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Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility

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Abstract. Epidemiological theory predicts that asymmetric transmission, susceptibility, and mortality within a community will drive pathogen and disease dynamics. These epidemiological asymmetries can result in apparent competition, where a highly infectious host reduces the abundance of less infectious or more susceptible members in a community via a shared pathogen. We show that the exotic pathogen *Phytophthora ramorum* and resulting disease, sudden oak death, cause apparent competition among canopy trees and that transmission differences among canopy trees drives patterns of disease severity in California coast redwood forests. *P. ramorum* ranges in its ability to infect, sporulate on, and cause mortality of infected hosts. A path analysis showed that the most prolific inoculum producer, California bay laurel (*Umbellularia californica*), had a greater impact on the mortality rate of tanoak (*Lithocarpus densiflorus*) than did other inoculum-supporting species. In stands experiencing high tanoak mortality, lack of negative impacts by *P. ramorum* on bay laurel may increase bay laurel density and subsequently result in positive feedback on pathogen populations. This study demonstrates the degree to which invasive, generalist pathogens can cause rapid changes in forest canopy composition and that differences in transmission can be more important than susceptibility in driving patterns of apparent competition.

Key words: apparent competition; community epidemiology; disease ecology; emerging infectious disease; forest pathogens; Lithocarpus densiflorus; Phytophthora ramorum; Quercus *spp.;* Sequoia sempervirens; *sudden oak death;* Umbellularia californica.

Introduction

Plant epidemiological theory predicts that generalist pathogens with asymmetric host impacts will alter plant communities by selectively removing the most susceptible species and by subsequently increasing the densities of the most infectious and/or tolerant species (Alexander and Holt 1998, Gilbert 2002, Burdon et al. 2006). These asymmetries provide a pathway for apparent competition mediated by generalist pathogens and shape communities by reversing or restructuring competitive relationships among species that may not otherwise strongly interact. Several investigators have demonstrated the importance of epidemiological asymmetries in driving apparent competition through theoretical approaches and field studies of diverse taxa (Holt 1977, Begon et al. 1992, Brown and Hastings 2003, Power and Mitchell 2004). Apparent competition is likely a general ecological phenomenon; however, predictions of pathogen-mediated community change often utilize the simplifying assumption that relative susceptibility, the likelihood of pathogen establishment within a host, is an appropriate indicator of transmission, the likelihood of pathogen spread from an infected host to an uninfected host (Holt 1977, Begon et al. 1992). This assumption may be inappropriate for many generalist pathogens because asymmetric host impacts influence host population dynamics, and transmission rate may change with altered community structure (Begon et al. 1992, Otten et al. 2005).

Controlled studies have demonstrated apparent competition as a mechanism for pathogen intensification with asymmetric host impacts and reservoir capacity as major drivers of disease dynamics (Power and Mitchell 2004; see also Borer et al. 2007). Generalist pathogens are likely to cause changes in forest community structure by infecting and removing the most susceptible hosts, but within a diverse host community, susceptibility to infection, host impacts, and infectiousness are likely to vary widely. The combination of pathogen tolerance and greater interspecific transmission by one species can reduce the abundance of a less tolerant neighbor (Holt 1977). Because epidemiological characteristics of generalist pathogens may be highly variable across hosts, different patterns of disease expression are likely to emerge across community composition, and disease in complex communities may result in interactive epidemiological processes. Specifically, long-term shifts within host communities can have positive or negative impacts

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on pathogen prevalence depending on how the overall susceptibility, reservoir capacity, or infectiousness of host composition is structured in a post-disease community (Ostfeld and Keesing 2000, Gilligan and van den Bosch 2008). Given these complexities, simple assumptions such as transmission–susceptibility equivalence may lead to highly erroneous predictions of disease impacts within heterogeneous communities.

