New Phytologist

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

Richard C. Cobb[1], Valerie T. Eviner[2] and David M. Rizzo[1]

[1]Department of Plant Pathology, University of California, One Shields Ave, Davis, CA 95616, USA; [2]Department of Plant Sciences, University of California, One Shields Ave, Davis, CA 95616, USA

Author for correspondence:
Richard C. Cobb
Tel: +1 530 754 9894
Email: rccobb@ucdavis.edu

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

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

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

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

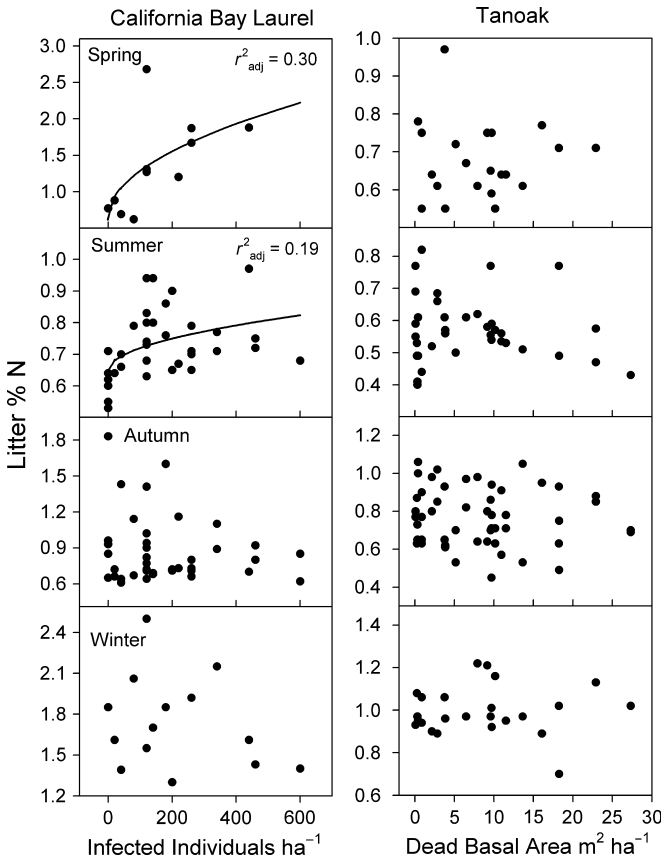

Fig. 1 Bay laurel (*Umbellularia californica*) and tanoak (*Notholithocarpus densiflorus*) litterfall %N vs prevalence of *Phytophthora ramorum* or cumulative dead tanoak basal area. When the relationship between infected hosts or mortality and litter nitrogen (N) concentration was significant ($P \leq 0.05$), 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

