

Temporal Fluctuations and the Role of Disturbance in Disease Progression of the Sudden Oak Death Epidemic¹

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Abstract

With its high host mortality and ability to cause landscape-scale alterations in forest cover and composition, sudden oak death (SOD) (etiological agent *Phytophthora ramorum*, Stramenopila, Oomycota) mirrors past forest disease epidemics such as Chestnut Blight and Dutch Elm Disease. In contrast with these past epidemics, however, the appearance of SOD converges with a time of significant advancement in the development of molecular genetic tools that allow the movement of individuals (or individual genotypes) to be tracked and the role of evolutionary processes in disease progression to be assessed. Such methods have been instrumental in reconstructing the likely origins and geographic pathways of spread of the pathogen. However, little is yet known about the local-scale processes contributing to disease maintenance and progression. Since 2008, we have surveyed a network of sampling plots in the San Francisco Bay Area multiple times per year in order to examine seasonal patterns in the incidence and population genetic structure of infection foci. A period of severe drought in 2007-2009, followed by a period of normal to above-normal precipitation in 2010-2011, provide a significant opportunity to examine the effects of abiotic disturbance on disease progression. Here, we present recent and ongoing research focused on assessing seasonal patterns in genotypic diversity of viable infections that can act as reservoirs of new infectious propagules, assessing the effect of drought as a potential agent of selection on pathogen genotypes, examining the relationship between infections on dead-end (*Quercus*) and amplifying (California bay laurel, *Umbellularia californica* (Hook. & Arn.) Nutt.) hosts, and assessing the possible role of competitive interactions with sympatric *Phytophthora* species (*P. pseudosyringae*, *P. nemorosa*) using culture-based surveys, culture-independent (qPCR) assays, and analyses of population genetic structure (based on variable microsatellite loci) to infer the underlying processes of pathogen demographic expansion, contraction, and spread.

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