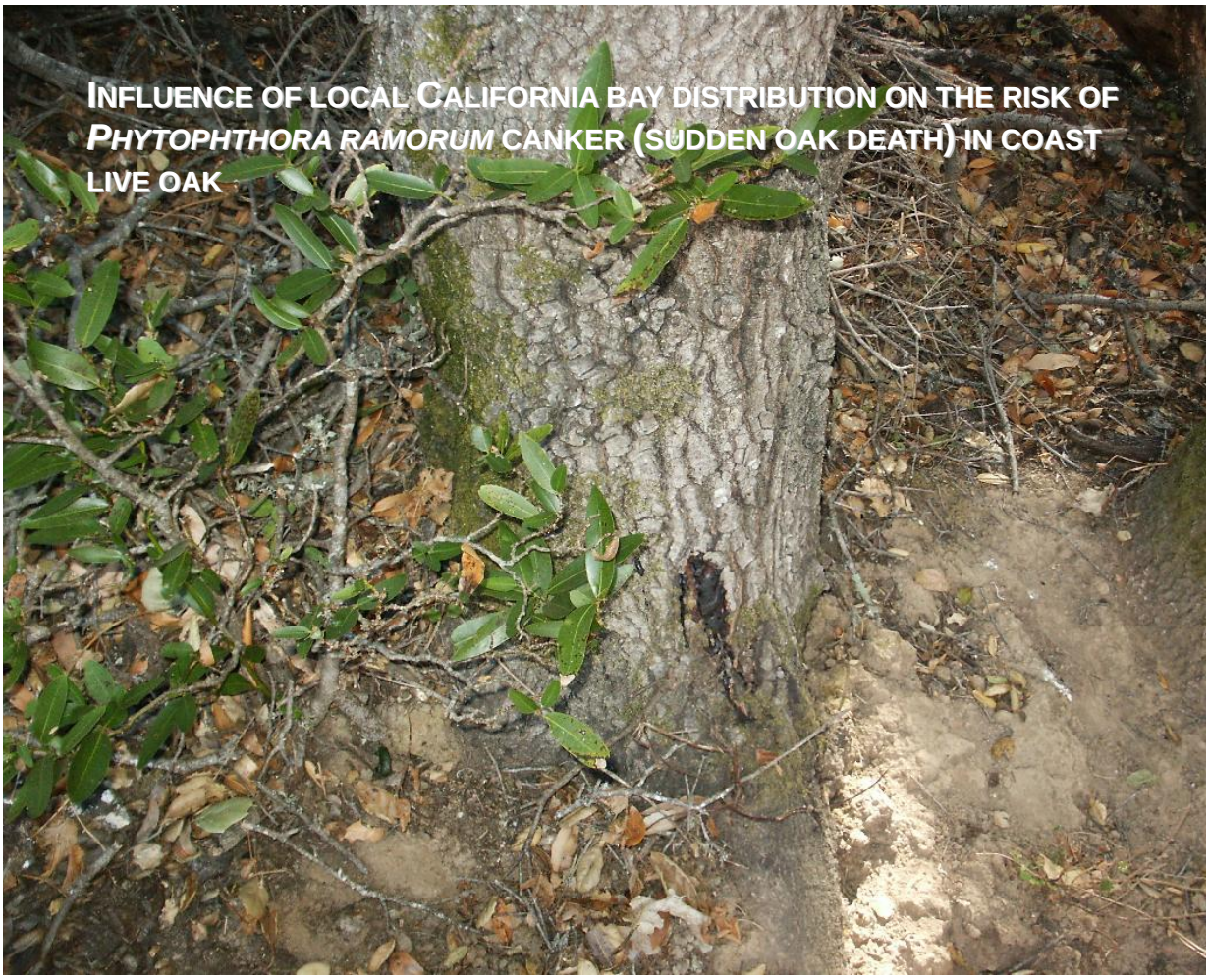

INFLUENCE OF LOCAL CALIFORNIA BAY DISTRIBUTION ON THE RISK OF *PHYTOPHTHORA RAMORUM* CANKER (SUDDEN OAK DEATH) IN COAST LIVE OAK

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April 27, 2007

Agreement No. 02-JV-11272138-063
Phytosphere Research Project No. 2002-0803

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Cover photo: Coast live oak, positive isolation for *P. ramorum* from canker under California bay in Novato, California.

ACKNOWLEDGEMENTS

This study was conducted with funding provided by the USDA Forest Service, Pacific Southwest Research Station and Phytosphere Research under cost share agreement 02-JV-11272138-063.

We would like to express our appreciation to the following persons who assisted us with various aspects of this project.

Dr. David Rizzo and members of his lab at the Department of Plant Pathology, U.C. Davis, provided selective media and lab space for isolations and assisted in the identification of *P. ramorum* isolates.

We also thank the following persons that assisted us in securing study locations. Dave Boyd and Patrick Robards (California State Parks) assisted us in arranging plot establishment at China Camp State Park. Mike Swezy (Marin Municipal Water District) provided valuable assistance in establishing plots on MMWD lands. Kent Julin (Marin County Fire Department) helped arrange contacts with private landowners in Marin County. Mischon Martin (Marin County Open Space District) helped us arrange for the plot location in Novato. We also thank the private landowners who allowed us to establish plots on their properties.

SUMMARY

Foliar infections in California bay (*Umbellularia californica*) are the most important known source of inoculum contributing to *Phytophthora ramorum* canker in coast live oak (*Quercus agrifolia*). This research addressed the question of whether there is a "safe" distance between California bay and coast live oak beyond which the risk of disease is acceptably low. We quantitatively evaluated bay cover and other factors in the neighborhoods around 247 coast live oaks in long-term research plots in mixed hardwood forests where *P. ramorum* canker has been prevalent since 2000.

Both the risk and severity of *P. ramorum* canker decreased as the minimum distance between California bay foliage and the oak trunk increased. Disease risk and severity were greatest at bay foliage-oak trunk distances of 1.5 m or less and were minimal at a distance of 10 m or more. Risk of tree mortality due to *P. ramorum* canker was highest for bay foliage-oak trunk distances less than 0.5 m.

Disease risk and disease severity increased as bay cover within 2.5 m of the trunk increased. Bay cover within 2.5 m of the trunk was a stronger predictor of disease risk and severity than the minimum bay-trunk distance. Bay foliage located south and west of an oak (prevailing wind directions during storms) had greater influence on disease incidence and severity than bay foliage that was north or east of the oak. The strength of this directional effect was greater for bay foliage 2.5-10 m from the oak trunk than for bay foliage within 2.5 m of the trunk.

For some oaks with *P. ramorum* canker, the presence of disease symptoms could not be readily explained by proximity to bay. Large amounts of poison oak (*Toxicodendron diversilobum*) vines climbing in the oak canopy or in adjacent trees appeared to be the most likely source of *P. ramorum* inoculum for these trees.

Based on timed counts of symptomatic bay leaves repeated at intervals between fall 2005 and fall 2006, bay foliar infection levels were minimal in fall and peaked in late spring and summer. Counts of infected bay leaves in fall 2005 were not correlated with counts from the same trees in either spring/summer 2006 or fall 2006. Spring/summer 2006 counts were correlated with fall 2006 counts.

Based on our results, the risk of developing *P. ramorum* canker in coast live oak appears to be minimal at bay foliage-oak trunk clearances of 10 m or more. The risk of *P. ramorum* canker and the severity of the disease can be greatly reduced, but not completely eliminated by (1) removing bay from within 2.5 m of the trunk of a susceptible oak; (2) extending bay foliage-oak trunk clearance to 5 m where possible, especially in the direction(s) from which storm winds blow; (3) pruning low branches to obtain up to 5 m of clearance in the lower canopy even if upper canopy bay branches are present at closer horizontal distances; and (4) eliminating poison oak climbing at canopy level within an oak or in adjacent tree within 2.5 m of the oak trunk .

Additional key words: *Umbellularia californica*, *Quercus agrifolia*, disease risk, disease severity, cover, clearance

INTRODUCTION

On coast live oak (*Quercus agrifolia*), *P. ramorum* canker or sudden oak death (SOD) exhibits a patchy distribution both within its range in California and within affected stands (Rizzo and others 2005). Some of this patchiness may be related to the length of time that has elapsed since the pathogen was introduced into the stand (Rizzo and others 2005, Swiecki and Bernhardt, in press). However, even within stands that have been heavily infested with *P.*

ramorum for at least 7 years, SOD has not become uniformly spread throughout the stands (Swiecki and Bernhardt 2006). This suggests that the epidemiology of the disease is strongly influenced by factors operating at a localized spatial scale.

Since 2000, we have been studying disease risk and progress in coast live oak in 128 long-term research plots in areas where *P. ramorum* canker is prevalent (Swiecki and Bernhardt 2006). Our analyses indicate that both tree- and plot-level factors are significant predictors of *P. ramorum* canker for coast live oak. A number of characteristics that are seen exclusively or primarily in relatively vigorous, fast growing trees (including high levels of canopy exposure, high stem water potential, unweathered bark in bark furrows, and greater bark thickness) are significantly associated with high disease risk (Swiecki and Bernhardt 2004, 2005). In addition, the presence and abundance of California bay (*Umbellularia californica*) within plots was identified as a significant plot-level predictor of disease risk in our initial data analyses (Swiecki and Bernhardt 2001). Several related variables, including counts of bay trees within the 8 m radius plot and plot bay cover are significant predictors of SOD risk (Swiecki and Bernhardt 2004), showing that disease risk increases with increasing bay density and cover within 8 m of a coast live oak.

Davidson and others (2002, 2005) showed that *P. ramorum* infects bay foliage and sporulates abundantly on it, but does not sporulate on coast live oak. The amount of *P. ramorum* inoculum dispersed from bay canopies decreased rapidly as the distance from the bay canopy source increased from 0 to 5 m or beyond (Davidson and others 2005). Tjosvold and others (2006) did not detect *P. ramorum* propagules more than 1 m away from infected rhododendron source plants, and infection of rhododendron trap plants was not observed more than 0.5 m from infected source plants.

Taken together, these studies indicate that bay foliage closest to a host oak is likely to make the largest contribution to disease risk. However, the studies do not allow us to determine a minimum "safe" bay foliage-oak distance for purposes of disease management.

