2004 RESURVEY OF PERMANENT PLOTS FOR MONITORING *PHYTOPHTHORA RAMORUM* CANKER (SUDDEN OAK DEATH) IN SONOMA COUNTY

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Cover: Mixed hardwood forest with coast live oak, California black oak, and California bay near Healdsburg, CA. This area was unaffected by *P. ramorum* in July 2004.

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EXECUTIVE SUMMARY

Sudden oak death (SOD), a lethal bark canker disease caused by *Phytophthora ramorum,* has the potential to severely impact many of the ecologically important woodlands and forests of Sonoma County, California. In 2001 we established permanent research/monitoring plots in Sonoma County woodland and forest types at risk due to *P. ramorum* canker and collected baseline data on these plots. This report presents the results from the 2004 resurvey of these plots. Within plots, we evaluated the occurrence and progression of symptoms caused by *P. ramorum,* assessed tree decline and mortality due to other agents, and looked at levels of tree failure and potential regeneration of affected species.

We resurveyed a total of 250 fixed-area plots (0.02 ha each) at 11 study locations in various portions of Sonoma County. The number of locations with confirmed *P. ramorum* infections of both SOD canker hosts (i.e., coast live oak, California black oak, and tanoak) and California bay increased from three in 2001 to four in 2004. At a fifth location, *P. ramorum* was confirmed from California bay foliage, but not from SOD canker hosts. Hence, *P. ramorum* became established within the study areas at two additional locations between 2001 and 2004.

Among all plots containing tanoak (73 plots), the percentage of plots with *P. ramorum* canker increased from 29% in 2001 to 40% in 2004. For plots containing California black oak (119 plots), the percentage of plots with *P. ramorum* canker symptoms increased from 2% in 2001 to 10% in 2004. The percentage of plots with coast live oak (114 plots) that had *P. ramorum* canker symptoms decreased slightly, from 9% in 2001 to 7% in 2004. 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 State Park.

Between 2001 and 2004, the percentage of trees with *P. ramorum* canker symptoms increased at three of the four locations that had symptomatic SOD canker hosts. The percentage of SOD canker hosts with *P. ramorum* canker symptoms increased from 0% to 6% at Annadel SP, 7% to 23% at Austin Creek SRA, and 44% to 51% at Jack London SP between 2001 and 2004. The remaining location (Sugarloaf Ridge SP) showed a slight drop (12% to 11%) in the percentage of symptomatic SOD canker hosts.

Mortality in SOD canker hosts due to both *P. ramorum* and other agents increased at 9 of the 11 study locations between 2001 and 2004. The percentage of the mortality increase due to *P. ramorum* was 27% for California black oak, 49% for coast live oak, and 30% for tanoak. For most locations, annualized background mortality unrelated to *P. ramorum* was less than 1% per year between 2001 and 2004. Over this period, mortality rates associated with *P. ramorum* exceeded background mortality at three locations and was equal to it at a fourth location. At tanoak-dominated Sonoma Coast State Beach, mortality associated with an unidentified bark canker greatly exceeded background mortality and was comparable to levels of mortality associated with *P. ramorum* at other locations. Although no pathogens were successfully isolated from tanoak cankers at Sonoma Coast SB, we isolated both *P. nemorosa* and *P. pseudosyringae* from symptomatic California bay foliage within plots.

Overall tree failure rates for the period 1999-2004 were significantly higher for California black oak (11.5%) than for tanoak (7.4%) or coast live oak (5.1%). Bole failures were the most common failure type among tanoaks and coast live oaks, whereas large branch failures and bole failures were equally common in California black oak. Failure rates were significantly higher among coast live oaks with *P. ramorum* canker symptoms than among asymptomatic trees. At two locations, tanoaks with *P. ramorum* canker also failed at a higher rate than asymptomatic tanoaks. Most of

the coast live oaks and tanoaks with *P. ramorum* symptoms were dead when they failed. Wood decay was the primary contributing factor in almost all observed failures.

Tanoak seedlings were present in nearly all plots with tanoak trees. All plots with tanoak mortality had tanoak seedlings which could potentially grow to replace dead trees. Coast live oak plots were less well-stocked with seedlings. Twenty five percent of plots with coast live oak mortality lacked coast live oak seedlings, and mean counts of coast live oak seedlings per plot were significantly lower in 2004 than in 2001. Less than half of all plots with California black oak trees had California black oak seedlings. Three-quarters of the plots with California black oak mortality lacked seedlings of this species. Regeneration of California black oak appears inadequate to maintain stand density even without the additional mortality due to *P. ramorum* in the surveyed woodlands.

INTRODUCTION

In summer 2001, with funding from the Sonoma County Fish and Wildlife Advisory Board, we established a set of permanent research / monitoring plots in Sonoma County woodlands and forests at risk due to *P. ramorum* canker (sudden oak death or SOD). We targeted woodland/forest types that were dominated by or had a substantial component of tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and/or California black oak (*Q. kelloggii*), all of which can be killed by *P. ramorum* canker.

The main objectives of the original project were as follows:

1. Establish a baseline for measuring vegetation change in specific habitat types that could result from increased disease incidence and mortality in tanoak, coast live oak, and black oak.

2. Evaluate patterns of *P. ramorum*-related mortality on a landscape scale in a variety of woodland and forest types. This information could be used to help develop models for predicting disease spread and progress in different parts of the county.

3. Measure background levels of disease and mortality associated with other agents to develop a complete picture of the health of Sonoma County forest/woodland resources and the potential management implications for maintaining these resources.

4. Provide background vegetation data for specific sites that could be coupled with subsequent wildlife use/population studies that can directly measure effects of SOD on wildlife and fisheries.

Information on initial plot establishment and basic disease and stand data are reported in Swiecki and Bernhardt (2001).

This report describes results from the resurvey of these plots in the summer of 2004. This resurvey is part of a project to estimate the overall impact of SOD on affected forest types and to monitor the spread of disease over time. Changes in disease status and general tree health that occurred in these plots between summer 2001 and summer 2004 are described in this report.

METHODS

Plot selection

In 2001, we established 250 plots at eleven locations throughout Sonoma County where tanoak, coast live oak, and/or California black oak were common (Figure 1, Table 1). Plots were established on public lands to the extent possible. At locations 4 and 6, plots are on private lands that are protected by conservation easements held by the Sonoma County Agricultural Preservation and Open Space District.

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 were present and the slope was navigable (generally no greater than about 70%). 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 coast live oak, California black oak, and/or tanoak were sampled, the pattern of actual plot positions often differs from the idealized sampling grid. In addition, the overall area represented by the sampled plots in each grid varied between locations, from about 4.7 to 14 ha (Table 1), depending on the distribution of the host trees at each location. The sampling plan is explained in detail in Swiecki and Bernhardt (2001).

Plot centers were originally established using GPS-specified coordinates (Swiecki and Bernhardt 2001). Distance and azimuth readings were taken to trees marked with 31 cm numbered aluminum tree tags in or near each plot to permit precise relocation of plot centers. The tree tags in each plot point towards the plot center. A hand held laser rangefinder (Leica® Disto Classic) was used to determine whether the trees were within the plot. Trees were included in plots if the edge of the main stem was within 8 m of the plot center. We used a handheld GPS receiver (Garmin® GPS 76) with a high-gain external Gilsson® GPS antenna mounted on a telescoping mast to relocate plot centers.

In each plot, we reevaluated the condition of all trees in the plots and certain trees located just beyond the plots as described below. We collected detailed disease data (Table 2) for up to three tagged canker host trees (coast live oak, California black oak, and tanoak) 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 recounted by species, canopy position (overstory or understory) and disease status with respect to both *P. ramorum* canker symptoms and decline or death due to other agents. In the 2004 resurvey, we used a flat measuring tape to estimate the diameter of the largest stem of all canker host trees in most plots to facilitate tracking the condition of individual trees over time. We also noted changes in the status of trees other than canker hosts in the plots (e.g., decline or mortality). Other plot data we evaluated included overall tree cover, California bay cover, the presence of *P. ramorum*-like foliar symptoms on California bay, poison oak cover, shrub species present, overall shrub cover, canker host regeneration, and the presence of other disease agents in the plot. Table 3 presents a detailed description of plot data collected in the initial survey and resurvey.

Isolations from symptomatic coast live oak, California black oak, tanoak, and California bay were made onto PARP media (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 which grew out on the isolation plates.

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

Location number	Location	Abbreviation	Number of plots	Approximate plot grid area[1] (ha)	Subject tree species
1	Jack London State Park	JLSP	24	8.2	Cal. black oak, tanoak, coast live oak
2	Sugarloaf Ridge State Park	SRSP	25	6.7	coast live oak
3	Lake Sonoma (Army Corps of Engineers)	LS	24	8.8	coast live oak, Cal. black oak
4	Weston (private land)	Weston	26	7.2	coast live oak, Cal. black oak
5	Austin Creek State Recreation Area	ACSRA	25	7.5	tanoak, Cal. black oak
6	Modini (private land)	Modini	25	14.0	Cal. black oak, coast live oak
7	Annadel State Park	ASP	24	7.5	Cal. black oak
8	Salt Point State Park	SPSP	18	5.5	tanoak
9	Helen Putnam Regional Park	HPRP	24	5.3	coast live oak, Cal. black oak
10	Foothill Regional Park	FRP	15	4.7	Cal. black oak, coast live oak
11	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.

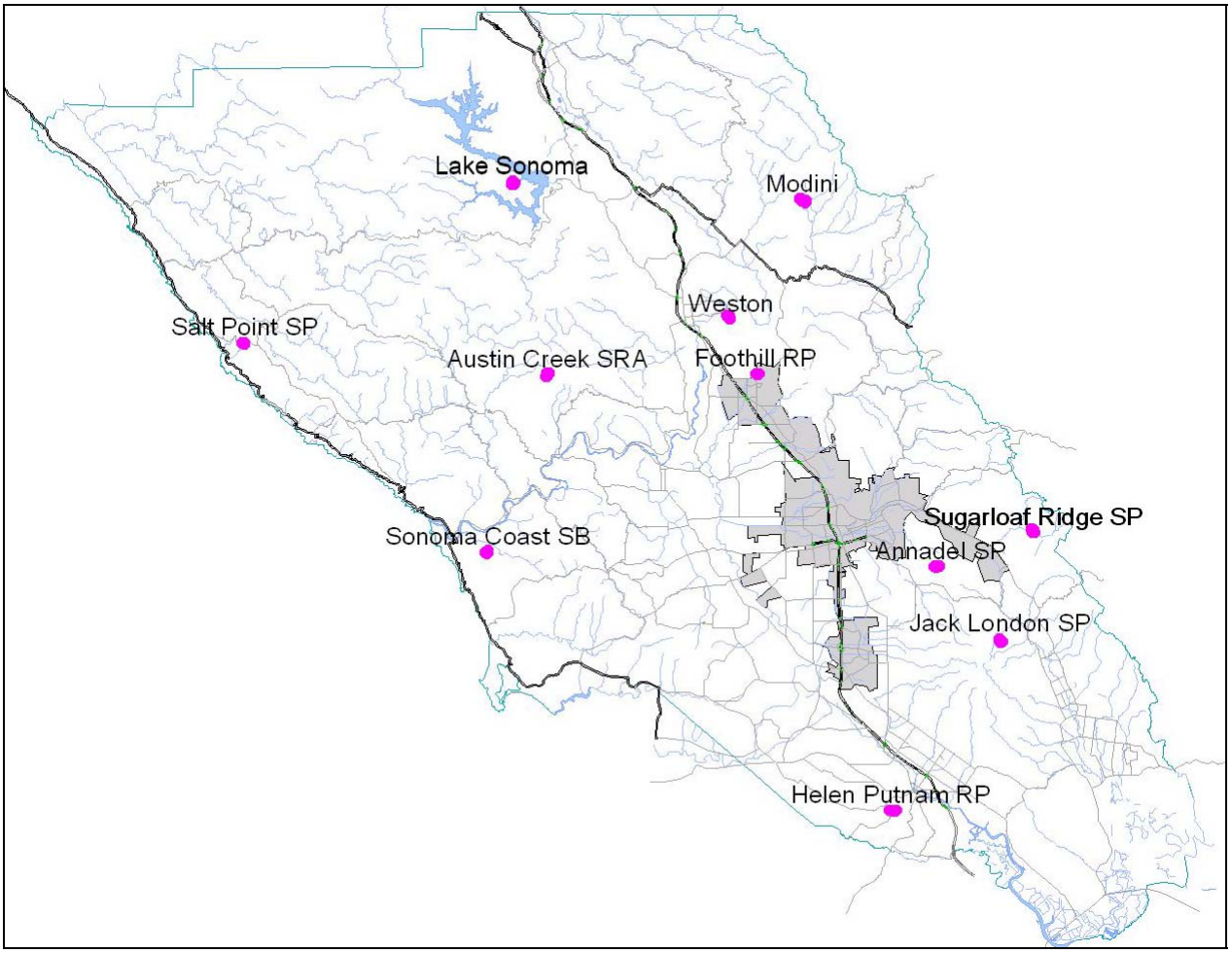

Figure 1. Geographic distribution of survey / monitoring locations within Sonoma County.

