Long-term Performance of Sudden Oak Death Management Treatments in Northern California Locations[1]

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Extended Abstract

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 multiple field studies to test strategies for reducing SOD impacts in stands of tanoak and susceptible oaks. In oak forests, California bay laurel (*Umbellularia californica*) is the primary source of *P. ramorum* inoculum that infects oaks, and proximity to bay greatly increases disease risk (Swiecki and Bernhardt 2008). 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*). Studies were conducted at four Midpeninsula Regional Open Space District preserves (OSP) on the San Francisco Peninsula (https://www.openspace.org/) where *P. ramorum* was present at the study start (fig. 1).

Area-wide removal was used in study areas where bay was present mostly as understory regeneration and small trees, and bay could be readily cleared from the entire plot area. Local bay removal was used to protect large high-value oaks where bay trees were large and abundant. The treatment objective was to eliminate or minimize bay canopy within 2.5 to 5 m of the oak trunk. The minimum horizontal clearance for each oak was measured from its trunk to a vertical plumb line that touched the edge of the nearest bay foliage. The plumb line was determined using a laser pointer attached to an angle gauge. In the Monte Bello OSP area-wide bay removal plot, minimum oak-bay clearance was 5.4 m, but almost all trees had at least 10 m of clearance. Area-wide removal at Rancho San Antonio OSP was conducted in smaller patches, leaving more edges close to bay; 10 of 42 monitored trees in the treated area had clearances less than 5 m. Among trees treated by local bay removal treatment at all locations, bay cover within 5 m of the trunk was always reduced, but 7 of 95 treated trees still had zero clearance to overstory bay canopy, and 7 other trees had clearances between 0.5 and 2.1 m. Remaining treated oaks had clearances of 2.8 m or more, with maximum clearances of 25 to 30 m. At two locations (fig.1, bottom), some oaks with suboptimal bay clearance were also treated with potassium phosphite

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applied by stem injection (15.3% a.i., Rancho San Antonio only) or by stem spray as described below for tanoaks.

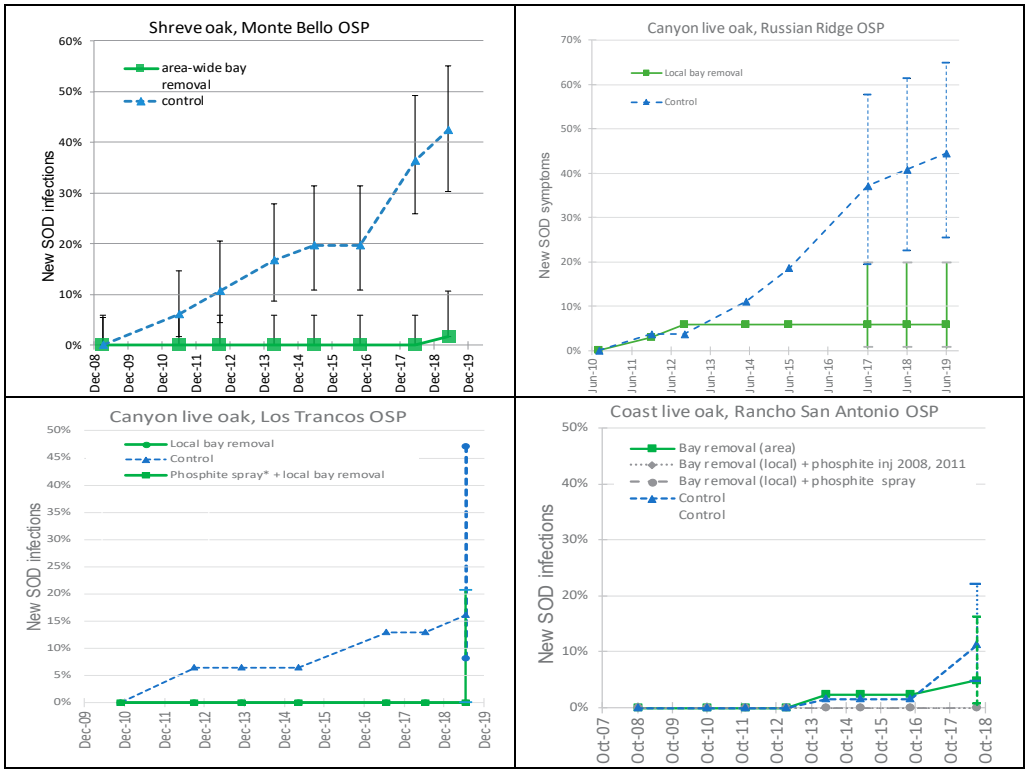

Figure 1—SOD disease progress in untreated control oaks and oaks treated by local or area-wide bay removal at four preserves. Note that scales vary between graphs. Error bars are exact binomial confidence limits. In two locations (bottom) some trees were also treated with phosphite through 2018.