Based on current understandings of the epidemiology of *P. ramorum* canker in mixed bay-oak woodlands, we hypothesized that the following factors were likely to influence the risk of *P. ramorum* infection in coast live oak:

1. The minimum distance between infected bay foliage and the trunk and/or canopy of a susceptible coast live oak.
2. The amount of bay foliage present at a given distance. Other factors being equal, higher amounts of foliage should produce greater amounts of inoculum.
3. The level of foliar infection present on the bay foliage, which is related to the genetic susceptibility of the bay as well as other local factors that influence the foliar disease epidemic on bay.
4. The direction of bay relative to the oak trunk. If inoculum is moved primarily by wind or rain splash, the prevailing wind direction during storm events could affect inoculum dispersal. Bay foliage located downwind from an oak could pose a greater disease risk than bay that is located an equal distance away in the upwind direction.
5. The vertical distribution of foliage with respect to the coast live oak canopy. Splashed and wind-blown inoculum is likely to spread greater horizontal distances if it is produced at greater heights in the canopy than if it is produced on foliage near the ground. In addition, inoculum that impacts the canopy of a coast live oak may be transported to the lower trunk via rainwater that runs down the stems. This transport pathway may not be important for inoculum produced on low understory bay foliage located away from the oak bole.

In order to assess the relative importance of these factors in determining the risk of *P. ramorum* canker in coast live oak, we conducted a study to determine whether various factors describing bay distribution in the neighborhood a coast live oak were predictors of *P. ramorum* canker presence and severity.

METHODS

Study sites and plots

The plots used for this study were established in September 2000 for a case-control study on factors influencing development of *P. ramorum* canker (Swiecki and Bernhardt 2001). Plots were established in areas where *P. ramorum* had been shown to be prevalent. The 10 locations used in this study are shown in *table 1*.

Table 1. Locations of plots and host species studied**.**

Location number	Location	County	Approximate latitude and longitude	Number of plots
1	Marin Municipal Water District (MMWD) watershed - Azalea Hill area	Marin	37.9723 N 122.6274 W	12
2	MMWD-Pumpkin Ridge south	Marin	37.9527 N 122.5949 W	16
3	MMWD-Pumpkin Ridge north	Marin	37.9599 N 122.5989 W	11
4	MMWD-Phoenix Lake area	Marin	37.9590 N 122.5770 W	11
5	China Camp SP - Miwok Meadows area	Marin	38.0044 N 122.4848 W	16
6	China Camp SP - SE Buckeye Point area	Marin	38.0044 N 122.4768W	12
7	Woodacre (Private land)	Marin	38.0175 N 122.6472 W	12
8	Lucas Valley (Private land)	Marin	38.0432 N 122.5996 W	12
10	Wall Road (Private land)	Napa	38.4092 N 122.4751 W	13
11	Novato (Private land)	Marin	38.0988 N 122.6273 W	13

At each study location, we established circular 8 m radius (0.02 ha) fixed-area plots, each of which was centered at a coast live oak tree. The tree-centered plots were spaced approximately 25 m apart although actual interplot spacing varied with vegetation and terrain. We marked the center tree in each plot with a numbered aluminum tree tag, and mapped the positions of other coast live oaks in the plots by recording distance and azimuth from the plot center tree. Trees in the plots were evaluated annually in September of 2000 through 2006 for symptoms of *P. ramorum* canker and other indicators of tree health (Swiecki and Bernhardt 2006). For this study, overall *P. ramorum* symptom status and estimated girdling due to *P. ramorum* cankers were evaluated as the primary disease outcomes.

P. ramorum symptom status was visually assessed using the following scale: (0) no symptoms; (1) early symptoms: bleeding cankers only; (2) late symptoms: cankers plus *Hypoxylon thouarsianum* sporulation and/or beetle boring; (3) dead as result of *P. ramorum*

infection. The disease status of some symptomatic trees was confirmed by isolating the pathogen from bark tissue pieces sampled from the canker margins. *P. ramorum* was the only *Phytophthora* sp. recovered from cankers at the study locations.

The percentage of the oak main stem that was girdled by *P. ramorum* cankers was estimated visually, based on bleeding, bark characteristics such as obvious necrosis or cracking, and, in some trees, limited chipping of bark to expose the canker margins. The overall girdling rating was derived by estimating the extent of all cankers in the lower 2 m of the bole and combining the affected percentage of the circumference as if all cankered areas were on the same stem cross section. Hence, cankers at different heights along the stem increase the girdling ranking only if they are horizontally offset around the stem circumference. We used the following 0 to 6 scale, the intervals of which are pretransformed using the arcsine transformation, to estimate the percent of stem circumference girdled: 0 = no girdling seen; 1 = <2.5 percent girdled; 2 = 2.5 to <20 percent girdled; 3 = 20 to <50 percent girdled; 4 = 50 to <80 percent girdled; 5 = 80 to <97.5 percent girdled; 6 = 97.5 to 100 percent girdled or tree dead due to *P. ramorum*.

Tree selection

We selected coast live oaks trees from the study plots to represent cases (trees with *P. ramorum* canker symptoms) or controls (trees lacking symptoms of *P. ramorum* canker). The symptom status of individual trees could be determined with a high degree of reliability because trees had been observed for disease symptoms and disease progress annually between 2000 and 2006. All symptomatic coast live oaks in the study plots, except for those with ambiguous disease symptoms, were selected as cases.

The initial pool of controls included all trees in the plots that were free of *P. ramorum* canker symptoms over the previous 7 years. In selecting controls, we also eliminated trees that had tree characteristics that previous models have shown to be associated with low disease risk (Swiecki and Bernhardt 2001, 2004). These included trees that were almost completely overtopped (low values for sky-exposed canopy), had very low ratings for unweathered tissue in bark fissures, and/or were in severe decline due to agents other than *P. ramorum* canker.

Trees were selected based on existing data sets prior to locating the trees in the field to avoid potential bias. Preselected trees were rejected in the field only if trees (either the selected oak or nearby bays) had failed and bay neighborhood prior to failure could not be reliably assessed. In all, 247 coast live oak trees were included in this study, 64 percent of which had symptoms of *P. ramorum* canker. The disease status of the trees in the study is shown in *table 2*.

Table 2. Disease status of coast live oak trees included in the study based on evaluations through September 2006. Early = bleeding cankers only; Late = cankers and associated sporulation of *Hypoxylon thouarsianum* and/or damage by wood boring beetles; Dead = entire tree killed by *P. ramorum* canker. Multistemmed trees with both live stems and stem(s) killed by *P. ramorum* were classified as having late disease symptoms.

P. ramorum canker status	Number of trees	Percent of study trees
Early symptoms	40	16
Late symptoms	58	23
Dead	59	24
All with *P. ramorum* symptoms	158	64
Asymptomatic	90	36
Total trees	247	

Evaluation of bay around oaks

In order to obtain basic data on patterns of bay distribution around oaks selected for the study, we conducted a pilot-level investigation on 37 coast live oaks in September and October 2005. We mapped the distribution of bay around each of these trees by aggregating the bay into zones defined by: the azimuth from the oak trunk to the start and end of the bay zone; the minimum distance from the trunk to bay foliage within the zone; and the depth of the zone along a line radiating from the center of the oak trunk. We counted the number of bay trees in each zone, measured the height of the bay above the oak base, and quantified bay foliar symptoms using 45-second timed counts of the number of leaves with apparent *P. ramorum* foliar symptoms. We also measured the average distance from the oak canopy edge to the oak trunk and the average extent of the oak trunk watershed within the zone. The edge of the oak trunk watershed was defined as the furthest branch that was continuously sloped back toward the trunk so that water flowing down branches could reach the trunk.

Based on a preliminary analysis of this data, we revised our data collection protocols to reduce the amount of time needed for assessments and analysis. Trees were assessed using the revised protocols between October 2005 and July 2006.

Under the revised protocols, we estimated bay cover within concentric rings centered around each oak tree included in the study. The rings were based on the following distance ranges from the oak trunk: <2.5 m, 2.5 to 5 m, 5 to 10 m, and 10 to 20 m (*figure 1*). Each distance ring was divided into four 90 degree arcs centered at each of the cardinal compass directions (*figure 1*). Within the three innermost rings, we estimated the bay cover in each quarter arc of the ring using the following quartile scale: 0 = no bay cover; 1 = 1 to 25 percent bay cover; 2 = 26 to 50 percent bay cover; 3 = 51 to 75 percent bay cover; 4 = more than 75 percent bay cover.

For the 10 to 20 m distance ring, only bay presence or absence in each arc was noted. Within all distance rings, we also noted the height classes of any bay present using the following relative height scale:

understory = bay foliage from ground level to half the height of the oak

codominant = bay foliage as high as the in upper half of oak canopy and up to 4 m taller than oak

overstory = bay foliage present at heights of 4 m or more above top of oak canopy

In most but not all cases, the overstory class consisted of bays located upslope from a study oak. One or more of the bay height classes could be present in each distance-direction ring arc.

Because overstory bay foliage almost always occurred in combination with codominant bay foliage, these two categories were consolidated into a combined overstory class.

Based on field observations of water flow along stems, it appeared that the total trunk watershed, as defined above, might overestimate the size of the catchment area from which *P. ramorum* inoculum could be channeled to the trunk. We therefore defined a smaller catchment area, the inner watershed, which included the portion of the canopy watershed that was likely to contribute the greatest amount of water flow down the main stem. The inner canopy watershed consisted of only first and second order branches that were at least one quarter the diameter of the trunk and that sloped back towards the trunk at angles no greater than 45 degrees from vertical. We measured the distance from the trunk to the edge of the total trunk watershed and the inner watershed in each of the four directional quadrants. We also recorded whether bay foliage overtopped the oak main stem; the minimum distance between bay foliage and the trunk and the direction that it occurred in; and the minimum distance from the oak total watershed and inner watershed to bay foliage, if this distance was less than the trunk-bay distance.

We used an angle gauge with an attached high intensity green laser pointer to project vertical lines into the canopy to help define the edges of distance rings and arcs and the oak trunk watershed edges. A hand-held Leica Disto™ laser rangefinder was used to measure distance to the oak trunk.

We also noted the presence, location, and amount of other foliar hosts of *P. ramorum*, such as tanoak or poison oak (*Toxicodendron diversilobum*), that might serve as alternative sources of inoculum.

