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Sudden oak death in redwood forests: Vegetation dynamics in the wake of tanoak decline

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ABSTRACT

Sudden oak death, an emerging disease caused by the exotic pathogen *Phytophthora ramorum*, is impacting coast redwood (*Sequoia sempervirens*) forests throughout coastal California. The most severely affected species, tanoak (*Lithocarpus densiflorus*), is currently widespread and abundant in the redwood ecosystem, but diseased areas are experiencing tremendous mortality and many lines of evidence suggest that sudden oak death could lead to the eventual extinction of this ecologically valuable species from redwood forests. Such a transition will likely result in a wide range of long-term impacts, and current mortality levels will undoubtedly cause many short-term impacts as well, several of which are already apparent. In this study, we focused on vegetation dynamics in the wake of sudden oak death-induced tanoak decline in redwood forests. We documented short-term impacts and explored potential long-term impacts by identifying variables that were correlated with tanoak abundance. In order to account for potentially confounding factors (e.g. slope position and aspect) and isolate the effects of tanoak mortality and total tanoak abundance, we utilized multivariate generalized linear models. Within this analytical framework, we investigated numerous response variables ranging from species richness to canopy cover to regeneration. The current impacts of sudden oak death-induced tanoak mortality included the following: decreased canopy cover, increased coarse woody debris, increased herb species richness, and an increase in the proportion of mature tanoak trees with basal sprouts. Interestingly, despite substantially reduced canopy cover, we discovered that a strong regenerative response was not occurring. Correlates of increasing tanoak abundance included the following: decreased herbaceous cover, decreased herb richness, decreased shrub richness, increased tanoak regeneration, and a decrease in the proportion of mature redwood trees with basal sprouts. In addition to these primary analyses, we conducted supplemental analyses aimed at predicting which tree species might eventually establish in diseased areas, and considered potential explanations for the current paucity of regeneration.

1. INTRODUCTION

Tanoak (*Lithocarpus densiflorus*) is widespread and abundant in coast redwood (*Sequoia sempervirens*) forests, and is believed to be an integral component of the structure and function of these unique ecosystems. As the most abundant hardwood species in conifer forests of California's coastal mountains, tanoak is found alongside redwood throughout the majority of the redwood range (Burns & Honkala 1990, Hunter et al. 1999, Noss 2000). However, the close association between redwood and tanoak may be relegated to history if sudden oak death (SOD), an emerging disease caused by the exotic pathogen *Phytophthora ramorum*, continues to spread throughout coastal California. Current research demonstrates drastic declines in tanoak populations and mounting evidence (e.g. field studies, genetic resistance trials, disease progression models) suggests that SOD could eventually drive tanoak to extinction in redwood forests (Rizzo et al. 2005, Maloney et al. 2005, McPherson et al. 2005, Meentemeyer et al. 2004).

Several tree species succumb to SOD, but tanoak is the most severely affected tree and the most abundant SOD-susceptible species in redwood forests. The extreme susceptibility of tanoak results from a combination of factors: **a.** little or no genetic resistance (greenhouse and field trials have failed to detect any individuals capable of surviving infection), **b.** susceptibility at all ages and size classes, and **c.** ability of tanoak foliage and twigs to support pathogen sporulation, facilitating eventual lethal infection of the bole (all other species that are killed by SOD require the presence of a secondary foliar host for infection of the bole). Furthermore, because many native species support sub-lethal foliar infections, including redwood, *P. ramorum* has almost certainly become a permanent resident of infested areas (Rizzo et al. 2005, Maloney et al. 2005, McPherson et al. 2005, Meentemeyer et al. 2004, Davidson et al. 2008). Tanoak also occurs in other community types (e.g. mixed evergreen forest, Douglas-fir forest; Burns & Honkala 1990), but the moist, mild climate that characterizes redwood forests appears to be especially conducive to pathogen spread and infection (Meentemeyer et al. 2004). Considered collectively, the unique properties of this pathosystem suggest it is incredibly unlikely that tanoak will ever become re-established in diseased redwood forest. Following most disturbances, including all those known to affect redwood forests (Lorimer et al. 2008), the affected species has the capacity to eventually return to similar structures and relative abundances (Oliver & Larson 1996, Picket & White 1985); thus SOD-induced tanoak mortality represents a particularly unique type of disturbance, one which is most often associated with exotic insects or pathogens (Lovett et al. 2006). Tanoak stumps often sprout prolifically following death of the main bole, and it is possible that root systems could be maintained indefinitely if adequate amounts of photosynthate are consistently produced prior to episodic SOD-induced sprout dieback; a similar situation has been occurring for approximately a century with the American chestnut, *Castanea dentata*, and the exotic disease chestnut blight, caused by *Cryphonectria parasitica* (Ellison et al. 2005). However, even if this unlikely scenario were to manifest and tanoak were to avoid absolute extirpation from redwood forests, it would still be rendered *functionally extinct* (sensu Ellison et al. 2005).

SOD-induced tanoak mortality has already affected several aspects of forest structure and composition, and this epidemic will almost certainly lead to longer-term impacts as well. Current mortality rates are estimated to be four times higher than historical levels, and diseased areas have been found to exhibit reduced canopy cover and increased levels of coarse woody debris (Rizzo et al. 2005, Swiecki & Bernhardt 2008, Moritz et al. 2008). Other short-term impacts might feasibly include increases in tree regeneration, expansion of shrub or herb cover, increases in species richness, disruption of mycorrhizal networks, exotic plant invasions, and accelerated erosion, but none of these potential impacts have received much research attention. Long-term impacts are likely to result from the loss, or at least severe decline, of tanoak in redwood forests. Estimation of such impacts will be dependent upon accurate prediction of the tree species that will replace tanoak, and an understanding of how these tree species will affect the abundance and distribution of other species (ranging from understory plants to fungi to wildlife) as well as ecosystem processes.

Species richness and composition are important factors in physical resistance, community resilience, ecosystem function, wildlife habitat, and a wide variety of other ecological attributes. In forested ecosystems, large established trees will likely have the greatest impact (Ellison et al. 2005), but juvenile trees, shrubs, and herbaceous plants may also serve important ecological functions (e.g. wildlife food sources, soil stabilization). The competitive balance of coexisting understory species can be affected by the species of trees comprising the canopy above; for instance, different tree species allow the passage of differing amounts of light, provide leaf litter with unique chemical and microbial properties, and support distinct mycorrhizal communities (Oliver & Larson 1996, Halpern et al. 2005). Hunter et al. (1999) found that light levels beneath tanoak canopies were lower than that of light levels beneath other co-occurring species (e.g. madrone). No research has been conducted on tanoak leaf litter, but it is likely that tanoak's broad leaves exhibit different chemical properties than the needles of redwood. As such, there are several reasons to believe that understory shrubs and herbs, as well as tree regeneration, may vary in response to the abundance of tanoak in redwood forests.

Tree regeneration represents the next generation of canopy trees and it is thus a key predictor of many features of future forests, including, of course, which tree species will comprise the next cohort. In the context of this study, we are especially interested in the question of whether functionally similar native tree species will be able to colonize and persist in areas previously occupied by tanoak. Or, alternatively, might redwood take sole advantage of tanoak's demise and increase its dominance? There is some evidence that the decline of tanoak could primarily benefit redwood, by increasing the growth rates of canopy trees and/or initiating new cohorts (Waring & O'Hara 2008, Moritz et al. 2008). These findings are not definitive and may not be representative of most diseased areas, but they should still be alarming given the tremendous value of tanoak's large nutritious acorns to many wildlife species (including bear, deer, and several rodent and bird species), in contrast to the limited value of redwood's small and light seeds (Burns & Honkala 1990; McDonald & Tappeiner 1987). If tanoak is not replaced by one or more functionally similar tree species, its loss could result in serious cascading impacts; for instance, acorns are a primary food source for the dusky footed woodrat (*Neotoma fuscipes*), which is in turn a primary food source for the northern spotted owl (*Strix occidentalis caurina*; Courtney et al. 2004)

In order to address the questions posed throughout this introductory section, our experimental approach was to install a network of plots throughout sites with varying degrees of SOD-induced tanoak mortality, covering the full spectrum of disease severity and tanoak abundance. Although all our data were collected in a single field season, we believe it is possible to infer many of the immediate impacts of SOD-induced tanoak mortality. Existing research indicates that the current patchy distribution of SOD in redwood forests, at scales of tens to hundreds of meters, is a result of historical and stochastic factors (Maloney et al. 2005, Rizzo et al. 2005, Moritz et al. 2008), as opposed to underlying biotic or abiotic conditions. In effect, SOD is in the process of performing a *natural removal experiment* (for a related argument in another ecosystem, see Leathwick & Austin 2001). As such, it is likely that many of the differences *correlated* with disease severity are in fact *caused* by SOD. In contrast, correlations between tanoak abundance and other variables of interest are quite possibly controlled by other factors that were not measured as part of this study (e.g. soil properties). Therefore, we do not definitively claim a causative role for tanoak abundance. However, we suggest that tanoak abundance is potentially responsible for many of our findings, and present relevant arguments where appropriate.

The primary objectives of this study were to:

1. assess the short-term impacts of SOD-induced tanoak mortality to regeneration, species richness and composition, percent cover of shrubs and herbs, canopy cover, and coarse woody debris

2. compare characteristics of redwood forests across the full range of tanoak abundance, in terms of regeneration, species richness and composition, percent cover of shrubs and herbs, canopy cover, and coarse woody debris

3. consider the potential long-term impacts of SOD-induced tanoak decline

2. METHODS

2.1. Site and plot selection

Given the premise that the current distribution of SOD in redwood forests is essentially stochastic, our sampling protocol began with the installation of a network of plots, randomly located throughout redwood forests with mixed levels of disease severity. Field research was conducted in three different counties at sites which a) contain redwood forest, and b) have been confirmed to be infested with *Phytophthora ramorum* (the causative agent of SOD). Field sites and counties are as follows: Santa Cruz (Henry Cowell Redwoods State Park & The Forest of Nisene Marks), Marin (Marin Municipal Watershed District), and Humboldt (Humboldt Redwoods State Park). The two Santa Cruz county sites are located very near each other and are ecologically similar, and are thus treated as one site in this report.

All plots consist of a 1/20 hectare (12.62 m radius) inner plot, as well as a buffer, which extends to a ¼ hectare area (28.21 m radius); these two elements are collectively referred to as an extended plot. Plots were located in second-growth redwood forest, satisfying the following criteria: **a.** sufficient redwood coverage (at least 25% redwood canopy cover in all four quadrants of the extended plot, estimated visually); **b.** between 50 and 200 meters from trails or roads (the lower limit imposed to avoid edge effects, and the upper limit to aid accessibility and decrease travel time in areas with rugged terrain); and **c.** on slopes less than 60% (31 degrees) for worker safety and minimization of erosion.

Most of our field plots were installed at randomly generated UTM coordinates, which were constrained to areas identified as redwood forest on at least one of several available GIS coverages (e.g. California GAP Analysis, CDF, and local vegetation layers). All randomly generated points are at least 500 meters from all other randomly generated points. When a random point failed to meet the above requirements, the plot was moved 50 meters in a randomly chosen cardinal direction. This procedure was repeated until an acceptable location was discovered; in several cases, it became clear that no reasonable number of iterations would produce a suitable location, and thus the random point was discarded entirely.

The majority of our plots were randomly located, via the method described above, but we also opted to add several supplemental plots; these plots were placed in subjectively selected areas, but precise plot locations were randomized. A preliminary assessment of our data (prior to the completion of fieldwork) displayed a general pattern of increasing dead tanoak abundance with increasing total tanoak abundance, a logical finding given that dead tanoak abundance is necessarily a function of total tanoak abundance ("abundance" will be defined in the data analysis section). Correspondingly, our random plots were missing the relatively rare areas with a great deal of tanoak, but very little tanoak mortality, especially in Marin County. In addition, we were not capturing many of the most severely diseased areas, which are relatively uncommon at the present time. As such, we opted to add supplemental plots in "representative" areas (i.e. no apparent site peculiarities) satisfying one or the other of these conditions. By supplementing our data set with these extreme conditions, we have captured the full range of possible conditions, and have achieved an enhanced statistical ability to detect patterns associated with sudden oak death-induced tanoak mortality (see figure 1). We also opted to add several supplemental plots with little or no tanoak, in order to improve our ability to examine correlates of tanoak abundance. In addition, we installed three supplemental plots in areas where previous SOD-related research had been conducted (Spencer 2004), so that some prior data would be available for comparison. With the addition of the supplemental plots, the total dataset consists of 65 plots.

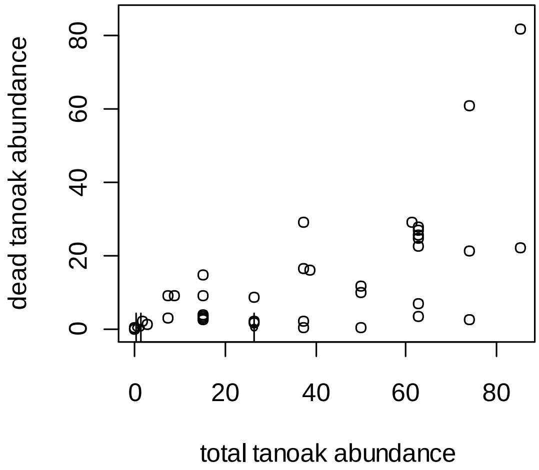

Figure 1a: Dead tanoak abundance plotted against total tanoak abundance; random plots only. N = 43.

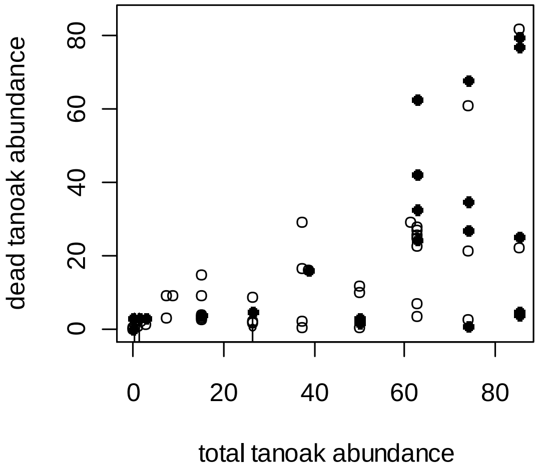

Figure 1b: Dead tanoak abundance plotted against total tanoak abundance; all plots – random (open circles) and supplemental (filled circles). N = 65.

2.2. Data Collection

Within each inner plot, we assigned ordinal cover class estimates (modified Braun-Blanquet) for all vascular plant species; cover classes were recorded as *r* (rare, <0.5%), *t* (<1%), *1* (1-5%), *2* (5-25%), *3* (25-50%), *4* (50-75%), *5* (75-95%) or *6* (95-100%). Tree species were separated into three categories: “juvenile” (seedlings/sprouts/saplings/young trees; all stems less than 10cm DBH), “mature” (all stems greater than or equal to 10cm DBH), and total (mature and juvenile combined). For each shrub species, average heights within the plot were recorded.

Summary cover classes were recorded within the inner plot for total canopy cover, shrub, herb, litter/duff, bare soil, and coarse woody debris (CWD). “Canopy” is tree foliage/branches greater than 5 meters high, including dead foliage on tanoak or other recently dead trees (i.e. anything that blocks sunlight). Herbs were defined as all non-woody vascular plant species. Shrubs and trees were separated by species, not by height, DBH, or other physical measures (for example, very large *Corylus cornuta* specimens were still considered shrubs). CWD was deemed to be any dead woody material that was greater than or equal to 3cm in diameter, and was less than half buried (excluding large chunks of bark, which was considered “litter”).

Because of our focus on recent tanoak mortality, tanoak cover classes included dead mature trees (standing or fallen), ignoring only those trees with highly decomposed wood (defined by whether or not the bole wood compacted when stepped upon). When dead tanoak trees had already fallen, pre-collapse cover classes were visualized and assigned, by examining the size and orientation of the fallen bole and canopy. Estimate of juvenile tanoak cover classes did not include dead individuals. Similarly, estimated cover classes for all individuals (mature and juvenile) of all other species only included living trees. However, as will be explained shortly, all standing dead mature trees of all species were measured and tallied. Finally, several cover classes were estimated for mature trees throughout the plot buffer: living redwood, total tanoak (live and dead), living tanoak, standing dead tanoak, broken/fallen dead tanoak.

For all standing trees greater than or equal to 10 cm DBH, the following variables were recorded: species, health status, diameter at breast height (DBH), and presence/absence of basal sprouts. In order to capture recent SOD-induced tanoak mortality, tanoak stems that were broken below breast height were also recorded, provided that the fallen bole wood was relatively intact (i.e. it did not compact when stepped upon); dead individuals of other tree species were not recorded if broken below breast height. Multi-stemmed trees that were split below breast height were counted as separate trees. A tree was considered to be sprouting from the base if sprouts less than 3 cm DBH were arising from the lowest 10 cm of the bole or within 10 cm of the bole on a root or in duff, litter, or soil (unless an individual stem was obviously a seedling). The health status of all species other than tanoak was recorded simply as alive or dead. Tanoak health and post-mortality deterioration were characterized in great detail, as is shown in Table 1.

- **A – Alive**
  - **A – Asymptomatic**
  - **S – Symptomatic**
    - **B – Bole** (only bole symptomatic)
    - **C – Canopy** (only canopy symptomatic)
      - **E – Early** (< 50% of leaves dead)
      - **A – Advanced** (> 50% of leaves dead)
    - **T – Tree** (both bole and canopy symptomatic)
      - **E* – Early** (< 50% of leaves dead)
      - **A* – Advanced** (> 50% of leaves dead)
  - * nature and degree of bole symptoms irrelevant

- **D – Dead**
  - **S – Standing**
    - **E – Early** (> 50% of leaves still on tree)
    - **I – Intermediate** (< 50% of leaves still on tree)
    - **A – Advanced** (all leaves fallen)
  - **B – Broken** (above breast height)
    - **5 – Broken** at a diameter of 5-10cm
    - **10 – Broken** at a diameter of 10-20cm
    - **20 – Broken** at a diameter of 20-40cm
    - **40 – Broken** at a diameter ≥ 40cm
  - **F – Fallen** (broken below breast height, any diameter)

Table 1. Tanoak health and post-mortality deterioration classes

Canopy cover was measured with a spherical densiometer. Standing at plot center and facing each of the four cardinal directions, the number of quarter squares open in the densiometer were counted (a quarter-square was considered open if sky was visible in more than half of the quarter-square). This process was repeated at 3 meters from plot center along transects in all 4 cardinal directions (N, E, S, W). These data were then averaged and converted to percent cover.

