Evaluation of Stem Water Potential and Other Tree and Stand Variables as Risk Factors for *Phytophthora ramorum* Canker Development in Coast Live Oak[1]

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Abstract

We conducted a case-control study to examine the role of water stress and various other factors on the development of *Phytophthora ramorum* cankers in symptomatic (case) and symptomless (control) coast live oak (*Quercus agrifolia*) and tanoak (*Lithocarpus densiflorus*). Midday stem water potential (SWP) in *Q. agrifolia* subject trees ranged from –0.25 to –3.1 megapascals (MPa). SWP was higher (indicating lower water stress) in cases than controls. Other variables significantly associated with disease in *Q. agrifolia* included tree canopy dieback, the amount of the canopy shaded by other trees, and the density of California bay in the plot. Cases were also significantly associated with counts of other plot trees with early *P. ramorum* canker symptoms, indicating that spatial aggregation of diseased trees occurs on the scale of the plot area (0.02 hectare). For *Q. agrifolia*, the incidence of decline and death related to *P. ramorum* in case plots was almost equal to rates of decline and recent mortality due to other agents. For *L. densiflorus*, decline and mortality related to *P. ramorum* was far more common than decline and mortality due to other agents.

Introduction

Phytophthora ramorum has been associated with elevated levels of mortality in tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and California black oak (*Q. kelloggii*) trees in a number of California coastal counties over the past few years (Garbelotto and others 2001, Rizzo and others 2002b). Early symptoms of the disease, which is commonly referred to as "sudden oak death," consist of bark cankers which typically produce a brown exudate. The sapwood-decaying fungus *Hypoxylon thouarsianum*, oak bark beetles (*Pseudopityophthorus* spp.), and ambrosia beetles (*Monarthrum* spp.) are commonly associated with *P. ramorum*-infected trees in later stages of decline. These agents also attack declining trees or branches that are not infected with *P. ramorum*

Very little was known about the epidemiology of this disease at the time this study was initiated. Water stress had been considered as a possible risk factor for disease development because affected trees are commonly found in highly competitive situations. Water stress occurring either before or after infection has been

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shown to increase the susceptibility of various plants to *Phytophthora* spp. (Sinclair and others 1987) and is also a predisposing factor for *Hypoxylon* infection (Sinclair and others 1987) and beetle attack.

To examine the role of water stress and various site and stand attributes on the development of *P. ramorum* bole cankers, we conducted a case-control study in areas where the disease was common. Case-control studies are designed to examine how past (retrospective) factors are related to the current health of individuals. In this study, we investigated whether water stress and various other tree and stand factors are risk factors for the early phase of the disease, i.e., the bleeding bark cankers that are associated with *P. ramorum* infections. Current tree and stand conditions were used as indicators of past conditions at the site.

Midday stem water potential (McCutchan and Shackel 1992) can be considered an indicator of preexisting water stress levels if *P. ramorum* infection does not substantially affect water transport or tree water potential. *P. ramorum* cankers affect the bark but generally do not affect substantial amounts of xylem tissue (Garbelotto and others 2001, Rizzo and others 2002b), at least in trees in early stages of disease that we selected as cases. Therefore, we assumed that case trees were not likely to exhibit changes in stem water potential brought about by infection with *P. ramorum*.

Due to limitations of time and funding, we concentrated our efforts on one host species, coast live oak. For comparative purposes, we also collected a limited amount of data on tanoak. This study addresses only the trunk cankers caused by *P. ramorum* on coast live oak, black oak, and tanoak. Foliar infections on tanoak, California bay *(Umbellularia californica),* and various hosts were not known at the time that this research was conducted

Methods

In this study, cases were defined as live trees with evident *P. ramorum* cankers but lacking fruiting of *H. thouarsianum* and evidence of beetle boring on the bole that is typical of *P. ramorum*-infected trees in late stages of decline. Hence, cases are characterized as having early disease symptoms only. Controls were trees lacking evident *P. ramorum* cankers, although it is possible that some controls could have been infected but still asymptomatic trees. Cases and controls were sampled within areas where the disease syndrome is prevalent. This reduces the likelihood that controls simply represent trees that have not been exposed to any *P. ramorum* inoculum, although this possibility cannot be ruled out for all controls.

Study Site Selection

During September 2000, we established plots and collected data at 12 study locations *(table 1)*. Study sites were selected on the basis of appropriate vegetation type, the presence of case and control coast live oak or tanoak trees in the study area, and absence of recent disturbances that might affect tree health.