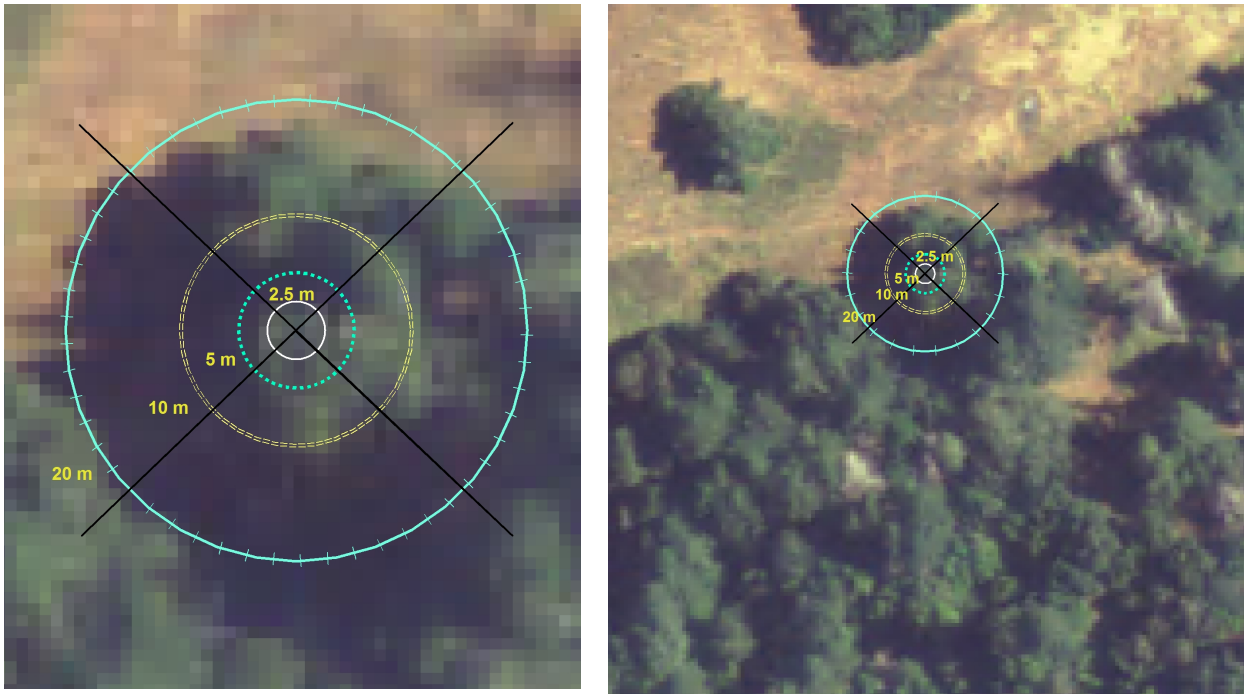

Figure 1. Layout of zones used for assessing bay cover around individual oaks. Distance rings are drawn to scale and are superimposed on an aerial image of a relatively large-canopied oak shown at higher (left) and lower (right) magnifications. Black lines represent the edges of the 90 degree arc ring segments.

Bay leaf symptom counts

As noted above, we used 45-second timed counts of symptomatic leaves to assess foliar disease levels in a total of 106 mapped bay zones around 37 coast live oaks in September and October 2005. The zones contained varying numbers of bay trees: 47 zones had one bay tree, 37 zones had clumps of two to three bay trees, and 22 zones had greater numbers (4 to 19) of bay trees. Counts of foliar symptoms in the same bay zones were made using the same methods between late May and early August 2006 and again in September 2006 for all zones. A subsample of the zones was also recounted in January 2006. All counts were made by the same observer for all trees and all sampling dates.

Statistical analyses

We used JMP® statistical software (SAS Institute Inc., Cary NC) for data analysis. Unless otherwise indicated, effects or differences are referred to as significant if $p \leq 0.05$. The square root transformation was applied to count variables (e.g., bay foliar symptom counts) prior to analysis.

We used analysis of variance (F-tests) or t-tests to compare means of continuous variables. For ordinal variables such as bay cover percentage ratings, the nonparametric Wilcoxon rank sum test was used to test the significance of differences. Differences between medians were tested using the nonparametric median test. Effects of sampling date and other variables on bay symptom counts were tested using repeated measures analysis of variance. We used linear regression to test for correlations between continuous variables. The nonparametric Spearman test was used to test for correlations between pairs of categorical variables.

Recursive partitioning was used to develop models and investigate interactions between predictors. Recursive partitioning splits data in a dichotomous fashion, with each partition chosen to maximize the difference in the responses between the two branches of the split. We also developed logistic regression models to examine the effects of factors on the binary disease outcome (tree is diseased, i.e., a case) and used generalized linear models to test relationships between various predictor variables and the girdling rank outcome.

RESULTS

Minimum distance to bay foliage

California bay was well-distributed throughout the mixed hardwood forests at the study locations. Only 6 of the coast live oak trees in the study (2.4 percent) did not have bay present within 20 m of the trunk. Figure 2 shows that the distributions for minimum distances from bay foliage to the oak trunk differed for coast live oaks with and without *P. ramorum* canker symptoms. Although both distributions are strongly left-skewed, the mean and median bay foliage-oak trunk distances were significantly greater for the controls than for the cases (*table 3*). In addition, the minimum distance between bay foliage and the total and the inner oak trunk watersheds differed significantly between cases and controls (*table 3*).

Poison oak is a known *P. ramorum* host, although inoculum production on this host has not been studied. Overall, five cases (three of which were dead in 2006) had substantial amounts of poison oak climbing in their canopies and three had canopy-level poison oak in adjacent trees at distances of 2.5 m or less from the trunk. Among controls, only one had poison oak climbing in the canopy and no others had canopy-level poison oak within 2.5 m of the trunk. The two cases with the greatest bay foliage-oak trunk distances (greater than 15 m) had extensive amounts of poison oak climbing in their canopies (*figure 2*). The other two cases with bay at distances of 9.7

and 12 m had extensive climbing poison oak in adjacent tree canopies that were within 1 m and 7.5 m of the oak trunk, respectively. If we exclude cases for which canopy-level poison oak could have served as a source of *P. ramorum* inoculum, the maximum bay foliage-oak trunk distance among cases is less than 10 m.

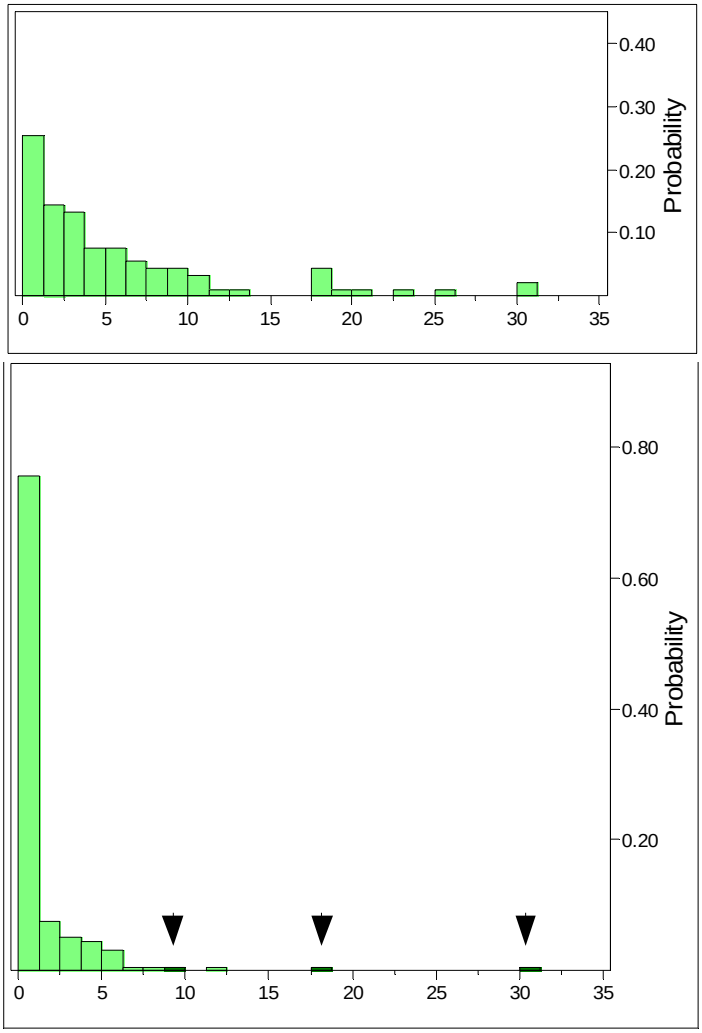

Figure 2. Minimum distance (m) between nearest bay foliage and coast live oak trunks for oaks without (top graph, n=90) or with (bottom graph, n=157) *P. ramorum* canker symptoms. Three dark shaded bars in the bottom graph (arrows) indicate trees with extensive poison oak growing in the canopy.

Table 3. Minimum mean and median distances between bay foliage and the trunk or trunk watershed edge for coast live oaks without (controls) of with (cases) symptoms of *P. ramorum* canker.

Minimum distance measurement	Statistic	Controls (asymptomatic)	Cases (*P. ramorum* canker symptoms)
	n	90	157
bay foliage-oak trunk (m)	mean	5.7	1.3 [a]
	median	3.3	0 [b]
bay foliage-total oak trunk watershed (m)	mean	4.8	0.8 [a]
	median	2.4	0 [b]
bay foliage-inner oak trunk watershed (m)	mean	5.0	1.1 [a]
	median	3.1	0 [b]

[a] Control and case means significantly different (P<0.0001) according to 1 tailed t-test.

[b] Control and case medians significantly different (P<0.0001) according to the median test.

We used recursive partition analysis to more closely examine the relationship between the minimum distance from bay foliage and the presence of *P. ramorum* canker symptoms in coast live oak. For the minimum bay foliage-oak trunk distance, the greatest difference in both percent infection and in average *P. ramorum* canker girdling rating was achieved by partitioning at a 1.5 m distance. For oaks with a minimum bay foliage-oak trunk distance of less than 1.5 m, *P. ramorum* canker incidence was 83 percent and average girdling rank was 3.8 (nearly 80 percent girdling). In oaks with a bay foliage-oak trunk distance greater than or equal to 1.5 m, *P. ramorum* canker incidence was 33 percent and average girdling rank was 1.3 (less than 20 percent girdling). Furthermore, coast live oaks with bay foliage within 1.5 m of the trunk were more likely to have advanced disease symptoms (late or dead) than oaks for which the bay foliage-oak trunk distance was greater than 1.5 m (*figure 3*).

We also used recursive partition models to examine the relationship between *P. ramorum*-related mortality and minimum bay foliage-oak trunk distance. A minimum bay foliage-oak trunk distance of 0.5 m provided the greatest difference in levels of mortality associated with *P. ramorum*. Among oaks with bay foliage closer than 0.5 m to the trunk, 41 percent had been killed by *P. ramorum.* Among trees with bay at least 0.5 m from the trunk, only 8 percent had been killed by *P. ramorum*. Based on a single variable logistic regression model (model p<0.0001) for SOD mortality, oaks with bay foliage within 0.5 m of the trunk were almost nine times more likely to have been killed by *P. ramorum* than trees with greater bay foliage-oak trunk distances (odds ratio=8.7; 95 percent confidence interval=4.2–20).

