Title

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

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Summary

1) Few studies have quantified the effects of pathogen caused tree mortality to ecosystem processes despite that generalist pathogen impacts differ across species and ecosystems.

2) We measured litterfall mass, litterfall chemistry, and soil N cycling associated with multiple hosts along a gradient of mortality caused by *Phytophthora ramorum*, the cause of sudden oak death.

3) 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 has moderate litterfall N (0.723%) and transmits infection but also suffers 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.

4) 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.

Key words: Emerging infectious disease, ecosystem ecology, redwood forests, community-pathogen feedback, N mineralization, nitrification, *Phytophthora ramorum*

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. In 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 and Hinkel 2011; McDowell et al. 2011). Generalized ecosystem theory predicts that pathogen outbreaks which 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 and Likens 2008). However, few field studies have quantified pathogen impacts to ecosystem processes which limits understanding of the affects 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 to ecosystem processes, several authors have described useful conceptual frameworks that link host and pathogen characteristics with mechanistic changes to functional process (Burdon et al. 2006; Lovett et al. 2006; Eviner and Likens 2008). The theoretical foundations of pathogen impacts on ecosystems are corollaries to insect outbreak, and field study of insect outbreak provides guidance in the formulation of hypotheses to study ecosystem-level pathogen impacts (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 due to the arrest of nutrient resorption (Morehouse et al. 2008; Griffin and 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 is likely driven by: (1) host characteristics including biomass, unique function (shade tolerance, N fixation, phenology), and (2) host epidemiological characteristics including susceptibility, competency to transmit infection, and consequences of infection to host health (Eviner and Chapin 2003; Ellison et al. 2005; Lovett et al. 2006; Eviner and Likens 2008). Although epidemiological models can be accurately applied across broad spatial scales (Gilligan and 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 which 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 to 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 also favorable to the pathogen (Davidson et al. 2011). In 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 is epidemiologically significant but, unlike redwood and bay laurel, tanoak tree boles are also susceptible and infection causes bole-cankers that can lead to mortality in as little as two years (Cobb et al. 2012b).

Predicting which exotic organisms are likely to establish and cause deleterious impacts to natural resources remains an important but tremendously challenging goal of ecology. Eradication of many wide-spread 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 pathogen outbreak. 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: 1) direct impacts of pathogens and host mortality on litterfall chemistry, 2) mortality driven changes to soil N cycling and litterfall dynamics, and 3) 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: 1) to examine the respective effects of pathogen prevalence in bay laurel and mortality in tanoak on litter N chemistry, 2) to quantify the effects of disease-caused mortality to soil N cycling, litterfall amounts and litterfall chemistry, and 3) 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 bark beetle killed trees (Morehouse et al. 2008; Griffin and Turner 2012). We also expected stands with high levels of *P. ramorum*-caused mortality would have increased soil N availability and mineralization rates compared to stands without 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 and Turner 2012). Lastly, we expected distinct litterfall chemistry and soil N dynamics associated with the principle *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 and 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.

Methods

Field sites and study design

We conducted measurements of litterfall from January 2007 to December 2009 (3 years) and soil N cycling from December 2007 to December 2009 (2 years) at two sites where disease and vegetation dynamics had been monitored during annual summer surveys from 2002-2007 (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, landuse, 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 are circular, 500 m^2, and randomly located with at least 100m between each plot (see Maloney et al. 2005). At the time of establishment, each stem greater than 1 cm diameter at breast height (dbh; 1.3 m height) was measured for diameter, mapped, and symptomatic tissue was returned to the laboratory for pathogen isolation in a *Phytophthora* selective medium (PARP; see 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 use the strict criteria of *P. ramorum* recovery via laboratory culturing as the criteria 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 could may remain living. Our study design is predicated on the expectation that changes in ecosystem processes would be 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 which had been killed by *P. ramorum* at the initiation of measurements (dead tanoak biomass; see 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. non-invaded 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 (c.f. Lovett et al. 2010). Prevalence of infection at the plot level ranged from 66-100% of bay laurel stems and 6-95% of tanoak stems. Many study plots are notable for almost complete tanoak mortality while other plots have suffered almost no mortality even though tanoak is basal area is 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 ecosystem function responses 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 of our study plots. This provided an independent assessment of the relative influence of dominant overstory species and tanoak mortality on soil N cycling (c.f. Freid et al. 1990; Finzi et al. 1998). Further detail regarding community, pathogen, and disease characteristics can be found in Table S.1 (Supplemental Information).

