EVALUATING AND MONITORING EFFECTS OF *PHYTOPHTHORA RAMORUM* CANKER (SUDDEN OAK DEATH) IN SONOMA COUNTY WOODLANDS AND FORESTS

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right - First author using GPS receiver with mast-mounted antenna to locate plot coordinates in black and coast live oak dominated woodland at Weston Ranch.

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EXECUTIVE SUMMARY

The recently described plant pathogen *Phytophthora ramorum* causes extensive bark cankers in coast live oak, black oak, and tanoak that are associated with premature mortality of these species. This bark canker disease, which is commonly referred to as "sudden oak death" (SOD) has the potential to severely impact many of the ecologically important woodlands and forests of Sonoma County. In this project, we established permanent research/monitoring plots in woodland and forest types at risk from *P. ramorum* canker to monitor disease progression and assess impacts of disease over time. We also assessed tree decline and mortality due to other agents.

We established a total of 250 fixed-area plots (0.02 ha each) at 11 study locations in various portions of Sonoma County. SOD host trees (i.e., coast live oak, black oak, and tanoak) lacked any symptoms of *P. ramorum* canker at four locations. Four locations had trees with SOD symptoms, but the presence of *P. ramorum* at these locations has not yet been confirmed by laboratory analysis. Three locations with SOD symptoms had preexisting laboratory confirmations for *P. ramorum*'s presence.

Among the seven locations with likely *P. ramorum* symptoms, apparent SOD infection rates ranged from 3% to 45%. Symptoms of *P. ramorum* canker were most common on tanoak and least common on black oak. For coast live oak but not tanoak, SOD symptoms were significantly more common among overstory trees than understory trees.

Among the 11 study locations, the incidence of decline and mortality of SOD hosts from agents other than *P. ramorum* ranged from 17% to 33%. In most locations, decline and mortality from these agents, mostly native wood decay fungi, is currently more important than SOD. However, we observed relatively little overlap between trees affected by *P. ramorum* and trees declining as a result of other pathogens. If *P. ramorum* is less likely to affect trees that are already in decline due to other factors, the overall impact of SOD will be increased in stands with high background levels of decline and mortality.

Seedlings of tanoak and coast live oak are typically present in the understory beneath these species. This increases the chances that tanoak and coast live oak mortality may be replaced by the same species. However, disease and competition with seedlings of other tree species may inhibit regeneration of tanoak and coast live oak. Black oak seedlings are relatively uncommon beneath black oak canopy. Black oak regeneration appears to be insufficient to maintain current densities at the study locations, including those in which *P. ramorum* is not currently a problem.

INTRODUCTION

The plant pathogenic water mold *Phytophthora ramorum* has been associated with elevated levels of mortality in tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and California black oak (*Q. kelloggii*) trees in a number of California coastal counties over the past few years (Garbelotto et al 2001). This pathogen causes bleeding bark cankers on the bole of these species and the uncommon Shreve oak (*Q. parvula* var. *shrevei)*. As these bark cankers expand and coalesce, they eventually girdle affected trees. *P. ramorum*-infected trees are commonly colonized by the sapwood-decaying fungus *Hypoxylon thouarsianum*, oak bark beetles (*Pseudopityophthorus* spp.), and ambrosia beetles (*Monarthrum* spp.) as they decline. Although the canker disease has been named "sudden oak death" (SOD), the time interval between the onset of visible disease symptoms and tree death may commonly be more than a year.

To date, bleeding stem cankers caused by *P. ramorum* have only been observed in the field on tanoak and oaks in the black oak subgenus, including coast live oak, California black oak, and Shreve oak. However, the pathogen also causes leaf spots and/or twig cankers on a number of native plants commonly associated with oaks and tanoak, including California bay (*Umbellularia californica*), madrone (*Arbutus menziesii*), huckleberry (*Vaccinium ovatum*), California buckeye (*Aesculus californica*), bigleaf maple (*Acer macrophyllum*), manzanita (*Arctostaphylos manzanita*), and other species. Stem cankers that occur on live oak and black oak are probably initiated by inoculum produced on at least some of these other hosts. At this time, there is no evidence to suggest that *P. ramorum* is transmitted directly from one oak to another. In tanoak, however, *P. ramorum* causes leaf and twig infections. Inoculum produced on these surfaces might contribute to cankers on the main stems of tanoak. The epidemiology of this disease is still under investigation.

Tanoak trees exhibiting symptoms consistent with SOD were reported in Marin County, CA in 1995 (Svihra 2001). By autumn 2000, SOD had become widespread in portions of Santa Cruz, Marin, Monterey, Napa, and Sonoma Counties. At the current time (December 2001), trees infected with *P. ramorum* have been confirmed in Solano, Alameda, and Mendocino counties and in southern Oregon. It is not clear how widely this disease will eventually spread, but further spread appears likely.

To provide information on how *P. ramorum* is likely to affect Sonoma County woodlands and forests, we initiated this project with the following objectives:

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

2. Evaluate current patterns of *P. ramorum*-related mortality on a landscape scale in a variety of woodland and forest types. This information can be used to help develop models for predicting disease spread and progression in different parts of the county. Such models may provide information that can be used to manage and possibly reduce disease impacts in natural stands.

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 can be coupled with subsequent wildlife use/population studies that can directly measure effects of the disease on wildlife and fisheries.

This report describes initial plot establishment and summarizes basic disease and stand data collected in the initial survey.

METHODS

Plot selection

We established plots at eleven locations in Sonoma County where tanoak, coast live oak, and/or black oak were common, as shown in Figure 1 and Table 1. Plots are located on public land 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 at each location are set up on a grid plan that allows for random sampling of the target area. Prior to visiting each study location, we contacted site owners or resource managers to obtain vegetation maps or general stand composition information. This allowed us to target areas for sampling that were likely to have one or more of the three host species of interest. We superimposed a plot grid based on UTM coordinates over the area of interest prior to visiting the site (Figure 2). Plot centers on the grid are at 50 m intervals as plotted on a topographic map, with the exception of the first location (Jack London SP) where 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, black oak, and/or tanoak were sampled, the pattern of actual plot positions differs from the idealized sampling grid (Figure 3). We attempted to sample about 24 plots per location, but fewer plots were sampled at some locations due to limitations of time and budget constraints. Field data collection required two days at all locations except Foothill Regional Park, where data were gathered in a single day. A total of 250 plots were established at the 11 locations between 5/19/01 and 7/26/01.

The UTM coordinates of each plot center were uploaded into a handheld GPS receiver (Garmin GPS 76) which was capable of differential correction using satellite-based WAAS signals. A high-gain external GPS antenna (Mighty Mouse II) mounted on a mast was used with the GPS receiver to improve reception under tree canopy. We used the GPS receiver to navigate to the plot centers. Due to uncertainty and drift in GPS-based coordinates, especially under poor satellite reception conditions, we established plot centers when the GPS reading indicated that we were within 5 meters of the uploaded plot center coordinates. After allowing GPS readings to stabilize, we stored the coordinates of the plot center as reported by the GPS receiver. These points were subsequently downloaded and plotted as the nominal plot coordinates. Positional uncertainty of these coordinates is generally 5 m or less, but may be greater in areas with dense redwood overstory where satellite reception was typically poor.

Plots are circular with a radius of 8 meters measured parallel to the ground slope (plot area 0.02 ha = 0.05 acre);. We demarcated the plot perimeter and determined which trees were in the plot by using a handheld laser rangefinder (Leica Disto Classic) aimed at the GPS mast, which was placed at the plot center. In each plot, we evaluated the condition of up to three *P. ramorum* canker host trees in detail (Table 2). These trees are referred to as

"tally trees". Tally trees were tagged with round, 31 mm diameter aluminum tree tags which point towards the plot center. For plots in which tally trees did not appear likely to persist for more than a few years, one or two nontarget tree species were tagged to increase the chance that the plot could be relocated at a later date. Using the laser rangefinder and a compass, we measured the distance and azimuth between tagged trees and the plot center to permit precise relocation of plot centers. In many plots that contained only one or two target species trees, we added tally trees outside the plot radius that were close to the plot edge. These trees are excluded from plot-based calculations, such as tree density.

All other trees in the plots were counted by species, canopy position (overstory or understory) and disease status. For coast live oak, black oak, and tanoak, the occurrence of *P. ramorum* canker symptoms was included in these evaluations. Other plot data we collected include shrub species present, overall tree and shrub cover ratings, plot slope and aspect, and the presence of other disease agents in the plot. Table 3 presents a detailed description of plot data collected.

Table 1. Locations of plots and host species studied.

Location number	Location	Number of plots	Subject tree species
1	Jack London State Park	24	Black oak, tanoak, coast live oak
2	Sugarloaf Ridge State Park	25	Coast live oak
3	Lake Sonoma (Army Corps of Engineers)	24	Coast live oak, black oak
4	Weston Ranch (private land)	26	Coast live oak, black oak
5	Austin Creek State Recreation Area	25	Tanoak, black oak
6	Modini Ranch (private land)	25	Black oak, coast live oak
7	Annadel State Park	24	Black oak
8	Salt Point State Park	18	Tanoak
9	Helen Putnam Regional Park	24	Coast live oak, black oak
10	Foothill Regional Park	15	Black oak, coast live oak
11	Sonoma Coast State Beach	21	Tanoak

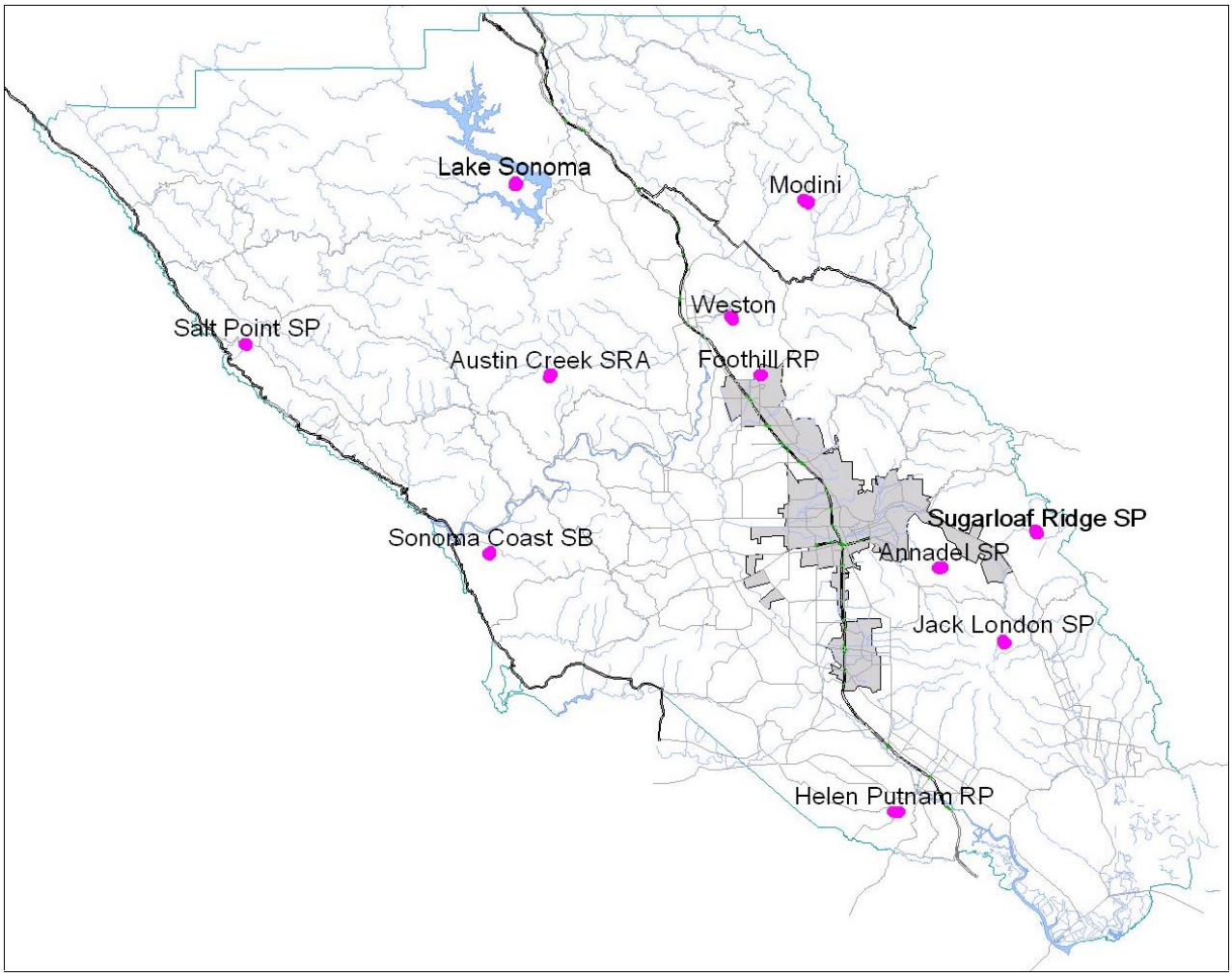

Figure 1. Distribution of survey / monitoring locations within Sonoma County.

