

Long Term Monitoring of the Ecological Impacts of Sudden Oak Death in Point Reyes National Seashore: 2007-2009

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EXECUTIVE SUMMARY

This report summarizes the first phase of what we hope will be a long-term study monitoring the effects of Sudden Oak Death on the redwood and Douglas-fir forests of Point Reyes National Seashore and the North District of the Golden Gate National Recreation Area. Permanent plots were established in 2007 to provide baseline data on the ecological impacts of Sudden Oak Death. These plots were re-measured in 2009. The results of this re-measurement and comparison between 2007 and 2009 data are presented here. Some of the major findings are summarized below:

- **Tanoak mortality increased throughout the park from 2007 to 2009**, in areas that were already experiencing considerable mortality in 2007 as well as in areas that were relatively unaffected.
- **The disease appears to have progressed more quickly in Douglas-fir forests than in redwood forests between 2007 and 2009.** In 2007, redwood forests were more severely impacted, but the increase in proportional tanoak mortality from 2007 to 2009 was higher in Douglas-fir forests, and by 2009, proportional mortality in the Douglas-fir vegetation type had surpassed that in the redwood vegetation type. However, dead trees in diseased redwood plots generally exhibited more advanced deterioration (e.g. structural failure of the bole), reflecting the fact that SOD became established earlier in this part of the park.
- Even if SOD-induced tanoak mortality is ultimately equivalent in redwood and Douglas-fir forests, disease impacts will differ in these two vegetation types, because of divergent baseline (i.e. pre-SOD) conditions and resulting differences in post-mortality vegetative characteristics. In particular, in areas with similar tanoak abundance, **Douglas-fir forests exhibited higher tree diversity and greater abundance of non-tanoak hardwoods (including California bay), demonstrating greater functional redundancy in this forest type, and suggesting that cascading community-level impacts are more likely in the redwood forest type.**
- At the plot scale, disease severity was not strongly related to any of the variables we tested (which included abundances of functional groups and individual tree species, average tree diameter, tree species richness, and several abiotic factors), with the exception of total tanoak basal area, which was positively related to dead tanoak basal area. **As such, our data suggest that current plot-level mortality patterns are primarily the result of introduction events and stochasticity, and that intact patches of abundant tanoak that are in close proximity to heavily impacted areas are likely to eventually experience considerable mortality.** However, the faster rate of mortality observed in the Douglas-fir forest type presents the possibility that stand-level factors (e.g. abundance of California bay, fog regime) may contribute to larger patterns of disease spread and severity.
- **In redwood forests, SOD-induced tanoak mortality has reduced canopy cover, reduced herbaceous cover, and increased non-tanoak hardwood regeneration (primarily Pacific madrone).** There is also some evidence that SOD has increased herb

species richness, total vascular plant species richness, redwood regeneration, Douglas-fir regeneration, and California bay regeneration. However, despite a statistically detectable regenerative response to tanoak mortality, current regeneration is not sufficient to fully re-occupy mortality gaps (assuming that tanoak regeneration will not persist).

- **In Douglas-fir forests, SOD-induced tanoak mortality has reduced canopy cover and increased herb species richness.** In addition, there is some evidence that SOD has increased total regeneration, tanoak regeneration, and California bay regeneration.
- **Dead and downed fuel loading increased in both redwood and Douglas-fir forest types, but increases were greater in redwood forests.** This is likely related to the more advanced degree of deterioration of dead trees in redwood stands.

1. INTRODUCTION

Sudden oak death (SOD) is a non-native forest disease which is caused by the pathogen *Phytophthora ramorum*. This pathogen causes extensive mortality of tanoak (*Notholithocarpus densiflorus* syn. *Lithocarpus densiflorus*), the most susceptible host, and also kills some oaks in the red oak group, including coast live oak (*Quercus agrifolia*; Rizzo et al. 2005). Sudden oak death symptoms were first noticed in Marin County in the mid-1990's, but it was not detected at Point Reyes National Seashore (PRNS) and the North District of Golden Gate National Recreation Area (GGNRA-n) until 2003. Since then it has spread throughout PRNS and GGNRA-n, killing many tanoaks and coast live oaks. According to US Forest Service estimates based on aerial surveys, close to 4,000 acres of the park are suffering from SOD-related mortality. Based upon random field sampling in PRNS and GGNRA-N, approximately 64% of redwood forests, 45% of coast live oak forests and 24% of Douglas-fir forests were infested as of 2007 (Moritz et al. 2008).

The widespread decline of SOD-affected species such as tanoak and coast live oak will almost certainly result in both short- and long-term ecological impacts. A significant decrease in acorn production is likely to affect native animal species that feed upon acorns, as well as to cascade up the food chain to keystone species such as the northern spotted owl (Courtney et al. 2004). Decline of tanoak and coast live oak may also lead to serious structural changes and the loss of crucial wildlife habitat. Major reductions in the populations of common tree species could facilitate the establishment of exotic plants that were previously unable to compete, as well as trigger shifts in native plant communities. Widespread mortality and short-term increases in fuel loads may also affect the frequency and/or severity of fire or other disturbances, and could even lead to alternative stable states. In addition, several recent publications have indicated the possibility of accelerated erosion and nutrient depletion, greater landslide risk, loss of native animal diversity, invasion by exotic animals and microorganisms, disruption of mycorrhizal networks, decline in aesthetic and recreational values, and local extinctions of host species (Meentemeyer et al. 2004, Davidson et al. 2005, Maloney et al. 2005, McPherson et al. 2005, Rizzo et al. 2005).

This report quantifies some of the impacts of SOD at PRNS and GGNRA-n, and details how these impacts have changed from 2007 to 2009. The data we present are from the first re-measurement phase of a long-term monitoring project aimed at quantifying the impacts of SOD-induced tree mortality. Permanent plots were established in redwood and Douglas-fir forests within PRNS and GGNRA-n in 2007 (Moritz et al. 2008). During the summer of 2009 these plots were revisited in order to assess changes to forest structure, composition, regeneration, and fuel loading.

The primary objectives of this research were to detect short-term impacts and predict long-term impacts of SOD-induced tanoak mortality within PRNS and GGNRA-n. This study focuses specifically on mortality of tanoak, as opposed to other SOD-impacted species such as coast live oak, because previous research has identified tanoak as the most susceptible species (Rizzo et al. 2005) and our observations from the first phase of this project (Moritz et al. 2008) indicate that tanoak is experiencing the most substantial mortality within the park. We collected data on mortality and SOD symptoms, forest structure, regeneration, species richness, and fuel loading, within plots randomly located in redwood (*Sequoia sempervirens*) or Douglas-fir (*Pseudotsuga menziesii*) forest types. Plots were stratified by both vegetation type (redwood vs.

Douglas-fir) and disease status (“Healthy” vs. “Diseased”), in order to assess and compare the impacts of SOD on both of these forest types.

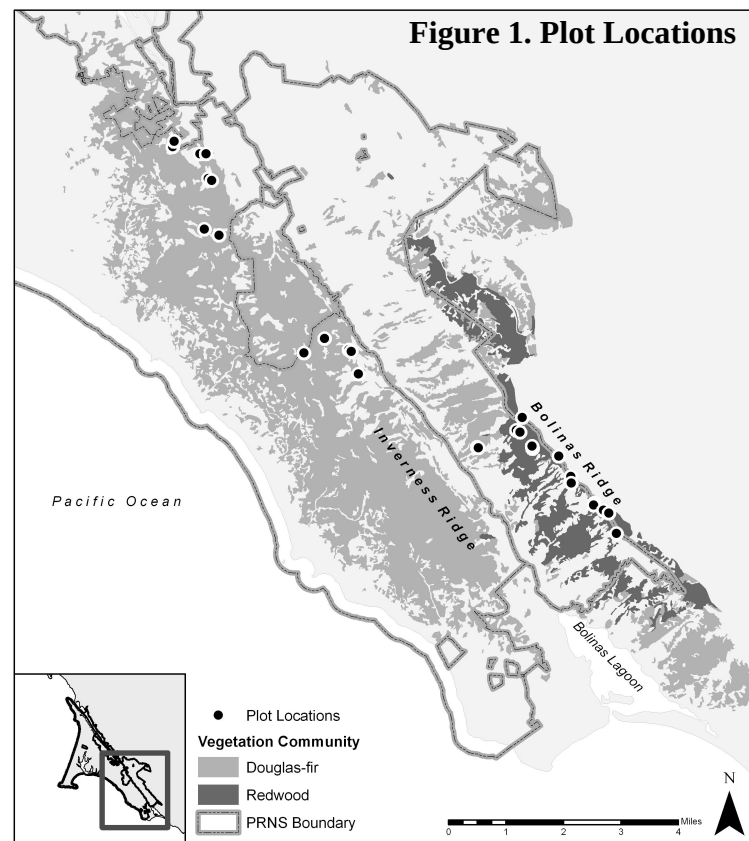
2. METHODS

2.1. Study Site

Point Reyes National Seashore (PRNS) is located on the California coast approximately 40 miles north of San Francisco (Figure 1). The National Park Service administers both Seashore lands and the North District of the Golden Gate National Recreation Area (GGNRA-n) as one unit, hereafter referred to collectively as PRNS, “the Seashore”, or “the park”. The 35,631 hectare (88,046 acre) Point Reyes peninsula makes up most of the area of the Seashore. Major geologic features include Inverness Ridge to the west and Bolinas Ridge to the east, which both run northwest to southeast and are separated by the San Andreas Fault. The Seashore straddles the fault, which is the boundary between the Pacific and North American tectonic plates. Primary vegetation types of PRNS include mixed evergreen, bishop pine, Douglas-fir, and redwood forests, coastal scrub, and California annual grasslands. Grazing for dairy and beef production, an historic land use, still occurs on portions of the Seashore. Climate at PRNS is Mediterranean in character, with dry summers and cool wet winters, and a significant fog-marine influence. Temperatures range approximately from 5-25° C (low 40’s to mid 70’s F) in the summer months, and from 3-18° C (high 30’s to mid 60’s F) in winter months. Average annual precipitation ranges from 51 cm (20 in) near the coast to as much as 102 cm (40 in) near the Bear Valley visitor center, with additional precipitation provided by fog drip.

2.2. Plot selection

Our area of interest was generally defined as forested land (either redwood or Douglas-fir associations), with a substantial tanoak component, where *P. ramorum* infection is either known or highly probable. The specific areas designated for potential sampling were identified as part of previous related research (the “rapid assessment” conducted in June and July of 2007; Moritz et al. 2008) and by using Geographic Information Systems (GIS) data. All potential plot locations are designated on the 1994 National Park Service vegetation data



layer as having vegetation associations with either Douglas-fir or redwood as the primary component (excluding Douglas fir-coyote brush association).

To establish plots, random starting points were selected within stratified vegetation types (Douglas-fir or redwood), and a rigorous in-field protocol was then followed to locate the closest acceptable sampling locations. Starting points consisted of combination of a) randomly-located plots from the related rapid assessment project referenced above or b) supplemental random locations. All points were at least 500 meters from all other points, at least 50 meters from trails or roads, and on slopes less than 60% (for worker safety and minimization of erosion). Each starting point served as a “plot-pair origin” (PPO), a location from which we initiated a radial search to establish a pair of “Healthy” (H) and “Diseased” (D) plots, or a sole H or D plot, in the event that a pairing was impossible from a given PPO. At each PPO, the nearest acceptable position, for an H or D plot (whichever condition was encountered first), became the first plot of the pair, and a new radial search was conducted from this first plot to identify the nearest location satisfying the opposite condition. If the second plot of the pair could not be located within a radius of 200 meters, the first plot became a stand-alone (unpaired) plot. If neither an H nor D plot could be identified within 200 meters of the PPO, the PPO was discarded. The PPO itself became the first of the paired plots if all criteria for one condition were met at this initial location.

The criteria used for the in-field plot locating protocol are as follows. Within all plots (redwood and Douglas-fir, H and D), **a.** the dominant species (redwood or Douglas-fir) must provide at least 25% canopy cover within a $\frac{1}{4}$ hectare circular area (28.21 m radius; hereafter referred to as the “extended plot”), estimated visually, **b.** at least 0.46 square meters (5 square feet) of basal area (BA) of the dominant species must be present within a $\frac{1}{20}$ hectare circular area (12.62 m radius; hereafter referred to as the “inner plot” or simply the “plot”), **c.** no individuals greater than 10 cm DBH (hereafter referred to as “mature”) of the other dominant species may be present within 20 meters of the plot center, and a maximum of two mature individuals may be present within the remainder of extended plot, **d.** no planted species (e.g. eucalyptus, Monterey pine) may be present within the extended plot, **e.** slope must not exceed 60%, **f.** paired plot centers will not be closer than 40 meters, and not farther than 200 meters from each other, **g.** plot centers will be at least 28.21 meters from roads or trails, **h.** the inner plot must contain at least 0.93 square meters (10 square feet) BA of total tanoak (any health condition); downed trees will not be counted toward the total tanoak BA (in any category) if they are considerably decayed (more than 50% of bark falling away from bole wood and/or bole wood compacts when stepped on).

Requirements for H plots were identical for redwood and Douglas-fir vegetation types; these criteria (which are described in detail in Appendix A) mandate that tanoak mortality and/or symptoms of SOD be scarce or non-existent, and thus these plots represent baseline conditions. Initially, we intended that criteria for D plots would also be identical across vegetation types, but this goal could not be achieved. In order to assess the full present impacts of SOD, an objective that we deemed necessary, we needed to capture the most severely diseased areas in both vegetation types; however, in 2007, SOD was much more established in the redwood forests of Bolinas Ridge than in the Douglas-fir forests of Inverness Ridge. As such, D Douglas-fir plots were permitted to have less tanoak mortality than D redwood plots, and thus diseased plots are not directly comparable across vegetation types. Specific criteria, which were established via an iterative process during our pilot plot-locating period, are detailed in Appendix A. All data that

are presented in this report were collected in the 1/20 hectare inner plot; the ¼ hectare extended plot area was considered for the purposes of plot selection only.

2.3. Data Collection

2.3.1. Plot-level metrics

In 2007 and 2009, modified Braun-Blanquet cover classes were used to estimate overall percent cover of canopy, shrubs, and herbs. “Canopy” was defined as all vegetation greater than 3 meters high. Herbs were defined as all non-woody vascular plant species. Shrubs and trees were separated by species, as opposed to physical measures such as height or DBH; for example, very large *Corylus cornuta* specimens were still considered shrubs. In addition, finer estimates of tree, shrub, and herb cover were made by assigning modified Braun-Blanquet cover classes to each species. Tree species were further designated as juvenile (seedlings, sprouts, or saplings) or “mature” (greater than or equal to 3 cm DBH; note that the “mature” classification does not necessarily indicate that a tree has begun to produce reproductive structures). Dead trees and shrubs (including tanoak) were not counted in cover classes, even if dead leaves were still clinging to twigs. However, total canopy cover included anything that blocked sunlight. Each species and cover class category was assigned one of the following: 0 (absent), r (rare, <0.5%), t (trace, 0.5-1%), 1 (1-5%), 2 (5-25%), 3 (25-50%), 4 (50-75%), 5 (75-95%) or 6 (95-100%). Canopy cover was also measured using a spherical densiometer; four measurements were taken at plot center, one in each cardinal direction. Throughout this report, canopy cover is expressed with both densiometer readings and visual estimates, because each of these methods have their merits; densiometer readings are more precise, but may produce misleading results if the area around plot center is considerably denser than the remainder of the plot. Finally, slope, aspect, and elevation were recorded in each plot.