Field litterfall and soil N cycling measurements

Three 1935.48 cm^2 plastic litter traps were established in each plot (~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 effectively air-dried litter between collections; we found no evidence of litter decomposition within our traps (e.g., discoloration, fungal hyphae). For the first two years of measurement, litter was collected eight times per year (every 4 to 8 weeks) until seasonal patterns of litterfall were established for each species; during the final year of measurements litter was collected every 12 weeks. Litter samples were air dried in the laboratory at 45 C^{o} for 48 hours when precipitation occurred between samplings and stored in paper bags 1-12 weeks before processing. 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 symptoms on a leaf-by-leaf basis for each sample collection by qualitatively assessing the proportion of necrotic tissue. After sorting, each sample was dried at 60 C^{o} for 48 hours, weighed, and archived for later chemical analysis. Litterfall chemistry was not measured for each sampling due to insufficient litterfall mass at some collection dates. Rather, after the two years of measurement it became clear quarterly-periods corresponding to winter (Jan-Mar), spring (Apr-Jun), summer (Jul-Sept), and autumn (Oct-Dec) 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 surface 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, 22 cm into

197 the mineral soil. The bottom 2 cm of soil was carefully removed and replaced with a nylon mesh
198 bag filled with ~10 g of IRN 150 ion exchange resin (Amberlite ^{TM}) and fitted with a rubber ring
199 which held the soil in the core. This yielded an open-top, open-bottom core which allowed free
200 water movement during the 10-28 week field incubation. A second core was used to sample the
201 top 20 cm of mineral soil and establish initial NH_4-N and NO_3-N concentration. For both
202 incubated and initial cores, the PVC tube was emptied in the field, soil samples were transported
203 back to the laboratory on ice, and processed within 48 hours. Each sample (incubated and initial)
204 was sieved to pass a 2 mm screen; a subsample was dried for 48 hours at 105 C^o to determine
205 moisture content and a second subsample was analyzed for inorganic N by gently shaking 10g of
206 field moist soil in 1M KCl for 0.5 hrs and filtering the extract through a 0.45µm pore-size glass-
207 fiber filter. NO_3-N and NH_4-N concentration of this extract was measured with a sulfanilamide
208 reaction after reduction in a copperized cadmium column and a salicylate method, respectively,
209 at the UC Davis Analytical Laboratory (QuikChem Methods 12-107-04-1-B and 12-107-06-2-A,
210 respectively; Lachat Instruments, Loveland, CO;).
211
212 Laboratory soil mineralization measurement
213 We conducted a laboratory incubation designed to examine the influence of individual
214 species and tanoak mortality on inorganic N availability and mineralization among species under
215 common environmental conditions. In April 2009, we selected eight redwood, healthy tanoak,
216 bay laurel, and tanoak where the main stem had been killed by *P. ramorum* (N = 32). These trees
217 were located at the Jack London site and chosen in sets of four such that each tree was between
218 10-40 m of the others in its set, and each set was separated by at least 150 m. We sampled the
219 surface 20 cm of mineral soil at eight locations within 2 m of each individual tree using a 6.60

cm diameter stainless steel soil-core and composited samples in the field. These samples were transported, processed, and analyzed with the same methods described for N mineralization measurements. Two subsamples for each tree were measured for initial soil moisture, KCL extractable NO_3-N and NH_4-N (64 total). Soil collection occurred within two days of significant rainfall, initial soil moisture content did not significantly differ among species, and soil moisture was at adequate levels to support microbial processes for the five week 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 ~50 g soil (sieved to pass a 2 mm screen) in 300 ml volume vented plastic sample cups (320 total). Microcosms were incubated at 22 C^o in a dark, climate-controlled space and two microcosms from each tree were destructively sampled every week for 5 weeks to estimate changes in N dynamics through time. Each microcosm was assessed for soil moisture, KCL extractable NO_3-N and NH_4-N. Tree-level data were taken as the average value from both microcosms and the two 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. Objective 1: To examine relationships between pathogen prevalence and litter %N for bay laurel, and mortality and litter %N for tanoak we employed a series of linear models for each season of collection where individual chemistry parameters (%C, %N, C/N) were the dependent variables and infection (number 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. Objective 2: We expected that disease caused changes in litterfall mass, N mass, and soil N

