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Apparent competition among host species and feedbacks on disease severity in the sudden oak death pathosystem

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Background/Question/Methods

Selective infection and mortality by generalist pathogens holds the potential to shift competitive interactions and radically transform community structure. *Phytophthora ramorum*, the causative agent of sudden oak death, has a broad host range but is highly virulent to only a few species. In California redwood forests, susceptibility to foliar *P. ramorum* infection varies from high in California bay laurel (*Umbellularia californica*) to low in redwood (*Sequoia sempervirens*) but neither species suffers mortality. In contrast, tanoak (*Lithocarpus densiflorus*), an important component of these forests, is highly susceptible but suffers extensive mortality due to stem cankers. We hypothesize that differing susceptibility among host species to *P. ramorum* results in apparent competition among hosts and causes feedback between removal of tanoak and disease intensification. Because high amounts of pathogen sporulation occurs on leaves of bay laurel, increased survival of this species is likely to increase disease severity in tanoak. Conversely, increased survival of redwood is likely to decrease inoculum load because redwood is much less susceptible and supports minimal sporulation. Over the course of six years, we monitored survival of 5769 trees spanning the current geographic distribution of *P. ramorum* in California to estimate effects of *P. ramorum* on competitive interactions and host survival.

Results/Conclusions

Principal components analysis showed orthogonal relationships among host species before the arrival of sudden oak death which were influenced by edaphic factors such as topographic moisture index, aspect, and soil texture. Multivariate regression analysis of stand structure showed positive relationships between the level of tanoak mortality over a 12-year period and the biomass of bay laurel and tanoak existing before the onset of the disease. Negative correlations between tanoak and bay laurel after the arrival of the disease are similar to the patterns expected if these species were competing for resources. The difference in the patterns before and after pathogen introduction suggest that sporulation pressure is important in structuring community composition in impacted stands, and consequently that *P. ramorum* results in negative effects of bay laurel on tanoak. Logistic regression showed increased survival of infected redwood and bay laurel compared to uninfected trees in small size classes suggesting that infection may increase survival of these hosts, presumably through reduced competition for resources. These changes in community structure are likely to increase disease intensity in stands with

niche structure favoring bay laurel and decrease disease intensity in those with niche structure favoring redwood.

Incorporating effects of landscape heterogeneity on pathogen dispersal into spatially-explicit disease models

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Background/Question/Methods

Many spatially-explicit disease models have determined the probability of an area becoming infected or the force of infection in a particular area by calculating the straight line distances to known sources of infection, and incorporating a Euclidean-based dispersal kernel into a predictive disease model. However, these Euclidean-based dispersal kernels may only be appropriate for describing patterns of dispersal in homogeneous environments. The dispersal of most pathogens is influenced by a variety of landscape features such as the distribution of hosts, which may render Euclidean distances a less meaningful measure of the probability of reaching a given site. In these situations, the least-cost distance between sites based on the distribution of hosts and host habitat may be more appropriate.

In this study, we used a geographic information system (GIS) to examine the degree to which considering spatial heterogeneity of host habitat increases predictive power of dispersal kernel models for the emerging infectious disease Sudden Oak Death. Because the Sudden Oak Death pathogen (*Phytophthora ramorum*) is moderately dispersal limited, we hypothesize that conventional Euclidean-based dispersal kernels should not perform as well as models that incorporate heterogeneity into the dispersal estimation process. We first used a map of host (i.e., woodland) and non-host (e.g., grassland, agricultural land, residential developments) vegetation (derived from ADAR multispectral aircraft imagery) to calculate Euclidean and least-cost distances between 86 previously established plots in Sonoma County, CA. We then used field data to model the number of infected leaves in each plot (a measure of disease severity) as a function of the local density of primary foliar hosts, abiotic variables (i.e., precipitation, temperature, canopy cover, solar radiation, and topographic moisture index), and the force of infection from surrounding plots. The force of infection was modeled as a negative exponential dispersal kernel using either straight line or least-cost distances between plots.

Results/Conclusions

Our results demonstrate that although kernels based on Euclidean distances perform reasonably well in predicting disease severity, models that incorporate spatial heterogeneity of host habitat through least-cost path modeling perform better and are more epidemiologically relevant.

Ecological and economic impacts of Sudden Oak Death in Oregon with an emphasis on barrier zones and quarantines

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Background/Question/Methods

Sudden Oak Death (SOD), caused by *Phytophthora ramorum*, is an invasive forest disease that was recently discovered in Oregon (2001), where it has infected at least 170 acres and caused mortality in tanoak as well as leaf and shoot blight in many other hosts. We performed tradeoff analyses of policies being implemented to control the spread of SOD in order to provide a context to policy makers and to improve the allocation of resources invested in control.