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

Location number	Location	County	Number of plots	Subject tree species
1	Marin Municipal Water District (MMWD) watershed - Azalea Hill area	Marin	12	coast live oak
2	MMWD-Pumpkin Ridge south	Marin	16	coast live oak
3	MMWD-Pumpkin Ridge north	Marin	11	coast live oak
4	MMWD-Phoenix Lake area	Marin	11	coast live oak
5	China Camp SP - Miwok Meadows area	Marin	16	coast live oak
6	China Camp SP - SE Buckeye Point area	Marin	12	coast live oak
7	Woodacre (Private land)	Marin	12	coast live oak
8	Lucas Valley (Private land)	Marin	12	coast live oak
9	Muir Woods NM / Mt. Tamalpias SP	Marin	10	tanoak
10	Wall Road (Private land)	Napa	13	coast live oak
11	Marin County Open Space land, Novato	Marin	13	coast live oak
12	Jack London SP	Sonoma	12	tanoak

Plot Selection

At each study location, we collected data on 10 to 16 circular 8 m radius (0.02 ha) fixed area plots, each of which was centered around a subject case or control tree. The number of plots per location was limited by the time constraints associated with stem water potential measurements and terrain. At each location, we entered into the stand, selected a random bearing and proceeded along that bearing to search for the nearest case or control tree. We attempted to space adjacent tree-centered plots approximately 25 m apart, but actual interplot spacing varied with vegetation and terrain. Distances between two adjacent plots ranged from 16.1 to 48.8 m (average 26.1 m). Plots at the various locations were distributed over areas ranging between about 0.75 and 1.6 ha. We attempted to alternate case and control plots, but if the alternate subject tree type did not exist within a 4 to 8 m search radius of the target point, the same type was repeated. We collected data on 75 control and 53 case plots for coast live oak, and 13 control and 9 case plots for tanoak. Overall, controls outnumber cases because trees showing early *P. ramorum* symptoms were often difficult to locate when following the sampling pattern we used.

Stem Water Potential Determinations

We collected midday stem water potential (SWP) readings on the center subject tree in each plot during the 2 hour peak midday period (1:00-3:00 P.M. PDT) following methods outlined by Shackel (2000). On each tree, two leaves or shoot tips with several leaves were sealed inside clear plastic bags and overbagged with larger opaque reflective plastic bags. We selected leaves that arose directly from the trunk, main branches near the trunk, or basal sprouts. Bags were left in place for 2 or more hours to allow leaf water potential to equilibrate to that of the subtending stem. Each leaf or shoot tip was then excised and placed into the pressure chamber while still sealed in the clear plastic bag. One or two SWP determinations were made per tree. In general, two valid SWP measurements from a single tree were within 0.05 to 0.1

megapascals (MPa) of each other. SWP measurements were made with a pump-up pressure chamber (PMS Instrument Co., Corvallis OR) fitted with a 4 inch diameter 40 bar (0.4 MPa) gauge with 1percent accuracy.

To estimate vapor pressure deficit (VPD) during the period that SWP readings were made, we recorded the minimum and maximum temperature and relative humidity values using a portable electronic thermohygrometer (Mannix TH Pen, model PTH8708). The thermohygrometer was placed in a ventilated shelter mounted on a mast and was positioned near the upper portion of the tree canopy layer during the observation period. VPD was calculated from the average of the recorded minimum and maximum temperature and relative humidity values.

Additional Tree and Plot Variables

In addition to SWP determinations, subject trees were rated for origin class (seed or coppice); stem count; stem diameter(s) at 137 cm diameter at breast height (DBH); and amount of crown exposure to overhead sunlight (using 0-6 scale described below). Counts of *P. ramorum* cankers were estimated from bleeding areas and girdling by *P. ramorum* cankers was rated by estimating the percent bole circumference girdled as if all cankered areas were projected on same cross section. The horizontal extent of cankers was estimated from the distribution of bleeding areas, alterations in bark appearance, and some limited chipping of outer bark to expose canker margins. We also assessed overall (chronic) canopy thinning (none, slight, definite), the presence of epicormic branches (none, few, numerous), and recent canopy dieback (0-6 scale below). Although assessment of actual levels of decay in standing trees is problematic, we rated decay impact (none, low, moderate, high) by assessing the probability that existing decay would have a significant negative impact on tree health or survival. The assessment of decay impact was based on the type(s) of decay present, location of decay within the tree, and the estimated extent of decay as rated by a trained observer. Additional details of the tree health rating methods are presented in Swiecki and Bernhardt (2001).

