Impacts of *Phytophthora ramorum* Canker and Other Agents in Sonoma County Forests[1]

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Abstract

To study impacts of sudden oak death (SOD), a lethal bark canker disease caused by *Phytophthora ramorum*, we established permanent plots in Sonoma County forest types at risk of SOD. Baseline stand and tree health data were collected in 2001 and the plots were reassessed in 2004. The 250 plots (0.02 ha each) were located at 11 study locations in stands containing *Quercus agrifolia*, *Q. kelloggii*, or *Lithocarpus densiflorus* as the dominant hardwood species. By 2004, *P. ramorum* canker symptoms developed at two locations that lacked symptoms in 2001, leading to new tree mortality at one of these locations. Between 2001 and 2004, plot level incidence of *P. ramorum* canker increased from 29 to 40 percent of plots containing *L. densiflorus* and from 2 to 10 percent in plots containing *Q. kelloggii*. Plots with *Q. agrifolia* showed a slight drop in *P. ramorum* canker (from 9 to 7 percent of plots) due to apparent symptom remission in trees at one location. Between 2001 and 2004, the percentage of trees with *P. ramorum* canker symptoms increased at three of four locations with symptomatic SOD canker hosts. Mortality due to both *P. ramorum* and other agents increased at 9 of 11 study locations between 2001 and 2004. Among SOD canker hosts that died during this period, mortality was due to *P. ramorum* in 4 of 16 *Q. kelloggii*, 7 of 18 *Q. agrifolia*, and 18 of 50 *L. densiflorus*. In most study locations, annual background mortality unrelated to *P. ramorum* was less than 1 percent per year between 2001 and 2004. Over this same period, mortality due to *P. ramorum* exceeded background mortality at three locations and equaled it at a fourth. At one *L. densiflorus* location, mortality associated with an unidentified bark canker was comparable to levels of mortality associated with *P. ramorum* at other locations. Only *P. nemorosa* and *P. pseudosyringae* were isolated at this location.

Keywords: California black oak, coast live oak, Lithocarpus*, mortality,* Quercus*, SOD, sudden oak death, tanoak.*

Introduction

Phytophthora ramorum has become established in native forests in a number of counties in Northern and Central California, including Sonoma County. Host trees that can be killed by *P. ramorum* canker (sudden oak death or SOD), including tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and California black oak (*Q. kelloggii*), are important components of many Sonoma County forests and woodlands. Elevated mortality of these SOD canker hosts due to *P. ramorum* canker has the potential to severely impact many important forest ecosystems in Sonoma and other counties.

In summer 2001, with funding provided by the Sonoma County Fish and Wildlife Advisory Board, we established a set of permanent research/monitoring

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plots in Sonoma County woodlands and forests at risk due to *P. ramorum* canker (Swiecki and Bernhardt 2001). Primary objectives of this initial project were to assess overall disease impacts in various forest types and establish baseline conditions against which future changes could be measured. Targeted stands were those with a major component of tanoak, coast live oak, and/or California black oak.

Once baseline conditions within plots had been established, subsequent assessments of the plots were to be used to document the rate and pattern of disease spread within stands and disease progress in individual trees. As part of a project funded by the USDA-Forest Service, the plots established in 2001 were reevaluated in the summer of 2004 (Swiecki and Bernhardt 2005). The objectives of this second project were to estimate the overall impact of SOD on affected forest types and to monitor the spread of disease over time. In this paper, we report on changes in disease distribution, tree mortality, and overall tree health in these plots between summer 2001 and summer 2004.

Methods

In the summer of 2001, we established 250 plots at eleven locations throughout Sonoma County where tanoak, coast live oak, and/or California black oak were common (*table 1*). Plots were circular with a radius of 8 m measured parallel to the ground slope (plot area 0.02 ha=0.05 acre). Plots at each location were established at vertices of a grid superimposed over a map of the location and are unbiased with respect to tree condition or the presence or absence of disease. The only requirements for establishing a plot at a grid intersection was that SOD canker host species (coast live oak, California black oak, and/or tanoak) were present and the slope was navigable (generally no greater than about 70 percent slope). The nominal spacing between grid points was 50 m as plotted on a topographic map, with the exception of the first location (Jack London SP), where the grid spacing was 60 m. Ground distances between plot centers are generally greater than 50 m because of ground slope. Because only plots containing SOD canker host were sampled, the pattern of sampled plots often differs from the idealized sampling grid. The overall area represented by the sampled plots in each grid varied from about 4.7 to 14 ha (*table 1*), depending on the distribution of the host trees at each location. The sampling plan is described in detail in Swiecki and Bernhardt (2001).

We used a handheld GPS receiver (Garmin® GPS 76) with a high-gain external Gilsson® GPS antenna mounted on a telescoping mast to geolocate the specified plot vertices during plot establishment in 2001 and to relocate plots in 2004. One to several trees in or near each plot were marked with numbered aluminum tree tags in 2001. We also recorded the distance and azimuth from tagged trees to each plot center to permit precise relocation of plot centers.

