AN ABSTRACT OF THE DISSERTATION OF

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Title: The Epidemiology of Sudden Oak Death in Oregon Forests

Abstract approved:

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The phytopathogen *Phytophthora ramorum* (Werres, DeCock & Man in't Veld), causal agent of Sudden Oak Death (SOD) of oaks (*Quercus* spp.) and tanoaks (*Notholithocarpus densiflorus* syn. *Lithocarpus densiflorus*), is established in coastal forests of the western United States. Since the discovery of SOD in the Douglas-fir / tanoak forests of southwest Oregon in 2001, a multiagency effort has ensued with the goal of fully eliminating *P. ramorum* from this originally small and isolated area. In this study we investigated the epidemiology of SOD in Oregon, particularly as it affects the success of the eradication program. Two approaches were taken to discern the mechanism of long distance dispersal: first, a landscape analysis of the spatial relationship between SOD sites and roads or streams, features associated with movement of infested soils, and, second, a local analysis to discern if understory infection is originating from soil or stream-borne inoculum. Using a restricted randomization test we concluded that SOD sites were no closer to roads than expected by chance, which is inconsistent with soil dispersal by people. While we found evidence that SOD sites were preferentially closer to waterways, inoculum had not moved away from streams into adjacent understory foliage. The local distribution of understory infection around SOD positive trees indicated that primary inoculum is infecting overstory canopies first, suggesting that *P. ramorum* is dispersing in air currents. Regression modeling indicated that weather

conditions two years before detection could explain variation in the maximum distance inoculum moved each year of the epidemic between 2001 and 2010. This two year delay between infection and detection has allowed ample time for infested sites to contribute to further spread. Model results were consistent with observations made the summer of 2011, when trees likely infected by secondary inoculum at non-eradicated sites developed symptoms but were still undetectable by aerial surveys. Due to the prevalence of infection on tanoak, opportunities for sporulation and infection occur more often in Oregon than in California. These data can explain the failure to eliminate *P. ramorum*. Nevertheless, we did find evidence that the eradication program has significantly reduced the potential size of the SOD epidemic in Oregon.

The Epidemiology of Sudden Oak Death in Oregon Forests

by
Ebba K. Peterson

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APPROVED:

Major Professor, representing Botany and Plant Pathology

Chair of the Department of Botany and Plant Pathology

Dean of the Graduate School

I understand that my dissertation will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

Ebba K. Peterson, Author

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The superior teacher demonstrates. The great teacher inspires."
– William Arthur Ward

I dedicate this manuscript, the cumulative product of my time spent
as a student of ecology, to my 7th grade biology teacher, Mrs. Bea Moore,
whose intelligence, quirkiness, enthusiasm and encouragement
inspired me and many others to a life of science.

THE EPIDEMIOLOGY OF SUDDEN OAK DEATH IN OREGON FORESTS

INTRODUCTION AND LITERATURE REVIEW

Phytophthora ramorum (Werres, DeCock & Man in't Veld) is an invasive pathogen currently causing harm to forest communities through the loss of tanoak and other ecologically important trees (Rizzo and Garbelotto 2003, McPherson et al. 2010, Ellison et al. 2005). This pathogen is capable of infecting over 100 species in 35 genera (APHIS 2011), causing non-lethal leaf blight or dieback in most hosts (Rizzo and Garbelotto 2003). On others, particularly tanoak (*Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon, & Oh, syn. *Lithocarpus densiflorus* (Hook. & Arn.) Rehd.) and coast live oak (*Quercus agrifolia* Nee), ***P. ramorum*** causes bleeding bole cankers and eventual tree death (Rizzo and Garbelotto 2003, McPherson et al. 2010). This disease, termed Sudden Oak Death (SOD), was first recognized in the mid-1990s as extensive tanoak mortality in the San Francisco Bay Area, California (Rizzo et al. 2005). It has since spread to forests in 13 coastal counties near San Francisco, CA, and two isolated locations in Humboldt County, CA and Curry County, OR (Rizzo et al. 2005, APHIS 2011).

P. ramorum was described as a new species infecting the branches of rhododendron in Germany and the Netherlands in 2001 (Werres et al. 2001), and was attributed to the decline of western American oaks and tanoaks in 2002 (Rizzo et al. 2002). As a heterothallic oomycete (Kingdom Stramenopila, formerly Protista), ***P. ramorum*** requires two mating types to produce oospores (Brasier and Kirk 2004, Werres and Kaminski 2007). American populations are predominantly the A2 mating type, which is further divided into two distinct lineages, named NA1 and NA2 (Grünwald et al. 2009). The A1 mating type is established in gardens and nurseries of Western Europe, and comprises the third recognized lineage, named EU1 (Grünwald et al. 2009). The NA1 lineage

dominates forest populations, although all three lineages, EU1, NA1 and NA2, have been recovered in American and Canadian nurseries (Goss et al. 2009b, 2011). Despite the presence of both mating types in these environments, there is no evidence for sexual recombination. Most likely, the mating types of *P. ramorum* have been genetically isolated long enough to render them functionally sterile (Goss et al. 2009a). Spore production for this species is thus limited to asexual means, including the production of sporangia or chlamydospores. Infection of hosts occurs through the production of motile, biflagellate zoospores borne from sporangia (Widmer 2009). The dependence of zoospores on water for movement towards infection courts, as well as the general lack of tolerance *Phytophthora* spp. have for dry conditions, have garnered this genus and other oomycetes the common name 'water molds.'

Phytophthora spp. are infamous for being especially aggressive pathogens. Other members in this genus have caused historical losses of important food crops, most notably the Irish potato famine caused by *P. infestans*. Other species are responsible for declines in Port-Orford cedar in southwest Oregon (*P. lateralis*) and the jarrah forests of Australia (*P. cinnamomi*) (Hansen et al. 2000, Cahill et al. 2008). Concerns regarding the extent of oak and tanoak mortality since SOD was first identified have resulted in a collaborative effort to understand and control *P. ramorum* in natural, landscaped, and nursery environments. This work aims to increase our understanding of the epidemiology of SOD, particularly of the epidemic established in Oregon forests.

History of *P. ramorum* in Oregon

Oregon's infestation was first identified in 2001 in the Douglas-fir / tanoak forests outside the coastal town of Brookings (Goheen et al. 2002). This area is characterized by a heterogeneous topography, as well as a variety of disturbances including fire, rural

development, and logging (Hansen et al. 2008). Numerous hosts of ***P. ramorum*** comprise the dominant plant communities in this area, and the Mediterranean climate is conducive to pathogen establishment (Rizzo et al. 2005). In Oregon forests, foliar infections are most readily observed on tanoak and Pacific rhododendron (*Rhododendron macrophyllum* D. Don ex G. Don). Koch's postulates have been completed on many other common native species, notably Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco), coast redwood (*Sequoia sempervirens* (D.Don) Endl.), California bay laurel (*Umbellularia californica* (Hook. & Arn.) Nutt.), big leaf maple (*Acer macrophyllum* Pursh), evergreen huckleberry (*Vaccinium ovatum* Pursh), poison oak (*Toxicodendron diversilobum* (Torr. & A. Gray) Greene), salmonberry (*Rubus spectabilis* Pursh) and madrone (*Arbutus menziesii* Pursh) (APHIS 2011, Hansen et al. 2005). ***P. ramorum*** can also be recovered from other common understory plants including sword fern (*Polystichum munitum* (Kaulf.) C. Presl), Oregon grape (*Berberis diversifolia* Pursh), and salal (*Gaultheria shallon* Pursh) (APHIS 2011, E.Hansen pers. com.). These minor hosts are typically only found infected in Oregon when located beneath symptomatic tanoak, presumably because of the presence of higher secondary inoculum loads produced from tanoak lesions (Hansen et al. 2008). Red alder (*Alnus rubra* Bong.) is also commonly present within the infested area but is not known to be susceptible to ***P. ramorum*** (APHIS 2011).

As an abundant and easily infected foliar and bole host, tanoak plays an important role in the establishment and spread of ***P. ramorum*** in Oregon. Within tanoak's coastal range from Santa Barbara County, CA to southwest Oregon, ***N. densiflorus*** is commonly found in the understory, as a codominant species mixed with emergent conifers, or in dense, pure tanoak stands (Tappeiner et al. 1990). This species also produces prolific basal sprouts at all ages (Tappeiner et al. 1990). Symptoms of ***P. ramorum*** infection on tanoak

typically include bleeding exudates on mature stems, under which lesion lines between healthy and infected tissues of the inner bark are common (Appendix A Fig. A.1, A.2). Foliar infection is usually apparent as dark lesions on the petioles of tanoak leaves, eventually extending into the sprout stem or midrib of the leaf (Rizzo and Garbelotto 2003) (Appendix A Fig. A.3). Bleeding stem cankers and sprout lesions provide the most characteristic indications of *P. ramorum* in the understory, although other *Phytophthora* species, particularly *P. nemorosa* and *P. pseudosyringae*, can cause similar symptoms on tanoak (Rizzo and Garbelotto 2003, Wickland et al. 2008). More characteristic of *P. ramorum* is extensive and rapid death of mature trees due to secondary attack by bark and ambrosia beetles, or water stress and reduced photosynthesis elicited by the presence of the pathogen, resulting in clusters of dead trees with identifiable red crowns (Rizzo and Garbelotto 2003, Parke et al. 2007, Manter et al. 2007) (Appendix A Fig. A.6). This symptom is vital to the monitoring of SOD in Oregon, which relies upon the aerial detection of these dead, overstory tanoaks.

At the time of first detection, the Oregon infestation was confined to a small area and successful eradication was deemed possible (Hansen et al. 2008). A collaborative effort between the Oregon Department of Forestry (ODF), Oregon Department of Agricultural (ODA), Oregon State University (OSU), and USDA Forest Service (USDA-FS, including Forest Health Protection and the Pacific Southwest Research Station) agencies ensued with the goal of fully eliminating *P. ramorum* from Oregon forests (Goheen et al. 2002). Under the eradication program, aerial surveys have been performed multiple times each year to identify recently deceased tanoak. Follow up ground surveys confirm the coordinates of potentially infected trees with a global position system (GPS) and collect samples of inner-bark, leaves and stems with characteristic symptoms of *Phytophthora* infection.

Symptomatic trees have been identified as positive for *P. ramorum* via culture or molecular diagnosis with PCR primers developed by Winton and Hansen (2001). After lab identification, SOD sites have been extensively surveyed and treated. Eradication protocol initially required the removal of all main hosts, usually accomplished with cutting and burning, within a buffer of 15 to 30 m away from the furthest symptomatic tree confirmed infected by *P. ramorum* (Hansen et al. 2008). Tanoaks with latent infection remained at the edges of eradicated areas, however, and subsequently the minimum treatment distance was increased to 100 m (Hansen et al. 2008). While most of the vegetation at the periphery of the eradication zone is not infected, this distance has proven effective at eliminating those few trees that had been exposed to local spread but were asymptomatic at the time of surveys. With support of the landowner, eradication boundaries have been greater at some locations.

Due to the rigor of aerial and ground surveys, and relative speed at which sites have been detected and treated, the eradication program has thus far prevented a regulatory quarantine of all of Curry County. Instead, quarantine boundaries have been defined around areas of known infection, and have grown only as the geographic distribution of *P. ramorum* has increased (Fig. 1.1) (Kanaskie et al. 2008, Hansen et al. 2008). Movement of inoculum to the edges or outside of the quarantine boundaries is relatively rare, however these long distance dispersal events have presented a real obstacle in controlling this epidemic.

New infestations have been detected every year since the eradication program started in 2001. Most new positive trees are found within 300 m of a confirmed positive site from any prior year, though some have occurred up to 4.2 km from the nearest known inoculum source (Hansen et al. 2008). For the first five years of the eradication program

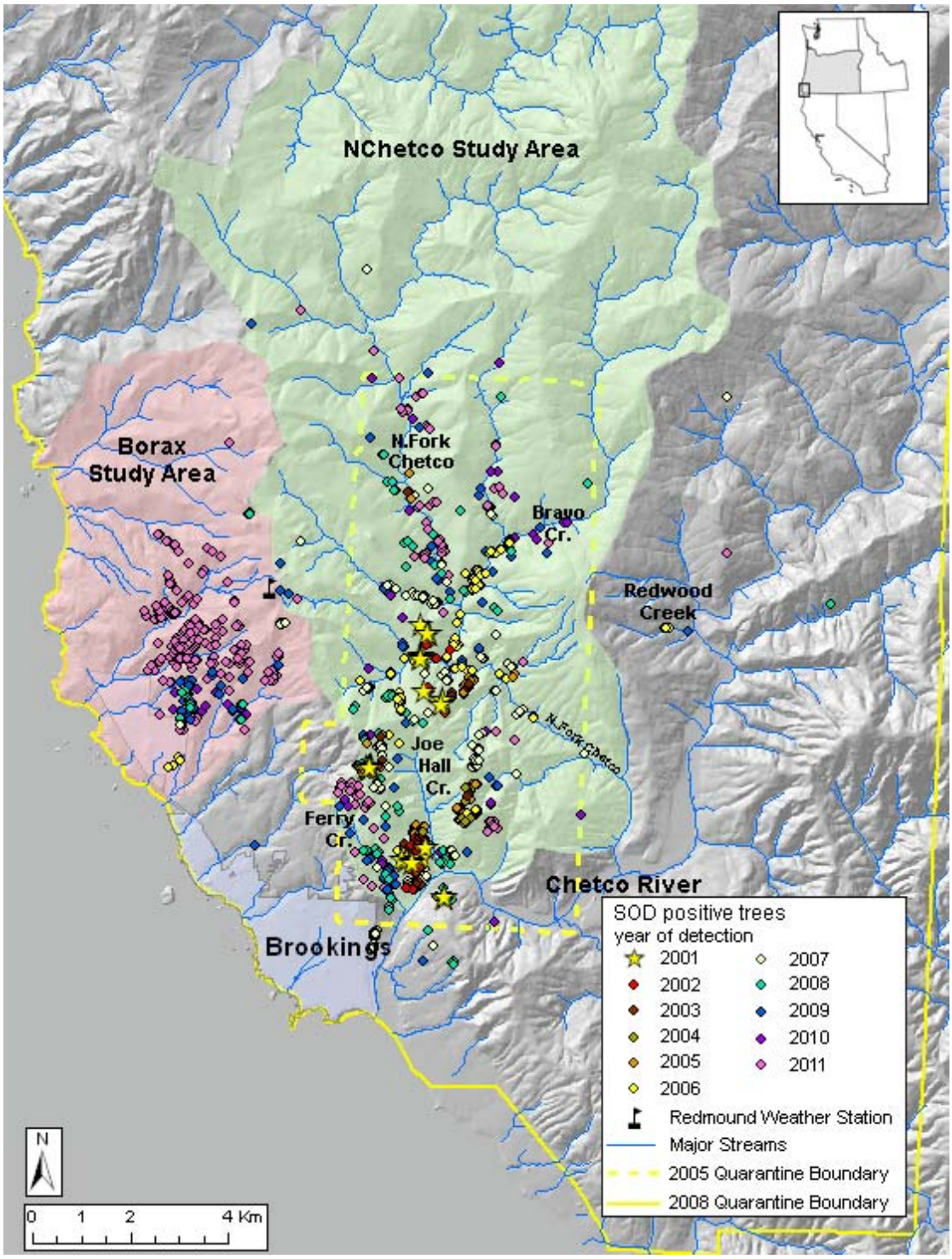


Fig. 1.1. Study region, showing all SOD positive trees identified between 2001 and 2011 within the 2008 quarantine zone, as well as the boundaries of the two study areas used in our spatial and temporal analyses: the North Fork Chetco, Joe Hall Creek and Ferry Creek watersheds (NChetco study area), and the infestation originating from the Borax site, first identified in 2006 (Borax study area).

P. ramorum was limited to the areas north of the original infections, especially within the watershed of the North Fork Chetco River and its tributary Bravo Creek (Fig. 1.1).

While five of the original nine sites detected in 2001 were located on small private parcels, most of the long distance spread has been up this drainage system (Fig. 1.1). Much of the North Chetco watershed is networked with dirt and gravel roads maintained by the South Coast Lumber Company (SCLC), on whose lands a significant amount of infection has been identified. Co-mixed with private SCLC property and accounting for most of the remaining infestation in the North Chetco watershed are lands owned by the USDI Bureau of Land Management (BLM). Despite model predictions of a near homogenous host distribution and climate suitability north and south of the originally infected area (Václavík et al. 2010, Kelly et al. 2007), *P. ramorum* has not spread significantly south of the Chetco River (Fig. 1.1).

Long distance dispersal events have surpassed the North Chetco watershed to Redwood Creek in the east and the Borax site in the west, both first identified in 2006 (Fig. 1.1). Due to the lack of genetic diversity amongst populations, the tracking of distinct *P. ramorum* genotypes has relied upon an analysis of microsatellite markers (short-sequence repeats, Ivors et al. 2006) Microsatellite analysis of Oregon's *P. ramorum* population has shown both the Redwood and Borax infestations are part of the same clonal population common to the North Fork area. Whereas multiple distinct populations comprise the infestation in Californian forests (Mascheretti et al. 2008), 66% of isolates recovered in Oregon forests between 2001 and 2004 have been a single multilocus genotype, PrOR1 (NA1 lineage, Prospero et al. 2007). The remaining isolates are all closely related and are of the same clonal line (Prospero et al. 2007). PrOR1 is unique to Oregon and is distinct from any populations recovered from nurseries or California forests, supporting a

hypothesis of a single introduction into Oregon forests (Prospero et al. 2009, Mascheretti et al. 2008). PrOR1 has continued to dominate the Oregon landscape, and no new major populations have yet been recovered (Britt and Hansen 2011). How primary inoculum reached the Redwood, Borax, and far northern sites from the original 2001 infections is unknown. The appearance of these long distance dispersal events has required a reevaluation of the feasibility of eradication, especially given the delay between initial infection and detection during which time dispersal is likely. These events have also highlighted our general lack of understanding of the dispersal dynamics of *P. ramorum* in natural ecosystems, a hindrance to all SOD control programs.

Dispersal of forest *Phytophthora* spp.

P. ramorum is a prolific producer of asexual spores in culture and *in planta* (Werres et al. 2001, Davidson et al. 2005, 2008). While chlamydospores are produced in abundance within plant tissues, because it is difficult to assess their viability their epidemiological role is unknown. Rather, the majority of SOD spread within forests has been attributed to sporangia dispersal. Sporangia of *P. ramorum* are caducous and are borne only from infections on twigs and leaves of foliar hosts, and may germinate directly or release motile zoospores to initiate new infections (Werres et al. 2001, Davidson et al. 2005, 2008, Tooley and Browning 2009, Denman et al. 2006). Davidson et al. (2005) documented recovery of inoculum from rain splash 15 m away from the nearest foliar host, though wind-driven rain may spread sporangia further distances. The mechanism of long distance dispersal, that is, the dispersal of inoculum into new stands without prior exposure to *P. ramorum*, is less well understood.

Current evidence from Californian studies suggests that soilborne inoculum can influence epidemic dynamics in natural ecosystems, either as a survival mechanism over the

summer or through human-assisted dispersal. During wet months, *P. ramorum* has been recovered from soils rinsed from the shoes of hikers leaving infested areas and from recreational trails in areas located near infested areas but lacking foliar hosts (Davidson et al. 2005, Cushman and Meentemeyer 2008). *P. ramorum* has also been recovered from Oregon soils after eradication of hosts has been completed (Goheen et al. 2008). Cushman and Meentemeyer (2008) rarely recovered *P. ramorum* further than 1 m from trails, indicating that human activity is not responsible for the movement of large amounts of inoculum off trails. Nevertheless, rain splash from inoculum-bearing soils can initiate infections on understory plants and low branches, from which it could spread into a stand (Fichtner et al. 2009). This indirect evidence has led to the hypothesis that *P. ramorum* is dispersed between stands in soils, transported predominantly by people (Cushman and Meentemeyer 2008, Davidson et al. 2005, 2008, Fichtner et al. 2007, 2009).

Associations between *Phytophthora* species and soils are well documented in forest and agricultural environments. Following the emergence of invasive *Phytophthora* spp. and their potential economic and environmental impacts, researchers have begun surveying soils for *Phytophthora* diversity. *P. parasitica, P. sojae, P. cinnamomi*, *P. lateralis* (sister species to *P. ramorum*, Ivors et al. 2004), *P. cambivora*, *P. cactorum*, *P. "citricola"*, *P. megasperma*, *P. palmivora, P. syringae, P. europaea, P. pseudosyringae*, *P. quercina, P. gonapodyides, P. uliginosa, P. cryptogea,* and *P. siskiyouensis* are examples of agricultural and forest *Phytophthoras* known to be present in soils, either as root pathogens or as inoculum that splashed from foliar infections into soils (Ristaino and Gumpertz 2000, Hansen et al. 2000, Jeffers and Aldwinckle 1988, Graham et al. 1998, Greslebin et al. 2005, Jung et al. 2003, Balci and Halmschlager 2003, Vettraino et al. 2002).

Similarly, *Phytophthora* species can be easily recovered from surface waters into which spore-bearing soils or vegetation have been introduced (Reeser et al. 2011). This is the basis for stream monitoring programs focusing on water bait sampling as an early indication of *P. ramorum* presence in the waterways around Brookings (Sutton et al. 2009). Movement of inoculum in streams or soils along trails or roads contributes to the spread of many *Phytophthora* species (Ristaino and Gumpertz 2000). As a result, in natural ecosystems disease at the landscape scale is typically spatially associated with roads, or downstream of where roads cross waterways (Jules et al. 2002, Kauffman and Jules 2006). These patterns have been especially apparent early in an epidemic when inoculum is originally introduced into a new area (Jules et al. 2002). It is unclear if the movement of infested soils can explain the dispersal patterns of *P. ramorum* in Oregon, especially if researchers have missed a significant amount of understory infection due to the dependence on overstory mortality as an indication of SOD presence.

Hansen et al. (2008) have postulated that aerial dissemination of sporangia can explain the long distance dispersal of *P. ramorum* in Oregon, as is possible for other agricultural oomycetes (Ristaino and Gumpertz 2000). As with soil movement, the hypothesis of aerial dispersal relies on indirect evidence. In Oregon, *P. ramorum* has moved considerable distances in areas of relatively minimal public access not easily explained by soil movement. The pattern of SOD on the landscape over the first seven years of the epidemic is also comparable to a disease gradient expected from aerial dispersal (Hansen et al. 2008). No direct evidence for aerial dispersal of *P. ramorum* has yet been observed, however *P. ramorum* does possess deciduous sporangia common to other aerially dispersing oomycetes (Werres et al. 2001). If confirmed, the potential for aerial dispersal will affect modeling of the long term development of the SOD epidemic and poses a

significant risk to forests in the vicinity of infested stands, especially as the size of the infested area is allowed to grow (Aylor 1999, Kot et al. 1996).

Concluding remarks and objectives

Once established, *P. ramorum* has proven difficult to manage. As the only host known to both support sporulation and develop SOD symptoms, tanoak is particularly difficult to protect from infection. Unfortunately, with plot-level tree mortality up to 100% this pathogen will change western forests (Davis et al. 2010). Potential impacts from the loss of tanoak include the loss of a food source for mammal, bird and mycorrhizal communities if alternative sources are absent, as well as changes in nutrient cycling and forest succession (Monohan and Koenig 2006, Bergmann and Garbelotto 2006, Cobb et al. 2010, Waring and O'Hara 2008). *P. ramorum* positive nurseries, as well as horticultural and forestry businesses in APHIS regulated counties, face increased operation costs from control and quarantine requirements (Dart and Chastanger 2007, Frankel 2008). The mortality associated with SOD also reduces land values in the urban-wildland interface due to lost aesthetics and changes in fire loading expected to exacerbate wildfire risk (Kovacks et al. 2011, Valacovich et al. 2011). Major gaps in our knowledge regarding long distance dispersal mechanisms of *P. ramorum* have hindered the success of the eradication program. Additionally, due to lack of a comparable control infestation we have been unable to quantify the extent to which the eradication has succeeded in reducing the potential extent of the SOD epidemic.

The primary objective of this study was to assess the spatial and temporal dynamics of the SOD epidemic in Oregon, especially as they pertain to mechanisms of long distance dispersal and the reductive effects of the eradication program. Additionally, we aimed to

describe ways in which the Oregon epidemic differs from that described in California, where the majority of epidemiology has been studied. Specifically, our objectives were:

1) Identify spatial patterns of infection indicating areas of increased risk of exposure to inoculum, and assess if these patterns are consistent with the movement of inoculum in soils.
2) Discern if the years in which the SOD epidemic greatly expanded can be explained by weather conditions during the year of primary inoculum production through the modeling of epidemic development, and to quantify the effect that a delay in eradication has had upon the extent of the infestation.
3) Determine if the timing of sporulation and symptom development are similar to that observed in California, with an emphasis on discerning the relative importance of tanoak and California bay laurel for the initial establishment of primary inoculum.

We hypothesized that spatial patterns of SOD are inconsistent with soil dispersal, as an indication of an aerial mechanism. Given an understanding of the environmental variables driving this epidemic, we hypothesized that during years of prompt eradication this program has reduced both the size of infested areas and potential dispersal distances. Any attempt at landscape-level management relies upon an assessment of where the highest risk is located, which inherently requires an understanding of all modes of long distance dispersal and other factors driving epidemic development. *P. ramorum* has spread with relatively minimal management in most of its range for over ten years, yet significantly large areas have yet to become infected (Rizzo et al. 2005). These data will aid the direction of limited resources to guide future management of *P. ramorum* in Oregon and Californian forests.

SPATIAL ANALYSIS OF SUDDEN OAK DEATH IN OREGON

INTRODUCTION

An understanding of the dominant dispersal mechanisms responsible for the spread and establishment of a pathogen is an essential component of any disease management system (Ristaino and Gumpertz 2000). For *P. ramorum*, short distance (up to 15 m) dispersal in rain splash has been well documented (Davidson et al. 2005). Nevertheless, the distances between new SOD sites in Oregon indicate that different dispersal mechanisms are responsible for disease spread at different scales. Long distance dispersal (LDD) within Oregon forests can likely be attributed to either the movement of soil on vehicles, boots and equipment, or the transport of spores in wind currents (Hansen et al. 2008). The later mechanism is unprecedented amongst forest *Phytophthora* species leading most researchers to focus their efforts on the epidemiological importance of soil movement. The spatial relationship between SOD sites to potential soil introduction points has yet to be investigated, but may indicate if roads or streams are providing opportunities for the movement of inoculum. We took two approaches to assess if soil movement can explain the dispersal of *P. ramorum* in Oregon forests: first, a landscape analysis to discern if SOD sites are closer to roads or streams than expected by chance, and, second, a local analysis to discern if understory infection originates from soil or stream-borne inoculum.

Landscape spatial patterns of *Phytophthora* species

The analysis of spatial patterns of disease is one approach used to discern which aspects of host, environment or pathogen heterogeneity are contributing to the development of an epidemic, and often the observed patchiness in pathogen distribution (Ristaino and Gumpertz 2000). Dispersal of inoculum from a source is inherently a spatial process, and thus is one major aspect contributing to the development of spatial structure of an

epidemic. As such, spatial pattern is often the measure targeted by studies seeking to understand the sources and spread of inoculum, especially as a means to prevent further establishment of a pathogen. The collection of spatial data is typically a labor intensive process, and relatively few studies have quantitatively assessed spatial structure in order to discern dispersal mechanisms. Those that have been performed typically focused on agricultural diseases, where heterogeneity in all aspects of the disease triangle model is more easily controlled (e.g. Larkin et al. 1995, Ristaino et al. 1994, Zwankhuizen et al. 1998, Jaime-Garcia et al. 2001).

Few studies have investigated the spatial structure of epidemics caused by *Phytophthora* spp. in heterogeneous, forest environments. Of those species known to be invasive in forest ecosystems, *P. lateralis*, *P. cambivora* and *P. cinnamomi* are the best studied examples. *P. lateralis* is an invasive pathogen of Port-Orford cedar (POC) in northern California and southwestern Oregon (Hansen et al. 2000). First identified in forests in 1952, *P. lateralis* has readily colonized riparian areas post introduction (Hansen et al. 2000, Jules et al. 2002). In contrast to the host specificity and restricted geographic range displayed by *P. lateralis*, *P. cambivora* and *P. cinnamomi* are generalists with a larger geographic distribution (Cahill et al. 2008, Jung et al. 2009, Saavedra et al. 2007, Balci et al. 2007, Vettraino et al. 2005). These species have been documented causing declines alone or in a complex with other *Phytophthora* species in many ecosystems, notably in the jarrah forests of Australia and of hardwoods in Europe and the United States (Cahill et al. 2008, Jung et al. 2009, Saavedra et al. 2007, Balci et al. 2007, Vettraino et al. 2005).

Soil movement significantly contributes to the spread of all three species, where roads and streams are the most common pathways contributing to the introduction of inoculum into new areas. As such, disease most often occurs closer to roads or waterways (Jules et

al. 2002, Kauffman and Jules 2006, Vannini et al. 2010, Weste and Taylor 1971). Typically, early studies identify these spatial patterns (e.g. Hansen et al. 2000, Saavedra et al. 2007), although additional work quantifying the degree of spatial structure is required to verify or refute these observations. Mortality caused by ***P. lateralis*** is associated either with roads, or, as a result of inoculum being introduced at stream crossings, downstream of where roads cross waterways (Jules et al. 2002). Correspondingly, proximity to roads and streams, or greater road density and use, increases the risk of exposure to ***P. lateralis*** inoculum (Jules et al. 2002, Kauffman and Jules 2006, Clark 2011). Similar results have been recorded for ***P. cambivora***, where disease severity and rates of tree mortality decrease as one moves away from water drainages located in areas where human activities are limited (Vannini et al. 2010).

Despite recent studies documenting the presence of ***P. ramorum*** in soils and waterways in Oregon (Goheen et al. 2008, Sutton et al. 2009, Reeser et al. 2011) and California (Davidson et al. 2005, Cushman and Meentemeyer 2008), it remains unclear if these pathways present a risk for the long distance dispersal of this pathogen in natural ecosystems (Hansen et al. 2008). Current evidence from California suggests that disease prevalence is related to the movement of infested soils by people, whereby greater public access may be responsible for increasing inoculum loads and risk for establishment of ***P. ramorum***. For example, Cushman and Meentemeyer (2008) have found that the severity of bay laurel infection in wooded areas increases with greater public access and increasing population density. Similarly, infection in Oregon is more prevalent in areas with a higher proportion of roads and public access, especially around Joe Hall Creek and Ferry Creek (Fig. 1.1). The severity or incidence of SOD, however, can be significantly augmented by changes in forest composition, connectivity or microclimate (Meentemeyer et al. 2008b, Ellis et al. 2010), as well as disease gradients away from the areas where

inoculum was first introduced (by any means). This makes the deduction of dispersal mechanism from disease patterns problematic, especially when the site of introduction cannot be adequately determined, as in most of California. In contrast, the early detection of SOD in Oregon allows us to conclude that *P. ramorum* was first introduced into the forests around Joe Hall and Ferry Creek. Subsequently we would expect to see greater establishment in these drainages, irrespective of the mechanism of long distance dispersal (Fig. 1.1).