dynamics would be a joint function of the maximum potential disease impact (initial tanoak basal area m^2 ha^{-1}) conditioned on 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; c.f. Gotelli and Ellison 2004) and the time-by-disease interactions are meaningful given that mortality occurs on an annual basis. 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 two years of measurement (c.f. Gotelli and Ellison 2004). These models also included a fixed effect of soil moisture measured in the initial cores to examine potential moisture limitation to microbes among plots. Objective 3: 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. Differences in litter %N, %C, 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. Variation 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 compared each tree-type individually with each of the others. For each linear model, normal distribution and homogeneous variance of the residuals was assessed with goodness of fit tests to the normal distribution and visual examination of heteroskedasticity; 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 the JMP® version 8 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 (Figure 1). The spring and early summer seasons also correspond with peak periods of *P. ramorum* sporulation and within-tree infection at the Jack London Site (see 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 (Figure 2). Bay laurel %N and C/N were not significantly related to prevalence of symptomatic leaves. Given that bay laurel contributed ~7-11% of overall litterfall N (Figure 2; Table S.2) 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 (Figure 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 (Figure 3). Litterfall amounts were positively associated with the respective pre-disease 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 $p < 0.05$; Figure 3; Table S.3). The estimates from the repeated measures model indicate *P. ramorum*-caused tanoak mortality resulted in up to 91% reduction in tanoak litterfall and up to 95% reduction of tanoak litterfall N in plots with the greatest amount of cumulative tanoak mortality (up to ~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 due to litter production from basal sprouts that frequently developed from *P. ramorum* killed tanoak stems. Compared to other species collected in our litter traps, tanoak had less seasonal variation (Figure 2). Even though redwood dominates litterfall N mass (65-78% of total), total litter N (stand-level) decreased with tanoak mortality (Figure 3) probably because tanoak litter has relatively high %N compared to 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 S.4; supplemental information).

311 Total and NO_3-N pools were significantly increased with disease but rates of nitrification
312 and mineralization were not affected (Figure 4). NO_3-N and total N concentration were
313 negatively associated with pre-disease tanoak basal area and positively associated with
314 cumulative dead tanoak basal area and soil moisture (Figure 4; Table S.5). Extractable inorganic
315 N pools were dominated by NO_3-N, and were often more than 60% nitrate. The shared patterns
316 of significance between NO_3-N and total N is mostly driven by this high proportion of NO_3-N
317 (total N = NO_3-N + NH_4-N). Similarly, nitrification rates were 80 to 100% of net N
318 mineralization for soils incubated in the field (Figure 4). Almost identical patterns between
319 nitrification and N mineralization were driven by the dominance of nitrification in N
320 mineralization rates of our study plots. Seasonal influences on soil N concentration and
321 mineralization were weak although the sampling duration also spanned a California-wide
322 drought from 2007-2008.
323
324 *Species effects on litterfall, litterfall chemistry, and soil N cycling: Objective 3*
325 Litter chemistry was markedly different among species. %N was greatest in bay laurel,
326 lowest in redwood, and intermediate in tanoak (Figure 2; $p < 0.05$ each contrast). Litter C/N
327 followed a similar pattern with the highest C/N in redwood, the lowest in bay laurel, and
328 intermediate values for tanoak. Redwood dominated the total litterfall mass in our plots with
329 amounts followed by tanoak, madrone, bay laurel, and other species (Figure 2; Table S.2).
330 Redwood litterfall was low during the spring and summer but peaked in the late autumn/early
331 winter (Figure 2). Tanoak and bay laurel litterfall tended to peak in the mid summer and early
332 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 levels in the winter and lowest levels 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 five week laboratory soil incubation (Figure 5). Tanoak had significantly lower extractable NO_3-N compared to bay laurel and dead tanoak. Soil NO_3-N availability from redwood was significantly higher compared to tanoak and tended to be lower than bay laurel or dead tanoak but these differences were not significant. Total N levels were similar between species and net rates of nitrification and N mineralization also did not differ among species (Figure 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 (Figure S.1). The overall patterns of N availability from the laboratory incubation were consistent with measurements made in the field. In both measurements 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 to these processes for many landscape-scale tree mortality events. Tanoak mortality 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 and perhaps greater-magnitude changes to these ecosystem features. Our study, along with several others, suggests disease-caused ecosystem changes can be driven primarily by mortality

and the resulting changes in plant community composition (Hobara et al. 2001; Lovett et al. 2006; Orwig et al. 2008; Cobb 2010). These results suggest 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 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 (Figure 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 species such as redwood or Douglas fir (Cobb et al. 2010) which frequently co-occur with tanoak. These species have notably lower litter quality compared to bay laurel or tanoak which is likely to result in slower litter decomposition and net accumulation of forest floor mass (Figure 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 has been a common ecosystem response following insect and pathogen outbreak (Hobara et al. 2001; Orwig et al. 2008; Morehouse et al. 2008; Lovett et al. 2010; Griffin and Turner 2012). Our study departs from this