Table 1—*Study locations and numbers of plots, approximate areas, and host species present at each.*

Location	Abbreviation	Number of plots	Approximate plot grid area[1] (ha)	SOD canker host species
Jack London State Park	JLSP	24	8.2	Cal. black oak, tanoak, coast live oak
Sugarloaf Ridge State Park	SRSP	25	6.7	coast live oak
Lake Sonoma (Army Corps of Engineers)	LS	24	8.8	coast live oak, Cal. black oak
Weston (private land)	Weston	26	7.2	coast live oak, Cal. black oak
Austin Creek State Recreation Area	ACSRA	25	7.5	tanoak, Cal. black oak
Modini (private land)	Modini	25	14.0	Cal. black oak, coast live oak
Annadel State Park	ASP	24	7.5	Cal. black oak
Salt Point State Park	SPSP	18	5.5	tanoak
Helen Putnam Regional Park	HPRP	24	5.3	coast live oak, Cal. black oak
Foothill Regional Park	FRP	15	4.7	Cal. black oak, coast live oak
Sonoma Coast State Beach	SCSB	21	6.4	tanoak

[1]Plot grid areas were estimated by drawing an irregular polygon around the plots at each location using ArcView® GIS software. Polygon edges were set approximately 30 m beyond plot centers.

Trees were included in plots if the edge of the main stem was within 8 m of the plot center. A hand-held laser rangefinder (Leica® Disto Classic) was used to determine which trees were within a plot. In each plot, we collected detailed disease data on up to three tagged SOD canker host trees in or near each plot. These trees are referred to as tally trees. Tally trees were sometimes located beyond the 8 m plot boundary in plots that had few live SOD canker host trees. These out-of-plot tally trees are considered only in calculations related to change in disease status and disease on a percentage basis, but are excluded from plot-based density calculations.

All other SOD canker host trees in the plots were categorized with respect to species, canopy position (overstory or understory) and disease status. We noted the type and extent of *P. ramorum* canker symptoms present and whether the tree was in decline or dead due to other agents. We also noted changes in the status of trees other than canker hosts in the plots (for example, decline or mortality). Other plot data evaluated included overall tree cover, California bay (*Umbellularia californica*) cover, the presence of *P. ramorum*-like foliar symptoms on California bay, poison oak (*Toxicodendron diversilobum*) cover, shrub species present, overall shrub cover, canker host regeneration, and the presence of other disease agents in the plot.

In 2001, some suspected *P. ramorum* cankers were sampled for pathogen isolation by Steven Swain (then with Sonoma County UC Cooperative Extension). Most of those isolations were conducted in the late summer of 2001. In the 2004 resurvey, we conducted additional sampling for pathogen isolation, primarily to (1) determine whether *P. ramorum* was present at locations where it had not been found

previously, (2) clarify the infection status of trees in known *P. ramorum*-infested areas that had ambiguous or atypical symptoms, or (3) determine whether other *Phytophthora* spp., including *P. nemorosa* and/or *P. pseudosyringae*, were present at locations with symptomatic trees. In particular, numerous symptomatic plants were sampled at Austin Creek in 2004 to determine whether any of the cankers at this site were caused by species other than *P. ramorum*.

Most isolations were made from stem cankers on SOD canker hosts or from California bay leaves, but a few other host species were also sampled (*table 2*). Tissue pieces from sampled plants were placed into PARP agar plates (Erwin and Ribeiro 1996) to confirm the presence of *P. ramorum* or other *Phytophthora* species. Plates were transported to the lab of Dr. David Rizzo at UC Davis for incubation and identification of fungi that grew out on the PARP plates.

Data Management and Analysis

Data summaries and analyses were prepared using JMP® statistical software, version 5.1.2 (SAS Inc., Cary NC). We used the likelihood ratio chi square test to test for independence of variables in two-by-two or larger contingency tables. We used paired t-tests to test for mean differences in continuous variables recorded for individual plots in 2001 and 2004. Unless otherwise indicated, effects or differences are referred to as significant if $p \leq 0.05$.

Results

The densities of SOD canker hosts at each study location are shown in *figure 1*. A total of 460 coast live oak, 373 California black oak, and 645 tanoak trees were included in the study. Tally trees, for which the most detailed health data was collected, included 229 coast live oaks, 251 California black oaks, and 180 tanoaks. As shown in *figure 1*, California bay, an epidemiologically important foliar host of *P. ramorum*, was present within plots at 7 of the 11 study locations.

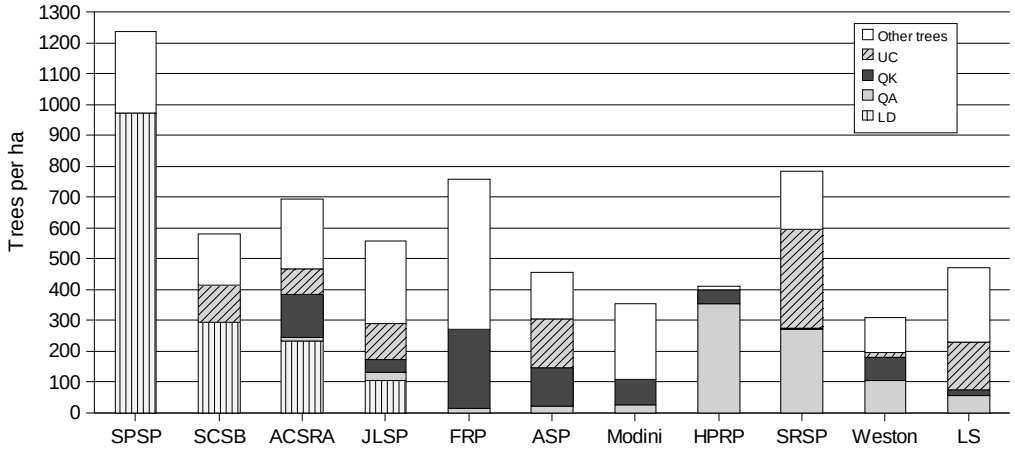

Figure 1—Composition of forests at each study location in trees/hectare. LD=*Lithocarpus densiflorus*; QA=*Q. agrifolia*; QK=*Q. kelloggii*; UC=*Umbellularia californica*; Other trees=other oak, hardwood, and/or conifer species.