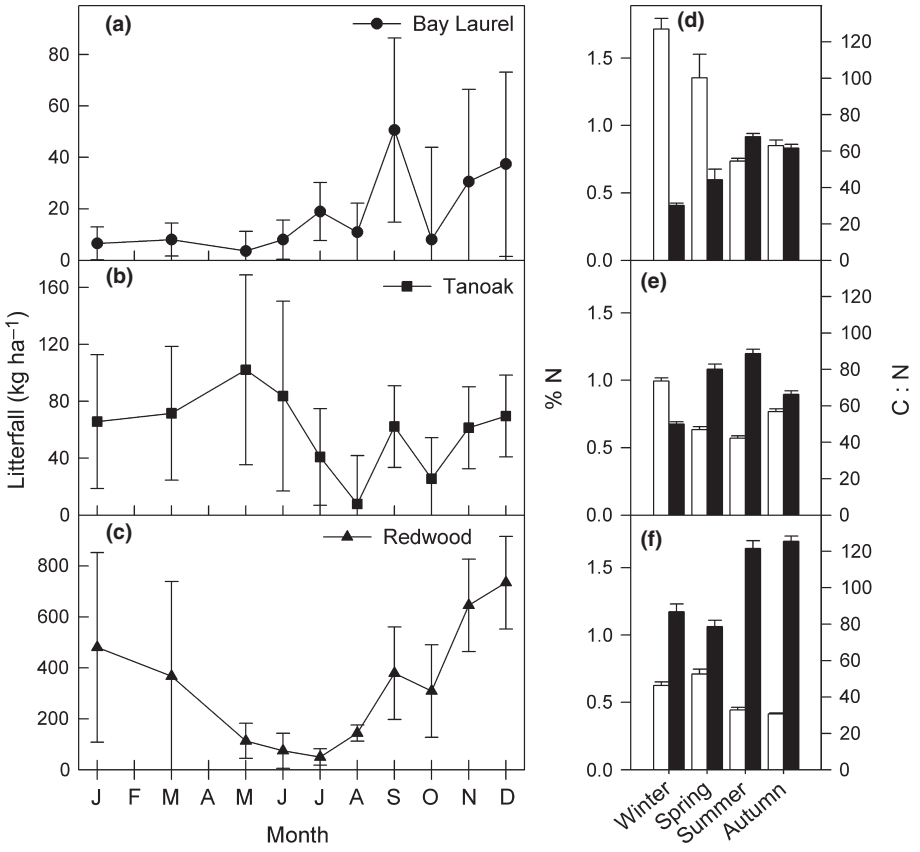

Fig. 2 Monthly litterfall mass (a–c) with seasonal values of carbon and nitrogen (d–f; open bars, %N; closed bars, C : N) for bay laurel (*Umbellularia californica*; top), tanoak (*Notholithocarpus densiflorus*; middle), and redwood (*Sequoia sempervirens*; bottom) from two redwood forests impacted by 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

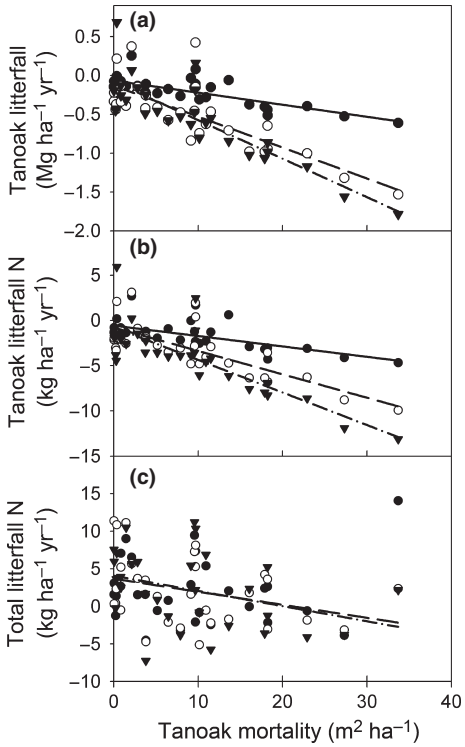

Fig. 3 Effects of sudden oak death-caused mortality (*x*-axis) on annual tanoak (*Notholithocarpus densiflorus*) litterfall mass (a), tanoak litterfall nitrogen (N; b), and total stand-level litterfall N (c). Closed circles with solid lines, 2007; open circles with dashed lines, 2008; triangles with 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.

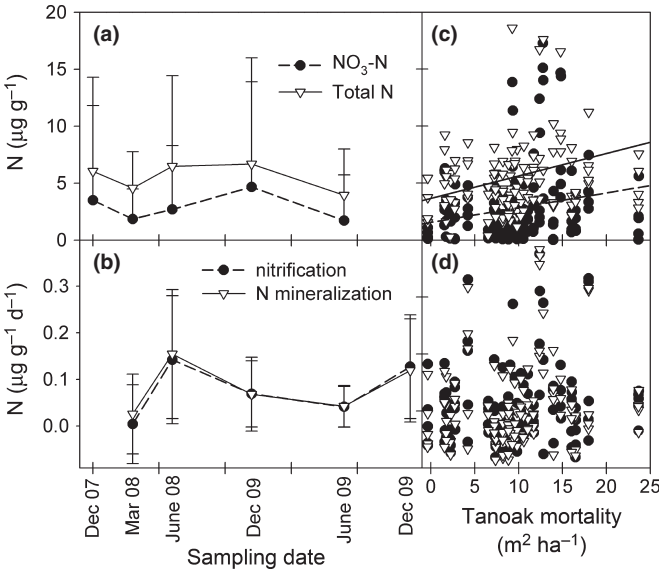

Fig. 4 Seasonal patterns and effects of sudden oak death on soil nitrogen (N). Seasonal patterns of inorganic N pool sizes (a) and rates of N mineralization and nitrification (b) are shown with sampling date on the *x*-axis. (c, d) Leverage plots from mixed linear models showing the effect of tanoak (*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