379 overall trend in that mortality increased NO_3-N availability but did not change cycling rates, a
380 result that was consistent in the laboratory as well as the field (Figures 4 and 5). The majority of
381 studies examining pathogen and insect impacts to ecosystems have also focused on outbreaks
382 which result in more uniform mortality or defoliation across a stand compared to sudden oak
383 death (Hobara et al. 2001; Russell et al. 2004; Morehouse et al. 2008; Orwig et al. 2008; Lovett
384 et al. 2010; Griffin and Turner 2012). In contrast, even in our study plots with the greatest
385 amount of tanoak mortality, the majority of biomass was in redwood, bay laurel, or other species
386 which are minimally impacted by the disease. Further, survival times of *P. ramorum* infected
387 tanoak trees can vary from 2-20 years because of differences in susceptibility within populations
388 and size-specific mortality rates (Hayden et al. 2011; Cobb et al. 2012b). The resulting spatial
389 and temporal variation in mortality may dampen impacts to soil N cycling because changes in
390 canopy structure are less severe relative to homogeneous disturbances or outbreaks (Cobb et al.
391 2012a). Comparatively, Gypsy moth (*Lymantria dispar*) outbreak can cause extensive defoliation
392 with low mortality relative to other outbreaks (Lovett et al. 2002; Russell et al. 2004); this
393 defoliation can increase litterfall and litterfall N without changing N mineralization or
394 availability (Russell et al. 2004). Our study supports the general expectation that the timing and
395 uniformity of mortality is an important control over the magnitude of changes to ecosystem
396 processes following outbreak (Ellison et al. 2005; Lovett et al. 2006; Eviner and Likens 2008)
397 even though our data do not confirm our initial hypothesis that disease would increase rates of
398 soil N mineralization.

399 Direct impacts of infection on host tissues had the least significant effect on ecosystem
400 processes at the spatial scale of our study (the ecosystem; Figure 1). Unlike bark-beetle caused
401 mortality, tanoak mortality was not associated with increased litterfall %N (c.f. Morehouse et al.

2008; Griffin and 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 (Figure 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 and 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 suggesting direct pathogen impacts did not drive these changes in litterfall chemistry. In contrast, Davidson et al. (2011) demonstrated increased rates of leaf shedding for infected vs. uninfected bay laurel leaves and suggested *P. ramorum* can accelerate leaf senescence by 3-4 years. 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* species 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* species may influence bay laurel litterfall %N without eliciting disease (c.f. Eviner and Likens 2008).

P. 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). Mortality from sudden oak death is largely driven by sporulation sources in conjunction with the distribution of

425 tanoak and susceptible oaks, the species which may be killed following *P. ramorum* infection
426 (Davidson et al. 2008; Meentemeyer et al. 2008; Lamsal et al. 2011). These patterns emerge
427 because *P. ramorum* virulence is high and resistance in tanoak is insufficient to protect many
428 tanoak populations from significant mortality (Rizzo et al. 2005; Hayden et al. 2011). Patterns of
429 mortality can be reasonably predicted for several other exotic pathogens and insects that are
430 actively spreading into naïve host populations and where community or landscape factors of
431 spread are well understood (Loo 2009; Lovett et al. 2010; Orwig et al. 2012). However,
432 predicting mortality is much more difficult for many regional tree mortality events because the
433 relationships between physiological stress and pathogen impacts are typically unknown for the
434 diverse and widespread native pathogenic flora of most temperate forests (Sinclair et al. 1987;
435 McDowell et al. 2011). Understanding how or when native pathogens and insects overcome
436 plant defenses, and what landscape, climatic, or management factors predispose hosts to greater
437 physiological stress (Raffa et al. 2008; Adams et al. 2009; McDowell et al. 2011) is likely to hold
438 greater potential to predict landscape-level tree mortality of these outbreaks.
439 For sudden oak death, many of the ecosystem changes we observed are tied to the
440 epidemiological roles of canopy tree species and their individual influences on ecosystem
441 processes. The mechanisms driving these affects included changes in host litter chemistry,
442 mortality, and shifts in community composition that are likely to be common among many
443 pathogen outbreaks in the same way that they are common drivers of ecosystem change
444 following insect outbreak. Although interactions between pathogens, hosts, and the environment
445 is a foundation of plant pathology (e.g. the disease triangle; Burdon et al. 2006), these
446 interactions are poorly understood for abundant, diverse, but broadly distributed weak pathogens
447 (Balci et al. 2007; Wickson et al. 2008; Hawkins and 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 substantially 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.

