Sudden Oak Death in Redwood Forests: Vegetation Dynamics in the Wake of Tanoak Decline[1]

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Introduction

Numerous lines of inquiry have concluded that tanoak (*Lithocarpus densiflorus*) will continue to experience drastic population declines and may even disappear entirely from redwood (*Sequoia sempervirens*) forests as a result of the exotic disease sudden oak death (SOD) (Maloney and others 2005, McPherson and others 2005, Meentemeyer and others 2004, Rizzo and others 2005). As the only species that can both compete for canopy space and tolerate the deep shade of redwood understories, tanoak is widespread and abundant in redwood forests, and is an integral component of the structure and function of these unique ecosystems. Tanoak is the most common hardwood species in conifer forests of California's coastal mountains, and is associated with redwood throughout the majority of the redwood range (Burns and Honkala 1990, Hunter and others 1999, Noss 2000). As such, the loss of tanoak from redwood forests is likely to result in significant impacts. The primary objectives of this study were to: (1) examine the short-term compositional and regenerative effects of SOD in redwood forests; (2) determine which compositional and regenerative variables are correlated with tanoak abundance; and (3) consider the long-term structural and compositional effects of SOD-induced tanoak decline in redwood forests.

Materials and Methods

In order to assess the effects of SOD-induced tanoak decline, measurements of forest structure, shrub and herb cover and composition, and regeneration were collected in 65 plots distributed throughout three infested counties. Most plots were randomly located within second-growth redwood forest, but some supplemental plots were subjectively installed in order to ensure adequate representation of the full range of tanoak abundance and disease severity. All field research was conducted in a single field season (summer 2008), and thus we do not have time series data, but we nonetheless believe it is possible to reliably infer short-term impacts of SOD-induced tanoak mortality. Given that the current highly patchy distribution of SOD in redwood forests is believed to be a result of introduction events and stochasticity (Maloney and others 2005, Moritz and others 2008, Rizzo and others 2005), as opposed to confounding biotic or abiotic variables, it is likely that differences correlated with SOD are caused by SOD. In contrast, relationships between total

tanoak abundance and other investigated variables could be controlled by other unmeasured factors (for example, soil nutrients), and thus we are hesitant to claim a causative role for tanoak abundance.

Field sites were located in the counties of Santa Cruz (The Forest of Nisene Marks and Henry Cowell Redwoods State Park), Marin (Marin Municipal Watershed District), and Humboldt (Humboldt Redwoods State Park). Data collection consisted of the following: (1) species, health status, diameter, and presence or absence of basal sprouts for all trees greater than 10 cm diameter at breast height; (2) cover and composition of shrubs and herbs; (3) tree regeneration tallies; (4) canopy cover and coarse woody debris; and (5) standard plot measurements such as slope, aspect, and elevation. Foliar samples were also collected in each plot to test for the presence of *Phytophthora ramorum*, the causative pathogen. Samples were processed by the University of California (UC) Berkeley forest pathology laboratory.

Data analysis consisted primarily of multivariate generalized linear modeling. This flexible technique allowed us to examine response variables with highly non-normal error distributions (for example, regeneration counts), and to isolate the effects of tanoak mortality and abundance while accounting for other uncontrolled variables. For all response variables, we specified the appropriate error distribution and started with the following full model: Y ~ dead tanoak abundance + total tanoak abundance + redwood abundance + slope + slope position + northness + Humboldt + Santa Cruz [Marin = baseline]. We then fit models with all possible subsets of predictors, and selected the "best" model (the model with the lowest AIC [Akaike's Information Criterion] value) for interpretation. In addition, we calculated simple averages of tanoak regeneration and mature tree abundance; for these calculations, we utilized randomly located plots only.

Results and Discussion

Our attempt to verify the presence of *P. ramorum* in diseased plots was unsuccessful. 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 2 very dry years, and dry conditions are known to decrease the likelihood of detection (Rizzo and others 2005); (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 and 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 (for example, competition, windthrow).

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.

Our results demonstrate the great abundance of tanoak in redwood forests within our study area, and also identify several variables that are related to SOD-induced tanoak mortality and/or total tanoak abundance. In all three counties, mean tanoak basal area, stem counts, and importance values were higher than all other hardwoods combined, as well as all conifers excluding redwood. In terms of regeneration, median tanoak regeneration exceeded all other species combined, in all three counties, and tanoak regeneration occurred in 100 percent of our randomly located plots; as such, tanoak is currently a ubiquitous component of the regeneration stratum in redwood forests.

