



## Management of *Phytophthora ramorum* in tanoak and oak stands

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### Summary

In this study we are evaluating two methods for controlling sudden oak death (SOD): potassium phosphite bark spray application on tanoak and removal of adjacent California bay for coast live oak and California black oak. Of two tanoak study sites that have been treated for 8 years, one location showed no effect of potassium phosphite and the other location has too little disease to date to draw a conclusion. Plots at two other locations have been treated for 6 years but disease levels have remained low and no conclusions can yet be drawn regarding efficacy. We hope to continue annual potassium phosphite applications until definitive results can be obtained at the three locations where disease levels are currently low.

Six years after plot establishment, disease levels have also remained relatively low in the oak plots, so absolute differences in disease incidence between treated and untreated trees are small. In the first year after bay removal, an initial increase in SOD incidence due to undetected latent infections was seen especially among coast live oaks. Since that time, SOD incidence has not changed among trees treated by bay removal. However, oaks without bay removal have shown a slow increase in SOD incidence. Weather conditions have been relatively unfavorable for SOD development in oaks since the plots were established in 2007 and were quite unfavorable in 2013 due to the dry spring conditions. The next evaluation of the oak plots could be deferred until after the next spring that has favorable conditions for *Phytophthora ramorum* infections.

### Introduction

The objective of this project is to test methods for managing *Phytophthora ramorum* canker (sudden oak death [SOD]) in tanoak and oak stands. We are testing different control strategies in tanoaks and susceptible oaks because disease epidemiology differs between these hosts. The plots we have established for this project are located in Napa, Solano, and Sonoma counties.

Because *P. ramorum* reproduces on tanoak, fungicide application is one of the few strategies that has the potential to protect tanoak stands. In this project, we are testing whether potassium phosphite, applied as a bark spray, can protect tanoak stands from *P. ramorum*. This material has been widely used to protect susceptible trees from SOD, even though this use is supported by very little field data. The goal of our study is to test phosphite efficacy in a variety of sites under conditions that are realistic from an implementation standpoint. Our application conditions are tightly calibrated and controlled, and have been optimized to provide a reliable test of efficacy. The design of phosphite-treated plots was conducted collaboratively with Matteo Garbelotto (UCB) and Yana Valachovic (UCCE Humboldt Co) and was intended to allow for comparisons between plots established by the three research teams.

For coast live oak and California black oak, we are testing whether localized removal of California bay can reduce the risk of *P. ramorum* infection to acceptably low levels. California bay is the primary source of *P. ramorum* spores that infect susceptible oaks in forests lacking tanoak. Disease epidemiology data from SOD-affected oak stands (Swiecki and Bernhardt 2007, 2008) indicates that *P. ramorum* infection risk and SOD severity increase as the minimum bay-oak clearance decreases. Oaks with bay foliage directly over or within 1.5 m of the trunk have the highest risk of infection. SOD risk and severity also increase as the total amount of bay cover within 2.5 to 5 m of the oak trunk increases. Our current study examines whether altering the bay neighborhood around individual trees can provide a significant reduction in SOD risk. Bay removal treatments in this study involve relatively minor amounts of bay clearing in the zone closest to the oak trunk, rather than area-wide bay removal.

## **1. Protection of tanoak stands using bark-band application of phosphite and understory thinning.**

### ***Methods***

#### **Plot locations**

The Phytosphere tanoak/phosphite plots are located in two geographic areas. All plots are in spatially grouped sets, with each set containing one phosphite treated plot and one or two paired control plots (Table 1). Three sets of plots are located in two rural subdivisions in northwestern Sonoma County (Seaview Ranch and Gualala Ranch), which are located about midway between Plantation (near Salt Point State Park) and Cazadero. The remaining two plot sets are on a property along Mill Creek, west of Healdsburg. To optimize treatment efficacy, understory tanoaks have been removed from treated plots. At locations SF and BL, separate controls with and without understory tanoak removal were included. At other locations understory tanoaks had been removed in both control and treated plots before the start of the study.

