Modeling when, where, and how to manage a forest epidemic, motivated by sudden oak death in California

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Edited by Simon A. Levin, Princeton University, Princeton, NJ, and approved March 25, 2016 (received for review February 10, 2016)

Sudden oak death, caused by *Phytophthora ramorum*, has killed millions of oak and tanoak in California since its first detection in 1995. Despite some localized small-scale management, there has been no large-scale attempt to slow the spread of the pathogen in California. Here we use a stochastic spatially explicit model parameterized using data on the spread of *P. ramorum* to investigate whether and how the epidemic can be controlled. We find that slowing the spread of *P. ramorum* is now not possible, and has been impossible for a number of years. However, despite extensive cryptic (i.e., presymptomatic) infection and frequent long-range transmission, effective exclusion of the pathogen from large parts of the state could, in principle, have been possible were it to have been started by 2002. This is the approximate date by which sufficient knowledge of *P. ramorum* epidemiology had accumulated for large-scale management to be realistic. The necessary expenditure would have been very large, but could have been greatly reduced by optimizing the radius within which infected sites are treated and careful selection of sites to treat. In particular, we find that a dynamic strategy treating sites on the epidemic wave front leads to optimal performance. We also find that "front loading" the budget, that is, treating very heavily at the start of the management program, would greatly improve control. Our work introduces a framework for quantifying the likelihood of success and risks of failure of management that can be applied to invading pests and pathogens threatening forests worldwide.

Phytophthora ramorum | constrained budget | landscape-scale stochastic epidemiological model | optimizing disease control | risk aversion

Introductions of new pathogens into previously uncolonized areas pose threats to trees in natural ecosystems, commercial woodlands, and urban environments. Rates of introduction are increasing (1), driven by changing climate (2) and altered patterns of travel and trade (3). Emerging epidemics cause direct economic loss from death and restricted growth of trees grown for timber and horticultural use (4). Other major impacts occur when susceptible trees play critical roles in ecosystem services (5).

Successful control of epidemics involves matching the scale of management with the inherent scale of spread (6). Early detection and timely removal of affected trees from a small number of newly infected sites soon after initial introduction(s) can prevent epidemics (7). Routine detection and control of emerging pathogens of woodland trees, however, are prone to significant logistical and epidemiological constraints. Detection and reporting can be delayed by incomplete and infrequent sampling of large areas of susceptible hosts (8), with broad pathogen host ranges often increasing the area that must be surveyed. Infected sites may be inaccessible or under multiple ownership (9). Long incubation periods for some pathogens mean that disease remains cryptic while infection continues to spread (10). Spread can also be over long distances (11), with extensive creation of new foci.

Difficulties in detection and management have undoubtedly contributed to high-profile failures of large-scale control programs for a number of tree diseases, including chestnut blight (12), white pine blister rust (13), Dutch elm disease (10), and citrus canker (14). Identifying when, where, why, and how to manage emerging epidemics at regional, state, or countrywide scales, and even whether or not it is feasible to do so, remains a major challenge (15). However, understanding whether management can eradicate a pathogen or restrict its spread to uninvaded locations is critical in identifying cost-effective control strategies. We show here how mathematical modeling can be used to do this, using sudden oak death (SOD) in California (CA) as an example.

SOD, caused by the oomycete *Phytophthora ramorum* (*PR*), has killed millions of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) in CA since first detection in 1995 (16). The epidemic has been intensively monitored (17), and much is now known about *PR* epidemiology. However, questions remain about the feasibility of statewide control, introducing uncertainty and confusion into identifying regional management objectives. The epidemic also provides an opportunity for retrospective analyses of how effective control scenarios could have been, had they been introduced at different stages in the epidemic.

Here we extend a previously tested, spatially explicit, stochastic, statewide model, resolved to 250 × 250-m resolution (18), to compare the range of outcomes for different management scenarios, addressing the following questions:

Could statewide prevention of continued pathogen spread be successful were it to start now, given the current size of the epidemic and the budget potentially available for treatment?

Could prevention of pathogen spread ever have been successful by management starting after the pathogen was sufficiently well characterized for control to have been realistic?

How could local and statewide deployment of management have been optimized?

The analyses also address the following generic questions about epidemic control under uncertainty:

Significance

We use sudden oak death in California to illustrate how mathematical modeling can be used to optimize control of established epidemics of invading pathogens in complex heterogeneous landscapes. We use our statewide model—which has been parameterized to pathogen spread data—to address a number of broadly applicable questions. How quickly must management start? When is an epidemic too large to prevent further spread effectively? How should local treatment be deployed? How does this depend on the budget and level of risk aversion? Where should treatment be targeted? How should expenditure be balanced on detection and treatment? What if the budget changes over time? The underlying principles are important for management of all plant disease epidemics in natural ecosystems.

Author contributions: N.J.C., R.C.C., R.K.M., D.M.R., and C.A.G. designed research; N.J.C. performed research; N.J.C. analyzed data; and N.J.C., R.C.C., R.K.M., D.M.R., and C.A.G. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1602153113/-/DCSupplemental.

How soon must control start for it to be effective?

When is an epidemic so large that control is impossible?

Which sites should be targeted when there are insufficient resources to treat all infected sites?

How extensively should sites be treated?

How does this depend on the budget and risk aversion?

How can costs of detection and treatment be balanced?

What is the effect of a budget that changes over time?

Over 100 tree and forest shrub species are known to be susceptible to *PR* infection (16). First reported in 1995 in coastal regions near San Francisco (19), the pathogen is transmitted locally via rain splash and over long distances via storms and human-mediated transport. Subsequent estimates of the initial introduction to natural ecosystems suggest that the first invasion occurred around 1990 (16). *PR* has spread widely in coastal CA since then: Coastal forests from southern Monterey County up to northern Humboldt County are affected. Billions of tanoak and oak trees, covering over 20 million ha of land, are potentially threatened (20). There are also large and growing epidemics in several countries in Europe (21).

The only treatment shown to be effective in reducing pathogen prevalence at the landscape scale is removal of host species (22), as has been practiced in the United Kingdom for a number of years (21). In North America, however, large-scale management has not been attempted. Nevertheless, an outbreak of SOD in Curry County, Oregon, has remained relatively small in comparison with CA due to active management by host removal since first detection in 2001 (23). The attempted eradication of *PR* in Oregon in the very earliest stage of that outbreak was only possible because the potential impacts were already clear from CA, and because its epidemiology was beginning to be characterized (22). We consider 2002 to be the earliest that statewide attempts to prevent further spread of *PR* in CA could, in principle, have been attempted. This was 1 y after the pathogen was first named (24), and the year in which the pathogen first came under European Community emergency control measures (21).

Results

Pathogen Spread Without Management. Our analyses predict that spread of *PR* within CA will accelerate after 2020 if measures to slow the spread are not attempted. This will be driven by the pathogen reaching the northwestern coast, where large regions of continuous host and suitable weather conditions facilitate spread (18). The estimated area infected by 2030 in CA is around 14,000 km^2, a nearly 10-fold increase from the predicted ~1,550 km^2 infected in 2014 (Fig. 1*B* and Movie S1). However, all estimates of future epidemic size reflect the inherent variability of pathogen spread (Fig. 1*C*), largely driven by the timing of long-distance dispersal to the northwest but also reflecting stochastic variability in pathogen bulking up and dispersal driven by fluctuating environmental drivers in a heterogeneous host landscape. A 95% prediction interval for the area infected by 2030 ranges from ~7,600 to ~19,600 km^2. Acceleration in spread means that an ever-increasing area is expected to be infected but in which the pathogen remains undetected. By 2030, we predict on average ~4,000 km^2 would have been colonized too recently to show crown mortality symptoms detectable via an aerial survey.

Slow the Spread Management Starting 2014. Initially, we consider performance of a baseline management strategy that removes *PR*-susceptible and -infected hosts within a radius of 375 m of detected foci of infection. Treated sites are randomly selected from the set of sites known to contain infection until a fixed budget per y is exhausted. Control starting in 2014 under this scenario has very little effect on the area lost by 2030, that is, the area ever affected by either disease or host removal (Fig. 2 and Movie S2). Even a very large budget allowing up to 200 km^2 to be treated annually—which we estimate would cost at least 100 million US dollars (USD) per y (*SI Appendix*)—has almost no effect on spread. The California Department of Forestry and Fire Protection (Cal Fire) allocated just over 90 million USD in fiscal year 2015–2016 to improve forest management for carbon sequestration and to implement drought mitigation, suggesting this level of expense is at least within the bounds of possibility. However, multiple and sometimes competing objectives must be addressed by Cal Fire, meaning that allocation of the full budget to slowing the spread of a single pathogen is very unlikely. Given the almost imperceptible effect on the epidemic of even such a large amount of management, we consider prevention of spread at the statewide scale to no longer be possible.

Slow the Spread Management Starting Before 2014. Were an attempt to restrict the natural spread of *PR* to have started earlier than 2014, it could have been more successful, although the requisite budget increases rapidly as the start date becomes later (Fig. 3*A*). Successful management can make it impossible to spend the full budget as the epidemic comes under control, and so the total area managed by 2030, which corresponds to the amount spent on treatment, is nonmonotone in budget and starting year (Fig. 3*B*). The distribution of area lost (Fig. 3*C*) again reveals the wide variability in outcome, a pattern that is replicated for the full range of budgets and starting years we considered.

Optimizing the Radius of Treatment. We consider how performance of management is affected by the treatment radius within which susceptible and infected hosts are removed around each detected site, focusing initially on a default budget that allows removal of up to 50 km^2/y. Unless otherwise stated, henceforth all results relate to management starting in 2002, the first date we consider control to have been realistic. Too small a treatment radius does

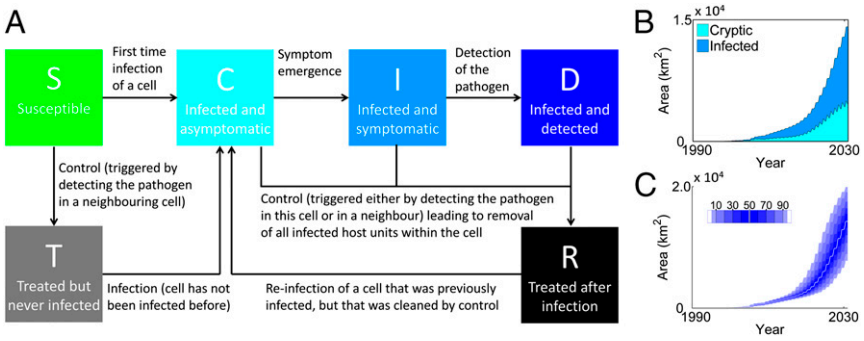

Fig. 1. Underlying epidemiological model and spread when there is no control. (*A*) Epidemiological model. (*B*) Median predicted area infected when management is not attempted, distinguishing symptomatic and cryptic infection. (*C*) Distribution of infected area without management, showing the variability in the area lost to disease. Shading shows the deciles and 5th and 95th percentiles; the white curve marks the median.

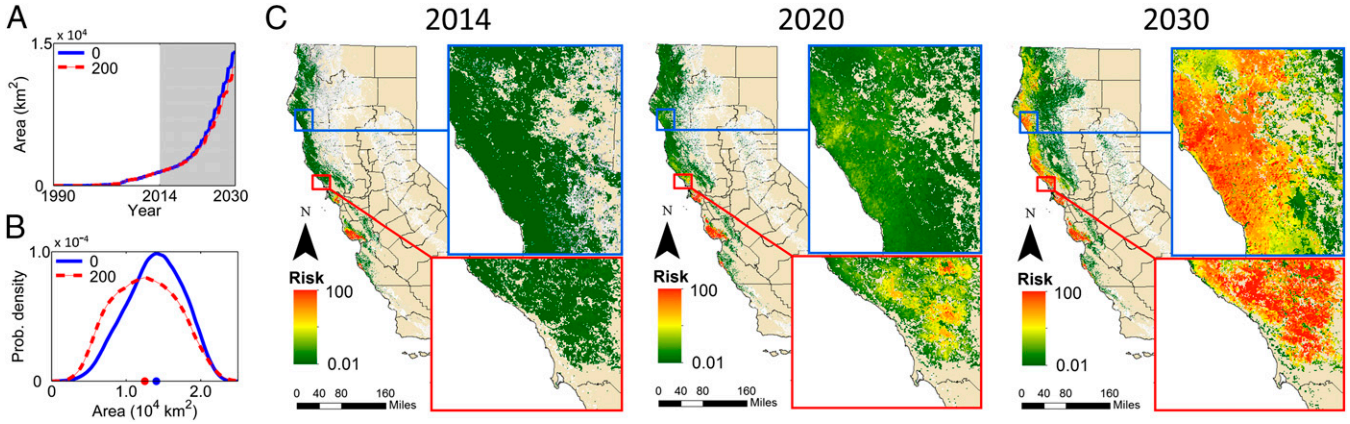

Fig. 2. Extensive treatment starting in 2014 does not contain the epidemic. (*A*) Median total area affected by infection or host removal when there is sufficient budget to treat up to 200 km^2/y, starting 2014 (red), compared with no management (blue). (*B*) Distributions of area affected by infection or host removal in 2030. (*C*) Predicted spread of infection for statewide management with budget allowing up to 200 km^2/y to be controlled (control starting 2014).

not account sufficiently for cryptic infection around each detected focus, whereas at too large a treatment radius, too many healthy trees are unnecessarily removed and/or an increased proportion of other infection remains untreated because of the limited budget (Fig. 4*A*). There is therefore an optimum in the area lost as the treatment radius is altered, at least if the objective is phrased in terms of minimizing the average epidemic size. In particular, at the optimal radius of 187.5 m, the median area lost is ∼6,800 km^2 (95% prediction interval ∼2,600 to ∼16,200 km^2; compare median of ∼7,900 km^2 with 95% interval ∼1,100 to ∼17,100 km^2 at the 375-m baseline).

Effect of Budget and Risk Aversion. The optimal treatment radius depends upon the budget. The more limited the resources, the more strongly treatment should focus on locations known to be infected by selecting a shorter treatment radius around each detected site, because this allows a larger number of distinct foci to be managed (Fig. 4*B*). Optimizing the treatment radius at different percentiles of the distribution of area lost reveals that shorter radii are also promoted when there is a high level of risk aversion (Fig. 4*C*). Again, a smaller treatment radius means that more known disease foci will be removed, which reduces the risk of failing to treat a site that goes on to cause many secondary infections.

Optimizing the Subset of Locations to Treat. We have assumed that foci to treat are selected randomly from the set of sites known to contain currently uncontrolled infection, analogous to independent action by stakeholders throughout infected areas. The selection of sites to treat can be improved (25–28). Logistical constraints surrounding movement of machinery and access to land make tackling highly infected regions an attractive default. However, this "infected" strategy (Movie S3) performs worse in terms of epidemic impact than the default "random" strategy (Fig. 5*A* and Movie S4). We considered a range of other possibilities, which in order of performance were focusing control on regions with large areas of host, irrespective of disease status ("host" strategy) (Movie S5), with high local rates of spread ["hazard" strategy (Movie S6), targeting areas with high basic reproductive number; R_0 (29)], or with large areas currently uninfected ("susceptible" strategy) (Movie S7).

However, the best-performing strategy we tested targets local control at and ahead of the northerly wave front of epidemic spread ("wave-front" strategy; Fig. 5*A* and Movie S8). This strategy reduces predicted area lost by 2030 to ∼2,400 km^2 (95% interval 2,600–7,400 km^2) at an optimal radius of 362.5 m (compare ∼6,800 km^2 with interval 2,600–16,200 km^2 at the optimal radius 187.5 m under the random strategy starting 2002) (Fig. 5 *A* and *D*). The increase in optimal treatment radius for the better-performing strategies was consistent across all prioritizations we tested. Selecting sites for management more effectively allows more extensive treatment around each one, because sites that have a greater impact are then treated, reducing the future spread of the epidemic.

The relative performance at the optimal treatment radius of the six strategies was consistent across all control budgets (Fig.

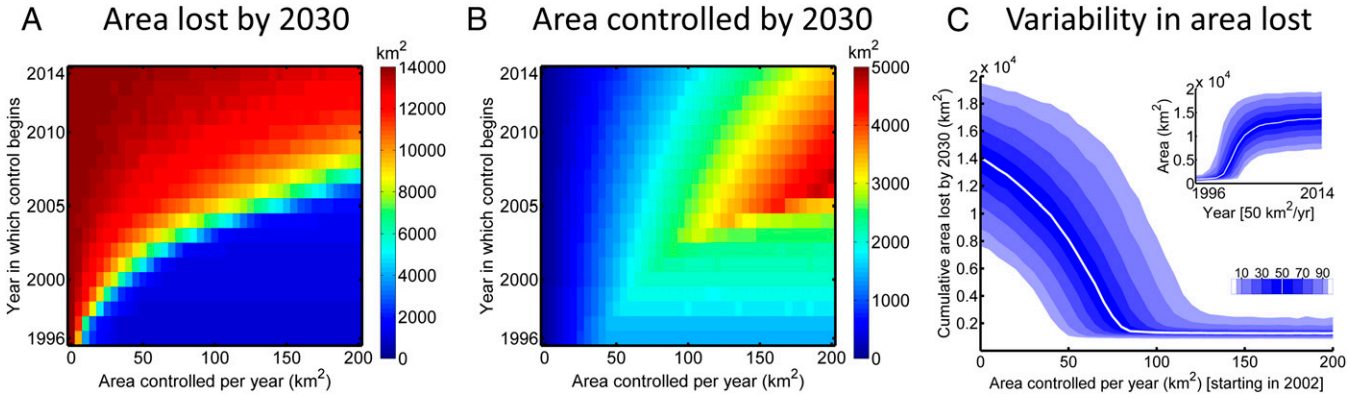

Fig. 3. Management efficacy depends on the starting date and the available budget. (*A*) Median area affected by infection or host removal by 2030. (*B*) Median area removed by treatment by 2030. (*C*) Area lost by 2030 as a function of the budget, for treatment that starts in 2002, showing the distribution of possible epidemic impacts. (*Inset*) Response to the year in which treatment begins, for a fixed budget that allows up to 50 km^2/y to be treated.

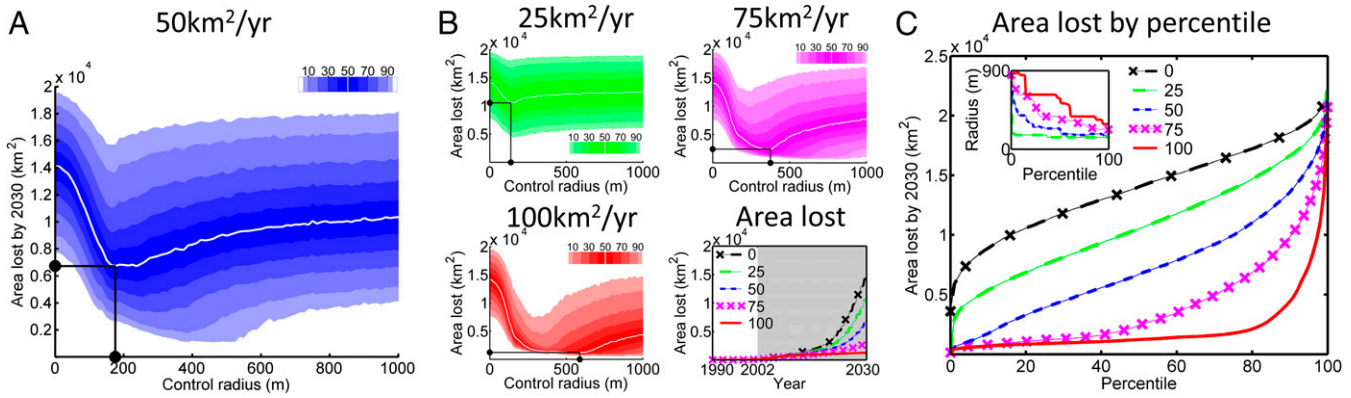

Fig. 4. Optimizing the local deployment of treatment. (*A*) Area lost by 2030 when treating up to 50 km^2/y (starting 2002). Shading shows the deciles and 5th and 95th percentiles; the white curve marks the median. (*B*) The distributions of area lost when treating 25 km^2/y (*Top Left*), 75 km^2/y (*Top Right*), and 100 km^2/y (*Bottom Left*). The median area lost when treating using the optimal radius (minimizing the median) is shown (*Bottom Right*). (*C*) Area lost at optimal radius as a function of the percentile of the distribution of area lost that is optimized. (*Inset*) Optimal radius by percentile.

5*B*) and starting dates (Fig. 5*C*) we considered, emphasizing that the ordering of these strategies is generic, at least for this epidemic. We note, however, that the performance of even the wave-front strategy degrades for control that starts later in the epidemic. Management starting in 2014 of up to 50 km^2/y leads to an average area lost of >7,000 km^2 by 2030. The epidemic remains out of control even when treating more effectively, and the ostensibly large reduction in area lost under the wave-front strategy starting 2014 corresponds only to a 7-y delay relative to not treating at all (*SI Appendix*, Fig. S8). Such poor performance even with the optimal strategy and comparatively large budget (equivalent to at least 25 million USD per y) reiterates our earlier contention that the epidemic is uncontrollable at the statewide scale for management starting today.

Budgets That Vary over Time. Resources that can be devoted to control are set by policymakers in response to a number of complex and time-dependent drivers, including public opinion, other demands on a limited budget for plant health, and the perceived and anticipated success of control. We therefore assessed management strategies for which the budget varies over time but that ensure the total budget over the period 2002–2030 is fixed (*SI Appendix*, Fig. S1*A*). It is better to devote larger resources to treatment early in the epidemic, particularly if any unused budget can be carried over to subsequent years, because the net rate of growth of the epidemic increases with its size, and so earlier treatment reduces the future growth rate (28).

Accounting for the Cost of Detection. Limited resources for management must be split between pathogen detection and treatment (30). Results thus far are contingent on extensive statewide surveys, repeated yearly. In practice, the cost of this would reduce the amount remaining to be spent on treatment. We therefore tested the effect of assuming the area that can be surveyed for disease symptoms is proportional to the fraction of the budget that is spent on detection, with the remainder devoted to management (*SI Appendix*, Fig. S1*B*). We find an optimum irrespective of the year treatment starts, and that a larger proportion of the budget should be devoted to detection for interventions that start earlier, because smaller amounts of treatment are then required and detection is more difficult when the epidemic is smaller.

Discussion

Deciding whether and how to control invasive pathogens are two of the principal challenges in epidemiology (15). Our results show how a stochastic, spatially explicit, epidemiological model can be used to integrate the current state of knowledge about the pathogen with detailed information on host topology and environmental drivers to predict the likely effects of management strategies. The model simulates epidemic spread through a large heterogeneous host landscape. It couples fine spatial resolution of 250 × 250 m for pathogen transmission with the facility to address local and statewide control of the advancing disease, allowing for spatial targeting of control within a limited budget. The model has been parameterized using data for *PR* spread in CA and successfully used to predict patterns of statewide spread (18). However, this is the first study, to our knowledge, to use a plant disease model calibrated to pathogen spread data and that predicts spread over such a large spatial scale to understand management. A version of the model has also been calibrated and validated for spread in the United Kingdom, in which context it is being used by plant health policymakers to inform management strategies, particularly the extent of host removal around infected larch stands (31).

We distinguish three phases of invasion and spread for an emerging epidemic.

> Phase 1: initial invasion that may go undetected and undiagnosed for some time and in some places; frequently, little is known about the causal agent and the potential for damage.
>
> Phase 2: the epidemic continues and is perceived to be a potentially serious threat; knowledge about the causal agent is sparse but there are sufficient data to construct models to begin to assess different strategies.
>
> Phase 3: the pathogen has spread far enough that eradication is no longer possible; local containment may still be an option.

Our analyses show that statewide action to eradicate or even slow the spread of *PR* is no longer feasible, even with a substantial budget, indicating that the epidemic in CA is now firmly in phase 3 (Figs. 1 and 2). Shifting management resources to restoring degraded forests and protecting ecological function at smaller scales would be more beneficial than attempting statewide control. Prevention of spread could, however, have been feasible starting earlier, with substantial losses prevented by earlier treatment (Fig. 3 *A* and *B*), indicating that the epidemic was still in phase 2 in 2002. However, the cost in 2002 would have been very high, and practical implementation would have required unprecedented cooperation among agencies and landowners.

The analyses for SOD in CA illustrate how epidemiological principles could be translated into practical application. One important principle is matching the scale of treatment with the inherent spatial and temporal scales of pathogen spread to achieve effective management (6, 15, 25–27, 32). Characterization of epidemic scales is complicated, as the epidemic advances through heterogeneous host populations subject to variability in

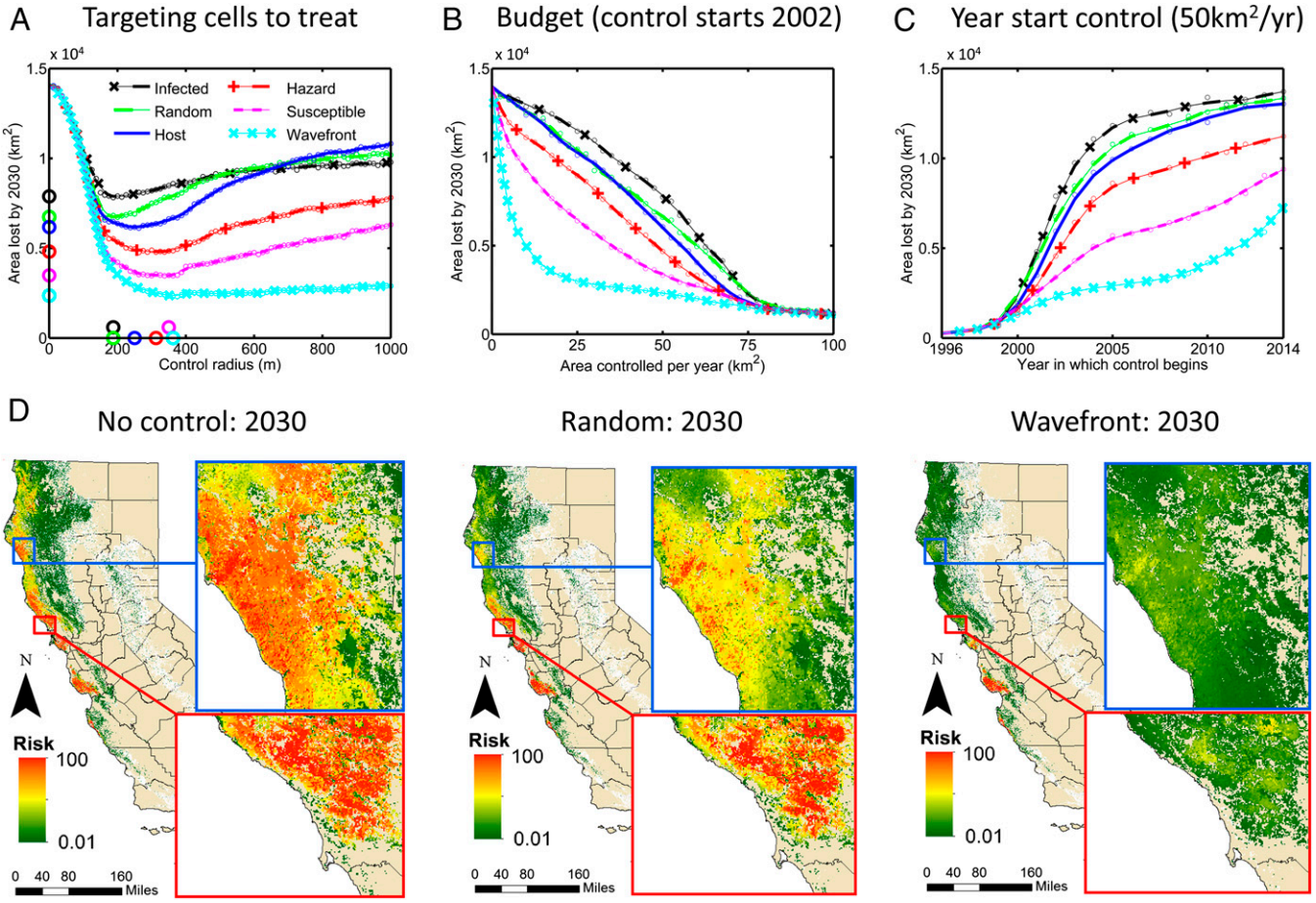

Fig. 5. Optimizing the set of locations to treat. (*A*) The median area lost by 2030 as a function of treatment radius, comparing strategies to prioritize disease foci for treatment. Management starts in 2002, with sufficient budget to treat 50 km^2/y. Circles show the optimal radius and minimum median area lost for each strategy. (*B*) Response of area lost to the budget, independently optimizing the radius for each strategy at each budget. In all cases, control starts in 2002. (*C*) The area lost by 2030 as a function of the year in which treatment starts, for treatment of up to 50 km^2/y, again optimizing the radius of treatment for each strategy for each year. (*D*) Maps showing the risk of infection in 2030 with no control (*Left*), treating with random selection of sites from the set of sites known to contain untreated infected (*Middle*), and for control, focusing treatment at and beyond the northward moving wave front of the epidemic (*Right*). Both treatment strategies are shown at their optimal radius, for treatment starting in 2002 and with sufficient budget to treat 50 km^2/y.

environmental drivers. Nevertheless, optimal scenarios can be derived for the median response, for example, the treatment radius that minimizes the impact of disease. Although optimal control radii have been derived using models at small spatial scales (7, 33), this is the first demonstration, to our knowledge, that the idea extends to landscape-scale control of plant disease within a fixed budget. Impact is assessed by totaling the area lost from disease and from removal of healthy trees around infected sites. However, the metric could readily be extended to a range of objective functions with different weightings for the individual components depending on perceived costs and benefits by different stakeholders (7, 34). Treatment radii can also be adjusted to allow for different degrees of risk aversion (35), for example, selecting the radius that corresponds to the 5th percentile (high risk aversion) through to the 95th percentile (low risk aversion) (compare Fig. 4).

Our results confirm that it would have been possible, in principle, to bring the epidemic under control by early removal of infected hosts. In practice, however, selection of sites for treatment within a limited budget is problematic. We considered a range of possibilities from random selection of sites for treatment, analogous to independent action by stakeholders in infected areas, through to highly structured centralized selection of sites according to model predictions (28, 36), the first assessment, to our knowledge, of such a prioritization for a plant disease. Model-informed selection proved superior to randomly choosing sites in terms of minimizing areas lost. The impacts of disease and treatment successively decreased (Fig. 5) by prioritizing areas where there are large host populations, where the potential for rapid local spread is high [i.e., selecting regions of high hazard (15, 18, 29)], and where the density of susceptible hosts available for infection is high. However, for the combination of host distribution, pathogen dispersal, epidemic progress, and environmental suitability for SOD in CA, local control at and ahead of the northerly wave front proved most effective (Fig. 5).

Finding and mapping newly infected sites are expensive for an epidemic spreading through a heterogeneous host landscape in which access is limited by terrain, private ownership, and resources (8). Previous work has shown how to optimize the balance between detection and treatment to maximize the cost-effectiveness when there is a shared budget for detection and treatment, but has focused exclusively on simple theoretical models (37, 38). Here we present the first test, to our knowledge, of the tradeoff between probability of detection and deployment of treatment in a realistic landscape. Our results show that there are optima that balance the expenditure on the identification of newly infected sites against the remaining resources available to treat the sites.