Plot variables recorded included plot slope and aspect; total basal area; tree counts by species; plot canopy cover; woody understory cover; disease status or recent mortality (estimated within the preceding 10 years) of other coast live oak, black oak, and tanoak trees in the plot; counts of regeneration of these 3 host species; and the presence of other pathogens of these species. Coast live oak, black oak, and tanoak trees other than the center subject tree are referred to as plot trees in this paper.

Other than basal area, which was assessed using a 5 m^2/ha BAF reticle, plot-related variables were assessed on an 8 m radius fixed-area plot centered at the subject tree. The measured variables were also used to calculate a number of additional variables for various analyses. We used plot slope, aspect, elevation, and latitude data to calculate the total annual insolation (solar radiation) that the plot would receive in the absence of shading from vegetation or nearby landforms. Insolation was calculated using a program developed by Dr. Tom Rumsey (Department of Biological and Agricultural Engineering, University of California, Davis) based on the Hottel estimation model (Duffie and Beckman 1991)

We used the following arcsine-transformed percentage scale for most ocular estimates of percentages: 0= not seen, 1= less than 2.5 percent, 2= 2.5 percent to 19

percent, 3= 20 percent to 49 percent, 4= 50 percent to 79 percent, 5= 80 percent to 97.4 percent, 6= more than 97.5 percent. *P. ramorum* girdling rank was derived by grouping percent girdling estimates into the following classes: 0= 0 percent, 1= 1 percent to 19 percent, 2= 20 percent to 39 percent, 3= 40 percent to 59 percent, 4= >59 percent.

Statistical Analyses

We used JMP® statistical software version 4.0.4 (SAS Inc., Cary NC) for data analysis.[3] Unless otherwise indicated, effects or differences are referred to as significant if $P \leq 0.05$. We fitted logistic regression models to the data to examine the effects of factors on the binary disease outcome (subject tree is diseased, i.e., a case). Although we constructed logistic regression models for both tanoak and coast live oak, only the latter models are reported here.

We screened possible predictor variables using univariate logistic regressions, examined correlations between predictor variables, and checked predictor variable distributions to ensure that models were not overly influenced by a few outlying observations. We developed multivariate models using a stepwise procedure. Factors were generally considered for entry into the multivariate models if odds ratios from univariate models were significant at $P \leq 0.10$. The significance level of each factor reported in the final models should be interpreted as if it were the last factor added to the model.

We used linear regression, analysis of variance, and analysis of covariance to test for associations between continuous outcomes (e.g., SWP) and continuous or categorical predictor variables. We also used analysis of variance (F-tests) or t-tests to test whether mean levels of continuous variables differed between cases and controls.

Results

Disease Levels within Plots

Overall densities of coast live oak, black oak, and tanoak did not differ between plots in which the subject tree was a case (case plots) and plots in which the subject tree was a control (control plots). However, plot trees with *P. ramorum* cankers were more numerous in case plots than control plots *(table 2)*. In a two-way analysis of variance on percent *P. ramorum* infection of plot trees (arcsine transformed data), plot type (case/control) was significant at P=0.01, species was significant at P=0.058, and the interaction term was not significant.

In contrast, rates of decline and death of coast live oak, black oak, and tanoak due to agents other than *P. ramorum (table 2)* did not differ significantly between case and control plots. For coast live oak in case plots, the percentage of trees declining or recently killed by other agents (33 percent) was not significantly different than the total percentage of trees affected by *P. ramorum* (25 percent). For tanoak, the percentage of trees affected by *P. ramorum* in case plots (56 percent) was far in excess of the background decline and mortality associated with other agents (6

[3] Mention of trade names or products is for information only and does not imply endorsement by the U.S. Department of Agriculture.

percent). Levels of *P. ramorum*-related mortality were also much higher in tanoak than in live oak *(table 2)*.