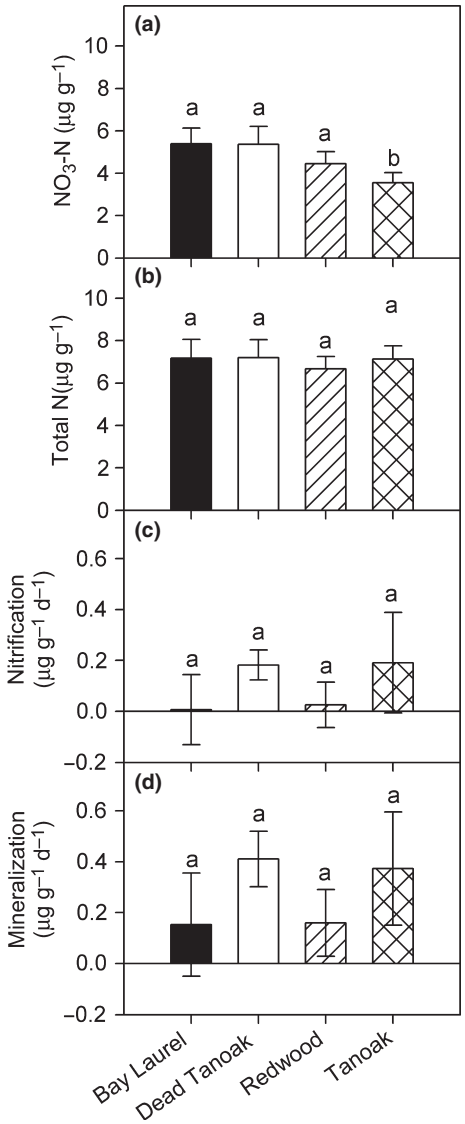

Fig. 5 Species-level effects on nitrogen (N) availability (extractable pools a, b) and cycling rates (c, d). Data are from incubation of soils collected immediately below the three focal species and tanoak (*Notholithocarpus densiflorus*) killed by *Phytophthora ramorum* (dead tanoak). Data are means ± 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.

References

Adams HD, Guardiola-Claramonte M, Barron-Gafford GA, Villegas JC, Breshears DD, Zou CB, Troch PA, Huxman TE. 2009. Temperature sensitivity of drought-induced tree mortality portends increased regional die-off under global change-type drought. *Proceedings of the National Academy of Sciences, USA* **106**: 7063–7066.

Balci Y, Balci S, Eggers J, MacDonald WL, Gottschalk KW, Juzwik J, Long R. 2007. *Phytophthora* spp. associated with forest soils in eastern and north-central U.S. oak ecosystems. *Plant Disease* **91**: 705–710.

Burdon JJ, Thrall PH, Ericson L. 2006. The current and future dynamics of disease in plant communities. *Annual Review of Phytopathology* **44**: 19–39.

Chapman SK, Whitham TG, Powell M. 2006. Herbivory differentially alters plant litter dynamics of evergreen and deciduous trees. *Oikos* **114**: 566–574.

Classen AT, Hart SC, Whitman TG, Cobb NS, Koch GW. 2005. Insect infestations linked to shifts in microclimate. *Soil Science Society of America Journal* **69**: 2049–2057.

Cobb RC. 2010. Species shift drives decomposition rates following invasion by hemlock woolly adelgid. *Oikos* **119**: 1291–1298.

Cobb RC, Chan MN, Meentemeyer RK, Rizzo DM. 2012a. Common factors drive disease and coarse woody debris dynamics in forests impacted by sudden oak death. *Ecosystems* **15**: 242–255.

Cobb RC, Filipe JAN, Meentemeyer RK, Gilligan CA, Rizzo DM. 2012b. Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests. *Journal of Ecology* **100**: 712–722.

Cobb RC, Meentemeyer RK, Rizzo DM. 2010. Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility. *Ecology* **91**: 327–333.

Davidson JM, Patterson HA, Rizzo DM. 2008. Sources of inoculum for *Phytophthora ramorum* in a redwood forest. *Phytopathology* **98**: 860–866.

Davidson JM, Patterson HA, Wickland AC, Fichtner EJ, Rizzo DM. 2011. Forest type influences transmission of *Phytophthora ramorum* in California oak woodlands. *Phytopathology* **101**: 492–501.

Davidson JM, Wickland AC, Patterson HA, Falk KR, Rizzo DM. 2005. Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. *Phytopathology* **95**: 587–596.

DiLeo MV, Bostock RM, Rizzo DM. 2009. *Phytophthora ramorum* does not cause physiologically significant systemic injury to California bay laurel, its primary reservoir host. *Phytopathology* **99**: 1307–1311.