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607 **Figure captions**

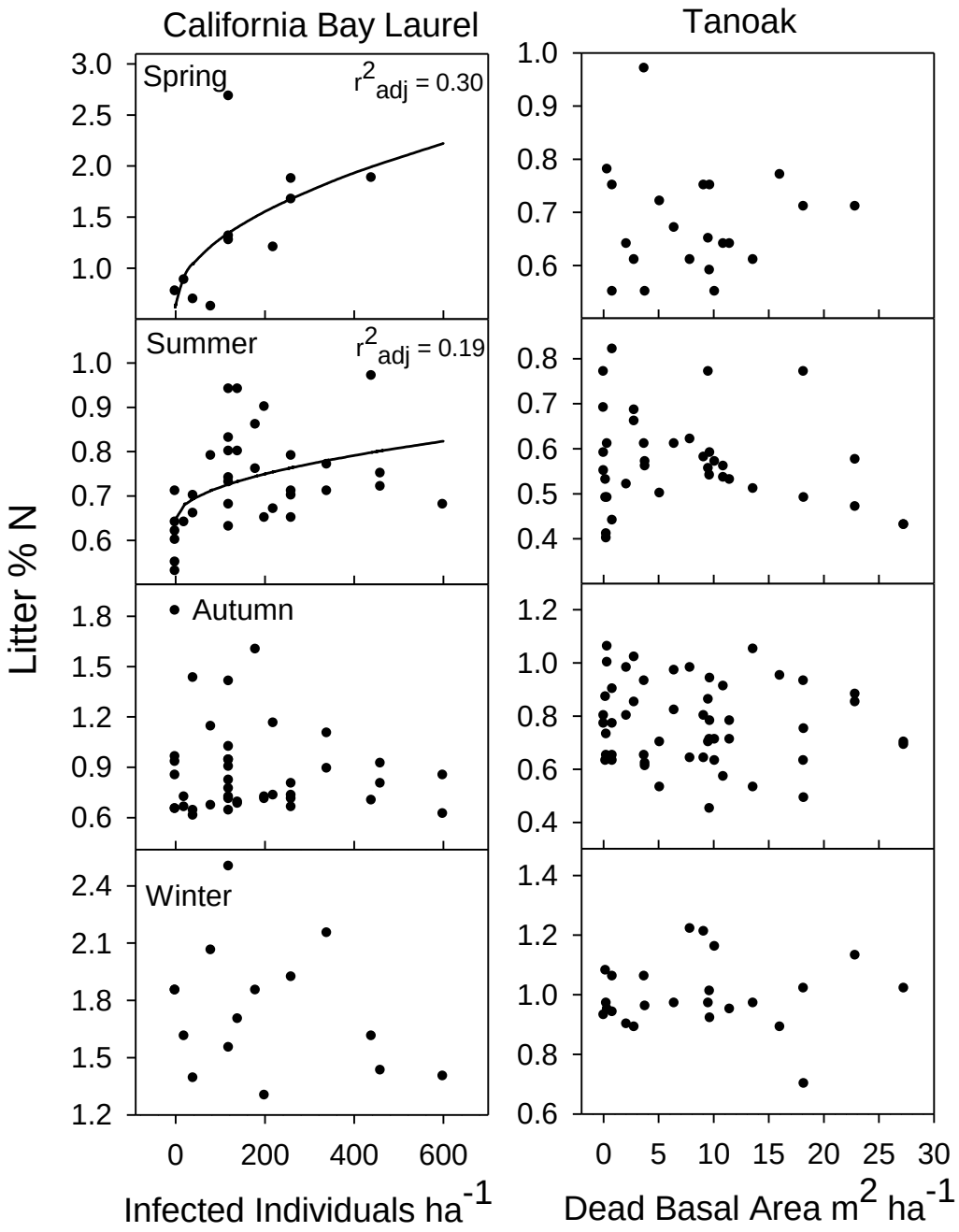
608 Figure 1. Litterfall %N vs. prevalence of *Phytophthora ramorum* in bay laurel or cumulative
609 dead tanoak basal area. When the relationship between infected hosts or mortality and litter N
610 concentration was significant ($p \leq 0.05$), the r^2 is reported along with the square root transformed
611 least squares fit. Note the differences in scale for bay laurel and tanoak (x-axis) as well as
612 differences in N concentration between seasons of measurement (y-axis).

613 Figure 2. Monthly litterfall mass (A-C) with seasonal values of carbon and nitrogen (D-F) for
614 bay laurel (top), tanoak (middle), and redwood (bottom) from two redwood forests impacted by
615 sudden oak death. Data are means from three years of litterfall monitoring presented with one
616 standard deviation for litterfall mass and standard error for seasonal litter chemistry. Note the
617 differences in scale in the panels of litterfall mass for each species. The month of collection is
618 abbreviated with the first letter.