Heterogeneity must be included in spatial models to make meaningful interpretations of spatial patterns (Fortin et al. 2000). For example, while Jules et al. (2002) was the first study to analyze the association between *P. lateralis* and stream crossings, other sources of heterogeneity influenced patterns of disease in relation to streams (Kauffman and Jules 2006). While proximity to streams was a strong determinate in infection probability, variation in tree size also significantly predicted pathogen spread. Larger POC trees, in this case, had roots reaching further from upslope locations into waterways, resulting in mortality of trees further away from streams amongst larger cohorts (Kauffman and Jules 2006). Heterogeneity in exposure to inoculum resulting from the initial introduction of *P. ramorum* in the southern end of its range confounds conclusions made on observation alone. For example, we are unable to determine if the severity of SOD in the southern range of the North Chetco-Joe Hall-Ferry Creek watersheds can be attributed to dispersal along roads, or if this pattern is a result of the chance introduction and subsequent intensification of *P. ramorum* in this area. The challenge, therefore, is to assess the distribution of *P. ramorum* for spatial dependence upon roads or streams, while retaining the overall landscape distribution resulting from this pathogen's history in Oregon.

Our approach assesses if the distribution of *P. ramorum* deviates from spatial independence to roads or streams by using a regionally restricted randomization test that retains the overall south to north distribution of this epidemic in Oregon. If the long distance dispersal of *P. ramorum* in Oregon forests can be explained by the movement of infested soils by vehicles, we hypothesize that SOD will occur closer to roads or streams than would be expected by chance. Lacking patterns expected of soil dispersal, we propose alternative methods are contributing to long distance movement, including aerial dispersal as one potential mechanism.

Understory infection

Dominant soil mediated dispersal mechanisms require that in early stages of local establishment, understory infection at the ground level must precede canopy infection. The landscape pattern of SOD in relation to roads and streams is inherently a process describing the mortality and detection of overstory, mature tanoak trees. Little is known about the importance of understory infection for the spread and establishment of *P. ramorum* in Oregon, especially on hosts where infection cannot be detected in aerial surveys, such as California bay laurel. Davidson et al. (2005) documented a strong dispersal gradient from infected trees resulting from local spread in rain splash, with consistent recovery of *P. ramorum* only up to 10 m from infected canopies. Provided we can assess the distribution of infection in the understory during early stages of local spread, we expect to see similarly strong dispersal gradients around the point of inoculum introduction. This provides a platform to discern whether understory infection indicates origins in the understory resulting from exposure to inoculum in soil or streams (a 'bottom-up' pathway) or from above as a result of aerial dispersal (a 'top-down' pathway) (Fig. 2.1). If exposure to understory inoculum in soils can explain the

a. bottom-up pathway

b. top-down pathway

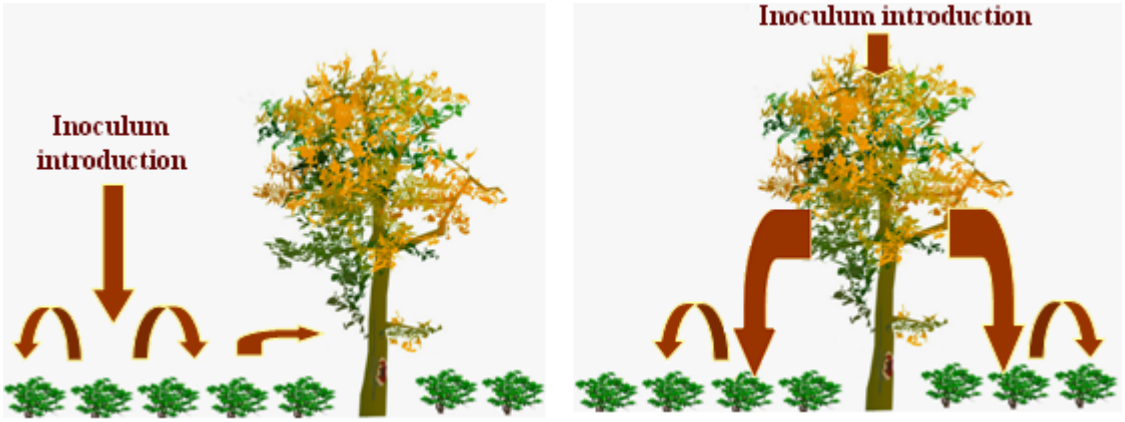


Fig. 2.1. Diagram illustrating the spatial pattern in understory foliage expected from soil dispersal (a, bottom-up pathway), or aerial dispersal (b, top-down pathway). Illustration adapted from Parke and Lucas 2008.

movement of *P. ramorum*, then we should observe understory infection occurring in patterns independent of overstory mortality; if exposure to stream-borne inoculum can explain the movement of *P. ramorum*, then we should observe understory infection occurring closer to streams. Assessing patterns of understory infection furthermore allows us to challenge an assumption central to the eradication program: the distribution of *P. ramorum* in Oregon can be detected and described by overstory mortality.

The extent of understory infection is not well documented and is difficult to measure. Due to the large quarantine area and relative rarity of *P. ramorum*, random understory surveys performed as part of the early detection effort are unlikely to find infection in the absence of overstory mortality. Instead, surveys of infected areas before extensive secondary spread allows for an opportunity to assess the likelihood of soils or streams being the primary source contributing to LDD. Understory dynamics were investigated in two studies: the first focusing on the role stream-borne inoculum plays in the spread of

P. ramorum, and the second identifying the distribution of understory infection in relation to SOD positive, overstory tanoak.

Distribution of infection in relation to streams

Stream detection is an important component of the early detection protocol for the SOD eradication program, particularly in watersheds on the periphery of the North Chetco watershed (Hansen et al. 2008, Sutton et al. 2009). Mesh bags of two leaves each of rhododendron and tanoak are floated in waterways for two weeks as a bait for motile *Phytophthora* zoospores. The presence of *Phytophthora* species on these leaves is determined both by isolation on selective media and by PCR (Sutton et al. 2009). These baits have yielded numerous *Phytophthora* species of which *P. gonapodyides*, *P.* taxon Pgchlamydo and *P. nemorosa* have been the most common, although recovery varies seasonally for most species (Reeser et al. 2011, Sutton et al. 2009). In streams draining from areas with an abundance of forest infection, *P. ramorum* can be recovered in all seasons of the year (Sutton et al. 2009).

Movement of inoculum from soils or foliage into waterways can contribute to the recovery of *P. ramorum* in stream baits. Despite the presence of *P. ramorum* in streams, however, it is unknown if this pathogen causes significant infection in streamside foliage, or if streamside infection is spreading away from waterways on adjacent understory plants. Lateral movement of *P. ramorum* away from streams could easily escape detection, especially if mature tanoaks are not located streamside to provide an opportunity for overstory identification. To assess this risk, we performed surveys to measure host and *Phytophthora* diversity along waterways downstream of previous SOD sites. In addition to these along-stream surveys, we also assessed if hosts and

Phytophthora spp. are present away from streams, as means to assess the likelihood of lateral spread of *P. ramorum*.

Movement of *P. lateralis* away from streams is a rare event relative to the abundance of streamside infection of POC (Jules et al. 2002, Kauffman and Jules 2006), and we hypothesize the movement of *P. ramorum* may be similarly uncommon. The expected presence of a disease gradient away from streams is consistent with the current distribution of *P. ramorum* in Oregon, given the infrequency in which new positive sites have been located along long infested waterways. Provided hosts are equally abundant adjacent to streams as away from streams, if stream-borne inoculum can explain the LDD of SOD, then *P. ramorum* will be recovered more often in streamside foliage than in foliage away from the stream edge due to increased risk of exposure to inoculum. In contrast, recovery of *P. ramorum* in vegetation preferentially away from streams, or only in close proximity to dead, overstory tanoak is an indication that stream-borne inoculum can be attributed to the pathogen moving only from upslope infections.

Distribution of infection in relation to overstory mortality

Infection originating from soils would produce patterns in the understory consistent with a bottom-up pathway, whereby secondary inoculum would spread focally in the understory from the point of introduction. This pattern would be difficult to assess without prior knowledge about where primary inoculum was introduced (Fig. 2.1a). Alternatively, primary inoculum may be aerially dispersed and infect the canopy of mature trees before being dispersed locally in rain splash. This second, top-down pathway is particularly testable, as it presumes the source of secondary inoculum responsible for understory infection is produced in tanoak canopies (Fig. 2.1b). In this case the greatest amount of understory infection should be closest to the source, the first

infected overstory tree. As an indication of aerial dispersal, we hypothesize the incidence of *P. ramorum* in the understory is spatially dependant on the overstory tree or clump of trees with the most advanced symptoms at a given site, presumed to be the 'first' trees infected and the source of secondary inoculum for that locale.

Justification and objectives

Control measures for soil-borne pathogens often focus on reducing the spread of infested soils, including closing roads at times deemed high risk for soil movement (e.g. *P. lateralis*, Hansen et al. 2000), or through the use of vehicle and equipment washing stations (e.g. *P. cinnamomi*, Cahill et al. 2008). Similar measures are suggested by California regulators for *P. ramorum*, whereby the public is advised to stay out of areas of wet soils, and clean personal clothing and equipment of soils when entering or leaving infested areas (Cushman and Meentemeyer 2008, California Oak Mortality Task Force (COMTF) website). It remains unclear if these measures have prevented the spread of *P. ramorum* between stands within California, or if road closures could have prevented the rare long distance dispersal events observed in Oregon.

The objective of this study is to discern if the movement of *P. ramorum* in Oregon forests is spatially dependant upon roads or streams, landscape features that are associated with the movement of soil-borne inoculum, particularly over long distances. These associations, however, must also be supported by the patterns of infection at smaller scales. That is, we would expect to see stream-borne inoculum to be causing infections closer to streams, or understory inoculum causing infections independent of overstory trees. Regardless of the mechanism, however, any association to landscape or local features may identify areas with a higher risk for establishment and priority for treatment.

METHODS

Landscape analysis: spatial dependence on roads and streams

Confirmation of SOD locations. Geographic coordinates for *P. ramorum* positive locations were recorded as part of the SOD eradication program. As part of the early detection protocol, dead tanoak trees are located in aerial surveys. Ground crews then locate identified trees, verify cause of death, and, when *P. ramorum* positive, extensively survey the area. For this study we used all *P. ramorum* positive coordinates (representing dead and symptomatic trees) identified between 2001 and 2010 within the original Ferry Creek, Joe Hall Creek and North Fork Chetco River drainages (hereafter called the NChetco study area), where the infestation is most extensive (Fig. 1.1).

All positives trees in close proximity were reduced to a single site coordinate defined as the centroid of all isolations located within 60 m of one another (Appendix B Fig. B.1). This minimizes the bias of over-sampled locations, and approximates the point of primary inoculum introduction. The use of the centroid instead of the point closest to the road or stream to describe the spatial dependence with these landscape features was selected for ease of computation. A subset of data using the point closest to the nearest road or stream was used to test validity of this method without significantly altering our results (data not shown). From the 709 positive trees within the NChetco study area we defined 294 sites for analysis (Table 2.1)

Table 2.1. Spatial relationships by year for all sites identified as positive for *P. ramorum* infection within the NChetco Study Area. A site was defined as the centroid of all trees located with 60 m of one another. Median distance to road or stream was calculated as horizontal distance to the closest road or stream for all sites in that year, or as the 10 year median for all sites detected between 2001 and 2010.

year of detection	# of new SOD positive trees	# of new sites	median distance to road (m)	median distance to stream (m)
2001	50	10	102	128
2002	79	17	73	94
2003	56	18	111	102
2004	28	10	62	160
2005	60	17	124	65
2006	128	46	111	45
2007	132	61	96	44
2008	79	50	104	89
2009	68	44	93	59
2010	29	21	141	91
All years:	709	294		
10 year median:			100	71

Topographical and landscape features. Using a 10 m digital elevation model obtained from the USDA National Resources Conservation Service (http://datagateway.nrcs.usda.gov/), we generated a stream network with the program tauDEM 5.0 (http://hydrology.usu.edu/taudem /taudem5.0/index.html). This was accomplished with the 'Stream Definition by Threshold' tool whereby a raster cell was classified as a waterway if it had a minimum contributing area of 300 upslope grid cells (threshold = 300). The resultant stream rasters were then converted to vector format and screened for anomalies before analysis. Road layers were obtained from the POC-GIS regional distribution maps compiled on September 6th, 2006 (source: E. Hansen). Of importance were the maps relating to the Rogue River Siskiyou region which includes

most logging roads and all major roads within this region. Roads not in this dataset but present in aerial photographs in 2005 or 2009 were manually added in ArcMap.
All datasets were projected in the OR NAD83 Lambert coordinate system and analyzed in ArcGIS (version 9.3; ESRI). To quantify the minimum distance of each of the 294 SOD sites to the nearest road or stream a spatial join was performed relating points (site) to each line feature (either roads or streams). This analysis created a field containing the minimum Euclidean distance between the point and the closest line. Twenty-five points were checked manually with the distance tool to verify accuracy.

Statistical analysis. We performed a restricted randomization procedure to test the null hypothesis that sites are no closer to roads or streams than would be expected by chance (spatial independence). A random data set was constructed in ArcMap with the goal of obtaining the overall south to north distribution of observed *P. ramorum* sites in order to account for the differences in exposure to the pathogen and variation in road or stream density as one moves north in the NChetco study area (Appendix B Fig. B.2, Fig. B.3). Random points were generated separately within 1-km wide regions spaced horizontally throughout the study area; the proportion of points created was identical to the proportion of SOD sites found in each region (Appendix B Fig. B.3). The total number of random points created over the entire NChetco study area was equal to the number of sites present multiplied by 5,000, generating 1.47×10^6 random points total. The distance of each random point to the nearest road and stream was calculated with a spatial join as with the true dataset.

All distances were compiled in Excel, then imported into MATLAB. Using code implemented in MATLAB we sampled 294 distances (each representing the distance of a random point to the nearest road) from the random dataset, then calculated the median

distance to the nearest road (Appendix B Fig. B.3, Fig. B.4). Median distance was preferred over the mean in order to reduce the effect of extreme outliers present in the random dataset. Due to computational constraints no attempt was made to enforce a minimum distance between random points. This was repeated 10,000 times, and all statistics were tabulated to generate a distribution of median distances to roads expected under the null hypothesis of spatial independence (Appendix B Fig. B.4).

Statistical likelihood of observing the true median distance under randomness was computed with a 1-tailed randomization test where pseudo-p = k/N; k = the number of random data sets which had median distances less than or equal to the true median distance to roads (those with a median distance closer than observed), N = the total number of randomizations performed (Manly 1991). An identical process using the same initial dataset of 1.47×10^6 random points, but a new set of 10,000 randomizations was performed to assess the spatial dependence of *P. ramorum* to streams.

Distribution of infection in relation to streams

To assess the risk of stream dispersal we hypothesized that for understory infection to be contributing to the movement of *P. ramorum* out of streams, two conditions must be met: first, foliar hosts must be present both adjacent to and away from waterways; second, *P. ramorum* must be recovered more commonly from hosts within the splash or flood line than in foliage away from the primary inoculum source. If we failed to recover *P. ramorum*, we furthermore must demonstrate that other *Phytophthora* spp. were capable of causing infection on stream associated hosts.

Survey location selection. Surveys were performed along major and minor streams known to contain *P. ramorum* inoculum. Starting locations preferentially included water bait stations established as part of the early detection network. To be considered, a bait station must have had either a *P. ramorum* culture positive, or, when culture negative, a PCR positive and known *P. ramorum* site at any location upstream. To increase our sample size and ensure equal representation of landowner (public or privately owned by SCLC) and stream size (main or side), additional random locations within the study area were used. These locations were along the same waterways as the bait stations and were downstream of recent infection. Potential starting points were excluded if we lacked the ability to survey 200 m without encountering a commercial clear cut or known active infection. Locations were also not surveyed if more than half the length of the survey would pass through an eradicated area on both sides of the stream. As this study was performed in areas at relatively high risk for *P. ramorum* infection and during the time of aerial surveys, July and August 2011, some transects were later confirmed to have infection on uphill vegetation after the surveys were completed.

Two sets of transects were completed at each of the 15 surveyed locations. The first comprised a survey of the understory and overstory vegetation along the stream (hereafter called the 'main transect'); the second focused only on major foliar host species located away from the stream ('side transect'). Additionally, any tanoak observable from the stream that displayed crown dieback or fading was inspected for symptoms of *P. ramorum* infection. When symptoms were present we sampled foliage and cankers and recorded the tree's location with a GPS.

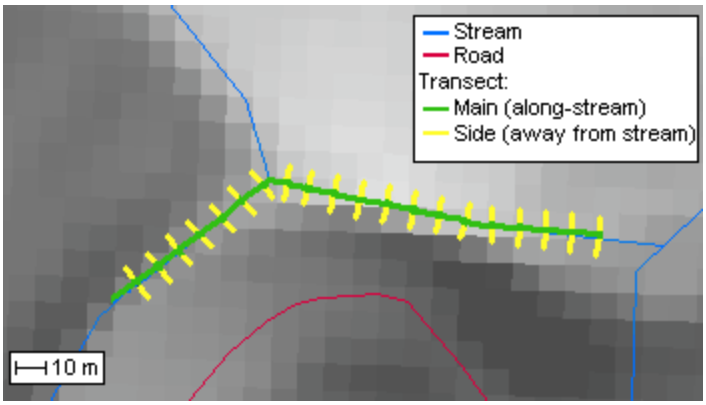


Fig. 2.2. Diagram demonstrating the configuration of main and stream transects used to assess host and *Phytophthora* spp. presence within or outside of the stream splash or flood line.

Main transect methods. To assess vegetation and pathogen abundance in streamside foliage, at each starting location we ran a 100 to 200 m long transect, total length depending upon the topography. In 10 m intervals the presence or absence of all major riparian plant species was recorded if its canopy fell within 2 m of the waters' edge (applicable to smaller streams) or bank (applicable to larger streams with gravel bars due to seasonal flooding) (Fig. 2.2). The species recorded included: understory tanoak, bay laurel, evergreen huckleberry and rhododendron, and overstory tanoak, red alder, big leaf maple and Douglas-fir. Despite the potential for asymptomatic infection, prior sampling in SOD sites before and after eradication have shown that *P. ramorum* is preferably isolated from symptomatic tissues, especially from key foliar hosts. In this study, we collected up to five symptomatic leaves each from tanoak and bay laurel as a means to detect *P. ramorum* or other *Phytophthora* spp.

Side transect methods. At each 10 m interval along the main transect, we started additional 2 m wide, 5 m long transects perpendicular to the stream (Fig. 2.2). 5 m long transects were performed on both sides of the stream and were corrected for slope. Side transects were not performed when the slope exceeded 120 degrees, or when located within a previously eradicated area. Tanoak and bay laurel located within the side transect but not the main transect were inspected for symptoms. As with the main transect, up to five leaves from each species were gathered for isolation. When cankers were present on mature tanoaks a bark sample was taken for isolation in lab.

Statistical analysis. A standard Bonferroni correction was applied to all statistical tests with a common hypothesis. Differences between understory tanoak, overstory tanoak, or bay abundance along streams (main transect) and away from streams (side transects) were tested with a Wilcoxon signed-rank test comparing proportion of stream segments to proportion of side transects in which each host was present at each site ($\alpha = 0.0167$).

P. nemorosa was the most common non-*P. ramorum* species we recovered, and was used as the best indicator of *Phytophthora* presence in foliage. Preferential isolation of *P. ramorum* or *P. nemorosa* from either tanoak or bay was analyzed with a Pearson chi-square statistic on contingency tables built separately for each pathogen ($\alpha = 0.025$). Preferential isolation of *P. nemorosa* from main and side transects was tested with a Wilcoxon signed-rank test comparing proportion of transect lengths in which a host was present and *P. nemorosa* was isolated, for *P. nemorosa* positive locations only. An identical analysis was attempted for main and side recovery of *P. ramorum*. All analyses were performed in S+ statistical software.

Distribution of infection in relation to overstory mortality (local distribution)

Survey location selection and sampling protocol. To assess the distribution of *P. ramorum* in understory vegetation around positive overstory tanoaks, we assumed that the first tree(s) to die at a SOD positive site was among the first infected by primary inoculum. Survey locations were selected from aerial maps depicting isolation density under the criteria of having an identifiable 'first' tree on which to base the spatial sampling. This allowed us to avoid having to scale sampling distances to account for relative size of the infested area; however, it also reduced the number of potential sites available for this study, as at the time of detection most sites were too large to approximate the point of introduction.

For each of the 7 locations surveyed as part of this study, four belt transects were constructed extending 20 m uphill, downhill, and laterally centered around the overstory tanoak identified as the first infected, adjusted for slope (Fig. 2.3). Each transect was 10 m wide, and was divided into 5 m by 5 m sections. In each 5 m^2 plot the presence or absence of rhododendron and tanoak sprouts was recorded. Up to 5 symptomatic leaf samples were taken from each host to determine if *P. ramorum* was present in the

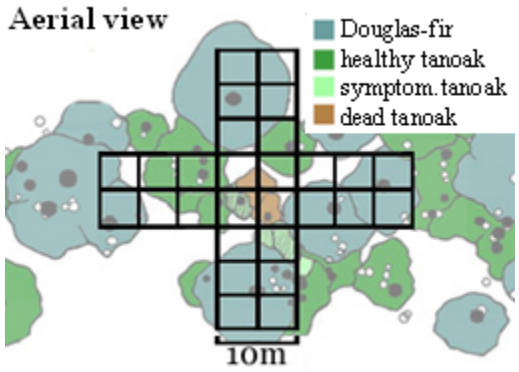


Fig. 2.3 Diagram of local survey methods, illustrating the orientation of transects used to assess the spatial relationship between understory infection and the 'first' dead tree within a site

understory at that location. Due to the infrequency with which we recovered *P. ramorum* understory at that location. Due to the infrequency with which we recovered *P. ramorum* from other foliar hosts (e.g. evergreen huckleberry) in earlier surveys, we limited this study to rhododendron and tanoak. California bay laurel was rarely encountered, but was recorded and sampled when present.

Statistical analysis. Recovery of *P. ramorum* in each distance interval (0 to 5 m, 5 to 10 m, 10 to 15 m, or 15 to 20 m) at each location was visualized as the proportion of plots with understory hosts present in which the pathogen was recovered. Statistical significance of a decline in recovery from the center of the transect was tested by first fitting a logistic regression model to the binary recovery response for each 5 m^2 plot against distance interval for each location. Fitted slope values from each model (n = 7) were used to test for a decline in recovery with increasing distance from the center of the transect with a two-sided, one-sample Wilcoxon signed-rank test (H_o: $\mu = 0$; $\alpha = 0.05$).

Isolation and identification of *Phytophthora* species

All vegetation samples were stored in a cooler for a maximum of four days and returned to Corvallis, Oregon. Within 5 days of collection one lesion per sample was plated onto cornmeal agar-ampicillin-rifampicin-pimaricin selective media (CARP) (Osterbauer 2004) and incubated in the dark for 7 to 12 days at 20^{o}C. Culture identification was based upon morphology of hyphae and spore structures (Appendix C). Any cultures lacking diagnostic features at the time of the first observation were incubated for another week and re-examined. In the local distribution study we only attempted to identify *P. ramorum* to species. We additionally identified *P. nemorosa* from isolates recovered from streams surveys. Any other *Phytophthoras* present were noted but not identified further.

RESULTS

Landscape analysis: spatial dependence on roads and streams

From the 709 positive isolations observed in the NChetco study area between 2001 and 2010 we identified 294 sites, ranging from <1 to 610 m to the nearest road (median = 100 m), and <1 to 414 m to the nearest stream (median = 71 m) (Table 2.1, Fig. 2.4a, b).

Of the 10,000 randomizations used to assess spatial dependence to roads, the average median distance to roads was 101 m; 4,733 randomizations had a median distance to road that was closer than observed. Sites were not significantly closer to roads than expected by chance (k/N = pseudo-p = 0.4733) (Fig. 2.5a). Of the 10,000 randomizations used to assess spatial dependence to streams, the average median distance to streams was 88 m; only 14 of the 10,000 randomizations were closer to streams than observed. Sites were significantly closer to streams than expected by chance (k/N = pseudo-p = 0.0014) (Fig. 2.5b).

Distribution of infection in relation to streams

Host distribution. To assess likelihood of dispersal from stream-borne inoculum we surveyed 15 locations, comprising a total of 2.78 km of main and 2.54 km of side transects (Appendix D Fig. D.1). All species except for Pacific rhododendron (*R. macrophyllum*) were present at most sites (Fig. 2.6). Red alder (*A. rubra*) was the most common overstory species along streams (Fig. 2.6).

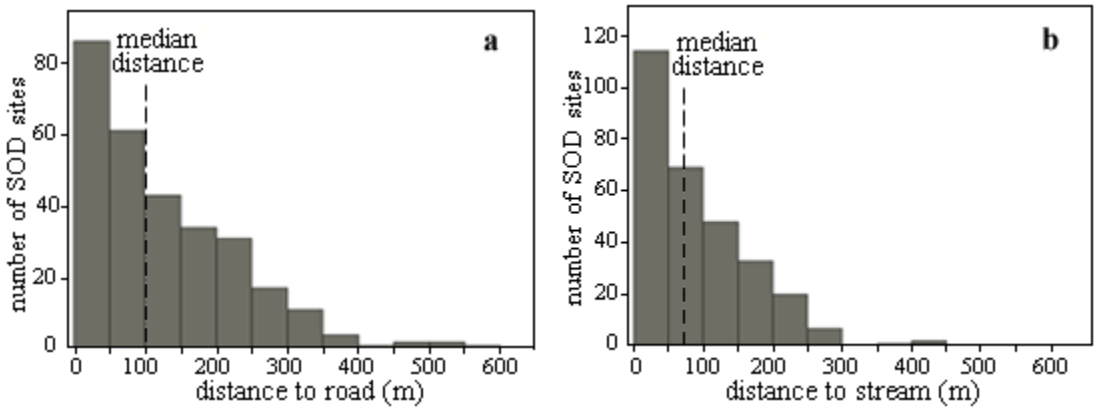


Fig. 2.4. Distribution of observed distances to the nearest road (a) or stream (b) for all SOD positive sites identified between 2001 to 2010 in the NChetco study area (n = 294).

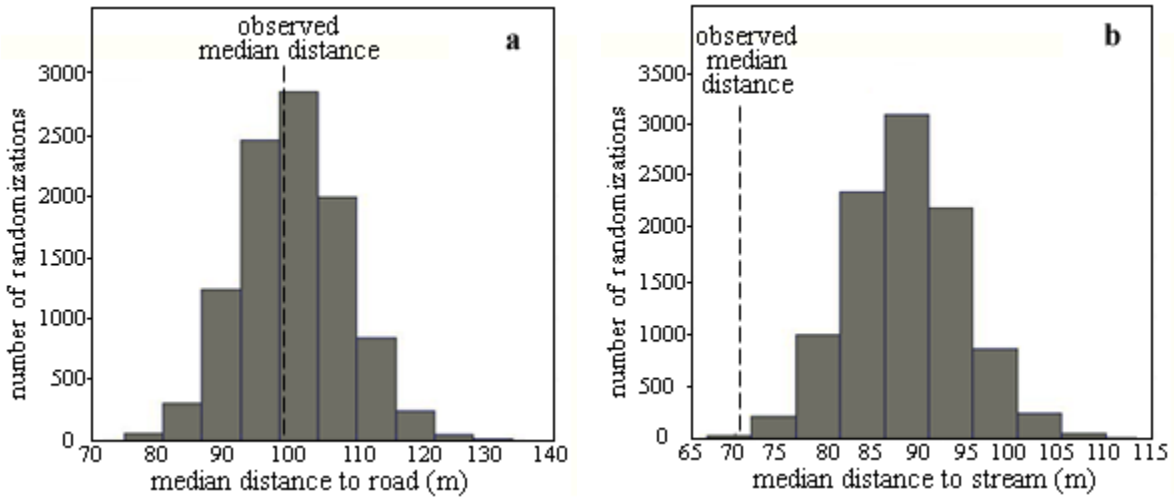


Fig. 2.5. Distributions of median distances to the nearest road (a) or stream (b) calculated for each of the 10,000 randomizations used to assess the spatial independence of SOD sites to roads or streams. The observed median distance to road or streams calculated for the actual 294 SOD positive sites is indicated by the dashed line.

California bay laurel was the most common foliar host at all locations (Fig. 2.6). There was significantly more bay present along the main transects than the side transects (z = 2.6126, p = 0.009). While not as common, tanoak was also present at all locations. Overstory tanoak was present in an average of 10.56% of the 10 m segments observed in the main stream transects (range by location: 0 to 61.54%), and 12.57% of all side transects (range by location: 0 to 24.36%) (Fig. 2.6). There was no significant difference in the abundance of understory tanoak (z = -0.2841, p = 0.7763) or overstory tanoak (z = -0.2841, p = 0.7547) between main and side transects.

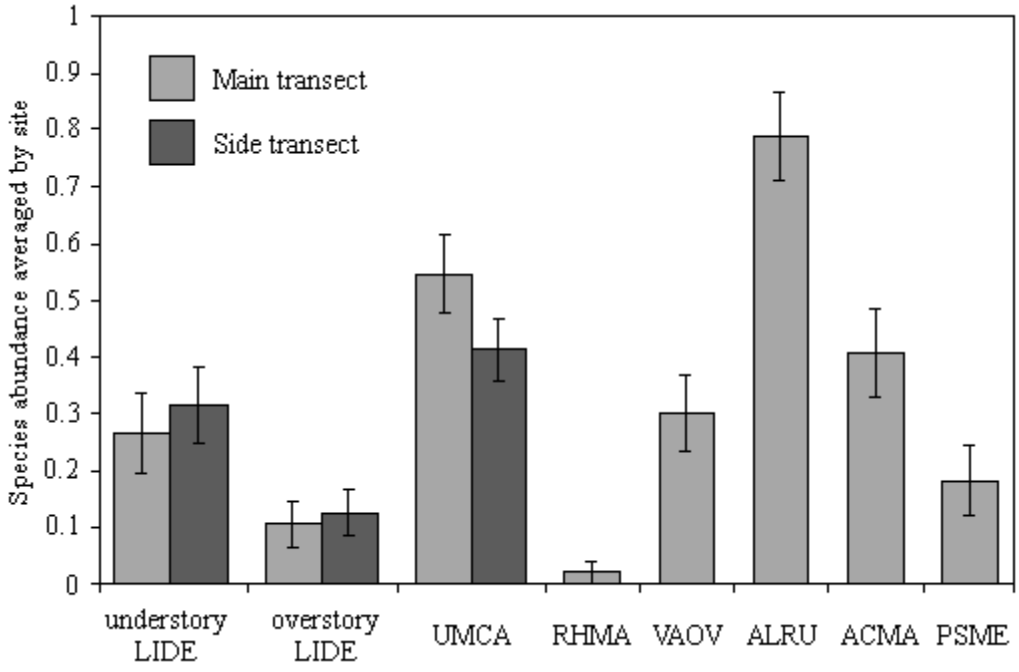


Fig. 2.6. Relative abundance of streamside vegetation. Abundance is quantified as either the average proportion of 10 m segments along the main transect or average proportion of side transects in which the host was present at each site. Error bars represent standard error. LIDE = ***N. densiflorus***; UMCA = ***U. californica***; RHMA = ***R. macrophyllum***; VAOV = ***V. ovatum***; ALRU = ***A. rubra***; ACMA = ***A. macrophyllum***; PSME = ***P. menziesii***.