**Table 1.** Overview of tanoak phosphite-treated and control plots.

Study site	Locality	Plots	Phosphite applications	Notes
SF	Seaview Ranch, Creighton Ridge area	1 phosphite treated+thinned 1 thinned control 1 nonthinned control	Dec 2005 May 2006 May 2007 May 2008 May 2009 May 2010 Oct 2011	Plots initially established 2005 (Kashia Band of Pomo Indians cooperating). Plots no longer being treated due to the high amount of disease in the treated plot.
BL	Gualala Ranch Creighton Ridge area	1 phosphite treated+thinned 1 thinned control 1 nonthinned control	Dec 2005 May 2006 May 2007 May 2008 May 2009 June 2010 Oct -Nov 2011 Jan 2013	Plots initially established 2005 (Kashia Band of Pomo Indians cooperating).
PC	Gualala Ranch Creighton Ridge area	1 phosphite treated 1 control	Jan 2007 May 2007 May 2008 May 2009 May 2010 Nov 2011 Jan 2013	Understory tanoak mostly pre-thinned by landowner. Some minor additional thinning was conducted in treated and nontreated plots.
FE	Mill Creek Road, Healdsburg	2 phosphite treated 2 control	Feb 2007 May 2007 May 2008 May 2009 April 2010 Nov 2011 Jan 2013	Understory tanoak mostly pre-thinned by landowner. Some minor additional thinning was conducted in treated and nontreated plots

Plots at the SF and BL sites (Table 1) were established in cooperation with the Kashia Band of Pomo Indians of Stewarts Point Rancheria, and most of the activities associated with those locations was previously conducted under a separate contract with the Kashia, with funding provided by USDA-FS State and Private Forestry. When that contract ended, continuation of research activities associated with those plots was transferred to this project.

#### Treatment methods

Phosphite was applied using a 15 L (4 gal) ShurFlo Propack electric backpack sprayer mounted on a modified mountain bike. For all applications, we banded the spray high on the stem (up to 6 m height) using a long telescoping spray wand. Placement of the spray high on the bole was designed to maximize phosphite uptake. To minimize the variation in the phosphite dose applied to trees of different sizes, we calculated a specific diameter-related spray volume for each stem. Methods used to calculate the spray volume for each stem were as described in our December 2007 progress report. Application equipment

was calibrated to apply a known spray volume per unit time, and spray applications for each tree were timed to apply the desired volume.

The phosphite-treated plots at locations BL, FE, and PC were retreated in January 2013. All bark spray applications were within a few percent of the target dose based on audit of materials mixed and left over after spraying. At all three locations, we added a subtreatment for the 2013 application. We randomly selected half of the single-stemmed trees in the sprayed plots and applied 1 L of spray mixture to the base of the tree (about bottom 30 cm of the trunk and the root flare area).

### **Disease evaluations**

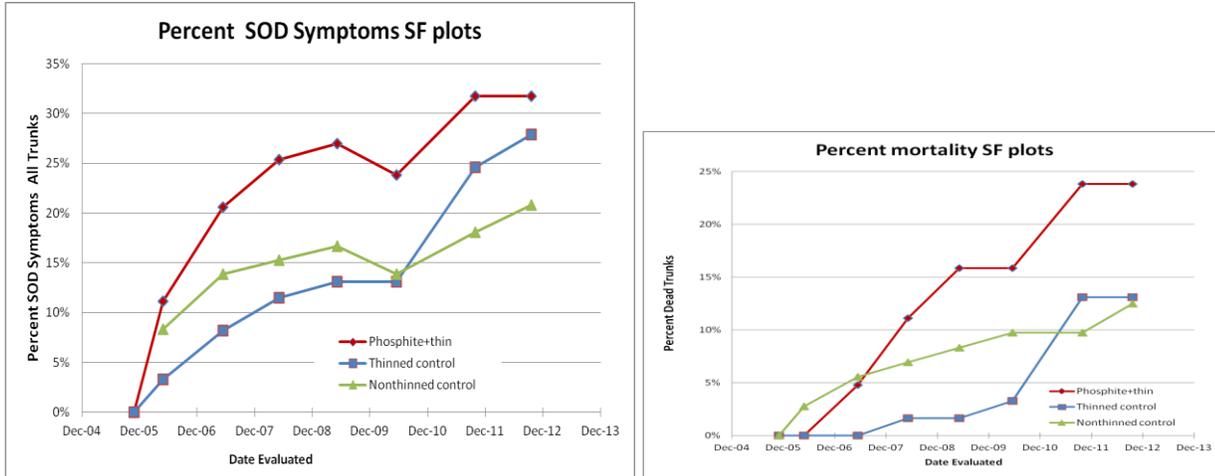
The 12 plots have 656 tanoak stems which have been individually evaluated for disease status and condition at least annually. Dead stems and stems that were nearly dead were omitted from the list of stems to be sprayed.