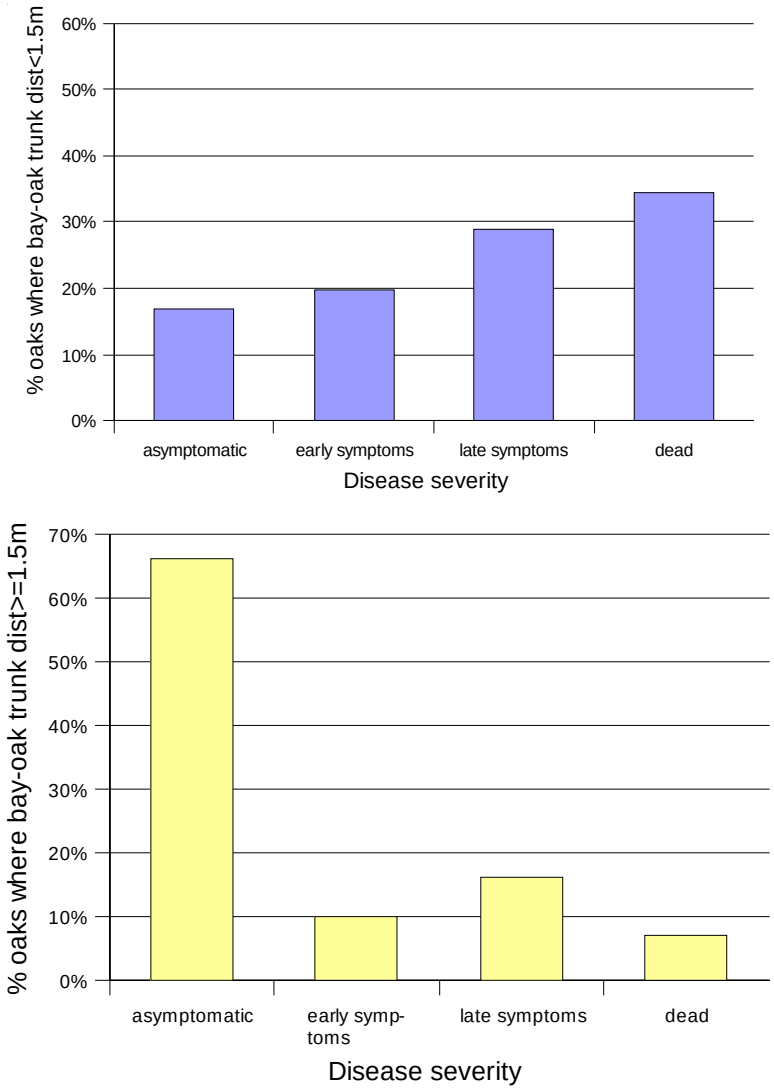

Figure 3. Frequency distribution of oaks in *P. ramorum* disease severity classes for trees with minimum bay foliage-oak trunk distances of less than 1.5 m (top graph, n=142) or 1.5 m or more (n=98 bottom graph). Trees with poison oak growing in the canopy or in adjacent trees within 1.5 m of the oak trunk are omitted from the graphs.

Figure 4 illustrates how the incidences of *P. ramorum* symptoms, *P. ramorum*-related mortality, and disease severity (based on girdling rating) decrease with increasing minimum bay foliage-oak trunk distance. For this graph, oaks with poison oak in the canopy or at canopy level within 1.5 m of the trunk have been omitted under the assumption that poison oak might serve as an alternative source of inoculum in these cases. Both the incidence of *P. ramorum* canker and average girdling rank decreased as the minimum bay foliage-oak trunk distance increased (*figure 4*). However, the incidence of *P. ramorum*-related mortality did not change significantly for distance classes greater than 0 m.

Only 15 trees in the sample had minimum bay foliage-oak trunk distances greater than 10 m, so all of these trees were aggregated in the highest distance class. The only symptomatic oak in

the >10 m minimum bay foliage-oak trunk distance class had a minimum bay foliage-oak trunk distance of 12 m and had canopy-level poison oak present 7.5 m from the trunk.

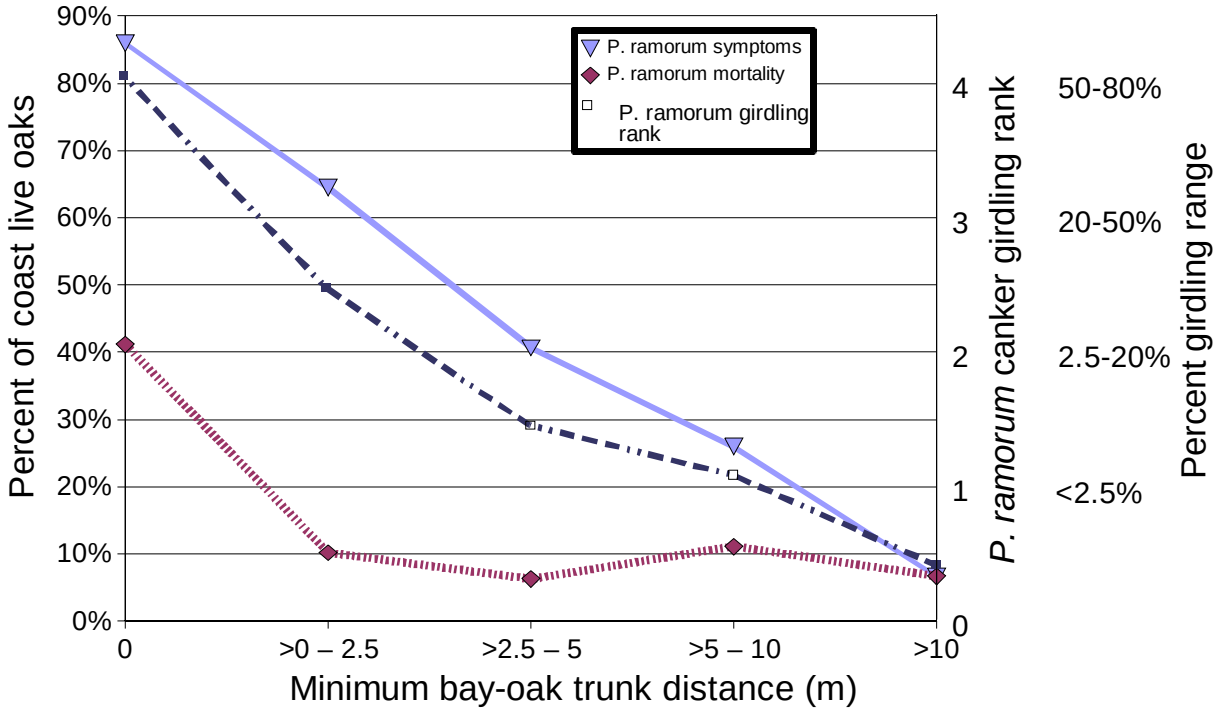

Figure 4. Percent of coast live oaks with *P. ramorum* symptoms and mortality due to *P. ramorum* (left scale), and average *P. ramorum* girdling rank (right scale) by minimum bay foliage-oak trunk distance class. Sample sizes for the distance classes from left to right are 107, 59, 32, 27, and 15. Trees with poison oak growing in the canopy or in the canopies of adjacent trees within 1.5 m of the oak trunk are omitted.

Interaction between bay distance and oak watershed size

Measurements of the extent of the total and inner trunk watershed in each cardinal direction were also used to estimate the areas of these two watersheds. Although the areas of the two watersheds for individual oaks were significantly correlated ($p<0.0001$), the R^2 value was only 0.219, due to the large amount of scatter in the data. This is related to the wide variation in tree branch structure among the study trees. Both the total and inner trunk watershed areas were also significantly correlated with the DBH of the largest stem ($p<0.0001$ for both), although the R^2 value for the correlation with total watershed area (0.342) was higher than that for the inner watershed (0.148).

Cases and controls did not differ significantly with respect to the area of the total or inner trunk watersheds or average DBH. For all study trees, the average radii of the inner and total trunk watersheds were 1.06 ± 0.71 m and 2.76 ± 1.40 m respectively.

We hypothesized that the minimum distance between bay foliage and the trunk watershed might be a better predictor of disease risk than bay foliage-oak trunk distance for trees with relatively large bay foliage-oak trunk distances. Among trees with bay foliage-oak trunk distances of 5 m or more (n=43), the minimum distance between the edge of the total oak watershed and the nearest bay foliage was significantly less ($p=0.0297$, two-tailed t-test) for

cases (average distance = 4.7 m) than for controls (average distance = 10.7 m). This subset of cases and controls also differed significantly with respect to the minimum distance between inner oak watershed and the nearest bay foliage ($p=0.0422$, two-tailed t-test) but not the minimum bay foliage-oak trunk distance ($p<0.0530$, two-tailed t-test). For these 43 study trees, the DBH of the largest stem was also significantly greater ($p=0.0039$, two-tailed t-test) for cases (average 56 cm) than controls (average 36 cm), but DBH was not significantly correlated with the minimum distances between bay foliage and the oak trunk or the total or inner oak watershed. These results suggest that for larger trees, considering only the distance between bay foliage and the trunk may underestimate disease risk.

We developed recursive partition models using the minimum distances between bay foliage and the oak trunk, total oak watershed, and inner oak watershed as candidate predictors. The data set for these analyses (n=240) excludes trees with poison oak in the canopy or at canopy level within 1.5 m of the trunk. The initial split in the recursive partition model for the case/control outcome was at a bay foliage-total oak watershed distance of 2.26 m or more (84 percent controls in this group). The group with bay foliage-total oak watershed distances less than of 2.26 m (77 percent cases) was then split at a minimum bay foliage-oak trunk distance of 1.3 m (<1.3 m = 83 percent cases; ≥ 1.3 m = 58 percent cases). For the girdling rating outcome, the initial split was made at a bay foliage-oak trunk distance of 1.5 m; trees in the ≥ 1.5 m group were subsequently split at a bay foliage-total oak watershed distance of 2.26 m. These results indicate that disease risk is elevated for trees with bay foliage-total oak watershed distance of less than 2.26 m even if the bay foliage-oak trunk distance is greater than about 1.3 to 1.5 m.