Presence of P. ramorum *and Other* Phytophthora *Species at Study Locations*

At the time of the original 2001 survey, sampling conducted by members of David Rizzo's lab at the University of California, Davis, had confirmed the presence of *P. ramorum* at three of the study locations: Jack London, Sugarloaf Ridge, and Austin Creek. No additional locations had positive confirmations of *P. ramorum* as the result of sampling of suspected *P. ramorum* cankers in 2001.

The results of the more extensive sampling conducted in 2004 are shown in *table 2*. In 2004, *P. ramorum* was the only *Phytophthora* species isolated from stem cankers or California bay foliage at the three locations (Austin Creek, Jack London, and Sugarloaf Ridge) that were known to have *P. ramorum* in 2001.

Two locations, Annadel and Lake Sonoma, lacked *P. ramorum* symptoms in 2001 but were confirmed as having *P. ramorum* present in 2004. At Annadel, several California black oak trees showed canker symptoms in 2004 typical of those caused by *P. ramorum*, and isolations confirmed its presence in trees in three plots. *P. ramorum* was also isolated from cankers on a coast live oak near a fourth plot. In addition, many California bay trees showed typical symptoms of leaf infection by *P. ramorum*, and *P. ramorum* was confirmed on California bay leaves from eight plots.

We recovered *P. ramorum* in 2004 from symptomatic California bay foliage from trees located along a small stream between plots at Lake Sonoma. Foliar symptoms in California bay were only observed in this portion of the Lake Sonoma plot grid, and no symptomatic oaks were observed in 2004. We had noted two California black oaks trees with atypical bleeding bark cankers near this area in 2001, but these cankers had become inactive by 2004 and did not appear to be typical of active or inactive *P. ramorum* cankers. Tissue sampled from one of these two trees in 2004 did not yield any *Phytophthora* spp.

Table 2—*Number of positive* Phytophthora *isolations out of the total number of units sampled in 2004 at various study locations. Sampling units were individual trees for stem cankers and single trees or localized groups of trees or plants for the foliar symptoms.* P. ramorum *was the only* Phytophthora *species present in positive isolations with the exception of the Sonoma Coast samples, which yielded both* P. pseudosyringae *and* P. nemorosa*.*

Location	California bay	Tanoak	California black oak	Coast live oak	Coast redwood	Douglas-fir
Salt Point		0/14[1]				
Sonoma Coast	3/4[2]	0/7				
Austin Creek	13/16	14/16	1/2		2/2	0/1
Jack London	1/1		0/1			
Annadel	8/9		3/3	1/1		
Helen Putnam				0/4		
Sugarloaf Ridge	2/2			1/4		
Weston	0/4			0/2		
Lake Sonoma	1/2		0/1			

[1]An additional isolation from California huckleberry (*Vaccinium ovatum*) was also negative.
[2]Leaves from one tree yielded *P. pseudosyringae*; leaves from two other trees yielded *P. nemorosa*.

Sonoma Coast was the third location where *Phytophthora* spp. was newly isolated in 2004. *P. nemorosa* and *P. pseudosyringae* were isolated from leaves of California bay, which is common at this location (*fig. 1*). However, *P. ramorum* was

not isolated from California bay leaves or tanoak cankers sampled at this location (*table 2*).

Bark cankers were present on about 20 percent of the tanoaks at Sonoma Coast *(fig. 2*), but canker symptoms on tanoaks at this location were generally not typical of those caused by *P. ramorum*. Many of the cankers that had recent bleeding in 2004 were small and did not appear to be very aggressive. Many cankers originally noted in 2001 appeared inactive in 2004, with no recent bleeding, and some had callus development at the old canker margin. In some trees, the bark was only affected to a shallow depth. The phloem tissues in these shallow cankers subsequently decayed to a light-colored, powdery consistency and eventually sloughed off, leaving a somewhat eroded appearance to the bark surface.

No other likely causes of the bark cankers at Sonoma Coast were identified. Other pathogens observed on tanoak at this location included *Cryphonectria gyrosa*, which causes stem cankers but is readily recognized by its distinctive sporulation, and the root pathogens *Armillaria* spp. and *Inonotus dryadeus*.

A small number of tanoaks at Salt Point had bark cankers similar to those seen at Sonoma Coast *(fig. 2*). These included the non-aggressive shallow cankers that decayed to form a light powdery material. We also observed numerous callused cankers with exposed wood in the center, similar to canker rot cankers seen in some oaks. None of the tanoak cankers sampled at this location yielded any *Phytophthora* spp. No California bay trees are present within the plot grid for sampling.

