

# Influence of Woodland Expansion (1942 to 2000) on the Establishment of *Phytophthora ramorum*<sup>1</sup>

Ross K. Meentemeyer,<sup>2</sup> Nathan E. Rank,<sup>3</sup> Brian L. Anacker,<sup>2,4</sup>  
David M. Rizzo,<sup>5</sup> and J. Hall Cushman<sup>3</sup>

## Abstract

Human land-use practices have resulted in dramatic alterations of forest ecosystems worldwide. By modifying transmission pathways and habitat structure, land use changes are being increasingly implicated in the emergence of infectious plant disease. In this research, we examined the effects of human-related land-cover change on the establishment of the invasive plant pathogen *Phytophthora ramorum*, causal agent of the forest disease sudden oak death. We hypothesized oak woodlands in coastal California have increased in density and expanded into grassland and shrubland areas over the last century due to fire suppression, leading to increased contagion of host vegetation and cooler forest microclimate conditions facilitating disease establishment.

To examine this hypothesis, we assessed forest structure, understory microclimate, and symptoms of infection on *P. ramorum* hosts over two years (2005 and 2006) in 102 15 x 15 m<sup>2</sup> plots within a 275 km<sup>2</sup> heterogeneous forest region of southeastern Sonoma county, California. Within a 150 m radius area around each plot, we mapped types of land cover (oak woodland, chaparral shrubland, grassland, vineyard, and development) in both 1942 and 2000 using detailed aerial photos. During this 58-year period, oak woodland host habitat significantly increased in area, while grassland and chaparral decreased. In addition, mean size of woodland patches substantially increased and the number of woodland patches decreased, leading to a marked decline in spatial heterogeneity of plant communities.

Woodland expansion was a significant predictor of disease severity, expressed as numbers of symptomatic stems and leaves of bay laurel (*Umbellularia californica*), the primary inoculum-producing host in mixed evergreen forests. Path analysis showed that woodland expansion resulted in larger forests with higher densities of the primary host trees (*U. californica*, *Quercus agrifolia*, *Q. kelloggii*) and cooler understory temperatures. Together, the positive effects of woodland size and host stem density and the negative effects of understory temperature explained 65 percent of the variation in the number of symptomatic bay laurel trees and 40 percent of the variation in the number of symptomatic leaves per plot. In conclusion, the enlargement of woodlands and closure of canopy gaps facilitated the

<sup>1</sup> A version of this paper was presented at the Sudden Oak Death Third Science Symposium, March 5–9, 2007, Santa Rosa, California.

<sup>2</sup> Department of Geography & Earth Sciences, University of North Carolina at Charlotte, NC 28223, USA.

<sup>3</sup> Department of Biology, Sonoma State University, Rohnert Park, CA 94928, USA.

<sup>4</sup> Department of Environmental Science and Policy, University of California, Davis, CA 95616, USA.

<sup>5</sup> Department of Plant Pathology, University of California, Davis, CA 95616, USA.

establishment of *P. ramorum* by increasing contagion of hosts and enhancing forest microclimate conditions. Epidemiological studies that incorporate land-use change are rare but may increase understanding of disease dynamics and improve our ability to manage invasive forest pathogens.

Key words: *Phytophthora ramorum*, invasive species, landscape pathology, woodland expansion, land-cover change.