Regeneration and Tanoak Mortality in Coast Redwood Stands Affected by Sudden Oak Death[1]

Benjamin S. Ramage[2], Kevin L. O'Hara[2], and Alison B. Forrestel[2]

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 (*Notholithocarpus densiflorus*), is currently widespread and abundant in the redwood ecosystem, but diseased areas have begun to experience considerable mortality. Tanoak, which is extremely valuable as food source to numerous wildlife species, is unlikely to successfully regenerate in these areas, and thus affected redwood forests are transitioning to a novel state. In this study, to predict which species might replace tanoak, we investigated regeneration patterns in heavily impacted stands in Marin County, California. Our main findings were: (1) despite reductions in canopy cover, there is no evidence that any species other than tanoak has exhibited a regenerative response to tanoak mortality, (2) the regeneration stratum was dominated by redwood and tanoak (other tree species were patchy and/or scarce), and (3) some severely affected areas lacked sufficient regeneration to fully re-occupy available growing space. Our results indicate that redwood is likely to initially re-occupy the majority of the ground relinquished by tanoak, but also provide evidence that longer-term trajectories have yet to be determined and may be highly responsive to management interventions.

Key words: *Lithocarpus densiflorus*, *Notholithocarpus densiflorus*, *Phytophthora ramorum*, regeneration, redwood, *Sequoia sempervirens*, sudden oak death, tanoak

Introduction

Tanoak (*Notholithocarpus densiflorus* syn. *Lithocarpus densiflorus*), a broadleaf evergreen in the Fagaceae family, is currently 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 and 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, from numerous field studies (for example, Maloney et al. 2005, McPherson et al. 2010, Ramage et al. 2010) and

[1] A longer version of this paper has been published. If you wish to cite this paper, please reference the original publication (Ramage, B.S.; O Hara, K.L.; Forrestel, A.B. 2011. **Forest transformation resulting from an exotic pathogen: regeneration and tanoak mortality in coast redwood stands affected by sudden oak death**. Canadian Journal of Forest Research 41: 763-772.).

[2] Dept. of Environmental Science, Policy, & Management, University of California, Berkeley, CA 94720-3114. Corresponding author: bsramage@berkeley.edu.

several disease progression models (for example, Meentemeyer et al. 2004), suggests that SOD could eventually drive tanoak to extinction in redwood forests.

Several tree species succumb to SOD, but tanoak is the most severely affected tree and the most abundant SOD-susceptible species in redwood forests (Burns and Honkala 1990, Rizzo et al. 2005). The extreme susceptibility of tanoak results from a combination of factors: (a) little or no genetic resistance, (b) susceptibility of 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 appear to require the presence of a secondary foliar host for bole infection; Maloney et al. 2005, Ramage et al. 2010, Rizzo et al. 2005). Furthermore, because many native species support sub-lethal foliar infections, including redwood (Davidson et al. 2008), *P. ramorum* has almost certainly become a permanent resident of infested areas, and thus it is unlikely that tanoak will ever return to pre-SOD abundances in diseased redwood forests. Tanoak stumps often sprout prolifically following death of the main bole (Cobb et al. 2010, Ramage et al. 2010), and it is feasible 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 scenario was to manifest, such an outcome would still represent a form of *functional extinction* (sensu Ellison et al. 2005).

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 and 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. Of particular concern is the question of whether functionally similar native tree species will be able to colonize and persist in areas previously occupied by tanoak. Tanoak regularly produces large nutritious acorns that are utilized by many wildlife species (for example, bear, deer, and several rodent and bird species), while redwood produces unpredictable crops of small and light seeds with limited wildlife value (Burns and Honkala 1990). If tanoak is not replaced by one or more functionally similar tree species (for example, a true oak 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).