Bay cover within distance rings

Analysis of bay cover data for the various distance rings around the cases and controls is complicated by correlations between these variables. The mean bay cover ratings from each of the distance rings show significant positive correlations with all other rings, although the highest correlations are seen between adjacent distance rings (*table 4*). In addition, minimum bay foliage-oak trunk distance is negatively correlated with bay cover ratings for each of the distance rings; the strongest correlations are seen for the distance zones closest to the oak trunk (*table 4*). These correlations are related to the overall spatial distribution of bays around coast live oaks in these plots. In addition, many of the bays in the plots have large enough canopies that they commonly span multiple distance rings, especially the innermost distance rings.

Table 4. Spearman's rho rank correlation coefficients for pairwise comparisons between bay distance and cover variables. All correlations shown are significant at $p<0.0001$.

Variable	0-2.5 m bay cover	2.5-5 m bay cover	5-10 m bay cover	10-20 m bay presence
Minimum bay foliage-oak trunk distance	-0.9095	-0.8047	-0.5744	-0.4004
0-2.5 m bay cover		0.8644	0.6113	0.3445
2.5-5 m bay cover			0.7626	0.3801
5-10 m bay cover				0.5764

One consequence of the strong correlations is that some of the variables are confounded. Some combinations of variable levels that are needed to differentiate between certain variables either are lacking or represented by too few points to be statistically meaningful. For example, among the 77 trees with a minimum bay foliage-oak trunk distance of 2.5 m or more, only 7 trees have an average bay cover rating of 1 or higher (at least 25 percent bay cover) in the 2.5 to 5 m

distance ring. Hence, the combination of no bay foliage close to the trunk but high amounts beyond 2.5 m is poorly represented in the data set. In comparison, 137 of the 170 trees with a minimum bay foliage-oak trunk distance of less than 2.5 m have at least 25 percent bay cover in the 2.5 to 5 m distance ring. Our ability to differentiate between the effects of bay cover within 2.5 m of the oak trunk and bay cover between 2.5 and 5 m from the oak trunk is limited because the bay cover within these two zones is highly concordant in the data set.

As shown in *figure 5,* the average bay cover ratings of cases are significantly greater than those of controls for all distance rings. These significant differences persist if oaks with minimum bay foliage-oak trunk distances of less than 0.5 m are omitted, although the significance level of the 10-20 m zone is slightly decreased (p=0.011, Wilcoxon rank sum test). Although the relative differences in bay cover between cases and controls become smaller as the distance from the oak increases (*figure 5*), it is difficult to separate the effects of bay cover in the different zones due to the high level of correlation between the distance classes (*table 4*).

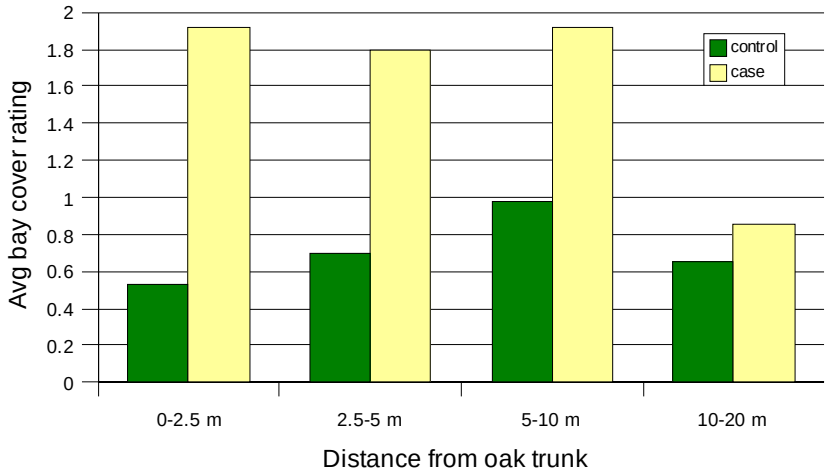

Figure 5. Average California bay cover ratings in distance rings around coast live oak controls (asymptomatic trees) and cases (trees with *P. ramorum* canker symptoms). For distance rings from 0 to 10 m, ratings were made using the quartile scale; an average rating of 1 indicates 1 to 25 percent bay cover, 2 indicates 26 to 50 percent bay cover. For the 10-20 m ring, only presence (1) or absence (0) was scored. An average rating of 1 in the 10-20 m ring indicates that bay is present at this distance in all four cardinal directions. All differences between cases and controls are significant at $p<0.0001$ according to a two-tailed t-test (distance rings from 0 to 10 m) or Wilcoxon rank sum test (10-20 m distance ring).

We used recursive partitioning to investigate the relative ability of bay cover variables to predict disease outcomes. *P. ramorum* canker girdling rank was used as the primary disease outcome because it takes into account both disease incidence and disease severity. Oaks with canopy-level poison oak within 1.5 m of the trunk were omitted as before, although the first two splits of the recursive partition model are nearly the same if these trees are included. Using the four variables in *table 4*, the initial partition was based on the average bay cover rating within 2.5 m of the oak trunk (*table 5)*. The cutting value is at an average bay cover rating of 0.775 within 2.5 m of the oak trunk, somewhat less than 25 percent bay cover within this ring. The next two splits were based on bay cover in more distant rings. The minimum bay foliage-oak distance was not used as a splitting criterion until the fourth partition (*table 5)*. In a recursive partition

model using *P. ramorum* canker presence as the outcome variable, the same initial split is made, although subsequent splits differ somewhat from those shown in *table 5*.

Overall, these models indicate that bay cover within 2.5 m of the oak trunk is the best single predictor of *P. ramorum* canker incidence and severity in these trees. Oaks with 25 percent cover or more in this zone showed the highest disease incidence and severity. However, higher levels of bay cover in further distance zones (to at least 10 m) also tend to increase disease incidence and severity, although the confounding of the data does not allow us to derive a robust estimate of the disease risk associated with bay cover in those farther zones.

Table 5. Recursive partition model for the *P. ramorum* girdling rank disease outcome. Candidate predictors were the four variables shown in *table 4*. Overall model R^2=0.334. Trees with poison oak within the canopy or at canopy level within 1.5 m of the trunk were excluded from the data set. Note that girdling ranks are non-linear (see methods).

Predictor variable *cutting value*			n	Mean *P. ramorum* girdling rank	*P. ramorum* incidence (percent)
bay cover rating <2.5 m *<0.775*	bay cover rating 5-10 m *<0.75*		37	0.38	13.5
	bay cover rating 5-10 m *≥0.75*		72	1.81	45.8
bay cover rating <2.5 m *≥0.775*	bay cover rating 2.5-5 m *≥1.775*		86	4.27	90.7
	bay cover rating 2.5-5 m *<1.775*	Min bay foliage-oak trunk dist *<0.5 m*	37	3.59	81.1
		Min bay foliage-oak trunk dist *≥0.5 m*	8	1.75	62.5

Effect of direction

In all of the study locations, the predominant wind direction during winter/spring precipitation events is from the south or west. If wind-blown rain contributes substantially to inoculum dispersal, we would hypothesize that the bay foliage located south or west of an oak would have a larger impact on disease risk than bay foliage located north or east of an oak.

To test this hypothesis, we first looked at the direction in which the minimum bay foliage-oak trunk distance occurred for oaks that had bay foliage-oak trunk distances greater than zero. For this analysis, we divided the directions into two categories: south, southwest, and west in one category (n=38) and the remaining directions (southeast through northwest, n=67) in the other. Using these two directional categories, the direction of the minimum bay foliage-oak trunk distance was a significant predictor of the binary disease outcome (likelihood ratio p=0.0338) with a greater percentage of cases (48 percent) than controls (28 percent) having minimum bay clearances to the south and west. In addition, *P. ramorum* girdling rank was significantly higher (2-tailed t-test p=0.0136) among oaks for which the minimum bay foliage-oak trunk distance was toward the south or west (average girdling rank 2.3) than in other directions (average girdling rank 1.2).

To further test the effect of direction on disease outcomes, we created new predictor variables based on averages of bay cover ratings from the distance ring segments in the south and west directions (S+W) and the east and north (E+N) directions. Bay cover ratings were averaged separately for the ring within 2.5 m of the oak trunk and across the two rings that spanned the 2.5 to 10 m distance from the oak trunk. The four resulting variables are significantly correlated with each other.

We constructed models for both the binary disease and girdling rank outcomes for oaks with a minimum bay foliage-oak trunk distance greater than zero, again omitting oaks with canopy-level poison oak within 1.5 m of the trunk (n=105). For the 0-2.5 m distance ring, average bay cover did not differ significantly between the S+W and the E+N directions. Average bay cover in both the S+W and the E+N directions were significant predictors of the binary disease and girdling rank outcomes, but the S+W direction variable was more highly significant for both outcomes. In addition, although both variables were significant in a generalized linear model for girdling rank ($p<0.0001$; parameter significance $p=0.0044$ for S+W, $p= 0.0266$ for E+N), only S+W 0-2.5 m bay cover was significant ($p=0.0006$, odds ratio 31.24) when both variables were included in a logistic regression model for the binary disease outcome (E+N 0-2.5 m bay cover $p=0.0533$). These analyses indicate that within the 0-2.5 m distance zone, bay cover south and west of the oak trunk may have a somewhat greater influence on disease incidence and severity than bay cover north and east of the oak.

We also tested whether the direction of bay cover in the 2.5-10 m distance ring influenced disease risk or severity. In these models, average bay cover in the 0-2.5 m distance ring was included as a predictor in the model; only oaks with canopy-level poison oak within 1.5 m of the trunk were omitted from the data set (n=240). In the 2.5-10 m distance ring, average bay cover was lower overall in the S+W direction (mean 1.33) than in the E+N direction (mean 1.63; two-tailed t test $p=0.0043$). Even so, in a logistic regression model for the binary disease outcome (*table 6*), average bay cover 2.5-10 m in the S+W direction was significant whereas E+N bay cover 2.5-10 m was not. Similarly, only average bay cover 0-2.5 m ($p<0.0006$) and S+W bay cover 2.5-10 m ($p=0.0026$) were significant predictors for the *P. ramorum* girdling rank outcome. Hence, when bay cover within 2.5 m of the oak trunk was accounted for in the model, an additional effect of bay cover in the 2.5-10 m distance ring is discernable, but only for bay cover south and west of the oak.

Table 6. Logistic regression model for the binary *P. ramorum* disease outcome (tree is symptomatic=case). Overall model $p<0.0001$, n=240. Trees with poison oak within the canopy or at canopy level within 1.5 m of the trunk were excluded from the data set.