Ellison AM, Bank MS, Clinton BD, Colburn EA, Elliott K, Ford CR, Foster DR, Kloeppel BD, Knoepp JD, Lovett GM. 2005. Loss of foundation species: consequences for the structure and dynamics of forested ecosystems. *Frontiers in Ecology and the Environment* **3**: 479–486.

Eviner VT, Chapin FS III. 2003. Functional matrix: a conceptual framework for predicting multiple plant effects on ecosystem processes. *Annual Review of Ecology, Evolution, and Systematics* **34**: 455–485.

Eviner VT, Likens GE. 2008. Effects of pathogens on terrestrial ecosystem function. In: Ostfeld RS, Keesing F, Eviner VT, eds. *Infectious disease ecology. Effects of ecosystems on disease and disease on ecosystems*. Princeton, NJ, USA: Princeton University Press, 260–283.

Filipe JAN, Cobb RC, Meentemeyer RK, Lee CA, Valachovic YS, Cook AR, Rizzo DM, Gilligan CA. 2012. Landscape epidemiology and control of pathogens with cryptic and long-distance dispersal: sudden oak death in northern Californian forests. *PLoS Computational Biology* **8**: e1002328.

Finzi AC, Van Breemen N, Canham CD. 1998. Canopy tree-soil interactions within temperate forests: species effects on soil carbon and nitrogen. *Ecological Applications* **8**: 440–446.

Fried JS, Boyle JR, Tappeiner JC II, Cromack K Jr. 1990. Effects of bigleaf maple on soils in Douglas-fir forests. *Canadian Journal of Forest Research* **20**: 259–266.

Gilligan CA, Van den Bosch F. 2008. Epidemiological models for invasion and persistence of pathogens. *Annual Review of Phytopathology* **46**: 385–418.

Gotelli NJ, Ellison AM. 2004. *A primer of ecological statistics*. Sunderland, MA, USA: Sinauer Associates Inc.

Griffin JM, Turner MG. 2012. Changes to the N cycle following bark beetle outbreaks in two contrasting conifer forest types. *Oecologia* **170**: 551–565.

Hawkins AE, Henkel TW. 2011. Native forest pathogens facilitate persistence of Douglas-fir in old-growth forests of northwestern California. *Canadian Journal of Forest Research* **41**: 1256–1266.

Hayden KJ, Nettel A, Dodd RS, Garbelotto M. 2011. Will all the trees fall? Variable resistance to an introduced forest disease in a highly susceptible host. *Forest Ecology and Management* **261**: 1781–1791.

Hicke JA, Allen CD, Desai AR, Dietze MC, Hall RJ, Hogg EH, Kashian DM, Moore D, Raffa KF, Sturrock RN. 2012. Effects of biotic disturbances on forest carbon cycling in the United States and Canada. *Global Change Biology* **18**: 7–34.

Hobara S, Tokuchi N, Ohte N, Koba K, Katsuyama M, Kim SJ, Nakanishi A. 2001. Mechanism of nitrate loss from a forested catchment following a small-scale, natural disturbance. *Canadian Journal of Forest Research* **31**: 1326–1335.

Holt RD, Dobson AP, Begon M, Bowers RG, Schauber EM. 2003. Parasite establishment in host communities. *Ecology Letters* **6**: 837–842.

Hunter MD. 2001. Insect population dynamics meets ecosystem ecology: effects of herbivory on soil nutrient dynamics. *Agricultural and Forest Entomology* **3**: 77–84.

Lamsal S, Cobb RC, Hall Cushman J, Meng Q, Rizzo DM, Meentemeyer RK. 2011. Spatial estimation of the density and carbon content of host populations for *Phytophthora ramorum* in California and Oregon. *Forest Ecology and Management* **262**: 989–998.

Loo JA. 2009. Ecological impacts of non-indigenous invasive fungi as forest pathogens. *Biological Invasions* **11**: 81–96.

Lovett GM, Arthur MA, Weathers KC, Griffin JM. 2010. Long-term changes in forest carbon and nitrogen cycling caused by an introduced pest/pathogen complex. *Ecosystems* **13**: 1188–1200.

Lovett GM, Canham CD, Arthur MA, Weathers KC, Fitzhugh RD. 2006. Forest ecosystem responses to exotic pests and pathogens in eastern North America. *BioScience* **56**: 395–405.