619 Figure 3. Affects of sudden oak death-caused mortality (x-axis) on tanoak annual tanoak litterfall
620 mass (A), tanoak litterfall N (B), and total stand-level litterfall N (C). Data are observed values
621 minus those expected if the stands had not been impacted by sudden oak death (see text and table
622 S.3 for more details). Negative values on the y-axis denote the amount of litterfall reduction
623 associated with a given amount of tanoak mortality. Data are total annual amounts for each plot
624 with least squares regression lines.

625 Figure 4. Seasonal patterns and affects of sudden oak death on soil N. Seasonal patterns of
626 inorganic N pool sizes (A) and rates of N mineralization and nitrification (B) are shown with
627 sampling date on the x-axis. Right panels; leverage plots from mixed linear models showing the
628 effect of tanoak mortality on N pool sizes (C) and rates of turnover (D). Least squares regression
629 lines are shown for statistically significant ($p \leq 0.05$) models.

630 Figure 5. Species level effects on N availability (extractable pools A and B) and cycling rates (C
631 and D). Data are results from incubation of soils collected immediately below the three focal
632 species and tanoak killed by *P. ramorum* (Dead Tanoak). Data are means with one standard
633 error. Results from a paired t-test analysis are presented above each bar with the significance
634 tests indicating differences across all possible pairs; different letters indicate statistically
635 different mean values ($p \leq 0.05$).



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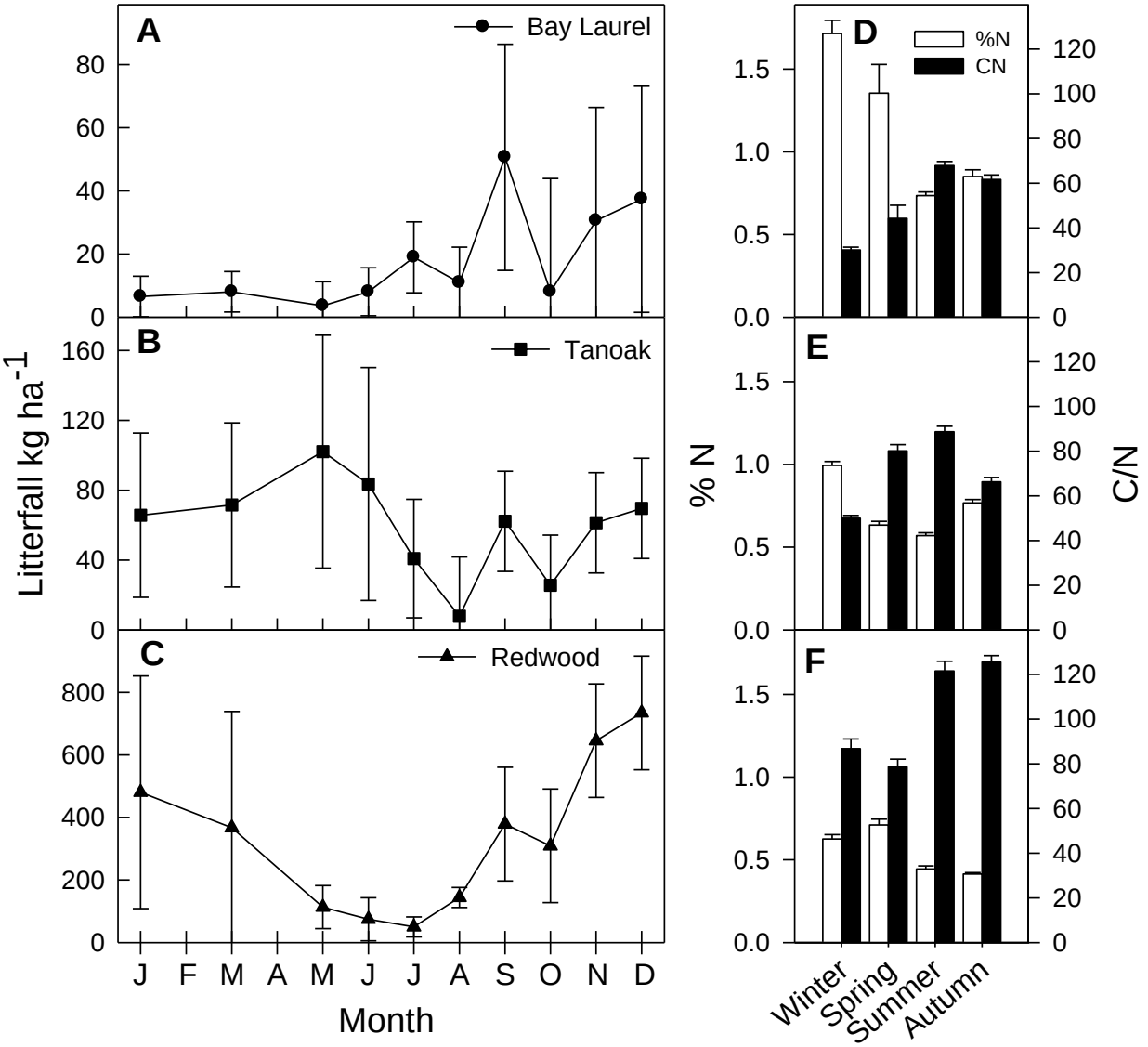

