Increasing Distance from California Bay Laurel Reduces the Risk and Severity of *Phytophthora ramorum* Canker in Coast Live Oak [1]

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Abstract

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 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. Bay cover within 2.5 m of the trunk was a stronger predictor of disease risk and severity than the minimum bay-trunk distance. These results suggest that removing bay from within 2.5 m of the trunk of a susceptible oak will greatly reduce, but not eliminate, the risk of disease. For some oaks with *P. ramorum* canker, the presence of disease symptoms could not be readily explained by proximity to bay, but 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 inoculum. 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 leaves in fall 2005 were not correlated with counts from the same trees in either spring/summer 2006 or fall 2006, but spring/summer 2006 counts were correlated with fall 2006 counts.

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

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(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), which 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 150 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 are significantly associated with high disease risk. 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 and sporulates abundantly on bay foliage, 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. This study was undertaken to determine whether it is possible to specify a bay foliage-oak distance beyond which the risk of disease is reduced to acceptably low levels.

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 SOD (Swiecki and Bernhardt 2001). Plots were established in mixed hardwood forests where *P. ramorum* symptoms were prevalent on coast live oak. The study locations were in Marin (nine locations) and Napa (one location) counties.

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. 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.

Phytophthora 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. *Phytophthora ramorum* was the only *Phytophthora* sp. recovered from cankers at all of 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. Cankers at different heights along the stem increase the girdling rating 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

Coast live oaks were selected from the study plots to represent cases (trees with SOD symptoms) or controls (trees lacking SOD symptoms). 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 in the study plots, except for those with ambiguous disease symptoms, were selected as cases.

Potential controls included all trees in the plots that were free of *P. ramorum* canker symptoms over the previous 7 years. In selecting controls, we 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 sky exposed canopy values), had very low ratings for unweathered tissue in bark fissures, and/or were in severe decline due to agents other than *P. ramorum*.

Trees were selected based on existing data sets to avoid potential bias. Preselected trees were rejected in the field only if major structural failures had occurred in either the selected oak or nearby bays and bay neighborhood prior to tree failure could not be reliably assessed. In all, 247 coast live oak trees were included in this study: 36 percent were asymptomatic, 16 percent had early symptoms, 23 percent had late symptoms, and 24 percent had been killed by *P. ramorum*.

Evaluation of California Bay Laurel Around Oaks

We estimated bay cover within concentric rings centered around each oak tree included in the study. The rings were based on the following distances from the oak

trunk: <2.5 m, 2.5 to 5 m, 5 to 10 m, and 10 to 20 m. Each distance ring was divided into four 90 degree arcs centered at each of the cardinal compass directions. 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 was noted. Bay cover was assessed between October 2005 and July 2006.

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. A hand-held Leica Disto™ laser was used to measure distance to the oak trunk. We also noted the presence, location, and amount of other foliar hosts of *P. ramorum* in the immediate neighborhood that might serve as alternative sources of inoculum, such as tanoak (*Lithocarpus densiflorus*) or poison oak (*Toxicodendron diversilobum*).

We assessed bay foliar infection level in 106 patches of bay canopy around 37 of the coast live oaks in the study. Contiguous patches of bay foliage, arising from either a single bay stem (47 zones) or multiple (2 to 19) stems were mapped based on distance and azimuth relative to the oak. We used 45-second timed counts of symptomatic leaves to assess foliar disease levels in the mapped bay zones in September and October 2005. Recounts of foliar symptoms in the same bay zones were made between late May and early August 2006 and again in September 2006; a small subsample 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 Inc., Cary NC) for data analysis. Unless otherwise indicated, effects or differences are referred to as significant if $p \leq 0.05$. 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. The square root transformation was applied to bay foliar symptom counts prior to analysis.

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, in other words, 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 six of the coast live oak trees in the study (2.4 percent) did not have bay present within 20 m of the trunk. Figure 1 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 in the controls (mean 5.7 m, median 3.3 m) than in the cases (mean 1.3 m, median 0 m).

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. All of the cases with bay foliage-oak trunk distances greater than 10 m either had extensive amounts of poison oak climbing in their canopies (fig. 1) or were within 7.5 m of such trees.