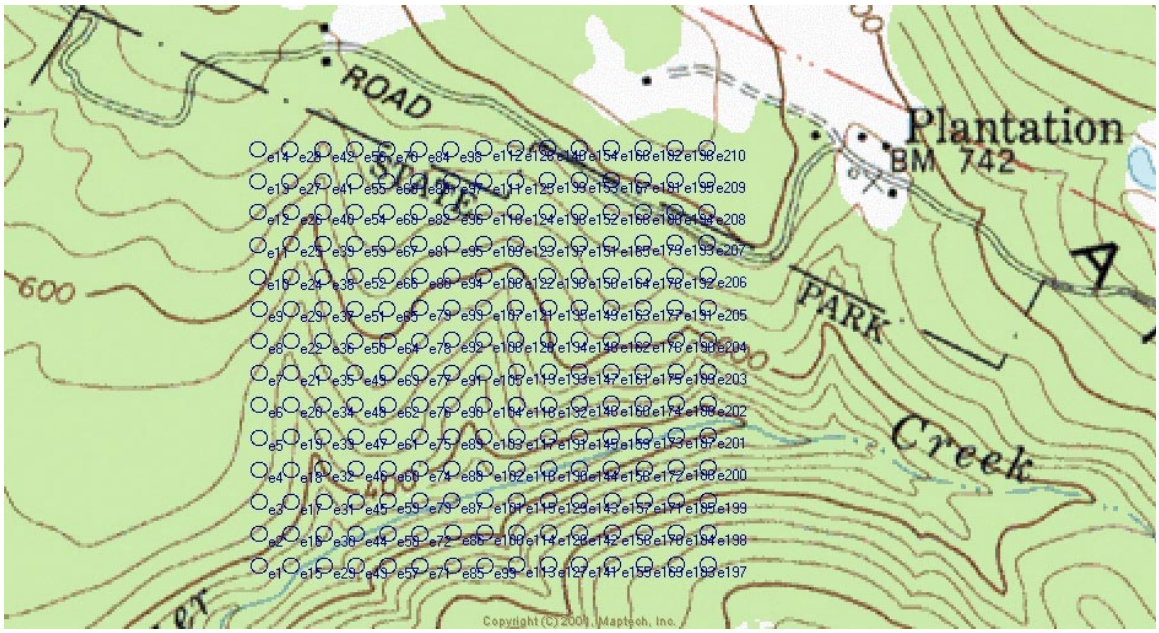

Figure 2. Example of a plot grid layout. Shown is the plot grid layout for Salt Point State Park. The grid was superimposed over an area shown as tan oak forest in a vegetation map supplied by State Park staff.

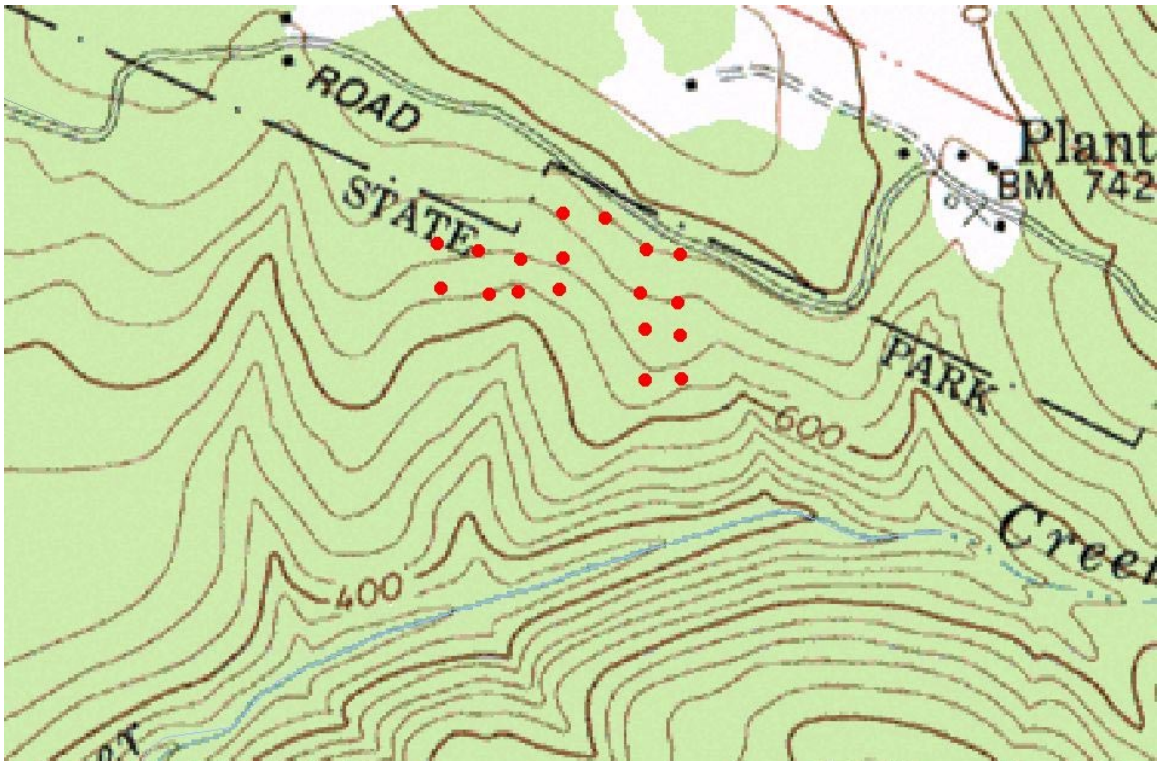

Figure 3. Actual plot locations for Salt Point State Park, based on coordinates recorded by a handheld GPS receiver.

Data management and analysis

Plot data are georeferenced to the plot locations to allow for use and analysis with GIS software. For each location, we acquired local aerial photo and USGS topographic map images which we 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 databases.

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 4.0.4 (SAS Inc., Cary NC).

Table 2. Tree variables measured for tally trees in each study plot

Variable	Method	Scale/units and notes
Tally tree species		*Q. agrifolia, Q. kelloggii, Q. wislizeni,* or *L. densiflorus*
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	inches (d-tape) or cm (flat tape); flat tape used where d-tape could not be (e.g., trunks covered with poison oak)
Number of stems from ground	count	stems/tree
Stems with *P. ramorum* symptoms	count	infected stems/tree
Dead stems	count	dead stems/tree
Tree dead / cause	visual assessment	Causes: (1). *Phytophthora;* (2). other agent(s); (3). unable to determine Tree scored as dead if all main stems are dead. Two causes (*Phytophthora* plus other agents) are scored if applicable.
Hypoxylon thouarsianum	presence of fruiting bodies	present / absent
Bark and/or ambrosia beetles in main stem	presence of boring dust and/or holes	present / absent
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 Assessment of actual levels of decay in standing trees is problematic. Decay impact rating (Swiecki and Bernhardt 2001) 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 study plots. All variables were measured in the 8 m radius fixed-area plots.

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 Trees do not have to be rooted within the plot
Count by tree health class relative to *Phytophthora ramorum* canker and other decline/mortality agents (SOD 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)	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 3 - late *P. ramorum* disease 4 - dead attributed to *P. ramorum* 5 - severe decline 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*
Count by general tree health class (trees other than SOD hosts[2])	tree count by species, subcategorized by symptom class and canopy position (overstory/understory)	Symptom classes: 1 - live 2 - decline 3 - dead
SOD host[2] regeneration	count or estimate if >10	regeneration: seedlings and saplings <3 cm dbh
Disease incidence in SOD host[2] regeneration	count or estimate percent if count > 10	Disease could be due to *P ramorum* or other agents
Dead SOD host[2] regeneration	count	Cause of mortality in regeneration was not determined
Regeneration of trees other than SOD 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
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* canker, i.e., coast live oak, black oak, and tanoak

RESULTS

The main species of interest in this study are coast live oak, black oak, and tanoak, all of which develop *P. ramorum* bark cankers. The prevalence of these species at each of the study locations is shown in Table 4. For the purposes of this report, these three species will be referred to as SOD host trees. A total of 425 coast live oak, 350 black oak, and 635 tanoak trees were observed in plots. Tally trees included 228 coast live oaks, 259 black oaks, and 179 tanoaks.

All oak species which are currently known to develop bark cankers due to *P. ramorum* are members of the black oak subgenus. Another species in the black oak subgenus, *Q. wislizeni* var. *frutescens* (a variety of interior live oak), was encountered at two study locations (Lake Sonoma and Austin Creek). Although infection of this species under field conditions by *P. ramorum* has not been reported, we consider this species to be a potential SOD host due to its close relatedness to coast live oak and Shreve oak. Therefore, we have included the 41 *Q. wislizeni* var. *frutescens* observed in the plots with other members of the black oak subgenus in Figure 4.

Current information indicates that various other oak species are not susceptible *P. ramorum* cankers. These include species in the white oak subgenus (valley oak, *Q. lobata;* blue oak, *Q. douglasii;* scrub oak, *Q. berberidifolia;* and Oregon white oak, *Q. garryana*) and a member of the intermediate oaks (canyon live oak, *Q. chrysolepis*). Because these oaks provide many of the same habitat values as the SOD hosts, they are handled as a separate group in Table 4 and Figure 4. A total of 231 of these other oaks were present in plots.

The remaining 1,189 trees in the plots consist of trees other than oaks or tanoak. Some of these species, including California bay and madrone, are known to sustain leaf and/or twig infections caused by *P. ramorum*. However, since the full list of potential foliar hosts is not known at this time, we have refrained from splitting these other species into *P. ramorum* hosts and non-hosts in the following summaries. 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 in some locations.