Data management and analysis

Plot data are georeferenced to the plot locations to allow for use and analysis with GIS software. For each location, USGS digital orthoquad images were used as a base map layer. We designed GIS-compatible databases for data storage and manipulation. Plot data and detailed data on individual tally trees are stored in separate database tables.

All data were checked for possible data entry errors prior to summary 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 2×2 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 $p \leq 0.05$.

Table 2. Tree variables measured for tally trees in each study plot. Variables reevaluated in 2004 are shown in bold.

Variable	Method	Scale/units and notes
Tally tree species		*L. densiflorus, Q. agrifolia, Q. kelloggii;* some *Q. wislizeni* included at Lake Sonoma
Origin class	visual assessment	seed or sprout
Distance to plot center	laser rangefinder	m; data used for plot center relocation
Azimuth to plot center	compass	degrees; data used for plot center relocation
DBH	d-tape or flat tape	cm (d-tape measurements in inches were converted); flat tape used where d-tape could not be (e.g., trunks covered with poison oak)
Number of stems from ground	count	number of stems (>3 cm DBH) per tree
Stems with *P. ramorum* symptoms	count	infected stems/tree
Dead stems	count	dead stems/tree
Tree dead / cause	visual assessment	Causes: (1). *P. ramorum;* (2). other agent(s); (3). unable to determine; (4) unidentified bark canker at SCSB and SPSP. Tree scored as dead only if all main stems are dead. Two causes (*P. ramorum* plus other agents) are scored if applicable.
Hypoxylon thouarsianum	presence of fruiting bodies	present / absent on main stem
Bark and/or ambrosia beetles in main stem	presence of boring dust and/or holes	present / absent on main stem
P. ramorum-related symptoms	visually assess symptoms present	1. No symptoms 2. Early - bleeding cankers only 3. Late - cankers plus beetles and/or *H. thouarsianum* 4. Dead as result of *P. ramorum* infection; evidence of bark cankers present
P. ramorum canker count	count	estimated on basis of external bleeding spots and limited inspection of canker margins
Percent girdling due to *P. ramorum* canker	visual estimate	pretransformed 0-6 scale[1] Percent of circumference affected estimated based on projection of cankered areas as if all were viewed on same cross section; some limited chipping of bark done to confirm horizontal extent of canker margins
Severe tree decline due to other agents	visual assessment	yes / no Trees scored as in decline if overall condition is poor enough that death within 10 years appeared likely.
Sky exposed canopy	visual	pretransformed 0-6 scale[1]; percent of canopy projection area with unobstructed access to direct overhead sunlight
Canopy thinning	visual estimate	0-2 scale: 0-none, 1-slight, 2-pronounced
Canopy dieback	visual estimate	pretransformed 0-6 scale[1] Based on percent dead crown volume
Decay impact	visual assessment	0-3 scale: 0-no , 1-low, 2-moderate, 3-high Decay impact rating assesses the probability that existing decay will have a significant negative impact on tree health or survival. Assessment of decay impact is 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.
Live basal sprouts	visual observation	presence/absence scored for dead trees only For summaries, trees are scored as dead if all main stems are dead even if some live basal sprouts are present

[1]The 0-6 scale is based on the following arcsine-transformed percentage scale:

0: Symptom not seen
1:< 2.5%
2: 2.5% to <20%
3: 20% to < 50%
4: 50% to < 80%
5: 80% to < 97.5%
6: 97.5% to 100%

Table 3. Plot and stand variables measured in 8 m radius fixed-area study plots. Variables reevaluated in 2004 are shown in bold.

Variable	Method	Scale/units and notes
Tree density / species composition	count by species	Trees have at least one stem at least 3 cm DBH located within 8 m of plot center; multi-stemmed trees count as single trees; coppiced redwoods separated by 1 m count as separate trees
Plot slope	clinometer	percent
Plot aspect	compass	degrees
Plot tree canopy cover	visual estimate	pretransformed 0-6 scale[1]
Plot shrub cover	visual estimate	pretransformed 0-6 scale[1]
Overstory canopy trees species in plot	visual assessment	list of species Base of tree does not need to be within the plot
Tree health class relative to *Phytophthora* stem canker and other decline/mortality agents (SOD canker hosts[2] only)	tree count by species, subcategorized by symptom class and canopy position (overstory/understory where overstory trees have sky-exposed canopy rating 2 or higher); stem diameters were also noted for individual trees in plots with many trees	Symptom classes are based on combinations of tree death causes, *P. ramorum* symptom classes, and severe decline ratings in Table 2: 1 - asymptomatic 2 - early *P. ramorum* disease (bleeding cankers only) 3 - late *P. ramorum* disease (cankers plus beetles and/or *H. thouarsianum*) 4 - dead attributed to *P. ramorum* 5 - severe decline (tree death likely within 10 years) due to other agents 6 - dead due to other agents 7 - dead but cause can't be determined 8 - early *P. ramorum* disease and severe decline due to other agents 9 - late *P. ramorum* disease and severe decline due to other agents 10 - dead attributed to both *P. ramorum* and other agents -Other decline/mortality agents do not include *H. thouarsianum* and bark or ambrosia beetles if they are associated with *P. ramorum*
General tree health class (trees other than SOD canker hosts[2])	tree count by species, subcategorized by symptom class and canopy position (overstory/understory)	Symptom classes: 1 – live 2 – decline (tree death likely within 10 years) 3 – dead
Phytophthora-type foliar symptoms on California bay[3]	visual observation	presence/absence
California bay cover[3]	visual estimate	pretransformed 0-6 scale[1], bay cover in plot, including regeneration
Poison oak cover[3]	visual estimate	pretransformed 0-6 scale[1]
Poison oak stature[3]	visual observation	low (less than 30 cm tall), ascending (30-100 cm tall), climbing
Tree failures[3]	visual observation; recorded for all main stem failures and branch failures >20 cm diameter at break	recorded by species, DBH, type of failure (branch, scaffold, bole, root crown, root), diameter at the break, condition of the tree at failure (live, dead), disease status of failed tree, estimated date of failure (prior to 2001 survey, after 2001 survey, within past 12 months).
SOD canker host[2] regeneration	count or estimate if >10	regeneration: seedlings and saplings <3 cm DBH
Dead SOD canker host[2] regeneration	count	Cause of mortality in regeneration was not determined
Regeneration of trees other than SOD canker hosts[2]	presence noted by species	regeneration: seedlings and saplings <3 cm DBH
Other pathogens/agents	note presence	listing of agents and symptoms observed, including various decay fungi, canker rot, root disease, *H. thouarsianum*, and beetles

Table 3 continued.

Variable	Method	Scale/units and notes
Woody understory species	note presence	list shrubs and woody vines present within plot; herbaceous species and grasses were not scored
Disturbance	Note type of disturbance	roads, trails, logging, etc. within plot or near edge of plot were noted

[1]The 0-6 scale is based on the following arcsine-transformed percentage scale:

0: Symptom not seen
1:< 2.5%
2: 2.5% to <20%
3: 20% to < 50%
4: 50% to < 80%
5: 80% to < 97.5%
6: 97.5% to 100%

[2]Hosts of *P. ramorum* stem canker, i.e., coast live oak, California black oak, and tanoak.

[3]Variable first evaluated in 2004 or evaluated in more detail in 2004.

RESULTS AND DISCUSSION

The main species of interest in this study are coast live oak, California black oak, and tanoak, all of which are susceptible to *P. ramorum* bark cankers and are collectively referred to as SOD canker hosts in this report. The prevalence of these species at each of the study locations is shown in Figure 2. A total of 460 coast live oak, 373 California black oak, and 645 tanoak trees were included in the study. Tally trees included 229 coast live oaks, 251 California black oaks, and 180 tanoaks.

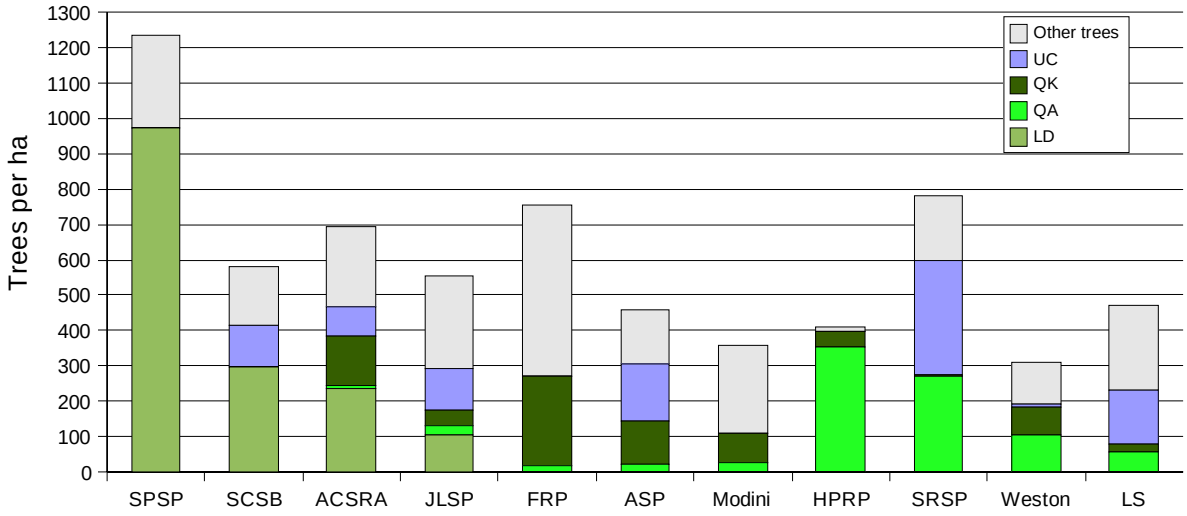

Figure 2. Composition of forests at each study location in trees/hectare. LD=*Lithocarpus densiflorus*; QA=*Q. agrifolia;* QK=*Q. kelloggii*); UC=*Umbellularia californica*; Other trees=other oak, hardwood, and/or conifer species.

Table 4 shows the major tree species contributing to the composition of each surveyed stand. We encountered only seven canyon live oak trees (*Q. chrysolepis*) in study plots; these are included in the SOD canker host category in Table 4. Interior live oak (*Q. wislizeni*) is listed separately because although *P. ramorum* cankers have not yet been reported under natural conditions on this species, it is susceptible when inoculated artificially. White oaks include *Q. lobata, Q. garryana, Q. douglasii,* and *Q. berberidifolia*. In addition to the species listed in Table 4, bigleaf maple (*Acer macrophyllum*), California buckeye (*Aesculus californica*), and California nutmeg (*Torreya californica*) trees were found in a few plots at some locations.

Table 4. Composition of the sampled area at each study location.

	Percent of trees within plots						
Location	SOD canker hosts[1]	Interior live oak	White oaks[2]	California bay	Madrone	Coast redwood	Douglas-fir
SPSP	79%	0%	0%	0%	1%	15%	5%
SCSB	51%	0%	0%	21%	0%	22%	7%
ACSRA	55%	1%	0%	12%	8%	8%	14%
JLSP	31%	0%	0%	21%	11%	11%	23%
FRP	36%	0%	50%	0%	15%	0%	0%
ASP	32%	0%	28%	35%	1%	0%	5%
Modini	31%	0%	15%	0%	22%	0%	24%
HPRP	96%	0%	4%	0%	0%	0%	0%
SRSP	35%	0%	3%	36%	6%	0%	11%
Weston	59%	0%	4%	4%	25%	0%	0%
LS	18%	18%	5%	33%	5%	11%	9%

[1]SOD canker hosts include. *Lithocarpus densiflorus, Q. agrifolia, Q. kelloggii*, and *Q. chrysolepi*s.

[2]White oaks include *Q. lobata, Q. garryana, Q. douglasii*, and *Q. berberidifolia*.

Presence of *P. ramorum* and other *Phytophthora* species at study locations

At the time of the original 2001 survey, sampling to confirm the presence of *P. ramorum* was limited due to both state regulatory restrictions and budget constraints. At that time, the presence of *P. ramorum* had been confirmed through sampling conducted by members of the Rizzo lab at UC Davis in three of the study locations: Jack London, Sugarloaf Ridge, and Austin Creek. Additional sampling of suspected *P. ramorum* cankers was conducted in 2001 by Steven Swain (then with Sonoma County UC Cooperative Extension). Most of those isolations were conducted in the late summer of 2001. No additional locations had positive confirmations of *P. ramorum* as the result of that sampling.