2.3.2. Stem mapping and characterization of individual trees

Stems of all trees ≥ 3 cm DBH were mapped in 2007 by measuring distance and direction from plot center. The following variables were also recorded for each tree (initially in 2007 and again in 2009): species, health status, and the presence or absence of basal and epicormic sprouts. In addition, DBH was recorded (2007 only), as well as height and height-to-live-crown (2009 only). For new 2009 recruits (stems less than 3 cm DBH in 2007, but greater than 3 cm DBH in 2009), all variables were measured and recorded, including those that were not assessed in 2009 for pre-existing trees. Boles that were split below DBH were counted as multiple stems. For all species other than tanoak and coast live oak, dead trees were only recorded if they had not broken below breast height. All dead tanoak and coast live oak trees (including those that were uprooted or broken below breast height) were recorded, provided that the bole wood did not compact when stepped upon; as such, SOD-susceptible trees that had died recently were included in our dataset, but long dead individuals (most of which were probably not killed by SOD) were omitted. Health and deterioration status of tanoak and coast live oak was recorded in detail (see Appendix B), while species that are not susceptible to SOD, as well as madrone, were recorded as simply Alive or Dead. Although madrone is susceptible to SOD, detailed health conditions were not recorded because madrone is currently being impacted by several other diseases in addition to SOD (Burns and Honkala 1990) and additional categorization would have been required for meaningful interpretation. Basal sprouts were considered to be any sprouts arising from the lowest 10 cm of the bole or within 10 cm of the bole in duff, litter, or soil.

2.3.3. Tree regeneration

In 2007 and 2009, counts of all seedlings/sprouts (height less than breast height) and saplings (height greater than breast height, but DBH less than 3 cm) were conducted for all tree species. Individuals as well as clumps were tallied. “Clumps” were defined as groups of seedlings, sprouts, or saplings separated from other seedlings, sprouts, or saplings (of the same species) by at least 20 cm. Also, when a single clump exceeded 60 cm along its longest axis, the clump was split in two at a natural break. Individual saplings were distinguished from those which occurred in conspecific seedling/sprout clumps (in all plots in 2009, but in only a subset of plots in 2007), in order to estimate the number of combined seedling/sprout/sapling clumps. Sprouts were counted only if litter, duff, or soil obscured the base of sprouts (i.e. sprouts visibly emerging from wood were not recorded in regeneration tallies). Saplings were uncommon throughout the entire study area, and thus, throughout the remainder of this report – unless otherwise noted – the word “regeneration” applies to seedlings/sprouts.

2.3.4. Fuel Loading

To estimate surface and ground fuels, two planar intercept transects (Brown 1974), 12.62 meters in length, were established, originating at plot center and radiating due north and due south. Along each transect, surface fuels were tallied in timelag-classes. The number of 1-hour fuels (< 0.64cm diam.) and 10-hour fuels (0.64 – 2.54cm diam.) that crossed the transect plane between 10.5 and 12.5 meters were counted, the number of 100 hour fuels (2.54cm – 7.62cm diam.) that crossed the transect plane between 8.5 and 12.5 meters were counted, and the number of 1000-hour fuels (> 7.62cm diam.) that crossed the transect plane between 0 and 12.5m were counted. If no 1000-hour fuels were intersected in the first 12.5 meters of the transect, it was extended to 20 meters. Surface fuels included all dead twigs, branches, stems and boles of trees and shrubs that had fallen and were on or above the ground. Fuels that were still attached to standing shrubs or trees were not counted. Additionally, the diameter (cm) and condition (sound or rotten) were noted for each 1000 hour fuel. In situations when a large tree occurred directly N or S of the plot center, blocking the transect, we moved the transect the minimum possible azimuth in a clockwise direction. When an unacceptable amount of poison-oak (*Toxicodendron diversilobum*) occurred between 8.5 and 12.5 on N or S transect, transects were rotated to radiate E and W instead. We recorded all such adjustments. At two points (6m and 9m), we measured surface fuel depth and the depth of ground fuels – litter (loose, recently fallen foliage and plant material) and duff (decomposing plant material above mineral soil). Where poison-oak or a root crown occurred at 6m, we moved the sampling location to 5m, and so on; where poison-oak or a root crown occurred at 9m, we moved to 10m, and so on. All such adjustments were recorded.

Counts of surface fuels were translated into tons/acre using Brown’s standard equations and composite coefficients for calculating fuel loading in the interior West (Brown 1974, Brown et al., 1982). English units (tons/acre) are presented here, since this is the convention currently used by PRNS. Ground fuel loading (litter and duff) was calculated using standard bulk density estimates. Ladder fuels were characterized by measuring the height-to-live-crown-base (m) for each stem >3cm DBH in the 12.62m radius plot using a hypsometer. Live crown was defined as live branching foliage extending horizontally from the bole of the tree in at least two quadrants, and/or continuously into the upper canopy of the tree.

2.4. Data Analysis

All plot-level analyses, unless otherwise noted, were conducted as Generalized Linear Mixed Models (GLMMs), with each H-D plot pair treated as a block (i.e. a random effect). Error distributions were examined and assigned individually for each response variable; for instance, a poisson distribution was specified for regeneration counts, a binomial distribution was specified for most cover estimates (because values must fall between 0 and 1), and a gaussian distribution was used for variables with normal and homoskedastic errors (e.g. most basal area metrics). These analyses were conducted with the function *glmmPQL*, from the package *MASS*, in the R statistical software, version 2.9.2, by The R Foundation for Statistical Computing (R Development Core Team 2009).

Prior to statistical analysis, several preliminary conversions were necessary. Braun-Blanquet cover classes were converted to the numerical midpoints of each class (0.25% was used for “r”). Densimeter readings were transformed to percent canopy cover as per manufacturer instructions. Aspect was converted to northness as per Zar (1999): northness = cosine ((aspect * pi) / 180). Diversity, as defined by Simpson’s diversity index, D , was calculated as: $D = \sum ([n_i \cdot (n_i - 1)] / [N \cdot (N - 1)])$, where n_i is the cover of the i th species (within a given functional group) in each plot, and N is the total cover (in the corresponding functional group) in each plot. In this formula, we have defined n_i and N with cover, instead of with counts of individuals, which is the more typical usage (Magurran 2004). We report diversity as $1 - D$, a common modification that facilitates more intuitive interpretation; on the modified scale, higher values indicate greater diversity (i.e. a higher probability that any two random points will intercept two different species). Evenness, as defined by Simpson’s measure of evenness, $E_{1/D}$, was calculated from D : $E_{1/D} = (1/D) / S$, where S is the number of species (within the functional group used to calculate D) in the plot. Higher values of this metric indicate greater equivalence of relative abundances; unlike Simpson’s diversity index, Simpson’s measure of evenness is not affected by species richness (Magurran 2004).

2.4.1. Baseline comparison of redwood and Douglas-fir vegetation types

In order to establish a basic framework for predicting and understanding the ways in which SOD might differentially impact redwood and Douglas-fir forests, we used two approaches to assess baseline (i.e. pre-SOD) distinctions between these two vegetation types. The first and most intuitive approach was a direct comparison between healthy redwood plots and healthy Douglas-fir plots, in which vegetation type was the only predictor variable (random effects were not specified in these models because each H plot occupied a unique block). In addition, we conducted analyses with our full dataset (H and D plots) in which vegetation type and tanoak mortality (total dead tanoak basal area in 2007) were both included as predictors, thereby enabling comparison of redwood and Douglas-fir forests after accounting for varying amounts of SOD-induced tanoak mortality. Blocks (plot pairs) were included as random effects in all of these models.

For both of these baseline approaches, 2007 response variable values were examined only, because several “healthy” plots had begun to experience considerable tanoak mortality by 2009. In analyses of basal area and stem counts, only trees that were alive in 2007 were included, with the exception of tanoak, for which all living and dead trees were included (in order to accurately reflect the considerable number of recently killed tanoak trees in diseased plots). In analyses of regeneration, saplings were omitted because a) we were mostly focused on recent regeneration, and b) sapling tallies were generally miniscule in comparison to

seedlings/sprouts and thereby inconsequential to our results; we considered conducting separate analyses with saplings, but because of the high numbers of zero values (and/or extreme overdispersion) in our dataset, analyses were generally not feasible or meaningful.

2.4.2. *Disease progression*

For the purposes of graphical display and interpretation, detailed health status categories (see Appendix B) were converted to five simplified classes: Alive Asymptomatic (AA; no bole or canopy symptoms), Alive Symptomatic (AS; one or more bole or canopy symptoms present), Dead Intact (DI; bole unbroken or broken at a diameter of less than 7.5 cm), Dead Broken (DB; bole broken at a diameter of 7.5 cm or greater, but not broken at or below 1.37 m height), and Dead Fallen (DF; broken at or below 1.37 m height, regardless of diameter at break point). Within each vegetation type and plot-level health status (H or D) combination (e.g. RW-D), we determined the proportion of trees (in terms of both stem counts and basal area) that belonged to each of the five simplified tree-level health status classes. In addition, these five classes were further compressed into two categories (living and dead), enabling an additional set of broad comparisons. Proportions were calculated (across all plots within each sampling strata) for both 2007 and 2009, in order to document disease progression trends over this two-year period. To test for significant differences in disease progression between plot strata, we conducted GLMMs in which vegetation type and plot-level health status were included as fixed categorical predictors, and each H-D plot pair was treated as a block (i.e. a random effect). Several metrics of mortality and disease progression were analyzed as response variables. Note that, in these GLMMs, proportional mortality was calculated within each plot (thus providing multiple values within each sampling strata), but that the proportional mortality displayed in disease progression figures (Figures 2 through 5) was calculated across all plots within each sampling strata.

2.4.3. *Exploration of factors that could be affecting disease severity*

In order to examine factors that may have increased the severity of SOD in our study system, we identified several variables that were almost certainly – at least at the time of our initial field sampling – unaffected by SOD, and tested the ability of these variables to predict tanoak mortality (which was quantified in four separate ways: the total amount of dead tanoak BA in 2007, the *percent* of tanoak BA that was dead in 2007, the change in dead tanoak basal area from 2007 to 2009, and the change from 2007 to 2009 in the *percent* of tanoak basal area that was dead). The following continuous predictor variables were examined: 1) BA and stem counts of total tanoak (living and dead combined), all tree species, all species other than tanoak, all hardwoods other than tanoak, the dominant species (redwood or Douglas-fir depending on vegetation type), and California bay; 2) the mean and median DBH for tanoak, as well as all tree species combined; 3) species richness of mature trees; and 4) slope, elevation, and northness. With each combination of explanatory and response variables (the response variable was defined separately as each of the four tanoak mortality metrics described above), we fit several model variants: A) redwood plots only; B) Douglas-fir plots only; C) redwood and Douglas-fir plots pooled (with vegetation type included as an additional predictor); and D) redwood and Douglas-fir plots pooled (with vegetation type *and* total tanoak basal area included as additional predictors). In addition, with each of the four tanoak mortality metrics, we fit a single categorical model with vegetation type and plot status (i.e. H or D) as predictors, to assess broad differences between our sampling strata. All models specified plot pair blocks as a random effect.

2.4.4. Disease impacts

In addition to the direct impacts of SOD (i.e. tanoak symptoms, mortality, and deterioration), which are assessed in the disease progression section, we explored many other variables that may have responded to SOD-induced tanoak decline. To test for any such effects, we conducted categorical comparisons of H and D plots, as well as several analyses in which tanoak mortality was represented by one of three continuous predictor variables: 1) the amount of dead tanoak basal area in 2009, 2) the amount of dead tanoak basal area in 2007, and 3) the amount of tanoak basal area that was “broken” or “fallen” in 2007 (in the redwood vegetation type only; see Appendix B for deterioration categories). These three predictor variations are intended to represent a continuum of tanoak mortality, ranging from that which we assume occurred several years prior to 2007 (i.e. broken/fallen by 2007, referred to as “early mortality”) to all the cumulative mortality that had occurred by 2009 (or at least that for which evidence existed). In Douglas-fir plots, the amount of tanoak basal area that had broken/fallen by 2007 was not examined, because very few trees in such condition existed in this vegetation type (13 out of 15 plots had values of zero for this metric). By examining each of these mortality metrics independently, we were able to consider potential lag times and/or mortality thresholds for each response variable.

With each mortality metric, we conducted two separate analyses: one in which total tanoak basal area was included as an additional model predictor, and one in which the given mortality metric was the only fixed effect. We included the former model variant because dead tanoak basal area was positively related to total tanoak basal area (see Results), and we deemed it important to account for any potentially confounding effects of total tanoak. The three continuous model variants in which total tanoak basal area was *not* included as a predictor variable are similar to the categorical plot comparison in the sense that they assess directly observable responses to tanoak mortality. In contrast, the model variants that account for the effects of total tanoak illuminate patterns that may only be apparent by comparing potentially non-existent areas (e.g. two plots with highly divergent levels of mortality, but equivalent levels of total tanoak). In all disease impact analyses, blocks (plot pairs) were included as random effects, and redwood and Douglas-fir plots were examined independently (i.e. two separate sets of analyses). In analyses of regeneration, saplings were omitted because a) we were mostly focused on recent regeneration, and b) sapling tallies were generally miniscule in comparison to seedlings/sprouts and thereby inconsequential to our results; we considered conducting separate analyses with saplings, but because of the high numbers of zero values (and/or extreme overdispersion) in our dataset, analyses were generally not feasible or meaningful.

3. RESULTS

3.1. Baseline comparison of redwood and Douglas-fir vegetation types

Several variables were significantly correlated with vegetation type (Tables 1 and 2). The following variables were positively associated with the Douglas-fir vegetation type, in comparisons utilizing healthy plots only, analyses utilizing all plots (and accounting for tanoak mortality), or both: herb cover, shrub richness, total vascular plant richness, mature tree diversity, total tree diversity (juvenile and mature combined), total vascular plant diversity, canopy cover (as measured with a densiometer, but not as estimated visually), BA of non-tanoak hardwoods and California bay, stem counts of non-tanoak hardwoods and California bay, and

regeneration of non-tanoak hardwoods and California bay. Note that the non-tanoak hardwood category includes California bay, but this species was also examined individually because other research has shown that SOD disease severity may increase with California bay abundance (Rizzo et al. 2005, McPherson et al. 2010). Herb richness displayed a borderline positive relationship (p-value between 0.05 and 0.10) with Douglas-fir plots, but only when all plots were included. Variables that were positively correlated with the redwood type vegetation type are as follows: herb evenness, shrub evenness, stem counts of conifers, and regeneration of conifers. Total basal area and total stem counts displayed a borderline positive relationship with redwood plots, but only when all plots were included.

