); we assumed densities of 0.41, 0.51, and 0.53 g cm^{-3} which reflect the average wood densities for woody debris in decay classes 1, 2, and 3 respectively. The selection of these wood density values reflects the assumption that decomposition rates will be more rapid for smaller diameter materials.

Data Analysis

We calculated overstory species structure for each plot. Stand metrics were restricted to live trees only and included basal area, density, and quadratic mean diameter:

$$\text{QMD} = \sqrt{\frac{\sum dbh^2}{n}}$$

This metric was selected because increases in average tree size, as measured by QMD, is listed as a goal of forest management by the California Department of Forestry and Fire Protection (CalFire) in its grants programs and policy publications (Forest Management Task Force 2021).

We analyzed the restoration and prevention experiments as completely independent datasets. Because the prevention study included only one treatment approach (piles without burning) we evaluated treatment effects with a series of one-way Analysis of Variance (ANOVA) models that include only the survey period (N=30 observations of 10 plots, df=27). These models contrast pre-treatment, post-treatment, and 5-year follow up measures and do not include any other factors or random effects. A second set of one-way ANOVA models were constructed to examine the responses of individual species (and the host "classes" that they represent; see Figure 1) to treatments by contrasting the pre-treatment and 5-year reassessment surveys (N=20 observations of 10 plots, df=18).

The restoration experiment followed the Before-After-Control-Impact (BACI) design with a random block variable (see Chevalier et al. 2019). Here, we employed a mixed linear modeling approach integrating treatment, time, and their interaction as a fixed effect. To test for differences in stand structure with and without post-treatment thinning, stand treatment and measurement period were integrated as nested random variables with post-treatment management as the fixed effect (N=12 for both sprout removal and no sprout removal treatments). For all models, we evaluated the assumptions of normal distribution of error and homogeneous variance across the range of the predictors and applied the square root

transformation on the dependent variable when necessary to meet these assumptions, specifically stand density and fine woody debris. Statistical significance was considered when $p \leq 0.05$.

Results

Prevention treatments resulted in significantly lower stand density and higher QMD in both post-treatment surveys compared to pre-treatment conditions (Figure 2; Table 1). Contrary to our expectations, we found no significant difference in stand basal area between pre- and post-treatment conditions (Table 1). The prevention treatment experiment also did not result in a significant decrease in fine fuel levels (Figure 2; Table 1). Within the prevention experiment, a species–by–species pre-post treatment comparison revealed that changes in stand density were driven by an ~80% reduction in tanoak stems; tanoak basal area was also reduced by treatment even though this did not result in statistically significant changes in basal area at the stand level (Figure 3; Table 1). Density, QMD, and basal area for Douglas-fir, redwood, and California bay laurel did not change at a statistically significant level with treatment (Table 1). Tanoak average diameter (QMD) was significantly increased by these hand crew treatments, and appears to have led to the statistically significant increase in average tree size at the stand level (Figure 3; Table 1).

Within the restoration experiment, all treatment types were initially highly effective in reducing stand density and increasing average tree diameter (QMD – Figure 4; Table 2). These treatment effects did not appear to incur a significant change in stand basal area (Table 2). Each of the individual restoration treatments altered our focal stand metrics in ways which were persistent five years after treatment. Each treatment remained significantly below pre-treatment levels and we found no evidence of significant differences between measurements of stand

structure between the post-treatment and 5-year post treatment assessments. Furthermore, we found no evidence that the body of treatments were significantly different in terms of density, QMD, or fine fuels during either post-treatment survey (immediate vs 5-year follow up) implying that treatment effects were similar in terms of these metrics despite the differences in treatment technique (Figure 4). We found ambiguous evidence ($p = 0.068$; Table 2) of lower basal area in the no-burn pile treatments relative to the untreated reference treatment and between this treatment and the piles with burns ($p = 0.044$) although these differences appear to result from pre-existing differences among plots prior to their establishment (Figure 4). Conditions in the untreated reference plots were consistent across surveys with the exception that fine fuels decreased in all treatments during the five-year timespan of the study.

Although treatment benefits were stable and persistent for five years in the restoration experiment, we found evidence that post-treatment sprout removal will play a role in maintaining these treatment conditions (Figure 5; Table 2). Across all treatments, basal sprout removal decreased stem recruitment by ~64 stems ha^{-1} yr^{-1} while also maintaining higher QMD (Table 2). Plots with basal sprout removal showed a weak trend of decreased basal area ($p = 0.062$; Table 2) even though follow-up basal sprout removal treatments target stems below the dbh measurement threshold ($\geq$ 1 cm dbh). We found no evidence that follow-up basal sprout removal influenced post-treatment fine fuel levels (Table 2).

Discussion

The experiments performed in this study were aimed at addressing two overlapping and interconnected problems facing contemporary western US forest management: wildfire and invasive biological drivers of tree mortality. Our study was specifically aimed at sudden oak

death, the most important biological driver of tree mortality in the broadleaved forests of coastal California. In the broadest sense, we sought to understand if aspects of both problems could be addressed simultaneously with common forest management interventions that may reduce live and dead fuels, and elicit stand changes which reduce biologically driven tree mortality. The initial results overwhelmingly point to positive impacts of management on both goals for the five-year post-treatment window of observation in both disease impacted (restoration treatments) and at-risk forests (prevention treatments). All treatments were effective in decreasing stem density, increasing average tree size, and accomplished these benefits without significantly decreasing basal area and prevention treatments significantly shifted stand composition in ways that are likely to delay *P. ramorum* establishment and possibly reduce mortality should the pathogen become established (Cobb et al. 2012b, 2017a).

The experimental treatments helped achieve key management goals

Our findings are a validation of calls to reduce stand densities in coastal broadleaved forests in service of fostering fire prevention and resiliency (Hurteau and North 2008, Forrestel et al. 2015, Forest Management Task Force 2021). The optimism generated by these experiments results from durable (up to at least five years) improvements to the stand density component of live fuels, beneficial changes in average tree diameter, and the accomplishment of these goals without significant costs to forest basal area. The restoration treatment may also have reduced fine dead fuels although we have overall lower confidence in this measurement given fine dead fuels decline in all treatments over time (across surveys) including in the untreated reference treatments. This pattern could not be attributed to differences in surveyors or other measurement error sources, meaning it may have resulted from an unidentified natural productivity dynamic

315 combined with decomposition; given this uncertainty we temper the confidence of this inference
316 without validation of the fine fuels data.