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

		Percent of trees within plots								
Location	Trees per hectare	coast live oak	black oak	tanoak	interior live oak	all other oaks[1]	CA bay	madrone	coast redwood	Douglas fir
Annadel	460	5%	28%	0%	0%	27%	35%	1%	0%	5%
Austin Creek	688	2%	19%	34%	1%	0%	12%	8%	8%	14%
Foothill	757	1%	35%	0%	0%	49%	0%	15%	0%	0%
H. Putnam	402	87%	10%	0%	0%	4%	0%	0%	0%	0%
J. London	529	6%	7%	16%	0%	0%	21%	12%	11%	24%
L. Sonoma	465	12%	6%	0%	17%	5%	33%	5%	12%	9%
Modini	352	6%	26%	0%	0%	16%	0%	22%	0%	23%
Salt Point	1269	0%	0%	79%	0%	0%	0%	2%	14%	5%
Sonoma Coast	571	0%	0%	50%	0%	0%	21%	0%	22%	7%
Sugarloaf	781	35%	0%	0%	0%	3%	42%	6%	0%	13%
Weston	306	33%	30%	0%	0%	0%	4%	26%	0%	0%

[1]All other oaks includes members of the white oak group (*Q. lobata, Q. douglasii, Q. berberidifolia*, and *Q. garryana*) and a member of the intermediate oaks (*Q. chrysolepis*). To date, none of these oaks have been shown to be susceptible to bark cankers caused by *P. ramorum.*

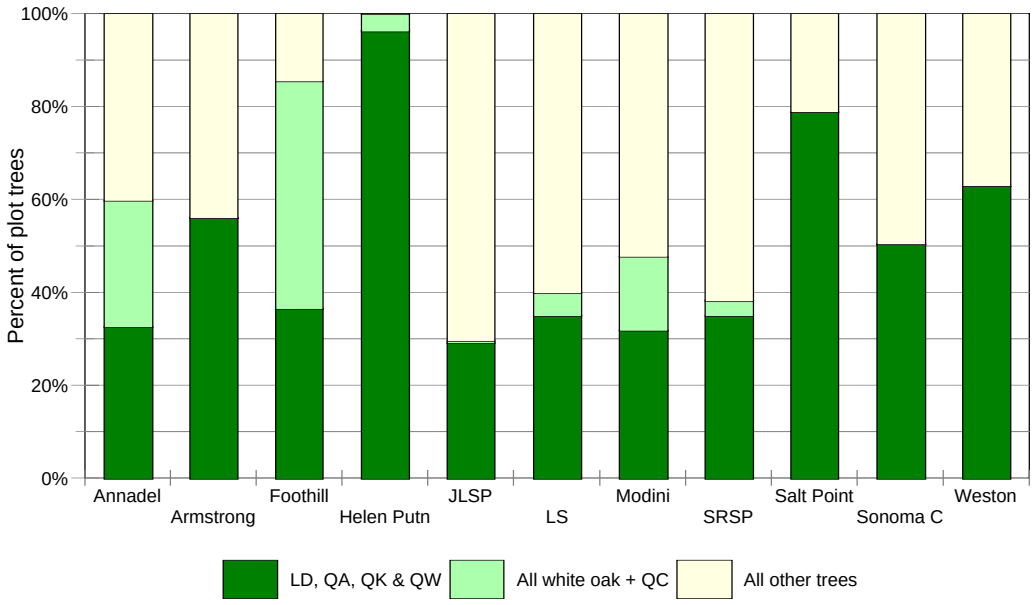

Figure 4. Percent of plot trees in three species groups: known and likely SOD hosts (tanoak plus the black oaks *Q. agrifolia, Q kelloggii*, and *Q. wislizeni*) [dark green]; other, apparently non-host oaks (*Q. chrysolepis, Q. berberidifolia, Q. douglasii, Q. garryana*, and *Q. lobata*) [light green]; and other non-oak tree species (including species such as California bay, madrone, and buckeye which may sustain leaf and twig infections caused by *P. ramorum*) [yellow]. JLSP=Jack London SP, LS=Lake Sonoma, SRSP=Sugarloaf Ridge SP.

Presence of *P. ramorum* at different study locations

We did not observe any trees with typical symptoms of *P. ramorum* canker at Modini Ranch, Weston Ranch, Annadel State Park, or Foothill Regional Park. At least some symptomatic trees were observed in plots at the remaining seven locations. The presence of *P. ramorum* canker at Jack London State Park, Sugarloaf Ridge State Park, and Austin Creek State Recreation Area had been confirmed by the lab of U.C. Davis Plant Pathologist David Rizzo before this project was initiated. Sampling and testing is currently underway by Sonoma County UCCE staff to determine whether canker symptoms at Sonoma Coast State Beach, Salt Point State Park, Helen Putnam Regional Park, and Lake Sonoma are due to *P. ramorum*.

Determining whether *P. ramorum* is present at the four unconfirmed sites is complicated by several factors. The most common diagnostic technique, isolation of the pathogen on selective media, frequently yields false negative results, especially in samples collected at certain times of the year. Although a positive isolation from these sites would provide definitive evidence for the presence of *P. ramorum*, a negative result would not rule out the possibility that the cankers are caused by *P. ramorum*. More sensitive DNA-based tests are not yet available for use with field-collected woody canker tissue. In addition, another, possibly unnamed *Phytophthora* species similar to *P. ilicis* has been associated with bleeding bark cankers on tanoak and coast live oak in some locations (D. Rizzo, personal communication). Hence, symptoms that appear typical of *P. ramorum* could actually be due to another *Phytophthora* species. While it may take some time before a definitive determination of the agent(s) associated with the bleeding bark cankers at the four unconfirmed locations can be made, in this report, data are summarized assuming that these symptomatic locations are positive for *P. ramorum*.

With the exception of Helen Putnam Regional Park, surveyed stands are generally contiguous with extensive woodlands or forests. The stand at Helen Putnam Regional Park is essentially a small remnant island of woodland that is separated by about 4.8 km (3 mi) from a more extensive woodland located to the south in Marin County. Within our study area, the Helen Putnam stand was composed almost exclusively of coast live oak and black oak. It lacked any overstory trees that are known to support twig and foliar infections of *P. ramorum,* although honeysuckle (*Lonicera* sp.), which sustains foliar infections, was common in the area. One possibility that warrants further investigation is whether the relatively few and generally small cankers seen at this site could have resulted from wind-borne *P. ramorum* inoculum originating in the infected stands to the south.

Although Sonoma County is within the declared Zone of Infestation for *P. ramorum*, our surveys show that the county has stands of host trees that are currently free of evident disease symptoms. One location (Annadel SP) is relatively close to areas where the disease is common whereas the remaining asymptomatic location (Modini, Weston, Foothill RP) are relatively distant from heavily diseased areas. The apparent lack of disease at the Annadel study area is of interest because the site possesses several characteristics that would seem to place it at a relatively high risk for disease. It has relatively high populations of California bay, the leaves of which support sporulation of *P. ramorum*. Annadel is also relatively close to other infested areas, and has other characteristics (trails heavily used by recreationists, proximity to residences) that increase the chance that *P. ramorum* might be introduced into the stand.

Future monitoring of the study plots will be necessary to determine whether unaffected stands remain free of disease, and if not, the pattern of disease introduction and spread in these areas. It would also be useful to relate differences in disease progress in different stands to factors such as stand density and composition (Table 4, Figure 4), moisture and temperature regimes, and other site characteristics to determine whether these factors can be used to predict the risk of disease spread.

Levels of disease by location

P. ramorum canker (SOD)

Figures 5A, 6A, and 7A show the numbers of SOD host trees exhibiting symptoms that appear to be due to *P. ramorum* infection for each study location. The totals are further subdivided by canopy position (overstory or understory) for each symptom class. If we exclude the four locations where no symptomatic trees were observed, the overall apparent infection rates for black oak, coast live oak, and tanoak are 3.4%, 8.9%, and 12.1%, respectively. If we limit our analysis to the three locations for which positive identifications of *P. ramorum* have been made, the apparent infection rates for the black oak, coast live oak, and tanoak are 3.6%, 12.9%, and 26.0%, respectively. These percentages appear to agree with laboratory studies showing that tanoak is the most and black oak the least susceptible of these three species to *P. ramorum* infection (D. Rizzo, personal communication).

Apparent infection rates vary widely between the study locations. At the time of the survey, the study area at Jack London SP was the most heavily impacted by *P. ramorum*. At this location, nearly 45% of all SOD hosts, including 66% of all surveyed tanoak, were symptomatic. Currently, the percentage of SOD hosts showing symptoms at other locations is much lower, ranging from about 3% to 21% of all SOD hosts at the other six locations where we observed symptoms typical of *P. ramorum* (Figure 8). Sites in which tanoak comprises a large portion of the SOD host tree population (Table 4) generally have higher rates of symptomatic SOD hosts than sites dominated by the less susceptible oak species.

One less obvious trend is that symptomatic coast live oak are significantly more likely to be found in the overstory than in the understory ($P = 0.0031$, 2-tail Fisher's exact test). At the five locations with both coast live oak and SOD symptoms, the apparent coast live oak infection rate among overstory trees was 12% compared with 2% for understory trees. Although 72% of the surveyed coast live oaks at these locations were overstory trees, 94% of the symptomatic trees were overstory trees. In a previous study (Swiecki and Bernhardt 2001) coast live oak trees with more than 50% sky-exposed canopy (Table 2) were at higher risk of developing *P. ramorum* infections than those with less sky-exposed canopy. This increased risk might be related to interception of wind-blown inoculum by these tree canopies or to tree growth rates as discussed in the following section.

In tanoak, apparent *P. ramorum* infection rates did not differ significantly between overstory and understory trees. For black oak, the number of symptomatic trees within plots (4) is too low to draw any meaningful conclusions.

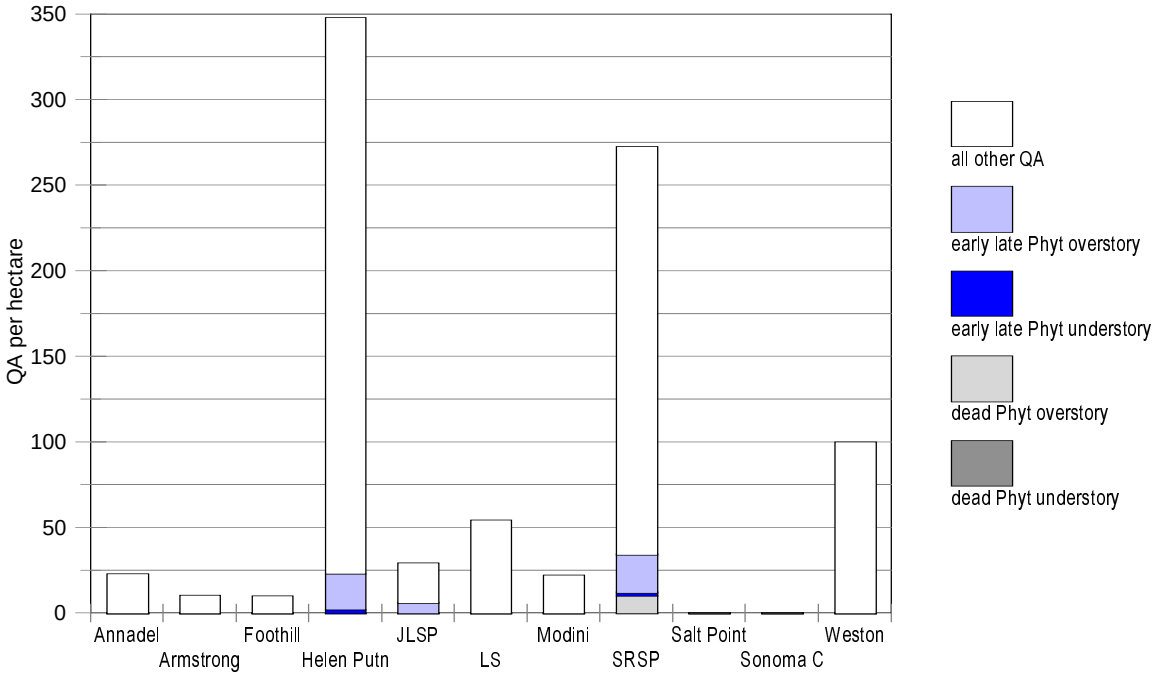

Figure 5A. Density of coast live oak (QA) trees with *P. ramorum*-related symptoms by canopy position at eleven locations in Sonoma County.

