Long-term Performance of Sudden Oak Death Management Treatments in Northern California Locations

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Sudden oak death (SOD), caused by *Phytophthora ramorum*, was first diagnosed as the cause of a lethal canker disease of coast live oak (*Quercus agrifolia*), California black oak (*Q. kelloggii*), and tanoak (*Notholithocarpus densiflorus*) in 2000. Between 2005 and 2009, we initiated a number of studies to test strategies for reducing SOD impacts in stands of tanoak and susceptible oaks. In oak forests, California bay (*Umbellularia californica*) is the primary source of *P. ramorum* inoculum that infects oaks. We evaluated the effectiveness of removing California bay, either on an area-wide basis or locally around individual oaks on the development and progress of SOD in coast live oak, Shreve oak (*Q. parvula* var. *shrevei*), and canyon live oak (*Q. chrysolepis*). Since these plots were initiated in 2008/2009, California bay removal treatments around susceptible oaks has proved to be very effective in preventing new infections in comparison to matched untreated controls.

Although the presence of California bay enhances disease development in tanoak stands, the development of *P. ramorum* inoculum on tanoak twigs and leaves requires different strategies for managing SOD in tanoak stands. The systemic chemical, potassium phosphite has been identified as a possible chemical treatment for control of SOD in susceptible oaks and tanoaks. Starting in 2005, we initiated a series of studies to determine if trunk spray applications of this chemical could prevent infection or suppress SOD development to a practical degree in tanoak stands. Stands selected for treatment were free of SOD but close to areas where *P. ramorum* was present, and California bay was not present in the treated or control plots. Annual applications of potassium phosphite initiated years before *P. ramorum* was detected in the study areas did not prevent treated trees from becoming infected with SOD. In treated stands that were invaded by *P. ramorum*, disease development and mortality were not suppressed by phosphite application. Even though *P. ramorum* had been detected in the vicinity of all study plots at the start of the studies, little or no progress of SOD into either treated or control plots has been detected in multiple locations. Drought conditions that persisted from 2012 through 2016 greatly slowed the advancement of *P. ramorum* in these extensive stands of susceptible tanoaks. Our data indicate that the invasion of tanoak stands by *P. ramorum*, even within areas considered to be infested, is subject to considerable variation over space and time and is strongly affected by rainfall. As a result, large differences in disease levels can develop between nearby plots due to chance alone that could incorrectly be attributed to treatment effects. Over short time horizons, tests showing no treatment effect are more readily interpreted than those that appear to show a treatment effect. A high-confidence protective treatment effect cannot be obtained without long-term observations that clearly show treated areas have resisted disease while all adjacent areas have succumbed.