317 We were surprised that these stand changes were consistent between the restoration and
318 prevention scenarios given that the prevention treatments allowed for greater removal of
319 biomass, and in fact, tanoak basal area was significantly decreased by the treatments (Figure 3).
320 Our stand-level experiments present a degree of novelty in that they are some of the first longer-
321 term (5 year) reports on sudden oak death management efficacy. However, the single site (case
322 study) nature of the prevention treatments, and low replication (3 sites) of the restoration
323 treatments require additional trials to ensure the encouraging results we report here are
324 generalizable and can be applied efficiently at a larger scale.

325 As a long-term goal, our focal treatments also aim to increase growth in less susceptible
326 species which, in this case, also have the highest timber value. Although we did not find
327 evidence of increased growth of Douglas-fir and redwood as measured by changes in basal area
328 between pre-treatment and 5-year post treatment measurements, basal area and density of these
329 species were not reduced suggesting that stand composition was shifted towards these species in
330 the prevention experiment. In the ideal treatment outcome, the extent of tanoak removal would
331 be positively associated with biomass accumulation in redwood and Douglas-fir in response to
332 increased light, water, and/or nutrient availability (Harrington and Tappeiner 2009, D'Amato et
333 al. 2013, Kolb et al. 2016). Although these benefits may emerge over a longer time horizon, this
334 growth response was not evident with stem diameter remeasurement after five years. A failure to
335 initially detect these effects could easily emerge due to error associated with diameter
336 remeasurement or a slowing of forest growth overall given that drought conditions dominated
337 during the study period (Williams et al. 2022).

Regardless, host reduction in terms of density and basal area was significant, commensurate with the treatment aims, durable over five years (Figure 3), and *P. ramorum* has yet to establish within the treatment area despite an established outbreak within ~100 m of the treatment boundaries. This nearby outbreak was one of the motivating factors for the managing agency to pursue treatments at our study location (prevention treatments) which is well within the expected pathogen dispersal range (Meentemeyer et al. 2008a, Eyre et al. 2013). Encouragingly, the treatment area has yet to be invaded by the pathogen although it is impossible to definitively ascribe the lack of invasion as evidence of treatment efficacy given the high degree of inoculum variability in time and space (Davidson et al. 2011, Eyre et al. 2013, Cunniffe et al. 2016, LeBoldus et al. 2022). However, the significant reduction of transmission-supporting tanoak in the prevention treatments is expected to slow rates of invasion as well as reduce future disease impacts should the pathogen become established (Cobb et al. 2017a, 2019, Filipe et al. 2019). Lastly, lower stand densities and higher average diameter resulting in all treatments suggests these stands will be more resilient to wildfire. These findings give further support to the goal of increasing treatment applications aimed at disease, and wildfire in a broad swath of coastal California broadleaved forests.

Prevention vs restoration: the cost problem

Forest treatment costs can vary considerably with market dynamics, equipment costs, labor rates, and forest-level factors such as density, slope, and distance to roads (Rummer 2008, Abbas et al. 2013). This study did not directly quantify or integrate treatment costs although these factors are likely to influence many aspects of forest management decision making ranging from stand-level decisions to policy making. Treatment costs in our study averaged 1000 USD per 0.405 ha in the prevention treatments and 10,000 to 12,000 USD per 0.405 ha in the

restoration treatments. The limited replication of these experiments at the landscape or regional scale limits how this information can be applied. Here, treatment intensity, timing, and location – rural in the prevention experiment vs peri-urban in the restoration experiment – likely influenced these costs and we are unable to account for these in a rigorous manner. However, the extreme stand densities and prolific large woody debris generated by sudden oak death are almost certain to increase costs and difficulty by slowing workers on foot or increasing machine hours, such as the masticators employed here (Valachovic et al. 2011, Cobb et al. 2012b, 2012a). While the lack of replication restricts the potential to apply these factors elsewhere, a reasonable speculation is that sudden oak death restoration treatments will almost certainly be substantially more expensive compared to prevention treatments when disease-caused changes in forest structure are as severe as the pre-treatment conditions in our restoration experiments (Figure 4). Additionally, previous work has demonstrated that these forest structure changes can increase fire intensity, fire-driven tree mortality, and fire-associated loss of soil carbon (Metz et al. 2011, 2013, Cobb et al. 2016). Thus, even when restoration treatments are substantially more expensive, these costs may be justified by the broader set of treatment benefits. In practical terms, either restoration or preventative treatments may not be affordable for public land management agencies and other organizations without supplemental funds or innovative financing (Madeira and Gartner 2018).

Basal resprouting and the prioritization problem

While all our focal forest treatments were durable in the five-year window of the study, we also found evidence that follow-up management will be required to maintain these treatment impacts over the longer term due to the frequency of recruitment via basal sprouting, a common adaptation to wildfire in these forests (Simler et al. 2018, Simler-Williamson et al. 2021).

Follow-up basal sprout removal treatments clearly reduced stem recruitment and helped maintain gains in average stem diameter in the restoration experiment (Figure 5). Although follow-up basal sprout removal was not applied in the prevention treatments, we found very little evidence that this intervention is needed to the same degree as in disease-impacted areas. Specifically, tanoak basal sprout recruitment was virtually absent in the prevention experiment during the first five years following treatment, twelve tanoak stems reached 1 cm in diameter in a total of 0.5 ha (1.2 ac) surveyed or 1.2 stems yr^{-1} ha^{-1} compared to ~ 64 stems yr^{-1} ha^{-1} of recruitment in the restoration treatments without follow up basal sprout control (Table 2). However, in both the restoration and prevention experimental contexts all individual genets which were rated as healthy prior to treatment were documented as resprouting during the post treatment and 5-year follow up measurements. In retrospect, a quantification of resprouting such as number of stems per genet, sprout height, or basal diameter would strengthen our capacity to infer treatment outcomes to tanoak recruitment. Overstory canopy cover is much more intact in the prevention experiment and may limit understory growth of resprouts, thus the necessity of follow-up treatment may be a legacy of disease impacts; this speculation could be tested with counts of basal resprouts or other measurements in future surveys. Without better assessment of resprouting we may only fully understand this aspect of treatment efficacy with later follow-up assessments such as a 10 or even 15-year post treatment evaluation. This information has potential management implications as removal of basal sprouts also appears to reduce local inoculum buildup which could be helpful for slowing invasion and subsequent mortality (Cobb et al. 2012b, Filipe et al. 2019).

