

An emergent disease causes directional changes in forest species composition in coastal California

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Abstract. Non-native forest pathogens can cause dramatic and long-lasting changes to the composition of forests, and these changes may have cascading impacts on community interactions and ecosystem functioning. *Phytophthora ramorum*, the causal agent of the emergent forest disease sudden oak death (SOD), has a wide host range, but mortality is concentrated in a few dominant tree species of coastal forests in California and Oregon. We examined interactions between *P. ramorum* and its hosts in redwood and mixed evergreen forest types over an 80,000 ha area in the Big Sur ecoregion of central California, an area that constitutes the southernmost range of the pathogen and includes forest stands on the advancing front of pathogen invasion. We established a network of 280 long-term forest monitoring plots to understand how host composition and forest structure facilitated pathogen invasion, and whether selective mortality from SOD has led to shifts in community composition. Infested and uninfested sites differed significantly in host composition due to both historical trends and disease impacts. A reconstruction of pre-disease forest composition showed that stands that eventually became infested with the pathogen tended to be more mature with larger stems than stands that remained pathogen-free, supporting the hypothesis of aerial dispersal by the pathogen across the landscape followed by local understory spread. The change in species composition in uninfested areas was minimal over the study period, while infested stands had large changes in composition, correlated with the loss of tanoak (*Notholithocarpus densiflorus*), signaling the potential for SOD to dramatically change coastal forests through selective removal of a dominant host. Forest diversity plays an important role in pathogen establishment and spread, and is in turn changed by pathogen impacts. Asymmetric competency among host species means that impacts of *P. ramorum* on forest diversity are shaped by the combination and dominance of hosts present in a stand.

Key words: Big Sur, California; diversity-disease risk; emerging infectious disease; mixed evergreen forests; pathogen-mediated competition; *Phytophthora ramorum*; redwood forests; sudden oak death; tanoak.

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INTRODUCTION

Non-native forest pathogens continue to establish in North American forests at a steady rate despite intense regulation and quarantine efforts

to prevent their introduction and establishment (Aukema et al. 2010). These invaders lead to significant ecological and economic costs (Pimentel et al. 2000, Holmes et al. 2009). The ecological effects of non-native pathogens can have a long-

lasting legacy, especially when a pathogen causes mortality to abundant tree species or species that play a foundational role in ecosystem functioning (Gilbert 2002, Ellison et al. 2005). The pathogens with the most potential to cause such transformative changes are those that are virulent, often host-specific, and cause mortality to dominant or unique host species (Lovett et al. 2006). The impacts of these pathogens may include shifts in species composition and cascading effects on ecosystem functioning and foodweb dynamics when species with key roles in the ecosystem are removed by the pathogen (Lovett et al. 2006). These changes may create novel ecosystems or interactions among species.

A recent review of pathogens and plant communities (Mordecai 2011) placed their interactions into a theory of species coexistence developed by Chesson (2000) where forces that promote plant species diversity may be stabilizing or equalizing (*sensu* Chesson 2000), and both types of forces may operate at different relative strengths. In this framework, pathogens that have stabilizing effects are ones whose impacts increase with host abundance, so that a host is more strongly limited by its pathogen than are its competitors, and plant coexistence is promoted. When pathogen impacts are negatively correlated with host abundance, the effect is destabilization via a feedback loop where rare species are more strongly impacted by pathogens, perhaps through pathogen spillover among shared hosts, than are common species. Equalizing forces promote diversity by reducing fitness differences among species, which may otherwise lead to competitive exclusion. The impacts of pathogens on plant community diversity thus depend on the degree to which pathogens are mediating competition by influencing fitness differences among species or are having host-specific impacts that negatively or positively correlate with host abundance to stabilize or destabilize species coexistence (Mordecai 2011).

Eviner and Likens (2008) outlined a framework for examining pathogens as drivers of ecosystem change based on the ways in which pathogens affect community composition. This framework combines principles from disturbance ecology with an understanding that even subtle shifts in species composition can have large effects on ecosystem functioning. Important considerations

for predicting the impact of pathogens on ecosystems include: the nature of the pathogen impact on the host; what life stages, functional groups or proportion of individuals are affected; and the temporal and spatial dimensions of the impact.

Neither Mordecai (2011) nor Eviner and Likens (2008) explicitly differentiated between native and exotic pathogens in considering pathogen impacts on communities, even though the most extreme examples of ecosystem or community change discussed in both reviews come from exotic, recently introduced pathogens causing widespread disturbance. There may be stark differences in the role that native vs. exotic pathogens play in regulating forest diversity (Rizzo 2005, Desprez-Loustau et al. 2007). Endemic pathogens that have co-evolved with their hosts may structure forest composition and mediate plant interactions and coexistence in any of the ways just described with varying levels of host resistance (Hansen and Goheen 2000). The importance of endemic pathogens in structuring forests can change, however, when any one of the components necessary for disease (pathogen, host, or environment) are altered by management decisions, such that suitable conditions for pathogen establishment and spread may become more widespread (Slaughter and Rizzo 1999, Hansen and Goheen 2000).

Non-native pathogens, in contrast, face susceptible hosts that have had little opportunity to evolve resistance, and there are several examples of how virulent, novel pathogens have caused transformative changes to forests, both in North America and worldwide. In North American forests, chestnut blight and beech bark disease are notable examples of forest diseases that have resulted in region-scale declines of susceptible species (Paillet 2002, Garnas et al. 2011). Other pathogens such as *Ophiostoma novo-ulmi* and *Phytophthora cinnamomi* have altered host populations on multiple continents (Weste and Marks 1987, Brasier and Buck 2001). White Pine blister rust caused by *Cronartium ribicola*, affects multiple species of pine in Asia, Europe, eastern North America and western North America (Geils et al. 2010). In recent years, southeastern forests have experienced mortality of redbay (*Persea borbonia*) by a newly described pathogen (*Raffaelea lauricola*) that causes laurel wilt and is vectored by an

exotic ambrosia beetle (*Xyleborus glabratus*) (Fraedrich et al. 2008, Harrington et al. 2008). These examples confirm theoretical expectations that exotic pathogens have the potential to be largely destabilizing to diversity, causing changes to species composition and associated ecosystem function (Eviner and Likens 2008, Mordecai 2011). Because forest diversity may mediate disease risk (Haas et al. 2011), understanding both the forest characteristics that facilitate invasion and the compositional and ecosystem changes caused by pathogens is important to predicting future patterns of disease establishment and spread.

Sudden oak death (SOD) is an emerging forest disease causing high levels of mortality in coastal forests of the western United States. Extensive tree mortality was first noticed in the mid-1990s in the San Francisco Bay Area, and *Phytophthora ramorum*, a non-native generalist pathogen, was identified as the causal agent in 2000 (Rizzo et al. 2002). Today the pathogen is known to occur across a range of 640 km in California, and in several disjunct zones in northwestern California and southwestern Oregon. Although the pathogen is a generalist, infecting over 100 species from more than 40 genera, mortality is concentrated in only a few species in the family Fagaceae. Potentially millions of trees have died throughout the pathogen's range since its discovery (Meentemeyer et al. 2008a), and as the pathogen becomes established in northern California, where host biomass and densities are relatively high, the cumulative biomass and number of hosts killed by the pathogen is likely to increase substantially (Lamsal et al. 2011, Meentemeyer et al. 2011).

The full ecological impact of historical analogues to SOD, such as the invasion of chestnut blight, is largely unknown, as quantitative baseline data on the ecology of the forests before pathogen arrival often do not exist (Loo 2009). In the case of *P. ramorum*'s recent invasion and spread, the patchy distribution of disease on the landscape permits comparison of infested and uninfested stands in close proximity at a landscape scale to understand the relationship between host abundance and pathogen establishment and impacts. The biology of the pathogen leads to several expectations about this relationship. Pathogen establishment requires the

presence of sporulating hosts (few species are competent hosts for pathogen sporulation), and the likelihood of *P. ramorum* establishment should increase with increasing abundance of these hosts at regional and local scales (Maloney et al. 2005, Meentemeyer et al. 2008a). Due to primarily short-distance dispersal (Davidson et al. 2005, Hansen et al. 2008, Mascheretti et al. 2008, 2009), the pathogen should initially be patchily distributed across the landscape, and stands of similar host composition may differ in infection status simply because of differences in exposure to dispersing spores. Asymmetric pathogen impacts among species (high mortality of selected species and no impact to other hosts) should lead to shifts in the species composition.

Here we use a large network of long-term forest monitoring plots across two forest types and wide topographical and climatic gradients in Big Sur, California (Fig. 1) to examine these expectations. This is an area with a gradient of SOD impacts and a high risk of predicted SOD spread (Meentemeyer et al. 2004, Meentemeyer et al. 2011). Specifically we examine several questions: (1) What forest characteristics favored establishment of *P. ramorum* across a diverse and heterogeneous landscape? (2) How does *P. ramorum* change forest composition, and are these changes potentially destabilizing to forest diversity? (3) What do the trajectories of compositional change in infested plots predict for future pathogen impacts? Linked together, these questions represent the most comprehensive study of *P. ramorum* impacts on shifts in plant community structure over a region-wide landscape.