The emerging plant disease, sudden oak death (SOD), has caused extensive mortality of oaks (*Quercus* spp.) and tanoak (*Lithocarpus densiflorus*) in coastal forests in California and Oregon since the mid-1990s (Rizzo et al. 2005). The disease is caused by *Phytophthora ramorum*, a generalist pathogen that also infects over 40 plant genera beyond oak. The diseases that this pathogen causes on this wide range of hosts are expressed in two ways: canker infections that may cause tree mortality and nonlethal foliar and twig infections (Rizzo et al. 2005). Transmission of the pathogen is primarily driven by spore production from foliar or twig infections rather than from lethal cankers on the main stem of trees (Davidson et al. 2005, Rizzo et al. 2005). In California redwood forests, bay laurel (*Umbellularia californica*) is an especially important foliar host of *P. ramorum* because it produces large amounts of inoculum in the form of sporangia from necrotic leaf spots (Davidson et al. 2005, 2008). Stand- and landscape-level surveys have demonstrated increased likelihood of pathogen establishment and disease in forests where bay laurel is present (e.g., Maloney et al. 2005, Meentemeyer et al. 2008*a*, *b*). Infection of redwood (*Sequoia sempervirens*) is much lower and results in substantially less sporulation from infected needles (Davidson et al. 2008). Regardless of susceptibility, neither species appears to suffer mortality following infection by *P. ramorum*. In contrast, tanoak is highly susceptible to stem infection and diseased trees often die within five years. Tanoak supports sporulation on twig cankers at an order of magnitude lower than bay laurel (Davidson et al. 2008).

Rizzo and Garbelotto (2003) suggested that asymmetric transmission and differential impacts between tanoak and bay laurel would result in apparent competition between the two species and pathogen populations could be magnified over time if SOD led to increased bay laurel populations. We tested the hypothesis that apparent competition is responsible for patterns of disease severity in coast redwood forests invaded by *P. ramorum*. We evaluated a 10-year data set of infection and mortality for effects of apparent competition mediated by *P. ramorum* with the expectation that disease severity patters would follow the relative likelihood of pathogen establishment within hosts (susceptibility) or likelihood of pathogen spread among infected hosts (transmission). We use species-specific infection and mortality rate as measures of susceptibility and host impact by the disease, respectively. We estimated pre-disease stand conditions, infection rate, and mortality rates over a five-year period of pathogen establishment and a five-year period immediately following when the disease intensified. If differences in transmission dominate patterns of disease severity, the impact of infection in bay laurel would be greater than the impact of infection in tanoak or redwood in accordance with differences in sporulation.

Study Area

Phytophthora ramorum has established across a contiguous zone of costal redwood forests from southern Mendocino County to southern Monterey County California, USA, with a patchy distribution at finer spatial scales (Meentemeyer et al. 2008*a*). Study plots were located across 14 sites that span the geographic extent of the disease and variation in host community composition (Maloney et al. 2005). The study sites are located in the southern and central distribution of redwood forests and were established within 75 km of initial *P. ramorum* introduction locations in coastal California (Mascheretti et al. 2008). All sites, except an old-growth stand at Henry Cowell State Park, have had at least moderate levels of harvesting of redwood in the past 150 years. Within redwood forests at each site, between 6 and 15 500-m^2 circular plots were randomly established at intervals $>$100 m. Sudden oak death was first observed in many of our study locations in 1998, and we use this date as a standard estimate of disease establishment, although the actual dates likely range from 1995 to 2000. The Big Basin State Park (Santa Cruz County) remained *P. ramorum* free for the duration of the study, which provided baseline mortality estimates for redwood and tanoak. Big Basin State Park does not differ from other study sites in any substantial way (e.g., soils, tree size distribution, logging history) except that bay laurel is not present within 100 m of study plots at this site.