Our study also suggests the community of researchers, managers, and policy makers can begin to shift to another challenging question to resolve: which stands and landscapes should be

prioritized for treatment? The cost difference between the prevention and restoration treatments in this study suggests costs will factor into treatment prioritization particularly in light of the extensive distribution of at-risk forests in California (Cunniffe et al. 2016, Cobb et al. 2020). Furthermore, both approaches could be applied in unison through prevention treatment(s) on the margins of ongoing eradication treatments actively applied in southwestern Oregon and several isolated outbreaks in Humboldt County (Valachovic et al. 2017, Cobb et al. 2020, LeBoldus et al. 2022). In contrast, millions of people live in or adjacent to the broadly distributed coastal forests which have endured ~50 million trees killed by *P. ramorum* and several weather-driven wildfire disasters (Penman et al. 2014, Williams et al. 2019). Thus, treatments which are substantially more expensive on a per unit area will often be justified because of the protection they confer to critical evacuation routes, other critical infrastructure, and population centers. For example, our restoration experiment is located within the densely populated landscape of the greater San Francisco Bay area. In combination with slope and road access, managers are certain to find situations where our restoration treatments will be justified irrespective of per ha cost differences as well as areas where prevention treatments should be prioritized, particularly where these can enhance carbon sequestration by limiting future disease emergence (Cobb et al. 2012b, Valachovic et al. 2017, Cobb 2022).

Conclusions

Phytophthora ramorum has been and will continue to be a powerful agent of change in coastal forests in California and Oregon. Our study demonstrates that common forest fuels and disease prevention treatments addressed many of the stand-level impacts of sudden oak death without incurring significant loss of standing basal area. The results are encouraging given that the various treatments did not have a significant effect on these benefits in the five-year

timescale of the study, thus operational, cost, or logistic constraints on some treatments, such as mastication, would appear less impactful in terms of benefits in light of these results. Questions remain about prioritization, overall costs vs benefits, and durability of prevention treatments relative to restoration for sudden oak death, but it is likely these can be addressed in a campaign of broader application of forest management and disease monitoring. This study strongly supports such a campaign given that many different treatment approaches and disease contexts resulted in forest structure and host reduction changes that are likely to advance forest disease, and wildfire management goals.

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Table 1. Paired contrast differences for the prevention experiment derived from Analysis of Variance models (ANOVA; where $p < 0.1$) with measurement period effects for select stand metrics and species-level contrasts with Tukey HSD p values in parentheses.

Inference level	Stand metric			
Stand *Contrast*	Stem density (stems ha^{-1})	Basal area (m^2 ha^{-1})	QMD (cm)	Fine woody debris (Mg ha^{-1})
Pre-treatment – Post-treatment	-974 (<0.001)	ns (0.48)	11.02 (<0.001)	ns (0.15)
Pre-treatment – 5 year reassessment	-1184 (<0.001)	ns (0.16)	13.39 (<0.001)	ns (0.98)
Post-treatment – 5 year reassessment	ns (0.37)	ns (0.76)	ns (0.47)	ns (0.2)
Tanoak†				
Pre-treatment – 5 year reassessment	-888 (<0.001)	-12.84 (0.024)	11.36 (0.009)	NA
Douglas-Fir†				
Pre-treatment – 5 year reassessment	ns (0.83)	ns (0.45)	ns (0.52)	NA
Redwood†				
Pre-treatment – 5 year reassessment	ns (0.93)	ns (0.98)	ns (0.96)	NA
California Bay Laurel†				
Pre-treatment – 5 year reassessment	ns (0.73)	ns (0.28)	ns (0.28)	NA

† The post-treatment measurement was dropped in species level models (df error = 18)

ns – non-statistically significant differences (omitted)

NA – metric does not apply to the species level

628 Table 2. Paired contrast differences for the restoration experiment derived from mixed linear
629 models of treatment effects (where $p < 0.1$) for select stand metrics with Tukey HSD p values in
630 parentheses.

Model	Stand metric			
Contrast	Stem density difference (stems ha^{-1})†	Basal area (m^2 ha^{-1})	QMD (cm)	Fine woody debris (Mg ha^{-1})†
Metric = Treatment + random(Block)				
Mastication (Centralized) – Untreated	-1647 (<0.001)	ns (0.63)	13.58 (0.048)	-4.76 (0.055)
Mastication (Distributed) – Untreated	-1897 (<0.001)	ns (0.36)	18.23 (<0.001)	-3.442 (0.036)
Piles (burned) – Untreated	-1763 (<0.001)	ns (1.0)	16.35 (0.003)	-7.9 (<0.001)
Piles (no burn) – Untreated	-1355 (0.003)	27.1 (0.068)	16.99 (0.003)	-5.585 (0.034)
Mastication (Centralized) – Mastication (Distributed)	ns (0.97)	ns (0.97)	ns (0.89)	ns (1.0)
Mastication (Centralized) – Piles (burned)	ns (0.99)	ns (0.97)	ns (0.99)	ns (0.59)
Mastication (Centralized) – Piles (no burn)	ns (0.97)	ns (0.97)	ns (0.98)	ns (1.0)
Piles (burned) – Piles (no burn)	ns (0.74)	-29.04 (0.044)	ns (1.0)	ns (0.41)
Metric = Treatment + random(Block)				
Basal sprout removed – No removal	-318 (0.021)	-3.35 (0.062)	7.71 (0.01)	ns (0.87)

631 ns – non-statistically significant differences (omitted)