METHODS

Pathosystem

Phytophthora ramorum is a generalist oomycete pathogen that is regulated by state, federal, and international agencies because of its broad host range, destructive impacts, and documented spread through the horticultural trade. In addition to the west coast of the US, the pathogen has established in the United Kingdom, where it spreads from infected rhododendron to beech in woodlands and causes widespread mortality in plantations of Japanese larch (Brasier and Webber 2010). The pathogen infects over 100 species of trees, shrubs, herbs, or ferns, but species differ

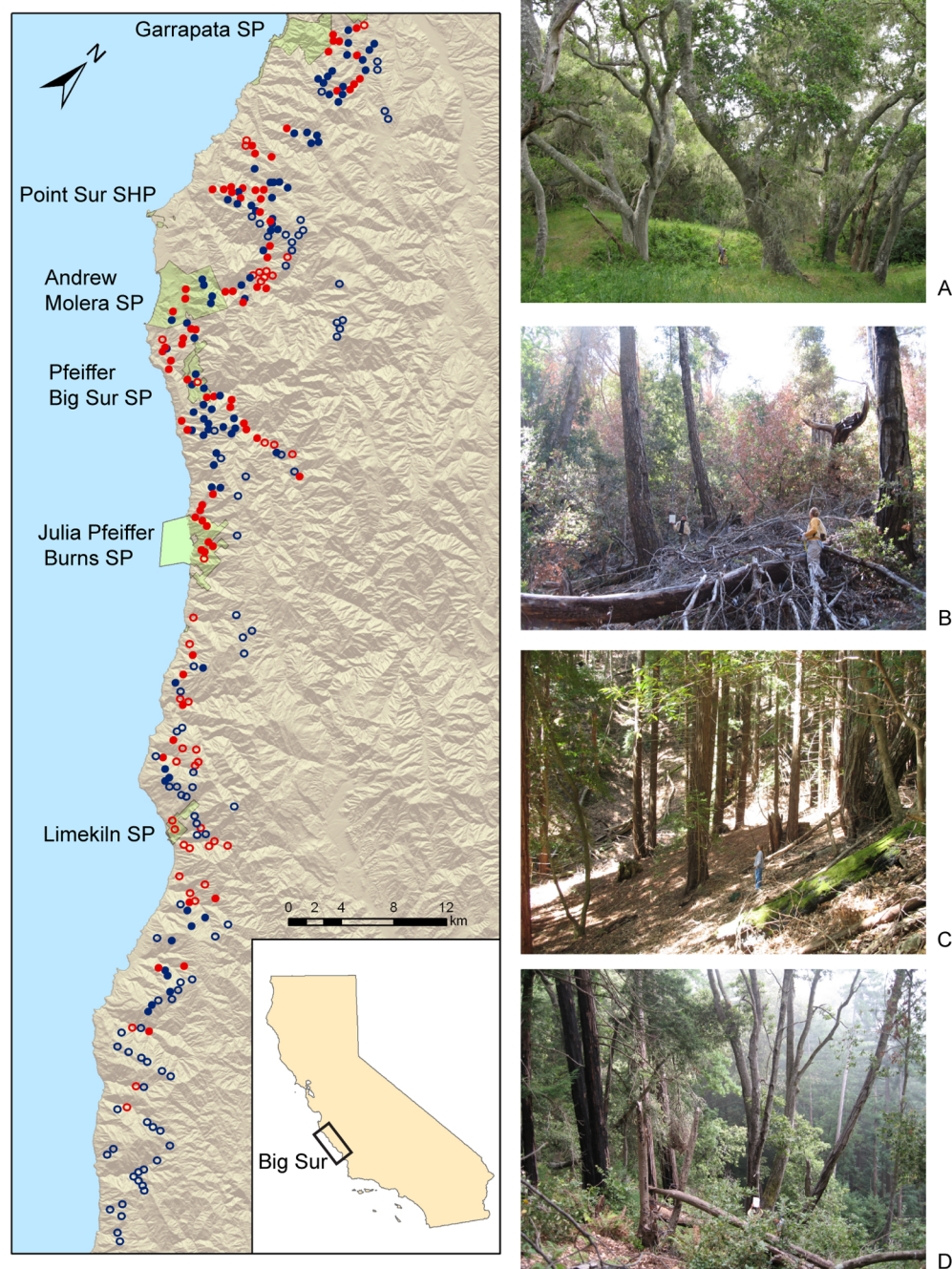


Fig. 1. Big Sur sudden oak death forest monitoring network. The 280 500-m² plots are distributed across ~80,000 ha on the western slopes of the Santa Lucia mountains in central California, spanning mixed evergreen and redwood forests (blue and red dots, respectively) and areas with and without *Phytophthora ramorum* (solid dots = infested, open dots = uninfested). Left panel: map of the study region with plot locations indicated. Right panel: typical host habitats; (A) uninfested mixed evergreen forest dominated by tanoak; (B) infested mixed evergreen forest with accumulated tanoak woody debris; (C) uninfested redwood forest; (D) infested redwood forest with dead tanoaks.

in their susceptibility, type of infection, and ability to support pathogen sporulation and spread, leading to asymmetries in host-pathogen interactions among host species (Rizzo et al. 2005). The range of *P. ramorum* spans several habitat types in the coastal forests of California and Oregon, including oak woodlands and conifer-tanoak forests (Rizzo and Garbelotto 2003, Lamsal et al. 2011). Most infections in natural systems are non-lethal foliar or twig infections. Infections that kill tanoak (*Notholithocarpus densiflorus*) and some oak species (*Quercus* spp.) in California occur through formation of bleeding cankers on the trunk of the tree that may eventually girdle the tree and/or allow for invasion by secondary pathogens or insects. The main sources of inoculum from competent hosts in California forests are leaf infections on California bay laurel (*Umbellularia californica*) or leaf and twig infections on tanoak; pathogen sporulation is most active during warm, wet springs (Davidson et al. 2005, Davidson et al. 2008, Davidson et al. 2011). Most dispersal of the pathogen occurs over distances <100 m, however rare long-range dispersal events are thought to happen up to 3 km (Davidson et al. 2005, Rizzo et al. 2005, Hansen et al. 2008, Mascheretti et al. 2008, Mascheretti et al. 2009, Meentemeyer et al. 2011).

Study region

The Big Sur ecoregion comprises the western slopes of the Santa Lucia Mountains in Monterey County, California. The region is very dissected topographically with elevational changes of over 1500 m in a distance of only 5 km from the coast. There are strong precipitation and climatic gradients from north to south and from the coast inland, supporting a heterogeneous assemblage of habitats and species (Davis and Borchert 2006). The vegetation is a diverse mosaic of forests, shrublands and grasslands. There are coastal sage scrub, chaparral, redwood forests, oak woodlands, and coastal prairies all within close proximity (Davis et al. 2010). Mixed evergreen forest dominated by hardwoods is the most common forest type, and redwood (*Sequoia sempervirens*) forests are generally limited to riparian areas. There are small patches of mixed-conifer forests (including Ponderosa Pine, *Pinus ponderosa*) at higher elevations. Almost 85%

of the area is under protection by the state or federal government or local conservation organizations; much of the forest is uninterrupted by human development.

Big Sur is one of the earliest sites of *P. ramorum* introduction, and also among the most heavily impacted areas in California (Rizzo and Garbelotto 2003, Maloney et al. 2005, Mascheretti et al. 2008, Mascheretti et al. 2009, Meentemeyer et al. 2008c). The spread of the pathogen across the landscape has been patchy, however, resulting in forest stands that span a range of disease impacts or durations since pathogen establishment, as well as numerous stands where *P. ramorum* has not been detected (Fig. 1; Meentemeyer et al. 2008c, Davis et al. 2010, Metz et al. 2011). Detailed stand-level data from plots across this gradient permit a rigorous examination of how host composition may impact disease establishment and impacts both in areas that have long been invaded by the pathogen and areas on the advancing front of the pathogen's range expansion.

Plot monitoring network and forest species composition

We established a network of 280 500-m² (0.05 ha) forest monitoring plots across approximately 80,000 ha in the Big Sur region during 2006–2007 to track the distribution and impacts of *P. ramorum* and to understand the interactions among the pathogen, its hosts and the environment (Fig. 1). Using a GIS, potential plot locations were assigned randomly across the Big Sur ecoregion so as to be more than 300 m distant from each other and to have multiple replicates across combinations of several stratifying factors. These included: two forest types where the pathogen and its hosts are found (redwood and mixed evergreen), a gradient of tree mortality as estimated using aerial imagery of standing dead trees in 2005 (Meentemeyer et al. 2008c), among public/private lands, across a range of fire histories, and across watersheds. Not all of the potential locations were accessible—either physically or because of landowner permission—leading to the final establishment of 280 plots between April 2006 and October 2007. The average distance to the nearest neighboring plot is 650 m $\pm$ 20 m (SE), with only 9 plots having a nearest neighbor slightly less than 300

Table 1. Dominance, density, and importance value (ranges 0–1) for live stems of six dominant species in (A) 83 infested and 83 uninfested mixed evergreen stands, and (B) 70 infested and 44 uninfested redwood stands. See Table S1 for the abundance of other species occurring in the plots. Values are means (SD) across plots.

Species	<i>P. ramorum</i> present			<i>P. ramorum</i> absent		
	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value
A) Mixed evergreen						
Tanoak	5.0 (11.9)	268.9 (456.5)	0.17 (0.27)	3.3 (9.2)	266.3 (909.3)	0.10 (0.23)
Bay	6.3 (8.7)	415.9 (523.8)	0.28 (0.29)	3.0 (6.9)	436.9 (846.4)	0.16 (0.23)
Redwood	0.0 (0.2)	3.9 (22.0)	0.00 (0.01)	0.0 (0.0)	0.2 (2.2)	0.00 (0.00)
Madrone	6.1 (12.0)	160.2 (431.0)	0.12 (0.18)	4.1 (9.6)	268.4 (640.2)	0.11 (0.21)
Coast live oak	11.4 (17.5)	88.9 (142.7)	0.20 (0.27)	13.7 (17.0)	252.0 (632.3)	0.30 (0.35)
Shreve's oak	6.4 (13.4)	217.8 (743.5)	0.13 (0.23)	3.2 (7.2)	301.2 (695.7)	0.10 (0.20)
B) Redwood						
Tanoak	11.3 (14.6)	860.6 (902.2)	0.33 (0.27)	8.0 (12.6)	907.7 (1301.4)	0.20 (0.22)
Bay	4.2 (8.5)	258.6 (682.9)	0.10 (0.15)	1.7 (3.8)	209.5 (498.6)	0.07 (0.1)
Redwood	82.8 (73.8)	486.9 (481.1)	0.52 (0.23)	111.5 (66.1)	1006.8 (933.7)	0.68 (0.2)
Madrone	0.3 (1.4)	8.3 (41.1)	0.01 (0.04)	0.4 (1.6)	27.3 (93.6)	0.01 (0.02)
Coast live oak	0.2 (1.0)	5.4 (28.7)	0.00 (0.01)	0.6 (2.6)	9.5 (32.7)	0.01 (0.03)
Shreve's oak	1.4 (5.5)	39.4 (124.5)	0.03 (0.10)	0.7 (3.1)	58.6 (288.3)	0.01 (0.05)

m. Plot locations may be viewed on GoogleEarth via SODMAP (<http://www.sodmap.org>).