Table 2—*Incidence of symptomatic trees and recent (10 year) tree mortality associated with* P. ramorum *or other agents in study plots. Totals exclude symptoms on the plot subject tree.*

Plot type	Species	Number of trees	Due to *P. ramorum*			Due to other agents	
			Early symptoms	Late symptoms	Dead	Severe decline	Dead
Control	Live oak	321	7.2 pct	4.0 pct	2.8 pct	17.4 pct	6.9 pct
	Tanoak	93	9.7 pct	2.2 pct	14.0 pct	4.3 pct	5.4 pct
	Black oak	7	14.3 pct	14.3 pct	0 pct	28.6 pct	0 pct
Case	Live oak	251	14.7 pct	4.8 pct	6.0 pct	27.1 pct	6.0 pct
	Tanoak	54	29.6 pct	3.7 pct	22.2 pct	5.6 pct	0 pct
	Black oak	11	18.2 pct	0 pct	0 pct	27.3 pct	36.4 pct

Stem Water Potential

SWP measurements varied widely among subject trees within locations. We developed an analysis of covariance model for coast live oak that accounted for about half of the variation in SWP *(table 3)*. SWP was negatively correlated with insolation and the percent of the tree canopy exposed to direct overhead sunlight (sky-exposed canopy). These results indicate that subject tree SWP is higher (i.e., water stress is lower) in plots that receive lower amounts of solar radiation (e.g., north-facing slopes) and in trees with heavily shaded canopies. SWP was also negatively correlated with VPD overall, but further analysis showed that the correlation was significant only among trees with more than 50 percent sky-exposed canopy (interaction term in *table 3*).

Table 3—*Analysis of covariance model for midday stem water potential (MPa) of coast live oak.*

Source	DF	F Ratio	Prob>F	Adjusted R^2	N
Overall model	6	21.67	<0.0001	0.517	117

Model terms	DF	F Ratio	Prob>F	Parameter estimate
Vapor pressure deficit (KPa)	1	5.06	0.0026	-0.101
Sky-exposed canopy	1	37.45	<0.0001	-0.172
Annual insolation (MJ/m^2)	1	10.33	0.0017	-0.000153
P. ramorum girdling rank	1	7.59	0.0069	0.0930
Decay impact rating	1	6.24	0.0140	0.120
Interaction: Sky-exposed canopy >50 pct [true] × (VPD-daily mean VPD)	1	5.58	0.0199	-0.108
Intercept				0.0920

SWP was also correlated with two tree health variables *(table 3)*. Variables describing *P. ramorum* presence or severity (canker count, percent girdling, girdling rank, case/control) were positively correlated with SWP. Girdling rank was the most highly significant of these variables and was included in the final model *(table 3)*.

Water stress, as measured by SWP, was lower in cases than controls and the severity of *P. ramorum* girdling generally increased as water stress decreased. This result is consistent with our assumption that early stages of *P. ramorum* infection seen in cases would not adversely affect SWP. The positive association between disease and high SWP suggests that, after adjusting for other factors in the model, trees located in relatively moist areas are at higher risk for disease than those located in drier sites

SWP was also positively correlated with decay impact ratings in the subject tree *(table 3)*. Hence, the development of wood decay in *Q. agrifolia* also appears to be favored by moist sites. However, other tree condition variables, including canopy thinning and dieback, were not correlated with SWP. This suggests that SWP is not elevated in diseased trees simply due to a reduction in the amount of transpiring leaf area.

Because SWP readings were taken on different days, we needed to adjust the observed SWP readings to account for the effect of VPD in order to create valid SWP variables for use in logistic regression models. Two variables successfully removed the effect of VPD on SWP. Of these the difference from SWP_{max} (maximum SWP from all trees on a given date – tree SWP) was more highly significant in multivariate models (discussed below) although the likelihood ratio for this variable was significant only at P=0.0533 in univariate logistic regression models for the case outcome.

Other Variables Associated with Occurrence of P. ramorum *Canker*

For both coast live oak and tanoak, the risk of *P. ramorum* canker in the subject tree was elevated if other symptomatic trees were present in the plot (univariate logistic regression, likelihood ratio P=0.0254). For coast live oak plots, the risk of early disease symptoms in a subject tree increased with the number of other plot trees showing early *P. ramorum* canker symptoms, i.e., bleeding but no evidence of beetle attack or *H. thouarsianum* fruiting (likelihood ratio P=0.0145). However, the number of plot trees with late *P. ramorum* canker symptoms and/or dead trees with *P. ramorum* canker symptoms were not significant predictors of disease in the subject tree. Furthermore, the number of plot trees showing late disease symptoms was not correlated with the number of plot trees showing early disease symptoms. Similar patterns were observed in tanoak plots

Evidence of bark and/or ambrosia beetle damage and fruiting bodies (stromata) of *H. thouarsianum* were found in 63 percent of coast live oak plots and 36 percent of tanoak plots. For live oak, the presence of beetle damage and *H. thouarsianum* fruiting within the plot were correlated, and the presence of these agents was associated with disease in the subject tree, the number of plot trees with *P. ramorum* cankers, and the number of declining and dead host trees due to causes other than *P. ramorum*.