Table 1. Comparison of redwood and Douglas-fir vegetation types: cover classes, species richness, evenness, and diversity. The number of positive symbols indicates the degree of significance: one symbol = $0.01 < p < 0.05$; two symbols = $0.001 < p < 0.01$; three symbols = $p < 0.001$. Parentheses indicate borderline effects (significance between 0.05 and 0.1). ¹direct comparison (vegetation type was the only predictor variable); ²accounting for tanoak mortality (vegetation type and 2007 dead tanoak BA were both specified as fixed predictors).

2007 values	H plots ¹		All plots ²	
	RW	DF	RW	DF
<u>Cover Classes</u>				
Herb		++		++
Shrub				
Juvenile Tree				
Exotic				
Canopy (Braun-Blanquet)				
Canopy (Densiometer)		++		+
<u>Richness</u>				
Herb				(+)
Shrub		++		+++
Juvenile Tree				
Mature Tree				
Tree (juv and mat)				
Exotic				
Total		(+)		++
<u>Evenness</u>				
Herb	(+)		+	
Shrub	+		+	
Juvenile Tree				
Mature Tree				
Tree (juv and mat)				
Exotic				
Total				
<u>Diversity</u>				
Herb				
Shrub				
Juvenile Tree				
Mature Tree		+		++
Tree (juv and mat)		(+)		++
Exotic				
Total		+		+++

Table 2. Comparison of redwood and Douglas-fir vegetation types: basal area, stem counts, regeneration, and fuels. See Table 1 for an explanation of symbols. ¹direct comparison (vegetation type was the only predictor variable); ²accounting for tanoak mortality (vegetation type and 2007 dead tanoak BA were both specified as predictors); ³results are qualitatively identical for individuals and clumps.

2007 values	<u>H plots</u> ¹		<u>All plots</u> ²	
	RW	DF	RW	DF
<u>Basal Area</u>				
Total			(+)	
Tanoak (living and dead)				
Conifer				
Non-tanoak hardwood		+		++
California bay		+		++
<u>Stem Counts</u>				
Total			(+)	
Tanoak (living and dead)				
Conifer	++		++	
Non-tanoak hardwood		+		++
California bay		+		+
<u>Regen (Individuals / Clumps) ³</u>				
Total				
Tanoak				
Conifer	++		+++	
Non-tanoak hardwood		++		++
California bay		++		++
<u>Fuels</u>				
One hour				D
Ten hour				I
Hundred hour				F
Thousand hour: Solid				F
Thousand hour: Rotten			N	E
Thousand hour: R and S			O	R
One to Hundred Hour				E
One to Thousand Hour				N
Litter				C
Duff		+		E
Litter and Duff		+		S
Total		+		

3.2. Disease progression

Sudden oak death continued to progress between 2007 and 2009. In all four sampling strata (RW-H, RW-D, DF-H, and DF-D), new mortality occurred and the number of asymptomatic trees decreased. As expected, after accounting for vegetation type, D plots had significantly higher levels of dead tanoak BA in 2007, as well as a significantly higher proportion of tanoak BA that was dead in 2007; however, changes between 2007 and 2009 (for both absolute and proportional metrics of dead BA) were completely unrelated to plot-level health classification. In contrast, the magnitude of these changes was strongly related to vegetation type. After accounting for plot-level health classification (i.e. H vs. D), redwood plots had significantly higher levels of dead tanoak BA in 2007, but Douglas-fir plots experienced a significantly higher increase in dead tanoak BA from 2007 to 2009. Douglas-fir plots also exhibited a significantly higher two-year increase in the *proportion* of tanoak BA that

was dead, while static 2007 values for proportional dead BA were unrelated to vegetation type.

In H redwood plots, the total proportion of dead tanoak stems and basal area increased by 0.04 and 0.09 respectively (Fig. 2). Similarly, in D redwood plots, the corresponding increases were 0.07 and 0.04 (Fig. 3). In contrast, Douglas-fir plots experienced much greater increases in the proportion of dead tanoak; stem counts and basal area increased by 0.19 and 0.37 in H plots (Fig. 4) and 0.20 and 0.41 in D plots (Fig. 5). While proportional mortality was comparable between redwood and Douglas-fir plots in 2007, Douglas-fir plots had clearly surpassed redwood plots by 2009; nearly 100% of tanoak basal area was dead in Douglas-fir D plots, while redwood D plots still retained approximately 25% of their pre-SOD tanoak BA, at the time of our most recent measurements.

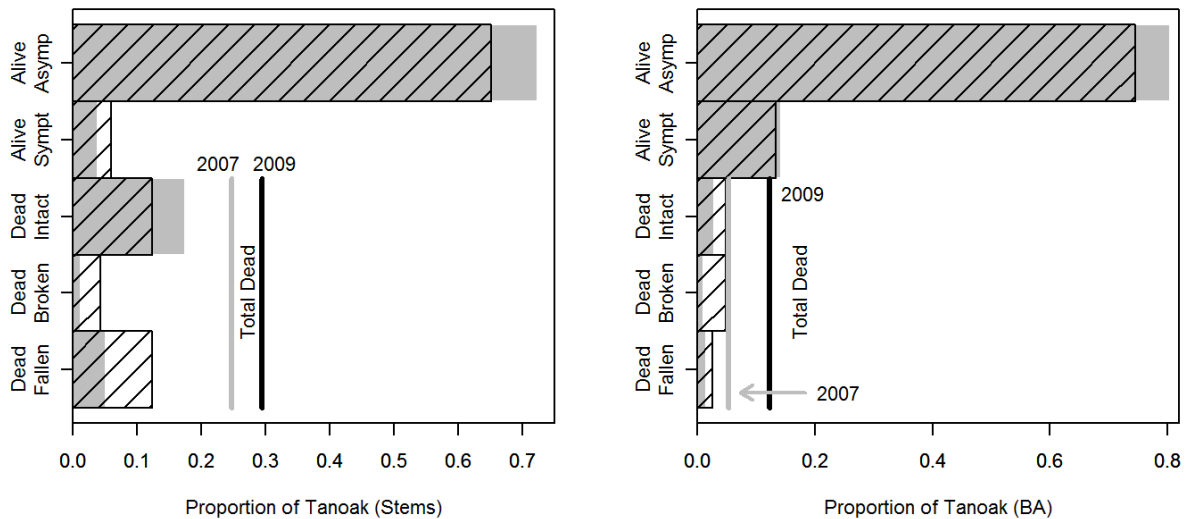


Figure 2. Proportion of tanoak stems (left) and basal area (right) belonging to different symptom and deterioration classes in H redwood plots. Solid gray bars = 2007; Hatched black bars = 2009.

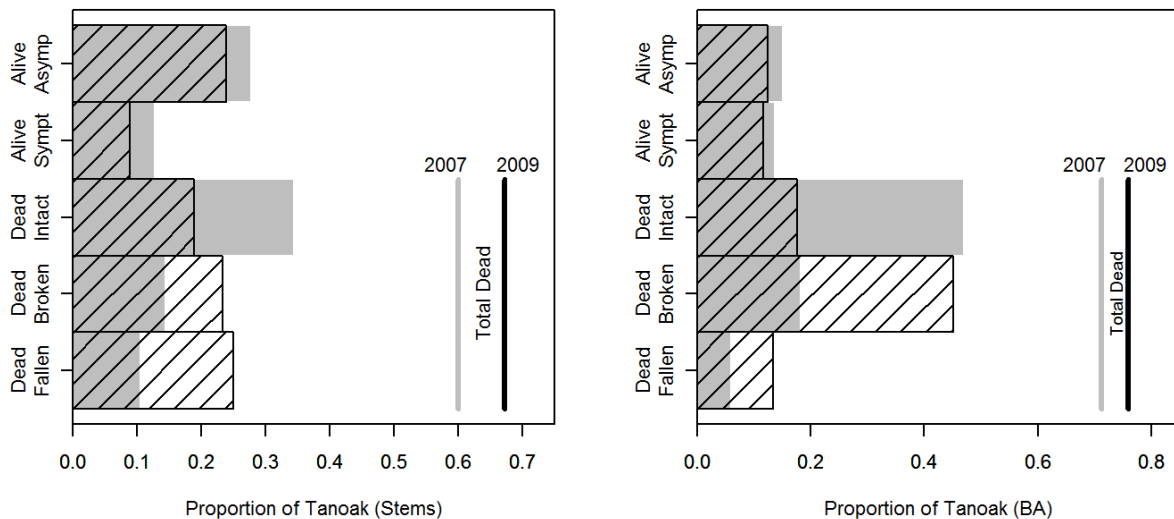


Figure 3. Proportion of tanoak stems (left) and basal area (right) belonging to different symptom and deterioration classes in D redwood plots. Solid gray bars = 2007; Hatched black bars = 2009.

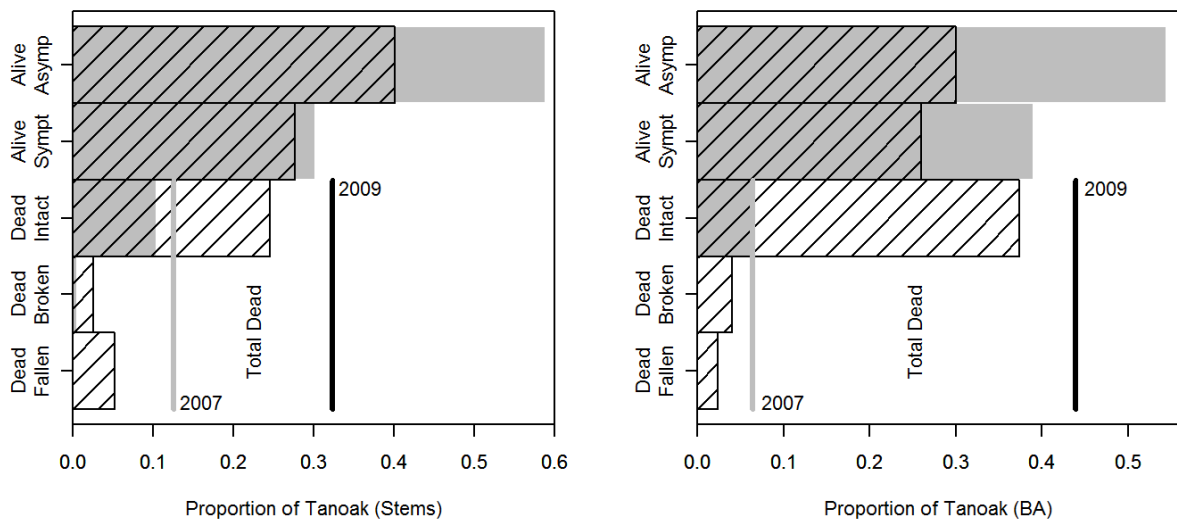


Figure 4. Proportion of tanoak stems (left) and basal area (right) belonging to different symptom and deterioration classes in H Douglas-fir plots. Solid gray bars = 2007; Hatched black bars = 2009.

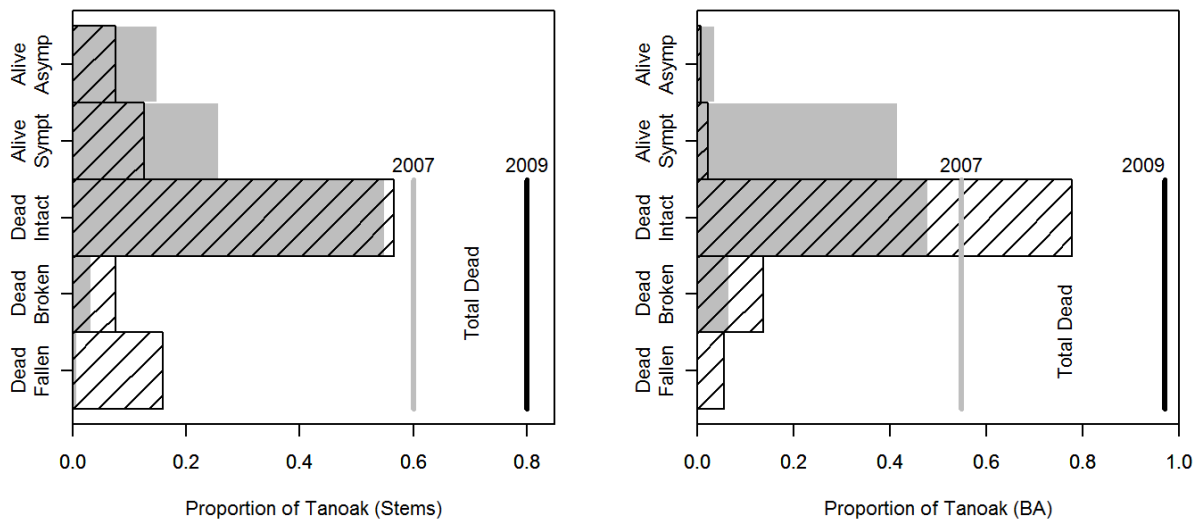


Figure 5. Proportion of tanoak stems (left) and basal area (right) belonging to different symptom and deterioration classes in D Douglas-fir plots. Solid gray bars = 2007; Hatched black bars = 2009.

While diseased redwood plots did not experience drastic increases in mortality, a large proportion of previously killed trees did begin to deteriorate (i.e. transition from “intact” to “broken” or “fallen”); such deterioration was not as pronounced in Douglas-fir plots, supporting observations by park staff that mortality begun earlier in the redwood forests of Bolinas Ridge than in the Douglas-fir forests of Inverness Ridge. From 2007 to 2009, the proportion of trees that were asymptomatic declined in all sampling strata, and the proportion of trees that were symptomatic increased in all strata except RW-H.

3.3. Additional factors that could be affecting disease severity

Several continuous predictors were related to tanoak mortality. Total tanoak basal area was a significant (p-value < .05) or borderline significant (p-value between 0.05 and 0.10) predictor of dead tanoak basal area in redwood plots as well as pooled redwood and Douglas-fir plots (model variant C), but not in Douglas-fir plots alone; total tanoak basal area was also significant or borderline significant in *all* variant D models (note that every variant D model included total tanoak basal area as a predictor). No other continuous predictors were significant in any of the models in which they were included, although elevation and median tanoak DBH exhibited borderline negative relationships in analyses with Douglas-fir plots only.

None of the continuous variables we tested were significant predictors of the *percent* of tanoak basal area that was dead in 2007. However, the following borderline positive relationships with percent tanoak mortality were detected: the basal area of hardwoods-excluding-tanoak in model variants C and D, and the basal area of California bay (which accounts for a considerable component of the basal area of all non-tanoak hardwoods) in model variant D only. No such relationships were detected in analyses utilizing only redwood or Douglas-fir plots.

