Community and Individual Effects on SOD Intensification in California Redwood Forests: Implications for Tanoak Persistence[1]

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Abstract

Processes operating across different spatial scales (for example, individual, community, landscape) influence disease dynamics. Understanding these processes and their interactions can yield general insights into disease control, disease dynamics within communities, and community response to disease. For *Phytophthora ramorum*, pathogen establishment and disease intensity are key drivers of deleterious impacts on ecosystems such as changes in fuel loads, tree mortality, and transformation of native plant communities. We studied infection rates of *P. ramorum* for major overstory species together with mortality rates caused by sudden oak death in tanoak (*Lithocarpus densiflorus*) in central coast redwood forests of California. Our analyses used a combination of individual and population-level estimates for these processes. Approximately 5800 trees were surveyed in 2002 and 2007 across 120 plots located at 14 sites from Sonoma to Monterey Counties (Maloney and others 2005). Our objectives were to examine rates of pathogen establishment amongst species and to identify key drivers of tanoak mortality. We used hierarchical path analysis to quantify community disease drivers (overstory species densities, prevalence of *P. ramorum* in infectious hosts) and survival analysis to examine characteristics that influence infection and mortality rates at the individual level (tree size, canopy position, species, post mortality sprouting). A simple theoretical model was constructed to examine the potential for tanoak persistence across redwood communities where *P. ramorum* has been naturalized. The model was parameterized from the path and survival analyses.

Previous study has shown the importance of the presence of California bay laurel (*Umbellularia californica*) and tanoak as factors predicting the likelihood of infestation (Maloney and others 2005). We hypothesized that disease severity at the stand level would be driven by the prevalence of infection within these two species. Furthermore, we expected that canopy position and tree size would cause infection and mortality rates to differ among tanoak trees. Our path analysis identified prevalence of infected tanoak and bay laurel as key drivers of tanoak mortality rate across our network of study plots. Pathogen prevalence in both species increased mortality rate, but the per-tree effect of bay laurel was 1.4 times greater than that of tanoak (Cobb and others, in press). High sporulation rate in bay laurel (potentially 10 times higher than tanoak; Davidson and others 2008) is the most likely driver of differences in mortality rate amongst our study plots, and suggests that risks posed by *P. ramorum* are best quantified among species transmission rates as opposed to among species susceptibility (Davidson and others 2008). Average time to infection differed among species. Within stands

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where *P. ramorum* had already become established, average time to infection was 8, 3, and 2 years for coast redwood (*Sequoia sempervirens*), tanoak, and bay laurel respectively. Considerable variation in time to infection was observed across plots and decreased with higher prevalence of infected bay laurel and tanoak, landscape level force of infection, and across canopy strata. Infection was more rapid in understory trees compared with trees in higher canopy strata. In contrast to rates of infection, median tanoak mortality rates following confirmed infection were more rapid in large trees compared with small trees. Once infection was confirmed in our study tanoak trees, trees died on average within 1 to 6 years, depending on tree size and number of infected bay laurel and tanoak within plots. Infection latency could bias these estimates towards an underestimate of post infection survival time. Survival times for all tanoak (regardless of confirmed infection status) resulted in death of tanoak on average between 4 to 15 years, depending upon other factors within plots. These estimates correspond closely with the time to infection following exposure (pathogen establishment within the stand) and post infection mortality. Estimated tanoak survival times were greatest for tanoak in stands dominated by conifers otherwise isolated from other infected bay laurel and tanoak. Within our study plots, trees with maximum expected survival were located in stands dominated by redwood and Douglas-fir (*Pseudotsuga menziesii*). Half of all tanoak biomass has been killed within our study plots during the first 8 years following disease establishment. However, 2.3 percent of the study population had estimated survival times greater than 28 years. This suggests potential exists for tanoak persistence at low densities and in stand conditions with low or no density of other species that support sporulation of *P. ramorum*, such as bay laurel and *Rhododendron* sp. Results from these statistical models are in line with results from our theoretical model (see below).

Stump sprouting from trees that have been killed by *P. ramorum* is common (43 percent of *P. ramorum*-killed tanoak stems) and 58 percent of sprouting basal sprouts had symptoms of *P. ramorum* infection. This suggests that of killed trees, 25 percent still provide an opportunity for vertical transmission via sprouting. Prolific basal sprouting also provides an additional pathway for pathogen persistence, especially in stands where tanoak is the sole supporter of sporulation. Post mortality basal sprouting may also help tanoak persist as an understory component of coast redwood forests. To test this hypothesis, we constructed a simple dynamic disease-competition model to investigate tanoak persistence with and without a co-occurring community member which supports sporulation. In our model, the co-occurring sporulation supporting species is similar to bay laurel in that infection does not alter the host mortality rate. The model suggests that in pure tanoak stands, tanoak persistence is likely, but surviving trees may not survive beyond 15 years which would relegate the species to the understory. In stands with sporulation supporting species such as bay laurel, tanoak is likely to persist only when post-mortality sprouting is high, but tanoak densities are expected to be much lower than in pure stands and tanoak would be relegated to the understory. This study demonstrates that sudden oak death differs from historical disturbances such as fire by selectively killing trees that are large and close to *P. ramorum* sporulating species. The disease is also likely to increase understory tanoak for the next several decades, but it remains uncertain whether these stand changes could impede regeneration of economically valuable conifer species. Forestry practices which increase conifer regeneration, such as thinning of tanoak and other sporulation supporting species, may also reduce the prevalence of sudden oak death and maintain tanoak as a component of coast redwood forest biodiversity.

Literature Cited

Cobb, R.C.; Meentemeyer, R.K. and Rizzo, D.M. [In press]. Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility. Ecology.

Davidson, J.M.; Patterson, H. and Rizzo, D.M. 2008. Sources of inoculum for *Phytophthora ramorum* in a redwood forest. Phytopathology. 98: 860–866.

Maloney, P.E.; Lynch, S.C.; Kane, S.F.; Jensen, C.E. and Rizzo, D.M. 2005.
Establishment of an emerging generalist pathogen in redwood forest communities. Journal of
Ecology. 93: 899–905.