### **Results**

Monitoring disease development on tanoaks within the study plots is our primary method for determining the efficacy of the phosphite treatment. We assessed the disease status of each tanoak stem in the plots prior to the start of phosphite treatments and have annually reassessed the stems to detect evidence of disease.

We assessed tree health in all treated plots and most control plots trees in September 2012. The control plots at the BL location were re-evaluated in January 2013. Data for this plot is included in the 2012 data points in the graphs presented below.

In the SF plots (Figure 1), which have developed substantial levels of disease, it appears that phosphite has had no effect on SOD incidence. Total SOD incidence has been higher within the phosphite treated plot since disease was first seen in the plots in early 2006. The phosphite-treated plot was closest to the point where SOD first developed in the stand, which accounts for the early onset of disease in this plot. Over time, SOD levels in the control plots have begun to approach those seen in the treated plot. However, rates of disease increase over time in the control plots have been similar to or less than the rate of disease increase in the treated plot. Analyses of disease data from this location by stem diameter showed no effect of phosphite application on SOD incidence for any trunk diameter class.



**Figure 1.** Percent of trees with SOD symptoms (left graph), and percent mortality (right graph) in SF plots, Sonoma County. Phosphite treated plot=63 trunks, thinned control=61 trunks, nonthinned control=72 trunks.. Plots were initially treated in December 2005.

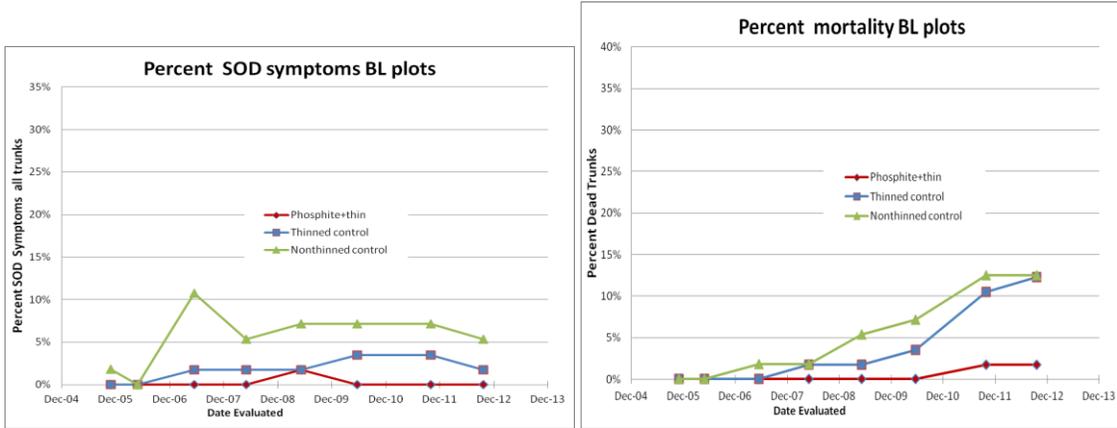
There can be a 1 to 2 year latent period between *P. ramorum* infection and canker symptom development in tanoak. Therefore, the initial increase in SOD seen in the phosphite-treated plot demonstrates that phosphite treatment was ineffective against established infections. However, increases in disease seen among phosphite treated trees from at least 2007 onward, especially the jump seen between 2010 and 2011, suggest that phosphite applications were also ineffective at preventing new infections and slowing the rate of mortality of infected trees.

As of the September 2012 evaluation, about 40% of the trees in the SF phosphite-treated plot had died due to SOD, were in late stages of SOD, or had been impacted by tree failures, and were therefore not suitable for further treatment. Furthermore, because most of the largest tanoaks in the plot have been killed by SOD or are in late disease stages, the amount of tanoak canopy cover in the plot has declined by more than 50%. Therefore, the rate of SOD development in the treated plot is likely to be influenced by its greatly reduced tanoak cover. Because effects of reduced tanoak cover are now confounded with any possible effect of phosphite, no additional phosphite treatments are planned at the SF location. However, we hope to continue observations on these plots for at least one more year to monitor further increases in disease in all plots.