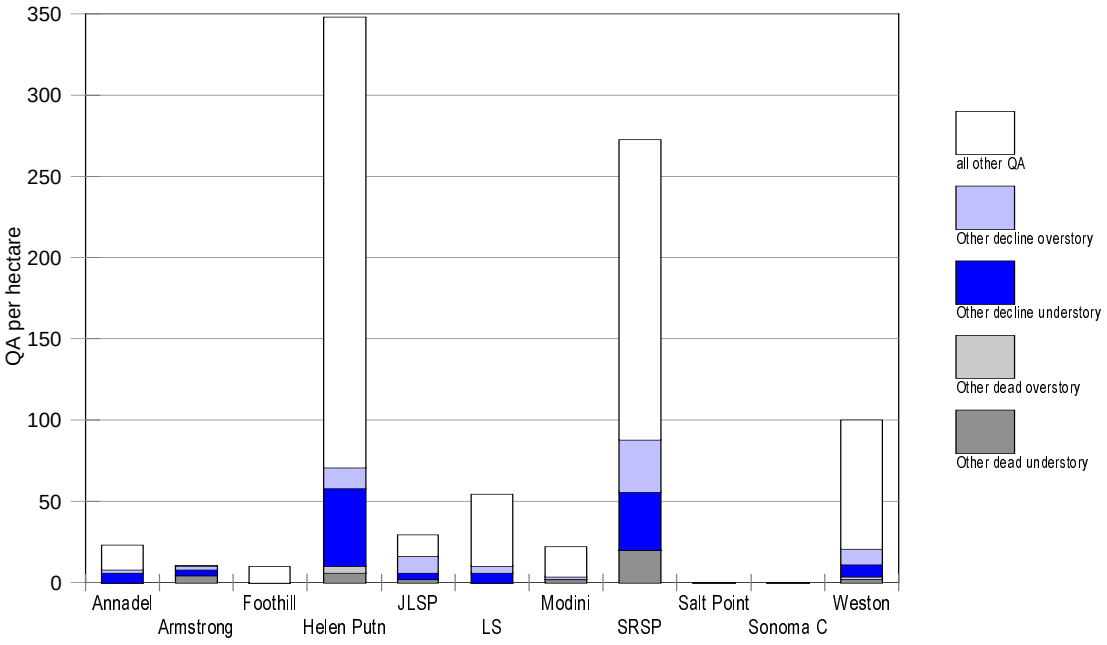

Figure 5B. Density of coast live oak (QA) trees in severe decline or recently dead (within past 10 years) due to causes other than SOD by canopy position at eleven locations in Sonoma County.

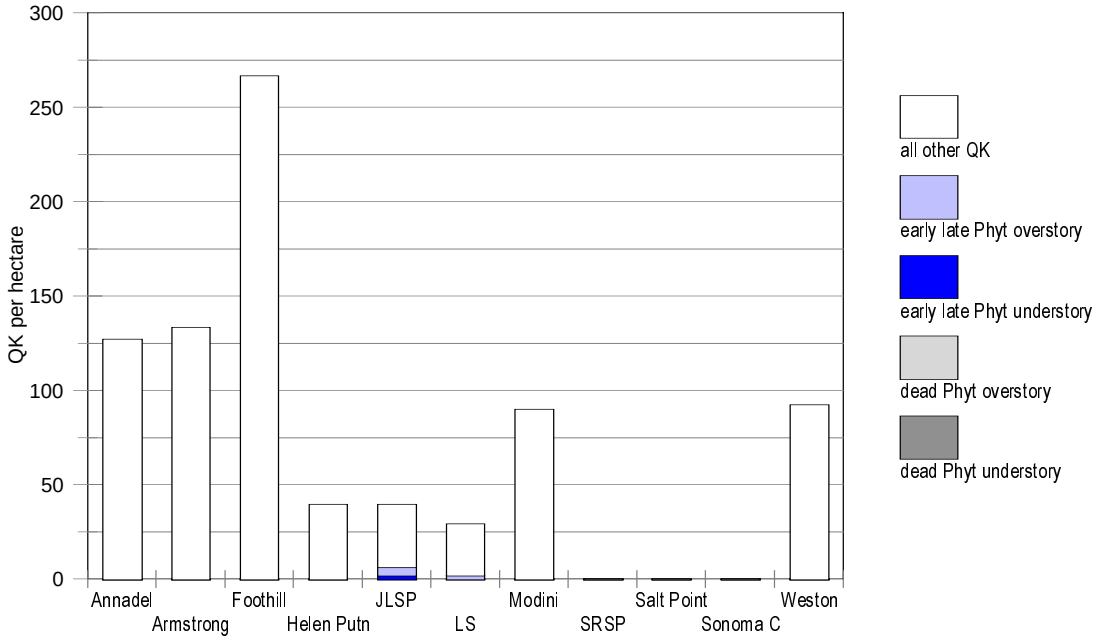

Figure 6A. Density of black oak (QK) trees with *P. ramorum*-related symptoms by canopy position at eleven locations in Sonoma County.

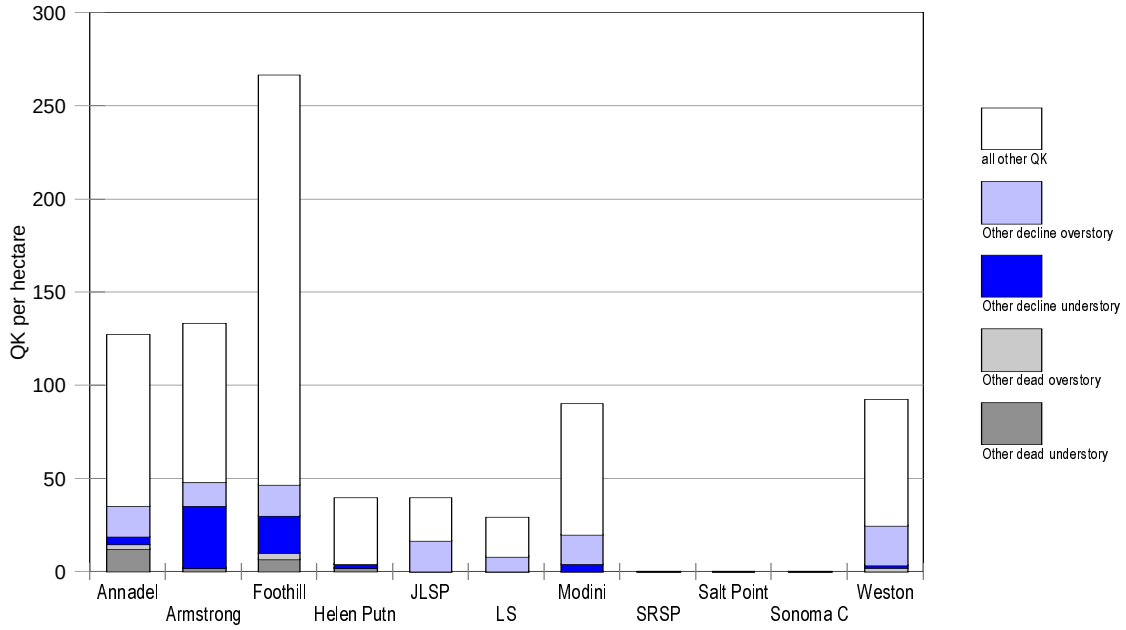

Figure 6B. Density of black oak (QK) trees in severe decline or recently dead (within past 10 years) due to causes other than SOD by canopy position at eleven locations in Sonoma County.

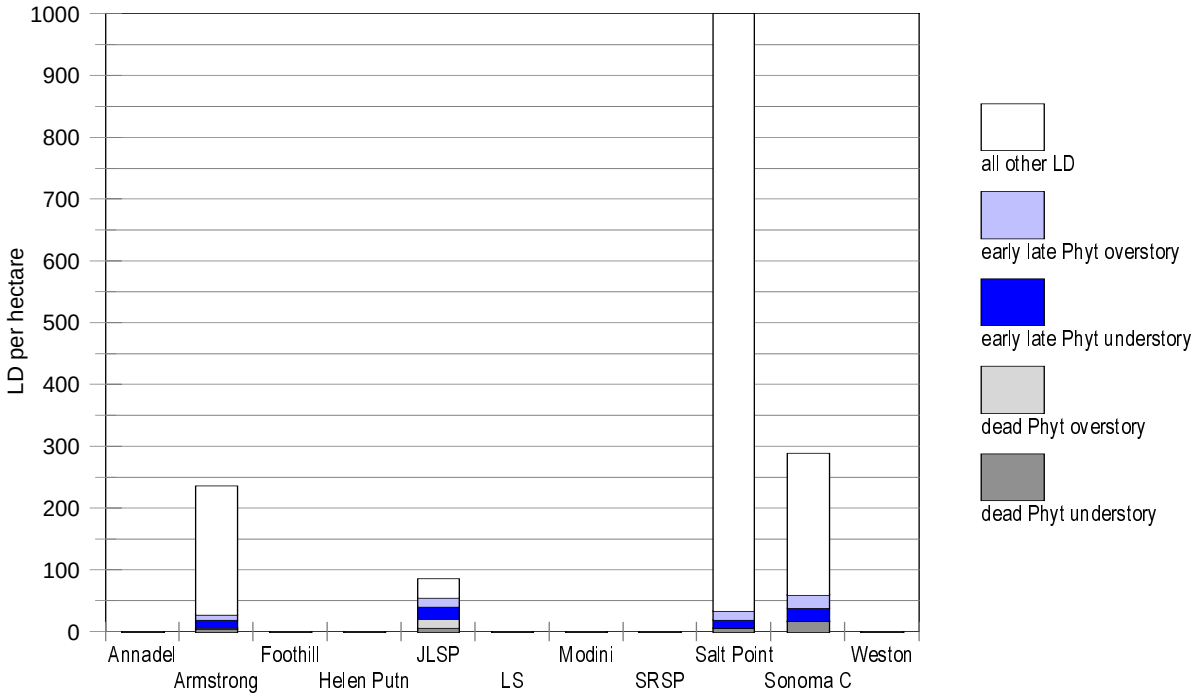

Figure 7A. Density of tanoak (LD) trees with *P. ramorum*-related symptoms by canopy position at eleven locations in Sonoma County.

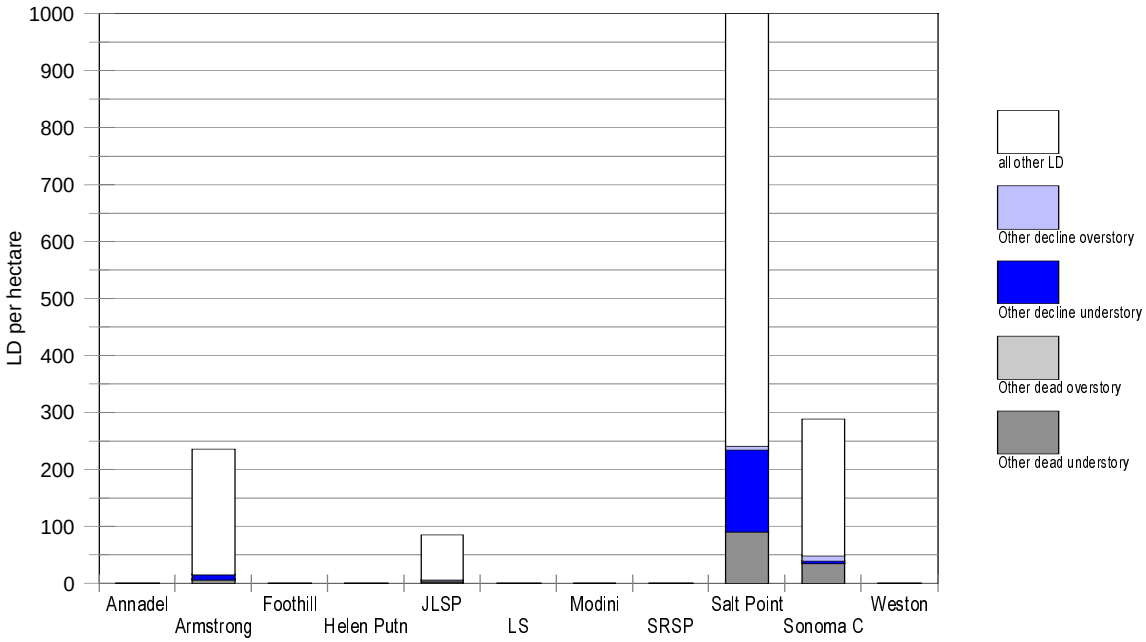

Figure 7B. Density of tanoak (LD) trees in severe decline or recently dead (within past 10 years) due to causes other than SOD by canopy position at eleven locations in Sonoma County.