We used recursive partition analysis to more closely examine the relationship between the minimum distance from bay foliage and the presence of *P. ramorum*

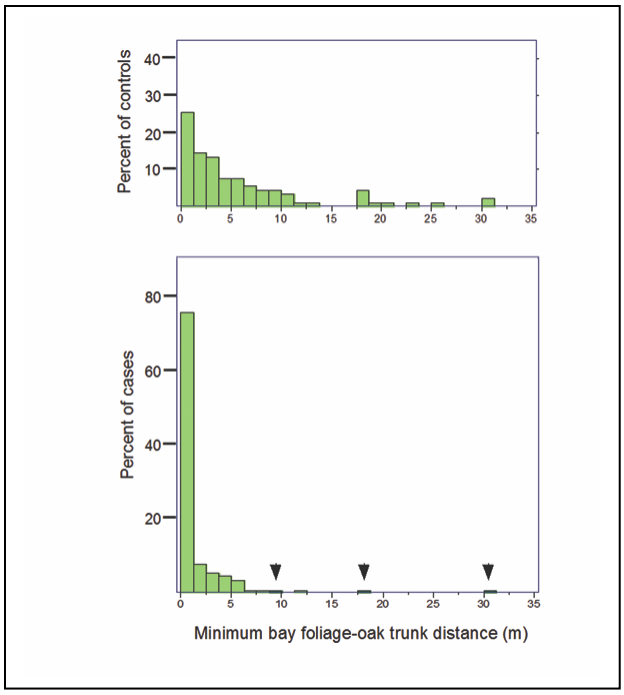

Figure 1—Minimum distance (m) between nearest bay foliage and coast live oak trunks for oaks without (top graph, n=90) or with (bottom graph, n=157) *P. ramorum* canker symptoms. Arrows in the bottom graph indicate three trees with extensive poison oak growing in the canopy (shaded bars).

canker symptoms in coast live oak. The greatest difference in both percent infection and in average *P. ramorum* canker girdling rank was achieved by partitioning at a minimum bay foliage-oak trunk distance of 1.5 m. 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.

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*. Based on a single variable logistic regression model (model $p<0.0001$) for 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 2 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. The only symptomatic oak in the >10 m minimum bay foliage-oak trunk distance class (n = 15) had a minimum bay foliage-oak trunk distance of 12 m and had canopy-level poison oak present 7.5 m from the trunk. Both the incidence of *P. ramorum* canker and average girdling rank decreased as the minimum bay foliage-oak trunk distance increased, but *P. ramorum*-related mortality did not change significantly for distance classes beyond 0 m (fig. 2).

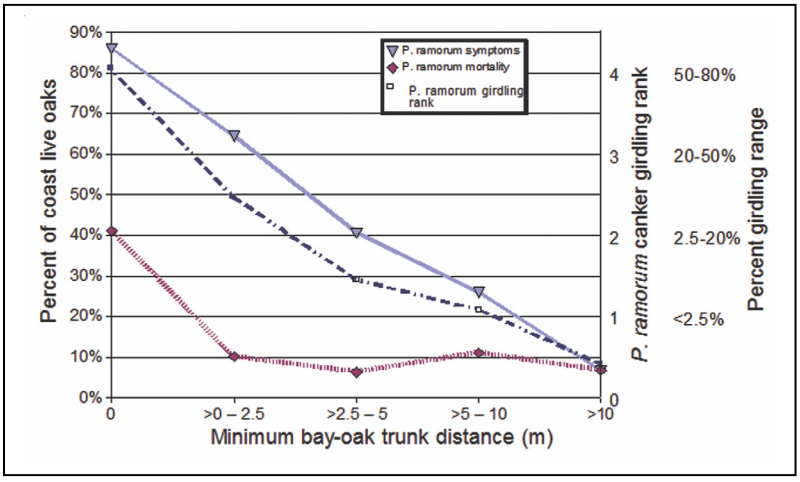

Figure 2—Percent of coast live oaks with *P. ramorum* symptoms and mortality due to *P. ramorum* (left scale), and average *P. ramorum* girdling rating (right scale) by minimum bay foliage-oak trunk distance class. Sample sizes for the distance classes from left to right are 107, 59, 32, 27, and 15. Trees with poison oak growing in the canopy or in adjacent trees within 1.5 m of the oak trunk are omitted.