Predictor	Parameter estimate	Likelihood ratio Chi Square	P level	Odds Ratio (95% CI)
average bay cover rating <2.5 m	0.465	7.125	0.0076	6.43 (1.62-28.2)
average bay cover rating 2.5-10 S+W	0.799	14.501	0.0001	24.48 (4.56-149.07)
average bay cover rating 2.5-10 E+N	0.216	1.255	0.2627	2.38 (0.52-10.83)

Effect of bay canopy height

To determine whether the vertical distribution of bay canopy present at various distances from an oak trunk has the potential to affect disease risk and severity, we noted the height classes

of bay canopy present in each directional sector in which bay cover was assessed in each distance ring. However, like the bay cover data, the bay height data are highly correlated and various height-distance combinations of interest are not represented at high frequencies, if at all. Consequently, the inferences we can draw from the data are limited.

For purposes of analysis, bay height classes within distance rings were consolidated into the following categories: no understory bay present, understory and overstory bay present, and no overstory bay present. In addition, because bay cover in the 0-2.5 m ring and S+W bay cover 2.5-10 m ring are significant predictors of disease, these variables were included in models to test the effect of bay height categories in the 0-2.5 m, 2.5-10 m and 10-20 m distance rings on disease risk.

In logistic regression models for the binary disease outcome and generalized linear models for the *P. ramorum* girdling rank outcome, bay cover variables were significant but height variables for the 0-2.5 m, 2.5-10 m and 10-20 m distance rings were not. Thus, when the effect of bay cover was accounted for, the vertical distribution of bay foliage within the distance rings was not a significant predictor of disease.

P. ramorum foliar symptoms on bay

Bay foliar symptoms were generally distributed in a nonuniform fashion within individual bay zones and among the bay zones surrounding a given oak. In general, symptomatic bay leaves were more common in the lower, typically shaded portions of the canopy than in the uppermost portions. In addition, symptoms were generally less common on open-grown trees, especially if they were relatively small and/or appeared water-stressed (leaves relatively small and somewhat chlorotic).

Figure 6 shows how the bay foliar symptom counts varied through a single year for 20 bay zones at two locations. The general pattern of seasonal variation is similar for all bay zones: counts were at or near minimum values in September/October and at maximum values in late spring/early summer. Reductions in the number of symptomatic leaves that occurred over the summer were due to early dehiscence of infected leaves. In some locations, many symptomatic leaves had become chlorotic by early May 2006 (*figure 7*), and these leaves had dropped by September. As shown in the bottom graph of *figure 6*, at least some bay zones showed strong decreases in foliar symptom counts between June and July, which suggests that infected leaves were dropping during this interval. Bay zones within a given location varied considerably with respect to the maximum and minimum counts observed over the year and the timing of increases and decreases in infected leaf counts (*figure 6*).

To determine how bay foliar symptom counts varied between locations, we selected the six locations for which midseason counts were made in the May-June interval, to reduce variation associated with the timing of the late spring-early summer evaluation. Repeated measures analysis of variance showed that symptomatic leaf counts varied significantly over time ($p<0.0001$), and by study location (time × location interaction $p<0.0001$; *figure 8*). However, counts did not differ significantly based on the number of trees within a bay zone.

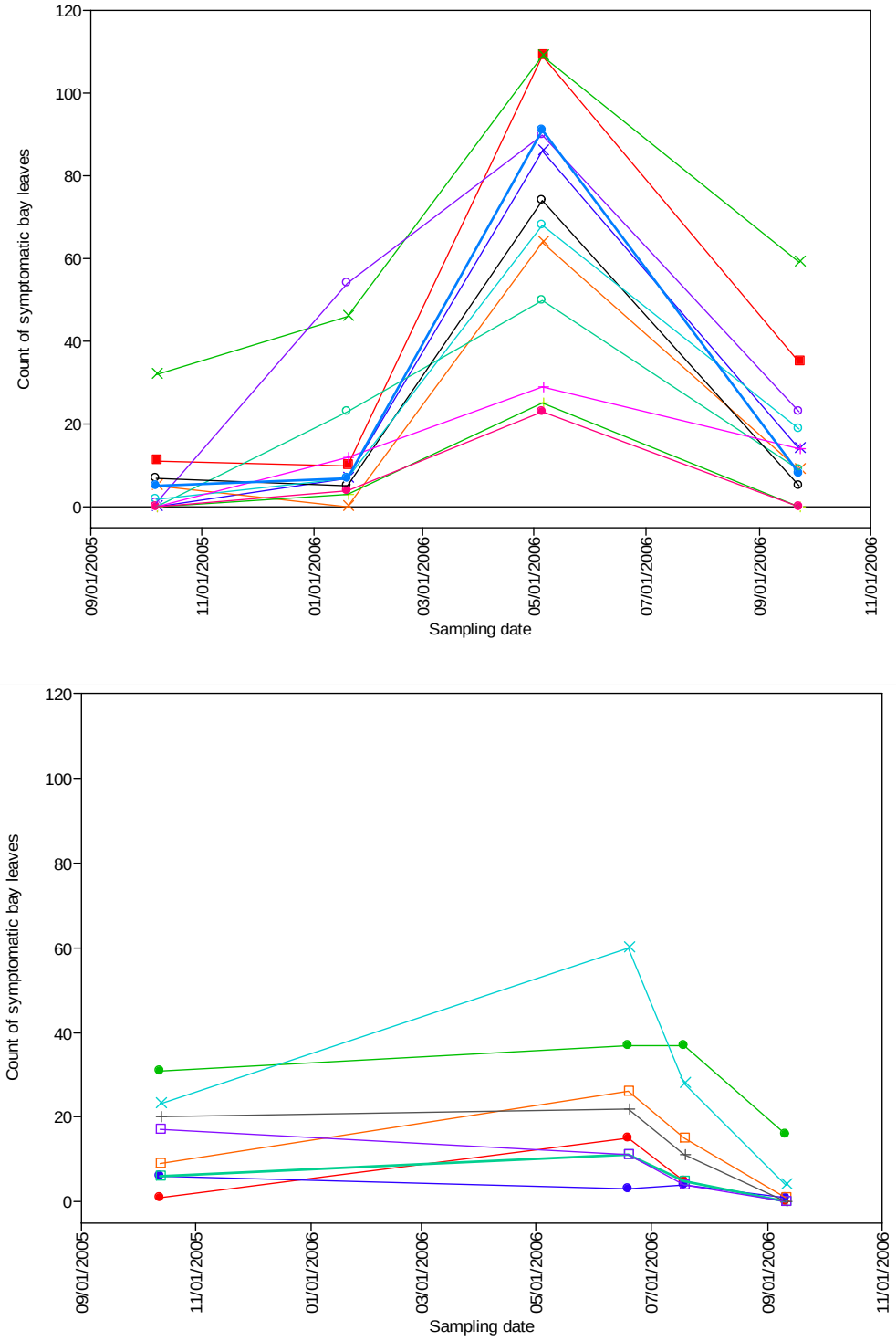

Figure 6. Number of infected bay leaves counted in a 45 second search period for 12 bay zones at location 5 (top) and 8 bay zones at location 7, assessed on four dates between September 2005 and October 2006. Connected points represent counts in the same bay zone. Within each graph, zones with the same symbol type are located around the same oak.

Figure 7. Bay leaves with foliar symptoms of *P. ramorum* infection at location 5 (Miwok Meadows) on 6 May 2006. Many of the symptomatic leaves were chlorotic at this point in the season and had been shed by the time trees were reassessed in fall 2006.

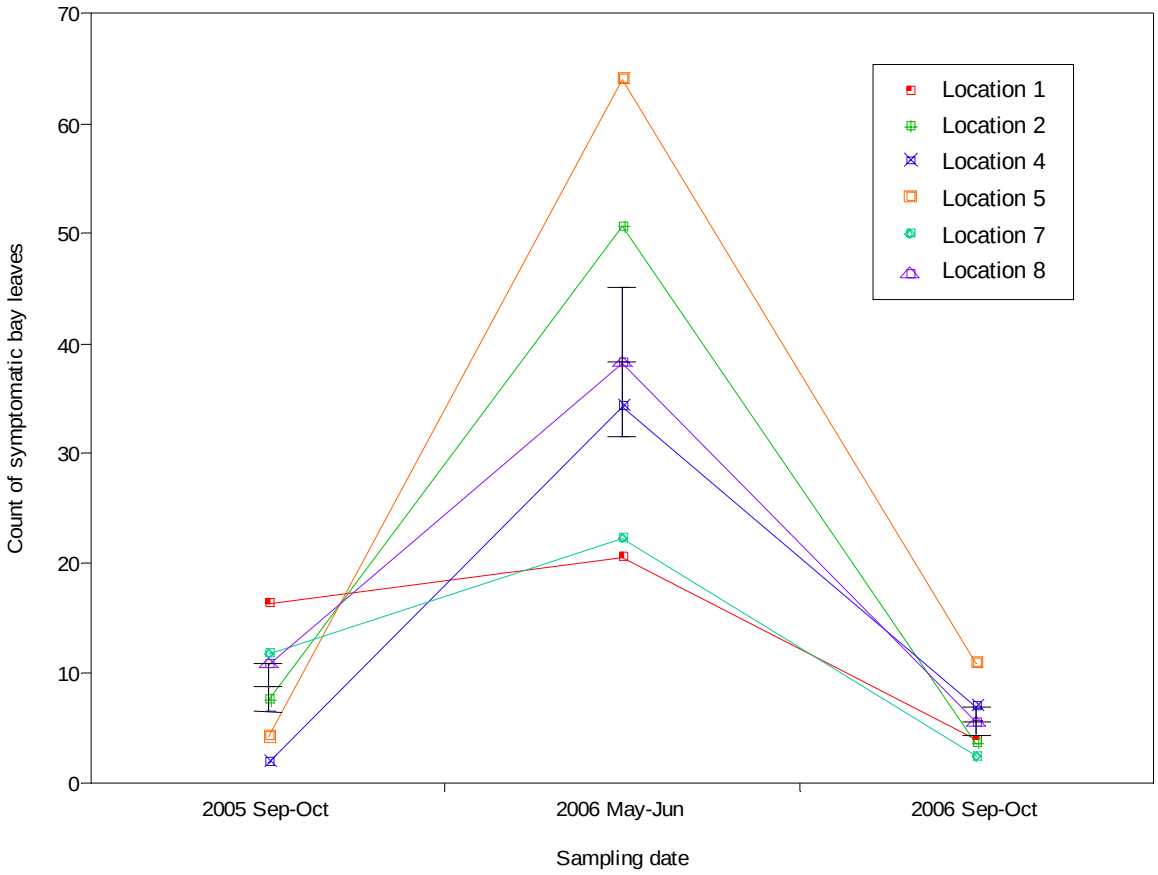

Figure 8. Mean number of infected bay leaves counted in a 45 second search period for bay zones at six locations assessed in September-October 2005 and 2006 and in May-June 2006. Black bars show overall means and standard errors for the sampling interval.

