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Unexpected redwood mortality from synergies between wildfire and an emerging infectious disease

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Abstract. An under-examined component of global change is the alteration of disturbance regimes due to warming climates, continued species invasions, and accelerated land-use change. These drivers of global change are themselves novel ecosystem disturbances that may interact with historically occurring disturbances in complex ways. Here we use the natural experiment presented by wildfires in redwood forests impacted by an emerging infectious disease to demonstrate unexpected synergies of novel disturbance interactions. The dominant tree, coast redwood (fire resistant without negative disease impacts), experienced unexpected synergistic increases in mortality when fire and disease co-occurred. The increased mortality risk, more than fourfold at the peak of the effect, was not predictable from impacts of either disturbance alone. Changes in fire behavior associated with changes to forest fuels that occurred through disease progression overwhelmed redwood's usual resilience to wildfire. Our results demonstrate the potential for interacting disturbances to initiate novel successional trajectories and compromise ecosystem resilience.

Key words: biological invasions; global change; interacting disturbances; Phytophthora ramorum; Sequoia sempervirens; *sudden oak death; synergy*.

Introduction

The disturbance regimes that structure ecosystems worldwide are changing rapidly due to warming climates, continued species invasions, and accelerated land-use change (Turner 2010). Shifts in disturbance frequency or severity as a result of any of these drivers of global change can have profound impacts on ecosystems, especially if the disturbance regime moves beyond the range of variation to which native species are adapted (Fraterrigo and Rusak 2008). Many drivers of global change are themselves novel disturbances that may interact with historically occurring disturbances in complex ways (Mack and D'Antonio 1998). For example, there have been several plant invaders that changed ecosystem fuel properties and thereby altered the timing, intensity, or type of fire in a system (D'Antonio and Vitousek 1992, Mack and D'Antonio 1998, Brooks et al. 2004). Multiple disturbances interact in additive, synergistic, or antagonistic ways, sometimes leading to "ecological surprises" and novel successional trajectories that change ecosystems in unanticipated ways (Paine et al. 1998, Darling and Côté 2008). Managing ecosystems in a world of global change will therefore require a detailed understanding of how altered disturbance regimes impact communities, and yet, despite these concerns, interactive disturbance regimes have been little studied relative to other components of global change (Turner 2010).

Introductions of nonnative pests and pathogens constitute biotic disturbances in many forest ecosystems (Aukema et al. 2010, Santini et al. 2013), creating concern that widespread pest-related tree mortality can lead to positive feedbacks with fire severity (Parker et al. 2006, Jenkins et al. 2008, but see Simard et al. 2011). Very little attention has been paid to the ways in which the tree mortality from nonnative diseases could augment fire hazard or severity. In many temperate and boreal landscapes, warming temperatures have been associated with increases in the frequency and duration of large wildfires (Gillett 2004, Pausas 2004, Westerling et al. 2006). As the spread of forest pathogens continues (Aukema et al. 2010, Santini et al. 2013), there is growing opportunity for disturbance interactions between fire and nonnative pathogens with unknown consequences for the composition and dynamics of native forests.

We capitalized on the rare opportunity to study potential interactions between a novel biotic disturbance and a large-scale natural disturbance when wildfires burned through California forests impacted by the emerging infectious disease, sudden oak death (SOD; Appendix A: Fig. A1). Caused by the introduced pathogen *Phytophthora ramorum*, SOD has led to

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widespread mortality of tanoak (*Notholithocarpus densiflorus* syn. *Lithocarpus densiflorus*) and several oak species (*Quercus* spp.) in coastal forests of California and Oregon since its emergence in the late 1990s (Rizzo and Garbelotto 2003, Metz et al. 2012). Tree species in these forests differ in their ability to withstand injury or resist mortality from disease or fire, and trees surviving one disturbance will vary in their ability to transmit pathogen inoculum (Davidson et al. 2008) and in the flammability of their tissues (Engber and Varner 2012). Therefore, either disturbance can cause changes to forest composition and influence the extent or severity of a subsequent disturbance. Variation in surface and crown fuel amount and quality at different stages of the disease epidemic create the potential for complex fire–disease interactions in the SOD pathosystem (Kuljian and Varner 2010, Metz et al. 2011, Valachovic et al. 2011).