Pathogen recovery. *Phytophthora* species were recovered from all locations. *P. nemorosa* was the most common species recovered, and was not isolated preferentially from either California bay laurel or tanoak ($\chi^2 = 1.79$, d.f. = 1, p = 0.181) (Fig. 2.7). *P. nemorosa* was recovered at 12 locations, from an average of 26.5% of main transect segments and 24.9% of side transect segments in which either host was present. There was no significant difference in recovery of *P. nemorosa* between main or side transects (z = 0.6676, p = 0.5044).

We isolated *P. ramorum* from 28 leaves collected at 4 sites (Fig. 2.6). All 4 sites were in close proximity to current, active infection (Appendix D Fig. D.2). *P. ramorum* was disproportionally isolated from tanoak (93% of all *P. ramorum* isolates; $\chi^2 = 72.5$,

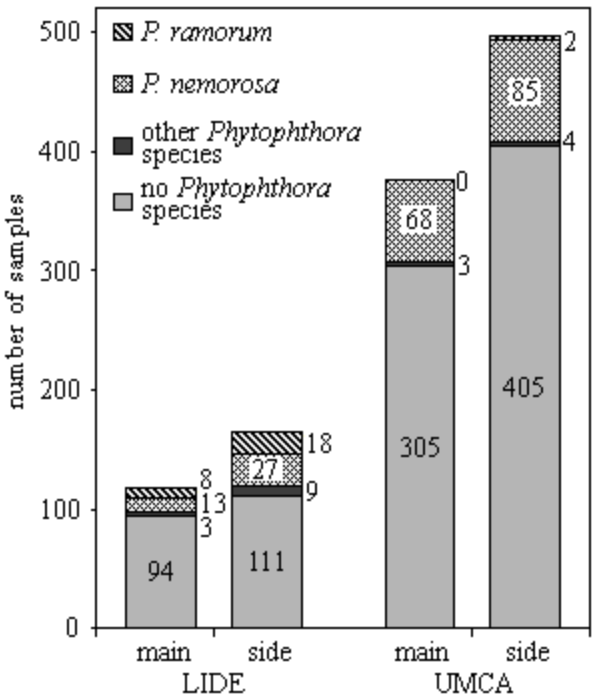


Fig. 2.7. *Phytophthora* spp. recovered in stream surveys from tanoak (LIDE) and California bay laurel (UMCA), separated by main and side transects. Total number of samples taken of tanoak = 118 from main and 165 from side transects; total number of samples taken of bay laurel = 376 from main and 496 from side transects.

d.f. = 1, $p < 0.0001$), and side transects (9.38% of main transect segments vs. 34.21% of side transects with hosts sampled within locations in which *P. ramorum* was recovered) (Fig. 2.7, Appendix D). Due to the small number of *P. ramorum* positive locations we lacked sufficient power to determine statistical significance between recovery of *P. ramorum* in main versus side transects.

Distribution of infection in relation to overstory mortality (local distribution)

Seven locations were sampled to assess the spatial relationship between overstory mortality and understory infection: one in 2007, four in 2008 and two in 2011, between the months of May and August. *P.* ramorum was recovered at all distance intervals away from the center of the site (at 0-5, 5-10, 10-15, or 15-20 m) at most locations. There is evidence for a dispersal gradient indicative of overstory sporulation: incidence was greatest closer to the center of each transect at most sites (Fig. 2.8). Fitted slope values were negative for all sites (range: -0.0335 to -2.4521), with a significantly negative trend between pathogen recovery and distance from the center of each transect ($p = 0.0156$).

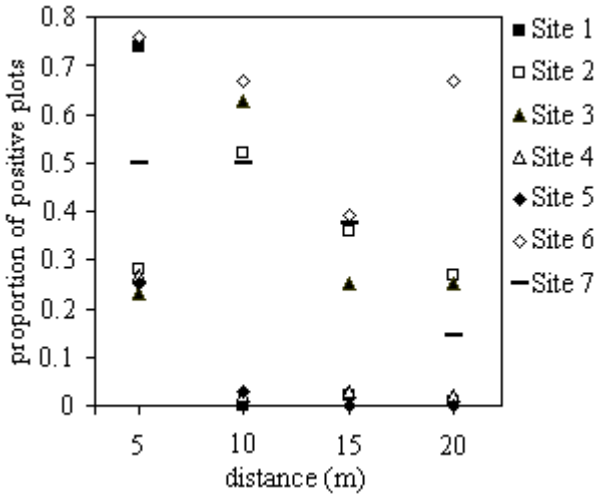


Fig. 2.8. Local distribution of *P. ramorum* in the understory around SOD positive trees. Pathogen presence is presented as proportion of 5 m^2 plots in which either tanoak or rhododendron were present and *P. ramorum* was recovered at each distance. Locations with an identical recovery at a given distance have been jiggered.

DISCUSSION

Roads as pathways for inoculum movement

Human activities along roadways have been implicated in facilitating the introduction of invasive organisms, including well documented examples of invasive *Phytophthora* species (Jules et al. 2002, Kauffmann and Jules 2006, Vannini et al. 2010). While movement of infested plants or firewood by people could have contributed to the initial introduction of *P. ramorum* into Oregon in the late 1990's or early 2000's, there is no evidence that *P. ramorum* has continued to spread into adjacent natural ecosystems by human-assisted pathways. Although 50% of SOD sites were within 100 m of the nearest roadway (Fig. 2.4a), sites were no more likely to occur closer to roads than expected by chance (Fig. 2.5a). This pattern is apparent despite year round access to roads within infested areas, providing opportunity for spread in both wet and dry seasons. The frequency with which we observe *P. ramorum* closer to roads is likely due to the overall high road density within this study area. Our conclusion of spatial independence to roads is supported, moreover, by the lack of roadside infection in preliminary surveys that have failed to isolate the pathogen from road soils or roadside vegetation within this study area (E.Hansen, unpublished data).

Soil inoculum is not absent from Oregon forests. Despite eradication, *P. ramorum* has been recovered in soils at least five years after mature tanoaks were removed from infested areas (Goheen et al. 2008). The persistence of inoculum, potentially as chlamydospores, in soils has been described for many *Phytophthora* species. Chlamydospores of *P. cinnamomi*, for example, can persist up to 6 years in soils in the absence of a host (Zentmyer and Mircetich 1966). Chlamydospores of *P. ramorum* apparently lose their viability upon drying, although they can persist in deep soils over dry summer months, provided they stay moist (Fichtner et al. 2007). Regardless, due to

our inability to reliably assess chlamydospore viability, their role as inoculum in sites post-eradication over multiple years is unknown.

We suspect most soil inoculum present at eradicated sites can be attributed to infection from tanoak sprouts that arise from cut stumps. Ideally, as part of the eradication, stems are killed with herbicides to prevent sprouting. Legislation has until recently prevented the use of pesticides on trees occurring within BLM property, however, and consequently tanoak sprouts can be found in abundance after eradication on these lands. These sprouts can be found infected, presumably from inoculum reservoirs in the soil. While we do not see significant infection resulting from soils along roads, soil inoculum may contribute to infection under a set of specific circumstances found at these locations: the availability of light after eradication has favored the production of new growth that is particularly susceptible; lacking canopy cover, the greater impact velocity of rain may increase the splash heights of inoculum from soil onto vegetation; additionally, inoculum loads in the soils are much greater at eradicated sites than what would be moved into new areas.

It remains possible that current management protocols have maintained inoculum levels below a threshold that could contribute to the spread of *P. ramorum* in soils between stands. This may explain why soils are implicated in the spread of *P. ramorum* in California, where the epidemic is more established, but not in Oregon. Regardless, dispersal of soil inoculum by hikers and animal traffic independent of roads may be relatively short ranged. For *P. lateralis*, foot and hoof traffic contributes more to inoculum movement out of streams than between watersheds (Jules et al. 2002). If soils have contributed to establishment of SOD in California we propose that it has only aided the local intensification of disease, not as a means of long distance dispersal and source of primary inoculum.

Streams as pathways for inoculum movement

While road dispersal cannot account for the landscape distribution of *P. ramorum* in Oregon, we did observe that SOD sites occurred significantly closer to waterways than expected by chance (Fig. 2.5b). *P. ramorum* can be recovered in stream baits in all seasons (Sutton et al. 2009), although this species is a weak saprotroph in aquatic environments (Aram and Rizzo 2011). We suspect active infection at undetected or eradication sites can serve as an inoculum source year round, posing a risk to stream-side vegetation. While *P. ramorum* has a significant spatial stream-association, infested sites are not necessarily located stream-side. Rather, 25% of sites were located greater than 140 m horizontal distance away from waterways (Fig. 2.4b). We investigated whether undetected understory foliar sporulation may be responsible for moving inoculum away from streams, a phenomenon that might be more readily observed for an aerial pathogen such as *P. ramorum* than *P. lateralis* or *P. cinnamomi*, which are predominantly root pathogens (Hansen et al. 2000, Shea et al. 1983).

Given that *P. ramorum* has been present in some streams included in our surveys for as long as six years, our hypothesis of stream dispersal would have predicted greater streamside infection than was observed. While understory tanoak, the best indicator of pathogen presence, was equally as common in transects adjacent to streams as away from streams, we recovered *P. ramorum* more commonly in vegetation in side transects away from the splash or flood line (Fig. 2.6, Fig. 2.7). All recoveries were found in areas of known, active SOD infection, and could be attributed to sporulation from upslope, overstory trees identified in the summer of 2011 (Appendix D Fig. D.2). In the absence of these overstory inoculum sources we failed to recover *P. ramorum*, even immediately downstream of positive samples (Appendix D Fig. D.2). These observations support our conclusion that streams are an inoculum sink rather than a source, and stream borne

inoculum is not responsible for significant long distance dispersal. Despite limited streamside infection by *P. ramorum*, stream baiting remains an important early indicator of pathogen presence for its potential to monitor large areas over the entire year and recover *P. ramorum* before overstory mortality is detected.

Lack of streamside infection is likely due to low risk of exposure. Streamside vegetation was within splash and flood line, though little foliage observed as part of this study was in direct contact with water. We propose the risk of stream-borne inoculum coming into contact with susceptible foliage is relatively minor, especially in contrast to *P. lateralis* infection of POC whose roots actually grow within the water (Kaufmann and Jules 2006). Alternatively, other *Phytophthoras*, but especially *P. nemorosa*, may be competitively excluding *P. ramorum* in this particular habitat. While we can find both species within a single site or tree, or occasionally the same lesion, little is known about the interaction between these two species. Nor do we understand the dispersal mechanism of this pathogen. *P. nemorosa* is not limited to streamside habitats, suggesting, like *P. ramorum*, the range of *P. nemorosa* is not limited by stream dispersal.

With data currently available we are unable to distinguish between other alternative hypotheses that can explain a strong stream association. Heterogeneity in environmental conditions may favor SOD closer to waterways. Hosts, especially tanoak, may be more common closer to streams. Alternatively, inoculum may be dispersing in wind channels, where the generally tendency for winds within a valley is to blow parallel to the valley axis without gaining vertical height (Eckman 1998). Host distribution is an unlikely contributor to this pattern. It is easy to distinguish overstory tanoak in aerial photographs, from which one can deduce that they are generally abundant at all elevations within the study area. We found overstory and understory tanoak at low

frequencies in both transect sets as part of our stream surveys (Fig. 2.6), indicating that while tanoak is regionally abundant (more so than can be detected in aerial photographs) it does not occur in dense stands in the immediate proximity of waterways. Rather, non-hosts (e.g. red alder) or hosts on which we do not observe a significant amount of infection (e.g. bay laurel or evergreen huckleberry) had greater exposure to inoculum than tanoak.

P. ramorum has demonstrated tolerance to a wide variety of environments in Oregon, though studies in California have shown a strong preference for moister conditions. Recovery of *P. ramorum* in soils, foliage, and rainwater is typically higher in wetter seasons and forest types (Davidson et al. 2011, Fichtner et al 2007). Changes in vegetation cover have also increased inoculum load and disease prevalence through a decrease in solar insolation and temperature (Meentemeyer et al. 2008b). We do expect these conditions to be more prevalent closer to streams, either due to topography or increased hardwood canopy cover (Chen et al. 1999, Rambo and North 2008). Additionally, fog is commonly observed settling in stream basins in this region, which likely provides a more favorable cooler and moister microclimate that increases chance of establishment and spread.

Local distribution and aerial dispersal

More indicative of an aerial mechanism without confounding environmental conditions is the strength of evidence for local infection resulting from top-down dispersal. In locations with early stages of local spread we had hypothesized that the pattern of understory infection could indicate whether inoculum had a soil-borne or an aerial-borne source. While the soil-borne source was undetectable since there would be no discernable way to determine the location of primary inoculum introduction, an aerial

mechanism should have produced a strong disease gradient from the first infected tree, as observed by Davidson et al. (2005).

Overall, there was a strong spatial dependence between understory infection and overstory mortality, as would be expected from local dispersal from tanoak canopies. A high proportion of plots were positive at 20 m only for one site which had, comparatively, higher recovery at all distances (site 6, Fig. 2.8). This distal infection could be associated with a second, dead overstory tree in the vicinity of the transect at site 6. As we observed spatial isolation of these new, small infected areas despite a high abundance of understory tanoak and other hosts in the forests surrounding these locations, we conclude that the sources of primary inoculum at these sites are best attributed to aerial inoculum.

The lack of association with roads, lack of evidence for dispersal from streams, and evidence for a top-down dispersal mechanism all support our alternative hypothesis of aerial dispersal. Aerial dispersal has not been conclusively demonstrated for any forest *Phytophthora*, although interest in aerial dispersal of sporangia has recently come to include two other species: the newly described ***P. pinifolia*** (Durán et al. 2008, E.Hansen pers.com.), and ***P. lateralis*** (Robin et al. 2010). While ***P. lateralis*** is known as a root pathogen of POC, it can cause foliar infections on low lying branches (Trione and Roth 1957). Recent infection in European windbreak plantings of POC has occurred not at ground level, but as cankers in the upper boles of trees (Robin et al. 2010). This circumstantial evidence supports the hypothesis that otherwise soil-bound *Phytophthoras* can disperse in air currents under specific circumstances.

Other lines of study have suggested aerial dispersal may play an important role in the development of the SOD epidemic not only in Oregon under the eradication program, but in California as well. Strong autocorrelation between genetic and geographic distance was detected at scales attributable to local dispersal in rain splash and the influence of forest composition, around 200 m; however, genetic similarly is also detected at larger scales, between 1 and 2 km (Mascheretti et al. 2008, Meentemeyer et al. 2004). Mascheretti et al. (2008) attributed spatial autocorrelation at 1 km to the movement of inoculum during times of strong winds and rain, but not under the same mechanism as other aerially dispersing oomycetes. This refers to the hygroscopic twisting of sporangiophores that occur during a drop in humidity, serving to release sporangia into dry, turbulent air currents (Leach 1982, Su et al. 2000, Aylor et al. 2001).

We have observed long distance dispersal (LDD) independent of roads at scales four times the 1 km Mascheretti et al. (2008) detected strong spatial autocorrelation. Such dispersal distances are unlikely during periods of heavy rain due to the likelihood of washout of spores closer to the source (Aylor 2003). Still, attempts made to trap sporangia in aerial spore traps have thus far been inconclusive. We suggest further studies be performed to identify if dispersal is occurring in dry currents (as with other oomycetes), exclusively in strong storms, or through some intermediate method. The environmental conditions supporting LDD may be rare. Regardless, distal sites have been observed every year of the epidemic, and have presented a real challenge to the management of this pathogen in Oregon.

The eradication effort has produced a spatially explicit dataset of the distribution of *P. ramorum*, and furthermore has eliminated confounding patterns resulting from multiple years of local spread. As such, all new SOD sites found within a single year can

reasonably be attributed to LDD. The elimination of local spread through the cutting and burning of infected trees at SOD sites, along with the particular configuration of the topography and lack of overall host heterogeneity in this study area has reduced the variation in spatial pattern that could be attributed to confounding factors. These methods may not have worked as well if *P. ramorum* had first been introduced into a different area. For example, initial investigations into the landscape distribution of the infestation originating from the Borax area produced similar, albeit statistically insignificant, results. The pattern may not be as strong in this area due to topography that was not oriented perpendicular to the coast as it is in the NChetco resulting in spread that, while to the north, was not confined to a single drainage system (Fig. 1.1). That this pattern was apparent, however, is significant given that the road and population density is much greater in the Borax area than in the NChetco area, which is accessed by privately owned, gated roads.

While the eradication program has aided the detection of these spatial patterns, it has also limited the scope of inference for both the stream and overstory surveys. Only a small number of locations were available for characterization under our criteria. For our local distribution study, most infestations were too large at the time of detection to be surveyed and, usually being located on the periphery of the quarantine zone, were top priorities for eradication treatment. Similarly, streams had to have been surveyed in heavily infested areas to ensure that stream inoculum was present, but significant portions of streamside forest had been altered by the eradication program at the time of this study. Selection of sites and small sample size may have affected our results, however our results are consistent with field observations in infested areas that, for reasons of practicality, we were unable to survey with these methods.

Conclusion

Our observations support our conclusion that the geographic expansion of *P. ramorum* would not have been slowed through more active road closures in the North Chetco area. Management aimed at preventing the movement of infested soils – trail and road closures or washing stations – will be ineffective at preventing the movement of inoculum into new stands once *P. ramorum* has established regionally. This recommendation is in sharp contrast to those made by researchers documenting roadside associations with other invasive *Phytophthoras*. Jules et al. (2002) found that while foot and animal traffic was responsible for moving inoculum of *P. lateralis* away from streams, vehicle traffic could best explain the introduction of inoculum into new watersheds, especially early in the epidemic. As such these authors suggested that watersheds without roads have a relatively minimal risk of exposure to inoculum (Jules et al. 2002). Unfortunately, this has not been our observation of the distribution of *P. ramorum* in Oregon.

Owing to the low probability of successful dispersal and infection over long distances, new distal infections resulting from aerial dispersal often appear sporadically and randomly distributed across the landscape (Aylor 2003). While these events are difficult to predict, they do occasionally happen at distances now documented up to 4.2 km. Any management decisions designed to limit spread or protect individual trees from infection (e.g. host-free zones or removal of adjacent foliar hosts) must take into account the possibility that inoculum may span greater distances than expected from splash dispersal or human movement, and commit to management practices that deal with these rare new foci as they develop.

TEMPORAL EPIDEMIOLOGY OF SUDDEN OAK DEATH DURING ERADICATION

INTRODUCTION

Despite concerted efforts to stop the expansion of SOD in Oregon, newly infected sites have been identified each year of the eradication program. Two aspects of this epidemic have been particularly inhibitory to the program's management objectives: the annual emergence of new infections at the periphery of known sites, and the occurrence of long and unpredictable jumps between sites. The eradication has operated under the presumption that by reducing the primary inoculum load, we could effectively limit both the size of new infected patches and the likelihood of long distance dispersal (LDD). Given the growing range in which we find *P. ramorum* (Fig. 3.1), it remains unclear if the eradication program has met these goals. The potential for aerial dispersal, a mechanism that is hard to control and even more difficult to predict, can partially account for our failure to completely control *P. ramorum*. This is compounded by the delay between initial infection and mortality, during which time LDD is likely. The length of this incubation period (defined here as the time between infection by primary inoculum and the development of crown mortality, the symptom on which aerial identification of new SOD sites depends upon), as well as how to manage variation in epidemic severity between years is unknown.

Modeling is a common approach utilized to develop hypotheses about the behavior of plant pathogens (Madden et al. 2007). In the case of invasive pathogens in heterogeneous environments, however, models are generally difficult to interpret. Early in establishment, an invasive species' distribution may not fully represent its fundamental niche. Estimates of potential geographic or host range could then be biased towards

where the pathogen was originally introduced. Variation in host distribution or susceptibility is hard to quantify at the scale needed to make management decisions or draw epidemiologically relevant conclusions. Additionally, environmental variables are easily over-fit in models due to the covariance between weather conditions within and between years (Holdenrieder et al. 2004).

The SOD epidemic in Oregon may be one of few non-agricultural examples in which this heterogeneity is manageable, and for which we have a substantial spatial and temporal dataset. Within the immediate area of current infection in Curry County, tanoak is abundant and is the dominant host responcible for spread (Hansen et al. 2008). Moreover, among the bole hosts, which include *Quercus* spp. as well *N. densiflorus*, tanoak presents the highest rates of mortality, but the least amount of inheritable resistance (Maloney et al. 2005, McPherson et al. 2005, Hayden et al. 2011). While there is some variation in susceptibility among tanoak populations (Hayden et al. 2011), differences in susceptibility to *P. ramorum* infection can be negligible relative to the influence of local environmental conditions (Anacker et al. 2008). Aggressiveness also differs among *P. ramorum* lineages (Manter et al. 2010), although as Oregon's infestation is comprised of one clonal population dominated by a single genotype we expect less variation in pathogenicity relative to other forest populations (Prospero et al. 2007).

Due to the elimination of local spread by the eradication program, the size of infested areas contributing to LDD has also been relatively constant between years. Regardless, weather conditions of a given year have likely favored the production of greater or lesser amounts of inoculum from these sources. Sporulation is one of the major factors contributing to the rate of spread of phytopathogens, and varies significantly with changes in the environment. Without significant change in host composition,

pathogenicity or treatment protocol, the range of infestation sizes and LDD in Oregon can reasonably be attributed to changes in weather conditions between years, particularly those conditions affecting the ability of *P. ramorum* to sporulate.

Sporulation by *P. ramorum* is one of the more thoroughly investigated aspects of SOD. Significant differences in the quantity of spores produced are observed between different foliar hosts (Davidson et al. 2008) and forest types (Davidson et al. 2011). Regardless, the recovery of inoculum from rain splash and foliar lesions has consistently captured greater spore loads with increased precipitation, and in forest types with moister microclimates (Davidson et al. 2008, 2011). The effects of temperature are less well understood, although recovery of inoculum from individual bay laurel lesions is greatest during late spring rains (Davidson et al. 2008). This effect was heightened during El Niño years when precipitation extended longer into the warm season (Davidson et al. 2008). Also in support of an influence of temperature, disease severity on California bay laurel is positively associated with maximum daily temperatures from December through May (Condeso and Meentemeyer 2008).

We have not been able to quantify inoculum in rain splash in Oregon as done in California because eradication protocol requires the treatment of infested sites, limiting our ability to deploy collection buckets at a given location for any appreciable amount of time. Instead, both the maximum distance moved and infestation size of a given year can be good indicators of inoculum quantity. Per standard dispersal and disease curves, we expect to observe disease at further distances and greater amounts of disease at a given distance with larger sources of primary inoculum (Madden et al. 2007). We hypothesize that greater dispersal distances should have occurred in years with conditions most conducive to sporulation. Correspondingly, years with less sporulation should have

resulted in reduced dispersal opportunity, producing shorter distances and fewer new sites. The size of an infestation in a given year will be augmented by the amount of primary inoculum establishing new sites, but also the amount of secondary inoculum contributing to local spread. In contrast, the maximum dispersal distance, as predicted by the length of the dispersal curve, will solely represent the amount of primary inoculum. Because of the incubation period, however, we expect a delay between the year of inoculum production and LDD and the detection of overstory mortality at a distal location. This delay is our best indication of the incubation period for *P. ramorum* in Oregon forests.

While the eradication program has not eliminated *P. ramorum* from Oregon forests, we need to describe the effect that it has had upon the potential extent of this epidemic. Specifically, we want to determine how well the elimination of local spread has reduced the size of primary inoculum sources contributing to LDD, and if this has reduced maximum dispersal distances and the overall size of newly detected infestations. We analyzed the epidemic in two areas with independent disease development: the original Ferry Creek, Joe Hall Creek and North Fork Chetco River drainages (hereafter called the 'NChetco study area'), and a second area originating from the smaller Borax site first identified in 2006 (hereafter called the 'Borax study area') (Fig. 3.1).

Both areas have been treated with the same general protocol during most of the time that *P. ramorum* has been in Oregon with one major exception: a delay in 2009 due to a temporary lack of funding to treat sites located on private property. While treatment would have normally ensued in the summer of 2009, eradication was delayed for most sites until the summer and fall a year later. This delay preferentially stalled the treatment in the Borax area, as funds were still available to treat infection found on BLM lands

located within the North Chetco watershed. Upon its restart, treatment was also preferentially directed to those non-treated sites located in the NChetco area because many of these sites were closer to the 2008 quarantine boundary and were of a higher priority for treatment (A.Kanaskie, ODF, pers. com.). This delay could have contributed to the dramatic increase in infested acreage observed in the Borax area in 2011 (Fig. 3.1). This conclusion is so far speculative as we are unsure if 2011 infection can be best explained by extended sporulation from trees detected (but not treated) in 2009, or if the 2011 increase resulted from unusually favorable weather and would still have been seen had the eradication proceeded normally. Otherwise, the extent of infection in 2011 provides a first indication that timely eradication has reduced the potential size of this epidemic.

In an effort to better understand the dynamics of *P. ramorum* in Oregon, we seek to describe how the development of SOD has changed over time. This will be accomplished by analyzing data gathered as part of the eradication program with the following objectives:

1) Build two models to describe annual variation in infestation size and maximum distance moved for sites identified in the NChetco and Borax areas between 2001 and 2010.
2) Deduce the length of time between initial infection by primary inoculum and overstory mortality through model parameters.
3) Determine if the extent of the infestation observed in 2011 deviates significantly from model predictions, and see if these deviations can be attributed to the 2009 delay. While we lack a true control for comparison, this will indicate if the eradication program has slowed the establishment and spread of *P. ramorum*.

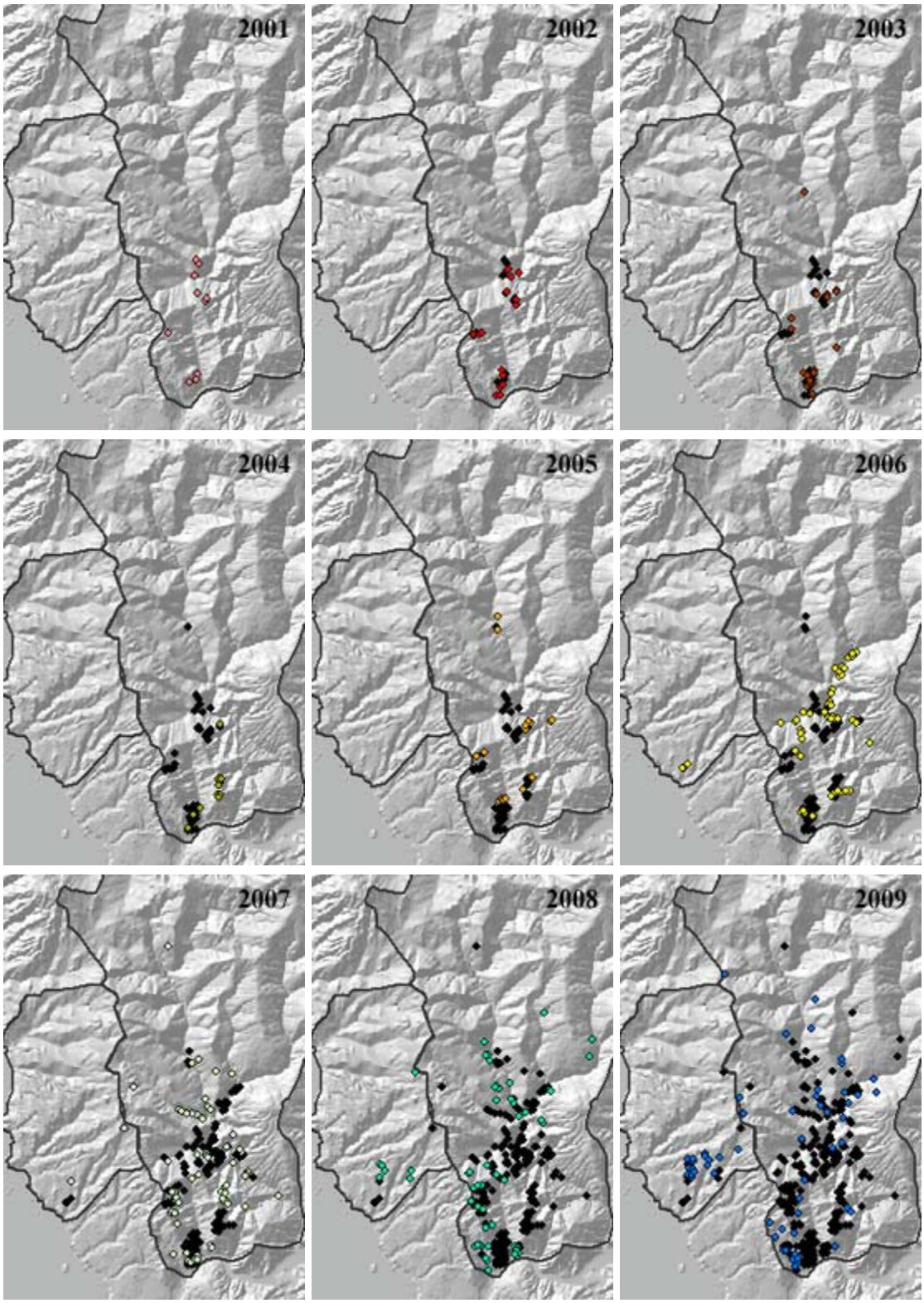


Fig. 3.1. Yearly progression of the SOD epidemic in the North Chetco and Borax study areas, depicting all new sites within a given year. (Continued on following page.)