Several continuous variables were significant predictors of the *change* in dead tanoak basal area between 2007 and 2009. Total tanoak basal area was significant (positive relationship) in all models except the model utilizing redwood plots only. The following variables were significantly inversely related to the two-year change in dead tanoak basal area in one or more models: median DBH of all species (DF plots only), California bay BA (model variant C), BA of non-tanoak hardwoods (model variant C), and BA of all species other than tanoak (model variants C and D). In addition, several variables exhibited borderline relationships (all negative) with the two-year change in dead tanoak basal area: BA of non-tanoak hardwoods BA (model variant D), stems of non-tanoak hardwoods (redwood plots only), stems of all species other than tanoak (model variant C), and elevation (model variant C).

The only significant continuous predictor of the change from 2007 to 2009 in the percent of dead tanoak BA was the BA of all species other than tanoak; this relationship was negative and was only significant in model variants C and D. Total tanoak BA was weakly positively associated (i.e. borderline significant) with the two-year change in the percent of dead tanoak BA, but only in the model with Douglas-fir plots alone. In addition, several variables exhibited borderline negative relationships with this response variable in one or more models: median DBH of all species (Douglas-fir plots only), BA of California bay (model variants C and D), stems of California bay (model variants C and D), and BA of non-tanoak hardwoods (model variant C).

3.4. Disease impacts

3.4.1. Cover classes

In redwood plots, 2009 canopy cover (estimated visually) was significantly lower in D plots than in H plots, and several continuous metrics of tanoak mortality were inversely related to this variable (Table 3; Fig. 6). Dead tanoak basal area in 2007 was a significant predictor in models with and without total tanoak basal area, while dead tanoak basal area in 2009 was significant only when total tanoak abundance was not included as a model predictor (although 2009 dead tanoak basal area and 2007 broken/fallen tanoak basal were both borderline significant in the reduced models). When measured with a densiometer, 2009 canopy cover was significantly lower in D plots than in H plots, and dead tanoak basal area in 2009 was inversely

related to this variable, but only when total tanoak was included a model predictor; 2007 dead tanoak basal area was borderline significant in the model including total tanoak. In models that did not include total tanoak as a predictor, no continuous metrics of tanoak mortality were predictive of 2009 canopy cover (measured with a densiometer). The change in canopy cover from 2007 to 2009 (whether estimated visually or measured with a densiometer) was not related to tanoak mortality in any categorical plot comparisons or continuous analyses.

Table 3. Cover class as a function of tanoak mortality (redwood plots). Each cell presents the results of a separate analysis. Positive and negative symbols indicate the direction of the relationship, and the number of these symbols indicates the degree of significance: one symbol = $0.01 < p < 0.05$; two symbols = $0.001 < p < 0.01$; three symbols = $p < 0.001$. Parentheses indicate borderline effects (significance between 0.05 and 0.1, or significance level of 0.05 is achieved only with or without outliers). Zeros indicate that the predictor significance level was greater than 0.1.

Veg = RW	(total tanoak incl. as a predictor)			(total tanoak <i>not</i> incl. as a predictor)			D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	
Herb: 09	(-)	--	-	--	--	(-)	--
Herb: 07-09	0	0	0	0	0	0	0
Shrub: 09	0	0	0	0	0	0	0
Shrub: 07-09	0	0	0	0	0	0	0
Juv Tree: 09	0	0	0	0	0	0	0
Juv Tr: 07-09	0	0	0	0	0	0	0
Exotic: 09	0	0	0	0	0	0	0
Exotic: 07-09	0	0	0	0	0	0	0
Canopy: 09	-	-	0	(-)	-	(-)	-
Canopy: 07-09	0	0	0	0	0	0	0
Densi: 09	-	(-)	0	0	0	0	-
Densi: 07-09	0	0	0	0	0	0	0

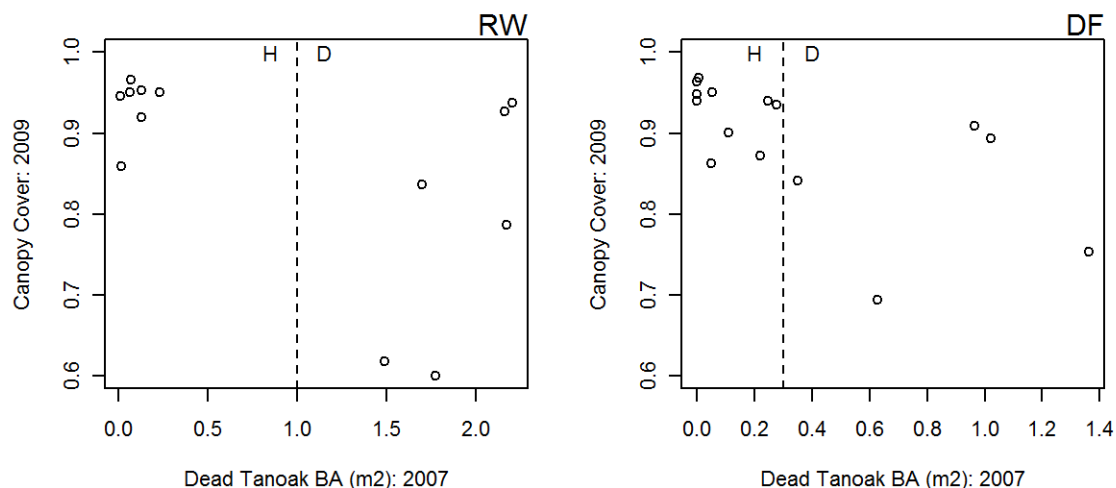


Figure 6. Canopy cover (2009) vs. dead tanoak basal area (2007) in redwood (left) and Douglas-fir (right) plots. The dashed line separates H and D plots within each vegetation type.

In Douglas-fir plots, 2009 canopy cover (estimated visually or measured with a densiometer) was significantly lower in D plots, and was inversely related to all continuous mortality metrics in the models that omitted total tanoak basal area (Table 4; Fig. 6). In models that included total tanoak abundance, 2007 dead tanoak basal area predicted both measures of 2009 canopy cover (visual estimates and densiometer readings), while 2009 dead tanoak basal area was a significant predictor of densiometer readings only (although the relationship was borderline significant with visual estimates of canopy cover). The change in canopy cover from 2007 to 2009 was also related to tanoak mortality in several continuous analyses, but this two-year change was not significantly related to categorical plot comparisons (although we have found suggestive evidence, i.e. a borderline relationship, that the reduction in densiometer-defined canopy cover from 2007 to 2009 was greater in D plots). In continuous analyses, 2009 dead tanoak basal area was a significant predictor of the two-year change in canopy cover (estimated visually) in the model without total tanoak and a borderline significant predictor in the model including total tanoak. When canopy cover was represented with densiometer readings, no continuous variables were significant predictors of the change in canopy cover from 2007 to 2009, but dead tanoak basal area in 2007 and 2009 were borderline predictors.

Table 4. Cover class as a function of tanoak mortality (Douglas-fir plots).

See Table 3 for a description of symbols.

Veg = DF	(total tanoak incl. as a predictor)		(total tanoak <i>not</i> incl. as a predictor)		D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	Ttl Dd: 09	Ttl Dd: 07	
Herb: 09	0	0	0	0	0
Herb: 07-09	0	0	0	0	0
Shrub: 09	0	0	0	0	0
Shrub: 07-09	0	0	0	0	0
Juv Tree: 09	0	0	0	0	0
Juv Tr: 07-09	0	0	0	0	0
Exotic: 09	0	0	0	0	0
Exotic: 07-09	0	0	0	0	0
Canopy: 09	(-)	-	-	-	-
Canopy: 07-09	(-)	0	-	0	0
Densi: 09	-	--	-	--	--
Densi: 07-09	0	0	(-)	(-)	(-)

Herbaceous cover in 2009 exhibited consistently negative relationships with tanoak mortality in the redwood vegetation type; herb cover was significantly lower in D plots than in H plots, and all continuous metrics were either significant or borderline significant predictors. Tanoak mortality was unrelated to 2009 herb cover in our Douglas-fir plots, and was unrelated to the two-year change in herb cover in both vegetation types. Cover classes in 2009, as well as two-year change values, for shrubs, juvenile trees, and exotic plants were all unrelated to tanoak mortality in both vegetation types.

3.4.2. Species richness

In redwood plots, 2009 herbaceous species richness was positively related to the amount of tanoak basal area that was dead in 2009 and 2007, but only when total tanoak was included as a model predictor (Table 5; Fig. 7). 2009 herb richness was not significantly related to tanoak

mortality in categorical plot comparisons or in continuous analyses that did not account for the effects of total tanoak. However, the change in herb richness from 2007 to 2009 exhibited borderline negative relationships with tanoak mortality in the categorical plot comparison and three continuous analyses: the two-year change in herb richness was weakly predicted (i.e. borderline significance) by 2007 dead tanoak basal area (in models with and without total tanoak) and by 2009 dead tanoak basal area (but only in the model without tanoak). Juvenile tree richness was not related to tanoak mortality in categorical comparisons, or in most continuous analyses; however, when total tanoak was not included as a model predictor, the amount of tanoak basal area that was broken/fallen in 2007 was positively related to the two-year change in juvenile tree richness. Total 2009 vascular plant species richness results were qualitatively identical to those for 2009 herbaceous species richness; however, unlike herb richness, two-year change values for total richness were completely unrelated to all metrics of tanoak mortality. Shrub richness and exotic species richness were unrelated to tanoak mortality in all categorical and continuous analyses.

Table 5. Richness as a function of tanoak mortality (redwood plots).

See Table 3 for a description of symbols.

Veg = RW	(total tanoak incl. as a predictor)			(total tanoak <i>not</i> incl. as a predictor)			D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	
Herb: 09	++	+	0	0	0	0	0
Herb: 07-09	0	(-)	0	(-)	(-)	0	(-)
Shrub: 09	0	0	0	0	0	0	0
Shrub: 07-09	0	0	0	0	0	0	0
Juv Tree: 09	0	0	0	0	0	0	0
Juv Tr: 07-09	0	0	0	0	0	+	0
Total: 09	++	++	0	0	0	0	0
Total: 07-09	0	0	0	0	0	0	0
Exotic: 09	0	0	0	0	0	0	0
Exotic: 07-09	0	0	0	0	0	0	0

Table 6. Richness as a function of tanoak mortality (Douglas-fir plots).

See Table 3 for a description of symbols.

Veg = DF	(total tanoak incl. as a predictor)		(total tanoak <i>not</i> incl. as a predictor)		D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	Ttl Dd: 09	Ttl Dd: 07	
Herb: 09	++	+	+	+	+
Herb: 07-09	0	0	0	0	0
Shrub: 09	0	0	0	0	0
Shrub: 07-09	0	0	0	0	0
Juv Tree: 09	0	0	0	0	0
Juv Tr: 07-09	0	0	0	0	0
Total: 09	++	+	0	+	+
Total: 07-09	0	0	0	0	0
Exotic: 09	0	0	0	0	0
Exotic: 07-09	0	0	0	0	0

In Douglas-fir plots, 2009 herbaceous species richness was positively related to tanoak mortality in categorical plot comparisons as well as several continuous analyses (Table 6; Fig. 7). With and without the inclusion of total tanoak as a model predictor, dead tanoak basal area in both 2007 and 2009 were predictive. Total vascular plant richness results were almost qualitatively identical to herb richness results, except that, when total tanoak was not included as a model predictor, 2009 dead tanoak basal area was not a significant predictor of 2009 total richness. Shrub richness, juvenile tree richness, and exotic species richness were unrelated to tanoak mortality in all categorical and continuous analyses.

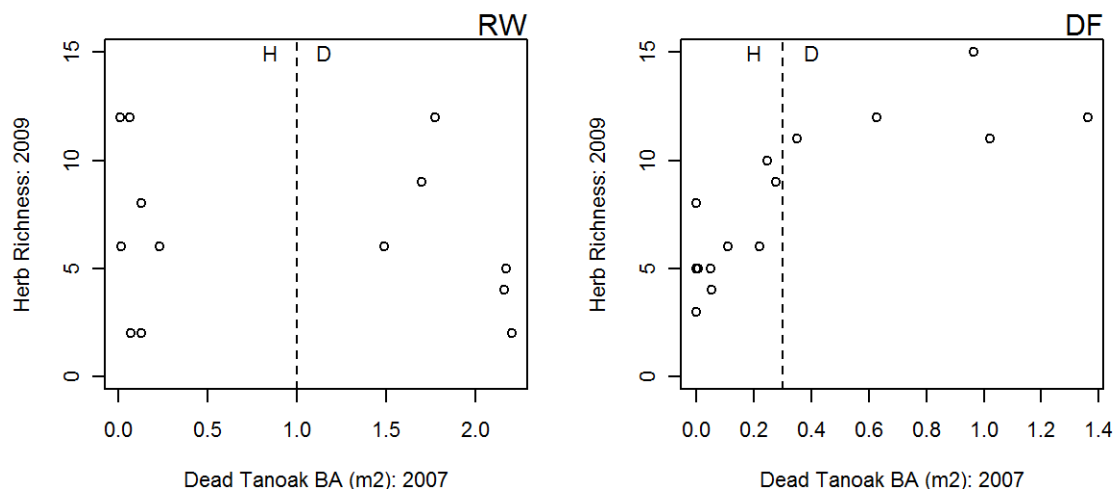


Figure 7. Herb richness (2009) vs. dead tanoak basal area (2007) in redwood (left) and Douglas-fir (right) plots. The dashed line separates H and D plots within each vegetation type.

3.4.3. Regeneration

In the redwood vegetation type, we found no evidence that total regeneration (i.e. regeneration of all species combined) was affected by SOD (Table 7). However, our results do indicate that tanoak mortality has stimulated a regenerative response in some functional groups and species. Regeneration of hardwoods-excluding-tanoak was significantly higher in areas with greater tanoak mortality. In continuous analyses, this pattern generally held regardless of whether regeneration was analyzed as individuals or as clumps, or as absolute 2009 numbers or as the two-year change from 2007 to 2009. In categorical plot comparisons, 2009 regeneration levels were higher in diseased plots, but a similar pattern in two-year change values was not definitive. The absolute number of regenerating conifer clumps in 2009 was significantly related to tanoak mortality, in both continuous and categorical analyses. In continuous analyses, all mortality metrics were significant except the amount of tanoak basal area that was broken/fallen in 2007 (with and without total tanoak basal included in the models). Numbers of regenerating conifer individuals were not related to tanoak mortality, nor were the two-year change values.

We did not find strong evidence that tanoak regeneration (which accounted for the vast majority of total regeneration in redwood plots) was related to mortality of mature tanoak trees, but there is weak evidence that the two-year change values of individuals (but not clumps) were negatively affected by SOD in our redwood plots (Table 7). The number of regenerating redwood clumps in 2009 was significantly related to tanoak mortality, in both continuous and categorical analyses (Fig. 8a). In continuous analyses, this relationship was only definitive when

total tanoak was not included in the model, and only with regard to total dead tanoak in 2007 and 2009 (the amount of broken/fallen tanoak in 2007 was not predictive). Numbers of regenerating redwood individuals were not related to tanoak mortality, nor were the two-year change values for individuals or clumps.