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 distance ring show significant positive correlations with all other rings, although the highest correlations are seen between adjacent distance rings (table 1). In addition, minimum bay foliage-oak trunk distance is negatively correlated with bay cover ratings for each of the distance rings, although the strongest correlations are seen for the distance zones closest to the oak trunk (table 1). These correlations are related to the overall spatial distribution of bays around coast live oaks and the fact that many of the bay canopies were large enough to span multiple distance rings.

Table 1—Spearman's rho rank correlation coefficients for pairwise comparisons between bay distance and cover variables. All correlations shown are significant at p<0.0001

	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 confounding of some variables: combinations that are needed to differentiate between effects of certain variables either are lacking or represented by too few points to be statistically meaningful. In particular, 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 this data set.

As shown in figure 3, the average bay cover ratings for 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 (fig. 3), 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 1).

We used recursive partitioning to investigate the relative ability of bay cover variables to predict disease outcomes. *P. ramorum* canker girdling rank, which takes both disease incidence and disease severity into account, was used as the disease outcome. Oaks with canopy-level poison oak within 1.5 m of the trunk were omitted, 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 1, the initial partition was based on the average bay cover rating within 2.5 m of the oak trunk (table 2). 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 2).

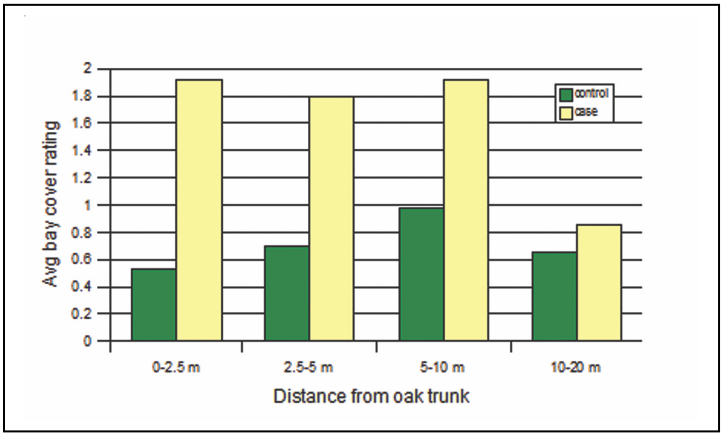

Figure 3—Average California bay cover ratings in distance rings around coast live oak controls and cases. For distance rings from 0 to 10 m, ratings were made using the quartile scale (maximum rating=4); for the 10-20 m ring, only presence (1) or absence (0) was scored. All differences between cases 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).

Table 2—Recursive partition model for the *P. ramorum* girdling rank disease outcome. Candidate predictors were the four variables shown in Table 1. 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 <2.5 m *< 0.775*	bay cover 5-10 m *<0.75*		37	0.38	13.5
	bay cover 5-10 m *≥ 0.75*		72	1.81	45.8
bay cover <2.5 m *≥ 0.775*	bay cover 2.5-5 m *≥ 1.775*		86	4.27	90.7
	bay cover 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

This model indicates 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% 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 the farther zones.

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 generally shaded lower portions of the canopy than in the more exposed 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 4 shows how bay foliar symptom counts varied through a single year for 12 bay zones at one location. The general pattern of seasonal variation was 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, and these leaves had dropped by September. 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 (fig. 4).

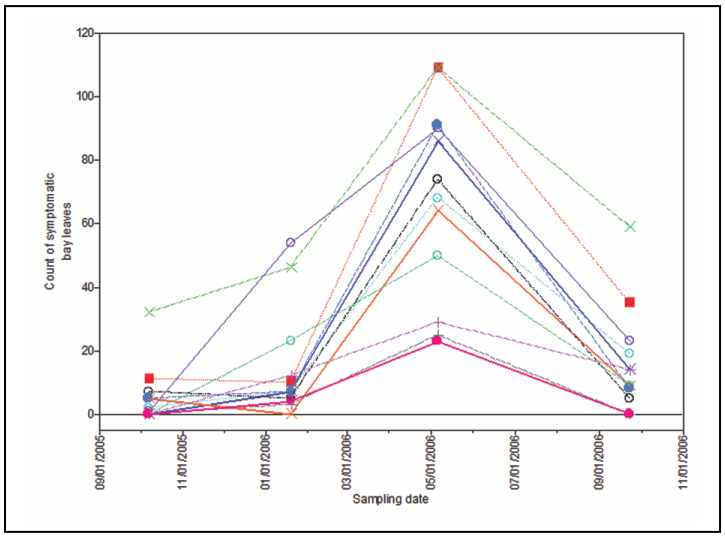

Figure 4—Number of infected bay leaves counted in a 45 second search period for 12 bay zones at location 5 assessed on four dates between September 2005 and October 2006. Connected points represent counts in the same bay zone. Within each graph, zones with the same symbol type are located around the same oak.