Field Measurements

Plots were established in 2001 and all stems $\geq$1 cm in diameter at breast height (dbh) were assessed for size (dbh), crown position, and pathogen infection. Field measurements included all overstory trees and much of the understory tree and shrub ecosystem components. Symptoms of *P. ramorum* were recorded for each individual, and symptomatic tissue was removed and brought to the laboratory for pathogen isolation using a *Phytophthora*-selective media (PARP; Davidson et al. 2005). Annually for each species, five randomly selected trees with previously confirmed infections and five uninfected trees were reassessed for infection and mortality. We conducted a full census of the study plots from July to October 2007. Mortality was assessed by visiting all trees in all plots and infection was quantified in all plots visited until the end of July (first 60 plots) and then discontinued to avoid biased assessments due to poor pathogen recoveries during the fall when the pathogen is difficult to isolate. Infection rates for the

remaining 60 plots were estimated from all confirmed infections between 2002 and 2006.

Data Analysis

We reconstructed pre-disease stand conditions by assuming all tanoak stems with evidence of *P. ramorum* stem cankers were living before 1998. Because the period of measurement varied slightly across plots and parameters, we calculated pathogen infection and mortality rates on a per capita basis and temporally standardized the estimates according to the survey procedures. These rates were angular transformed (Krebs 1999):

$$p' = 1 - \frac{1}{2}\left[\arcsin\sqrt{\left(\frac{S_{ti}}{1+N_t}\right)^{t/t_i}} + \arcsin\sqrt{\left(\frac{1+S_{ti}}{1+N_t}\right)^{t/t_i}}\right]$$

where p' is the per capita infection or death rate in degrees, N_t is the initial susceptible population size, S_{ti} is the number of individuals surviving to time t_i, and $t = 1$ year. We back-transformed p' values into proportions using $p = (\sin p')^2$ after converting p' into radians. Per capita rates of infection and mortality were calculated separately for each species within the pathogen establishment (1998–2002) and disease intensification periods (2003–2007). While this approach does not provide as much insight into temporal dynamics afforded by traditional disease progress curves, it is highly appropriate in light of survey frequency and data collection procedures. To test if per capita infection and mortality rates differed among species within plots, we used t tests for paired data employing the analysis platform JMP (SAS 2007).

We constructed a confirmatory path analysis based on current understanding of *Phytophthora ramorum* biology. This technique was selected because it is appropriate to multi-level structural hypothesizes and has been successfully implemented with host–parasite systems (Thomas et al. 2007). We tested for impacts of pathogen prevalence in the three main overstory species on tanoak mortality rate and feedback on mortality rates of bay laurel and redwood. We accounted for the hierarchical structure of our data set by constructing mixed models with random temporal and spatial (site) effects and then assessed pathogen prevalence in overstory host species and mortality feedback as fixed effects using the nlme package in R (R Development Core Team 2008). These models follow the general form

$$p' = \sum_{i=1}^{n} \delta_i \beta_{it} x_i + (1|\text{time}) + (1|\text{site}) + \varepsilon$$

where the intercepts vary across time and sites. Mortality rate (p') is a function of the prevalence of infected individuals of species i at time t ($\beta_{it}x_i$) parameters δ_i, and a normally distributed error term ε. Following the general method of Shipley (2009), we fit one confirmatory path model and assessed model fit utilizing direct separation tests ("d-sep") of implied conditional independence of driving variables. Model adequacy was based on the C statistic (Appendix: Table A1) which follows a chi-square distribution and suggests an inadequate fit of the model to the data when $P < 0.05$ (Shipley 2009). Our objective was to develop a model that was parsimonious, had adequate fit to the data, and assessed the relative importance of bay laurel, tanoak, and redwood on disease intensity and mortality feedback. The model was revised if variables lacked implied conditional independence and consequently resulted in C statistic values with $P < 0.05$. Paths were evaluated for statistical significance with t tests and unstandardized coefficients were reported for statistically significant paths. Residuals for each response variable in the path model were saved and assessed for univariate normality in the error term; prevalence data were subsequently square-root transformed following the addition of 0.5 to meet the assumptions of normal distribution and homoscedasticity of error terms. Residual spatial autocorrelation in mortality rates was assessed based on Moran's I values calculated with distance-weighted residuals and was found to be nonsignificant for bay laurel and redwood and very low for tanoak (Appendix: Table A2). All statistical tests were considered significant at $P \leq 0.05$.

