

Stand Structure and Local Distribution of *Phytophthora ramorum* in Oregon Forests

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Introduction

The *Phytophthora ramorum* eradication program in effect in Oregon has allowed for the rapid detection of new infection foci, typically before they develop within each stand and expand into adjacent sites. Yet despite gallant efforts, new locations that previously harbored no apparent infection have been identified each year since the original detection in 2001. Multiple factors may influence why and where each new infection occurs including, but not limited to, stand characteristics such as dominant overstory vegetation, age and stem density (which influence microclimate variations within the stand) (Shaw, 2004), the distribution of host and non-host species, and the various possible dispersal mechanisms of the pathogen. Previous studies in California have found that landscape patterns of disease severity are poorly explained by variation at small scales (Condeso and Meentemeyer, 2007), however the disease intensity and stand composition differ greatly in Oregon. This study was performed first to examine stand characteristics and local distribution of *P. ramorum* around the first tree detected, if not the first tree infected, in a remote location. We further set out to document and characterize a site with infestation that has developed to an extent previously unseen in Oregon.

Site Characteristics

The two sites of this study, North Bean Creek (NBC) and site 2748, are located within a mile of each other on land privately owned by South Coast Lumber Company near Brookings, OR. Bordering fir plantation, both sites are composed of mixed Douglas fir (*Pseudotsuga menziesii*), alder (*Alnus rubra*), and tanoak (*Lithocarpus densiflorus*). The topography of the sites are similar, both uphill from the nearest road and occupying east facing slopes with similar pitches (35-45 degrees), elevations (approximately 1050-1200 feet above sea level), and locations relative to the top of their respective ridges. Both sites were first detected and confirmed positive for *P. ramorum* this current season, summer 2007.

Methods

To assess the distribution of understory vegetation likely to contribute to the spread of *P. ramorum*, notably *Rhododendron macrophyllum* and *Vaccinium ovatum*, a grid was established either around positive trees (site 2748; 30m x 40m) or through the area of worst infestation (NBC; 25m x 60m). For each 5m² block within the grid presence or absence of foliar hosts was noted (*R. macrophyllum*, *V. ovatum*, *L. densiflorus* sprouts, and *Umbellularia californica* only). Symptomatic tissue samples were taken from every grid and *P. ramorum* presence was confirmed through culture on agar. The stands were surveyed between July 25th and August 9th, 2007.

Stand composition and canopy structure was assessed by running a belt transect (10m x 50m for NBC and 10m x 40m for site 2748) nested within the vegetation plot. For all tree species, tree height and length of crown were measured, as well as distance traveled along the length and width of the transect. Distances were later corrected for changes in elevation for depiction. Disease severity on tanoak was rated by estimating crown condition (% browned to the nearest 10%), and recording presence or absence of symptoms associated with *P. ramorum* (bleeding, frass, symptomatic basal or stem sprouts).

Works cited:

Condeso, TE, Meentemeyer, RK. 2007. Effects of landscape heterogeneity on the emerging forest disease sudden oak death. *Journal of Ecology* 95:364-375
Davidson JM, Wickland AC, Patterson, H, Falk K, Rizzo DM. 2005. Transmission of *Phytophthora ramorum* in mixed-evergreen forests of California. *Phytopathology* 95:587-96
Shaw, DC. 2004. Vertical organization of canopy biota. In "Forest Canopies 2nd ed." (Lowman, M.D., Rinker, H.B., Eds.), pp. 73-101. Academic Press, San Diego.

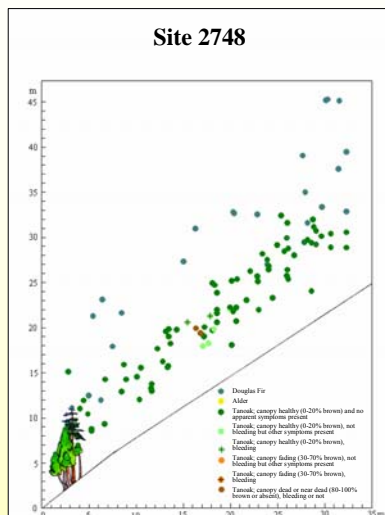


Figure 1a. Representation of the upper crown of individual trees (dbh>5cm). Scale represents vertical and horizontal distance traveled, corrected for change in elevation.

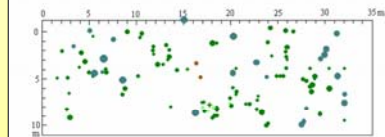


Figure 1b. Stem map, as from above, for each tree. Circle size represents relative dbh of each individual tree (not to scale).

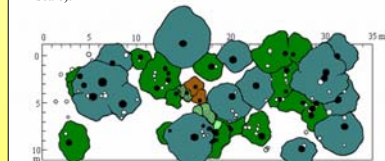


Figure 1c. Above crown projections for all trees either dominant or greater than 9m tall. (●) stems for which a canopy is drawn. (○) stems either understory or unmeasured. Canopies of stump sprouts were not distinguished individually but are represented by a single, continuous crown.