Within every plot, each standing live or dead stem ≥ 1 cm diameter at breast height (DBH) was mapped, identified to species, measured for stem diameter, and checked for a variety of pathogen symptoms (standardized methods of Maloney et al. 2005, Cobb et al. 2010, Haas et al. 2011, Metz et al. 2011). The designation of the mixed evergreen or redwood forest type—mapped from aerial imagery—was ground-truthed during plot establishment based on canopies dominated by hardwoods or redwoods, respectively. A 2009 census of tree mortality and recruitment in these plots was used to confirm trajectories of pathogen impacts inferred from the 2006–2007 plot establishment data. We include data from the 31 plots (of 115 censused in 2009) that had not burned in major wildfires in 2008, so as to isolate the impacts of the pathogen from those of fire. Other sympatric species of *Phytophthora* can also cause disease symptoms that appear similar to those of *P. ramorum*, so diagnosis of pathogen presence in each survey was confirmed via

culturing of symptomatic tissue or DNA sequencing following the methods of Davidson et al. (2005, 2008).

Most species in these forests produce basal sprouts that lead to multi-stemmed trees. Our measurements are taken on each stem (≥ 1 cm DBH) and summed across stems or trees (multiple stems joined at ground level are considered the same tree) to obtain plot-level values as our units of replication. We used plot-level importance value (IV) as the measure of species abundance in order to account for both frequency and dominance of basal area as species in this forest vary greatly in growth form and canopy position (Table 1):

$$IV = 0.5 \times (\text{relative density} + \text{relative dominance}). \quad (1)$$

Relative density is the number of a species' stems in a plot divided by the total number of stems, and relative dominance is the species' summed basal area in a plot divided by the total plot basal area. IV ranges from 0 to 1 for each species in a

plot.

We calculated the IV of each species in each plot at three time points. First, we created a conservative estimate of reconstructed pre-disease forest composition by summing all live and dead stems standing at plot establishment in 2006–2007, thereby undoing mortality that had occurred within recent years. This is a conservative estimate because it does not include mortality where trees may have died and already fallen to the ground. Second, we used live stems only to quantify the current composition of the stands (current defined as the state in 2006–2007 at the establishment of the plot network, including disease impacts as of that year). Finally, for plots that were re-sampled in 2009, we calculated the IV for each species using live stems in 2009 (using DBHs measured at plot establishment) and newly recruited stems, which were measured in 2009. For all three time points, we divided the plots into two groups based on their infestation status at the 2006–2007 baseline survey.

Statistical analyses

Our analyses proceeded in three broad steps. First, we asked how the abundance of major hosts varied in the reconstructed pre-disease stand composition to understand how species composition may have influenced disease establishment. Second, we examined the trajectories of species compositional change from the reconstructed state to the current state in 2006–2007 to understand the impacts of the disease, comparing plots where *P. ramorum* was present and absent. Third, we confirmed the generality of these trajectories by testing whether a subset of plots censused in 2009 experienced continued directional change in species' abundances.

Reconstructed pre-disease stand differences.—We compared the abundance of dominant species between plots that, by 2006–2007, had become infested with *P. ramorum* and those that remained pathogen-free. Here, disease status in 2006–2007 is the grouping variable for the plots, but the desired biological inference is whether there were host compositional differences conducive to the pathogen that led to this disease status. We calculated an effect size for reconstructed basal area (summing live and dead stems) to understand the magnitude of the difference in species abundances relative to background levels in

uninfested plots:

$$\text{Host Abundance Effect Size} = \frac{(\text{infested} - \text{uninfested})}{\text{uninfested}} \quad (2)$$

Above, infested or uninfested indicate the mean across plots of the reconstructed, pre-disease basal area of dominant species for infested or uninfested plots. We used the SE of the group means and error propagation to calculate a SE for the effect size. When the 95% CI for the effect size (calculated using $1.96 \times \text{SE}$) did not include 0, the variable of interest was significantly greater (or lower) in infested plots than uninfested.

Disease impacts as of 2006–2007 and trajectories of change.—We conducted a factorial analysis of variance using distance matrices of species composition upon plot establishment (2006–2007) in all 280 plots (also called permutational or nonparametric MANOVA; McArdle and Anderson 2001). Pathogen status (infested vs. uninfested by *P. ramorum*) and forest type (redwood vs. mixed evergreen) were crossed factors in the model. This test asked whether current species composition (2006–2007) varied among the different combinations of forest type and pathogen status, and confirmed whether ground-truthed designations of habitat type were suitable for distinguishing forest types.

For each habitat type, we examined the trajectories of species compositional change from the reconstructed pre-disease state to 2006–2007 using non-metric multidimensional scaling (NMDS) and Bray-Curtis dissimilarity matrices. We included points representing both the current and reconstructed stand composition for each plot in the ordination (and 2009 values, where appropriate) so that we could track compositional changes in multivariate species space (Laurance et al. 2006, Feeley et al. 2011). We overlaid pathogen infestation status, species abundances, and plot environmental characteristics onto the ordinations to interpret the relationship between species composition and pathogen establishment and impacts. We also used techniques from circular statistics to examine the hypothesis that vectors representing species compositional change between the reconstructed state and 2006–2007 would be large and in a non-random direction for infested stands compared to vectors

for uninfested stands. The details of the ordination methods and circular statistics are described in the supplementary information in the Appendix.

We compared the percent mortality of dominant species between infested and uninfested plots using a disease impacts effect size, calculated as above. Finally, we tested whether the distributions of stem DBHs differed between infested and uninfested stands in the current and reconstructed states using the Kolmogorov-Smirnov test of equality of distributions.

Continued compositional change to 2009.—The reconstructed pre-disease state and the current disease impacts were both assessed with a single time point of data—the baseline census in 2006–2007. To understand whether further observations of mortality and recruitment supported our inferences of pathogen impacts to species composition, we examined the change in IV between the reconstructed state and the 2009 census (for the 31 plots where these data were available) for the dominant species in each habitat. This analysis confirmed whether the trajectories observed in the ordination analyses were borne out in future field observations and included mortality and new stem recruitment occurring between plot establishment and 2009.

All analyses were conducted in the statistical programming language R (R Development Core Team 2009).

RESULTS

In total we measured over 12,000 multi-stemmed trees from 36 species, representing approximately 4,000 dead stems and 24,000 live stems. Eighty-three of the 166 mixed evergreen plots were infested with *P. ramorum*; 70 of 114 redwood plots were infested at the time of plot establishment (Fig. 1). There was great variability in the number of stems or basal area among species in plots (Appendix: Table A1). Six species were consistently more abundant than others, occurring in more than one third of the plots in either habitat type. These were bay laurel, coast live oak, tanoak, madrone (*Arbutus menziesii*), and Shreve's oak (*Quercus parvula* var. *shrevei*) in mixed evergreen forests and redwood, tanoak, and bay laurel in redwood forests (Table 1). Each of these species is considered to be a host for *P.*

ramorum though sporulation competency and susceptibility to lethal bole cankers varies among species.

The proportion of dead stems in a plot varied among the dominant species. Mortality for canker hosts (tanoak, coast live oak, and Shreve's oak) was greater in infested plots relative to background mortality levels in uninfested plots, and mortality differed by size class (Fig. 2). On average, a greater proportion of large stem diameter tanoaks died in infested plots compared to uninfested plots, but mortality in the smallest size classes ($1 \text{ cm} \leq \text{DBH} < 5 \text{ cm}$) did not differ with pathogen presence. Coast live oak mortality was higher in infested plots than uninfested plots for all but the largest stem diameter size classes. For Shreve's oak, mortality rate differences were most pronounced in an intermediate size class, with DBH between 5–10 cm. Bay, redwood and madrone, none of which are killed by SOD, showed qualitatively similar mortality rates between infested and uninfested plots (Fig. 2).

Reconstructed pre-disease stand differences

In both habitat types, the reconstructed, pre-disease composition of stands that eventually became infested with *P. ramorum* differed from stands that remained pathogen-free (Appendix: Figs. A1 and A2). In particular, the total basal area of sporulating hosts (tanoak and/or bay laurel) was significantly greater in infested plots than uninfested plots in both habitats (Fig. 3). Tanoak basal area in plots that became infested was approximately 75% and 150% higher on average (for redwood and mixed evergreen plots, respectively) than the background levels in plots that remained pathogen-free. Reconstructed bay laurel basal area observed in plots that became infested was >100% of the background level of bay laurel abundance in uninfested mixed evergreen stands. Plots where the pathogen eventually established also had significantly less redwood basal area than pathogen-free plots in redwood stands.

Disease impacts as of 2006–2007 and trajectories of change

Upon plot establishment in 2006–2007, the ground-truthed designation of redwood or mixed evergreen forest types corresponded well to differences in species composition in the plots.