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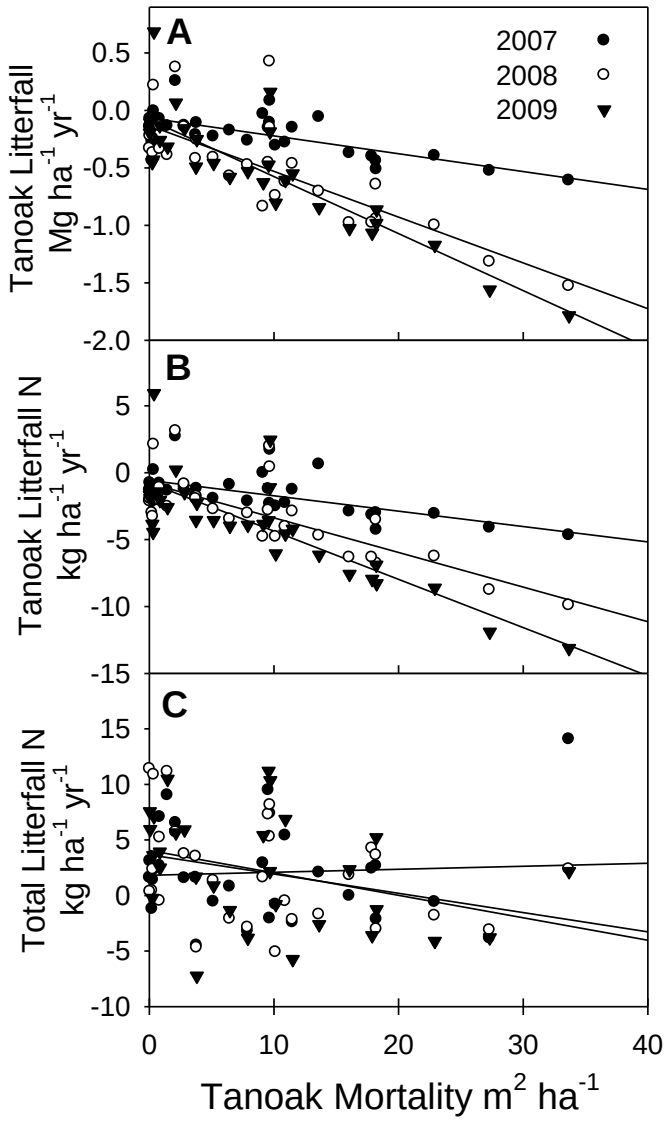