Table 7. Regeneration as a function of tanoak mortality (redwood plots).

See Table 3 for a description of symbols.

Veg = RW	(total tanoak incl. as a predictor)			(total tanoak <i>not</i> incl. as a predictor)			D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	
Total Ind: 09	0	0	0	0	0	0	0
Total Cl: 09	0	0	0	0	0	0	0
Total Ind: 07-09	0	0	0	0	0	0	0
Total Cl: 07-09	(+)	0	0	0	0	0	0
HW Ind: 09	+	+	(+)	+	+	+++	+
HW Cl: 09	+	+	(+)	+	+	+++	+
HW Ind: 07-09	+	+	++	+	+	+++	0
HW Cl: 07-09	+	+	++	+	+	+++	(+)
Con Ind: 09	0	0	0	0	0	0	0
Con Cl: 09	+	+	0	+	+	0	+
Con Ind: 07-09	0	0	0	0	0	0	0
Con Cl: 07-09	0	0	0	0	0	0	0
Lide Ind: 09	0	0	0	0	0	0	0
Lide Cl: 09	0	0	0	0	0	0	0
Lide Ind: 07-09	0	(-)	0	(-)	(-)	0	0
Lide Cl: 07-09	0	0	0	0	0	0	0
Sese Ind: 09	0	0	0	0	0	0	0
Sese Cl: 09	0	(+)	0	+	++	0	+
Sese Ind: 07-09	0	0	0	0	0	0	0
Sese Cl: 07-09	0	0	0	0	0	0	0
Psme Ind: 09	0	0	+++	0	0	0	0
Psme Cl: 09	0	0	++	0	0	0	0
Psme Ind: 07-09	0	0	(+)	0	0	0	0
Psme Cl: 07-09	(+)	0	(+)	0	0	0	0
Umca Ind: 09	0	0	(+)	(+)	(+)	+	(+)
Umca Cl: 09	0	0	0	0	0	(+)	0
Umca Ind: 07-09	0	0	0	0	0	0	0
Umca Cl: 07-09	0	0	0	0	0	0	0
Arme Ind: 09	++	+	+++	+	+	0	+
Arme Cl: 09	++	+	+++	+	+	0	+
Arme Ind: 07-09	++	+	+++	+	+	0	+
Arme Cl: 07-09	++	+	+++	+	+	0	+

In redwood plots, Douglas-fir regeneration (individuals and clumps) was significantly related only to the amount of tanoak basal area that was broken/fallen in 2007, and only in the continuous analysis in which total tanoak basal area was included as a model predictor (Table 7). Because plots with more dead tanoak also tend to have more total tanoak, and total tanoak basal

area has a negative effect on Douglas-fir regeneration (results not shown), a positive relationship between tanoak mortality and Douglas-fir regeneration is illuminated only after accounting for total tanoak levels. While our data predict that areas with greater tanoak mortality (assuming equivalent total tanoak basal area) will have more Douglas-fir recruitment, it is important to emphasize that our actual diseased plots did not have more regeneration than our actual healthy plots in 2009.

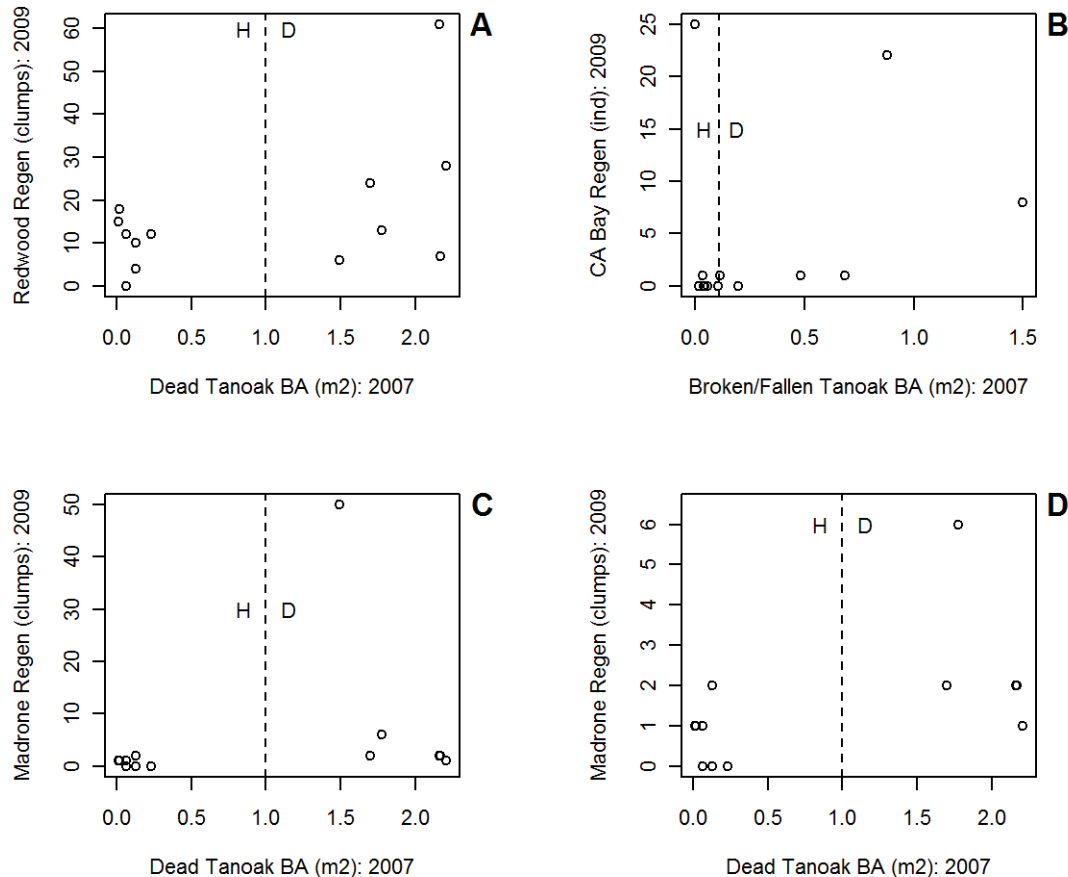


Figure 8. Regeneration (2009) vs. dead tanoak basal area (total dead in 2007 or broken/fallen dead in 2007) in redwood plots. The dashed line separates H and D plots.

California bay regeneration (counts of individuals only) was related to the amount of tanoak basal area that was broken/fallen in redwood plots in 2007 (Fig. 8b), but this relationship was only definitive when total tanoak basal area was not included as a model predictor. Pacific madrone regeneration (2009 values and two-year changes, as well as individuals and clumps) was positively related to tanoak mortality in both continuous and categorical analyses (Fig. 8c/d). In continuous analyses, all mortality metrics were significant except the amount of tanoak basal area that was broken/fallen in 2007 (and only without total tanoak basal included in the model). Because there was one obvious outlier in counts of Pacific madrone (in terms of individuals as well as clumps), we ran all analyses again after replacing this outlying value (50 individuals; 56 clumps) with the second largest value (6, in the case of both individuals and clumps); after this substitution, the majority of analyses were qualitatively identical.

In the Douglas-fir vegetation type, total regeneration did not differ between healthy and diseased plots, but continuous analyses yielded a few significant relationships (Table 8). When total tanoak basal area was not included as a model predictor, the total number of regenerating individuals in 2009 was positively related to the amount of tanoak basal area that was dead in 2009, but not to dead basal area in 2007. In addition, the change in total individuals (from 2007 to 2009) was positively related to the amount of tanoak basal area that was dead in 2009.

Table 8. Regeneration as a function of tanoak mortality (Douglas-fir plots).

See Table 3 for a description of symbols.

Veg = DF	(total tanoak incl. as a predictor)		(total tanoak <i>not</i> incl. as a predictor)		D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	Ttl Dd: 09	Ttl Dd: 07	
Total Ind: 09	0	0	++	0	0
Total Cl: 09	0	0	0	0	0
Total Ind: 07-09	0	0	+	0	0
Total Cl: 07-09	0	0	0	0	0
HW Ind: 09	0	0	+	0	0
HW Cl: 09	0	0	0	0	0
HW Ind: 07-09	+	0	++	0	(+)
HW Cl: 07-09	+	(+)	0	(+)	(+)
Lide Ind: 09	0	0	++	0	0
Lide Cl: 09	0	0	(+)	0	0
Lide Ind: 07-09	0	0	+	0	0
Lide Cl: 07-09	0	0	0	0	0
Psme Ind: 09	0	0	0	0	0
Psme Cl: 09	0	0	0	0	0
Psme Ind: 07-09	0	0	0	0	(+)
Psme Cl: 07-09	0	0	0	0	(+)
Umca Ind: 09	0	0	+	0	0
Umca Cl: 09	0	0	0	0	0
Umca Ind: 07-09	+	0	++	0	(+)
Umca Cl: 07-09	(+)	(+)	0	(+)	(+)

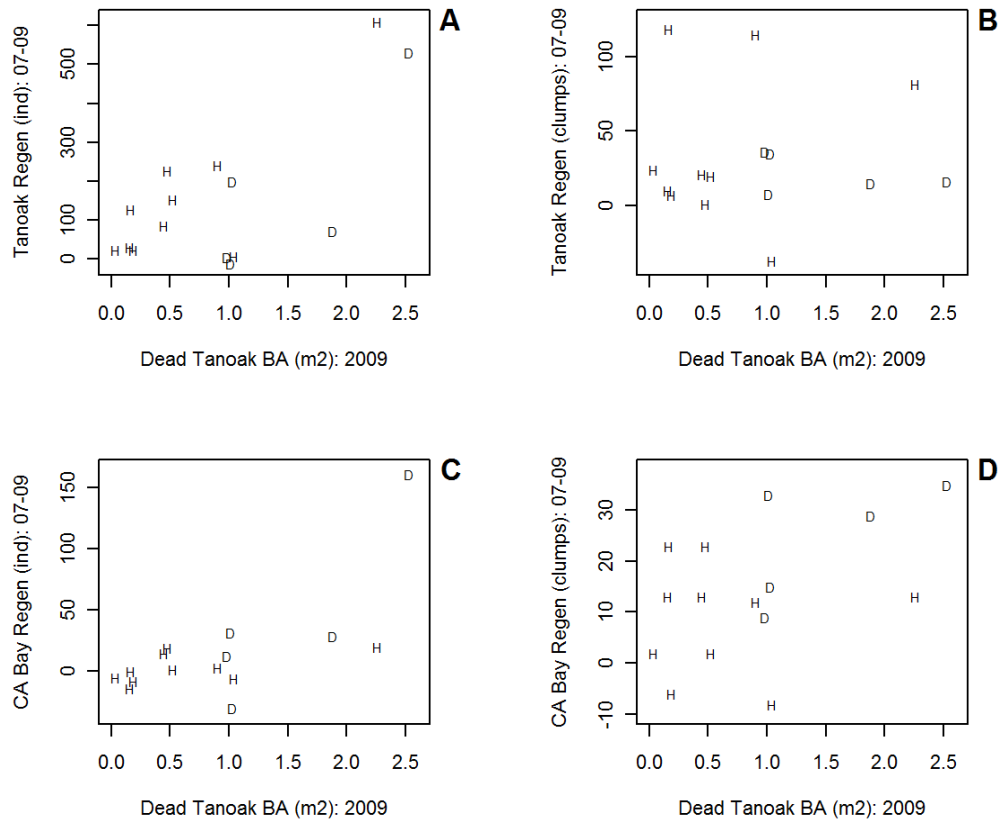


Figure 9. Regeneration (2009) vs. dead tanoak basal area (2009) in Douglas-fir plots. H and D plots are identified with the letters “H” and “D”.

Regeneration of hardwoods-excluding-tanoak was not definitively different between healthy and diseased plots, but continuous analyses did yield several significant results. In models with total tanoak, the amount of dead tanoak basal area in 2009 predicted two-year change values (but not 2009 values) for individuals and clumps, while in models without total tanoak, the amount of dead tanoak basal area in 2009 predicted regenerating individuals (2009 and two-year change values), but did not predict clumps.

Tanoak regeneration (which accounted for the vast majority of total regeneration in Douglas-fir plots) was unrelated to plot strata in the Douglas-fir vegetation type, but in continuous models not including total tanoak as a predictor, regeneration of tanoak individuals (2009 counts as well as two-year change values) was positively related to the amount of tanoak basal area that was dead in 2009 (Table 8; Fig. 9a/b). Douglas-fir regeneration was not significantly related to tanoak mortality in any of our categorical or continuous analyses. California bay regeneration results were nearly identical to hardwood-excluding-tanoak results (the former accounts for the vast majority of the latter in our Douglas-fir plots; Fig. 9c/d).

3.4.4. Fuels and fire hazards

Dead and downed fuel loading increased across all size classes in both redwood and Douglas-fir forest types (Fig. 10 and 11). Increases were largest in the redwood plots and in particular in the 1000 hour fuel class. Tanoak mortality was a significant predictor of the increase in 1000 hour fuels in redwood plots (Table 9). Douglas-fir plots experienced smaller

increases in dead and downed fuel loading and these increases were limited primarily to the smaller fuel classes. Tanoak mortality was a significant predictor of increases in fuel loading in Douglas-fir plots in the 1 and 10 hour fuel classes (Table 10). Changes in the litter and duff depths were subject to measurement errors in 2007 and 2009 and are therefore not the focus of this analysis.

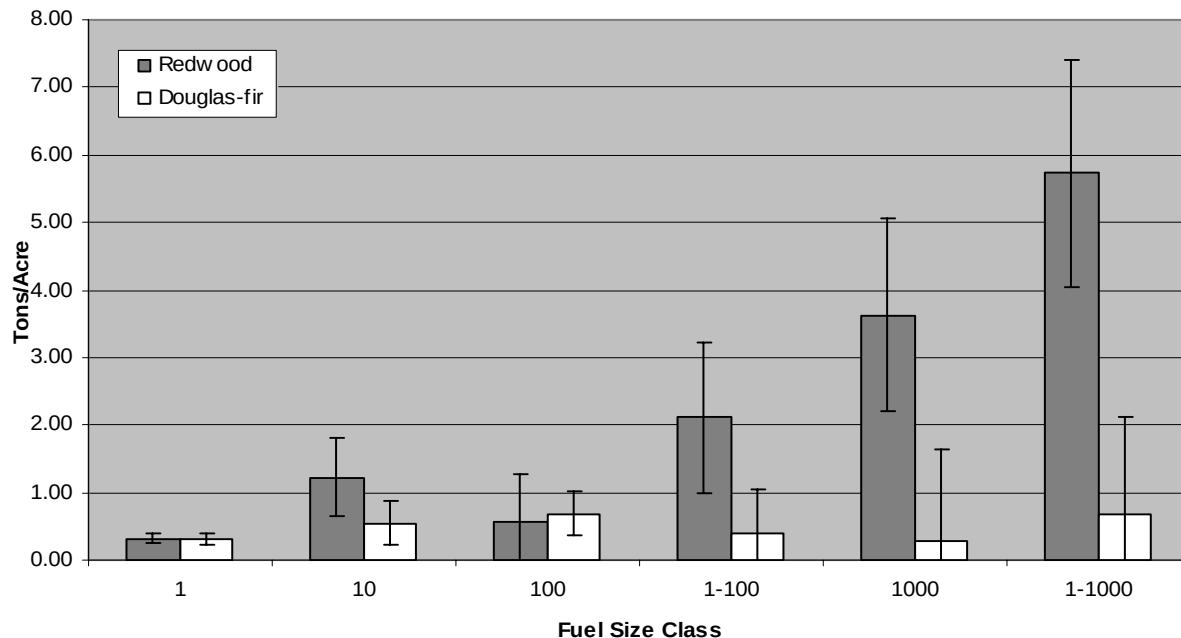


Figure 10. Change in fuel loading from 2007 to 2009 in redwood versus Douglas-fir plots.

