

Examining the Relationship between Fire History and Sudden Oak Death Patterns: A Case Study in Sonoma County¹

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Abstract

Fire is often integral to forest ecology and can affect forest disease dynamics. Sudden oak death has spread across a large, fire-prone portion of California, killing large numbers of oaks and tanoaks and infecting most associated woody plants. Building on our earlier study of fire-disease dynamics, we examined spatial patterns of confirmed infections in relation to past fire history for a single county using a geographic information system (GIS). The region analyzed here is Sonoma County, where infections have been documented in past burn areas and exposure to the disease is thought to be extensive. We found that the disease is extremely rare inside recently burned areas (since 1950), although the relationship in this region is somewhat weaker than previous findings across a much larger area. This highlights how factors, such as variation in vegetation, climate, and patterns of severity, within burn areas are important to consider in the landscape pathology of sudden oak death. Despite confirmation of earlier findings at a different scale of analysis, it remains to be seen whether the observed negative relationship between current infection locations and past fires is due to a fire-related mechanism, a surrogate of fire, and/or an artifact of the datasets used. Regardless, it is likely that interactions between the disease and fire will vary with local differences in host species composition, the stage of disease progression, and the scales of space and time used in analyses.

Key words: spatial pattern analysis, landscape pathology, fire management, forest disease epidemiology

Introduction

Sudden oak death (SOD), a disease caused by the pathogen *Phytophthora ramorum*, has reached epidemic proportions over the last decade across landscapes of northern coastal California. Based on climatic factors and host distributions, there are large portions of the state at risk for the disease, including ecosystems of the Sierra Nevada foothills (Meentemeyer

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and others 2004, Guo and others 2005). Suitable habitat for the disease may exist over extensive areas in the western U.S. and beyond, and there is great concern over the cascading ecological effects that will occur in affected areas (Rizzo and others 2002, Rizzo and Garbelotto 2003). A variety of modeling tools exist for predicting the spatial-temporal dynamics of plant disease epidemics (van Maanen and Xu 2003), although presently not enough is known about SOD to parameterize such models.

Forest diseases involve interactions among pathogens and hosts in heterogeneous, dynamic environments (Lundquist and Klopfenstein 2001), all of which are components of the plant “disease triangle.” This conceptual model illustrates a fundamental paradigm in plant pathology: that is, the existence of a disease caused by a biotic agent requires the interaction of a pathogen, susceptible hosts, and an environment favorable to disease development (Stevens 1960). Recently there has been increasing awareness of landscape-scale factors in forest diseases, exemplified by the emergence of “landscape pathology” as a field of study (Holdenrieder and others 2004). This approach is an extension of the traditional disease triangle that explicitly includes coarse-scale landscape patterns and processes as part of disease dynamics. Examples include landscape fragmentation effects on pathogen dispersal and how vegetation diversity patterns might limit the availability of susceptible hosts (Holdenrieder and others 2004).

Fire is one of the dominant pattern-forming forces on terrestrial landscapes (Bond and others 2005), and it is an important natural disturbance in ecosystems currently impacted by SOD. Our goal has, therefore, been to understand the strength and possible causes of a relationship between past fires and current disease patterns (Moritz and Odion 2005). Given how little is known about the landscape pathology of SOD and the possible role of fire, here we reanalyze fire-disease patterns in a specific area, to see if our earlier findings hold at a finer spatial scale. We also discuss how geographic variation in fire and processes it affects at different scales of space and time may influence the landscape pathology of the disease. While there are many ways fire can influence the landscape pathology of the disease, specific mechanisms must be identified to confirm that the observed spatial relationship between current *P. ramorum* infections and long unburned areas is caused by fire.

Case Study: Sonoma County

The analysis presented in this paper is an extension of previous analyses (*fig. 1a*), which are described elsewhere in detail (Moritz and Odion 2005). In our earlier work we found a strong and consistent negative relationship between *P. ramorum* infections across northern California and the locations of fires since 1950, and this relationship was robust under different scenarios of susceptibility in host abundance and elimination of spatially dependent sample observations. Notably, we also found that the reduced probability of infections within areas burned in the past was not due to undersampling in previously burned areas, supporting the hypothesis that past fire occurrence is somehow important in the spread of the disease. Sonoma County contained the portion of the study area in which the relationship appeared to

be weakest, because it was the only region in which infections were found in past burned areas. Sonoma County is also thought to be an area that has been completely exposed to *P. ramorum*, which may not be the case for all susceptible landscapes examined in our earlier study (D. Rizzo, personal communication). In this paper we examine Sonoma County in particular (*fig. 1b*) to address possible concerns about variation in pathogen exposure across a study area, and to test the fire-disease relationship in a region where it appears to be least robust. This case study will also allow for a finer spatial scale of analysis; however, the temporal scale will remain the same as in Moritz and Odion (2005).