632 † Parameter was square root transformed

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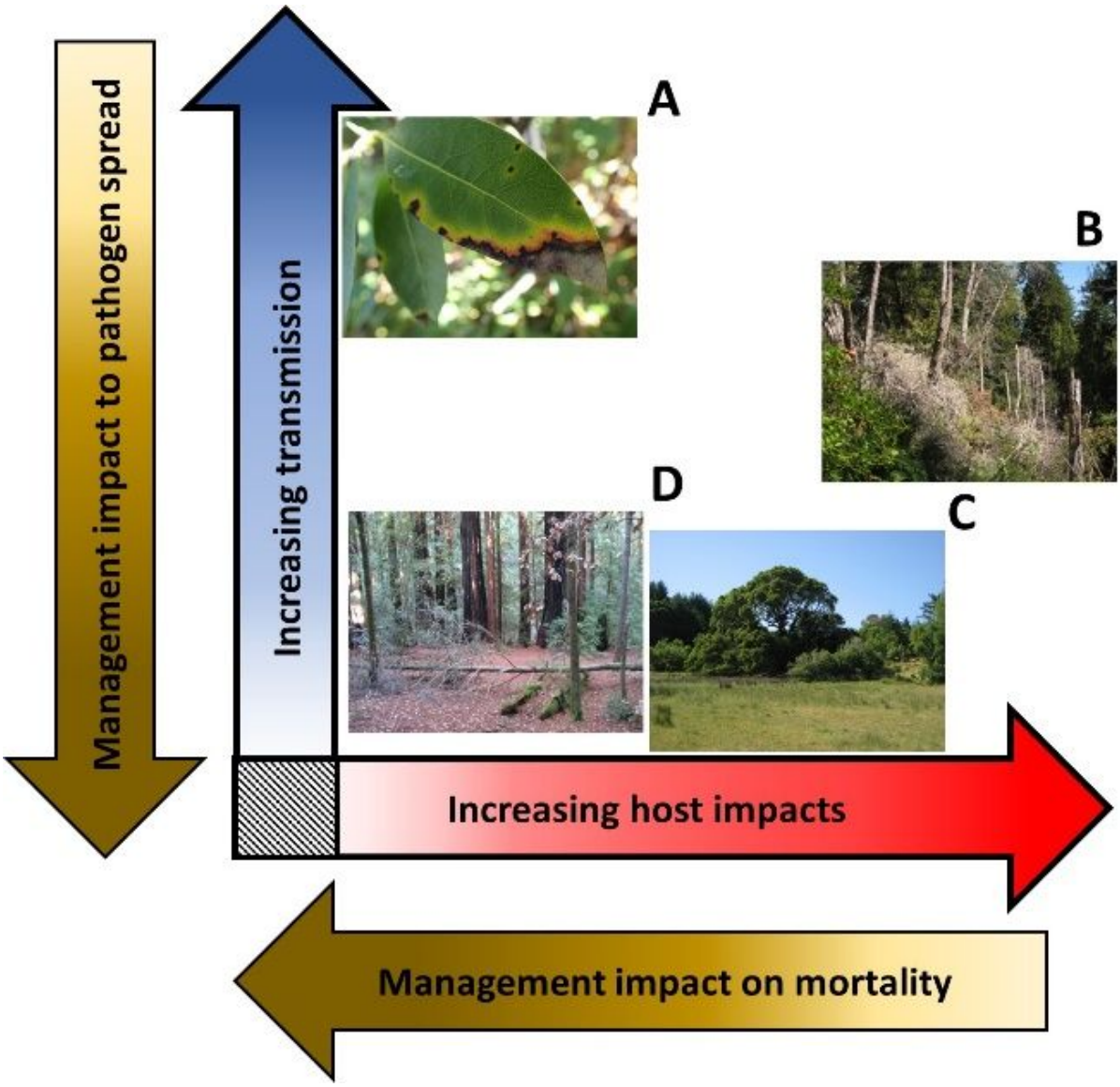

Figure 1. *Phytophthora ramorum* transmission and host impacts are uncorrelated when compared across several host classes. California bay laurel (A) supports high rates of transmission with little or no host impacts while tanoak (B) supports significant transmission and suffers the highest levels of mortality. On the low extremes of transmission, red oaks (primarily coast live oak, C) suffer mortality but do not support sporulation and a range of overstory species, particularly redwood (D), support little or no sporulation and suffer no significant host impacts. Management may target either or both goals of reducing transmission and mortality by shifting stand composition among these four broad host classes.

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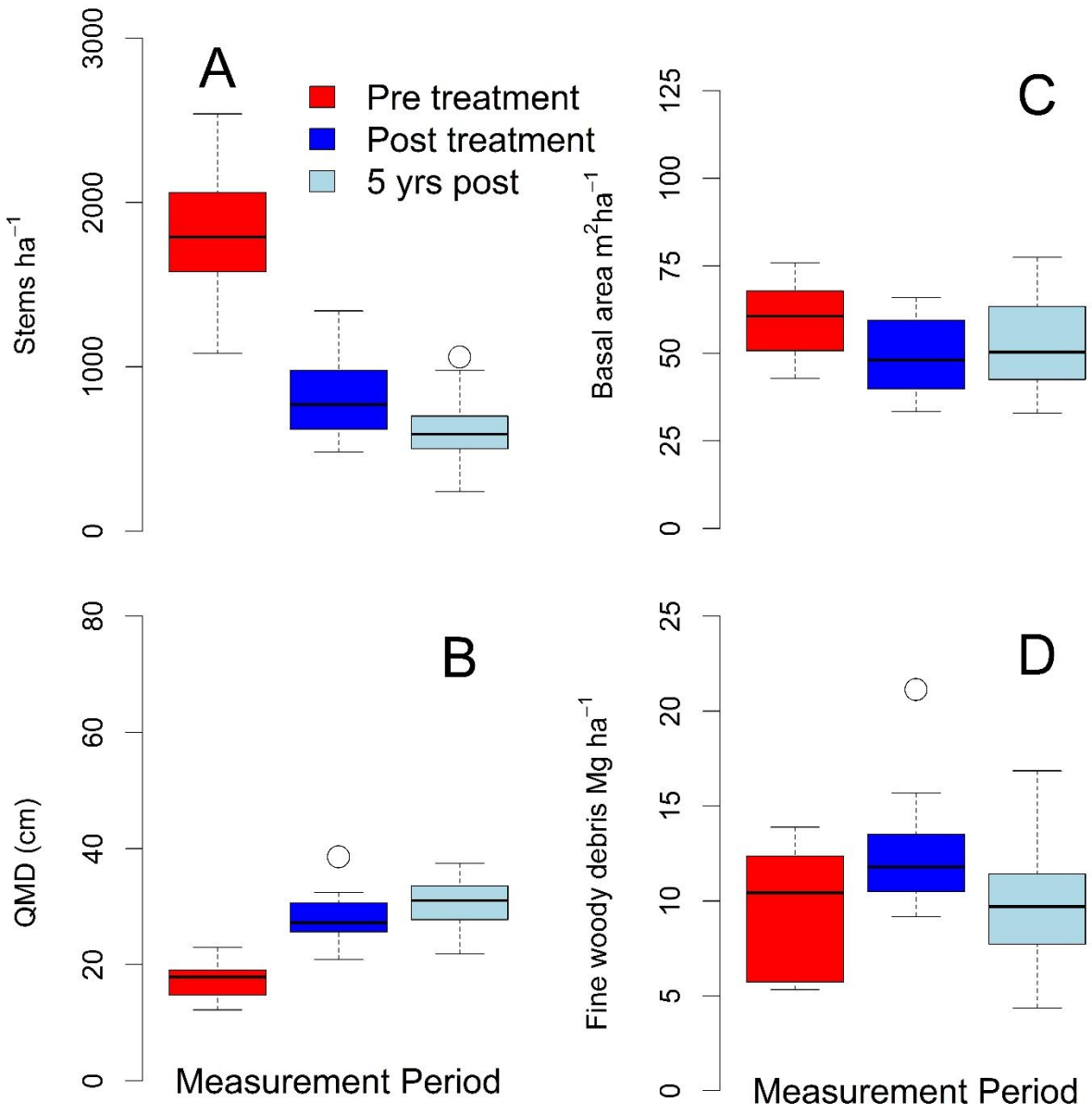