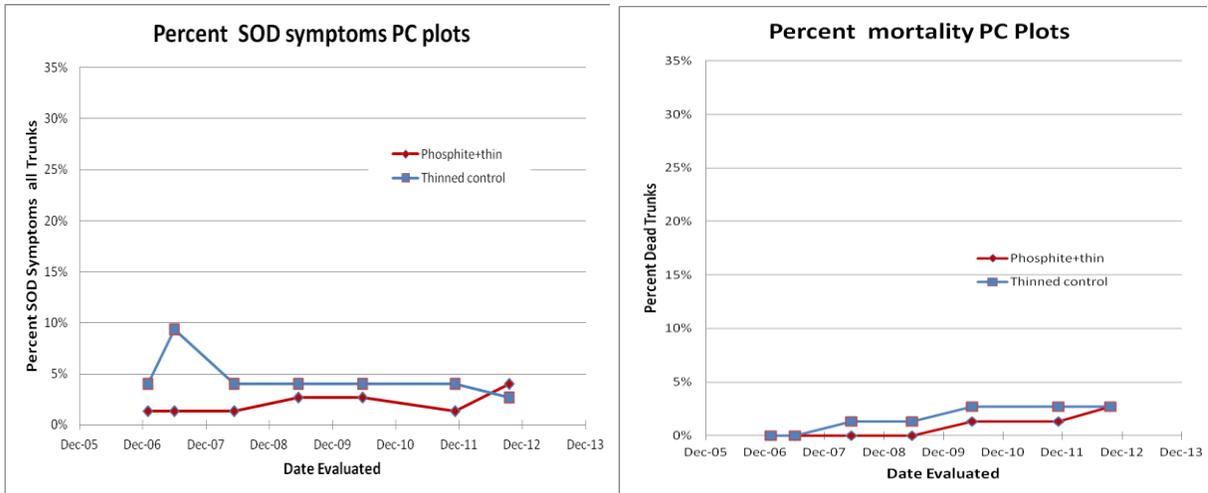
Currently, SOD levels in the plots at BL, FE, and PC are too low to draw any conclusions about phosphite efficacy to control SOD (Figures 2 - 4). High amounts of spatial variation are seen in SOD development within stands, especially when disease levels are low, so differences that seem to be related to treatments may be simply due to chance. For these three plot locations, further monitoring and annual potassium phosphite applications are needed until definitive results can be obtained.

Mortality rates also remain low overall in these three locations (Figures 2 - 4). In addition to SOD-caused mortality, we have observed tanoak mortality that is associated

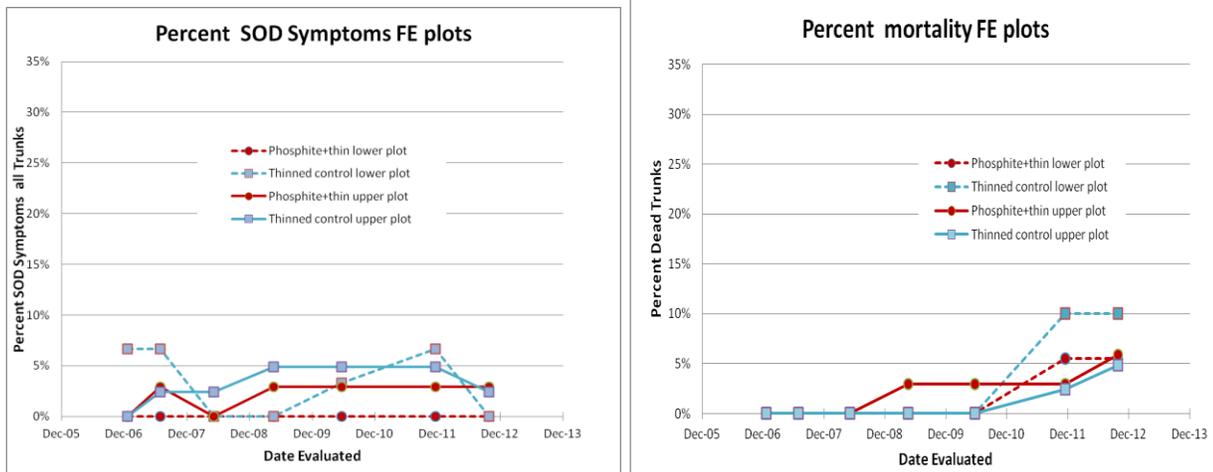
with decay fungi. This mortality is more common among smaller trunks of multitrunked trees



**Figure 2.** Percent of trees with SOD symptoms (left graph), and percent mortality (right graph) in BL plots, Sonoma County. Phosphite treated plot=58 trunks, thinned control =57 trunks, nonthinned control=56 trunks. Plots were initially treated in December 2005.