Methods and Data

The infection database used for this study contains confirmed *P. ramorum* infection locations as of October 2002, reported through the California Oak Mortality Task Force (COMTF) (Kelly and others 2004). Only those samples testing positive and not originating from a nursery were used in our analyses, which were restricted to Sonoma County (*fig. 1b*). We used the mapped fire history in a GIS format from the California Department of Forestry and Fire Protection (CDF)⁴ and merged this with a supplemental CDF coverage of management burns. We then used a GIS to identify portions of the landscape that had burned in the relatively recent past (i.e., since 1950) and those we considered “long unburned” for further analysis. It is worth noting that these mapped fire databases omit many of the smaller fires that have occurred in the past (in other words < ~120 ha, based on official documentation); however, these fires cumulatively account for a very small proportion of the landscape burned over time. In addition, many areas burned in past smaller fires have been subsequently reburned in larger, mapped events, so omission of small fires in the database should not bias analyses.

⁴ See <http://frap.cdf.ca.gov/data/frapgisdata/select.asp> for additional details and metadata documentation.

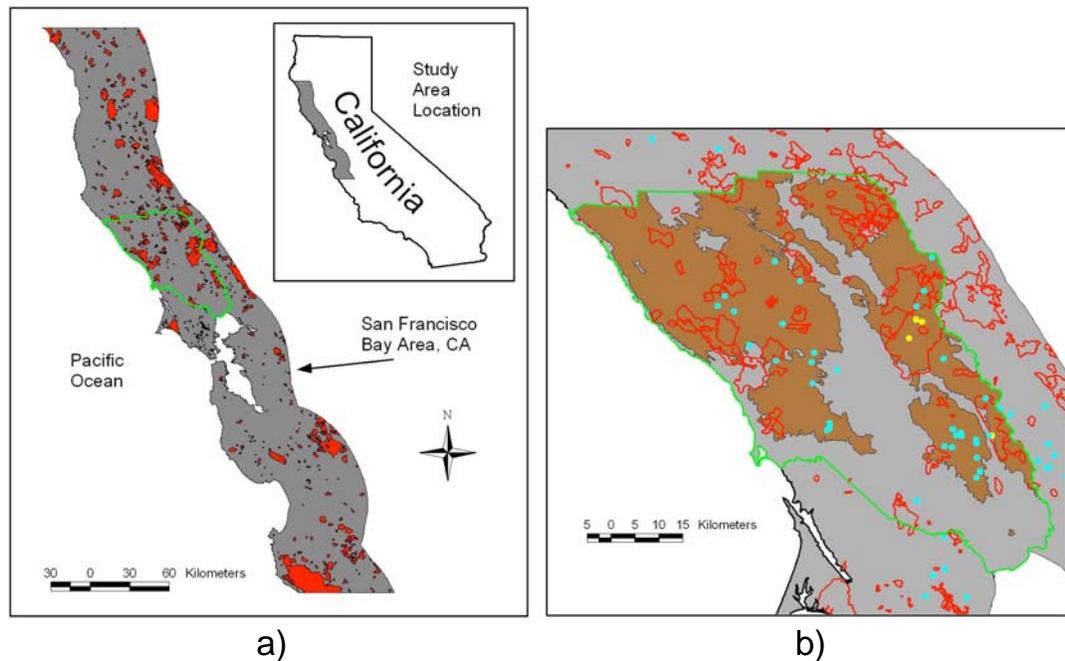


Figure 1—Map of study area. Panel a) shows the portion of California analyzed in Moritz and Odion (2005), which extended approximately 61 km inland from the Pacific coastline; mapped fire history (1950-2000) is shown in red, and Sonoma County is highlighted with a green border. Panel b) shows the study area of Sonoma County analyzed here, with landscape susceptibility scenario L3 in brown, past fire perimeters in red, and positive *P. ramorum* infection locations shown as points; infections points located in past burns are shown in yellow, while others are in light blue. See text for details about infection sampling and vegetation scenarios used in analyses.

As in Moritz and Odion (2005), we examined the strength and consistency of the fire-disease relationship in Sonoma County using different sampling and landscape susceptibility scenarios. We did this because the original COMTF database had been populated by sampling symptomatic plants on an *ad hoc* basis, so it was possible that an observed relationship between infections and past fires could be due to how one defined the study area for analysis, or artifacts of sampling. We found that the original sampling effort was actually biased somewhat toward sampling in past burned areas (Moritz and Odion 2005), which makes the consistent negative relationship in fire-disease patterns even more striking. Although we discuss the issue of possible sampling bias later in this paper (see Assumptions and Limitations section), we do not reanalyze the sampling effort itself (in other words including samples testing negative) further.