Other diseases and interactions with SOD

As shown in Figures 5B, 6B, 7B, 8, and 9, tree decline and death due to agents other than *P. ramorum* are common among SOD hosts in the surveyed stands. Among the seven locations with apparent *P. ramorum* canker symptoms, only two (Jack London SP and Sonoma Coast SP) had apparent *P. ramorum* infection levels that were comparable to "background" levels of disease associated with other agents (Figure 8). Tanoak comprises the majority of the SOD hosts in both of these locations (Table 4). For locations in which less susceptible coast live oak and black oak represent the bulk of the SOD hosts, *P. ramorum* canker was much less prevalent than decline due to other agents.

Considering only SOD host trees lacking *P. ramorum* canker symptoms, between 17% and 33% of these trees were in severe decline or had died within the past 10 years because of agents other than *P. ramorum*. Canker rot, commonly caused by *Inonotus* spp., was the most common cause of decline and mortality overall. Canker rot symptoms were scored in 67% of all surveyed plots. By comparison, root disease symptoms were scored in only 5% of surveyed plots. After *H. thouarsianum*, whose fruiting bodies were observed in 26% of all sampled plots, fruiting bodies of *Phellinus* spp. (10% of plots) and *Inonotus* spp. (8% of plots) were most common among the wood decay fungi. However, the frequency of detection based on fruiting bodies is likely to be well below the actual frequency of occurrence for these fungi.

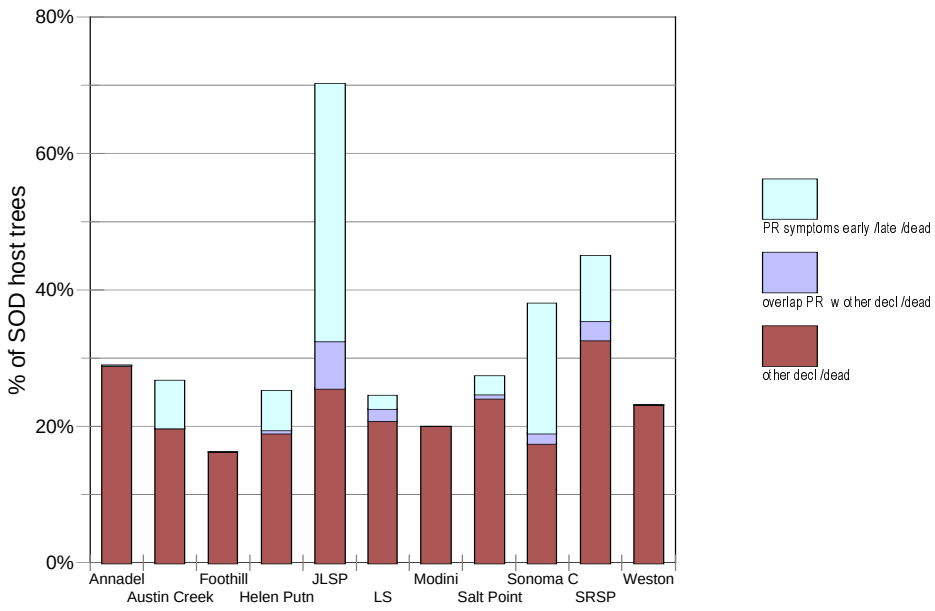

Figure 8. Percent of known SOD host trees (coast live oak, black oak and tanoak) at each location exhibiting symptoms of *P. ramorum* infection (PR) or showing severe decline or recent mortality due to other causes. The presence of *P. ramorum* has not been confirmed at LS (Lake Sonoma), Salt Point, Helen Putnam, and Sonoma Coast.

As shown in Figures 5B, 6B, and 7B, many of the trees that are dead or declining due to agents other than *P. ramorum* are understory trees. Especially for coast live oak and black oak, these are often overtopped and/or suppressed trees and competition is frequently a

contributing factor to decline. However, not all of these declining and dead trees are suppressed understory trees. Many are large, relatively old overstory trees that have been under attack by pathogens such as canker rot fungi and other wood decay fungi for many years.

One possibility raised by these data is that especially for coast live oak and black oak, *P. ramorum* canker may be more common on relatively vigorous trees. We observed very few SOD host trees that were affected by both *P. ramorum* and other decline/mortality agents (Figure 8). It may be that severely declining trees are less likely to be infected by *P. ramorum* because they are growing slowly. The relative dearth of *P. ramorum*-infected understory coast live oaks (Figure 5A) could likewise be due to poor growth rates in these individuals compared with trees with greater access to sunlight. Other research (Swiecki and Bernhardt 2001, 2002) indicates that coast live oaks that experience relatively high levels of water stress late in the season have a reduced risk of *P. ramorum* infection. Water-stressed trees grow slower than their less-stressed companions. For live oak, and possibly black oak, it is conceivable that bark characteristics of faster-growing trees (e.g., bark fissures, thickness, concentrations of tannins or other biochemical factors) render them more susceptible to *P. ramorum* infection and/or affect the rate of pathogen expansion. Further investigation into this question seems warranted because a relationship between growth rate and disease susceptibility would have major management implications.

Figure 9 illustrates the relative impacts of SOD and other diseases on overall stand condition at the 11 locations. The percentage of all trees (SOD hosts and others) that were dead or in decline due to factors other than *P. ramorum* ranged from about 11% to 22% at the 11 locations with an overall average of 17%. If SOD-affected trees are included with the dead and declining tree totals, the percent of trees affected ranges from 11% to 29%, and the overall average for all locations is increased to 21%. In heavily diseased sites where highly susceptible tanoaks constitute a large proportion of the stand (e.g., Jack London SP, Sonoma Coast SB), *P. ramorum* has already had a major impact on stand density and composition, and the impact of this pathogen is likely to increase. None of the other locations has more than 6% of the plot trees affected by *P. ramorum* (Figure 9).

Impacts of SOD in these forests differ in several ways from those of native pathogens. *P. ramorum*-related decline generally proceeds more quickly than decline related to wood decay fungi. This has implications for habitat value. For example, a tree in slow decline due to infection by the canker rot pathogen *Inonotus andersonii* will contribute coarse woody debris to the understory over a period of many years as branches periodically fail. In addition, the tree will provide an abundance of cavities as well as healthy branches and foliage over an extended period. Although a tree killed by *P. ramorum* will provide some habitat value, decay is likely to proceed more quickly, reducing the time span during which various habitat elements are made available. In addition, SOD appears to increase decline and mortality rates among trees that are otherwise generally healthy (Figure 8), potentially depleting the population of mature trees much more quickly than would occur as the result of native diseases.

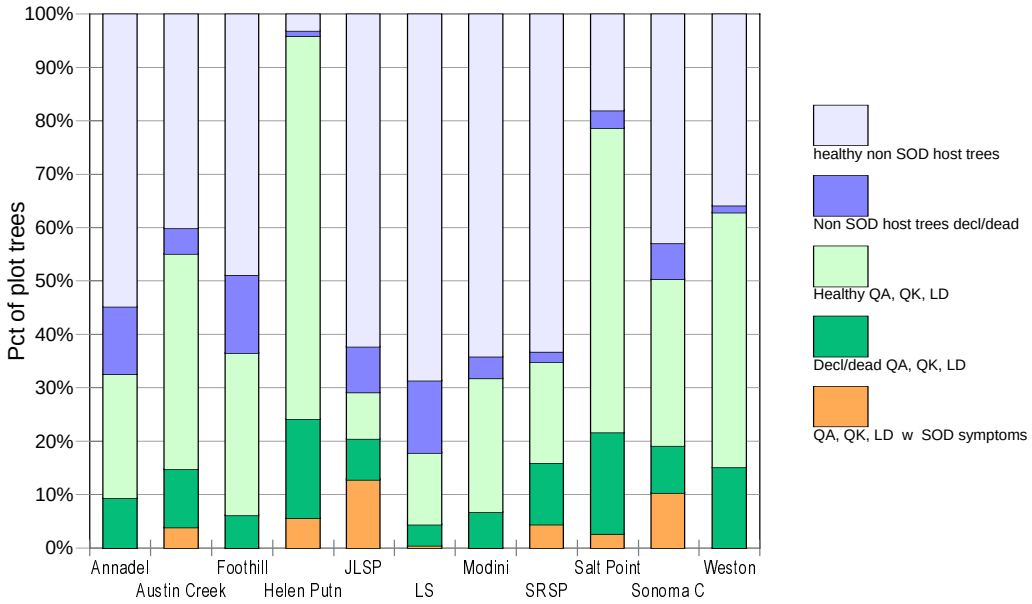

Figure 9. Impact of sudden oak death and other diseases on the health of forest stands at 11 locations in Sonoma County. Green and red shades represent SOD host trees (coast live oak, black oak, and tanoak) and blue shades all other tree species (including other oaks).

Distribution of SOD within study locations

Plot maps showing the distribution of plots with symptoms typical of *P. ramorum* canker for each location are shown in Figures 11 through 21 at the end of this section. All figures are shown at the same scale. At several locations, canker symptoms on specific trees were not completely typical of *P. ramorum* cankers. However, the complete range of variation possible in *P. ramorum* cankers is not known at this time. Plots in which the only symptomatic tree(s) had ambiguous symptoms are denoted with yellow triangles in the figures.

Even at the location with the greatest amount of disease (Jack London SP), disease symptoms were not uniformly distributed across the surveyed area (Figure 15). Plots with diseased trees tend to show some degree of clustering at most locations where disease is present (Figures 12, 14, 15, 16, 18, 19, 20). Within plots, we recorded data on stand composition, density, understory species, plot slope and aspect, and other factors that could be related to observed differences in disease incidence. We have previously shown that certain plot factors are correlated with the risk of *P. ramorum* canker (Swiecki and Bernhardt 2001, 2002). An analysis of the data from this study would provide additional information on plot factors that may be correlated with disease, but no funding is currently available to conduct these analyses. Similarly, we have also not analyzed the data for the 682 tally trees to examine individual tree factors that may be related to disease severity.

Regeneration

Mortality due to disease or other factors has the potential to have long-term effects on forest stand composition and hence its value as wildlife habitat. 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 the presence of tree species regeneration in the understory of plots where SOD host densities have or will be impacted due to mortality by both SOD and other agents.

Dead oak and tanoak seedlings can be difficult to detect or identify as seedlings, so levels of seedling mortality noted below are approximate at best and probably underestimate actual mortality rates. Also, seedlings of deciduous oaks such as black oak are difficult to observe if foliage senesces early due to drought stress. Hence, seedling densities of such species may be underreported in a one-time survey.

Tanoak

Among the 75 plots with tanoak overstory and/or understory trees, 74 (98.7%) also had tanoak regeneration in the understory. Tanoak regeneration was 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. Tanoak regeneration was generally abundant; 75% of the plots had 10 or more seedlings and 13% of the plots had 50 or more. Disease symptoms, some of which are probably attributable to *P. ramorum,* were seen in tanoak regeneration in 38% of all plots with tanoak, and dead tanoak seedlings were observed in 24% of these plots. Disease symptoms were seen in about 16% of all tanoak seedlings.

Dead, declining, or *P. ramorum*-diseased tanoak trees were present in 56 plots (75% of all plots with tanoak). Declining or dead tanoak were in the overstory in just over half of these plots (29/56). All 56 of the plots that have or are likely to sustain a loss of tanoak trees due to disease had tanoak regeneration in the understory. Most of these plots (71.4%) also had regeneration of other tree species, most commonly California bay (44.6%), coast redwood (30.4%), Douglas fir (30.4%), and madrone (14%). Oak seedlings were present only in two (3.6%) of these plots. Seedlings of as many as four other tree species were found in some plots.