After accounting for the effects of all other potential model predictors (total tanoak abundance, redwood abundance, slope, slope position, northness, and sampling site), SOD-induced tanoak mortality was positively associated with coarse woody debris, herbaceous species richness, the percent of total mature stems with basal sprouts, and the percent of mature tanoak stems with basal sprouts. SOD–induced tanoak mortality was negatively associated with total canopy cover. In addition, weak positive relationships (significance levels between 0.05 and 0.1) were found with regard to the percent of mature redwood stems with basal sprouts and the percent of mature hardwood (excluding tanoak) stems with basal sprouts. We discovered no effects of SOD-induced tanoak mortality on herbaceous cover, shrub cover, shrub richness, mature tree richness, or juvenile tree richness.

Notably, we did not detect a regenerative response to SOD-induced tanoak mortality (in terms of absolute counts, as opposed to the percent of mature stems with basal sprouts) for any regeneration category (total, tanoak, redwood, hardwoods excluding tanoak, conifers excluding redwood, and all species excluding tanoak and redwood). This result was surprising given the frequently observed – and often dramatic – deterioration of the canopy, a process that had already begun 7 years prior to field sampling in at least some of our plots (as documented by Spencer 2004). It is also worth noting that the plots with historical records were not as severely impacted as many other plots, and thus it is likely that some plots had been experiencing increased light levels for well over 7 years.

After accounting for the effects of all other potential model predictors (tanoak mortality, redwood abundance, slope, slope position, northness, and sampling site), total tanoak abundance was negatively associated with herbaceous cover, herbaceous richness, shrub richness, mature tree richness, the percent of mature redwood stems with basal sprouts, and the percent of mature non-redwood non-tanoak stems with basal sprouts. Total tanoak abundance was positively associated with tanoak regeneration, suggesting a tendency towards self-replacement in the absence of SOD.

In summary, our results suggest that as diseased tanoaks die and deteriorate, the forest canopy opens, coarse woody debris levels increase, and herbaceous species experience increased seedling recruitment and/or vegetative expansion. In addition, basal sprout incidence may weakly increase for some tree species, but a pronounced

and definitive regenerative response is unlikely to occur, at least in the first several years after mortality. Given the current paucity of regeneration in areas with high mortality, we have little evidence with which to predict what tree species will eventually occupy SOD-induced tanoak mortality gaps. Our data also illuminate correlations between total tanoak abundance and several variables of interest, but such relationships may be controlled by other variables that were not measured as part of this research. Long-term impacts of SOD-induced tanoak decline will depend upon the extent to which tanoak abundance controls other key variables, and how such effects will interact with short-term mortality patterns, tree regeneration, and resulting ecological trajectories.

Literature Cited

Burns, R.M. and Honkala, B.H. (eds.). 1990. Silvics of North America. Volume 1, Conifers. Agriculture Handbook 654. Washington, D.C.: U.S. Department of Agriculture, Forest Service. 675 p.

Hunter, J.C.; Parker, V.T. and Barbour, M.G. 1999. Understory light and gap dynamics in an old-growth forested watershed in coastal California. Madrono. 46: 1–6.

Maloney, P.E.; Lynch, S.C.; Kane, S.F. [and others]. 2005. Establishment of an emerging generalist pathogen in redwood forest communities. Journal of Ecology. 93: 899–905.

McPherson, B.A.; Mori, S.R.; Wood, D.L. [and others]. 2005. Sudden oak death in California: disease progression in oaks and tanoaks. Forest Ecology and Management. 213: 71–89.

Meentemeyer, R.; Rizzo, D.; Mark, W. [and others]. 2004. Mapping the risk of establishment and spread of sudden oak death in California. Forest Ecology and Management. 200: 195–214.

Moritz, M.; Moody, T.; Ramage, B.S. and Forestel, A. 2008. Spatial distribution and impacts of *Phytophthora ramorum* and sudden oak death in Point Reyes National Seashore. National Park Service Report. [Pages unknown].

Noss, R.F. (ed.). 2000. The Redwood forest: history, ecology and conservation of the coast redwood. Washington, DC: Island Press. 340 p.

Rizzo, D.M.; Garbelotto, M. and Hansen, E.M. 2005. *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. Annual Review of Phytopathology. 43: 309–335.

Spencer, M. 2004. A spatial analysis of the *Phytophthora ramorum* symptom spread using second-order and GIS based analyses. Chapter 1 of Doctoral Dissertation. Berkeley, CA: University of California at Berkeley. [Pages unknown]. PH.D Dissertation.

Swiecki, T.J. and Bernhardt, E.A. 2006. A field guide to insects and diseases of California oaks. Gen. Tech. Rep. PSW-GTR-197. Albany, CA: Pacific Southwest Research Station, Forest Service, U.S. Department of Agriculture. 151 p.