Ecosystems
DOI: 10.1007/s10021-011-9506-y

ECOSYSTEMS

Common Factors Drive Disease and Coarse Woody Debris Dynamics in Forests Impacted by Sudden Oak Death

Richard C. Cobb,[1,2]* Maggie N. Chan,[2] Ross K. Meentemeyer,[3] and David M. Rizzo[1,2]

[1]*Graduate Group in Ecology, University of California, One Shields Ave., Davis, California 95616, USA;* [2]*Department of Plant Pathology, University of California, One Shields Ave., Davis, California 95616, USA;* [3]*Department of Geography and Earth Sciences, University of North Carolina, 9201 University City Boulevard, Charlotte, North Carolina 28223, USA*

Abstract

Disease ecology has made important steps in describing how epidemiological processes control the impact of pathogens on populations and communities but fewer field or theoretical studies address disease effects at the ecosystem level. We demonstrate that the same epidemiological mechanisms drive disease intensity and coarse woody debris (CWD) dynamics in natural forest ecosystems impacted by an emerging disease. Sudden oak death (causal agent, *Phytophthora ramorum*) has caused mortality of tanoak (*Notholithocarpus densiflorus*) on a spatial scale and rate comparable to other major North American forest diseases caused by invasive pathogens. In pathogen invaded stands, mean CWD masses were 22.4 Mg ha^{-1} of standing dead tanoak (snags) and 11.5 Mg ha^{-1} in logs compared to 0.27 and 1.16 Mg ha^{-1} of snags and logs in an uninvaded stand. Within invaded stands variation in CWD mass and accumulation rates were largely driven by the distribution of pre-disease tanoak biomass and the densities of infected tanoak and California bay laurel (*Umbellularia californica*) which jointly determine *P. ramorum* sporulation and disease emergence rates. In a narrow range of community and host characteristics sudden oak death can result in woody debris dynamics similar to discrete disturbances such as fire and forest harvest but it is more common to have lower maximum amounts with slower rates of accumulation than these better studied disturbances. Our results indicate that models of CWD dynamics need to integrate epidemiological processes to predict realistic ecosystem impacts and lead to management applications for forest pathogens.

Key words: *Phytophthora ramorum*; emerging infectious disease; disease ecology; tanoak; coarse woody debris; decomposition.

Introduction

A developing body of research highlights the mechanisms by which pathogens, parasites, and insect herbivores alter ecosystem function. Pathogen outbreak can cause shifts in community

Received 26 May 2011; accepted 21 October 2011

Electronic supplementary material: The online version of this article (doi:10.1007/s10021-011-9506-y) contains supplementary material, which is available to authorized users.

Author Contributions: RC and RM conceived and designed study, performed research, analyzed data, designed methods and models, and wrote the paper. MC performed research, analyzed data, designed methods and models, and wrote the paper. DR conceived and designed study, analyzed data, designed methods and models, and wrote the paper.

**Corresponding author; e-mail:* rccobb@ucdavis.edu

Published online: 10 December 2011

composition, the distribution of biomass, and forest canopy characteristics that in turn alter annual net primary productivity, nutrient cycling, and the accumulation and decomposition of organic matter (Ellison and others 2005; Lovett and others 2006; Eviner and Likens 2008). Understanding when and to what degree pathogen outbreak alters these processes requires integration of epidemiological understanding into field and theoretical ecosystem studies. Epidemiology has many parallels to the broader field of ecology in that predicting disease outbreak relies on understanding how pathogens interact with the environment and their hosts. For example, variation in host contact structure and susceptibility influence individual tree response to pathogens as well as spatial and temporal patterns of mortality (Hansen and Goheen 2000; Rizzo and others 2000; Kauffman and Jules 2006). For generalist pathogens, community composition determines the frequency of hosts that support high levels of sporulation and in turn the rates of pathogen spread within stands (Burdon and others 2006; Cobb and others 2010). Of direct importance to ecosystem ecology, the local dominance, biomass, or functional importance of species killed following infection determines the magnitude and nature of ecosystem change (Matson and Boone 1984; Lovett and others 2006; Ruess and others 2009). In attempting to combine epidemiological and ecosystem understanding, it is important to recognize that pathogen impacts range from dramatic population declines to removals of single individuals and may occur over many years (Eviner and Likens 2008). In contrast, many ecosystem models consider disturbances which are spatially homogeneous, discrete in time, and cause tree mortality in single or multiple but unrelated mortality events. Although these models are excellent tools for examining ecosystem changes for events such as fire and forest harvest, they are yet to integrate the spatiotemporal variability characteristic of outbreak.

In forest ecosystems, coarse woody debris (CWD; dead wood greater than 10–20 cm diameter) is an important store of nutrients, C, and sources of C flux (Harmon and others 1986; Janisch and Harmon 2002). In many forests, these structures are also important wildlife habitat, substrate for tree recruitment, and influence fire impacts (Sturtevant and others 1997; Schlegel and Donoso 2008; Metz and others 2011); consequently they are often the focus of management (Spies and others 1988; Valachovic and others 2011). Several field studies demonstrate that insect and disease outbreak can increase CWD accumulation rate and amount (Lang 1985; Sturtevant and others 1997; Edman and others 2007; D'Amato and others 2008; Swei and others 2011) but little effort has been made to develop models of disease and CWD dynamics.

Some of the first models of CWD dynamics were designed to address fir-waves in New Hampshire (Lambert and others 1980) where wind exposure leads to declines in tree vigor and concentrated mortality in defined zones that travel up mountain elevation gradients (Marchand and others 1986). Early CWD models also address acute insect outbreak (Lang 1985) but both fir-wave and acute insect outbreak are events where mortality is concentrated in a short period or is spatially homogeneous. Subsequent models have focused on other common but temporally discrete disturbances such as forest harvest and wildfire (Spies and others 1988; Tinker and Knight 2000; Harmon 2009). Discrete disturbance models are unlikely to fit disease outbreaks where environmental stochasticity, the distribution of host species, and asymmetric pathogen impacts create complex spatial and temporal variability in tree mortality (Hansen and Goheen 2000; Rizzo and others 2000; Edman and others 2007; Meentemeyer and others 2008b). Moreover, impacts of disease are likely to stretch CWD accumulation over longer periods compared to discrete events, such as fire, harvest, and wind (Lang 1985; Tinker and Knight 2000; Edman and others 2007). Figure 1 shows a simple but mathematically rigorous conceptual model of CWD dynamics for a forest pathogen (compare Lang 1985; Harmon 2009; see Supplementary materials for mathematical details). In this model, variation in infection rate and subsequent mortality within a size-structured tree population results in very different magnitudes, timing, and duration of dead biomass. In contrast, mortality is treated as single or multiple pulses in discrete disturbance models; this treatment of mortality is unlikely to fit outbreaks that cause continuous mortality over years or decades especially for disease outbreaks where annual mortality is determined by interacting epidemiological drivers (Eviner and Likens 2008).