Regeneration in SOD-induced mortality gaps is likely to differ from regeneration in areas not experiencing mortality, and – because SOD gaps represent a novel occurrence – unexpected patterns may emerge. Features such as standing dead trees and debris piles may attract birds and thereby increase the input of bird-dispersed seeds into disturbed areas (Rost et al. 2009). Seedling and sprout survival and growth rates may increase following the death of mature trees via: (a) improved photosynthetic capacity, which can occur with even small reductions in canopy cover, and/or (b) a reduction in the intensity of competition for water and soil nutrients (Oliver and Larson 1996, Smith et al. 1997); increased basal sprouting incidence could also occur due to bole damage from falling trees. These mechanisms

vary among species (Grubb et al. 1977) and thus relative abundances of regeneration in mortality gaps are likely to differ from the surrounding matrix. In the case of redwood, studies have shown that basal sprout survival and growth rates were greater in higher light environments (O'Hara and Berrill 2010, O'Hara et al. 2007), and that a greater proportion of established redwood stems had basal sprouts in an area experiencing SOD-induced tanoak mortality than in an unaffected area (Waring and O'Hara 2008).

Our specific objectives were to: (1) test the hypothesis that regeneration is positively associated with tanoak mortality, (2) identify the species that are regenerating in the greatest numbers in areas severely affected by sudden oak death, and (3) consider the potential implications of the observed patterns.

Methods

Fieldwork was conducted at the Marin Municipal Water District (MMWD), which occupies approximately 8500 ha of protected land on the northern slope of Mount Tamalpais in Marin County, California. Redwood forest, primarily second-growth stands that originated at the end of the 19th century or beginning of the 20th century, is scattered throughout the watershed, covering a wide range of slopes, slope positions, and aspects. Unusual levels of tanoak mortality were first observed in Marin County in 1994, and SOD has been causing extensive tanoak mortality at MMWD since at least 2000 (McPherson et al. 2010, Rizzo et al. 2002). Most study plots (1/20 ha; 12.62 m radius) were randomly located (in redwood forest), but we also used a stratification protocol to ensure adequate representation of: (a) areas with high tanoak abundance but little or no tanoak mortality, and (b) areas with very high levels of tanoak mortality. Additional details are provided in the full version of this paper (Ramage et al. 2011).

Data for mature trees ($\geq$ 10 cm diameter at breast height; 1.37 m height; DBH) were collected during the summer of 2008. Within each plot, we recorded species and DBH for all tree species, as well as health and deterioration status for all tanoaks; details are provided in the full version of this paper (Ramage et al. 2011). In 2008, we also collected one sample per plot of symptomatic tanoak (5 to 10 leaves and 2 to 3 twigs) and/or California bay (*Umbellularia californica*; 5 to 10 leaves), to test for the presence of *P. ramorum* via polymerase chain reaction (PCR); methods are described in Hayden et al. (2006) and results are presented and discussed in the full version of this paper (Ramage et al. 2011). Regeneration and canopy cover data were collected during the summer of 2010. Counts of all seedlings (< 1.37 m height), basal sprouts (< 1.37 m height), saplings (seed or sprout origin stems $\geq$ 1.37 m height and < 3 cm DBH), and juvenile trees (3to 10 cm DBH) were conducted for all tree species in two randomly selected quadrants per plot (for example, NW and SE). Canopy cover was measured with a spherical densiometer at five points per plot (plot center and 3 m in each cardinal direction) and values were averaged.

To limit any potentially confounding effects of tanoak abundance, all plots with less than the median basal area (BA) of total tanoak (living and dead trees combined, calculated with the randomly located plots only; 14.4 m^2 per ha) were excluded from all analyses. Using this dataset (n = 16 plots), we tested the effects of tanoak mortality on canopy cover and tree regeneration with generalized linear models. For each response variable, we fit models in which either dead tanoak BA (in 2008) or

the number of dead tanoak stems (in 2008) was specified as the sole predictor variable (with and without squared terms). Response variables, data for all of which were collected in 2010, consisted of canopy cover and all measures of regeneration (seedlings, basal sprouts, saplings, and juvenile trees) for several species groups (all species combined, all non-tanoak species, non-tanoak hardwoods, and non-redwood conifers), as well as each tree species individually: tanoak, redwood, pacific madrone (*Arbutus menziesii*), California bay, bigleaf maple (*Acer macrophyllum*), Douglas-fir (*Pseudotsuga menziesii*), and California nutmeg (*Torreya californica)*.