Counts of all seedlings/sprouts (height less than breast height), saplings (taller than breast height, but less than 3 cm DBH), and juvenile trees (DBH 3 cm to 10 cm) were conducted for all tree species in two randomly selected quadrants per plot. Sprouts were only counted if litter, duff, or soil obscured the base of the sprout; this criterion provided a quick and simple method for tallying numbers of sprouts, but note that it differs somewhat from the basal sprouting variable associated with mature trees (as explained above, a mature tree was considered to be sprouting from the base if sprouts were arising from the lowest 10 cm of the bole or within 10 cm of the bole on any medium). Individuals as well as clumps were tallied. "Clumps" were defined as groups of individuals (seedlings/sprouts, saplings, or juvenile trees) separated by at least 30 cm from other conspecific individuals in the same regeneration class. When a single clump exceeded 50 cm along its longest axis, the clump was split in two at a natural break. Solo saplings were distinguished from those which occurred in seedling/sprout clumps. Dead regeneration was not counted in any size category.

In all analyses presented in this report, regeneration is quantified as the total number of clumps of seedlings/sprouts and saplings (seedlings/sprout clumps + solo sapling clumps).

Clumps were analyzed instead of individuals because we believe this metric is more indicative of the number of mature stems that may eventually inhabit an area (e.g. it is obvious that a clump of basal sprouts with 30 individuals will not lead to the establishment of 30 mature trees). Tallies of individuals and clumps are highly correlated for all species, and all results were similar when we tried our analyses with individuals, so this decision is fairly trivial with respect to our overall findings. Saplings and juvenile trees were not examined independently because of very low numbers of occurrences, and juvenile trees were not included in combined clump tallies because solo juvenile trees were not distinguished from those occurring in groups with seedlings/sprouts or saplings.

At each plot, several abiotic characteristics were recorded. One of five primary slope positions was assigned (Alluvial, Lower, Mid, Upper, or Ridge), and one of three secondary slope positions was assigned (Draw, Mid, or Hump). Slope shape (Convex, Flat, or Concave), and aspect were also recorded. For the purposes of analysis, northness was calculated from aspect as per Zar (1999): northness = cosine((aspect * pi)/180).

2.3. Sampling and Testing of Symptomatic Leaves and Twigs

Symptomatic samples of tanoak (leaves and twigs) and/or California bay (leaves only) were collected in all plots, in order to test for the presence of *Phytophthora ramorum*. In many cases, California bay was not available, but tanoak was present in all but one plot. Samples were tested via polymerase chain reaction analysis by the UC Berkeley Forest Pathology Laboratory.

2.4. Data Analysis

To estimate average abundances of tanoak and other tree species or functional groups, we examined randomly located plots only. As such, these estimates are completely unaffected by our subjective efforts to sample the full range of tanoak abundance and disease severity. Average basal area and stem count values are given as means, while regeneration values are presented as medians because a few plots exhibited extremely high outlying values; in addition, we present data on the proportion of plots in which regeneration of species or functional groups is present. To convert per-plot values to per-hectare values, basal area and stem counts were multiplied by 20 (plot area is 1/20 ha), and regeneration counts were multiplied by 40 (because regeneration was only tallied in half of each plot).

Our primary analyses examined the predictive roles of tanoak mortality and tanoak abundance, after accounting for the effects of redwood abundance, sampling site, and several abiotic factors. Response variables included measures of richness, cover, regeneration, and abundance of functional groups of tree species. Stand development stage was not included as a potential predictor because all plots were borderline stem exclusion/understory re-initiation/young multi-strata, and thus we are not very confident in the accuracy or meaningfulness of our classifications. Furthermore, univariate analyses showed entirely insignificant relationships between our stand development stage classifications and all measures of total tanoak and dead tanoak abundance (all p-values greater than .35), and it is therefore unlikely that our primary variables of interest are confounded by stand development stage. In Appendix A, we explore other factors that could be confounded with tanoak mortality, including the presence or absence of California bay, and explain why we believe that the full model specified below is sufficient. Site index was not measured, but our abiotic variables (slope, slope position, and northness) should serve as rough proxies. All primary analyses began with the following full model:

Y ~ dead tanoak abundance + total tanoak abundance + redwood abundance + slope + slope position +
northness + Humboldt + Santa Cruz [Marin = baseline]

Abundance is quantified as a measure of cover that includes the inner plot area as well as the buffer zone: (inner plot cover + buffer cover) / 2. This metric provides equal overall weight to the inner plot and buffer areas, but because the buffer area is larger than the inner plot, inner plot conditions are given more weight per unit of area.

For each response variable, all possible subsets of predictor variables were fit as generalized linear models (GLM), and the model with the lowest Akaike Information Criterion (AIC) value was selected as the "best model". Unless otherwise noted, all multivariate GLM analyses began with the full model shown above. When the response variable was continuous and the residuals were normally distributed and homoskedastic, a Gaussian error distribution was specified. If transformations of the response variable could not satisfy these assumptions, which was often the case with count data such as regeneration tallies, a Poisson error distribution with a log link function was specified. A quasipoisson error distribution, which does not impact parameter estimates but does reduce significance levels, was used instead of a Poisson error distribution when the dispersion parameter was greater than 1 (i.e. for extremely skewed distributions). When the response variable represented the probability of an event occurring, a binomial error distribution with a logit link function (i.e. logistic regression) was used. A quasibinomial error distribution, which does not impact parameter estimates but does reduce significance levels, was used instead of a binomial error distribution when the dispersion parameter was greater than 1. Partial residual plots showed no clear evidence of curvature and thus no squared terms were introduced. Analyses with outliers (i.e. Cook's D values greater than 1), were re-executed after excluding these outliers, and results from these models were compared; all instances in which outlier exclusion yielded qualitatively different results are indicated. Several secondary analyses were also conducted. In these secondary analyses, all statistical methods were identical to the primary analyses described above, but the full set of possible predictors was modified. These modified models are presented along with the results.

In addition to the standard generalized linear model output (e.g. slope estimates, standard deviations, and significance levels), we calculated several other metrics. A p-value for the overall model was calculated with a chi-squared test, and a maximum likelihood equivalent to the ordinary least squares r2, termed "r2.L", was calculated as specified in Quinn and Keough (2002):

$$r2.L = 1 - [\ (\text{residual deviance}/-2)\ /\ (\text{null deviance}/-2)\ ]$$

For each predictor, we also calculated the following metrics: partial r2.L and maximum impact. Partial r2.L is a measure of the explanatory power of each predictor, after accounting for the effects of the other predictors in the model; it is calculated by comparing the deviance of the best model with the deviance of a model excluding the predictor in question (i.e. the reduced model). Partial r2.L was calculated as specified by Quinn and Keough (2002):

$$\text{partial } r2.L = (\text{reduced model residual deviance} - \text{best model residual deviance})\ /\ \text{reduced model residual deviance}$$

Maximum impact was calculated with the following formula:

max impact = (the predicted value of the response variable at the *maximum* value of the predictor in question, while holding all other predictors at their mean values)
– (the predicted value of the response variable at the *minimum* value of the predictor in question, while holding all other predictors at their mean values).

All maximum impact values are reported on the raw response variable scale, even if transformations were necessary for model fitting.

Partial regression plots (i.e. partial residual plots), which are described in Quinn and Keough (2002) are also provided for several variables. Partial regression plots visually display the effect of the predictor of interest, *after accounting for the effects of all explanatory variables*. The y-axis values are the residuals from a model fitting the response variable against all predictors except the predictor of interest, and the x-axis values are the residuals from a model fitting the predictor of interest against all other predictors. After calculation of these two sets of residuals, a new model was fit and a line representing the predicted values was added to the partial regression plot. We display all partial regression plots on the raw data scale of the response variable, and all predicted values (the fitted line) are drawn with the appropriate shape for the link function. For instance, if the response variable was log-transformed for analysis, the residuals were back-transformed and the fitted line assumed an exponential shape. As another example, when the error distribution was binomial or quasibinomial, the fitted line assumed a characteristic logistic "S" form (provided that the parameter estimates were sufficiently strong; but note that in most cases, the relationships were fairly weak, and at best a hint of the "S" shape can be seen). These partial regression plots may be unfamiliar to many readers, and thus we provide the following simplified explanation: our partial regression plots visually display the effects of the predictor of interest, after accounting for the effects of all other model predictors, *on the raw scale of the response variable*. Our partial regression plots are intended to portray the shape of the relationship and the residual cloud, while the accompanying tables (which include standard multiple regression output) should be used for quantitative interpretation. The statistical software R, version 2.8.0, by The R Foundation for Statistical Computing, was used to conduct all analyses and create all figures.

2.5. Note about the use of dead tanoak abundance as the sole metric of disease severity

In all analyses, we utilize dead tanoak abundance as the primary metric of disease severity. We also considered using other metrics (which we termed "mortality indices") that incorporated the magnitude of tanoak mortality, as well as the approximate length of time that the plot has been undergoing disease-induced changes (e.g. reduced canopy cover). In brief, these approaches weighted the basal area of each dead tanoak stem with its deterioration class. For instance, a fallen tanoak with a given basal area would contribute more to the plot-level mortality index than a standing dead tanoak with equivalent basal area. However, we discovered a very tight relationship between our mortality indices and dead tanoak abundance, and as such, in the interest of parsimony, we have opted to utilize dead tanoak abundance as our primary measure of disease severity. This unexpectedly tight correlation is interesting in its own right, suggesting predictable patterns of disease progression, but it is not a subject that we explore in this paper.

3. RESULTS

3.1. Abundance of tanoak in redwood forests: mature trees and regeneration

Within our randomly located plots, mean tanoak basal area and stem counts were consistently higher than all other hardwoods combined, as well as all conifers excluding redwood (although the difference between tanoak basal area and all non-redwood conifer basal area in Santa Cruz was very small). And mean tanoak stem counts were actually higher than all non-redwood, non-tanoak species (conifers and hardwoods combined) in all counties. In terms of both basal area and stem counts, tanoak is a considerable component of redwood forests within our study area.

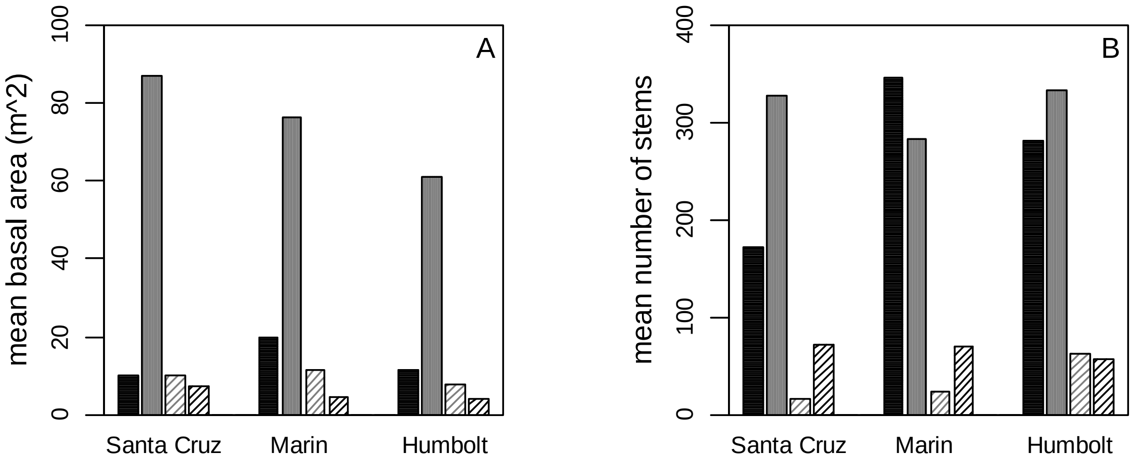

Figure 3: Abundance, by county. A: mean basal area (square meters) per hectare; B: mean number of stems per hectare. Solid black bars = tanoak; solid grey bars = redwood; grey hatching = conifers excluding redwood; black hatching = hardwoods excluding tanoak.

Tanoak regeneration, whether measured continuously (as individuals or clumps), or as the proportion of plots in which it was present, is a major component of the regeneration stratum. Within our randomly located plots, tanoak individuals and clumps outnumbered redwood, all other hardwoods combined, and all conifers excluding redwood, in all counties. In addition, note that tanoak regeneration was present in 100% of these plots. This highly shade-tolerant species thus exists as a ubiquitous and abundant constituent of advance regeneration in the redwood forests throughout our study areas. Note that median values are displayed, as opposed to mean, because a few plots with extreme values had very large and misleading effects on mean values. For instance, one plot contained over 1,100 regenerating redwood individuals (seedlings/sprouts and saplings combined), while the plot with the next highest redwood regeneration had only 157 individuals.

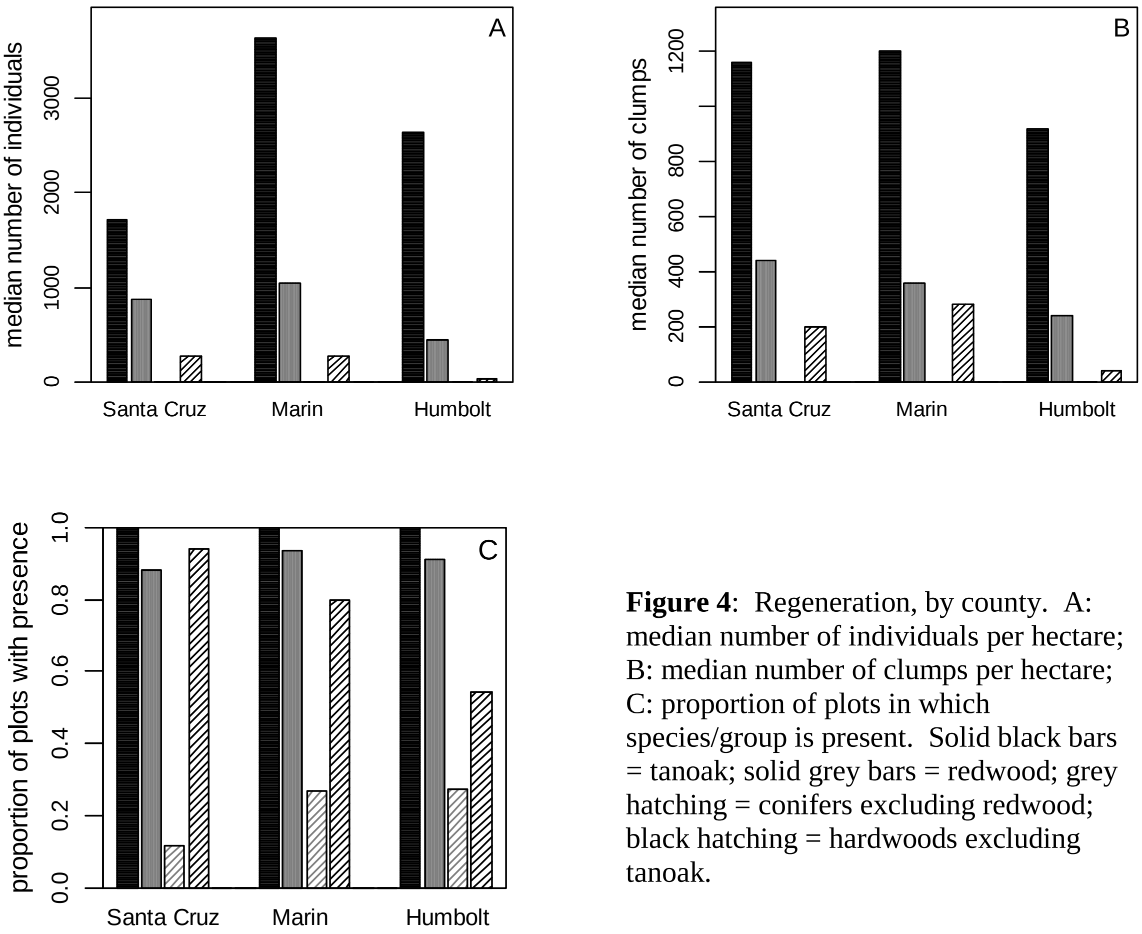

Figure 4: Regeneration, by county. A: median number of individuals per hectare; B: median number of clumps per hectare; C: proportion of plots in which species/group is present. Solid black bars = tanoak; solid grey bars = redwood; grey hatching = conifers excluding redwood; black hatching = hardwoods excluding tanoak.

3.2. Verification of Phytophthora ramorum presence

Our attempt to verify the presence of *P. ramorum* in diseased plots was unsuccessful; results indicate very poor correspondence between pathogen verification and high levels of tanoak mortality. However, several factors suggest that these results are unreliable, and thus we proceed with the assumption that most tanoak mortality has resulted from SOD. This assumption is examined in more detail in the discussion section.

3.3. Impacts of tanoak mortality and correlates of tanoak abundance

After accounting for the effects of redwood abundance, sampling site, slope, slope position, and northness, several response variables displayed significant relationships with tanoak mortality and tanoak abundance (table 2).