Since 2006, we have been monitoring the spread and impacts of SOD across the 80 000 ha of the Big Sur ecoregion along California's central coast (Haas et al. 2011, Metz et al. 2012; Appendix A: Fig. A1). In 2008, two wildfires together burned more than 70 000 ha, including more than 40% of the plots in our network, which included a gradient of disease impacts varying both in levels of disease-related mortality and time since establishment of the pathogen (Meentemeyer et al. 2008, Metz et al. 2011). We combined extensive prefire data on forest structure and disease impacts with postfire assessments of burn severity and tree mortality to evaluate the potential for synergies between an emerging forest disease and wildfire in coast redwood forests. We focused on the three tree species that together account for more than 95% of the basal area in these forests: the iconic coast redwood (*Sequoia sempervirens*), tanoak, and California bay laurel (*Umbellularia californica*). These species differ in their vulnerability to injury or mortality by disease (Rizzo et al. 2005) and fire (Stuart and Stephens 2006). Specifically, we addressed the following three questions about potential disease–fire interactions: (1) Did the presence of *P. ramorum* increase the risk of fire-related mortality for redwood, bay laurel, and tanoak? Further, if *P. ramorum* presence did affect mortality, was the stage of disease progression a better predictor of mortality than pathogen presence alone? (2) Were the stand-level consequences of co-occurring disturbances predictable from the individual effects of either disease or disturbance occurring alone? (3) What were the potential mechanisms leading to synergistic interactions between disease and fire? Results have implications for coastal California forests where SOD and fire interact and, more broadly, in forests where novel disturbance interactions will be increasingly common.

Methods

We have been monitoring forest composition and disease spread using 280 500-m^2 plots in Big Sur, Monterey County, California, USA, since 2006–2007 to understand potential feedbacks between *P. ramorum*, its hosts, and the environment (Haas et al. 2011, Metz et al. 2012; Appendix A: Fig. A1). Big Sur, at the southernmost limit of *P. ramorum*'s current range, has a heterogeneous topography and diverse flora occurring in a mosaic of grasslands, shrublands, and forests along the western slopes of the Santa Lucia Mountains. Redwood forests are generally confined to riparian areas, and the average fire return interval is thought to be 24 years (Davis and Borchert 2006). Infested plots in our network were likely invaded by *P. ramorum* anywhere from 3 to 15 years before plot establishment in 2006–2007. The exact introduction event into Big Sur is unknown, but SOD mortality was first observed in Monterey and neighboring counties in the mid-1990s (Maloney et al. 2005).

Each plot was assigned a disease progression index, as follows, using mortality of tanoak and oaks that suffer lethal infections from *P. ramorum*. Upon plot establishment, we scored each standing dead tree as newly dead (retained leaves and fine twigs) or having died several years previous (with loss of fine twigs and fragmentation of the main stem). The earliest disease stage thus comprised infested plots with no host mortality (but with standing live hosts), followed by plots where more than one-third of the dead host basal area was newly dead, and then by late-stage plots where more than two-thirds of host mortality was classified as older mortality. Plots advanced in the index if downed log volume surpassed the mean host log volume observed in uninfested plots. This system led to plots separated into four groups ranked 0 (disease detected, no impacts) to 3 (older invasion, most hosts dead and fallen). We combined the first two categories to obtain appropriate sample sizes for stands in the early, intermediate, or late stage of *P. ramorum* invasion.

In 2008, the Basin Complex (~65 000 ha) and Chalk (~6400 ha) fires, burned 121 of the 280 500-m^2 monitoring plots in this network (Appendix A: Fig. A1) The fires followed two years of drought and burned over a month each, spanning a wide variation in corresponding fire weather (Appendix A: Fig. A1). The fires provided a natural experiment to understand the impacts of interacting disturbances on forest dynamics (Metz et al. 2011). We re-surveyed 46 burned redwood plots in 2009 (23 each infested, uninfested) (Appendix A: Fig. A1). Every live or dead stem $\geq$1 cm dbh was identified, mapped, measured, and assessed for pathogen infection upon plot establishment in 2006–2007 and reassessed for survival in 2009, the first growing season after the 2008 fires. For 29 of these plots (19 infested, 10 uninfested) we also collected tree-level indicators of burn severity (e.g., stem char height, percent crown scorch, and changes to soil structure) immediately following the Basin Fire in September–October 2008 (Metz et al. 2011; see Plate 1).