No other disease or damage agents that we recorded were significantly associated with cases. Of the other agents that are associated with oak mortality, canker rots (typically caused by *Inonotus* spp.) were the most common. Canker rot symptoms and/or fruiting bodies were observed in 63 percent of coast live oak and 18 percent of tanoak plots. Fruiting bodies of *Phellinus* spp., *Ganoderma* spp., and *Laetiporus sulphureus* were observed in about 11 percent, 2 percent, and 1 percent

respectively of the coast live oak plots. Particularly in live oak, *P. ramorum* cankers sometimes occurred in trees that were declining due to canker rot infection or other agents that are not normally associated with *P. ramorum*-related mortality.

Other subject tree factors that were positively associated with disease in univariate models included the canopy dieback rating (likelihood ratio P=0.0410), sky-exposed canopy rating (likelihood ratio P=0.0012), and number of stems (likelihood ratio P=0.0061). Among plot variables, only the count of California bay trees in the plot showed a significant positive association with disease in the subject tree (likelihood ratio P=0.0024).

Multivariate Logistic Regression Models for P. ramorum *Canker in Coast Live Oak*

Many of the individual predictor variables discussed above are correlated or interrelated in various ways. We constructed multivariate logistic regression models to gain insight into the relative strength of various predictor variables and the degree to which predictor variables can be substituted for each other. In general, highly collinear variables cannot be fitted into the same model. Given several related and highly correlated variables, we selected the variable that improved overall model fit the best. Inclusion of a factor in a model does not necessarily imply a causal relationship between the factor and the outcome. Predictor variables included in a model may in fact be outcomes that are influenced by the same underlying (and possibly unmeasured) factors that influence disease risk.

We first fitted a multivariate model that included all significant predictors. We then eliminated two predictors in this model (California bay density, cross-sectional area of largest stem) whose significance in the model was dependent on a few extremely high outliers in their data distributions. This model *(table 4)* correctly classified 77 percent of the subject trees as cases or controls, using P(outcome)>0.5 as the criterion for predicting either outcome (case or control). The model was more successful at predicting controls (87 percent correctly classified) than cases (65 percent correctly classified).

This model is moderately successful at predicting disease in subject trees, but requires information on the incidence of *P. ramorum* cankers on plot trees, SWP of the subject tree, and canopy dieback rating. Such a model would not be of much use for predicting disease risk in an unsurveyed stand because of the detailed site and tree observations are required. Therefore, we also constructed a reduced model *(table 4)* that omits these more data-intensive variables. The reduced model correctly classified 71 percent of the subject trees as cases or controls, and correctly assigned 75 percent of the cases and 70 percent of the controls. Note that California bay density was significant in this model even with high outliers for this variable omitted. Bay density and SWP are correlated to a high degree, so typically only one or the other could be included in a given multivariate model. The reduced model suggests that multistemmed coast live oaks with high amounts of sky exposure that are surrounded by high numbers of California bay trees may be at increased risk of *P. ramorum* canker.

Table 4—*Multivariate logistic regression model parameter estimates for the binary disease outcome (case) for coast live oak.*

Predictor variables	Full model[1]		Reduced model[2]	
	Likelihood Ratio Prob>χ^2	Odds ratio (95 pct confidence interval)	Likelihood Ratio Prob>χ^2	Odds ratio (95 pct confidence interval)
More than 2 stems [true]	0.0009	14.6 (2.77 - 124)	0.0011	11.5 (2.48 - 87.2)
Sky-exposed canopy >50 pct [true]	<0.0001	10.6 (3.67 - 36.0)	0.0001	5.69 (2.30 - 15.90
Difference from SWP_{max} (MPa)	0.0057	0.0430 (0.00347 - 0.415)		
Canopy dieback >20 pct [true]	0.0062	3.66 (1.43 - 10.2)		
Count of trees with early *P. ramorum* symptoms	0.0239	8.96 (1.34 - 65.8)		
Count of California bay trees			0.0223	8.42 (1.35 - 63.4)

[1] Overall model likelihood ratio P<0.0001, n=127 (one record omitted due to missing data for one variable).