Especially at Sonoma Coast, many trees with extensive cankers were subsequently attacked by ambrosia beetles (*Monarthrum* spp.) and the sapwood-decaying fungus *Hypoxylon thouarsianum* (*fig. 2*). Tree mortality associated with these stem cankers increased substantially at both Salt Point and Sonoma Coast between 2001 and 2004.

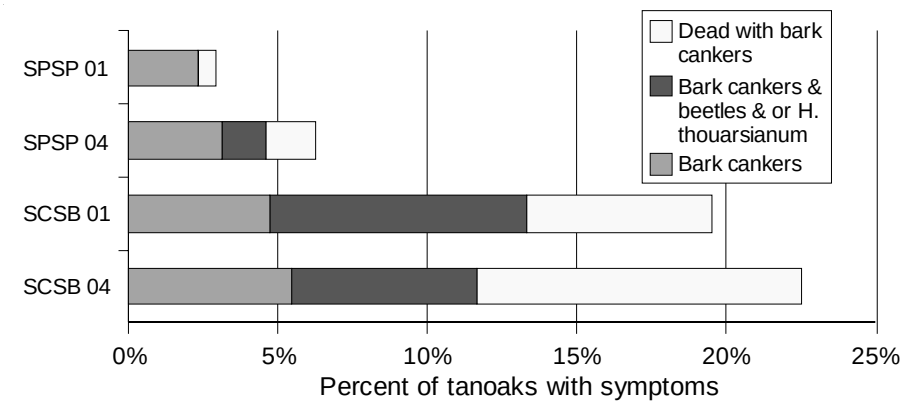

Figure 2—Incidence of symptoms in tanoak associated with an unidentified bark canker at Salt Point (SPSP) and Sonoma Coast (SCSB) in 2001 and 2004. Symptom classes are analogous to the early, late, and dead classification system used for *P. ramorum* canker *(fig. 3*).

Changes in P. ramorum *Disease Levels*

The proportion of trees with *P. ramorum* canker symptoms increased at the two tanoak locations with known *P. ramorum* infestations, and in the new disease front at Annadel (*fig. 3*). The increase in the proportion of symptomatic trees was most dramatic at Austin Creek, nearly tripling since 2001. However, the proportion of symptomatic trees at Sugarloaf Ridge actually declined slightly between 2001 and 2004 due to the apparent remission of canker symptoms in some trees. All four locations showed an increase in the proportion of trees killed by *P. ramorum* (*fig. 3*).

Across the four locations shown in *figure 3*, the percentage of plots that had trees with *P. ramorum* canker symptoms increased from 22 to 38 percent. The percent of plots with *P. ramorum* canker symptoms on California black oak and tanoak increased, but the percentage of plots with *P. ramorum* canker symptoms on coast live oak decreased slightly between 2001 and 2004 (*table 3*). The slight drop in the incidence of *P. ramorum* canker symptoms in coast live oak was due to apparent symptom remission in two trees at Sugarloaf Ridge, which were the only symptomatic trees in their respective plots.

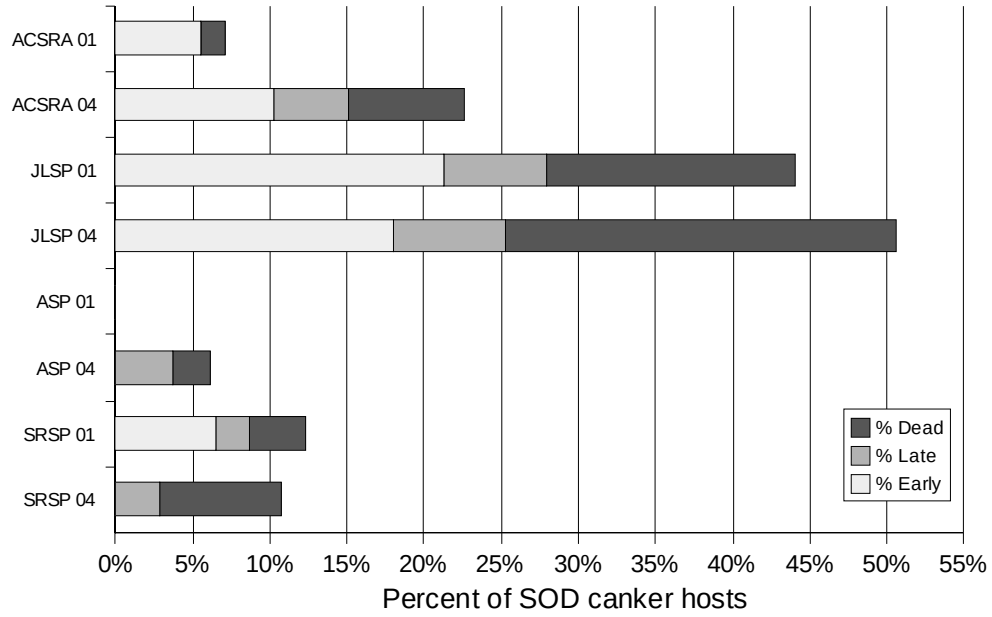

Figure 3—Changes in *P. ramorum* symptom classes between 2001 and 2004 at Austin Creek (ACSRA), Jack London (JLSP), Annadel (ASP), and Sugarloaf Ridge (SRSP). Early = bleeding cankers only; Late = cankers and associated sporulation of *Hypoxylon thouarsianum* and/or damage by wood boring beetles, primarily ambrosia beetles (*Monarthrum* spp.); 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.