This study examines CWD dynamics in forests impacted by the emerging forest disease sudden oak death in Mediterranean ecosystems in California (Rizzo and others 2005). The causative pathogen, *Phytophthora ramorum*, can infect over a hundred plant species, but these hosts are highly variable in their competence to transmit the pathogen and the consequence of infection on host health (Rizzo and others 2005; Davidson and others 2008). For example, leaf infections on California bay laurel (*Umbellularia californica*) support high

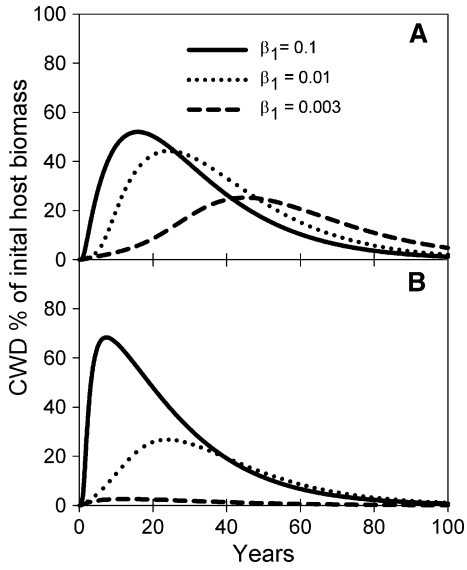

Figure 1. Results of a simple size structured epidemiological and CWD dynamics model demonstrating the sensitivity of woody debris accumulation rates and maximum levels to differences in infection rates. For each realization of the model initial host biomass is equal. **A** Mortality rates are held constant while within species infection rates (β_1) are varied. **B** Mortality rates increase with increasing tree size and values β_1 are the same as **A**. Slower infection rates lead to greater amounts of time required to reach maximum CWD levels and overall lower maximum amounts. Increased mortality rate in larger individuals in combination with high infection rate approximates the typical pattern of CWD dynamics following a discrete disturbance such as wildfire or forest harvest (**B**). The model is a system of linked ordinary differential equations without spatial structure. The explicit model and parameter values are reported in the Supplemental information.

levels of sporulation but with no known deleterious impacts at the individual or population level in this species (DiLeo and others 2009; Cobb and others 2010). In contrast, significant sporulation occurs on twig and leaf infections in tanoak (*Notholithocarpus densiflorus*) but, bole infections on this species disrupt sapwood function and lead to rapid tree mortality (Parke and others 2007; Davidson and others 2008; McPherson and others 2010). Sudden oak death is highly selective against tanoak which dies rapidly following infection; once infected, large tanoak have more rapid mortality rates compared to small trees (McPherson and others 2010; Cobb and others, unpublished data). Forest community composition influences both pathogen establishment and disease impacts. At the landscape scale, high bay laurel and tanoak densities increase the likelihood of pathogen establishment (Maloney and others 2005; Condeso and Meentemeyer 2007) whereas within stands, tanoak mortality is positively associated with the prevalence of infection in bay laurel and tanoak (Cobb and others 2010; Davis and others 2010).

In light of the important contributions to ecosystem structure played by woody debris, we used sudden oak death as a case study to examine the importance of epidemiological processes on CWD dynamics. We assessed disease severity and accumulation of CWD mass across a range of community composition and environmental conditions that affect the rate of disease emergence. Specifically, we ask (1) what is the significance of sudden oak death to CWD accumulation in natural forest ecosystems? and (2) what are the epidemiological drivers of CWD amounts and rates of accumulation across heterogeneous landscapes? Answers to these questions are needed to integrate epidemiological mechanisms into models of CWD dynamics and to increase the realism of these models for predicting ecosystem impacts of forest disease. We avoid fitting our data to a temporally explicit model (for example, Figure 1; Harmon and others 1986) because *P. ramorum* has not been established in California forests long enough to observe peak woody debris levels. Instead, we examine mechanisms driving CWD accumulation to determine the degree to which woody debris dynamics moderated by disease are analogous to discrete disturbances and to identify how CWD models can better accommodate the nature of epidemiological processes. We address this objective with detailed measurements of pathogen prevalence, vegetation composition, tree mortality, and tree-fall via 6 years of annual surveys (2002–2007) and a one-time census of logs and snags (standing dead trees) during 2007–2008. We hypothesize that rates and overall amounts of CWD accumulation are driven by epidemiological parameters, which may either approximate or proceed slower than discrete disturbances (Figure 1).

Study Area, Landscape, and Disease History

Phytophthora ramorum is well established in coast redwood and mixed evergreen forests in coastal California from the Big Sur region to Mendocino County with isolated outbreaks in Humboldt County and in Curry County, Oregon (Rizzo and others 2005). Variation in community composition, especially the local prevalence of bay laurel and tanoak, has resulted in heterogeneous disease intensity across California (Maloney and others 2005; Meentemeyer and others 2008a, b; Davis and

others 2010; Metz and others 2011). Tanoak biomass and the likelihood of *P. ramorum* establishment are highest in northern California suggesting that sudden oak death impacts will become more extensive in the coming decades (Lamsal and others 2011; Meentemeyer and others 2011). In 2002, we established one-hundred and twenty 500 m^2 plots in 14 redwood (*Sequoia sempervirens*) dominated sites to study the influence of plant community composition on disease progression and resulting ecosystem impacts (Maloney and others 2005; Figure 2). This plot network was established near initial infection centers (Marin-Sonoma county, Santa Cruz, and Big Sur) to minimize differences in pathogen invasion history. *P. ramorum* was established in all but one study site (Big Basin State Park) within 2 years of plot establishment (Maloney and others 2005; Cobb and others 2010). Evidence of past fire is present at all study sites and all sites have experienced some forest harvest except an old-growth stand at Henry Cowell Redwoods State Park. We characterized moisture availability for each plot by deriving a topographical moisture index (TMI) in a GIS using a 30-m USGS digital elevation model, the amount of up-slope drainage area, and the overall slope of the 30-m cell (see Meentemeyer and others 2008b).

Field Surveys and Laboratory Methods

Complete censuses of all stems greater than 1 cm dbh were conducted in 2002 and 2007 between May and October. From 2003 to 2006 we conducted partial surveys of each plot; five trees with and without previously confirmed *P. ramorum* infections were assessed for infection and mortality (Maloney and others 2005; Cobb and others 2010). Tree fall events, snag decomposition class, and snag height were recorded during the final census between June and October 2007. Snag height was assessed with visual estimates; visual estimates were unavoidable given the dense canopy and variable topography of our study plots. To minimize error associated with visual estimates of height, each surveyor was trained using trees of known height and each snag was separately evaluated by two observers. We quantified potential error associated with visual estimates by remeasuring a subset of trees in fall 2010 with a laser range finder and conducted a formal analysis of error propagation (see data analysis). Snag decomposition class was assigned using a five class scale adapted to overstory species in redwood forests with special emphasis on tanoak (Supplemental information).

Between November 2007 and July 2008 we conducted a complete census of all logs with diameter and length greater than 10 cm and 1 m, respectively. We measured within-plot log length and diameter at the large and small ends of each log. Decomposition class was assessed every 1–2 m on the log and assigned an overall decomposition class on a five class rating scale (Supplemental information). Approximately 20% of surveyed logs were sampled for assessment of wood density except at Henry Cowell Redwoods State Park where cultural and esthetic conservation precluded the cutting of logs. Using hand saws, we sampled wood from the bark to the pith and removed half-circle cookies including from logs in the first decomposition class. Use of hand saws precluded measurement of very large, undecomposed logs but median size of sampled logs was 22 cm and included large (50–94 cm) diameter logs in decomposition classes 3–5. A subset of trees had especially well-resolved mortality and fall dates, these logs were sampled for woody density analysis even when they came to rest outside of the plot and were used in estimates of log decomposition. To estimate initial tanoak wood density we collected complete cross sections from four live tanoak stems with diameters between 10 and 15 cm sampled from the mid-peninsula water management district (K. Aram, unpublished data) and four recently wind-thrown trees at the Jack London study site. We mapped the position of each log in half of the study plots at the Jack London site and reassessed decomposition class and wood density in December 2010. We were unable to census logs in the 30 most southern study plots due to the 2008 Basin Complex Fire which impacted these plots between the surveys for snags and logs.