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 $p<0.0001$). Counts did not differ significantly based on the number of trees within a bay zone.

Symptomatic bay leaf counts of individual bay zones made in fall 2005 were not significantly correlated with counts made in fall 2006 (n=106), January 2006 (n=15), May-June 2006 (n=80), or July-August 2006 (n=33). However, symptomatic bay leaf counts from May-June 2006 and September 2006 were significantly correlated (n=80, $p<0.0001$, $R^2=0.411$; square root-transformed counts) as were July-August 2006 and September 2006 counts (n=33, $p=0.0011$, $R^2=0.294$; 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). The average symptomatic bay leaf counts for the zones surrounding these oaks were not significant predictors of either the binary disease outcome or the *P. ramorum* canker girdling rank outcome.

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 at bay foliage-oak trunk distances between 0 and 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. 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.

Where 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. 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-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 most coast live oaks. 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 bay leaf area, bay cover is more directly related to potential levels of inoculum production than is bay foliage-trunk distance.

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, such as poison oak, may be important in some situations. Alternatively, some trees may be so highly susceptible to *P. ramorum* infection that small amounts of inoculum can initiate successful and sometimes lethal infections.

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, we expect that levels of foliar infection in bay would correlate with disease risk in a prospective study that examines the initiation of disease in healthy oaks. 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.

Bay foliar infection levels in patches of bay foliage were correlated within a single year, and showed a decline in infection level over time as symptomatic leaves dropped. This indicates that foliar symptom counts need to be made over a sufficiently short time interval to minimize variation due to seasonal loss of symptomatic leaves.

Rank and others (these proceedings) have shown significant correlations between bay foliar counts for individual trees made in late spring/early summer of 2004 and 2005, when symptom levels are near their maximum. Their data are not directly comparable to ours due to differences in assessment methodology, characteristics of the study locations, and timing of the assessments. Although foliar infection levels in patches of bay foliage can be correlated over relatively short time intervals, the correlation is likely to break down over successively longer time intervals. Especially in stands that have high levels of *P. ramorum*-related mortality, the change in microclimate over time due to the loss of tree canopy could result in substantial changes in the potential for bay foliar infection.

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 SOD. 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, which may not be financially feasible or consistent with other forest management objectives or landowner preferences.

If both disease incidence and severity are considered, bay foliage located within about 2.5 m of the trunk pose the greatest risk to coast live oak. At minimum, removal of bay foliage from this zone should substantially decrease the risk of both disease and mortality due to *P. ramorum*. Bay present at distances between 2.5 and 10 m may also increase disease risk, especially if it is located in the direction of prevailing storm winds (Swiecki and Bernhardt 2007). However, our current data set lacks examples of situations where the no bay cover is found within 2.5 m of the oak trunk but high bay cover levels are found beyond this point, so it is not possible to quantify the disease risk associated with bay foliage that is exclusively found in the 2.5 to 10 m range. For purposes of management, each doubling of clearance distance quadruples the area that needs to be cleared, so incremental reductions in disease risk need to be weighed against the increased cost of developing additional clearance.

We recently initiated a study in which selective bay removal and pruning is being used to create localized areas free of bay foliage near oak trunks. Based on the analyses presented here, we used the following prescription for selective bay removal around individual oaks. We believe this 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, including removal of small understory bay seedlings and saplings within at least 2 m of the oak trunk.

- Where feasible with a minimum of additional bay removal, extend the clearance to 5 m, especially toward the normal storm wind direction (Swiecki and Bernhardt 2007)

- Where it is difficult to completely remove bay in the 2.5 to 5 m distance range, remove low bay canopy by pruning low branches.

- Cut stems of poison oak that are climbing into the canopy of the oak or adjacent trees to provide at least 2.5 m of horizontal distance between canopy-level poison oak and 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 may not be sufficient to completely prevent disease in all treated trees. In addition, it may not be feasible 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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