The conventional approach to dealing with emerging epidemics involves scaling up of the budget for control as the perception of the risk from the epidemic becomes more widely understood. This

was certainly the case during the Dutch elm disease epidemic in the United Kingdom in the 1970s (10) and also during the 50-y attempt to control white pine blister rust in the United States (13). Indeed, for SOD, the budget for control of the isolated epidemic in southwestern Oregon increased over time as the outbreak became larger (23). We show here, however, how front loading of the budget with greater expenditure in the earlier years is likely to be markedly more effective in bringing epidemics under control, increasing long-term cost-efficiency. However, in practice it might be difficult for a policymaker to justify a very large expenditure on an epidemic that currently remains small, particularly because estimates of future sizes are often subject to considerable uncertainty early in epidemics (39).

For SOD in CA, our analyses show—for the first time, to our knowledge—that statewide action to slow epidemic spread has not been feasible for some years. Management efforts to reduce impacts at local scales must now be the focus. However, we have shown how management starting 2002 could, in principle, have been successful. Our work illustrates how mathematical models can be used to optimize and assess the likely feasibility of management of established plant disease, particularly when there is a limited budget. It therefore complements a body of work on foot-and-mouth disease (25–27, 36), which also focuses on model-based optimization of large-scale management for an epidemic with long-distance dispersal. Particular challenges for SOD, however, include the broad host range of the pathogen, difficulties surrounding detection leading to very extensive cryptic infection, and the lack of any realistic control options other than host removal (e.g., no vaccination). The challenge—but also the opportunity—presented by increasing rates of introductions of plant pathogens (2, 40) and increasing acceptance of models by policymakers (31) is now to more routinely use the insights from models in the early stages of invading epidemics, when carefully optimized management can still make a difference.

Materials and Methods

Our stochastic epidemic model (Fig. 1*A*) tracks the density of *PR*-infected host across CA at 250 × 250-m spatial resolution, extending the model developed by Meentemeyer et al. (18) to include detection and treatment. The model (18) was parameterized using data on pathogen spread at local and statewide scales and validated by predicting the infection status of positive and negative sites surveyed by the California Oak Mortality Task Force (41). The AUC (area under the receiver operating curve) value was 0.89, indicating very good performance. The model allows for spatial heterogeneity in host density, multiple host species with different susceptibility and infectivity (16), short- and long-range dispersal (42), and environmental drivers to affect transmission (19). Further details are in *SI Appendix*.

ACKNOWLEDGMENTS. We thank Richard Stutt, Stephen Parnell, and Matthew Castle for discussions; and Mark Calleja for administering a computing cluster. We acknowledge funding from the BBSRC, DEFRA, NSF, USDA, and Gordon and Betty Moore Foundation.

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Biodiversity Conservation in the Face of Dramatic Forest Disease: An Integrated Conservation Strategy for Tanoak (*Notholithocarpus densiflorus*) Threatened by Sudden Oak Death

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Source: Madroño, 60(2):151-164. 2013.
Published By: California Botanical Society
DOI: http://dx.doi.org/10.3120/0024-9637-60.2.151
URL: http://www.bioone.org/doi/full/10.3120/0024-9637-60.2.151

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MADROÑO, Vol. 60, No. 2, pp. 151–164, 2013

BIODIVERSITY CONSERVATION IN THE FACE OF DRAMATIC FOREST DISEASE: AN INTEGRATED CONSERVATION STRATEGY FOR TANOAK (*NOTHOLITHOCARPUS DENSIFLORUS*) THREATENED BY SUDDEN OAK DEATH

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ABSTRACT

Non-native diseases of dominant tree species have diminished North American forest biodiversity, structure, and ecosystem function over the last 150 years. Since the mid-1990s, coastal California forests have suffered extensive decline of the endemic overstory tree tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae), following the emergence of the exotic pathogen *Phythophthora ramorum* and the resulting disease sudden oak death. There are two central challenges to protecting tanoak: 1) the pathogen *P. ramorum* has multiple pathways of spread and is thus very difficult to eradicate, and 2) the low economic valuation of tanoak obscures the cultural and ecological importance of this species. However, both modeling and field studies have shown that pathogen-centric management and host-centric preventative treatments are effective methods to reduce rates of spread, local pathogen prevalence, and to increase protection of individual trees. These management strategies are not mutually exclusive, but we lack precise understanding of the timing and extent to apply each strategy in order to minimize disease and the subsequent accumulation of fuels, loss of obligate flora and fauna, or destruction of culturally important stands. Recent work identifying heritable disease resistance traits, ameliorative treatments that reduce pathogen populations, and silvicultural treatments that shift stand composition hold promise for increasing the resiliency of tanoak populations. We suggest distinct strategies for pathogen invaded and uninvaded areas, place these in the context of local management goals, and suggest a management strategy and associated research priorities to retain the biodiversity and cultural values associated with tanoak.

Key Words: California Floristic Provence, disease ecology, genetic diversity, pathogen-caused extinction, pathogen management, *Phytophthora ramorum*, restoration, tanoak population decline.

To the detriment of cultural, economic, and silvicultural interests, North America has experienced multiple declines of dominant tree species following the introduction and spread of exotic pathogens, many of which are innocuous microbes or weak pathogens in their native ranges (Loo 2009). The quarantine of known pathogens and other techniques to avoid introduction of pathogens into novel environments are important actions to reduce pathogen spread on international and inter-continental scales (Brasier 2008). However, the host range, invasion history, and persistence in the environment of many forest pathogens suggest these invasive organisms have, or will become, naturalized in their new ranges. This challenging natural resource problem demands well-crafted management efforts to avoid the most extensive or severe impacts of forest pathogens.

The pathogen *Phytophthora ramorum* S. Werres, A.W.A.M. de Cock (Werres et al. 2001) exemplifies the destructiveness, epidemiological complexity, and difficulty in managing the most problematic invasive microorganisms (de Castro and Bolker 2005; Smith et al. 2006). *Phytophthora ramorum* was first introduced into California and spread into wildlands via infected nursery stock in Santa Cruz and Marin Counties from a yet unknown native range (Garbelotto and Hayden 2012; Grünwald et al. 2012). The pathogen subsequently spread to Big Sur (Monterey County) and Humboldt County, probably on infected plants, and has since become established in central-California coastal forests. Establishment of *P. ramorum* has been rapid during the ~20 years since its arrival; spread of the pathogen is aerial with extensive local dispersal (20–50 m) and less frequent but consistent long-distance events of 1–3 km (Hansen et al. 2008; Meentemeyer et al. 2008a; Garbelotto and Hayden 2012). *Phytophthora ramorum* causes the disease sudden oak death (Rizzo et al. 2002) which has been responsible for a regional-scale population decline of tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae); formerly *Lithocarpus densiflorus* (Manos et al. 2008), as well as the mortality of hundreds of thousands of oak trees (*Quercus* spp.) from the red oak clade, primarily coast live oak (*Q. agrifolia* Née) throughout the invaded areas of California (Meentemeyer et al 2008b; Davis et al. 2010; Metz et al. 2012).

Phytophthora ramorum has a broad host range, with over 140 known native and non-native plants including many trees, shrubs, forbs, grasses, and ferns common to the California floristic province (Grünwald et al. 2012). In California, sporulation occurs on multiple hosts, the most important of which is the common forest tree California bay laurel (*Umbellularia californica*, Hook. and Arn.). Sporulation also occurs on tanoak twig lesions at levels sufficient to spread the pathogen as well as cause bole-lesions which are the principal infections that lead to mortality in tanoak and oak. In California forests, sporulation can be an order of magnitude greater on bay laurel and peak amounts for both species occur during warm spring rain events (Davidson et al. 2005, 2008; Mascheretti et al. 2008). In California, both tanoak and bay laurel are central to pathogen spread within stands and across landscapes although risk of pathogen establishment and spread rates are more strongly affected by bay laurel density (Meentemeyer et al. 2008a; Davidson et al. 2011). Host impacts are strikingly different across species: *P. ramorum* infection often leads to tanoak mortality, but has no known deleterious impacts on bay laurel at either the individual or the population level (DiLeo et al. 2009; Cobb et al. 2010). In California, the stand-level densities of tanoak and bay laurel greatly influence tanoak mortality rate and population decline (Cobb et al. 2012b). Pathogen dynamics are somewhat different in Oregon forests where sporulation occurs year-round and bay laurel does not play a significant role in spread at the landscape scale (Hansen et al. 2008). However, in both Oregon and California forests, tanoak population decline is expected across much of its endemic range (Fig. 1; Meentemeyer et al. 2004; Václavík et al. 2010; Lamsal et al. 2011; Meentemeyer et al. 2011; Dillon et al. this volume).

Independent studies using repeated measurements of individual tanoak show that mortality rate of infected trees increases with size (McPherson et al. 2010; Cobb et al. 2012b). Although the mechanism for this increase is not known, the pathogen's selective removal of large tanoak leads to rapid, stand-level loss of tanoak biomass in *P. ramorum* invaded stands (Ramage et al. 2011; Cobb et al. 2012b; Metz et al. 2012). Basic biodiversity information such as acorn production rates, knowledge of species obligate to tanoak, and other ecological functions provisioned by mature tanoak trees have not been reported in the peer-reviewed literature. However, ecological functions of individual trees often increase with tree biomass, which scales exponentially with tree size (Lamsal et al. 2011; Cobb et al. 2012a). Selective removal of large trees is likely to accelerate loss of ecosystem characteristics directly associated with tanoak, such as ectomycorrhizal associations or provisioning of habitat for other native flora and fauna which rely on or are obligate to tanoak (Rizzo et al. 2005; Bergemann and Garbelotto 2006; Cobb et al. 2012b; Wright and Dodd this volume).

The distribution of tanoak varies across the landscapes of the California floristic province (Fig. 1), increasing in prevalence with latitude up to the northern range limit of the species in

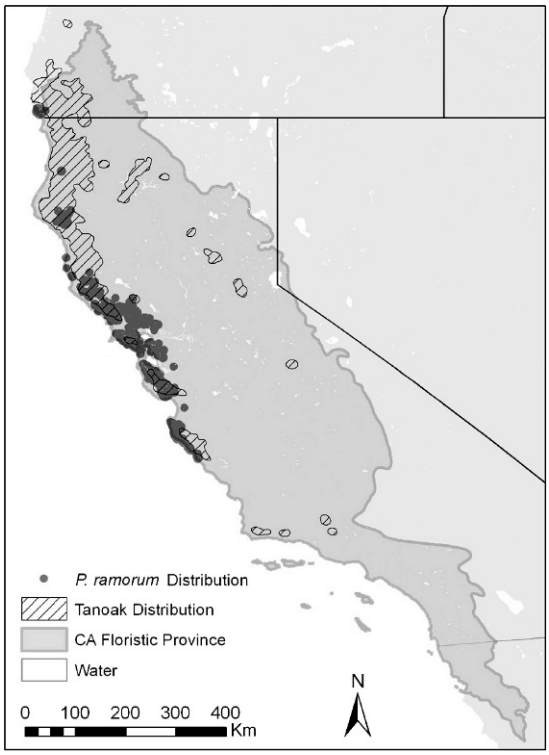

FIG. 1. The California floristic province with approximate extent of *P. ramorum* invasion and tanoak distribution. *Phytophthora ramorum* points are confirmed infected trees; the map was generated with publically available data accessed in January 25, 2013 from oakmapper.org and suddenoakdeath.org.

southwestern Oregon. Several notable disjunct populations are located in the northern Sierra Nevada foothills and in small, isolated populations in Santa Barbara County, CA (Fig. 1; Lamsal et al. 2011; Meentemeyer et al. 2011; Dillon et al. this volume). While *P. ramorum* establishment and disease emergence have been extensive in central coastal California, *P. ramorum* has not yet invaded the majority of the tanoak range (Meentemeyer et al. 2011). However, the pathogen is expected to invade culturally important, highly susceptible North Coast forests over the next several decades reflecting the opportunity and importance of planning and employing a proactive strategy to reduce disease impacts.

Phytophthora ramorum populations in newly invaded areas are often below thresholds where the pathogen can be detected using landscape-scale surveys. These cryptic invasions play an important role in landscape-level spread, and represent a critical challenge to eradication attempts and other pathogen-centric disease management strategies (Filipe et al. 2012). For many invasive pathogens, rapid responses that match the scale of the outbreak increase the likelihood of successful eradication or the efficacy of slowing pathogen spread (Gilligan and van den Bosch 2008; Filipe et al. 2012). This requires rigorous study to identify effective management actions and minimize unintended impacts that are counter to the overall goal. Our intent here is not to review individual management techniques and their efficacy, rather we seek to bring together different management actions and place them in the context of local disease conditions and management goals. We begin by highlighting several historical and contemporary mistakes in forest disease management and emphasize the importance of incorporating historical lessons into sudden oak death management. We suggest how judicious use of established techniques can alter disease conditions and increase tanoak population resiliency across different forest types and community assemblages. We use the term 'resiliency' to mean retention of tanoak and the ecological functions provisioned by this tree and recognize that specific management objectives will vary among ownerships, agencies and situations. Part of our goal is to aid in the selection of when, where, and how to increase tanoak resiliency with existing management actions. We stress that management resources, land-owner cooperation, and chemical treatments will often be insufficient to control pathogen spread and disease emergence. Rather, when management actions aim to retain tanoak as a component of forest structure and composition, we advocate for integration of host-centric actions into management strategies. These strategies rely on increasing tanoak resiliency through stand manipulation to reduce sporulation, increasing distance among tanoak to reduce the probability of transmission, and identification of resistance in tanoak populations. Lastly, we stress that continuous feedback among sudden oak death research, management, and pathogen monitoring is needed to maintain tanoak as a component of biodiversity in many forests and to reduce the spread of *P. ramorum*.

LEARNING FROM FAILURE

The consequences of invasion of *Cryphonectria parasitica* (Murrill) Barr and the resulting disease, chestnut blight, is a notorious example of disease causing North American overstory tree loss (Anagnostakis 1987). Experience with chestnut blight can inform present-day challenges posed by exotic pathogens, especially *P. ramorum*. *Cryphonectria parasitica* is a generalist pathogen, selectively removes American chestnut (*Castanea dentata* Marsh.) from forest stands, and readily disperses across local and regional scales (Loo 2009). Within 30 years of introduction by the pathogen, these characteristics caused a widespread

decline of American chestnut and loss of the considerable ecological, cultural, and economic value that was associated with this species (Anagnostakis 1987; Freinkel 2007). The rate and extent of chestnut decline was unprecedented when the disease emerged circa 1900, and remains as one of the most infamous diseases in North American forest history because of the significant economic and cultural impacts caused by the epidemic (Freinkel 2007).

Chestnut blight is also notable as a case of failed management that stemmed from decisions with incomplete knowledge of pathogen biology and host relationships. For example, the state of Pennsylvania initiated an ambitious plan to remove infected chestnut trees and create a barrier zone with the intention of restricting pathogen spread into the western portion of the state (Freinkel 2007). However, understanding of *C. parasitica* transmission pathways, estimates of pathogen spread rate, and the capacity to detect cryptic infection were all inadequate and pathogen spread was unabated. A consequence of dedicated and extensive chestnut cutting was, at a minimum, an acceleration of chestnut removal from the overstory of these forests, an action that may have even expedited pathogen spread (Freinkel 2007). Today, American chestnut remains common in eastern forests as an understory shrub, but overstory trees and their associated ecological functions such as provisioning of large, nutritious, low-tannin nuts, have been lost from most of the species' former range (Paillet 2002). A full accounting of changes to native flora and fauna is hampered by the lack of natural history investigations of species associated with the tree prior to the epidemic. Although other species replaced American chestnut in the overstory, loss of chestnut probably impacted small mammal and invertebrate populations and may have resulted in the extinction of several insects obligate to this overstory tree (Orwig 2002; Ellison et al. 2005).

Regrettably, chestnut blight is not the sole example of misdirected or ineffective management actions. Eastern hemlock (*Tsuga canadensis* L.) forests are in decline across its native range from Georgia to southern Vermont and New Hampshire due to the invasion and subsequent regional-scale outbreak of hemlock woolly adelgid (*Adelges tsugae* Annand), an exotic insect pest (Fitzpatrick et al. 2012; Orwig et al. 2012). Eastern hemlock decline has been accelerated in southern New England by pre-emptive hemlock harvesting, regardless of stand invasion status (Orwig et al. 2002). From a conservation perspective, indiscriminant host tree removal will accelerate population declines of trees threatened by insect or disease outbreak as well as degrade the genetic diversity of remaining populations (Foster and Orwig 2006; Broadhurst et al. 2008).

Natural resource management relies on adaptive management to address imprecise knowledge and to avoid the field's own version of "the twin traps of overtreatment and therapeutic nihilism" (e.g., the Hippocratic Oath, Edelstein 1943). The backbone of this approach is feedback among researchers, managers, and policy makers who together establish experimental treatments, quantify their efficacy, and direct resources. An analogous framework was not in place to address either chestnut blight or much of the management response to the decline of eastern hemlock populations. In the case of sudden oak death, an adaptive management approach is more likely to identify treatments which judiciously use limited management resources (Filipe et al. 2012) and avoid loss of genetic diversity (Hayden et al. 2011; Kjær et al. 2012) in comparison to reactive actions such as the attempt to construct a barrier to arrest the spread of chestnut blight (Freinkel 2007). The benefits of this approach are already very clear; one of the most important initial breakthroughs regarding sudden oak death was the discovery that *P. ramorum* was the cause of widespread oak and tanoak mortality (Rizzo et al. 2002) which laid to rest spurious hypotheses that the mortality was caused by insects or native pathogens. Adaptive management further relies on clear articulation of management objectives while also recognizing the need to evaluate treatments to achieve these objectives. The extent of the *P. ramorum* invasion suggests management goals must be comprehensive enough to match the magnitude of the disease and recognize technical and practical limitations in order to identify where, when, and in what combination available techniques will be most effective in slowing pathogen spread and maintaining the cultural and ecological value of tanoak.

Tanoak Mortality: Patterns and Impacts

Tanoak has low timber value and the economic value is further lowered because of competition with commercial timber species, i.e., Douglas fir, *Pseudotsuga menziesii* (Mirb.) Franco (Harrington and Tappeinier 2009). Before the 1950s, tanoak-bark extracted tannins were important to California's leather industry, but the advent of synthetic tanning compounds has shifted the economic perspective on this tree (Alexander and Lee 2010; Bowcutt 2011). Prior to the emergence of sudden oak death, most recent research involving tanoak focused on quantifying and reducing competition with coniferous species central to commercial timber production often through the use of herbicides (Harrington and Tappeiner 2009; Bowcutt 2011). Little basic research has been conducted on tanoak and the measurement of growth characteristics, their relationship with disease resistance, and descrip-

tion of flora and fauna obligate to or associated with tanoak have only recently been undertaken (Bergemann and Garbelotto 2006; Cobb et al. 2012b; Wright and Dodd this volume). Although the risk of pathogen-driven, regional-scale removal of tanoak from the forest overstory is becoming increasingly clear, we face a critical lack of understanding as to what and how many species may be threatened by the widespread mortality of this common tree.

Even though *P. ramorum* has not yet invaded most of the tanoak range (Meentemeyer et al. 2011), the pathogen has significantly impacted tanoak from the Big Sur region (Monterey County, CA) north to Sonoma County, CA. In these regions, sudden oak death has caused widespread loss of overstory tanoak trees, significant shifts in overall forest composition, and outright extirpation of tanoak from some stands (Meentemeyer et al. 2008b; Ramage et al. 2011; Cobb et al. 2012b; Metz et al. 2012). These forests occur across topographically, environmentally, and biologically diverse landscapes where several different conifer and broad-leafed trees are likely to experience increases in density, biomass, and dominance as a result of the decline of tanoak populations. Metz et al. (2012) and Cobb et al. (2010) examined sudden oak death-impacted California stands ranging from relatively dry, mixed-evergreen forests to relatively cool and moist redwood-dominated forests; in both studies, tanoak mortality was associated with increased dominance of bay laurel. Given that *P. ramorum* establishment and disease emergence is associated with bay laurel abundance (Maloney et al. 2005; Meentemeyer et al. 2008a; Cobb et al. 2010), bay laurel is often situated to benefit from tanoak population decline. This shift in species composition has the additional impact of improving habitat for *P. ramorum*, suggesting that the pathogen will persist even if tanoak becomes locally extinct (Cobb et al. 2012b).

Additional shifts in species composition due to sudden oak death are also likely in North Coast forests from Mendocino County to Curry County, OR. While the current distribution and prevalence of *P. ramorum* in the North Coast is lower compared to the central coast where the pathogen was initially introduced (Fig. 1), pathogen invasion and associated tanoak mortality have mobilized management actions by state, private, and federal land managers (Rizzo et al. 2005; Valachovic et al. 2008; Goheen et al. 2009). In North Coast forests, Douglas-fir and redwood (*Sequoia sempervirens* [Lamb. ex D. Don] Endl.) have greater importance in terms of density, biomass, and basis in local economies; these characteristics are likely to favor an increase in dominance by these species in areas following tanoak mortality. Notably, forests in Del Norte County, CA and Curry County, OR are notable for their lower abundance of bay laurel especially compared to forests in the central coast, especially Big Sur (Monterey County) and Sonoma County (Lamsal et al. 2011). *Phytophthora ramorum* is likely to invade and cause disease even when tanoak is the sole sporulation-supporting species (Rizzo et al. 2005; Goheen et al. 2009; Meentemeyer et al. 2011) although mortality rates are likely to eventually slow because of a negative feedback on pathogen populations when hosts die (Cobb et al. 2012b). Additionally, pathogen-killed trees or cut tanoak often develop prolific basal sprouts (Harrington and Tappeiner 2009; Cobb et al. 2010; Ramage et al. 2011), which may be sufficient to perpetuate pathogen populations and maintain spread in stands that do not include bay laurel (Cobb et al. 2012b).

Sudden oak death also has far-reaching economic, cultural, and ecological impacts. Spread of *P. ramorum* via ornamental nursery plants was responsible for the initial introduction to California and continues to play a role in spread within the United States and internationally (Garbelotto and Hayden 2012; Grünwald et al. 2012). Regulation of nursery stock, a costly but necessary management action, has been initiated to address this pathway of pathogen spread (see also Alexander and Lee 2010). For individual property owners, the loss of oak and tanoak directly impacts property values (Kovacs et al. 2011), and increased disease-caused fuel loadings at wildland-urban interfaces could augment the risk of property loss during wildfire (Metz et al. 2011; Valachovic et al. 2011). Similarly to American chestnut, tanoak has historically been an important component of human nutrition and local culture; tanoak resources retain significant cultural importance to many Native American communities in California (Bowcutt 2011). From a functional ecology perspective, tanoak is the sole ectomycorrhizal host in many forests (Rizzo et al. 2005) and supports ectomycorrhizal diversity on par with *Quercus* species (Bergemann and Garbelotto 2006). Tanoak-wildlife associations are poorly described, but the large nutritious acorns are likely used by many species and a recently described insect pollination pathway suggests yet undocumented insect communities and ecological interactions that may be impacted by the loss of tanoak (Wright and Dodd this volume).

Disease Treatments: Models and Experiments

A central problem of managing emergent pathogens is that the available biological or epidemiological understanding is usually insufficient to control disease at the earliest stages of

outbreak. When epidemiological understanding is flawed, seemingly appropriate management actions may actually increase pathogen spread and disease intensity (Ndeffo Mabah and Gilligan 2010), or simply be insufficient to control pathogen spread despite large investments of management resources (Filipe et al. 2012). Research is essential to addressing these inefficiencies and maximizing the benefits of disease management. Epidemiological models can help guide management by estimating the efficacy of treatments and forecasting the spread and impacts of pathogens based on current data. Of equal importance are field experiments designed to test treatment efficacy and model structure. Field experiments are also essential in establishing a feedback among treatment application, monitoring, and research efforts, including modeling, which comprise adaptive management. For example, field studies improve the understanding of pathogen biology and epidemiology, which can be applied in models to identify the most appropriate treatments thus generating hypotheses that are testable with further field experiments (Sniezko 2006; Gilligan and van den Bosch 2008).

Models of disease risk based on host distribution, environmental conditions, and pathogen spread are useful tools for understanding where and when tanoak mortality will occur. The initial risk models for sudden oak death in California and Oregon (Meentemeyer et al. 2004; Václavík et al. 2010) identified locations that are optimal for pathogen establishment. These models have been reliable tools because the underlying epidemiological assumptions have subsequently been demonstrated to be biologically accurate (Meentemeyer et al. 2012). More recent models incorporate spread following establishment in a given set of locations (Meentemeyer et al. 2011; Filipe et al. 2012). These dynamical models enable estimation of a probable time of invasion for specific tanoak populations and suggest that the majority of the tanoak range is at risk of pathogen invasion and disease emergence over the next several decades. In conjunction with tanoak distribution models (Lamsal et al. 2011), these *P. ramorum* spread models hold promise to predict the timing of disease emergence, risk of tanoak population decline (Dillon et al. this volume), and changes in ecosystem functions such as carbon cycling (Cobb et al. 2012a). Further development of models and datasets to identify levels of biodiversity, cultural, or ecological value would be useful as an overlay with risk models (e.g., Meentemeyer et al. 2004; Václavík et al. 2010) to aid in identification of specific stands where resources for tanoak conservation should be focused.

Management of isolated outbreaks of *P. ramorum* has largely relied on removal of infected hosts to reduce inoculum. These treatments are often combined with removal of susceptible hosts in the surrounding area in an attempt to account for undetected infections (treatment buffers) and have been implemented in southern Humboldt County, Redwood Valley (northern Humboldt County), and Curry County, OR (Hansen et al. 2008; Valachovic et al. 2008). These treatments, when done at an early disease stage, are clearly effective in reducing local pathogen prevalence and the likelihood of spread within treated stands (Hansen et al. 2008; Goheen et al. 2009), suggesting overall rates of spread in the landscape are slowed by aggressive and rapid interventions (Filipe et al. 2012). However, in each case the pathogen has not been eradicated by the treatments and spread throughout the landscape has continued. Undetected, or cryptic, infections are an important source of this continued spread into uninfected stands (Filipe et al. 2012). The broad *P. ramorum* host range, asymmetric impacts on hosts (some hosts support sporulation but are not killed by infections), cryptic infections, and survival in habitats such as soil and watercourses make eradication from a region exceedingly difficult (Hansen et al. 2008; Goheen et al. 2009). However, disease management need not attain 100% pathogen reduction to be effective. Slowing pathogen spread within the landscape is valuable, as it provides time to plan and proactively manage for ecosystem impacts, such as increased fuel loads, stress to biodiversity associated with tanoak, and the accompanying loss of acorn production.

As new isolated outbreaks emerge in northern California and southern Oregon it will become necessary to focus limited management resources to achieve specific cultural and biological conservation goals. Inoculum-reduction treatments such as attempts to locally eradicate the pathogen are expensive to apply and demand significant investments from land managers, researchers, and field staff (e.g., Valachovic et al. 2008) suggesting a shift from pathogen-centric to host-centric management will be more effective to reaching conservation goals when an individual outbreak reaches some yet undefined threshold. However, limiting spread into specific uninvaded landscapes will be aided by attempting eradication of isolated outbreaks as these treatments are likely to reduce inoculum (Filipe et al. 2012). For example, the Redwood Valley outbreak is strategically important given its proximity to culturally and ecologically valuable tanoak stands on tribal lands, within Redwood National Park, and an other nearby public and private lands with large tanoak populations. Pathogen-centric treatments in Redwood Valley have likely slowed *P. ramorum* spread into these valuable nearby areas by reducing inoculum loads. However, further removal of isolated infections outside of the treated area may be needed to attain maximum inoculum reduction or as part of

host-centric management that increases the resiliency of these tanoak populations and protects their associated biodiversity, ecological function, and cultural value. When to shift from pathogen-centric to host-centric management or identifying the optimal balance of each approach is an outstanding question for land managers working to address
P. ramorum and other forest disease outbreaks.

Epidemiological models can inform both pathogen-centric and host-centric management actions (Fig. 2). Often, treatments for each management type will use the same techniques (stand thinning, infected host removal), but the timing and location of treatments may differ. Some insights into the design of these treatments can be gleaned from stand-level epidemiological models which often describe the amount of inoculum that can reach a given individual host through the concept of force of infection (FOI) (for examples, see Meentemeyer et al. 2011; Cobb et al 2012a; Filipe et al. 2012). FOI often has two components: the number of infected hosts weighted by a dispersal kernel (a function of relative distance between hosts), and the rate at which each infected individual transmits the pathogen to susceptible individuals. Sporulation rates vary across host species and are dependent on environmental conditions (rainfall, temperature), therefore formulations of FOI may include species specific pathogen spread factors as well as environmental variation. In addition, the length of time an individual remains infectious, known as the infectious period, also determines the number of individuals which spread a pathogen. Infectious period is poorly quantified for bay laurel and tanoak, but models demonstrate that this parameter can influence rates of pathogen spread at stand-to-landscape scales as well as the persistence of the pathogen within invaded stands (Cobb et al. 2012b; Filipe et al. 2012). The rate of spread and tanoak mortality within a population or landscape increases with FOI and management actions can be directed at one or more of its components. Therefore, the achievement of specific management goals will be improved by understanding which components of FOI are being acted upon by a given treatment.

In general, eradication treatments reduce the number of hosts spreading the pathogen but do not change the rate of spread from each infected host. This is important because infected individuals that remain untreated support continued local and regional pathogen spread (Hansen et al. 2008; Filipe et al. 2012). Prophylactic chemical treatments, such as phosphonate injections, have been shown to reduce the susceptibility of individual tanoak against *P. ramorum* (Garbelotto and Schmidt 2009); while further research is needed on the efficacy, longevity, phytotoxicity and environmental interactions with phosphonate, in general protection of individual trees has been shown to be effective in slowing spread within some populations. Genetic variation within host species may also reduce susceptibility and subsequent pathogen spread. Bay laurel populations in Oregon (known locally as ''myrtlewood'') are not as abundant, susceptible, and consequently not as important in spreading *P. ramorum* as they are in much of California (Hansen et al. 2005; Hüberli et al. 2011), suggesting genetically based differences among host populations may influence disease severity in different regions. Similarly, Hayden et al. (2010) found evidence of resistance to *P. ramorum* infection within tanoak populations, indicating that patterns of resistance could influence tanoak susceptibility or mortality within stands. Similarly to chemical protection treatments, further work is needed to understand how resistance influences pathogen spread rates.