In the 2004 resurvey, isolations were performed primarily to (1) determine whether *P. ramorum* was present at additional locations, (2) determine whether other *Phytophthora* spp., including *P. nemorosa* and/or *P. pseudosyringae,* were present at locations with symptomatic trees, and (3) clarify the infection status of trees in known infested areas when symptoms were ambiguous or atypical. Numerous isolations were made at Austin Creek by Djibo Zanzot 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 5).

Table 5. Number of positive *Phytophthora* isolations out of the total number of units sampled in 2004 at the 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 recovered in all positive isolations except at Sonoma Coast as noted below.

Location	California bay	Tanoak	California black oak	Coast live oak	Coast redwood	Douglas -fir	*Vaccinium ovatum*
Salt Point		0/14					0/1
Sonoma Coast	3/4[1]	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]Leaves from one tree yielded *P. pseudosyringae*, leaves from two other trees yielded *P. nemorosa*.

An overview of our results documenting *P. ramorum, P. nemorosa,* and *P. pseudosyringae* in the study locations is presented in Table 6. In 2004, only *P. ramorum* was 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. Although few samples were taken at Jack London and Sugarloaf Ridge, plots at Austin Creek were sampled extensively and failed to yield any *Phytophthora* other than *P. ramorum*.

Table 6. Presence of *P. ramorum*, *P. nemorosa* and *P. pseudosyringae* at the 11 study locations in 2001 and 2004 as confirmed by isolations from SOD canker hosts and California bay. + = one or more positive isolations, — = all attempted isolations negative, 0 = no isolations attempted due to lack of suitable symptomatic material. Positive isolations in 2001 were conducted by personnel from the laboratory of David Rizzo, UC Davis.

Pathogen species:	*P. ramorum*			*P. nemorosa*		*P. pseudosyringae*	
Year confirmed:	2001	2004		2004		2004	
Location	SOD canker host	SOD canker host	California bay	SOD canker host	California bay	SOD canker host	California bay
Salt Point		—	0	—	0	—	0
Sonoma Coast		—	—	—	+	—	+
Austin Creek	+	+	+	—	—	—	—
Jack London	+	+	+	—	—	—	—
Foothill		0	0	0	0	0	0
Annadel		+	+	—	—	—	—
Modini		0	0	0	0	0	0
Helen Putnam		—	0	—	0	—	0
Sugarloaf Ridge	+	+	+	—	—	—	—
Weston		—	—	—	—	—	—
Lake Sonoma		—	+	—	—	—	—

Annadel and Lake Sonoma were the only two locations that lacked *P. ramorum* symptoms in 2001 that 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. To our knowledge, these are the first reports of *P. ramorum* on SOD canker hosts at this park. In addition, many California bay trees showed typical symptoms of

leaf infection by *P. ramorum*, and *P. ramorum* was confirmed from California bay leaves in 8 plots. *P.ramorum* was first confirmed in the park on bay leaves in July 2003 (Allison Wickland, personal communication; http://kellylab.berkeley.edu/OakMapper/viewer.htm).

At Lake Sonoma, two trees with atypical bleeding bark cankers were noted in 2001. 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 did not yield any *Phytophthora* spp. However, we observed foliar symptoms in California bay located along a small stream between plots (Figure 3). *P. ramorum* was recovered from the symptomatic California bay foliage at this site. Foliar symptoms in California bay were only observed in this portion of the Lake Sonoma plot grid and no symptomatic oaks were observed.

Based on its limited distribution, we believe that the observed infestation at Lake Sonoma is 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 (Figure 4). It appears that the *P. ramorum* infestation probably became established when infested materials (soil, foliage, etc.) were transported to the area by humans and were introduced into the creek either via the trail or the parking lot.

Figure 3. Left to right, California bay leaves at Lake Sonoma positive for *P. ramorum*, from Weston Ranch negative for *P. ramorum*, and from Sonoma Coast State Beach positive for *P. pseudosyringae.*

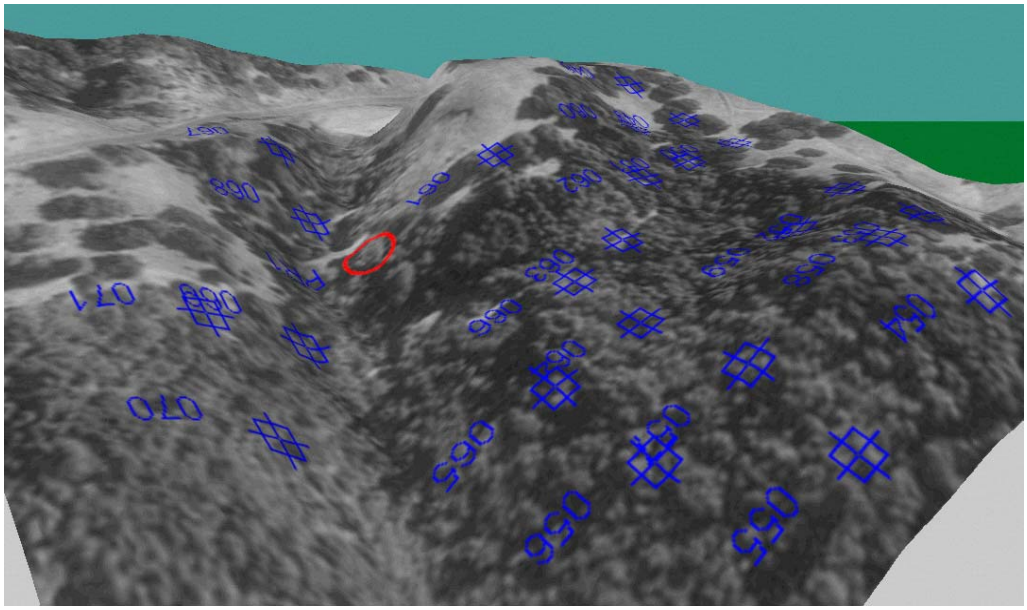

Figure 4. Three-dimensional representation (vertical exaggeration 1.5x) of the Lake Sonoma plot grid (numbered blue symbols) and the location of the *P. ramorum* infestation (red circle) detected in 2004. A park road (Rockpile Road) and a parking area (bright area near road) are visible upslope from the known infested area.

Sonoma Coast was the third location where *Phytophthora* spp. were newly isolated in 2004. *P. nemorosa* and *P. pseudosyringae* (Figure 3) were isolated from leaves of California bay, which is common at this location (Table 3). However, *P. ramorum* was not isolated from California bay leaves at this location. *P. nemorosa* and *P. pseudosyringae* have only been recognized in the last few years (Hansen et al 2003, Jung et al 2003), and it is possible that they have been present at this location for an extended time.

Tanoak mortality at Sonoma Coast was relatively high (Figures 5, 14) and bark cankers were present on about 20% of the trees (Figure 5). Most canker symptoms on tanoak at Sonoma Coast were not typical of those caused by *P. ramorum* (Figure 6). Many of the cankers that had recent bleeding in 2004 were small and did not appear to be very aggressive (Figure 6). 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 (Figure 6) and eventually sloughed off, leaving a somewhat eroded appearance to the bark surface.

No *Phytophthora* spp. were recovered from the few relatively active bleeding cankers that were observed on tanoak at Sonoma Coast. Given the low number of cankers sampled (Table 5) and the fact that many of these cankers were not at an optimal stage for isolation, it is impossible to rule out *P. nemorosa* and/or *P. pseudosyringae* as possible causes of the observed cankers. No other likely causes of the bark cankers were identified. Other pathogens observed on tanoak at Sonoma Coast included *Cryphonectria gyrosa,* which causes stem cankers but is readily recognized by its distinctive sporulation, and the root pathogens *Armillaria* spp. and *Inonotus dryadeus*.

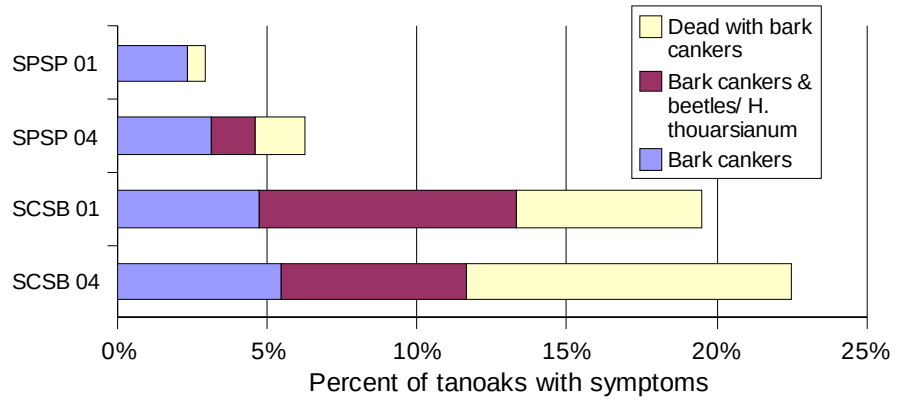

Figure 5. Incidence of symptoms in tanoak associated with an unidentified bark canker at Salt Point (SPSP) and Sonoma Coast (SCSB) in 2001 and 2004. Symptom classes are analogous to those used for *P. ramorum* canker (i.e., early, late, dead).

A small number of tanoaks at Salt Point had bark cankers similar to those seen at Sonoma Coast (Figure 5, Figure 7). 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., and no California bay trees are present within the plot grid for sampling.

We did not observe any trees with bleeding bark cankers at Modini or Foothill. Neither of these locations contained California bay trees. We did not observe any symptoms on other potential hosts, so no isolations were done at these locations. At Weston and Helen Putnam we observed a few bleeding cankers that were not typical of *P. ramorum,* and no *Phytophthora* spp. were recovered from the sampled trees (Table 6). California bay was relatively uncommon in most of the plot grid at Weston (Table 4) although it is common within the general vicinity. A few California bay leaves at Weston had possible *Phytophthora* symptoms (Figure 3), but no *Phytophthora* spp. were isolated from California bay leaves from this location. In the plot grid at Helen Putnam, California bay was represented only by a few small seedlings, and no *P. ramorum* foliar symptoms were observed on these.

P. ramorum, P. nemorosa, and *P. pseudosyringae* were only confirmed to be present at locations that also had substantial amounts of California bay within the plots (Table 4). Although California bay was present at Weston, it was a minor component of the woodland within most of the plot grid (Figure 2, Table 4). Tanoak also sustains foliar and twig infections which can produce *P. ramorum* inoculum. However, no *Phytophthora* spp. were recovered from trees at Salt Point, which had the highest tanoak density of the study locations but lacked California bay within the plot grid (Figure 2).

Figure 6. Bark cankers on living tanoak trees at Sonoma Coast. All cankers developed after the 2001 survey. Top and lower left: active cankers with recent bleeding; isolations from these cankers in 2004 were negative. Lower right – inactive canker in which the outer bark has decayed to a light colored, powdery residue.

Figure 7. Bark canker symptoms on living tanoak trees at Salt Point State Park. Upper left, cankers present in 2001, isolation negative; bottom right-new cankers since 2001, isolation negative, right - tree between plots, no information on status in 2001, isolation negative.

Overall change in disease symptom classes at locations with confirmed *P. ramorum*

The proportion of trees with *P. ramorum* canker symptoms increased at the two tanoak locations with known *P. ramorum* infestations, and in the newly expanding disease front at Annadel (Figure 8). 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* (Figure 8).

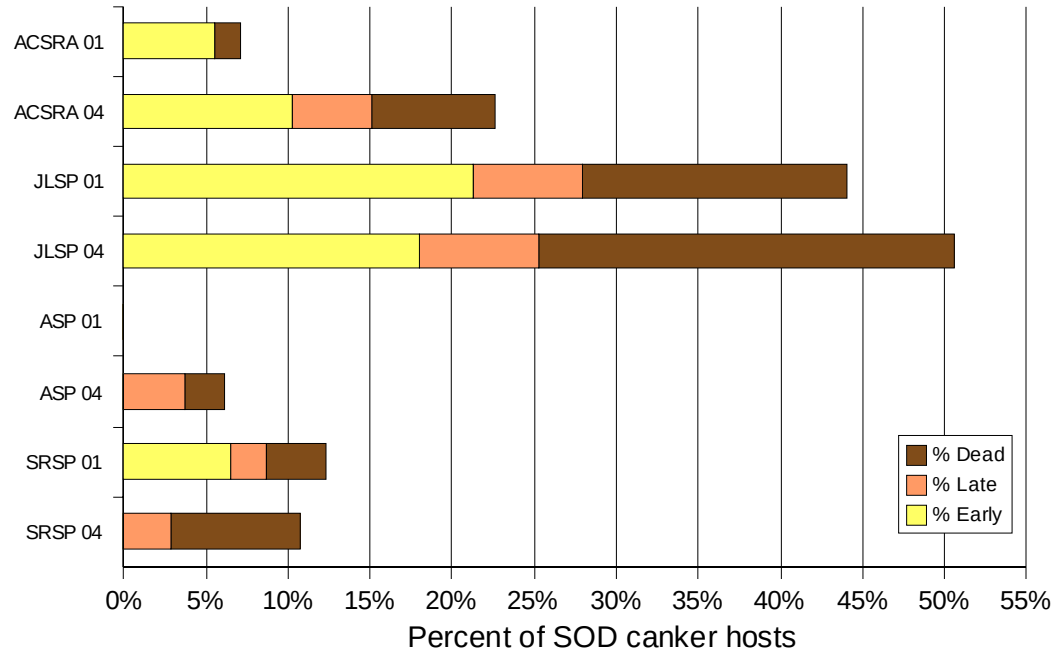

Figure 8. Changes in *P. ramorum* symptom classes between 2001 and 2004 at Austin Creek (ACSRA), Jack London (JLSP), Annadel (ASP), and Sugarloaf Ridge (SRSP). Early = bleeding cankers only; Late = cankers and associated sporulation of *Hypoxylon thouarsianum* and/or damage by wood boring beetles, primarily ambrosia beetles; Dead = entire tree killed by *P. ramorum* canker. Multistemmed trees with both live stems and stem(s) killed by *P. ramorum* are classified as having late disease symptoms.