**Figure 3.** Percent of trees with SOD symptoms (left graph), and percent dead stems (right graph), in PC plots, Sonoma County. Phosphite treated plot=75 trunks, thinned control=75 trunks. Plots were initially treated in January 2007.



**Figure 4.** Percent of trees with SOD symptoms (left graph), and percent mortality (right graph) in FE plots, Sonoma County. FE1 is paired with FE2; FE3 is paired with FE4. Phosphite treated lower plot=36 trunks, thinned control lower plot=30 trunks, phosphite treated upper plot=34 trunks, thinned control upper plot=41 trunks. Sonoma County. Plots were initially treated in Jan/Feb 2007.

## 2. Protection of oaks using selective removal of California bay.

### Methods

In this portion of the project, we used a matched pairs design to study the effect of localized bay removal around SOD-susceptible oaks (coast live oak or California black oak). The oaks within the pairs were matched to the degree possible for known factors that influence disease risk, especially the amount of bay in the immediate vicinity of the trunk. One tree of each pair was designated as the control and was not altered in any way. For the other (treated) tree, we removed bay from the zone nearest to the trunk. We tried to achieve a minimum bay foliage-oak trunk clearance of 2.5 m. Where it could be achieved without excessive effort, we increased the minimum clearance up to about 5 m, especially in the direction of the prevailing storm winds (generally south and west of the tree). If present, poison oak climbing in the tree canopy was killed by cutting stems at ground level.

Bay foliage-oak clearance was defined as the minimum distance between vertical lines that were even with the edge of bay canopy and the closest surface of the lower oak trunk. We used a 500 mW green laser attached to an angle gauge to project the vertical line at the edge of bay canopy nearest to the oak trunk and used a laser rangefinder to measure the horizontal distance from this vertical line to the oak trunk. Bay foliage-oak trunk clearance was generally achieved by removing small-diameter bays close to the oak and/or bay branches from bays located farther from the oak. In some cases, very high bay canopy could not be reached using our pole pruner (above about 8 m) and the bay stems were too large to fell. In such cases, we removed as much of the lower, shaded bay canopy within the target clearance zone as possible. We have previously observed that

bay foliar symptoms of *P. ramorum* are generally much more common on lower foliage than it is on leaves at the top of the canopy.

In addition to measuring the minimum bay foliage-oak clearance, we also assessed the bay neighborhood around each oak. We scored bay canopy cover within 2.5 m of the oak trunk and within 5 m of the oak trunk using a 0-4 scale (0=no bay canopy, 1=1-25% cover, 2=26-50% cover, 3=51-75% cover, 4=more than 75% cover). We also recorded whether bay was present within 5-10 m and 10-20 m from the oak trunk. The presence of bay foliar symptoms was also noted for the zones from 0-2.5 m and from 2.5-5 m from the oak trunk.

Locations used in this study are in Sonoma, Napa, and Solano Counties. At the start of the study, *P. ramorum* was present at all of the study locations and was causing symptoms on bay and at least some oaks. The locations included in the study to date are summarized in Table 2.

**Table 2.** Number of bay removal study pairs at study locations.

Location	County	Coast live oak pairs	California black oak pairs	Initial bay removal date
Wall Rd.	Napa	7	--	Jan 2007
Annadel SP	Sonoma	7	6	Feb 2007
JT/GVR	Napa/Solano	11	--	March 2007
Jacobs Ranch	Sonoma	5	4	May 2007
SA	Sonoma	1	8	November 2007
<b>Total pairs</b>		31	18	

### Tree health evaluations

At the time that tree health was reassessed, both disease status and the cover of bay in the oak's local neighborhood were evaluated. For disease evaluations, we assessed whether *P. ramorum* canker symptoms were present. If cankers were present, other parameters were noted, including recent bleeding; degree of stem girdling by the *P. ramorum* canker, beetles, and *Annulohypoxylon thouarsianum*; and the location of cankers on the trunk (by compass points). Overall tree health ratings (canopy dieback, thinning, decay impact, non-SOD decline) were also scored.

California bay cover within the vicinity of the tree was reevaluated by measuring the minimum horizontal bay foliage-oak trunk clearance and direction, and estimating bay cover within the distance zones described above. Bay foliar symptoms were also recorded as described above.