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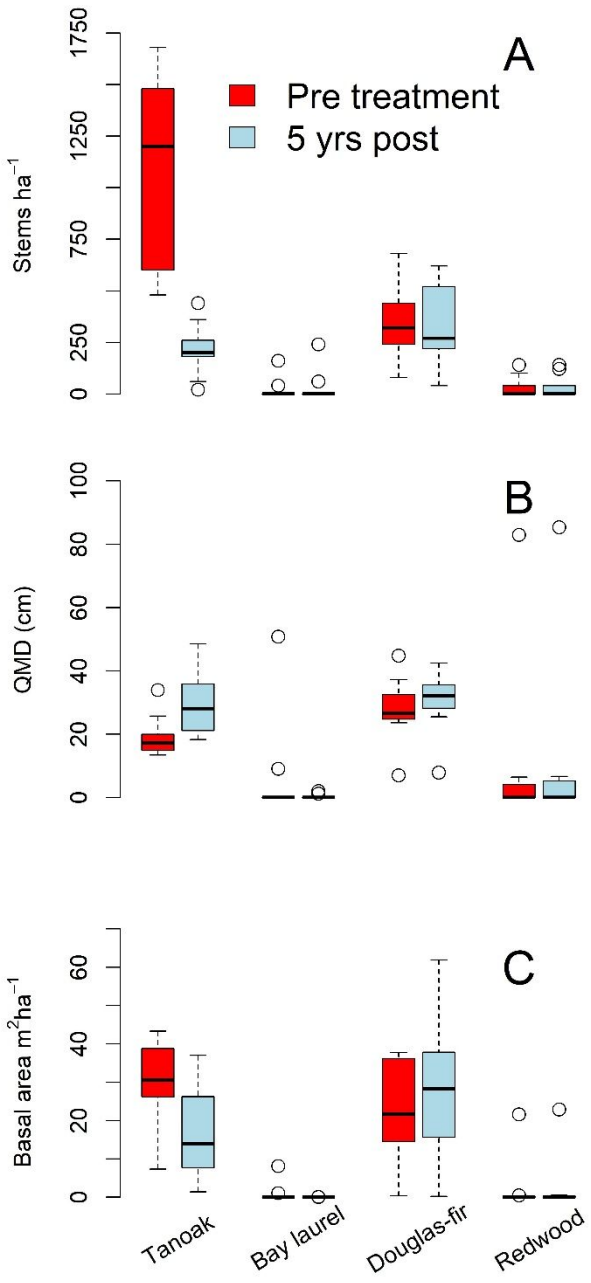
646 Figure 2. Prevention experiment forest structure contrasting pretreatment, immediate treatment
647 effects ("Post Treatment", measured within 1- 6 months post treatment), and five years post-
648 treatment ("5 yrs post") structure in terms of stand density (A), quadradic mean diameter (QMD
649 -B), basal area (C), and fine woody debris (fine fuels D). A single treatment type, hand crew
650 thinning without pile burning was applied at a single site: the Lacks Creek prevention experiment
651 in Humboldt County, CA (N=10).



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653 Figure 3. Prevention experiment treatment effects by species five years after treatments. A single
654 treatment type, hand crew thinning without pile burning was applied at a single site: the Lacks
655 Creek prevention experiment in Humboldt County, CA (N=10). The treatment was evaluated for
656 its impacts to *P. ramorum* host species (tanoak and California bay laurel) and local timber
657 species (Douglas-fir and Redwood) in terms of density (A), quadratic mean diameter (QMD –
658 B), and stand basal area (C). The treatment evaluation measurement is omitted for clarity.