Recent modeling results suggest the degree to which management actions reduce *P. ramorum* infection rates can directly influence the number of tanoak that maintain an overstory canopy position in *P. ramorum* invaded forests (Fig. 3). Following a line of tested epidemiological models (Filipe and Gibson 2001; Gilligan and van den Bosch 2008; Ndeffo Mabah and Gilligan 2010; Filipe et al. 2012), Cobb et al. (2012a) developed a stand level model of disease outbreak with dynamic vegetation composition and tanoak size structure. These models demonstrate that a desired outcome of host-centric management may be feasible: retention of tanoak, including large tanoak, even though *P. ramorum* has invaded the stand. The model shows a critical level of tanoak density (tanoak stems ha^{-1}) below which the pathogen is not able to maintain spread from host to host and consequently, does not diminish tanoak populations or remove overstory tanoak. These results assume bay laurel is not present in, or has been removed from the stand and that remaining tanoak trees are evenly distributed across the stand. The models also suggest that slowed host-to-host infection rates result in better retention of the habitat and ecosystem function provided by tanoak (Fig. 2). Slower infection rates could be achieved by increasing tanoak resistance or stand-level application of effective chemical protection. However, identification of tanoak resistance prior to treatments is currently limited by the lack of molecular-resistance markers; this technical limitation must be overcome to use resistance measurements in broader-scale preemptive treatments. Regardless, the epidemic threshold predictions of the model (e.g., Fig. 3) should still be tested with field experiments, including current phosphonate applications (Garbelotto and Schmidt 2009) and tanoak thinning treatments to understand under which conditions these

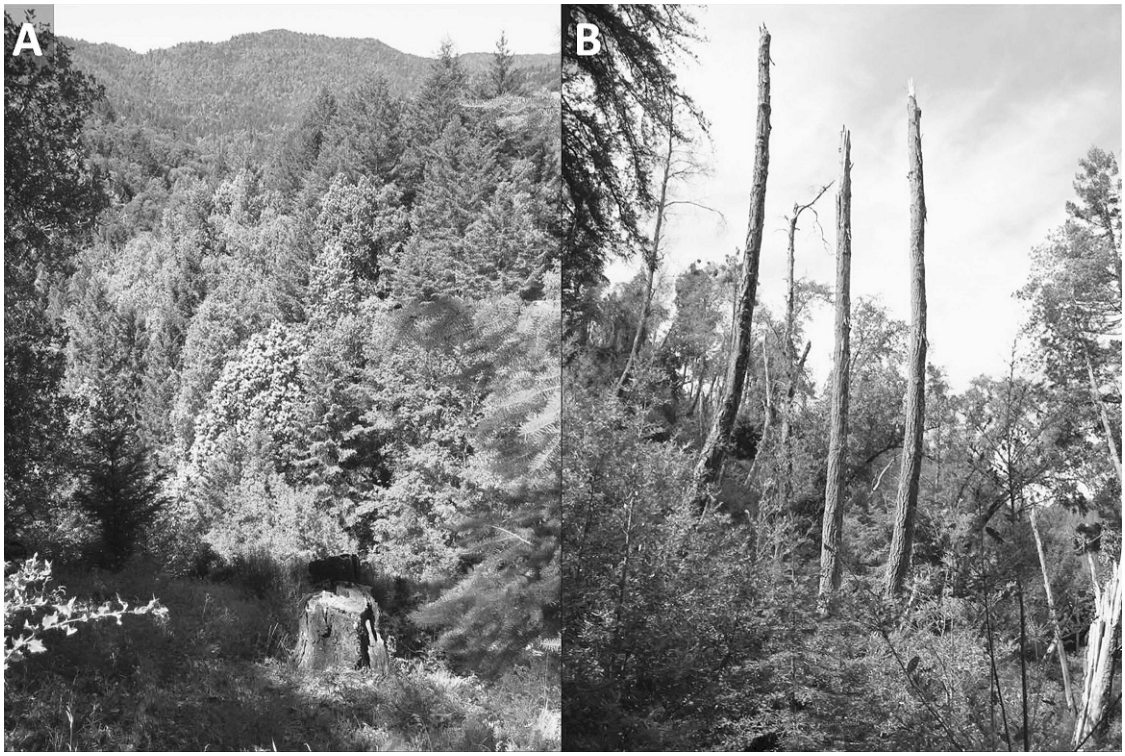

FIG. 2. Landscapes and stands that have not been invaded by *Phytophthora ramorum* (A) offer different opportunities and require different management approaches compared to invaded forests (B) when the goal is to retain tanoak and its associated flora and fauna. Variation in the likelihood of pathogen invasion into pathogen-free landscapes and stands is largely driven by proximity to pathogen invaded stands and community composition. Community composition can be manipulated to slow invasion and disease impacts. Disease impacted stands (e.g., B) require a host-centric management approach that avoids removal of surviving tanoak, protects potential genetically based resistance, and retains ecological functions and species obligate to tanoak. In both cases, successful conservation of tanoak will require continuous feedback between research and management to establish baselines, test hypotheses, and determine the efficacy of management actions.

density thresholds are overcome through long-distance dispersal events (Filipe and Gibson 2001; Gilligan and van den Bosch 2008), sporulation from species not thought to be epidemiologically significant, or human-mediated transport of infected plants (Cobb et al. 2012b; Filipe et al. 2012).

MANAGEMENT GOALS DEFINED: CONSERVE BIODIVERSITY ASSOCIATED WITH TANOAK

Phytophthora ramorum has, and will continue to alter the distribution, prevalence, and biomass of tanoak across California and Oregon. A difficult reality of this disease is that much of this tanoak loss has and will occur regardless of the amount and efficacy of management actions. However, the available management tools and current understanding of the disease may be sufficient to avoid the worst possible outcomes such as the removal of large trees and consequent loss of ecological function (cf., Orwig 2002; Ellison et al. 2005). Management efficacy should be measured at least in part by the success in retaining biodiversity and function associated with tanoak; retention of tanoak is likely to be easier in community or environmental conditions where pathogen spread is lower. Currently, many culturally and ecologically significant tanoak populations have yet to be invaded by *P. ramorum* (Lamsal et al. 2011; Meentemeyer et al. 2011), and timely actions have the potential to reduce future impacts of this disease.

Invaded Stands and Landscapes

Invasion status is a useful first distinction for management efforts and techniques as *P. ramorum* invasion of a stand eliminates the usefulness of many treatments (Fig. 2). For example, prophylactic treatments are ineffective on trees that are already infected (Garbelotto and Schmidt 2009). Similarly, limiting inoculum influx into stands where the pathogen is already established may have a minimal effect in reducing disease because most inoculum is produced

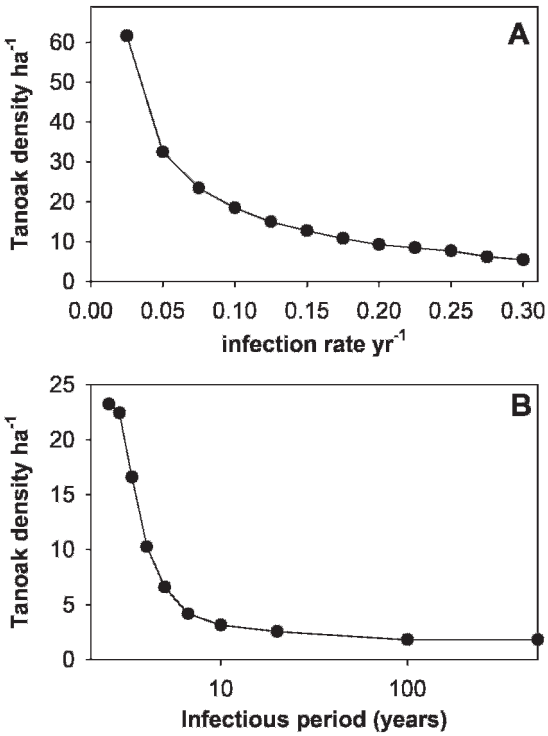

FIG. 3. The epidemiological characteristics of *Phytophthora ramorum* affect the threshold density of large tanoak trees (all trees > 30 cm diameter at 1.3 m) below which *P. ramorum* outbreak cannot be sustained in a parameterized epidemiological model (Cobb et al. 2012b). (A) The epidemic thresholds for tanoak density decreases with increasing infection rate. (B) Similarly, the density of large tanoak decreases with increasing infectious period (log scale; infection rate was kept constant at 0.3).

locally (Davidson et al. 2005; Mascheretti et al. 2008; Cobb et al. 2012b). *Phytophthora ramorum*-invaded stands are potential pathways of spread and the quarantine of infected, sporulation-supporting plants from these stands is a judicious action to reduce, though not eliminate, this pathway of long-distance pathogen spread.

Managers increasingly face isolated outbreaks that threaten to spread into the wider landscape. In Oregon, emphasis has been placed on monitoring and rapid implementation of eradication-type treatments within a designated quarantine zone (Hansen et al. 2008). In northern Humboldt County, the isolated outbreak in Redwood Valley was also addressed with a rapid eradication attempt response, but discovery of the pathogen outside of the treatment area raises the question of what follow-up treatments are needed to maximize the benefit of the initial intervention. The model designed by Filipe et al. (2012) was used to examine a number of landscape-level treatments including inoculum-reduction treatments (such as eradication attempts) at the initial invasion or preemptive host removal ahead of the infection, and construction of a host-free barrier at the landscape-scale. Among these treatments, preemptive removal of hosts ahead of the initial invasion and removal of infected hosts at the initial invasion, similar to
the strategy in Oregon, were found to be most effective in slowing *P. ramorum* spread as long as the treatments were applied early during the epidemic and the scale of treatments matched the scale of the invaded area. The landscape-barrier treatments were not effective in slowing the spread or protecting particular areas because continued spread from undetected infections, or long-distance dispersal across the barrier overcame these treatments.

In regions with the most extensive *P. ramorum* invasion, eradication attempts are a poor choice of management. In highly invaded stands from Big Sur to Sonoma County, *P. ramorum* populations are so large and widely established that any benefit from local removal of infected trees would be overcome by reinvasion from adjacent stands. Eradication attempts always represent a tradeoff between causing tree mortality through cutting and herbicides vs. allowing the pathogen to cause tree mortality. In highly invaded landscapes, management should be aimed at reducing disease impacts and protecting high-value individual stands or trees. For example, in the fire-prone Big Sur landscape, high-density tanoak stands are problematic because sudden oak death generates significant amounts of dead fuels (Metz et al. 2011; Cobb et al. 2012a). Stands with accumulated dead fuels also suffered greater amounts of soil damage during the 2008 Basin Fire (Metz et al. 2011; Big Sur). These patterns suggest treatments which increase resiliency of tanoak populations to *P. ramorum* while also reducing dead fuels may have substantial benefits in terms of reducing ecological costs caused by wildfire in disease impacted forests. Disease-generated fuels are dependent on the rate of mortality and the amount of tanoak biomass present before invasion. This suggests preemptive reduction of tanoak density combined with bay laurel removal is likely to slow disease-driven dead fuel accumulation and reduce maximum amounts (Valachovic et al. 2011; Cobb et al. 2012a). These treatments could easily be structured to retain specific trees such as larger or more resistant individuals and increase spacing between individual trees in order to reduce within-stand spread. However, the effects of increasing stand openness are unresolved; pathogen dispersal may be less impeded in more open stands or, conversely, microclimate conditions could be less suitable to sporulation and infection (Rizzo et al. 2005). This uncertainty reflects the need for further experimental treatments to

understand the interactions of management actions with the epidemiology of *P. ramorum*. The Big Sur region is a strong candidate for experimental fuel and disease reduction treatments given the frequency of fire and the extent of sudden oak death in this region (Rizzo et al. 2005; Meentemeyer et al. 2008b; Metz et al. 2011).

Uninvaded Stands and Landscapes

The most effective strategy to prevent impacts of sudden oak death is to prevent *P. ramorum* invasions into new, at risk environments altogether. However, the history of *P. ramorum* is notable for numerous and often surprising invasions (Werres et al. 2001; Rizzo et al. 2005; Hansen et al. 2008; Brasier and Webber 2010) and current models suggest many stands and landscapes will be invaded in the coming decades (Meentemeyer et al. 2011). Proactive management of uninvaded stands may be much easier to implement where manipulation of stand and community structure are already planned to achieve management goals apart from disease. Where ever possible, host-centric management should aim to achieve multiple goals (Fig. 2).

The design, implementation, and evaluation of preemptive disease treatments are difficult from a research perspective because invasion dynamics are idiosyncratic. Without pathogen invasion of a stand–which is never guaranteed–the efficacy of a preemptive treatment cannot be evaluated. At the same time, intentional pathogen introduction to uninvaded areas must be ruled out on ethical grounds. We suggest two actions that help overcome this practical problem. First, disease management should be put in the context of long-term management goals, such as the reduction of fuel loads, growth of timber species, desired community composition or canopy structure, and/or enhancement of biodiversity. Second, further effort should be allocated to developing models for management evaluation. Models provide insight into the consequences of landscape-level management when experiments at this spatial and/or temporal scale are not possible (Meentemeyer et al. 2012). Lastly, to implement treatments for emerging outbreaks, we caution that management goals must be consistent with the priorities and level of cooperation within the local community (Alexander and Lee 2010).

Genetically-Based Resistance

Genetically-based resistance to *P. ramorum* in tanoak has the potential to be of great practical value. Stands with greater innate resistance are likely to experience lower rates of pathogen spread, fewer disease impacts, and have larger tanoak populations following challenge by *P. ramorum* (Fig. 3). In a study quantifying patterns of tanoak resistance, Hayden et al. (2010; 2011) found variation in susceptibility within tanoak populations, but fairly equivalent susceptibility among populations. If this pattern is broadly representative of tanoak genetic patterns, individual stands are unlikely to resist invasion by *P. ramorum* on the basis of genetic composition alone. However, resources from within stands could be used to maximize tanoak population resiliency in conjunction with other treatments, or to develop less susceptible tanoak growing stock suitable for restoration in *P. ramorum*-invaded areas and preemptive planting treatments in uninvaded landscapes. Further work is needed to increase the ease and rate of identifying tanoak resistance, to determine if rates of sporulation differ in less susceptible trees, and to develop tanoak suitable for restoration.

Where the management goal is to retain biodiversity and function associated with tanoak, treatments need not conserve every living tanoak. Rather, these treatments must retain tanoak populations large enough to maintain both the species and its valued ecological functions. Moreover, sampling and conservation efforts should be targeted to retain genetic diversity across regions of known tanoak differentiation. A genetic analysis of neutral markers by Nettel et al. (2009) has shown deep divisions in chloroplast markers between central coastal California tanoak populations and northern coastal-California/Klamath/Sierra tanoak populations, along with evidence of considerable pollen flow among populations. The variance structure in neutral nuclear genetic markers was similar to the variance in resistance measured on leaves cut from wild trees, with more variance within populations than there was among populations (Hayden et al. 2011). In pathogen-invaded landscapes, disease may be a useful force to select for more resistant tanoak. Hayden et al. (2011) suggested that greater prevalence of resistance at some sites could reflect a selection effect of the pathogen, and McPherson et al. (2010) reported the survival of several mature tanoaks after nearly a decade of monitoring in a *P. ramorum*-invaded forest. These results argue for retention of tanoak where *P. ramorum* has already killed a large portion of the tanoak population. In highly disease impacted stands, treatments which remove surviving trees would clearly exacerbate the loss of tanoak and may reduce genetic resources important for developing molecular markers of resistance and restoration planting stock.

Protect High Conservation Value Stands

Targeted conservation acquisitions, such as land purchases or conservation easements, are

often used to protect rare species or habitat and could be effective in some cases for retaining high-value tanoak populations, such as uniquely located or isolated stands threatened by development. For example, the most southern tanoak populations occur in Santa Barbara County in small and relatively high-elevation stands and a larger geographically-separated tanoak population occurs in the foothills of the central to northern Sierra Nevada range (Fig. 1). In both cases, these tanoak populations hold important genetic diversity, and have lower likelihood of *P. ramorum* invasion because of their isolation, poorer environmental conditions for pathogen sporulation, and low spread risk from adjacent stands (Nettel et al. 2009; Dodd et al. 2010; Meentemeyer et al. 2011). However easements can restrict available management tools needed to slow pathogen spread or protect individual trees and will not be appropriate for many sites or as a method to protect large tanoak populations.

Conclusions–Guidelines for Tanoak Conservation

The goal of tanoak conservation requires an adaptive management approach where management actions and research are designed and conducted with synergistic feedback. We suggest a set of overarching management and research guidelines to achieve this objective.

1. Define the management goals for *P. ramorum* and integrate tanoak conservation as part of a broader vision of landscape management melding treatments for fire, wildlife, aesthetics, and other stand-level goals.
2. Continue efforts to develop field, lab, and molecular tools to identify resistance to *P. ramorum* and develop methodologies to identify and map resistance in the field. Combine this information with stand-to-landscape level epidemiological models to identify tanoak stands with the greatest resiliency to disease.
3. Conduct field experiments to evaluate the efficacy of host-centric management treatments to retain tanoak in *P. ramorum* invaded stands. In combination, conduct epidemiological and bio-economic analysis to identify the optimal timing to shift from pathogen-centric management (eradication and similar slow-the-spread treatments) to host-centric management aimed at retaining tanoak in invaded lands.
4. In uninvaded stands or landscapes where tanoak is highly valued, increase the resiliency of these populations by reducing the potential for pathogen spread (i.e., reduce bay and tanoak densities) while retaining the most resistant tanoak individuals.
5. Develop host-centric, ameliorative and proactive treatments that reduce the ecological costs of disease for: fire prone forests, regions with the greatest potential ecological impact of disease (i.e., areas with high tanoak importance and biomass), and lands where tanoak has the greatest cultural value.
6. Work with the public to increase appreciation and interest in tanoak and to encourage further participation in pathogen monitoring and disease management (cf., Alexander and Lee 2010).
7. Increase collaboration among state and federal agencies, and public-private partnerships to support management in tanoak stands.
8. Expect the unexpected. *Phytophthora ramorum* is a remarkably well-adapted pathogen for which the native host or geographic range has not yet been identified. The importance of pathogen characteristics often becomes clear only after a new management problem has emerged.

Acknowledgments

Katherine Hayden and Joao Filipe contributed to this paper as second authors. The authors are grateful for comments and criticism to a previous version of this paper from four anonymous reviewers. The paper has also been improved by discussions with H. Mehl, M. Metz, and B. Twieg. We thank C. DeLong for designing Fig. 1. This work was funded by NSF grant DEB EF-0622770 as part of the joint NSF-NIH Ecology of Infectious Disease program, the Gordon and Betty Moore Foundation, and the USDA Forest Service, Pacific Southwest Research Station. JANF and CAG were funded by DEFRA (United Kingdom) and USDA.

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New Phytologist

Mortality and community changes drive sudden oak death impacts on litterfall and soil nitrogen cycling

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Received: *18 January 2013*
Accepted: *15 May 2013*

New Phytologist (2013) **200:** 422–431
doi: 10.1111/nph.12370

Key words: community–pathogen feedback, ecosystem ecology, emerging infectious disease, nitrification, nitrogen mineralization, *Phytophthora ramorum*, redwood forests.

Summary

- Few studies have quantified pathogen impacts to ecosystem processes, despite the fact that pathogens cause or contribute to regional-scale tree mortality.
- We measured litterfall mass, litterfall chemistry, and soil nitrogen (N) cycling associated with multiple hosts along a gradient of mortality caused by *Phytophthora ramorum*, the cause of sudden oak death.
- In redwood forests, the epidemiological and ecological characteristics of the major overstory species determine disease patterns and the magnitude and nature of ecosystem change. Bay laurel (*Umbellularia californica*) has high litterfall N (0.992%), greater soil extractable NO_3–N, and transmits infection without suffering mortality. Tanoak (*Notholithocarpus densiflorus*) has moderate litterfall N (0.723%) and transmits infection while suffering extensive mortality that leads to higher extractable soil NO_3–N. Redwood (*Sequoia sempervirens*) has relatively low litterfall N (0.519%), does not suffer mortality or transmit the pathogen, but dominates forest biomass.
- The strongest impact of pathogen-caused mortality was the potential shift in species composition, which will alter litterfall chemistry, patterns and dynamics of litterfall mass, and increase soil NO_3–N availability. Patterns of *P. ramorum* spread and consequent mortality are closely associated with bay laurel abundances, suggesting this species will drive both disease emergence and subsequent ecosystem function.

Introduction

Pathogens are powerful ecological and evolutionary forces that can rapidly influence the structure of plant communities through landscape-to-regional tree population declines (Holt *et al.*, 2003; Burdon *et al.*, 2006; Loo, 2009). Both native and exotic pathogens can be important causes of tree mortality, but the respective drivers and dynamics of outbreak may be very different. Widespread tree mortality can be triggered when pathogens are introduced to naïve host populations where natural enemies and host defenses are absent or ineffective. By contrast, widespread tree mortality caused by native pathogens or insects may follow host distribution shifts, changes in management, and weather- or climatic-driven increases in pest or pathogen populations (Raffa *et al.*, 2008; Worrall *et al.*, 2010; Hawkins & Henkel, 2011; McDowell *et al.*, 2011). Generalized ecosystem theory predicts that pathogen outbreaks that alter host or community characteristics will, in turn, alter ecosystem processes such as N cycling, litterfall dynamics, and decomposition (Ellison *et al.*, 2005; Lovett *et al.*, 2006; Eviner & Likens, 2008). However, few field studies have quantified pathogen impacts to ecosystem processes, which limits understanding of the effects of pathogens on landscape-level biogeochemistry and the implication of these impacts on global change (Hicke *et al.*, 2012).

In contrast to the lack of empirical studies of pathogen impacts on ecosystem processes, several authors have described useful conceptual frameworks that link host and pathogen characteristics with mechanistic changes to functional processes (Burdon *et al.*, 2006; Lovett *et al.*, 2006; Eviner & Likens, 2008). The theoretical foundations of pathogen impacts on ecosystems are corollaries to insect outbreak, and ecosystem-level field studies of insect outbreak provide guidance to similar studies of disease (Hunter, 2001; Hicke *et al.*, 2012). For example, foliar chemistry changes caused by foliar-feeding insects have been linked to altered litterfall chemistry and decomposition rates (Lovett *et al.*, 2002; Russell *et al.*, 2004; Chapman *et al.*, 2006). Similarly, bark beetle outbreak has been shown to increase litterfall %N under dead trees, presumably as a result of the arrest of nutrient resorption (Morehouse *et al.*, 2008; Griffin & Turner, 2012). Mortality-related canopy damage can alter microclimate and subsequent rates of soil N cycling (Classen *et al.*, 2005; Orwig *et al.*, 2008), while shifts in species composition can cause long-term shifts in fundamental ecosystem processes that control N and C dynamics (Ruess *et al.*, 2009; Cobb, 2010; Lovett *et al.*, 2010).

Pathogens infect different host tissues (leaves, tree boles, roots), cause selective mortality among canopy species, and may lead to species shifts within communities, suggesting that epidemiological processes drive variation in ecosystem function

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during, and well after, the emergence of disease (Burdon *et al.*, 2006; Lovett *et al.*, 2006). At the local scale, the timing and extent of ecosystem change are likely driven by host characteristics, including biomass, unique function (shade tolerance, N fixation, phenology), and host epidemiological characteristics, including susceptibility, competency to transmit infection, and consequences of infection to host health (Eviner & Chapin, 2003; Ellison *et al.*, 2005; Lovett *et al.*, 2006; Eviner & Likens, 2008). Although epidemiological models can be accurately applied across broad spatial scales (Gilligan & Van den Bosch, 2008; Meentemeyer *et al.*, 2011; Filipe *et al.*, 2012), the lack of data on ecosystem-level pathogen impacts limits our ability to test and accurately apply these models in analyses of C or N cycling in landscapes shaped by disease (Lovett *et al.*, 2006; Hicke *et al.*, 2012).

Phytophthora ramorum, an oomycete pathogen that causes the forest disease sudden oak death, is an example of an exotic pathogen of unknown origin which has resulted in region-scale tree mortality and ecosystem change (Rizzo *et al.*, 2005; Cobb *et al.*, 2012a). *P. ramorum* has a broad host range, but susceptibility, competency to transmit infection, and impacts on host health vary independently across hosts. For example, coast redwood (*Sequoia sempervirens*) foliage has low-to-moderate susceptibility, supports little sporulation, and the tree does not suffer mortality following infection (Davidson *et al.*, 2005; Maloney *et al.*, 2005). Redwood has very little influence on the spread and impacts of *P. ramorum*, but is common in cool, wet environments that are also favorable to the pathogen (Davidson *et al.*, 2011). By contrast, susceptibility and sporulation from California bay laurel (*Umbellularia californica*) foliage is high and drives pathogen spread at stand-to-landscape scales, but infection has no known negative impacts on bay laurel health (Davidson *et al.*, 2008; DiLeo *et al.*, 2009; Meentemeyer *et al.*, 2011). Susceptibility and sporulation from tanoak (*Notholithocarpus densiflorus*) twigs and foliage are epidemiologically significant, but unlike redwood and bay laurel, tanoak tree boles are also susceptible and infection causes bole cankers that can lead to stem death in as little as 2 yr (Cobb *et al.*, 2012b).

Predicting which exotic organisms are likely to establish and cause deleterious impacts to natural resources remains an important but challenging goal of ecology. Eradication of many widespread exotic pathogens is unrealistic, and further introduction of damaging microorganisms is virtually certain to continue (Balci *et al.*, 2007; Loo, 2009; Santini *et al.*, 2012). This increases the importance of understanding ecosystem-level impacts caused by disease. In this study, we focus on three mechanisms by which pathogens may alter ecosystem processes that have been previously documented as drivers of ecosystem change during insect outbreak: direct impacts of pathogens and host mortality on litterfall chemistry; mortality-driven changes to soil N cycling and litterfall dynamics; and the long-term implications of pathogen-mediated community changes to litterfall and soil N cycling. Our field study has three objectives which parallel these mechanisms: to examine the respective effects of pathogen prevalence in bay laurel and mortality in tanoak on litter N chemistry; to quantify the effects of disease-caused mortality to soil N cycling, litterfall amounts and litterfall chemistry; and to describe litter and soil N dynamics associated with each of the major overstory species in redwood forests impacted by sudden oak death. At the individual plant level, we hypothesized that *P. ramorum* infection would increase bay laurel %N and mortality would increase tanoak litter %N, given previous work demonstrating that infection increases bay laurel leaf senescence rates (Davidson *et al.*, 2011) and litterfall N increases in trees killed by bark beetle (Morehouse *et al.*, 2008; Griffin & Turner, 2012). We also expected that soil N availability and mineralization rates would increase with *P. ramorum*-caused mortality given that other disease and insect-caused tree mortality has been demonstrated to alter these soil N dynamics (Hobara *et al.*, 2001; Morehouse *et al.*, 2008; Orwig *et al.*, 2008; Lovett *et al.*, 2010; Griffin & Turner, 2012). Lastly, we expected distinct litterfall chemistry and soil N dynamics associated with the principal *P. ramorum* host species, given that species identity is a critical control over litter chemistry and soil N dynamics (Fried *et al.*, 1990; Finzi *et al.*, 1998; Eviner & Chapin, 2003; Cobb, 2010). We accomplish these objectives by combining litterfall and soil N cycling measured across a gradient of pathogen prevalence and tanoak mortality with a controlled study of species influences on soil N dynamics.

Materials and Methods

Field sites and study design

We conducted measurements of litterfall from January 2007 to December 2009 (3 yr) and soil N cycling from December 2007 to December 2009 (2 yr) at two sites where disease and vegetation dynamics had been monitored during annual summer surveys from 2002 to 2007 (Cobb *et al.*, 2012b). From a pool of potential study sites, we selected Jack London State Park (Jack London) and the Marin Municipal Water District (MMWD), located in Sonoma and Marin Counties (CA, USA), respectively. Both sites are notable for species composition, land use, and disease history characteristic of the broader region. Plots were selected so that soil types were common at each site: Goulding clay loam at Jack London and a Tocaloma–McMullin complex at MMWD. In 2002, 30 plots were established at each site; study plots were circular, 500 m^2, and randomly located with at least 100 m between each plot (Maloney *et al.*, 2005). At the time of establishment, each stem > 1 cm diameter at breast height (dbh; 1.3 m height) was measured for diameter and mapped, and symptomatic tissue was then returned to the laboratory for pathogen isolation in a *Phytophthora* selective medium (Davidson *et al.*, 2008). In the autumn of 2006, we identified a subset of these plots (15 at each site) that span the range of pathogen prevalence (number of infected hosts) and disease severity (tanoak mortality) at each site. We used the strict criteria of *P. ramorum* recovery via laboratory culturing for considering an individual infected; however, mortality was assessed at the stem level, meaning that stems could have been killed by *P. ramorum* yet a multi-stemmed or resprouting individual might remain alive.

Our study design is predicated on the expectation that changes in ecosystem processes are a function of pathogen prevalence, the

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local amount of host biomass that could be killed by the pathogen (tanoak biomass), and the cumulative host biomass that had been killed by *P. ramorum* at the initiation of measurements (dead tanoak biomass; Lovett *et al.*, 2006). Specifically, the selected plots range in initial tanoak basal area from 0.12 to 35.5 $m^2\,ha^{-1}$ and cumulative mortality from 0.05 to 33.7 $m^2\,ha^{-1}$. We forego a two-level pathogen invaded vs noninvaded design in favor of relating the amount of variation in ecosystem processes to infection (prevalence of infected hosts) and mortality (dead tanoak basal area), given the initial tanoak basal area (cf. Lovett *et al.*, 2010). Prevalences of infection at the plot level ranged from 66 to 100% of bay laurel stems and from 6 to 95% of tanoak stems. Many study plots are notable for almost 100% tanoak mortality, while other plots have suffered almost no mortality, even though the tanoak basal area was substantial (11–15 m^2 ha) and pathogen populations have been present since the initial survey in 2002 (Maloney *et al.*, 2005). This variation forms a gradient of disease impacts across plots with different host composition. Our study shares some of the same limitations of space-for-time designs, in that it does not distinguish between responses of the disease to ecosystem function and the responses of ecosystem function to disease. To address this circularity, we conducted a second measurement of N cycling under common temperature and moisture conditions in the laboratory using soils collected from redwood, bay laurel, tanoak, and recently killed tanoak trees located outside our study plots. This provided an independent assessment of the relative influence of dominant overstory species and tanoak mortality on soil N cycling (cf. Fried *et al.*, 1990; Finzi *et al.*, 1998). Further detail regarding community, pathogen, and disease characteristics can be found in Supporting Information, Table S1.

Field litterfall and soil N cycling measurements

Three 1935.48 cm^2 plastic litter traps were established in each plot (*c.* 0.58 m^2 collection area) in January 2007 and July 2007 at the Jack London and MMWD sites, respectively. Large holes were cut into the trap floor, traps were lined with 1 mm mesh screen, and the trap was elevated 10–15 cm above the forest floor surface. This design allows free flow of precipitation and air which air-dried litter between collections; we found no evidence of litter decomposition within our traps (e.g. discoloration, fungal hyphae). For the first 2 yr of measurement, litter was collected eight times yr^{-1} (every 4–8 wk) until seasonal patterns of litterfall were established for each species; during the final year of measurements, litter was collected every 12 wk. Litter samples were air-dried in the laboratory and stored in paper bags 1–12 wk before processing; when precipitation occurred between sampling dates, litter samples were first dried at 45°C for 48 h. Foliar litter was sorted by major overstory species (redwood, tanoak, bay laurel, madrone – *Arbutus menziesii*, Douglas fir – *Pseudotsuga menziesii*) and the remaining material was sorted, without regard to species, into woody litter and all other material which included fruit, flower parts, herbaceous plant litter, bryophytes, seeds, and occasionally insect bodies. Bay laurel foliar litterfall was further assessed for the frequency of *P. ramorum* symptoms on a leaf-by-leaf basis for each sample. After sorting, each sample was dried at 60°C for 48 h, weighed, and archived for later chemical analysis. Litterfall chemistry was not measured for each sampling, because of insufficient litterfall mass at some collection dates. Rather, after the 2 yr of measurement, it became clear that quarterly periods corresponding to winter (January–March), spring (April–June), summer (July–September), and autumn (October–December) reflect the major seasonal changes in litterfall mass for tanoak, bay laurel, and redwood in our study plots. Therefore, we composited, analyzed C and N concentration, and calculated litterfall N mass on this quarterly basis.