To determine which species are beginning to replace tanoak, we examined tanoak mortality and species-specific regeneration patterns in areas heavily impacted by SOD (plots with $\geq$ 300 dead stems per ha and/or $\geq$ 15 m^2 dead BA per ha), termed "severe" plots (n=8), and we present relevant data for these plots. We also re-executed all of the models described above using only the "severe" plots. Finally, we identified three plots in which the total density of non-tanoak seedlings (in 2010) was less than the density of dead tanoak stems (in 2008), termed "regen-deficient" plots. Additional analytical details are provided in the full version of this paper (Ramage et al. 2011).

Results

Effects of tanoak mortality on canopy cover and regeneration

Canopy cover in 2010 was significantly affected by tanoak mortality in 2008 (BA: $p < 0.0001$; stems: $p = 0.0014$). Squared terms were not significant in either model; predicted values are curved because of the logit transformation prior to model fitting and subsequent back-transformation prior to plotting (*fig. 1*). In the plots with the greatest mortality, canopy cover was below 60 percent, while canopy cover was generally above 90 percent in plots with little or no mortality.

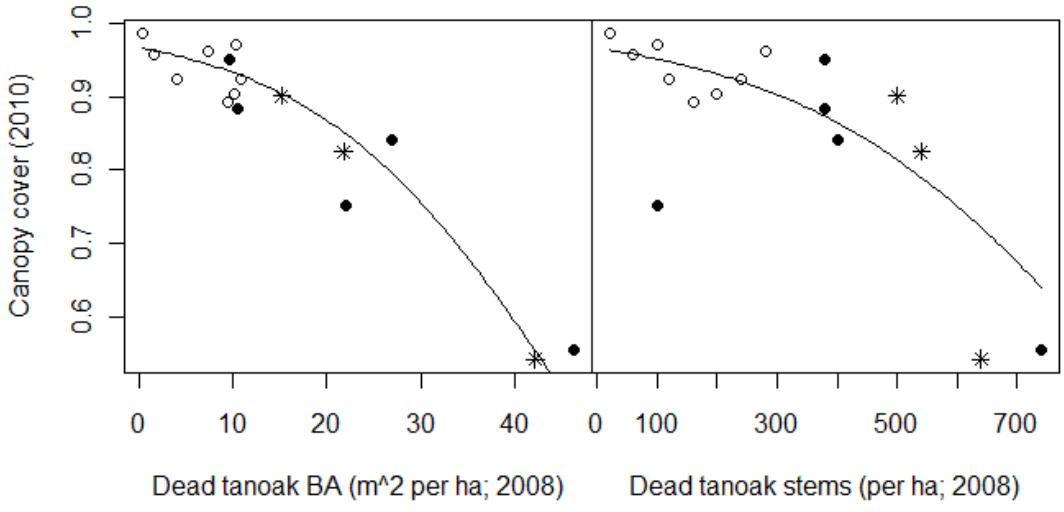

Figure 1—Canopy cover (2010) as a function of tanoak mortality (2008). Solid circles and asterisks were deemed severely impacted ("severe" plots); asterisks indicate "regen-deficient" plots; open circles represent all other plots (i.e., not "severe" or "regen-deficient").

Dead tanoak BA (in 2008) did not affect the density of tanoak seedlings, basal sprouts, or juvenile trees (in 2010), but the density of tanoak saplings was positively related to dead tanoak BA ($p = 0.0105$). The density of dead tanoak stems (in 2008) did not affect the density of tanoak seedlings, basal sprouts, or saplings (in 2010), but the density of tanoak juvenile trees was negatively related to dead tanoak stems ($p = 0.0007$). Squared terms were not significant in either model; predicted values are curved because of the log transformation prior to model fitting and subsequent back-transformation prior to plotting.

Tanoak mortality (in 2008) was entirely unrelated to regeneration (in 2010) of all species other than tanoak, including redwood. This was true across both tanoak mortality metrics (BA and stem counts) and all regeneration categories (seedlings, basal sprouts, saplings, and juvenile trees), and regardless of whether each species was analyzed individually or pooled into functional groups (non-tanoak hardwoods, non-redwood conifers). Similarly, tanoak mortality was unrelated to total regeneration (all species including tanoak and redwood), as well as all non-tanoak species. We also re-executed all these analyses using only the eight "severe" plots; all results were qualitatively identical, except for the relationship between tanoak juvenile trees and dead tanoak stems, which was insignificant in the "severe" analysis.

