Interacting Disturbances: Did Sudden Oak Death Mortality in Big Sur Worsen the Impacts of the 2008 Basin Complex Wildfire?[1]

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Introduction

In late June 2008, a large, dry lightning storm ignited thousands of fires across California. The largest of these fires became the Basin-Indians Complex Fire in Big Sur, along the State's central coast. The fire burned over 240,000 acres (USDA Forest Service 2008) and required over a month of intense firefighting operations to contain the perimeter. Media reports and anecdotal accounts from firefighters linked the intensity of the fire and difficulty of firefighting operations to increased fuels from tree deaths caused by an emergent forest disease, sudden oak death (SOD).

Coastal California forests have experienced extensive mortality from the pathogen *Phytophthora ramorum*, causal agent of SOD (Rizzo and others 2005). The forests of Big Sur are among the most impacted by *P. ramorum*, with 100 percent of tanoaks in some stands infected by the pathogen and hundreds of thousands of dead host trees across the region (Maloney and others 2005). Big Sur is among the earliest sites of *P. ramorum* infection in California, and the pathogen has spread and become established throughout great portions of the region (Meentemeyer and others 2008).

We used an extensive network of forest monitoring plots in Big Sur to examine the potential interactions between these two important disturbance agents, a destructive exotic pathogen and wildfire. We used pre-fire data on tree mortality and pathogen distribution and post-fire surveys of burn severity to ask: i) How did pre-fire fuel loads vary among areas that differ in pathogen presence or impacts? and ii) Was burn severity higher in areas that had previously experienced higher SOD mortality? Ongoing research will track longer-term impacts of the fire on forest structure and recovery.

Materials and Methods

In 2006 and 2007, we established 280 intensive long-term monitoring plots across Big Sur as part of ongoing research to examine the changes in the forest community and environment that might result in positive or negative feedback between the

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pathogen, its various hosts, and the physical environment. The plots are randomly stratified among watersheds, in two forest types (redwood-tanoak or mixed evergreen), on public and private lands, and in areas with and without the presence of *P. ramorum*. In each 500 m^2 plot we quantified disease incidence, levels of tree mortality, amount of coarse woody debris, and various other biological and physical characteristics of the forest. Ninety eight of these monitoring plots were within the perimeter of the 2008 Basin Complex fire.

We used measures of standing dead basal area and downed log volume for both host and non-host tree species to understand pre-fire fuel loads in the region. Every standing dead stem ≥ 1 cm dbh (diameter at breast height, 1.3 m) was identified to species, measured for dbh, and classified according to an estimated time since death based on the presence or absence of fine fuels (leaves and fine twigs) or the fracturing and falling of major canopy branches. Downed logs ≥ 20 cm in diameter were identified to species and measured in 0.5 m length increments and 5 cm width increments to obtain a cylindrical volume.

In September and October, 2008, following containment of the Basin Complex Fire, we conducted a rapid response survey of 61 monitoring plots to assess burn severity using characteristics likely to disappear with the onset of California's winter rains. These included 30 mixed evergreen plots (nine uninfested, 21 infested) and 31 redwood-tanoak plots (10 uninfested, 21 infested). We rated plot-level burn severity using the Composite Burn Index (CBI), a rating from zero to three of the damage to several forest strata (soil, herbs, shrubs, intermediate trees, and dominant trees). We also took quantitative measures of soil and tree damage at eight random locations in each plot. We measured soil damage by assessing the depths of deposited ash, consumed litter and duff, and destroyed soil. We also measured the height of bole charring and canopy scorching or torching on the tree nearest the soil sample point.

We compared pre-fire fuel loads (standing dead basal area or downed log volume) from the full network of plots for lethal host species (tanoak, *Lithocarpus densiflorus*; coast live oak, *Quercus agrifolia*; and Shreve's oak, *Q. parvula* var. *shrevei*) using one-sided Mann-Whitney U tests, hypothesizing that host fuel loads were higher in plots where *P. ramorum* was present. Similarly, we compared burn severity in each forest type between infested and uninfested plots, hypothesizing that burn severity was worse where the disease was present. We used linear regressions of burn severity against fuel abundance (for lethal host species alone or all species) across both forest types and disease presence/absence to examine whether increasing fuels increased burn severity. We also separately analyzed the relationship in the 27 plots that contained recent SOD mortality and compared the relationship to that found in the 15 infested plots with older mortality. All our measures of burn severity were highly correlated, so we used the CBI as the outcome variable in the analyses presented here.