	Dead Tanoak	Total Tanoak	Redwood
Canopy Openness (100 - Canopy Cover)	+ + +	0	0
Cover: Coarse Woody Debris	+ + +	0	0
Cover: Herb	0	-	0
Cover: Shrub	0	0	- -
Richness: Herb	+ +	- - -	0
Richness: Shrub	0	- -	(-)
Richness: Mature Trees	0	-	- - -
Richness: Juvenile Trees	0	0	0
% of total stems sprouting	+ +	0	0
% of tanoak stems sprouting	+ + +	0	0
% of redwood stems sprouting	(+)	-	0
% of hardwood-stems-excl-tanoak sprt	(+)	0	0
Regen: total	0	(+)	- -
Regen: tanoak	(-)	+ + +	- - -
Regen: redwood	0	0	+
Regen: hardwood-excl-tanoak	0	(-)	- -
Regen: conifer-excl-redwood	0	(-)	0
Regen: all-excl-tanoak-and-redwood	0	-	- -

Table 2: Summary of results (after accounting for the effects of sampling site, slope, slope position, and northness). Each row represents a separate multiple GLM, in which the response variable has been fitted against the subset of predictors with the lowest AIC value. 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, or that the predictor was omitted from the best model. Redwood effects are shown for comparison with total tanoak effects

3.3.1. Cover classes: canopy, coarse woody debris, herb, and shrub

Our data indicate that SOD-induced tanoak mortality strongly reduced canopy cover ($p < .0001$) and strongly increased coarse woody debris ($p < .0001$). After accounting for other model predictors, tanoak mortality decreased canopy cover (which was analyzed as “canopy openness” [100 – canopy cover] in order to satisfy statistical assumptions) by an estimated 29% (note that percent cover is the unit of all cover class variables) across the full range of this predictor (Table 3, Figure 5). Average canopy cover in plots with little or no tanoak mortality was near 100%, indicating that tanoak mortality (which explains 53% of the residual variation in canopy cover) created significant canopy gaps and seriously altered the light environment of affected plots. And not surprisingly, as these dead tanoak trees fell and opened up the canopy, they also increased levels of coarse woody debris. After accounting for other model predictors, tanoak morality increased coarse woody debris cover by an estimated 36% across the full range of this predictor, explaining 32% of residual variation (Table 4, Figure 6). As these numbers indicate, areas with high mortality tend to be choked with fallen boles and branches, often rendering movement difficult. Herb cover was not impacted by tanoak mortality but was negatively associated with total tanoak abundance ($p = .0109$, partial r2.L = .10, max impact = -9.54). Shrub cover was not related to tanoak mortality or total tanoak abundance but was negatively correlated with redwood abundance (p = .0035, partial r2.L = .14, max impact = -7.95).

With regard to percent cover of individual shrub and herb taxa, very few exhibited significant relationships with tanoak mortality or total tanoak abundance. Of the 16 shrub species in our dataset, none were related to tanoak mortality, and only two were related to tanoak abundance. Poison-oak (*Taxodium diversilobium*; p < .0001, partial r2.L = .28, max impact = -1.39) and California hazelnut (*Corylus cornuta*; p = .0301, partial r2.L = 24, max impact = -3.00) cover were both negatively associated with tanoak abundance, but note that the maximum impact estimates were very low. Of the 45 herb taxa in our dataset, only five exhibited significant relationships, and the majority of these maximum impact estimates were also very low (often below one percent cover). Carex (*Carex* sp.) cover was positively associated with tanoak mortality (p = .0043, partial r2.L = .17, max impact = 4.54) and negatively associated with tanoak abundance (p = .0006, partial r2.L = .30, max impact = -1.00). California harebell (*Campanula prenanthoides*) cover was positively associated with tanoak mortality (p = .0482, partial r2.L = .08, max impact = 0.31) and weakly negatively associated with tanoak abundance (p = .0914, partial r2.L = .06, max impact = -0.08). The following taxa were unrelated to tanoak mortality, but all three were negatively associated with tanoak abundance: bedstraw (*Galium* sp.; p = .0032, partial r2.L = .19, max impact = -0.54), California wood fern (*Dryopteris arguta*; p = .0359, partial r2.L = .10, max impact = -0.35), and a grass species or genus that we could not positively identify (p = .0437, partial r2.L = .35, max impact = -1.98)

3.3.2. Species Richness

Herb richness was strongly related to both tanoak mortality (p = .0051) and total tanoak abundance (p = .0007). After accounting for other model predictors, tanoak mortality was estimated to increase herb richness by 7.09 species across the full range of this predictor, explaining 13% of residual variation (Table 5, Figure 7a). We interpret this result as evidence that herbaceous species requiring higher light levels (i.e. early seral species) are recruiting in mortality gaps. In contrast, total tanoak abundance was estimated to decrease herb richness by 6.54 species across its full range, explaining 18% of residual variation (Table 5, Figure 7b), and perhaps indicating that tanoak has an inhibitory effect on some herb species. In conjunction with the previous result, this finding suggests that if tree species other than tanoak become established on these infested sites, the current increase in herb species richness may persist even after eventual closure of the canopy, despite the fact that species identities are likely to change.

Shrub richness was strongly related to total tanoak abundance (p = .0016) and weakly related to redwood abundance (p = .0508). After accounting for other model predictors, total tanoak abundance decreased shrub richness by an estimated 2.14 species across the full range of this predictor, explaining 15% of residual variation (Table 6, Figure 8a). Redwood abundance also may have decreased shrub richness, but any such effects appear much weaker than the effects of tanoak. After accounting for other model predictors, redwood abundance was estimated to decrease shrub richness by 0.62 species across the full range of this predictor, explaining only 6% of residual variation (Table 6, Figure 8b). Richness of mature trees was unrelated to tanoak mortality but was negatively correlated with redwood abundance (p < .0001, partial r2.L = .26, max impact = -2.11) and, to a lesser extent, with total tanoak abundance (p = .0342, partial r2.L = .07, max impact = -0.83). Juvenile tree richness was not related to any of our variables of interest.

Table 3. Results from the “best model” for log(canopy openness).

y = log(Canopy Openness)	family: Gaussian		signif = <.0001		r2.L = 0.5250	
x	est	SE	signif		partial r2.L	max imp
dead tanoak abund.	0.032	0.004	<.0001	***	0.53	29.46
slope (deg)	-0.018	0.010	0.0607	'	0.06	-2.47

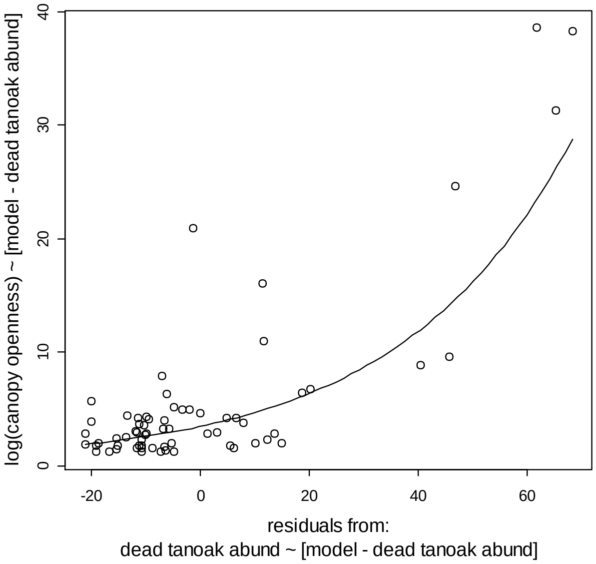

Figure 5. Partial regression plot for log(canopy openness) vs. dead tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 4. Results from the "best model" for log(coarse woody debris cover).

y = log(Cover: CWD)		family: Gaussian	signif = <.0001			r2.L = 0.3258
x	est	SE	signif		partial r2.L	max imp
Humboldt	0.572	0.253	0.027	*	0.08	4.37
dead tanoak abund.	0.028	0.005	<.0001	***	0.32	36.27

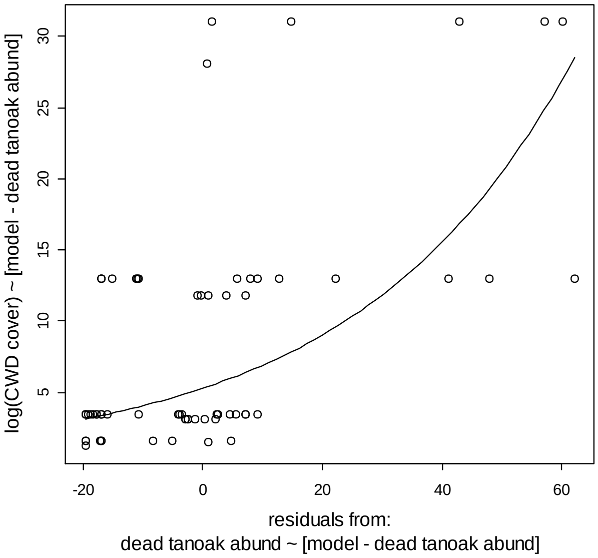

Figure 6. Partial regression plot for log(coarse woody debris cover) vs. dead tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 5. Results from the “best model” for herb richness.

y = Richness: Herb	family: Gaussian		signif = 0.0010			r2.L = 0.2575
x	est	SE	signif		partial r2.L	max imp
total tanoak abund.	-0.077	0.022	0.0007	***	0.18	-6.54
dead tanoak abund.	0.086	0.030	0.0051	**	0.13	7.09
northness	1.502	0.841	0.0794	'	0.05	3.01
Santa Cruz	1.481	1.044	0.1611		0.03	1.49
slope (deg)	0.093	0.061	0.1317		0.04	2.99

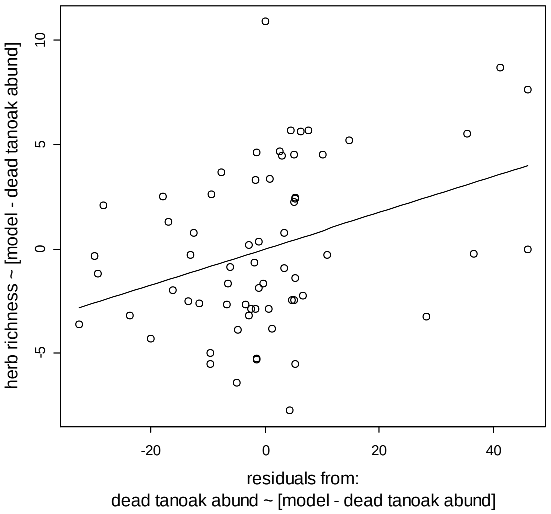

Figure 7a. Partial regression plot for herb richness vs. dead tanoak abund.

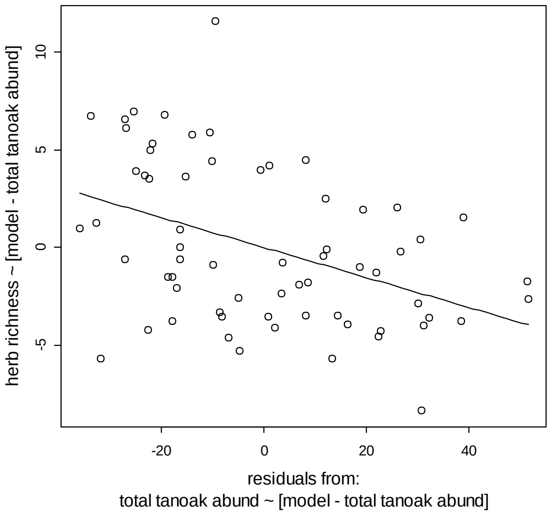

Figure 7b. Partial regression plot for herb richness vs. total tanoak abund.

Table 6. Results from the “best model” for shrub richness.

y = Richness: Shrub	family: Gaussian		signif = 0.0020			r2.L = 0.1949
x	est	SE	signif		partial r2.L	max imp
total tanoak abund.	-0.025	0.008	0.0016	**	0.15	-2.14
Santa Cruz	0.625	0.383	0.1082		0.04	0.62
redwood abund.	-0.023	0.012	0.0508	'	0.06	-1.52

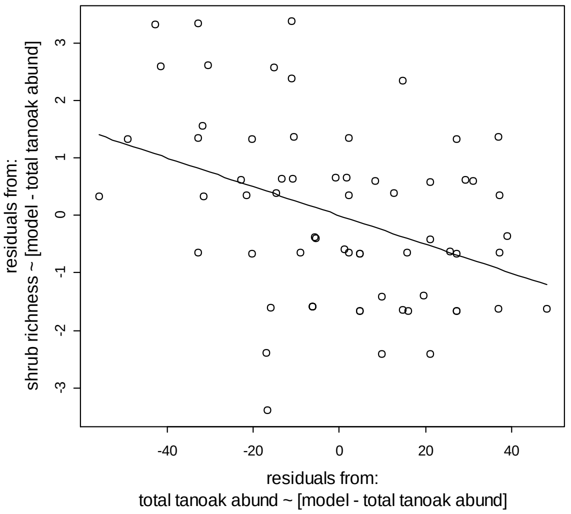

Figure 8a. Partial regression plot for shrub richness vs. total tanoak abund.

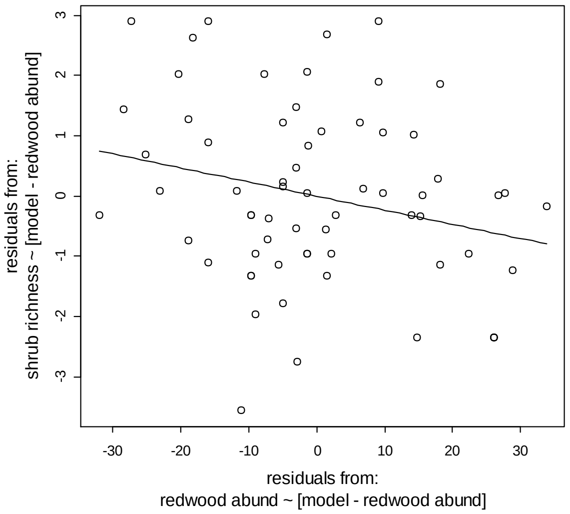

Figure 8b. Partial regression plot for shrub richness vs. total redwood abund.

3.3.3. Regeneration

At the time of our field measurements, tanoak mortality had not stimulated a strong regenerative response. There is no evidence that tanoak mortality had increased the number of regenerating clumps (or individuals; results not shown) for any species or functional group. Of all the regeneration variables we analyzed, the only definitive response was an increase in the percent of total stems exhibiting basal sprouts (p = .0037, partial r2.L = .13, max impact = .29) and the percent of tanoak stems exhibiting basal sprouts (p < .0001, partial r2.L = .32, max impact = .35; see Table 7 and Figure 9), with the former trend apparently driven primarily by the latter. Some of the sprouting response of tanoak may have been due to increased light levels, but it is likely that SOD was directly responsible for the increased sprouting observed in diseased plots. Individual stem analyses (data not presented) revealed that top-killed tanoaks exhibited significantly higher percentages of basal sprouting than tanoaks with living canopies, and that symptomatic trees exhibited significantly higher percentages of basal sprouting than asymptomatic trees; these findings are in agreement with unpublished findings of other SOD researchers (e.g. David Rizzo; personal communication). There is also weak evidence that tanoak mortality increased the percent of redwood stems exhibiting basal sprouts (p = .0625, partial r2.L = .05, max impact = .32; see Table 8 and Figure 10a) and the percent of hardwoods-excl-tanoak exhibiting basal sprouts (p = .0536, partial r2.L = .13, max impact = .83; see Table 9 and Figure 11). But it is worth emphasizing that, even if all existing hardwood-excl-tanoak stems were to sprout in response to tanoak mortality, they would not be able to occupy all of tanoak's current growing space; across all our sampled plots, tanoak had a mean of 304 mature stems/ha, while the mean for hardwoods-excl-tanoak was only 54 mature stem/ha.

If all tanoak sprouts in diseased areas eventually die (which is a definite possibility given tanoak's apparent lack of resistance and susceptibility at all ages), and non-tanoak regeneration does not increase, such areas will lack a strong regenerating cohort. This is not to say that non-tanoak regeneration was completely absent in diseased areas, but rather that regeneration levels in these mortality gaps were no higher than in closed canopy forest with healthy tanoak populations. However, regeneration levels of species other than tanoak were very low throughout our entire study area; across all our sampled plots, the median values for regenerating individuals were as follows (expressed in individuals/ha): hardwoods-excl-tanoak = 160, conifers-excl-redwood = 0, all-excl-tanoak-and-redwood = 200, and redwood = 880. In stark contrast, the median value of regenerating tanoak was 3,120 individuals/ha. While the redwood figure is more respectable than the other functional groups, our field observations indicated that the vast majority of redwood regeneration was in the form of basal sprouts, with seed origin regeneration appearing to be much lower (and entirely absent in many or even most plots). And given that redwood and tanoak are very clumpy (Waring & O'Hara 2008), redwood did not appear poised to claim available growing space. As such, a strong and as-of-yet-non-existent regenerative response to tanoak mortality will be necessary for any other tree species to become established on former tanoak sites.

Several regeneration variables were related to total tanoak abundance and/or redwood abundance. Total tanoak abundance was negatively related to the percent of redwood stems sprouting (p = .0180, partial r2.L = .09, max impact = -.28; see Table 8 and Figure 10b) as well as regeneration of all-species-excluding-tanoak-and-redwood (p = .0458, partial r2.L = .09, max impact = -14.19), weakly negatively associated with regeneration of hardwoods-excluding-tanoak (p = .0605, partial r2.L = .08, max impact = -11.60) as well as conifers-excluding-redwood (p = .0871, partial r2.L = .28, max impact = -0.63), and weakly positively associated with total regeneration (p = .0544, partial r2.L = .06, max impact = 37.40). In addition, total tanoak abundance was strongly related to tanoak regeneration (p < .0001); after accounting for other model predictors, total tanoak abundance increased tanoak regeneration by an estimated 91 clumps across the full range of this predictor, explaining 43% of residual variation (Table 10, Figure 12a).

Redwood abundance was negatively related to total regeneration (p = .0086, partial r2.L = .11, max impact = -52.37), tanoak regeneration (p = .0010, partial r2.L = .17, max impact = -28.79; see Table _ and Figure 12b), regeneration of hardwoods-excluding-tanoak (p = .0060, partial r2.L = .17, max impact = -11.72), and regeneration of all-species-excluding-tanoak-and-redwood (p = .0038, partial r2.L = .18, max impact = -14.31). Redwood abundance was positively related to redwood regeneration (p = .0110); after accounting for other model predictors, redwood abundance increased redwood regeneration by an estimated 15 clumps across the full range of this predictor, but explains only 10% of residual variation (Table 11, Figure 13).