Our examination of potential disease–fire synergies focused on redwood, tanoak, and bay laurel, which

account for 76.3% ± 2.5% (mean ± SE), 15.1% ± 2.2%, and 4.4% ± 0.8%, respectively, of the basal area in redwood stands in our plot network. Coast redwood is known to be resilient to either disturbance. Redwood's thick bark, flammable litter, and vigorous branch and stem sprouting all confer resistance to the periodic fires that occur in California forests (Stuart and Stephens 2006, Lorimer et al. 2009, Ramage et al. 2010). Redwoods have epidemiologically negligible contributions to pathogen sporulation, suffer no known negative impacts from leaf infections, and may even benefit from SOD by the removal from the canopy of its main competitor, tanoak (Davidson et al. 2008, Waring and O'Hara 2008, Cobb et al. 2010, Metz et al. 2012). Tanoak and bay laurel are both epidemiologically important hosts that support sporulation and transmission of the pathogen, but only tanoak dies from SOD (Davidson et al. 2008), and both species are moderately sensitive to fire (Stuart and Stephens 2006).

Our analyses proceeded in three steps. First, we asked whether pathogen presence increased the fire mortality risk of stems of different sizes using generalized linear mixed-effects models with binomial errors for each species separately. We included the random effects of individual trees nested within study plots to account for the non-independence of stems within the same tree (trees of all three species often have multiple stems >1 cm dbh) or within the same plot. When there was a significant effect of disease presence, we also substituted a three-level categorical variable of disease stage to assess whether changing fuels through disease progression (Metz et al. 2011) were better predictors of tree mortality than disease presence alone (comparing models using AIC). Here, early-stage disease impacts were combined with uninfested plots because of the lag between pathogen invasion and changes to fuels caused by host mortality.

Second, we asked how stand-level basal area lost to fire-caused mortality from the co-occurrence of both SOD and fire compared to expectations from loss in stands that experienced either disturbance alone. We averaged the percent basal area for stems ≤100 cm dbh (negligible mortality occurred for larger stems; Fig. 1) that was alive at plot establishment (2006–2007) but dead postfire (2009) across plots that experienced SOD and fire alone or together. We used the addition theorem of probability to create a null mortality expectation under the co-occurrence of two independent disturbance agents: wildfire and SOD. Given that a stem killed by either SOD or fire cannot be re-killed by the other agent and because both agents have the same potential maximal effect (i.e., 100% mortality in a stand), the first agent kills some fraction (A) of basal area, and the second disturbance's fractional mortality (B) applies to the surviving fraction ($1 - A$). Thus, the expected joint fractional mortality of the two agents is $A + (B \times [1 - A])$, and significant departures above or below this value represent synergistic or antagonistic disturbance interactions, respectively. This additive response is a common null model in other fields, though terminology varies widely (e.g., Greco et al. 1995, Sih et al. 1998). We calculated the expected mortality using mean values of single-disturbance plots and confidence intervals (95% CI) from Gaussian error propagation. We asked whether the mean observed basal area lost to mortality in burned, infested plots fell within the 95% CI of the null expectation of additive interactions. The magnitude of the synergy was calculated using an effect size for differences between observed (obs) mortality with co-occurring disturbances and expected (exp) mortality as: ([obs − exp]/obs).