In a study of old growth redwood forests in the California Central Coast region, Hunter et al (1999) showed that tanoak seedlings accumulate in the understory. Although shade tolerant, tanoak seedlings grow quickly in response to overhead light when canopy gaps form and can outcompete Douglas fir and redwood seedlings in canopy gaps (Burns and Honkala 1990). Although tanoak seedlings are currently plentiful and fairly consistent in plots with tanoak overstory, high levels of seedling disease could reduce the chance that tanoak mortality will be replaced by recruited tanoak seedlings.

Unless high proportions of tanoak seedlings resistant to *P. ramorum* are present in understory seedling populations, it seems unlikely tanoak regeneration will be as successful in stands affected by *P. ramorum* as it has been in the past. If practical levels of resistance to *P. ramorum* do not occur in tanoak populations, it is possible that this species

could be nearly eliminated in some areas. However, the strong selection pressure posed by *P. ramorum* against susceptible tanoak genotypes could in time result in the development of more resistant or tolerant tanoak populations if genes for resistance are present. Such selection would occur quicker if disease resistance in seedlings is correlated with mature tree resistance.

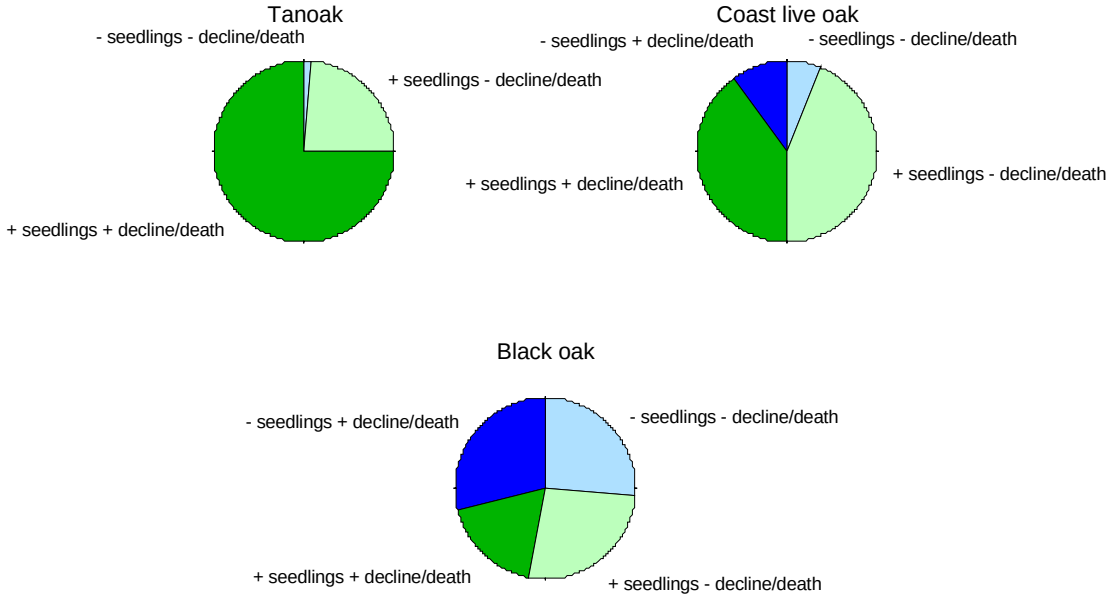

Figure 10. Plots with same-species regeneration and/or tree decline or mortality (due to any cause) for tanoak, coast live oak, and black oak. Decline/death includes all tree with *P. ramorum* canker symptoms as well as trees with severe decline or recent (in past 10 years) mortality from other agents. Regeneration includes any seedling or sapling with DBH< 3 cm. Plots with same-species regeneration are shown in green, those without in blue; plots with decline or mortality are indicated with darker colors.

Coast live oak

Coast live oak was present in 127 plots, of which 106 (83%) also had coast live oak seedlings. Regeneration consisted primarily of small seedlings less than 15 cm tall, including both recently-established seedlings and older, persistent seedling-sprouts. Seedling populations were variable. High numbers of coast live oak seedlings were sometimes present; 18 plots (14%) had 100 or more seedlings. Dead seedlings were observed in only 5% of the plots. Disease symptoms, presumably due to agents other than *P. ramorum*, were scored for only a single seedling.

About half of the plots with coast live oak (63/127) also had dead, declining, or *P. ramorum*-diseased trees, and in the majority of these plots (43/63) the diseased trees were in the overstory. Coast live oak seedlings were present in 79% of these 63 plots. Seedlings of other oaks and/or tanoak were present in 51% of these plots, while seedlings of other non-oak tree species were present in 73% of these plots. Other than coast live oak, the most common seedling species in these plots were bay (71% of plots), Douglas fir (54% of plots), madrone (24%of plots), and tanoak (19% of plots). Only 1 of these 63 plots had no tree seedlings of any sort in the understory, and some plots had seedlings of as many as five other tree species.

Coast live oak regeneration is not consistently present in the understory of plots where mortality of this species is occurring. Although some self-replacement of coast live oak is likely, the presence of generally larger seedlings of other tree species in these plots may reduce the success of coast live oak regeneration. Increased rates of coast live oak mortality due to SOD will exacerbate this situation, because reduced coast live oak acorn production will further reduce live oak seedling populations in the understory. Although other oak species may replace coast live oak in some plots, bay and Douglas fir appear to be the most likely replacements in many areas.

Black oak

Black oak was present in 135 plots; only 60 of these (44%) had black oak regeneration. Black oak regeneration consisted entirely of small seedlings or seedling-sprouts, mostly less than 20 cm tall. We did not observe black oaks in the intermediate size class between trees and seedlings. In contrast with the two species described above, seedling populations in black oak plots were generally low. Four or fewer black oak seedlings were present in 90% of the plots. Two plots had approximately 50 seedlings, which was the highest density we recorded for this species. The remaining plots had 16 or fewer seedlings. We did not observe dead or diseased seedlings.

Less than half of the plots with black oak (63/135) had dead, declining, or *P. ramorum*-diseased trees. Overstory black oaks were dead or diseased in about 75% of these plots (47/63). Black oak regeneration was observed in 38% of these 63 plots. Seedlings of other oaks and/or tanoak were present in 75% of these plots, whereas seedlings of other non-oak tree species were present in 89% of these plots. Besides black oak, the most common seedling species in these plots were bay (68% of plots), Douglas fir (65% of plots), coast live oak (40% of plots), madrone (32% of plots), and tanoak (21% of plots). Up to eight different tree species' seedlings were present in a single plot. Two black oak plots (3%) lacked any tree seedlings.

Of the three species studied, black oak was the least likely to have its seedlings present in its own understory. In much of the surveyed area, it appears unlikely that black oak mortality will be replaced by regeneration of this species, resulting in a decrease in black oak density at least over the near term. Coast live oak and tanoak may replace black oak in some plots, but bay and Douglas fir are probably more likely to increase in dominance as a result of black oak attrition.

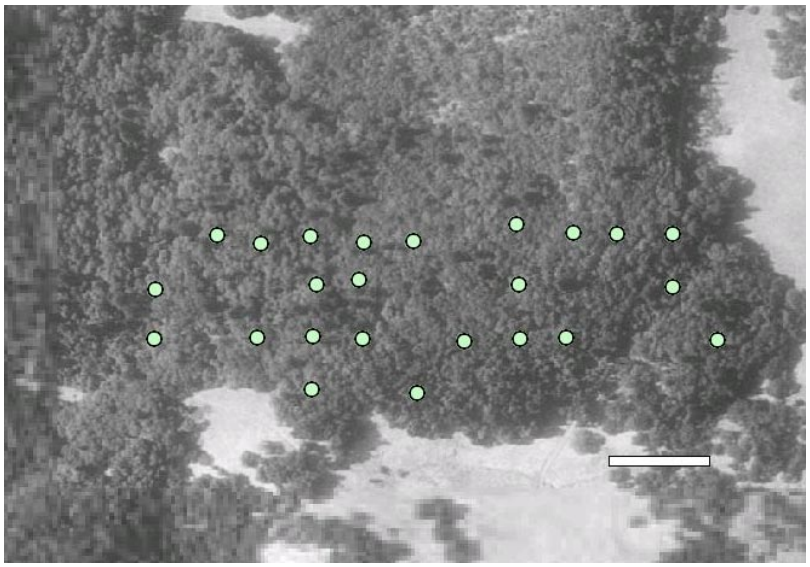

Figure 11. Plot locations at Annadel State Park. No *P. ramorum*-infected trees were seen in plots at this location. Scale bar at lower right is 100 m long. The same scale is used for Figures 11-21.

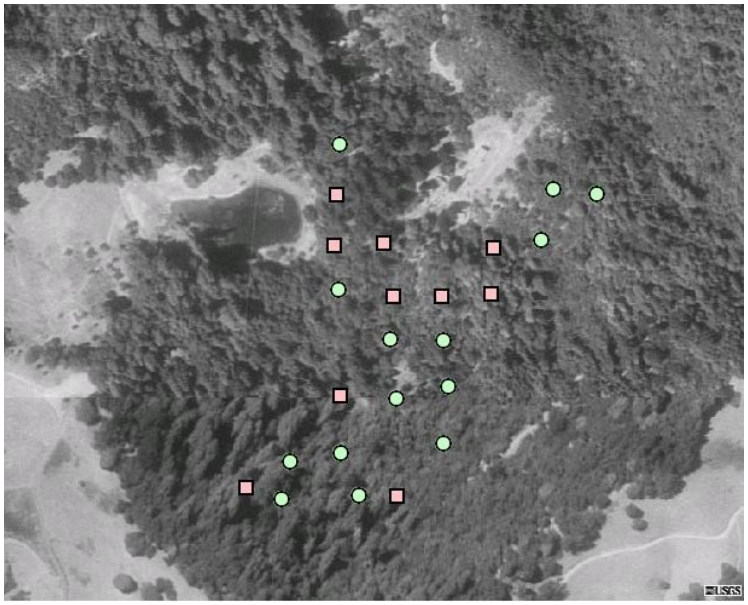

Figure 12. Plot locations at Austin Creek State Recreation Area. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. *P. ramorum* had previously been confirmed by UC researchers at this location.

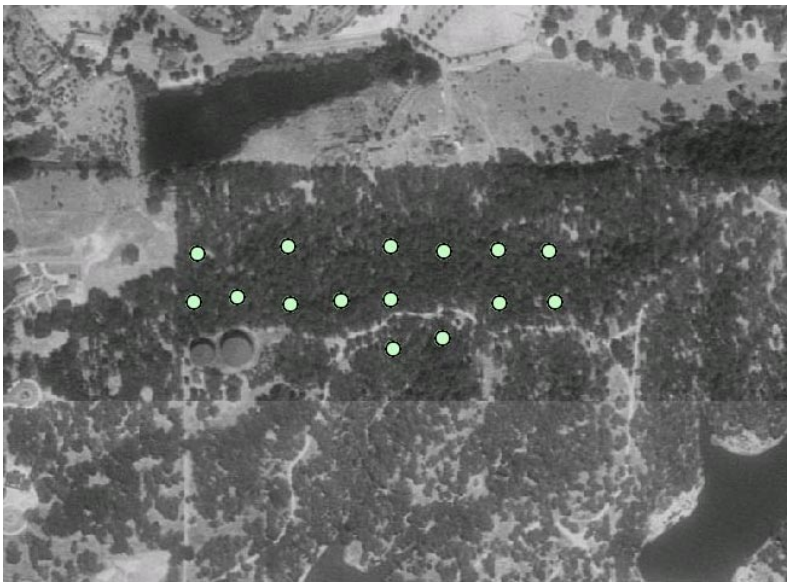

Figure 13. Plot locations at Foothill Regional Park. No *P. ramorum*-infected trees were seen in plots at this location.