We assessed soil net N mineralization and net nitrification of the top 20 cm of mineral soil with a field incubation of intact soil cores. At two locations in each plot, we removed the forest floor layer and drove a 27-cm-long, 5.08-cm-diameter PVC tube into the mineral soil to a depth of 22 cm. The bottom 2 cm of soil was carefully removed and replaced with a nylon mesh bag filled with *c.* 10 g of IRN 150 ion exchange resin (Amberlite IRN 150, Rohm and Haas, Philadelphia, PA, USA) and fitted with a rubber ring which held the soil in the core. This yielded an open-top, open-bottom core, which allowed free water movement during the 10–28 wk of field incubation. A second core was used to sample the top 20 cm of mineral soil and establish initial NH_4–N and NO_3–N concentration. For both incubated and initial cores, the PVC tube was emptied in the field, and soil samples were transported back to the laboratory on ice, and processed within 48 h. Each sample (incubated and initial) was sieved to pass a 2 mm screen; a subsample was dried for 48 h at 105°C to determine moisture content and a second subsample was analyzed for inorganic N by gently shaking 10 g of field moist soil in 1 M KCl for 0.5 h and filtering the extract through a 0.45 µm pore-size glass-fiber filter. NO_3–N and NH_4–N concentrations of this extract were measured with a sulfanilamide reaction after reduction in a copperized cadmium column and a salicylate method, respectively, at the UC Davis Analytical Laboratory (QuikChem Methods 12-107-04-1-B and 12-107-06-2-A, respectively; Lachat Instruments, Loveland, CO, USA).

Laboratory soil mineralization measurement

We conducted a laboratory incubation designed to examine the influence of individual species and tanoak mortality on inorganic N availability and mineralization under common environmental conditions. In April 2009, we selected eight redwood, bay laurel, healthy tanoak, and tanoak trees in which the main stem had been killed by *P. ramorum* ($n = 32$). These trees were located at the Jack London site and chosen in sets of four such that each tree was between 10 and 40 m from the others in its set, and each set was separated by at least 150 m. We sampled the top 20 cm of mineral soil at eight locations within 2 m of each individual tree using a 6.6-cm-diameter stainless steel soil core and composited samples in the field. These samples were transported, processed, and analyzed using the same methods described for N mineralization measurements. Two subsamples for each tree were measured for initial soil moisture, NO_3–N and NH_4–N (64 total). Soil collection occurred within 2 d of significant rainfall, initial soil

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moisture content did not significantly differ among species, and soil moisture content was adequate to support microbial processes for the 5 wk incubation (range 0.40–0.49 g g^{-1}); therefore soils were incubated at field moisture. For each tree, we created 10 replicate soil microcosms of *c.* 50 g soil (sieved to pass a 2 mm screen) in 300 ml vented plastic sample cups (320 in total). Microcosms were incubated at 22°C in a dark, climate-controlled space and two microcosms from each tree were destructively sampled every week for 5 wk to estimate changes in N dynamics through time. Each microcosm was assessed for soil moisture, NO_3–N and NH_4–N with the same methods used for intact soil cores. Tree-level data were the average values from both microcosms at each time point, including the initial measurements ($n = 192$).

Data analysis

We assessed the effects of disease and pathogen prevalence on litterfall and soil N cycling with a series of linear models. In order to examine the relationships between pathogen prevalence and litter %N for bay laurel, and mortality and litter %N for tanoak (objective 1), we employed a series of linear models for each season of collection where individual chemistry parameters (%N and C : N) were the dependent variables and infection (number of infected bay laurel) or mortality (dead basal area, $m^2\,ha^{-1}$) was the independent variable. An identical model was used to assess bay laurel litter chemistry and frequency of *P. ramorum* symptoms within individual samples. We expected that disease-caused changes in litterfall mass, N mass, and soil N dynamics (objective 2) would be a joint function of the maximum potential disease impact (initial tanoak basal area, $m^2\,ha^{-1}$) and the cumulative tanoak biomass killed by the pathogen (dead tanoak basal area, $m^2\,ha^{-1}$). We analyzed annual litterfall mass and N amounts with a set of multivariate repeated-measures ANOVA models for redwood, tanoak, bay laurel, tanoak litter N, and total (stand-level) litter N ($n = 90$). We selected this ANOVA model because our litterfall parameters were measured on a limited number of well defined categories (annual litterfall; cf. Gotelli & Ellison, 2004) and the time × disease interactions on an annual basis have a straightforward interpretation. Here, the dependent variable (Y) for each species or litterfall component (i) at time t was modeled as a function of the independent variables (X_i) conditioned on species-specific parameters ($b_{i,t}$), the respective annual estimated mean $\bar{Y}_{i,t}$, and a normally distributed error term (ε): $Y_{i,t} = \bar{Y}_{i,t} + \sum X_i b_{i,t} + \varepsilon$. Models of soil N responses to tanoak mortality were similar to those for litterfall, except we used a mixed model with time parameterized as a random effect given that the timing of sampling was irregular throughout the 2 yr of measurement (cf. Gotelli & Ellison, 2004). These models also included a fixed effect of soil moisture measured in the initial cores to examine potential moisture limitation to microbes. We assessed the potential impacts of species shifts by describing litter C : N and local soil N dynamics associated with the major overstory species in our study plots (objective 3). Differences in litter %N and C : N content among species were assessed with a one-way mixed-model ANOVA where species was the main effect and sampling date was a random effect; when the main effect was significant, differences among species were assessed with Tukey's HSD test. For our laboratory comparison of species effects on soil N dynamics, we were able to employ a matched-pairs *t*-test for all possible pairs on the basis that each subject was grouped into individual blocks. Variations in NO_3–N, total N pool sizes, net nitrification and N mineralization rates associated with species identity and dead tanoak were assessed with identical models that individually compared each tree type with each of the others. For each linear model, normal distribution and homogeneous variance of the residuals were assessed with goodness-of-fit tests to the normal distribution and visual evaluation of heteroscedasticity; for the paired *t*-test analysis, normal distribution was tested for each variable. Square-root transformation was required for litterfall chemistry, field-based soil N measurements, and soil N pool sizes for the laboratory study. Analysis was performed with JMP® version 8 (SAS Institute, Cary, NC, USA), with the critical value of $P \leq 0.05$ for statistical significance.

Results

Direct pathogen impacts on litter chemistry (objective 1)

The plot-level prevalence of infected tanoak and bay laurel was not significantly associated with annual litterfall mass in either species (data not shown). However, a modest, positive effect of litterfall %N and prevalence of infection was found for bay laurel during the spring and summer, but not during autumn and winter (Fig. 1). The spring and early summer seasons also correspond to peak periods of *P. ramorum* sporulation and within-tree infection at the Jack London site (Davidson *et al.*, 2011). We found a similar, negative, and statistically significant relationship between bay laurel litterfall C : N and prevalence of infection (not shown). The spring collections were also notable for relatively high %N concentrations and low C : N relative to the other three seasons, but this period also had the lowest mass of bay laurel litterfall (Fig. 2). Bay laurel %N and C : N were not significantly related to frequency of symptomatic leaves in our litter traps. Given that bay laurel contributed *c.* 7–11% of overall litterfall N (Fig. 2; Table S2) and that litterfall amounts were low when the pathogen may elevate foliar %N (decrease C : N), this pathogen effect on the total N transfer from the canopy to the forest floor is subtle. In contrast to bay laurel, no relationship between tanoak litterfall %N or C : N and tanoak mortality was found for any season of comparison (Fig. 1; C : N not shown).

Mortality impacts on litterfall and soil N cycling (objective 2)

Disease had significant effects on the mass of tanoak litterfall, tanoak litterfall N, and total foliar litterfall N (Fig. 3). Litterfall amounts were positively associated with the respective predisease basal area for each species. But for tanoak, litterfall mass and N were also negatively associated with cumulative tanoak mortality, and the magnitude of these reductions was also variable across years (interaction $P < 0.05$; Fig. 3; Table S3). The estimates from the repeated-measures model indicate that *P. ramorum*-caused

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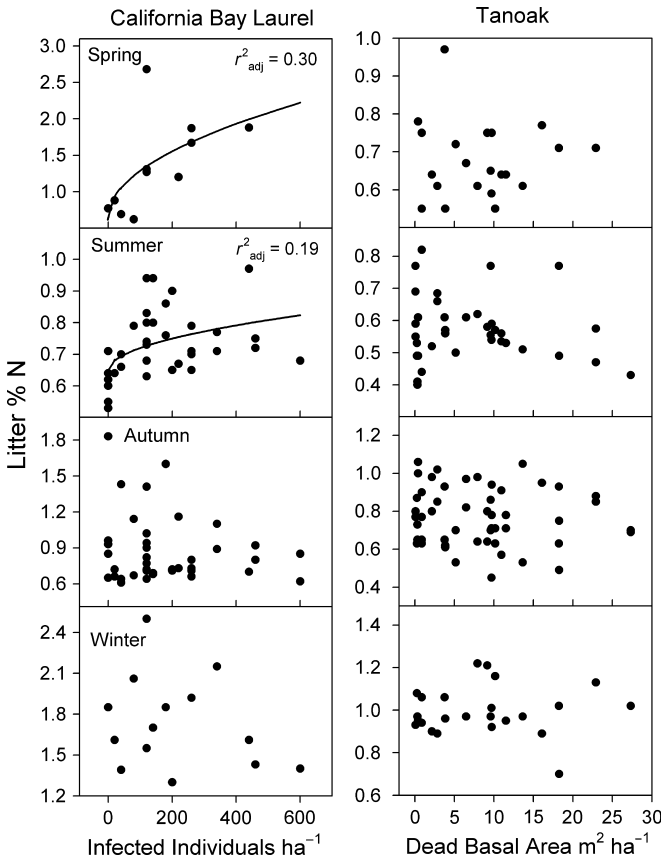

Fig. 1 Bay laurel (*Umbellularia californica*) and tanoak (*Notholithocarpus densiflorus*) litterfall %N vs prevalence of *Phytophthora ramorum* or cumulative dead tanoak basal area. When the relationship between infected hosts or mortality and litter nitrogen (N) concentration was significant ($P \leq 0.05$), the r^2 is reported along with the square-root-transformed least-squares fit. Note the differences in scale of N concentration between species and seasons of measurement.

tanoak mortality resulted in up to 91% reduction of tanoak litterfall and up to 95% reduction of tanoak litterfall N in plots with the greatest amount of cumulative tanoak mortality (up to *c.* 33 m^2 ha^{-1} basal area). Even when tanoak mortality was extensive, tanoak foliar litter (and tanoak foliar litter N) was still part of the overall litterfall mass, as a result of litter production from basal sprouts that frequently developed from *P. ramorum*-killed tanoak stems. Compared with other species collected in our litter traps, tanoak showed less seasonal variation (Fig. 2). Even though redwood dominates litterfall N mass (65–78% of total), total litter N (stand-level) decreased with tanoak mortality (Fig. 3). This effect is probably caused by the relatively high %N of tanoak litter compared with redwood. Total foliar litterfall, woody litter, and total litterfall (e.g. foliage, woody litter, and other materials) were not significantly associated with disease and were relatively insensitive to forest structure across our plots (Table S4).

Total and NO_3–N pools were significantly increased with disease, but rates of nitrification and mineralization were not affected (Fig. 4). NO_3–N and total N concentrations were negatively associated with predisease tanoak basal area and positively associated with cumulative dead tanoak basal area and soil moisture (Fig. 4; Table S5). Extractable inorganic N pools were dominated by NO_3–N, and were often > 60% nitrate. The shared patterns of significance between NO_3–N and total N were mostly driven by this high proportion of NO_3–N (total N = NO_3–N + NH_4–N). Similarly, nitrification rates were 80–100% of net N mineralization for soils incubated in the field (Fig. 4). Almost identical patterns between nitrification and N mineralization were driven by the dominance of nitrification in N mineralization rates of our study plots. Seasonal influences on soil N concentration and mineralization were weak, although the sampling duration also spanned a California-wide drought from 2007 to 2008.

Species effects on litterfall, litterfall chemistry, and soil N cycling (objective 3)

Litter chemistry was markedly different among species. %N was greatest in bay laurel, lowest in redwood, and intermediate in tanoak (Fig. 2; $P < 0.05$ each contrast). Litter C : N followed a similar pattern, with the highest C : N in redwood, the lowest in bay laurel, and intermediate values for tanoak. Redwood dominated the total litterfall mass in our plots, followed by tanoak, madrone, bay laurel, and other species (Fig. 2; Table S2). Redwood litterfall was low during the spring and summer but peaked in the late autumn/early winter (Fig. 2). Tanoak and bay laurel litterfall tended to peak in the mid-summer and early autumn, several months earlier than redwood. Despite the significant differences in litterfall chemistry among species, all three followed a similar seasonal pattern of %N (and C : N), with highest concentrations in the winter and lowest concentrations during peak litterfall in summer or autumn.

Species identity significantly affected NO_3–N availability but did not influence any other soil N cycling parameter during the 5 wk laboratory soil incubation (Fig. 5). Tanoak had significantly lower extractable NO_3–N compared with bay laurel and dead tanoak. Soil NO_3–N availability from redwood was significantly higher compared with tanoak and tended to be lower than in bay laurel or dead tanoak, but these differences were not significant. Total N concentrations were similar between species, and net rates of nitrification and N mineralization did not differ among species either (Fig. 5). Soil moisture declined over the course of the incubation to an average of 0.29 g g^{-1} (± 0.02 SE) and net mineralization rates became less variable (Fig. S1). The overall patterns of N availability from the laboratory incubation were consistent with measurements made in the field. Tanoak mortality was positively associated with NO_3–N availability, but no changes in mineralization or nitrification rates were found in either set of measurements.

Discussion

This study demonstrates the potential for sudden oak death to alter litterfall and soil N availability in redwood forests and provides general, *a priori* expectations of impacts on these processes for many landscape-scale tree mortality events. Tanoak mortality

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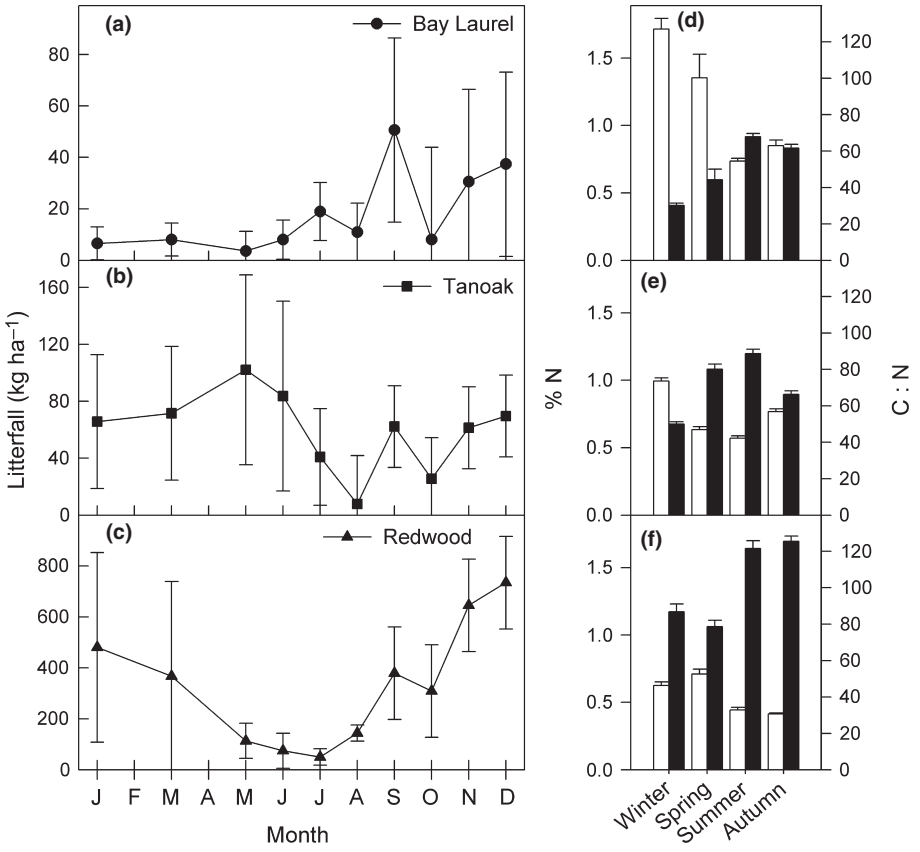

Fig. 2 Monthly litterfall mass (a–c) with seasonal values of carbon and nitrogen (d–f; open bars, %N; closed bars, C : N) for bay laurel (*Umbellularia californica*; top), tanoak (*Notholithocarpus densiflorus*; middle), and redwood (*Sequoia sempervirens*; bottom) from two redwood forests impacted by sudden oak death. Data are means from 3 yr of litterfall monitoring presented ± 1 SE for litterfall mass and +1 SE for seasonal litter chemistry. Note the differences in scale in the panels of litterfall mass for each species. The month of collection is abbreviated to the first letter.

had the greatest short-term impacts on litterfall dynamics and N availability in our redwood-dominated study sites, but directional shifts in community composition mediated by *P. ramorum* will have longer-term changes (and perhaps of a greater magnitude) on these ecosystem features. Our study, along with several others, suggests that disease-caused ecosystem changes can be driven primarily by mortality and the resulting changes in plant community composition (Hobara *et al.*, 2001; Orwig *et al.*, 2008; Cobb, 2010; Lovett *et al.*, 2010). These results suggest that patterns of landscape-scale tanoak mortality and species shifts (Meentemeyer *et al.*, 2008; Metz *et al.*, 2012) are an appropriate basis for predicting changes in NO_3–N availability and litterfall dynamics for sudden oak death.

An emerging consensus of field and modeling studies demonstrate the importance of sporulation sources, especially bay laurel, on rates of *P. ramorum* spread and emergence of sudden oak death (Davidson *et al.*, 2005, 2008, 2011; Maloney *et al.*, 2005; Meentemeyer *et al.*, 2008, 2011; Cobb *et al.*, 2012a). Landscape-level data show increased dominance of bay laurel under many conditions, especially when this species co-occurs with tanoak and redwood (Cobb *et al.*, 2010; Metz *et al.*, 2012). Shifts to greater dominance of bay laurel will increase litterfall %N as well as soil NO_3–N concentration (Figs 2, 5); this increase in litter % N is likely to increase overall litter decomposition rates as well (Chapman *et al.*, 2006; Cobb, 2010). Notably, tanoak mortality can be extensive even when bay laurel is not present within a stand, because sporulation on tanoak is sufficient to cause mortality (Ramage *et al.*, 2011; Cobb *et al.*, 2012b; Metz *et al.*, 2012). In these stands, sudden oak death is likely to favor common species with low-susceptibility, especially redwood and Douglas fir (Cobb *et al.*, 2010) that frequently co-occur with tanoak. These species have notably lower litter quality than bay laurel or tanoak, which is likely to result in slower litter decomposition and net accumulation of forest floor mass (Fig. 2; Valachovic *et al.*, 2004). In either scenario, shifts in species abundance are most likely to drive long-term changes to soil N availability and litterfall dynamics in *P. ramorum*-invaded forests.

Increased rates of soil N cycling and NO_3–N availability have been a common ecosystem response following insect and pathogen outbreak (Hobara *et al.*, 2001; Morehouse *et al.*, 2008; Orwig *et al.*, 2008; Lovett *et al.*, 2010; Griffin & Turner, 2012). Our study departs from this overall trend in that mortality increased NO_3–N availability but did not change cycling rates, a result that was consistent in the laboratory as well as in the field (Figs 4, 5). The majority of studies examining pathogen and insect impacts on ecosystems have focused on outbreaks that result in a more uniform mortality or defoliation across a stand than frequently occurs in sudden oak death (Hobara *et al.*, 2001; Russell *et al.*, 2004; Morehouse *et al.*, 2008; Orwig *et al.*, 2008; Lovett *et al.*, 2010; Griffin & Turner, 2012). By contrast, even in our study plots with the greatest amount of tanoak mortality, the majority of biomass was often redwood, bay laurel, or other species which are minimally impacted by the disease. Further, survival times of *P. ramorum*-infected tanoak trees can vary from

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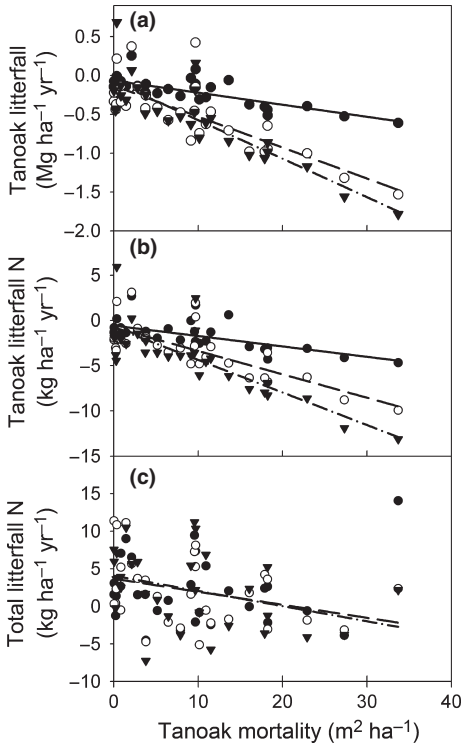

Fig. 3 Effects of sudden oak death-caused mortality (*x*-axis) on annual tanoak (*Notholithocarpus densiflorus*) litterfall mass (a), tanoak litterfall nitrogen (N; b), and total stand-level litterfall N (c). Closed circles with solid lines, 2007; open circles with dashed lines, 2008; triangles with dash-dot lines, 2009. Data are observed values minus those expected if the stands had not been impacted by sudden oak death (see text and Table S3 for more details). Negative values on the *y*-axis denote the amount of litterfall reduction associated with a given amount of tanoak mortality. Data are presented with least-squares regression lines.

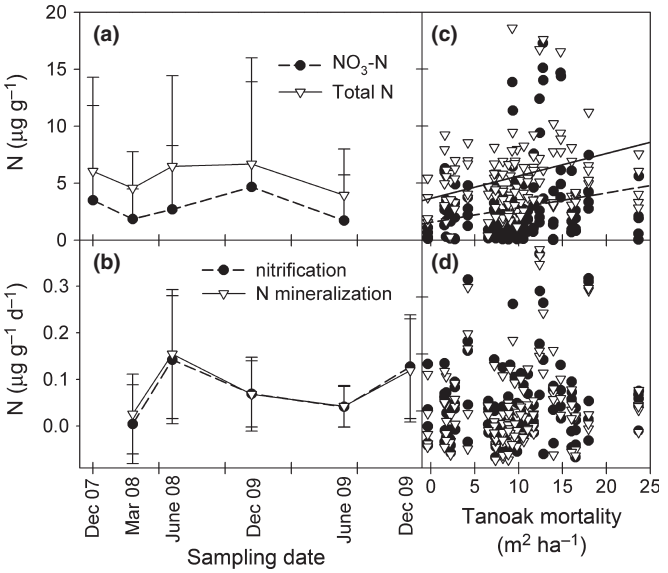

Fig. 4 Seasonal patterns and effects of sudden oak death on soil nitrogen (N). Seasonal patterns of inorganic N pool sizes (a) and rates of N mineralization and nitrification (b) are shown with sampling date on the *x*-axis. (c, d) Leverage plots from mixed linear models showing the effect of tanoak (*Notholithocarpus densiflorus*) mortality on N pool sizes (c) and rates of turnover (d). Data in (a) and (b) are means ± 1 SE; least-squares regression lines are shown for statistically significant ($P \leq 0.05$) models.

2 to 20 yr because of differences in susceptibility within populations and size-specific mortality rates (Hayden *et al.*, 2011; Cobb *et al.*, 2012b). The resulting spatial and temporal variation in mortality may dampen impacts on soil N cycling because changes in canopy structure are less severe relative to homogeneous disturbances or outbreaks (Cobb *et al.*, 2012a). Comparatively, Gypsy moth (*Lymantria dispar*) outbreak can cause extensive defoliation with low mortality relative to other outbreaks (Lovett *et al.*, 2002; Russell *et al.*, 2004); this defoliation can increase litterfall and litterfall N without changing N mineralization or availability (Russell *et al.*, 2004). Our study supports the general expectation that the timing and uniformity of mortality are important controls over the magnitude of changes to ecosystem processes following an outbreak (Ellison *et al.*, 2005; Lovett *et al.*, 2006; Eviner & Likens, 2008), even though our data do not confirm our initial hypothesis that disease would increase rates of soil N mineralization.

Direct impacts of infection on host tissues had the least significant effect on ecosystem processes at the spatial scale of our study (the ecosystem; Fig. 1). Unlike bark beetle-caused mortality, tanoak mortality was not associated with increased litterfall %N (cf. Morehouse *et al.*, 2008; Griffin & Turner, 2012), which may also reflect the heterogeneous timing of tanoak mortality in *P. ramorum*-invaded stands (Cobb *et al.*, 2012b). The modest positive association between bay laurel %N and prevalence of infection during the spring and summer seasons (Fig. 1) could be driven by changes in plant chemistry induced by infection or by increased shedding of infected foliage (Hunter, 2001; Lovett *et al.*, 2006; Eviner & Likens, 2008). In bay laurel, *P. ramorum* infection reduces photosynthetic leaf area but does not change photosynthetic rates (DiLeo *et al.*, 2009). Additionally, the prevalence of symptoms within individual bay laurel litter samples was not significantly associated with litterfall %N or C : N, which might be expected if the pathogen induced these changes in litterfall chemistry. By contrast, Davidson *et al.* (2011) demonstrated increased rates of leaf shedding for infected vs uninfected bay laurel leaves and suggested that *P. ramorum* can accelerate leaf senescence by 3–4 yr. Increased litterfall %N is likely when leaf senescence occurs before nutrient reabsorption is maximized in evergreen species including bay laurel (Lovett *et al.*, 2002; Chapman *et al.*, 2006). Although this increase in litter N was small, it could be spatially extensive if other broadly distributed *Phythophthora* pathogens such as *P. nemorosa* and *P. pseudosyringae* also increase bay laurel leaf senescence rate. These other *Phythophthora* spp. are weak pathogens on tanoak, but have similar ecology to *P. ramorum* on bay laurel and a more extensive geographic range (Wickland *et al.*, 2008). All three *Phythophthora* spp. may influence bay laurel litterfall %N without eliciting disease (cf. Eviner & Likens, 2008).

Phytophthora ramorum–tanoak interactions form a relatively tractable host–pathogen system from which it is possible to build local to regional predictive models of outbreak and subsequent tree mortality (Meentemeyer *et al.*, 2011; Cobb *et al.*, 2012b; Filipe *et al.*, 2012). Landscape-level mortality from sudden oak death is largely driven by sporulation sources in conjunction with

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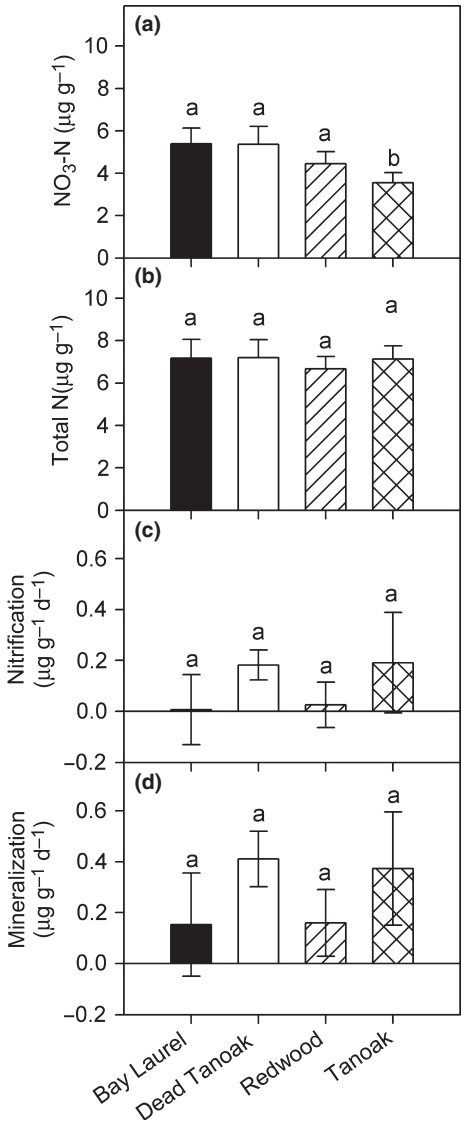

Fig. 5 Species-level effects on nitrogen (N) availability (extractable pools a, b) and cycling rates (c, d). Data are from incubation of soils collected immediately below the three focal species and tanoak (*Notholithocarpus densiflorus*) killed by *Phytophthora ramorum* (dead tanoak). Data are means ± 1 SE. Results from a paired *t*-test analysis are presented above each bar; different letters indicate statistically different mean values ($P \leq 0.05$) across all possible pairs.

the distribution of tanoak and susceptible oaks, the species that are frequently killed following *P. ramorum* infection (Davidson *et al.*, 2008; Meentemeyer *et al.*, 2008; Lamsal *et al.*, 2011). These patterns emerge because *P. ramorum* virulence is high and resistance in tanoak is insufficient to protect many tanoak populations from significant mortality (Rizzo *et al.*, 2005; Hayden *et al.*, 2011). Patterns of mortality can be reasonably predicted for several other exotic pathogens and insects that are actively spreading into naïve host populations and where community or landscape factors of spread are well understood (Loo, 2009; Lovett *et al.*, 2010; Orwig *et al.*, 2012). However, predicting mortality is much more difficult for many regional tree mortality events, because the relationships between physiological stress and pathogen impacts are typically unknown for the diverse and widespread native pathogenic flora of most temperate forests (Sinclair *et al.*, 1987; McDowell *et al.*, 2011). Understanding how or when native pathogens and insects overcome plant defenses, and what landscape, climatic, or management factors predispose hosts to greater physiological stress (Raffa *et al.*, 2008; Adams *et al.*, 2009; McDowell *et al.*, 2011) will greatly aid the prediction of landscape-level tree mortality and resulting ecosystem changes.

For sudden oak death, many of the ecosystem changes we observed are tied to the epidemiological roles of canopy tree species and their individual influences on ecosystem processes. The mechanisms driving these effects included changes in host litter chemistry, mortality, and shifts in community composition that are likely to be common among many pathogen outbreaks in the same way that they are common drivers of ecosystem change following insect outbreak. Although interactions among pathogens, hosts, and the environment are a foundation of plant pathology (e.g. the disease triangle; Burdon *et al.*, 2006), these interactions are poorly understood for abundant, diverse, but broadly distributed weak pathogens (Balci *et al.*, 2007; Wickland *et al.*, 2008; Hawkins & Henkel, 2011). This lack of understanding hinders prediction of tree mortality incited by regional drought but contributed to by pathogens (Worrall *et al.*, 2010; McDowell *et al.*, 2011). However, when pathogens or insects incite or contribute to major tree die-offs, the longest lasting and greatest magnitude ecosystem impacts can be reasonably predicted by understanding patterns of mortality and subsequent changes in species composition.