Table 7. Results from the “best model” for percent of tanoak stems sprouting.

y = % of tanoak stems sprouting		family: quasibinomial			signif = <.0001	r2.L = 0.4188
x	est	SE	signif		partial r2.L	max imp
dead tanoak abund.	0.023	0.005	<.0001	***	0.32	0.35
northness	0.453	0.242	0.0669	'	0.06	0.22
redwood abund.	-0.017	0.011	0.1465		0.04	-0.26
slope (deg)	-0.033	0.016	0.0502	'	0.07	-0.24
slope position	-0.531	0.138	0.0004	***	0.23	-0.46

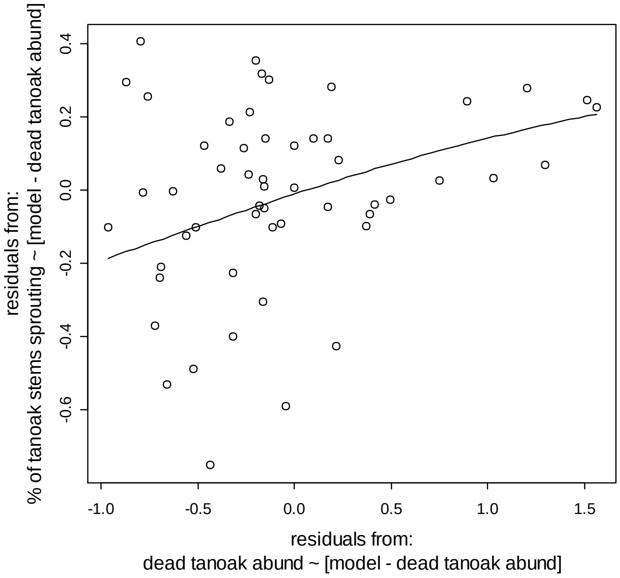

Figure 9. Partial regression plot for percent of tanoak stems sprouting vs. dead tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 8. Results from the “best model” for percent of redwood stems sprouting.

y = % of redwood stems sprouting		family: quasibinomial			signif = <.0001	r2.L = 0.4144
x	est	SE	signif		partial r2.L	max imp
total tanoak abund.	-0.014	0.006	0.0180	*	0.09	-0.28
dead tanoak abund.	0.016	0.008	0.0625	'	0.05	0.32
Santa Cruz	0.694	0.239	0.0053	**	0.12	0.17
slope (deg)	0.061	0.014	0.0001	***	0.24	0.44
slope position	0.177	0.123	0.1562		0.03	0.17

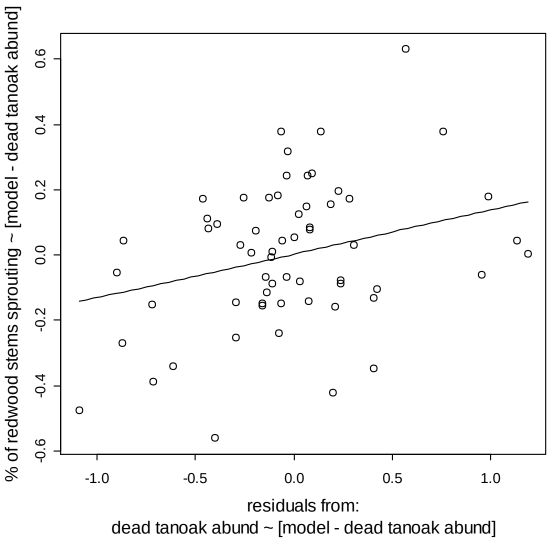

Figure 10a. Partial regression plot for percent of redwood stems sprouting vs. dead tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

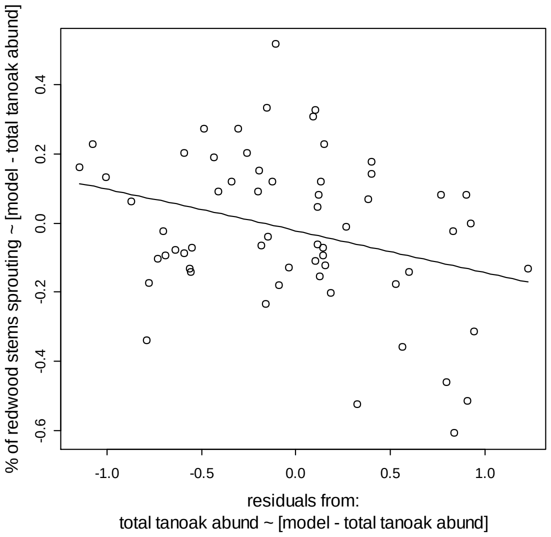

Figure 10b. Partial regression plot for percent of redwood stems sprouting vs. total tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 9. Results from the "best model" for percent of hardwood-stems-excluding-tanoak sprouting.

y = % of hardwood-stems-excl-tanoak sprouting		family: quasibinomial			signif = 0.1474	r2.L = 0.1976
x	est	SE	signif		partial r2.L	max imp
total tanoak abund.	-0.021	0.017	0.2267		0.05	-0.40
dead tanoak abund.	0.077	0.038	0.0536	'	0.13	0.83
northness	-0.527	0.408	0.2066		0.05	-0.25
slope (deg)	-0.052	0.039	0.1870		0.05	-0.39
slope position	0.477	0.397	0.2404		0.04	0.43

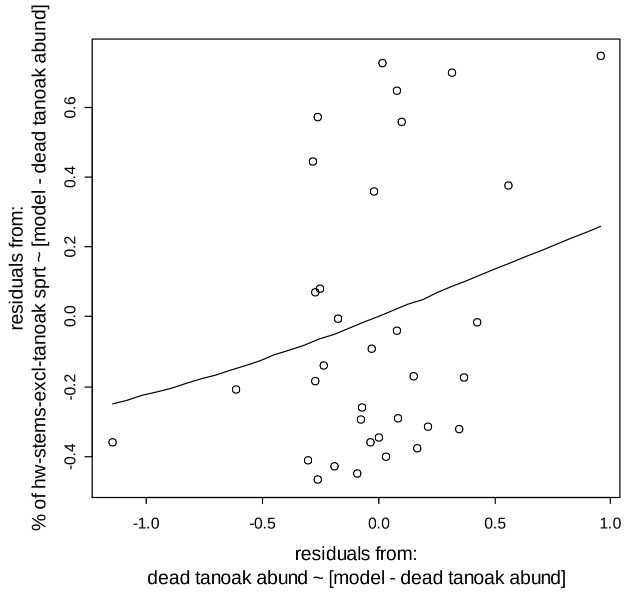

Figure 11. Partial regression plot for percent of hardwood-stems-excl-tanoak sprouting vs. dead tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 10. Results from the “best model” for log(tanoak regen clumps).

y = log(tanoak regen clumps)		family: Gaussian		signif = <.0001		r2.L = 0.6777
x	est	SE	signif		partial r2.L	max imp
total tanoak abund.	0.030	0.004	<.0001	***	0.43	90.72
dead tanok abund.	-0.009	0.005	0.0815	'	0.05	-15.97
Santa Cruz	0.257	0.175	0.1477		0.04	7.07
redwood abund.	-0.019	0.005	0.0010	***	0.17	-28.79
slope position	-0.226	0.093	0.0179	*	0.09	-24.24

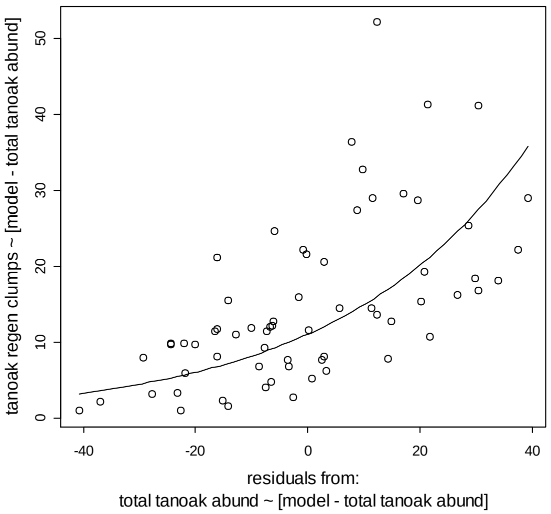

Figure 12a. Partial regression plot for tanoak regen clumps vs. total tanoak abund. Residuals and predicted values are displayed on the original scale of the response variable.

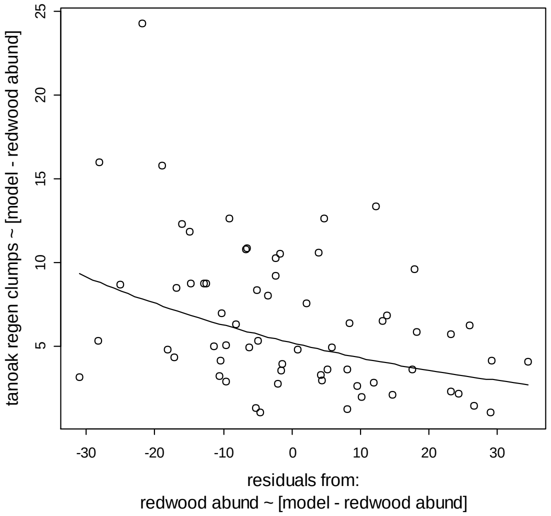

Figure 12b. Partial regression plot for tanoak regen clumps vs. redwood abund. Residuals and predicted values are displayed on the original scale of the response variable.

Table 11. Results from the “best model” for log(redwood regen clumps).

y = log(redwood regen clumps)		family: Gaussian			signif: 0.0086	r2.L = 0.1606
x	est	SE	signif		partial r2.L	max imp
redwood abund.	0.021	0.008	0.0110	*	0.10	15.28
slope (deg)	0.029	0.017	0.1056		0.04	8.02
slope position	0.294	0.149	0.0524	'	0.06	11.17

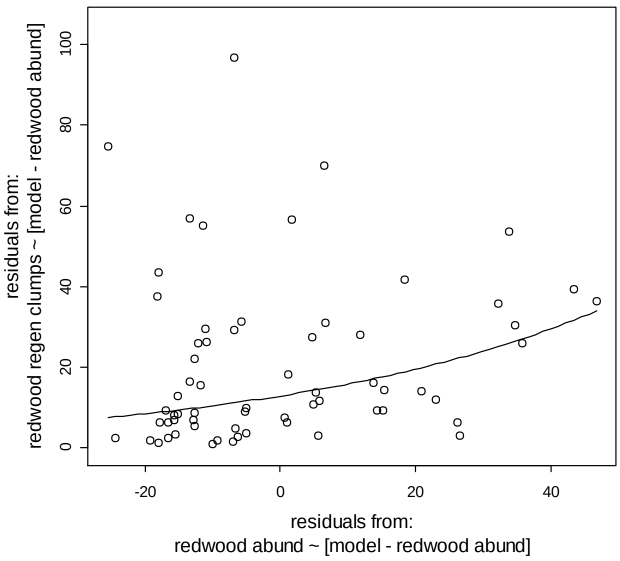

Figure 13. Partial regression plot for redwood regen clumps vs. redwood abund. Residuals and predicted values are displayed on the original scale of the response variable.

3.4. Examination of potential tanoak replacement species

Given that there were no significant relationships between disease severity (i.e. dead tanoak abund) and regenerating individuals or clumps of broad functional groups, here we examine regeneration patterns of particular species, and also take a closer look at some of the factors that may help to predict which species will eventually replace tanoak. First, since redwood and tanoak both showed evidence of self-replacement (i.e. positive relationships between regeneration and abundance of mature con-specific trees), we executed GLMs with the response variable defined as the number of regeneration clumps of each individual species (or functional group), and the full model predictors consisting of the same eight predictors utilized in the primary analyses above, plus an additional predictor: the mature tree abundance of the species or functional group corresponding to the response variable. Note that this additional predictor would be redundant in the case of redwood and tanoak, because redwood and tanoak abundances are already included within the original set of eight predictors. Second, we examined the impacts to regeneration of abiotic factors and sampling site, along with dead tanoak, total tanoak, and redwood abundance. In the interest of brevity, a summary table is provided (table 12), but full reports of model parameters are not included.

There is no evidence that any species or functional group was exhibiting a regenerative response to tanoak mortality. Aside from tanoak and redwood, California bay and interior live oak are the only species that showed any evidence of self-replacement within our study area (and both were only borderline significant). In terms of functional groups, there was a strong self-replacement trend for hardwoods-excl-tanoak (perhaps driven by the combined effects of interior live oak and California bay), but no evidence of self-replacement for conifers-excl-redwood. Regeneration of some species and functional groups were also related to abiotic factors and/or sampling site (table 12); a more detailed discussion is provided in Appendix B.

In an attempt to predict potential long-term changes to SOD-infested redwood forests, and to look for discrepancies between niches of established trees and regeneration, we also examined the effects of abiotic factors and sampling site on the abundances of mature trees. In these GLMs, the abundances of mature tree species or functional groups were fitted against a full model consisting of northness, slope position, slope (deg), Santa Cruz, and Humboldt. Results of these models (which are summarized in table 13) demonstrate that several species and functional groups were related to abiotic factors and/or sampling site; a more detailed discussion is provided in Appendix B. It is also worth pointing out that several discrepancies existed between regeneration and established trees, with regard to associated abiotic variables (compare tables 12 and 13). For instance, tanoak regeneration was associated with lower slope positions, but mature tanoak trees were strongly associated with upper slope positions, perhaps reflecting the tendency of tanoak's large heavy acorns to roll downhill. A more thorough discussion of these discrepancies is provided in the supplemental material.

Table 12. Summary of regeneration relationships. Each row represents a separate multiple GLM, in which the number of regenerating clumps (of each species or functional group) has been fitted against the predictors. 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, or that the predictor was omitted from the best model. The following species were not analyzed due to an insufficient number of occurrences: pacific madrone, canyon live oak, Scouler's willow, hollyleaf cherry, California nutmeg, and grand fir.

[1] or mature members of functional group

REGEN-ERATION	north-ness	slope position	slope (deg)	dead tanoak abund	total tanoak abund	redwood abund	Santa Cruz	Humboldt	mature con-specific[1]
bigleaf maple	0	0	0	0	0	0	0	0	0
interior live oak	0	0	(-)	0	(-)	0	0	0	(+)
California bay	0	0	+ +	0	0	(+)	-	0	(+)
Douglas-fir	0	0	0	0	0	0	0	0	0
tanoak	0	-	0	(-)	+ + +	- - -	0	0	redundant
redwood	0	(+)	0	0	0	+	0	0	redundant
hw-excl-tanoak	- - -	- - -	0	0	0	0	- -	- - -	+ + +
con-excl-redwood	-	0	0	0	(-)	0	-	0	0

Table 13. Summary of mature tree abundance relationships. Each row represents a separate multiple GLM, in which the abundance of each species or functional group has been fitted against the predictors. 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, or that the predictor was omitted from the best model. Abundances of all individual species were analyzed as mature tree cover classes, while abundances of the two functional groups were analyzed as BA (because cover classes were not recorded for functional groups). The following species were not analyzed due to an insufficient number of occurrences: canyon live oak, Scouler's willow, hollyleaf cherry, California nutmeg, and grand fir.

MATURE TREES	northness	slope position	slope (deg)	Santa Cruz	Humboldt
bigleaf maple	0	(-)	0	0	0
interior live oak	- - -	+ + +	- - -	0	0
California bay	(+)	0	+ +	(-)	0
Pacific madrone	0	0	0	0	0
Douglas-fir	0	0	0	0	(+)
tanoak	0	+ + +	0	0	0
redwood	0	- -	0	0	0
hw-excl-tanoak	-	0	0	0	0
con-excl-redwood	-	0	0	0	0

4. DISCUSSION

Several important variables appear to be related to SOD-induced tanoak mortality and/or total tanoak abundance. Because the current distribution of SOD in redwood forests is believed to be a result of stochastic factors and introduction events, as opposed to underlying biotic or abiotic conditions, we believe it is generally reasonable to suggest that differences *correlated* with tanoak mortality are in fact *caused* by tanoak mortality. And with regard to our specific data, all such findings seem logical and comprehensible within a broader theoretical framework. In other words, it is entirely feasible that high levels of mortality could impact all of the variables for which we detected significant relationships: canopy cover, coarse woody debris, herbaceous species richness, percent cover of two herb taxa, and the percent of established stems with basal sprouts. In summary, as diseased tanoaks die and collapse, canopy cover decreases, fallen boles and branches increase coarse woody debris, and higher light levels stimulate the recruitment and/or expansion of herbaceous species, as well as the production of basal sprouts in some tree species. What is perhaps more interesting than these somewhat predictable findings, is the current lack of a strong regenerative response, a topic which we discuss below in great detail.

Correlations between total tanoak abundance and other variables are quite possibly controlled by factors that were not measured as part of this study. As such, we do not definitively assert a causative role for tanoak abundance, except with regard to tanoak regeneration, which should obviously be related to the abundance of mature conspecifics. Variables that were definitively related to total tanoak abundance were herbaceous cover, herbaceous species richness, shrub species richness, percent cover of several shrub and herb taxa, mature tree species richness, the percent of established redwood stems with basal sprouts, and the pooled regeneration of all species other than redwood and tanoak; all of these associations were negative. It is entirely conceivable that all of these relationships are caused by the effects of tanoak, but we cannot reasonably reject the possibility of confounding factors. For instance, tanoak may have inhibitory effects (e.g. allelopathic exudates) on herbaceous cover and richness of herb and shrub taxa, but it is also feasible that sites on which tanoak trees thrive are characterized by other factors (e.g. soil properties) that are inhospitable to many shrub and herb species. Very little is known about possible mechanisms of tanoak inhibition, and thus more research is clearly needed to elucidate the patterns we have discovered.

We were unable to verify the presence of *P. ramorum* in many plots with high mortality levels, but there are compelling reasons to suspect that these results represent false negatives: **a.** our sampling period followed two very dry years, and dry conditions are known to decrease the likelihood of detection (Rizzo et al. 2005, Moritz et al. 2008); **b.** two plots which tested negative were confirmed positive in a previous study that was conducted in a wetter year (Spencer 2004); **c.** several plots that tested negative exhibited very severe levels of tanoak mortality and displayed other characteristic symptoms of SOD as well (in all of these cases, infestation has been confirmed in the general area); and **d.** no other agent is known to cause such severe and widespread mortality of tanoak (Swiecki & Bernhardt 2006). In fact, because the four most severely impacted plots all returned negative results, we suggest that greater light penetration (and presumably lower humidity levels) may actually make it more difficult to detect the pathogen in severely diseased areas, at least in dry years. These results also suggest that the dry sampling year may have produced false negatives in plots with lower levels of mortality. As such, we consider these results unreliable and assert that most tanoak mortality has probably resulted from SOD. It is also worth noting that we are unable to prove every dead tanoak we encountered was killed by SOD; in fact it is highly likely that some individuals died of other causes (e.g. competition, windthrow). As such, impacts that we loosely attribute to SOD are actually, in a strict sense, impacts of tanoak mortality. However, since severe tanoak mortality is not associated with any disturbance other than SOD, and all of the effects that we have detected are most apparent at very high levels of mortality, we believe it is reasonable to assign a causative role to SOD.