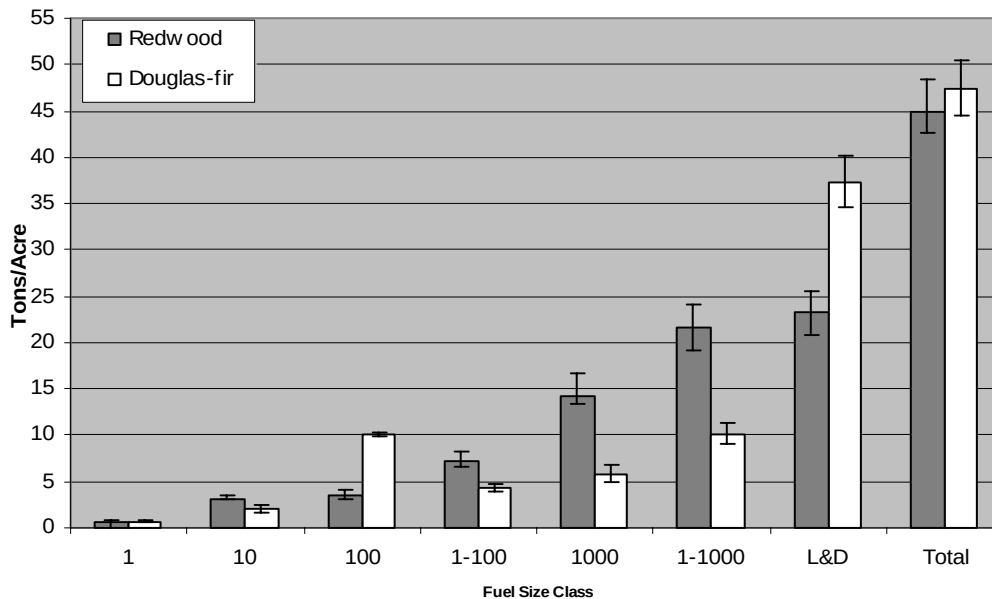


Figure 11. Total fuel loading in redwood versus Douglas-fir plots in 2009.

Table 9. Fuel loading as a function of tanoak mortality (redwood plots).

See Table 3 for a description of symbols.

Veg = RW	(total tanoak incl. as a predictor)			(total tanoak <i>not</i> incl. as a predictor)			D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	Ttl Dd: 09	Ttl Dd: 07	B/F Dd: 07	
1 hr: 09	0	0	0	0	0	0	0
1 hr: 07-09	0	0	+	0	0	0	0
10 hr: 09	0	0	0	0	0	0	0
10 hr: 07-09	0	0	0	0	0	0	0
100 hr: 09	0	0	0	0	0	0	0
100 hr: 07-09	0	0	0	0	0	0	0
1000 hr S: 09	0	0	0	(+)	(+)	(+)	(+)
1000 hr S: 07-09	0	0	0	0	0	0	0
1000 hr R: 09	0	0	0	0	0	0	0
1000 hr R: 07-09	+	+	+	+	(+)	+	+
1000 hr: 09	0	0	0	+	(+)	(+)	+
1000 hr: 07-09	0	0	0	0	0	0	0
1-100 hr: 09	0	0	0	0	0	0	0
1-100 hr: 07-09	0	0	0	0	0	0	0
1-1000 hr: 09	(+)	0	0	+	+	(+)	+
1-1000 hr: 07-09	0	0	0	0	0	0	0
Litter: 09	0	0	0	0	0	0	0
Litter: 07-09	0	0	0	0	0	0	0
Duff: 09	(-)	-	0	0	0	0	0
Duff: 07-09	0	(-)	0	0	0	0	0
L & D: 09	0	0	0	0	0	0	0
L & D: 07-09	0	0	0	0	0	0	0
Total: 09	0	0	0	(+)	(+)	0	+
Total: 07-09	0	0	0	0	0	0	0

Table 10. Fuel loading as a function of tanoak mortality (Douglas-fir plots).

See Table 3 for a description of symbols.

Veg = DF	(total tanoak incl. as a predictor)		(total tanoak <i>not</i> incl. as a predictor)		D plot (vs. H plot)
	Ttl Dd: 09	Ttl Dd: 07	Ttl Dd: 09	Ttl Dd: 07	
1 hr: 09	0	0	0	(+)	0
1 hr: 07-09	0	+	0	+	(+)
10 hr: 09	+	0	+	(+)	+
10 hr: 07-09	0	(+)	+	(+)	(+)
100 hr: 09	0	0	0	0	0
100 hr: 07-09	0	0	0	0	0
1000 hr S: 09	0	0	0	0	0
1000 hr S: 07-09	-	0	0	0	0
1000 hr R: 09	0	0	0	0	0
1000 hr R: 07-09	0	0	0	0	0
1000 hr: 09	0	0	0	0	0
1000 hr: 07-09	-	0	0	0	0
1-100 hr: 09	(+)	0	0	0	(+)
1-100 hr: 07-09	0	0	(+)	0	0
1-1000 hr: 09	0	0	0	0	0
1-1000 hr: 07-09	0	0	0	0	0
Litter: 09	0	0	0	0	0
Litter: 07-09	0	0	0	0	0
Duff: 09	0	0	0	0	0
Duff: 07-09	+	++	0	++	++
L & D: 09	0	0	0	0	0
L & D: 07-09	0	++	0	+	(+)
Total : 09	0	0	0	0	0
Total : 07-09	0	+	0	0	0

4. DISCUSSION

4.1. Baseline comparison of redwood and Douglas-fir vegetation types

Many variables distinguish the redwood and Douglas-fir forests of Point Reyes National Seashore. Our field observations indicate that the redwood forests of Bolinas Ridge generally exhibit much more tanoak than the Douglas-fir forests of Inverness Ridge, but due to our plot selection protocol (which prescribed a minimum tanoak basal area) our data specifically assess differences between areas with similar tanoak components. We found that the Douglas-fir vegetation type, in comparison to the redwood vegetation type, exhibited greater herb cover, reduced herb evenness, greater shrub richness, reduced shrub evenness, greater total vascular plant richness, greater mature tree diversity, greater total tree diversity (juvenile and mature combined), greater total vascular plant diversity, greater canopy cover, greater abundance of mature non-tanoak hardwoods (pooled), greater abundance of mature California bay, greater abundance of non-tanoak hardwood regeneration, greater abundance of California bay regeneration, reduced abundance of mature conifers (in terms of stem counts, but not basal area),

and reduced abundance of conifer regeneration. The non-tanoak hardwood category (which includes California bay) provides a metric of functional redundancy within these two vegetation types, while the data for California bay alone was included in order to examine why SOD appears to be progressing so quickly through the Douglas-fir forests of Inverness Ridge (see next section).

If we assume that tanoak mortality will be ultimately equivalent in all areas of the park (i.e. approach 100%), impacts to redwood and Douglas-fir forests will diverge for at least two reasons. First, because tanoak is more abundant in redwood forest, the direct impacts of SOD will be greater in this vegetation type. Second, because of the compositional differences we detected between our redwood and Douglas-fir plots, areas with similar tanoak components will also exhibit divergent residual vegetation, which will likely lead to differing impacts to other trophic levels and ecosystem processes, as well as distinct responses to this novel disturbance. For instance, redwood forests exhibit less functional redundancy (i.e. less abundance of non-tanoak hardwoods) and less richness and/or diversity of several other functional groups as well, and thus the loss of tanoak will probably create a greater risk of cascading impacts in this vegetation type. Furthermore, our redwood plots exhibit less non-tanoak hardwood regeneration and more conifer regeneration than our Douglas-fir plots, suggesting a long-term trajectory in which conifers (primarily redwood) expand their dominance in the redwood forests of Bolinas Ridge while non-tanoak hardwoods exploit the growing space vacated by tanoak in the Douglas-fir forests of Inverness Ridge. In summary, our field observations and statistical findings suggest that – given a scenario of 100% tanoak mortality in both vegetation types – both short- and long-term impacts will be greater in redwood forest. However, as discussed in the section below, the assumption of eventually equivalent proportional mortality rates in both vegetation types may not be realistic.

4.2. Disease progression

From 2007 to 2009, new mortality occurred throughout the park in all four sampling strata. During plot installation, which occurred in 2007, pooled proportional tanoak mortality in D plots was equal (in terms of stem counts) and higher (in terms of basal area) in redwood forest as compared to Douglas-fir forest. However, from 2007 to 2009, mortality occurred at a much faster rate in the latter vegetation type, and by 2009, the pooled proportional mortality of tanoak in D Douglas-fir plots had surpassed that in D redwood plots, in terms of stem counts (0.80 vs. 0.67) and basal area (0.97 vs. 0.76). In H plots, trends were similar and increases in proportional mortality were again much greater in the Douglas-fir vegetation type, but cumulative mortality remained lower in H plots than in D plots.

Given that we do not know with certainty when SOD-induced tanoak mortality began, we cannot calculate mortality rates over the entire period of disease impact, but we can calculate mortality rates within our sampling window. For example, of the tanoak trees that were alive in D redwood plots in 2007, 15.7% had died by 2009, yielding an annual non-compounded mortality rate of 7.9%. And of the tanoak trees that were alive in D Douglas-fir plots in 2007, 45.5% had died by 2009, yielding the astoundingly high annual non-compounded mortality rate of 22.7%. This rate was undoubtedly influenced by our 2007 plot selection protocol, which required a minimum basal area of tanoak that was either dead or exhibiting advanced symptoms (Appendix A); due to the recent onset of disease on Inverness Ridge, these criteria had the effect of locating D Douglas-fir plots in areas that contained a large amount of tanoak basal area that was symptomatic but still alive in 2007. Across all four of our sampling strata (redwood and

Douglas-fir, H and D), the annual non-compounded mortality rate was 8.1%. In comparison, according to Barrett (2006), prior to the introduction of SOD, the statewide natural mortality rate for tanoak trees $\geq 12\text{cm}$ DBH was approximately 0.36% per year.

The mortality rates we have discovered in diseased areas of PRNS are also higher, especially in the Douglas-fir vegetation type, than the rates reported for other SOD-impacted areas. In the Marin Municipal Watershed District (which is adjacent to PRNS), between 2000 and 2008, McPherson et al. (2010) calculated a non-compounded tanoak mortality rate of 5.4% per year (for trees that were a. $\geq 5\text{cm}$ DBH, b. asymptomatic at first measurement, and c. ultimately symptomatic); this rate would be higher if trees that were symptomatic in 2000 and/or trees that died but never exhibited SOD symptoms were also included, but the authors do not provide this statistic. However, they do indicate that, by 2008, 51% of all tanoak trees that were alive in 2000 had died. In a study that spanned redwood forests from Monterey to Sonoma counties, but only encompassed one full year (2002 to 2003), Maloney et al. (2005) found an annual tanoak mortality rate of 6.0% for trees that were $\geq 1\text{cm}$ DBH and deemed to be infected with *P. ramorum* (this rate would have been higher if all tanoak deaths were included). We do not know the extent to which the very high annual mortality rates we have discovered are a result of a. a limited time frame that was especially conducive to mortality, b. a location that is very favorable to disease intensification, or c. our specific field sampling and calculation methods. Nonetheless, it appears that the rate of tanoak mortality we have documented at PRNS is among the highest that has been observed.

SOD became established in the redwood forests of Bolinas Ridge several years prior to the Douglas-fir forests of Inverness Ridge (this claim is supported by our data documenting more advanced deterioration of dead trees in redwood plots, our 2007 field observations of mortality patterns throughout the park, and earlier observations by park staff). However, our data clearly demonstrate that mortality is occurring much more rapidly within the Douglas-fir vegetation type. Several factors could explain this phenomenon. Total inoculum loads may be higher in Douglas-fir forests as result of community characteristics. California bay, which is more abundant in our Douglas-fir plots, supports a higher level of sporulation than any other known host (Davidson et al. 2008), and it also feasible that other plants (many of which have not been thoroughly investigated) could be contributing to the magnitude of *P. ramorum* spores being produced in this vegetation type. In addition, it is possible that microclimate differences between Inverness and Bolinas Ridges could contribute to the observed differences in mortality rates. Sporulation of *P. ramorum* is most prolific with high humidity and mild temperatures (Davidson et al. 2005), suggesting that Inverness Ridge may be providing ideal conditions for pathogen spread. Finally, although we do not have any evidence to support this hypothesis, it is conceivable that the tanoak population on Inverness Ridge is more genetically susceptible than the tanoak on Bolinas Ridge. Our experimental design does not facilitate direct testing of disease spread at the extra-plot scale, but given the increases in mortality in the H Douglas-fir sampling strata (in which plots exhibited little or no mortality in 2007), we surmise that spread has been occurring at scales beyond what we captured within our plots.

The proportion of tanoak trees that were asymptomatic decreased from 2007 to 2009 in all sampling strata (in terms of both basal area and stem counts), and the proportion of tanoak trees that exhibited symptoms also decreased (with the exception of stem counts in H redwood plots, which showed a small increase). These results suggest that most of the mortality that occurred between 2007 and 2009 was due to infections that occurred prior to 2007 (or at least infections that did not yield symptoms until after the summer of 2007), or that most infections

that occurred after the summer of 2007 had already proven fatal by our 2009 sampling period. However, summary data for changes in the proportion of symptomatic trees are hard to interpret because we cannot necessarily assume that all trees will progress from asymptomatic to symptomatic to dead; for instance, trees may die rapidly without showing symptoms or potentially even recover from a symptomatic state and appear asymptomatic in the future (McPherson et al. 2010). Additional clarification of such patterns would be revealed by analysis of health status transitions on an individual tree basis. Similarly, incorporating data on incubation period (i.e. the expected time from infection to symptom development and/or tree death), in conjunction with inter-annual weather patterns, may lead to interesting insights. We plan to pursue both of these initiatives as part of a future project.

In redwood plots, increases in mortality were fairly consistent whether measured as stem counts or basal area, indicating that tree death was not strongly correlated with DBH. In contrast, proportional mortality increases in Douglas-fir plots were much greater in terms of basal area than stems counts, indicating that larger trees experienced more mortality than smaller trees. Greater likelihood of SOD-induced mortality and/or infection for tanoaks with larger diameters has also been documented by other researchers (McPherson et al. 2010, Cobb et al. 2008).