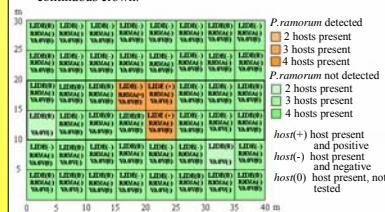


Figure 1d. Understory vegetation sample grid representing presence or absence of the four foliar hosts and *P. ramorum* in each 5m² block. LIDE = *L. densiflorus*, RHMA = *R. macrophyllum*, VAOV = *V. ovatum*, UMCA = *U. californica*. The canopy transect is represented in bold.

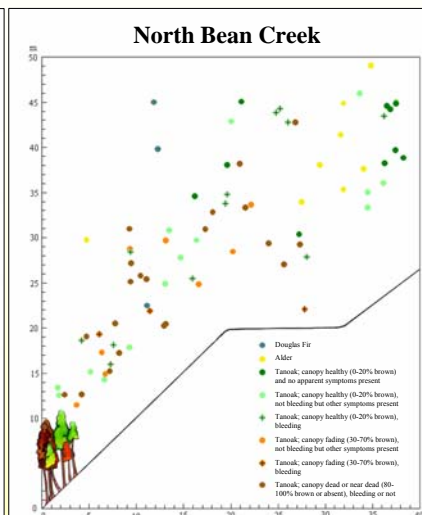


Figure 2a. Representation of the upper crown of individual trees (dbh>7cm). Scale represents vertical and horizontal distance traveled, corrected for change in elevation.

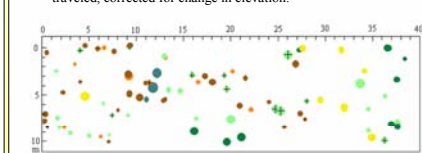


Figure 2b. Stem map, as from above, for each tree. Circle size represents relative dbh of each individual tree (not to scale).

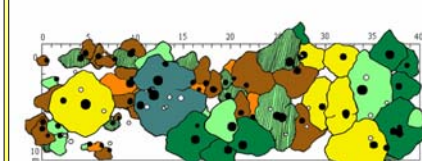


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Results

Stand composition and canopy structure

Stems were more dense at site 2748 though individual trees were much smaller, especially amongst tanoak (table 1). Tanoak comprised a greater proportion of the upper canopy and total basal area in NBC (table 1). In contrast, at site 2748 there was a marked stratification between the emergent Douglas firs, which were distributed throughout the transect, and the codominant tanoaks. While many stems were dominant, nearly all tanoak crowns were adjacent to taller Douglas fir crowns (fig. 1a); a notable exception is the few known positive trees (fig. 1c). Trees at various stages of disease were not distributed amongst different dbh sizes, tree heights, or crown volumes at either site (fig. 1a, fig. 2a.; data not shown).

Distribution of understory vegetation and infection

Both sites had a continuous understory of host vegetation (fig. 1d, fig. 2d). *P. ramorum* was recovered from leaf tissue in all but two 5m² blocks within NBC (68 samples taken; fig. 2d). Despite extensive sampling within the grid (86 samples taken) *P. ramorum* was only recovered within 5m of positive trees (fig. 1d). *U. californica* was present only at NBC, though only as single, small understory trees.

Table 1. Summary of stand structure at either site.

Species	No. live trees counted		Tree density (stems/ha)		Total basal area (m ² /ha)		Mean tree height (m)		Mean canopy volume (m ³)	
	Upper Bean Creek	site 2748	Upper Bean Creek	site 2748	Upper Bean Creek	site 2748	Upper Bean Creek	site 2748	Upper Bean Creek	site 2748
<i>Lithocarpus densiflorus</i>	76	91	1520	2275	35.6	16.7	13.8	7.6	84.0	31.0
<i>Alnus rubra</i>	8	0	160	0	7.1	0	20.5	0	211.0	0
<i>Pseudotsuga menziesii</i>	3	23	60	575	5.7	19.1	24.0	14.8	508.0	204.0
Total for all species	87	114	1740	2850	48.4	35.8				

¹ Bean Creek 500m² plot, all tree greater than 7cm DBH, site 2748 400m² plot, all trees greater than 5cm DBH
² For all trees at both sites either with emergent canopies or over 9m tall

Conclusions

Our results are consistent with previous observations that the development of disease in Oregon is dependant upon the infection of mature tanoak. Site 2748 is a typical location: highly restricted, local infection centered around isolated trees whose disease developed from inoculum produced in an unknown source. While other studies have focused upon movement of infested soil to explain the original introduction of inoculum into remote areas (Davidson *et al.* 2005), the location of this site is not consistent with movement by either people or vehicles. Other possibilities include movement by animals, either from below or above, or the aerial dispersal of inoculum. Further studies are needed to determine whether the observed pattern reflects the dispersal method of the pathogen or microclimate variation that favors initial establishment (or both).

It is likely not for the lack of host continuity or topographical differences that the severity of disease in either site differs so drastically, and which other factors best explain this remain unclear. Nevertheless, this study highlights the potential for the diseases caused by *P. ramorum* to reach epidemic proportions in Oregon.

Acknowledgments: Dr. David Shaw (Oregon State University), Alan Kanaskie (Oregon Department of Forestry), South Coast Lumber Company. Funding was provided by USDA Forest Service. Contact: petersoe@science.oregonstate.edu