An important consideration that we have not directly examined is the amount of time that has passed since the onset of SOD-induced tanoak mortality. In other words, how long has the understory been exposed to a higher light environment? Given that all our research was conducted in a single field season, our data are not instructive. However, in three of our supplemental plots, which were placed in areas sampled as part of previous epidemiologically-focused SOD research (Spencer 2004), we can confirm that SOD-induced tanoak mortality was underway by 2001. Only one of these three plots is among the ten most severely impacted plots in our dataset, and thus many of our plots have probably been experiencing SOD-induced tanoak mortality, and associated increases in light levels, for well over seven years.

Despite several years of canopy deterioration and increased light penetration, there is no definitive evidence of a regenerative response to SOD-induced tanoak mortality (aside from tanoak basal sprouts, which are unlikely to survive). However, we did detect two relationships with borderline significance levels, both of which represent the percent of established trees with basal sprouts, as opposed to absolute regeneration counts. Dead tanoak abundance and the percent of redwood stems sprouting were positively related ($p = .0625$), with a predicted 0.32 increase in the probability of basal sprouting across the full range of dead tanoak abundance. This result is in rough agreement with the work of Waring and O'Hara (2008), which compared a single pair of healthy and diseased *sites*, each composed of several plots. Waring and O'Hara detected a greater percent of redwood stems with basal sprouts in the diseased site, but the statistical significance level ($p = .038$) was not much less than what we detected, and the difference in probabilities between sites was only 0.11. Taken together, these findings suggest that tanoak mortality is causing an increase in the percent of redwood stems with basal sprouts, but that this response is weak and inconsistent. Furthermore, the long-term potential for these sprouts to occupy growing space vacated by tanoak is rather low because of the generally clumped nature of redwood and tanoak patches, the proximity of redwood basal sprouts to established redwood trees, and the strong vertical growth habit of redwood. We also detected a weak positive relationship between dead tanoak abundance and the percent of hardwoods-excl-tanoak exhibiting basal sprouts ($p = .0536$), but due to the general paucity of established hardwood-excluding-tanoak stems, even a one hundred percent basal sprouting response would do little to occupy tanoak's former growing space. Due to the clumped nature of redwood and the general deficiency of other tree species, it is clear that full re-occupation of diseased sites will require seedling recruitment. However, current regeneration is not sufficient to replace the tanoak that is being lost and, in terms of regeneration counts (as opposed to the percent of established stems with basal sprouts), we have found no evidence of a regenerative response to tanoak mortality.

If SOD eventually removes all tanoak from our study sites, what species will replace it? And how will these species vary among areas with different abiotic conditions? Of course, it is possible – and even probable – that some small patches vacated by tanoak will be occupied by lateral branch extension and increased growth rates of existing neighbors. When occurring in second-growth forests, this scenario represents an accelerated transition towards the structural –

but not necessarily the compositional – characteristics of old-growth redwood forests (Keyes 2005, Waring 2005). It is also feasible that some areas will convert, at least temporarily, to shrub dominated communities; this should be most likely to occur in large patches with minimal tree regeneration. But for the time being, let us put these scenarios aside and consider what tree species are most likely to regenerate in and occupy SOD-induced canopy gaps (via response of an existing but previously suppressed regeneration stratum, initiation of new basal and/or root sprouts, or post-mortality seedling recruitment). As mentioned above, current regeneration does not appear sufficient to fully occupy the growing space that is being vacated by tanoak, and thus one or more of the following conditions are likely to be true: **a.** insufficient time has passed for a full regenerative response (e.g. stochastic factors in recent years have not been conducive to seed production, seed dispersal, and/or seedling establishment; fallen tanoak leaves have created a thick but transient litter layer; redwood basal sprouts have not yet physiologically responded to increased light levels), **b.** permanent (i.e. long-term) barriers to dispersal have prevented, and will continue to prevent, seeds from reaching SOD-induced canopy gaps, and/or **c.** unidentified constraints exist that will indefinitely inhibit recruitment of other tree species (e.g. soil chemistry, soil texture).

If the current paucity of regeneration is simply a result of an insufficient passage of time, what species do are data suggest might regenerate, and which might survive to maturity? Let us temporarily assume that dispersal limitation is not an issue, and that no environmental constraints will inhibit the establishment of any species in our regional species pool; in other words, assume that uncommon tree species of redwood forests are uncommon only as a result of the pre-SOD competitive superiority of tanoak, and that tanoak has equally exclusionary effects on all other tree species. Under this scenario, we would expect species and functional groups to become established on the same types of sites on which they are currently present. Interior live oak would be most likely to replace tanoak on relatively flat, south-facing, upper slopes (table 13); however, southern Humboldt county is the current northern extent of this species (Pavlik et al. 2006), and thus it should not be expected to replace tanoak in northern redwood forests. California bay would be most likely to occupy steeper sites, with a weak preference for northerly aspects, and slightly reduced abundances in Santa Cruz county as compared to our other two field sites (table 13); because this species has a shade tolerance similar to tanoak and an ability to thrive in a very wide range of moisture conditions (Burns & Honkala 1990, Hunter et al. 1999), and is relatively unharmed by *P. ramorum* (while producing copious amounts of spores on its foliage and thereby contributing to the severity of SOD-induced tanoak mortality; Rizzo et al. 2005), California bay may be well-positioned to move into many areas previously occupied by tanoak. According to our data, pacific madrone would come to occupy tanoak-vacated sites with apparently no relation to the abiotic variables we tested (table 13); however, others have reported that this species is not quite as shade tolerant as tanoak and tends to occur on slightly drier and more open sites (Hunter et al. 1999, Burns & Honkala 1990). Furthermore, madrone is currently suffering from several diseases, including SOD (Burns & Honkala 1990, Rizzo et al. 2005), and may therefore be unlikely to establish in much of tanoak's domain. Bigleaf maple might be slightly more apt to replace tanoak at lower slope positions (table 13), especially in the southern portion of the redwood range, where the association with alluvial flats and lower slope positions is most pronounced (Burns & Honkala 1990). Douglas-fir may have an advantage in Humboldt county (table 13). While Douglas-fir recruitment might be expected in the increased light environment of SOD mortality gaps, this relatively shade-intolerant species will probably not be able to persist as a subcanopy tree, although it may compete with redwood for canopy positions

(Burns & Honkala 1990). In broader terms, all trees other than redwood and tanoak (i.e. hardwoods-excl-tanoak and conifers-excl-redwood) are more common on south-facing slopes (table 13), perhaps suggesting that north-facing tanoak mortality gaps are most likely to be occupied by redwood. In addition, there are several species that appear sporadically in the redwood forests within our sampled range, but are too uncommon to facilitate any meaningful predictions as to where they may replace tanoak: canyon live oak, Scouler's willow, hollyleaf cherry, California nutmeg, and grand fir.

There is probably some truth to the above scenario (an insufficient passage of time), but given the general paucity of mature seed-producing trees (other than redwood and tanoak) in the redwood forests within our study area, we suggest that dispersal limitation may continue to be a critical factor. At the scale of our plots, our data show definitive evidence of self-replacement (a proxy for dispersal limitation) for tanoak and redwood, and suggestive evidence ($0.05 < p < 0.1$) of self-replacement for interior live oak and California bay. We have not found evidence of self-replacement for any other tree species, but this may be a result of the very low number of occurrences (with regard to regeneration and/or mature trees) of all other species, thus reducing statistical power to detect such patterns. It is not surprising that canopy gaps lack a clear regenerative response when we consider that **a.** redwood and tanoak often occur in discrete small patches (Waring and O'Hara 2008), **b.** redwood is notoriously poor at seed production and seedling establishment, **c.** tanoak regeneration is susceptible to SOD, and **d.** the vast majorities of basal area and mature stems within our study area are comprised of redwood and tanoak. As such, these gaps may remain open for quite some time, waiting for a rare viable redwood seed, a Douglas-fir seed floating on the wind, an interior live oak acorn rolling downhill in a storm, a California bay fruit dropped by a passing bird, or perhaps even a tanoak acorn with genetic resistance to SOD.

The third scenario that could explain the current paucity of regeneration in SOD-induced tanoak mortality gaps is that unidentified, and relatively permanent, constraints are inhibiting recruitment of other tree species. It is possible that redwood and tanoak are tolerant of particular abiotic conditions (e.g. soil properties) that other native tree species cannot endure. It is also feasible that patches of tanoak within redwood forests occur on sites that even redwood cannot tolerate (i.e. tanoak is maintained by fine scale environmental heterogeneity); however, this seems unlikely given the generally successful efforts of foresters to plant redwood in patches previously dominated by tanoak. Our data yield no insights into properties that might prevent the establish of other tree species in tanoak mortality gaps, and we are unaware of any relevant research. Furthermore, many native tree species do occasionally occur alongside tanoak (in healthy and diseased areas), thus reducing the likelihood of significant abiotic inhibition. As such, we believe it is unlikely that permanent abiotic constraints are solely responsible for the current lack of a regenerative response. However, we do recognize that subtle abiotic constraints could be interacting with transient stochastic factors (e.g. coarse soils combined with low precipitation).

Another possibility to consider – a scenario that does not explain the current paucity of regeneration, but may be important for long-term community assembly processes – is that the loss of tanoak will fundamentally alter the competitive environment of redwood forests, creating a *niche opportunity* (Shea & Chesson 2002), and allowing for the widespread establishment of one or more currently uncommon species. It is often assumed that redwood forests are relatively poor in tree species diversity because of the strong competitive effects of redwood, but there is surprisingly little evidence to support this conjecture. Given that tanoak is a nearly ubiquitous associate of redwood (Burns & Honkala 1990, Noss 2000), we cannot discount the possibility that tanoak is competitively excluding one or more species otherwise capable of persisting in redwood forests. If this is the case, and if tanoak's inhibitory effects are more effective at excluding particular tree species, current species distributions may have little relevance to the composition of future redwood forests. However, it may be possible to accurately predict which species will be most effective at sequestering tanoak's former niche by examining the functional traits of tanoak, redwood, and potential replacement species. For example, a cursory overview of well-documented traits suggests that canyon live oak has potential to thrive in areas currently dominated by tanoak. Although this species is currently uncommon in redwood forests, it is highly shade tolerant and has a deep root system similar to that of tanoak, but distinct from the fairly shallow root systems of redwood (Burns & Honkala 1990). This gross oversimplification implies that tanoak is able to coexist with redwood simply by tolerating deep shade and accessing water and soil nutrients that are beyond the reach of redwood roots. While shade tolerance and root zone stratification are undoubtedly important for species coexistence in forests (Balandier et al. 2006), competitive relations in redwood forests are almost certainly affected by additional variables, including several that should be treated as continuous (as opposed to categorical) variables and many that vary across the geographic range of redwood. As such, effective prediction of tanoak replacement species is likely to require a quantitative multivariate functional traits approach (e.g. Fukami et al. 2005), rather than a simple classification based upon broad functional groups.

Given the absence of a strong regenerative response to SOD-induced tanoak mortality, there is currently great potential for the intentional establishment of other tree species. Land managers who wish to minimize the impacts of tanoak decline (e.g. trophic cascades resulting from the loss of tanoak acorns, reduced *resistance* and/or *resilience* in the face of future threats; sensu Millar et al. 2007) should consider experimenting with the establishment of functionally similar tree species in heavily impacted areas. Such efforts could optionally focus upon species at or near the northern extent of their range, in anticipation of generally warming climatic conditions (i.e. assisted migration, sensu Loarie et al. 2008). By choosing to direct ecological trajectories, managers may successfully alter long-term characteristics such as species composition and stand structure, but such actions will be most efficient in the early stages of community assembly (Thompson et al. 2001). Present conditions could transition to a period of rapid shrub expansion and/or dense recruitment of undesirable tree species, and thus the current abundance of available growing space in heavily impacted areas may characterize a relatively short window of opportunity. Furthermore, because SOD-induced tanoak mortality gaps exhibit some unique properties (e.g. large tanoak sprout clumps arising from root systems of diseased trees), and novel ecosystems are likely to present unfamiliar and unforeseen challenges (Hobbs et al. 2006), it is prudent to assume that successful plantings may require considerable experimentation. As an alternative approach, managers may opt to actively maintain the open nature of these sites, so that if SOD-resistant tanoak genotypes are discovered, these genotypes can be readily reintroduced into areas where tanoak previously dominated.

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APPENDIX A: *What factors could be increasing the severity of tanoak mortality?*

To test our claim (which is supported by Maloney et al. 2005 and Moritz et al. 2008) that tanoak mortality is essentially randomly distributed throughout redwood forests, we examined the abilities of several variables to predict dead tanoak basal area and dead tanoak stem counts. In both cases, we started with the full model, calculated the AIC for all possible combinations of predictors, and selected the model with the lowest AIC (i.e. the "best model"). All models were fit as generalized linear models with a quasipoisson error distribution. Full model predictors were selected to represent fixed abiotic variables and biotic variables that are very unlikely to have responded considerably to SOD-induced tanoak mortality at this point in time: slope, slope position, northness, sampling site, mature redwood abundance, total mature tanoak abundance, and abundance of mature hardwoods-excluding-tanoak and mature conifers-excluding-redwood. In addition, the presence or absence of mature California bay trees was included because this tree species has been linked to disease severity at broad scales (Maloney et al. 2005, Rizzo et al. 2005). No other individual species (aside from redwood and tanoak, which are by far the most dominant species) were considered as potential explanatory variables. Measures of herbaceous species, shrubs, and juvenile tree abundances were not considered because these variables are at least as likely to have to responded *to* tanoak mortality, as opposed to playing a potentially causative role in the SOD epidemic. Full and best models, for dead tanoak basal area as well as dead tanoak stem counts, are displayed below. Significant predictors (p < .05) are shown in bold type, and the direction of the effect is indicated by the symbols in parentheses.

FULL: Dead tanoak BA ~ slope + slope position + northness + Humboldt + Santa Cruz + mature redwood BA + total mature tanoak BA + mature hardwoods-excluding-tanoak BA + mature conifers-excluding-redwood BA + presence/absence of mature CA bay

BEST: Dead tanoak BA ~ slope + slope position + northness + Humboldt + **total mature tanoak BA (+)** + mature hardwoods-excluding-tanoak BA + **presence/absence of mature CA bay (+)**

FULL: Dead tanoak stems ~ slope + slope position + northness + Humboldt + Santa Cruz + mature redwood stems + total mature tanoak stems + mature hardwoods-excluding-tanoak stems + mature conifers-excluding-redwood stems + presence/absence of mature CA bay

BEST: Dead tanoak stems ~ slope + slope position + northness + Humboldt + Santa Cruz + mature redwood stems + **total mature tanoak stems (+)** + mature hardwoods-excluding-tanoak stems + **presence/absence of mature CA bay (+)**

Whether measured in basal area or stem counts, there is some evidence that the presence of mature California bay was associated with increased tanoak mortality, although the explanatory power was low in both cases (partial r2.L was 0.07 for basal area and 0.14 for stems). In addition, it is worth pointing out that the four most severely diseased plots in our dataset contained no mature California bay trees, and our field observations suggest that mature California bay trees were not present within at least several hundred meters of these severely diseased plots. As such, while California bay may have a positive impact on disease severity, it is clear that extreme SOD-induced tanoak mortality is not dependent upon this tree species, at least in redwood forests. This should not be surprising given that the foliage of both tanoak and redwood also foster *P. ramorum* spore production (Davidson et al. 2008). Aside from California Bay presence/absence and total tanoak abundance, no other tested explanatory variables showed any significant relationships with disease severity.

Because the presence or absence of California bay was significantly associated with tanoak mortality (and these variables were thus confounded, although to a very small degree), we re-executed all analyses in Table 2 for which tanoak mortality was found to be significant (or borderline significant), with the presence of mature California bay included in the full model. In these analyses, California bay presence was included in several "best models", but no qualitative changes resulted and all quantitative impacts were very small (results not shown). As such, we are relatively confident in asserting that trends correlated with tanoak mortality were indeed caused by tanoak mortality.

APPENDIX B: *Detailed examination of potential tanoak replacement species*

Aside from tanoak and redwood, California bay and interior live oak are the only species that showed any evidence of self-replacement within our study area (and both were only borderline significant); there were no associations between mature trees and regeneration for bigleaf maple or Douglas-fir. In terms of functional groups, there was a strong self-replacement trend for hardwoods-excl-tanoak (perhaps driven by the combined effects of interior live oak and California bay), but no evidence of self-replacement for conifers-excl-redwood. Beyond self-replacement patterns, interior live oak regeneration was weakly associated with flatter slopes and less total tanoak abundance, while California bay regeneration was strongly associated with steeper slopes, weakly associated with greater redwood abundance, and significantly less common in our Santa Cruz county plots. Bigleaf maple and Douglas-fir regeneration occurred more or less randomly throughout our study area, with respect to the variables we analyzed. Regeneration of hardwoods-excl-tanoak was strongly associated with south-facing slopes and lower slope positions, and most common in our Marin county plots; note that total tanoak abundance and redwood abundance were related to hardwood-excl-tanoak regeneration only if mature members of this functional group were not included as an explanatory variable. Regeneration of conifers-excl-redwood was associated with south-facing slopes, weakly negatively associated with total tanoak abundance, and significantly less common in our Santa Cruz county plots. Tanoak regeneration was positively associated with lower slope positions, strongly negatively associated with redwood abundance, and weakly negatively correlated with disease severity (i.e. dead tanoak abundance). Redwood regeneration was weakly associated with upper slope positions. The following species were not analyzed due to an insufficient number of occurrences: pacific madrone, canyon live oak, Scouler's willow, hollyleaf cherry, California nutmeg, and grand fir.