For the Sonoma study area, we employed three landscape susceptibility scenarios. These were based on the abundance of susceptible host species in vegetation units mapped for the California Gap Analysis Project (Davis and others 1998), which contain floristic detail on the extent of vegetation types (in other words primary, secondary, and tertiary coverages) within

mapped polygons. Details of these scenarios are given in Moritz and Odion (2005), although the following provides an overview of the screening categories resulting from characteristics of the dominant mapped vegetation types: L1 specifies that susceptible vegetation dominates the primary, secondary, or tertiary types; L2 requires that either the primary or secondary vegetation types are of susceptible species; and L3 means that the primary cover type must be mapped as susceptible vegetation⁵. This approach produced a gradient of increasingly restrictive screens, progressively excluding areas that might fall near urban and/or agricultural landscapes.

Plant diseases can result in clustered symptoms and infections, and many samples from the COMTF database were collected in close proximity. For these reasons, it is likely that many of these samples do not constitute spatially independent observations. To create an alternative subsampling scenario to address possible spatial dependence issues in sampling, we aggregated closely spaced sample points into “sites.” Based on published information on clustering of *P. ramorum* symptoms at 100 to 300 m (Kelly and Meentemeyer 2002), we chose a conservative buffer distance of 400 m. For the COMTF positive infections in Sonoma County, this aggregation process resulted in n=89 sample points and n=34 sample sites.

To investigate the spatial relationship between positive infection locations and fire history, given the two sampling scenarios and three landscape susceptibility scenarios, we tested the null hypothesis that the proportion of infections in previously burned areas is not lower than would be expected by chance. The expected value was based on the proportion of each landscape susceptibility scenario (L1-L3) that had burned since 1950 within the boundaries of Sonoma County. We used a test statistic based on the normal approximation to the binomial distribution, representing the portion of a normal distribution lying beyond a given normal deviate (Zar 1999).

Results and Discussion for Sonoma

Similar to our earlier findings statewide, in Sonoma County we found fewer positive *P. ramorum* infections in areas burned in the relatively recent past (in other words since 1950). When original COMTF sampling points were compared to mapped fire history patterns, there were consistently far fewer infections in burns than expected, based on the proportion of Sonoma County that had burned in the past (top half of Table 1). After aggregating closely spaced sample points into sites, the negative relationship between infection locations and past fires is not as statistically strong; however, the relationship is still weakly significant for landscape susceptibility scenario L3, the most spatially restrictive vegetation screen (last row of Table 1). One caveat worth mentioning is that the relatively low number of observations for sites (n≈30) raises questions about the statistical power of this test (in other words normal approximation to the binomial).

⁵ Primary, secondary, and tertiary refer to the 3 vegetation communities that dominate, in order of their spatial extent, each mapped unit on the landscape.

Table 1—Results for spatial analyses of whether infection locations occur less frequently in previously burned areas than expected; study area is limited to Sonoma County.

POINTS	Landscape area (ha)	Landscape burned (ha)	Proportion (expected)	Infections in scenario	Infections burned	Proportion (observed)	P-value
Scenario L1:	294751	54343	0.1844	85	7	0.0824	0.0067*
Scenario L2:	290666	53933	0.1856	83	7	0.0843	0.0088*
Scenario L3:	245716	50344	0.2049	82	6	0.0732	0.0016*

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Scenario L1:	294751	54343	0.1844	31	4	0.1290	0.2134
Scenario L2:	290666	53933	0.1856	30	4	0.1333	0.2310
Scenario L3:	245716	50344	0.2049	29	3	0.1034	0.0880*

* Significantly lower than expected, $\alpha = .05$ ** Weakly significant, $\alpha = .10$

In comparison to our earlier analyses across a larger area (*fig 1a*), evidence for a negative relationship between SOD and fire history is not as strong in Sonoma County. The weaker statistical results for Sonoma County were to be expected, given that this is the only county in which any COMTF positive infections were found in past burned areas. In a statistical sense, this is at least partially offset by the fact that more of Sonoma County has burned since 1950 (18 to 20 percent; Table 1) than in our earlier study area (13 to 15 percent; Moritz and Odion 2005). Regardless, several of the scenarios analyzed for Sonoma County did still show a significant apparent avoidance of the disease in places that have burned in the last 50 years.

Assumptions and Limitations

We have already described several possible fire-related mechanisms for the observed negative relationship between past fire locations and current *P. ramorum* infections (Moritz and Odion 2005). In short, there are many fire-related factors that can affect the three sides of the plant disease triangle, such as changes in host densities, plant stress levels due to mineral nutrition, or effects of the chemical environment on the pathogen. The observed negative spatial relationship may be caused by fire-related mechanisms, and we have ongoing research in this area. However, it may also be due to something closely correlated with fire patterns and/or an artifact of the datasets themselves. Here we focus on several important caveats about the data used in this study and other possibly confounding factors.

