Wildfire Limits the Occurrence, Frequency, and Impacts of *Phytophthora ramorum* in the Coastal Forests of Big Sur, CA

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Ecological disturbances have clear potential to shape the dynamics of native and introduced plant diseases, via their impacts on the spatial pattern of hosts, host composition, pathogen survival, and microclimatic conditions. Yet, studies examining potential interactions between disease and disturbance are rare, or primarily focus on pathogen occurrence, rather than the additional processes of spread, infection, and host mortality that determine disease impacts. Given that anthropogenic activities have both altered disturbance regimes across the western United States and introduced ecologically-damaging, non-native plant pathogens, understanding how disease dynamics may interact with or be determined by historical and changing disturbance regimes may be of significant conservation and management importance.

The emerging infectious disease sudden oak death (SOD) impacts fire-prone coastal forests in California and Oregon, and previous studies suggest that historical and recent fire history may influence the occurrence of SOD's causal agent, *Phytophthora ramorum* (Moritz and Odion 2005, Beh et al. 2012). In this study, we leveraged a ten-year forest monitoring dataset tracking the impacts of *P. ramorum* across the Big Sur region to explore the mechanisms underlying the relationship between fire and disease. We analyzed how both long-term fire history and recent wildfires influence pathogen presence, but also other metrics of disease, including infestation intensity, re-invasion, and severity of SOD host mortality.

We found that areas that burned more frequently over the last sixty-five years were less likely to contain California bay laurel (*Umbelluria californica*) trees and had reduced SOD host basal area. In turn, more frequently burned plots were less likely to contain *P. ramorum*, had lower rates of host infection, and exhibited decreased rates of host stem mortality. Immediately following the 2008 Basin Complex fire in this region, previous research recorded a reduction in *P. ramorum* occurrence in burned areas, with the pathogen primarily persisting in surviving, intact California bay laurel canopies (Beh et al. 2012). Up to 7 years following fire, we find no evidence that regenerating host vegetation plays an epidemiologically significant role and find that infestation intensity is primarily determined by this legacy of surviving hosts. Further, infection by *P. ramorum* does not appear to strongly determine host mortality in these recently-burned areas, suggesting that wildfire may reduce *P. ramorum* propagule pressure or alter microclimates to reduce SOD severity. Overall, these results suggest that severe wildfire reduces not only *P. ramorum* occurrence, but also its spread, infestation intensity, and mortality impacts, which could have significant implications for predictions of future SOD dynamics under climate change and increasing wildfire ignitions from human activities in this region.

References

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