In an attempt to predict potential long-term changes to SOD-infested redwood forests, and to look for discrepancies between niches of established trees and regeneration, we also examined the effects of abiotic factors and sampling site on the abundances of mature trees. In these GLMs, the abundances of mature tree species or functional groups were fitted against a full model consisting of northness, slope position, slope (deg), Santa Cruz, and Humboldt. Mature interior live oak trees were strongly associated with several abiotic variables; this species was most common on flat slopes, at upper slope positions, with southerly aspects. Mature California bay trees were most abundant on steeper slopes, and were weakly associated with northerly aspects and sites other than Santa Cruz. Mature bigleaf maple trees were weakly associated with lower slope positions, and mature Douglas-fir trees were weakly associated with Humboldt county. Mature pacific madrone trees did not appear to be affected by any of our analyzed variables. Hardwoods-excl-tanoak and conifers-excl-redwood were both associated with more southerly aspects, suggesting that north-facing slopes are comprised of little other than redwood and tanoak. Mature tanoak trees were strongly associated with upper slope positions; however, it is important to point out that tanoak also occurred frequently at mid slope positions, and occasionally at lower slope positions. Similarly, mature redwood trees were most associated with lower slope positions, but occurred frequently on mid and upper slope positions. The following species were not analyzed due to an insufficient number of occurrences: canyon live oak, Scouler's willow, hollyleaf cherry, California nutmeg, and grand fir.

With regard to associated abiotic variables, several discrepancies existed between regeneration and established trees. Interior live oak regeneration was not related to aspect or slope position, but mature trees were strongly associated with southerly aspects and upper slopes, perhaps suggesting that seedlings and saplings on northerly aspects or lower slope positions are unlikely to survive. Similarly, tanoak regeneration was associated with lower slope positions, but mature tanoak trees were strongly associated with upper slope positions. The discrepancy between slope positions of regeneration and mature trees, for both interior live oak and tanoak, perhaps reflects the tendency of their large heavy acorns to roll downhill. In contrast, redwood regeneration was weakly associated with upper slope positions, while mature redwood trees were most common on lower slopes; this pattern perhaps reflects the ability of light redwood seeds to disperse upslope from tall established trees on lower slopes. Regeneration of hardwoods-excl-tanoak was strongly associated with lower slope positions, but mature hardwood-excl-tanoak trees did not appear to be affected by slope position, perhaps reflecting the heavy-seeded nature of many hardwood inhabitants of redwood forest (e.g. interior live oak, California bay, pacific madrone). The abundances of mature hardwood-excl-tanoak and conifer-excl-redwood trees were unrelated to study site, but regeneration of both of these functional groups was reduced in Santa Cruz county, and regeneration of hardwoods-excl-tanoak was also reduced in Humboldt county, indicating that regeneration of hardwoods-excl-tanoak may have been unusually high in our Marin county plots. All other discrepancies exhibited only borderline significance levels.

FINAL PROJECT REPORT: Save-the-Redwoods League Grant (SRL-049-O'Hara, 2007) – PART 2 OF 2
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Sudden oak death–induced tanoak mortality in coast redwood forests: Current and predicted impacts to stand structure

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ABSTRACT

Tanoak (*Lithocarpus densiflorus*) is one of the most widespread and abundant associates of coast redwood (*Sequoia sempervirens*), but no previous initiatives have examined the spatially explicit structural relationships between these two species, and very few have considered any structural attributes of tanoak. Understanding the structural contribution of tanoak is especially important, at present, because of the severe decline this tree species is currently experiencing. The emerging disease sudden oak death, caused by the exotic pathogen *Phytophthora ramorum*, is decimating tanoak populations throughout the entire latitudinal range of redwood, and is threatening the eventual extirpation of tanoak from redwood forests. In this study, we examined the current structural impacts of sudden oak death-induced tanoak mortality, predicted the immediate impacts of 100% tanoak mortality, and considered the long-term structural impacts of tanoak decline in redwood forests. By stratifying sample plots by tanoak abundance and disease severity, and utilizing a stand reconstruction technique to assess pre-mortality structural characteristics, we were able to detect and/or predict several trends, including some that are spatially-explicit. We found that residual trees in diseased plots were more aggregated than trees in unaffected plots, and we predicted that the loss of tanoak will lead to the following short-term changes: greater average diameter, greater average height, greater average height-to-live-crown, greater average crown length, greater average diameter difference between nearest neighbors, greater average height difference between nearest neighbors, and greater average crown length difference between nearest neighbors. In addition, plots without tanoak – as compared to plots with tanoak – exhibited greater average diameter, greater average diameter difference between nearest neighbors, greater average height difference between nearest neighbors, and greater average crown length difference between nearest neighbors. We also conducted a preliminary exploration of how sudden oak death-induced structural changes compare with typical old-growth characteristics, and how this disease may affect the structure of old-growth forests.

INTRODUCTION

Tanoak (*Lithocarpus densiflorus*) is widespread and abundant in coast redwood (*Sequoia sempervirens*) forests, and is believed to be an integral component of the structure and function of these unique ecosystems (Burns & Honkala 1990, Hunter et al. 1999, Noss 2000). As the most abundant hardwood species in conifer forests of California's coastal mountains, tanoak is found alongside redwood throughout the majority of the redwood range (Burns & Honkala 1990, Hunter et al. 1999, Noss 2000). However, the close association between redwood and tanoak may be relegated to history if sudden oak death (SOD) continues to spread throughout coastal California. This emerging forest disease, which is caused by the exotic pathogen *Phytophthora ramorum*, is currently threatening several native tree species, but tanoak is the most severely impacted. Current research demonstrates drastic declines in tanoak populations and mounting evidence (e.g. field studies, genetic resistance trials, disease progression models) suggests that SOD could eventually drive tanoak to extinction in redwood forests (Rizzo et al. 2005, Maloney et al. 2005, McPherson et al. 2005, Meentemeyer et al. 2004, Davidson et al. 2008).

Forest stand structure is often a key determinant of physiognomic resistance, compositional resilience, ecosystem function, wildlife habitat, biodiversity, hydrologic processes, micro-climatic conditions, and regeneration patterns (Oliver & Larson 1996, Zenner & Hibbs 2000, Kint et al. 2003, Pommerening 2002, Ishii et al. 2004, McElhinny et al. 2005, Chang 2006). Stands with greater vertical and/or horizontal heterogeneity are generally believed to support a higher number of species and to be more productive than compositionally similar stands with more uniform structures (Pommerening 2002, Ishii et al. 2004, McElhinny et al. 2005). In addition, stands with two or more canopy strata are thought to be *resistant* and/or *resilient* (sensu Millar et al. 2007) to a broader spectrum of disturbances that single-stratum stands (O'Hara 2006). Stand structure has been found to affect many hydrologic variables, including water uptake and transpiration (Ford & Vose 2007), runoff and water yield (Fernandez et al. 2006), water chemistry (Staelens et al. 2005), and canopy interception and throughfall (Andre et al. 2008). Wildlife habitat is greatly affected by stand structure, and in some cases, structural characteristics may be more relevant than tree species composition; for instance, throughout the western United States, northern goshawks (*Accipiter gentilis*) tend to select stands with large trees and closed canopies, with little apparent concern for the component tree species (Greenwald et al. 2005). In redwood forests, marbled murrelets (*Brachyramphus marmoratus*) nest exclusively in tall trees with large limbs, but do not appear to discriminate between redwood and other conifers capable of achieving very large sizes, such as Douglas-fir (*Pseudotsuga menziesii*; Noss 2000). In general, redwood forests with greater structural diversity are believed to support a greater number of vertebrate species (Noss 2000).

While several non-spatial structural attributes (e.g. total basal area, standard deviation of height) have tremendous predictive value, spatially explicit metrics are often necessary to explain ecological processes and functions. Horizontal positioning of trees is particularly predictive of light levels and regeneration patterns (Kint et al. 2003, Oliver & Larson 1996), as well as several hydrological phenomena. For instance, at similar stocking levels, canopy interception and throughfall rates are affected by the spatial pattern of stems, as well as variations in the vertical structure of neighboring trees (Andre et al. 2008, Chang 2006, Oliver & Larson 1996). Species diversity may also be affected by spatially explicit factors; for example, following timber harvest in western Oregon and Washington states, Louda et al. (2004) found richness and identity of detectable ectomycorrhizal fungi species differed between dispersed and aggregated retention units. In redwood forests, horizontally heterogeneous stands are believed to support a greater number of species than more homogenous stands, and growth rates of young trees are generally greater in canopy gaps (Noss 2000, O'Hara et al. 2007). However, very few data exist documenting the specific implications of horizontal spatial patterns in redwood ecosystems.

Despite the widely recognized importance of three-dimensional structural characteristics, and the abundance of tanoak in redwood forests, there has been no effort to quantify tanoak's contribution to structural diversity. Tanoak often forms a persistent lower canopy layer in redwood forests (Hunter et al. 1999), and thus it is likely that tanoak significantly influences overall stand structure. As a prolific producer of large and highly nutritious acorns (Burns & Honkala 1990), the ecological value of tanoak is undeniable, but its structural worth is not well understood. Does tanoak exhibit unique structural characteristics? Or are its physical contours relatively indistinguishable from those of other tree species, or even suppressed redwood individuals?

In order to address these questions, we installed a network of plots, throughout infested sites, stratified as follows: (a) little or no tanoak [no-tanoak], (b) abundant tanoak with little or no SOD [healthy], and (c) abundant tanoak with considerable SOD-induced tanoak mortality [diseased]. Although all of our data were collected in a single field season, we believe it is possible to assess many of the structural impacts of SOD-induced tanoak mortality. Existing research indicates that the current patchy distribution of SOD in redwood forests, at scales of tens to hundreds of meters, is a result of historical and stochastic factors (Maloney et al. 2005, Rizzo et al. 2005, Moritz et al. 2008), as opposed to underlying biotic or abiotic conditions. As such, it is likely that consistent differences between healthy and diseased areas are caused by SOD. In contrast, differences between areas with and without tanoak may be controlled by other factors (e.g. soil properties), and thus we do not definitively claim tanoak presence as a causative factor.

In addition to direct comparisons between our three sampling strata, we used a stand reconstruction method to model the structural attributes of pre-disease plots, and we project future conditions assuming eventual 100% tanoak mortality. With these reconstructions and projections, we examine predicted trends over time and compare past and future conditions with plots lacking tanoak. Finally, we provide a preliminary exploration of how SOD-induced structural changes compare with typical old-growth characteristics, as well as how SOD may impact old-growth forests with a substantial tanoak component. We consider a wide range of structural variables, including several spatially explicit metrics. The primary objectives of this study were to examine the current structural impacts of SOD-induced tanoak mortality, predict the immediate impacts of 100% tanoak mortality, and consider the long-term structural impacts of tanoak decline in redwood forests.

METHODS

Research was conducted in three different coastal California counties, at field sites which contain redwood forests and are infested with *Phytophthora ramorum* (the causative agent of SOD). Specific study locations were as follows: Henry Cowell Redwoods State Park (Santa Cruz county), Marin Municipal Watershed District (Marin county), and Humboldt Redwoods State Park (Humboldt county). Within these three sites, we installed a total of 13 plots (23 meter radius; 1/6 hectare) in areas satisfying the following criteria: (a) sufficient redwood coverage (at least 25% redwood canopy cover in all four quadrants of a ¼ hectare extended plot); (b) between 50 and 200 meters from trails or roads; and (c) slopes less than 60% (31 degrees). Plots were subjectively located within representative areas capturing the extremes of tanoak abundance and disease severity: (a) little or no tanoak [“no-tanoak”; NT]; (b) abundant tanoak with little or no SOD [“healthy”; H]; or (c) abundant tanoak with severe SOD [“diseased”; D]. We did not verify the presence of *P. ramorum* in our diseased plots, but we are confident that these plots were indeed infested because (a) mortality levels were very high and no other agent is known to cause such severe and widespread mortality of tanoak (Swiecki & Bernhardt 2006), (b) presence of the pathogen has been previously confirmed in the general vicinity of all diseased plots (Moritz et al. 2008, Maloney et al. 2005, Spencer et al. 2004), and (c) characteristic symptoms of SOD (see Rizzo et al. 2005) were common. Areas deemed “representative” were identified as part of previous related research, and precise plot locations were randomized. Within our study area, redwood forest with high abundances of hardwoods other than tanoak was very uncommon, and thus our NT plots were located in areas with a small or non-existent hardwood component. Most plots (11) were installed in second-growth forest, but two were installed in old-growth forest in order to facilitate a preliminary examination of variables related to old-growth status. Key characteristics of all plots are provided in Table 1.

Table 1: Key characteristics of sample plots.

ID [1]	Plot [2]	County	Status	Second-growth / Old-growth	Slope Position	Slope (deg)
1	HCR-S1	Santa Cruz	Diseased	Second-growth	Mid	16
2	HCR-S5	Santa Cruz	No Tanoak	Second-growth	Lower	15
3	HCR-S6	Santa Cruz	Healthy	Second-growth	Mid	8
4	HRSP-S5	Humboldt	Healthy	Old-growth	Lower	14
5	HRSP-S7	Humboldt	Healthy	Second-growth	Lower	12
6	HRSP-S8	Humboldt	No Tanoak	Second-growth	Alluvial	2
7	HRSP-S9	Humboldt	No Tanoak	Old-growth	Alluvial	1
8	MMWD-MS-43	Marin	Diseased	Second-growth	Ridge	19
9	MMWD-S1	Marin	Healthy	Second-growth	Mid	22
10	MMWD-S2	Marin	No Tanoak	Second-growth	Ridge	15
11	MMWD-S4	Marin	No Tanoak	Second-growth	Upper	17
12	MMWD-S7	Marin	Healthy	Second-growth	Ridge	10
13	MMWD-S9	Marin	Diseased	Second-growth	Ridge	18

[1] ID numbers correspond to labels on Figures 4 through 6. [2] Plot names correspond to Figures 1 through 3.

All trees greater than or equal to 10 cm diameter-at-breast-height (DBH) were mapped by recording distance and azimuth from plot center (at breast height), and the following variables were recorded for each tree: species, health status, DBH, height, and height-to-live-crown (HLC). Multi-stemmed trees that were split below breast height were counted as separate trees. Crown ratio and crown length were subsequently calculated with height and HLC for each tree. In order to capture recent SOD-induced tanoak mortality, tanoak stems that were broken below breast height were recorded, provided that the fallen bole wood was relatively intact (i.e. it did not compact when stepped upon); in such cases, we estimated pre-death DBH, distance, and azimuth. Dead individuals of other tree species were not recorded if broken below breast height. In order to reconstruct height (for tanoak trees that were dead and broken/fallen) and HLC (for all dead tanoak trees), we used all living tanoak trees in our dataset to construct models fitting height and HLC against DBH. Simple linear models were used for both height and HLC because visual analysis showed such fits to adequately approximate these relationships. Height and HLC were not reconstructed for any species other than tanoak because this study focuses specifically on SOD-induced tanoak mortality and, for all other species, the percentage of stems that were dead was very low (e.g. redwood) and/or the total number of occurrences was very low (e.g. pacific madrone, *Arbutus menziesii*).

Individual tree-based variables were then used to calculate several plot-level metrics, which included basic structural attributes (simple totals and means), as well as spatially explicit measures of structural complexity: mean nearest neighbor differences (the plot-level mean difference between each tree and its nearest neighbor, with respect to several variables of interest), and the Clark & Evans aggregation index (Bailey & Gatrell 1995; Kint et al. 2003). The Clark & Evans aggregation index expresses the ratio of the average distance between each tree and its nearest neighbor to the average distance expected under a random distribution of points. For all spatially explicit metrics, we imposed a buffer (i.e. a guard area) of three meters, meaning that all focal points were required to be at least three meters from the plot boundary, while secondary points (e.g. nearest neighbors) could be selected from all points within the plot.

Data analysis, which relied upon plot-level summary statistics, consisted of comparisons of healthy, diseased, and no-tanoak plots, as well as reconstructed pre-SOD conditions (0% tanoak mortality) and projected future conditions (100% tanoak mortality); in addition, we examined predicted trends over time within each plot, and qualitatively explored relationships between second-growth and old-growth plots. We used Tukey's Honestly Significant Difference (HSD) tests to identify differences among two separate sets of sample groups. The first HSD test (which we refer to as *observed*) assessed current differences between sampling strata (healthy, diseased, and no-tanoak), and the second HSD test (which we refer to as *inferred*) assessed differences between the following three groups: reconstructed 0% tanoak mortality plots (healthy and diseased plots combined), projected 100% tanoak mortality plots (healthy and diseased plots combined), and no-tanoak plots. In addition, the difference between 0% and 100% tanoak mortality was calculated for each plot individually, and a one-sample t-test was conducted to determine if these intra-plot differences, collectively, were significantly different from zero (referred to as *predicted*). Dead stems of species other than tanoak were excluded from all analyses. The statistical software R, version 2.8.0, by The R Foundation for Statistical Computing, was used to conduct all analyses and create all figures; the supplemental package Spatstat, version 1.14-9, was used for all spatially explicit calculations.

RESULTS

To enhance interpretation of our quantitative results, we here provide horizontal and vertical illustrations of representative second-growth plots from each sampling strata (Fig. 1 through 3).