California bay removal treatments around susceptible oaks was effective in preventing new infections compared to matched untreated controls (fig. 1, upper graphs). Early SOD symptoms in canyon live oaks are commonly lacking or very cryptic (Swiecki and others 2016); symptoms on this oak species at Russian Ridge by 2012 (fig. 1, top right) were likely from infections occurring before the start of the study in December 2009. Drought conditions from 2006-2010 and 2011-2016 suppressed disease progress, especially at Rancho San Antonio OSP, the warmest and driest location (fig. 1, bottom right). Disease incidence in control plots increased sharply in years following the wet winters of 2016-17 and 2018-19, whereas only two trees from treated areas developed symptoms over this period. One of these was in dense poison oak (*Toxicodendron diversilobum*), a *P. ramorum* host, and the other was at the base of a steep slope with bay canopy. Adding potassium phosphite treatments to oaks treated with bay removal (fig. 1, lower graphs) provided no additional benefit beyond bay removal alone.

Inoculum from California bay greatly enhances disease development in tanoak stands but *P. ramorum* sporulates readily on tanoak twigs and leaves. Hence, measures beyond bay removal are needed to manage SOD in tanoak stands. Stem application of the systemic chemical

potassium phosphite has been identified as a possible treatment for preventing SOD in susceptible oaks and tanoaks (Garbelotto and Schmidt 2007, 2009). 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. Phosphite application, which needs to be repeated indefinitely, would need to suppress SOD very substantially to justify the treatment cost. Minor suppression of SOD incidence over short time periods, even if statistically significant, is not likely to be of practical importance in managing affected stands.

At four locations in Sonoma County and two in San Mateo County, plots were established that were initially free of SOD but close to areas with tanoak mortality from *P. ramorum*. California bay was not present in treated or control plots. Trunk spray applications of aqueous potassium phosphite (22.36% a.i.) with Pentra-Bark® surfactant (2.3% v/v) were made at 6-month intervals the first year and annually thereafter. To optimize phosphite activity, spray volumes were scaled to tree diameter. Spray was banded on the trunk starting at a height of about 6 m and applied downward to favor absorption through the thinner bark and maximize potential for absorption as residues were remobilized by rain (Swiecki and Bernhardt 2017). Treating trees in contiguous blocks, especially large blocks, should also optimize disease suppression if phosphite translocated to the canopy suppresses sporulation. The Sonoma County plots (locations SF, BL, PC, and FE) were relatively small, mostly about 30-75 tanoak stems per plot (average area 0.063 ha). The San Mateo plots were much larger, with 233 treated and 243 control tanoaks (1.35 and 1.37 ha, respectively) at Skyline and 159 treated and 166 control tanoaks (0.35 ha for both) at El Corte de Madera OSP. At these locations, groups of trees monitored as controls were distributed around a single large treated plot.

Some of the phosphite treated trees at all locations, have developed SOD, even in plots where annual applications of potassium phosphite were initiated many years before *P. ramorum* was detected in the plots, indicating that the treatment did not completely prevent infection. In two study locations (Skyline and SF), phosphite-treated plots had significantly higher SOD incidence than the adjacent control plots, starting from the time that SOD was first detected in the plots. SOD incidence in the phosphite-treated plots was 32% at both Skyline and SF when phosphite treatments were terminated after 5 and 6 years of applications, respectively. The large Skyline plots were dominated by large-diameter tanoaks (average 45 cm DBH), whereas the SF plots had a mixture of large and small diameter trees (average 24 cm DBH).

Drought conditions that persisted from 2012 through 2016 inhibited the advance of *P. ramorum* into plots at the remaining study locations. SOD incidence at El Corte de Madera and FE plots was between 0 and 5% at their last evaluations in 2018. Following the wet winters of 2016-17 and 2018-19, an increase in SOD was observed at the BL and PC locations in 2019, which are dominated by smaller tanoaks (average DBH in treated plots 15 and 19 cm, respectively). The incidence of SOD in the control plots at BL and PC currently exceeds that seen in the phosphite-treated plots. However, because the initial invasion of tanoak stands by *P. ramorum* is very spotty on a local scale these early differences may not represent actual treatment effects.

In the SF and Skyline plots described above, SOD incidence was initially greater in phosphite-treated plots and remained so during the study observation intervals (6 and 4 years, respectively, from first observed trunk cankers). Because phosphite treatment is unlikely to increase SOD incidence, results from these plots illustrate that persistent differences in disease incidence can develop between closely spaced plots due to chance alone. In plots such as these, in which phosphite treatment clearly did not prevent extensive infection, the lack of a treatment effect can be interpreted with confidence over a relatively short time interval. However, because control plots may initially experience much higher levels of SOD than treated plots by chance alone, it is necessary to withhold judgement on potential positive treatment effects in tanoak stands until enough time has elapsed for disease levels to even out within the stand. To provide proof of an actual treatment effect, treated plots need to maintain very low disease levels after multiple years that are favorable for infection while disease levels in adjacent untreated areas continue to increase.

The observed lack of phosphite efficacy may be related to insufficient phosphite accumulation in target tissues (phloem of lower bole) or inadequate uptake. Stem diameter could be a factor influencing phosphite uptake in tanoaks treated with trunk spray applications. Although we scaled the applied phosphite dose by tree diameter and applied phosphite as high as feasible on the stems, the thicker layer of dead outer bark that occurs on larger trees may inhibit phosphite absorption. Phosphite might provide some protection to small diameter tanoaks that have little or no dead outer bark, though this has not yet been demonstrated in our plots. If SOD is partially suppressed in small tanoak, land managers would need to determine if any reduction in mortality of small tanoaks justifies the recurring expense of treatment, especially if the treatment is likely to become ineffective as the trees increase in size.

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