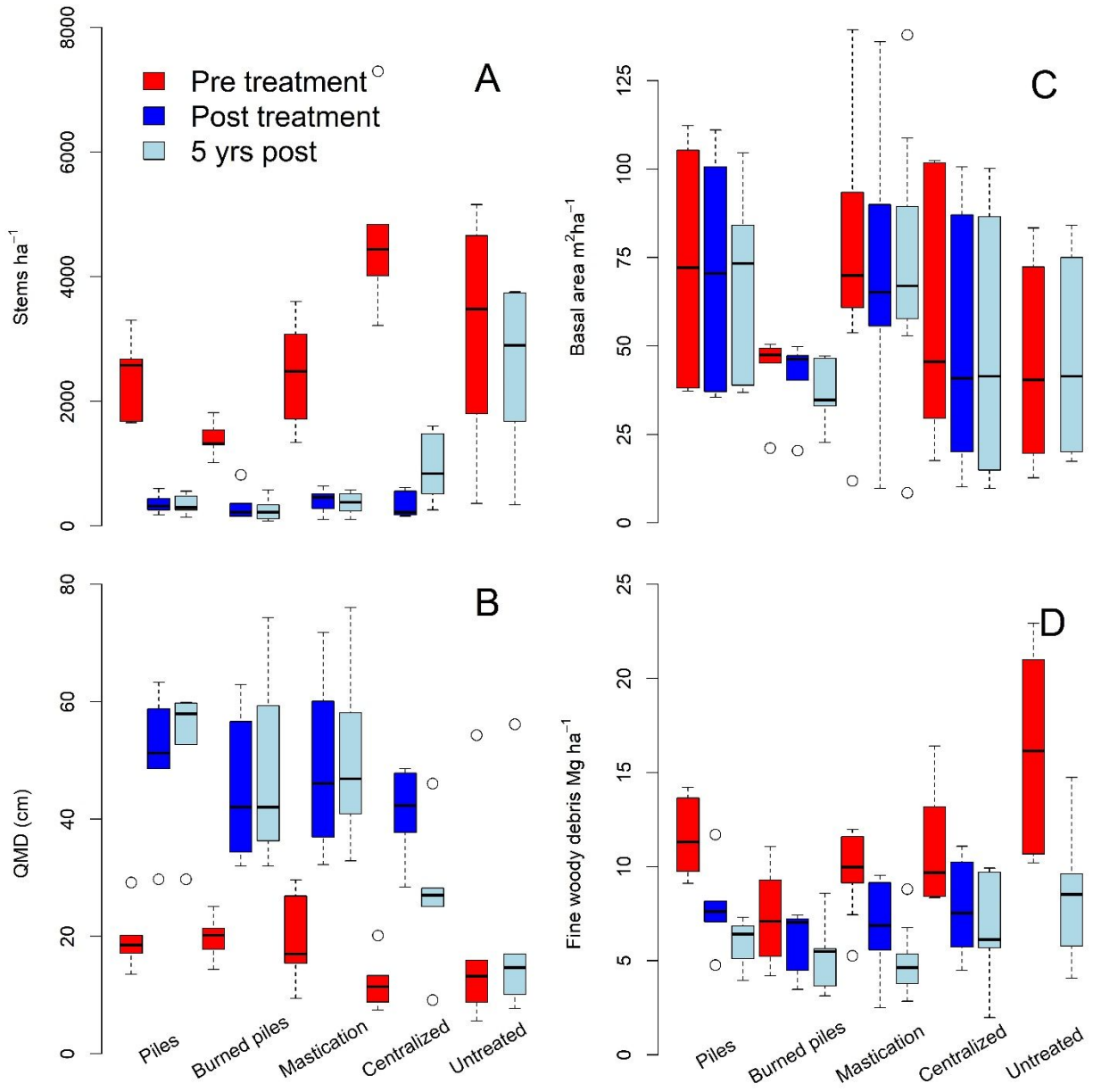


Figure 4. Restoration experiment treatment changes in forest stand structure between pre treatment, treatment, and a post treatment assessment five years following treatment represented by stand density (A), quadratic mean diameter (QMD – B), basal area (C), and fine woody debris (fine fuels - D).

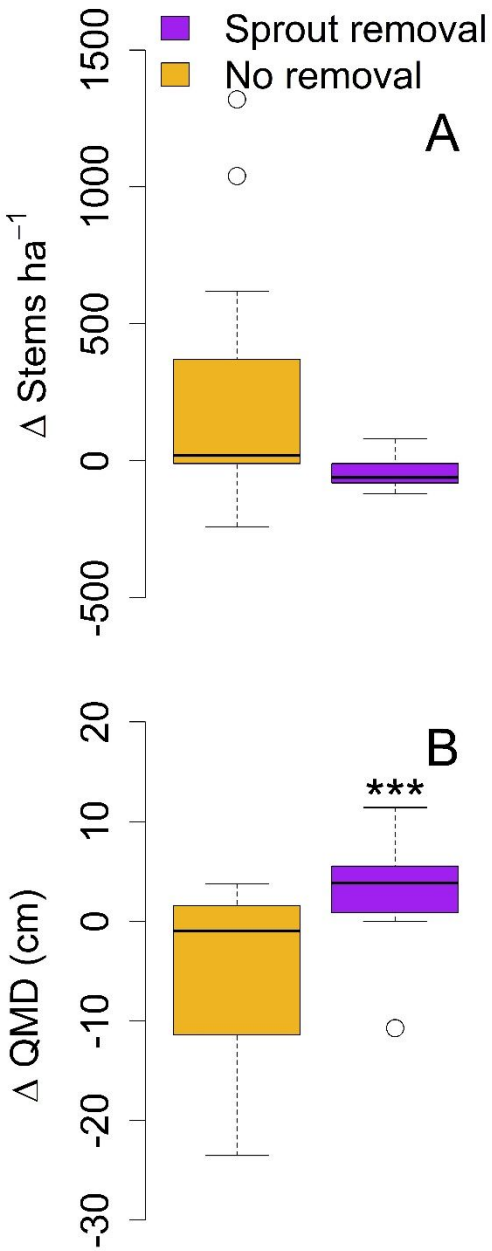
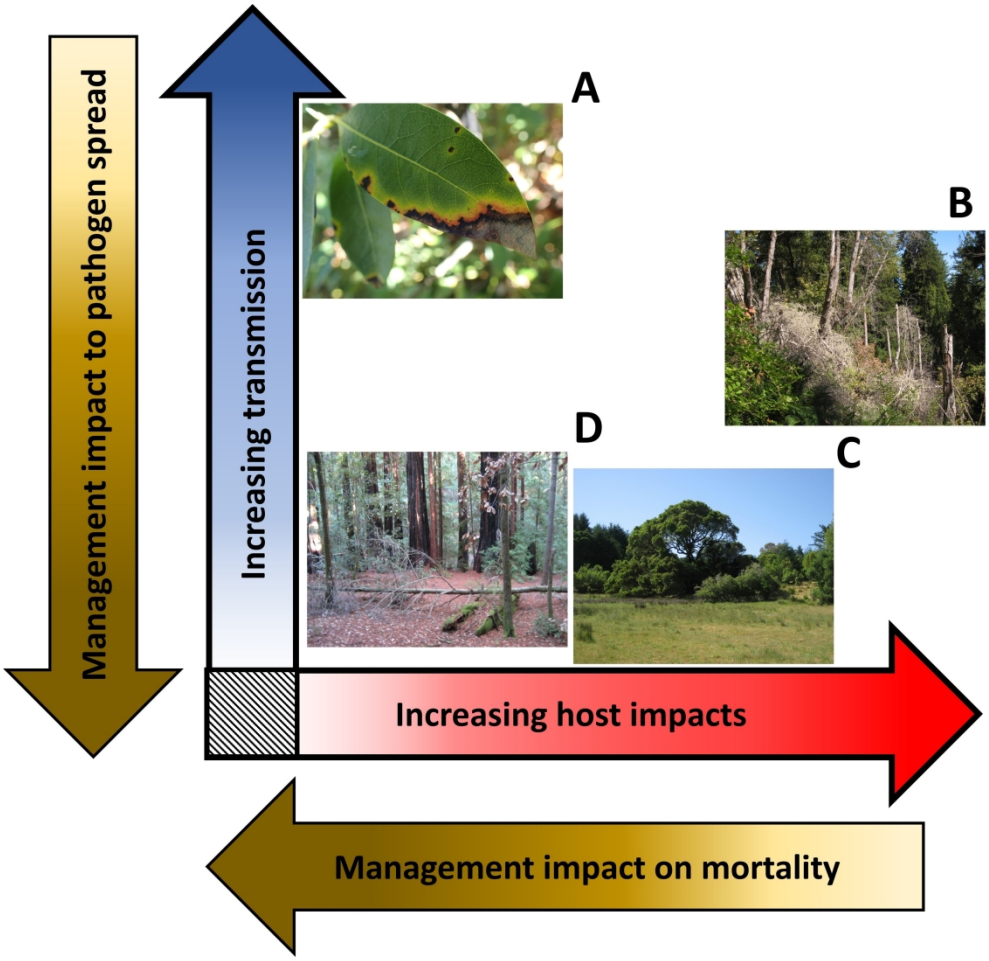