[2] Overall model likelihood ratio P<0.0001, n=125. Three plots with outlying high California bay counts (28-32 bay/plot) were omitted from the analysis. Overall model significance and significance of this factor are increased if the outliers are included.

Discussion

The primary objective of this study was to examine the relationship between water stress and *P. ramorum* infection. We found a significant positive association between disease and high SWP, which does not support our original hypothesis that disease might be more common or severe in water-stressed trees. Instead, the data are consistent with the hypothesis that trees located in relatively moist areas are at higher risk for disease than those located in drier sites.

One or more of several possible explanations could explain why disease risk may be elevated for trees in moist sites. Because *Phytophthora* spp. are highly dependent on wet conditions for disease development, environmental conditions associated with moist sites could be especially favorable for the pathogen. For example, prolonged dew periods or extended periods of soil saturation might occur in these areas and could favor sporangium production, zoospore motility and germination, and/or infection. High soil moisture might also alter host susceptibility. For instance, coast live oaks growing in wetter sites might experience relatively rapid trunk growth, possibly increasing the amount of growth cracks in the bark or otherwise rendering the bark more susceptible to infection.

Furthermore, it is now clear that *P. ramorum* causes foliar infections on many species (Rizzo and others 2002b) and such infections may be important sources of inoculum. Therefore, the association between disease and moist sites may be related to the presence of more foliar *P. ramorum* hosts and/or greater levels of foliar disease in such sites. Our analyses indicated that the density of California bay within the plot showed a significant positive association with disease. *P. ramorum* can cause foliar infections on California bay (Rizzo and others 2002a) although this was not known at the time we originally reported this association (Swiecki and Bernhardt 2001).

Because California bay is more common in relatively moist or mesic sites within coast live oak woodlands (Griffin and Critchfield 1976), we cannot cleanly separate the possible effects of high site moisture per se and inoculum production on

California bay leaves. The interaction between these factors may actually be the basis of the increased disease risk associated with these two predictors. In addition, because California bay produces relatively dense evergreen shade, the presence of high bay populations might also help create favorable microclimate conditions for disease development, for instance by slowing the rate at which stems dry. Further investigation will be needed to explore the how these interrelated factors influence disease risk.

P. ramorum-infected trees are more common in case plots than control plots *(table 2)* and disease risk in the subject tree is elevated if other plot trees are also symptomatic *(table 4)*. From these results, we infer that infected trees are spatially aggregated, at least on the scale of the plot size used in this study (0.02 ha). However, the lack of correlation between late disease symptoms in plot trees and early disease symptoms in the subject tree or other plot trees leads us to conclude that bole cankers present within a plot (i.e., within an 8 m radius) may not be an important source of inoculum for the initiation of new cankers.

The overall fit of our two multivariate models *(table 4)* was reasonably good despite the limited number of predictors variable included. The magnitudes of the effects in the models are quite substantial, although the confidence intervals for the odds ratios are large due to the limited sample size. We believe that these factors or closely related factors should be considered in further attempts to model *P. ramorum* disease risk in coast live oak. Nonetheless, our models do not include variables that may be important in disease development, such as the genetic resistance level of individual trees. In addition, the data set used to develop the model may include a number of control trees that are infected but had not yet developed visible symptoms. A follow-up survey of these trees has recently been completed, and information about the change in disease status of control trees should allow us to improve our disease risk models.

Areas sampled in this study were limited to those where *P. ramorum* canker was common. Therefore disease levels in these plots may be greater than would be expected overall across affected areas. Furthermore, because plots were selected based on the occurrence of a case or control tree and were not random, they do not provide unbiased estimates of disease levels in the sampled stands. Nonetheless, the relative levels of disease and mortality associated with *P. ramorum* and other agents are worth noting.

P. ramorum has doubled the mortality of coast live oak in case plots, whereas mortality in control plots has been increased by about 50 percent to date. The effect on tanoak is even more pronounced, because this species is both more susceptible to *P. ramorum* and exhibits relatively low rates of mortality due to other causes *(table 2)*. Overall tanoak mortality rates we observed (about 22 percent in case plots and 19 percent in control plots) are well above the rates reported by Hunter (1997). In a stand in Mendocino County, he recorded 6 percent mortality among tanoaks <20 cm DBH and 9 percent mortality among tanoaks >20 cm DBH over a 14 year period (1981-1995). *P. ramorum* canker thus has the potential to substantially alter the composition of affected stands.

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