Acknowledgements

We thank E. Fichtner, S. Lynch, and H. Mehl for their field and laboratory support of this research. We thank M. Metz, the Rizzo lab group, and three anonymous reviewers for helpful comments on earlier versions of this manuscript. Additionally, we are grateful to the California State Parks and the Marin Municipal Water District for facilitating this research on their lands. This work was funded by NSF grant DEB EF-0622770 as part of the joint NSF-NIH Ecology of Infectious Disease program, the Gordon and Betty Moore Foundation, and the USDA Pacific Southwest Research Station.

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Supporting Information

Additional supporting information may be found in the online version of this article.

Fig. S1 Temporal patterns from a laboratory N mineralization measurement.

Table S1 Study site, plant, community, soil, and disease characteristics

Table S2 Summary annual litterfall amounts in two redwood forests

Table S3 Litterfall and litterfall N repeated-measures MANOVA parameter values

Table S4 Total litterfall and woody litterfall repeated-measures MANOVA parameter values

Table S5 Parameter values from linear models of study plot soil N dynamics

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Range-Wide Threats to a Foundation Tree Species from Disturbance Interactions

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Richard C. Cobb , Margaret R. Metz and David M. Rizzo
Source: Madroño, 60(2):139-150. 2013.
Published By: California Botanical Society
DOI: http://dx.doi.org/10.3120/0024-9637-60.2.139
URL: http://www.bioone.org/doi/full/10.3120/0024-9637-60.2.139

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MADROÑO, Vol. 60, No. 2, pp. 139–150, 2013

RANGE-WIDE THREATS TO A FOUNDATION TREE SPECIES FROM DISTURBANCE INTERACTIONS

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ABSTRACT

The geographic range of tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae), encompasses tremendous physiographic variability, diverse plant communities, and complex disturbance regimes (e.g., development, timber harvest, and wildfire) that now also include serious threats posed by the invasive forest pathogen *Phytophthora ramorum* S. Werres, A.W.A.M. de Cock. Knowing where these disturbance factors interact is critical for developing comprehensive strategies for conserving the abundance, structure, and function of at-risk tanoak communities. In this study, we present a rule-based spatial model of the range-wide threat to tanoak populations from four disturbance factors that were parameterized to encode their additive effects and two-way interactions. Within a GIS, we mapped threats posed by silvicultural activities; disease caused by *P. ramorum*; human development; and altered fire regimes across the geographic range of tanoak, and we integrated spatially coinciding disturbances to quantify and map the additive and interacting threats to tanoak. We classified the majority of tanoak's range at low risk (3.7 million ha) from disturbance interactions, with smaller areas at intermediate (222,795 ha), and high (10,905 ha) risk. Elevated risk levels resulted from the interaction of disease and silviculture factors over small extents in northern California and southwest Oregon that included parts of Hoopa and Yurok tribal lands. Our results illustrate tanoak populations at risk from these interacting disturbances based on one set of hypothesized relationships. The model can be extended to other species affected by these factors, used as a guide for future research, and is a point of departure for developing a comprehensive understanding of threats to tanoak populations. Identifying the geographic location of disturbance interactions and risks to foundation species such as tanoak is critical for prioritizing and targeting conservation treatments with limited resources.

Key Words: Decision support system, fire, forest ecosystems, foundation species, landscape epidemiology, *Notholithocarpus densiflorus*, sudden oak death, tree disease.

Ecological disturbance regimes play an integral role in ecosystem dynamics by altering resource availability, modifying ecosystem structures, and creating new landscape spatial patterns (e.g., Mou et al. 1993; Spies et al. 1994; Turner et al. 2003). Increasing global connectivity, population growth, and climate change are rapidly altering disturbance regimes, resulting in the emergence of novel disturbance interactions with pronounced impacts to socio-ecological systems from local to global scales (Turner 2010). Interacting disturbance regimes that alter the abundance and structure of foundation species effectively disrupt the fundamental ecosystem processes that they support and stabilize, such as clean water, decomposition, and carbon sequestration (Chapin et al. 1997; Ellison et al. 2005), which are vital for maintaining the physical, social, and economic health of human populations (Costanza et al. 1997). Recognition of the relationships and impacts of disturbance interactions by local, regional, and global stakeholders is necessary to manage for the resiliency of foundation species and their functions (Folke et al. 2004). Research explicitly addressing the effects of interacting disturbances in forest ecosystems has recently increased (e.g., Bebi et al. 2003; Buma and Wessman 2011; Metz et al. 2011), but we still understand relatively little about their impacts. The landscape heterogeneity and spatial extent over which forest disturbances occur challenges the development of ecosystem conservation strategies, presenting a pressing need to develop tools that engage and guide stakeholders in achieving conservation objectives across these broad areas.

Accessibility to and familiarity with geographic information system (GIS) technologies (e.g., GPS-enabled smartphones and online mapping) has become more widespread in recent years, increasing the potential to effectively bring stakeholders together in addressing complex conservation management problems. Spatially explicit models developed using GIS can be

utilized as spatial decision support systems to identify the geographic location and potential severity of disturbance events, providing an essential tool for threat assessment and management. This adaptable framework can be applied to identify at-risk populations when the models are built using known biological relationships and sound ecological theory, even when knowledge of the precise relationships is scant. For example, Andersen et al. (2004) developed a risk model of threats to biodiversity across a large, heterogeneous landscape by examining relative risk of land use factors on several resident taxa, and Meentemeyer et al. (2004) similarly mapped the risk of establishment and spread of a forest pathogen (*Phytophthora ramorum* S. Werres, A.W.A.M. de Cock) across California. Mapping the threat to the abundance and structure of foundation species from disturbance interactions helps guide stakeholders in developing and implementing effective conservation strategies that protect vital ecosystem services.

Tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae), is a foundation species threatened with functional extinction by multiple interacting disturbances throughout large portions of its range. This tree is, a dominant component of the ecosystems it inhabits (Cobb et al. this volume), has unique ecological characteristics in coastal California forests (Bergemann and Garbelotto 2006; Wright and Dodd this volume), and as the lone representative of its genus it is a significant contributor to regional as well as global biodiversity. This shade-tolerant tree can form multistoried forest canopies with other dominant overstory species, providing important stand structure for wildlife, such as the spotted owl (*Strix occidentalis*; LaHaye et al. 1997; North et al. 1999). Tanoak acorns are a traditionally important nutrition source for Native Americans, and the trees were a principal source of bark-extracted tannins until the advent of chemical tanning compounds in the 1950′s (Bowcutt 2011). Since the collapse of the tanoak bark market, perspective on this species has shifted from an important forest commodity to an impediment to production of more valuable timber species, such as redwood, *Sequoia sempervirens* (Lamb. ex D. Don) Endl., and Douglas-fir, *Pseudotsuga menziesii* (Mirb.) Franco. Since the 1950′s, most applied forest research on tanoak has focused on techniques to reduce competition with timber species, primarily through herbicide applications to reduce tanoak prevalence and biomass in forests managed for timber production (Harrington and Tappeiner 2009; Bowcutt 2011). In the last ten years, *Phytophthora ramorum*, and the resulting disease sudden oak death, has emerged as a major cause of tanoak mortality in central coastal California, and increasingly in northern coastal California and southwest Oregon (Rizzo et al. 2005; Meentemeyer et al. 2008; Václavík et al. 2010). *Phytophthora ramorum* has a large array of hosts, but susceptibility, impacts of infection on host health, and the competency of hosts to transmit infection varies dramatically over the 137 regulated native and exotic host species (APHIS 2012).

Silviculture, disease, development, and altered fire regimes are arguably the major disturbances threatening the abundance, function, and persistence of tanoak throughout its range. Silvicultural practices have explicitly suppressed tanoak to promote the growth of conifer species; human population expansion has resulted in conversion of forested land to development; and fire regimes have been altered from historic baselines (Havlina et al. 2010). In addition to these disturbances, tanoak is being severely impacted by *P. ramorum* on local to regional scales (Meentemeyer et al. 2008; Meentemeyer et al. 2011; Cobb et al. 2012b). Tanoak readily supports sporulation of *P. ramorum* and can be rapidly killed following infection by this pathogen (Hansen et al. 2008). Several other common native species also support sporulation, but do not die following infection, most notably California bay laurel, *Umbellularia californica* (Hook. & Arn.) Nutt. (DiLeo et al. 2009). Thus, the distribution of bay laurel and tanoak strongly influences the risk of pathogen establishment and disease emergence (Meentemeyer et al. 2004; Davidson et al. 2008; Cobb et al. 2010). The geographic variation and extent of each of these disturbances present major challenges to the conservation of tanoak ecosystems.

We present a spatially explicit model to quantify and map the area threatened by these four disturbances and their interactions across the geographic range of tanoak. This hypothesis-based modeling approach is readily integrated with adaptive management strategies. Thus, as knowledge of the system grows, the parameters of our model can be adjusted in accordance with evolving goals and the efficacy of treatments.

Geographic Range

The geographic range of tanoak stretches contiguously along the Pacific coast from a four-county area in southwestern Oregon in the north, to Monterey County, CA, in the south, with disjunct populations occurring in the Sierra Nevada foothills to the east. Our analysis excluded an isolated population occurring near Santa Barbara, CA (Tappeiner et al. 1990), because the data we used for tanoak abundance and area estimates (Lamsal et al. 2011) were incomplete in this region. Tanoak's geographic range possesses tremendous physiographic variability and complex disturbance regimes, and is broadly characterized by a Mediterranean-type

Table 1. The Weights Assigned to Individual Disturbance Factors and the Interactions in the Initial Model Based on our Interpretations of Relevant Literature and Expert Observations. Each disturbance factor was first standardized to a 0–3 ranking so that assigned weights used in the modeling calculations reflect their relative importance as a threat to tanoak.

Disturbance	Weights
silviculture	30
disease	25
development	10
fire	5
Interaction	**Interaction weight**
disease x fire	2
disease x silviculture	2
disease x development	1
fire x development	1
development x silviculture	1
fire x silviculture	1

climate with cool, wet winters and warm, dry summers. Tanoak occurs from sea level to roughly 2190 m, with greater abundance in forests on the leeward side of the Coast Range (Lamsal et al. 2011).

Model Development

We developed a rule-based spatial model to quantify and map the relative threat to tanoak populations from four disturbance factors occurring throughout tanoak's geographic range. This heuristic approach is akin to a mental shortcut, where empirically undefined relationships can be hypothesized and examined. In order to parameterize the relative threat to tanoak from each disturbance factor, we classified the threat level of each factor at a particular location into an integer ranking system from zero to three, with zero representing no threat and three representing high threat. At each location, we assigned each factor a low (1), intermediate (2), or high (3) threat ranking by breaking the range of values greater than zero into three equal intervals. We also assigned a weight value to each ranked disturbance factor indicating its relative importance as a threat to tanoak (Table 1). We based these weight values on our interpretations of published research and expert knowledge evaluating the impacts of each disturbance to tanoak. They can be altered within the modeling framework to explore other hypotheses of the impacts to tanoak from these disturbances. We calculated multiplicative two-way disturbance interactions for each location, assigning an additional exponential term to weight the interaction that we hypothesized as representing the greatest threat to tanoak (Table 1). For each location, we then selected the highest-valued interaction for inclusion in the final threat calculation to visualize and highlight areas with the most at-risk populations for the set of hypotheses (i.e., weights) being examined.

The equation used to calculate potential threat to tanoak from these disturbance factors is the sum of the products of each factor's rank and importance weight, plus the highest valued interaction at each mapped location (grid cell):

$$P_j = \sum_{i}^{n} W_i R_{ij} + [W_i^a R_{ij}^a \times W_i^b R_{ij}^b]^y \qquad (1)$$

where P is the calculated risk for a grid cell in the model output, W_i is the weight of the ith disturbance factor, R_{ij} is the rank value of the ith factor at location j, and y is the weight assigned to the interaction of two factors. The interaction weight (y) is determined by which pair of weighted factors, $W_i^a R_{ij}^a$ and $W_i^b R_{ij}^b$, occur together at a given location (Table 1). The superscripts of these parameters (a and b) ensure that a factor is never multiplied by itself. We developed maps of each disturbance factor and overlaid them with a tanoak abundance surface (Lamsal et al. 2011). We then applied the interaction model (Eq. 1) in a GIS environment to generate a map of at-risk tanoak populations from these interacting disturbances across the geographic range. We classified the model output into a 1–3 threat-level ranking by breaking the calculated values at equal intervals. Similar to the individual factors, we qualitatively labeled the threat levels as 'Low' (1), 'Intermediate' (2), and 'High' (3). The lack of zero values in the final map output demonstrates that at least one factor was present at every estimated tanoak location.

Quantifying Number of Tanoak at Risk

We quantified the area and number of tanoak trees in each threat category by intersecting the output of equation 1 with maps of tanoak density produced by Lamsal et al. (2011). These data were further organized by county to aid to regional decision making. We then multiplied the county-level tanoak areas (hectares) in each threat level by the average number of tanoak per hectare in each county derived from data provided by Lamsal et al. (2011). This produced the estimated number of tanoak in each threat category for each county in the study system. We report on a limited number of areas in this paper, with more detailed county summaries available from the authors.

Disturbance Factors

Silviculture

Beginning in the early to mid-20th century, silviculture in Oregon and California strongly

favored softwood conifer species at the expense of hardwood species, especially tanoak (Bowcutt 2011). Most notable are broad-scale applications of herbicide to reduce tanoak competition with timber species in these forests. Tanoak vigorously sprouts following cutting and can reduce the growth of planted or naturally regenerating conifers (Harrington and Tappeiner 1997; Lorimer et al. 2009). Herbicide applications are effective in reducing tanoak cover and increasing the growth and dominance of coniferous timber species (Tappeiner et al. 1987; Harrington and Tappeiner 2009). When applied as a broadcast spray from aircraft, or at very high efficiencies by ground crews, it is reasonable to expect these practices would result in functional extinction of tanoak at local scales. For these reasons, the silviculture risk factor received a weight of 30, the highest weight (Table 1).

We developed the silviculture risk factor layer using Forest Inventory and Analysis (FIA) (USDA 2008) data for plots in California and Oregon where tanoak was reported (n = 565). FIA surveys record evidence of silvicultural treatments that affect areas of one acre or more; however, they do not specify herbicide application. We assumed the following about these data: 1. silviculture activities included suppression of undesirable species, i.e., tanoak, 2. a greater number of treatments is equivalent to greater threat to tanoak populations, and 3. recent treatments were more efficient and effective, whereas older treatments may have been overcome by recolonization from tanoak in adjacent stands (Tappeiner and McDonald 1984). We used all recorded treatment types with the exception of ''Firewood or local use cut,'' which we interpreted as unlikely to target tanoak (or any species) for removal or suppression. We ranked locations according to the number of recorded treatments weighted by the timing of those treatments. We used 20-year intervals to capture increased activity surrounding timber harvests as well as related treatments during intervening years. This process assigned the highest risk from silviculture to locations where treatments were both persistent and more recent. We used these scores to create a map of silviculture intensity with values ranging from 0–10, which we reclassified into 0–3 rankings by splitting non-zero values at equal intervals.

Disease

Phytophthora ramorum is an unprecedented pathogen in terms of its capacity to impact the abundance, structure, and function of tanoak communities. The mortality rate of *P. ramorum* infected trees (especially tanoak but also *Quercus* species) increases with tree size (Ramage et al. 2011; Cobb et al. 2012b), leading to rapid declines in tanoak biomass, dominance, and ecological function. We assigned the disease factor a weight of 25, slightly lower than silviculture because its impact on tanoak is slower, more heterogeneous, and highly dependent on other landscape and vegetation characteristics (Haas et al. 2011). For the disease disturbance factor, we used two previously developed maps detailing the risk of *P. ramorum* establishment and spread for Oregon (Václavík et al. 2010) and California (Meentemeyer et al. 2004). These studies used heuristic models incorporating host indices and climate factors derived from known infestations to characterize disease risk throughout each landscape. We created a mosaic of these independent risk layers with values ranging from 0–100, and reclassified this map along equal intervals into 0–3 rankings.

Development

Development impacts tanoak abundance, function, and persistence through conversion of forests to developed landscapes characterized by mixtures of impervious surfaces, soil, and vegetation (including forest remnants, planted lawns, shrubs, and trees). In addition to this direct impact, development also increases human activity in extant wildland areas. We interpreted the direct threat to tanoak from development to be relatively less than from disease and silviculture factors, but greater than from fire and assigned it a weight of 10 (Table 1). To estimate tanoak area at risk from development, we produced a development density layer for the geographic range using 2006 data from the National Land Cover Database (NLCD) (Fry et al. 2011).

The NLCD classification system breaks its level 1 ''Developed'' class into four sub-categories based on a ratio of human-made impervious surfaces to vegetation present within 30 m × 30 m pixels as mapped from Landsat Enhanced Thematic Mapper (ETM+) imagery. Thus, as the relative proportion of impervious surfaces increases, the development ''intensity'' increases from a ''developed low intensity'' to ''developed high intensity'' category. We reclassified the NLCD low to high development intensity categories into our ranking system so that one, two, and three represented low, moderate, and high respectively, with the assumption that higher development intensity presents a greater threat to tanoak. We reclassified all remaining undeveloped NLCD classes to zero. We resampled this layer to 100 m × 100 m cells to match the smallest grain size of other spatial data being utilized. We then generated a development density surface by summing all rank values (0–3) within a 500 m radius (i.e., 1 km × 1 km rectangular neighborhood) of each grid cell location. This process effectively spreads some

development risk into adjacent undeveloped areas. We then reclassified the resulting development density map (values ranging from 0–75) into our common 0–3 ranking system using equal intervals.

Fire

Tanoak is a species well adapted to survive and recover from fire. Mature trees have thick, fire resistant bark, and it vigorously regenerates from basal sprouts following mortality of the above ground portion of the tree (Tappeiner et al. 1990). These characteristics enable tanoak to persist and often thrive in a wide range of fire regimes, including areas of low fire frequency (Hunter 1997), albeit with varying functional roles. We hypothesized that fire regimes altered from historic baselines as well as increases in potential fire severity from higher fuel loads result in greater threat to tanoak populations. Thus, we developed the fire disturbance factor to incorporate departure from historic fire regimes and potential fire severity. We represented departure from historic fire regimes using Fire Regime and Condition Class (FRCC) layers created for Oregon (USDA 2010) and California (CDF 2003). FRCC is an interagency, standardized tool for determining the degree of departure from reference condition vegetation, fuels, and disturbance regimes (Havlina et al. 2010). FRCC layers consist of ranked categories that quantify the difference between current vegetation conditions and fire frequency from historic reference conditions. Three ordinal categories rank the degree of departure from reference conditions in addition to a 'not applicable' category based on land cover type. We represented potential fire severity using fuel risk layers developed for Oregon (ODF 2006) and California (CDF 2005). The fuel risk layers were similarly categorized with three ordinal rankings of fuel risk and one indicating 'not applicable' due to land cover type. We recoded these existing categories of the fire regime departure and fuel risk layers to the 0–3 ranking scheme. We then summed the two layers and reclassified the resulting values (ranging from 0–6) to the 0–3 rankings. Given tanoak adaptations to fire and its occurrence and persistence under a variety of fire regimes, we assigned the fire factor the lowest weight of 5 (Table 1).

Disturbance Interactions

We calculated two-way multiplicative interactions for all possible disturbance pairs at a given location weighted by the assigned exponent (y, Eq. 1). In locations where more than two factors overlapped, we selected only the highest-ranking interaction for calculating the threat value at that location. Using this approach, we assumed that one set of disturbance interactions is the dominant threat to tanoak at a given location for the set of hypotheses (i.e., factor and interaction weights) being examined. Below, we describe our reasoning behind the weights assigned to each interaction parameter in our initial model (Table 1).

Disease x Fire

We assigned the disease-fire interaction a weight of two, reflecting our hypothesis that the threat to tanoak increases where these factors coincide. Importantly, *P. ramorum* has been shown to decrease average tanoak size (Davis et al. 2010; Cobb et al. 2012b), and tree size is closely associated with the likelihood of post-fire tree survival (Hengst and Dawson 1994; Kobziar et al. 2006). This disease also increases fuel loads (Valachovic et al. 2011; Cobb et al. 2012a), which are associated with increased soil damage following fire (Metz et al. 2011). Thus, we hypothesize that tanoak in disease-impacted areas are more susceptible to fire-caused mortality, and that dead material from disease would increase fire severity (particularly ground fire), further impacting tanoak recovery. Notably, slower or reduced tanoak recovery would decrease sources of *P. ramorum* inoculum.

Disease x Silviculture

We assigned the interaction of disease and silviculture factors a weight of two. This hypothesis is supported by research demonstrating the effectiveness of herbicide applications in causing tanoak mortality and reducing tanoak dominance (Tappeiner et al. 1987; Harrington and Tappeiner 2009), and the broad patterns of tanoak mortality (Meentemeyer et al. 2008) and reduced average tree size in disease impacted forests (Cobb et al. 2012b). In stands where these disturbances coincide, they have the potential to permanently remove large, mature tanoak trees. More broadly, actions such as salvage harvesting can increase the decline of dominant tree species already impacted by landscape level outbreaks of insects or pathogens (Kizlinski et al. 2002; Foster and Orwig 2006; Freinkel 2007). Tanoak generally has little or a restricted specialty market value, and so salvage harvesting is unlikely to occur for this species. However, herbicide use has a similar effect on tanoak populations except that it may be more efficient in reducing tanoak biomass (Tappeiner and McDonald 1984). Forestry practices may also decrease local tanoak populations to levels where *P. ramorum* is unable to invade stands, but these stands would likely be devoid of ecological functions unique to tanoak (Cobb et al. 2012b; Wright and Dodd this volume).

Development x Fire

This term aims to characterize the threat to tanoak from increased fire frequency related to development density. Syphard et al. (2007) showed that fire incidence is greatest at the wildland-urban interface; however, we did not have explicit evidence for this relationship within the geographic range of tanoak. It is also likely that these fires would be aggressively combated resulting in smaller fires with shorter burn times compared to more remote areas. Thus, we assigned the interaction of these factors a weight of one (Table 1).

Development x Disease

This term represents risk to tanoak based on the relationship between higher development density and increasing likelihood of disease introduction events. While development alone has no direct physical impact in this case, Cushman and Meentemeyer (2008) showed an increased probability of *P. ramorum* occurrence in forests near higher population densities, suggesting that roads, larger populations, urban and suburban landscaping, and heavier use of wildland recreation areas provide additional spatial pathways for pathogen movement and introductions. However, we did not interpret this relationship as producing a significant impact relative to other interactions and so assigned it a weight of one.

Development x Silviculture

Silviculture and development require infrastructure (e.g., roads) for transportation and accessibility. This infrastructure enables further silviculture, recreation, and development activities. We hypothesized that this interaction would nominally increase the threat to tanoak and so assigned it a weight of one.

Fire x Silviculture

While silviculture reduces average tree size and therefore predisposes individual stems to fire-caused mortality, we also hypothesize that it reduces the risk of wildfire ignition and may reduce potential severity. Additionally, tanoak adaptations producing robust sprouting and growth following fire or timber harvest allow for an increased likelihood of tanoak persistence when these factors interact, though most mature trees may be removed. According to these postulations, we assigned this interaction a weight of one.

Sensitivity Analysis

We tested model sensitivity to interaction parameters by varying interaction weights in a series of model runs. We ran the model with all interaction weights set to zero, which produced threat values for only the additive part of the model. While this sums the weighted rank values occurring at each location it does not provide insight into local factor interactions. We then ran the model with all the interaction weights set to one (equally weighted) and used multiple iterations to examine how results changed when each interaction term was assigned a weight of two while holding all other factors at one. These tests produced no zero values, indicating that at least one factor was present at each location in our map of tanoak distribution (Lamsal et al. 2011). Using equal intervals, we reclassified the resulting

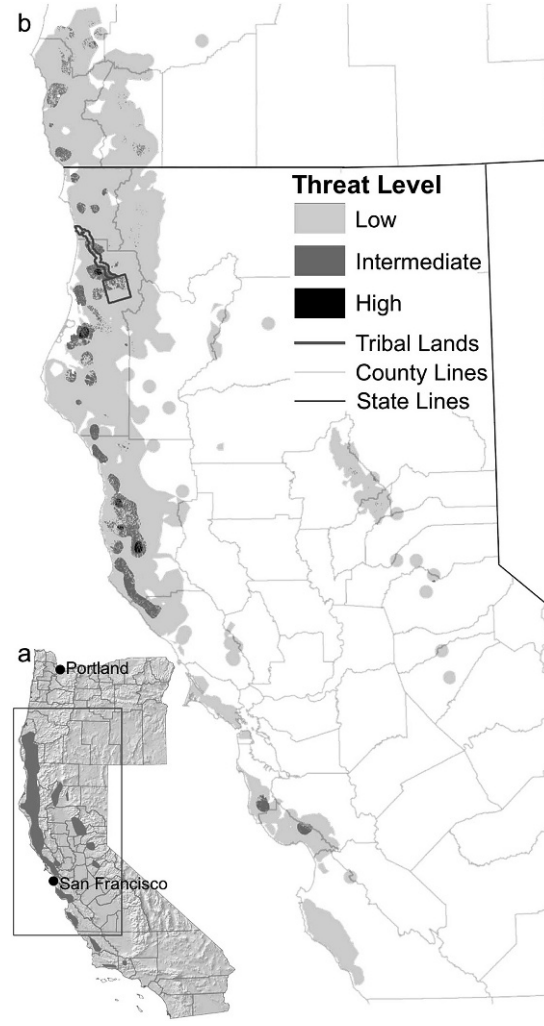

FIG. 1. The spatial distribution of (a) tanoak and (b) threats from weighted interacting disturbances (see Table 1) across tanoak's geographic range. Tanoak range adapted from Tappeiner (1990). Tanoak populations facing elevated threats were concentrated in Humboldt and Mendocino Counties, and partially located on Hoopa and Yurok tribal lands.

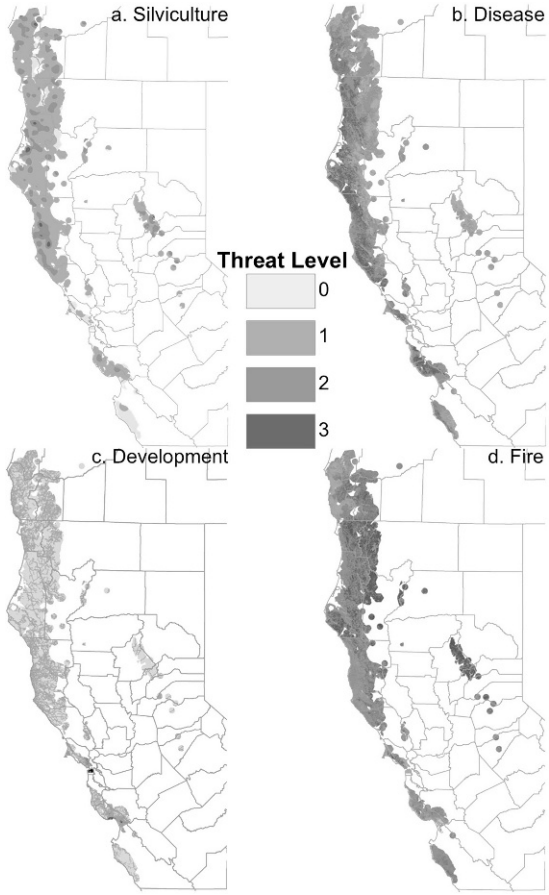

FIG. 2. Four disturbance factors overlaid on estimated tanoak area. Each map shows the classified threat level to tanoak from each disturbance factor across tanoak's geographic range, ranked zero to three ('none', 'low', 'intermediate', and 'high', respectively).

range of values from each model run into low (1), intermediate (2), and high (3) threat levels.

RESULTS

Risk from Disturbance Interactions

Across its geographic range tanoak predominantly faces low to intermediate threats from disturbance interactions, with smaller areas at high risk. The weights listed in Table 1 resulted in a threat map (Fig. 1) with 10,905 hectares (<1%) of estimated tanoak area at high risk, 222,795 ha (5.6% of estimated tanoak area) at intermediate risk, and over 3.7 million ha (94% of estimated tanoak area) at low risk from disturbance interactions. Elevated fire and silviculture risk factors overlap in the Sierra Nevada, whereas disease risk was low throughout much of this region (Fig. 2a, b, d). The influence of development on disturbance interaction risk was observed primarily in the San Francisco Bay Area, where it coincided with low values for other disturbance factors (Fig. 2c). Tanoak faces intermediate and/or high threats from disturbance interactions in 20 of the 30 counties within its geographic range. Using this model formulation, areas classified as intermediate and high threat occurred predominantly where disease and silviculture factors overlapped (Figs. 1 and 2a, 2b). The intermediate and high categories covered 233,700 ha containing an estimated 134.4 million tanoak, with 108.9 million (81%) of these trees located in Humboldt and Mendocino counties, CA. Some of the elevated threat categories are located in areas where tanoak has high cultural importance, including on Hoopa and Yurok tribal lands (Fig. 1).

Sensitivity analysis of the interaction weights demonstrated general robustness of the total amount of tanoak area classified into low and intermediate threat levels, with these two categories accounting for 91% to 99% of the total tanoak range across model runs with different interaction weights. With interaction weights all held at zero (the sum of weighted factors only), nine percent of tanoak area was at high risk, 73% at intermediate risk, and 18% at low risk. With all interaction weights set to one (the sum of the weighted factors plus the highest valued interaction at each location), six percent of tanoak area was at high risk, 24% at intermediate risk, and 70% at low risk. Results from these analyses, respectively, highlight areas where overlapping disturbance factors accumulate and where higher weighted factors coincide. In sensitivity analyses with each interaction weight increased to two while holding others constant at one, we found the disease-silviculture interaction produced the same result as the weighting scheme of the initial model (Table 1). The fire-silviculture interaction resulted in values similar to those produced by our initial model parameters: 6419 ha (<1%) at high risk, 222,790 ha (5.6%) at intermediate risk, and 3.7 million ha (94%) at low risk. Most significantly, the disease-fire interaction was most sensitive to changes in the interaction weight parameter and resulted in five percent of tanoak area at high risk, 31% at intermediate risk, and 64% at low risk. Development impacts on risk were generally small and consistently resulted in >99% of tanoak area in the low threat category. Of the three disturbance interactions that included development, the disease-development interaction resulted in the greatest area in intermediate (9990 ha) and high (1593 ha) threat levels.

DISCUSSION

Mapping the geographic distribution of disturbance factors that threaten foundation species is essential for understanding and managing popu-

lation and ecosystem impacts (Holdenreider et al. 2004; Ellison et al. 2005). Since many landscapes are influenced by multiple disturbances, spatially explicit tools identifying areas at risk from disturbance interactions are critical to conservation of threatened populations. These tools can be used for prioritizing limited resources for efficient and effective conservation of at-risk species.