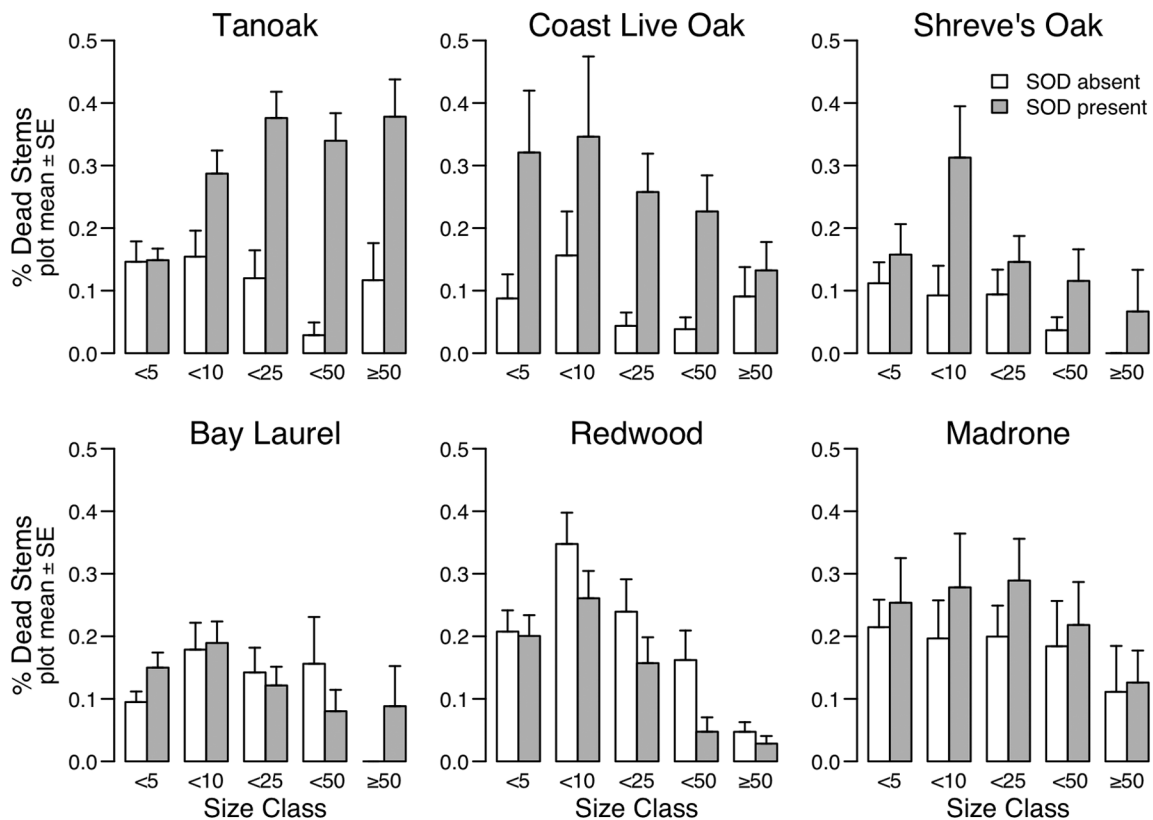


Fig. 2. Stem mortality for six dominant species. The number of dead stems is expressed as a percentage of total stems per species in each of five size classes (1 to <5 cm, 5 to <10 cm, 10 to <25 cm, 25 to <50 cm, and $\geq$ 50 cm DBH). Bars average across both mixed evergreen and redwood forest plots. Host species in the top row suffer lethal canker infections from SOD, and those in the bottom row have non-lethal infections.

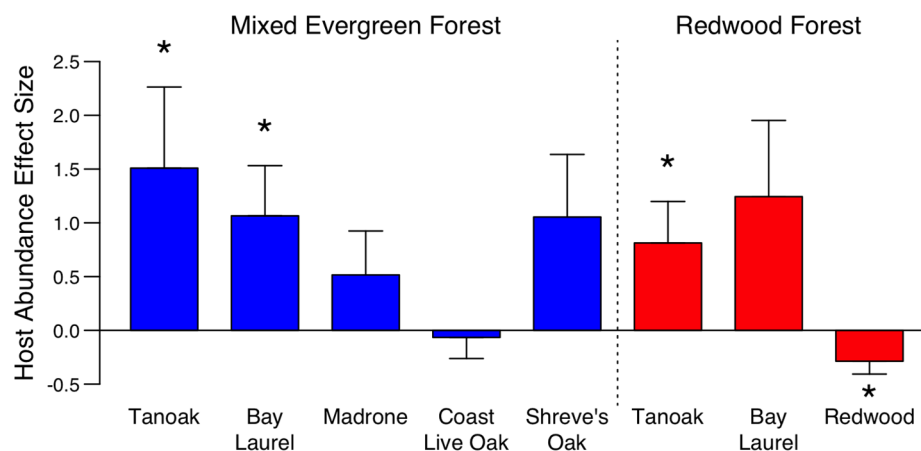


Fig. 3. SOD established in stands with more sporulating hosts. Host abundance effect size for the difference in total basal area (live and dead standing stems) between infested and uninfested plots, relative to levels in uninfested plots. Bars are plot means with SE bars calculated using error propagation. An effect size of 0 means there is no difference, on average, between infested and uninfested stands. An asterisk indicates the 95% CI does not overlap zero.

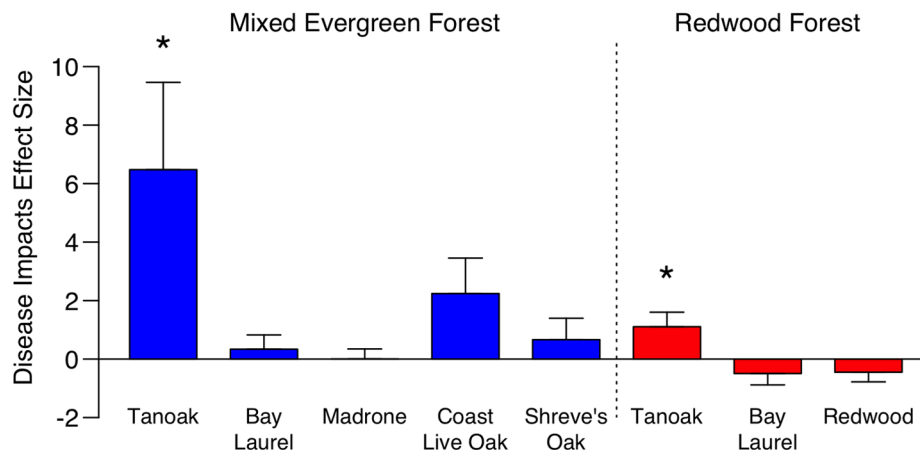


Fig. 4. Tanoak and oak mortality rates are higher in infested stands. Disease impacts effect size for the difference in percent dead basal area between infested and uninfested plots, relative to levels in uninfested plots. Bars, error bars, and asterisks are as in Fig. 3.

The species most correlated with the difference in the forest types were redwood, which dominated the redwood forest type, and madrone or coast live oak in the canopy of the mixed evergreen forests. The key sporulating hosts (bay laurel and tanoak) were important components of both habitat types (Table 1). Both pathogen presence and forest type (but not their interaction; $P = 0.06$) were significant correlates of species composition with the greatest compositional differences across the entire plot network occurring between habitat types (forest type $F = 125.88$, $P = 0.0001$, explains 30.5% of model variance; pathogen $F = 8.64$, $P = 0.0001$, explains 2.1% of variance).

In ordinations of plot species composition in each forest type, the axes were correlated with the abundance of several of the dominant species, and compositional change over time was large and directional in plots where *P. ramorum* established and began killing hosts (see Appendix). These multivariate results were corroborated by comparisons of disease impacts on the mortality of individual species between infested and uninfested states. Tanoak mortality was elevated 674% and 103% above the background mortality in uninfested plots in mixed evergreen and redwood stands, respectively (Fig. 4), while all other species did not differ significantly in percent mortality between infested and uninfested stands. Coast live oak also had elevated mortality in infested stands relative to

uninfested (Fig. 4), but the increase was variable enough that the 95% CI bound 0.

The DBH distributions of all six dominant species were skewed towards larger stems in infested plots (Appendix: Figs. A3 and A4), with significant differences in the distributions between infested and uninfested plots for both the current and reconstructed state. For tanoak only, there was also a significant difference in size distributions between the reconstructed and current states in infested plots (changing from larger stems pre-disease to smaller stems as the pathogen caused mortality, especially in larger stems), but not in uninfested plots. No other species differed in their size distribution between the reconstructed and current states.

Continued compositional change to 2009

P. ramorum caused directional and substantial compositional changes to forest stands (Fig. 5; Appendix: Figs. A1 and A2). Between the reconstructed state before establishment of the plot network and the 2009 census of stem mortality and recruitment, tanoak experienced a significant decrease in importance value in both habitat types in infested stands, and redwood increased its IV in the redwood plots (Fig. 5). Shreve's oak also had a significant decline in IV in infested mixed evergreen stands. Species' IV did not change in uninfested stands across this time period.

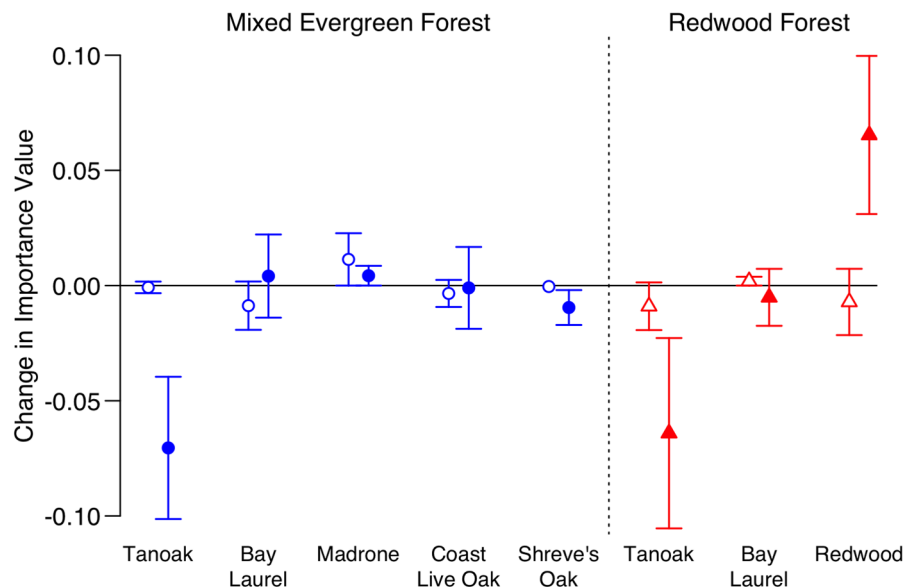


Fig. 5. Tanoak importance value declined from pre-disease state to 2009. The average ($\pm$ SE) change in importance value in 31 plots recensused in 2009 for infested (solid symbols) and uninfested (open symbols) stands. Change was measured in each plot as the species' IV in 2009 minus the IV in the reconstructed, pre-disease state.