Severely impacted areas: regeneration and mortality

In the eight "severe" plots (those with $\geq$ 300 dead stems and/or $\geq$ 15 m^2 dead BA, per ha), the regeneration stratum was generally dominated by tanoak and redwood, but seedlings of other species were present at higher levels in certain areas. The median density of tanoak seedlings, basal sprouts, saplings, and juvenile trees was 1960, 4600, 180, and 0 per ha, respectively. Corresponding values for redwood were 1380, 1500, 100, and 40. Redwood accounted for 100 percent of non-tanoak basal sprouts, saplings, and juvenile trees, and the majority of non-tanoak seedlings in most plots (median non-tanoak seedlings = 1740; median redwood seedlings = 1380). Douglas-fir seedlings occurred in three of the eight severely impacted plots and exceeded densities of 3500 per ha in two of these plots. Pacific madrone and California bay seedlings were each present in four severely impacted plots, but their densities never exceeded 600 and 160 per ha, respectively. Seedlings of California nutmeg and bigleaf maple each occurred in only one plot, at densities of 240 and 40 per ha, respectively. Complete regeneration data for all "severe" plots are provided in *Table 1* of the full version of this paper (Ramage et al. 2011).

In these plots, the median amount of dead tanoak (absolute value and percent of total), in terms of BA and stem counts, was 21.9 (m^2 per ha; 66.4 percent) and 450 (per ha; 68.0 percent), respectively. As quantified by percent dead, the most severely impacted plot exhibited mortality exceeding 90 percent, in terms of both stem counts and BA. When quantified with absolute mortality, the most severely impacted plot contained 46.4 m^2 dead BA per ha and 740 dead stems per ha. The median amount of dead tanoak that was *broken/fallen* (bole broken at a diameter of $\geq$ 5 cm), in terms of BA and stem counts, was 15.8 (75.2 percent of dead) and 270 (79.1 percent of dead), respectively.

Three plots (which we refer to as "regen-deficient") exhibited a total density of non-tanoak seedlings that was less than the density of dead tanoak stems. Regeneration in these plots was consistently dominated by tanoak and redwood, but numbers were highly variable between plots and regeneration categories. With regard to other tree species, no basal sprouts, saplings, or juvenile trees were present in any plot, and seedlings were very uncommon. Seedlings of California bay (160 per ha) and bigleaf maple (40 per ha), the only other species present, occurred in only one plot each, both at densities insufficient to replace the number of tanoak trees that had already died by 2008. Densities of redwood basal sprouts, saplings, and juvenile trees, as well as tanoak regeneration (all categories combined), exceeded densities of dead tanoak stems in most (in the case of redwood) or all (in the case of tanoak) plots, but it is unlikely that these sources of regeneration will be able to fully re-occupy mortality gaps; this statement is justified in the discussion.

In the three "regen-deficient" plots, canopy cover was highly variable (54.2, 82.5, and 90.1), as was dead tanoak BA (42.2, 21.8, and 15.2 m^2 per ha). The density of dead tanoak stems was more consistent (640, 540, and 500 per ha), as was the percent of total tanoak that was dead, whether quantified by BA (93.0, 95.6, and 82.6 percent) or stem counts (86.5, 93.1, and 80.6 percent). The percent of dead tanoak that had broken/fallen was also consistently high, whether quantified by BA (81.5, 91.7, and 76.3 percent) or stem counts (78.1, 88.9, and 80.0 percent), suggesting that much of this mortality occurred well before our 2008 measurements.

Discussion

Broad regeneration patterns

Despite a significant reduction in canopy cover, our data suggest that SOD-impacted redwood forests in our study area are not exhibiting a regenerative response to tanoak mortality; tree regeneration was abundant in some mortality gaps, but regeneration levels were generally equivalent in severely impacted areas and relatively unaffected areas. These results were consistent across all mortality metrics, regeneration categories, and species (with the exception of tanoak saplings, which increased with dead tanoak basal area, and tanoak juvenile trees, which exhibited a negative relationship with dead tanoak stem density). Redwood and tanoak dominated the regeneration stratum in heavily diseased areas, and throughout the entire study area, while regeneration of other tree species was present only in isolated patches, and typically in very low densities.