4.3. Factors affecting disease severity

Aside from total tanoak basal area, no variables were able to significantly predict the abundance of dead tanoak basal area in 2007, in any of the models in which they were included. However, elevation and median tanoak DBH displayed borderline negative relationships in analyses with Douglas-fir plots only. With regard to elevation, we believe this result is not indicative of a meaningful ecological constraint, but rather is due to the fact that, in 2007, SOD had only recently become established in the Douglas-fir forests west of highway one, and had not yet spread up to the higher portions of Inverness Ridge. The borderline negative association between median tanoak DBH and dead tanoak basal area, in Douglas-fir forests, perhaps indicates that sporulation on smaller tanoaks helps to drive infection and mortality of larger trees in this vegetation type, but any such effect appears to be minimal. As such, out of the variables we examined, the only definitive predictor of dead tanoak basal area appears to be total tanoak basal area. No variables were significant predictors of the *percent* of tanoak that was dead in 2007, but in some model variants, this metric of disease severity was weakly positively associated with the basal area of all non-tanoak hardwoods combined and the basal area of California bay (which is also included in the former combined grouping).

Several variables were significant predictors of the *change* in dead tanoak basal area between 2007 and 2009. This mortality metric was positively related to total tanoak basal area in all model variants except the variant utilizing redwood plots only; the lack of such a relationship in our redwood plots makes sense given that a) in diseased redwood plots, a very high percentage of total tanoak basal area was already dead in 2007, and b) in comparison to Douglas-fir plots, redwood plots did not experience much mortality between 2007 and 2009. In Douglas-fir plots, median DBH (of all species combined) was negatively related to the two-year change in tanoak mortality, suggesting that the disease may intensify more rapidly in environments with many small stems (e.g. tanoak and/or California bay) in this vegetation type. This hypothesis is consistent with two of the relationships noted above: the weak negative association between median tanoak DBH and 2007 dead tanoak basal area in Douglas-fir plots, and the weak positive association between the *percent* of tanoak basal area that was dead in 2007 and the basal area of

California bay. However, in contrast, we found that California bay basal area was negatively related to the two-year *change* in dead tanoak basal area (but in only one of the four model variants; it was insignificant in the other three variants). In both model variants in which redwood and Douglas-fir plots were pooled, the combined basal area of all species other than tanoak was negatively related to the change in dead tanoak basal area from 2007 to 2009. This finding may simply reflect the fact that total basal area must be finite in any given area (we found a borderline negative association between tanoak basal area and the combined basal area of all species other than tanoak; results not shown), and that – as described above – dead tanoak basal area (2007 values as well as two-year change values) was positively related to total tanoak basal area. We have also found suggestive evidence that several other variables may be related to the two-year change in tanoak mortality, but we have not attempted to interpret many of the relationships that exhibited only borderline significance and/or were only apparent in one of the four model variants (see Results section).

The only significant predictor of the two-year *change* in the *percent* of dead tanoak basal area was the basal area of all species other than tanoak; this relationship was negative and was only significant in model variants that pooled plots in both vegetation types. This relationship is identical to the relationship between this predictor variable and the two-year change in *absolute* dead tanoak basal area, and our interpretations are the same (see above). Several other predictor variables also exhibited relationships with the two-year *change* in the *percent* of dead tanoak basal area that were similar to the two-year *change* in *absolute* dead tanoak basal area: a) total tanoak basal area was weakly positively associated with the two-year change in the percent of dead tanoak basal area, but only in the model with Douglas-fir plots alone; b) median DBH (of all species) was weakly negatively associated with the two-year change in the percent of dead tanoak basal area, but only in the model with Douglas-fir plots alone; and c) basal area of California bay was weakly negatively associated with the two-year change in the percent of dead tanoak basal area, in the two model variants that pooled redwood and Douglas-fir plots. We have also found suggestive evidence that a few other variables may be related to the two-year change in the percent of tanoak basal area that was dead, but we have not attempted to interpret some of the relationships that exhibited only borderline significance and/or were only apparent in one of the four model variants (see Results section).

California bay abundance has proven to be a strong predictor of SOD-induced mortality in drier ecosystems (e.g. mixed evergreen forest, oak woodland), especially with regard to coast live oak (McPherson et al. 2010), but if this species is playing a role in the epidemiology of SOD in the moist, frequently fog-shrouded, conifer forests of PRNS, the effect is not easily detectable at the scale of our plots. California bay is clearly not essential for severe tanoak mortality in the redwood vegetation type, where it is entirely absent from many of our most severely diseased plots. In the Douglas-fir vegetation type, nearly all plots contain California bay, but our results display weak and/or contradictory relationships between disease severity and the abundance of this species. As such, within PRNS, California bay abundance is a poor predictor of disease severity at the plot scale, but - as discussed in the section above - it is possible that California bay sporulation is contributing to disease severity at a larger scale, at least in some parts of the park. Our results also suggest that there may be some link between disease severity and median DBH (of tanoak and/or other species), but such relationships are not consistent across mortality metrics, model variants, or vegetation types; if stand structure is playing a role in SOD epidemiology (i.e. small stems contributing to mortality of larger trees), this effect is not particularly pronounced at the plot scale. In terms of the absolute amount of dead tanoak basal

area (as compared to the percent of tanoak basal area that was dead), total tanoak basal area was the only strong and consistent predictor. In summary, after accounting for total tanoak basal area (which necessarily constrains dead tanoak basal area, at least at high levels of mortality), we posit that current mortality patterns within the park are primarily the result of introduction events and stochasticity; a similar argument was made by Maloney et al. (2005) with regard to redwood forests from Monterey to Sonoma counties. Unfortunately, this suggests that disease expansion and progression should be expected to continue, and that relatively unaffected areas with a significant tanoak component will probably be impacted eventually, although it is possible that rates of disease progression will be somewhat affected by California bay abundance and/or stand structure.

4.4. Disease Impacts

4.4.1 Cover classes

SOD-induced tanoak mortality has led to a significant reduction in canopy cover in both the redwood and Douglas-fir vegetation types; this claim is supported by several different analyses, as well as our field observations. In 2009, the mean canopy cover (as quantified with a spherical densiometer) was 93.5% in H redwood plots and 78.4% in D redwood plots. However, the mean value for D plots is somewhat misleading because several plots with considerable canopy deterioration contained clumps of intact trees near plot center (where canopy cover readings were taken) and/or had high redwood canopies above a severely impacted lower canopy stratum composed primarily of tanoak. If light levels were quantified directly, as opposed to canopy cover (which does not account for the density of foliage), or if measurements had been taken at multiple locations throughout each plot, we suspect that our data would have shown a greater divergence between H and D plots. We also estimated canopy cover with ordinal Braun-Blanquet cover classes; unlike the densiometer approach, this method was not affected by canopy cover at any particular point in the plot, but it was still very sensitive to a high thin layer of foliage. Furthermore, the cover class method was characterized by a great deal of imprecision (e.g. only two classes spanned the entire range from 50-95%). For these reasons, our cover class estimates are not any more revealing than our densiometer readings. As numerical evidence of the degree to which SOD has impacted canopy cover in at least some plots, the two D plots with the greatest crown deterioration (densiometer method) had canopy cover values of 60.0% and 61.8%. In contrast, the two lowest canopy cover values for H plots were 86.0% and 91.9%.

In Douglas-fir plots, the mean canopy cover was 81.8% in D plots and 92.8% in H plots. Some of the concerns regarding quantification of canopy cover discussed above with regard to our redwood plots may also apply in the Douglas-fir vegetation type, but any misleading characteristics of the data are not yet readily apparent. Due to the earlier stage of disease progression in our Douglas-fir plots, most dead tanoak trees are still standing and still have many of their dead leaves attached to their canopies. As such, canopy cover may decline rapidly over the next several years as dead leaves fall and canopies collapse. In the Douglas-fir vegetation type, the two lowest canopy cover values for D plots were 69.3% and 75.3%, while the two lowest canopy cover values for H plots were 86.2% and 87.3%.

In the redwood vegetation type, 2009 herbaceous cover exhibited consistently negative relationships with tanoak mortality in the redwood vegetation type, but we could not detect any relationships between tanoak mortality and the change in herb cover from 2007 to 2009. These results suggest that diseased areas have experienced a decline in herbaceous cover since the introduction of SOD, but that a relatively small proportion of this decline occurred between 2007

and 2009; however, we also recognize that our ordinal class-based method of quantifying cover was not very precise, making it difficult to detect changes over only a two-year period. Several different mechanisms could potentially explain a decline in herbaceous cover in diseased areas; for instance, one or more herb species that were present in redwood forest prior to tanoak mortality may be a) intolerant of the increased light levels that characterize mortality gaps, b) suffering as a direct result of infection by *P. ramorum*, and/or c) experiencing increased herbivory. Tanoak mortality was unrelated to herb cover in our Douglas-fir plots, which is not surprising given the relatively recent establishment and intensification of SOD in this vegetation type. Shrub cover, juvenile tree cover, and exotic plant cover were all unrelated to tanoak mortality in both vegetation types, suggesting that SOD has not – at least as of 2009 – led to a major structural transformation of the understory or facilitated an invasion process. However, our data do suggest that compositional shifts and subtle changes to understory structure are already underway.

4.4.2. *Species richness*

There is evidence that SOD-induced tanoak mortality is increasing herbaceous species richness in redwood and Douglas-fir forests. In redwood plots, 2009 herbaceous species richness was positively related to the amount of tanoak basal area that was dead in 2009 and the amount that was dead in 2007, but only after accounting for the effects of total tanoak abundance. Herb richness was not higher in D plots than H plots, but since plots with greater tanoak mortality also tended to have greater total tanoak basal area, and total tanoak basal area was negatively related to herb richness (results not shown), the positive relationship between tanoak mortality and herb richness was only apparent after accounting for total tanoak basal area. Early mortality (i.e. trees that were broken/fallen by 2007) was not predictive in any model, and plots with the highest levels of total mortality in 2007 and 2009 did not necessarily exhibit the highest levels of early mortality (results not shown), suggesting that a) significant recruitment of previously absent herb species requires crossing a mortality threshold, and b) this threshold was generally not exceeded solely by those trees that had already broken or fallen by 2007. However, given our finding of decreased herbaceous cover in areas with greater mortality (see above), it is also possible that – by 2009 – some shade-loving herb species had begun to disappear from plots with considerable early mortality; if this hypothesis is true, herb richness may peak several years after the onset of significant mortality, after the establishment of shade-intolerant species, but before the disappearance of shade-loving species.

In the redwood vegetation type, the two-year change (2007 to 2009) in juvenile tree richness increased with the amount of tanoak basal area that was broken/fallen in 2007, but only when total tanoak abundance was not included as a model predictor. All other analyses with juvenile tree richness (including all those utilizing absolute 2009 values, as opposed to two-year change values) were entirely unrelated to tanoak mortality. As such, while it is possible that the relationship between early mortality and tree species recruitment represents a real ecological phenomena (e.g. a year of abundant seed production by one or more tree species occurring prior to 2007), the lack of statistical significance in all other related analyses suggests the possibility that this result could be nothing more than a statistical anomaly. Shrub richness exhibited no relationships with tanoak mortality in any analyses, and – in support of our earlier suggestion that SOD is not currently facilitating invasion of the conifer forests of PRNS, even in the heavily impacted redwood forests of Bolinas Ridge – exotic species richness was also entirely unrelated to tanoak mortality. The effects of tanoak mortality on total vascular plant richness were nearly

identical to the effects on herb species richness; the latter comprises a substantial portion of the former and thus the increase in total richness probably simply reflects the increase in herb richness.

In the Douglas-fir vegetation type, 2009 herbaceous species richness increased with tanoak mortality in all analyses, strongly suggesting that tanoak mortality has led to herb species recruitment. However, we found no connection between tanoak mortality and the two-year change in herb richness, and we also detected several positive relationships between tanoak mortality and 2007 herb species richness (results not shown), despite the fact that 2007 mortality levels were quite low in Douglas-fir plots. Such observations present the possibility that diseased areas may have had higher herb richness prior to the establishment of SOD, but because 2007 relationships were not as strong as 2009 relationships (results not shown), it is conceivable that some herb species in Douglas-fir forests could simply be very sensitive to slightly increased light levels. Shrub richness, juvenile tree richness, and exotic species richness were unrelated to tanoak mortality in all analyses. Total vascular plant richness results were nearly identical to herb richness results, probably reflecting the fact that the latter comprises a substantial fraction of the former.

4.4.3. Regeneration

In the redwood vegetation type, we found evidence that SOD-induced tanoak mortality had stimulated a regenerative response in some functional groups and species. Our results very consistently demonstrated that regeneration of hardwoods-excluding-tanoak was greater in areas with higher levels of tanoak mortality. This pattern generally held regardless of whether regeneration was quantified as individuals or clumps, or as static 2009 values or two-year change values. These results appear to be primarily influenced by Pacific madrone regeneration, which accounted for a mean of 67% (calculated within each plot and then averaged across all plots) of the regeneration of hardwoods-excluding-tanoak (individuals in 2009) in the redwood vegetation type. Relationships with tanoak mortality were very similar for the species and the functional group to which it belongs, and the consistency across all response variable expressions (individuals vs. counts, 2009 vs. two-year change values) can be explained by the fact that the vast majority of Pacific madrone clumps consisted of only one individual, and only one plot contained any Pacific madrone regeneration in 2007. It is possible that the sudden spike in Pacific madrone regeneration occurred because a threshold light level was recently crossed in several plots, but because conditions vary considerably between D plots, we suspect that a heavy seed crop may have matured between our 2007 and 2009 field measurements. Regardless of the mechanism, it may be tempting to infer that this species is poised to exploit the growing space made available by the death of tanoak in the redwood vegetation type, but there are two important qualifications that must be emphasized: a) despite the strong statistical relationship between tanoak mortality and Pacific madrone regeneration, the median number of regenerating madrone individuals in D plots was only two, and b) the vast majority of Pacific madrone regeneration was in the form of very small seedlings, and thus long-term survival of these recruits should not be assumed.

There is some evidence that California bay regeneration (in 2009) was greater in areas of redwood forest with more tanoak mortality, but results were not very strong or consistent. Relationships between California bay regeneration and tanoak mortality are similar if 2007 regeneration values are used (results not shown), which might indicate that a relatively early, albeit weak, regenerative response occurred, but it is important to recognize that this pattern

could also be a subtle sign of reverse causation. While we did not find definitive evidence that disease severity is linked to the basal area of California bay trees within our sampling plots (see above), it is possible that plots with greater numbers of California bay seedlings have more mature representatives of this species in the general vicinity, and are within the range of frequent *P. ramorum* spore dispersal. However, because California bay seeds are dispersed by a variety of animals, some of which might be attracted to mortality gaps, we cannot conclude that regeneration is positive proof of proximate adults, and thus we remain uncertain about the importance of California bay in the moist redwood forests of Bolinas Ridge. Furthermore, although there is suggestive evidence of a pattern, any such relationship is rather weak, and the median number of regenerating California bay individuals in redwood plots in 2009 was very low (one in D plots, and zero in H plots).