DISCUSSION

We examined the distribution of an emergent forest pathogen and its hosts along the central California coast to understand how *P. ramorum* affects species composition in forests across a large (~80,000 ha) heterogeneous landscape. In each of two habitat types, the species composition of infested stands differed significantly from that of uninfested stands. These differences were due to both historical trends (i.e., the reconstructed stands differed in diversity and structure between currently infested and uninfested stands) and disease impacts (i.e., infested stands experienced greatly elevated mortality). Over a relatively short time period, infested plots had greater shifts in species composition than uninfested plots from the pre-disease state (before approximately 1995–2000) to the state in 2006–2007 (upon establishment of the monitoring network) or 2009. The changes in infested plots were also directional—correlated with loss of tanoak—whereas the changes in uninfested plots were much smaller as well as random with respect to species abundance. The continued trajectory of these changes was confirmed in

2009, where infested stands had a significant decrease in tanoak importance in both forest types, and infested redwood stands had an increase in redwood importance.

Conditions favoring pathogen invasion

Our reconstructions of stand composition indicated that areas that eventually became infested had more and larger individuals of the competent sporulating hosts, tanoak and bay laurel. This is consistent with results observed elsewhere tying the likelihood of *P. ramorum* establishment and spread to the abundance of bay laurel and tanoak at several scales (Maloney et al. 2005, Meentemeyer et al. 2008a, Swiecki and Bernhardt 2010, Meentemeyer et al. 2011, Haas et al. 2011). Interestingly, all six of the focal, dominant species had larger stems in infested plots than uninfested plots, suggesting that the pathogen has tended to establish in mature stands. This may explain the observation found here (for mixed evergreen forests; Appendix: Fig. A1) and throughout California by Moritz and Odion (2005) that infested areas have experienced fewer fires since 1950 than areas without the disease. Dominant or late seral trees have the

opportunity to grow larger as stands mature following fire events. If the pathogen is more likely to establish in mature stands, time since fire would be expected to correlate with pathogen presence, though it is unlikely this correlation is due to any direct impact of the fire on susceptibility to *P. ramorum*. Rather, fire history and pathogen presence correlate in similar ways to vegetation size structure and species composition. Stands differ in their exposure to inoculum, even with identical stand composition or fire history. Fire suppression can allow wooded areas to expand or increase connectivity and create pathogen-conducive microclimates (Condeso and Meentemeyer 2007, Meentemeyer et al. 2008b). Additionally, fire suppression tends to occur near inhabited areas, and these are also the areas with the greatest opportunity for introduction or spread of the pathogen through transport of infected plant material or soils. Alternatively, all of the dominant species examined here sort along temperature and moisture gradients (Davis et al. 2010), and the pathogen may respond to the same micro-climate or productivity gradients that permit trees, particularly individuals of competent hosts, to grow very large.

Our results suggest a possible progression of disease dynamics in Big Sur. *P. ramorum* appeared to have been introduced to the region in the mid-1990s (Maloney et al. 2005), likely on ornamental plants with secondary spread across the heterogeneous landscape via other mechanisms (Mascheretti et al. 2008, 2009). Not all areas would have had exposure to an inoculum source, and stands with very similar composition (Appendix: Figs. A1 and A2) may have differed in infestation status by 2006–2007 as a result of patchy dispersal across the landscape, not differences in host abundance. As the pathogen has spread, however, it became established in more mature stands, with larger stems, and especially in stands that had significantly greater abundance of sporulating hosts (bay laurel or tanoak) than other stands (Fig. 3). In redwood forests, uninfested stands also had a larger component of redwoods, which may physically intercept aerially dispersing spores with their large and dense canopies and act functionally as a dead-end host for inoculum spread in those stands.

Taken together, these observations suggest the heterogeneity of SOD impacts across the Big Sur

landscape (Meentemeyer et al. 2008c, Metz et al. 2011) may have arisen from three processes occurring in sequence at varying times since introduction of the pathogen to the region: (1) long-distance aerial dispersal of the pathogen to new stands, (2) infections initiating in the canopy and spreading short distances to understory trees, and (3) local progression of disease impacts through time. Hansen et al. (2008) proposed that spores formed in the upper crowns of trees are more easily lofted and dispersed across the canopy by large wind and rain events. In Oregon, where eradication efforts lead to the removal of all neighboring trees in infested areas, new infections are found on foliage and twigs in tree canopies before stem cankers develop, and understory trees (e.g., rhododendron or huckleberry) are only found infected when in the immediate neighborhood of trees that have developed cankers (Hansen et al. 2008).

Big Sur is the southernmost region in coastal California that has been invaded by *P. ramorum*. Our plot network is large enough to encompass some of the first areas where *P. ramorum* became established in Big Sur, as well as including areas of recent pathogen range expansion to the north and south (Fig. 1). In contrast, previous ecological studies of SOD mortality have primarily focused on small areas with limited sampling replication that have long been invaded by *P. ramorum* and where uninfested and infested forests do not exist for comparison (e.g., McPherson et al. 2005, Waring and O'Hara 2008, Brown and Allen-Diaz 2009, McPherson et al. 2010, Ramage and O'Hara 2010, Swiecki and Bernhardt 2010, Ramage et al. 2012). This region-wide plot network has allowed us to encompass each of the three disease processes proposed above as well as a diversity of plant assemblages that host *P. ramorum*.

Implications of the pathogen for forest diversity and function

A number of studies have demonstrated a “dilution effect” of diversity, whereby increased community diversity lessens some aspect of disease prevalence or risk (Keessing et al. 2006, 2010) with most examples coming from zoonotic diseases. In Big Sur, Haas et al. (2011) found a negative association between forest diversity and prevalence of SOD infection symptoms. Our

results suggest two possible mechanisms for this putative protective effect of diversity on disease risk. First, the increased abundance of large, non-competent hosts observed in uninfested sites could increase interception of aerially dispersed spores and prevent pathogen establishment. Epidemiological models have demonstrated that isolated tanoaks in a diverse neighborhood of noncompetent hosts may escape infection because encounters with spores are reduced (Cobb et al. 2012b). Second, because species richness can vary with stem density due to sampling effects, it is possible that the mature stands with fewer, larger stems that were more conducive to pathogen invasion also tended towards lower diversity, potentially contributing to the negative diversity-disease correlation observed by Haas et al. (2011). If this aspect of stand composition is related to the time since previous fires as discussed above, then there exists the possibility that wildfire can mediate the disease-diversity relationship. An exciting future area of research is to investigate how this novel biotic disturbance interacts with endemic disturbances to impact forest diversity and future disease risk (Metz et al. 2011, Beh et al. 2012, Meentemeyer et al. 2012).

The species- and size-selective mortality caused by *P. ramorum* (Fig. 2) over a large, diverse, and heterogeneous area suggests that this pathogen can cause significant changes to forest composition and ecosystem processes (Eviner and Likens 2008), even without complete extinction of tanoak. The preferential loss of large, canopy tanoaks to SOD-caused mortality followed by prolific re-sprouting of small stems is expected to retain tanoak as an understory component of these forests but changes both the structure and reproductive potential of trees in the stand, similar to the situation with the American chestnut in eastern North American forests (Paillet 2002). The species that will increase dominance following loss of tanoak do not share the same functional roles that tanoak plays in coastal forests, including, for example, provisioning of acorns as an important food source (Monahan and Koenig 2006) or unique leaf litter chemistry and contributions to nutrient cycling (R. C. Cobb and D. M. Rizzo, *unpublished data*). All these characteristics of SOD's impact on forest stands indicate large changes to ecosystem functioning in the short- and long-term (Eviner

and Likens 2008).

Whether the forest composition changes we observed affect forest diversity through stabilizing or equalizing forces (sensu Chesson 2000 and Mordecai 2011) depends on the components of the system examined. Loss of oak species in the presence of sporulating host neighbors, independent of oak abundance, suggests an important role for pathogen spillover and priority effects in destabilizing coexistence of these oaks and their neighbors, as described by Mordecai (2011). Because *P. ramorum* has a wide host range across a very heterogeneous area, however, there are several other disease-host interactions that may be more important in understanding the impacts of *P. ramorum* on the diversity of coastal CA forests. Notably, our results demonstrate that *P. ramorum* had the earliest and strongest impacts in stands where tanoak was relatively much more dominant than in uninfested stands. High tanoak mortality from *P. ramorum* could then increase forest diversity by reducing fitness of this dominant, much as Mordecai (2011) suggests eastern forests were changed following Chestnut blight (though the true impact of chestnut blight on the diversity of these forests also depends on the pre-disease composition, which may not be well documented). Whether tanoak mortality ultimately results in equalizing fitness among tanoak and its neighbors depends both on the composition of the neighborhood and the relative fitness advantage held by tanoak in the absence of the disease. Results from our plot network demonstrate that the dominant species in these forest types occur in a variety of assemblages with different abundance distributions. In stands where bay laurel and tanoak are equally abundant, reduction of tanoak by SOD could increase bay laurel dominance through pathogen-mediated competition (Cobb et al. 2010), with perhaps little change in the relative abundance of other species. Alternatively, where tanoak is the sole sporulating host, forest diversity may be promoted in the presence of *P. ramorum* because tanoak is strongly regulated by the disease. Further feedbacks between host species and phenotypic diversification of *P. ramorum* may also affect ecological dynamics over the long-term (Kasuga et al. 2012).

