Phytophthora ramorum canker (sudden oak death) in coast live oak and tanoak, 2000-2007: factors affecting disease risk, disease progression, and failure potential

2007-2008 Contract Year Annual Report

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Cover photo: Douglas-fir and madrone regeneration in a canopy gap formed by mortality of a coast live oak killed by *Phytophthora ramorum*. Due to recruitment of saplings in gaps such as this, Douglas-fir density (stems greater than 3 cm DBH) has increased by 31% since 2001 in study plots.

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SUMMARY

Additional key words: disease progression, survival, tree failure, regeneration

This report discusses findings after eight years of observations in a case-control study examining the role of tree and site factors on the development of *Phytophthora ramorum* stem canker (sudden oak death) in coast live oak (*Quercus agrifolia*) and tanoak (*Lithocarpus densiflorus*). In September of each year from 2000 through 2007, we collected data on *P. ramorum* symptoms, tree condition, midday stem water potential (SWP), and various other factors in 150 circular plots (8 m radius). Each plot was centered around a case (symptomatic) or control (asymptomatic) plot center tree. Plots were located at 12 locations in the California counties of Marin, Sonoma, and Napa in areas where *P. ramorum* canker was prevalent in 2000. At ten locations the predominant canker host was coast live oak, and at the other two locations the canker host was tanoak.

Across all locations, the percentage of trees with *P. ramorum* canker increased between September 2000 and September 2007. The overall percentage of coast live oaks with *P. ramorum* canker symptoms increased from 23% in 2000 to 32% in 2007. Most of the increase in disease incidence occurred between 2005 and 2007. This three-year peak of new disease was associated with abundant late season rains that provided favorable conditions for disease spread in the springs of 2005 and 2006. Between 2000 and 2007, tanoaks showed a significantly larger increase in disease incidence, from 31% to 48%. The percentage of newly symptomatic trees was greater for tanoak than coast live oak in all years except 2005.

Although *P. ramorum* was established at all study locations in 2000, disease incidence at the various location varied widely by 2007, ranging from 8% to 62%. Mortality due to *P. ramorum* also varied widely between locations and was higher at the two tanoak locations than at any of the coast live oak locations. Relatively stable differences in disease incidence between nearby coast live oak locations were mainly associated with differences in California bay cover rather than weather and climate variables.

Among other common forest species in plots containing coast live oak, California bay population numbers have remained relatively constant over the study period. Douglas-fir density increased by 31% between 2001 and 2007 due to sapling recruitment. Madrone populations decreased 7% overall. Three locations had relatively high levels of madrone mortality, ranging from 17 to 31 percent. We have determined that *P. cinnamomi* is associated with declining madrone and bay at one of these locations.

Over three quarters of the initial substantial failures in SOD-affected coast live oaks occurred in dead trees or dead stems of live trees. If only living coast live oak trees with *P. ramorum* canker are considered, more than half of the observed initial failures occurred in live stems or branches. However, almost all of these live trees had advanced to the late disease stage, characterized by invasion of cankered areas by secondary decay fungi (primarily *Hypoxylon thouarsianum* and *Phellinus gilvus*) and wood-boring beetles. Very few failures have been observed in trees with only bleeding cankers (early disease stage). The overwhelming majority of all recorded failures through 2007 have occurred in trees that had *P. ramorum* canker symptoms at the start of the study in 2000. Relatively few trees that have become symptomatic since 2000 had failed by 2007.

Data for the period from September 2000 to September 2007 indicate that *P. ramorum* has not impacted seedling populations of either coast live oak or tanoak. Seedling populations in

coast live oak plots have varied widely between and within locations over time. Despite fluctuations during the intervening years, seedling densities in 2007 did not differ significantly from those observed in 2000. Tanoak seedling densities have not fluctuated as widely as coast live oak seedling numbers and also showed no significant change between 2000 and 2007.

We made timed counts of *P. ramorum* foliar symptoms on California bay at intervals from September 2005 through April 2008 to determine how foliar infection levels changed over time in specific patches or zones of bay foliage. All monitored zones showed peak foliar infection levels in mid 2006 following the abundant and extended rainfall of spring 2006. Foliar infection levels dropped significantly by fall 2006 and remained low into spring 2008. A few monitored bay zones maintained relatively high foliar infection levels into 2008, suggesting that long-term inoculum carryover in bay may be spatially variable. Although repeated bay foliar symptom counts from fall 2005 were not correlated with any subsequent counts, counts from spring and fall 2006 were correlated with each other and with counts made in spring and fall of 2007. Correlations between bay foliar symptom counts may therefore depend to some degree on whether foliar disease levels are increasing or decreasing over the observation period.