Laboratory Measurements

Wood samples were stored at 5°C and processed for wood density measurements within 3 days of collection. The volume of each piece was measured twice by water displacement in a graduated cylinder; although this method can have high error rates we found that it had lower levels of root mean square error (RMSE = 0.21 cm^3) from repeated measurement compared to measurements with a top-loading balance (RMSE = 8.25 cm^3). Water absorption in a few of the most highly decomposed wood samples required wrapping the sample in Parafilm® to limit water absorption and density changes. All samples were dried at 60°C to a constant mass (consistently within 72 h) and a subset (20%) was further dried at 105°C to a constant

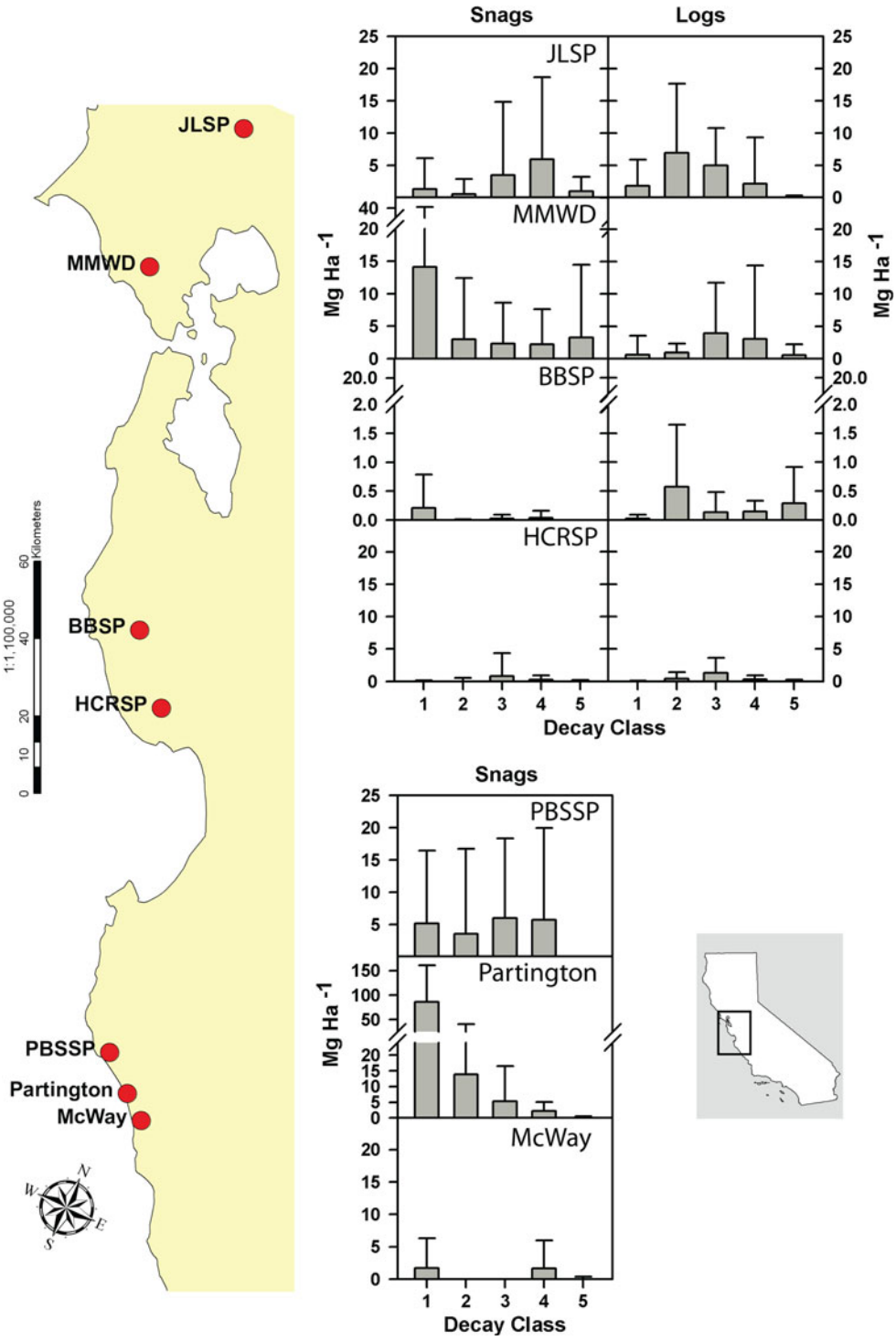

Figure 2. Distribution of tanoak snag and log mass across study sites in central-coastal California. Data are means and standard deviations for each decomposition class. Decomposition class represents different levels of decay in snags compared to logs (see text). Study sites within 5 km were grouped into single panels. Note differences of the *y*-axis scales for some study sites.

mass to correct for residual moisture across samples.

CWD Mass Calculations

Snag volume was estimated assuming constant snag taper and standard shapes across levels of CWD-flux (Supplemental information). Wood samples for density measurements could not be safely removed from snags. Instead, we assessed snag decomposition class using a surveyor's pin at ground level (0–2.5 m) and related these to the same observations made for logs: snag class 1 had characteristics of undecomposed tanoak, snag class

2 was comparable to class 1 logs, snag classes 3 and 4 were comparable to class 2 logs and class 5 was comparable to class 3 logs. We assumed that snag wood density values were equivalent to the wood density measured in the respective log decomposition class (Table 1). Log volume was calculated using Huber's, Smalian's, and frustum of cone equations which resulted in similar volume estimates ($r^2 > 0.99$ for each pair). We selected the volume equation for each log separately to reduce bias: when the proportional difference of log diameter measurements was less than 30% we used Smalian's equation, Huber's between 30 and 60%, and frustum of cone equations for those greater than 60%. Smalian's formula was mostly used in short pieces of CWD, Huber's for long pieces, and the frustum of a cone only occasionally. Snag and log mass was calculated by multiplying volume by mean wood density for the respective decomposition class.

We developed allometric biomass equations for tanoak using height–diameter relationships and initial tanoak wood density. These equations were used to estimate pre-disease standing tanoak biomass and were validated against estimates from general equations for mixed hardwoods developed by Jenkins and others (2004). We used initial tanoak biomass to derive an estimate of wood movement from the canopy to the forest floor which we term CWD-flux. Our calculation of CWD-flux differs from 'fragmentation', a more common term in the woody debris literature (see Harmon and others 1986). Here:

$$\text{CWD-flux} = \text{Initial Tanoak Mass} - \text{Standing Tanoak Mass}$$

where Initial Tanoak Mass is calculated using our allometric equations and Standing Tanoak Mass integrates decomposition class and snag height (described above). The equation does not correct for snag decomposition, thus CWD-flux is an index of wood movement rather than an estimate of the actual amount. Although anecdotal accounts suggest *P. ramorum* was established in our study plots by 1998, the exact dates are unknown (see Maloney and others 2005; Cobb and others 2010). Given this uncertainty, we calculate the CWD-flux in terms of Mg ha^{-1} rather than a rate (for example, Mg ha^{-1} y^{-1}).

