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

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Abstract

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

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

Introduction

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

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(Preston and others 2016). In turn, these impacts can alter biogeochemical processes by changing litterfall chemistry, amount, and timing as well as physical factors, including temperature, moisture, and light interception at the forest floor (Ruess and others 2009; Lovett and others 2010; Cobb and others 2013). Despite well-founded expectations of disease-driven changes to carbon cycling, few studies have quantified pathogen effects on forest communities and associated ecosystem processes; exceptions tend to focus on pronounced mortality events (Hicke and others 2012). However, many pathogens impact their hosts without causing mortality, creating potential for cryptic but spatially extensive changes to ecosystem processes (Hansen 1999; Ostry and Laflamme 2008). These effects are most likely to emerge through impacts of broad host-range generalist pathogens which may cause a range of disease severity within landscapes or communities (Eviner and Likens 2008).

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

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

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

Methods

Field Sites and Litter Collection

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

Field and Laboratory Methods

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

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

Data Analysis

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

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

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

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

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

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

Results

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

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

Table 1. Initial Litter Chemical Characteristics

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

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

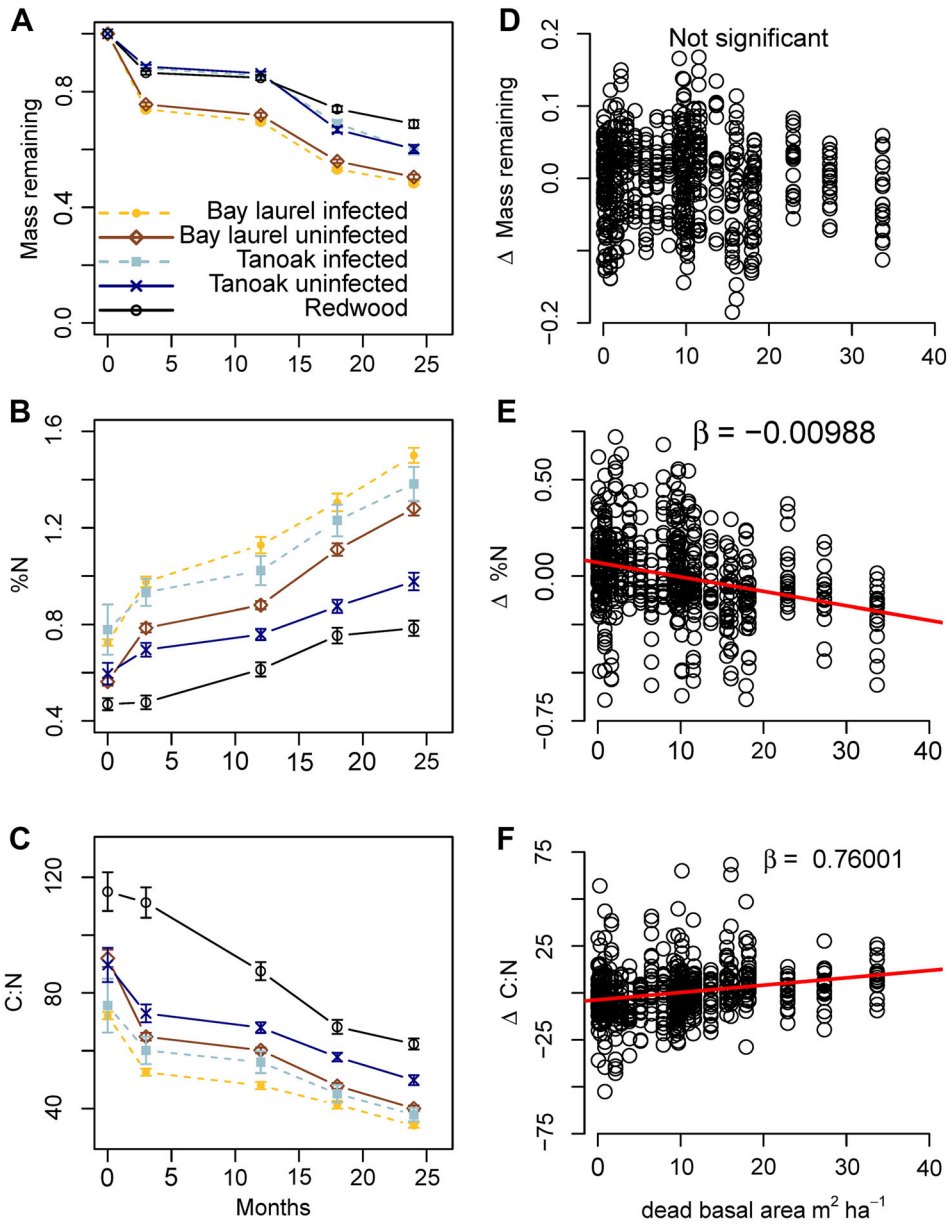

Figure 1. Effects of species identity, infection status, and canopy mortality on litter decomposition, the three most important hosts of *Phytophthora ramorum* in California redwood forests. **A–C** Effects of species and infection status for mass loss, %N, and C:N, respectively. **D–F** Effect of tanoak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

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

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

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

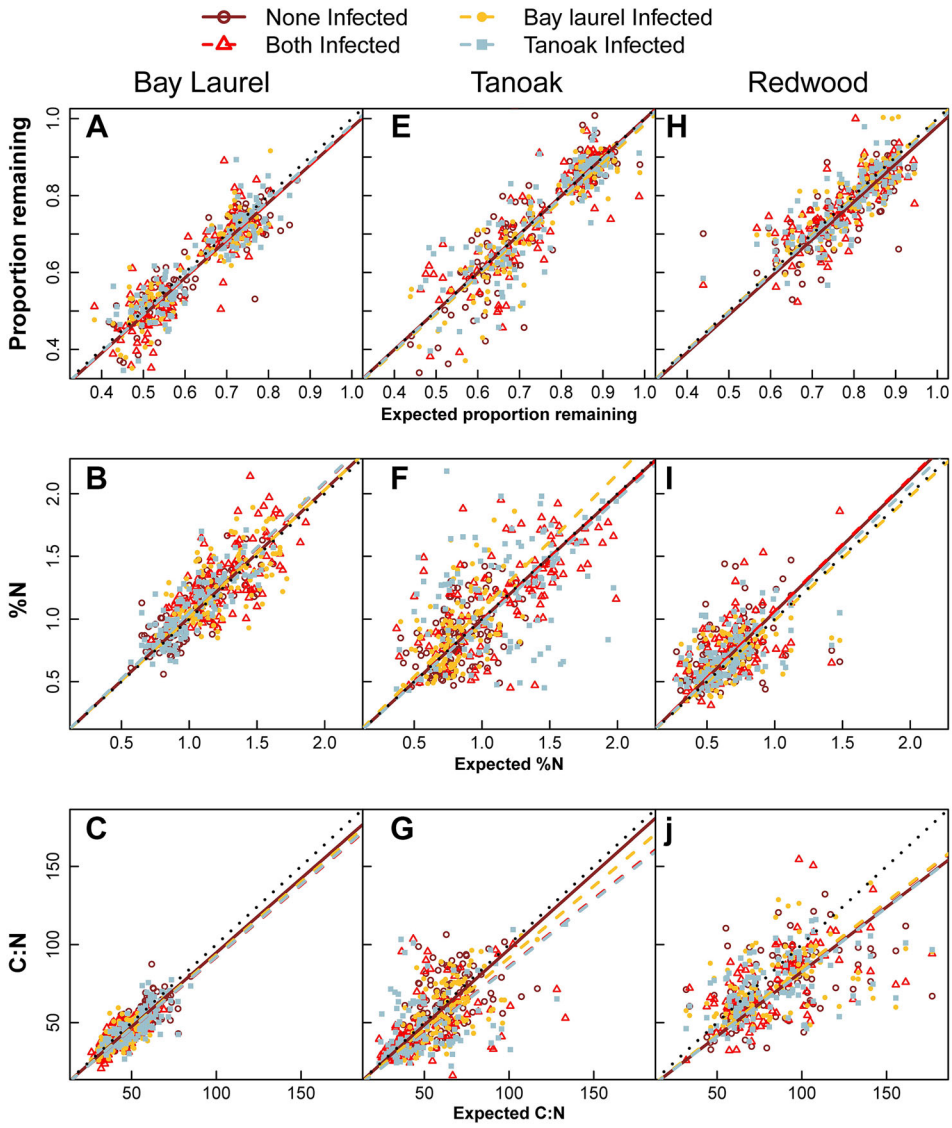

Figure 2. Non-additive effects on bay laurel, tanoak, and redwood litter decomposition across a full-factorial combination of bay laurel and tanoak from infected and uninfected sources. In each case, the *dotted one-to-one line* is the expected relationship from each litter-type decaying alone. Expected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