Since European settlement, harvesting of tanoak bark, and the subsequent increasing application of herbicides by industrial forestry interests to favor more marketable conifer species (Bowcutt 2011), undoubtedly altered the structure and function of tanoak forests. Coinciding with these processes was an increasing human population resulting in development of forest and wildlands, and alteration of fire regimes to favor conifer species that were valued over tanoak in post-1950 timber markets. Remarkably, tanoak has shown substantial resilience under these adverse conditions, but the introduction of *P. ramorum* into tanoak ecosystems presents a new and significant threat to this species. Although diseases can increase extinction risk (Smith et al. 2006), it is unlikely that tanoak could be eliminated by this pathogen alone. Species extinction most often occurs when multiple stressors coincide to reduce at-risk populations to unsustainable levels (de Castro and Bolker 2005; Smith et al. 2006). Analogously, disturbance interactions, especially novel ones such as those resulting from impacts of an emergent pathogen like *P. ramorum* (e.g., Metz et al. 2011), may increase the likelihood of stand-level tanoak extirpation. Thus, the functional extinction of tanoak due to the removal of all or most large trees over broad areas may be more likely to occur where disturbances interact (cf., American chestnut blight, Paillet 2002; jarrah dieback, Podger 1972).

Cobb et al. (2012b, this volume) indicated that significantly more *P. ramorum*-caused tanoak mortality is likely to occur over the coming decades. This is largely due to the epidemiological role of tanoak in driving pathogen spread and disease emergence, and the high abundance of tanoak in climates favorable to *P. ramorum* (Meentemeyer et al. 2011). Annual variability of temperature and precipitation significantly impact the likelihood of pathogen establishment and spread (Rizzo et al. 2005; Davidson et al. 2011), which was reflected in the observed difference in risk of disease establishment between coastal and inland landscapes (Fig. 2b; Meentemeyer et al. 2004; Václavík et al. 2010).

Global to regional climatic changes are forecasted to influence fire incidence, and changes in fire frequency and intensity could affect tanoak resilience. Moritz et al. (2012) projected significant increases of fire frequency in the near future across much of the globe, including the west coast of North America. A significant portion of tanoak's geographic range coincided with intermediate and high threat categories for the fire factor in our analysis, emphasizing the threat to tanoak from altered fire regimes and increased fuel loads (Fig. 2d). The forecasted changes to fire regimes throughout tanoak's geographic range would increase the probability of interactions with other disturbances and consequently the threat to tanoak populations.

With this model, we identified locations where interacting disturbances have the potential to threaten tanoak populations. Knowing the geographic variation of disturbance interactions is fundamental for developing and implementing management strategies that are landscape appropriate. Managers and researchers can often identify the dominant disturbance influencing a landscape, but these events do not occur in a vacuum and individual disturbances can shape the nature and intensity of other events (Turner 2010). This model provides the capacity to identify, target, and test management treatments in the context of multiple disturbances and their interactions. For example, areas at high risk from interactions of silviculture and disease factors in Mendocino and Humboldt counties could be reduced through judicious use of "slow-the-spread" actions such as proactive thinning of smaller tanoak and California bay laurel. Meanwhile, maintaining tanoak contribution to ecosystem function in the face of silviculture could be accomplished by retaining large tanoak in stands managed for timber. Tanoak in the Sierra Nevada is primarily threatened by fire and silviculture interactions, again suggesting that retention of large tanoak in stands managed for timber would be appropriate to enhance habitat as well as maintain tanoak resilience to fire.

Using models to guide decision-making requires recognition of model assumptions and limitations, principally that results (in this case mapped disturbance interaction threats to tanoak) are often sensitive to the values of the input parameters. The weighting of disturbances and the interactions using the initial model parameters (Table 1) resulted in a map where the interaction of silviculture and disease factors produced intermediate and high threat levels to tanoak over a relatively small portion of tanoak's geographic range (Fig. 1). This essentially shows that high intensity silviculture and disease factors are concentrated in a few smaller areas. Also, these two factors could potentially be the most addressable by management action and minor alteration to forestry practices in these areas. Our sensitivity analysis demonstrated that the model is robust with respect to parameter values for disease and fire interactions. Further, these factors resulted in the greatest total tanoak area

at intermediate and high threat levels (>1.4 million ha, or 36%). This is indicative of the role of tanoak as a host of *P. ramorum* as well as the potential alteration to fire regimes in tanoak ecosystems following the establishment of *P. ramorum*. We emphasize that the results of our model represent threats based on hypothesized relationships among these disturbances. Field measurements are necessary to validate these expected outcomes and provide appropriate model updates for further predictions, such as actual measurements of tanoak mortality from each factor across a wide range of environments.

Apposite model interpretation is especially important when results are used to inform management actions, because misconception of either inputs or outcomes could lead to decisions that are contrary to stakeholder objectives. Through careful analysis, diverse management goals may be accomplished by applying more effective, or "designer" treatments to areas with distinct threats. For example, forests at low risk and currently unaffected by disturbances may be most appropriate for the establishment of refuge tanoak populations. Areas facing intermediate threat levels that also border regions with higher threat levels (Fig. 1) may be ideal for treatments that slow pathogen invasion into adjacent stands. The effective threat from interacting disturbances is temporally implicit, and the actual impacts to tanoak are dependent on the state of a stand, as well as the order and timing of disturbance events (Lorimer et al. 2009; Turner 2010). Therefore, the timing of treatments is an essential consideration. For example, fuel load reduction activities could also address disease risk by reducing densities of bay laurel and tanoak, but these treatments would be most beneficial when applied between July and the onset of winter rains to avoid introduction or spread of the *P. ramorum* pathogen (Davidson et al. 2008, 2011). Also regarding disease, management efficacy decreases with time since *P. ramorum* arrival (Filipe et al. 2012), highlighting the importance of treatments that may prevent establishment as well as rapid responses to new invasions in order to mitigate impacts (Meentemeyer et al. 2012). These actions can provide time and space needed to implement further treatments that reduce the cost of disease (and potential interactions with other disturbances) to local communities (Kovacs et al. 2011; Cobb et al. this volume). Lastly, policy changes favoring retention of high value tanoak habitat, especially in locations at high risk from disturbance interactions could be effective at reducing the rate and extent of tanoak population decline as well as maintaining biodiversity and ecosystem function.

Although the maps of areas at-risk from disturbance interactions are static, the databases used to produce them are typically dynamic as new data is acquired and analyzed over time. As new discoveries are made and our knowledge of disturbance interactions and their impacts evolves, model parameters can be updated and results tested in order to maintain reliability of recommendations. Through integration into an adaptive management framework, updates can be quickly applied, enabling new strategies to be developed and implemented in a timely and effective manner. The model framework may also be similarly applied to examine the spatial variation of threats to other species from disturbance interactions.

Conclusions

With increasingly limited resources it is important to rapidly identify target areas where management actions will have the greatest chance of achieving objectives. We propose that this model of threats to tanoak from interacting disturbances could be used as part of an adaptive management plan to bring stakeholders together in prioritizing and achieving conservation of the abundance, structure, and function of tanoak trees and ecosystems. Tanoak is by all accounts a resilient species, persisting and sometimes thriving under a variety of pressures. By applying knowledge and tools currently available, this resiliency can be enhanced, tanoak mortality may be reduced, and the vital services provided by tanoak ecosystems can be conserved for the health and prosperity of current and future generations.

Acknowledgments

We are grateful to Monica Dorning and four anonymous reviewers for helpful comments on previous versions of this manuscript. This research was supported by a grant from the National Science Foundation (EF-0622770) as part of the joint NSF-NIH Ecology of Infectious Diseases program, the Gordon and Betty Moore Foundation, and the USDA Forest Service, Pacific Southwest Research Station.

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Ecosystems (2016) 19: 1478–1490
DOI: 10.1007/s10021-016-0017-8

ECOSYSTEMS
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Litter Chemistry, Community Shift, and Non-additive Effects Drive Litter Decomposition Changes Following Invasion by a Generalist Pathogen

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Abstract

Forest pathogens have strong potential to shape ecosystem function by altering litterfall, microclimate, and changing community structure. We quantified changes in litter decomposition from a set of distinct diseases caused by *Phytophthora ramorum*, an exotic generalist pathogen. *Phytophthora ramorum* causes leaf blight and increased litterfall %N, but no mortality on California bay laurel (*Umbellularia californica*), a common overstory tree that accumulates high levels of infection. Lethal twig and bole cankers on tanoak (*Notholithocarpus densiflorus*) lead to the disease sudden oak death which creates canopy openings and alters litterfall in mixed-species forests dominated by redwood (*Sequoia sempervirens*) which is minimally susceptible. Species identity had the greatest effect on mass loss and N dynamics with the most rapid rates in bay laurel, slowest in redwood, and intermediate in tanoak. Decomposing litter from infected sources had increased N accumulation, and although these changes were of lower magnitude relative to species identity, the region-scale invasion of *P. ramorum* suggests that this effect could occur over an extensive area. Canopy mortality was a significant and slowing influence on litter N dynamics in all species and also dampened non-additive effects within mixed litter bags. Redwood—the lowest quality litter—demonstrated non-additive interactions with consistently lower C:N when decomposed in mixed litter bags, but this effect did not alter the entire mixture. Mortality and subsequent changes in community composition have the greatest magnitude effects on litter decomposition for sudden oak death, but our study implies that different and sometimes cryptic mechanisms will drive decomposition changes for other forest diseases.

Key words: *Phytophthora ramorum*; sudden oak death; tanoak; California bay laurel; tree mortality; forest disease; ecosystem function; carbon; nitrogen.

Introduction

Pathogens, regardless of indigenous and exotic origin, are important agents shaping carbon cycling in forest ecosystems (Lovett and others 2006; Hicke and others 2012). Disease emergence may alter carbon cycling by killing trees, reducing plant growth, redistributing plant resources into chemically distinct pathogen biomass, or through pathogen damage to specific plant tissues, such as leaves

Received 4 January 2016; accepted 20 May 2016;
published online 11 July 2016
Electronic supplementary material: The online version of this article (doi:10.1007/s10021-016-0017-8) contains supplementary material, which is available to authorized users.
Author contributions RCC and DMR conceived and designed the study as well as designed the field methods, analyzed the data, and wrote the paper. In addition, RCC performed the field and laboratory portion of the research.
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(Preston and others 2016). In turn, these impacts can alter biogeochemical processes by changing litterfall chemistry, amount, and timing as well as physical factors, including temperature, moisture, and light interception at the forest floor (Ruess and others 2009; Lovett and others 2010; Cobb and others 2013). Despite well-founded expectations of disease-driven changes to carbon cycling, few studies have quantified pathogen effects on forest communities and associated ecosystem processes; exceptions tend to focus on pronounced mortality events (Hicke and others 2012). However, many pathogens impact their hosts without causing mortality, creating potential for cryptic but spatially extensive changes to ecosystem processes (Hansen 1999; Ostry and Laflamme 2008). These effects are most likely to emerge through impacts of broad host-range generalist pathogens which may cause a range of disease severity within landscapes or communities (Eviner and Likens 2008).

This study aims to improve understanding of pathogen influences on litter decomposition, an important component of forest-level carbon cycling. Ecosystem-level changes have been more extensively quantified for insect outbreak and this knowledge provides a basis for predictions of pathogen effects on functional processes. For example, insect outbreak has been shown to change litter chemistry, microclimate, and competitive interactions that subsequently increase or decrease litter decomposition rates (Chapman and others 2003; Classen and others 2005; Cobb 2010). Similar ecosystem-level disease impacts can be expected to cause analogous changes to litter decomposition (Eviner and Likens 2008). However, pathogens differ from insects in terms of life history, dispersal, and nutrient acquisition (Hansen 1999; Hunter 2002) implying that an insect-pathogen analogy may not always be an accurate framework for predicting disease impacts to ecosystems. In particular, differential host impacts by generalist or multi-host pathogens have strong potential to drive disease effects on ecosystem processes (Holt and others 2003). For example, the heteroecious pathogen white pine blister rust (*Cronartium ribicola*) is a foliar disease on *Ribes* (sp.), but a stem and bole canker on *Pinus* (sp.) meaning that disease-driven effects on litter decomposition, such as altered litter chemistry or mortality-driven changes to microclimate, will differ among hosts and over the course of the disease cycle. Generalist pathogens, such as *Phytophthora cinnamomi*, cause diseases ranging from minor root infections to rapid mortality (Shearer and others 2007) on scales spanning stands to landscapes. Broad host-range insects, such as Gypsy moth (*Lymantria dispar*), will also alter litter decomposition in many host trees and at variable severities (Gandhi and Herms 2010). However, extreme population fluctuation of many damaging insects limits their analogy to *P. cinnamomi* or other root pathogens, such as *Armillaria* and *Heterobasidion* species, which cause mortality and shape community structure continuously over decades (Rizzo and others 2000; Shearer and others 2007). It remains unclear if biological differences among insects and pathogens can be linked in a single generalized framework of outbreak impacts to litter decomposition.

We quantify the implications of variable disease impacts on litter decomposition by focusing on a single pathogen which causes several distinct diseases. *Phytophthora ramorum*, an exotic oomycete pathogen of unknown origin, has caused landscape-scale tree mortality in mixed-species forests in coastal California and Oregon (Rizzo and others 2005; Garbelotto and Hayden 2012). The most overt of these diseases, sudden oak death, has resulted in extensive mortality of tanoak (*Notholithocarpus densiflorus*) and coast live oak (*Quercus agrifolia*) in California and Oregon beginning circa 1995. Tanoak is severely impacted in coast redwood (*Sequoia sempervirens*) forests because of favorable microclimate for the pathogen and a potent combination of high host susceptibility, prolific within-host sporulation on twig and leaf infections, and development of fatal cankers from bole infections (Davidson and others 2005; Rizzo and others 2005). Disease outbreak can lead to rapid loss of tanoak biomass, altered litter chemistry, and shifts in litterfall amounts and dynamics (Cobb and others 2012, 2013). However, *P. ramorum* also causes an innocuous, but widespread, leaf blight on a broad range of native woody and non-woody plants similar to diseases caused by other widespread exotic *Phytophthora* species that rarely kill trees (Wickland and others 2008; Hansen and others 2012). Foliage of California bay laurel (*Umbellularia californica*), a common overstory tree, is highly susceptible leading to prevalent infection within the canopy which increases mortality rates of neighboring tanoak. However, negative impacts to bay laurel health have never been documented (Davidson and others 2008; DiLeo and others 2009; Cobb and others 2010). *Phytophthora ramorum* infection in bay laurel increases foliar %N (Cobb and others 2013), implying that this region-scale invasion may alter decomposition over the long term and across a broad spatial area, including stands, where the pathogen does not cause tree mortality.

We tested and qualitatively compared four potential mechanisms controlling litter decomposition in coast redwood forests impacted by *Phytophthora ramorum*: (1) species effects and potential community composition changes, (2) infection-associated differences in litter chemistry, (3) physical-environmental change associated with canopy mortality, and (4) non-additive interactions emerging from mixing of different species with and without prior exposure to the pathogen. If infection leads to increases in litter quality (for example, decreased lignin or increased N), pathogen invasion should be accompanied by increases in decomposition rate. Similarly, changes in species composition should be expected to result in new baseline decomposition rates which reflect litter chemistry in the altered community (Hunter 2002; Lovett and others 2006, 2010; Eviner and Likens 2008). We expected non-additive interactions would be dependent on changes in litter chemistry, either via direct impacts of infection or shifts in species composition (Schweitzer and others 2005; Cobb 2010; Lummer and others 2012). Changes in the physical environment accompany the death of canopy trees, such as altered forest floor temperature and moisture availability, may also directly impact decomposition (Classen and others 2005; Cobb and others 2006). We conducted a two-year study of litter decomposition in two *P. ramorum* invaded redwood forests to evaluate these expectations and to assess the relative importance of each mechanism of decomposition change.

Methods

Field Sites and Litter Collection

We conducted litter decomposition measurements concurrently with a study of litterfall and soil N cycling at two sites, Jack London State Park (Jack London) and the Marin Municipal Water District (MMWD) located in Sonoma and Marin Counties, respectively (Cobb and others 2013). The sites are located within 45 km and both occur in a typically Mediterranean climate that receives approximately 1200–1500 mm y^{-1} during cool wet winters interspersed with annual summer droughts. A map of the study sites can be found in the supplemental information. We selected a subset of 15 plots from a pool of 30 at each site for litter decomposition measurements. The subset of plots was selected to maintain common soil types, cover the range of tanoak mortality and pathogen prevalence within each study site, and exclude plots without each of the three focal study species. Study plots were established in 2002 to study pathogen spread and disease impacts by mapping and measuring all stems with diameter at breast height (dbh) greater than 1 cm. Each plot is circular with 500 m^2 area; annual plot surveys from 2002 to 2007 provide estimates of infection and mortality rates (Cobb and others 2012). These data were used to calculate cumulative basal area killed by the disease in 2007, the year we initiated of our litter decomposition study. Tanoak biomass scales exponentially with individual tree basal area and provides a comparable metric of ecosystem-level disease severity among locations. Previous studies have found basal area to be associated with disease-driven changes to canopy structure and litterfall rates which in turn are likely to influence microclimate factors that determine litter decomposition rates (Cobb and others 2012, 2013). We collected a single season of autumnal litterfall in each plot which was subsequently used to construct litter bags. To ensure adequate amounts of litter material for this study, we collected supplemental litterfall immediately below dead tanoak and highly infected bay laurel at each site during September 2007. Plots with no known tanoak infections were present at each site; additional litter was collected in these plots also during September 2007 to augment the pool of uninfected reference litter. High pathogen prevalence in bay laurel at each site precluded collection of local uninfected litter; instead litter from the Big Creek UC Reserve (Monterey, Co.) was collected simultaneously and employed as a reference from uninfected source trees. This inclusion of off-site litter is justified by the previous study showing positive associations between bay laurel foliar N and infection prevalence across sites (Cobb and others 2013). Redwood litter was collected at each site and assumed to be uninfected given that infection prevalence is low within this species (Davidson and others 2005; Cobb and others 2010).

Field and Laboratory Methods

We constructed 1080 litter nylon decomposition bags with 1 mm mesh. Bags were filled with about 3.0 g air dry foliar litter in pure culture or equal part mixes of each species (~1.0 g). Four samples of the initial litter were saved for each litter type (species, infection status, and site), dried at 60°C for 48 h to determine initial moisture content, and analyzed at the UC Davis Analytical Lab (http://anlab.ucdavis.edu/) using a total combustion method for C:N on a TruSpec C and N Analyzer and lignin measured gravimetrically following extrac-

tion with 72% H_2SO_4 and ashing of acid detergent fiber residue. This method of lignin determination also provides an estimate of cellulose concentration which we do not report. Our design produced five types of litter bags containing a single species: redwood or either infected or uninfected bay laurel and tanoak. We also constructed four types of three-species mixed litter bags, where bay laurel and/or tanoak were from infected and/or uninfected sources and decomposed in mixture with a site-specific redwood litter. Our design produced one full set of litterbags per plot. Bags were placed at 270° and 8 m from the plot center. Bags were deployed at both sites in mid-December 2007 (less than three months after litterfall) and recovered after 3, 6, 12, and 24 months. Each bag was air dried for several weeks, opened, and gently cleaned of debris using a brush and deionized water to remove mineral soil adhered to litter surfaces; mixed-species bags were also sorted to species. Cleaned litter samples were immediately placed in a drying oven at 60°C for 48 h and weighed. Animal damage, excessive mineral soil contamination, and vandalism did not occur in our study resulting in a complete recovery of all 1080 bags. Each sample was ground to pass an 850 μm mesh screen and analyzed for C and N content.

Data Analysis

Statistical models were constructed to examine the significance of pathogen-associated effects on litter decomposition and facilitate comparison among four pathways of change: (1) effects of foliar chemistry differences at the species level, (2) effects of host-level changes in foliar chemistry, (3) effects of canopy damage, and (4) effects of non-additive interactions driven by pathogen-caused changes in foliar chemistry. Prior to construction of these models, differences in the initial litter chemistry were determined with a one-way ANOVA, including a single variable combining species and infection status ($N = 36$). In conjunction, exponential decomposition constants (k) were calculated using non-linear regression of changes in mass loss over time fitted to a single-parameter exponential function (Adair and others 2010) for each species and treatment, including dynamics within mixed litter bags when appropriate. Non-additive decomposition dynamics were calculated for each analysis parameter i (mass loss, k values, N, and C:N) by calculating the differences (Δ) for each species or litter type (j) between observed and expected values at the respective collection time point (t):

$$\Delta_{j,i,t} = \text{observed}_{i,t} - \text{expected}_{i,t} \quad (1)$$

where the expected values are those of the respective litter-type decaying in a single-species litter bag. Although Eq. (1) provides insight into non-additive dynamics within mixed litter bags, it does not provide a test of whether mixing litter types changes the decomposition of the entire litter mixture which could have implications for ecosystem-level C and N dynamics (Ball and others 2008). Therefore, non-additive dynamics of the overall litter mixtures were estimated by a similar calculation except that expected values for the overall dynamics of a mixed litter bag were the sum of the respective species or litter-type decaying alone:

$$\Delta_{i,t} = \text{observed}_{i,t} - \sum_{j=1}^{3} \text{expected}_{j,i,t}. \quad (2)$$

Expected values for the overall mixed litter bags were adjusted for minor differences in the initial litter mass (~0.01 g) between species. Expected k values for the overall mixed bags were calculated by first using Eq. (2) to estimate expected mass loss values for the overall mixed litter bag and then fitting these values to the single-parameter (k) decomposition models. For both equations, expected C:N ratios were calculated by first determining the C and N masses (either observed or expected) for a respective time point and then finding the C:N ratio, as opposed to summing ratios directly. Differences in decomposition dynamics among species in pure cultures and overall mixed litter cultures were analyzed with a mixed linear model by including site as a random factor and time, dead tanoak basal area, and litter type as fixed effects. This model structure was applied to three subsets of our overall data set that reflect key mechanisms by which disease may influence decomposition processes: (1) overall differences in decomposition dynamics among species and infection status ($N = 600$), (2) differences from expected values (non-additive dynamics) for species decomposed within mixed litter bags ($N = 1440$), and (3) overall changes in mixed litter bag decomposition due to non-additive dynamics ($N = 480$). Effects of canopy mortality on decomposition were included and tested within each model, this provided estimation of two and three-way interactions among non-additive dynamics, canopy damage, and time for data subsets 2 and 3. When significant, mortality effects were examined further by extracting the partial residuals for the mortality effect; this yields the multiple regression equivalent of comparing independent vs dependent

variable(s) in a single-parameter regression (linear) model. For each subset of data, identical models were applied to analyze mass loss, %N, and C:N. k values were analyzed with the same model except that time is part of the k parameter estimation rather than a component of the statistical model. When fixed effects were significant, a Tukey's multiple linear contrast was implemented to estimate specific treatment differences. Each model was evaluated for normal error distribution and homogeneous variance across levels of the predictor variables, but no transformations were necessary to meet these assumptions. All analyses were performed in R version 3.1.1 with statistical significance considered, where $P \leq 0.05$; partial residuals were calculated using the package 'visreg' version 2.2-2.

Results

The initial litter chemistry differed among species and infection status with the most consistent significant differences occurring between bay laurel and redwood. Bay laurel and tanoak from infected sources had significantly greater %N compared to redwood ($P = 0.023$ and 0.004, respectively), but the respective litter from uninfected sources of each species was not significantly different from redwood ($P = 0.87$ and 0.51, respectively). C:N levels followed the same patterns of significance as %N (Table 1). Within tanoak and bay laurel, neither %N or C:N significantly differed between litters from infected versus uninfected sources. Lignin amounts were more variable across species and infection status. Bay laurel (infected and uninfected) had lower lignin than tanoak and redwood ($P < 0.001$ each contrast) with the exception of bay laurel and tanoak from uninfected sources which were not significantly different ($P = 0.141$). Tanoak from uninfected sources had significantly lower lignin content compared to tanoak from infected sources ($P = 0.044$), but tanoak lignin levels were not significantly different from redwood irrespective of infection status ($P > 0.05$ each contrast).

Decomposition was highly seasonal with the greatest amounts occurring during the wet season of each year (Nov-Mar) and minimal during the annual summer drought (April-Oct); this resulted in a distinctly stepped pattern of decomposition, particularly for litter mass loss (Figure 1). Decomposition dynamics tended to mirror the respective quality of the initial litterfall chemistry; bay laurel was most rapid in terms of mass loss, k values, and N change, tanoak was intermediate, and redwood had the slowest rates. Mass loss of bay laurel was significantly greater compared to tanoak and redwood ($P < 0.001$, both contrasts), but the latter two species did not significantly differ ($P = 0.084$). Infection status did not significantly affect mass loss rate for either bay laurel or tanoak ($P > 0.93$, each contrast). The same patterns of decomposition and statistical significance were found for decomposition constants (k values). Chemistry of decomposing litter generally reflected the initial chemical differences among litter types, and these differences became more pronounced over time. C:N and %N followed similar patterns with bay laurel litter having the greatest N accumulation over time (lowest C:N) and redwood having the least N accumulation; tanoak was intermediate to the other species (Figure 1). Infection status influenced the variation of %N in decomposing litter within species, but only resulted in significant differences vs. litter from uninfected sources for tanoak ($P < 0.001$) and not bay laurel ($P = 0.059$). However, infection status resulted in significant C:N differences within both bay laurel ($P = 0.048$) and tanoak ($P < 0.001$). Variation due to infection status was great enough that the general pattern of differences among species was not always significant. Infected bay laurel and uninfected bay laurel were not significantly different from infected tanoak for either %N or C:N ($P > 0.05$, each contrast). Other differences were dependent on the chemical parameter; C:N of uninfected bay laurel

Table 1. Initial Litter Chemical Characteristics

Species	Status	% N	C:N	% lignin
Bay Laurel	Uninfected	0.56 (0.02)^{ab}	92.0 (3.0)^{ab}	17.1 (2.0)^{ab}
	Infected	0.73 (0.01)^{b}	72.1 (1.2)^{a}	15.0 (1.1)^{a}
Tanoak	Uninfected	0.60 (0.04)^{ab}	89.7 (6.0)^{ab}	20.7 (2.1)^{bc}
	Infected	0.78 (0.10)^{b}	75.6 (9.3)^{a}	24.3 (3.6)^{d}
Redwood	Uninfected	0.47 (0.03)^{a}	115.0 (6.7)^{b}	23.5 (2.4)^{cd}

Data are means with one standard error in parenthesis. Values not connected by a common letter are significantly different at $P \leq 0.05$.

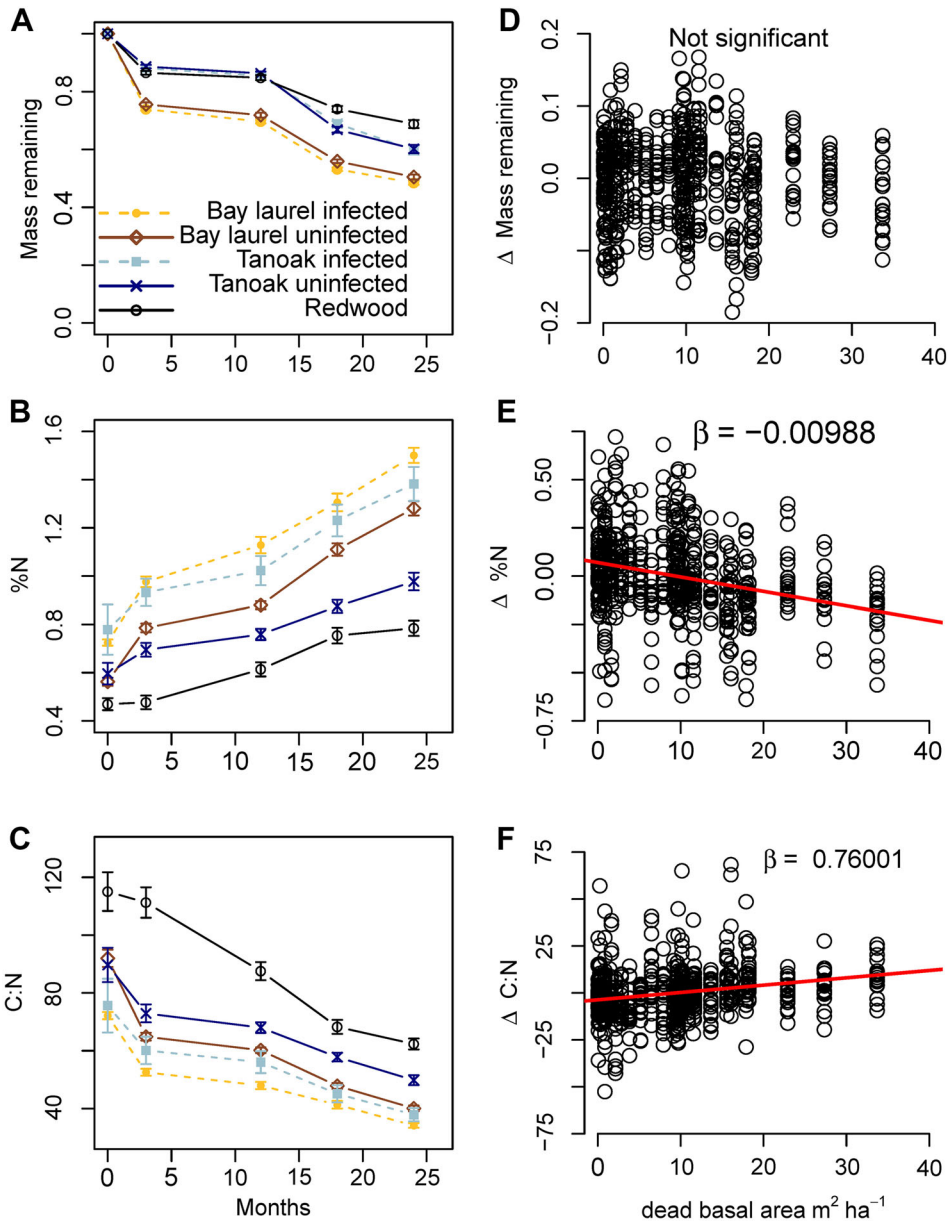

Figure 1. Effects of species identity, infection status, and canopy mortality on litter decomposition, the three most important hosts of *Phytophthora ramorum* in California redwood forests. **A–C** Effects of species and infection status for mass loss, %N, and C:N, respectively. **D–F** Effect of tanoak canopy mortality on the respective aspect of decomposition; points are the partial residuals for canopy mortality accounting for the effects of all other factors in the model. Least-squares parameter estimates are shown when significant.

and tanoak were significantly different from redwood ($P < 0.001$ both contrasts), but %N was not ($P = 0.14$ and 0.72).

Canopy mortality significantly affected %N and C:N of decomposing litter, but not mass loss (Figure 1). Litter N accumulation, both in terms of changes in %N and C:N, decreased linearly with dead tanoak basal area (C:N increased with increasing dead basal area). With two exceptions, all litter-type * canopy mortality and litter-type * canopy mortality * time interactions were not significant in our statistical models. Significant interactions were found in tanoak litter from uninfected sources and redwood; for these litter types, canopy damage significantly lowered C:N with increasing dead tanoak basal area indicating that slowed N accumulation in these litter types was at a low magnitude compared to other litter types. These positive species * dead tanoak basal area interactions ($P = 0.038$ and 0.001, respectively) revealed that the slowing effect of dead tanoak basal area on changes in C:N was moderated over time for the two lowest quality litter types included in the study (Table 1). Overall, the effect of canopy mortality was a significant influence on litter N dynamics, but small relative to variation due to species and infection status (Figure 1).