Results/Conclusions

P. ramorum is currently distributed within a 162 square mile quarantine area in SW Oregon, where 1400 acres have been treated to limit SOD spread through eradication of host plants, at a total direct cost of \$2 million. The disease has also been discovered at 55 nurseries in Oregon (as of 2005), likely causing an additional \$727,100 in direct losses to the nurseries. Impacts to Oregon forests through tree mortality are thought to be limited to the tanoak forests in SW Oregon, where tanoaks are easily replaced ecologically by other hardwoods. Commercial forest landowners actively replace tanoak with conifers due to the low economic value of tanoak. Therefore, the major economic impacts of SOD to Oregon will come not from ecological losses, but instead from state, national, and international quarantines that control the treatment, movement, and export of *P. ramorum* hosts and their products, such as trees and lumber, floral greens, nursery plants, and Christmas trees. These policies would increase production costs and limit market access for affected commercial producers. The best size for a quarantine zone involves trading off between the benefits of a larger zone—for example, a lower chance of spread beyond the zone—with the costs of a larger zone—such as, increased numbers of businesses within the zone, higher enforcement/monitoring costs, lower effectiveness of enforcement, and spread within the quarantine zone. Similarly, a preliminary tradeoff analysis of a California host reduction zone to stop the northward spread of SOD indicates the level of avoided costs necessary to make the current zone size and investment worthwhile.

Spread of sudden oak death: Application of stochastic epidemic modeling to realistic landscapes

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Background/Question/Methods

As the number of emerging infectious diseases (EIDs) continues to rise, prediction of

disease outbreaks is critical for effective management and prevention of epidemics, especially in complex spatially heterogeneous landscapes. Epidemiological models of Susceptible-Infectious (SI) transitions increasingly incorporate effects of spatial heterogeneity, but are rarely applied to realistic landscapes of host availability, infection pressure, and abiotic conditions, limiting our ability to map hotspots for disease outbreaks and assess the geography of disease dynamics. In this paper, we describe and validate a stochastic epidemic model, applied to temporally and spatially heterogeneous landscape data mapped in a GIS, to predict the spread of the invasive forest pathogen *Phytophthora ramorum*, causal agent of the EID sudden oak death. Previously collected field and lab data were used to parameterize key system variables that affect establishment and spread of *P. ramorum*, including weather conditions, host infectiousness and availability, and a statistically-estimated dispersal kernel.

Results/Conclusions

Implemented on a weekly time step (1990-2030), the model considers three distinct epidemiological processes to forecast disease spread across the state of California at a spatial resolution of 250 m: production of inoculum by infected cells, dispersal of inoculum, and probability of infection. We established 784 field plots across the pathogen's potential geographic range to examine the correspondence between predicted spread of disease and observed spatial distribution of disease incidence in 2006. Model predictions have a high degree of correspondence with field observations of disease distribution and identify numerous forest ecosystems at high risk of infection. The nature of prediction errors are examined by ecoregion, plant community, and climate, and we discuss how application of epidemiological models to realistic landscapes in a GIS fosters understanding of spatially-explicit processes and designing management strategies to control spread.

Effects of sudden oak death on plant community structure and regeneration in the Big Sur ecoregion of California

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Background/Question/Methods

Sudden oak death is an emerging infectious forest disease caused by the recently discovered generalist pathogen *Phytophthora ramorum*. Lethal infections are concentrated in several ecologically important species, including tanoak (*Lithocarpus densiflorus*) and various oak species in the genus *Quercus*. The disease has killed potentially millions of trees in coastal forests of California and may be dramatically changing the ecological dynamics and biodiversity of these systems. In 2006 and 2007 we installed 280 500-m² forest monitoring plots on public and private lands in several watersheds throughout the Big Sur ecoregion to assess impacts of sudden oak death on stand composition and dynamics of coastal forests in this global biodiversity hotspot. The plots were randomly located in two forest types (mixed-evergreen oak woodlands, redwood-tanoak) across differing levels of fire history and disease severity. In each plot,

we mapped and measured all standing live and dead stems > 1 cm DBH, counted seedlings and advanced regeneration, and quantified plot-level measures of coarse woody debris, vegetation cover, canopy height and prevalence of *P. ramorum*.

Results/Conclusions

Approximately 20% of all host trees in the region were killed since the establishment of this disease in the mid-1990's, with levels of tanoak mortality exceeding more than 60% in some stands. The composition of understory seedlings differed with latitude and proximity to the coast, gradients that reflect variation in the abiotic environment and history of disease spread in the region. Community analyses examining differences in the advanced regeneration among plots with varying levels of *P. ramorum* infection and mortality revealed a significant correlation between understory composition and the amount and species identity of standing dead basal area. The total basal area of live tanoak and oak species also predicted the composition of understory seedlings. The high levels of tree mortality observed in Big Sur will greatly change the abiotic and biotic characteristics found in local forest stands. This has the potential to profoundly impact the trajectory of natural forest regeneration in infected stands compared to uninfected stands and affect regional biodiversity as disease continues to spread.