Patterns of disease distribution and spread

Across all locations, the percentage of plots that had trees with *P. ramorum* canker symptoms increased from 22% to 38%. Plots with California black oak and tanoak showed substantial increases in the percent of plots with *P. ramorum* canker symptoms, but the percentage of plots with coast live oak that had *P. ramorum* canker symptoms decreased slightly between 2001 and 2004 (Table 7). 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.

Table 7. 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%

The four locations that had confirmed *P. ramorum* symptoms in canker hosts showed different patterns of disease across the plot grid. Differences between locations were associated with the initial extent and intensity of disease observed in the 2001 survey and the prevalence of California bay with *P. ramorum* foliar symptoms observed in 2004.

Annadel State Park

P. ramorum canker symptoms were not observed at Annadel in 2001 but were confirmed on California black oak and coast live oak and on California bay in the 2004 survey. *P. ramorum* canker symptoms were found in California black oaks in four plots on the west end of the plot grid (Figure 9) and on a coast live oak on the east end of the grid. We observed foliar *P. ramorum* symptoms on California bay in all of the plots with *P. ramorum* canker symptoms (Figure 9), in adjacent plots without symptomatic oaks, and in a few scattered plots on the east side of the grid (Figure 9). The *P. ramorum*-infected coast live oak was about 19 m from the easternmost plot with symptomatic California bay foliage.

Based on the pattern of disease at this location, it appears that the *P. ramorum* disease front is spreading outward from a disease center on the west end of the plot grid, but the disease has also recently become established on California bay in some spots toward the east end. Park trails are located near the disease center on the west and near at least one of the plots with California bay symptoms on the east end of the plot grid. Pedestrian and bicycle traffic along these trails may have played a role in the spread of the pathogen within the plot grid.

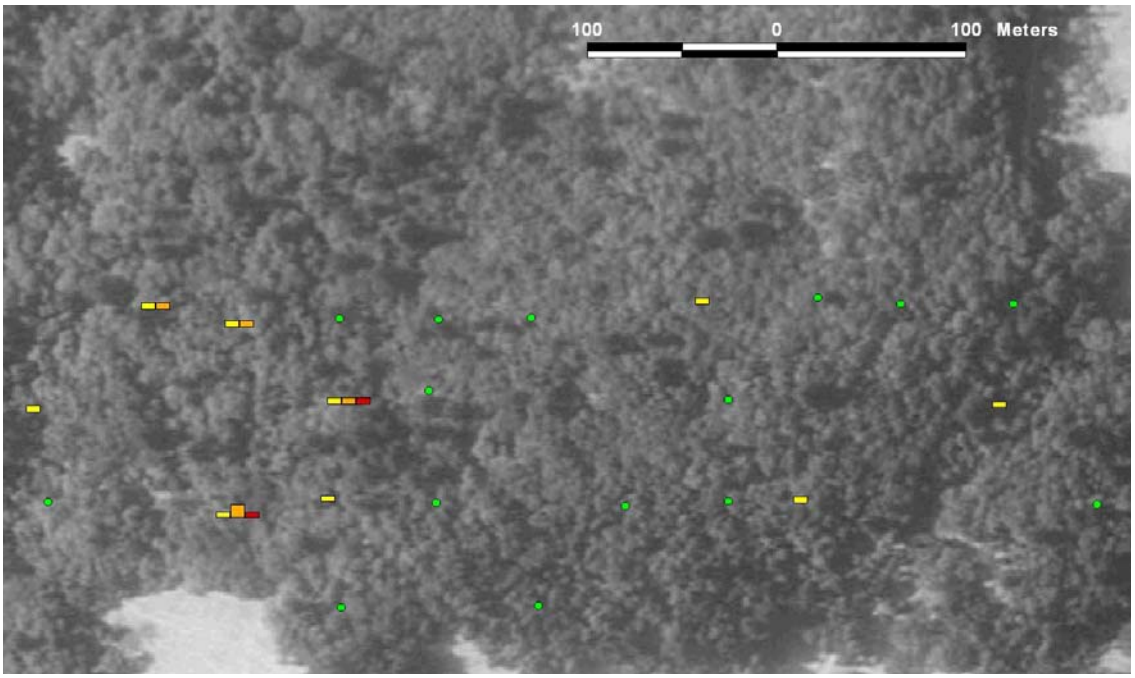

Figure 9. Distribution of *P. ramorum* symptoms in Annadel plots in 2004. Orange bars indicate the number of symptomatic SOD canker hosts and red bars indicate the number of SOD canker hosts apparently killed by *P. ramorum*. SOD canker host counts include trees in plots and additional tagged trees beyond plot edges, if any. The minimum bar height shown indicates one tree. Yellow bars indicate California bay with foliar *P. ramorum* symptoms within the plot; green circles indicate plots with asymptomatic California bay. California bay was present in all plots.

Sugarloaf Ridge State Park

P. ramorum canker symptoms and foliar symptoms on California bay were clustered in the southern portion of the plot grid at Sugarloaf Ridge (Figure 10). A single plot with both stem cankers and foliar symptoms is located on the north end of the grid. Disease spread at this location was minimal at best. All plots that had trees with *P. ramorum* canker symptoms in 2004 also had symptomatic trees in 2001. The number of symptomatic trees increased in two plots, stayed the

same in two plots, and decreased in three plots, resulting in a slight decline in the percentage of symptomatic trees overall at this location (Figure 8). Despite the low rate of new infection, disease progressed in existing infected trees, resulting in additional *P. ramorum*-related mortality in four of the seven plots with symptomatic SOD canker hosts (Figure 10, right).

The lack of new infections and the apparent remission of canker symptoms in some trees is similar to what we have observed over the same period in many of our SOD research plots in coast live oak-dominated woodlands in Marin and Napa Counties (Swiecki and Bernhardt 2004, in press). Sugarloaf Ridge is relatively dry and has a relatively open stand structure in much of the plot grid area, which may limit inoculum production and dispersal. In addition, recent inoculum production on California bay appears to have been insufficient to initiate many new stem cankers on oaks.

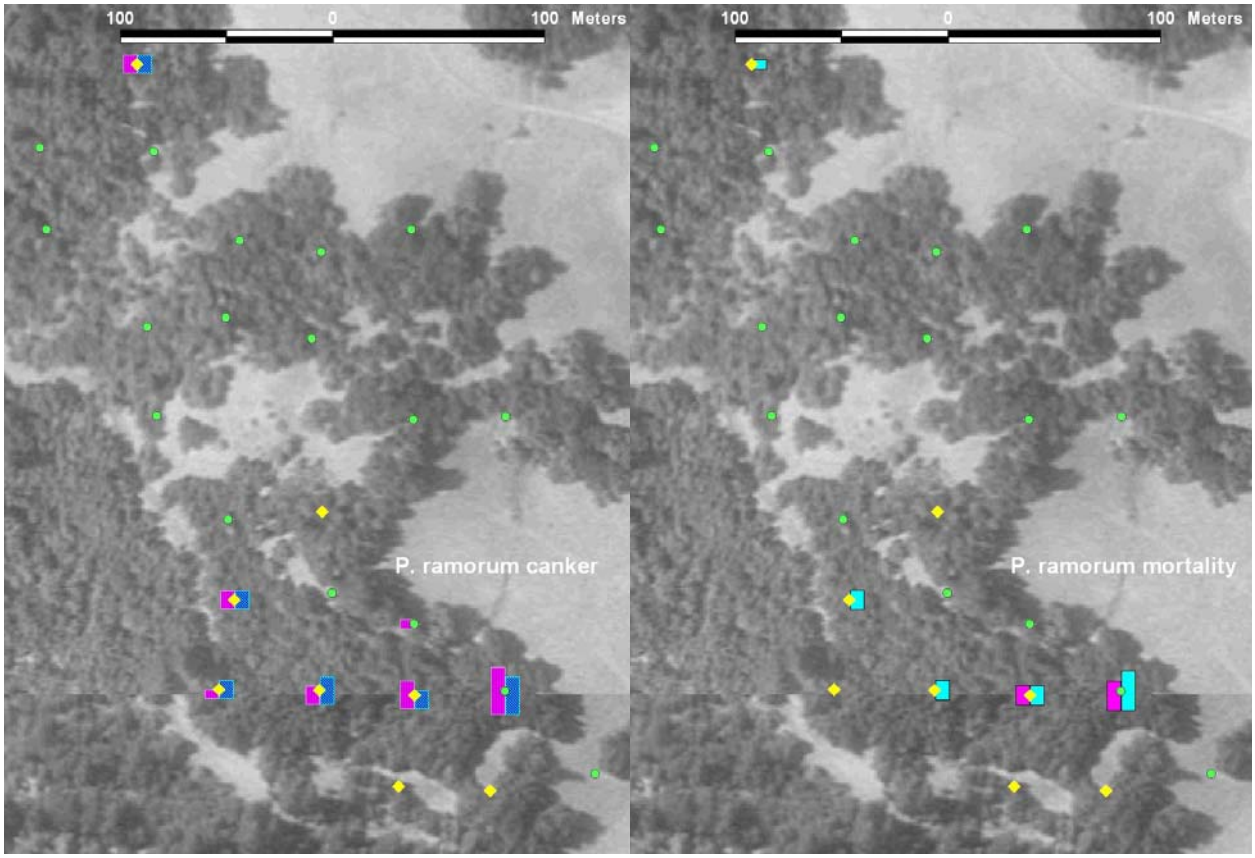

Figure 10. Distribution of *P. ramorum* symptoms in Sugarloaf Ridge plots in 2001 (magenta bars) and 2004 (blue and teal bars). Bars indicate the number of symptomatic SOD canker hosts (left) or the number of SOD canker hosts apparently killed by *P. ramorum* (right). SOD canker host counts include trees in plots and additional tagged trees beyond plot edges, if any. The minimum bar height shown indicates one tree. Green circles indicate locations of plots with asymptomatic California bay. Yellow diamonds indicate the presence of California bay with foliar *P. ramorum* symptoms within the plot. All plots had some California bay present.

A trail passes through the center of the overall plot grid, originating at the park road just south of the grid and passing by the symptomatic plot at the north end. At this site it also seems likely that trail users may have played a role in the original introduction and later spread of the pathogen.

Jack London State Park

At Jack London, *P. ramorum* canker symptoms were found throughout much of the plot grid area in 2001. This location had the highest disease incidence among the sites with *P. ramorum* canker in both 2001 and 2004 (Figure 8). Only modest increases in disease incidence were seen at this location between 2001 and 2004 (Figure 11, top).

By 2004, canker symptoms appeared in two additional plots, but were not detected in one previously affected plot, leading to a net increase of one additional plot with canker symptoms since 2001 (Figure 11, top). All plots at this location had California bay with foliar *P. ramorum* symptoms in 2004. Among plots that had trees with *P. ramorum* canker symptoms in 2001 or 2004, the number of trees with canker symptoms increased in five plots, decreased in one plot, and was unchanged in 10 plots. An additional eight plots had no symptoms on SOD canker hosts in either year, so overall, 19 of 24 plots at this location (79%) showed no new infections among SOD canker hosts between 2001 and 2004. As seen at Sugarloaf Ridge, disease progressed in many of the trees that were symptomatic in 2001, leading to additional SOD-related tree mortality in six plots by 2004 (Figure 11, bottom).

Different stages of the *P. ramorum* disease epidemic are represented at Jack London and Annadel. The *P. ramorum* epidemic in the Annadel plots is at an early stage, and, as of 2004, was probably limited by inoculum production and distribution. In comparison, *P. ramorum* inoculum was distributed throughout the Jack London plot grid area and had been for some time before the 2001 survey. At Jack London, a high percentage of the SOD canker hosts have already been infected and many have been killed. The epidemic at this location may be limited by a lack of hosts that are at a high risk of infection. At least some of the remaining nonsymptomatic SOD canker hosts may be more resistant to infection or are farther from inoculum sources. Despite differences in the stage of the epidemic at the two locations, Jack London and Annadel showed similar incremental increases in overall disease incidence between 2001 and 2004 (Figure 8).