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

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

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

Discussion

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

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

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

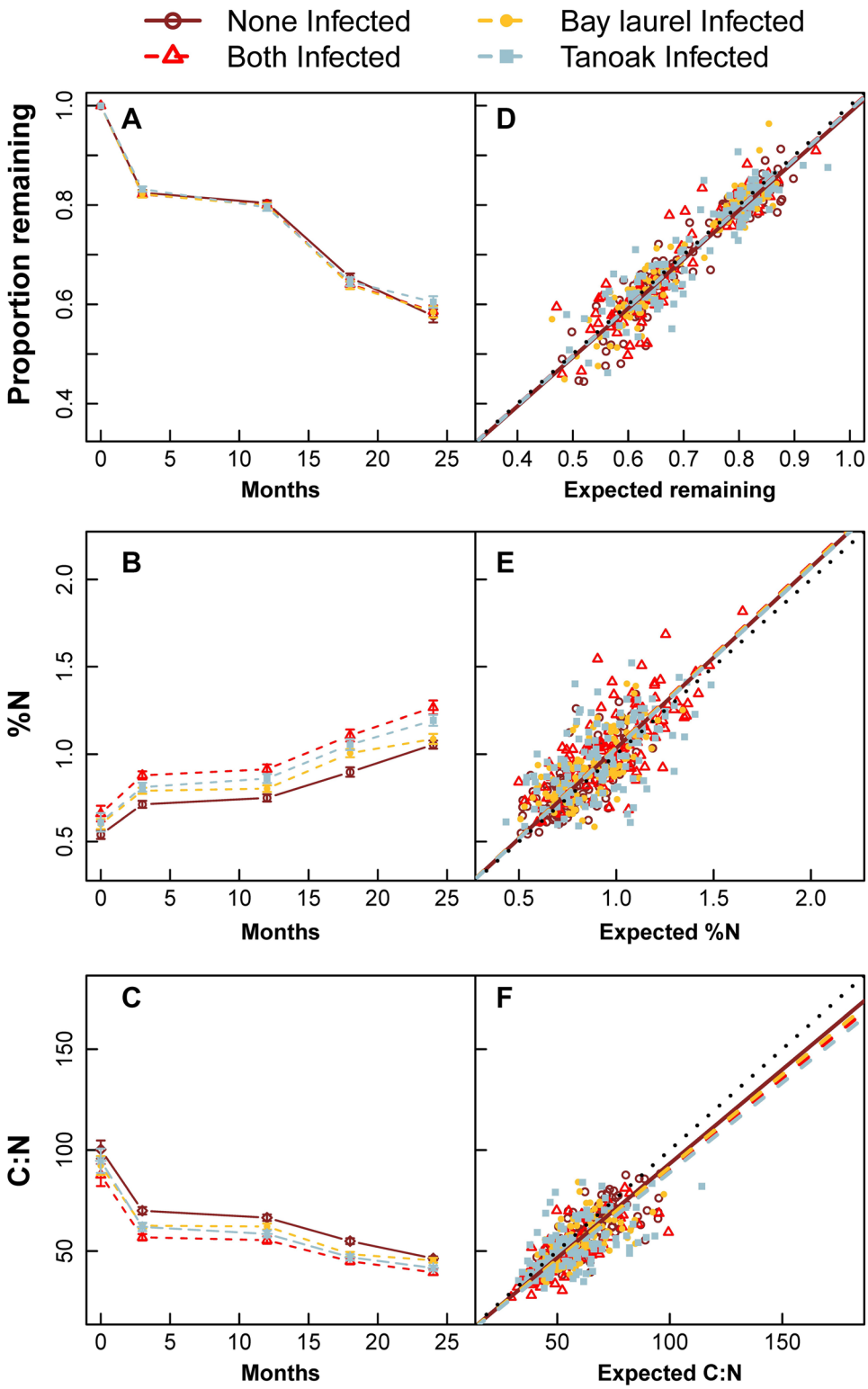

Figure 3. Dynamics of the overall mixed litter bags with treatments spanning a full-factorial combination of bay laurel and tanoak from infected and uninfected sources (**A**–**C**). Each litter bag contained the same proportion of each litter type (1/3), but the infection status of bay laurel and tanoak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

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

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

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

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

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

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

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

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

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

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