Decomposition Rate Estimates

Wood decomposition rate constants (k y^{-1}) were estimated as proportional changes in wood density over time by fitting a non-linear first-order exponential decomposition model following principles outlined by Adair and others (2010). Snag and log decomposition rates were estimated only when the mortality date of the individual log or snag was documented during annual surveys (2002–2007; 68 logs and 321 snags). Wood density was assumed to be the mean value measured for each decomposition class except when wood density was measured directly (7 of 68 logs and 0 of 321 snags). Most log decomposition rates are based on measurements at Jack London State Park site where mortality dates were better resolved (83% of samples) but snag decomposition times were acquired more evenly across sites.

Data Analysis

We calculated first order, second moment error propagation to quantify uncertainty arising from error in height and wood density measurements used to derive snag biomass. We used the Gaussian

Table 1. Wood Density (g cm^{-3}) of the Most Frequent Species Contributing to Coarse Woody Debris (CWD) in Redwood Forests Impacted by Sudden Oak Death

Species	Wood decomposition class					*N*
	1	2	3	4	5	
Tanoak	0.51 ± 0.03^{A}	0.48 ± 0.03^{A}	0.38 ± 0.02^{B}	0.24 ± 0.02^{C}	0.24 ± 0.03^{C}	99
Redwood	nm	0.42 ± 0.04^{A}	0.37 ± 0.03^{AB}	0.33 ± 0.03^{AB}	0.26 ± 0.04^{B}	50
Bay laurel	nm	0.47 ± 0.05^{A}	0.40 ± 0.05^{AB}	0.22 ± 0.03^{C}	0.22 ± 0.06^{BC}	30
Madrone	nm	0.56 ± 0.04^{A}	0.50 ± 0.03^{A}	0.28 ± 0.03^{B}	nm	25
Coast live oak	nm	0.53 ± 0.05^{A}	0.40 ± 0.03^{AB}	0.30 ± 0.04^{B}	nm	30

Species are ranked according to the relative frequency at which they were encountered. N indicates the total number of logs sampled. Letters indicate statistically significant differences ($P < 0.05$) among decomposition class within individual species from a Tukey's HSD means comparison test. Data are least squares means with one standard error. See text for species Latin names.
nm wood density not measured due to low frequency of the species in the respective decomposition class.

error propagation method with the assumption that snag height and wood density are uncorrelated. The explicit form of the error estimates in addition to an analysis of uncertainty at the individual snag and plot level can be found in the Supplemental materials.

Wood density measurements were examined for covariance with decomposition class, log size, site, and log length. Decomposition class was the sole significant factor influencing wood density across species, thus other variables were dropped and least squares means and variances were calculated with a linear model of wood density as the dependent variable and decomposition class (ordinal) as the independent variable. Differences in decomposition class were examined with Tukey's honestly significant difference (HSD) means comparison when overall effects were significant. Mean values were used even when wood density did not differ among decomposition classes to facilitate error analysis and comparisons of our data to other five-class CWD schemes.

Two sets of linear models were constructed to examine the role of tanoak biomass and community level pathogen prevalence on CWD amounts and input rates. Plot level log, snag, and CWD-flux masses were examined with a series of mixed models that parameterized study site as a random variable and stand characteristics of interest as fixed variables. Rates of CWD generation were quantified on a per-tree basis with general linear models (GLM) using the glm() command in R (R Development Core Team 2008). In these models, recruitment into snags or logs (via tree/snag fall) was the binomially distributed dependent variable linked to the linear predictor variables via the logit function. In these models, newly generated snags are trees which died and remained standing whereas newly generated logs were created by tree and snag fall. It is important to recognize that our GLM models do not describe tanoak mortality per se but that recruitment of snags and logs are each components of a larger mortality process. Tree/snag fall was documented as part of annual surveys, but CWD-flux was measured only during the final census. Therefore, we estimate rates of snag generation and tree/snag fall but not CWD-flux because our data have better temporal resolution for these two processes. GLM model adequacy was assessed with receiver operator characteristic (ROC) values where ROC > 0.70 is considered acceptable model performance. Response and independent variables were transformed when necessary to address heteroscedastic variance and normal error distribution in the residuals. We used a critical α value of $P \leq 0.05$ for all statistical analyses.

Results

Wood Density and Biomass Uncertainty

Initial tanoak wood density averaged 0.598 g cm^{-3} with $\pm$ 0.021 standard error ($N = 8$). Wood density declined with advancing decomposition class in all species (Table 1). Redwood, bay laurel, pacific madrone (*Arbutus menziesii*), and coast live oak (*Quercus agrifolia*) were not encountered in sufficient frequency to estimate wood density in all decomposition classes for each respective species. Tanoak was the most frequent component of CWD pools. We found close agreement between our allometrically derived initial biomass estimates and those using equations from Jenkins and others (2004). Differences between our allometric models of tanoak biomass and those from Jenkins and others were predominantly due to differences in initial wood density. Error analysis showed that uncertainty in initial biomass and snag biomass increased with tree/snag height but never exceeded 11.3% of the total for any particular tree or snag. This resulted in plot level uncertainty of 5.7 and 8.6% for snag and initial biomass, respectively (see Supplemental materials for details).

Amounts and Variation in Woody Debris

Considerable variation in the amount of tanoak CWD occurred among and within sites. Although snag, log, and CWD-flux masses were 22.4, 11.5, and 20.0 Mg ha^{-1} variances for each of these measurements were much greater. Standard deviations of snag, log, and CWD-flux masses were 42.1, 16.3, and 44.4 Mg ha^{-1}, respectively. Across sites, snags dominated tanoak CWD amounts (Figures 2, 3). Within sites the distribution of tanoak CWD biomass was highly variable; in some sites CWD was concentrated in less decomposed snags (Figure 2; Partington Canyon) whereas at other sites snag and log masses were comparable and more evenly distributed across decomposition classes (Figure 2; Jack London). At the pathogen-free Big Basin site, the majority of tanoak biomass was in live trees (Figure 3) and overall CWD levels were much lower compared to sites invaded by *P. ramorum*. Amounts of tanoak CWD increased with the amount of dead tanoak basal area and the prevalence of infected bay laurel (Table 2). CWD-flux was negatively associated with snag mass and positively associated with log mass. Overall, amounts of CWD at the time of the survey were

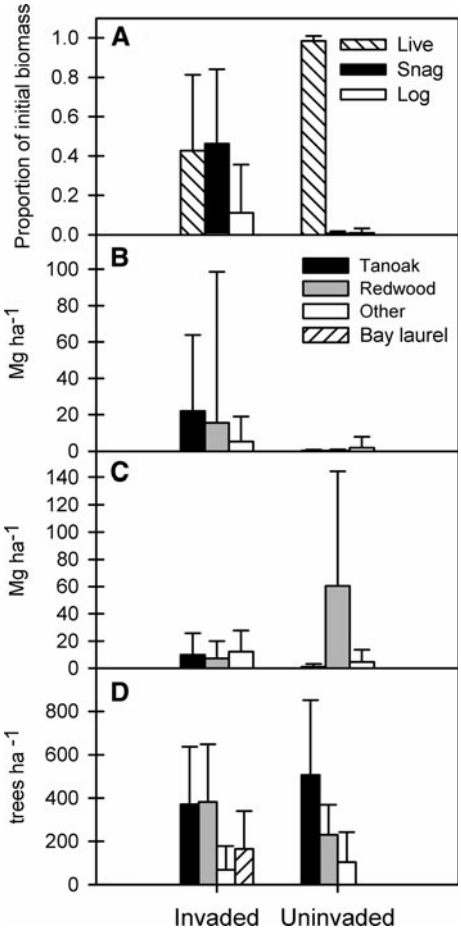

Figure 3. Distribution of tanoak biomass among live, snag, and log pools (**A**), snag and log mass for all species (**B**, **C**, respectively), and tree density (**D**) for *P. ramorum* invaded and uninvaded study plots. Bay laurel woody debris was included in the ''Other'' species category in **B** and **C**. Data are means with one standard deviation.

more strongly influenced by the amount of dead tanoak than pathogen prevalence in tanoak and bay laurel (Table 2). TMI did not significantly influence CWD amounts.