(Continued)

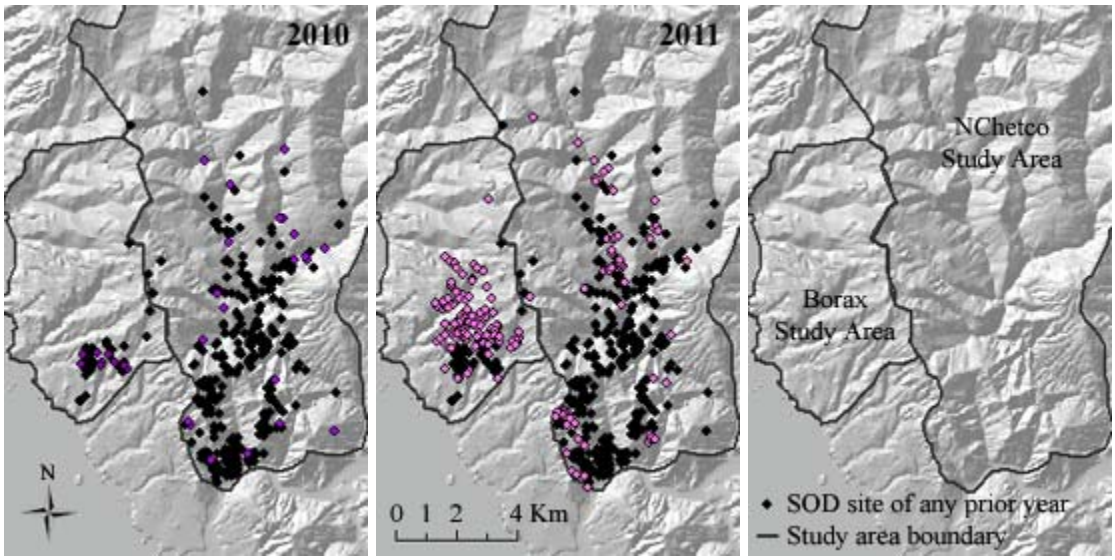


Fig. 3.1. Yearly progression of the SOD epidemic in the North Chetco and Borax study areas, depicting all new sites within a given year.

METHODS

Characterization of epidemic variables

Change in the SOD epidemic over time was characterized by quantifying the total size of the infested area and range of long distance movement in annual intervals. These variables were calculated in ArcMap (version 9.3, ESRI) separately for the NChetco and Borax study areas, and included all isolations identified before 31 August, 2011 (Fig. 3.1). Movement of *P. ramorum* over the ridges defining the North Chetco watershed has been rare over this pathogen's history in Oregon. As such, we assumed that all infestations located on the western side of the ridge defining the NChetco study area were attributable to the expansion of the Borax infestation.

Infestation size. The size of the annual infested area was assessed by creating 30 m buffers around the coordinates of all *P. ramorum* positive trees. This distance was

selected to approximate the area in which we find the majority of trees with canopy infections as a result of secondary, within-site spread. Trees with green foliage and bleeding cankers and canopy infection are easily found adjacent to dead tanoaks, and are often how we confirm the presence of SOD in an area (*P. ramorum* being poorly recovered from dead stems). We dissolved all buffers by year of detection and study area, and then calculated the total size of each polygon in hectares.

Maximum distance moved (LDD). To assess the maximum range of LDD, we first reduced all adjacent positive trees to site coordinates defined as the centroid of all points located within 60 m of one another. We calculated the dispersal distance moved between years by performing spatial joins of sites in each year to sites detected in any year prior within the same study area (e.g. between all sites detected in 2006 to all sites detected between 2001 and 2005 within the NChetco study area). This analysis calculates the distance of each site in the year of interest to the closest potential inoculum source.

Weather data

Weather data was obtained from the Red Mound remote access weather station (RAWS) maintained by the BLM (latitude: 42°07'24" N, longitude: 124°18'02" W; elevation: 534 m; http://www.raws.dri.edu/index.html) (Fig. 1.1). Average daily maximum temperature (°C), average daily precipitation (mm), and average daily humidity (%) from 1 October 1998 to 30 June 2011 were compiled into autumn (October through December), winter (January through March), spring (April through June), and summer (July through September) averages.

Modeling of epidemic development

Multiple linear regression was used to identify weather conditions that can model infestation size and LDD between 2001 and 2010. Explanatory variables included the seasonal weather conditions of the year of detection, and one and two years before detection (Table 3.1). We chose not to include weather variables greater than two years before detection to reduce confounding results emanating from covariances between weather variables. A preliminary analysis indicated that conditions three years before did not significantly increase the explanatory power of our models (data not shown). We have little reason to suspect that crown mortality is delayed much longer than three years given the relatively small size of most sites when detected, indicating that the pathogen has had limited time to spread locally. Infestation size of one year and two years before detection was also considered as explanatory variables (Table 3.1).

We used multiple selection procedures to identify weather and epidemic variables that best explain variation in infestation size and maximum distance moved. A full model was built with all weather and epidemic variables with correlation coefficients stronger than ±0.2, and, due to their reported role in California epidemiology, average spring precipitation and maximum winter temperature of one and two years before detection (regardless of correlation coefficient) (Table 3.1).

All models were evaluated by comparing adjusted r^2 and AIC values to identify those with the highest explanatory power. Using stepwise regression we sequentially deleted non-significant variables. Any model with AIC values within 2 units of the best model (lowest AIC) was considered as a potential candidate. To test for a significant effect of location (Borax and NChetco), study area was included as an additional variable in the final model, then rejected or accepted with an extra sums of squares F-test.

Table 3.1. Potential weather and epidemic variables considered as explanatory variables for regression modeling of maximum distance moved and infestation size by year. Data is not shown for weather variables the year of detection, or relative humidity and summer variables in any year as no variables from these categories were included in final models. Those variables included in the initial full regression model are in bold.

variable code	description	maximum distance moved	infestation size
epidemic variables		r^2	r^2
InfestYB4	infestation size year before	**(0.370)**^{2}	**(0.713)**^{2}
Infest2YB4	infestation size two years before	(0.200)^2	(0.335)^2
weather conditions one year before detection*			
AuMaxTYB4	autumn maximum temperature	(-0.185)^2	(-0.148)^2
AuPrecipYB4	autumn precipitation	(0.167)^2	(-0.033)^2
WMaxTYB4	winter maximum temperature	**(-0.553)**^{2}	**(-0.184)**^{2}
WPrecipYB4	winter precipitation	(0.449)^2	(-0.048)^2
SpMaxTYB4	spring maximum temperature	(-0.060)^2	(-0.168)^2
SpPrecipYB4	spring precipitation	**(0.244)**^{2}	**(0.059)**^{2}
weather conditions two years before detection			
AuMaxT2YB4	autumn maximum temperature	**(0.247)**^{2}	(0.195)^2
AuPrecip2YB4	autumn precipitation	(0.179)^2	(0.073)^2
WMaxT2YB4	winter maximum temperature	**(0.573)**^{2}	**(0.295)**^{2}
WPrecip2YB4	winter precipitation	**(-0.289)**^{2}	(-0.091)^2
SpMaxT2YB4	spring maximum temperature	**(-0.327)**^{2}	(0.059)^2
SpPrecip2YB4	spring precipitation	**(0.662)**^{2}	**(0.254)**^{2}

* Example: for a tree detected in spring or summer of 2009, one year before detection is defined as average daily conditions from Oct. – Dec. (autumn) 2007, Jan. – March (winter) 2008, or April – June (spring) 2008.

We forced the intercept at 0 for all regressions, and observed residual and quantile plots of the final models to ensure that assumptions of linearity and normalcy were met. Final models with the best predictive capacity were used to calculate infestation size and maximum distance moved expected in 2011 in each of the study areas. Significant deviation between observed and expected values was assessed by calculating 95% confidence intervals for all model parameters to estimate the upper and lower boundaries

considered significant at $\alpha = 0.025$. All analyses were performed in S+ and R statistical software packages.

RESULTS

Modeling of epidemic development

For all sites detected between 2001 and 2010, the majority of sites closest to new detections were identified the previous year in both the NChetco and Borax study areas (Fig. 3.2). The years 2006 and 2007 had, overall, the most apparent infection between 2001 and 2010, with larger infestation sizes and greater distances moved than observed in previous years (Table 3.2a, Fig. 3.3, Fig. 3.4). The extent of infestation in the NChetco area in 2011 was comparable to previous years (e.g. 2006), which contrasts with the increase in infested acreage observed in the Borax area in 2011 (Table 3.2, Fig. 3.3, Fig. 3.4).

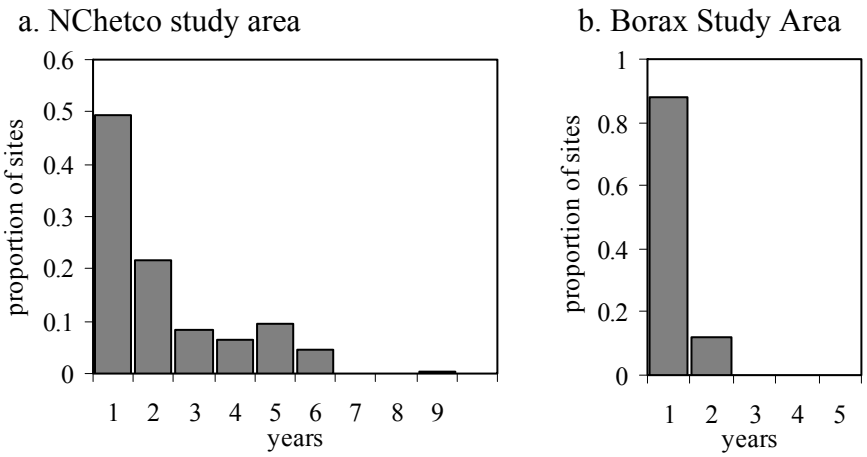


Fig. 3.2. Length of time between the detection of a new SOD site and the detection of the closest site of any previous year. Data is presented for all sites identified between 2001 and 2010 in either the NChetco (a; n = 294) or Borax (b; n = 37) study areas.

Table 3.2. Yearly characterization of the SOD epidemic size and range between 2001 and 2011, segregated by year of detection and location. Mean and maximum distance moved represent the distance between all SOD sites found in one year to the closest SOD sites of any previous year.

a. NChetco Study Area

year of detection	area of infestation (ha)	mean distance moved (m)	maximum distance moved (m)
2001	5.06	n/a	n/a
2002	10.15	256	573
2003	10.88	425	2,712
2004	5.70	225	564
2005	9.11	263	914
2006	22.38	647	2,192
2007	25.81	529	4,261
2008	18.19	430	2,449
2009	15.58	398	2,634
2010	7.61	377	1,259
2011	22.05	283	1,042
Total:	153		

b. Borax Study Area

year of detection	area of infestation (ha)	mean distance moved (m)	maximum distance moved (m)
2006	3.11	n/a	n/a
2007	1.54	2,098	3,493
2008	3.68	970	2,351
2009	6.98	347	858
2010	3.06	153	249
2011	45.42	828	2,071
Total:	62		

While the range of LDD increased in distance for the first three years of the epidemic, the maximum distance moved has not consistently increased over time in the NChetco area (Table 3.2a, Fig 3.3). The longest jumps for both the NChetco and Borax areas were observed in 2007 (Table 3.2, Fig. 3.3). This year had the largest total new infested acreage in the NChetco, but the smallest total new infested acreage in the Borax area (Table 3.2, Fig. 3.4). The largest infested acreage in the Borax study area was observed in 2011 (Table 3.2b, Fig. 3.4).

Maximum distance moved was best explained by weather variables from two years before detection (Table 3.3a, Appendix E). We were unable to determine if LDD was best determined by autumn maximum temperature, winter maximum temperature, or spring precipitation of two years before detection (or as a combination of these three

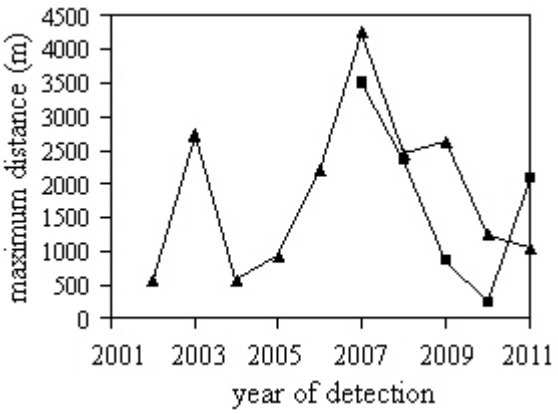


Fig. 3.3. Maximum distance between new sites in each year to the closest site of any previous year in the NChetco (▲) and Borax (■) study areas. As the Borax area was first detected in 2006, the first LDD event was recorded in 2007.

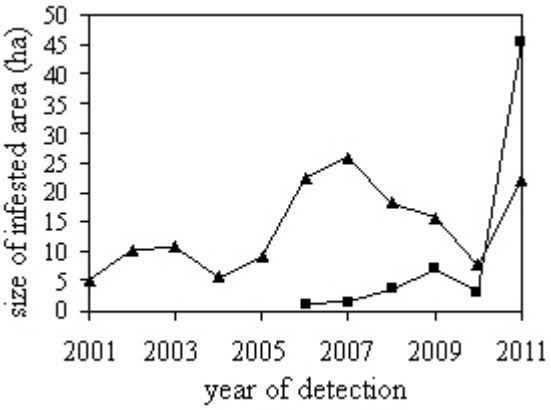


Fig. 3.4. Total size of newly detected infested areas (ha) by year in the NChetco (▲) and Borax (■) study areas.

variables) as weather variables were highly correlated with each other (Table 3.4). Evidence was strongest for spring precipitation of two years before detection, which also had the strongest correlation with maximum distance (Table 3.1, Table 3.3a). Infestation size of the year before and winter maximum temperature of two years before detection were marginally significant ($p = 0.0545$ and $p = 0.0567$, respectively) in our final preferred model: MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + InfestYB4 ($p < 0.0001$; adj. $r^2 = 0.8876$) (Table 3.3a). Study area was not a significant explanatory variable for maximum distance moved ($F = 0.767$, d.f. = 2,7, $p = 0.50$).

Infestation size was best modeled by explanatory variables of the year before detection, particularly spring precipitation and infestation size (Table 3.3b, Appendix F). The inclusion of study area did not increase our ability to model infestation size ($F = 0.603$, d.f. = 11,9, $p = 0.57$). Our final preferred model was: InfestArea ~ -1 + InfestYB4 + SpPrecipYB4 ($p < 0.0001$; adj. $r^2 = 0.8622$) (Table 3.3b).

Table 3.3 Reduced models built to describe maximum distance moved (a) and infestation size (b) in the NChetco and Borax study areas between 2001 and 2010. Both final models are significant at $p < 0.0001$.

a. Maximum distance moved (LDD)

model	β coefficient				Adj. r^2	AIC
	SpPrecip 2YB4	WMaxT 2YB4	FmaxT 2YB4	InfestYB4		
MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + FMaxT2YB4 + InfestYB4‡	369.82*	507.60^{ns}	-445.91*	60.20^{ns}	0.8876	214.99
MaxDistance ~ -1 + SpPrecip2YB4	416.31***	–	–	–	0.8249	217.70
MaxDistance ~ -1 + WMaxT2YB4	–	167.75***	–	–	0.7353	223.07
MaxDistance ~ -1 + FMaxT2YB4	–	–	134.82***	–	0.6988	224.75

^{ns} not significant, *p < 0.05; **p < 0.01; ***p < 0.001

‡ final preferred model (note: WMaxT2YB4 and InfestYB4 are marginally significant at p = 0.0567 and p= 0.0545, respectively)

b. Infestation size

model	β coefficient		Adj. r^2	AIC
	InfestYB4	SpPrecip YB4		
InfestArea ~ -1 + InfestYB4 + SpPrecipYB4‡	0.64*	3.14*	0.8622	81.72
InfestArea ~ -1 + InfestYB4	0.91***	–	0.8058	85.31
InfestArea ~ -1 + SpPrecipYB4	–	2.56**	0.6806	91.77

^{ns} not significant,*p < 0.05; **p < 0.01; ***p < 0.001

‡ final preferred model

Table 3.4. Correlation tables showing the relationships between all weather variables included in the initial full models built to explain variation in maximum distance moved (a) or infestation size (b) of the NChetco and Borax areas between 2001 and 2010. The variables included in each final model are in bold.

a. Maximum distance moved

	WMaxT YB4	SpPrecip YB4	AuMaxT 2YB4	WMaxT 2YB4	WPrecip 2YB4	SpPrecip 2YB4	SpMaxT 2YB4
WMaxTYB4	1.00	-	-	-	-	-	-
SpPrecipYB4	0.11	1.00	-	-	-	-	-
AuMaxT2YB4	-0.57	0.09	1.00	-	-	-	-
WMaxT2YB4	-0.57	0.07	0.53	1.00	-	-	-
WPrecip2YB4	0.19	-0.32	0.20	-0.50	1.00	-	-
SpPrecip2YB4	0.42	-0.42	-0.33	-0.76	0.77	1.00	-
SpMaxT2YB4	-0.72	0.01	0.73	0.57	0.08	-0.24	1.00

b. Infestation size

	WMaxT YB4	SpPrecip YB4	WMaxT 2YB4	SpPrecip 2YB4
WMaxTYB4	1.00	-	-	-
SpPrecipYB4	0.25	1.00	-	-
WMaxT2YB4	-0.57	0.23	1.00	-
SpPrecip2YB4	-0.72	0.13	0.57	1.00

Table 3.5. Observed and predicted values for maximum distance moved (a) and infestation size (b) in 2011. Confidence intervals (CI) represent the bounds considered significant at $\alpha = 0.025$)

a. Model: MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + FMaxT2YB4 + InfestYB4

study area	observed (m)	expected (m)	obs/exp	95% CI (lower)	95% CI (upper)
NChetco	1,042	2,088	0.50	835	3,342
Borax	2,071	1,822	3.14	495	3,150

b. Model: InfestArea ~ -1 + InfestYB4 + SpPrecipYB4

study area	observed (ha)	expected (ha)	obs/exp	95% CI (lower)	95% CI (upper)
NChetco	22.05	17.00	1.30	7.65	26.28
Borax	45.42	14.15	3.21	3.86	24.45

Neither maximum distance moved or infestation size observed in 2011 in the NChetco study area were significantly different than expected by our final models (Table 3.5a,b). The maximum distance moved was not significantly different than expected in the Borax study area (Table 3.5a); the size of the infestation, however, was significantly larger than predicted (Table 3.5b).

DISCUSSION

In the theory of how epidemics develop over time and space, the velocity of a disease modeled by a negative power law curve should increase over time and be related to the size of the infestation contributing inoculum or total inoculum load (Sackett and Mundt 2005b, Cowger et al. 2005, Aylor 2003). This is assuming that conditions favoring sporulation and the distribution of hosts are constant over the range of observations. In contrast, infestation size was not, by itself, the best indicator of the amount of inoculum produced in the year contributing to the LDD of *P. ramorum* in Oregon. Rather,

differences in weather conditions between years exerted additional influence on dispersal distance. This is especially probable given the longest jumps were observed in the same year for both the Borax and NChetco areas, and this occurred when the Borax area had the smallest infestation size (Fig. 3.3, Fig. 3.4).

We are unable to conclusively discern which weather variables or seasons contribute most to long distance spread; however, we are able to conclude that the conditions best explaining maximum distance moved occurred two years before the detection of new distal sites. This indicates that the incubation period between inoculum introduction and overstory mortality is, on average, two years (Fig. 3.5). This interval is consistent with modeled decline of tanoak in California, where survival analysis has predicted a median time to death after infection of 1.9 years when accompanied by bark beetle attack (McPherson et al. 2010). Two years allows ample time for *P. ramorum* to establish locally, as well as contribute to long distance dispersal the year that infection is present but not detectable by aerial surveys (hereafter referred to as the 'incubation year').

While weather variables of two years before contribute to LDD, maximum distance moved is also associated with the size of the infested area the year before detection (Table 3.3a). The length of time between the death of the trees providing inoculum and those receiving inoculum need not be representative of the incubation period, so long as the time between infection and detection is similar, albeit offset, between the source and sink trees. In our proposed model, while the delay between infection and mortality is two years within a site, the length of time between the deaths of the source and sink trees will only be offset by a single year (Fig. 3.5). For example, inoculum produced from a site in 2008 will infect trees that most likely will die in 2010; those trees that were the source of this inoculum will most likely die in 2009, an observed delay of only one year (Fig. 3.5).

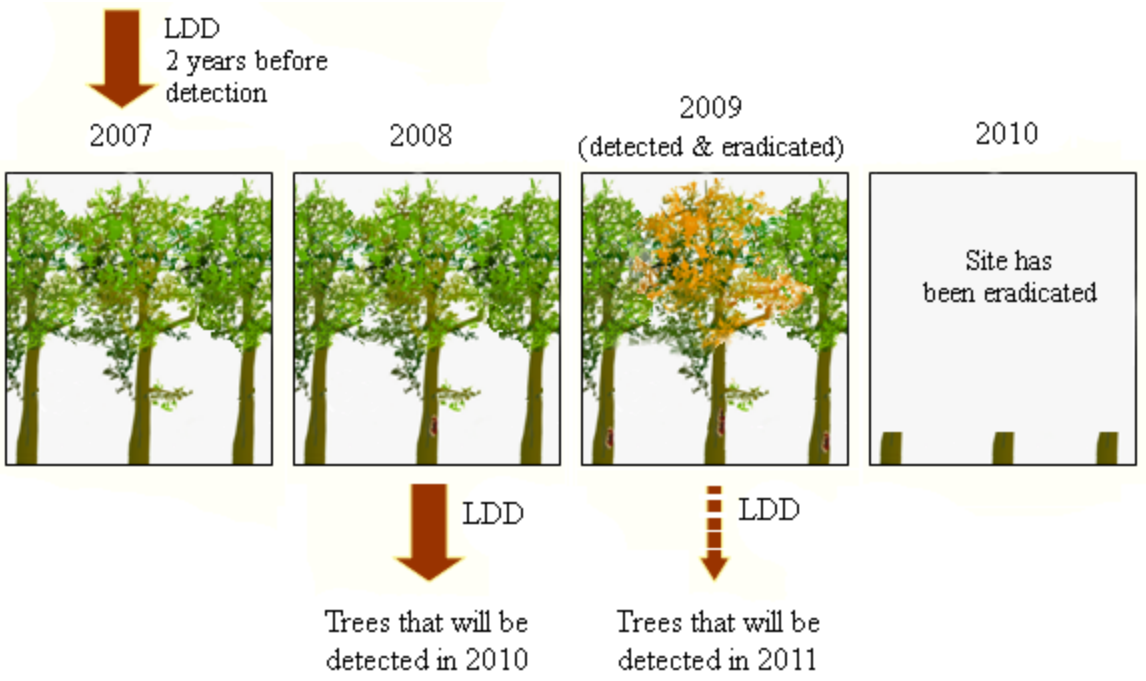


Fig 3.5. Proposed temporal epidemiology of *P. ramorum* in Oregon forests during eradication, showing the delay between initial infection and detection of overstory mortality of two full years. Per our model, trees that would have been detected and eradicated in 2009 are the most likely source of tree mortality detected in 2010, which were infected by inoculum produced in 2008. Illustration adapted from Parke and Lucas (2008).

This model incorporates features of expected epidemiology, whereby spores contributing to the establishment of most new sites are most likely produced at sources closer in space and in time. Closer sites are the most likely sources of primary inoculum, as dispersal gradients result in greater numbers of new infections closer to the source than further away (Fitt et al. 1987). Provided the only time period significantly contributing to LDD occurs during the incubation year (2008 in the example of Fig. 3.5), our model predicts that most new sites should be closest to a site detected the year before, as was observed in both the NChetco and Borax areas (Fig 3.2a, b). We assume the incubation year is the most likely contributor to LDD, and argue that this is valid given the need for sites to develop locally before being able to contribute the larger inoculum loads required for

LDD. Ideally, these sites are then eradicated before contributing significantly to further spread. Absent the eradication, however, trees that became infected through secondary inoculum dispersed in rain splash are likely the stronger contributors to LDD as the trees infected the first few years of establishment start to die.

Undoubtedly, the nearest site is not always the source of primary inoculum. Alternatively, variation in time to death or inoculum produced during the year of detection could also account for a time to mortality greater than a single year. For example, if inoculum produced in 2009 from a site detected and eradicated that same year resulted in the establishment of new sites, these would most likely be observed in 2011, a difference of 2 years. These alternative conditions can account for those sites in which the closest site of any previous year was identified multiple years before detection, although they occur with a decreasing frequency in both study areas (Fig 3.2a, b).

Of the weather variables identified as most likely contributors to the LDD of *P. ramorum*, average spring precipitation and average maximum winter temperature of two years before detection are both consistent with known epidemiology of SOD. We also detected the potential for autumn conditions to contribute to LDD, which is a deviation from conditions thought to be conducive to sporulation in California. Differences in the timing of sporulation between California and Oregon are expected given the differences in host dynamics and climates in each location. In the redwood / tanoak or mixed evergreen forests of California's epidemic, bay laurel is the dominant host supporting the sporulation needed for establishment (McPherson et al. 2010, Cobb et al. 2010, Maloney et al. 2005, Meentemeyer et al. 2008b). While sporulation is abundant in the late spring, inoculum production on bay laurel does not noticeably occur during autumn rains (Davidson et al. 2008, 2011).

Due to the extent of bay infection in Californian sites where *P. ramorum* is significantly more established, the modeling of the epidemic in California is biased towards those conditions favoring sporulation from bay laurel. For example, prior models built to predicatively map areas at high risk for *P. ramorum* establishment have used weather conditions only between December and May (Meentemeyer et al. 2008a, Václavík et al. 2010). In contrast, the epidemic in Oregon is driven by infection of tanoak (Hansen et al. 2008), which shows no significant difference in the ability to support sporulation between wet and dry years, or at the start and end of the rainy season (Davidson et al. 2008). In addition to the expected recovery of spores during spring and winter rains, inoculum is recovered from collection buckets placed underneath infected tanoak from October through December in Oregon (Hansen et al. 2008). Tanoak is also thought to be susceptible during most times of the year (E.Hansen pers.com.), allowing for the potential for autumn sporulation to contribute to LDD.

During the incubation year, weather conditions contributing to greater dispersal distances are also contributing to local spread. Our models predicted that weather conditions the year before detection are strong contributors to the size of an infestation, although the best determinate was the infestation size of the year prior (Table 3.3b). We propose that the size of the infested area in a given year is determined more by the number of LDD events contributing to new sites, than by the rate of local spread within a site. This is especially likely given how we calculated infestation size. Due to inconsistent sampling intensity between sites over the course of the eradication program, using the number of SOD positive trees would bias our observations towards more heavily sampled years or locations. Instead, we chose to estimate site size by creating a boundary around the coordinates of positive samples, which are generally taken to estimate the outer boundaries of symptomatic plants. The use of buffers to estimate total infestation size is

corroborated by spatially explicit sampling around infested sites, and accounts for asymptomatic infection on the periphery of sites as a result of local spread. It does, however, over-simplify our estimation of the actual number of trees contributing inoculum, presenting a source of error in our models.

If the number of sites identified in a given year is a function of the number of LDD events that occur two years prior, collectively these events would produce new infections with greater dispersion than a year in which LDD occurs less frequently. Provided all sites in a given year spread locally at close to the same rates regardless of their initial size, this would result in larger infestation summed over the entire study area. Local spread may be augmented by weather conditions the year before detection (as determined, for example, by spring precipitation the year before, which was retained in our model, Table 3.3). Regardless, as infestation size of the year prior was a strong determinant in both epidemic models, this suggests that by reducing the size of inoculum sources contributing the spread of *P. ramorum*, the eradication program has significantly reduced both the maximum distance moved and the size of SOD sites in a given year.

We identified a change in the progression of the epidemic observed in the Borax area in 2011 (Fig. 3.3, Fig. 3.4), and can speculate that sporulation in 2009 can account for infection detected in this year (Fig. 3.5). The significant increase in infestation size in the Borax area is likely due to the increased size of the area contributing to spread, specifically the sporulation from trees infected by secondary inoculum at sites detected in 2009. Under eradication conditions, these trees would not have normally contributed to further spread (Fig. 3.5). Leaving adjacent trees to sporulate in the Borax area in 2009 has only compounded the extent of this epidemic.

The events of 2009 were not the first time that trees had been left without treatment, either because of minor delays in funding or because some sites were wrongly identified as negative for *P. ramorum*. Yet we have not seen such a dramatic increase in infestation area immediately around these locations as was observed in 2011. Minor delays did not extend a whole year, as happened in the Borax area in 2009, suggesting that they were treated before contributing significantly to spread. The misidentification of sites has been uncommon over the 10 years that the eradication program has operated, and the lack of their contribution is predominantly due to their small size. Larger inoculum sources have a greater potential to contribute to epidemic development relative to smaller sources (Madden et al. 2007). We suspect that the difference in size between one missed site and the extent of the infestation left standing in the entire Borax area in 2009 can account for the disparity in epidemic development observed two years later in either scenario. This also explains why the small, isolated distal sites on the extreme northern margins of the NChetco area have not as dramatically contributed to spread and establishment of *P. ramorum* as the much larger infestation further south.

The inclusion of infestation size of prior years in our models, and significantly larger infestation size and longer (though insignificant) dispersal distances observed in the Borax area in 2011 following the delay in treatment are strong evidence that the eradication has reduced the spread of this epidemic. As infections are left standing without eradication in the southern end of the quarantine boundary, we expect to see an increase in the amount of infection in following years, especially on the periphery of known established areas. We also expect to see the pathogen move further distances as the size of infested areas is allowed to grow. Both responses, however, will be significantly augmented by weather conditions that affect the spread and establishment of *P. ramorum*.

SUMMER SPORULATION AND SYMPTOM DEVELOPMENT

INTRODUCTION

Numerous studies have suggested that the dry, warm Mediterranean summers initiate a dormancy period for *P. ramorum* in forest ecosystems (Davidson et al. 2011, 2008, Fichtner et al. 2007, 2009). This includes the reduced recovery of *P. ramorum* from soils, leaf litter, and attached bay laurel leaves over summer months, presumably due to increasing temperatures and declining moisture (Fichtner et al. 2007, DiLeo et al. 2008, Davidson et al. 2011). While symptoms continue to develop after the spring rains have ended, spore production stops in drier years by May despite occasional summer rains (Davidson et al. 2008) (Appendix G Fig. G.1). With the onset of autumn rain, inoculum production in California does not noticeably start again until January (Davidson et al. 2011) (Appendix G Fig. G.1). The recovery of *P. ramorum* in soils is correspondingly delayed, potentially because of the need to break dormancy after over-summering (Fichtner et al. 2007).

This strong seasonality is observed in both mixed-evergreen and in redwoods forests, the two forest types most commonly associated with SOD in California (Davidson et al. 2011). Sporulation at both forest types, however, was assessed as recovery of inoculum from California bay laurel (*U. californica*). Consistent among most epidemiological studies is the conclusion that bay laurel is the dominant host supporting the sporulation contributing to disease establishment and severity (McPherson et al. 2010, Cobb et al. 2010, Maloney et al. 2005, Meentemeyer et al. 2008b). That *U. californica* does not suffer significant injury from *P. ramorum* exacerbates spread within a site (DiLeo et al. 2009, Cobb et al. 2010).