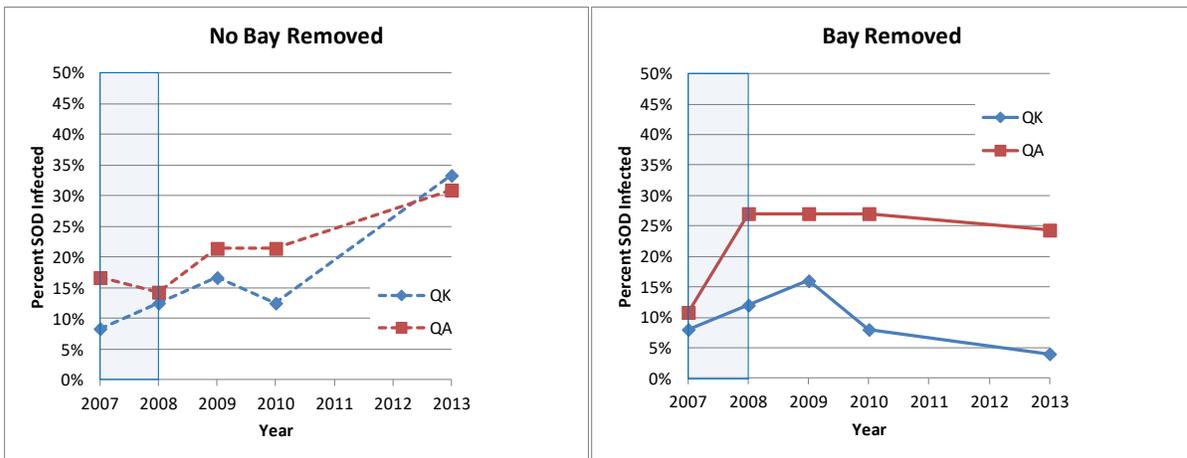
**California bay sprouts.** At each location, a number of bay stumps around treated oaks have been monitored for sprout regrowth. The stumps were relocated using distance and azimuth readings from tagged study trees. None of the stumps in this study have been treated with herbicides to suppress resprouting. For each stump, we recorded the height of the tallest shoot and the basal diameter of the thickest shoot. The presence of browsing

damage was also noted. Sprouts that had grown tall enough to constitute a potential source of inoculum were recut after measuring.

We evaluated study trees at Annadel State Park in October 2012. Assessment of other sites occurred between March 25 and April 10, 2013. In graphs below, the Annadel data is included with 2013 data.

### Results

All trees initially chosen for inclusion in this study were surrounded by high levels of bay and had little or no clearance from bay. Trees chosen for inclusion in the study in 2007 were free of SOD symptoms (88 trees) or had only early symptoms with small bleeding cankers (11 of the trees). The first post-treatment disease evaluation occurred in June 2008. Because the latent period for SOD in oaks is typically at least 1 to 2 years new symptoms observed in June 2008 most likely resulted from infection that occurred prior to treatment, probably during the wet years of 2005 and 2006. This is especially likely because 2007 was a very dry year with a dry spring which was not favorable for SOD infections. To account for the latent infections that existed at the start of the study, we have used SOD levels in 2008 as the pre-treatment baseline for this study. Overall results are shown in Figure 5.