Table 3—*Percent of plots with SOD symptoms on canker host species in 2001 and 2004.*

Year	California black oak (119 plots)	coast live oak (114 plots)	tanoak (73 plots)
2001	2	9	27
2004	10	7	40

In comparing the spatial distribution of disease in 2001 and 2004, it appeared that disease distribution was relatively static at Jack London and Sugarloaf, and was spreading from one or more disease loci within the plot grids at Annadel and Austin Creek. At all four locations, symptoms on California bay were more widely distributed than were *P. ramorum* cankers on the canker host species. Furthermore, plots with *P. ramorum* canker were commonly adjacent within the plot grid to other plots with symptomatic trees, especially in the 2004 evaluation. This pattern was especially evident at Austin Creek. Between 2001 and 2004, disease appeared to expand outward from areas that had infected trees in 2001 *(fig. 4*).

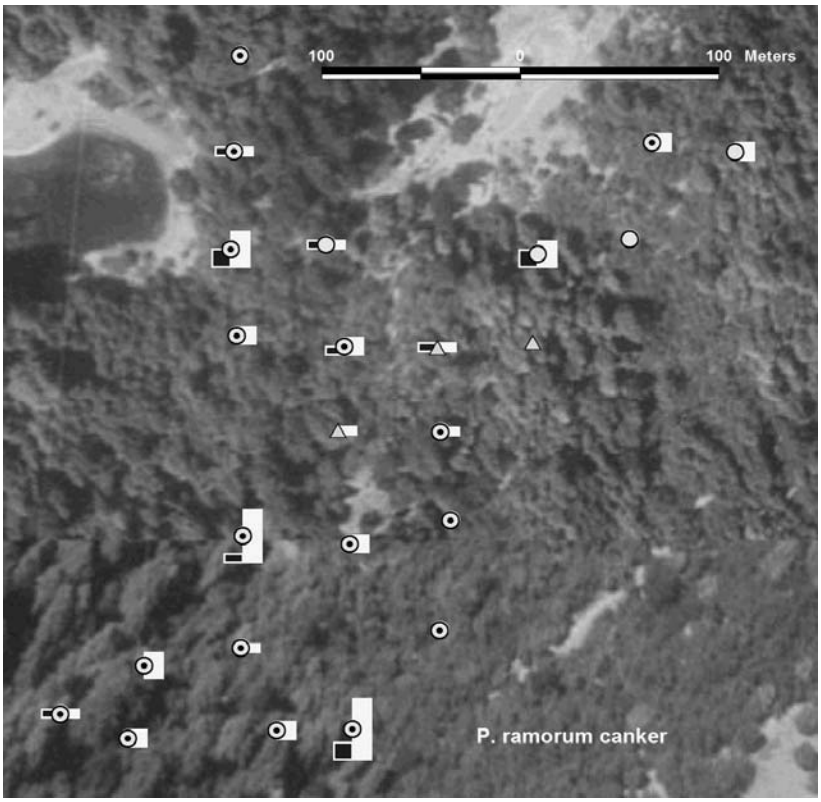

Figure 4—Distribution of *P. ramorum* symptoms in Austin Creek plots in 2001 (dark bar to left of symbol) and 2004 (light bar to right of symbol). Bars indicate the number of symptomatic SOD canker hosts, including trees in plots and additional tagged trees beyond plot edges. The minimum bar height shown indicates one tree. Symbols indicate plot locations. Plots containing California bay are denoted with circles; a black dot in the center of the circle indicates that *P. ramorum* symptoms were present on California bay foliage. Plots without California bay are denoted with triangles.

Mortality of SOD Canker Host Trees

Between 2001 and 2004, mortality increased among all SOD canker hosts *(fig. 5*) and the percentage of plots with mortality also increased (*table 4*). Between the 2001 and 2004 evaluations, the number of dead California black oak trees more than doubled, from 13 to 29, whereas coast live oak and tanoak showed smaller relative increases in mortality. *P. ramorum* contributed to the increase in mortality between 2001 and 2004 in each species, accounting for 4 of 16 dead California black oaks, 7 of 18 dead

coast live oaks, and 18 of 50 dead tanoaks. Among all mortality rated in 2004 (that is, all trees estimated to have died after 1991), *P. ramorum* was responsible for 5 of 29 dead California black oaks, 12 of 41 dead coast live oaks, and 31 of 116 dead tanoaks.

Mortality due to agents other than *Phytophthora* spp. was mostly due to wood decay fungi. Important pathogens we observed on oaks and/or tanoaks in plots included *Inonotus andersonii*, *I. dryophilus*, *I. dryadeus*, *Ganoderma* spp., *Laetiporus gilbertsonii*, and *Armillaria* spp.

Table 4—*Percent of plots with dead trees of SOD canker host species in 2001 and 2004.*

Year	California black oak (114 plots)	coast live oak (119 plots)	tanoak (73 plots)
2001	10	18	49
2004	20	25	67

Levels of mortality due to both *P. ramorum* and other agents varied between study locations. Tree mortality due to *P. ramorum* canker greatly exceeded mortality due to other causes at both Austin Creek and Jack London *(fig. 5*). At Sugarloaf Ridge, mortality due to *P. ramorum* canker approached, but was less than, mortality due to other agents. At Annadel, where *P. ramorum* canker symptoms first appeared in the 2004 evaluation, mortality due to other causes was still much more prevalent than mortality due to *P. ramorum*.