Within mixed litter bags non-additive dynamics primarily occurred in redwood, but interactions with canopy damage conditioned these effects.

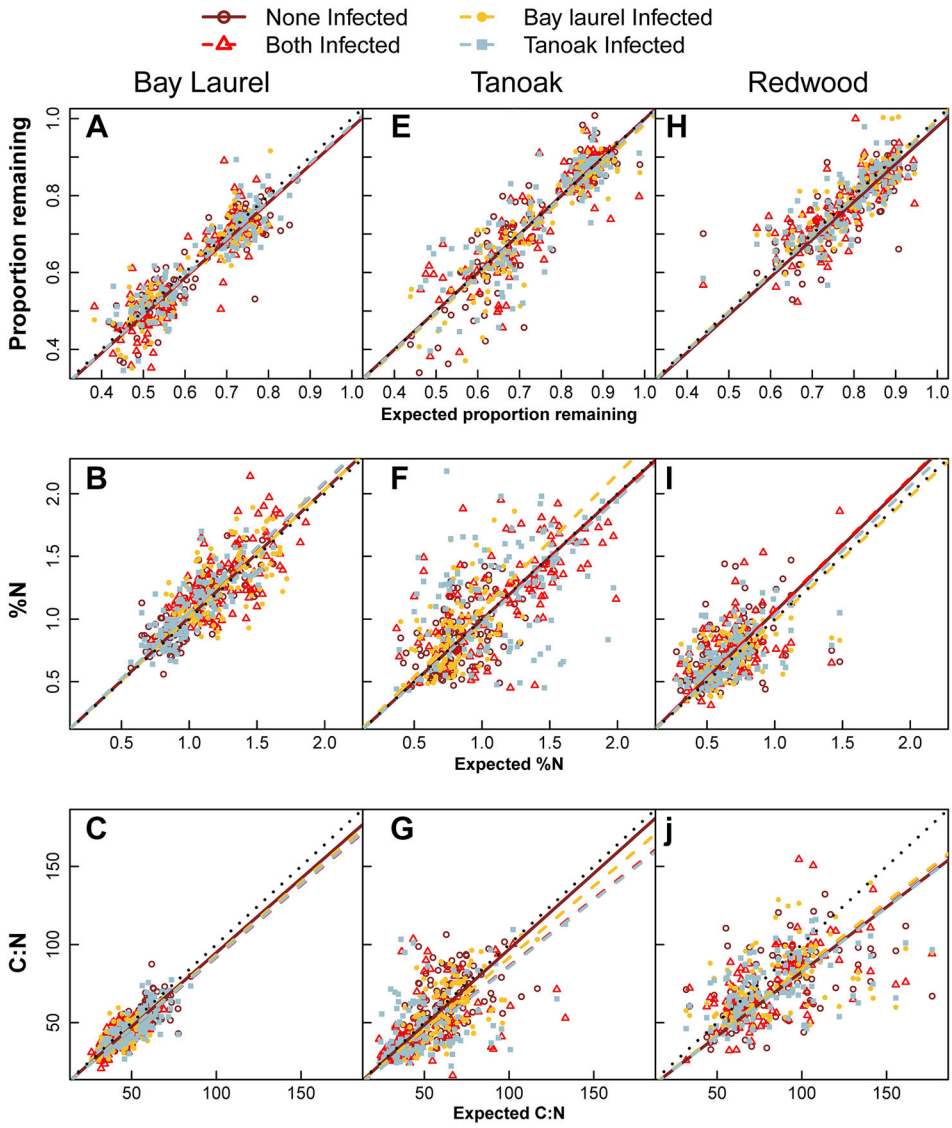

Figure 2. Non-additive effects on bay laurel, tanoak, and redwood litter decomposition across a full-factorial combination of bay laurel and tanoak from infected and uninfected sources. In each case, the *dotted one-to-one line* is the expected relationship from each litter-type decaying alone. Expected values were calculated using Eq. (1) for each species and decomposition parameter.

Models of mass loss provided no evidence of non-additive dynamics for any species (Figure 2), across time, or levels of canopy damage. Patterns of k values generally followed those of mass loss with the exception that k values for redwood (lowest quality litter) incubated with infected bay laurel ($P = 0.012$; highest quality litter) were significantly lower than those of single-species redwood litter bags. Extent of dead tanoak basal area alone did not influence non-additive effects on k values, but several significant interactions suggested positive effects of dead basal area on decomposition rates (k values) in some litter mixes. Infected bay laurel litter incubated with infected tanoak had higher k values (more rapid decomposition rate) with increasing dead tanoak basal area ($P = 0.026$). The same trend was found for uninfected bay laurel litter decomposing with infected tanoak, although, here, the interaction was not significant ($P = 0.099$). In addition, redwood incubated with infected bay laurel had increasing k values with increasing dead tanoak basal area ($P = 0.015$). Non-additive dynamics were more frequent for %N and C:N. Redwood, the lowest quality litter, was the only species to demonstrate consistent non-additive dynamics with consistently lower C:N values relative to expected values for all mixed litter treatments ($P < 0.001$, all contrasts; Figure 2). However, the magnitude of these departures from expected values was not different among litter treatments, suggesting that the degree of non-additive effects for redwood C:N dynamics was independent of the overall litter mixture quality. Dead tanoak basal area dampened non-additive dynamics as indicated by significantly slower changes in C:N relative to each litter-type

decaying in pure culture across plots with increasing mortality ($P = 0.025$); these patterns were also implied by %N data, although the effects were not statistically significant ($P = 0.083$). However, significant statistical interactions between dead tanoak basal area and time ($P = 0.047$, C:N; $P = 0.029$, %N) reflect that C:N and %N values reached values similar to expected by the end of measurements, suggesting that the interactions of non-additive dynamics and changes in the physical environment occurred early in the incubation of the litter bags and were no longer evident at the end of measurements. Although these non-additive C:N dynamics were evident for all three species, the magnitude of change was greatest for redwood, the lowest quality litter type (Figure 2).

Mixed litter incubations followed patterns similar to those from single-species litter bags. Litter mixing treatments did not significantly affect mass loss, k values, or %N; however, C:N was significantly higher in litterbags with both tanoak and bay laurel from uninfected sources relative to each of the other treatments ($P < 0.043$, each contrast; Figure 3). Decomposition rates and dynamics of mixed litter bags (entire mixture) showed no evidence of non-additive dynamics for any treatment or chemical parameter, including C:N. In addition, none of the interactions with dead tanoak basal area that were detected for individual species were found for the overall mixtures. These patterns suggest that the differences in C:N among different litter mixtures (Figure 3) were additive effects of the litter types composing each mixed litter treatment (Figure 1).

Discussion

Our study highlights a relatively underappreciated characteristic of forest diseases: a single generalist pathogen can be responsible for multiple diseases that affect decomposition by different mechanisms. This variation creates a mosaic of effects and drivers which range from subtle to substantial. For *P. ramorum* in California redwood forests, widespread mortality and shifts in species composition will have the most substantial and long-lasting effects on litter decomposition. However, pathogen effects on bay laurel foliar chemistry (Cobb and others 2013) are likely to have low-magnitude, persistent, and spatially extensive effects on decomposition in these ecosystems due to the susceptibility and broad distribution of this important overstory tree. Similar low-magnitude changes to decomposition can be expected for spatially extensive *Phytophthora* pathogens which are endemic or actively invading natural ecosystems worldwide (Hansen 1999; Hansen and others 2012). This kind of subtle but widespread effect will go undetected in most ecosystems given the lack of study on pathogens which do not cause extensive mortality (Desprez-Loustau and others 2007; Ostry and Laflamme 2008).

Individual species litter chemistry has substantial control over litter decomposition, and this study suggests that *P. ramorum*-mediated shifts in tree-species composition will have the largest magnitude impacts for this disease. The magnitude of differences among species was greater than any other mechanism we measured (Table 1; Figure 1) and changes in litterfall chemistry associated with altered overstory composition will result in the long-term effects, assuming that these canopy gaps are eventually colonized by trees which are minimally impacted *P. ramorum*. The importance of species shifts during insect or pathogen outbreak has been demonstrated both through field study and synthesis (Chapman and others 2003; Eviner and Likens 2008; Cobb 2010; Gandhi and Herms 2010; Lovett and others 2010). Our redwood-dominated study systems are mixed-species communities that include disparate overstory tree competency to transmit the pathogen as well as disparate pathogen impacts among these hosts (Davidson and others 2008). This epidemiological variation creates broadly predictable patterns of mortality, where susceptible hosts in locations with high inoculum pressure experience the most rapid and extensive mortality (Holt and others 2003; Cobb and others 2010). In the case of *P. ramorum*, rapid tanoak mortality in stands with high inoculum pressure from bay laurel creates canopy space for the establishment of either bay laurel, redwood, or other species. Sudden oak death emerged in these forests in the last 20 years and the timing of overstory tree establishment as well as the replacement species remains unclear (Ramage and others 2011; Cobb and others 2012; Metz and others 2012). However, our data set indicates that when redwood establishes in these new canopy gaps, litter quality and decomposition rates will slow; in contrast, when bay laurel increases in dominance, litter decomposition will accelerate (Figure 1). For other forest diseases, understanding patterns of mortality and species-driven shifts in litter chemistry will provide robust predictions of litter decomposition change in terms of magnitude, timing, and duration.

In our study of sudden oak death, canopy mortality had a linear, slowing effect on N dynamics in decomposing litter (Figure 1). Outbreak-driven

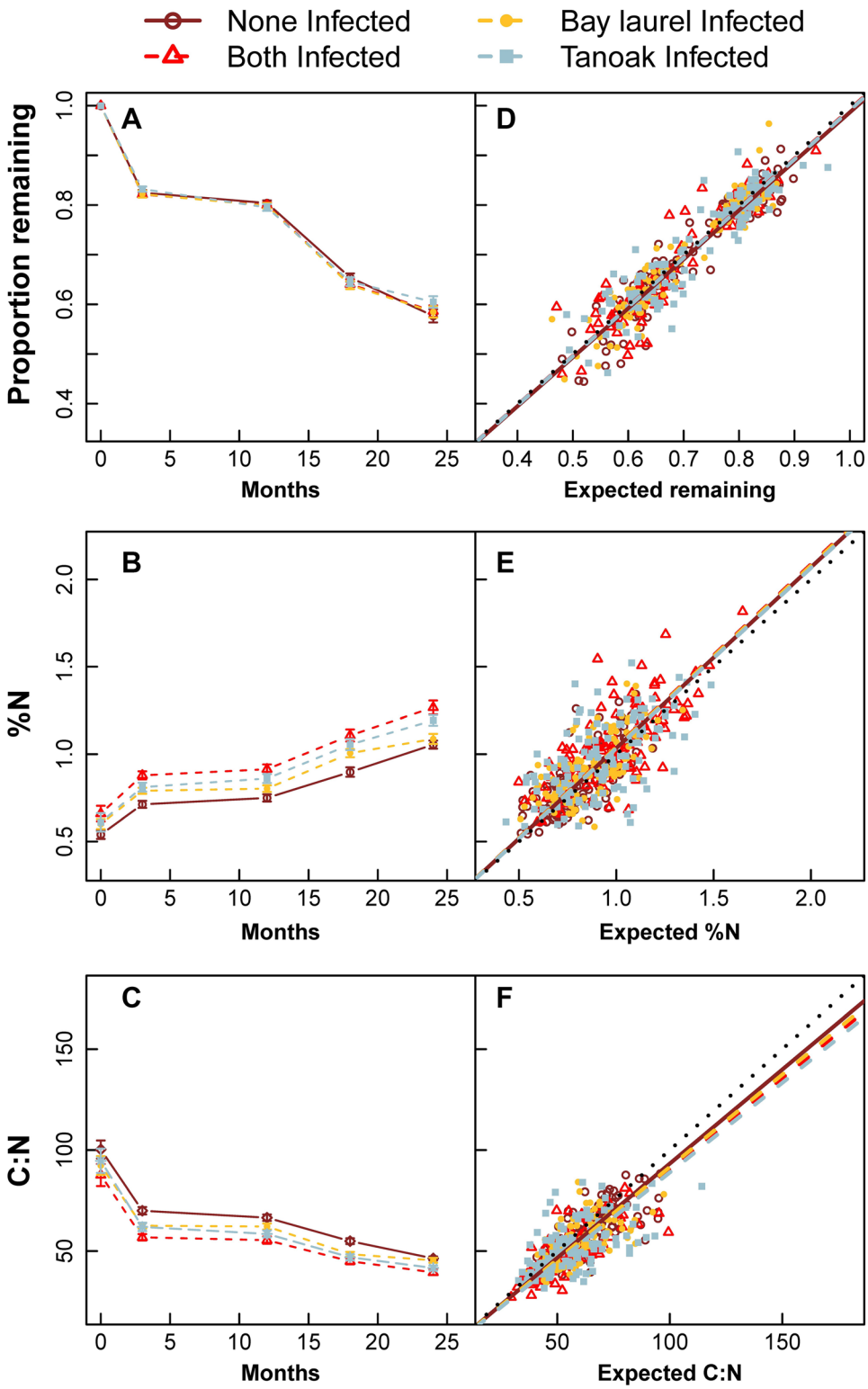

Figure 3. Dynamics of the overall mixed litter bags with treatments spanning a full-factorial combination of bay laurel and tanoak from infected and uninfected sources (**A**–**C**). Each litter bag contained the same proportion of each litter type (1/3), but the infection status of bay laurel and tanoak was varied among treatments. The analysis of non-additive dynamics is shown in panels **D**–**F,** where expected values were calculated using Eq. (2) for each species and decomposition parameter.

canopy mortality has been shown to increase (Classen and others 2005) or decrease (Cobb and others 2006) soil moisture and accelerate or slow (respectively) subsequent decomposition rates. The distinct stepped pattern of mass loss across all species is the characteristic of strong seasonal moisture limitation in California Mediterranean ecosystems (Hart and others 1992). Although we did not directly measure soil moisture as part of this study, these negative relationships are consistent with

canopy mortality causing decreased moisture availability to decomposers. Positive litter-type * host mortality and time * host mortality interactions suggest that such moisture limitations would have been lower for some litter types or may have decreased over time, as litter became buried in the forest floor. Extensive tanoak basal sprouting accompanies canopy mortality which creates a low-statured and dense stand relative to uninvaded forests (Ramage and others 2011; Cobb and others 2012; Metz and others 2012) including in the study plots utilized here. Extensive resprouting maintains *P. ramorum* even when bay laurel is not present and may also slow recruitment of non-basal sprouting species even when they have lower susceptibility (Harrington and Tappeiner 2009; Cobb and others 2012). Presuming that the slower litter N dynamics found in this study are due to lower moisture availability, we expect these changes will persist until overstory trees are reestablished which in turn will be slowed by competition from resprouting tanoak. Given that these effects were linear, our data suggest that the importance of microclimate change on litter decomposition for other forest diseases will depend on the extent and rate of canopy mortality in addition to canopy structural changes that determine the duration of these impacts (Lovett and others 2006; Eviner and Likens 2008).

Previous research using the same plots employed in this study showed a modest and seasonally dependent increase in bay laurel litterfall %N with increased *P. ramorum* prevalence (Cobb and others 2013). Infected bay laurel leaves have accelerated abscission rates compared to uninfected leaves (Davidson and others 2011); shedding of attacked leaves is a plant defense response to insects that may affect litter decomposition rates (Hunter 2002). In this study, increased litter %N may be a plant defense tradeoff dynamic, where bay laurel leaves senesce prior to resorption of all available nutrients and gain the benefit of reducing leaves with blight caused by *P. ramorum*. Recent synthesis has identified parasite biomass as a potential influence on biogeochemical dynamics (Preston and others 2016), especially when parasite tissues have high biomass or relatively high N concentration and very low concentrations of recalcitrant compounds, such as lignin. These effects are likely for hemiparasitic plant parasites, such as mistletoes and root disease pathogens, including *Armillaria* and *Heterobasidion* species, which can accumulate large amounts of biomass with chemistries very distinct from their hosts. An assessment of *P. ramorum* biomass N as a component of bay laurel litterfall N is not possible with our data set, although *P. ramorum* is a weak saprotroph, suggesting that pathogen biomass is primarily located in a relatively small portion of the leaf (Davidson and others 2005). Arguably, *P. ramorum* has effects at the ecosystem scale which greatly exceed the organism's biomass relative to mistletoes and root diseases. Overall, these direct effects of *P. ramorum* on litter chemistry were of relatively low magnitude (Cobb and others 2013) and similar to the impacts of hemlock woolly adelgid (*Adelges tsugae*), an exotic piercing-sucking foliar insect of eastern hemlock that causes ecosystem changes disproportionate to its biomass (Rubino and others 2015).

Inference on bay laurel litter decomposition and leaf-level chemistry changes is constrained by our use of litter from an independent site, where *P. ramorum* and other common bay laurel leaf *Phytophthora*, such as *P. nemorosa* and *P. pseudosyringae*, had never been recovered at the time of collection (Wickland and others 2008). Although the use of bay laurel litter from an independent site could introduce unaccountable site-specific variation into our statistical models, there is no evidence that this actually confounded our inferences given the consistency of litter chemistry patterns across data sets (compare Cobb and others 2013). Of course, legal restrictions and ethical principles preclude a full-factorial transplant of infected bay laurel litter or experimental introduction of this damaging invasive pathogen to uninvaded forests. Therefore, our data should be viewed as the best surrogate for idealized experiments and leveraged as predictions of litter decomposition changes by future *Phytophthora* invasions. Tanoak litter %N and C:N also did not differ between infected and uninfected sources, again in agreement with litterfall data from these sites. In contrast, lignin differed significantly among species and between tanoak litter from infected vs uninfected sources (Table 1); this chemical parameter was not assessed in the previous litterfall study (Cobb and others 2013). Dead tanoak retains foliage for several years following mortality, a characteristic of the disease which determines the distribution and dynamics of canopy fuels (Kuljian and Varner 2010). Higher tanoak lignin content from infected sources is consistent with several years of slow litter decomposition in the canopy prior to senescence, collection, and incubation in litterbags. Therefore, these patterns may be a byproduct of bole infections in the plant, rather than induction of chemical changes in response to infection. For both tanoak and bay laurel, mass loss was unaffected by infection status, but N accumulation (%N, C:N) was

greater for both species when litter was from infected sources (Figure 1). Assuming that higher initial %N is the mechanism driving that these changes in infected bay laurel litter and that changes in tanoak are due to canopy leaf retention by dead trees and consequent increases in lignin concentration, we can make the following predictions: infection in bay laurel will cause a low-magnitude but potentially widespread and long-term change by altering litter decomposition N dynamics, while infection in tanoak will cause a similar but short-term change that will persist for the duration of canopy mortality and leaf retention in dead trees.

Although non-additive interactions have been demonstrated for insect outbreaks, mixtures of genotypes, and canopy species removals which alter the chemical composition of the initial litter mixes (Schweitzer and others 2005; Ball and others 2008; Cobb 2010), we found modest and context-dependent evidence of non-additive interactions in sudden oak death impacted redwood forests. Non-additive mixed litter interactions were primarily found in redwood, our lowest quality litter, and were only significant for C:N dynamics (Figure 1). These non-additive interactions occurred irrespective of the infection status or chemical quality of the other leaf litters suggesting that non-additive interactions affect redwood litter decomposition whenever it is mixed with bay laurel and tanoak. Given that redwood co-occurs with bay laurel and or tanoak in most of the species' range, these non-additive interactions are likely widespread within individual stands and across the range of these forests. Microbial community differences between mixed and single-species litter bags (Chapman and others 2013) and N transfer from relatively high-quality litter to low-quality litter during decomposition (Berglund and others 2013) are potential mechanisms for these patterns. However, we did not quantify these mechanisms and caution that non-additive dynamics can be context-dependent (Quested and others 2005) and that N transfer has also been shown to flow from low-quality litter to high-quality litter in mixtures (Lummer and others 2012). Although we found evidence that non-additive dynamics were sensitive to changes in the physical environment, these also became less evident as litter became more decomposed and were insufficient to change the dynamics of the entire litter mixture (Figure 3). In contrast to litter removal experiments, our study did not compare the effects of tanoak removal (Ball and others 2008) primarily, because tanoak remains a significant portion of the litterfall even when canopy mortality is extensive (Cobb and others 2013). If these non-additive dynamics are driven by mixing relatively high-quality litter (such as bay laurel and other similar species) with redwood, these interactions will be maintained even if tanoak is locally extirpated.

Ecosystem impacts of disease have received relatively little ecosystem-level study which forces ecologists to borrow expectations from studies of other disturbances, particularly insect outbreak (compare Eviner and Likens 2008; Hicke and others 2012; Preston and others 2016). Although this is a reasonable point of departure, spatial patterns and timing of mortality caused by *P. ramorum* are distinct from many insects where ecosystem-level studies have been conducted, particularly low diversity forests, such as mountain pine beetle (*Dendroctonus ponderosae*) outbreak in lodgepole pine (*Pinus contorta*) and hemlock woolly adelgid in eastern hemlock forests (Edburg and others 2012; Orwig and others 2012). Although *P. ramorum*-caused mortality significantly impacts forest structure and function, the majority of aboveground biomass in redwood forests will not be killed directly by the pathogen (Cobb and others 2010; Metz and others 2012). Dispersed mortality across relatively diverse forests is likely to reduce the importance of microclimate effects on litter decomposition as spatial scale is increased (Edburg and others 2012). In contrast to sudden oak death, root diseases often cause spatially concentrated and relatively uniform mortality that can dramatically alter understory conditions (Rizzo and others 2000; Hansen and others 2012). For these diseases, changes in microclimate should be expected to have greater influence on litter decomposition compared to our study of *P. ramorum* in redwood forests. In other cases, biological similarities of insect and pathogen impacts can provide simple and accurate generalizations. Passively dispersed foliar pathogens and insects are likely to alter decomposition by common mechanisms. For example, hemlock woolly adelgid appears to modestly alter eastern hemlock (*Tsuga canadensis*) foliar chemistry and subsequent C:N dynamics in decomposing litter (Cobb and others 2006), but canopy mortality and species shifts induce the greatest magnitude changes in ecosystem processes (Orwig and others 2008; Cobb 2010). Finally, insect-pathogen complexes can alter forest composition and carbon dynamics (Lovett and others 2010) and predictions of ecosystem change must integrate the biology of each damaging agent as well as the characteristics of the impacted forest. Further quantification of changes and explicit comparisons of pathogen

versus insect impacts on ecosystem processes will improve predictions of how and when decomposition and other aspects of carbon cycling are altered by outbreak (Hicke and others 2012).

Phytophthora ramorum and other destructive, broad host-range host plant pathogens, alter ecosystems through a suite of ecosystem-level effects that quickly become baseline conditions, as these organisms become entrenched in a new range. Understanding these changes underlies predictions of the ecological costs incurred by forest diseases as well as meaningful management responses to their impacts. In the case of *P. ramorum*, widespread but low-magnitude changes to decomposition driven by altered foliar chemistry likely approximate impacts to decomposition caused by other widespread forest *Phytophthora* species and invasive fungi (Desprez-Loustau and others 2007; Wickland and others 2008; Hansen and others 2012). These changes create new baseline conditions that are unlikely to be affected by management actions for pathogens with broad host ranges and long-term survival in the environment. In contrast, changes to species composition and canopy mortality that underlie the greatest *P. ramorum* impacts to litter decomposition could be shaped or moderated by informed management to reestablish overstory trees.

ACKNOWLEDGEMENTS

We thank H. Mehl and C. Shoemaker for their field and laboratory support of this research. We thank J. Ashander and S. Ibanez for feedback on the statistical analysis and Gary Lovett and two anonymous reviewers for helpful comments on earlier versions of this manuscript. We are grateful to the California State Parks and the Marin Municipal Water District for facilitating this research on their lands. This work was funded by NSF Grant DEB EF-0622770 as part of the joint NSF-NIH Ecology of Infectious Disease program, the Gordon and Betty Moore Foundation, and the USDA Forest Service Pacific Southwest Research Station.

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Resiliency or restoration: management of sudden oak death before and after outbreak

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Abstract

Forests at risk to diseases caused by invasive *Phytophthora* pathogens can be grouped into two broad classes: those already invaded by the focal pathogen where disease has emerged or those at significant risk of invasion and subsequent emergence of disease. This dichotomy represents distinct management scenarios – treating after or before disease emerges – with a set of epidemiological, ecological, and practical management characteristics that determine optimal actions and associated costs. Here we present the initial outcomes from two management experiments aimed at restoring forest structure or protecting against changes to forest structure following invasion of *Phytophthora ramorum,* the cause of sudden oak death (SOD). We conducted a stand-level restoration experiment on Mount Tamalpais (Marin County, CA) at three sites where the disease has killed most overstory tanoak and dramatically increased understory vegetation density and fuels. A separate experiment in an uninvaded, at risk forest was conducted near Lacks Creek, in Humboldt County. This treatment is an attempt to increase forest resiliency to catastrophic loss of tanoak, increased fuels associated with tree mortality, and densification of the understory that are expected to accompany disease in these stands within the coming decade. Restoration experiments employed two types of mastication of understory vegetation and hand-crew thinning with pile burning; resiliency experiments employed hand crews. Treatments were compared to a set of reference conditions representing overly dense stands where intervention is needed and extensively managed stands that serve as management targets. Both restoration and resiliency treatments greatly reduced density of key sporulation supporting hosts with modest-to-minimal effects on stand basal area. Prior land use, especially past harvesting, was a primary factor determining treatment costs and potential disease impacts in treated vs untreated stands. Although both restoration and resiliency management strategies are likely to reduce disease impacts, treatment costs vary substantially and will greatly influence when and where each approach is optimal.

Introduction

Several biological characteristics of forest pathogens can interact to cause destructive and persistent diseases. Pathogen destructiveness is often a function of virulence, here defined in the evolutionary sense: the ability to infect and negatively impact host physiological or reproductive function (Desprez-Loustau et al. 2016). Invasion, infection, or pathogen growth – each a scale-dependent process – can determine pathogen impacts such as loss of growth and mortality. In contrast, persistence can result from a combination of factors including survival as a saprotroph, a broad host range (generalism) combined with tolerance (host amelioration of negative impacts) in some hosts (Best et al. 2014), and/or formation of long-lived survival structures (Smith et al. 2006). Disease destructiveness and persistence may interact in evolutionary and ecologically complex ways such as diseases that are persistent but not destructive, destructive but not persistent, and most consequentially, destructive and persistent disease. The latter

category includes some of the most problematic non-native and invasive forest pathogens: *Phytophthora cinnamomi* Rands (jarrah dieback and many other diseases), *Phytophthora ramorum* Werres, de Cock & Man in't Veld (sudden oak death), and *Cryphonectria parasitica* (Murrill) M.E. Barr (chestnut blight). Destructive and persistent forest diseases – especially those which can rapidly invade host populations – are some of the most difficult to prepare for or control because many of the characteristics which render a pathogen persistent complicate eradication (Mbah and Gilligan 2010, Filipe et al. 2012, Cobb et al. 2013b). Although challenging, these disease characteristics should not be misconstrued as cause for dismissal of timely, active and focused management actions. Many forest resources with economic, cultural, or ecological value may be protected – or future disease impacts significantly reduced – through actions founded on an understanding of the underlying factors that drive disease.

A broad host range in combination with host tolerance to infection can complicate management prescriptions and detection efforts, particularly when a host supports cryptic or innocuous infections on leaves or roots. The large number of individual leaves and roots as well as the distribution of infections in difficult-to-observe locations such as below ground or in the canopy, render perfect detection unrealistic more often than not. However, early detection and removal of inoculum sources in isolated locations can significantly slow disease progression (Hansen et al. 2008, Cobb et al. 2013b, Cunniffe et al. 2016). Further, invasion and disease emergence rates are influenced by stand host composition and densities, suggesting that proactive management aimed at reducing invasion rate and potential disease impacts in high risk areas could be a cost-efficient way to lessen future impacts. Here, we refer to this strategy as "resiliency management". Broadly, this strategy recognizes that many *Phytophthora* pathogens are persistent within sites and aims to alter environmental conditions or host densities/composition in ways that slow pathogen spread and disease emergence. Given that these actions are very likely to result in some alteration of stand structure and composition, informed actions would take into account additional management goals such as fuels reduction, timber growth, or other objectives.

Many forest pathogens are also remarkable for their realized impacts after disease emergence. The previously listed examples of non-native pathogens demonstrate a clear and alarming power to transform species distribution and ecosystems including region-scale host tree decline or loss and a reduction of stand-level carbon sequestration. For example, chestnut blight has caused a continental-scale loss of a previously common overstory tree; this disease transformed forest structure, composition, and eliminated a highly valuable timber species (Paillet 2003). *Phytophthora cinnamomi* has altered species composition and resulted in ecosystem transformation on multiple continents (Shearer et al. 2007, Corcobado et al. 2014). *Phytophthora ramorum* is notable for both direct disease impacts (Meentemeyer et al. 2011) as well as the degree that sudden oak death changes fire intensity, fire related mortality, reduces carbon sequestration and shifts forest structure and composition at the landscape-scale (Metz et al. 2012, 2013, Cobb et al. 2013a). The extent of stands with severe disease impacts indicates the need for development of "restoration management" – treatments to restore ecosystem functions lost due to disease and to break epidemiological feedback that maintains dense, contiguous fuels and restricts recruitment of resilent species such as redwood.

Our goal here is to describe, compare, and evaluate resiliency and restoration management actions using sudden oak death/*Phytophthora ramorum* as our focal disease. Sudden oak death has been an exceptionally destructive invasive disease in California and Oregon coastal forests where it kills trees at landscape and regional scales in multiple forest types (Meentemeyer et al. 2008, Cobb et al. 2012b, Metz et al. 2012). Tanoak (*Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. Oh) and several coastal red oak species (primarily coast live oak, *Quercus agrifolia* Née) can be killed in as little as two years following bole infection (Cobb et al. 2012b). Mortality rates accelerate with increasing tree size, which compounds disease impacts including fuels accumulation and loss of living biomass (Cobb et al. 2012a, 2012b). Sudden oak death is likely to impact an order of magnitude more individual trees and acres of forest land in CA and OR in the coming decades. These potential impacts stem from high levels

of host biomass and density as well as a conducive environment for the pathogen in regions where there is currently only limited invasion (Lamsal et al. 2011, Meentemeyer et al. 2011). Furthermore, a collection of case studies and modeling studies illustrates the difficulty and likely impossibility of eradication at regional and landscape scales (Hansen et al. 2008, Filipe et al. 2012, Valachovic et al. 2013a, Cunniffe et al. 2016). *Phytophthora ramorum* is a management challenge in both impacted and at risk stands as these conditions are abundant in CA and OR. Furthermore, *P. ramorum*'s rapid invasion rate, substantial ecological impacts, and broad host range place it squarely within the category of persistent and destructive forest diseases for which it is most important to understand and devise responses.

Forest disease management does not occur in a vacuum and managers are often confronted with concurrent issues and concerns including wildfire, insect outbreak, and soil erosion. Historical logging and past disturbances also alter forest structure, resources, and disturbance dynamics (Hirt 1996, Colombaroli and Gavin 2010, Trumbore et al. 2015) often creating a spate of management challenges related to forest health and timber production. Further, the need for forest management investment is occurring in the context of inadequate agency budgets to address these challenges (MacCleery 2008) meaning that management will be most effective it achieves multiple aims. What are the management goals in the absence of disease? In California and similar ecological-sociological environments, where management priorities are often directed at reducing potential wildfire impacts, maintaining water quality and quantity, protecting or enhancing biodiversity, and improving access to public lands, these issues must be balanced against or articulated with, actions aimed at disease.