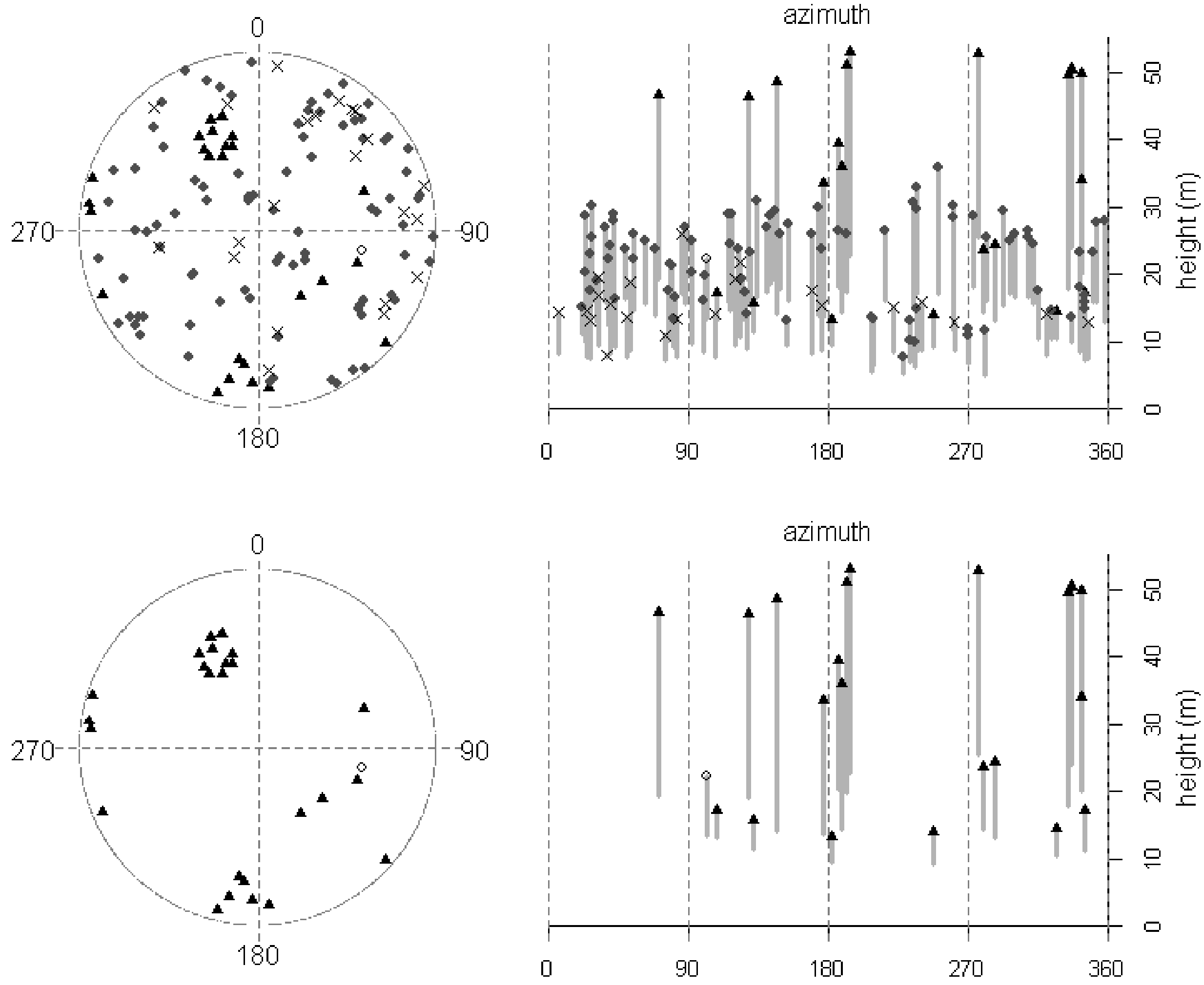

Figure 1: A representative healthy plot (MMWD-S7; see Table 1). Trees are mapped (left) and represented vertically (right; heights are plotted against azimuth, thus “unrolling” the 360 degree view from plot center, and achieving a two-dimensional image by flattening all distances from plot center into a single plane). The top set of graphs displays all trees, including reconstructed dead tanoaks, and the bottom set illustrates this plot after the removal of all tanoaks (both living and dead). Symbols represent the following: “X”s = dead tanoak; gray circles = living tanoak; black triangles = redwood; open circles = hardwoods other than tanoak; no conifers other than redwood were present in this plot. Gray bars display the length of the live crown (the symbol at the top of each bar is the height and the lower limit of each bar is the height-to-live-crown).

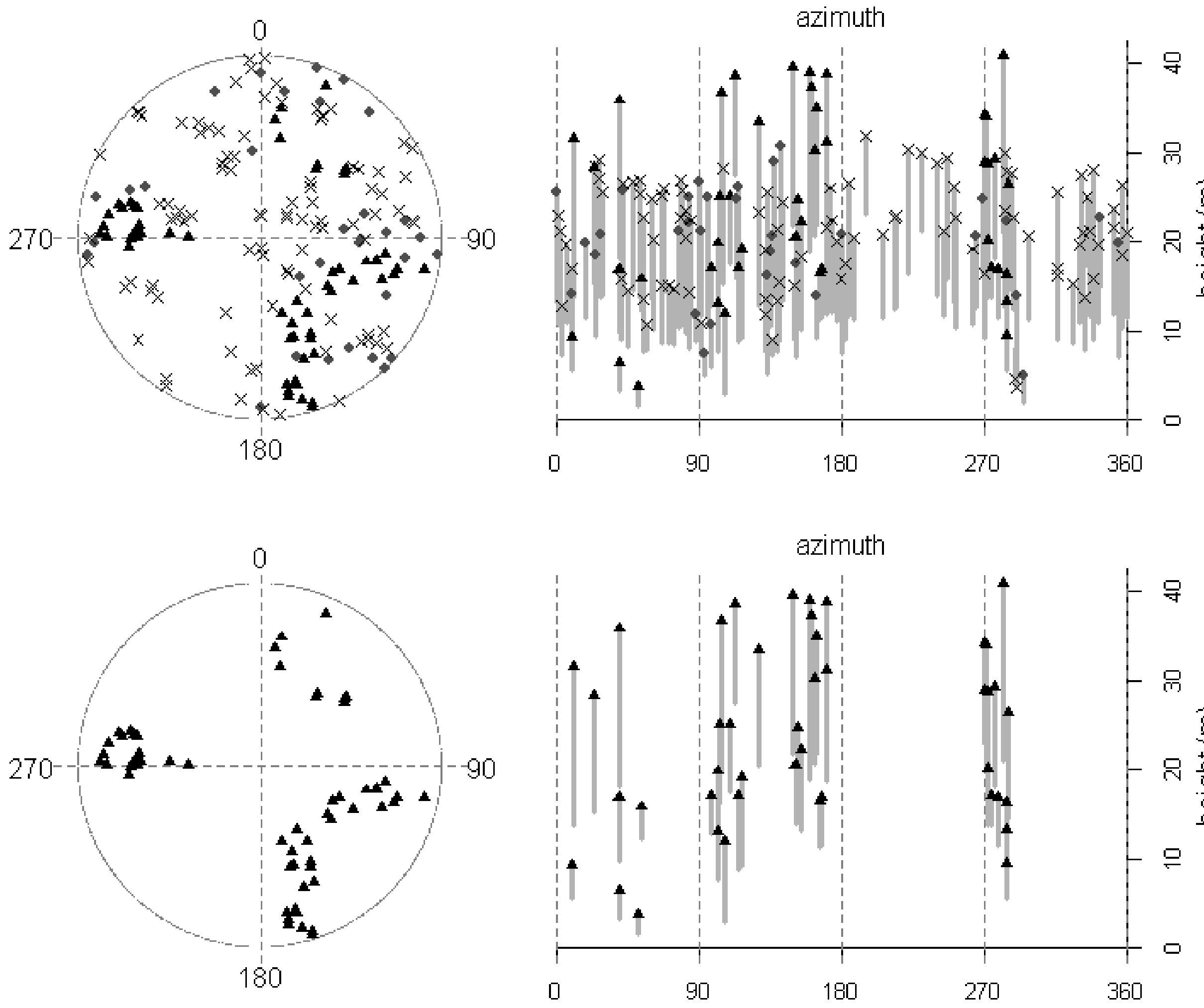

Figure 2: A representative diseased plot (MMWD-S9; see Table 1). Trees are mapped (left) and represented vertically (right; heights are plotted against azimuth, thus "unrolling" the 360 degree view from plot center, and achieving a two-dimensional image by flattening all distances from plot center into a single plane). The top set of graphs displays all trees, including reconstructed dead tanoaks, and the bottom set illustrates this plot after the removal of all tanoaks (both living and dead). Symbols represent the following: "X"s = dead tanoak; gray circles = living tanoak; black triangles = redwood; no tree species other than redwood and tanoak were present in this plot. Gray bars display the length of the live crown (the symbol at the top of each bar is the height and the lower limit of each bar is the height-to-live-crown).

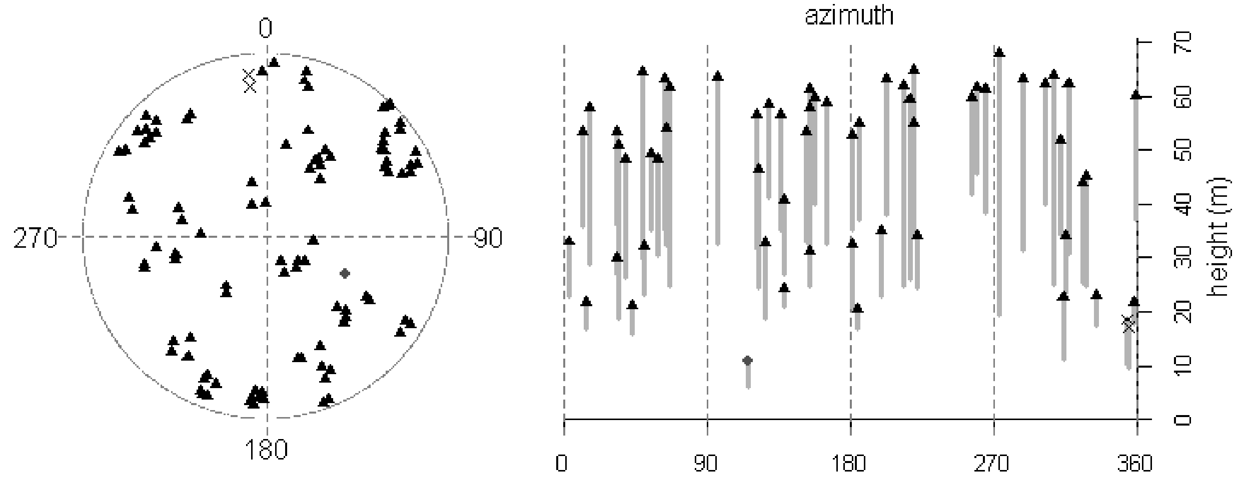

Figure 3: A representative no-tanoak plot (HCR-S5; see Table 1). Trees are mapped (left) and represented vertically (right; heights are plotted against azimuth, thus “unrolling” the 360 degree view from plot center, and achieving a two-dimensional image by flattening all distances from plot center into a single plane). Symbols represent the following: “X”s = dead tanoak; gray circles = living tanoak; black triangles = redwood; no tree species other than redwood and tanoak were present in this plot. Gray bars display the length of the live crown (the symbol at the top of each bar is the height and the lower limit of each bar is the height-to-live-crown).

Many structural variables were significantly related to tanoak presence and/or tanoak mortality (Table 2). The loss of tanoak should obviously result in an immediate reduction in both stem counts and BA (by a predicted average of 73 stems and 3.96 square meters per plot), but our results also show that 0% tanoak mortality reconstructions tended to have *more* stems and *less* BA than plots without tanoak. And similarly, we found that mean DBH was predicted to increase with the loss of tanoak, and that 0% mortality reconstructions had lower mean DBH than 100% mortality projections and plots without tanoak. These findings clearly demonstrate tanoak’s smaller average DBH relative to redwood, as well as its tendency to form dense stands in redwood forests (see Figures 1 through 3).

Mean height, mean HLC, and mean crown length were all predicted to increase with the removal of tanoak from our sample plots, but Tukey’s HSD tests failed to detect any differences between observed or inferred groups. As an example, mean height within each plot was predicted to increase by an average of 5.7 meters, but because of large variations within each sampling stratum, no differences between groups were apparent (Figure 4). Mean crown ratio exhibited no observed, inferred, or predicted relationships with tanoak presence or mortality (Figure 5).

Several spatially explicit measures of structural complexity were related to tanoak presence and/or SOD-induced tanoak mortality. Mean DBH difference between nearest neighbors was predicted to increase with the loss of tanoak, and 0% mortality reconstructions had lower values than 100% mortality projections and plots without tanoak; in addition, mean DBH difference between nearest neighbors was observed to be higher in no-tanoak plots than in either healthy or diseased plots. Mean height difference between nearest neighbors was predicted to increase with the removal of tanoak, and 0% mortality reconstructions had lower values than plots without tanoak (Figure 4). Mean crown length difference between nearest neighbors exhibited a qualitatively identical trend to that found for mean height difference between nearest neighbors. Mean HLC difference between nearest neighbors exhibited no observed, inferred, or predicted relationships with tanoak presence or mortality. Mean crown ratio difference between nearest neighbors displayed similarly non-significant patterns, but there was a borderline positive trend predicted with the loss of tanoak (Figure 5).

Spatial aggregation of stems was strongly impacted by tanoak mortality. Plots were predicted to become "clumpier" (lower Clark & Evans aggregation index values) with the loss of tanoak, and 100% mortality projections exhibited more clustering than 0% mortality reconstructions (Figure 6; these patterns are also clearly evident in Figures 1 through 3). Additionally, diseased plots were more clustered than healthy plots, indicating that – unlike most of our predictions – this impact had already occurred at the time of our field measurements. Aggregation of stems within no-tanoak plots was not significantly different from any group with tanoak. In a separate analysis, we found that the removal of redwood did *not* increase or decrease "clumpiness" (results not shown), demonstrating that the dispersion of tanoak was essentially random in our study plots (see Figures 1 and 2 for representative patterns).

Table 2: Summary of main results. The "*predicted* intra-plot changes" column expresses the results of one-sample t-tests assessing whether differences between 0% and 100% tanoak mortality are significantly different from zero, and if so, the predicted direction of change (see methods section). The number of asterisks indicates the significance level: one symbol = 0.01 < p < 0.05; two symbols = p < 0.01; parentheses indicate borderline significance (0.10 < p < 0.05). The "group comparisons" column displays all significant relationships resulting from Tukey's HSD tests (see methods section); *inferred* (*i*) and *observed* (*o*) relationships are distinguished with parenthetical notations. All analyses consider second-growth plots only.

Variable	***predicted* intra-plot changes (0% → 100% mort.)**	**group comparisons (*inferred* & *observed* relationships)**
Total Stems	decrease **	0% mort. > 100% mort. (*i*); 0% mort. > no-tanoak (*i*)
Total BA	decrease **	0% mort. < no-tanoak (*i*); 100% mort. < no-tanoak (*i*); diseased < no-tanoak (*o*)
Mean DBH	increase **	0% mort. < 100% mort.(*i*); 0% mort. < no-tanoak (*i*)
Mean Height	increase **	no significant relationships
Mean HLC	increase *	no significant relationships
Mean Crown Length	increase *	no significant relationships
Mean Crown Ratio	not significant	no significant relationships
Mean NN Diffs: DBH	increase *	0% mort. < 100% mort.(*i*); 0% mort. < no-tanoak (*i*); healthy < no-tanoak (*o*); diseased < no-tanoak (*o*)
Mean NN Diffs: Height	increase *	0% mort. < no-tanoak (*i*)
Mean NN Diffs: HLC	not significant	no significant relationships
Mean NN Diffs: Crown Length	increase *	0% mort. < no-tanoak (*i*)
Mean NN Diffs: Crown Ratio	(increase)	no significant relationships
C&E Aggregation Index	decrease **	0% mort. > 100% mort. (*i*); healthy > diseased (*o*)

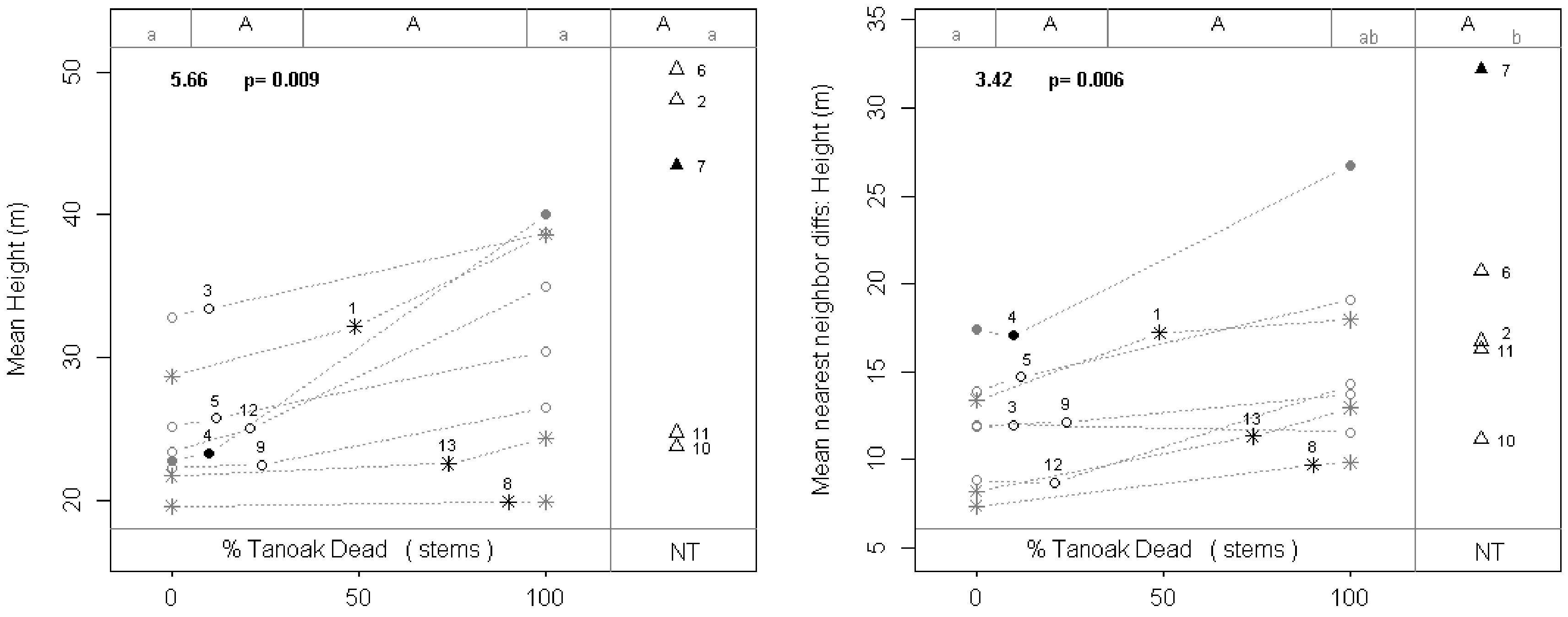

Figure 4: Mean height (left) and mean height difference between nearest neighbors (right) as a function of plot status and disease progression. Black numbered symbols represent present conditions of all 13 sampled plots: circles = healthy plots (H); stars = diseased plots (D), triangles = plots without tanoak (NT); open symbols = second growth plots; closed symbols = old growth plots. Grey symbols represent reconstructed pre-SOD conditions (0% tanoak dead) and projected future conditions (100% tanoak dead) for all plots with tanoak. Grey dotted lines connect each plot's present condition to its respective pre-SOD and future conditions, thereby displaying predicted transitions for each plot. Plot ID numbers correspond to Table 1. The results of several analyses, all of which exclude the two old growth plots, are also displayed. Letters at the top of the figure indicate significant differences between groups, as determined by two separate Tukey's HSD tests. The first test (of *observed* impacts), which assessed the predictive ability of present plot status, is represented by black capital letters corresponding to H, D, and NT plots (from left to right). The second test (of *inferred* impacts), which is represented by grey lower case letters, compared the following three groups (from left to right): reconstructed 0% tanoak mortality plots, projected 100% tanoak mortality plots, and present NT plots. All significance class letters are positioned directly above the group that is referenced. In addition, the *predicted* difference between 0% and 100% tanoak mortality was calculated for each plot individually, and a one-sample t-test was conducted to determine if these differences, collectively, were significantly different from zero. Near the top of the plotting area, the mean of these intra-plot differences and the corresponding p-value are displayed.

