

The Effects of Sudden Oak Death and Wildfire on Forest Composition and Dynamics in the Big Sur Ecoregion of Coastal California

Margaret R. Metz,^{1,2} Kerri M. Frangioso,¹ Ross K. Meentemeyer,³ and David M. Rizzo¹

Introduction

Sudden oak death (SOD), caused by *Phytophthora ramorum*, is an emerging forest disease associated with extensive tree mortality in coastal California forests (Rizzo et al. 2005). *P. ramorum* is a generalist pathogen that infects many hosts, but hosts differ in their ability to transmit the disease and in the impacts caused by the disease. In coast redwood forests, tanoak (*Notholithocarpus densiflorus*) is the main host dying from SOD and the main source of pathogen inoculum. SOD leads to compositional changes in these habitats through selective mortality of tanoak.

The effect of SOD on forest dynamics does not happen in isolation from other disturbances. Rather, fire is an important part of the natural disturbance regime in California's coastal forests. Elevated mortality by the disease may lead to increased fuel loads in these forests, leading to the potential for SOD to interact with wildfire severity (Valachovic et al. 2011). In 2008, wildfires in SOD-impacted forests presented a natural experiment to examine the interaction between these two disturbances (Metz et al. 2011).

We used a long-term network of forest monitoring plots in the Big Sur region of central California to examine the separate and joint impacts of an emergent forest disease and wildfire on forest dynamics and diversity. The network contains burned and unburned areas, and areas with and without *P. ramorum*. Specifically, we asked: 1) Did redwood stands that became infested by *P. ramorum* differ in species composition from uninfested areas? 2) How is *P. ramorum* changing species composition in redwood forests? 3) How did mortality from SOD impact fire severity in burned areas? 4) Was fire mortality selective by species?

Methods

The Big Sur region represents the southern limit of the ranges of both SOD and the coast redwood. Big Sur forests were a relatively early site of invasion by *P. ramorum* and are among the most impacted by the pathogen (Meentemeyer et al. 2008). In Big Sur, the historical and current distribution patterns of redwood consists of scattered stands, primarily in wetter canyon areas near the coasts. The redwood habitat is usually found in close proximity to a variety of other habitat types, including oak woodlands, chaparral, or grasslands, leading to a very heterogeneous matrix of habitat types. Forest and disease dynamics in this landscape can therefore differ dramatically from what is observed further north in the ranges of redwoods and

¹ Department of Plant Pathology, 1 Shields Ave., University of California, Davis, CA 95616.

² Corresponding author: mrmetz@ucdavis.edu.

³ Department of Geography and Earth Sciences, University of North Carolina, Charlotte, NC 28223.

P. ramorum, where the climate is cooler and wetter and redwood stands exist in much larger swaths.

We established a network of 280 monitoring plots throughout the Big Sur ecoregion in coastal California in 2006 and 2007 to examine the impacts of SOD on forest dynamics. These plots were located in two habitat types, mixed-evergreen forests (n=164) and redwood-tanoak forests (n=116). Within each plot, we marked, measured, and assessed for pathogen symptoms and health every live or dead stem ≥ 1 cm DBH. We also quantified the volume of large coarse woody debris, and the vegetation cover of all herbs, shrubs and trees. Here, we present results based on the 116 redwood-tanoak plots. We used live basal area to represent current plot composition or species abundance during a given census. Total basal area, calculated by summing the basal area of live and dead standing stems, was used to re-construct pre-epidemic stand conditions, without considering disease-related or other mortality.

Two wildfires in 2008 (the Basin Complex and the Chalk fire) burned over a third of this network, providing the opportunity to compare forest dynamics in uninfested and infested areas, both within and outside burned areas, using pre-fire data on fuel availability and disease impacts and post-fire data on burn severity and tree mortality. Standing dead basal area and the volume of downed logs were used as measures of large woody fuel abundance prior to the fire. We conducted a survey of burn severity in 2008 (Metz et al. 2011), using the Composite Burn Index, and surveyed for tree mortality in 2009.