Historically harvest has occurred extensively throughout the distribution of redwood forests as well as much of the Klamath mountains (Hirt 1996, Lorimer et al. 2009). Rapid and prolific tanoak resprouting in response to harvesting can lead to long-term shifts in stand structure, including shifting stands from dominance by high economic value conifers to tanoak (Harrington and Tappeiner 2009). Extensive harvesting in the last century occurred in the context of broad-scale fire suppression contributing to increased fire intensity due to increased forest density and fuels (Colombaroli and Gavin 2010). All of these land use and land management changes favor dominance of tanoak, particularly in northern coastal California where the greatest density and biomass of tanoak occur (Lamsal et al. 2011). Tanoak is a relatively low-timber value species (Harrington and Tappeiner 2009). Increased tanoak and other hardwood species dominance along with land management policy changes and depletion of high value old-growth timber stocks has contributed to mill closures and a decline in the importance of timber management in many areas (Hirt 1996, MacCleery 2008, Bowcutt 2011). Changes in demand and policy, as well as technological innovations that increase hardwood timber value could increase future timber management within the region.

The emergence of sudden oak death in forests with substantial tanoak results in changes in forest structure that pose multiple resource problems. *Phytophthora ramorum* kills tanoak by infecting the bole and eventually disrupting vascular function (Parke et al. 2007). These infections kill the above ground portion of trees, but basal sprouting from these individuals can be so extensive as to create a new forest structure where understory tanoak densities are very high with interspersed overstory trees, such as redwood (*Sequoia sempervirens* (D. Don) Endl.) and Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco), that do not suffer mortality following infection (Cobb et al. 2012b, Metz et al. 2012). In stands dominated by tanoak, infection can result in substantial amounts of dead fuels in the canopy and on the soil surface (Valachovic et al. 2011, Cobb et al. 2012a). An increase in canopy fuels is positively associated with greater fire intensity (Metz et al. 2011) while increased fuels at the soil surface can decrease tree survival, soil nutrients, and soil carbon during wildfire (Metz et al. 2013, Cobb et al. 2016). The persistence of *P. ramorum* at the forest level is maintained either in California bay laurel (*Umbellularia californica* (Hook. & Arn.) Nutt.) or in resprouting tanoak which facilitates rapid pathogen population reestablishment after events that are unfavorable to the pathogen such as fire or drought (Beh et al. 2012, Eyre et al. 2013). These disease-caused changes in forest structure also result in reduced rates of nitrogen transfer to the

forest floor, reduced carbon storage, and increased carbon efflux from some soil carbon pools (Cobb et al. 2013a, Cobb and Rizzo 2016). High densities of understory tanoak inhibit establishment of Douglas-fir (*Pseudotsuga menziesii*) (Harrington and Tappeiner 2009) suggesting the cycle of tanoak mortality and dense basal sprouting could limit conifer reestablishment in the overstory. These factors suggest intervention is needed to restore fire safe conditions as well as ecosystem functions compromised by disease.

Both pre- and post-*P. ramorum* invasion tanoak forests are problematic in California and Oregon. Here we contrast the pre- and post-treatment conditions for restoration- and resiliency-focused management actions aimed at sudden oak death. The effects of treatments on stand structure were compared to a set of reference conditions that span best- and worst-case scenarios for management. We then forecast expected treatment benefits using a field parameterized epidemiological model and discuss these expected disease benefits in light of treatment effects on fuels, above ground carbon, and forest structure.

Study sites and methods

Restoration management

We conducted a full-factorial replicated restoration management experiment on lands managed by the Marin Municipal Water District (MMWD), a public utility established in 1912. The MMWD was one of the first wildland areas to be invaded by *P. ramorum* (circa 1995) and is valuable for understanding the longer-term consequences of sudden oak death in Douglas-fir-tanoak and redwood forests, two of the most commonly impacted coast range forest types. The water district lands are centered on Mount Tamalpais, a prominent peak north of San Francisco, which provides drinking water for approximately 186,000 people in Marin County and is a recreation resource for millions of people in the greater San Francisco Bay Area. The water district manages approximately 8755 ha (21,635 ac) of land, several reservoirs, and water treatment and conveyance infrastructure. Wildlands managed by the MMWD include grassland, chaparral, and forested ecosystems including redwood, Douglas fir-tanoak, and a variety of mixed-evergreen hardwood forests, many of which include a significant California bay laurel component. Here we focus on forest communities that include tanoak, as these are the most severely disease-transformed forests in the region. *Phytophthora ramorum* challenges two central responsibilities of the MMWD: to provide: 1) high quality and reliable drinking water and 2) fire prevention in the form of fuels management.

Figure 1. *Phytophthora* restoration management on Mount Tamalpais (Marin County, CA). (A) Dense resprouting of tanoak has developed over the ~20 years since disease emergence. Resprouting tanoak maintains the pathogen and perpetuates a low statured stand. (B) Thinning and pile-burning and mastication were applied to intervene in this cycle. (C) Treated (foreground) and untreated conditions. Panel B photo credit E. Gunnison.

We conducted restoration experiments in redwood-tanoak and Douglas fir-tanoak forests impacted by *P. ramorum* (Figure 1). Heavily disease-impacted areas were identified with at least 2 ha (~5 ac) of suitable forest for restoration. Criteria included slopes flat enough to use heavy machinery, extensive tanoak mortality, and no risk of impacting species of concern. This resulted in treatments at three locations: Bolinas Ridge, a 6 ha (~15 ac), heavily impacted redwood forest; Laurel Dell, a 4 ha, heavily impacted Douglas fir-tanoak forest; and San Geronimo Ridge, a 2 ha, heavily impacted Douglas fir-tanoak forest. This design provided ~6 ha of treated area in each forest type and also met our criteria of applying uniform treatment in 0.405 ha square units (1 ac). Within each site, five non-overlapping 0.405 ha blocks were identified and randomly assigned to one of several treatments. In redwood forests, we applied two types of mechanical understory mastication with or without follow-up removal of resprouting by tanoak and other species. Mastication was conducted with an excavator with a masticating head or a skid-steer with a masticating-head forestry attachment. Mastication was applied in two ways: solely with heavy machinery or in conjunction with hand-crews who concentrated materials in a centralized location for later mastication by a skid-steer. Half of the treated plots were then randomly assigned for hand-crew stump sprout removal one year later (results not shown). Within each treatment block, one plot was left as an untreated reference. In both types of mastication, trees up to 5cm dbh were masticated into 3-8 cm length fragments. Mastication also typically pushed over dead standing trees up to ~20 cm dbh and ground them in place; sound dead standing trees were generally left in place. In the Douglas-fir-tanoak sites, treatments consisted of mastication with a skid-steer or traditional hand-crew piling with or without pile burning. Mastication treatments were analogous in redwood forests, while hand-crew work allowed cutting of trees up to 25 cm dbh. In this study, we examine only the three treatments with the greatest potential to result in initial differences in stand density and basal area: 1) mastication with heavy machinery only (mast), 2) an alternative mastication treatment that employed machinery and hand crews (alt mast), and 3) hand crew-only treatments which placed cut materials in piles (pile).

Figure 2. *Phytophthora* resiliency management. (A) Inadequate silvicultural investments can lead to high fuels and dense host conditions (Friday Ridge, Humboldt Co., CA). (B) Stand treatments to reduce host density, fuels, and increase the prevalence of non-host species is a common approach to proactive management (Lacks Creek, CA). (C) Deliberate hardwood silviculture is highly valuable for demonstrating desired stand conditions for many agencies such as lower fuels as well as fewer large tanoak and conifers. Panel C shows an example from the Yurok Indian Reservation (Humboldt, Co.) where a legacy of intentional cutting and repeated use of fire has created resilient conditions at the stand level.

Resiliency management

A single forest-level experiment was applied on the Lacks Creek Management Unit, a 1,820 ha multi-use landholding (~4,500 ac) near the community of Redwood Valley (Humboldt County, CA) managed by the Bureau of Land Management (BLM), where cutting of redwood and Douglas-fir has occurred across

most of the area. Although management could favor timber growth and harvest, on this property most management actions focus on habitat conservation/restoration, enhancing public access, and fuels management (Raoush 2010). In 2010, *P. ramorum* was identified as the cause of tanoak mortality in the nearby rural community of Redwood Valley, which triggered an attempt to eradicate a geographically isolated outbreak (Valachovic et al. 2013b). Similar to several other attempts to eradicate *P. ramorum* from wildlands, eradication treatments greatly reduced pathogen populations and appeared to slow pathogen spread, but did not achieve eradication, and the pathogen remains poised to invade the abundant surrounding tanoak populations. Past conifer harvest in the Lacks Creek area increased tanoak densities in parts of the study area (Figure 2) creating a need to protect stands against the substantial impacts observed at sites similar to our MMWD study area.

The BLM identified a continuous ~60 ha area with a relatively high density of tanoak and located close to known wildland infections (within ~200 m). This area had also been identified as potentially benefiting from thinning treatments to restructure species composition and reduce fuels. Species composition is variable across the study site with pockets of relatively dense tanoak or Douglas-fir. Scattered California bay laurel was also located throughout the treatment area, primarily in drainages. Treatments were conducted solely with hand crews with a tree removal limit of ~40cm dbh. Tree removals were restricted to significant *P. ramorum* hosts (tanoak and California bay laurel) but in practice removed trees were almost exclusively tanoak. By design this created thinning levels which are highly variable across the treatment area and primarily dependent on pre-treatment tanoak density. Removed tanoak were piled for later burning; thinning levels aimed to retain a closed overstory canopy to limit post-treatment resprouting. No herbicides – for example to reduce stump sprouting – were applied during these treatments.

We employed two different reference conditions to compare and evaluate treatment effects. Two untreated stands adjacent to the Lacks Creek treatment were identified and measured to provide long-term experimental controls. However, the high degree of variation in tanoak density in the broader landscape and the need to identify restoration targets led us to establish a second set of reference measurements in stands that reflected these conditions. To compare stand treatments vs potential long-term management targets, we measured plots (N = 10) in actively managed tanoak stands on the nearby Hoopa and Yurok reservations where tanoak thinning and burning treatments have been applied over the last several decades. An additional set of reference measurements were established in plots in high-density tanoak stands on Friday Ridge (Willow Creek, CA; N = 8), a previously clear-cut site which reflects the frequent high-density tanoak conditions that develop following Douglas-fir harvesting (Figure 2).

Field measurements

A common set of measurements was conducted at all of the sites. Study plots were randomly located within sites with a minimum distance of 100-200 m between plots. Each plot was circular and 500 m^2, within which each tree was mapped and every stem $\geq$ 1cm dbh measured for diameter, identified to species, and given a health rating. Symptoms of *P. ramorum* infection were recorded when present and occasionally returned to the laboratory for isolation on a *Phytophthora* selective media (see Davidson et al. 2005) to confirm if infection had occurred (primarily in the uninvaded resiliency study sites). Plots were established and measured prior to treatment application; a follow-up survey occurred for each experiment within 6 months post treatment to quantify changes in forest structure and host composition. Each plot was also assessed for pre- and post-treatment fuel levels, coarse woody debris, and soil carbon and nutrient pools (data not shown). The restoration treatments (Mount Tamalpais) included 30 individual 0.405 ha blocks and six control plots (one per five-plot block). The resiliency study included 10 study plots in the treated area, two reference plots in adjacent stands, eight reference study plots at the high tanoak density Friday Ridge reference site, and an additional 10 reference study plots in actively managed stands on Hoopa and Yurok lands (30 total). The resiliency experiment study plots were overlaid with a variable radius plot (basal area factor 5) to facilitate comparisons to the Friday Ridge study site where

study plots were 201 m^2 and Hoopa and Yurok plots where only variable radius plots were conducted at five locations within a 2 ha study plot.

Model application

We applied the SODDr (Sudden Oak Death Dynamics in R) model to estimate potential treatment benefits following restoration and resiliency experiments. The SODDr model is a dynamic, multi-species, size-structured, and spatially explicit, susceptible-infective (SI) host-pathogen model of SOD dynamics, parameterized based on a 6-year dataset of SOD spread (infection and mortality) in redwood forests (see Cobb et al. 2012b, 2013b). The current iteration of the model is implemented in R and available at https://github.com/noamross/SODDr.

We calculated median stand densities for each treatment type for both restoration and resiliency treatments and parameterized the SODDr model with the respective proportions of tanoak, California bay laurel, and other species – primarily redwood and Douglas-fir – that have a minimal epidemiological role, so call 'non-competent' species. Proportional species composition was calculated using the species densities within each treatment, rather than average density across all treatments, since the highly disparate tanoak densities across treatments would likely bias model results towards slow pathogen spread and limited or no disease emergence in treated stands. This could bias the model results toward excessively rapid pathogen spread. Therefore, we restrict our model interpretations to relative differences in disease dynamics between treated and untreated conditions. Although SODDr is a size-specific model, we employ a single uniform size class for the purposes of this study to facilitate comparisons across sites.

Results

Restoration treatments

Each restoration treatment type resulted in a dramatic reduction of stem density (Figure 3). Although there were clear differences in pre-treatment stand densities among treatment types – particularly the hand-crew treatment area had lower initial stem densities (Figure 3 A&B; "pre-pile") – each treatment type resulted in post-treatment stem densities which did not statistically differ. In contrast, stand basal area was not significantly different between pre- and post-treatment measurements (Figure 3) and reflects the relatively low contribution of tanoak resprouts to total stand basal area. This was true for all treatment types and is demonstrated in Figure 3 which contrasts basal area and stem densities for standard mastication ("mast"), mastication with joint machinery and hand crews ("alt mast"), or hand crew treatments ("pile"). Specifically, mastication, alt mastication, and pile treatments reduced density by 96.3, 83.1, and 90.7%, respectively. In contrast, basal area was reduced by 7.9, 14.1, and 3.7% for the same treatments. Treatments dramatically reduced the stem density of tanoak while having little effect on density of other species, which reflects the dominance of tanoak in small size classes that developed in the 20 years of disease progression at Mount Tamalpais.

Model projections suggest dramatic increases in tanoak survival following restoration treatments (Figure 4). Differences between treatment types are difficult to discern in Figure 4 because post-treatment tanoak densities were similar (and very low). Forecasted disease dynamics for pre-treatment stand conditions (untreated stands), showed more variation than treated stands and reflects variation in tanoak densities prior to treatment in our study sites. However, rapid and extensive mortality was common among untreated stand conditions in the model which agrees with observations from the field plots that *P. ramorum* transforms stand structure to a dense understory with scattered less susceptible trees such as redwood or Douglas-fir in the overstory.

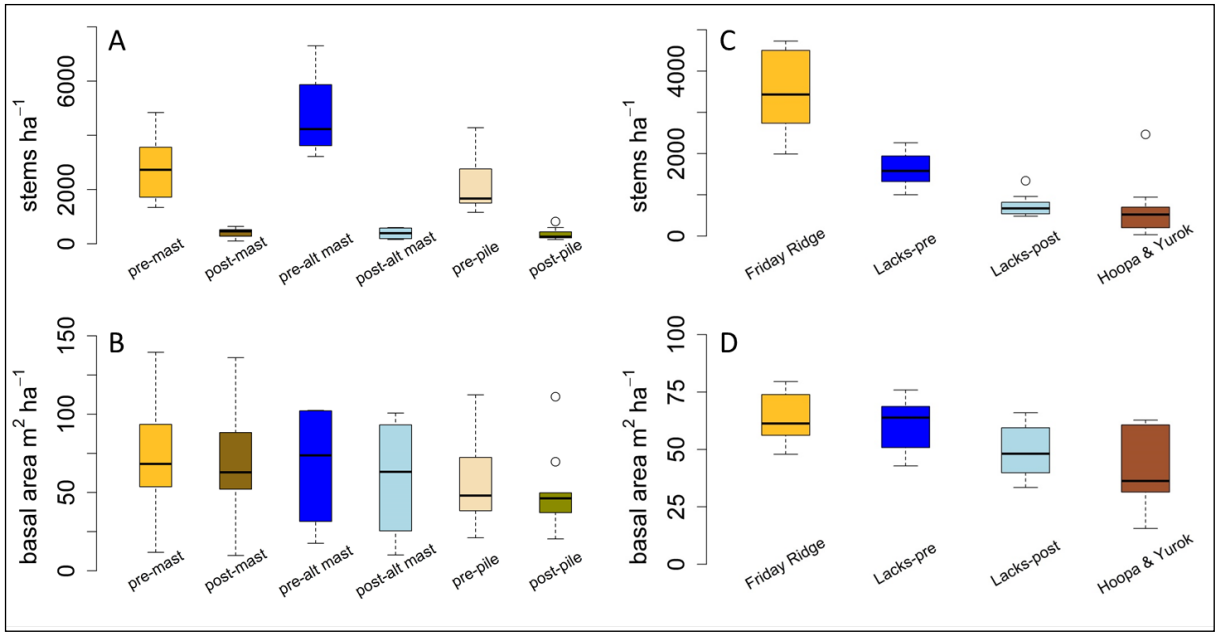

Figure 3. Comparison of *Phytophthora* restoration and resiliency treatment effects. Restoration of disease impacted forests on Mount. Tamalpais (Marin Co., CA) are shown with pre- and post- treatment changes in stem densities (A) and basal area (B). Resiliency treatments were conducted at the Lacks Creek field site ("Lacks") with the untreated Friday Ridge and treated Hoopa and Yurok sites used as reference comparisons (see text); resiliency treatment stand densities (C) and basal area (D) are presented for comparison with restoration treatment effects.

Resiliency treatments

At Lacks Creek, resiliency treatments reduced tanoak densities relative to pre-treatment conditions but not as dramatically compared to the restoration experiment (Figure 3C). Specifically, the pile-treatments at Lacks Creek resulted in a 42.4% decrease in stand density. However, basal area was reduced 24.6% in the pile treatments (Figure 3D). The differences in treatment effects reflect differences pre-treatment distribution of size between the forest conditions, disease-impacted stands have high densities of trees under 5cm dbh and hence density can be dramatically reduced with little effect on basal area. Further, in the forest conditions of the resiliency treatment sties much of the tanoak component is present in the overstory, including in the high-density Friday Ridge study site. Given that these treatments could remove tanoak up to ~40 cm dbh, some loss of basal area is unsurprising. Basal area was similar but density differed by an order of magnitude between the post-clear cut Friday Ridge site and the tribal plots where a range of fuels, prescribed burning, and other tanoak-focused silviculture has been applied. Tribal plots have been managed for specific tanoak resources, often provisioned by large trees; thus the differences in structure (Figure 3) reflect the dominance of smaller diameter overstory trees at Friday Ridge (< 40cm dbh) and larger tanoak (>50cm dbh) trees on many of the tribal plots. Lacks Creek post treatment densities and basal area were 19.5 and 75.4% of the same stand parameters at the post-clear cut Friday Ridge study site but were 128.3 and 132.6% compared to the Hoopa/Yurok study plots. Multiple stand treatments have been necessary to create the structure found on our Hoopa/Yurok study plots and a long-term commitment to management at Lack Creek or in forest conditions similar to our Friday Ridge site will be needed to create a similar structure. In addition to changes in density and basal area, the Lacks Creek experimental treatment removed all bay laurel individuals which should have a substantial benefit in terms of pathogen invasion rate.

Model projections suggest a clear benefit of thinning treatments in terms of reduced mortality rates in addition to a delay of tanoak demographic decline (Figure 4). However, the model results show tanoak populations were still in decline in the resiliency scenario at the end of the 100-year simulation suggesting these treatments will slow rather than arrest disease progression over the longer term. A comparison of the reference sites suggests a tanoak density threshold in the model where feedback between tanoak populations and the *P. ramorum* pathogen results in a stable tanoak population. It must be noted that the model is not direct evidence that these thresholds exist in nature and the reference sites have not yet been invaded by *P. ramorum*. Unlike the MMWD restoration experiments, these model results cannot yet be validated from field data, as long-term measurements of post-treatment dynamics are not yet available.

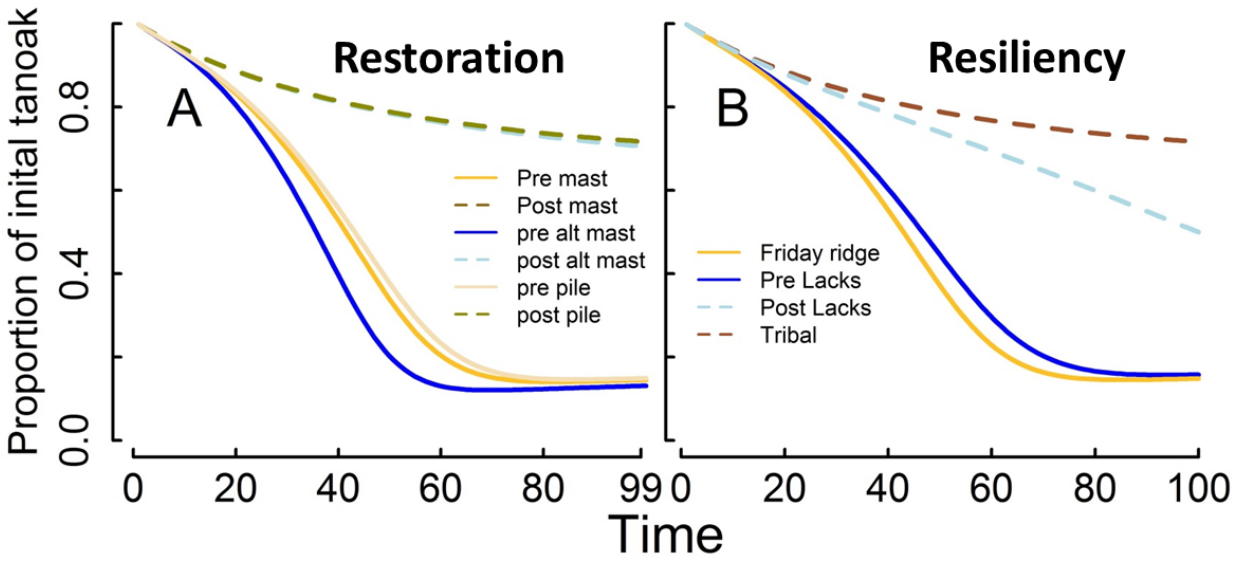

Figure 4. Effects of management actions on disease progression estimated from an epidemiological model (SODDr). For both (A) restoration and (B) resiliency treatments, pretreatment or untreated stands are shown with solid lines while treated stands are shown with dashed lines. For the post mastication treatment simulation ("Post mast" brown dashed line), results were virtually identical to the other treatments and are obscured by the other lines. In resiliency treatments the Friday ridge study site and plots on Hoopa and Yurok, "Tribal" lands are included as high tanoak density and low tanoak density silvicultural outcomes. In these simulations, a single uniform infection and mortality rate were applied to the tanoak population.

Discussion

With over a billion individual tanoak stems at risk from *P. ramorum* in California and Oregon (Lamsal et al. 2011), the scale of potential impacts from sudden oak death is daunting however, only a fraction of at-risk trees and stands have been impacted. This set of conditions creates a set of pressing management problems for Coast Range forests for the next several decades: what levels of protection can be applied to uninvaded stands while resources are also being directed towards restoring degraded disease-impacted stands? Current spread models suggest that rates of invasion will peak in the next 10-20 years (Meentemeyer et al. 2011, Cunniffe et al. 2016); given that stand-level inoculum buildup and mortality are thought to occur from several years to a decade after initial invasion, the ecological impacts of the disease will become realized over the next half-century (Cobb et al. 2012b, Filipe et al. 2012). Although invasion and disease emergence are both rapid and extensive, these model estimates suggest adequate time is available to design, test, and coordinate deployment of the most effective treatments. Currently,

most stand experiments have focused on local and regional eradication efforts (Hansen et al. 2008, Valachovic et al. 2013b; Valachovic *this volume*), this study seeks to expand management experiments into a broader range of stand conditions and objectives. Our study also represents one of the first attempts to design a sudden oak death restoration treatment, specifically in stands where the disease has been present for up to two decades. These stand conditions are frequent in the San Francisco Bay Area, Big Sur, southern Humboldt County, and parts of Curry County, Oregon, suggesting the need to expand and improve restoration approaches will increase in the coming years.

Implication for forest carbon sequestration

Restoration treatments at MMWD resulted in a dramatic change in stand structure (Figure 1; Figure 3). However, for carbon sequestration goals, since basal area is conserved, treatments had a nominal effect on above ground carbon storage. The reduction of density without biomass loss also suggests these treatments will increase water outflow by decreasing transpiration at the stand-level. Treatments are likely to cause a short-term decrease in fine carbon production (leaf litter and fine root production), but this loss to ecosystem carbon input is likely to be overcome by longer-term increases in overstory tree growth and productivity (Cobb et al. 2010, 2013a). An additional immediate treatment benefit is suggested by the model results which forecast increased survivorship of scattered overstory tanoak which, despite extensive disease, still occur throughout the area (Figure 1 & 4). However, increased survival of small-diameter tanoak would be detrimental to our longer-term management goals because these individuals are likely to be reinfected and contribute to the perpetuated dominance of understory tanoak stems. In fact, treatment maintenance in the form of repeated removal of resprouts from stumps will be necessary to avoid the return of undesirable high-density understory tanoak with high infection levels. Where tanoak densities can be maintained, increased survival of overstory tanoak individuals could be important for maintenance of ectomycorrhizal fungi in these stands as well as acorn production. Tanoak is the sole acorn-producing species or ectomycorrhizal host in the redwood study sites, meaning that its complete loss would reduce biodiversity as well as remove this source of below ground carbon input. Helpfully, none of the restoration treatments produced significant differences in post- treatment tanoak basal area indicating that these treatments do not accelerate loss of overstory tanoak. Our restoration treatment experiment suggests any of the specific techniques (mechanical, hand-crew) are effective and the decision of specific treatment type can be optimized for logistical and cost constraints.

Resilience treatments also altered stand structure with greatly reduced tanoak densities and decreased disease impacts within the model (Figure 3; Figure 4). Hand-crew pile treatments at Lacks Creek are distinguished from the restoration treatments at MMWD (Mount Tamalpais) by the larger average size of removed tanoaks at Lacks Creek. In addition to having an immediate greater impact on stand carbon, larger diameters create two challenges: fewer individual trees can be removed, and considerably more dead material is generated by the treatment (Figure 2). The scale of treating high density post-harvest tanoak stands is also apparent when contrasting stand conditions between the Friday Ridge site and the Hoopa/Yurok sites. The high densities of overstory trees at Friday Ridge cannot be easily converted to the more park-like, low density, large average tree size conditions of the tribal study plots. Efforts by Hoopa and Yurok forestry professionals and tribal members have transformed or maintained these stand conditions over tens to hundreds of years (Waterman 1920, Bowcutt 2013). But the lesson of these plots is clear: a long-term commitment to management can have a significant benefit to multiple forest management goals. Considering sudden oak death more narrowly, the potential benefits of slowed pathogen spread and reduced disease emergence through reduced tanoak densities is also strongly suggested by the SODDr model results (Figure 4). The model results are especially striking when comparing potential disease impacts between the Friday Ridge stand and the Hoopa/Yurok sites. We again emphasize that both stands are reference conditions with the former reflecting degraded stands and the latter reflecting a potential hardwood management goal. Although transforming problematic stand conditions of post-clear cut sites including Friday Ridge will almost certainly require multiple treatments and a long-term commitment, this effort has the additional benefit of addressing multiple resource

problems simultaneously (e.g., increasing timber growth and carbon sequestration rates, reducing crown fuel continuity, and increasing stand diversity). Initial treatments are complicated by multiple factors, but removal of cut tanoak material is especially challenging. This material has a very low market price dictated by biomass fuel and firewood prices. Lacking incentive for material removal and cost recuperation will increase the difficulty of treating these stands and must be overcome before resiliency treatments can be applied more broadly. However, the epidemiological model results suggest that these treatments can greatly reduce potential disease-related mortality which would increase stand-level carbon sequestration.

Treatment financial costs

Treatment cost differences between restoration and resiliency treatments in our study were substantial and represent a serious caution to decisions about when and where to apply treatments. Restoration treatments in the most difficult stand conditions (high density and steep slopes) reached up to $24,710 per ha ($10,000 ac). Total costs will increase over time depending on the intensity of follow-up treatments. In contrast, the hand-crew thinning employed for resiliency treatments cost an average of $2,471per ha ($1,000 ac). Resiliency treatments initially cost less due to considerably lower pre-treatment stand densities. In some locations tanoak was a relatively minor component compared to Douglas-fir; this facilitated treatment completion even though larger diameter trees were cut into ~1m lengths and piled. Our study suggests proactive treatments may be substantially less expensive to apply. As previously noted, sudden oak death impacts are forecast to be realized over the course of the next several decades, meaning that a proactive campaign to reduce future impacts could yield longer term cost savings while also addressing the non-disease issues of overly-dense stands and fuels accumulation. In both resiliency and restoration treatments, reduced total and understory stand density is likely to result in reduced fire risk. This suggests that fuels reduction programs hold promise to reduce disease risk, particularly when they can be focused strategically to include stands with a high capacity for pathogen spread (Cobb et al. 2013b, Cunniffe et al. 2016).

Conclusions

Restoration and resiliency treatments have received less critical study compared to eradication and quarantine efforts for many forest *Phytophthora* species. Restoration and resiliency treatments appear to be useful techniques for reducing or avoiding the problematic stand conditions generated by *P. ramorum* and sudden oak death in California. This study reports the effects of disease treatments for both management scenarios in terms of changes in stand composition and structure. Treatments greatly reduced tanoak density without substantial changes in basal area, effects that reflect the extensive removal of small stems. Proof of treatment efficacy on disease dynamics is not available at the early stage of these experiments, but estimates from a dynamic epidemiological model suggest pathogen spread and subsequent mortality rates will be greatly reduced by stand treatments. These expectations need evaluation with empirical data collected through further monitoring to understand the overall efficacy of the respective treatment strategies. Both treatments hold potential to also address co-occurring management challenges including fuels accumulation, loss of biodiversity, and carbon storage. In California, rates and patterns of *P. ramorum* invasion suggest a window of several decades is available further optimize the cost-efficiency of these treatments and to apply them at a broader scale. Given the potential scale and extent of disease impacts, as well as the important ecosystem attributes represented by tanoak, a greater emphasis on development and application of these techniques is likely to yield considerable benefits to biodiversity conservation, forest carbon storage, and creation of fire-safe forests through a reduction of overall disease impacts.

Acknowledgements

We thank the Six Rivers National Forest, Yurok Tribe, and Hoopa Tribe for permission to conduct these experiments or measurements on their lands. California Department of Forestry and Fire Protection, California Department of Corrections, California State Parks, Institute for Sustainable Forestry, and Able Forestry were all very helpful in implementing these management experiments. Funding was provided by USDA Forest Service, Forest Health Protection; USDA Forest Service, Pacific Southwest Research Station; American Recovery and Reinvestment Act; and CAL FIRE, Green House Gas Reduction Fund. We are grateful for comments from two anonymous reviewers that improved a previous version of this paper.

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