Figure 5. Post restoration basal sprout mitigation impacts represented by absolute changes to stems per ha (A), and quadratic mean diameter (QMD – B) five years after treatment application. Statistically significant differences between treatments are indicated by "***" ($p< 0.001$).



Phytophthora ramorum transmission and host impacts are uncorrelated when compared across several host classes. California bay laurel (A) supports high rates of transmission with little or no host impacts while tanoak (B) supports significant transmission and suffers the highest levels of mortality. On the low extremes of transmission, red oaks (primarily coast live oak, C) suffer mortality but do not support sporulation and a range of overstory species, particularly redwood (D), support little or no sporulation and suffer no significant host impacts. Management may target either or both goals of reducing transmission and mortality by shifting stand composition among these four broad host classes.

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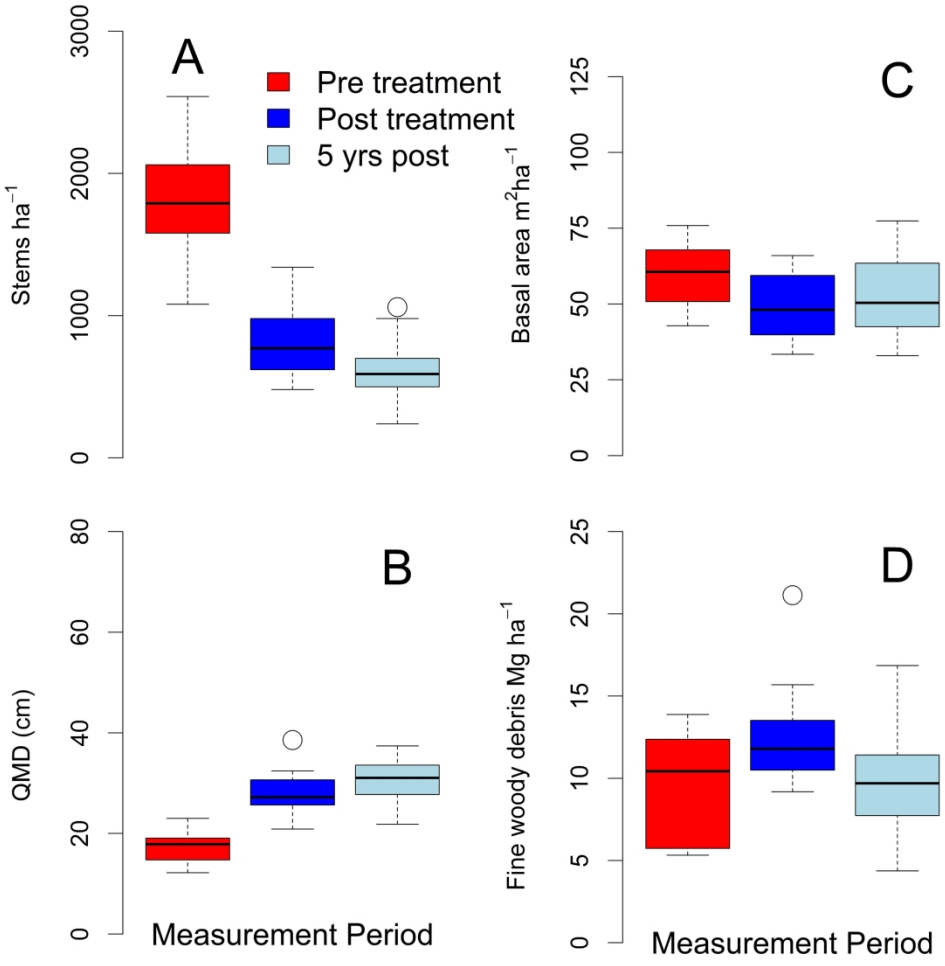

Prevention experiment forest structure contrasting pretreatment, immediate treatment effects ("Post Treatment", measured within 1- 6 months post treatment), and five years post-treatment ("5 yrs post") structure in terms of stand density (A), quadradic mean diameter (QMD -B), basal area (C), and fine woody debris (fine fuels D). A single treatment type, hand crew thinning without pile burning was applied at a single site: the Lacks Creek prevention experiment in Humboldt County, CA (N=10).

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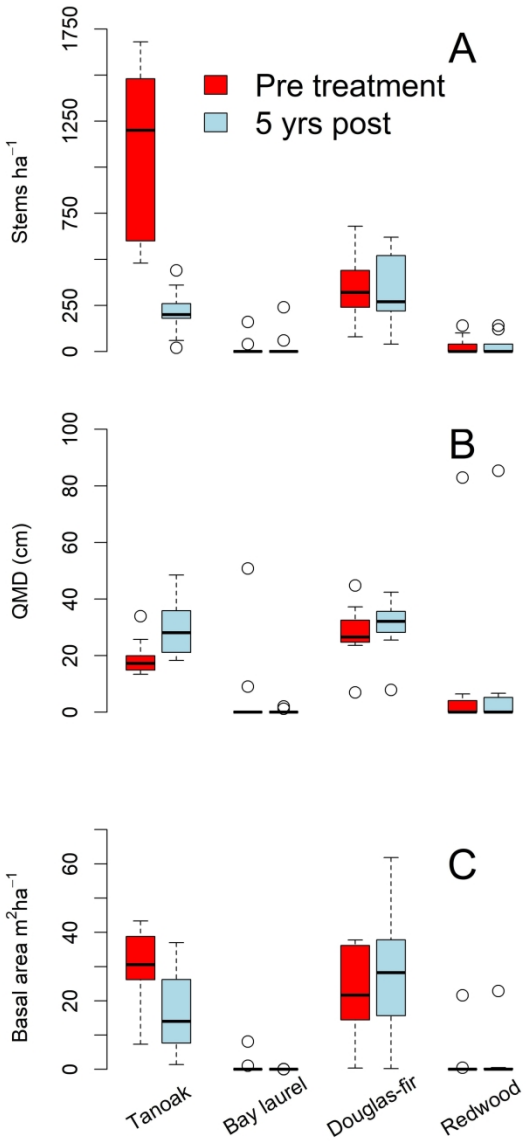