Results

Of the 116 redwood plots, 70 were infested with *P. ramorum* in 2006 and 2007. There was a gradient among plots in dominance of tanoak or redwood, and some plots also contained another sporulating host, bay laurel. The species composition of infested plots differed significantly from that of uninfested stands. Stands that became infested had significantly greater total basal area (live and dead stems) of the sporulating hosts tanoak and bay laurel, and significantly lower abundance of redwood. At the same time, tanoak experienced far greater mortality in infested plots than in uninfested plots, and there was a trend towards lower mortality in bay and redwood in infested plots.

Infested plots had significantly greater abundance of standing dead snags and large surface fuels. However, burn severity did not vary according to pathogen presence alone, but rather with the abundance and type of fuels that occur at different stages of the disease progression (Metz et al. 2011). In areas with new SOD-caused mortality, where dead trees still retain a crown of dry, dead material, burn severity increased with increasing SOD impacts. Similarly, in areas where the disease had progressed further and surface fuels had accumulated, soil and substrate burn severity increased significantly with greater volume of host logs. However, the abundance of dead trees and logs were not significant predictors of burn severity across the entire network, when infested and uninfested areas were examined together.

For trees that had been alive upon plot establishment in 2006 and 2007, the amount of basal area that was dead in 2009 was significantly higher in burned areas than unburned areas, regardless of the presence/absence of SOD. Outside of the burned areas, mortality was negligible except for canker hosts in infested areas,

where mortality is increased by SOD. Within the burned area, fire-caused mortality varied greatly among canker host species and non-host or foliar host species (which do not have lethal infections). Canker host mortality, primarily driven by tanoak mortality, was as high as 34 percent in burned, uninfested areas and 57 percent in burned, infested areas, on average, with great variation (difference between groups was nearly significant; $t=-1.9669$, $df=36.98$, $p=0.057$). Mortality of non-canker hosts (primarily redwoods, but also bay laurel) was much lower than for canker hosts. However, even non-canker host mortality was elevated in infested stands (16 percent) relative to uninfested stands (6 percent), indicating the potential for SOD mortality to worsen the effects of wildfire, although the difference was not significant ($t=-1.6008$, $df=27.67$, $p=0.12$).

Discussion

Both sudden oak death and the 2008 wildfires have been altering the species composition in Big Sur redwood forests through selective mortality (Maloney et al. 2005, Metz et al. 2011). Tanoak is dying in large numbers in areas infested with *P. ramorum*, and the 2008 wildfires caused greater mortality in canker hosts such as tanoaks relative to other dominant species in these stands (primarily redwoods). These changes in composition may lead to long-lasting impacts to forest diversity, dynamics, and structure in areas impacted by SOD in these fire-prone systems.

When trying to understand the impact of SOD on California's coastal forests, it is very important to understand that *P. ramorum* is an important biotic disturbance that has been introduced into a system that already had a significant disturbance regime consisting primarily of periodic wildfires. Thus, the full impacts of either disturbance cannot be examined in isolation. The relationship between disease impacts and burn severity is not straightforward. Our surveys of tree mortality 1 year following the fire suggest that mortality of all species is higher in infested areas.

We hypothesize that long-term forest composition will depend on the joint and interacting effects of disease and fire. Changes to tanoak abundance, whether caused by SOD or fire, will have important implications for establishment of *P. ramorum* and disease dynamics, especially in stands where tanoak is the sole sporulating host; stands with increasing redwood abundance are less likely to be infested. Within burned areas, SOD impacts may be elevating redwood mortality, perhaps through longer fire residence times or continued smoldering of surface fuels at the base of large trees that may otherwise be somewhat resistant to a fire that passes through quickly. This leads to the potential for SOD and fire to interact to create longer-lasting impacts to the composition of these forests. Further investigation into these interactions is ongoing.

References

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