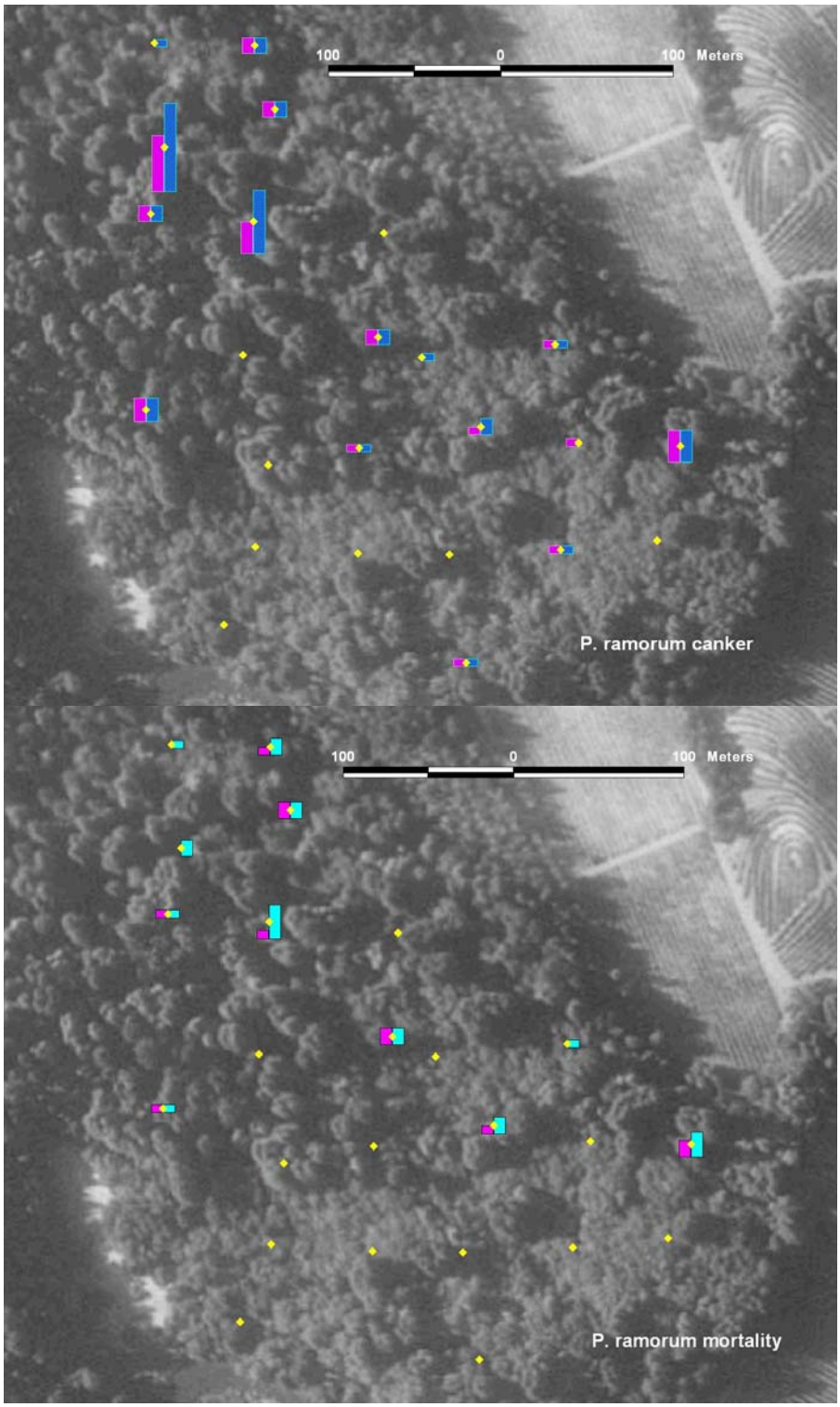

Figure 11. Distribution of *P. ramorum* symptoms in Jack London plots in 2001 (magenta bars) and 2004 (blue and teal bars). Bars indicate the number of symptomatic SOD canker hosts (top) or the number of SOD canker hosts apparently killed by *P. ramorum* (bottom). SOD canker host counts include trees in plots and additional tagged trees beyond plot edges, if any. The minimum bar height shown indicates one tree. Yellow diamonds indicate the presence of California bay with foliar *P. ramorum* symptoms within the plot. All plots had some California bay present.

Austin Creek State Recreation Area

Among the four locations with confirmed *P. ramorum* canker symptoms, Austin Creek showed the greatest increase in disease incidence between 2001 and 2004 (Figure 8). The proportion of plots with *P. ramorum* canker symptoms more than doubled from 9 of 24 (37.5%) in 2001 to 19 of 24 (79.2%) in 2004 (Figure 12, top). Among the 19 plots that had trees with *P. ramorum* canker symptoms in 2004, 15 showed an increase in the number of trees with canker symptoms and four showed no change in disease incidence (Figure 12, top). The number of symptomatic trees did not decrease in any plot. Tree mortality attributed to *P. ramorum* canker occurred in one-third of the plots between 2001 and 2004 (Figure 12, bottom).

Compared with the other three locations with confirmed *P. ramorum* canker symptoms, Austin Creek had the highest tanoak density (Figure 2) and the lowest California bay density (Figure 2, Table 4). It was the only location of these four that had plots without California bay. Furthermore, not all plots with California bay had foliar *P. ramorum* symptoms.

At this location, disease progress among the SOD canker hosts appeared to be at an exponential phase during the 2001-2004 interval. Considering results at the other locations, it seems likely that disease progress among the SOD canker hosts in a newly infected stand commonly follows 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 we suspect was the situation seen at Annadel in 2004. As the foliar disease epidemic spreads throughout a stand, disease incidence can increase rapidly under favorable conditions, as seen at Austin Creek. However, once most of the trees with the highest disease risk have been infected, the rate of new infections tails off, as seen at Jack London. 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 are relatively unfavorable for disease development.

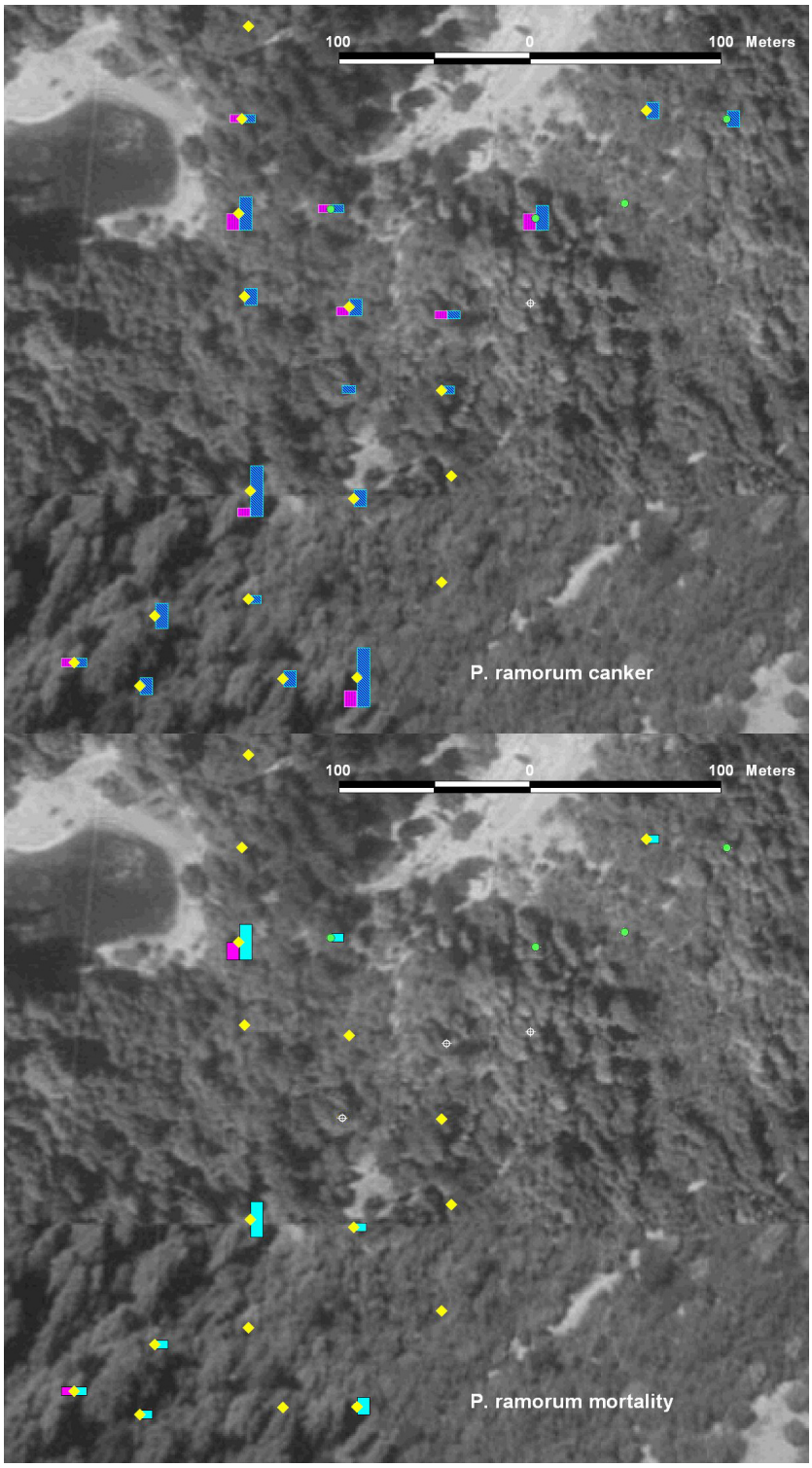

Figure 12. Distribution of *P. ramorum* symptoms in Austin Creek plots in 2001 (magenta bars) and 2004 (blue and teal bars). Bars indicate the number of symptomatic SOD canker hosts (top image) or the number of SOD canker hosts apparently killed by *P. ramorum* (bottom image). SOD canker host counts include trees in plots and additional tagged trees beyond plot edges, if any. The minimum bar height shown indicates one tree. Yellow diamonds indicate the presence of California bay with foliar *P. ramorum* symptoms within the plot. Plots with white ⌖ symbol did not have California bay present.

Overall changes in tree mortality

Between 2001 and 2004, mortality increased among all SOD canker hosts (Figure 13) and the percentage of plots with mortality also increased (Table 8). Between the 2001 and 2004 evaluations, the number of dead California black oak trees more than doubled, whereas coast live oak and tanoak showed smaller relative increases in mortality (Figure 13). *Phytophthora ramorum* contributed to the increase in mortality in each species, accounting for 27%, 49%, and 30% of the increase in mortality for California black oak, coast live oak, and tanoak, respectively. Among all mortality rated in 2004 (i.e., trees estimated to have died after 1991), *P. ramorum* accounted for 17% of the dead California black oaks, 29% of the dead coast live oaks, and 27% of dead tanoaks.

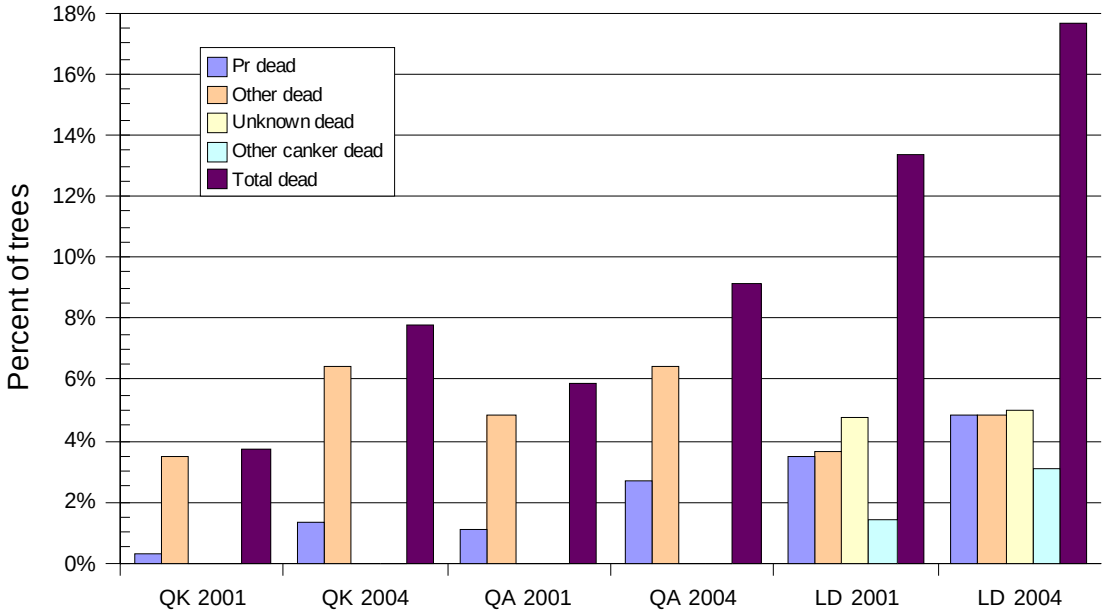

Figure 13. Changes in mortality levels among California black oak (QK), coast live oak (QA) and tanoak (LD) in 2001 compared to 2004. Pr dead= dead due to *P. ramorum*; Other dead= dead due to various causes, mostly wood decay fungi and root disease; Unknown dead=dead trees that could not be assigned to other groups with confidence; Other canker dead=dead tanoak trees affected by a bark canker other than *P. ramorum*, noted only at Sonoma Coast and Salt Point.