While sporulation is undoubtedly important once bay laurel becomes infected, little is known about the role this host plays in the initial establishment of *P. ramorum* when primary inoculum loads are relatively small. In the Douglas-fir / tanoak forests around Brookings, OR, many sites have little to no bay presence. When present, *U. californica* has not been found harboring significant infection, at least in comparison to the tanoak at that location. Studies have also shown that under low but consistent inoculum levels (for example, along waterways bearing inoculum), California bay laurel leaves are not commonly found infected by *P. ramorum*. Rather, infection is preferentially found on tanoak (*N. densiflorus*). As most SOD sites in Oregon are the result of new infections originating from primary inoculum, we expect to see early-infection dynamics playing a significantly stronger role in Oregon epidemiology than in California.

It has been our observation has been that *P. ramorum* is more aggressive on tanoak compared to most other hosts, and that tanoak is the host responsible for the initial establishment of this pathogen. Tanoak is common in coastal forests within its range, though most epidemiology has not focused on this host as a strong contributor to spread and establishment of *P. ramorum*. This is predominantly because tanoak is not as prolific a producer of sporangia as bay laurel (Davidson et al. 2008). Although the number of sporangia can be 3-4 times less than the maximum measured from bay, spore production can occur under a much wider range of conditions from tanoak (Davidson et al. 2008). Recovery of inoculum from individual tanoak twigs showed no significant difference between wet or dry years, or between the start and end of the rainy season, although the amount of overall sporulation recovered was low (Davidson et al 2008). The difference in timing of sporulation on these hosts has strong implications for the modeling of epidemic development, particularly if sporulation outside of the times attributed to bay laurel is contributing to new infection.

Early reports on the recovery of inoculum in baited bucket traps have shown inoculum may be produced in both early autumn and during brief summer rains in Oregon, a phenomenon not observed in California (Hansen et al. 2008, Davidson et al. 2008). If sporulation, infection, and summer recovery from tanoak lesions do persist over the drier summer month, this may account for a lack of strong dormancy for *P. ramorum* in tanoak. Thus far, we have been unsure if inoculum produced during the summer can contribute to either local or long distance spread. Sporangia, the spore most likely contributing to dispersal, are particularly sensitive to moisture and lose viability upon extended periods of drying (Mitzubiti et al. 2000). Similarly, zoospores require leaf moisture to initiate infection (Jeger and Pautasso 2008).

Various aspects of the epidemic in Oregon have thus far been unanswerable due to the eradication program. Particularly, we have been unable to investigate how disease develops within a stand and if the patterns of symptom development mirror those documented in California. If these patterns are not similar, then we need to reevaluate how well models built to describe the Oregon epidemic predict risk of establishment given that these models were built predominantly with California epidemiological data (Meentemeyer et al. 2004, Meentemeyer et al. 2008a, Václavík et al. 2010).

The spring and summer of 2011, now ten years after SOD was first found in Oregon forests, presented a change in treatment protocol and research opportunities within infested areas. The decision was made early in the season to not treat new 2011 infestations within the core area of the epidemic, instead allocating resources to the regions on the periphery of the quarantine area. While monitoring of sporulation has continued since its first report (Hansen et al. 2008), we now have the opportunity to study

local spread and infection, and document stand decline during the first season without treatment. This affords us the capacity to thoroughly compare Oregon epidemiology to patterns described in California. This work represents results from a preliminary survey of forest stands in Oregon, with the following objectives:

1) Assess if patterns of sporulation and symptom development differ from those observed in Californian redwood or mixed-evergreen forests.
2) Discern if there is any evidence for summer infection during periods of summer sporulation.
3) Assess if recovery of *P. ramorum* from tanoak foliage, our presumed best indicator of pathogen presence in the understory, declines over the summer months, or if incidence of *P. ramorum* infection differs between tanoak and bay laurel.

METHODS

Sporulation monitoring

Monitoring of inoculum captured in collection buckets has continued since its initial description by Hansen et al. (2008). Bait leaves of rhododendron and tanoak are placed in plastic bags secured in screened, 4 L buckets. Buckets are filled with a small amount of water to prevent drying, and are then placed beneath infected tanoak trees for a period of approximately two weeks, after which the leaf baits are collected and the bags replaced. The leaves are then returned to lab to determine if *P. ramorum* or other *Phytophthora* species are present via isolation in selective media and / or PCR (Hansen et al. 2008). Only *P. ramorum* was identified to species. This method does not allow for direct quantification of inoculum, however relative sporulation was calculated as the

number of positive buckets divided by the total number of buckets deployed over the collection time period.

To assess seasonal changes in sporulation we analyzed recovery in buckets deployed between 31 October 2006 to 23 April 2008, and 30 October 2008 to 30 September 2011. As sufficient sporulation was detected after herbicide treatments (data not shown), we used all data from bait locations that had at least one positive detection and bucket collection dates until sites were burned. To summarize recovery of inoculum over these 5 years, recovery dates were segregated by season: autumn (October – December), winter (January – March), spring (April – June), and summer (July – September).

An additional analysis was performed to assess differences in recovery of inoculum in collection buckets placed underneath infected tanoak or bay laurel. Buckets were placed underneath *P. ramorum* positive California bay laurel trees retained at some eradicated sites. Buckets were installed 1 February 2011, and were collected with the same protocol over the following spring, summer, and start of autumn. To compare sporulation from *U. californica* to that from *N. densiflorus*, we used all buckets placed under tanoak in untreated areas from 1 February until 26 October 2011.

Weather data

Weather data was obtained from the Red Mound remote access weather station (RAWS) maintained by the BLM (latitude: 42°07'24" N, longitude: 124°18'02" W; elevation: 534 m; http://www.raws.dri.edu/index.html) (Fig1.1). Because each baiting period was not a consistent length (range: 9 to 22 days; average = 14 days) we averaged daily precipitation (mm) data over each collection period.

Symptom development and summer infection

Symptom development on tanoak sprouts and stems was studied in three connected tanoak patches on private-access land in the Borax study area in the summer of 2011 (Fig. 1.1). Each area had one to five deceased tanoak trees first identified and confirmed as infected by *P. ramorum* in early 2011, but minimal understory or overstory symptom development in May 2011.

Tanoaks were considered for random selection if they had a minimum of five basal sprouts or branch tips without symptoms on this and last year's growth at the time of the first observation, 13 June 2011. For each individual we tagged five asymptomatic sprouts and measured the length of the expansion of new growth. Diameter at breast height (dbh) was taken, then trees were assessed for crown fading or flaring, and bleeding exudate indicative of inner bark cankers, then ranked on an ordinal scale from 0 – 5 indicating the condition of their basal sprouts: 0 = no sprout symptoms present; 1 = one or two sprouts symptomatic; 2 = up to ½ sprouts are symptomatic; 3 = ½ to ¾ of sprouts are symptomatic; 4 = greater than ¾ sprouts are symptomatic; 5 = most sprouts are dead. A total of 90 trees and 450 sprouts were tagged. Bleed height from the soil line was measured to the nearest 0.3 m.

Every four weeks until mid-September all trees were reevaluated for symptoms (4 assessment dates total). We measured the length of new growth for each sprout, and re-assessed tree and sprout health. If new growth was symptomatic but the lesion did not extend to the bud scar the sprout was collected to determine if the lesion could be attributed to infection by *P. ramorum* via culture in lab. So long as we could confirm that infection did not move from older tissues, new growth that expanded and developed lesions between observations periods was considered evidence for summer infection.

Summer pathogen recovery

To discern if *P. ramorum* is consistently recovered from tanoak lesions into and during the summer months, or if there are other *Phytophthora* species found infecting tanoak in this study area, we sampled 20 to 25 symptomatic sprouts from random trees within the study area in two week intervals between 28 May and 8 September 2011 (7 collection periods total). On the last collection period we also gathered symptomatic bay leaves in the understory of infected tanoak to determine the extent of *P. ramorum* infection on bay, or if other pathogens are found infecting California bay laurel at this locale.

Isolation and identification of *Phytophthora* species

All leaf and stem samples were stored in a cooler for a maximum of four days and returned to Corvallis, OR. Within 5 days of collection lesions were plated onto corn meal-ampicillin-rifampicin-pimaricin selective media (CARP) (Osterbauer 2004) and incubated in the dark for 7 to 12 days at 20^{o}C. Culture identification was based upon morphology of hyphae and spore structures (Appendix C). Any cultures lacking diagnostic propagules at the time of the first observation were incubated for another week and re-examined. Only *P. ramorum* and *P. nemorosa* were identified to species; other *Phytophthoras* present were noted but not identified any further.

As part of the summer infection study, a section of new growth 1 cm below the lesion edge was also plated to confirm independent infection of expanding foliage during the summer months (Appendix A Fig. A.5). A subset of culture negative stem sections were stored in 2 ml microfuge tubes at -20 ^{o}C and then sequenced with multiplex polymerase chain reaction (PCR) using primers developed by Winton and Hansen (2001).

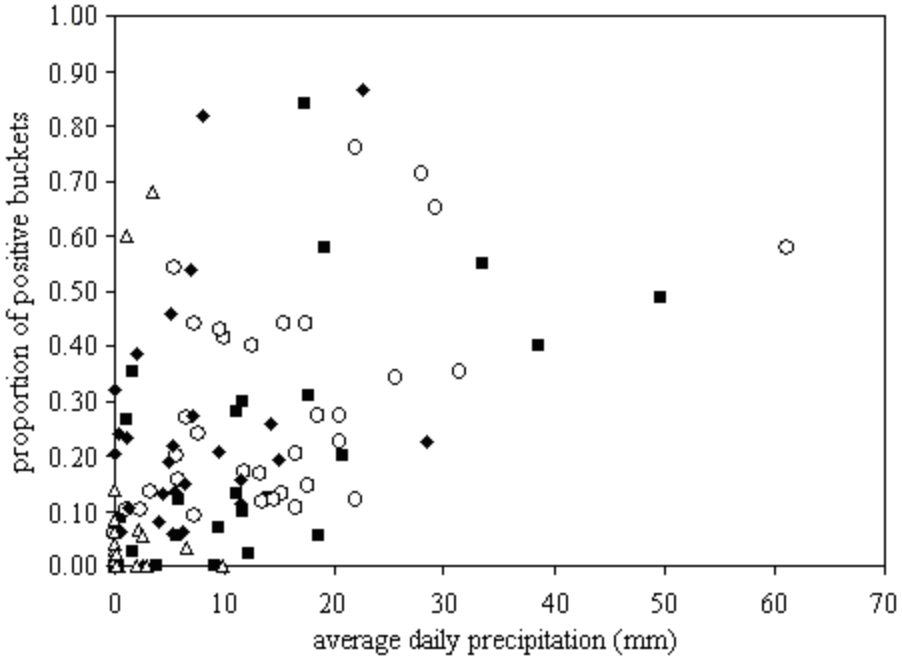


Fig. 4.1. Summary of proportion of positive buckets collected between 31 October 2006 to 23 April 2008, and 30 October 2008 to 30 September 2011. The proportion of positive buckets is the number of buckets from which *P. ramorum* was recovered / the total number of buckets deployed over each baiting period in all pre-burn areas that had at least one positive detection. Recovery is plotted against the average daily precipitation (mm) during each baiting period. Recovery dates are segregated by season: October – December (■), January – March (○), April – June (◆), and July – September (△).

RESULTS

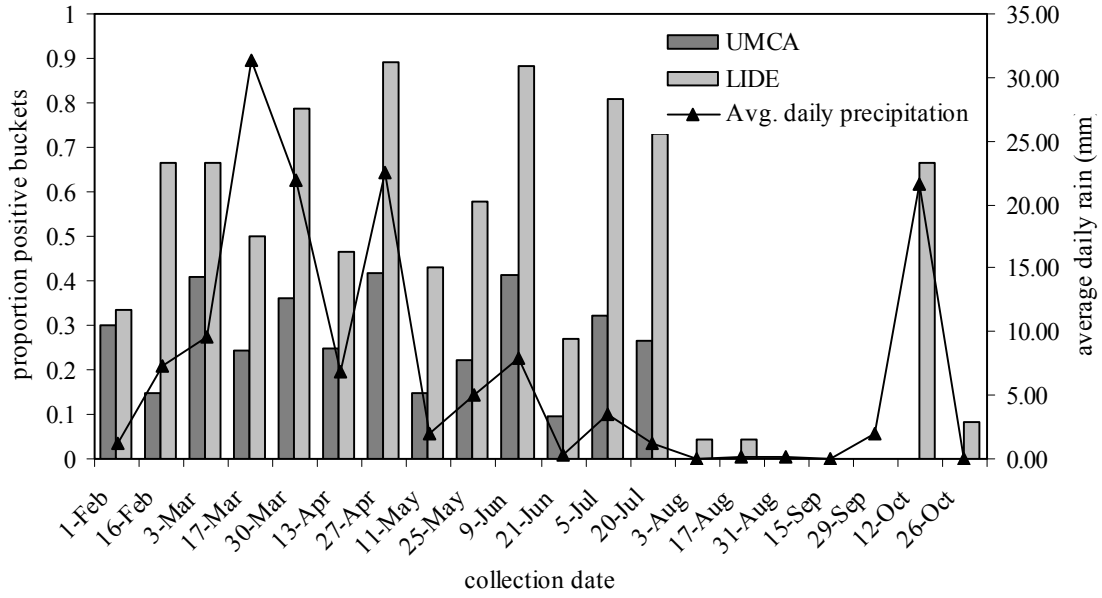
Sporulation monitoring

From October 2006 to September 2011, there was a significant, positive correlation between total precipitation over the collection period and the proportion of positive buckets (spearman rank correlation $r = 0.58$, $p < 0.0001$). Inoculum was collected during all seasons of the year, including during brief summer rains (Fig. 4.1, Appendix G Fig. G.2a-e). We observed a high proportion of positive buckets in some collection periods with low amounts of precipitation in all seasons of the year (Fig. 4.1).

During the baiting periods in 2011, *P. ramorum* was recovered from rain traps underneath both California bay laurel and tanoak (Fig. 4.2a). Recovery was greater from tanoak for all collection periods. Of the 474 buckets deployed between 1 February and 26 October 2011 (range: 12 to 28 buckets per collection period), *P. ramorum* was isolated from 211 (44.52%) buckets placed underneath infected tanoak (Fig. 4.2a). No other *Phytophthora* spp. were recovered from underneath tanoak (Fig. 4.2b). Over this same time period 632 buckets were deployed underneath infected California bay laurel (range: 10 to 40 buckets per collection period), from which multiple *Phytophthora* species were recovered during most collection periods. *P. ramorum* was recovered from 108 (17.09%) of these buckets (Fig. 4.2a); unidentified *Phytophthora* spp. were identified in an additional 100 (15.82%) buckets (Fig. 4.2b).

In 2011, recovery from both hosts declined over the summer with a decline in precipitation. Only three collection dates failed to yield any *Phytophthora* spp.: 31 August, 15 September, and 29 September 2011 (Fig. 4.2a,b). There was measurable precipitation over this time period (average daily rain = 0.11, 0.07, and 2.05 mm, respectively). During rain events in the early autumn *P. ramorum* was recovered from tanoak, but not bay laurel (Fig. 4.2a).

a. recovery of *P. ramorum*



b. recovery of other *Phytophthora* spp.

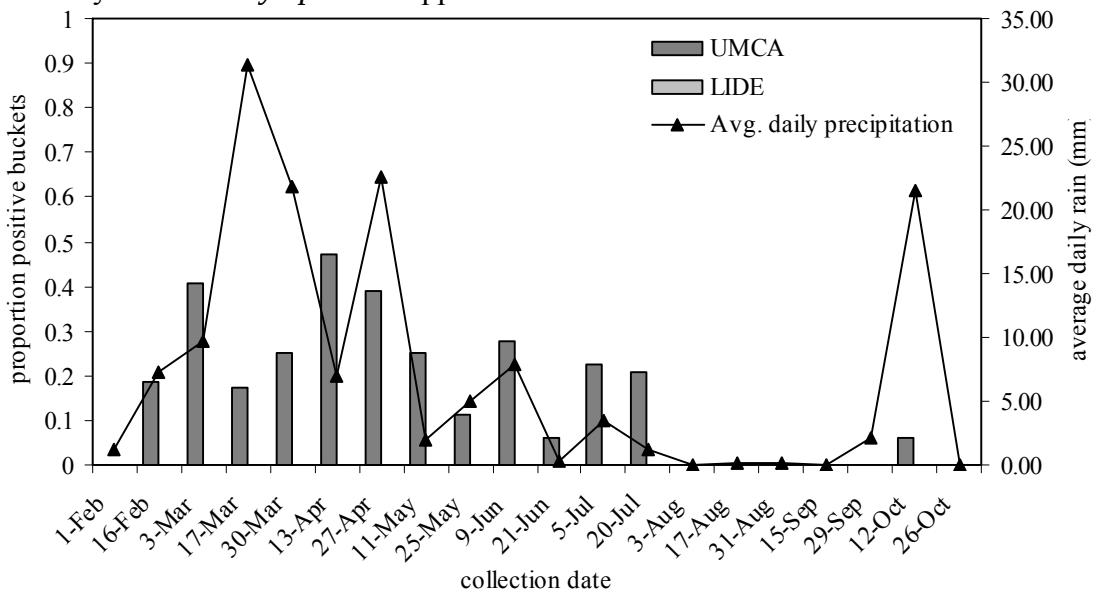


Fig. 4.2. Recovery of *Phytophthora* spp. from baited buckets placed underneath *P. ramorum* infected but non-treated tanoak (LIDE) or California bay laurel (UMCA) over each baiting period between 1 February 2011 and 26 October 2011. Recovery is presented as proportion buckets in which we isolated either *P. ramorum* (a) or other *Phytophthora* spp. (b) from leaf baits infected by inoculum in rain splash, plotted against daily precipitation (▲).

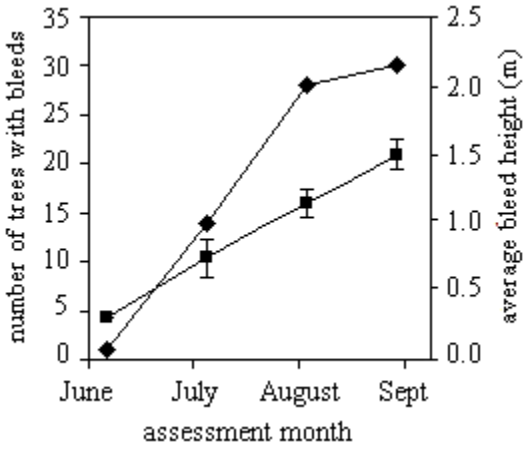


Fig. 4.3. Development of symptoms on mature tanoak trees between June and September 2011. Tree health was assessed monthly for the presence of bleeds along the main stem (◆) and maximum height of bleeds when present (■). Bars represent standard error.

Symptom development and summer infection

At the start of the summer observation study in June 2011, 34 of the original 90 tanoaks had no sprout symptoms; only 1 tree had minor bleeds < 0.3 m from the soil line. The number and height of bleeds increased over time (Fig. 4.3). By the last assessment date, 30 of the 74 trees with a dbh greater than 10 cm developed bleeds (average bleed height = 1.5 m, maximum height = 3 m) (Fig. 4.3).

A greater proportion of trees with larger dbhs developed bleeds by the September assessment (Table 4.1). Average sprout status (from 0 to 5) increased for every size cohort, regardless of initial ranking (Table 4.1, Fig. 4.4). All trees had healthy crowns at the beginning and end of the study.

Table 4.1. Symptom development of tanoak stems for each size cohort. Percentage of stems in each size cohort that developed bleeds and change in tree sprout status was measured every four weeks from 14 June to 6 September 2011.

stem dbh (cm)	# stems	% with bleeds in Sept.	average sprout status (s.e.)	
			June	Sept.
<10	16	n.a.	1.00(0.32)	1.75(0.36)
10.1 - 20	19	15.79	1.42(0.30)	2.84(0.37)
20.1 - 30	27	37.04	1.74(1.26)	3.18(0.29)
>30.1	28	60.71	1.46(0.28)	2.71(0.51)

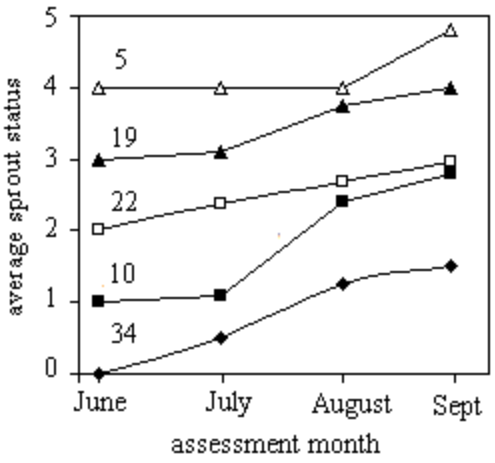


Fig. 4.4. Change in tree sprout status over the summer, separated by sprout status at the start of the observation period of June 2011: 0, no sprouts symptomatic (◆); 1, one or two sprout symptomatic (■); 2, up to ½ of sprouts are symptomatic (□); 3, ½ - ¾ of sprouts are symptomatic (▲); 4, greater than ¾ sprouts are symptomatic (△); 5, most sprouts are dead. Numbers indicate the number of sprouts at each status at the beginning of the study (total = 90 trees)

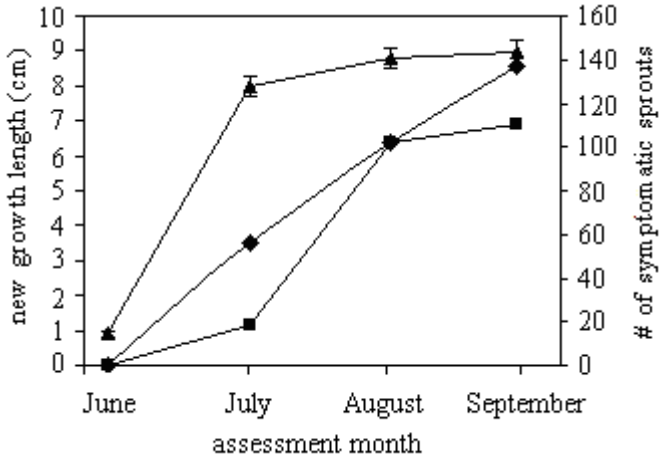


Fig. 4.5. Average new sprout length (cm) for all sprouts that expanded during the summer assessment (▲), and number of sprouts that developed lesions on this years (■) and last years (◆) growth. Bars represent standard error.

New growth expanded on 354 of the 450 tagged sprouts, predominantly between the first and second assessment dates (14 June and 13 July, respectively) (Fig. 4.5). 206 (45.78%) of sprouts developed petiole or twig lesions along either this or last year's growth. Most new growth developed symptoms within 1 month of expansion and rapidly died with infection; last year's sprout growth developed symptoms continuously over the course of the summer (Fig. 4.4). Commonly the new growth would die and form a shepherd's crook due to infection on older tissues. We did find symptomatic new growth without apparent infection on last year's tissues, although at the time of each observation most of these sprouts had symptoms extending the whole length of new stems.

Only a few sprouts had developed symptoms recently enough to allow us to identify the potential point of introduction. We sampled the new growth of 36 of these sprouts to verify independent infection of new growth after expansion. Independent infection of

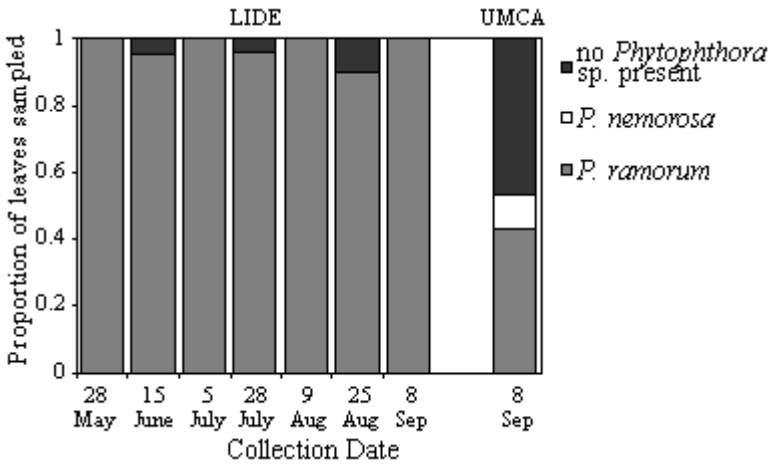


Fig. 4.6. Isolation of *Phytophthora* spp. over the summer of 2011 from tanoak (LIDE) and California bay laurel (UMCA). Only ***P. ramorum*** and ***P. nemorosa*** were recovered.

expanding sprouts was verified on most symptomatic stems in culture: 77% of tissues sampled 1 cm below the lesions were culture negative. ***P. ramorum*** was absent from these segments in all PCR analyses. The remaining 33% of sprout segments that were culture positive were isolated from asymptomatic tissue too close to the leading edge of infection. Spring rains and sporulation continued during the time of expansion, so we could not discern if infection on new growth originated from isolated summer rains (Fig. 4.2, Fig. 4.5).

Summer pathogen recovery

P. ramorum was recovered from 142 of the 146 tanoak sprouts sampled over all collection periods. No other *Phytophthora* species were recovered from tanoak during this study. Of the 109 California bay laurel samples taken, we isolated ***P. ramorum*** from 47 leaves, and ***P. nemorosa*** from 11 leaves. Nearly half of bay leaves collected (51 of 109) yielded no *Phytophthora* species (Fig. 4.6)

DISCUSSION

Our observations are consistent with Californian epidemiology inasmuch as recovery of *P. ramorum* inoculum increases during periods with greater daily rain. The overall trend between average daily precipitation and recovery of *P. ramorum* was positive; with some exceptions, recovery over drier collection periods in all seasons tended to be lower (Fig. 4.1, Appendix G). We did, however, observe a number of baiting periods with a high proportion of positive buckets despite small amounts of rain, even in the summer time (Fig. 4.1). Many, but not all, of these observations were made between October 2010 and September 2011 (Fig. 4.2, Appendix G Fig. G.2e). Most likely the 2010/2011 results can be attributed to the placement of bucket traps in the untreated areas of the Borax infestation, which has a significant amount of untreated tanoak infection and secondary spread of *P. ramorum* (Fig. 1.1). Although this may make the 2011 trapping season less comparable to years before, it does indicate that sporulation is possible under a wider range of conditions than previously suspected.

All forests within the current range of SOD experience dry summers, although overall there is evidence for a shorter period of summer dormancy in Oregon, if dormancy is achieved at all. While sporulation ceases by late spring in California (Davidson et al. 2008), we recovered inoculum as late as the end of July from California bay laurel, or mid-August from tanoak (Fig. 4.2a,b). Although the rainy season starts at approximately the same time in both states, between November and December, inoculum production in California does not noticeably increase until January (Davidson et al. 2011). In contrast, the months between November and January recorded some of the highest overall recovery of inoculum in Oregon (Appendix G Fig. G.2.a,c,e). Isolated rain events in October of 2011 were adequate to capture inoculum produced from tanoak (Fig. 4.2a).

Consistent with Californian observations, *P. ramorum* was not recovered underneath infected bay laurel during this same period (Fig. 4.2b).

Cumulative rain of less than 5 mm over a baiting period was sufficient for traps underneath tanoak trees to collect inoculum, for example during the baiting periods collected on August 3 or 17, 2011 (Fig. 4.2a). Unfortunately, we are unable to discern if sporulation occurred strictly during the rain event that brought inoculum down into the bucket, or during the weather conditions preceding precipitation. It has been postulated that heavy dew or fog could provide sufficient leaf moisture to support sporulation from tanoak, although with our current monitoring methods we can only reliably detect sporulation in rain splash. Unfortunately, preliminary attempts to trap sporangia of *P. ramorum* in fog traps or aerial spore traps have thus far proven inconclusive.

The method employed in Oregon to monitor sporulation has proven to be remarkably sensitive to spore production during times of limited precipitation, probably due to the presence of water and a bait leaf allowing for infection and persistence of the pathogen. Although we cannot directly quantify inoculum, this approach is better at assessing the presence or absence of *P. ramorum* than the method employed in California. Instead of baiting rain traps and plating the baits for a measure of incidence, Davidson et al. (2008, 2011) filtered rain captured in buckets, then plated the filters and counted colony forming units as a way to assess sporulation. This allows for better resolution of inoculum quantity, especially under higher inoculum loads. Assessing sporulation as only the proportion of positive buckets makes a crude estimate of sporulation quantity as it is likely more prone to correlation with the amount of precipitation and could be saturated when sporulation is high. As such, those methods employed in Oregon may be better used as a management tool to identify if inoculum is present (before or after treatment,

for example). Regardless, our technique may also better represent sporulation at the end of spring or start of autumn, sporulation that would have been missed with Californian methods.

Both the extension of late spring rains and abundance of tanoak infection are contributing to a shorter period of inactivity over the summer months. While recovery of inoculum underneath bay laurel displayed recovery patterns akin to those observed in California, recovery from tanoak ensued with the autumn rains. We are uncertain whether this is related to actual sporulation, or differences in retention of leaves harboring *P. ramorum* infection. Davidson et al. (2011) has shown that bay laurel leaves infected by *P. ramorum* are preferentially shed over the summer months, especially in drier climates. In contrast, tanoak sprouts are retained with infection (pers. obs.). Given that recovery of *P. ramorum* from tanoak foliage was as consistent at the start of the summer as the end (Fig. 4.6), we suggest that *P. ramorum* never undergoes a strict dormancy while in tanoak foliage. If infection on tanoak does enter a dormancy, then the process of breaking dormancy may not be as significantly delayed as observed in bay laurel in California.

As spring rains extended into the time period new growth was expanding without a significant interruption, we failed to identify if isolated summer rain events can contribute to new infection (Fig. 4.2, 4.5). Having verified new independent infection on sprouts that expanded between the July and August assessment dates, we did, however, document that infection is possible as late as July (Fig. 4.5). Environmental conditions during the autumn, winter and spring (but not summer) seasons have been shown to explain differences in maximum distance moved or infestation size of a given year, suggesting that sporulation during these seasons may be more important drivers of epidemic development. Importantly, we did not observe a difference in development of

symptoms between new growth or sprouts that were a year old (Fig. 4.5). It is likely that infection is not limited to times of the year when new tissues are available. Regardless, we conclude that July infection under the low-inoculum conditions we observed over the summer is representative of the ease with which this host is infected by *P. ramorum*, making tanoak the most likely source and recipient of inoculum at distant locations.

Despite differences in timing of sporulation, symptom expression is similar between Oregon and California. In both locations, the development of symptomatic tissues lags behind the rainy season, with maximum symptom expression occurring between June and August. We observed an increase in symptom development over the summer assessments in all disease measures: the development of symptoms on new and year old sprouts (Fig. 4.5), tree sprout status (Fig. 4.4), and number and height of bleeds on mature stems (Fig. 4.3). The propensity for larger trees to develop bleeds (Table 4.1) is also consistent with observations of tanoak in California (McPherson et al. 2005, 2010). Given the relative absence of disease at the start of this study, we conclude that symptoms that developed over this observation period were the result of recent infection, most likely from secondary inoculum produced by the few initially infected trees in the study area. Significantly, no trees died over this summer, leading us to suspect that these trees are in their incubation year and are contributing to long distance dispersal. To be consistent with our temporal model of SOD in Oregon, we expect those trees that developed symptoms in 2011 will most likely die within the next year.