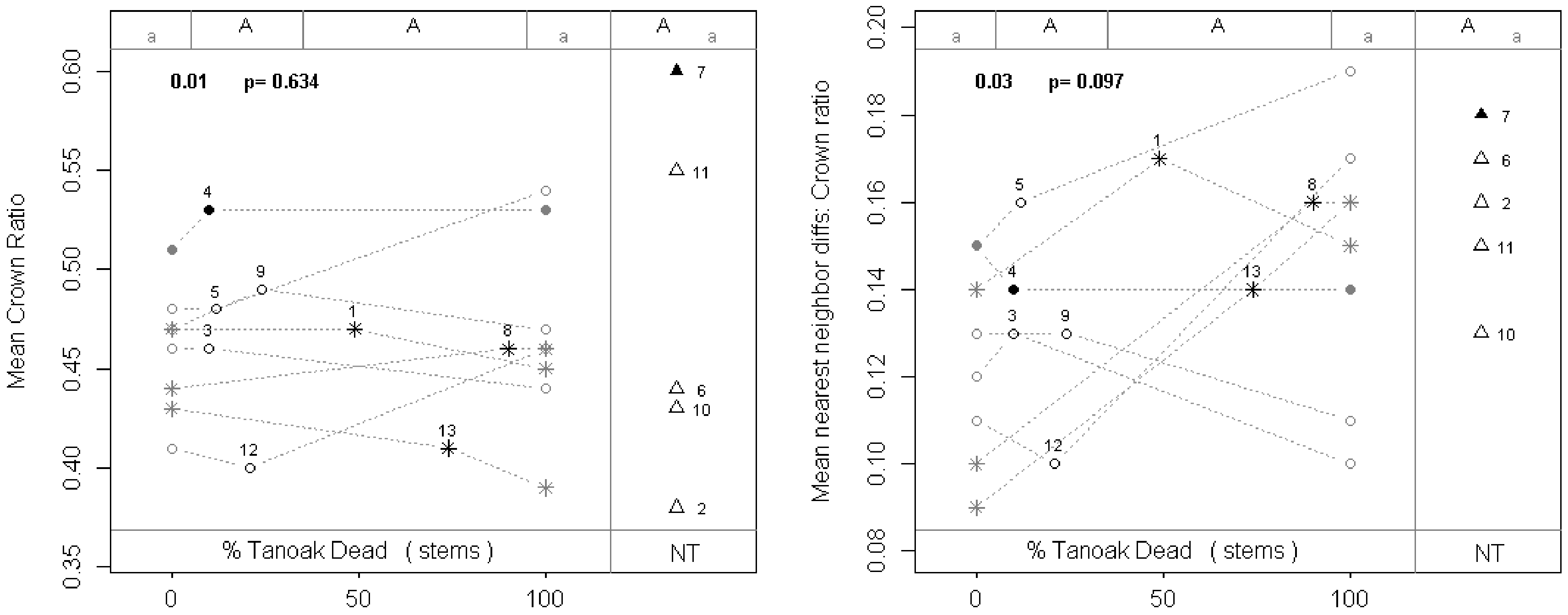

Figure 5: Mean crown ratio (left) and mean crown ratio difference between nearest neighbors (right) as a function of plot status and disease progression. See the caption to Figure 4 for a full explanation of symbols and analyses.

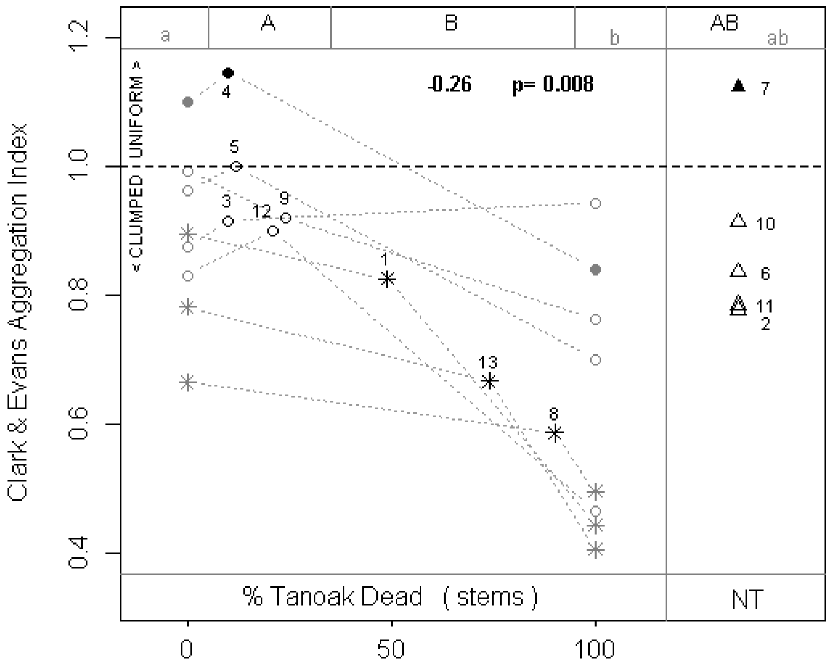

Figure 6: Clark &Evans aggregation index as a function of plot status and disease progression. See the caption to Figure 4 for a full explanation of symbols and analyses.

Most variables displayed generally accepted distinctions between old-growth and second-growth redwood forests (Table 3), demonstrating that our two old-growth plots were fairly representative; for instance, both old-growth plots were less aggregated (higher Clark & Evans values) than comparable (i.e. H or NT) second-growth plots (Figure 6, and all nearest neighbor differences were higher in old-growth plots than in comparable second-growth plots (see Figures 4 and 5 for examples). Following the projected loss of tanoak from second-growth redwood forests, the values of most structural variables were predicted to shift towards old-growth values (H-OG and/or NT-OG), or to exhibit inconsistent relationships with old-growth (H-OG and/or NT-OG). Only one variable, Clark & Evans aggregation index, was predicted to diverge from both H-OG and NT-OG. If SOD becomes established in old-growth redwood forests (H-OG), our results suggest that the structural impacts will be similar to the impacts predicted for second-growth forests; for all variables for which statistically significant changes within our second-growth plots were predicted (from 0% mortality reconstructions to 100% mortality projections), the old-growth plot was predicted to undergo qualitatively identical changes. All results regarding old-growth plots should be interpreted with caution because only two old-growth plots were sampled and no statistical tests were conducted.

Table 3: Anecdotal relationships between second-growth and old-growth. In the "Healthy" and "No Tanoak" columns, relationships are displayed for metrics in which all second-growth plot values (within the specified sampling strata) were consistently higher or consistently lower than the old-growth plot. In the "SG to *H*-OG?" column, "towards" indicates that the removal of tanoak was predicted to shift values of the given metric (for second-growth plots) towards the value of the healthy old-growth plot, while "away" indicates a predicted shift in the opposite direction of the healthy old-growth value. In the "SG to *NT*-OG?" column, "towards" indicates that the removal of tanoak was predicted to shift values of the given metric (for second-growth plots) towards the value of the no-tanoak old-growth plot, while "away" indicates a predicted shift in the opposite direction of the "no-tanoak" old-growth value. In the "OG vs SG trend" column, "similar" indicates that the removal of tanoak from the healthy old-growth plot was predicted to have qualitatively identical impacts (i.e. same direction of change) as the removal of tanoak from second-growth plots. Zeros indicate no clear pattern between second-growth and old-growth.

Variable	**Healthy**	**No Tanoak**	**SG to *H*-OG?** (0% to 100%)	**SG to *NT*-OG?** (0% to 100%)	**OG vs. SG trend** (0% to 100%)
Total Stems	SG > OG	SG > OG	towards	towards	similar
Total BA	0	SG < OG	0	away	similar
Mean DBH	0	SG < OG	0	towards	similar
Mean Height	0	0	0	0	similar
Mean HLC	SG > OG	0	away	0	similar
Mean Crown Length	0	SG < OG	0	towards	similar
Mean Crown Ratio	SG < OG	SG < OG	0	0	0
Mean NN Diffs: DBH	SG < OG	SG < OG	towards	towards	similar
Mean NN Diffs: Height	SG < OG	SG < OG	towards	towards	similar
Mean NN Diffs: HLC	SG < OG	SG < OG	towards	towards	similar
Mean NN Diffs: Crown Length	SG < OG	SG < OG	towards	towards	similar
Mean NN Diffs: Crown Ratio	SG < OG	SG < OG	0	0	0
C & E Aggregation Index	SG < OG	SG < OG	away	away	similar

DISCUSSION

Our analyses indicate that several important structural characteristics are likely to be affected by the loss of tanoak from redwood forests, and we were able to detect a current difference in “clumpiness” between healthy and diseased plots. Predicted reductions in total stem counts and total basal are a necessary immediate impact of SOD-induced tanoak mortality, and predicted instantaneous increases in mean DBH, mean height, mean HLC, and mean crown length are consistent with the larger size of redwood relative to tanoak. However, if new cohorts eventually regenerate in areas experiencing high levels of tanoak mortality (a regenerative response was not evident at the time of field sampling; unpublished data), all of these basic structural attributes should rapidly shift towards their pre-SOD values.

We initially hypothesized that tanoak might be increasing the structural complexity of redwood forests, but we have found no evidence to support this hypothesis. On the contrary, the two measures of vertical complexity that were significantly related to tanoak (mean height difference between nearest neighbors and mean crown length difference between nearest neighbors) exhibited decreased diversity when tanoak was present (0% reconstruction as compared to no-tanoak plots), and predicted increases immediately following the loss of tanoak. Similarly, mean DBH difference between nearest neighbors, which could be considered a form of horizontal structural complexity, was negatively affected by tanoak. Plots without tanoak exhibited higher values than healthy plots, diseased plots, and 0% mortality reconstructions, and the loss of tanoak was predicted to result in an immediate increase in mean DBH difference between nearest neighbors. All of these structural complexity metrics should also be affected by future regeneration, but unlike the basic structural attributes discussed above, a new cohort should cause these spatially explicit variables to diverge even farther from their pre-SOD values.

With respect to several attributes (DBH, height, and crown length), average nearest neighbor differences appear to be reduced by the fairly dense lower canopy layer of similarly sized tanoak trees (e.g. Figures 1 and 2). However, these metrics of structural diversity may not capture all ecologically relevant structural characteristics; while patches of similarly sized trees will decrease neighbor nearest differences, they may increase other structural quantification metrics (e.g. “patch-types”; sensu Zenner & Hibbs 2000). For instance, the juxtaposition of a plot with a dense sub-canopy layer (e.g. Figure 1) and a plot with a less contiguous lower canopy (e.g. Figure 3) may be more relevant to some wildlife species than the contrasting characteristics of neighboring trees. Relevant metrics would necessarily require a coarser resolution (spatial grain), rendering any such analyses meaningless – or at least unreliable – at the scale of our plots.

Several observations suggest that SOD-induced tanoak mortality may be accelerating the emergence of old-growth structural attributes in second-growth redwood stands. As our results show, tanoak mortality should lead to immediate as well as long-term increases in vertical complexity, an attribute that is generally characteristic of old-growth stands (Noss 2000, Sillett & Van Pelt 2007). Our preliminary comparison of second-growth and old-growth plots suggested that, with the removal of tanoak, most structural metrics in second-growth plots should move closer to old-growth values (Table 3). In addition, another study concluded that SOD-induced tanoak mortality was increasing growth rates and basal sprout regeneration of neighboring redwood trees (Waring & O’Hara 2008). A notable exception to the potential shift towards old-growth characteristics was the predicted and observed increases in clumpiness. Old-growth redwood stands tend to exhibit much more uniform dispersion patterns than second-growth stands, which are characterized by dense clusters of trees surrounding cut stumps (Waring & O'Hara 2008). However, this short-term increase in horizontal structural complexity could lead to a longer-term acceleration towards old-growth characteristics. Given that the current distribution of SOD is very patchy at many scales, and that the dispersion of tanoak within redwood forests is similarly non-uniform, the structural impacts of SOD may be analogous to a large-scale form of variable density thinning. This method, which aims to increase structural complexity as well as growth rates of residual trees, has been proposed as a strategy to accelerate the transition from second-growth to old-growth in many forest types (Franklin et al. 2007), including coast redwood (O'Hara et al. 2009). While the structural impacts of SOD-induced tanoak mortality may therefore be desirable to natural resource managers and casual observers alike, the compositional impacts should be much more unsettling. Tanoak is currently widespread and abundant in old-growth redwood stands, especially on upland sites, and thus the loss of this species is likely to profoundly change the ecology of these forests.

Our inability to observe many significant differences between healthy and diseased plots (in contrast to the large number of significant predictions and inferences) may suggest that many expected changes have yet to occur, but it is also possible that this lack of evidence was due to small sample sizes; for instance, healthy and diseased plots (present condition) consisted of four and three replicates, respectively, while 0% mortality reconstructions and 100% mortality projections consisted of seven replicates each. As such, we have not dismissed the possibility that many expected changes were already occurring at the time of our field measurements. Similarly, structural variables that appeared to be entirely unrelated to tanoak presence and SOD-induced tanoak mortality might have exhibited subtle differences had sample sizes been larger.

Although our reconstructions (0% tanoak mortality prior to SOD) and projections (100% tanoak mortality in the future) are somewhat unrealistic, these assumptions are definitely within the realm of possibility; background mortality levels for tanoak (i.e. on uninfested sites) are very low, and SOD-induced tanoak mortality is already approaching 100% in some localized areas (Moritz et al. 2008). However, it is important to emphasize that our 100% tanoak mortality projections assess the structural conditions that would exist if all remaining tanoak trees were to die immediately; in reality it could take many years for projected mortality levels to occur, allowing other trees to recruit in the interim period. Similarly, but with respect to our 0% tanoak mortality reconstructions, some recruitment may have occurred in the time that has passed between tree death and field measurements. It is also worth noting that our models using DBH for vertical reconstruction do not capture all of the variation in height and HLC (r^2 equals .53 and .30, respectively), and thus reconstructed structural characteristics do not perfectly represent pre-SOD conditions. Depending upon diameter distributions, as well as precise locations of reconstructed tanoaks, estimated height and HLC values could serve to over- or under-estimate the actual structural complexity that existed prior to tanoak mortality. Our inferences and predictions should thus be viewed as a preliminary assessment of the structural changes that may occur as a result of SOD. More complicated procedures (e.g. simulations of disease progression, recruitment, and/or growth responses) could be used in conjunction with our results in order to more accurately forecast future stand structures.

Plots that currently lack tanoak may or may not be representative of the structures that will emerge in the wake of SOD. If tanoak's current distribution is mostly due to dispersal limitation or stochastic factors, structures similar to those characterizing plots without tanoak may develop in infested areas. However, it is probable that underlying abiotic factors (e.g. soil conditions) and/or disturbance regimes (e.g. floods and silt deposition) affect the abundance of tanoak and other tree species within redwood forests (Burns & Honkala 1990, Lorimer et al. 2008). As such, we cannot definitely distinguish between the direct effects of tanoak presence and other potentially confounding factors. For instance, if areas where tanoak is currently thriving are inherently supportive of a lower canopy layer of hardwoods or other smaller statured trees, then pre-SOD structures may eventually re-emerge after the loss of tanoak. On the other hand, tanoak may be the only tree species with the ability to effectively compete with redwood (i.e. maintain high relative abundance levels) on some sites. Given this scenario, infested areas would be more likely to acquire structural characteristics that resemble areas currently devoid of tanoak.

While our data have revealed some interesting patterns, several key questions remain: How long will the immediate structural characteristics of SOD-induced tanoak mortality persist? Will infested stands remain open and aggregated far into the future or will a new cohort of trees quickly establish? Will areas vacated by tanoak begin to resemble areas currently devoid of tanoak, or will entirely new structures emerge? Changes to forest structure have been linked to trophic cascades and various ecosystem processes (Oliver & Larson 1996, Zenner & Hibbs 2000, Kint et al. 2003, Pommerening 2002, Ishii et al. 2004, McElhinny et al. 2005, Chang 2006), and thus these questions should be of great interest to a wide swath of society, encompassing land managers, recreational forest users, and anyone dependent upon ecosystem services from redwood forests. While the structural impacts of SOD-induced tanoak decline may be considerable, the compositional impacts of this emerging disease may be even greater. In redwood forests, species richness is believed to be higher in areas where other tree species, especially those bearing fruits or nuts, are relatively abundant (Noss 2000), and tanoak acorns in particular are known to sustain a wide range of wildlife species (Burns & Honkala 1990). As such, if tanoak is not replaced by one or more functionally similar tree species, redwood forests – which are already relatively species-poor – may experience severe reductions in biodiversity. The potential for trophic cascades and other compositional impacts, in conjunction with the structural impacts we have documented and/or predicted, suggests that redwood forests are currently experiencing profound and lasting ecological change.

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