Conclusions

The widespread and rapid mortality caused by *P. ramorum* is a disconcerting example of a non-native, virulent pathogen causing species-specific impacts to dominant trees with the potential to substantially transform the composition of forests across a large and diverse region. All our evidence at this time points to destabilization of diversity (*sensu* Chesson 2000, Mordecai 2011) by this pathogen, whether through density-independent spillover to susceptible hosts (e.g., coast live oak) or dramatic changes in fitness to a dominant host (e.g., tanoak). Although pathogen establishment, spread, and impacts are related to tanoak abundance, we found a non-random, directional change in species composition directly related to elevated, rapid mortality of tanoak, without regard to its initial abundance. We thus have no evidence at this time to support the existence of long-term stabilizing effects of *P. ramorum* through density-dependent regulation of tanoak, which provides a stark contrast to the ways in which native pathogens promote diversity through density-dependent, host-specific pathogen impacts (Mordecai 2011).

These compositional changes to the forest are dramatic, and have far-reaching effects on ecosystem functioning in coastal forests impacted by SOD (Eviner and Likens 2008). Species composition affects the prevalence of disease (Haas et al. 2011), pathogen survival during or re-invasion following wildfire (Beh et al. 2012), carbon cycling (Cobb et al. 2012a), among numerous other processes. All of these changes can have feedbacks that further alter species composition and the interactions between the pathogen and its hosts. The asymmetric competency and vulnerability among host species means that impacts of *P. ramorum* on forest diversity will lead to feedbacks between diversity and disease, shaped by the combination and dominance of hosts present in a stand.

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SUPPLEMENTAL MATERIAL

APPENDIX

ORDINATION METHODS

The 280 forest monitoring plots contained a diversity of tree assemblages. For each habitat type, we examined the trajectories of multivariate species compositional change from the reconstructed pre-disease state to 2006–2007 using non-metric multidimensional scaling (NMDS) and Bray-Curtis dissimilarity matrices. We performed multiple ordinations with random starts and assessed a scree plot of the stress values (a measure of rank-order correlation between Bray-Curtis distance and distance in ordination space) vs. dimensions of each ordination and chose the minimum dimensionality beyond which reductions in stress were minimal (McCune and Grace 2002). Based on this, we chose a 3-dimensional ordination for mixed evergreen plots ($n = 166$, stress = 13.93) and a 2-dimensional ordination for redwood plots ($n = 114$, stress = 7.83).

We overlaid the infestation status of each forest plot on the NMDS figures to visualize the potential relationship between species composition and pathogen presence; an ellipse encircling each group's mean and 95% CI about the mean is also plotted. We overlaid vectors representing the abundance (IV) of common species, when the IV was significantly correlated with the NMDS

space (these data were a subset of the species used to create the ordination, so a significant correlation is generally expected). Overlaid vectors represent the gradient of abundance for a species across ordination space, with the angle of the vector indicating the direction of most rapid change in IV, and the length of the vector corresponding to the strength of correlation of the species abundance with the ordination configuration. We similarly overlaid any environmental characteristics of the monitoring plot that were significantly correlated with the NMDS axes; these environmental variables included the location (UTM coordinates easting or northing), elevation, slope, or aspect of the plot and the number of fires occurring in a plot since 1950. Comparing these vectors to the direction of the compositional offset between infested and uninfested plots allowed an understanding of whether infested plots had, for example, more sporulating hosts or different environmental conditions than uninfested plots.

We used several techniques to understand potential mechanisms for the difference in species composition between infested and uninfested plots (see *Methods*). Using the ordinations, we examined the trajectory of a plot in multivariate species space from the reconstructed state to

the current state, with the hypothesis that infested stands will have changed more than uninfested plots, which have not experienced elevated and selective mortality from SOD. In particular we expected this change to be primarily a loss of tanoak, which we examined by plotting the contour surface of tanoak abundance onto the NMDS plots. We took the vectors connecting a plot's reconstructed state and current state and translated them to a common origin to compare the length and direction of the vectors for infested and uninfested plots (for the mixed evergreen ordination, this was done in all three 2-dimensional combinations of NMDS axes). We used a one-tailed *t*-test to compare the length of vectors between the two groups, hypothesizing the vectors to be longer for infested plots. We used techniques from circular statistics to examine the directionality of these changes. Rayleigh's test was employed to test the null that the vectors in each group were distributed uniformly on a circle, with the alternative hypothesis for the infested plots that the distribution was unimodal (Zar 1999) and centered on the direction of decreasing tanoak abundance (taken from the vectors of species abundance described above).

All analyses were conducted in the statistical programming language R (R Development Core Team 2009). The ordinations were made with the *metaMDS* routine in the R package *vegan* (Oksanen et al. 2010), which uses repeated random starts to converge on a stable solution and rotates the final solution to principal components so that variance is maximized along the first NMDS axis. The *envfit* and *ordisurf* routines in *vegan* were used to overlay species and environment vectors on the ordination. The R package *circular* was used for the circular statistics to analyze the vectors of compositional change (Agostinelli and Lund 2011).

ORDINATION RESULTS

In ordinations of plot species composition in each forest type, the axes were correlated with the abundance of several of the dominant species, and compositional change over time was large and directional in plots where *P. ramorum* established and began killing hosts. For mixed evergreen forests, separation of plots

along the first NMDS axis correlated strongly with the abundance of tanoak and coast live oak, and the second axis represented differences in dominance by bay laurel or madrone (Fig. A1A, B). The offset in the group means from uninfested to infested (represented by the 95% CI ellipses in Fig. A1A, B) occurred along axes 2 and 3. Along axis 2, this correlated with lower abundance of coast live oak and higher abundance of bay laurel and Shreve's oak, but was orthogonal to differences in tanoak abundance. Along axis 3, the species composition offsets between uninfested and infested sites correlated with an increase in tanoak importance value (Fig. A1). The vectors representing the move in ordination space of a plot from the reconstructed, pre-disease species composition to the current state in 2006–2007 were significantly longer for infested plots than uninfested plots in the mixed evergreen stands, indicating greater compositional change where the pathogen was present (for *t*-tests of vectors projected in NMDS axes 1 and 2, 1 and 3, or 2 and 3, all $P < 0.0001$; Fig. A1). For axes 1 and 2, the distribution of infested vectors was not uniform and was centered around the vector of decreasing tanoak abundance (Rayleigh's statistic = 0.22, $P = 0.002$); uninfested plots also had a non-uniform distribution (Rayleigh's statistic = 0.20, $P = 0.038$), but the mean angle was not related to tanoak abundance ($P = 0.78$). On NMDS axes 1 and 3, vectors of uninfested plots were uniformly distributed, and those for infested plots were significantly centered around decreasing tanoak abundance (Rayleigh's statistic = 0.17, $P = 0.038$). On NMDS axes 2 and 3, vectors of uninfested plots were non-uniformly distributed, but the mean angle was not related to tanoak abundance; vectors of infested plots were uniformly distributed.

In redwood plots, the first NMDS axis showed the separation among plots in the relative dominance of redwood vs. tanoak (Fig. A2). The second axis was correlated with the abundance of bay laurel and several secondary species (madrone, coast live oak, Shreve's oak) that occurred only infrequently in this habitat type. In redwood plots, the offset in group means from uninfested to infested plots directly corresponded to decreasing abundance of redwood and some increase in bay laurel abundance, but was