Results

Infection rates increased for all species over time (Fig. 1). Per capita infection rates were not significantly different between bay laurel and tanoak during pathogen establishment ($P = 0.33$, df = 85) or disease intensification ($P = 0.64$, df = 64) but were lower in redwood compared to bay laurel and tanoak during both periods. Overall, mortality rates were generally low for all species except tanoak during pathogen establishment and increased for all species in infected sites during disease intensification (Fig. 1). Mortality rates for tanoak were 6.4 and 5.7 times higher compared to bay laurel and redwood, respectively, during pathogen establishment ($P < 0.001$, df = 85; $P < 0.001$, df = 118). At the uninfected site (Big Basin State Park, California, USA), mortality rates of uninfected redwood and tanoak were not significantly different from each other for either the 1998–2002 and 2003–2007 periods ($P = 0.71$, df = 9; $P = 0.07$, df = 9). At infected sites, bay laurel mortality rates did not significantly differ from redwood during pathogen establishment ($P = 0.079$, df = 85) but were 20% lower compared to redwood during disease intensification ($P = 0.023$, df = 85). Falling tanoak was the most common cause of mortality in bay laurel and redwood during disease intensification and primarily occurred in understory trees (data not shown). Tanoak mortality rates remained 2.6 and 1.9 times higher compared to bay laurel and redwood mortality rates during disease intensification ($P < 0.001$, df = 84; $P < 0.001$, df = 118). Tanoak mortality resulted in proportional increases in bay laurel and redwood relative densities (Fig. 1).