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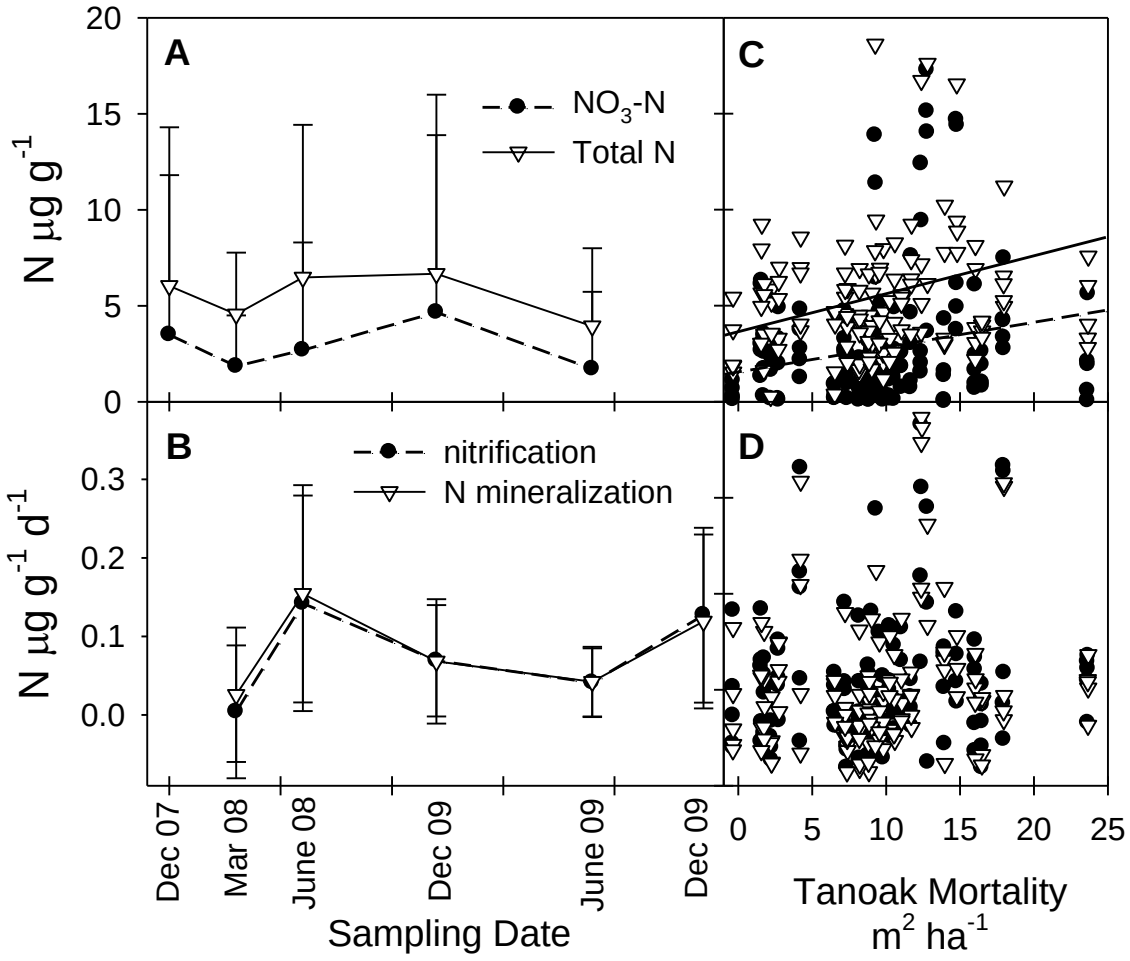

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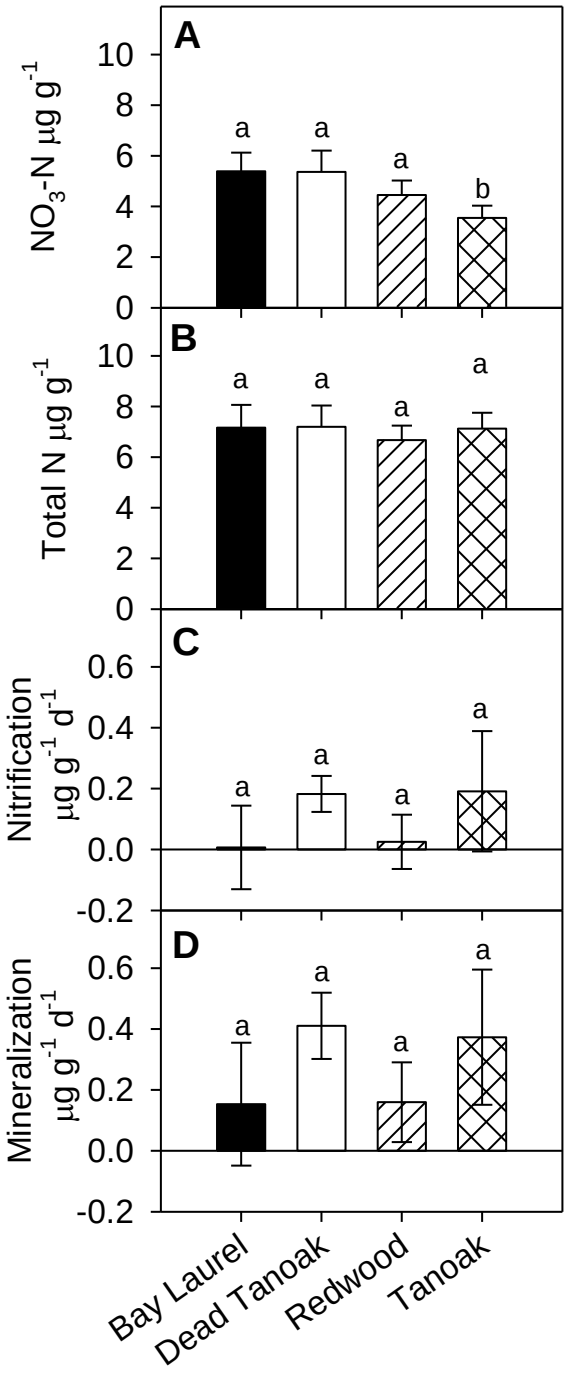

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