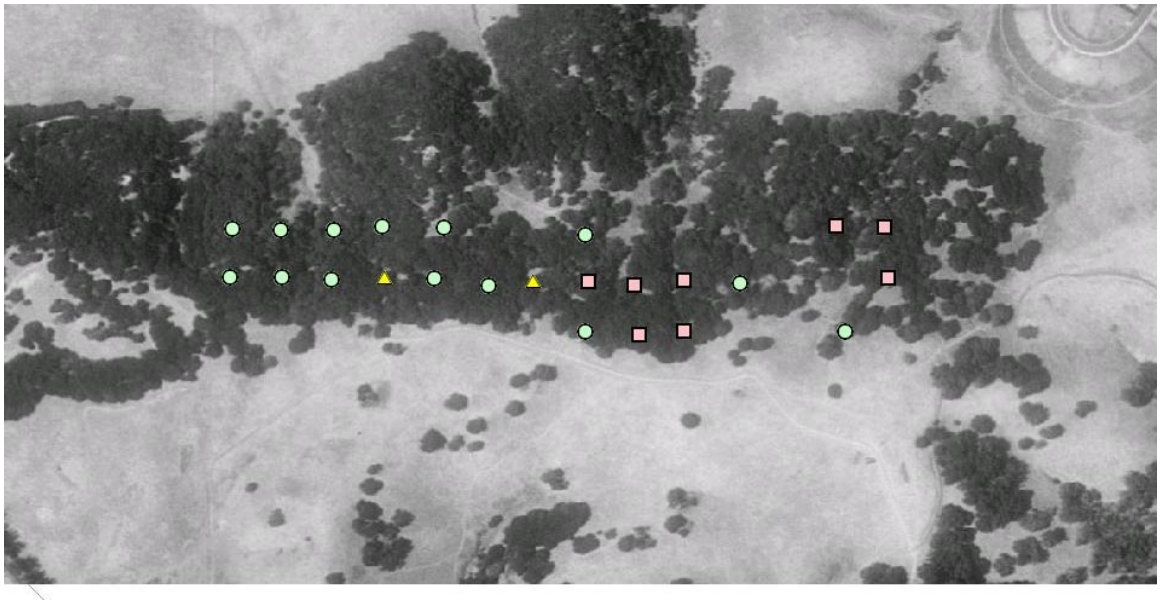

Figure 14. Plot locations at Helen Putnam Regional Park. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. Yellow triangles represent plots in which the only symptomatic tree(s) had ambiguous symptoms, i.e., cankers that were not entirely typical of *P. ramorum*. *P. ramorum* has not been confirmed at this location.

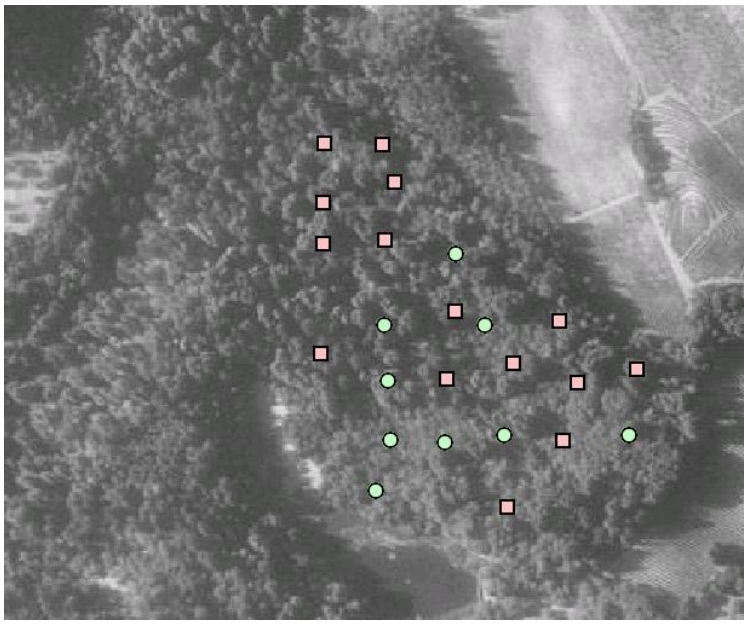

Figure 15. Plot locations at Jack London State Park. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. *P. ramorum* had previously been confirmed by UC researchers at this location.

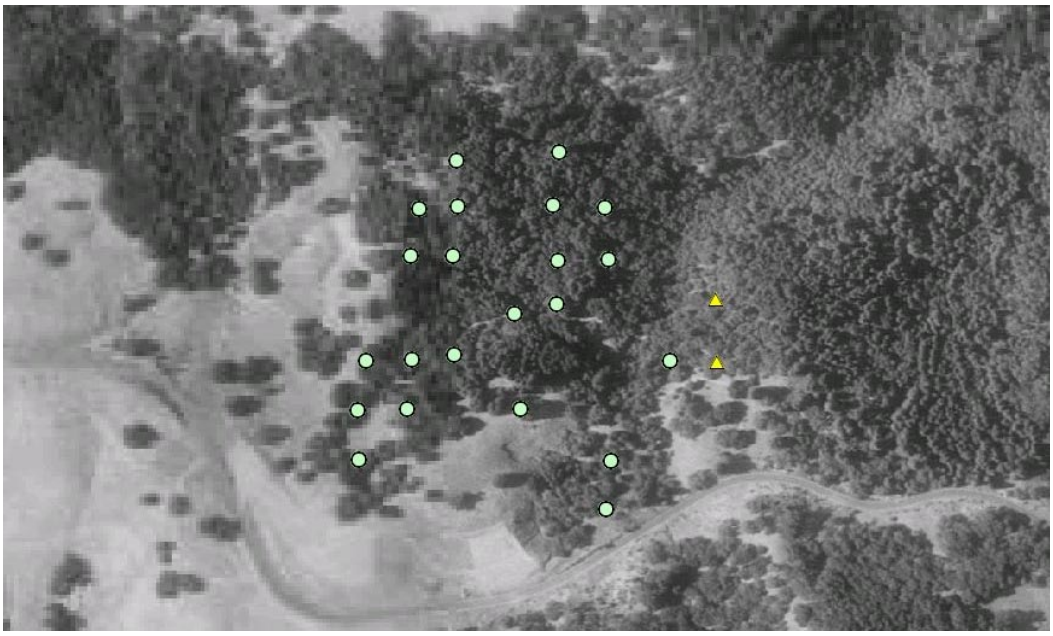

Figure 16. Plot locations at Lake Sonoma. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. Yellow triangles represent plots in which the only symptomatic tree(s) had ambiguous symptoms, i.e., cankers that were not entirely typical of *P. ramorum*. *P. ramorum* has not been confirmed at this location.

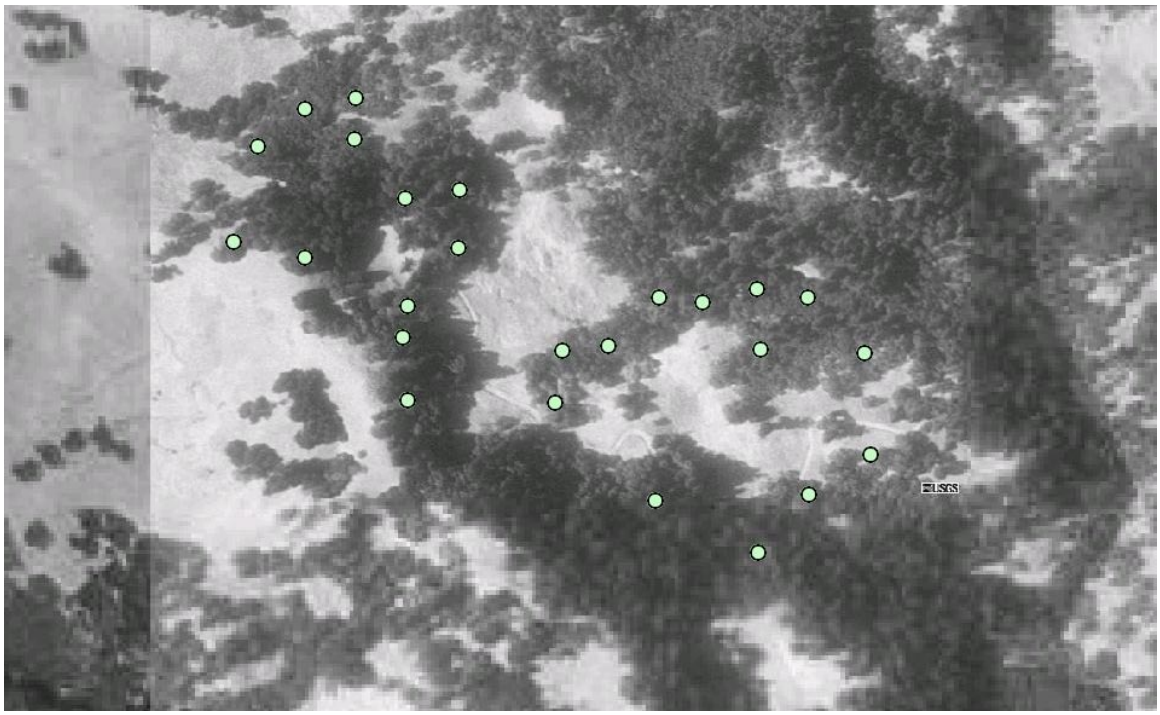

Figure 17. Plot locations at Modini Ranch. No *P. ramorum*-infected trees were seen in plots at this location.

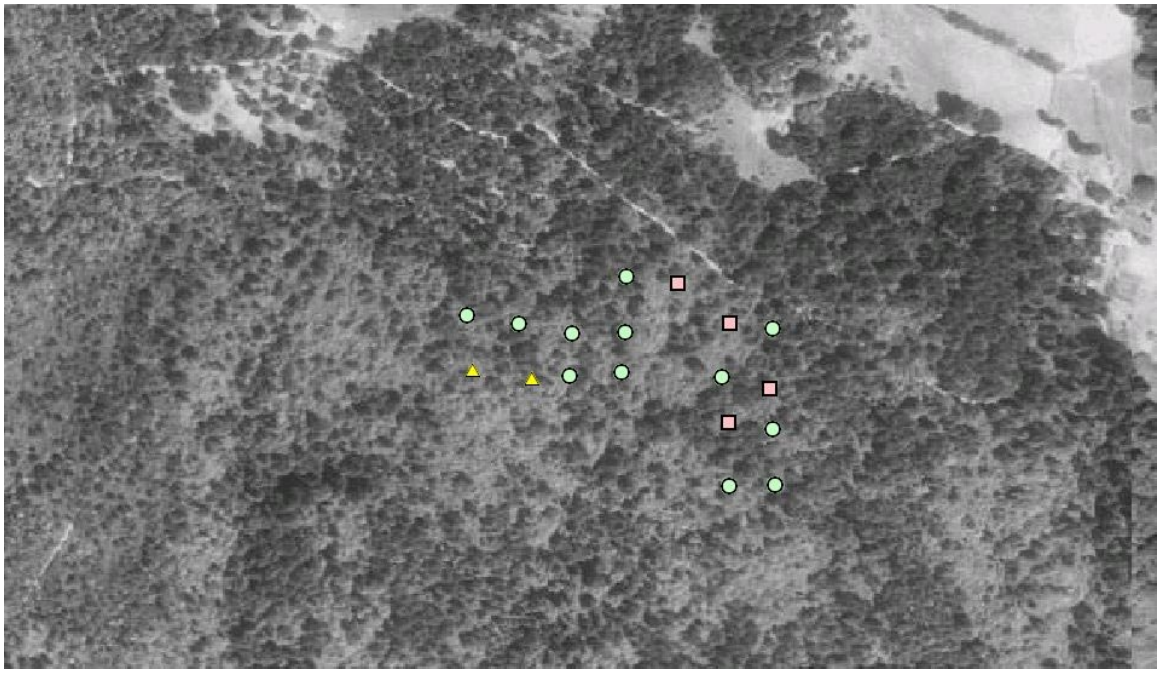

Figure 18. Plot locations at Salt Point State Park. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. Yellow triangles represent plots in which the only symptomatic tree(s) had ambiguous symptoms, i.e., cankers that were not entirely typical of *P. ramorum*. *P. ramorum* has not been confirmed at this location.