The emphasis that the epidemic in Oregon is being driven by tanoak rather than bay laurel has been a cornerstone in understanding Oregon epidemiology. Recovery of *P. ramorum* over this summer was consistent with our experience of this pathogen in

Oregon: *P. ramorum* is more aggressive and is isolated more readily from tanoak than bay laurel, at least under the early stages of spread. All isolations made from rain baits placed underneath infected tanoak canopies at multiple sites throughout *P. ramorum*'s current range, as well as those isolates recovered from understory foliage collected in the Borax area, were positive for *P. ramorum* and no other species (Fig. 4.2a,b, Fig. 4.6). While *P. nemorosa* is present infecting foliage where we gathered symptomatic sprouts as part of this study (Fig. 4.6), if this species is infecting tanoak it is doing so at a frequency too low to be detected with our sample size. The greater aggressiveness of *P. ramorum* on tanoak further suggests that initial establishment is dependent upon this host.

The lack of bay infection under current conditions also supports this conclusion. Our observations have been that despite bay laurel's exposure to moderate levels of *P. ramorum* inoculum, infection is less common on bay than tanoak. Less than half of the symptomatic leaves collected as part of our study were found to be infected by *P. ramorum* (Fig. 4.6). It is possible that *P. ramorum* was dormant in leaves in which we failed to isolate a pathogen, or that leaves infected by *P. ramorum* had been shed by the time we gathered foliage. Still, the species diversity and frequency with which we recovered *P. ramorum* from leaves was similar to that recovered from buckets underneath bay laurel trees during the entire baiting period (Fig. 4.2a,b).

Common garden experiments have shown that though there is significant variation in bay laurel susceptibility between and within populations, seasonal conditions can affect susceptibility to *P. ramorum* in the field (Hüberli et al. 2011, Anacker et al. 2008). We cannot discern whether the observed infrequency of *P. ramorum* infection on bay laurel can be attributed to lower inoculum densities, inheritable lower susceptibility of Oregon

populations to *P. ramorum*, subtle variation in weather patterns, competition from other *Phytophthora* spp., or some combination of these factors. Regardless, under the current conditions in Oregon, establishment of disease is not dependent upon bay laurel. Hüberli et al. (2011) suggested that due to the lower susceptibility of the Oregon population of bay laurel to *P. ramorum*, the epidemic in Oregon would not proceed as quickly as has been observed in California. This conclusion is not supported by our observations over the summer of 2011, whereby areas with a low abundance of bay laurel developed extensive symptoms comparable to that observed in California.

The niche models built to predict disease severity or map areas at greater risk for SOD establishment have included the weather conditions between the months of December and May as the best predictors of inoculum production. Given that these models were originally built to describe the epidemic in California, they have the weighted risk of establishment towards areas in which bay is present, and under conditions in which bay is know to support appreciable amounts of sporulation (Meentemeyer et al. 2004, Meentemeyer et al. 2008a, Václavík et al. 2010). The concessions made to adapt these models to Oregon have been to weight tanoak presence as a more significant contributor to risk than bay laurel (Václavík et al. 2010). Due to the over-prediction of bay laurel in map layers used to estimate host range in the Brookings area (pers. obs.), some areas may be classified as higher risk than may be expected under the current conditions we observe in Oregon. Further studies are needed, however, to determine if bay infection is expected to increase and contribute more to the establishment of *P. ramorum* once the eradication stops controlling inoculum loads.

GENERAL DISCUSSION

The past century's expansion of global trade in plants and, inadvertently, their pests, has resulted in the introduction of tree diseases having major impacts on native forest ecosystems. Some of these exotic pathogens, for example chestnut blight, caused by *Cryphonectria parasitica*, white pine blister rust, caused by *Cronartium ribicola*, or root rot of Port-orford cedar, caused by *Phytophthora lateralis*, have threatened populations of a single tree species (Anagnostakis 1987, Geils et al. 2010, Hansen et al. 2000). Others, for example root rot caused by *Phytophthora cinnamomi*, threatened to infect whole communities of plants (Cahill et al. 2008). Irrespective of host range, these exotic pathogens impact native and human ecosystems through the loss of ecologically, structurally and culturally important trees. Combined with the threats from climate change and human encroachment, these invasives have the capacity to change landscapes.

By their nature, invasives are difficult to control. Management of invasive pathogens in natural ecosystems is especially difficult given the patchy and extensive distribution of hosts and disease, difficult to access lands, and multiple mechanisms of spread which are difficult to assess in early stages of invasion processes. Management approaches addressing invasive forest pathogens have been widely varied, including: the introductions of avirulence imparting viruses or competitive fungi (*C. parasitica*, Anagnostakis 2001; *C. ribicola*, Maloy 1997), removal of alternative hosts (*C. ribicola*, Maloy 1997), the breeding of natural or introduced resistance in hosts (*C. parasitica*, Griffen et al. 2005, Diskin et al. 2006; *C. ribicola*, King et al. 2010; *P. lateralis*, Sniezko and Hansen 2003), or the prevention of introduction of inoculum into new host populations (*P. lateralis*, Hansen et al. 2000; *P. cinnamomi*, Cahill et al. 2008). None of

these measures have succeeded in eradicating targeted pathogens. Rather, persistence of invasive pathogens after establishment favors more adaptive management approaches, requiring long term commitment of land managers and funding agencies. Unfortunately, new introductions erode limited funding for evasive and eradicative control strategies.

Phytophthora ramorum, causal agent of sudden oak death (SOD), is one recent example of this continuing exotic pathogen problem. Although ***P. ramorum*** causes mortality on only a few host species, as a generalist pathogen with non-descript symptoms this species is difficult to manage in nursery, landscaped, and natural environments. Different approaches have been considered and implemented: in California, where the disease is established on its largest scale, active management has focused on preventing the movement of soils and plants bearing inoculum into new areas, treatment of individual trees with phosphate-based fungicides, removal of foliar hosts adjacent to terminal hosts (those hosts that succumb to SOD but do not support sporulation), and potential breeding of natural resistance in some *Quercus* species (Garbelotto and Schmidt 2009, Dodd et al. 2005, COMTF website). To prevent the further spread north from the heavily infested areas of central California, a barrier zone at the Van Duzen River was also constructed with the goal of removing the main hosts responsible for local spread (Frankel 2008). The most ambitious control measure thus far has been the eradication program in southwest Oregon.

During the initial emergence of invasive pathogens into new ecosystems, little is known about how the complex of environmental, host, and pathogen dynamics influence the rate at which an epidemic proceeds (Holdenrieder et al. 2004). The speed at which SOD research has progressed to answer epidemiological questions since trees first started

dying in the San Francisco Bay Area in the mid-1990's has been commendable. *P. ramorum* was described as a new species in 2001, the same year that SOD was detected in Oregon (Werres et al. 2001). Since that time an appreciable amount of information has accumulated about the population structure and community dynamics contributing to local spread and establishment of *P. ramorum*. The goal of fully eliminating *P. ramorum* from Oregon forests was expected to be difficult, especially since very little was known about *P. ramorum* in 2001. With the hindsight of ten years of effort, eradication has proven more challenging than anticipated. The length of time between initial infection and detection, and potential for aerial dispersal are two unsuspected factors contributing to the continued expansion of SOD.

The long distance spread of *P. ramorum* northward up the North Fork of the Chetco River and Bravo Creek, and the jump of *P. ramorum* to the east and west in 2006 were significant events that all but eliminated the possibility of full eradication. This movement of *P. ramorum* on the landscape has been shown to be inconsistent with the operating assumption that *P. ramorum* was dispersed long distances in infested soils, predominantly by people. Forest *Phytophthora* spp. had not, at the time, known to be dispersed by any means other than in soil, water, or plant material. The movement of some agricultural oomycetes can be attributed to dispersal in drying winds whereby the estimated upper limits of dispersal distances range from 3 km for *Bremia lactucae* (lettuce downy mildew), to 50 km for *P. infestans* (potato late blight), to greater than 500 km for *Peronospora tabacina* (blue mold of tobacco) (Ristaino and Gumpertz 2000, Wu et al. 2001, Aylor 2003, Aylor and Taylor 1982).

We remain unsure if *P. ramorum* disperses by a means mechanically similar to these other pathogens, or if dispersal at the km scale is the result of movement in fog, or in

rare, strong storms. Various studies have suggested that due to the morphology of *P. ramorum* sporangiophores on some hosts, aerial dispersal in unlikely (Mascheretti et al. 2008, Moralejo et al. 2006). Evidence in support of aerial dispersal is found in the spatial distribution of disease and the recovery of *P. ramorum* from the upper branches of overstory tanoak trees; however, we have been unable to trap sporangia in spore traps used to assess the movement of classic aerial dispersers. *P. ramorum* is potentially one species whose morphology and life cycle is less conducive to aerial dispersal when compared to *P. infestans* or *P. tabacina*. There are other oomycetes, though, for which aerial dispersal is rarer and spatial patterns of disease strongly suggest that soil or water-borne inoculum is more important for spread (e.g. *Phytophthora capsici* as investigated by Granke et al. 2009, or *P. lateralis* and *P. cinnamomi*). In contrast, it has been our observation that the range and frequency at which aerial dispersal is possible has contributed significantly to the establishment of *P. ramorum* in Oregon. Aerial dispersal of *P. ramorum* has implications not only for monitoring and quarantine protocols, but on the modeling of epidemic development on the landscape.

While some models built to describe the epidemic in California have included a term meant to gauge the relative risk of dispersal from close versus distant inoculum sources, assessments of the landscape-scale risk of establishment by *P. ramorum* have focused on environmental suitability and host distribution rather than specific dispersal mechanisms (Meentemeyer et al. 2004, Meentemeyer et al. 2008a). Dispersal is an important component to model accuracy, however, especially early in the invasive process (Václavík and Meentemeyer 2009). For aerial dispersal, most new infections occur close to the source, although some inoculum can move considerable distances as modeled by a fat-tailed dispersal curve (Kot et al. 1996, Sackett and Mundt 2005a, Aylor 1990, Wingen et al. 2007). With this model a disproportionally larger amount of inoculum lands closer

to the source relative to that expected from a negative exponential curve, however the chance that inoculum may move greater distance is much larger with a fat-tailed curve. Models incorporating a specific dispersal function should not use a negative exponential curve as this will overestimate the risk of infection at median distances away from the source, while underestimating the risk at distant locations (Sackett and Mundt 2005b).

Early models incorporating establishment risk as a function of dispersal have included exponential curves, although the most recent model (Meentemeyer et al. 2011) has tested the fit of various curves and have found that a fat-tailed curve better modeled the SOD epidemic. In and of itself this is not significant, although the results of Meentemeyer et al. (2011) lends another piece of information, which in combination with this work and the preliminary disease curves of Hansen et al. (2008), supports our hypothesis of aerial dispersal. As a result of expected dispersal gradients of inoculum attributable to aerial dispersal, the development of an epidemic resulting from aerial movement results in invasions that accelerate over time (Mundt et al. 2009, Sackett and Mundt 2005b). Within the limits defined by spore production at the source, spore survival and infection after transport, and host availability (Aylor 2003), we expect that the rate at which the SOD epidemic progresses will continue to grow over time, especially as areas of the infestation are left untreated. This may be observed as either increasing distances that *P. ramorum* is able to spread, or an increasingly rapid rate at which *P. ramorum* will colonize the periphery of heavily infested areas, so long as hosts are available. Fortunately, dispersal of *P. ramorum* appears to be heavily influenced by weather conditions. Hopefully this will allow additional time to implement more thorough control measures in years with smaller infestation sizes and ranges.

There are three lingering questions about the dispersal mechanisms responsible for spread and their impact on success of the eradication. First, if *P. ramorum* had been dispersed solely in soils, would the eradication have succeeded in its original goal of full elimination of SOD from Oregon? When *P. ramorum* was found infecting tanoak trees in 2001, 5 of the original 9 sites were on rural residential properties and thus posed a higher risk for spread of soils by people (Hansen et al. 2008). Given the loss of viability of *P. ramorum* in dry soils (Fichtner et al. 2007), the risk of movement away from these areas could have been negated with properly established and enforced quarantine boundaries during wet months, especially since at all but one site infection was found greater than 100 m from houses (Hansen et al. 2008). What made the eradication possible was that at the time of first detection, the cause of SOD had been identified and the initial infested areas were small. This contrasts with the situation in California where by the time *P. ramorum* was identified it had already established beyond the range of significant management.

Second, if wind patterns contribute to the northern spread of *P. ramorum*, when do we observe winds from the south? Winter and spring sporulation is thought to be the main contributor to spread, during which times average daily wind is blowing more often from the south (Fig. 5.1). While dominant wind patterns indicate that dispersal would contribute to a northerly spread more often in the winter months, a quarter to a third of the days during this time period were blowing from the north. Additionally, we see sporulation during all times of the year, and cannot rule out the possibility that spores produced during late spring or summer months are not contributing to long distance dispersal.

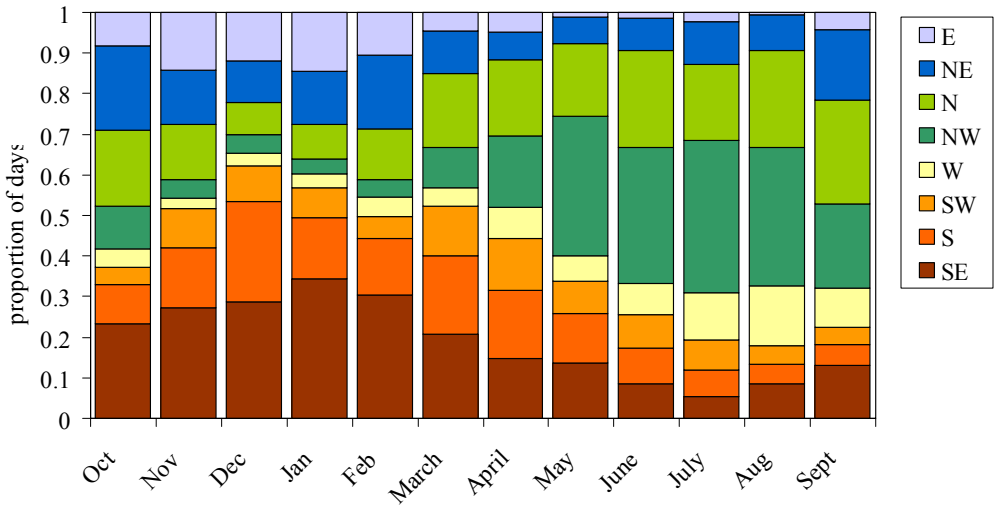


Fig. 5.1. Summary of wind direction by month, calculated as proportion of days in which the average daily wind direction was blowing from each of the following directions by month between 1 October 2001 to 30 September 2011: N = 337.6-360° or 0-22.5°, NE = 22.6-67.5°, E = 67.6-112.5°, SE = 112.6-157.5°, S = 157.6-202.5°, SW = 202.6-247.5°, W = 247.6-292.5°, and NW = 292.6-337.5°. Warm colors indicate directions that are, overall, blowing from the south; cool colors indicate directions that are blowing from the north. Data: Redmound RAWS weather station.

It is less likely that summer sporulation is contributing to significant long distance movement due to increased solar radiation and lack of moisture over summer months. Both environmental conditions serve to decrease inoculum amounts, either through poorer spore production or increased spore mortality, resulting in shorter dispersal distances (Aylor 2003). Regardless, average daily wind direction is not the best indicator of times of high risk of spread, especially if sporulation contributing to aerial dispersal occurs only during a particular time of day. For other aerially dispersing oomycetes, spores are typically produced during cooler, wetter periods over the evening, and are then released in response to increasing light and decreasing humidity late morning (Leach 1982, Su et al. 2000, Aylor et al. 2001). Hourly changes in wind, cloud cover, or precipitation are not well represented in a daily average assessment of weather

conditions, although these conditions strongly influence the potential for successful dispersal. Nor would this apply to a storm-driven mechanism. Until we can document the precise timing of sporulation, how the particular climate and wind patterns of the North Chetco area influences the movement of sporangia will allude us.

Finally, does aerial dispersal pose a risk over the entire range of *P. ramorum*, or in landscaped or nursery environments? It is difficult to extrapolate the situation in Oregon to other areas, especially since the Oregon landscape is so extensively populated by an easily infected host. There is no reason not to suspect that at least some of the spread of *P. ramorum* in California is due to aerial dispersal, although with the extent of local spread and greater host heterogeneity it would be more difficult to detect a spatial pattern as we observed in Oregon. Aerial dispersal poses a risk to any susceptible population close to inoculum sources, although it very well may be that episodes of aerial dispersal are rare. Regardless, these events have prevented the eradication program from fully meeting its goals, especially in light of the recent development of a new site considerably outside of the 2008 quarantine boundary.

In addition to having a significantly larger than expected extent of infestation in the Borax study area, the sites discovered in 2011 have also set a record in maximum distance moved. In the late summer of 2011 a new site at Cape Sebastian State Park was identified as positive for *P. ramorum*, 18 km from the nearest known infection. Preliminary microsatellite analysis has shown that the isolates recovered from Cape Sebastian are of the same clonal population established in the North Chetco-Borax area. Cape Sebastian was not included in our analysis as we cannot discern whether this new site originated from the North Chetco or Borax area. How inoculum reached Cape Sebastian is unknown, although it is possible that the delay in 2009 responsible for the

significantly larger infestation size in the Borax area could have also produced a significantly further dispersal distance. Although 2010 marked the last year that infection was treated in the extreme southern end of *P. ramorum's* range, the Cape Sebastian site will continue to be treated with the same eradication protocol that has been implemented until recently. This provides us, for the first time, an opportunity to compare the development of the SOD epidemic in Oregon with and without treatment.

So long as the eradication protocol of the Cape Sebastian site proceeds as normal and SOD infection further south does not contribute more inoculum to this area, we expect that the regression models built to describe variation in maximum distance moved and infestation size will apply to this new area. Based on weather and epidemic variables of the years prior, we can use these models to predict the extent of the epidemic we expect to see in next the year. This allows the ODF to gauge the amount of resources needed for the upcoming year. Monitoring of SOD over its entire range in Oregon will hopefully continue in order to assess how the range of long distance dispersal or infestation size changes in the absence of control in the south. This will provide further data to determine the effectiveness of the eradication program.

Ending remarks

Our understanding of the pathosystem of *P. ramorum* in western United States forests and nurseries has evolved considerably since this pathogen's description in 2001. This pathogen has forced ecologists and commercial and private landowners to assess the value of tanoak, a tree once considered expendable for its general abundance and current lack of commercial use, but which may become the chestnut of the west coast (Bowcutt 2011, Hayden et al. 2011). It has required the collaboration and quick commitment of resources of multiple agencies. It has forced us to reevaluate, test, and verify the

accuracy of interstate horticultural commerce and has tested the limits of international trade and other countries' trust of APHIS quarantine boundaries.

As an extension of our research, this pathogen has forced us to reevaluate much of what we know about *Phytophthora* pathosystems in general. We have redefined how we look at all *Phytophthora* spp. in forests and waterways. Monitoring for this pathogen has enlightened us to their relative abundance, be they invasive, presumed non-native (but not invasive), or native. Our empirical observations have suggested this *Phytophthora* is capable of aerial dispersal, a precedent amongst forest-infecting species in this genus. The monitoring efforts that made this work possible have been aided, in no small part, by the collaboration of local public and councils, state agencies, and federal institutions, but also by the seamless integration of new technology with good, old fashioned forest pathology. Indeed, never before has an invasive forest epidemic occurred with the availability of monitoring tools being utilized to describe this epidemic so soon in its development. While the eradication program has not fully eliminated *P. ramorum* from Oregon, it appears to have significantly delayed its expansion, benefiting businesses and public alike.

The introduction of invasive pathogens will continue to be a problem so long as hosts bearing pathogens are moved into foreign environments. The mitigation of their impact, especially in this case, was dependant upon the understanding that control and monitoring programs have significant benefit to the general public, legislative agencies, and the researchers seeking to understand the epidemiology of these pathogens. The SOD eradication program would have not been possible without this understanding. In this regard the management of *P. ramorum* in Oregon has been a success and has set a precedent for how invasive forest pathogens may be managed in the future.

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APPENDICES

Appendix A. Field symptoms of *P. ramorum* infection

Fig. A.1. Bleeding exudates on the outside of mature tanoak stems (source: Parke and Lucas 2008)

Fig. A.2. Inner-bark canker on tanoak, showing margin between diseased (dark) and healthy (light) tissue (source: Parke and Lucas 2008)

Fig. A.3. Lesions on tanoak sprout stems and petioles characteristic of *P. ramorum*. (E.Peterson)

Fig. A.4. Leaf lesions indicating potential infection by *P. ramorum* on California bay laurel (source: Parke and Lucas 2008)

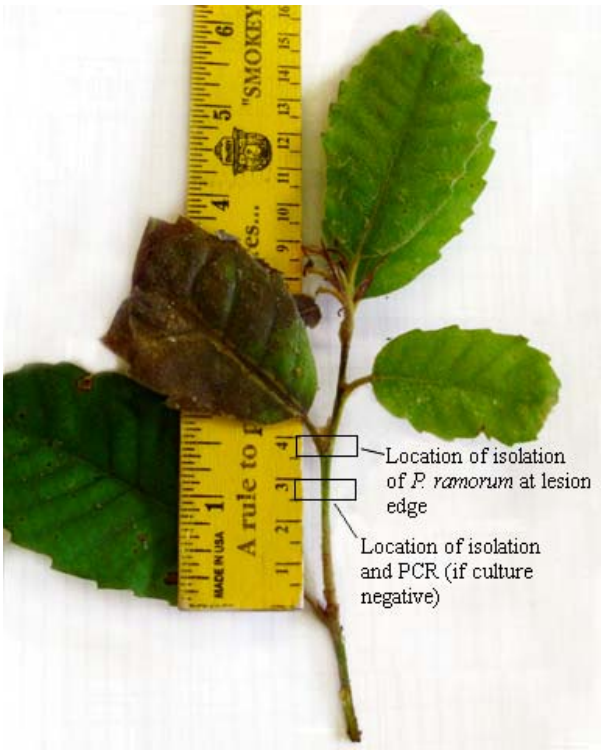


Fig. A.5. Symptoms on newly expanded sprout tissues. The two boxes refer to locations where we plated stem tissue to isolate *Phytophthora* spp. The upper box is at the lower margin of the lesion edge; the lower box was approx. 1 cm below the lesion edge, and was plated to verify independent infection of new tissues in the summer of 2011. These latter segments were later verified by PCR analysis when culture negative. (E.Peterson)

Fig. A.6. Crown flaring of overstory tanoak, the symptom detected by overstory surveys. This picture was taken within the Borax area in the summer of 2011. (E.Peterson)

Appendix B. Figures and MATLAB code to accompany the test for spatial independence

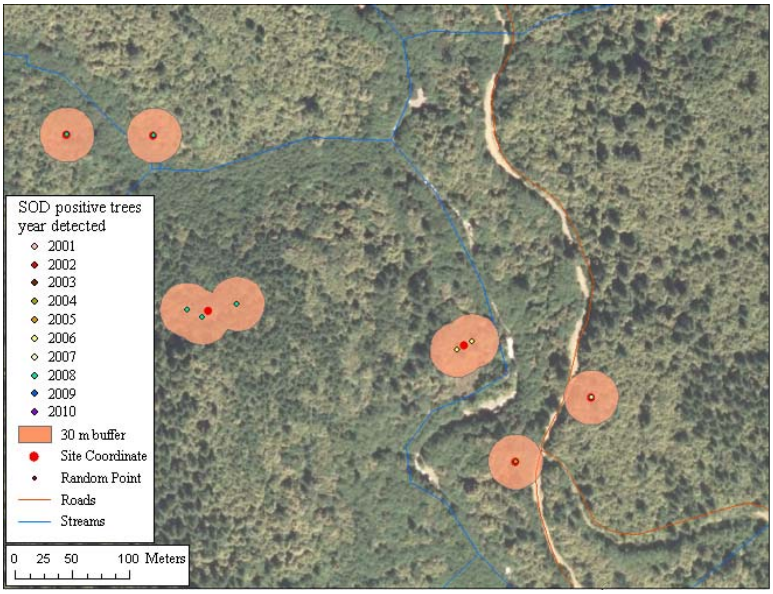


Fig. B.1. Example of how proximal trees identified as positive for *P. ramorum* infection were collapsed into site coordinates. A 'site' was defined as the centroid of all positives within 60 m of one another; when only one positive isolation was made in the immediate area the site coordinate equals that of the point.

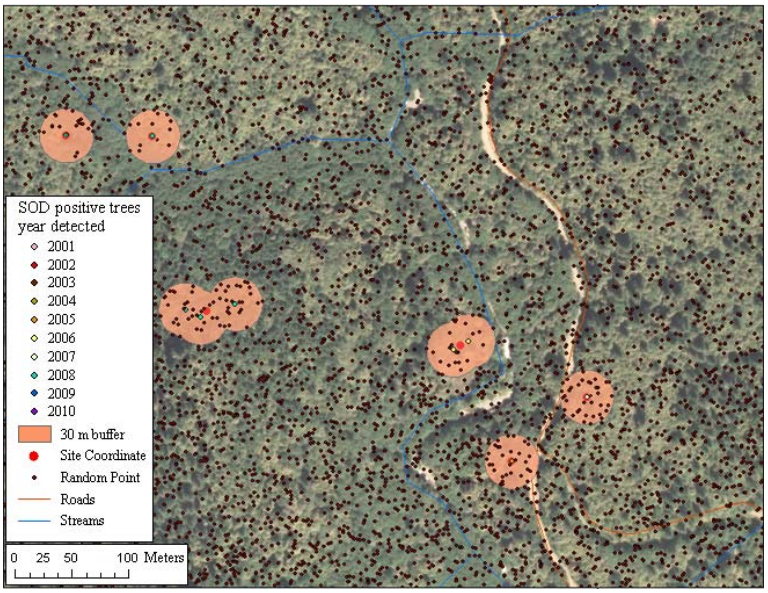


Fig. B.2. Example of the distribution of random points created to assess spatial independence to roads and streams, as produced with the 'Create Random Points' tool in ArcMap. The distance to the nearest road and the nearest stream was calculated for each random point; this data was then collated in Excel and imported into MATLAB for the randomization test.

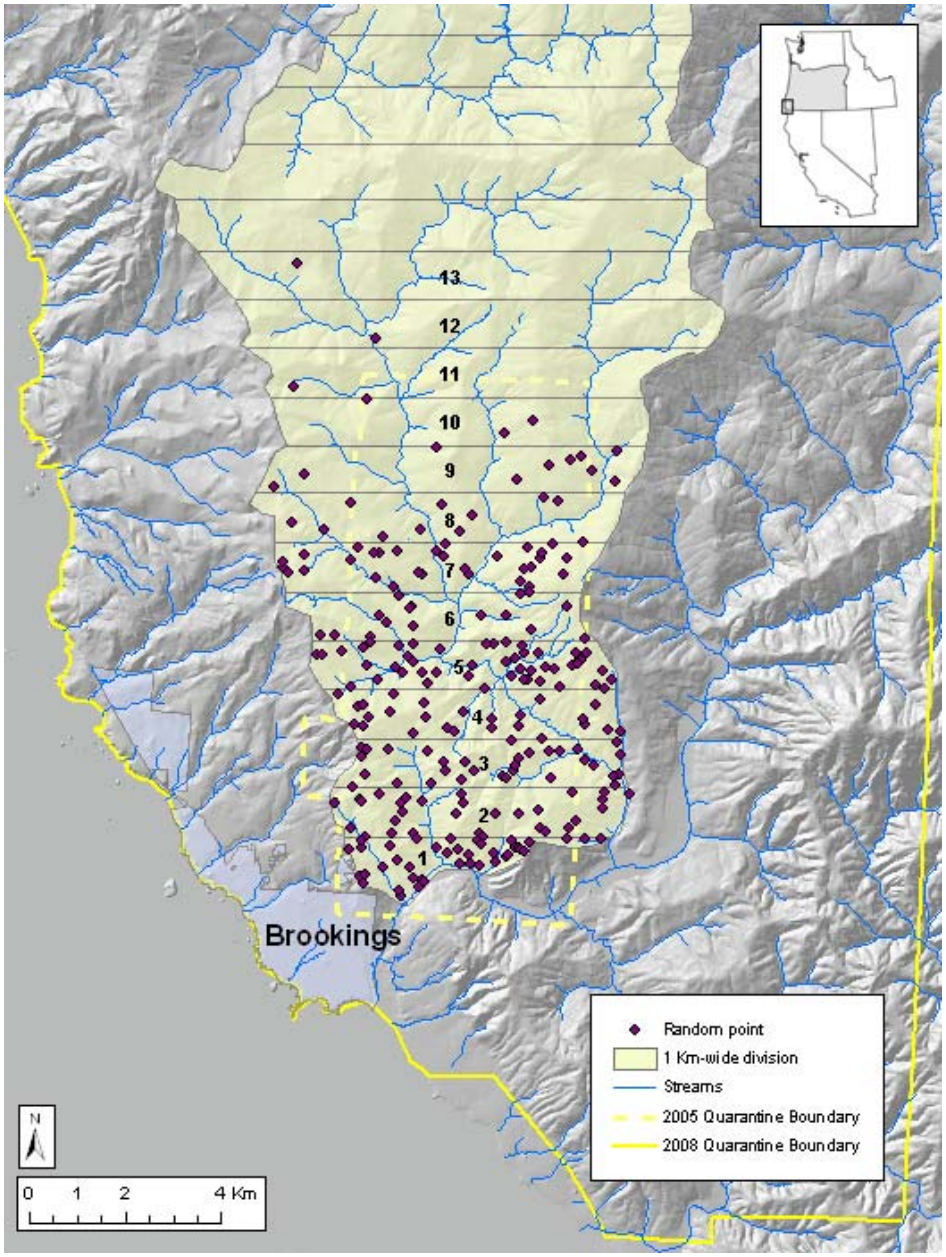


Fig. B.3. Regions used in randomization tests.
Example of the distribution of points selected for 1 of the 10,000 randomizations performed to assess spatial independence to roads; n = 294, median distance to road = 98.56 m. In order to retain the north to south distribution of observed *P. ramorum* infections, random points were created individually in each of the 13 1-km wide divisions of the NChetco study area, where the number of points generated was equal to the number of observed sites in that division times 5 000. Points were then selected randomly from each division. For example, 10 *P. ramorum* sites were identified between 2001 and 2010 in division #9; 10 of the 50,000 random points created in the division #9 polygon were sampled per randomization. All points from each division were combined into a single dataset from which median distance to road was calculated.