The overall mortality percentages shown in *figure 5* include trees rated in the 2001 survey as having died within the previous 10 years. Hence, the data for 2004 bars in these figures represent 13-year estimated mortality. In contrast, mortality occurring between the 2001 and 2004 evaluations does not include uncertainties associated with the year of mortality of trees that were dead in 2001. We used both 2001to 2004 observed mortality and the 1991 to 2001 estimated mortality rates to calculate annual mortality rates for all locations *(fig. 6*).

Estimated annual background mortality rates for the 2001 to 2004 interval were close to or somewhat higher than those for the 1991 to 2001 interval for most locations. Two locations, Foothill and Helen Putnam, had no new mortality among SOD canker hosts between 2001 and 2004. Overall, the background mortality rate was about 1 percent or less per year for both time intervals at all locations. The differences in background mortality rates between and within locations shown in *figure 6* were not significant. In all locations, some tree mortality was associated with wood decay fungi, especially canker rot fungi such as *I. andersonii* and *I. dryophilu*s, and root disease fungi such as *Ganoderma*. This background mortality excludes mortality due to *P. ramorum* canker and the unknown bark canker at Sonoma Coast and Salt Point.

Annual *P. ramorum* mortality rates were higher for 2001 to 2004 than for 1991to 2001 at all four locations with *P. ramorum* canker *(fig. 6*). For these four locations, the annual mortality due to *P. ramorum* canker for the period 2001 to 2004 (2.03 percent per year) was significantly greater than for 1991 to 2001 (0.42 percent per year; likelihood ratio p=0.02). Furthermore, annual 2001 to 2004 mortality due to *P. ramorum* exceeded background mortality during this period at Jack London, Austin Creek, and Sugarloaf Ridge, and equaled it at Annadel *(fig. 6*).

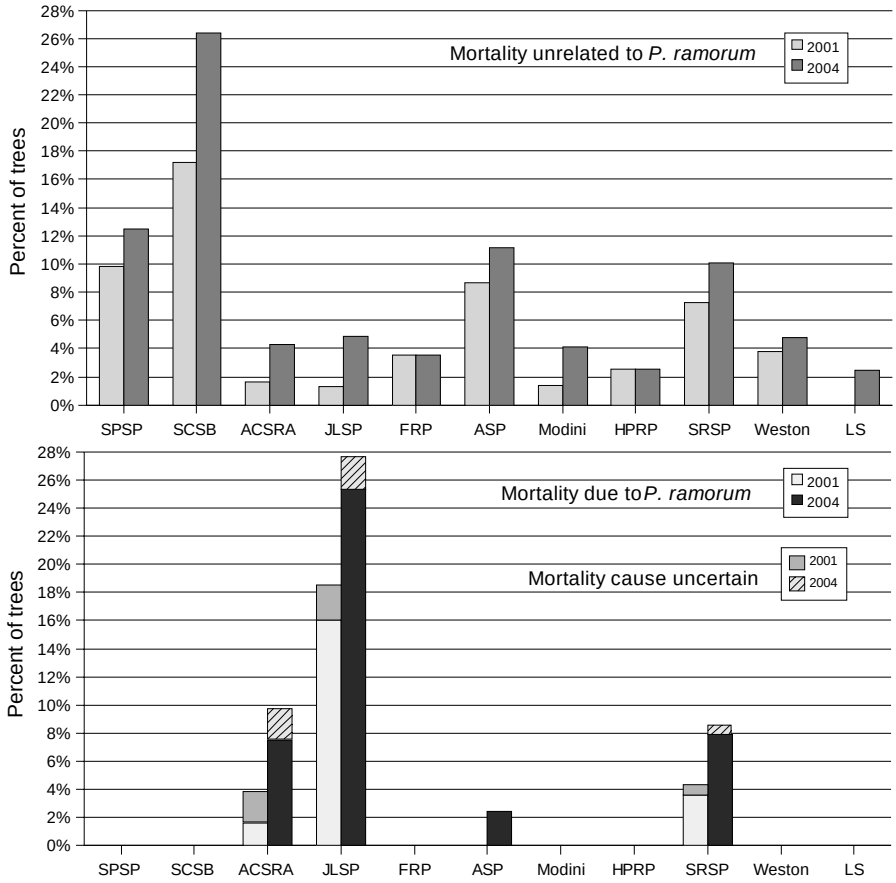

Figure 5—Cumulative mortality among SOD canker host trees due to *P. ramorum* (bottom) and other causes (top) in 2001 and 2004 by location. Mortality in the 2001 survey includes trees rated as having died within the previous 10 years, i.e., mortality since 1991. Total mortality in 2004 covers the period 1991 to 2004. Location abbreviations are shown in *table 1*.

As shown in *figure 6*, recent (2001 to 2004) rates of mortality ascribed to the unidentified bark canker at Sonoma Coast are similar to those due to *P. ramorum* at Austin Creek. Sonoma Coast also had high levels of mortality in 2001 to 2004 that could not be definitively assigned to a given cause (unknown bar in *figure 6*). If some or most of this mortality is also related to the unidentified canker disease, it would further increase the relative importance of this mortality category.