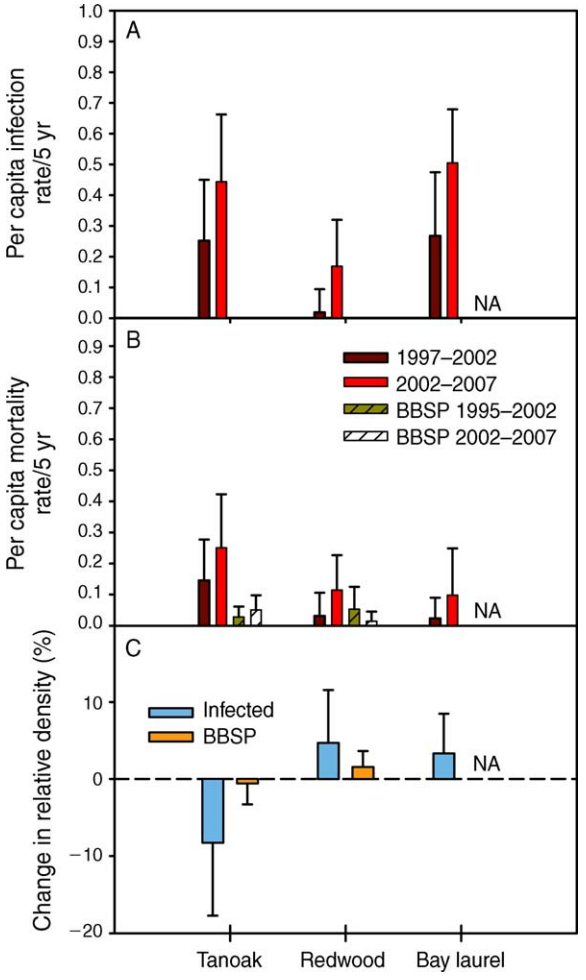

FIG. 1. Sudden oak death disease (SOD) dynamics and impacts in coast redwood forests in California, USA. Per capita (A) infection and (B) mortality rates for tanoak (*Lithocarpus densiflorus*), redwood (*Sequoia sempervirens*), and bay laurel (*Umbellularia californica*) were calculated for two periods of disease invasion: pathogen establishment (1998–2002) and disease intensification (2003–2007). The Big Basin State Park (striped bars; Santa Cruz County, California, USA) remained free of *Phytophthora ramorum* infection during the study and provided baseline mortality estimates for redwood and tanoak. (C) Change in relative species densities was calculated after 10 years of SOD. Data are means + SD. Baseline mortality rates for tanoak and redwood were estimated from a single pathogen-free site (Big Basin State Park [BBSP], $N = 10$), which has no bay laurel within the study plots (NA).

Our confirmatory path analysis revealed effects of apparent competition between bay laurel and tanoak mediated by *P. ramorum* (Fig. 2). Prevalence of infection in redwood did not result in any significant impacts on tanoak mortality rate. The per tree impact of infected bay laurel was 1.4 times greater than the impact of an infected tanoak on tanoak per capita mortality rate. At the scale of our 500-m^2 plots, this lead to a 1.3% (range 0.5–2.4%) increase in tanoak mortality for each additional infected bay laurel over a five year period. In contrast, increasing the prevalence of infected tanoak by a single tree resulted in a 0.9% (range 0.02–1.8%) increase in tanoak mortality for the same period. Densities for both species range from 0 to 25 stems per plot. Although mortality rates increased for redwood and bay laurel during disease intensification, tanoak mortality rate had no significant impacts on the per capita mortality rate of either species. Direct separation (d-sep) tests suggested that bay laurel per capita mortality rates lack independence from prevalence of infection in bay laurel, but the revised path model (Fig. 2) did not reveal statistically significant feedbacks on bay laurel survival.

DISCUSSION

This study shows that differences in transmission among host species drive patterns of apparent competition for the forest disease sudden oak death. The per individual impact of infection was greatest for bay laurel and secondly for tanoak, while infection in redwood had no significant impacts on tanoak mortality. These patterns follow relative levels of sporulation in these overstory species and suggest sporulation patterns are the best proxy for transmission of *Phytophthora ramorum* (Davidson et al. 2005, 2008). Although tanoak and bay laurel have similar susceptibility to the pathogen (Fig. 1), host-specific differences in transmission are important drivers of disease severity in this system. Apparent competition is an important contributor to mortality in these forests, and in conjunction with additive effects of transmission among tanoak, apparent competition is likely to lead to selective removal of tanoak from the overstory of many stands. Sporulation on tanoak and bay laurel is highly dependent on spring rain events and suggests that the strength of apparent competition in these forests is dependent on year-to-year weather conditions (Rizzo et al. 2005, Davidson et al. 2008). Overall, this analysis reveals that the pre-disease distribution of hosts is a useful predictor of disease intensity when transmission potential of the host community is incorporated into forecasts of risk. We expect the long observation period of our data set facilitated detection of this pattern. On shorter time scales, environmental stochasticity and the prevalence of infection within hosts may play an important role in determining patterns of disease severity (Gilligan 2002, Rizzo et al. 2005).

Separate parameters for infection and transmission are fundamental to basic epidemiological models, and we caution against using simple measures of susceptibility when making estimates of the potential range or impact of emerging infectious diseases such as sudden oak death (SOD). Susceptible hosts, such as many oaks, represent an extreme case; several oaks are susceptible to *P. ramorum* and die from infection, but no oaks are known to support sporulation. Thus, oak distribution alone provides little insight into spread risk of *P. ramorum*. However, apparent competition is likely to