In some plots redwood CWD exceeded that of tanoak, or tanoak combined with all other species (Figure 3). Although tanoak stem densities are comparable to redwood in our study plots, redwood biomass greatly exceeds tanoak in most cases (data not shown). Occasional large dead redwood trees were an important but inconsistent part of woody debris in these forests. For example, one 300 cm dbh snag at Henry Cowell State Park, a *P. ramorum* invaded site, had an estimated mass of 41.2 Mg which resulted in very large amounts when expanded to a ha^{-1} basis. The mortality of this individual was not associated with sudden oak death. Differences in redwood CWD between sites were largely determined by the presence of large redwood snags or logs that predate *P. ramorum* invasion.

Table 2. Linear Mixed Models Relating CWD Amounts (Mg ha^{-1}) Surveyed in 2007 and 2008 with Prevalence of Infected Hosts, Edaphic Characteristics, and Tanoak Biomass

Model Parameter	Coefficient	r^2 *P* value
CWD-flux‡		*0.54*
Intercept	ns	0.96
Dead tanoak m^2 ha^{-1}	0.36 (0.02)	<0.001
TMI†	ns	0.17
Bay laurel ha^{-1}	4.07×10^{-3} (1.36×10^{-3})	0.004
Tanoak ha^{-1}	ns	0.38
Snag mass‡		*0.85*
Intercept	ns	0.71
CWD-flux‡	−0.51 (0.08)	<0.001
Dead tanoak m^2 ha^{-1}	0.49 (0.04)	<0.001
TMI†	ns	0.60
Bay laurel ha^{-1}	ns	0.72
Tanoak ha^{-1}	ns	0.72
Log mass‡		*0.41*
Intercept	ns	0.79
CWD-flux‡	0.31 (0.15)	0.046
Dead tanoak m^2 ha^{-1}	ns	0.96
TMI†	ns	0.48
Bay laurel ha^{-1}	ns	0.96
Tanoak ha^{-1}	ns	0.14

Study site was parameterized as a random variable. Bay laurel and tanoak variables are the tally of individuals with infections confirmed by isolation of P. ramorum from symptomatic tissue. CWD-flux is an index of the biomass which had transitioned to the forest floor via tree/snag fall and snag fragmentation (see text). Coefficients and standard errors (in parentheses) were reported when covariates were statistically significant ($P < 0.05$). r^2 values are in italics.
ns parameter not statistically significant.
‡*Data were square root transformed.*
†*Topographic moisture index (TMI) is a unitless index describing plot-level topographically controlled moisture status (see text).*

Tanoak CWD Accumulation and Decomposition Rates

Snag and log recruitment rates were influenced by individual tree size, community level pathogen prevalence, and site factors (Table 3). Most log recruitment via tree/snag fall was from previously killed trees; 62% of logs originated from trees which were dead during the initial survey in 2002. Larger tanoak trees were significantly more likely to be recruited into snags compared to small trees but tree size decreased the probability of tree/snag fall (Table 3). These patterns suggest that large trees die more rapidly following infection but remain standing

Table 3. General Linear Models of CWD Input Rates from Recruitment of Snags and Generation of Logs via Tree/Snag Fall in Redwood Forests Impacted by Sudden Oak Death

Model Parameter	Coefficient	ROC *P* value
Snag recruitment		*0.749*
Intercept	−3.28 (0.28)	<0.001
dbh cm	0.043 (0.004)	<0.001
Bay laurel ha^{-1}‡	0.026 (0.012)	0.027
Tanoak ha^{-1}‡	0.056 (0.011)	<0.001
Redwood ha^{-1}‡	ns	0.15
TMI†	ns	0.12
Site	1.66:0	<0.001
Tree/snag fall		*0.741*
Intercept	−3.22 (0.37)	<0.001
dbh cm	−0.021 (0.005)	<0.001
Bay laurel ha^{-1}‡	0.073 (0.012)	<0.001
Tanoak ha^{-1}‡	ns	0.73
Redwood ha^{-1}‡	−0.049 (0.012)	<0.001
TMI†	0.076 (0.03)	0.011
Site	2.56:0	<0.001

Bay laurel and tanoak variables are the tally of these species with infections confirmed by isolation of P. ramorum from symptomatic tissue. Standard errors for parameter estimates appear in parentheses when coefficients were statistically significant ($P < 0.05$). Values for site variables are presented as a range. ROC values are in italics.
‡*Data were square root transformed.*
†*Topographic moisture index (TMI) is a unitless index describing topographically controlled plot moisture status (see text).*

longer following mortality. This pattern may explain why CWD amounts were greater in snags compared to logs during the field survey (Figure 3). Prevalence of infection in bay laurel increased input rates of CWD both for snags and logs whereas prevalence of infected tanoak increased input rates for snags but had no significant effect on accumulation of logs. Redwood density had no significant effect on snag generation but was negatively associated with tree/snag fall (Table 3). TMI was negatively associated with snag recruitment rate and positively associated with tree/snag fall suggesting that local soil and topographical factors are important in determining the rate at which trees and snags transition to the forest floor.

Our non-linear model estimates of k constants were 0.067 y^{-1} (±0.005) for logs and 0.04 y^{-1} for snags (±0.001 std error) suggesting that tanoak log decomposition rates are higher than snag decomposition rates. These models explained relatively low proportions of the variance (r^2 = 0.12 and 0.08 for logs and snags, respectively) likely due to assumed wood density values rather than direct measurements. A reassessment of decomposition rates from repeated measurement of mapped logs and snags (surveyed in 2007 and 2010) at the Jack London site yielded different rate estimates: k = 0.092 y^{-1} ± 0.018 for logs and k = 0.01 y^{-1} ± 0.004 (std error) for snags.

Discussion

We have shown that sudden oak death increases CWD in *P. ramorum* invaded redwood forests and that the combined influences of epidemiological drivers and pre-disease host biomass determine the amount and rate at which these materials are generated. In Figure 4, we show example study plots which reflect the differential effects that epidemiological drivers and host biomass have on forest woody debris. CWD amounts can be different than the rates at which they are generated by sudden oak death; specifically, conditions which lead to the most rapid tanoak mortality are not equivalent to conditions which lead to the greatest amounts of CWD (Figure 4). We expect this pattern is general to other forest diseases caused by generalist pathogens, especially where transmission is different among hosts. In our example, low density tanoak growing with high densities of bay laurel have the most rapid rate of mortality and CWD generation, but stands with high biomass of tanoak will lead to the greatest amounts of CWD even when the epidemiological characteristics of the stand lead to slower mortality rates (Figure 4). For sudden oak death, amounts and rates of CWD generated can be reasonably predicted with measurements of tanoak density, bay laurel density, and tanoak tree sizes which are relatively easy to acquire (Tables 2, 3).