This study is retrospective, and most of the *P. ramorum* infections in cases had developed at least several years prior to 2005; some cases had been symptomatic in 2000. Hence, symptomatic bay leaf counts measured in 2005 and/or 2006 are not likely to be good predictors of disease outcomes unless counts are highly correlated from year to year. However, symptomatic bay leaf counts from individual bay zones made in fall 2005 and fall 2006 were not significantly correlated (n=106, *figure 9*). Furthermore, symptomatic bay leaf counts from fall 2005 were not significantly correlated with counts made in January 2006 (n=15), May-June 2006 (n=80), or July-August 2006 (n=33). Symptomatic bay leaf counts from May-June 2006 and September 2006 were significantly correlated (n=80, $p<0.0001$, $R^2=0.411$ for square root-transformed counts; *figure 10*) as were July-August 2006 and September 2006 counts (n=33, $p=0.0011$, $R^2=0.294$, for square root-transformed counts).

We also calculated an overall average count for all bay zones around each of the 37 coast live oaks included in this portion of the study. These averages are analogous to averages for a plot centered around each oak. As was seen for the correlations on individual bay zones, average counts of symptomatic bay leaves for zones surrounding individual oaks were not correlated between September 2005 and September 2006 (n=37) or between September 2005 and May-June 2006 (n=28), but May-June 2006 counts were significantly correlated with September 2006

counts (p=0.0007, R^2=0.365, n=28 for square root-transformed means of counts). Average symptomatic bay leaf counts for the zones surrounding these trees were not significant predictors of either the binary disease outcome (*P. ramorum* canker present/absent) or the *P. ramorum* canker girdling rank outcome in either recursive partition or logistic regression models.

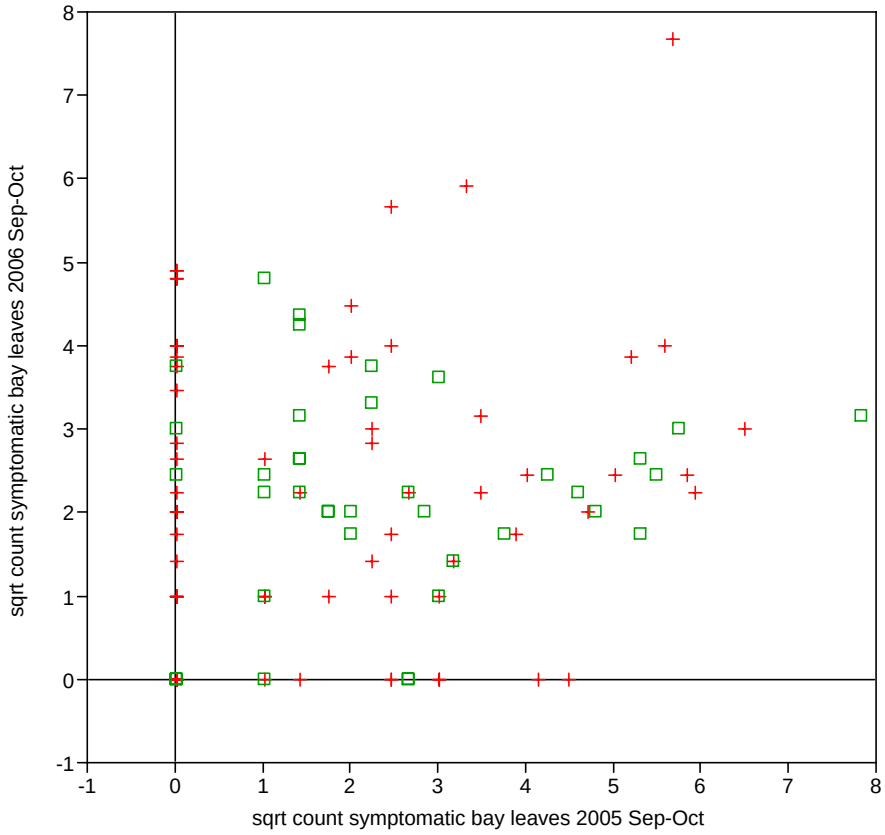

Figure 9. Square root of the number of symptomatic bay leaves counted in 45 seconds in individual bay zones in September-October 2006 and September-October 2006 (n=106). Bay zones near oaks without *P. ramorum* canker symptoms are denoted by green squares (□); zones near oaks with *P. ramorum* canker symptoms are shown with red crosses (+).

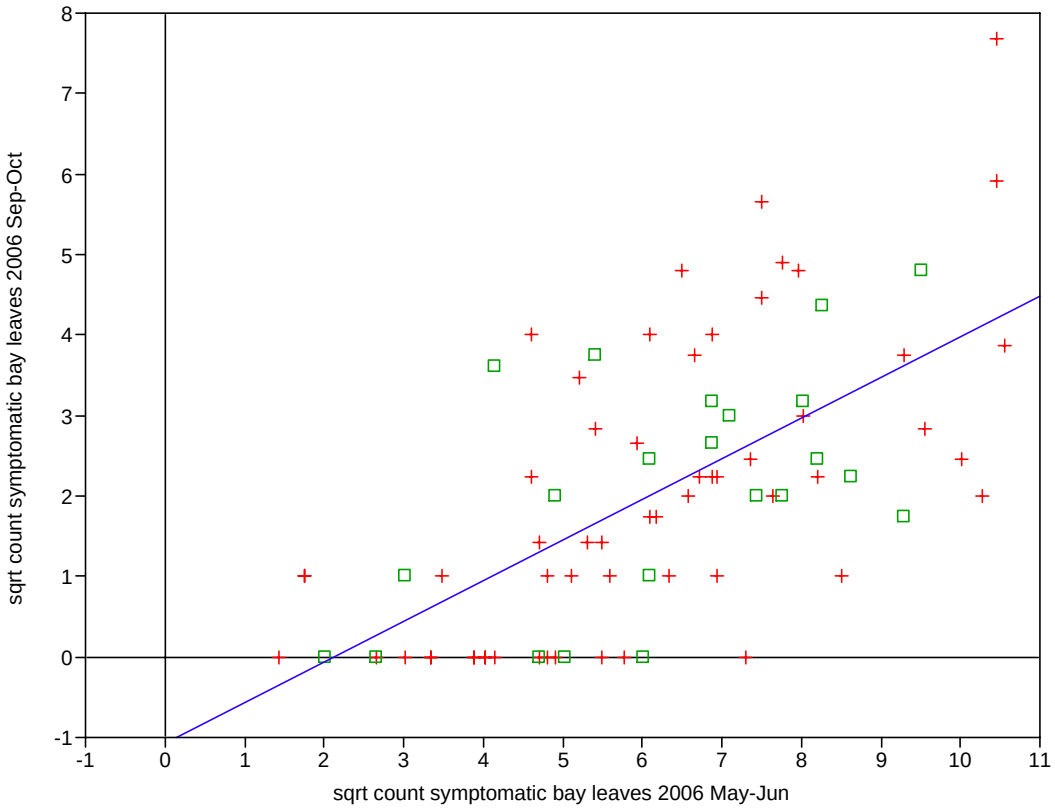

Figure 10. Square root of the number of symptomatic bay leaves counted in 45 seconds in individual bay zones in May-June 2006 and September-October 2006. Bay zones near oaks without *P. ramorum* canker symptoms are denoted by green squares (□); zones near oaks with *P. ramorum* canker symptoms are shown with red crosses (+). Regression line R^2=0.411, p<0.0001.

DISCUSSION

Bay variables that influence disease risk

For the coast live oaks in this study, both the risk of *P. ramorum* infection and the severity of *P. ramorum* canker symptoms increased as the horizontal distance between bay foliage and the oak trunk decreased. The risk of disease, severe symptom development, and mortality were highest in trees with minimum bay foliage-oak trunk distances of about 0 to 1.5 m. This distance is similar to the range of splash dispersal of *P. ramorum* observed by Tjosvold and others (2006) from infected container-grown rhododendrons. Similarly, most propagules of other *Phytophthora* species (Timmer and others 2000) and other pathogens (Grove and Biggs 2006) dispersed by splashing from plant surfaces impact within 1 to 2 m of the inoculum source in the absence of high winds.

If bay foliage is present within about 1.5 m of the oak trunk, *P. ramorum* inoculum can impact the trunk via droplets splashed from infected leaves or water that directly runs off bay foliage and drips on the trunk. These processes are likely to deliver much greater amounts of inoculum to the oak trunk than would be deposited via wind-blown droplets. Davidson and others (2005) showed that the highest numbers of *P. ramorum* propagules dispersed under

natural conditions from infected bay canopy at a forest edge were found directly under bay canopy. Progressively fewer propagules were detected at distances of 5, 10, or 15 m from bay canopy. These greater distances involve dispersal of droplets by wind across unobstructed airspace. Although wind-blown drips and splash droplets can carry inoculum well beyond the 1.5 m range, inoculum concentration falls off rapidly with increasing distance. For splash dispersed inoculum, the decline in inoculum concentration with increasing distance from the source generally follows power law or exponential models (Ahimera and others 2004, Huber and others 1996), which are characterized by steep declines in inoculum concentration within the first meter from the source.

Given that the highest risk and severity of *P. ramorum* canker were associated with short bay foliage-oak trunk distances where inoculum concentrations would be quite high, we conclude that relatively high *P. ramorum* inoculum concentrations are typically required to initiate severe symptom development in coast live oak. This conclusion is further supported by the fact that bay cover within 2.5 m of the oak trunk is a stronger predictor of disease risk and severity in coast live oak than is the minimum bay foliage-oak trunk distance. Because bay cover ratings are related to the amount of bay leaf area present at a given distance from the trunk, bay cover is more directly related to potential levels of inoculum production than is bay foliage-trunk distance.

Severe disease in coast live oak was associated with high levels of bay cover in the distance rings closest to the trunk (*table 5*). Furthermore, the risk of mortality due to *P. ramorum* was highest for those trees that had bay within 0.5 m of the trunk, but was decreased to a similarly low level for all greater minimum bay foliage-oak trunk distance classes (*figure 4*). These results suggest that greater disease severity and more rapid tree decline (*figure 3*) are most likely to develop among trees that are exposed to higher amounts of inoculum and/or are exposed to significant amounts of inoculum repeatedly, as would be the case for trees with little or no clearance from bay and high amounts of bay in the immediate neighborhood of the oak trunk.