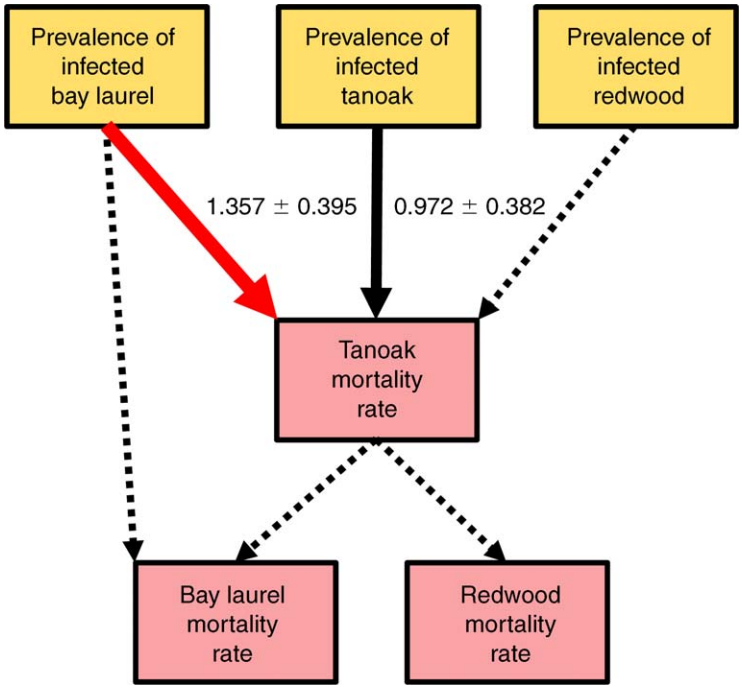

FIG. 2. Effects of *Phytophthora ramorum* mediated competition among bay laurel (*Umbellularia californica*), tanoak (*Lithocarpus densiflorus*), and coast redwood (*Sequoia sempervirens*) in forests impacted by sudden oak death. Confirmatory multilevel path analysis revealed statistically significant ($P \leq 0.05$) effects of apparent competition between bay laurel and tanoak (bold red arrow), intraspecies impacts of tanoak (bold black arrow), and nonsignificance of other hypothesized drivers (dotted line arrows). Values are nonstandardized regression parameters with one standard error for square-root transformed prevalence data.

occur between species such as coast live oak (*Quercus agrifolia*) and bay laurel due to the same epidemiological characteristics that drive disease severity in coast redwood forests, specifically differences in transmission and the impact of pathogens within hosts (Rizzo et al. 2005). In central California, presence of bay laurel is associated with higher risk of pathogen establishment (Maloney et al. 2005, Meentemeyer et al. 2008*a*), with additional local-scale variation in pathogen dynamics driven by variations in forest microclimate (Meentemeyer et al. 2008*b*) and bay laurel genotype (Anacker et al. 2008). An analogous community dependency on pathogen transmission is known from Lyme disease (Ostfeld and Keesing 2000, LoGiudice et al. 2003); communities with increased densities of the most competent reservoir host (white-footed mouse, *Peromyscus leucopus*) increased the prevalence of infectious vectors (black-legged tick, *Ixodes scapularis*). For both Lyme disease and SOD, an increased density of low transmission species at the community level is likely to reduce pathogen prevalence; however, different epidemiological processes may drive disease severity caused by plant pathogens. General models of plant disease show that secondary infection is important to pathogen persistence within plant populations (Gubbins et al. 2000); secondary infection is also a likely driver of higher tanoak mortality rate in the presence of infected bay laurel. Collectively, these results show that generalization of disease risk or severity based solely on susceptibility may result in erroneous predictions of disease spread and impact.