Table 8. Percent of plots with dead trees of SOD canker host species in 2001 and 2004.

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

Mortality at different study locations

Levels of mortality due to both *P. ramorum* and other agents varied between study locations. Tree mortality due to *P. ramorum* canker greatly exceed mortality due to other causes at both Austin Creek and Jack London (Figure 14). 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 within the past 3 years, mortality due to other causes was still much more prevalent than mortality due to *P. ramorum*. As shown in Figure 5, high levels of

mortality at Sonoma Coast, and to a lesser degree at Salt Point, were associated with the prevalence of a bark canker apparently caused by agent(s) other than *P. ramorum*.

The overall mortality percentages shown in Figures 13 and 14 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 recorded between the 2001 and 2004 surveys is observed mortality over a three-year period and does not include uncertainties associated with the original 2001 mortality estimates. We used both the 2001-2004 observed mortality and the 1991-2001 estimated mortality rates to calculate annual mortality rates for all locations (Figure 15). Two locations, Foothill and Helen Putnam, had no new mortality among SOD canker hosts between 2001 and 2004 (Figures 14, 15).

In all locations, some tree mortality was associated with wood decay fungi, especially canker rot fungi, and root disease. This mortality, which excludes mortality due to *P. ramorum* canker and the unknown canker at Sonoma Coast and Salt Point, is referred to as background mortality. Estimated annual background mortality rates for the 2001-2004 interval were close to or somewhat higher than those for the 1991-2001 interval. Overall, the background mortality rate was about 1% or less per year for both time intervals at all locations. Given the overall sample size, the differences in background mortality rates between and within locations were not significant (Figure 15).

Annualized *P. ramorum* mortality rates were higher for 2001-2004 than for 1991-2001 at all four locations with *P. ramorum* canker (Figure 15). For these four locations, the overall annual mortality rate due to *P. ramorum* canker for the period 2001-2004 (2.03% per year) was significantly greater than for 1991-2001 (0.42% per year; likelihood ratio $p=0.02$). Given the low rate of new infections at Sugarloaf Ridge (Figure 10) and Jack London (Figure 11), it is likely that annualized *P. ramorum* mortality rates will decrease at these locations over the next few years. In contrast, due to new infections at Austin Creek (Figure 12) and Annadel (Figure 9), annualized *P. ramorum*-related mortality rates are likely to stay constant or increase over the next few years.

As shown in Figure 15, recent (2001-2004) 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 2001-2004 that could not be definitively assigned to a given cause (unknown bar in Figure 15). 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. If most of the cankers at this location are actually caused by *P. nemorosa* and /or *P. pseudosyringae,* both of which occur on California bay at this location, these data 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 is common at Sonoma Coast, but absent at the Salt Point study location.

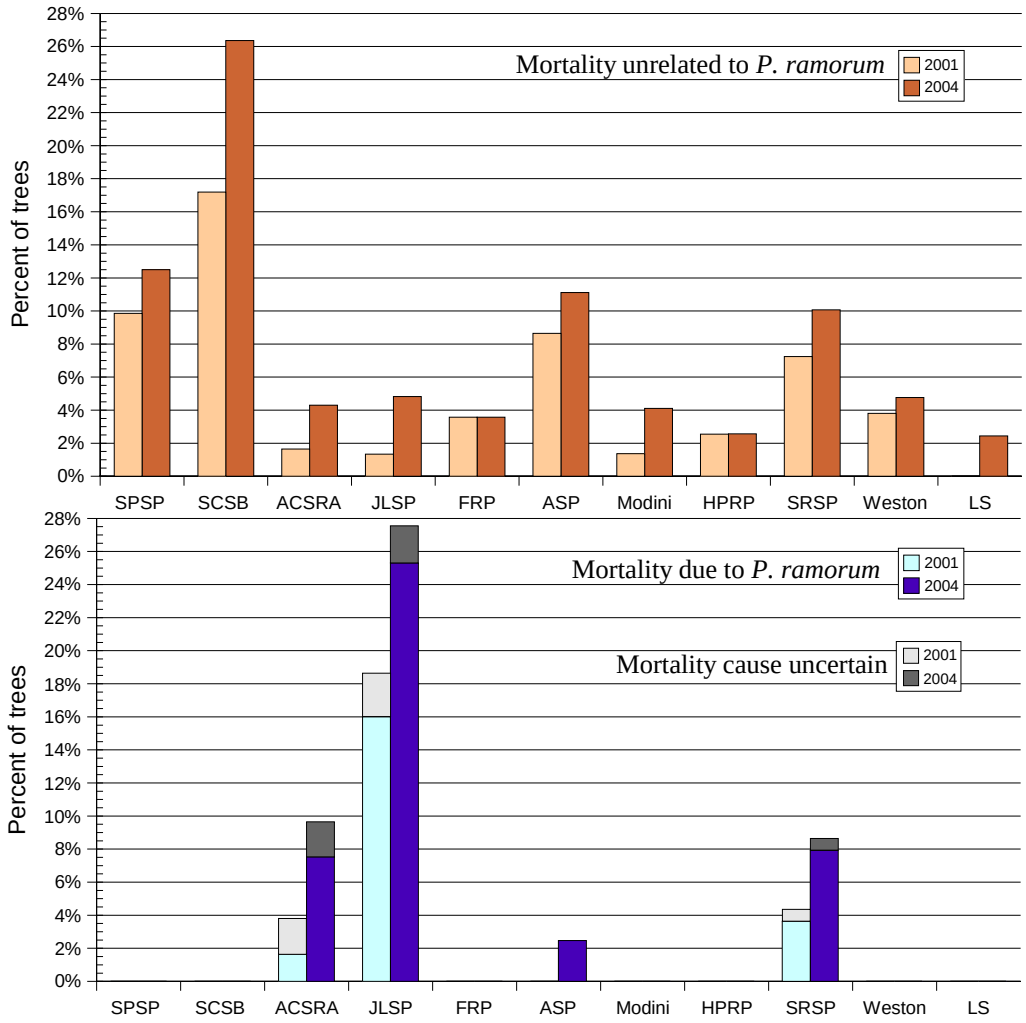

Figure 14. Overall mortality among SOD canker host trees due to *P. ramorum* (bottom) and other causes (top) in 2001 and 2004 by location. Mortality includes trees rated in the 2001 survey as having died within the previous 10 years, i.e., mortality since 1991. Location abbreviations are shown in Table 1.

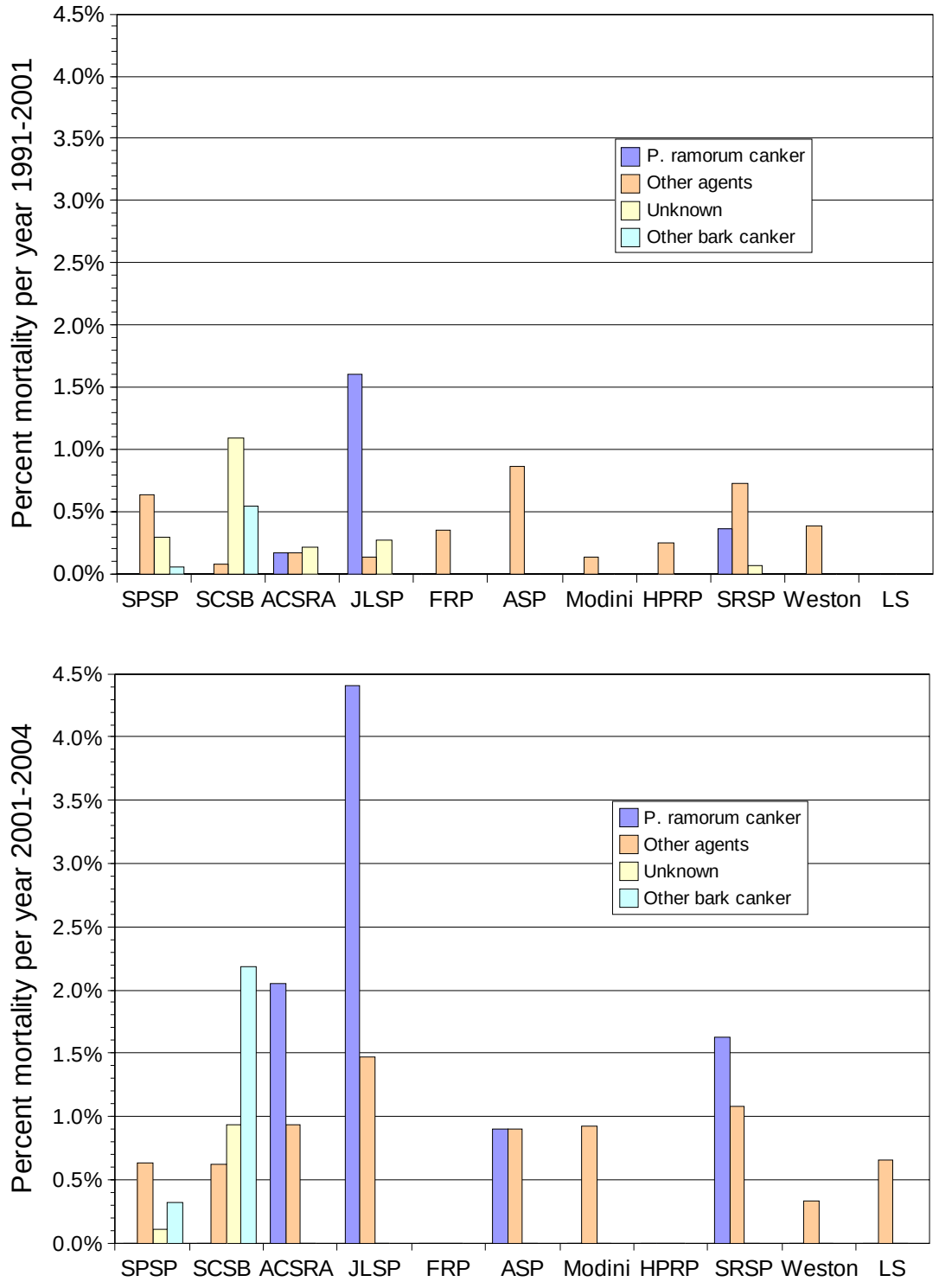

Figure 15. Annualized mortality rates (percent mortality per year) of SOD canker host trees by cause and study location for the periods 1991-2001 (top) and 2001-2004 (bottom). Location abbreviations are shown in Table 1.

Tree decline due to agents other than *P. ramorum*

As noted above, background mortality in the sampled stands is associated with a variety of pathogens, most of which are presumed to be native or long naturalized. Most of these agents are wood decay fungi, and trees affected by these pathogens tend to decline noticeably for a number of years before dying. We scored trees as being in decline if their condition appeared due to wood decay fungi and was poor enough that tree death appeared likely to occur within 10 years.

The overall incidence of decline due to these native pathogens was 16% among the SOD canker hosts. The incidence of decline differed significantly between species and study locations (likelihood ratio $p < 0.0001$). The incidence of decline was similar for California black oak (19%) and coast live oak (22%), but was much lower in tanoak (8%). The highest incidences of decline were seen at Sugarloaf Ridge (27%) and Jack London (25%). Decline was lowest at tanoak-dominated Sonoma Coast (7%), but this location had high amounts of disease and mortality associated with the unidentified canker disease (Figures 5, 14).

The percent of declining trees decreased by about 3% to 4% for all three SOD canker hosts between 2001 and 2004. The reduction in the number of declining trees was almost entirely due to the mortality of declining trees. Given that the expected survival of declining trees is 10 years or less by definition, it is not surprising that a number of these trees died between the two surveys. In addition, very few trees that were rated as asymptomatic in 2001 were rated as declining in 2004. This is consistent with the fact that most trees affected by canker rot and other wood decay fungi show a gradual deterioration in condition, so that most trees in good or "pre-decline" condition in 2001 would be unlikely to be characterized as in decline by 2004.