Fig B.4. MATLAB script used to assess spatial independence to roads and streams. The following MATLAB script was used to select random points and test the null hypothesis that SOD positive sites are located no more closely related to roads than would be expected by chance. All text proceeded by the '%' symbol are meant to explain each line of code.

```
%Define inputs
ColumnNum = [1 2 3 4 5 6 7 8 9 10 11 12 13];      % 1 km wide division identification number;
                                                  % 1 = furthest south, 13 = furthest north (fig. 3.)
FociNum = [57 33 36 30 47 19 34 17 12 4 3 1 1];   % number of SOD positive sites in each division
DistRand;                                         % 'DistRand' is a matrix of numbers with 13
                                                    % columns, each column containing
                                                    % 5,000*FociNum(x) for x = 1 to 13 values.
                                                    % Each value in this matrix is the distance to
                                                    % the  nearest road for a random point in (m).
                                                    % Column 1 corresponds to division 1, etc.
                                                    % Distances were generated in ArcMap,
                                                    % compiled in Excel, then imported into
                                                    %MATLAB.
RepNum = 10000;                                   % total number of randomizations to perform
PolyNum = 13;                                     % total number of divisions
MedianRoad = 100.14;                              % true median distance to roads in meters.



%Algorithm to select and store median distances to roads for each randomization
MedianHolder = [];                          % start of the algorithm
for rep = 1:RepNum;                         % for randomization 1 through 10000
  RandHold = [];                            % create a vector 'RandHold' for each randomization
where:
      for t = 1:PolyNum;                    % t equals a vector of numbers from 1 to 13,
          n = FociNum(t);                   % n equals the # of SOD positive sites in the t^th division,
          b = n*5000;                       % b equals the # of random points in the t^th division,
          r = randperm(b);                  % and r equals a vector of all numbers between 1 and b in
                                              % random order; e.g. r = [34  5  17…b^th value].
          rand = r(1:n);                    % rand is the first through the n^th numbers in vector r;
                                              % e.g. rand = [34  5  17... n^th number of r]
          Holder = [];                      % reset Hold for each division of each randomization
          for tt = 1:n;                     % tt is a vector from 1 to n
            posit = rand(tt);               % posit equals the tt^th value of the vector 'rand';
                                              % e.g. for tt = 1, posit = rand(1) = [34]
            distance = DistRand(posit,t);   % select the value at the 'posit' row in the 't' column of
                                              % DistRand, the distance to road for the random point in
                                              % the t^th division
            Holder = [Holder, distance];    % sequentially adds the value 'distance' in the vector
                                              % 'Holder.'  At the end of the active algorithm 'Holder'
                                              % includes all distances for all random points in the one
           end;                             % division being modeled.
          RandHold = [RandHold, Holder];    % a list of the distances for 1 complete randomization
      end;
  MedianHolder = [MedianHolder, median(RandHold)];      % consecutively holds the media
end;                                                      %distance to roads for all random
end;                                                      % points in each randomization
                                                          %  Size(MedianHolder) = 10,000
```

Fig. B.4. contintued

```
%Algorithm to calculate the statistical likelihood of spatial independence
NumLess = [];                          % start of the algorithm
for t = 1:RepNum;                      % t equals a vector from 1 to the # of randomizations
   if MedianHolder(t) <= MedianRoad;   % if the t^th value of MedianHolder is equal to or less than
       num = 1;                        %  the true median distance to roads then num = 1.
   else;                               % if it is greater that the true median then num = 0.
       num = 0;
   end;
   NumLess = [NumLess, num];           % repeat for all values of MedianHolder and save as vector
end;                                   %  'NumLess'; those randomizations that were assigned
                                       %  the value '1' were closer to roads than the observed
                                       %  data set.


%Called output
mean(MedianHolder)                     % average median distance to roads for all randomizations
hist(MedianHolder)                     % generate a histogram of median distances to roads
                                       %  expected  under the null hypothesis of spatial
                                       %  independence
k = sum(NumLess)                       % total number of randomizations for which
                                       %MedianHolder(t)  is less than or equal to (closer than)
                                       %observed.
p = k / RepNum                         % 1 sided p-value = k/N
```

Appendix C. Culture identification of *Phytophthora* spp.

P. ramorum

P. ramorum is one of the few *Phytophthora* species isolated in Oregon that may be reliably identified to species based on morphological traits alone (Reeser et al. 2011). This species has characteristic hyphae, and produces pigmented chlamydospores and clusters of ellipsoid, semi-papillate sporangia in abundance 1-2 weeks after plating (Werres et al. 2001). As only one mating type is found in Oregon forests, oogonia were neither expected nor observed (Prospero et al. 2007, 2009).

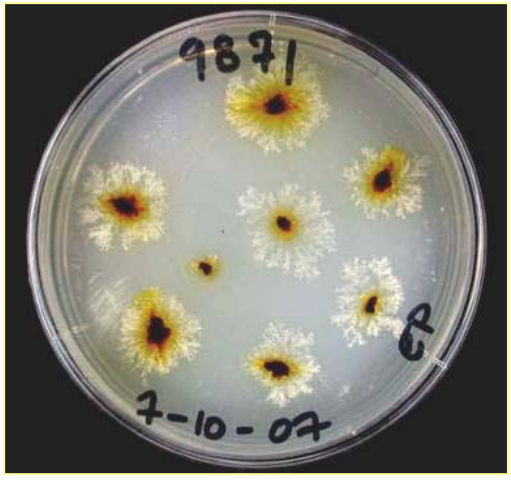


Fig. C.1. Culture morphology of *P. ramorum* on CARP. Isolated from tanoak bark.

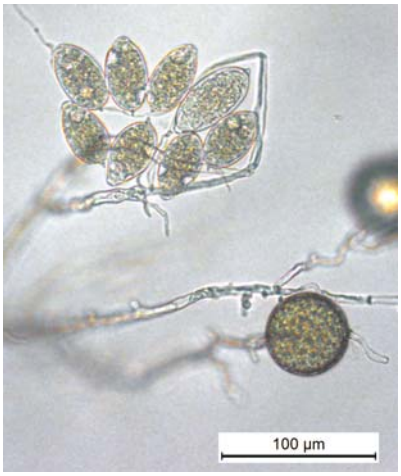


Fig. C.2. Spore structures of *P. ramorum* Sporangia with short pedicels and chlamydospore of *P. ramorum*.

Fig. C.3. Characteristically irregular, forked hyphae that pinches at hyphal junctions and chlamydospore of *P. ramorum*.

P. nemorosa

The appearance of amphigynous antheridia and oogonia, strong right angles at hyphael junctions, and round hyphael swellings were used as indicators of ***P. nemorosa***. Occasionally semi-papillate sporangia were observed. This species is easily confused with a new species that until recently has wrongly been identified as ***P. nemorosa*** (P. Reeser, personal com.). These two species are difficult to distinguish in culture, but under circumstances where hyphael swellings were much more developed and the culture had only a few oospores at 2 weeks the culture was classified as an 'other' ***Phytophthora***.

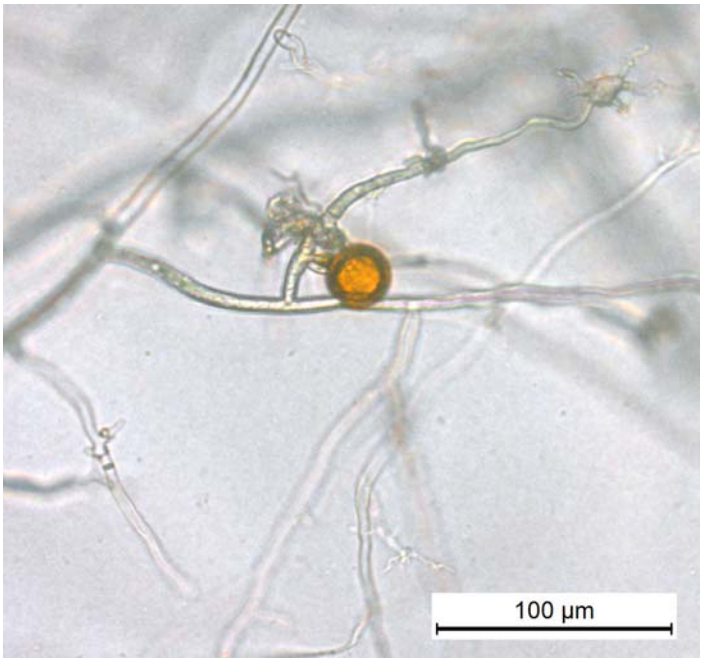


Fig. C.4. Hyphae with characteristic right angles and pigmented oospore of ***P. nemorosa***.

Fig. C.5. Detail of hyphal swelling and amphigynous antheridia and oogonia of ***P. nemorosa***.

Other *Phytophthora* species

Any *Phytophthora* species with lacking hyphae and spore characteristic of ***P. ramorum*** or ***P. nemorosa*** describe above were classified as 'other.' Below are some examples of hyphae or spores or some of these cultures for example.

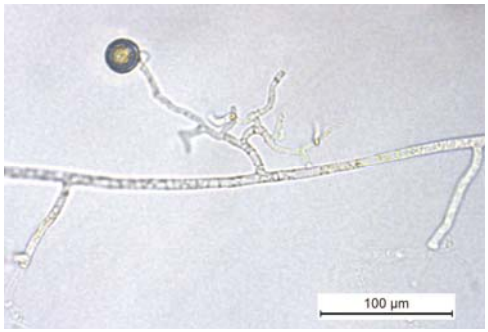


Fig. C.6. Isolated tanoak foliage as part of the stream surveys.

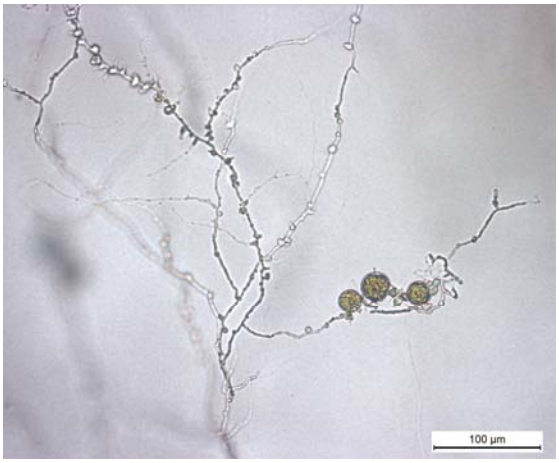


Fig. C.7. Isolated from tanoak foliage as part of the stream surveys.

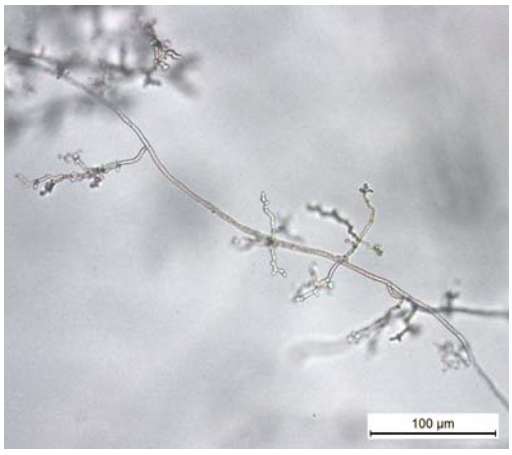


Fig. C.8. Isolated from tanoak foliage as part of the stream surveys.

Appendix D. Additional maps of stream and overstory surveys

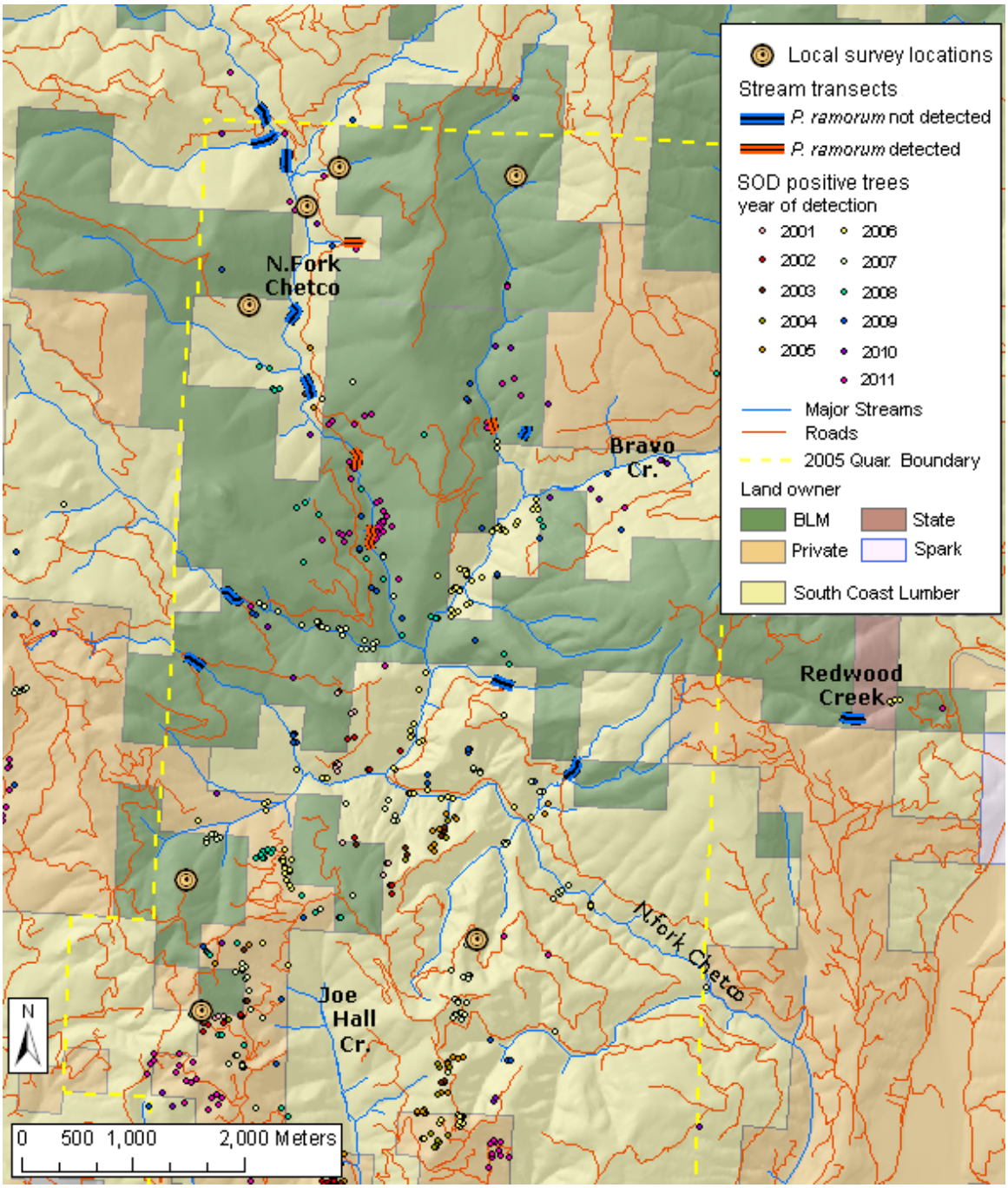


Fig. D.1. Map of locations of stream and local surveys. Streams were sampled between 26 July 2011 to 6 September 2011, indicating *P. ramorum* recovery and landownership (n = 15). Local surveys around *P. ramorum* positive trees were performed over in 2007, 2008, and 2011 (n = 7).

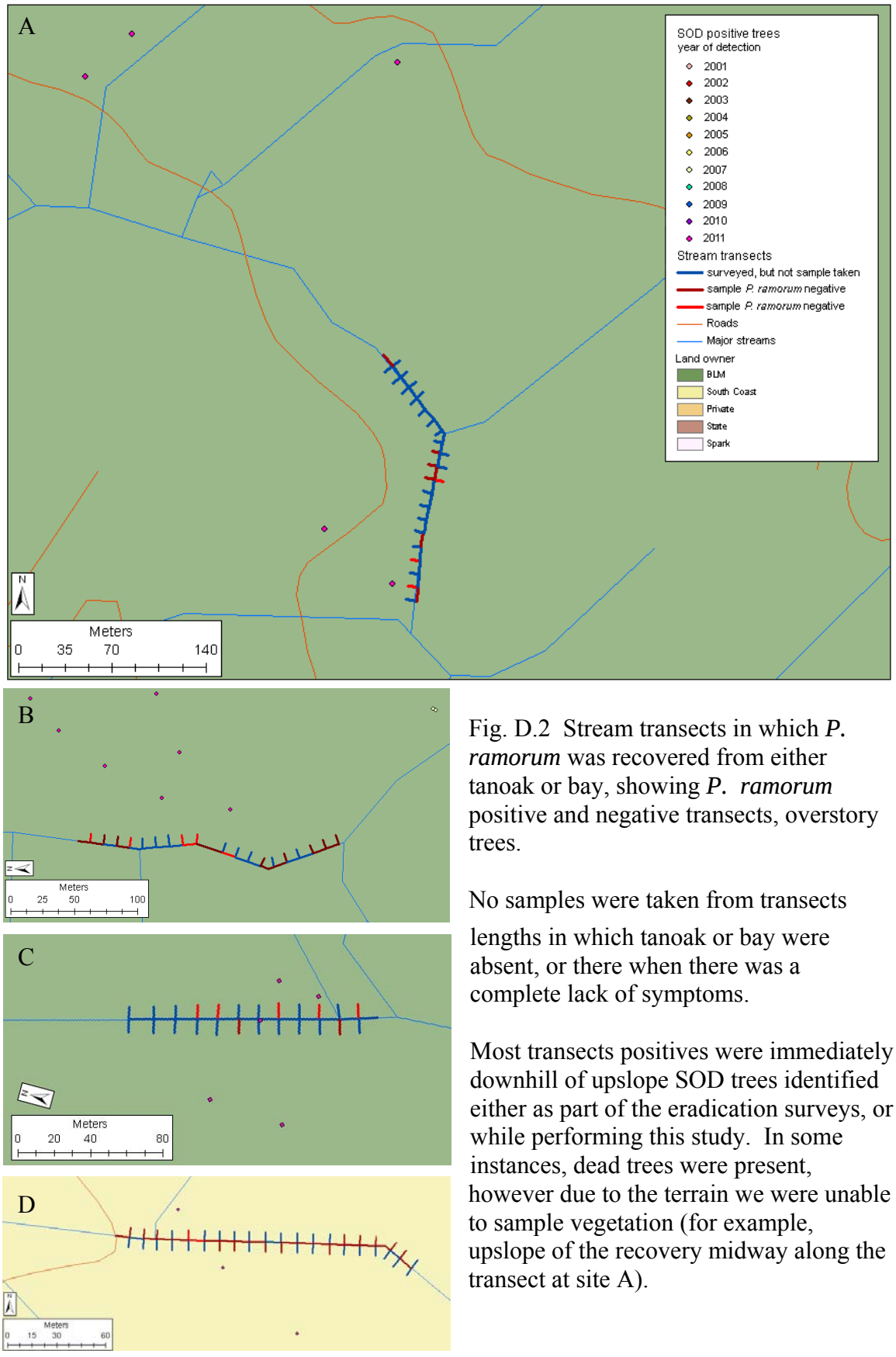


Fig. D.2 Stream transects in which *P. ramorum* was recovered from either tanoak or bay, showing *P. ramorum* positive and negative transects, overstory trees.

No samples were taken from transects lengths in which tanoak or bay were absent, or there when there was a complete lack of symptoms.

Most transects positives were immediately downhill of upslope SOD trees identified either as part of the eradication surveys, or while performing this study. In some instances, dead trees were present, however due to the terrain we were unable to sample vegetation (for example, upslope of the recovery midway along the transect at site A).

Appendix E. Model selection pathway to explain variation in maximum distance moved

Progression from full to reduced models built to explain variation in maximum distance moved each year of the epidemic between 2001 and 2010. Preliminary analysis eliminated summer weather conditions as potential explanatory variables of maximum distance. The initial full model was built using all weather variables for winter, spring, and autumn of one and two years before detection with r coefficients greater than 0.2; variables were sequentially eliminated and the resultant model reevaluated. The eliminated variable in each model is indicated in bold and by a (*).

Any models with AIC values within 2 units of the lowest calculated were considered as statistically indistinguishable from one another. We preferred the simplest model with the best explanatory power. The final model is re-listed with fitted residual and normal quantile plots.

Table E.1 Summary of all assessed models used to describe maximum distance moved

model #	model	p-value	Adj r^2	AIC
1	MaxDistance ~ -1+ SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpPrecipYB4 + WPrecipYB4 + WMaxTYB4 + WPrecip2YB4 + SpMaxT2YB4 + InfestYB4	0.0237	0.8496	217.438
2	MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SPrecipYB4 + WPrecipYB4 + WMaxTYB4 + SMaxT2YB4 + InfestYB4	0.0064	0.8765	215.783
3	MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpPrecipYB4 + WMaxTYB4 + SpMaxT2YB4 + InfestYB4	0.0015	0.8961	213.905
4	MaxDistance ~ -1+ SAvgPrecip2YB4+ WMaxT2YB4+ FMaxT2YB4+ SAvgPrecipYB4+ SMaxT2YB4+ InfestYB4	0.0005	0.8961	213.911
5	MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpMaxT2YB4 + InfestYB4	0.0002	0.89	214.382
6	MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + InfestYB4	<0.0001	0.8876	214.199
7	MaxDistance ~ -1 + SAvgPrecip2YB4	<0.0001	0.8249	217.698
8	MaxDistance ~ -1 + WMaxT2YB4	<0.0001	0.7353	223.069
9	MaxDistance ~ -1 + AuMaxT2YB4	0.0001	0.6988	224.75
10	MaxDistance ~ -1 + InfestYB4	0.0006	0.611	228.076
final preferred model:				
6	MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + InfestYB4	<0.0001	0.8876	214.199

1. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpPrecipYB4 + WPrecipYB4 + WMaxTYB4 + WPrecip2YB4 + SpMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	789.0012	393.0045	2.0076	0.1151
WMaxT2YB4	1265.3212	804.8285	1.5722	0.1910
AuMaxT2YB4	-2157.4924	1283.1209	-1.6814	0.1680
SpPrecipYB4	-507.9770	424.5020	-1.1966	0.2975
WPrecipYB4	47.5198	140.1051	0.3392	0.7515
WMaxTYB4	356.9466	524.8195	0.6801	0.5337
***WPrecip2YB4**	**55.0240**	**167.7055**	**0.3281**	**0.7593**
SpMaxT2YB4	594.3008	427.0059	1.3918	0.2364
InfestYB4	47.8852	33.2361	1.4408	0.2231

Residual standard error: 866.2 on 4 degrees of freedom
Multiple R-Squared: 0.9537 Adjusted R-squared: 0.8496
F-statistic: 9.16 on 9 and 4 degrees of freedom, the p-value is 0.02376

2. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SAvgPrecip2YB4 + WMaxT2YB4 + FMaxT2YB4 + SAvgPrecipYB4 + WAvgPrecipYB4 + WMaxTYB4 + SMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	775.3624	354.2143	2.1890	0.0802
WMaxT2YB4	1065.0206	475.3568	2.2405	0.0752
AuMaxT2YB4	-1877.9665	869.6416	-2.1595	0.0832
SpPrecipYB4	-427.7644	314.5463	-1.3599	0.2320
***WPrecipYB4**	**23.4541**	**108.1955**	**0.2168**	**0.8370**
WMaxTYB4	323.1785	466.4508	0.6928	0.5193
SpMaxT2YB4	557.4563	373.4070	1.4929	0.1957
InfestYB4	49.2314	29.8942	1.6469	0.1605

Residual standard error: 785.1 on 5 degrees of freedom
Multiple R-Squared: 0.9525 Adjusted R-squared: 0.8765
F-statistic: 12.53 on 8 and 5 degrees of freedom, the p-value is 0.006409

3. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpPrecipYB4 + WMaxTYB4 + SpMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	804.7879	300.0664	2.6820	0.0364
WMaxT2YB4	1104.1464	403.3244	2.7376	0.0338
AuMaxT2YB4	-1900.4993	791.8744	-2.4000	0.0533
SpPrecipYB4	-440.5754	283.3487	-1.5549	0.1710
***WMaxTYB4**	**372.8507**	**372.6275**	**1.0006**	**0.3557**
SpMaxT2YB4	524.7844	313.3340	1.6748	0.1450
InfestYB4	50.7452	26.6589	1.9035	0.1057

Residual standard error: 720.1 on 6 degrees of freedom
Multiple R-Squared: 0.952 Adjusted R-squared: 0.8961
F-statistic: 17.01 on 7 and 6 degrees of freedom, the p-value is 0.001454

4. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpPrecipYB4 + SpMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	549.4687	157.9057	3.4797	0.0103
WMaxT2YB4	881.6725	336.5331	2.6199	0.0344
AuMaxT2YB4	-1376.0945	593.6784	-2.3179	0.0536
***SpPrecipYB4**	**-230.9431**	**190.7869**	**-1.2105**	**0.2654**
SpMaxT2YB4	518.0137	313.2877	1.6535	0.1422
InfestYB4	53.1780	26.5500	2.0029	0.0852

Residual standard error: 720.2 on 7 degrees of freedom
Multiple R-Squared: 0.944 Adjusted R-squared: 0.8961
F-statistic: 19.68 on 6 and 7 degrees of freedom, the p-value is 0.0004696

5. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + SpMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	452.2233	139.8392	3.2339	0.0120
WMaxT2YB4	590.2135	241.8557	2.4404	0.0405
AuMaxT2YB4	-826.5161	393.4859	-2.1005	0.0689
***SpMaxT2YB4**	**235.8733**	**215.3381**	**1.0954**	**0.3052**
InfestYB4	56.4834	27.1663	2.0792	0.0712

Residual standard error: 740.8 on 8 degrees of freedom
Multiple R-Squared: 0.9323 Adjusted R-squared: 0.89
F-statistic: 22.04 on 5 and 8 degrees of freedom, the p-value is 0.0001742

6. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 + InfestYB4, data = LinReg.1YB4, na.action = na.exclude)

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	369.8170	119.1791	3.1030	0.0127
WMaxT2YB4	507.5988	232.3318	2.1848	0.0567
AuMaxT2YB4	-445.9054	186.6800	-2.3886	0.0407
InfestYB4	60.2034	27.2508	2.2092	0.0545

Residual standard error: 749 on 9 degrees of freedom
Multiple R-Squared: 0.9222 Adjusted R-squared: 0.8876
F-statistic: 26.66 on 4 and 9 degrees of freedom, the p-value is 0.00005273

7. *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4, data = LinReg.1YB4, na.action = na.exclude)
Residuals:

Min	1Q	Median	3Q	Max
-1878	-515.4	218.2	638.5	1187

Coefficients:

	Value	Std. Error	t value	Pr(>|t|)
SpPrecip2YB4	416.3147	52.7696	7.8893	0.0000

Residual standard error: 934.7 on 12 degrees of freedom
Multiple R-Squared: 0.8384 Adjusted R-squared: 0.8249
F-statistic: 62.24 on 1 and 12 degrees of freedom, the p-value is 4.335e-006

8. *** Linear Model ***

```
Call: lm(formula = MaxDistance ~ -1 + WMaxT2YB4, data = LinReg.1YB4, na.action
= na.exclude)
Residuals:
   Min    1Q Median    3Q  Max
 -1471 -1212  157.1 705.4 2021

Coefficients:
             Value Std. Error  t value Pr(>|t|)
WMaxT2YB4 167.7539  27.5351     6.0924   0.0001

Residual standard error: 1149 on 12 degrees of freedom
Multiple R-Squared: 0.7557      Adjusted R-squared: 0.7353
F-statistic: 37.12 on 1 and 12 degrees of freedom, the p-value is 0.00005398
```

9. *** Linear Model ***

```
Call: lm(formula = MaxDistance ~ -1 + AuMaxT2YB4, data = LinReg.1YB4, na.action
= na.exclude)
Residuals:
   Min    1Q Median    3Q  Max
 -1539 -1206  443.3 737.4 2294

Coefficients:
             Value Std. Error  t value Pr(>|t|)
AuMaxT2YB4 134.8199  24.1519     5.5822   0.0001

Residual standard error: 1226 on 12 degrees of freedom
Multiple R-Squared: 0.722      Adjusted R-squared: 0.6988
F-statistic: 31.16 on 1 and 12 degrees of freedom, the p-value is 0.0001195
```

10. *** Linear Model ***

```
Call: lm(formula = MaxDistance ~ -1 + InfestYB4, data = LinReg.1YB4, na.action
= na.exclude)
Residuals:
   Min     1Q Median   3Q  Max
 -1124 -716.8    125 1163 3340

Coefficients:
             Value Std. Error  t value Pr(>|t|)
InfestYB4 138.4455  29.9164     4.6277   0.0006

Residual standard error: 1393 on 12 degrees of freedom
Multiple R-Squared: 0.6409      Adjusted R-squared: 0.611
F-statistic: 21.42 on 1 and 12 degrees of freedom, the p-value is 0.0005824
```

Final model used to describe variation in maximum distance moved:

```
6.     *** Linear Model ***

Call: lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 +
InfestYB4, data = LinReg.1YB4, na.action = na.exclude)
Residuals:
    Min     1Q Median  3Q  Max
 -977.4 -510.7 -56.79 377 1307

Coefficients:
                  Value Std. Error   t value  Pr(>|t|)
  SpPrecip2YB4  369.8170  119.1791     3.1030    0.0127
     WMaxT2YB4  507.5988  232.3318     2.1848    0.0567
    AuMaxT2YB4 -445.9054  186.6800    -2.3886    0.0407
     InfestYB4   60.2034   27.2508     2.2092    0.0545

Residual standard error: 749 on 9 degrees of freedom
Multiple R-Squared: 0.9222      Adjusted R-squared: 0.8876
F-statistic: 26.66 on 4 and 9 degrees of freedom, the p-value is 0.00005273

Analysis of Variance Table
Terms added sequentially (first to last)
              Df Sum of Sq  Mean Sq  F Value     Pr(F)
  SpPrecip2YB4  1  54380816 54380816 96.94003 0.0000041
     WMaxT2YB4  1     92420    92420  0.16475 0.6943011
    AuMaxT2YB4  1   2605419  2605419  4.64446 0.0595404
     InfestYB4  1   2737957  2737957  4.88072 0.0545132
     Residuals  9   5048764   560974
```

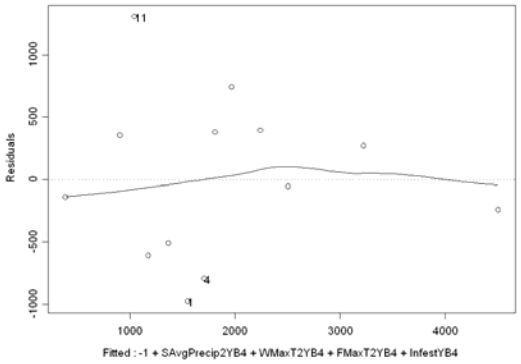


Figure E.1 Residual plot for final model of maximum distance.