Lovett GM, Christenson LM, Groffman PM, Jones CG, Hart JE, Mitchell MJ. 2002. Insect defoliation and nitrogen cycling in forests. *BioScience* **52**: 335–341.

Maloney PE, Lynch SC, Kane SF, Jensen CE, Rizzo DM. 2005. Establishment of an emerging generalist pathogen in redwood forest communities. *Journal of Ecology* **93**: 899–905.

McDowell NG, Beerling DJ, Breshears DD, Fisher RA, Raffa KF, Stitt M. 2011. The interdependence of mechanisms underlying climate-driven vegetation mortality. *Trends in Ecology & Evolution* **26**: 523–532.

Meentemeyer RK, Cunniffe NJ, Cook AR, Filipe JAN, Hunter RD, Rizzo DM, Gilligan CA. 2011. Epidemiological modeling of invasion in heterogeneous landscapes: spread of sudden oak death in California (1990–2030). *Ecosphere* **2**: art17.

Meentemeyer RK, Rank NE, Shoemaker DA, Oneal CB, Wickland AC, Frangioso KM, Rizzo DM. 2008. Impact of sudden oak death on tree mortality in the Big Sur ecoregion of California. *Biological Invasions* **10**: 1243–1255.

Metz MR, Frangioso KM, Wickland AC, Meentemeyer RK, Rizzo DM. 2012. An emergent disease causes directional changes in forest species composition in coastal California. *Ecosphere* **3**: art86.

Morehouse K, Johns T, Kaye J, Kaye M. 2008. Carbon and nitrogen cycling immediately following bark beetle outbreaks in southwestern ponderosa pine forests. *Forest Ecology and Management* **255**: 2698–2708.

Orwig DA, Cobb RC, D'Amato AW, Kizlinski ML, Foster DR. 2008. Multi-year ecosystem response to hemlock woolly adelgid infestation in southern New England forests. *Canadian Journal of Forest Research* **38**: 834–843.

Orwig DA, Thompson JR, Povak NA, Manner M, Niebyl D, Foster DR. 2012. A foundation tree at the precipice: *Tsuga canadensis* health after the arrival of *Adelges tsugae* in central New England. *Ecosphere* **3**: art10.

Raffa KF, Aukema BH, Bentz BJ, Carroll AL, Hicke JA, Turner MG, Romme WH. 2008. Cross-scale drivers of natural disturbances prone to anthropogenic amplification: the dynamics of bark beetle eruptions. *BioScience* **58**: 501–517.

Ramage BS, O'Hara KL, Forrestel AB. 2011. Forest transformation resulting from an exotic pathogen: regeneration and tanoak mortality in coast redwood stands affected by sudden oak death. *Canadian Journal of Forest Research* **41**: 763–772.

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

Ruess RW, McFarland JM, Trummer LM, Rohrs-Richey JK. 2009. Disease-mediated declines in N-fixation inputs by *Alnus tenuifolia* to early-successional floodplains in interior and south-central Alaska. *Ecosystems* **12**: 489–502.

Russell CA, Kosola KR, Paul EA, Robertson GP. 2004. Nitrogen cycling in poplar stands defoliated by insects. *Biogeochemistry* **68**: 365–381.

Santini A, Ghelardini L, Pace C, Desprez-Loustau ML, Capretti P, Chandelier A, Cech T, Chira D, Diamandis S, Gaitniekis T. 2012. Biogeographical patterns and determinants of invasion by forest pathogens in Europe. *New Phytologist* **197**: 238–250.

Sinclair WA, Lyon HH, Johnson WT. 1987. *Diseases of trees and shrubs*. Ithaca, NY, USA: Cornell University Press.

Valachovic YS, Caldwell BA, Cromack K Jr, Griffiths RP. 2004. Leaf litter chemistry controls on decomposition of Pacific Northwest trees and woody shrubs. *Canadian Journal of Forest Research* **34**: 2131–2147.

Wickland AC, Jensen CE, Rizzo DM. 2008. Geographic distribution, disease symptoms and pathogenicity of *Phytophthora nemorosa* and *Phytophthora pseudosyringae* in California, USA. *Forest Pathology* **38**: 288–298.

Worrall JJ, Marchetti SB, Egeland L, Mask RA, Eager T, Howell B. 2010. Effects and etiology of sudden aspen decline in southwestern Colorado, USA. *Forest Ecology and Management* **260**: 638–648.

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

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting information supplied by the authors. Any queries (other than missing material) should be directed to the *New Phytologist* Central Office.