Tree failures

In 2004, we recorded data on failures of SOD canker hosts in plots that were estimated to have occurred within the previous five years. All bole and root failures were recorded. Branch failures were recorded only if the failed branch was 20 cm or greater in diameter at the point of the break.

Overall five-year failure rates for California black oak (11.5%) were significantly higher than for tanoak (7.4%, likelihood ratio $p = 0.03$) or coast live oak (5.1%, likelihood ratio $p = 0.0007$). Bole failures were the most common failure type among tanoaks and coast live oaks, whereas large branch failures and bole failures were equally common in California black oak (Figure 16). Branch failures were most common on old, large diameter California black oaks and coast live oaks and were normally associated with canker rot fungi or other wood decay fungi. Among trees with scored branch failures, the average trunk DBH was 59 cm ($sd = 47$, $n = 8$) for coast live oak and 50 cm ($sd = 18$, $n = 28$) for California black oak. These mean diameters are significantly greater (t-test $p < 0.01$) than the average trunk DBH for all trees without failures (37 cm, $sd = 17$, $n = 184$ for coast live oak; 39 cm, $sd = 20$, $n = 185$ for California black oak). Stem diameters include only the largest trunk for multistemmed trees.

Root diseases caused by *Armillaria*, *Ganoderma*, and possibly other unidentified fungi were commonly associated with root failure. At Sonoma Coast, which has a relatively high frequency of root failures (Figure 17), we observed *Inonotus dryadeus* fruiting at the base of a large tanoak between plots. In addition to root pathogens, at least four root failures were partially or wholly related to unstable soils. In several steep plots at Sonoma Coast, root failures were clustered because soil structural failure associated with an initial root failure precipitated the failure of adjoining trees. No root failures were seen in coast live oak, which is consistent with the low rate

of root failure observed in our previous study on failure in this species (Swiecki and Bernhardt 2003).

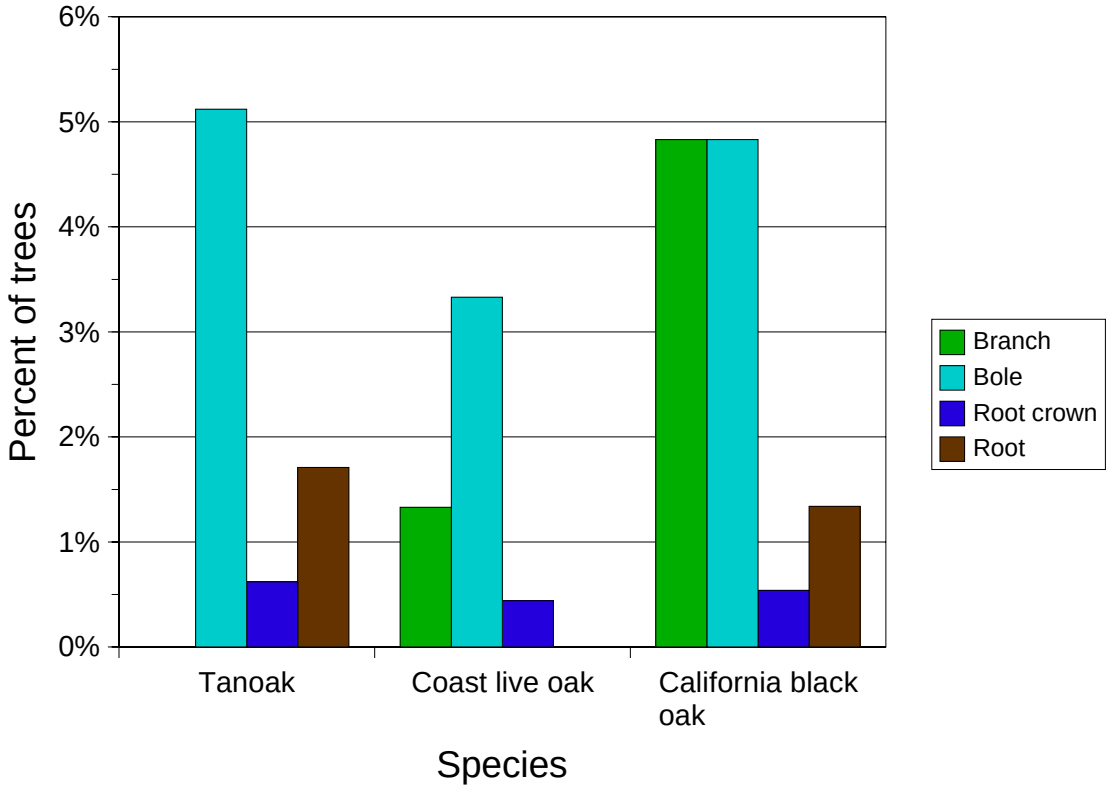

Figure 16. Frequency of different failure types in SOD canker hosts at all study locations. All failures are estimated to have occurred between 1999 and 2004 and include all bole, root crown and root failures and branch failures with a minimum diameter of 20 cm at the break.

The highest recent failure rates were observed at Sonoma Coast and Jack London (Figure 17), where tanoak was the dominant canker host. High failure rates were also seen at Annadel and Modini, where black oak is the most common canker host. The lowest failure rates, less than 5% (Figure 17), were observed at Salt Point, Helen Putnam, and Foothill, where the dominant canker hosts were tanoak, coast live oak, and California black oak, respectively (Figure 1). Compared with other locations, these three locations were dominated by relatively young, small-diameter trees.

The relationship between failure and *P. ramorum* canker differed by species. None of the California black oak failures occurred among the 14 trees with *P. ramorum* canker, even though 5 of these trees were dead in 2004. However, 11 of these 14 California black oaks developed *P. ramorum* canker symptoms after 2001 and 4 of the five dead trees died after 2001. Hence, these trees may not have been dead long enough to develop a high failure potential.

In contrast, 8 of the 17 (46%) coast live oaks with *P. ramorum* canker symptoms had failed by 2004. Seven of the eight failed coast live oaks were killed by *P. ramorum* canker before they failed. The failure rate among symptomatic coast live oaks was significantly higher (likelihood ratio $p < 0.0001$) than the 3.5% failure rate seen among coast live oaks without *P. ramorum* canker symptoms. This elevated failure rate associated with *P. ramorum* canker is consistent with results

from another study on SOD-affected coast live oaks in Marin County (Swiecki and Bernhardt 2003, Swiecki et al in press).

In tanoak, the failure rate among trees with *P. ramorum* canker symptoms (9/73 = 12.3%) was not significantly higher than the overall failure rate among trees without *P. ramorum* canker (39/572 = 6.8%). However, the pool of tanoaks without *P. ramorum* canker includes trees at Sonoma Coast, which has a high incidence of an unidentified canker disease (Figure 5). The high failure rate among tanoaks at Sonoma Coast (Figure 17) was at least partly associated with this other stem canker disease. If the analysis of tanoak failures is restricted to the two tanoak locations with confirmed *P. ramorum* canker (Jack London and Austin Creek), the failure rate among tanoaks with *P. ramorum* canker symptoms (9/73 = 12.3%) is significantly higher than the failure rate among asymptomatic tanoaks (1/92 = 1.1%; likelihood ratio p = 0.0017). Eight of the nine failures in tanoaks with *P. ramorum* canker occurred in trees that were dead at the time of failure.

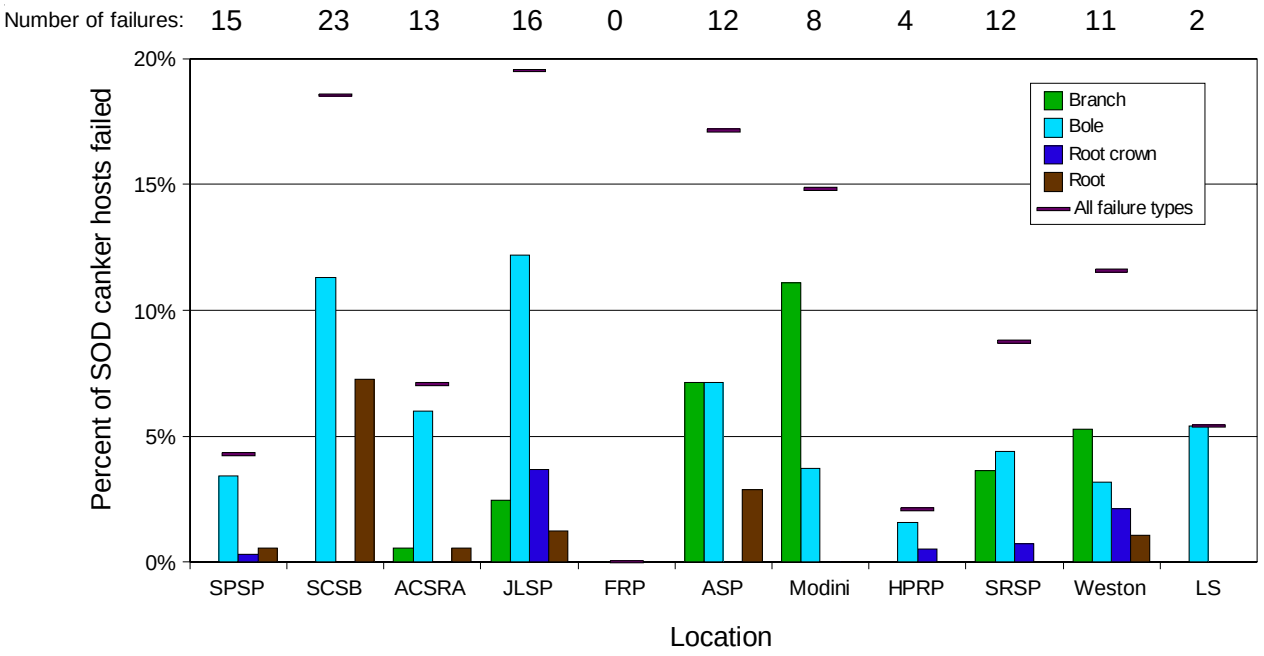

Figure 17. Percent of failed SOD canker hosts (tanoak, coast live oak, and California black oak) at study locations by failure type. All failures are estimated to have occurred between 1999 and 2004 and include branch failures with a minimum diameter of 20 cm at the break and all bole, root crown and root failures. The number of failures at each location are listed above the top line of the graph.

Wood decay was rated as contributing to failure in 96% of the observed failures, and was the primary factor related to failure in all three species (Table 9). Cavities and structural defects were more commonly associated with failures in the oaks than in tanoak (Table 9). Beetle boring was present in 52% of the failed trees overall, but was more common in failed coast live oaks than in failed tanoaks or California black oaks (Table 9). Although it was commonly associated with failures, beetle boring was seldom extensive enough to be considered a factor that contributed to the observed failures. Four of the six failures in which beetles were rated as a contributing factor occurred in trees killed by *P. ramorum* canker.

Canker rot symptoms were present in the overwhelming majority of failed oaks, but were only occasionally observed in tanoak (Table 9). Fruiting of the canker rot fungus *Inonotus andersonii* was associated with 14% of the failures in the oak species, but was not observed on tanoak. *Hypoxylon thouarsianum* was the most commonly observed wood decay fungus overall (Table 9),

and was associated with 11 of the 17 failures that occurred in trees with *P. ramorum* canker. Other wood decay fungi associated with tree failures included *Armillaria gallica*, *Ganoderma applanatum*, *G. lucidum*, *Hericium erinaceus*, *Inonotus dryophilus,* and *Laetiporus gilbertsonii*.

Table 9. Factors contributing to or associated with observed failures in tanoak, coast live oak, and California black oak

	Tanoak	Coast live oak	California black oak
Number of trees with failures	48	23	43
Factors contributing to failure:	Percent of failures		
Decay	98	100	93
Cavities	4	22	37
Beetle boring	2	22	0
Structural defects	4	13	12
Agents associated with failure:	Percent of failures		
Canker rot	8	74	88
Hypoxylon thouarsianum	25	61	12
Inonotus andersonii	0	17	12
Phellinus spp.	10	13	7
Beetles	54	87	30

Overall, factors associated with failure in coast live oak were similar to what we previously reported for a large sample of coast live oaks from SOD-affected woodlands in Marin County (Swiecki and Bernhardt 2003, Swiecki et al in press). Failure patterns in California black oak appear to be similar to those seen in coast live oak, with a few differences, such as the higher frequency of root failure (Figure 16) and the lower frequency and importance of beetle boring (Table 9) Some of these differences are probably associated with the fact that failures of California black oak affected by *P. ramorum* canker have not yet been observed in these plots. Tanoak failures appear to differ in several ways from failures seen in the two oak species. These differences (Figure 16, Table 9) probably reflect differences between tanoak and the two oak species with respect to tree growth form, forest types, and associated decay organisms.