The mean amount of tanoak CWD in stands impacted by sudden oak death was 34.0 Mg ha^{-1}, or a 32.5 Mg ha^{-1} increase compared to the pathogen free stand. This amount is lower than masses generated by temporally discrete events such as fire, forest harvest, and herbicide application (Spies and others 1988; Tinker and Knight 2000; Valachovic and others 2011). Additionally, CWD masses were much lower than total aboveground biomass of these forests (Sillett and others 2010). Given that only about 50% of total tanoak biomass had been killed by the 2007 survey, it is likely that woody debris will continue to accumulate and that 10 years after the onset of disease CWD levels may not have reached maximum amounts. Our results do not challenge the validity of discrete disturbance models of CWD accumulation (Harmon and others 1986; Spies and others 1988; Harmon 2009) which yield insight into the frequent and widespread ecosystem impacts of fire and harvest. Rather when these models are applied to spatially and temporally

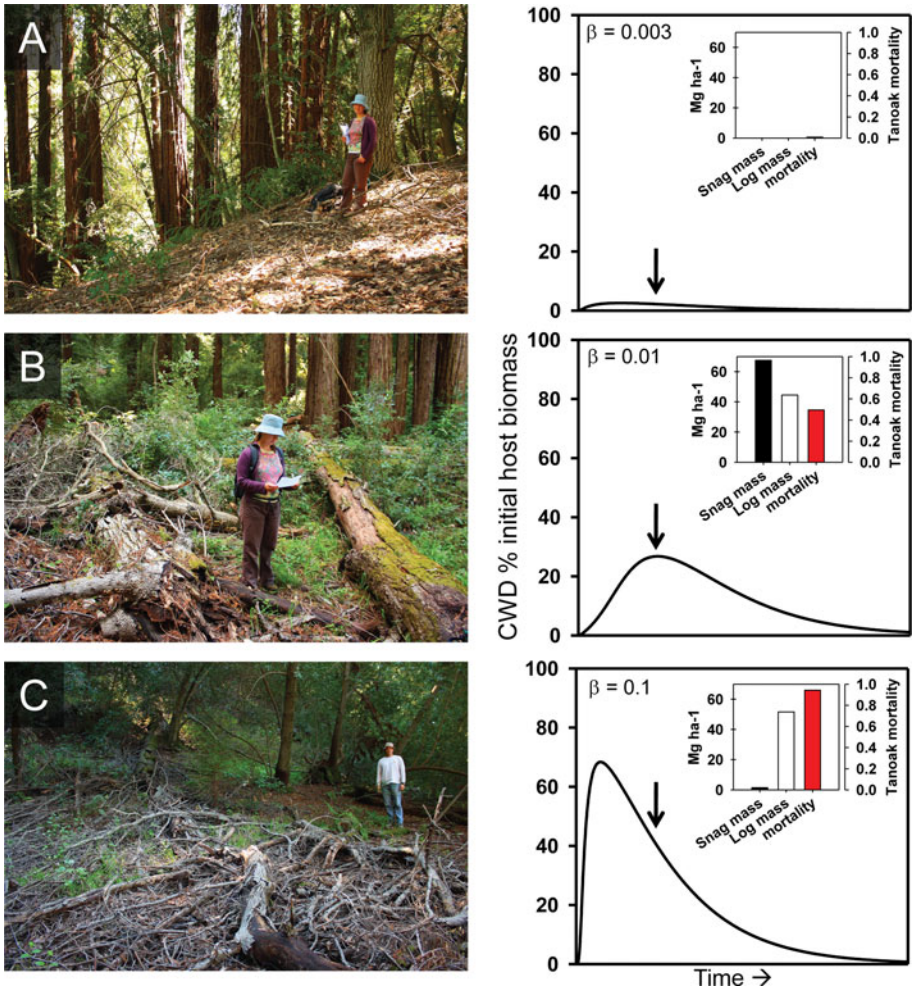

Figure 4. Example study plots illustrating the disparity between rates and amounts of coarse woody debris (CWD) generated by the forest pathogen *Phytophthora ramorum* in coast redwood forests. All photos are from the Jack London State Park study site (Glen Ellen, CA) taken at the plot boundary facing the center. Each photo is presented with a representative outcome of the conceptual model (Figure 1) with the infection rate parameter (β) set at low, moderate, and high rates, respectively. Values of β were chosen based on the local density of infected tanoak and bay laurel but not tanoak biomass. Disease progression is presented with the measured amount of snag mass, log mass, and the proportion of tanoak basal area killed by the pathogen (in *red*) in the *inset*. In Plot 7 (**A**), epidemiological characteristics are poor for disease emergence and CWD amounts are low. In Plot 27 (**B**), the pathogen has generated substantial amounts of CWD but moderate mortality rates (50% tanoak basal area killed) suggest CWD amounts have not reached maximum. In plot 18 (**C**), the pathogen rapidly killed most tanoak biomass (95% basal area killed) but total CWD amounts are lower compared to plots with higher initial tanoak biomass (for example, **B**). *Arrows* in each panel indicate an inferred state of disease progression from the amount of observed mortality. *P. ramorum* presence has been confirmed in each of these study plots continuously since 2002 (Maloney and others 2005).

variable pathogen outbreak our results show that integration of epidemiological processes, particularly factors which determine transmission and host biomass (c.f. Lovett and others 2006) will yield more realistic estimates of CWD accumulation rate and maximum amounts.

Tree mortality over years or decades will slow CWD accumulation compared to temporally discrete disturbances (Figure 1; Lambert and others 1980), especially for pathogens which cause selective species removal or when mortality rates differ among individuals. Variation in mortality rates is likely to diversify CWD among decomposition classes because of differences in the length of time individual pieces of wood have decomposed; this effect of outbreak was documented in stands impacted by spruce budworm (*Choristoneura fumiferana*), mortality caused by the heart-rot pathogen *Phellinus chrysoloma*, and beech bark disease (Sturtevant and others 1997; Edman and others 2007;

D'Amato and others 2008). In contrast to periodic outbreaks such as spruce budworm, *P. ramorum* is persistent within impacted stands, mortality is constant or increasing over the scale of a decade, and inter-annual climate variability is critical to rates of pathogen spread (Davidson and others 2008; Davis and others 2010; Cobb and others 2010). This temporal variability may extend mortality and CWD accumulation from *P. ramorum* over a greater period compared to other pathogens especially those causing root disease which expand or spread from discrete contact zones (Hansen and Goheen 2000; Rizzo and others 2000; Kauffman and Jules 2006).