Third, when synergistic disturbance impacts were detected, we used tree-level burn severity measures (percentage of crown volume scorched, trunk char height, and soil damage) to understand probable mechanisms for alteration of fire behavior by disease. We fit several logistic regression models of tree survival in 29 plots from the 2008 postfire burn severity assessment (Metz et al. 2011). We only modeled the survival of the largest dbh live stem per tree because burn severity markers had been assessed at the tree, not stem, level. We first calculated mortality predictions using standard equations based on bark thickness and percentage of crown volume scorched (Hood et al. 2007). We then fit to our data a logistic regression model of stem mortality similar to standard equations by using those same predictor variables and four other models for comparison of other factors known to affect tree mortality following fire: (1) additional fire injury indicators (soil scorch rating and height of char on tree bole [Metz et al. 2011]); (2) binary pathogen absence/presence; (3) disease impacts (categorical stage of disease invasion); and (4) topographic environment (plot slope and aspect) and stand structure (live basal area, dead basal area, coarse woody debris volume). We compared the fit and predictive power of the models using AIC and the area under the receiver-operating curve (ROC) for all trees and separately for infested and uninfested stands. These models all predicted one-year mortality although the traditional equations (Hood et al. 2007) are intended to predict three-year mortality. Importantly, the observed one-year mortality met or exceeded the three-year mortality predictions, providing a conservative assessment of potential synergies (i.e., three-year mortality would be even greater than one-year mortality).

Results

Coast redwood suffered a remarkable increase in mortality as a result of synergistic interactions of wildfire and disease despite its resilience to either disturbance alone. Fire-related redwood mortality increased significantly in the presence of the disease, with the stage of disease progression a better mortality predictor than pathogen presence/absence alone (Table 1). Mortality risk for redwood stems in stands at the

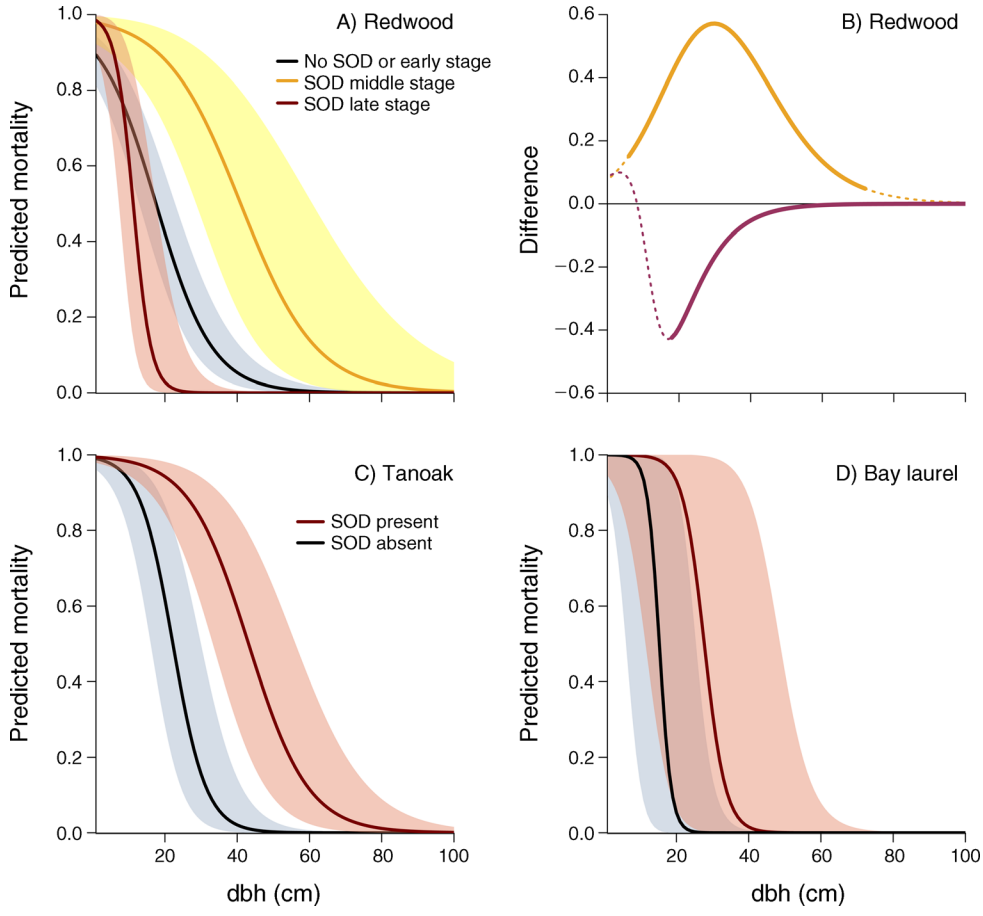

FIG. 1. Fitted probabilities of mortality risk (and 95% confidence interval envelope) between plot establishment prefire (2006–2007) and one year following wildfire (2009) for stems of increasing diameter with sudden oak death (SOD) presence or the disease stage for (A, B) redwood, (C) tanoak, and (D) bay laurel. Panel (B) shows the difference in redwood mortality risk by disease stage relative to uninfested or early infested stands, where the lines are the difference between the orange (intermediate-stage) or red (late-stage) lines and black line from panel (A) and are solid when the 95% CI of the diseased stands does not overlap the line from the undiseased plots.