Regeneration

Mortality due to disease or other factors has the potential to have long-term effects on forest species composition. All of the stands we studied had multiple tree canopy species, and gaps formed through tree mortality could be filled by a number of different species, possibly resulting in a shift in the dominance of canopy species. To examine these relationships, we looked at regeneration of SOD canker hosts in 2001 and 2004.

The number of plots with seedlings (plants less than 3 cm DBH) of the three SOD canker hosts did not change significantly between 2001 and 2004 (Figure 18). Actual seedling counts within plots showed more variation between 2001 and 2004, with the largest changes seen in coast live oak (Table 10). The number of plots that lost coast live oak seedlings over this period was more than twice the number of plots that gained coast live oak seedlings (Figure 19), leading to an overall decrease in the mean number of coast live oak seedlings per plot (Table 10). Numbers of plots that gained and lost tanoak and California black oak seedlings were similar, and the average numbers of tanoak and California black oak seedlings did not change significantly between 2001 and 2004.

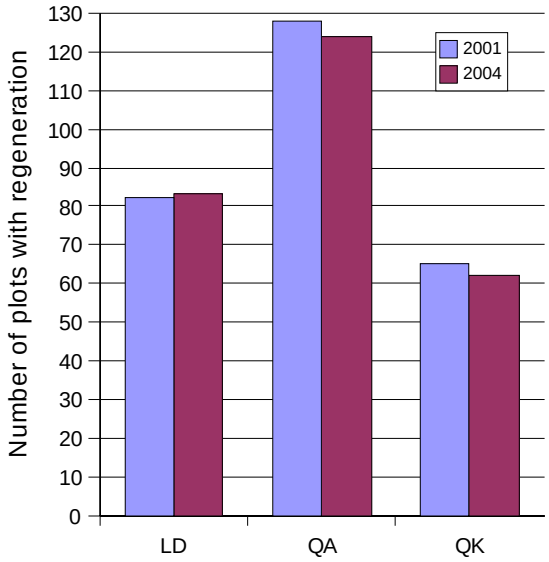

Figure 18. Number of plots with seedlings (<3 cm DBH) of tanoak (LD), coast live oak (QA), California black oak (QK) and in 2001 and 2004.

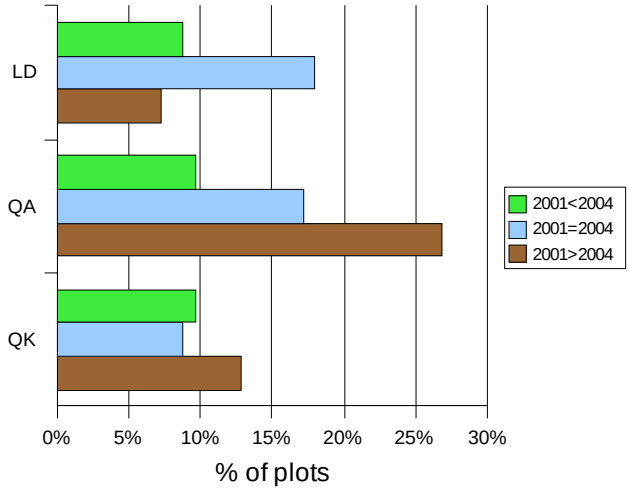

Figure 19. Percent of plots of coast live oak (QA), California black oak (QK) or tanoak (LD) that showed increases (2001<2004), decreases (2002>2004) or no change (2001=2004) in the number of seedlings (<3 cm DBH) in the two surveys. Totals exclude plots lacking seedlings in both 2001 and 2004.

Table 10. Mean counts of seedlings (<3 cm DBH) per plot by species in plots that had seedlings of the species present in 2001 and/or 2004.

Species	Plots	2001 mean seedlings/plot (SD)	2004 mean seedlings/plot (SD)
Tanoak	85	22.0 (19.0)	23.1 (20.7)
Coast live oak[1]	119	19.3 (26.9)	11.5[2] (18.1)
California black oak	78	3.7 (8.0)	2.7 (4.1)

[1]Coast live oak means exclude 15 plots with estimated regeneration counts greater than 100 per plot in 2004. Estimated counts in these plots were the same in both 2001 and 2004.

[2] Significantly different from 2001 mean at p<0.0001 (two-tailed paired t-test).

Tanoak seedlings were present in a range of size classes, but much of this regeneration consisted of "seedling-sprouts" (Burns and Honkala 1990). These are small shrubby plants that form from seedlings whose shoots have periodically died back to the ground and been replaced by new

shoots arising from the root crown. Most of these seedling-sprouts are no more than 30 to 60 cm tall and many are multistemmed. The number of tanoak seedlings and seedling-sprouts per plot was relatively high overall (Table 10). Among plots with tanoak regeneration, only 10% had nine or fewer and 10% had 60 or more seedlings and seedling-sprouts.

Coast live oak and California black oak advance regeneration typically consists of much smaller seedlings. Many of these persist for multiple years, which is why seedlings counts in many plots did not change between 2001 and 2004. California black oak seedlings were not present in high numbers in any of the surveyed plots in 2004 and numbers of seedlings did not change much between the two surveys in most plots. The highest number of California black oak seedlings per plot in 2004 was 24 and only 10% of the plots with California black oak regeneration had more than 15 seedlings. However, two plots at Weston had an estimated 50 seedlings in 2001, which were reduced to counts of 15 and two by 2004.

Coast live oak seedlings counts were much more variable than California black oak seedling counts. In 2004, 15 plots had coast live oak seedlings counts between 100 and 1000. Fourteen of these 15 plots were at Helen Putnam, which had exceptionally high numbers of coast live oak seedlings compared to other locations. Coast live oak seedling counts also showed the greatest decreases between 2001 and 2004; 18 plots showed losses of between 20 and 100 seedlings over this period. It appears that large numbers of coast live oak seedling can become established in years with high acorn production. Many of these new seedlings do not persist long, leading to wide fluctations in coast live oak seedling counts over time.

Estimated mortality levels among tanoak regeneration were similar in 2001 and 2004. Twenty two plots contained dead tanoak regeneration in 2004 compared to 18 plots in 2001. Dead coast live oak seedlings were observed in five plots in 2004 compared to six plots in 2001. No dead California black oak seedlings were observed in either 2001 or 2004.

In a strict sense, regeneration involves replacement of existing trees that die. For oaks and tanoaks, most of this replacement occurs via persistent seedlings or advance regeneration that has become established in the understory but is typically not released until a gap in the canopy is created or suppressive effects of overstory trees are otherwise reduced. We examined the balance between understory seedlings and mortality of the same species within plots to determine whether seedlings of SOD canker hosts were indeed present where they were needed to replace trees killed by *P. ramorum* canker or other agents (Figure 20).

Although 67% of plots with tanoak had one or more dead trees, all of these plots also had tanoak seedlings. If these seedlings survive and can be recruited to the tree size class, mortality of overstory trees could be replaced. Due to increased mortality of tanoak trees in 2004 compared with 2001, a greater percentage of tanoak plots currently need to have existing seedlings grow into trees in order to maintain existing stand density. This has happened to some extent as 14 new tanoak trees were added to plots because their DBH had increased by 2004 to over 3 cm.

In comparison, only 25% of the plots with coast live oaks had one or more dead trees in 2004, a slight increase from 2001. Coast live oak regeneration was present in 75% of these plots. Overall, 6.3% of all coast live oak plots had recent mortality but no regeneration of this species. The proportion of plots with coast live oak mortality but lacking regeneration had increased since 2001 (Figure 20).

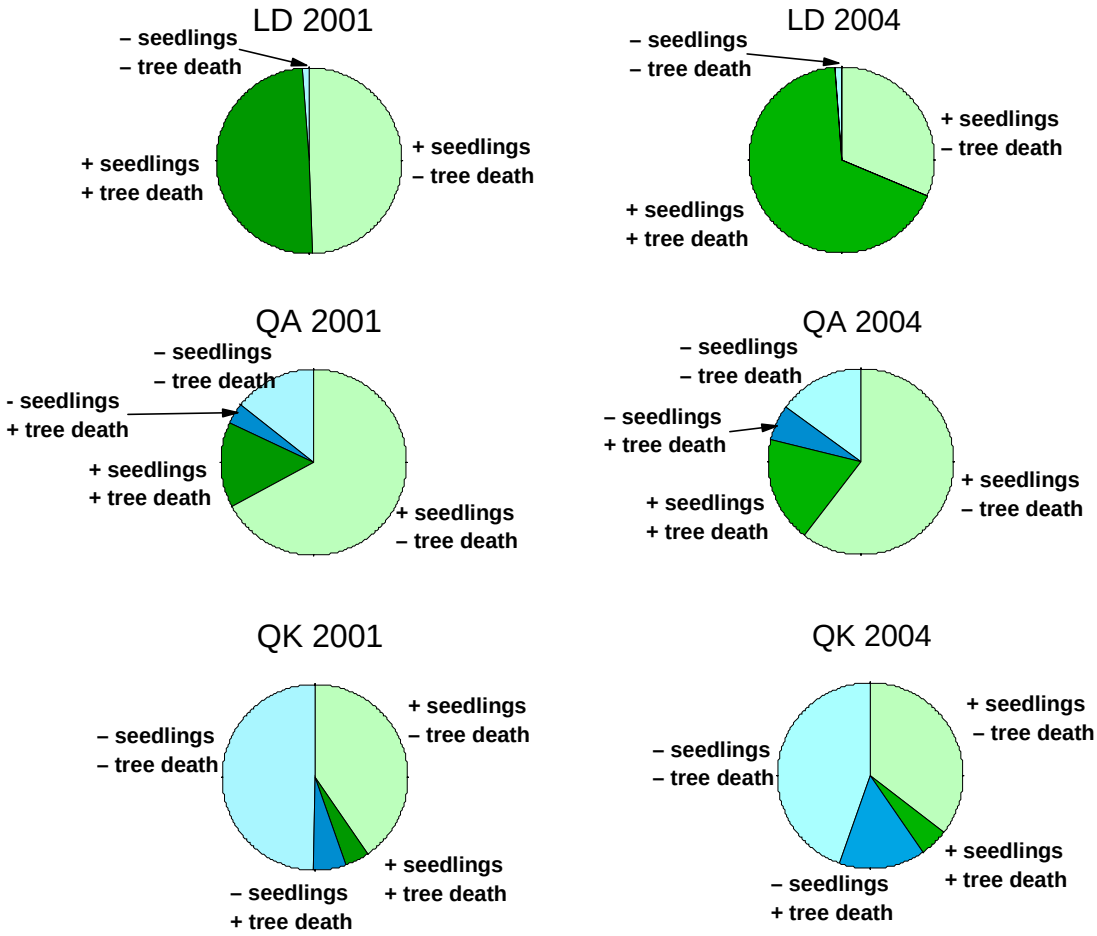

Figure 20. Percent of plots with and without tree mortality and regeneration of the same species in 2001 and 2004. LD=tanoak (73 plots), QA=coast live oak (114 plots), QK=California black oak (119 plots). Plots with regeneration are shown in green; plots without regeneration are shown in blue. Plots with dead trees are shown in dark shades and those without tree death are shown in lighter shades.

The regeneration situation was much worse for California black oak. In both 2001 and 2004, fewer than half of the plots with California black oak had seedlings of this species (Figure 20). As the amount of California black oak tree mortality has increased between 2001 and 2004, the percentage of plots with mortality and lacking regeneration has increased from 6% to 15%. Only 25% of the plots with dead California black oak trees had regeneration of this species.

For other forest canopy species, regeneration in plots was not counted but only noted as present or absent. These data did not indicate any significant changes between 2001 and 2004 with respect to the presence of regeneration of the non-canker host canopy species. California bay regeneration was the most common of these species, occurring in 64% of the study plots. Other species frequently encountered in the regeneration size class included Douglas-fir (49% of plots), madrone (28% of plots), coast redwood (12% of plots), and bigleaf maple (9% of plots), all of which are susceptible to various types of foliar or twig blight caused by *P. ramorum*.

The regeneration data gathered to date indicate that among the SOD canker hosts, California black oak populations are currently at greatest risk of decline as the result of non-replaced mortality in Sonoma County woodlands. Due to sparse seedling production by California black oak, even low levels of mortality due to *P. ramorum* is likely to result in long-term losses in density of this

species. Furthermore, current levels of regeneration appear to be inadequate to offset tree mortality due to agents other than *P. ramorum*.

In contrast, even though tanoak seedlings are susceptible to *P. ramorum* canker, there is no evidence to date that *P. ramorum* has depleted tanoak regeneration in the understory in these plots. However, continued long-term monitoring will be needed to determine whether tanoak seedlings will be able to recruit to the tree size class in areas with high levels of *P. ramorum* inoculum. Small diameter tanoak stems are susceptible to *P. ramorum* (Rizzo et al 2002). Ultimately, the ability of tanoak to regenerate in areas affected by *P. ramorum* may depend on the prevalence of resistant genotypes within the pool of advance regeneration.

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