Cobb et al. (2010) suggested that California bay may benefit more than any other tree species from SOD-induced tanoak mortality in redwood forests, because of similarities in growth form and size between tanoak and California bay, as well as positive feedbacks between inoculum loads and the abundance of California bay (which supports the most prolific sporulation of any host, but is not killed by *P. ramorum*; Davidson et al. 2008). However, at our study site, current regeneration patterns do not support this hypothesis. Although California bay seedlings occurred in 50 percent of "severe" plots, their densities were uniformly low (never exceeding 160 per ha, with a median of 20), and California bay basal sprouts, saplings, and young trees were entirely absent from all "severe" plots. In contrast, redwood regeneration occurred in all of these plots, and redwood seedlings alone had a median density of 1380 per ha (69 times that of California bay), suggesting that redwood is

currently much better poised to claim the space previously held by tanoak. Regeneration patterns may differ in other affected areas (for example, stands with a greater abundance of mature California bay), but given that no previous studies have comprehensively examined tree regeneration in SOD-impacted redwood forests, researchers and land managers should consider the possibility that our findings will be applicable beyond our study area.

Deficiencies in regeneration

Although broad patterns indicate that regeneration is sufficient to replace dead tanoaks, we have identified some patches (1/20 ha in size) in which the density of dead tanoak stems exceeded the density of non-tanoak seedlings. Tanoak regeneration and other forms of redwood regeneration (basal sprouts, saplings, and juvenile trees) were abundant in some of these plots, but we have deemed these plots deficient in regeneration for the following reasons: (a) *P. ramorum* is established in all SOD-impacted areas and tanoak regeneration is thus unlikely to survive to maturity (Cobb et al. 2010), and (b) redwood basal sprouts will not be able to fully re-occupy large mortality gaps because these sprouts necessarily emerge at the base of existing redwood trees, which tend to exist in dense discrete clumps in second-growth redwood-tanoak forest (Douhovnikoff et al. 2004, Ramage and O'Hara 2010), and generally exhibit a strong vertical growth habit. The same rationale applies in large part to redwood saplings and young trees, many of which were associated with established trees and most likely of sprout origin. While additional regeneration will almost certainly appear in the future, an insufficient passage of time does not appear to fully explain the paucity of regeneration in some mortality gaps, or the corresponding absence of a regenerative response to tanoak mortality throughout the study area; in the full version of this paper (Ramage et al. 2011), we provide a justification for this assertion, as well as a thorough examination of mechanisms that may be inhibiting recruitment in SOD-impacted areas.

Tanoak regeneration patterns

The positive relationship between tanoak saplings and tanoak mortality (dead basal area) probably reflects: (a) increased growth rates of advance regeneration in mortality gaps, and/or (b) the initial tendency of tanoaks that are top-killed by SOD, as well as tanoaks that are infected but still living, to sprout vigorously (Cobb et al. 2010, Ramage et al. 2010). The absence of a relationship between tanoak mortality and tanoak seedlings or basal sprouts may: (a) indicate a balance between disease-induced recruitment and mortality within these regeneration classes, and/or (b) reflect the fact that tanoak seedlings and sprouts (which are extremely shade tolerant; Burns and Honkala 1990) are often abundant in healthy stands. The strong negative relationship between tanoak juvenile stems and tanoak mortality (dead stems) suggests that individuals in this size class (3 to 10 cm DBH) are suffering high rates of SOD-induced mortality and/or not recruiting in diseased areas.

Conclusions and management considerations

SOD may ultimately create a *niche opportunity* (an opportunity for an absent or uncommon species to invade or increase in abundance; sensu Shea and Chesson 2002), but we have not discovered evidence that this phenomenon is occurring in the redwood forests of our study area. Rather, tree species other than redwood and tanoak

have made only small and highly variable incursions into mortality gaps, and some areas appear to lack sufficient regeneration for full re-occupancy of growing space, demonstrating that the future composition of SOD-impacted redwood forests is still far from certain. The ultimate ability of potential tanoak replacement species to co-exist with redwood in areas previously dominated by tanoak may only be apparent if and when such species are able to recruit in high numbers; at present, dispersal and recruitment limitation (both of which may be highly stochastic) are likely the dominant community assembly processes, but as these species begin to actively compete in areas previously occupied by tanoak, deterministic niche-related processes may become more important. For instance, tanoak develops a deep taproot (Burns and Honkala 1990), a characteristic that likely helps it to co-exist with redwood (which does not develop a taproot; Burns and Honkala 1990); this divergence in root morphology suggests that other deeply rooted tree species may be best equipped to compete with redwood in the absence of tanoak.