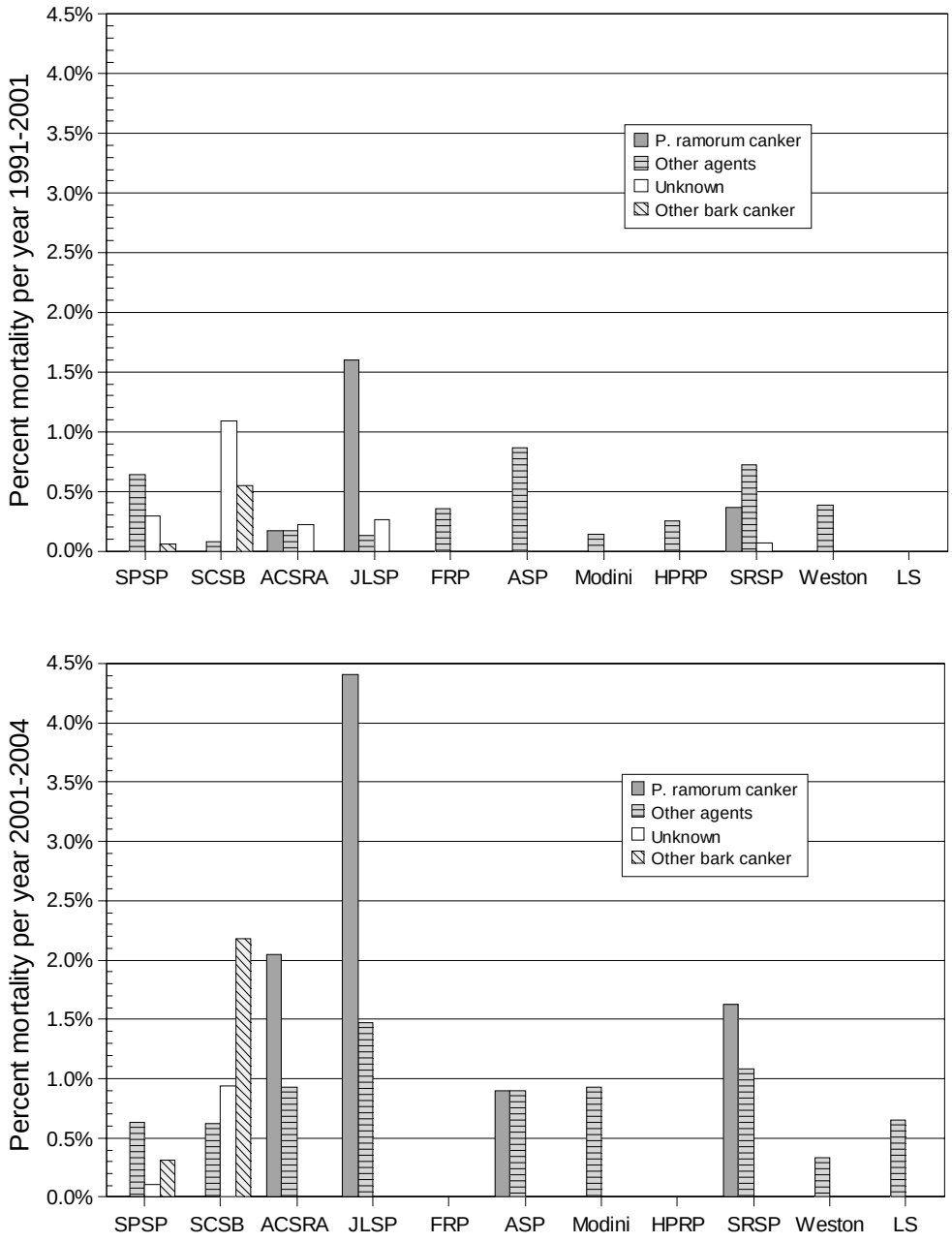

Figure 6—Annual mortality (percent mortality per year) of SOD canker host trees by cause and study location for the periods 1991 to 2001 (top) and 2001 to 2004 (bottom). Location abbreviations are shown in *table 1*.

Discussion

Between 2001 and 2004, *P. ramorum* first appeared within our study areas at two locations: Annadel and Lake Sonoma. Although *P. ramorum* was first confirmed at Annadel SP on bay leaves in July 2003 (Allison Wickland, personal communication; http://kellylab.berkeley.edu/OakMapper/viewer.htm), our isolations were the first reports of *P. ramorum* on SOD canker hosts at this location. Based on the first known appearance of symptoms in canker hosts, it appears that *P. ramorum* had been

introduced into Annadel at a later point than it had been at Jack London and Sugarloaf Ridge, two nearby state parks.

Based on its limited distribution, we believe that the observed infestation at Lake Sonoma is also of relatively recent origin. The infested area is located along a creek at the point where it is crossed by a constructed trail that is used by hikers, equestrians, and bicyclists. An unpaved parking lot used primarily for horse trailer parking and equestrian events is located adjacent to the creek upstream from the point where the infestation was detected. It is likely that *P. ramorum* became established in the creek area from infested materials (such as soil or foliage) that were transported to the area by humans and introduced into the creek either via the trail or the parking lot.