P. ramorum transmission and its influence on CWD accumulation rate was most strongly influenced by the prevalence of infection in bay laurel and secondly by prevalence in tanoak (Table 3). Although the importance of tanoak and bay laurel on *P. ramorum* spread among sites, tanoak infection rate, and tanoak mortality are well documented (Meentemeyer and others 2008a; Cobb and others 2010; Meentemeyer and others 2011) this study demonstrates that the same epidemiological mechanisms and drivers determine changes to an important ecosystem level pool of organic matter. Following infection, mortality rate increases with tree size (McPherson and others 2010) indicating that CWD generation rates will be influenced by tanoak size-distribution. When aboveground biomass is concentrated in large diameter tanoak and local densities of bay laurel are high, rates of CWD accumulation may be rapid enough to approximate discrete disturbances; tanoak biomass, prevalence of infected bay laurel, and patterns of snag accumulation at the Partington Canyon site fit this expectation (Figure 2). Although sudden oak death increased overall CWD amounts, that the disease lead to a range of CWD input rates and amounts is an important insight into how ecosystems are altered by pathogens (Figure 4). At times these rates and amounts were comparable to discrete outbreaks such as spruce budworm but frequently the rates are slower with few comparable examples in the literature of CWD dynamics (Figure 4). Although our study is not the first to model CWD dynamics from forest pathogens (Edman and others 2007), it may be the first to document changes in CWD following invasion of an invasive pathogen and serves as an important comparison to outbreak of other native and exotic pathogens. Pathogen transmission from sporulation supporting species is a critical driver of sudden oak death impacts (Tables 2, 3). To develop CWD models for other forest disease outbreaks, researchers and managers need to identify the central epidemiological mechanisms driving patterns of disease severity such as contact structure, susceptibility, and rates of mortality following infection (Hansen and Goheen 2000; Rizzo and others 2000; Kauffman and Jules 2006).

Our estimates of k values are within the range of reported values for Mediterranean type ecosystems and expected values from global models of wood decomposition (0.03–0.07; Harmon and others 1986; Chambers and others 2000). Given the low amount of variation explained by our models, our survey should be viewed as an initial attempt to estimate tanoak wood decomposition rates. The clearest pattern in our measurements of wood decomposition was that rates were faster in logs compared to snags. Snag decomposition rates are frequently lower compared to logs in dry-climate forests where moisture limitation of decomposition is greater in standing woody debris (Harmon 2009). Chronosequence type measurements, such as our study-wide estimates of k, lack the precision of repeated measurements of wood decomposition. Although we also made repeated measurement of snags and logs at Jack London State Park, these data are over a relatively short 3-year period and integrate underlying edaphic variation from only a single site. These limitations probably underlie the discrepancy between measured k y^{-1} values. Both the repeated measurements of tanoak logs at Jack London, as well as the study-wide log decomposition estimates suggest tanoak decomposition is fairly rapid but controlled measurements in the field are needed to estimate the parameter values with a higher degree of confidence. This study suggests factors controlling tree fall will influence rates of tanoak woody debris decomposition and duration of elevated CWD levels in forests impacted by sudden oak death. Adaptation of CWD models to other forest diseases needs to consider tree/snag fall rates, especially when wood decomposition rates differ greatly between snags and logs. However, pathogens can have very different influences on tree/snag fall rates, even for pathogens which kill trees via root infection (compare Hansen and Goheen 2000; Rizzo and others 2000; Kauffman and Jules 2006).

In this study bay laurel densities were positively associated with tree/snag fall rates (Table 3), but the underlying epidemiological mechanism for this pattern is less clear. This pattern may simply reflect underlying edaphic effects on fall rates which increased with our measure of edaphically mediated moisture availability (TMI; Table 3). However, bay laurel densities and basal area were not correlated with TMI (data not shown) suggesting that

any effects of bay laurel on snag fall are not confounded with edaphic factors. McPherson and others (2010) found that tanoak mortality and treefall rates were positively associated with the expression of symptoms from secondary pathogens and pests, such as *Hypoxylon thouarsianum* and ambrosia beetles (Coleoptera, Curculionidae). *H. thouarsianum* is a weak pathogen which directly attacks sapwood, begins rapid growth while tanoak trees are in decline, and is very common on *P. ramorum* infected trees (Rizzo and others 2005). Disruption of sapwood function is known to increase decomposition rates by sapwood inhabiting fungi (Boddy and Rayner 1983) and given that *P. ramorum* directly interferes with sapwood function (Parke and others 2007), increasing prevalence of *P. ramorum* in bay laurel may simply be correlated with increased tanoak sapwood decomposition and tree fall because of more rapid attack by secondary pathogens and insects in disease impacted stands (McPherson and others 2010).

Tanoak was the dominant species accounting for CWD mass across coast redwood forests invaded by *P. ramorum* but second-growth redwood forests often have moderate levels of canopy diversity in the central California study area and species such as Douglas-fir (*Pseudotsuga menziesii*), Pacific madrone, and coast live oak were also significant contributors to the CWD pool (see other species; Figure 3). Large amounts of wood accumulation in individual redwood trees are an important mechanism of CWD accumulation in these forests (Sillett and others 2010). Large individual snags and logs can lead to heterogeneous but large pools of CWD, especially in old-growth forests with infrequent or localized canopy disturbance (D'Amato and others 2008; Schlegel and Donoso 2008). With one exception, our study sites are second growth redwood forests and had much lower ($\sim$58 Mg ha^{-1} CWD) mass compared to 262 Mg ha^{-1} documented by Busing and Fujimori (2005) in old-growth redwood forests. However, we also measured large levels of variation in redwood CWD which reflect the importance of occasional large redwood snags and logs on overall CWD amounts and patterns of variation (Figure 3). There is no evidence that sudden oak death will increase redwood mortality (Davidson and others 2008; Cobb and others 2010), therefore we expect CWD levels of redwood and other species reported here are representative of second growth redwood forests. In stands with low tanoak biomass or bay laurel abundance, sudden oak death may not significantly increase CWD, especially in older redwood forests where woody debris pools may be concentrated in large slowly decaying redwood snags and logs (Busing and Fujimori 2005; Sillett and others 2010).

The influence of epidemiological drivers on rates of CWD accumulation by sudden oak death illustrates the importance of integrating epidemiological processes into predictions of ecosystem level changes following disease outbreak (Lovett and others 2006; Eviner and Likens 2008). Our study also suggests that the accumulation of woody debris from forest disease can occur over the scale of years or decades, an important temporal difference of pathogen impacts compared to fire and forest harvest. Efforts to adapt CWD models to pathogen outbreak will benefit from integration of epidemiological drivers, especially transmission and susceptibility. Such efforts will increase the temporal and spatial predictive power of these models and consequently improve management applications to assess fire hazards, control disease, and manage wildlife habitat (Metz and others 2011; Swei and others 2011; Valachovic and others 2011). Our study confirms predictions that host specificity, virulence, and the importance (biomass or functional characteristics) of the most impacted species are central to determining the impacts of insect or pathogen outbreak on ecosystem processes (Lovett and others 2006; Eviner and Likens 2008). For generalist pathogens such as *P. ramorum*, community context and its impact on pathogen spread and rate of tree decline must also be considered to fully understand the patterns and ecosystem changes caused by emerging infectious disease.

ACKNOWLEDGMENTS

H. Mehl, S. Lynch, A. Westbrook, K. Ireland, C. DeLong, C. Shoemaker, and A. Oguchi provided field and laboratory support for this research. We thank two anonymous reviewers, Margaret Metz, Val Eviner, Gary Lovett, and Faith Fogel for helpful comments on earlier versions of this paper. Additionally, we are grateful to the California State Parks and the Marin Municipal Water District for granting access to the study sites. This research was funded by the NSF-NIH Ecology of Infectious Diseases program EF-0622770, the Gordon and Betty Moore Foundation, and the USDA Pacific Southwest Research Station.

REFERENCES

Adair EC, Hobbie SE, Hobbie RK. 2010. Single pool exponential decomposition models: potential pitfalls in their use in ecological studies. Ecology 91:1225–36.

