Novel Interactions between Wildfire and Sudden Oak Death Influence Sexual and Asexual Regeneration in Coast Redwood Forests[1]

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Abstract

Novel interactions between compounded disturbances can leave lasting ecological legacies on communities and alter regeneration trajectories. Sudden oak death (SOD), caused by *Phytophthora ramorum,* is a biotic disturbance, an emerging disease causing widespread oak and tanoak mortality in California's coastal forests. In these redwood-tanoak forests, wildfire is a keystone process, shaping stand structure and composition. Dominant tree species can resprout rapidly after being "top-killed" by fire, in addition to regenerating via seed. Yet, in SOD-impacted areas, fuels generated by disease-related tree mortality may alter fire behavior and consequent stand recovery. In this system, tree species differ in resprouting capacity, patterns of seedling regeneration, effectiveness as hosts for *P. ramorum,* and susceptibility to fire and SOD.

In 2006 and 2007, we established 280 forest plots to monitor the impacts of SOD in the Big Sur region. The 2008 Basin Complex fires burned across infested and disease-free plots in this area, generating an opportunity to investigate impacts of a novel disturbance interaction on forest regeneration. Following the fire, burned and unburned forest plots were repeatedly sampled to assess tree mortality, pathogen presence, microclimate, and regeneration, including resprout and seedling recruitment across a gradient of SOD impacts. We investigated how interactions between fire and disease influence forest regeneration trajectories, and in turn, how regeneration in host tree species impacts post-fire disease dynamics and prevalence of *P. ramorum*.

Tanoaks in mid-stage SOD-infested areas were more likely to suffer complete (belowground) post-fire mortality, likely due to altered fuels and increased fire severity in disease-impacted areas. Pre-fire tree size and stand-level interactions between SOD and fire significantly influenced the abundance of resprouting regeneration measured in tanoaks that survived top-killing. In plots impacted by fire and SOD, individual tanoak resprouting vigor was significantly greater than expected by our model. This suggests that the pathogen is not measurably hindering re-growth of tanoak (a susceptible host) in burned, disease-impacted areas, despite reinvasion of *P. ramorum*. We hypothesize that this is due to decreased host connectivity and severe microclimates in burned areas. This also suggests that tanoaks surviving a bottleneck generated by SOD and fire experience reduced competition, responding with increased resprouting.

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Successful recruitment via seedlings is less common than recruitment via vegetative reproduction in this system. Following wildfire, seedling survival was similar across infested and uninfested areas, and post-fire seedling communities were similar regardless of SOD disease history, suggesting that wildfire, as a disturbance, may have a stronger overall influence on these sexual regeneration patterns. Areas impacted by SOD show pulses in tanoak and oak seedling recruitment associated with periods of disease-related tree mortality. The presence of *P. ramorum* did not significantly reduce seedlings and saplings' likelihoods of survival, echoing previous results that suggest that smaller trees may be less readily impacted by SOD.

Patterns of regeneration also influenced post-fire recovery of *P. ramorum*. In burned areas, the pathogen was significantly more likely to be recovered in larger bay laurel sprouting clusters and in areas with more surviving canopy cover, suggesting that resprouting and microclimate may play a key role in post-fire disease reinvasion. These results suggest that systems dominated by asexual regeneration may be surprisingly resilient to compounded disturbances, and despite reinvasion, *P. ramorum's* impacts may be temporarily reduced by fire-related changes to the stand.