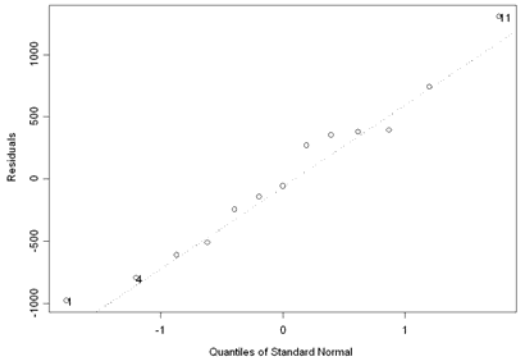


Figure E.2 Quantile plot for final model of maximum distance.

Table E.2 Model variables used to predict maximum distance moved observed in 2011.

model variable	study area	
	NChetco study area	Borax study area
SpPrecip2YB4	3.1	3.1
WMaxT2YB4	13.67	13.67
AuMaxT2YB4	14.47	14.47
InfestYB4	7.49	3.06

```
> MaxD.lm = lm(MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 +AuMaxT2YB4 +
InfestYB4)
> MaxD.lm

Call:
lm(formula = MaxDistance ~ -1 + SpPrecip2YB4 + WMaxT2YB4 + AuMaxT2YB4 +
InfestYB4)

Coefficients:
SpPrecip2YB4       WMaxT2YB4       AuMaxT2YB4       InfestYB4
   369.66           509.93          -447.73           60.19
>
> newdata = data.frame(SpPrecip2YB4 = 3.10 , WMaxT2YB4 = 13.67 ,
AuMaxT2YB4 = 14.47 , InfestYB4 = 7.49)
> newdata
  SpPrecip2YB4 WMaxT2YB4 AuMaxT2YB4 InfestYB4
1      3.1       13.67     14.47      7.49
>
>predict(MaxD.lm, newdata, interval="confidence")  #NChetco
       fit                lwr                upr
1     2088.861           835.7596           3341.963

>
>
> newdata= data.frame(SpPrecip2YB4 = 3.10 , WMaxT2YB4 = 13.67 ,
AuMaxT2YB4 = 14.47 , InfestYB4 = 3.06)
> newdata
  SpPrecip2YB4 WMaxT2YB4 AuMaxT2YB4 InfestYB4
1     3.1        13.67     14.47     3.06
>
> predict(MaxD.lm, newdata, interval="confidence") #Borax
             fit                lwr                upr
1            1822.237           494.5697           3149.903
>
```

Appendix F. Model selection pathway to explain variation in infestation size

Progression from full to reduced models built to explain variation in infestation size of each year of the epidemic between 2001 and 2010. Preliminary analysis eliminated summer weather conditions as potential explanatory variables of infestation size. The initial full model was built using all weather variables for winter, spring, and autumn of one and two years before detection with r coefficients greater than 0.2; variables were sequentially eliminated and the resultant model reevaluated. The eliminated variable in each model is indicated in bold and by a (*).

Any models with AIC values within 2 units of the lowest calculated were considered as statistically indistinguishable from one another. We preferred the simplest model with the best explanatory power. The final model is re-listed with fitted residual and normal quantile plots.

Table F.1 Summary of all assessed models used to describe infestation size

model #	model	p-value	Adj. r^2	AIC
1	InfestArea ~ -1 + AuPrecipYof + SpPrecipYof + WMaxTYB4 + SpPrecipYB4 + WMaxT2YB4 + SpPrecip2YB4 + InfestYB4	0.0068	0.8224	87.14
2	InfestArea ~ -1 + AuPrecipYof + WMaxTYB4 + SpPrecipYB4 + WMaxT2YB4 + SpPrecip2YB4 + InfestYB4	0.0017	0.8473	85.17
3	InfestArea ~ -1 + WMaxTYB4 + SpPrecipYB4 + WMaxT2YB4 + SpPrecip2YB4 + InfestYB4	0.0004	0.8634	83.47
4	InfestArea ~ -1 + WMaxTYB4 + SpPrecipYB4 + WMaxT2YB4 + InfestYB4	0.0001	0.8715	82.2
5	InfestArea ~ -1 + WMaxTYB4 + SpPrecipYB4 + InfestYB4	<0.0001	0.8624	82.46
6	InfestArea ~ -1 + SpPrecip2YB4 + SpPrecipYB4 + InfestYB4	0.0001	0.8489	83.67
7	InfestArea ~ -1 + SpPrecipYB4 + InfestYB4	<0.0001	0.8622	81.72
8	InfestArea ~ -1 + SpPrecip2YB4 + InfestYB4	<0.0001	0.8299	84.45
9	InfestArea ~ -1 + WMaxT2YB4 + InfestYB4	<0.0001	0.8318	84.31
10	InfestArea ~ -1 + SpPrecipYB4	0.0002	0.6806	91.77
11	InfestArea ~ -1 + SpPrecip2YB4	0.0005	0.6093	94.40
12	InfestArea ~ -1 + WMaxT2YB4	0.0002	0.6862	91.55
13	InfestArea ~ -1 + InfestYB4	<0.0001	0.8058	85.31
final preferred model:				
7	InfestArea ~ -1 + SpPrecipYB4 + InfestYB4	<0.0001	0.8622	81.72

```
1. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + AuPrecipYof + SpPrecipYof + WMaxTYB4 +
SpPrecipYB4 + WMaxT2YB4 + SpPrecip2YB4 + InfestYB4, data = LinReg.1YB4,
na.action = na.exclude)
Residuals:
    Min    1Q Median    3Q   Max
 -7.782 -1.07  1.004 1.832 5.237

Coefficients:
                Value Std. Error t value Pr(>|t|)
 FpPrecipYof   -0.3725  0.9656    -0.3857  0.7130
*SpPrecipYof    0.0937  0.6885     0.1361  0.8962
    WMaxTYB4   -1.7855  1.3527    -1.3199  0.2350
 SpPrecipYB4    1.7724  1.0427     1.6999  0.1401
   WMaxT2YB4    2.1687  2.2579     0.9605  0.3739
SpPrecip2YB4   -0.6226  1.1660    -0.5340  0.6125
   InfestYB4    0.6891  0.2036     3.3840  0.0148

Residual standard error: 5.494 on 6 degrees of freedom
Multiple R-Squared: 0.918      Adjusted R-squared: 0.8224
F-statistic: 9.6 on 7 and 6 degrees of freedom, the p-value is 0.006789



2. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + FAvgPrecipYof + WMaxTYB4 + SAvgPrecipYB4 +
WMaxT2YB4 + SAvgPrecip2YB4 + InfestYB4, data = LinReg.1YB4, na.action =
na.exclude)
Residuals:
    Min     1Q Median    3Q   Max
 -7.616 -1.236 0.9166 2.051 5.295

Coefficients:
                Value Std. Error t value Pr(>|t|)
  *AuPrecipYof -0.3520  0.8845    -0.3980  0.7025
     WMaxTYB4  -1.7044  1.1262    -1.5134  0.1739
   SpPrecipYB4  1.7228  0.9058     1.9020  0.0989
     WMaxT2YB4  2.1391  2.0839     1.0265  0.3388
  SpPrecip2YB4 -0.6696  1.0329    -0.6483  0.5375
     InfestYB4  0.6866  0.1881     3.6510  0.0082

Residual standard error: 5.094 on 7 degrees of freedom
Multiple R-Squared: 0.9178      Adjusted R-squared: 0.8473
F-statistic: 13.02 on 6 and 7 degrees of freedom, the p-value is 0.001729
```

```
3. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + WMaxTYB4 + SAvgPrecipYB4 + WMaxT2YB4 +
SAvgPrecip2YB4 + InfestYB4, data =
       LinReg.1YB4, na.action = na.exclude)
Residuals:
    Min     1Q Median    3Q   Max
 -7.649 -1.136   1.32 2.047 5.836

Coefficients:
                Value Std. Error t value Pr(>|t|)
       WMaxTYB4 -1.4371  0.8551    -1.6806  0.1313
    SpPrecipYB4  1.7174  0.8567     2.0046  0.0799
      WMaxT2YB4  1.4498  1.0962     1.3225  0.2225
  *SpPrecip2YB4 -0.6675  0.9770    -0.6832  0.5138
      InfestYB4  0.6977  0.1759     3.9665  0.0041

Residual standard error: 4.819 on 8 degrees of freedom
Multiple R-Squared: 0.9159      Adjusted R-squared: 0.8634
F-statistic: 17.43 on 5 and 8 degrees of freedom, the p-value is 0.0004064


4.     *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + WMaxTYB4 + SAvgPrecipYB4 + WMaxT2YB4 +
InfestYB4, data = LinReg.1YB4,
       na.action = na.exclude)
Residuals:
    Min     1Q Median    3Q  Max
 -8.199 -1.954 0.8372 1.845 7.41

Coefficients:
                Value Std. Error t value Pr(>|t|)
     WMaxTYB4 -1.0747  0.6505    -1.6520  0.1329
  SpPrecipYB4  1.6095  0.8167     1.9707  0.0803
   *WMaxT2YB4  0.8627  0.6601     1.3069  0.2237
    InfestYB4  0.6910  0.1703     4.0565  0.0029

Residual standard error: 4.674 on 9 degrees of freedom
Multiple R-Squared: 0.911      Adjusted R-squared: 0.8715
F-statistic: 23.03 on 4 and 9 degrees of freedom, the p-value is 0.00009541


5.     *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + WMaxTYB4 + SAvgPrecipYB4 + InfestYB4, data
= LinReg.1YB4, na.action =
       na.exclude)
Residuals:
    Min   1Q Median    3Q   Max
 -7.385 -3.2  1.347 3.509 6.139

Coefficients:
                Value Std. Error t value Pr(>|t|)
    *WMaxTYB4 -0.3393  0.3378    -1.0046  0.3388
  SpPrecipYB4  1.8247  0.8278     2.2043  0.0521
    InfestYB4  0.7172  0.1751     4.0969  0.0022

Residual standard error: 4.837 on 10 degrees of freedom
Multiple R-Squared: 0.8941      Adjusted R-squared: 0.8624
F-statistic: 28.15 on 3 and 10 degrees of freedom, the p-value is 0.00003439
```

```
6. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecip2YB4 + SAvgPrecipYB4 +
InfestYB4, data = LinReg.1YB4, na.action
       = na.exclude)
Residuals:
    Min     1Q Median    3Q   Max
 -6.917 -4.773  1.117 2.343 8.185

Coefficients:
               Value Std. Error t value Pr(>|t|)
   SpPrecip2YB4 0.1031 0.5976     0.1725  0.8665
    SpPrecipYB4 1.0563 0.6844     1.5434  0.1538
      InfestYB4 0.6261 0.1703     3.6772  0.0043

Residual standard error: 5.067 on 10 degrees of freedom
Multiple R-Squared: 0.8838      Adjusted R-squared: 0.8489
F-statistic: 25.35 on 3 and 10 degrees of freedom, the p-value is 0.00005454



7. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecipYB4 + InfestYB4, data =
LinReg.1YB4, na.action = na.exclude)
Residuals:
    Min     1Q Median    3Q  Max
 -7.189 -4.902  1.268 2.549 7.71

Coefficients:
              Value Std. Error t value Pr(>|t|)
  SpPrecipYB4 1.1386 0.4680     2.4330  0.0332
    InfestYB4 0.6350 0.1548     4.1011  0.0018

Residual standard error: 4.839 on 11 degrees of freedom
Multiple R-Squared: 0.8834      Adjusted R-squared: 0.8622
F-statistic: 41.68 on 2 and 11 degrees of freedom, the p-value is 7.347e-006



8.     *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecip2YB4 + InfestYB4, data =
LinReg.1YB4, na.action = na.exclude)
Residuals:
    Min     1Q   Median    3Q   Max
 -5.065 -3.818 -0.04009 1.563 14.17

Coefficients:
               Value Std. Error t value Pr(>|t|)
  SpPrecip2YB4 0.7468 0.4541     1.6447  0.1283
     InfestYB4 0.7031 0.1727     4.0708  0.0018

Residual standard error: 5.376 on 11 degrees of freedom
Multiple R-Squared: 0.8561      Adjusted R-squared: 0.8299
F-statistic: 32.72 on 2 and 11 degrees of freedom, the p-value is 0.00002341
```

```
9. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + WMaxT2YB4 + InfestYB4, data = LinReg.1YB4,
na.action = na.exclude)
Residuals:
    Min     1Q  Median     3Q   Max
 -6.275 -4.045 -0.6029 0.7754 12.02

Coefficients:
           Value Std. Error t value Pr(>|t|)
WMaxT2YB4 0.3642 0.2156     1.6895  0.1192
InfestYB4 0.6518 0.1932     3.3741  0.0062

Residual standard error: 5.347 on 11 degrees of freedom
Multiple R-Squared: 0.8577      Adjusted R-squared: 0.8318
F-statistic: 33.14 on 2 and 11 degrees of freedom, the p-value is 0.00002205



10.    *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecipYB4, data = LinReg.1YB4,
na.action = na.exclude)
Residuals:
    Min    1Q Median    3Q   Max
 -12.19 -4.88  2.273 4.468 12.08

Coefficients:
               Value Std. Error t value Pr(>|t|)
  SpPrecipYB4 2.5618 0.4781     5.3579  0.0002

Residual standard error: 7.367 on 12 degrees of freedom
Multiple R-Squared: 0.7052      Adjusted R-squared: 0.6806
F-statistic: 28.71 on 1 and 12 degrees of freedom, the p-value is 0.0001714



11.    *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecip2YB4, data = LinReg.1YB4,
na.action = na.exclude)
Residuals:
    Min     1Q Median   3Q   Max
 -15.01 -2.035 0.3106 6.82 17.26

Coefficients:
                Value Std. Error t value Pr(>|t|)
SAvgPrecip2YB4 2.1217 0.4601     4.6119  0.0006

Residual standard error: 8.149 on 12 degrees of freedom
Multiple R-Squared: 0.6393      Adjusted R-squared: 0.6093
F-statistic: 21.27 on 1 and 12 degrees of freedom, the p-value is 0.0005985
```

```
12.    *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + WMaxT2YB4, data = LinReg.1YB4, na.action =
na.exclude)
Residuals:
    Min     1Q Median    3Q   Max
 -11.13 -4.826 -2.122 4.314 13.14

Coefficients:
           Value Std. Error t value Pr(>|t|)
WMaxT2YB4 0.9492 0.1750     5.4245  0.0002

Residual standard error: 7.303 on 12 degrees of freedom
Multiple R-Squared: 0.7103      Adjusted R-squared: 0.6862
F-statistic: 29.43 on 1 and 12 degrees of freedom, the p-value is 0.0001539



13. *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + InfestYB4, data = LinReg.1YB4, na.action =
na.exclude)
Residuals:
    Min     1Q Median    3Q   Max
 -6.628 -3.323  1.598 3.896 14.05

Coefficients:
           Value Std. Error t value Pr(>|t|)
InfestYB4 0.9143 0.1234     7.4116  0.0000

Residual standard error: 5.745 on 12 degrees of freedom
Multiple R-Squared: 0.8207      Adjusted R-squared: 0.8058
F-statistic: 54.93 on 1 and 12 degrees of freedom, the p-value is 8.146e-006
```

Final model used to describe variation in infestation size:

```
7.     *** Linear Model ***

Call: lm(formula = InfestArea ~ -1 + SAvgPrecipYB4 + InfestYB4, data =
LinReg.1YB4, na.action = na.exclude)
Residuals:
    Min     1Q Median    3Q  Max
 -7.189 -4.902  1.268 2.549 7.71

Coefficients:
              Value Std. Error t value Pr(>|t|)
  SpPrecipYB4 1.1386 0.4680     2.4330  0.0332
    InfestYB4 0.6350 0.1548     4.1011  0.0018

Residual standard error: 4.839 on 11 degrees of freedom
Multiple R-Squared: 0.8834      Adjusted R-squared: 0.8622
F-statistic: 41.68 on 2 and 11 degrees of freedom, the p-value is 7.347e-006

Analysis of Variance Table

Response: InfestArea

Terms added sequentially (first to last)
              Df Sum of Sq  Mean Sq  F Value       Pr(F)
  SpPrecipYB4  1  1558.086 1558.086 66.55051 0.000005422
    InfestYB4  1   393.771  393.771 16.81912 0.001756075
    Residuals 11   257.533   23.412
```

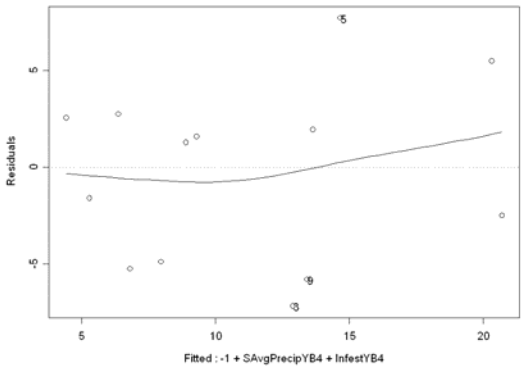


Figure F.1 Residual plot for final model of infestation size.

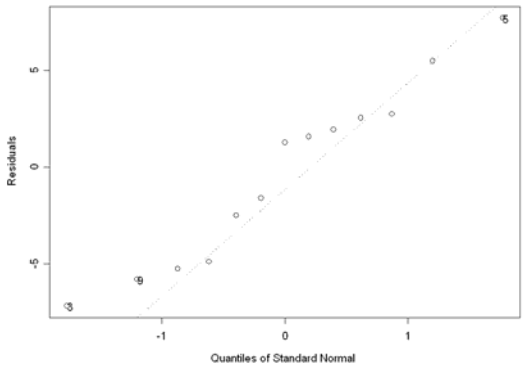


Figure F.2 Quantile plot for final model of infestation size.

Table F.2 Model variables used to predict infestation size observed in 2011.

model variable	study area	
	NChetco study area	Borax study area
InfestYB4	7.49	3.06
SpPrecipYB4	10.72	10.72

```
> LinReg <- read.csv(file="LinReg_1YB4.csv",sep=",",head=TRUE)
>attach(LinReg)
> LinReg
site year NumNewIsol NumNewSites InfestArea MeanDistance MaxDistance ….
1  Nchetco 2002        79         17    10.153      256.33     573.76    ….
2  Nchetco 2003        56         18    10.882      425.07    2712.72    ….

> infest.lm = lm(InfestArea ~ -1 + InfestYB4 + SpPrecipYB4)
> infest.lm

Call:
lm(formula = InfestArea ~ -1 + InfestYB4 + SpPrecipYB4)

Coefficients:
   InfestYB4  SpPrecipYB4
       0.635        1.139
>
> newdata = data.frame(InfestYB4 = 7.49, SpPrecipYB4 = 10.72)
> newdata
            InfestYB4          SpPrecipYB4
1           7.49                10.72
>
> predict(infest.lm, newdata, interval="confidence")  #NChetco
             fit                lwr                upr
1           16.96649           7.654958           26.27803
>
> newdata= data.frame(InfestYB4 = 3.06, SpPrecipYB4 = 10.72)
> newdata
            InfestYB4           SpPrecipYB4
1           3.06                10.72
> predict(infest.lm, newdata, interval="confidence")  #Borax
            fit                lwr                upr
1           14.15346           3.857484           24.44943
```

Appendix G. Yearly recovery of *P. ramorum* in collection buckets.

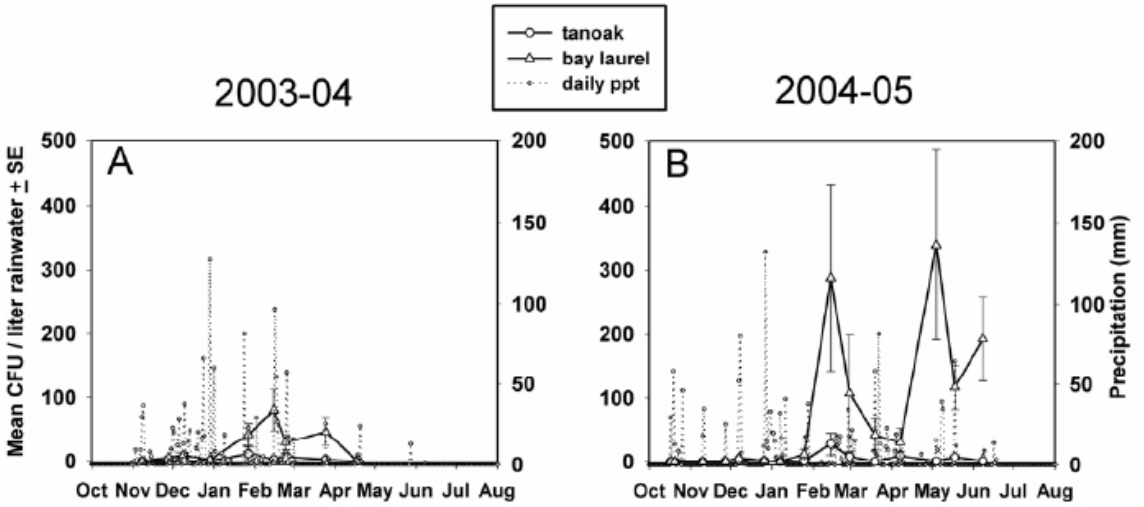
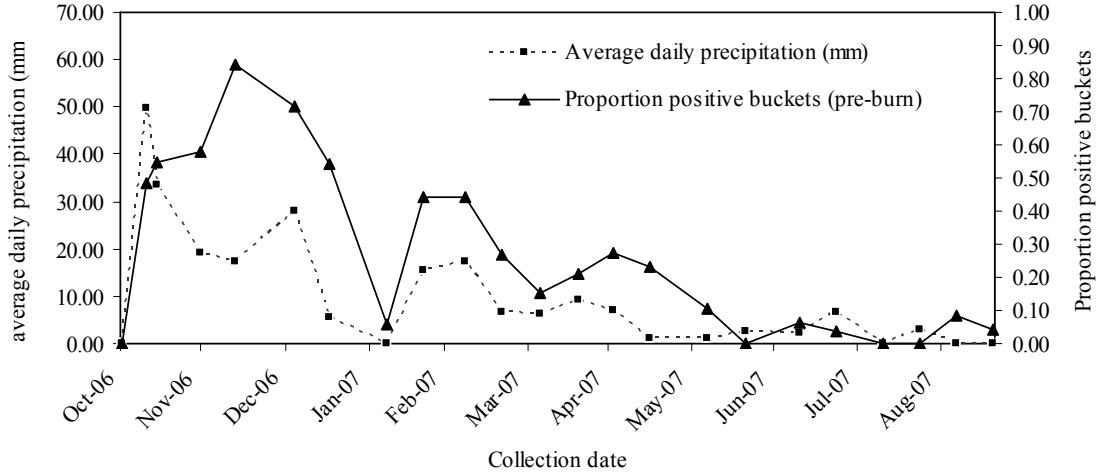
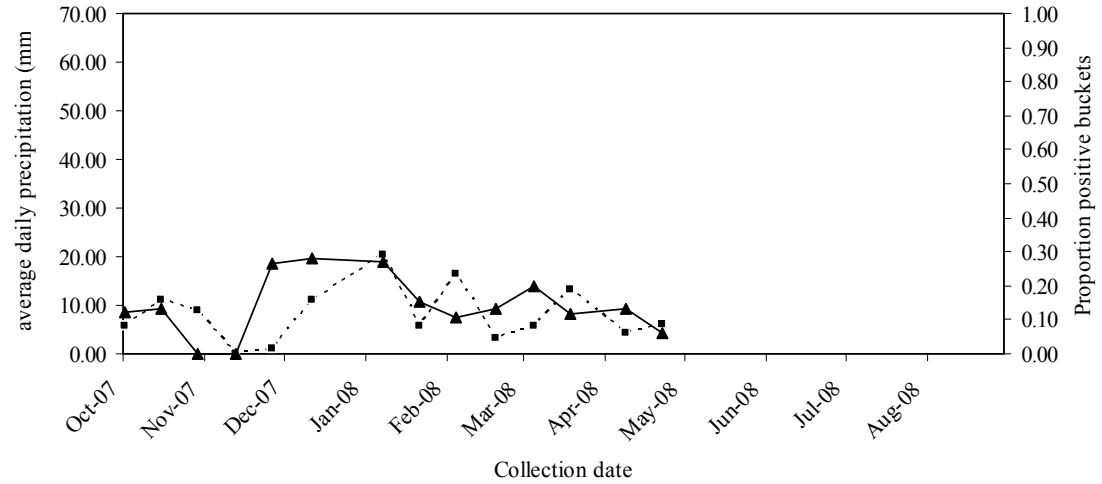


Fig. G.1. Recovery patterns of *P. ramorum* from rain water in California. Mean number of colony forming units (CFU)/liter rainwater standard error collected under bay laurel (△) or tanoak (○) during the seasons with low (2003 – 2004) and high (2004 – 2005) spring rainfall in a Californian tanoak-redwood-bay laurel forest. Rain was trapped during discrete rain events, then filtered and plated on selective media to quantify CFU. Reprinted from Davidson et al. (2005).

a. Oct. 2006 through Sept. 2007



b. Oct. 2007 through Sept. 2008



c. Oct. 2008 through Sept. 2009

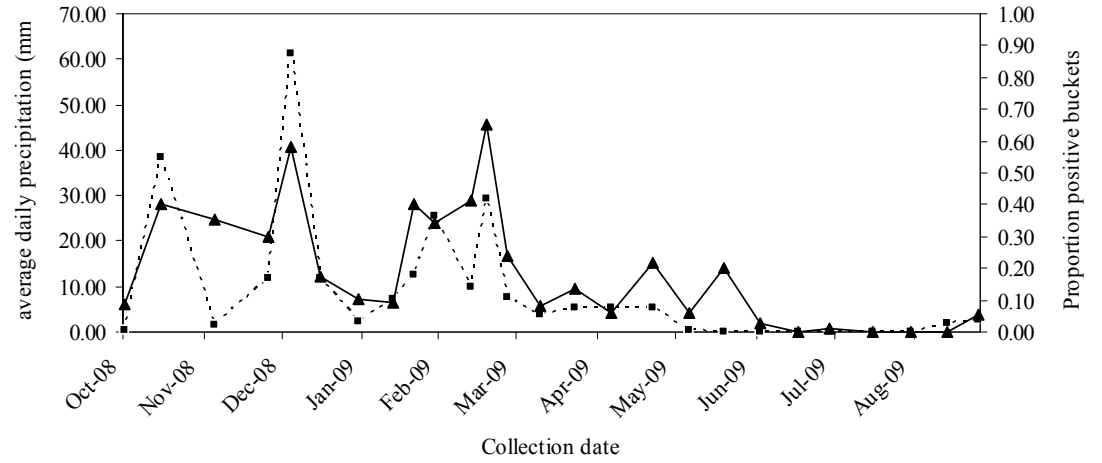
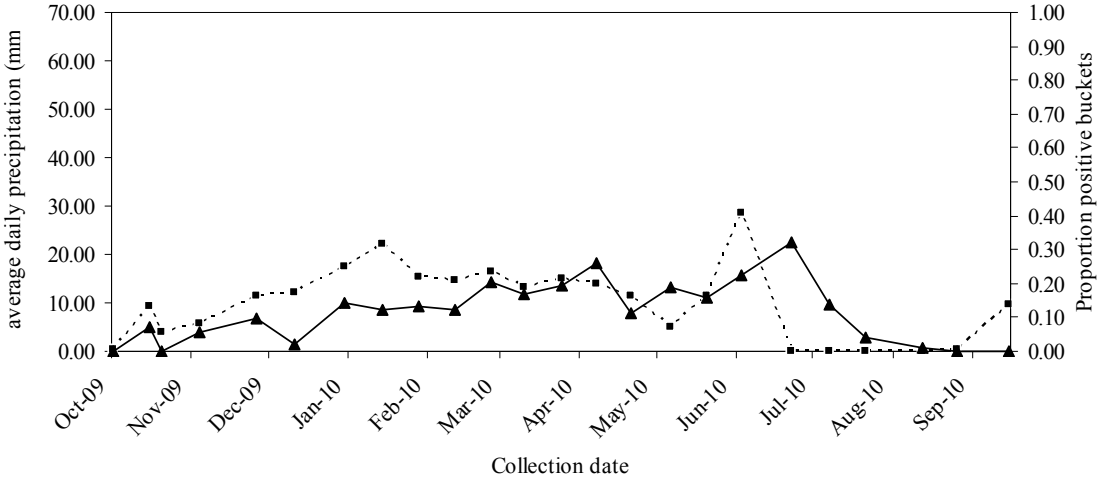


Fig. G.2. Recovery of inoculum in baited rain traps in between Oregon, 31 October 2006 to 23 April 2008, and 30 October 2008 to 30 September 2011. (Continued on following page).

(Continued)

d. Oct. 2009 through Sept. 2010



e. Oct. 2010 through Sept. 2011

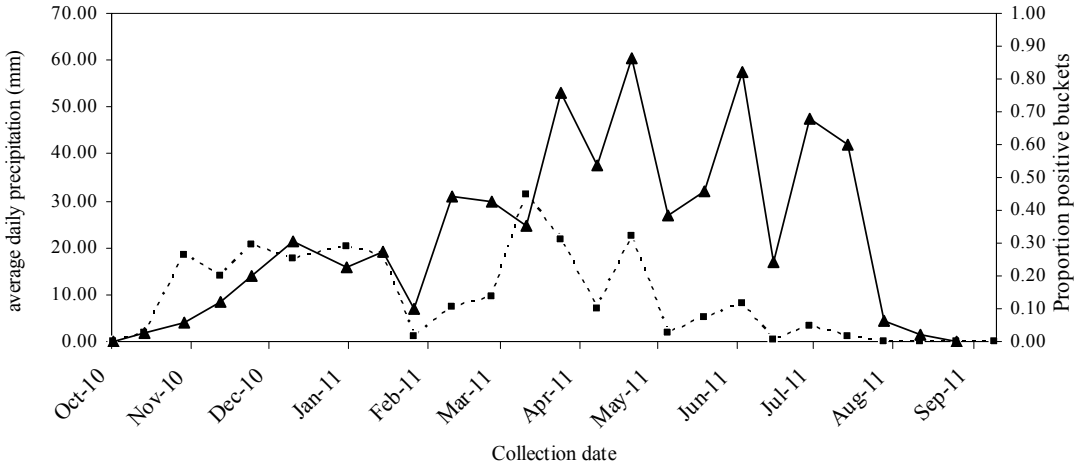


Fig. G.2. Recovery of inoculum in baited rain traps in between Oregon, 31 October 2006 to 23 April 2008, and 30 October 2008 to 30 September 2011. Precipitation is presented as average daily rain over the collection period. Proportion of positive buckets are displayed for bucket recovery at all sites that were untreated over the collection period, or for bucket recovery at all sites that were either untreated, or treated with herbicides but not yet burned. Note: not all collection dates had sites that were untreated.