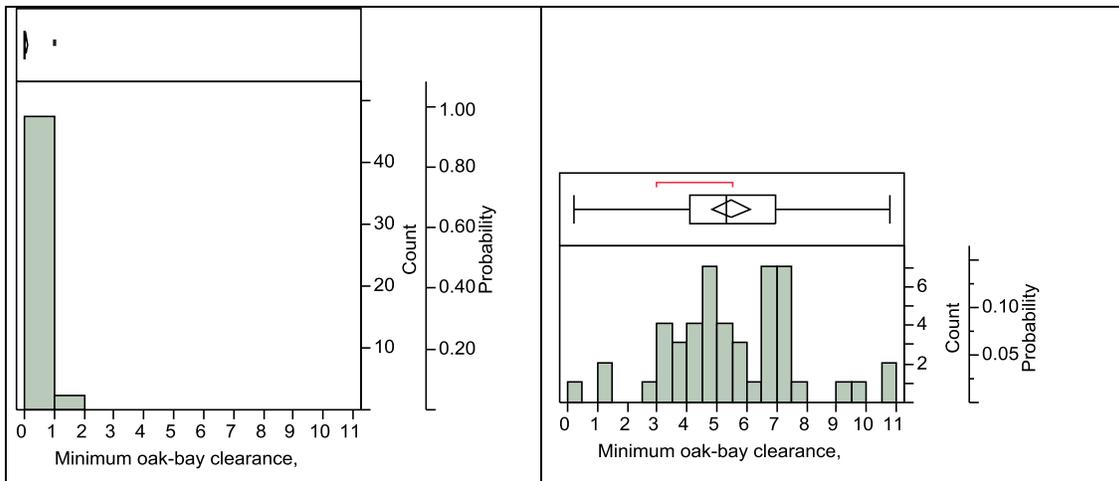


**Figure 5.** Change in SOD incidence among coast live oaks (QA) and California black oaks (QK), without (left) or with (right) nearby California bay removed. The disease baseline year for assessing treatments effects is 2008 (blue vertical line). Changes in disease status before 2008 (shaded area) are likely the result of latent infections that occurred before treatments were initiated.

The effect of bay removal treatments on SOD were similar for both coast live oak and California black oaks. The incidence of SOD infection has increased since 2008 among control oaks, especially those that had high amounts of California bay canopy over or near their trunks. Disease levels have remained constant, or have declined (due to disease remission) among oaks treated by removing California bay canopy over and near their trunks (figure 5). Although the overall difference in 2013 SOD incidence between coast live oaks in the bay removal and control group are small, the change in incidence between

2008 and 2013 shows a clear positive effect of bay removal. For California black oak, both 2013 SOD incidence and the 2008-2013 change in incidence show a clear positive effect of bay removal.

We did not visit the plots in 2011 or 2012. During the 2008, 2009 and 2010 evaluations, we cut additional bay branches or stems where practical to both maximize and stabilize oak-bay clearances. These adjustments appear to have helped. Among oaks with bay removal, the average loss in oak-bay clearance from 2010 to 2013 was 0.19 m, a small loss in clearance on average. Most oaks lost less than 0.5 m of clearance, and the greatest loss was 1.8 m of clearance. In contrast, average clearance losses were 0.33 m (2.8 m maximum) for 2008-2009 and 0.26 m (2.3 m maximum) for 2009-2010. The mean clearance for the bay removal treatment was 5.62 m in 2010 and 5.46 m in 2013. Half of the treated oaks have clearances of 5.3 m or more, whereas almost all control trees have no clearance from bays (Figure 6)



**Figure 6.** Minimum oak-bay clearances for oaks in the California bay removal (right) and control (left) groups at the 2013 evaluation. The target minimum clearance in the bay removal treatment was 2.5 meters. For a few treated oaks, final clearances were below the target due to high bay canopy that could not be removed; understory and lower canopy bay foliage near the oaks was removed.

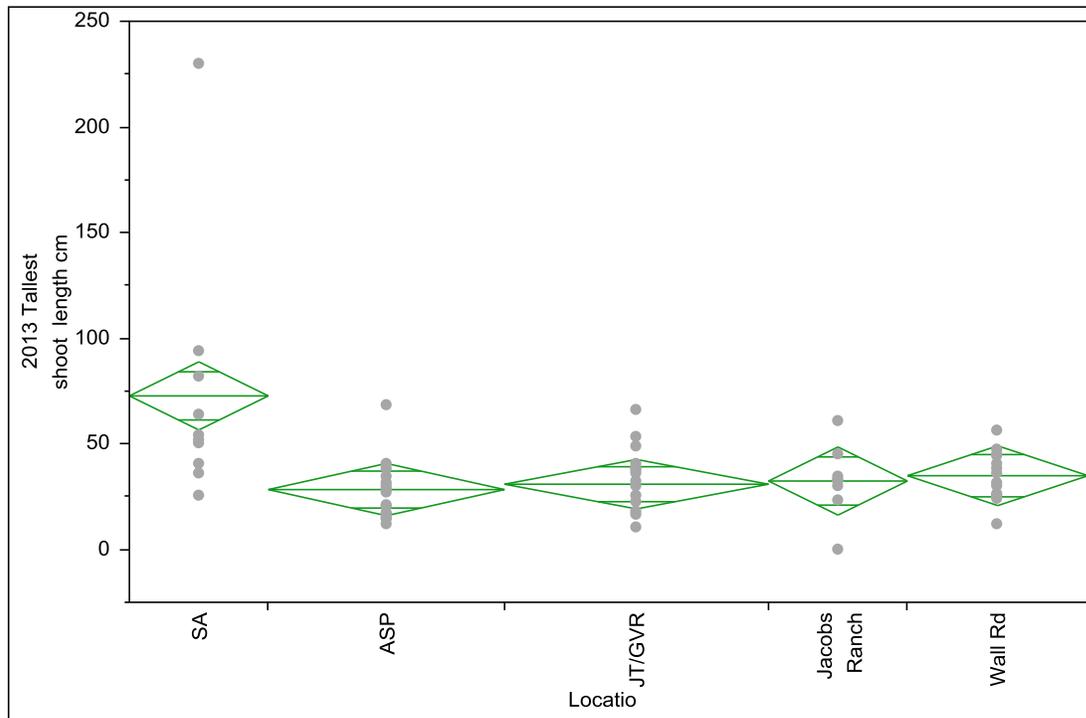
Given the dry spring conditions in 2013, we expect that few, if any new infections would have been initiated this year. Any new cases of SOD that develop by early 2014 would be associated with latent infections from spring 2012 or possibly 2011. Given the rate at which bay clearance has decreased and the lack of favorable disease conditions in 2013, it may be possible to defer reevaluation of the plots until late 2014 or 2015 without substantial loss in data quality. This may be necessary because no funding has yet been identified for continuing this portion of the study.