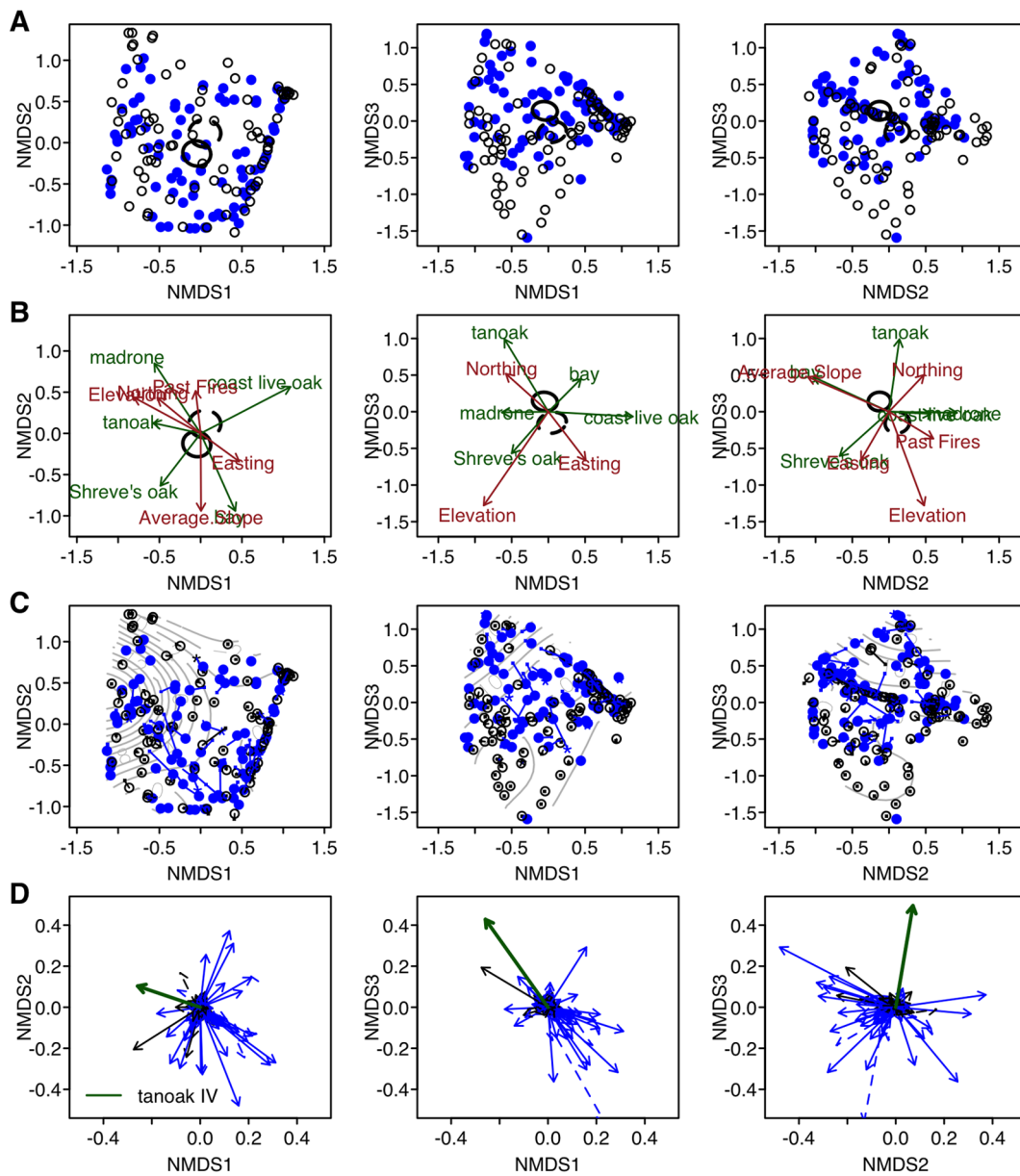


Fig. A1. Species composition of mixed evergreen plots. The importance values (IV) of 37 species were ordinated using NMDS with three dimensions; distances between points indicate similarity of community composition. For all combinations of the three NMDS axes there are: (A) Species composition of 166 mixed evergreen plots in 2006–2007 (open dots = uninfested, solid dots = infested). The 95% CI about the mean of the infested or uninfested plots is shown with a solid or dashed line, respectively. (B) Vectors indicating environmental characteristics (brown) and abundance of dominant species (green) that are significantly correlated with the ordination space with group centroids indicated as previously. (C) Trajectories of compositional change (blue = infested, black = uninfested) are indicated by segments that begin with the reconstructed stand composition (if all standing dead stems are considered alive) and end with the 2006–2007 stand composition (using only live standing stems) or the 2009 composition for 16 plots (6 uninfested, 10 infested). Gray contour lines indicate live basal area ($\text{m}^2 \text{ha}^{-1}$) of tanoak in plots in 2006–2007. (D) The trajectory vectors translated to a common origin for comparison of vector length and direction. Dotted lines indicate continued community change from 2006–2007 to 2009. The direction of increasing tanoak abundance is in green.

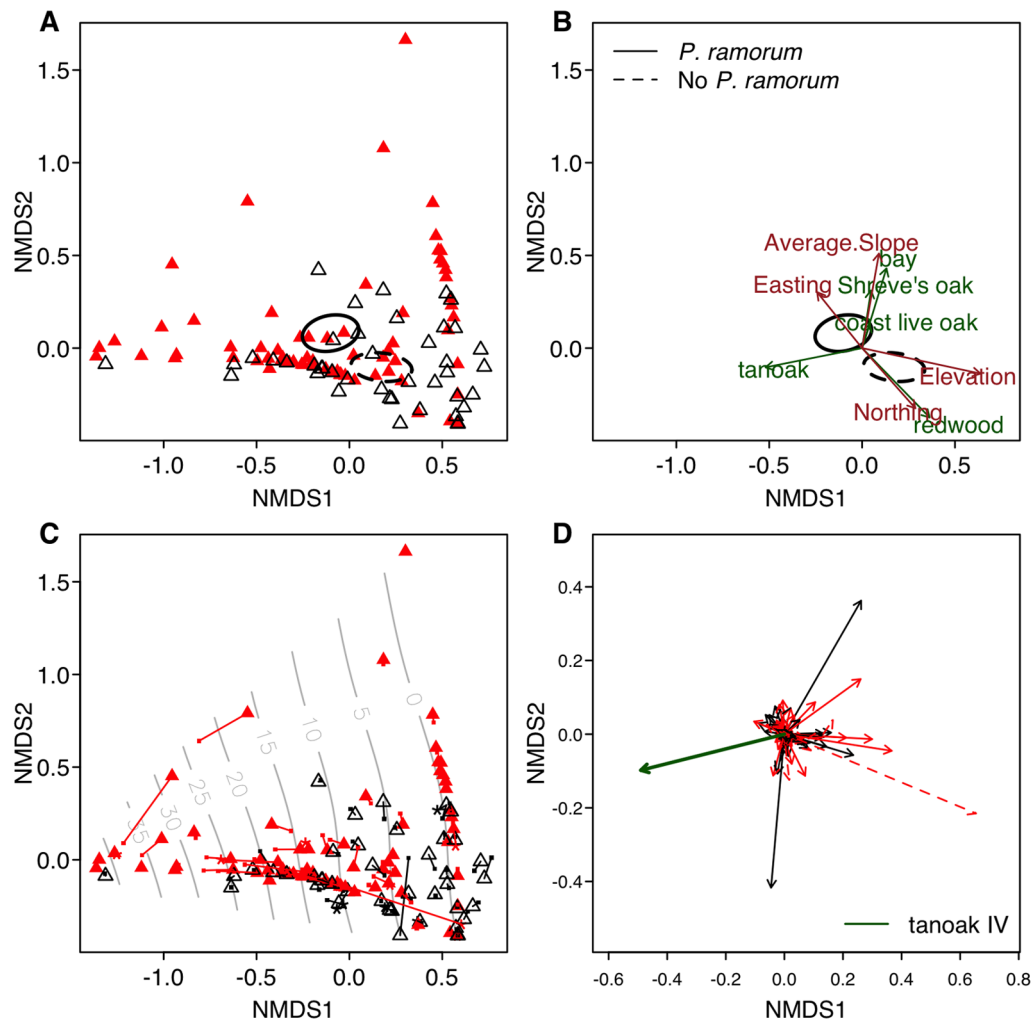


Fig. A2. Species composition of redwood plots. The importance values (IV) of 21 species were ordinated using NMDS with two dimensions; distances between points indicate similarity of community composition. Symbols are as in Fig. A1. (A) Species composition of 114 redwood plots. (B) Vectors indicating environmental characteristics and abundance of dominant species (green) that are significantly correlated with the ordination space. (C) Trajectories of compositional change (red = infested, black = uninfested), including change to 2009 for 15 plots (7 uninfested, 8 infested). (D) The trajectory vectors translated to a common origin for comparison of vector length and direction.

also mostly orthogonal to the abundance of tanoak (Fig. A2A, B). The vectors of compositional change from the reconstructed, pre-disease species composition to the current state in 2006–2007 were significantly longer for infested plots than uninfested plots in redwood stands, as in mixed evergreen ($P < 0.05$; Fig. A2). The angles of the vectors for both infested and uninfested plots were not statistically distinguishable from a

uniform distribution (infested, $P = 0.13$; uninfested, $P = 0.78$). However, for the subset of plots re-sampled in 2009, the vectors from the reconstructed state to the 2009 composition were not uniform in infested plots, centered around decreasing tanoak abundance ($P = 0.009$), while vectors in uninfested plots had a uniform angular distribution ($P = 0.90$).

Table A1. Dominance, density, and importance value (range 0–1) for live stems of species in infested and uninfested stands of (A) mixed evergreen and (B) redwood forests. Values are means (SD) across plots.