Numerous long-term impacts may result from SOD-induced tanoak decline (for example, trophic cascades resulting from the loss of tanoak acorns, reduced *resistance* and/or *resilience* in the face of future threats; sensu Suding et al. 2004). Land managers who wish to minimize the threat of such impacts should consider the intentional establishment of other native 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. 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). Furthermore, because SOD-induced tanoak mortality gaps are a novel occurrence, and novel ecosystems are likely to present unfamiliar and unforeseen challenges (Hobbs et al. 2006), it is prudent to assume that successful plantings (or other mitigation actions) may require considerable experimentation. As an alternative approach, managers may opt to actively maintain the open nature of these sites, so that if and when SOD-resistant tanoak genotypes are discovered, these genotypes can be readily reintroduced into areas where tanoak previously dominated.

Acknowledgments

We thank Save the Redwoods League for providing funding for this research and the University of California-Berkeley Forest Pathology Laboratory for molecular testing for *Phytophthora ramorum* in our study plots. In addition, we thank the Marin Municipal Water District for allowing this study to be conducted on their land, and two anonymous reviewers for valuable feedback. Finally, we are very grateful for the excellent work of our dedicated field assistants (Ben Ewing, Karla Martinez, and the many others who donated their time).

References

Burns, R.M.; Honkala, B.H., technical coordinators. 1990. **Silvics of North America: 1. Conifers; 2. Hardwoods.** Agriculture Handbook 654. Washington, DC: U.S. Department of Agriculture. 673 p.

Cobb, R.C; Meentemeyer, R.K.; Rizzo, D.M. 2010. **Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility.** Ecology 91: 327-333.

Courtney, S.P.; Blakesley, J.A.; Bigley, R.E.; Cody, M.L.; Dumbacher, J.P.; Fleischer, R.C.; Franklin, A.B.; Franklin, J.F.; Gutiérrez, R.J.; Marzluff, J.M.; Sztukowski, L. 2004. **Scientific evaluation of the status of the northern spotted owl [online]. Final report.** Sustainable Ecosystems Institute, Portland, Oregon. http://www.sei.org/owl/finalreport/finalreport.htm [accessed 19 May 2010].

Davidson, J.M.; Patterson, H.A.; Rizzo, D.M. 2008. **Sources of inoculum for *Phytophthora ramorum* in a redwood forest.** Phytopathology 98: 860-866.

Douhovnikoff, V.; Cheng, A.M.; Dodd, R.S. 2004. **Incidence, size, and spatial structure of clones in second-growth stands of coast redwood, *Sequoia sempervirens* (Cuppressaceae).** American Journal of Botany 91: 1140-1146.

Ellison, A.M.; Bank, M.S.; Clinton, B.D.; Colburn, E.A.; Elliot, K.; Ford, C.R.; Foster, D.R.; Kloeppel, B.D.; Knoepp, J.D.; Lovett, G.M.; Mohan, J.; Orwig, D.A.; Rodenhouse, N.L.; Sobczak, W.V.; Stinson, K.A.; Stone, J.K.; Swan, C.M.; Thompson, J.; von Holle, B.; Webster, J.R. 2005. **Loss of foundation species: consequences for the structure and dynamics of forested ecosystems.** Frontiers in Ecology and the Environment 3: 479-486.

Grubb, P.J. 1977. **The maintenance of species-richness in plant communities: the importance of the regeneration niche.** Biological Reviews 52: 107-145.

Hayden, K.; Ivors, K.; Wilkinson, C.; Garbelotto, M. 2006. **TaqMan chemistry for *Phytophthora ramorum* detection and quantification, with a comparison of diagnostic methods**. Phytopathology 96: 846-854.