We have found some evidence that tanoak mortality has increased conifer regeneration, in terms of clumps but not individuals, in redwood forests. Relatively recent mortality (i.e. trees that were not broken or fallen by 2007) was a significant predictor of the number of clumps present in 2009, but early mortality was unrelated to regeneration of this functional group, suggesting that 2009 conifer regeneration was little affected by tanoak mortality that occurred more than several years prior to 2007. Two-year changes were statistically unrelated to tanoak mortality, but the number of conifer clumps increased in all redwood D plots from 2007 to 2009, and results from analyses utilizing 2007 clump values (results not shown) were similar to, but not as strong as, results utilizing 2009 values. As such, we infer that a regenerative response by conifers began prior to the summer of 2007, and that new clumps continued to recruit after our initial field measurements, surviving at least until our 2009 field season.

Redwood regeneration results were very similar to pooled conifer results, which makes sense given that regenerating redwood individuals accounted for 82% of all conifer individuals, and 78% of all conifer clumps, in the redwood vegetation type in 2009 (calculated within each plot and then averaged across all plots). Redwood clumps were sometimes composed of a large number of individual sprouts, which probably rendered it more difficult to detect changes in the number of individuals than in the number of clumps (for redwood individually as well as for the conifer functional group). Given that no more than a few individuals within a redwood sprout clump are likely to grow into mature trees, we suggest that the clump may be a more biologically meaningful metric for this species. It is also very important to point out that redwood regeneration was tightly coupled to the number of mature redwood stems within a plot (Pearson's correlation coefficients for individuals and clumps, in 2007 and 2009, were 0.73, 0.70, 0.81, and 0.78, respectively). Redwood seedlings and root sprouts were very rare in all plots, with nearly all redwood regeneration occurring as sprouts at the base of existing trees, casting some doubt on the hypothesis that tanoak mortality has led to an increase in the number of regenerating redwood clumps. However, because we found no connection between tanoak mortality and the number of mature redwood stems (results not shown), our results collectively suggest that redwood regeneration is a function of existing redwood trees as well as tanoak mortality.

In redwood plots, Douglas-fir regeneration exhibited a significant positive relationship with tanoak mortality, but only with the amount of tanoak basal area that was broken/fallen in 2007, and only in analyses that accounted for total tanoak basal area. Because plots with more dead tanoak also tend to have more total tanoak, and total tanoak basal area has a negative effect on Douglas-fir regeneration (results not shown), a positive relationship between tanoak mortality and Douglas-fir regeneration is illuminated only after accounting for total tanoak levels. As

such, although our data predict that areas with greater tanoak mortality (assuming equivalent total tanoak basal area) will have more Douglas-fir recruitment, our D plots did not have more regeneration than our H plots.

We have not found strong evidence that tanoak regeneration is related to mortality of mature tanoak trees in the redwood vegetation type, but there is weak evidence that the two-year change values of individuals (but not clumps) were negatively affected by SOD. These results suggest that sprout clumps arising from the bases of SOD-killed trees may be beginning to thin, either as a direct result of SOD infection or as a result of competition between clumps, but that root systems of top-killed trees have not begun to die in appreciable numbers. Finally, we have found no evidence that total regeneration (all tree species pooled) is related to tanoak mortality.

Relationships between regeneration and tanoak mortality in our Douglas-fir plots were quite different from those detected in our redwood plots. Some of these discrepancies were probably related to inherent differences between these two forest types, while others probably resulted from differences in the stage of disease progression. In the Douglas-fir vegetation type, there is some evidence that total regeneration has increased as a result of SOD-induced tanoak mortality (although results were not very consistent across related analyses); regeneration of individuals (2009 values and two-year change values) was positively related to the amount of tanoak basal area that was dead in 2009, but not to dead basal area in 2007, suggesting that a regenerative response may have occurred primarily as a result of mortality that occurred after our 2007 sampling period (e.g. the necessary threshold had not been crossed by 2007). Note that the predictive ability of the amount of basal area that was broken/fallen in 2007 was not examined in our Douglas-fir plots, because nearly all dead tanoak trees in this vegetation type were still structurally intact in 2007.

There is also evidence that regeneration of hardwoods-excluding-tanoak has increased in response to tanoak mortality in our Douglas-fir plots, especially between 2007 and 2009 (analyses utilizing two-year change values were more consistent than analyses utilizing static 2009 values). General findings for this functional group were similar to results for total regeneration, suggesting that trends of the latter may have been strongly influenced by the former; for instance, mortality in 2009 was more predictive of hardwoods-excluding-tanoak regeneration than was mortality in 2007, lending more support to the supposition that a regenerative response occurred as a result of mortality that occurred after our 2007 sampling period. However, regeneration of tanoak was also positively related to tanoak mortality in some analyses, and results were very similar to those for total regeneration, suggesting that total regeneration patterns may have been primarily driven by tanoak (which accounted for 78% of total individuals and 65% of total clumps in 2009; calculated within each plot and then averaged across all plots). If this is the case, the assumption that increased levels of light (or other resources) have increased total regeneration must be examined; tanoak trees that are dead or dying as a result of SOD often produce a tremendous amount of basal sprouts (field observations), so increased regeneration may be a direct result of SOD infection as opposed to an indirect result of resource liberation. This interpretation also explains why the number of individuals was significantly related to tanoak mortality, while the number of clumps was not (for tanoak regeneration as well as total regeneration).

Douglas-fir regeneration was unrelated to tanoak mortality; results for the conifer functional group would be identical because Douglas-fir was the only regenerating conifer we encountered in the Douglas-fir vegetation type. California bay regeneration results were nearly identical to hardwood-excluding-tanoak results, which is not surprising given that the former

accounts for the vast majority of the latter in our Douglas-fir plots (94% of individuals and 96% of clumps in 2009, calculated within each plot and then averaged across all plots); as such, the above interpretations of this functional group might apply solely to this individual species. California bay may have responded rapidly to recent mortality with the production of basal sprouts, which are sometimes dense and abundant (field observations). If additional light penetration, or damage to California bay canopies caused by falling tanoak trees, stimulates growth of dense sprout clumps, this phenomenon would also explain why counts of individuals were more closely related to tanoak mortality than counts of clumps.

4.4.4. Fuel Loading

Fuel loading increased across both vegetation types and across all size classes. The most substantial increases were in 1000 hour fuels in redwood plots. This is no doubt related to the fact that approximately 25% of the tanoak basal area on diseased redwood plots changed from 'dead intact' in 2007 to 'dead broken' in 2009. These large numbers of falling dead tanoaks likely contributed to increased fuels in all size classes, but particularly in the largest size class. This increase in 1000 hour fuels is likely a good indicator of what will happen in Douglas-fir plots in future years as tanoaks that have already died decay further and eventually fall down. Although 1000 hour fuels are not generally considered to be the primary agents of fire spread or the main contributors to flame lengths or heat produced at the flaming front (Rothermel 1972), they would have significant ecological impacts in the case of a wildfire. Increased 1000 hour fuels would lead to increased residence time of fire and overall increases in heat output which can impact plant survival, seed banks and soils among other factors (Neary et al. 1999).

Increases associated with disease progression appeared in smaller fuels in Douglas-fir plots. This is likely related to the fact that Douglas-fir plots are in an earlier phase of disease progression than redwood plots. These increases could increase potential fire spread as well as flame lengths and heat output of the flaming front. It is interesting to note that fuel loading in the small size classes increased by similar amounts in Douglas-fir and redwood plots; increases in these classes for redwood plots were *not* significantly related to disease progression.

4.5. Scope of Inference

Because we opted to impose a rigid minimum on the amount of tanoak basal area within each plot, as opposed to sampling randomly within Douglas-fir and redwood vegetation types, our data do not provide an estimate of the abundance and distribution of tanoak throughout the park. This approach was utilized in order to standardize tanoak abundance across strata, thereby increasing our ability to detect the specific impacts of SOD in two different vegetation types. In order to maximize our chances of detecting the influence of SOD-induced tanoak mortality, we defined a minimum acceptable tanoak component that may be higher than the average abundance of tanoak throughout the sampled vegetation types. Therefore, any inferences we make may not apply to redwood forest or Douglas-fir forest in general, but only to areas with a similar minimum tanoak component.

4.6. Management Implications and Conclusions

Sudden Oak Death is a reality that managers at PRNS and other land managers along the California coast will likely have to deal with for the foreseeable future. Although the distribution of the disease at PRNS continues to be patchy at the local scale, it is present throughout PRNS. It is reasonable to assume that all susceptible forest types will be affected by this disease and that

any local management actions should take the presence of *P. ramorum* into account. Given that, there are several management approaches that the Seashore should consider:

- **Stop the Spread** – Although this disease is already present through PRNS, both staff and visitors have an important role to play in keeping this disease from spreading to regions with susceptible forest types that remain uninfested including the north coast of California, the Sierra Nevada, and the eastern US. Staff should maintain and increase efforts to disinfect equipment after working in infested areas and prior to working in other parks. Additionally, efforts should be made to increase visitor awareness of the disease and what they can do to stop its spread.
- **Visitor Education** – PRNS is in a unique position to be able to educate visitors about Sudden Oak Death. This opportunity could be used not only to inform people about the risk of spreading Sudden Oak Death, but also about invasive organisms more generally.
- **Protect High Value Trees** – Prophylactic treatments such as AgriFos are available and are currently being used by PRNS to protect trees with high cultural or aesthetic value from SOD. This solution is not realistic to implement in wildland areas, but is being used around the park visitor center and Morgan horse ranch.
- **Tanoak Resistance** – Efforts have been ongoing in the research community to develop resistant tanoaks which could then be used for outplanting. PRNS should support these efforts to the greatest practical extent.
- **Ongoing Monitoring** – Long-term monitoring efforts provide important baseline information about how SOD is impacting coastal ecosystems. Efforts such as these would also be likely to serve as early detection systems if issues of concern, such as increased prevalence of invasive species, were to arise in infested forests.
- **Future Research** – There are many more research needs associated with SOD including but not limited to fire risk modeling, impacts to soil microorganisms, impacts on wildlife species such as Northern Spotted Owls, and the viability of experimental plantings to restore forests impacted by this disease.

This study captures two years of disease progression in the Douglas-fir and redwood forests of PRNS. Even in this short window, it yielded some surprising results, such as faster disease progression in Douglas-fir than redwood forests. However, it is likely that many of the impacts of SOD-induced tanoak mortality have yet to become apparent. Future monitoring will provide important information as the impacts of SOD continue to unfold. We have found that nearly every stand of tanoak within PRNS is already impacted by SOD, and thus we believe that emphasis should be placed on understanding the effects of this disease, and considering restorative and/or ameliorative management actions.

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Appendix A:

Criteria for “Healthy” and “Diseased” plots in redwood and Douglas-fir vegetation types

All H plots (redwood and Douglas-fir) were required to contain **a.** at least 0.74 square meters (8 square feet) BA of *green* tanoak (defined below), **b.** at least 0.19 square meters (2 square feet) BA of *green* tanoak in each of 2 separate plot quadrants (i.e. it was unacceptable for all *green* tanoak to be in a single quadrant), and **c.** at most 0.28 square meters (3 square feet) BA of dead tanoak, only 0.19 square meters (2 square feet) of which may include trees which have shed all of their dead leaves, and only 0.09 square meters (1 square foot) of which may be broken or fallen. *Green* trees were defined as having a full canopy with at least 50% of leaves green. Other symptoms were considered irrelevant for the purposes of plot location. For example, a tree with boring dust, *Hypoxylon*, and/or bleeding was counted toward *green* BA provided it had a relatively full canopy. We decided to ignore bole symptoms in our plot selection criteria because of the difficulty in finding locations without bole symptoms, and because of our focus on forest response and impacts (e.g. regeneration, fuel loading). In areas where the vast majority of tanoak trees retained full green canopies, we assumed that light and moisture levels on the forest floor were not appreciably altered, and response to SOD-induced tanoak mortality had not yet occurred, at least not to any considerable extent. Therefore, we assert that plots in which some bole symptoms were present can still be interpreted as exhibiting “baseline conditions”, for the purposes of this study.

Redwood D plots were required to contain **a.** at least 0.65 square meters (7 square feet) BA of dead tanoak, excluding standing dead trees with more than 50% of leaves still clinging to canopy branches (standing dead tanoak with most leaves still attached to the tree were excluded here to insure that tree death has already increased light levels within the plot), **b.** at least 0.19 square meters (2 square feet) BA of dead tanoak (again excluding standing dead trees with more than 50% of leaves still clinging to canopy branches) in each of two separate plot quadrants (i.e. it was unacceptable for all dead tanoak to be in a single quadrant), and **c.** at least 0.28 square meters (3 square feet) BA of broken or fallen dead tanoak. We did not impose any upper or lower limit to *green* tanoak BA in D redwood plots.

Douglas-fir D plots were selected to contain **a.** at least 0.65 square meters (7 square feet) BA of tanoak that was dead (standing, broken, or fallen) or showing advanced canopy symptoms (at least 50% of canopy leaves dead), and **b.** at least 0.19 square meters (2 square feet) BA of tanoak that is dead or showing advanced canopy symptoms in at least two separate plot quadrants. We did not impose any upper or lower limit to *green* tanoak BA in D Douglas-fir plots.

Comparisons of “baseline conditions” (H plots) between redwood and Douglas-fir plots are valid and directly interpretable. Likewise, H plots can be compared with D plots within a forest type to infer changes resulting from SOD, although relative impacts may be affected by both disease severity and vegetation type. D plots cannot be directly compared across forest types, because of differences in disease criteria between Douglas-fir D and redwood D plots, and hence the confounding of disease severity with vegetation classification.

Appendix B:

Symptom and deterioration classes for tanoak and coast live oak

A – Alive

A - Asymptomatic

S – Symptomatic

B – Bole (only bole symptomatic¹)

C – Canopy (only canopy symptomatic)

E – Early (less than 50% of leaves dead)

A – Advanced (more than 50% of leaves dead)

T – Tree (both bole and canopy symptomatic)

E – Early (less than 50% of leaves dead²)

A – Advanced (more than 50% of leaves dead²)

D – Dead

S – Standing (i.e. “intact”)

E – Early ($\geq 50\%$ of leaves still attached)

I – Intermediate ($< 50\%$ of leaves attached)

A – Advanced (all leaves fallen)

B – Broken

7.5 – Broken at a diameter of 7.5 – 15 cm

15 – Broken at a diameter of 15 – 30 cm

30 – Broken at a diameter of 30 – 60 cm

60 – Broken at a diameter of ≥ 60 cm

F – Fallen (root ball pulled out of ground)

¹ Bole symptoms: bleeding, *Hypoxylon*, boring dust, and/or cankers

² nature and degree of bole symptoms irrelevant