intermediate disease stage was significantly increased above that in uninfested plots, where there is a mixture of recent and old standing dead host trees and some accumulation of surface woody fuels (Fig. 1A). At the peak of this effect, stems 30 cm dbh had a greater than fourfold increase in mortality, from 17% in uninfested stands to 74% in infested stands in the intermediate stage of disease progression (Fig. 1B). The size at which redwood trees outgrew significant vulnerability to fire-caused mortality also increased. For example, stems that were $\geq$41 cm dbh in uninfested stands had $<$5% mortality risk, but the threshold stem size for this same level of mortality risk increased to $\geq$72 cm dbh in stands that were in the intermediate stage of the disease progression. There was a somewhat reduced mortality risk for stems in late-stage disease stands where the vast majority of host trees had died, fallen to accumulate on the ground, and had been decomposing (Fig. 1A). Tanoak mortality risk was elevated in burned diseased stands relative to burned disease-free stands, and bay mortality risk did not depend on the presence of the pathogen (Table 1, Fig. 1C and D). Fire-related mortality of all species decreased with increasing stem girth regardless of disease presence (Table 1, Fig. 1), and there was negligible mortality for large trees $\geq$100 cm dbh.

Consistent with our understanding of its biology, plot-level redwood basal area loss between 2006–2007 and 2009 (pre- and postfire, respectively) for stems $\leq$100 cm dbh was low in stands experiencing either SOD or fire alone (Fig. 2). Surprisingly, mortality for redwoods in stands experiencing both disturbances was significantly higher than expected from additive effects of either disturbance alone, demonstrating synergistic increases when SOD and fire co-occur (Fig. 2). The observed basal area loss was ~200% greater than the expected loss if there were no synergies between fire and disease. This result was even more remarkable because these same synergies were not observed in fire-sensitive

TABLE 1. Results of generalized linear mixed-effects models of mortality for models including binary disease presence/absence or disease stage for redwood (1043 redwood stems from 44 plots), tanoak (2346 stems from 38 plots), and bay laurel (270 stems from 23 plots).

Effect	Disease presence/absence			Disease stage	
	Redwood	Tanoak	Bay	Redwood	Tanoak
Random effects (SD)					
Tree(Plot)	0.852	2.418	5.718	0.974	2.858
Plot	0.954	2.068	5.246	1.200	2.687
Fixed effects (odds ratio)					
Intercept (SOD absent)	**0.441**	**33.415**	9.837	**0.429**	**32.017**
Tree size	**0.879**	**0.805**	**0.544**	**0.880**	**0.779**
SOD	**3.841**	2.671	24.167		
Size × SOD	1.029	**1.100**	1.312		
SOD-intermediate				**11.606**	**22.377**
SOD-late				**0.013**	3.161
Size × SOD-intermediate				1.033	**1.146**
Size × SOD-late				**0.759**	**1.141**
Evaluating model fit					
AIC	695	**906**	150	**676**	1890
ROC	0.98	0.99	1.00	0.98	0.98

Notes: The standard deviation of random effects of tree nested within plot are followed by the odds ratio for the parameter estimates, which are in boldface type when the predictor was significant. When a significant effect of SOD presence in a stand was detected, we also ran a model with disease stage and compared Akaike's information criterion (AIC) and the area under the receiver-operating curve (ROC). The AIC assesses the fit of the model and can be compared among alternative models: a lower value reflects a better fit (e.g., between models that had only disease presence/absence vs. disease progression stage). The ROC value assesses how well the model discerns between mortality outcomes (tree survived or died).

tanoak or bay laurel that are also dominant in these plots (Table 1, Figs. 1 and 2).