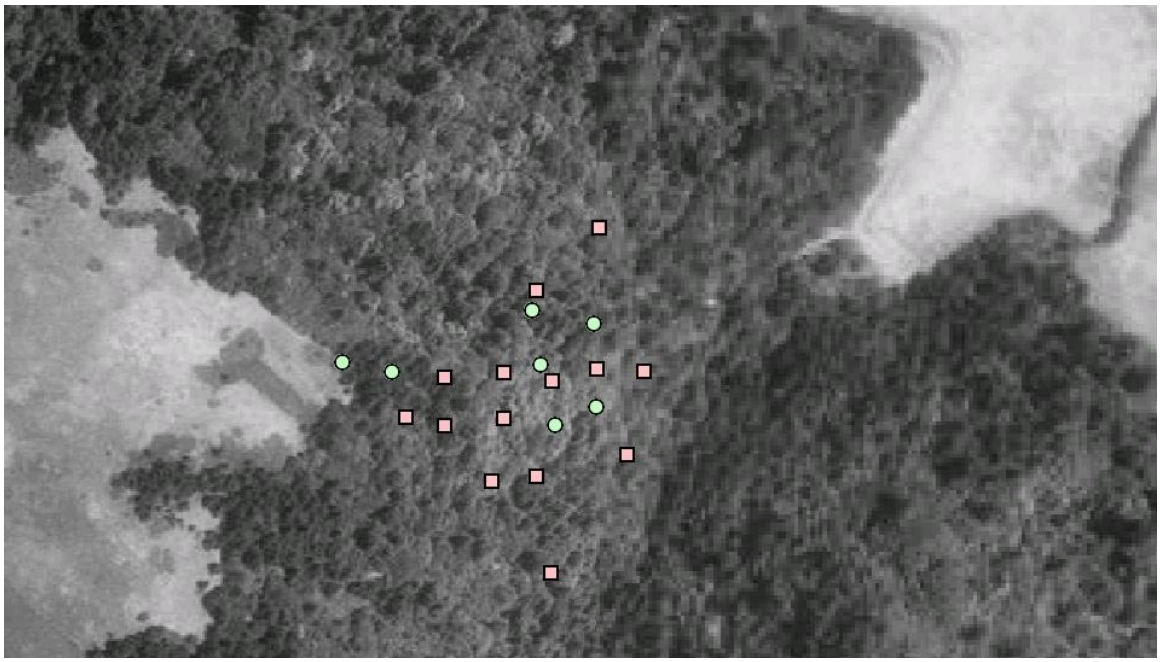

Figure 19. Plot locations at Sonoma Coast State Beach. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. *P. ramorum* has not been confirmed at this location.

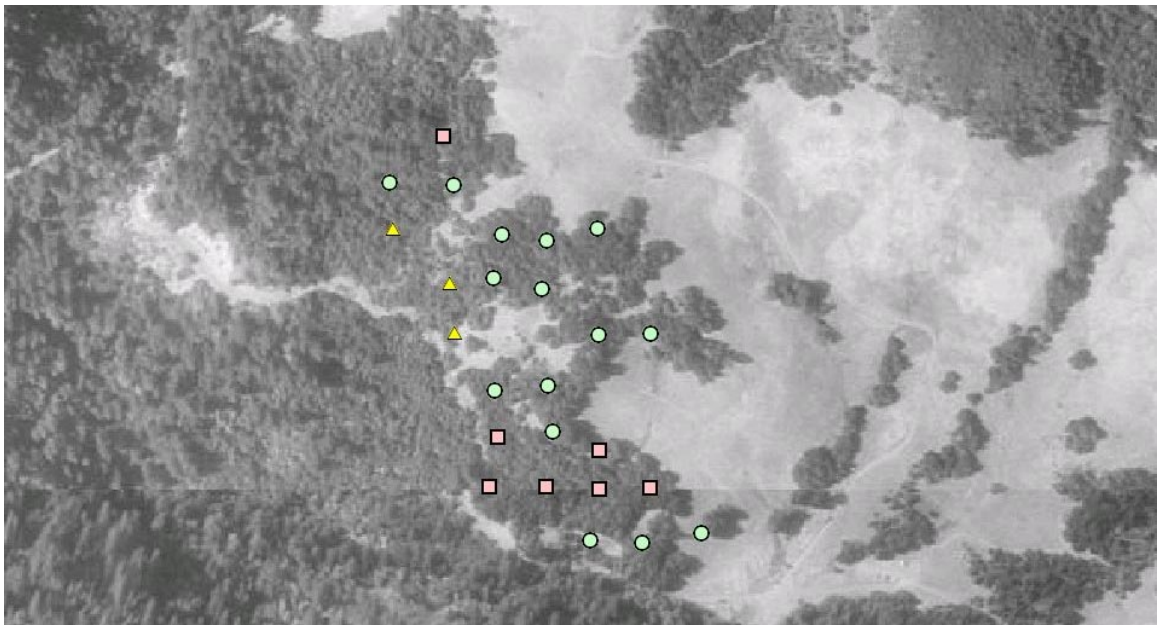

Figure 20. Plot locations at Sugarloaf Ridge State Park. Pink squares represent plots which contained trees with typical symptoms of *P. ramorum* infection. Green circles indicate plots in which no trees showed *P. ramorum* symptoms. Yellow triangles represent plots in which the only symptomatic tree(s) had ambiguous symptoms, i.e., cankers that were not entirely typical of *P. ramorum*. *P. ramorum* has been confirmed at this location by UC researchers.

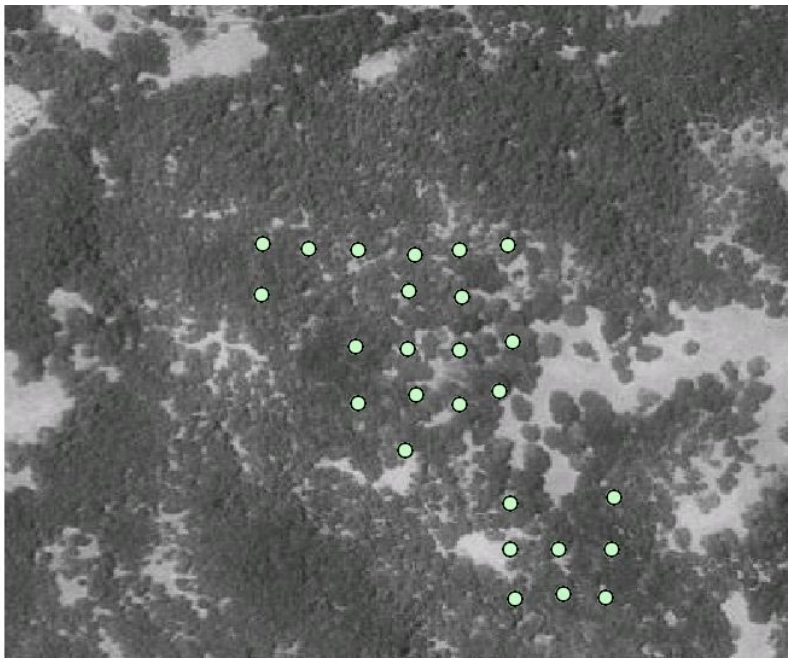

Figure 21. Plot locations at Weston Ranch. No *P. ramorum*-infected trees were seen in plots at this location.

SUMMARY AND CONCLUSIONS

In interpreting our results, one should bear in mind that our results apply specifically to portions of sampled stands that contain the SOD hosts of interest, i.e., tanoak, coast live oak, and black oak. It is precisely in these areas that the direct impacts of SOD will occur. However, average disease levels in our plots are not valid estimators of disease levels countywide. Furthermore, because we limited our sampling to portions of stands with SOD hosts, our data typically do not reflect the composition of the entire stand.

Perhaps the most basic finding of this survey is that stands containing coast live oak and black oak that lack any symptoms of *P. ramorum* are still found in portions of Sonoma County. This has obvious implications for disease management within the county. We believe that efforts to reduce the possible transport of infected host material into noninfected stands within the county should remain a high priority.

Human activities, including the transport of infected plant material, are suspected to be a likely source of disease spread to areas that are widely separated from existing diseased areas. We observed that in several locations, apparent infection rates tended to diminish with increasing distance from roads, campgrounds, or other areas frequented by vehicles. Human-mediated transport of *P. ramorum* is especially significant because disease spread via wind-blown spores is likely to occur over a limited distance. Furthermore, spread via invertebrate or vertebrate vectors has not yet been demonstrated to occur. Even if insects or animals do contribute to the spread of this pathogen, humans are likely to be more efficient vectors of the disease over long distances than other possible vectors.

We found that in most study areas where *P. ramorum* occurs, disease levels in SOD hosts are generally low (Figure 8) and the distribution of symptomatic trees is spotty. From this,

we may infer that either conditions favorable for disease spread and development vary across the landscape and/or the distribution of inoculum has not been very uniform or efficient to date. Variations in disease incidence and intensity could be related to local variations in environmental conditions, the spatial distribution of SOD host trees with high levels of disease resistance within stands, and/or local differences in stand structure or composition with respect to SOD hosts, *P. ramorum* foliar hosts, and non-hosts.

If conditions for local disease spread occur in these stands and disease is mainly limited by the amount of time that has passed since the introduction of the pathogen, we can expect the impact of *P. ramorum* canker to increase over time in many locations. Because disease progress may be influenced by the various factors mentioned above, it is not possible to predict the final outcome of the disease epidemic in these stands. The plots we have established provide a valuable resource for tracking disease progress and examining factors that may speed or retard the progress of the epidemic.

In Sonoma County, tanoak populations in various forest associations appear to be at the greatest risk from *P. ramorum* over the near term. If other tanoak stands attain the high disease levels currently observed at Jack London SP, the impact on tanoak forests will be considerable. Given our current understanding of *P. ramorum*'s environmental preferences, climatic conditions and moisture regimes favorable for SOD development are likely to occur in most areas where tanoak occurs. Substantial amounts of tanoak decline, apparently due to agents other than *P. ramorum,* were seen at Salt Point SP (Figure 7B). Hence, other agents may contribute significantly to tanoak loss in some locations.

Currently, *P. ramorum* is having little impact on black oak in the stands that we studied. However, even without major impacts due to SOD, black oak populations seem likely to decline in many of the study locations because of low rates of regeneration and high levels of tree decline associated with other pathogens (Figure 10). Because levels of background decline and mortality are high and regeneration is low, any additional mortality associated with *P. ramorum* will have a disproportionately large impact.

While natural regeneration of coast live oak is more consistent and plentiful than black oak, the incidence of SOD in coast live oak is also greater than that observed in black oak. Mortality due to *P. ramorum* is expected to add to typically high levels of background decline and mortality, resulting in decreased coast live oak tree densities in many of the study areas. Over the long term, much of this coast live oak mortality could be replaced by seedlings of this species that are recruited to fill resulting canopy gaps. However, it remains to be seen whether coast live oak seedlings can be successfully recruited in the presence of the competing seedling growth of other tree species.

Resurvey of the established plots will provide information on disease progress that cannot be determined from a one-time survey. In some locations where disease levels are high, such as Jack London SP, it may be worthwhile to revisit plots as early as one year after the initial survey because rates of change are likely to be rapid. Furthermore, loss of tagged trees due to tree failure or tree removal could make some plots impossible to relocate if the initial resurvey interval is longer than a year or two in these stands. In stands that are currently free of SOD symptoms, resurvey need not occur for at least two years unless the current wet season initiates a substantial increase in the range of the disease.

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