Prevention experiment treatment effects by species five years after treatments. A single treatment type, hand crew thinning without pile burning was applied at a single site: the Lacks Creek prevention experiment in Humboldt County, CA (N=10). The treatment was evaluated for its impacts to P. ramorum host species (tanoak and California bay laurel) and local timber species (Douglas-fir and Redwood) in terms of density (A), quadratic mean diameter (QMD – B), and stand basal area (C). The treatment evaluation measurement is omitted for clarity.

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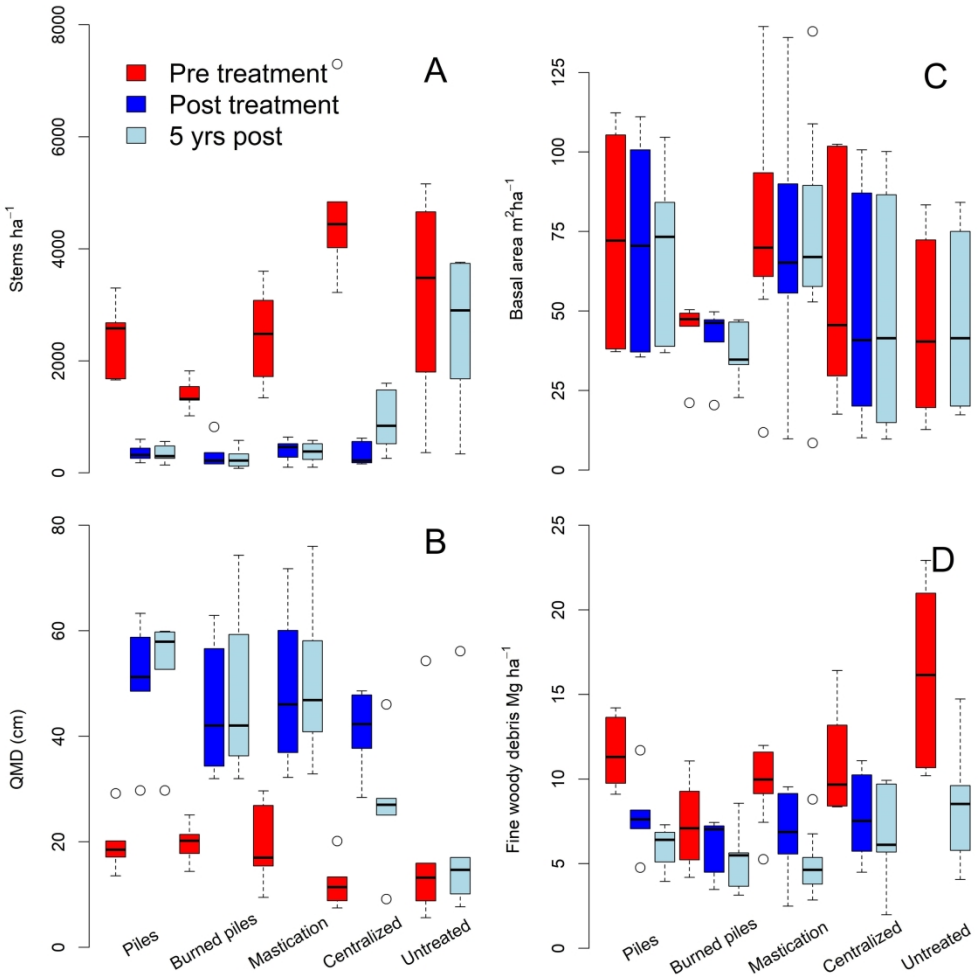
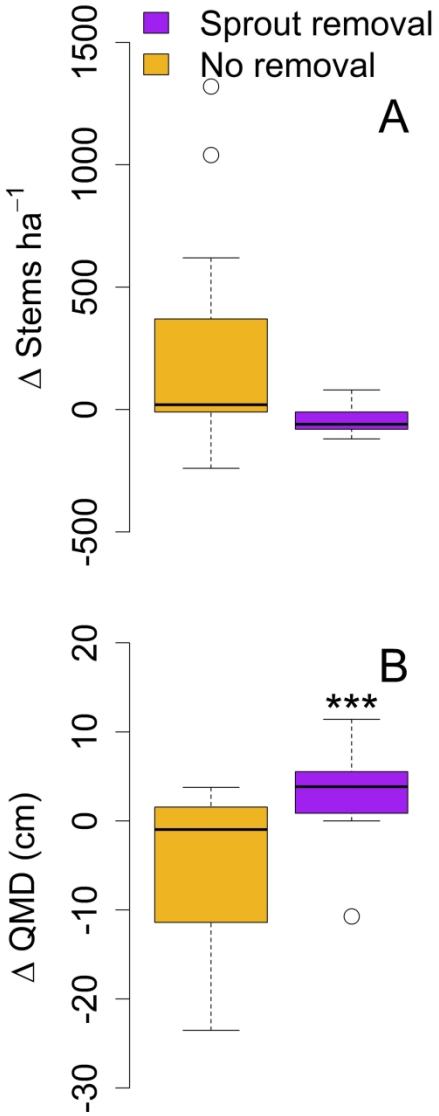


Restoration experiment treatment changes in forest stand structure between pre treatment, treatment, and a post treatment assessment five years following treatment represented by stand density (A), quadratic mean diameter (QMD – B), basal area (C), and fine woody debris (fine fuels - D).

262x262mm (300 x 300 DPI)



Post restoration basal sprout mitigation impacts represented by absolute changes to stems per ha (A), and quadratic mean diameter (QMD – B) five years after treatment application. Statistically significant differences between treatments are indicated by "***" (p< 0.001).

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