### California bay resprouting from stumps

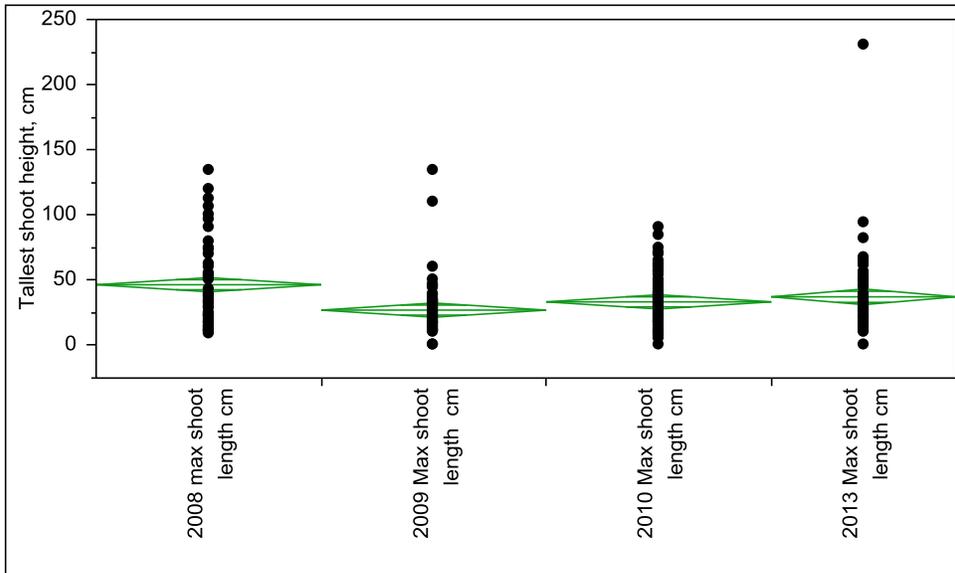
Sprouts from the monitored stumps continue to be strongly suppressed by deer browsing (Figure 7). As seen in previous years, bay resprouts have grown taller at the SA location compared to the other locations. On average, sprout heights have not changed

significantly between 2010 and 2013 (Figure 8). The tall outlier in the 2013 data is at the SA location (Figure 8). We cut 54% of the bay shoots after measurement in 2013. Because we were unsure when we might be able to return to these plots, we removed bay stump sprouts within a meter of bay-removal oaks even if the shoots were relatively small. The smallest shoot we removed was 18 cm tall and the tallest was 2.3 m tall.

At other study locations, cut stumps were treated with glyphosate, which has killed many of the stumps and provided greater sprout suppression. Among the 84 monitored stumps in this study, only one had died by 2013. However, the degree of suppression provided by browsing has been acceptable for most bay stumps other than those located very close to the oaks. If it is desirable to reduce stump herbicide treatment applications, it may be possible to restrict herbicide treatments to stumps located close (within about 2 m) to oaks, very large stumps (which tend to produce vigorous sprouts, and stumps located in areas that may be difficult for deer to access (e.g., in constricted or very brushy areas).



**Figure 7.** Maximum height (cm) in 2013 of bay sprouts from stumps of trees cut in 2007. Location means are shown by the horizontal line in center of each diamond.



**Figure 8.** Maximum height (cm) of bay resprouts in 2008, 2009, 2010 and 2013 from stumps cut in 2007. Means are shown by the horizontal line in each diamond.