Species	<i>P. ramorum</i> present			<i>P. ramorum</i> absent		
	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value
A) Mixed evergreen						
Redwood	0	3.9	0	0	0.2	0
(<i>Sequoia sempervirens</i>)	(0.2)	(22)	(0.01)	(0)	(2.2)	(0)
Tanoak	5	268.9	0.17	3.3	266.3	0.1
(<i>Notholithocarpus densiflorus</i>)	(11.9)	(456.5)	(0.27)	(9.2)	(909.3)	(0.23)
Bay laurel	6.3	415.9	0.28	3	436.9	0.16
(<i>Umbellularia californica</i>)	(8.7)	(523.8)	(0.29)	(6.9)	(846.4)	(0.23)
Coast live oak	11.4	88.9	0.2	13.7	252	0.3
(<i>Quercus agrifolia</i>)	(17.5)	(142.7)	(0.27)	(17)	(632.3)	(0.35)
Shreve's oak	6.4	217.8	0.13	3.2	301.2	0.1
(<i>Quercus parvula</i> var. <i>shrevei</i>)	(13.4)	(743.5)	(0.23)	(7.2)	(695.7)	(0.2)
Madrone	6.1	160.2	0.12	4.1	268.4	0.11
(<i>Arbutus menziesii</i>)	(12)	(431)	(0.18)	(9.6)	(640.2)	(0.21)
Canyon live oak	3.3	30.8	0.04	5.1	364.3	0.12
(<i>Quercus chrysolepis</i>)	(12.9)	(74.3)	(0.15)	(16.7)	(856.3)	(0.23)
Poison oak	0	6.5	0	0	11.3	0
(<i>Toxicodendron diversilobum</i>)	(0)	(16)	(0.01)	(0)	(28)	(0.01)
Hairy ceanothus	0.1	20.5	0.01	0.1	22.2	0.01
(<i>Ceanothus oliganthus</i>)	(0.4)	(100.8)	(0.03)	(0.2)	(77.5)	(0.02)
Toyon	0.1	8.9	0.01	0	9.2	0
(<i>Heteromeles arbutifolia</i>)	(0.3)	(26.7)	(0.02)	(0.1)	(23)	(0.01)
Big leaf maple	0.4	8.2	0.01	0	0.5	0
(<i>Acer macrophyllum</i>)	(1.4)	(33.5)	(0.05)	(0.3)	(3.1)	(0)
Ponderosa pine	0.1	8.4	0	2	37.8	0.03
(<i>Pinus ponderosa</i>)	(0.3)	(41.8)	(0.01)	(7.9)	(183.1)	(0.09)
Douglas fir	0	4.3	0	0.9	5.3	0.01
(<i>Pseudotsuga menziesii</i>)	(0.1)	(39.5)	(0.02)	(4.4)	(23.8)	(0.06)
California coffeeberry	0	1	0	0	2.7	0
(<i>Rhamnus californica</i>)	(0)	(4.3)	(0)	(0)	(10.3)	(0.01)
Black oak	0.5	3.4	0.01	0.9	8	0.02
(<i>Quercus kelloggii</i>)	(3.2)	(21.6)	(0.04)	(4)	(29.3)	(0.09)
Coulter pine	0	0	0	0.4	11.8	0.01
(<i>Pinus coulteri</i>)	(0)	(0)	(0)	(1.4)	(48.1)	(0.04)
Honeysuckle	0	0.7	0	0	1.2	0
(<i>Lonicera hispidula</i>)	(0)	(3.8)	(0)	(0)	(4.8)	(0)
Blue-blossom	0	0	0	0	5.8	0
(<i>Ceanothus thyrsiflorus</i>)	(0)	(0)	(0)	(0.3)	(34.4)	(0.02)
B) Redwood						
Redwood	82.8	486.9	0.52	111.5	1006.8	0.68
(<i>Sequoia sempervirens</i>)	(73.8)	(481.1)	(0.23)	(66.1)	(933.7)	(0.2)
Tanoak	11.3	860.6	0.33	8	907.7	0.2
(<i>Notholithocarpus densiflorus</i>)	(14.6)	(902.2)	(0.27)	(12.6)	(1301.4)	(0.22)
Bay laurel	4.2	258.6	0.1	1.7	209.5	0.07
(<i>Umbellularia californica</i>)	(8.5)	(682.9)	(0.15)	(3.8)	(498.6)	(0.1)
Coast live oak	0.2	5.4	0.0	0.6	9.5	0.01
(<i>Quercus agrifolia</i>)	(1)	(28.7)	(0.01)	(2.6)	(32.7)	(0.03)
Shreve's oak	1.4	39.4	0.03	0.7	58.6	0.01
(<i>Quercus parvula</i> var. <i>shrevei</i>)	(5.5)	(124.5)	(0.1)	(3.1)	(288.3)	(0.05)
Madrone	0.3	8.3	0.01	0.4	27.3	0.01
(<i>Arbutus menziesii</i>)	(1.4)	(41.1)	(0.04)	(1.6)	(93.6)	(0.02)
Canyon live oak	0	0.3	0	0	1.8	0.0
(<i>Quercus chrysolepis</i>)		(2.4)			(12.1)	(0.01)
Poison oak	0	0.9	0	0	1.8	0
(<i>Toxicodendron diversilobum</i>)		(4.1)			(5.8)	
Hairy ceanothus	0	2.6	0	0.1	19.5	0.01
(<i>Ceanothus oliganthus</i>)		(17.3)		(0.2)	(60.3)	(0.02)
Toyon	0	0.6	0	0	0.5	0
(<i>Heteromeles arbutifolia</i>)		(3.4)			(3)	
Big leaf maple	0.1	5.1	0.0	0.4	10.5	0.01
(<i>Acer macrophyllum</i>)	(0.7)	(17.6)	(0.01)	(1.4)	(34.6)	(0.03)
Ponderosa pine	0	0	0	0	0	0
(<i>Pinus ponderosa</i>)						
Douglas fir	0	0	0	1.1	4.5	0.01

Table A1. Continued.

Species	<i>P. ramorum</i> present			<i>P. ramorum</i> absent		
	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value	Basal area (m ² ha ⁻¹)	Stems (ha ⁻¹)	Importance value
<i>(Pseudotsuga menziesii)</i>				(3.5)	(14.2)	(0.02)
California coffeeberry <i>(Rhamnus californica)</i>	0	0.3 (2.4)	0	0	0	0
Black oak <i>(Quercus kelloggii)</i>	0	0	0	0	0	0
Coulter pine <i>(Pinus coulteri)</i>	0	0	0	0	0	0
Honeysuckle <i>(Lonicera hispidula)</i>	0	0.9 (5.3)	0	0	0	0
Blue-blossom <i>(Ceanothus thyrsiflorus)</i>	0	0	0	0.0 (0.1)	7.3 (29.3)	0.0 (0.01)

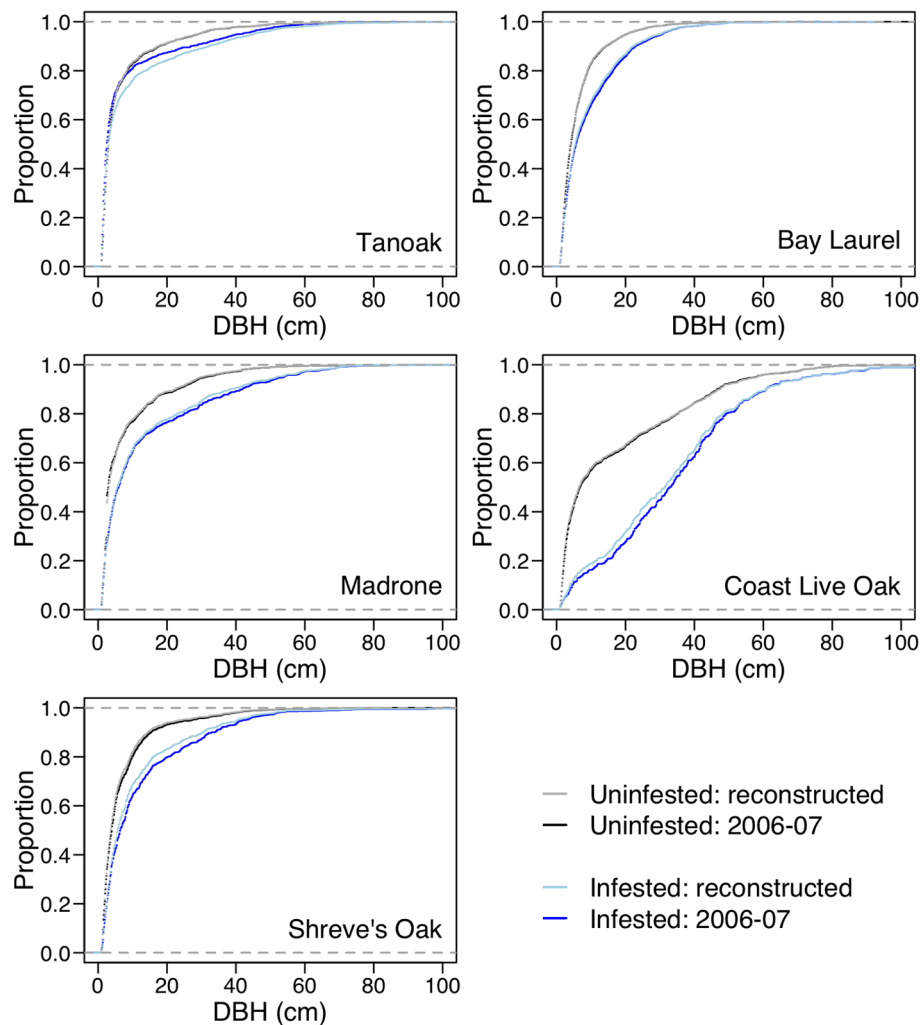


Fig. A3. Stem DBH distributions across infested and uninfested stands in mixed evergreen forests. The cumulative proportion of stems in increasing DBH size classes is shown for five dominant species in both the reconstructed and current states using all stems that occur in these plots.

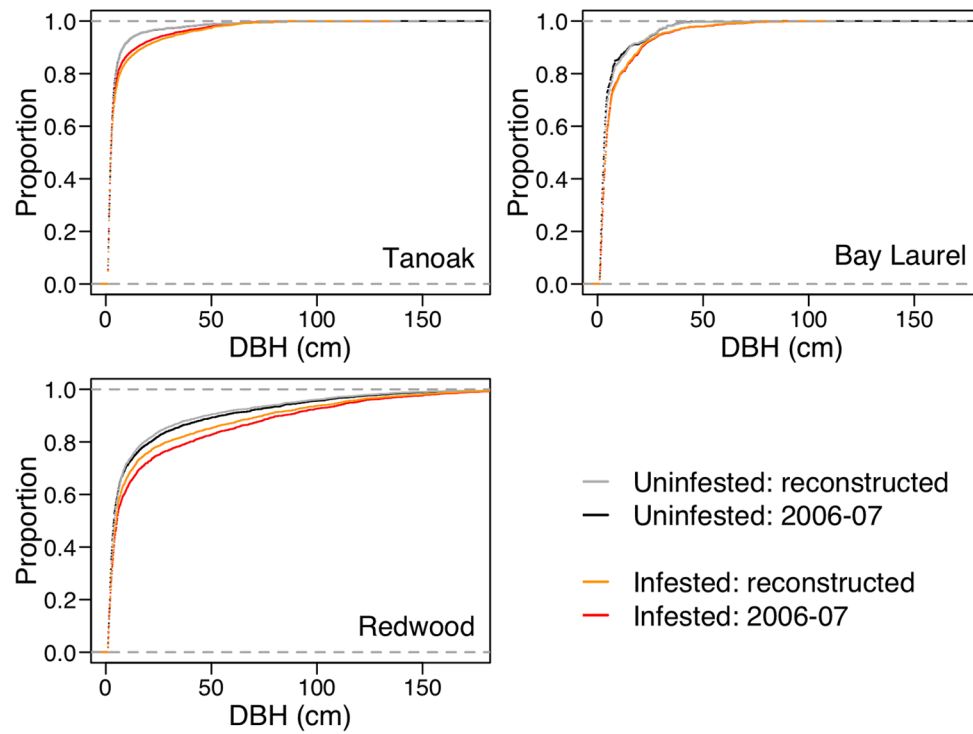


Fig. A4. Stem DBH distributions across infested and unfested stands in redwood forests. The cumulative proportion of stems in increasing DBH size classes is shown for three dominant species in both the reconstructed and current states using all stems that occur in these plots.