To understand the potential mechanisms of increased mortality, we examined indicators of fire injury experienced by trees and characteristics of the forest neighborhood, including stand structure, disease impacts, and topography. The synergistic mortality observed for redwoods in burned, infested stands meant that standard tree-mortality models (Hood et al. 2007) using bark thickness and crown scorch failed to adequately predict redwood mortality in infested plots, but performed well for uninfested plots (Appendix B: Table B1). Large trees suffered higher mortality in infested plots than predicted by the models. In all models, crown scorch volume was the only consistent significant predictor of tree mortality. Redwood crowns in inter-

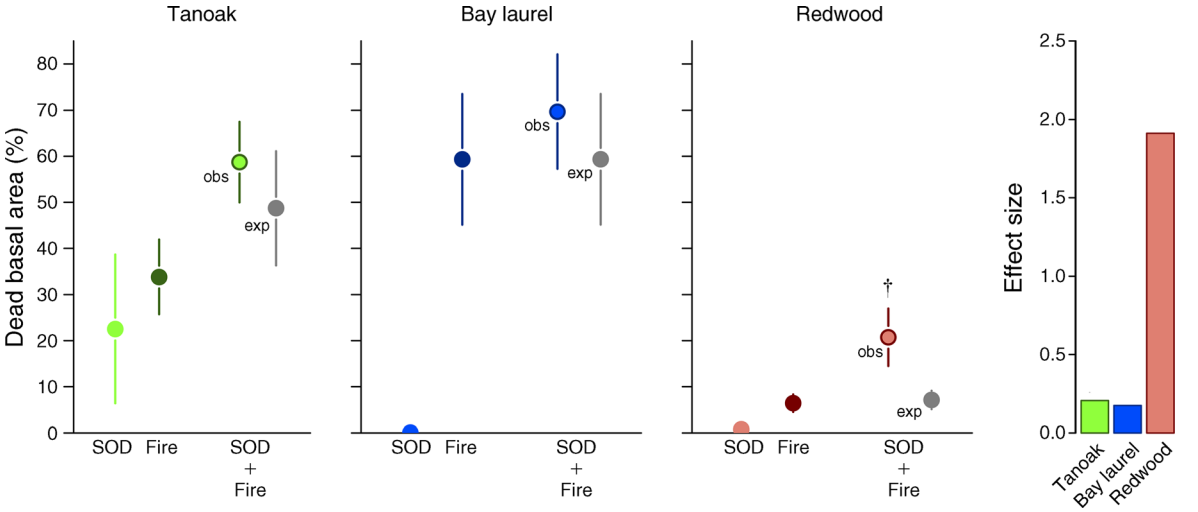

FIG. 2. Loss of basal area (mean ± SE) between 2006–2007 and 2009 for trees <100 cm dbh only in plots that experienced SOD alone, fire alone, or both disturbances together. The expected (exp) loss of basal area for both disturbances (gray points) based on the observed (obs) additive effects of either disturbance alone is shown for each species with propagated SE bars. The dagger (†) indicates that observed mortality from both disturbances is not within the 95% CI of the expected mortality. The synergistic effect size ([obs − exp]/obs) for departures from additivity is shown in the right-most panel. The observed mortality in redwood under both disturbances represents an almost 200% increase above the expected basal area loss.

PLATE 1. Burned understory of a redwood forest in Big Sur, California, USA, during the 2008 Basin Fire. Photo credit: ©Howard Kuljian.

mediate-stage infested stands had 43.5% ± 10.1% (mean ± SE) volume scorched, on average, corresponding to scorch heights reaching 38 m, with a mean height of 15.9 ± 2.8 m that was double the heights observed in early or late stage diseased stands. Inclusion of other variables did improve the fit and prediction ability of the model (Table B1). In particular, including other mechanisms of tree injury (trunk char and soil damage) or disease stage categories (early, intermediate, late) in the model improved mortality predictions, suggesting disease impacts altered fire behavior and thus tree injury.