Results

Host fuels were significantly higher in plots with *P. ramorum* than in plots without the pathogen, as measured by host standing dead basal area and the volume of downed host logs. Despite great differences in host mortality, burn severity (CBI) did not differ between infested and uninfested plots in either forest type. The CBI also

showed no relationship with the amount of standing dead host basal area across all 61 plots.

We did observe the hypothesized increase in burn severity with increasing fuel loads when we examined the different types of fuels that occur over time as the pathogen becomes established in an area. Burn damage to the soil layer (as represented by the soil stratum component of the overall CBI) significantly increased with an increase in the volume of host logs. In plots with recent SOD mortality, where dead trees still possessed leaves and fine twigs when surveyed in 2006 and 2007, standing dead basal area (of all species) was a significant predictor of the overall burn severity (CBI), such that greater mortality led to increased burn severity across both forest types. In infested plots where mortality was older, and the trees had lost their fine fuels, there was no such relationship, however.

Discussion

The Basin Fire provided a unique opportunity to examine the potential for *P. ramorum* to have cascading effects on forest communities through interactions with wildfire. We hypothesized that SOD mortality would increase fire severity, but our results demonstrate that the relationship is complex, with great variability in SOD impacts and in burn severity across the region. Although host mortality increased significantly when the pathogen was present, burn severity showed little relationship with pathogen presence. We found that increasing fuel abundance did predict increasing burn severity when fuels were examined separately by type, however.

The effects of SOD on forest structure develop over several years as *P. ramorum* is dispersed to a new area, becomes established and begins to kill trees (Rizzo and others 2005, McPherson and others 2005). The quantity and quality of available fuel will correspondingly vary. For example, recently dead host trees may retain their leaves and fine branches for a year or more, resulting in a canopy full of very dry and highly flammable fuels. With time, these fine fuels will fall to the ground and decompose, and the larger branches in the canopy will also begin to fragment and fall. This will result in greater surface fuels in an area, and the rate of decomposition of these fuels will vary among species and habitat conditions.

Our results indicate that the timing of the fire in regard to the progression of the disease is an important predictor of burn severity because differences among fuel types were more important indicators of damage than pathogen presence alone. We found increased soil damage in plots with greater volumes of large downed logs. In higher severity plots, the fire consumed more of the litter or duff and destroyed soil to greater depths. The heat transfer to the soil causing this damage may also kill tree fine roots, and the soil damage will likely affect water flow and soil erosion throughout the plot. We also found that the abundance of standing dead biomass predicted burn severity only in areas that had recent SOD mortality, likely due to the presence of dry, fine canopy fuels, whereas no such relationship existed in plots where the pathogen had been established for longer periods of time.

Although there was an important and detectable relationship between SOD and fire severity, there remains much unexplained variation in fire severity. Fire behavior and spread depends on fuel availability, habitat characteristics and the climatic conditions

occurring on the day of the fire (Rothermel 1983). Big Sur contains a complex mosaic of habitat types, has steep topography, and has large temperature and moisture gradients. Mortality from SOD across the landscape varies by region and forest type (Maloney and others 2005, Meentemeyer and others 2008). The processes that determined fire severity during the Basin Complex fire likely occurred at multiple scales and were dependent on many factors, only one of which is the SOD mortality measured at the scale of our plots.

There are very few examples in the literature of post-fire ecological effects that are based on both pre- and post-fire data; most studies are based solely on post-fire examination of the landscape with no previous data (Jenkins and others 2008). Even rarer is the opportunity to examine interactions between a large wildfire and a destructive biological invasion. Ongoing research will expand our surveys of burn severity to a greater number of study plots and track tree mortality, forest regeneration, and pathogen survivorship and spread.

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