These findings suggest that even within counties or regions in which *P. ramorum* is present, preventing transport of the pathogen material into apparently non-infested areas may be an important method for reducing disease impacts. Current regulatory programs at the state and federal level are directed at the prevention of spread from infested counties to other counties or states. Within infested regions, local programs to minimize or prevent intra-county and intra-regional pathogen spread may be needed.

Increases in tree mortality were observed in all locations where *P. ramorum* was found. Given the low rate of new infections at Sugarloaf Ridge and Jack London, it is likely that annual *P. ramorum* mortality rates will decrease at these locations over the next few years. In contrast, due to new infections at Austin Creek and Annadel, annual *P. ramorum*-related mortality rates are likely to stay constant or increase over the next few years.

Different stages of disease spread and progress were seen among the locations with *P. ramorum* infestations. As of the 2004 assessment, Lake Sonoma appeared to represent a site of very recent pathogen introduction; the pathogen had not yet become widely established on California bay and canker hosts were not yet affected. At Annadel, *P. ramorum* was more widely established in 2004, but the disease epidemic in the plot area appeared to be at an early stage. *P. ramorum* had begun to affect some canker hosts as well as California bay, but the epidemic was probably still limited by inoculum production and distribution. At Austin Creek, the pathogen was established in various parts of the plot grid by 2001, and disease progress among the SOD canker hosts appeared to be at an exponential phase between 2001 and 2004. Jack London represents a later phase of the epidemic, in which the pathogen had become well established throughout the plot grid. At this site, many of the trees with the highest disease risk had already been infected by 2001, so the rate of new infections was relatively low between 2001 and 2004.

These results suggest that disease progress among the SOD canker hosts in a newly infested stand may follow a sigmoidal disease progress curve. The percentage of newly-diseased trees probably increases slowly after the initial introduction of the pathogen due to limited inoculum density. As the foliar disease epidemic spreads throughout a stand, disease incidence in canker hosts can increase rapidly under favorable conditions. As the number of uninfected susceptible hosts decreases, the epidemic becomes limited by the lack of suitable hosts. The disease situation at Sugarloaf Ridge may either represent this latter stage of the epidemic or a stalling of the epidemic in the early phase due to environmental conditions that were relatively unfavorable for disease development.

Inoculum production by *P. ramorum* is favored by late spring rainfall, although levels and temporal patterns of inoculum production differ somewhat between mesic tanoak stands and more xeric oak stands (Dave Rizzo, personal communication). Based on our review of weather station data (Western Regional Climate Center 2006), most portions of Sonoma County had average to below average rainfall without high amounts of late spring rain between our 2001 and 2004 evaluations. Disease spread under these conditions may have been relatively low compared with what would be expected under more favorable conditions, such as those that occurred in spring 2005 and 2006. Informal observations indicate that disease levels have increased dramatically in portions of Sonoma County since 2004. Reassessment of these plots in 2007 would allow us to compare disease progress during periods with different levels of inoculum production.

P. ramorum, *P. nemorosa*, and *P. pseudosyringae* were only confirmed at locations that also had substantial amounts of California bay within the plots *(fig. 1*). Previous research has shown a strong epidemiological link between California bay proximity and coast live oak infection rates by *P. ramorum* (Swiecki and Bernhardt 2002). The presence of other foliar hosts, with the possible exception of poison oak (Swiecki and Bernhardt 2003), has not been correlated with infection of California black oak or coast live oak. Futhermore, *P. ramorum* does not normally sporulate on infected bark cankers of its oak hosts (Davidson and others 2005). Hence, California black oaks and coast live oaks at the Helen Putnam, Modini, and Foothill study locations and much of the Weston location may be very unlikely to develop *P. ramorum* canker under current stand conditions. Disease risk at sites containing tanoak is not completely related to California bay presence since tanoak sustains foliar and twig infections that can produce *P. ramorum* inoculum (Rizzo and others 2002). Therefore, tanoak stands in Sonoma County are likely to be at risk from *P. ramorum* for the foreseeable future.

The cause of the bark canker symptoms seen at Sonoma Coast and Salt Point remains uncertain. *P. nemorosa* and *P. pseudosyringae* were isolated from bay leaves in the plots at Sonoma Coast, but we were unable to recover any *Phytophthora* spp. from active bleeding cankers at either location. However, given the relatively low numbers of isolations performed, we cannot rule out *P. nemorosa* and/or *P. pseudosyringae* as possible causes of the cankers. *P. ramorum* also cannot be entirely ruled out as a possibility at this point. However, the fact that *P. ramorum* was not recovered from symptomatic trees in either 2001 or 2004 strongly suggests that this species was either not present or was not associated with the sampled cankers.

P. nemorosa and *P. pseudosyringae* are recently-described species (Hansen and others 2003, Jung and others 2003) and further analysis is needed to determine how long these species have been present in California forests. If most of the cankers at Sonoma Coast are actually caused by one or both of these species, it would imply that these agents may have impacts comparable to *P. ramorum* in at least some locations. The greater importance of this canker disease at Sonoma Coast relative to Salt Point may be related to the fact that California bay, which is also a host of *P. nemorosa* and *P. pseudosyringae*, is common at Sonoma Coast but absent at the Salt Point study location *(fig. 1*).

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