Infection in bay laurel and redwood results in minor tissue damage with no indication of increased mortality at this stage of disease progression (Davidson et al. 2005). Recent work by DiLeo et al. (2009) showed that *P. ramorum* infection in bay laurel has only a minimal impact on photosynthetic efficiency and no impact on leaf area of infected seedlings. In our study, neither redwood nor bay laurel exhibited increased mortality rate in response to the level of pathogen prevalence (Fig. 2). The low cost of infection in redwood and bay laurel is likely to initiate a shift to greater dominance of these species in the near term (Alexander and Holt 1998, Gilbert 2002, Rizzo et al. 2005). Similarity in canopy position between tanoak and bay laurel suggests bay laurel is more suited to exploit increased light availability compared to redwood (sensu Kohyama 1993). Although mortality, mostly associated with disease-related tree fall, increased through time for both bay laurel and redwood, our path analysis suggests that as disease intensified, neither species is significantly favored to survive the conditions in disease impacted stands. For understory trees that survive a period of increased tanoak tree fall, increases in resources are likely to benefit bay laurel and redwood fairly equally. However, negative feedbacks may over time mediate competition

between bay laurel and redwood and canopy dominance in these forests. For example, bay laurel is a preferred forage of black-tailed deer (*Odocoileus hemionus columbianus*) suggesting herbivory may favor canopy recruitment of redwood in regions with high deer populations and buildup of native pathogens such as *Armillaria mellea* could increase mortality of both species over time (Rizzo et al. 2005).

Community shifts have important ramifications for pathogen populations (Gilbert 2002, Burdon et al. 2006); specifically, reduced tanoak densities or increased redwood densities will decrease pathogen populations while increased bay laurel density will increase pathogen populations. Given that sporulation can be much higher on bay laurel, a complete replacement of tanoak by bay laurel would greatly magnify pathogen populations, but a simple species replacement is unlikely for moderate canopy diversity forests such as coast redwood and others containing tanoak. Increases in low-susceptibility, low-transmission species such as Douglas-fir (*Pseudotsuga menziesii*) are likely in much of the region impacted by SOD due to changes in the fire regime (Hunter and Barbour 2001). Additionally, sprouting in cut or killed tanoak is prolific and often cited as a limitation to regeneration of economically valuable conifers such as redwood (Tappeiner and McDonald 1984). Of the trees surveyed in this study, 43% of dead tanoak stems had basal sprouts and of these 58% were symptomatic of *P. ramorum* infection. Tanoak is unlikely to reestablish in the overstory because of sustained inoculum production from neighboring bay laurel trees or infected basal sprouts. However, sprouting individuals may occupy space and limit establishment of new individuals from other species (Bond and Midgley 2001). Tanoak persistence as an understory or lower canopy strata species may be possible in the novel conditions created by *P. ramorum*. A historical precedent is the impact that *Cryphonectria parasitica* had on American chestnut (*Castanea dentata*). *C. parasitica* altered conditions in eastern forests by relegating chestnut to the understory but did not extirpate the species from eastern forests (Paillet 2002). If increased tanoak sprouting limits seedling establishment, changes in canopy composition will likely be determined by overstory species, which can maximize capture of liberated resources. In coast redwood forests, bay laurel is the most likely beneficiary of overstory tanoak removal (sensu Kohyama 1993). Extirpation of tanoak from coast redwood forests is unlikely in the next 10–20 years, due to high rates of post-mortality sprouting. However, this regeneration mechanism is also likely to provide a reservoir for *P. ramorum* persistence and possibly outbreak of SOD in the absence of apparent competition from bay laurel.

We have shown that differences in transmission can drive patterns of apparent competition even when transmission within the most impacted host is sufficient to cause disease. Forecasts of apparent competition will benefit from assuming that transmission among hosts is a dominant driver of disease severity rather than considering measures of susceptibility alone. At a minimum, the assumption of transmission–susceptibility equivalence warrants assessment before making predictions based on susceptibility. Community composition can greatly influence the potential for pathogen spillover and risk of disease outbreak for plant, zoonotic, and human diseases with highly disparate impacts on economies and public health (LoGiudice et al. 2003, Power and Mitchell 2004). Regardless of host taxa, differences in transmission within the host community are likely to drive apparent competition and severity of many diseases.

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APPENDIX

Diagnostic analyses of the multilevel confirmatory path analysis in Fig. 2 (*Ecological Archives* E091-024-A1).