Boddy L, Rayner ADM. 1983. Origins of decomposition in living deciduous trees: the role of moisture content and a

re-appraisal of the expanded concept of tree decomposition. New Phytol 94:623–41.

Burdon JJ, Thrall PH, Ericson L. 2006. The current and future dynamics of disease in plant communities. Annu Rev Phytopathol 44:19–39.

Busing RT, Fujimori T. 2005. Biomass, production and woody detritus in an old coast redwood (*Sequoia sempervirens*) forest. Plant Ecol 177:177–88.

Chambers JQ, Higuchi N, Schimel JP, Ferreira LV, Melack JM. 2000. Decomposition and carbon cycling of dead trees in tropical forests of the central Amazon. Oecologia 122:380–8.

Cobb RC, Meentemeyer RK, Rizzo DM. 2010. Apparent competition in canopy trees determined by transmission rather than susceptibility. Ecology 91:327–33.

Condeso TE, Meentemeyer RK. 2007. Effects of landscape heterogeneity on the emerging forest disease Sudden Oak Death. J Ecol 95:364–75.

D'Amato AW, Orwig DA, Foster DR. 2008. The influence of successional processes and disturbance on the structure of *Tsuga canadensis* forests. Ecol Appl 18:1182–99.

Davidson JM, Patterson H, Rizzo DM. 2008. Sources of inoculum for *Phytophthora ramorum* in a redwood forest. Phytopathology 98:860–6.

Davis FW, Borchert M, Meentemeyer RK, Flint A, Rizzo DM. 2010. Pre-impact forest composition and ongoing tree mortality associated with sudden oak death in the Big Sur region; California. For Ecol Manag 254:2342–54.

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 99:1307–11.

Edman M, Jönsson M, Gunnar Jonsson B. 2007. Fungi and wind strongly influence the temporal availability of logs in an old-growth spruce forest. Ecol Appl 17:482–90.

Ellison AM, Bank MS, Clinton BD et al. 2005. Loss of foundation species: consequences for the structure and dynamics of forested ecosystems. Front Ecol Environ 9:479–86.

Eviner VT, Likens GE. 2008. Effects of pathogens on terrestrial ecosystem function. In: Ostfeld RS, Keesing F, Eviner VT, Eds. Infectious disease ecology. Princeton, NJ: Princeton University Press.

Hansen EM, Goheen EM. 2000. *Phellinus weirii* and other native root pathogens as determinants of forest structure and process in western north America. Annu Rev Phytopathol 38:515–39.

Harmon ME. 2009. Woody detritus mass and its contribution to carbon dynamics of old-growth forest: the temporal context. In: Wirth C, Gleixner G, Heimann M, Eds. Old-growth forests: function, fate, value, ecological studies, Vol. 207. New York: Springer.

Harmon ME, Franklin JF, Swanson FJ et al. 1986. Ecology of coarse woody debris in temperate ecosystems. Adv Ecol Res 15:133–302.

Janisch JE, Harmon ME. 2002. Successional changes in live and dead wood carbon stores: implications for net ecosystem productivity. Tree Physiol 22:77–89.

Jenkins JC, Chojnacky DC, Heath LS, Birdsey RA. 2004. Comprehensive database of diameter-based biomass regressions for north American tree species. USDA-FS Gen Tec Report NE-319.

Kauffman MJ, Jules ES. 2006. Heterogeneity shapes invasion: host size and environment influence susceptibility to a non-native pathogen. Ecol Appl 16:166–75.

Lambert RL, Lang GE, Reiners WA. 1980. Loss of mass and chemical change in decaying boles of a subalpine balsam fir forest. Ecology 61:1460–73.

Lamsal S, Cobb RC, Cushman H, 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. For Ecol Manag 262:989–98.

Lang GE. 1985. Forest turnover and the dynamics of bole wood litter in subalpine balsam fir forest. Can J For Res 15:262–8.

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.

Maloney PE, Lynch S, Kane S, Jensen CE, Rizzo DM. 2005. Establishment of an emerging generalist pathogen in redwood forest communities. J Ecol 93:899–905.

Marchand PJ, Goulet FL, Harrington TC. 1986. Death by attrition: a hypothesis for wave mortality of subalpine *Abies balsamea*. Can J For Res 16:591–6.

Matson PA, Boone RD. 1984. Natural disturbance and nitrogen mineralization: wave-form dieback of mountain hemlock in the Oregon Cascades. Ecology 65:1511–16.

McPherson BA, Mori SR, Wood DL, Kelly M, Storer AJ, Svihra P, Standiford RB. 2010. Responses of oaks and tanoak to the sudden oak death pathogen after 8 y of monitoring in two coastal California forests. For Ecol Manag 259:2248–55.

Meentemeyer RK, Anacker BL, Mark W, Rizzo DM. 2008a. Early detection of emerging forest disease using dispersal estimation and ecological niche modeling. Ecol Appl 18:377–90.

Meentemeyer RK, Rank NE, Shoemaker D, Oneal C, Rizzo DM. 2008b. Impacts of sudden oak death on tree mortality in the Big Sur ecoregion of California. Biol Invasions 10:1243–55.

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.

Metz MR, Frangioso KM, Meentemeyer RK, Rizzo DM. 2011. Interacting disturbances: wildfire affected by stage of forest disease invasion. Ecol Appl 21:313–20.

Parke JL, Oh E, Voelker S, Hansen EM, Buckles G, Lachenbruch B. 2007. *Phytophthora ramorum* colonizes tanoak xylem and is associated with reduced stem water transport. Phytopathology 97:1558–67.

R Development Core Team. 2008. R: A language and environment for statistical computing. Vienna: R Foundation for Statistical Computing.

Rizzo DM, Slaughter GW, Parmeter JR. 2000. Enlargement of canopy gaps associated with a fungal pathogen in Yosemite Valley, California. Can J For Res 30:1501–10.

Rizzo DM, Garbelotto M, Hansen EM. 2005. *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. Annu Rev Phytopathol 43:309–35.

Ruess RW, McFarland JM, Trummer LM, Rohrs-Richey KR. 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.

Schlegel BC, Donoso PJ. 2008. Effects of forest type and stand structure on coarse woody debris in old-growth rainforests in the Valdivian Andes, south-central Chile. For Ecol Manag 255:1906–14.

Sillett SC, Van Pelt R, Koch GW, Ambrose AR, Carroll AL, Antoine ME, Mifsud BM. 2010. Increasing wood production through old age in tall trees. For Ecol Manag 259:976–94.

Spies TA, Franklin JF, Thomas TB. 1988. Coarse woody debris in Douglas-fir forests of western Oregon and Washington. Ecology 69:1689–702.

Sturtevant BR, Bissonette JA, Long JN, Roberts DW. 1997. Coarse woody debris as a function of age, stand structure, and disturbance in boreal Newfoundland. Ecol Appl 7:702–12.

Swei A, Ostfeld RS, Lane RS, Briggs CJ. 2011. Effects of an invasive forest pathogen on abundance of ticks and their vertebrate hosts in a California Lyme disease focus. Oecologia 166:91–100.

Tinker DB, Knight DH. 2000. Coarse woody debris following fire and logging in Wyoming lodgepole pine forests. Ecosystems 3:472–83.

Valachovic YS, Lee CA, Scanlon H, Varner JM, Glebocki R, Graham BD, Rizzo DM. 2011. Sudden oak death-caused changes to surface fuel loading and potential fire behavior in Douglas-fir-tanoak forests. For Ecol Manag 261:1973–86.