Repeated exposure to high numbers of infective *P. ramorum* propagules may overwhelm host defense mechanisms in coast live oak and lead to severe disease development. Lower or more intermittent doses of inoculum may be associated with limited and slower-progressing infections that develop in some coast live oaks (Swiecki and Bernhardt 2005). If this is the case, it may be possibly to slow disease development in oaks that have only a few small cankers by removing bay, which should lessen chances of reinfection in subsequent years and may increase the effectiveness of host defense reactions.

Based on our analyses of the direction of bay canopy relative to the oak trunk, it appears that longer-range dispersal of inoculum associated with wind-driven rain also increases disease risk in coast live oak to some degree. This factor needs to be considered in developing an effective buffer distance between coast live oak and bay canopy. It may be important to increase bay foliage-oak trunk clearance in the direction that winds blow from during spring storm events, generally the south and west for most of northern California.

In contrast, we did not demonstrate a significant relationship between bay canopy height and disease risk in oak. It is possible that bay canopy height is related to disease risk in some fashion, but this relationship was not obvious due to the specific vertical architecture of the canopy in our plots, the correlations between canopy cover and height distribution, and/or the specific height strata that we used to model vertical canopy distribution. It is also possible that within the broad height strata we used, bay canopy height does not affect disease risk. Any possible enhanced inoculum dispersal associated with tall bay canopy could be offset by lower

inoculum production that apparently occurs in the uppermost portions of bay canopies. Alternatively, enhanced interception of inoculum by nonsusceptible plant surfaces (e.g., coast live oak foliage) high in the canopy could reduce the efficiency with which inoculum from high bay canopy is transported to the oak trunk.

Although severe disease and mortality due to *P. ramorum* is most commonly associated with high amounts of bay cover adjacent to the oak trunk, disease sometimes develops in trees that do not fit this profile. This suggests that alternative sources of inoculum may be important in some situations, and/or that some trees are so highly susceptible that small amounts of inoculum can initiate successful and sometimes lethal infections.

Poison oak, which is known to be susceptible to both foliar and stem infections by *P. ramorum* (http://nature.berkeley.edu/comtf/html/host_plant_lists.html) may be an important alternate source of inoculum in some situations, particularly when it climbs into and grows extensively through oak canopies. If poison oak supports even moderate levels of spore production on its leaves and/or twigs, substantial amounts of *P. ramorum* inoculum could be splashed from poison oak in the oak canopy to the oak trunk. The seasonal production of inoculum on poison oak and the potential for variation in inoculum production between different poison oak genotypes remain to be investigated. Nonetheless, from a management perspective, elimination of climbing poison oak vines within or adjacent to susceptible oaks is probably a prudent practice. Aerial poison oak vines can be killed simply by severing the ascending stems, which are typically few in number in well-established plants.

Some apparently anomalous infections do not appear to be directly associated with significant amounts of nearby bay or poison oak. These may represent infections associated with spores produced on other host plants, introduction of propagules via animal vectors, or other sources. It seems likely that oaks with anomalous infections are likely to be highly susceptible genotypes that may be infected when exposed to relatively low levels of inoculum. To date, such anomalous infections appear to occur at relatively low frequencies. We have observed only a few such trees among over 650 coast live oaks in this set of plots.

Without a better understanding of the factors associated with disease in these trees, it is not possible to determine which portion of the tree population is at risk, so the best management actions to protect these trees cannot be specified. However, if other management actions, such as localized bay removal, are implemented, it is possible that the relative importance of these anomalous sources of infection may increase. This possibility emphasizes the need for careful monitoring of disease management studies and projects to provide better information about disease epidemiology in altered stands.

Foliar infection levels in bay

Because disease risk in coast live oak appears to be highly correlated with the level of inoculum produced on bay close to the oak, levels of foliar infection in bay should correlate with disease risk. However, since this is a retrospective study, we were unable to observe *P. ramorum* infection levels in bay that existed at the time the oaks became infected. If bay foliar infection levels within specific patches of bay canopy were highly correlated from year to year, infection levels measured in any given year might still be a useful predictor of *P. ramorum* canker risk. However, our data on bay foliar infection levels failed to show either clear year to year correlations in foliar symptom levels or any significant relationship between foliar symptom levels and disease on adjacent oaks.

Dehiscence of infected bay leaves decreases observed foliar disease levels over time. Based on this and our own field observations in plots over multiple years, we expected that apparent bay infection levels would vary over the season and possibly from year to year. It was not clear, however, to what degree the bay foliar infection levels noted in a given section of bay canopy at any given time would be correlated with infection levels measured at a different time. Our bay foliar symptom counts, spanning a single year from fall 2005 to fall 2006, showed only within-year correlations for the period from peak symptom expression in late spring/early summer to fall of the same year. This indicates that trees with high infection levels in spring/summer tend to have more symptomatic leaves remaining in the fall. However, counts made in the fall 2006 and spring 2006 were not correlated, suggesting that infection levels in the fall do not necessarily predict infection levels in the following spring. Furthermore, we did not detect a significant year to year correlation based on counts made in the fall in two successive years. Hence, it appears that bay foliar infection levels measured in a given year is not likely to be a useful proxy for bay foliar infection levels that developed in previous years.

Nathan Rank and colleagues at Sonoma State University (in press) have shown significant correlations between bay foliar counts for individual trees made in late spring/early summer of 2004 and 2005, when symptom levels were near their maximum. Their data are not directly comparable to ours since we have not yet assessed bay foliar symptoms from spring to spring in two successive years. Furthermore, our assessment methodology differs somewhat from that used by the Sonoma State researchers (e.g., 45 second vs. 90 second counts) and our study locations differ from theirs geographically and with respect to the length of time that *P. ramorum* has been established in the area.

Our data suggest that assessments of bay foliar infection levels are not likely to be useful disease predictors in retrospective studies, but they could be better predictors of disease in oaks in prospective studies. Our data also show that symptom counts need to be made over a sufficiently short time interval to minimize variation due to seasonal loss of symptomatic leaves.

Management considerations

Due to the confounding of several variables, we are only partially able to address the question as to what constitutes a "safe" bay foliage-oak distance with respect to the risk of *P. ramorum* canker. As shown in *figure 4*, *P. ramorum* canker in coast live oak is minimal at bay foliage-oak trunk distances of 10 m or more. Sources of inoculum other than bay may be involved when *P. ramorum*-infected oaks are more than 10 m from bay foliage.

While it is probably possible to prevent nearly all *P. ramorum* infections in coast live oak by clearing all bay within 10 m of the oak trunk, this strategy is probably best suited for protecting a relatively few individual high-value trees at a given site. In many locations, obtaining 10 m of clearance from all susceptible oaks would require nearly complete removal of bay from a stand. Large bays are commonly codominant with oaks and other hardwood species and the canopies are interlocked to varying degrees. Extensive removal or complete eradication of bay from such stands without causing severe damage to oak canopies can be a difficult and expensive proposition. Furthermore, removal of large amounts of bay may not be consistent with other forest management objectives or landowner preferences. For example, bay is an important plant in the traditional culture of a number of Native American tribes in California. Although some management of bay canopy to protect oaks from *P. ramorum* canker may be acceptable, widespread elimination of bay is unlikely to be feasible or acceptable in many mixed hardwood stands.

Although 10 m is a reasonably safe bay foliage-oak distance, it is probably not the minimum safe distance. Figures 2, 3, and 4 show that some coast live oaks that have no separation from bay have remained asymptomatic through 2006 in areas where *P. ramorum* was well established in 2000. Clearly, if oaks possess some level of resistance and/or inoculum production on bay is limited, disease may not develop even if the bay foliage-oak distance is zero.

The challenge is to determine a bay foliage-oak distance at which the risk of disease development in susceptible oaks is low, and the amount of bay removed is minimized. For purposes of management, it is important to remember that the area to be cleared of bay foliage increases as a function of the square of the clearance radius. Hence, bay foliage-oak trunk clearances of 2.5, 5, and 10 m correspond to cleared areas of about 20, 79, and 314 m^2, respectively, around the oak trunk. Because bay foliage within a given distance ring may arise from trees rooted beyond the ring, the actual area to be manipulated will generally be greater than these nominal areas.

Considering both disease incidence and severity, bay foliage within about 1.5 m of the oak trunk poses the greatest disease risk to coast live oak. Removal of bay foliage from this zone should substantially decrease the risk of both disease and mortality due to *P. ramorum*. However, based on our analyses of bay cover, we propose that the minimum clearance between the bay foliage and the oak trunk should be 2.5 m. Whether it is necessary to extend the zone of bay foliage clearance beyond this distance cannot be completely addressed by analyses of our current data set. The data set lacks examples of situations where no bay cover is found within 2.5 m of the oak trunk but high bay cover levels are found beyond this point simply because this type of canopy juxtaposition does not occur in the stands we studied. Analyses related to the effects of the oak trunk watershed and directional effects suggest that bay foliage-oak trunk clearances greater than 2.5 m may be desirable to reduce disease risk to acceptably low levels in large-canopied oaks or when substantial amounts of bay foliage are present in the downwind direction at distances between 2.5 and 10 m from the oak trunk.

We have recently initiated a PSW-funded study in which we are creating modified bay environments by selective bay removal and pruning around individual oaks. Based on the analyses presented here, we are implementing the following selective bay removal prescription around individual oaks, which we believe represents a reasonable balance between minimizing disease risk and the cost of bay removal.

- Establish a minimum of 2.5 m of horizontal clearance between bay foliage and the oak trunk.
- Small understory bay seedlings and saplings should be removed for a distance of at least 2 m from around the oak trunk.
- Where feasible with a minimum of additional bay removal, extend the minimum clearance to 5 m.
- Within the 2.5 to 5 m distance range, emphasize additional clearance to the south and west of the oak (i.e., the normal storm wind direction).
- Where complete bay removal is difficult to obtain in the 2.5 to 5 m distance range, remove low bay canopy by pruning low branches.
- Cut stems of poison oak climbing into the canopy of the oak or any adjacent tree which supports canopy-level poison oak within 2.5 m (horizontal distance) of the oak trunk.

Implementing this prescription around an individual oak should significantly reduce the likelihood that the oak will develop or be killed by *P. ramorum* canker, but it may not be sufficient to completely prevent disease in all treated trees. In addition, it may not be feasible or

economical to implement this prescription for all trees in a stand, especially where very large bays are present. This prescription is most appropriate for reducing potential disease impacts in stands where adequate clearances can be established around asymptomatic oaks by removing and/or pruning relatively small-diameter bays. The new study we have initiated will evaluate the efficacy of this prescription.

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