Hobbs, R.J.; Arico, S.; Aronson, J.; Baron, J.S.; Bridgewater, P.; Cramer, V.A.; Epstein, P.R.; Ewel, J.J.; Klink, C.A.; Lugo, A.E.; Norton, D.; Ojima, D.; Richardson, D.M.; Sanderson, E.W.; Valladares, F.; Vila, M.; Zamora, R.; Zobel, M. 2006. **Novel ecosystems: theoretical and management aspects of the new ecological world order.** Global Ecology and Biogeography 15: 1-7.

Hunter, J.C.; Parker, V.T.; 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.; Jensen, C.E.; Rizzo, D.M. 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.; Kelly, M.; Storer, A.J.; Svihra, P.; Standiford, R.B. 2010. **Responses of oaks and tanoaks to the sudden oak death pathogen after 8 years of monitoring in two coastal California forests.** Forest Ecology and Management 259: 2248–2255.

Meentemeyer, R.; Rizzo, D.M.; Mark, W.; Lotz, E. 2004**. Mapping the risk of establishment and spread of sudden oak death in California.** Forest Ecology and Management 200: 195-214.

Noss, R.F., editor. 2000. **The redwood forest: history, ecology and conservation of the coast redwood.** Washington, DC: Island Press. 366 p.

O'Hara, K.L.; Berrill, J. 2010. **Dynamics of coast redwood sprout clump development in variable light environments.** Journal of Forest Research 15: 131-139.

O'Hara, K.L.; Stancioiu, P.T.; Spencer, M.A. 2007. **Understory stump sprout development under variable canopy density and leaf area in coast redwood.** Forest Ecology and Management 244: 76-85.

Oliver, C.D.; Larson, B.C. 1996. **Forest stand dynamics.** New York: John Wiley & Sons, Inc. 520 p.

Pickett, S.T.A.; White, P.S. 1985. **The ecology of natural disturbance and patch dynamics.** San Diego: Academic Press. 472 p.

Ramage, B.S.; O'Hara, K.L.; Forrestel, A.B. 2011. **Forest transformation resulting from an exotic pathogen: regeneration and tanoak mortality in coast redwood stands affected by sudden oak death.** Canadian Journal of Forest Research 41: 763-772.

Ramage, B.S.; Forrestel, A.B.; Moritz, M.A.; O'Hara, K.L. 2010. **Long term monitoring of the ecological impacts of sudden oak death in Point Reyes National Seashore: 2007-2009.** National Park Service Report. http://nature.berkeley.edu/~bsramage/Ramage_et_al_2010--SOD_at_Pt_Reyes.pdf [accessed 20 August 2010].

Ramage, B.S.; O'Hara, K.L. 2010. **Sudden oak death-induced tanoak mortality in coast redwood forests: current and predicted impacts to stand structure.** Forests 1: 114-130.

Rizzo, D.M.; Garbelotto, M.; 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.

Rizzo, D.M.; Garbelotto, M.; Davidson, J.M.; Slaughter, G.W.; Koike, S.T. 2002. ***Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and *Lithocarpus densiflorus* in California.** Plant Disease 86: 205-214.

Rost, J.; Pons, P.; Bas, J. 2009. **Can salvage logging affect seed dispersal by birds into burned forests?** Acta Oecologica 35: 763-768.

Shea, K.; Chesson, P. 2002. **Community ecology theory as a framework for biological invasions.** Trends in Ecology and Evolution 17: 170-176.

Smith, D.M.; Larson, B.C.; Kelty, M.J.; Ashton, P.M.S. 1997. **The practice of silviculture: applied forest ecology.** New York: John Wiley & Sons, Inc. 560 p.

Suding, K.N.; Gross, K.L.; Houseman, G.R. 2004. **Alternative states and positive feedbacks in restoration ecology.** Trends in Ecology and Evolution 19: 46-53.

Thompson, J.N.; Reichman, O.J.; Morin, P.J.; Polis, G.A.; Power, M.E.; Sterner, R.W.; Couch, C.A.; Gough, L.; Holt, R.; Hooper, D.U.; Keesing, F.; Lovell, C.R.; Milne, B.T.; Molles, M.C.; Roberts, D.W.; Strauss, S.Y. 2001. **Frontiers of ecology.** BioScience 51: 15-24.

Waring, K.M.; O'Hara, K.L. 2008. **Redwood/tanoak stand development and response to tanoak mortality caused by *Phytophthora ramorum*.** Forest Ecology and Management 255: 2650-2658.