As mentioned above, one concern about the observed relationship between past fires and current infection locations is that it could be caused by a sampling bias with respect to

previously burned areas. We addressed this issue in Moritz and Odion (2005) by examining the overall sampling effort, using a subset of the COMTF sample points from the California Department of Food and Agriculture (CDFA). The CDFA subset contained sampling locations taken for symptomatic plants, regardless of infection status (in other words both positive and negative for *P. ramorum*), and we tested these to see if there had been undersampling with respect to fire history. In our analysis we found that symptomatic samples were being taken roughly as often in past burned areas as one would expect by chance, and even significantly more often in some scenarios. This is based on the assumption that the CDFA data were representative of the overall sampling effort. While this might be additional evidence to support a hypothesis that burned areas are less susceptible to *P. ramorum*, there are additional considerations. If a substantial number of samples found to be negative for *P. ramorum* were erroneous, which may be the case (M. Garbelotto personal communication), then *P. ramorum* could be much more common in areas burned since 1950 than the confirmed positive infection data suggest. Without good estimates of how often one might expect false positives in different parts of the study area, among different host species, and in burned versus unburned areas, it is difficult to assess how strongly this issue may influence findings (in other words by raising the number of additional positive infections in previously burned areas).

Regardless of uncertainty due to inconsistent identification of *P. ramorum* in samples, a CDFA sample testing negative was about twice as likely to have been taken in a past burned area as a positive (data not shown). It is interesting that more sampling may have been done in burned areas (Moritz and Odion 2005), and, for whatever reason, it appears there are many plants that are symptomatic for *P. ramorum* in places that have burned relatively recently. Perhaps the most likely reason for concentrated sampling for SOD in burns and failure to document *P. ramorum* is that much similar mortality may have occurred there as a result of bark beetles, which can increase after fire. In fact, McPherson and others (these proceedings) reported more rapid mortality in bark beetle infested hosts, and the trees they monitored were in or near very recent burn areas. In addition, many symptomatic samples may test positive for other plant diseases (for example, *Phytophthora* spp.), which could be more likely to occur in areas burned since 1950.

A further complication is that human activities that may affect both fire and the disease are not randomly dispersed on the landscape: 1) Fire patterns, in particular the locations of ignitions, can often be associated with the road network; 2) Fire suppression efforts typically utilize the road network and are often strongest near areas of human habitation; and 3) Fire size and frequency can be affected by fragmentation of natural vegetation patterns due to both urban and rural development. Despite our findings of sampling patterns being unbiased with respect to fire history, one might still question whether fire-disease dynamics are somehow strongly driven by human population patterns, road locations, and/or access constraints. Our landscape susceptibility scenarios L1-L3 indirectly address this concern, as they progressively exclude portions of the landscape that may be affected by human activities. The negative relationship between SOD and fire history was consistently statistically significant across these analyses (Moritz and Odion 2005), as were several of the scenarios examined here for Sonoma County.

Conclusions

Just as there is spatial variation in the risk of *P. ramorum* infections based on patterns of host distribution and climatic factors (Meentemeyer and others 2004, Guo and others 2005), there will be natural spatial variation in many different fire regime parameters (in other words fire intensity, size, season, frequency) within and between different ecosystems. The spatial variability of fire at microscales can even be considerable. For example, continuous temperature measurements by sensors less than 0.5 m apart can vary as greatly as among sensors several meters apart, with overall levels of heating at the soil surface varying by over an order of magnitude (Odion and Davis 2000). Factors such as the hydrophobicity of soil, organic matter consumption, and thermal alteration of soil chemistry may, therefore, show considerable fire-related variation at relatively fine spatial scales. Such variation may interface with the life history of *P. ramorum* dispersed to new areas, for example by human activities (Cushman and Meentemeyer, these proceedings). It is also likely that the factors that drive ongoing disease progression in a given location are different from those that may control the initial invasibility of an ecosystem, and fire-related effects on the disease will probably vary by the host species in question. We may therefore expect substantial variation in how a fire-disease relationship manifests itself, depending on the stage of disease progression and the scales of space and time used in examining the landscape pathology of SOD.

Spatial analysis of Sonoma County's fire history and locations of confirmed *P. ramorum* infections from the COMTF database supports our earlier findings of an apparently lower probability of *P. ramorum* in places that have burned in the relatively recent past. Nonetheless, further research is needed to determine whether this relationship is due to a fire-related mechanism, a surrogate of fire, and/or an artifact of available datasets. The differences between prescribed burns and past wildfires must also be understood before advocating fire as a tool in managing *P. ramorum* infections (Moritz and Odion 2004). Little is known about fire-disease dynamics in the ecosystems affected by SOD and the landscape pathology of this disease is likely to be complex.

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