DISCUSSION

Synergistic interactions between a nonnative pathogen and the historically occurring disturbance of wildfire led to unexpected mortality increases in the iconic coast redwood. Elevated fire-related redwood mortality in the presence of *P. ramorum* could not have been due to any direct effect of the pathogen on redwood, an inconsequential foliar host (Davidson et al. 2008), but rather the ways in which SOD altered forest structure and fuel availability with consequences for fire severity. Invasion by *P. ramorum* changes both intrinsic fuel quality and extrinsic fuel arrangement or quantity (sensu Brooks et al. 2004) in redwood stands. The relationship between SOD and fire-caused redwood mortality was best predicted by inclusion of the disease-stage variable rather than mere presence/absence of the pathogen, supporting the hypothesis that fuel variation among disease stages is important for fire behavior and effects (Kuljian and Varner 2010, Metz et al. 2011, Valachovic et al. 2011). The dynamics of this spatially and

temporally patchy disease generated an availability of aerial and surface fuels that elevated mortality in coast redwood, a normally fire-resistant tree species. Tanoak and bay laurel did not experience synergistic increases in fire-related mortality when the disease occurred, likely because these species were already quite vulnerable to mortality from fire, and altered fire behavior could only cause marginal increases in these already high rates.

While elevated redwood mortality under co-occurring disturbances was not predictable from the mortality observed when either disturbance occurred alone, there are several potential mechanisms to explain this synergy. These include local increases in fire line intensity caused by torching of standing, disease-killed tanoaks (Kuljian and Varner 2010) or the acceleration of fuel drying and subsequent increases in fire line intensity because of reduced canopy shade from pathogen-related tree mortality. In the early stages of disease progression, standing trees were killed and initially retained their dead foliage, constituting crown fuels with very low fuel moisture (Kuljian and Varner 2010). Our mortality models (Table B1) consistently identified crown scorch as the primary factor in elevated redwood mortality, suggesting that standing dead fuels were the principal contributor to elevated mortality. This supports the hypothesis that fires in this phase of the disease likely ignited the crowns of the standing dead tanoaks, resulting in localized collateral crown scorching of intermixed redwoods. As the disease progressed, branches and stems of killed hosts fell to the forest floor, comprising a well-aerated fuel bed that increased surface fire line intensity, likely resulting in more bole charring of remnant redwoods. This hypothesis is supported by the high crown scorch heights measured in intermediate-stage stands. Using equations that relate scorch height to fire line intensity (Van Wagner 1973), we conservatively estimate substantially higher fire line intensities (maximum, 4100 kW/m; median, 920 kW/m) in the intermediate-stage sites than the uninfected/early (maximum, 1680 kW/m; median, 290 kW/m) and late-stage (maximum 825 kW/m; median, 325 kW/m) infestations (Table B2). With time and decay of fallen tanoaks, the fuel quality (of decayed wood and the increased compaction of the fuel bed) of these decomposing coarse fuels would have decreased, potentially explaining the depression of crown scorch and resulting fire severity we observed in the latter stages of *P. ramorum* invasion. The accuracy of redwood mortality predictions was improved when other indicators of tree injury or stage of disease were included, reflecting the close link between these synergistic effects and the complexity of heterogeneous fuel development across a landscape at different stages of pathogen invasion.

There is growing concern that synergistic interactions among multiple stressors are common, and that novel disturbance interactions challenge an ecosystem's resilience or recovery to pre-disturbance states (Darling and Côté 2008, Buma and Wessman 2011). Redwood forests with a significant tanoak component have historically experienced recurrent wildfires (Stephens and Fry 2005) and occur throughout the potential range of *P. ramorum* and its competent hosts. The pathogen continues to expand its distribution in fire-prone areas of California (and Oregon), increasing the likelihood that other forests will experience the synergistic impacts of both disturbances. Increased fire-related redwood mortality in the presence of *P. ramorum* will alter the structure, composition, and stand dynamics of redwood forests, which already are of great conservation concern. Understanding interactions between the disease and wildfire is central to the management of coast redwood forests and may be a hint of the unexpected consequences that can occur in other fire-prone wildlands where nonnative pathogens are invading.

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Supplemental Material

Appendix A

Map of the Big Sur study region with monitoring plot locations and fire perimeters (*Ecological Archives* E094-201-A1).

Appendix B

Understanding fire-related redwood mortality through model comparisons of fire severity indicators and estimates of fire line intensities for stands in different stages of the disease progression (*Ecological Archives* E094-201-A2).