INTRODUCTION

Phytophthora ramorum, the causal agent of sudden oak death (SOD), causes bleeding bark cankers on the main stems of tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and California black oak (*Q. kelloggii*) (Garbelotto and others 2001, Rizzo and others 2002). The bark cankers can expand over time and eventually girdle susceptible trees (Rizzo and others 2002). The sapwood-decaying fungus *Hypoxylon thouarsianum*, ambrosia beetles (*Monarthrum* spp.), and oak bark beetles (*Pseudopityophthorus* spp.) are commonly associated with *P. ramorum*-infected trees in later stages of decline (Garbelotto and others 2001).

We initiated a long-term study to follow disease progress and evaluate disease risk factors in the summer of 2000, shortly after *P. ramorum* (then unnamed) was identified as the cause of SOD. Most of the trees in the study are coast live oaks, but we collected parallel data on tanoaks at two locations for comparative purposes. We used a case-control study design to test whether various tree factors and plot/stand factors were related to the development of *P. ramorum* bole cankers in coast live oaks in areas where the disease was common.

Models based on results from the first three years of this project (Swiecki and Bernhardt 2001, 2002ab) were the first to document that California bay (*Umbellularia californica*) cover and density near coast live oak are significantly correlated with disease risk. Other variables that are positive predictors of disease risk in coast live oak include high canopy dominance (tree canopy sky exposure), low levels of water stress (as assessed by measuring stem water potential [SWP]), larger stem diameter, multiple main stems, and the absence of tree decline associated with other disease agents. Based on the effects of these variables in disease risk models, we inferred that trees with faster growth rates (associated with larger diameter, higher SWP, greater sky exposure, lack of decline from other agents) had an elevated risk of developing *P. ramorum* canker. More recently (Swiecki and Bernhardt 2005), we showed that disease risk in coast live oak also increases with bark thickness and the amount of bark fissures that show evidence of recent expansion. These risk factors add further support to the concept that fast-growing trees are more likely to develop *P. ramorum* canker.

In this report, we have updated our observations on disease progress, mortality, and failure in trees affected by *P. ramorum* canker. Annual observations of disease incidence and severity in these plots have allowed us to obtain a clearer picture of the length of time that elapses between the onset of symptoms and tree failure or mortality. These observations also help to document how infection potential varies between years in coast live oak and tanoak.

METHODS

Study site selection

During September 2000, we established plots at 12 study locations (*table 1*, *fig. 1*). Study sites were selected on the basis of appropriate vegetation type (adequate representation of coast live oak or tanoak), the presence of cases (trees with symptoms of *P. ramorum* canker) and controls (asymptomatic trees) in the study area, and absence of recent disturbances that might affect tree health (e.g., root-damaging construction). Plots were established in areas where *P. ramorum* had been shown to be prevalent. Coast live oak was the subject host species at 10 of the 12 locations; tanoak was the subject species at the remaining two locations.

Table 1. Locations of plots and host species studied.

Location number	Location	County	Approximate latitude and longitude	Number of plots	Subject tree species
1	Marin Municipal Water District (MMWD) watershed - Azalea Hill area	Marin	37.9723 N 122.6274 W	12	coast live oak
2	MMWD-Pumpkin Ridge south	Marin	37.9527 N 122.5949 W	16	coast live oak
3	MMWD-Pumpkin Ridge north	Marin	37.9599 N 122.5989 W	11	coast live oak
4	MMWD-Phoenix Lake area	Marin	37.9590 N 122.5770 W	11	coast live oak
5	China Camp SP - Miwok Meadows area	Marin	38.0044 N 122.4848 W	16	coast live oak
6	China Camp SP - SE Buckeye Point area	Marin	38.0044 N 122.4768W	12	coast live oak
7	Woodacre (Private land)	Marin	38.0175 N 122.6472 W	12	coast live oak
8	Lucas Valley (Private land)	Marin	38.0432 N 122.5996 W	12	coast live oak
9	Muir Woods NM / Mt. Tamalpias SP	Marin	37.9024 N 122.5839 W	10	tanoak
10	Wall Road (Private land)	Napa	38.4092 N 122.4751 W	13	coast live oak
11	Novato (Private land)[1]	Marin	38.0988 N 122.6273 W	13	coast live oak
12	Jack London SP	Sonoma	38.3450 N 122.5616 W	12	tanoak

[1] This site was previously listed as being on Marin County Open Space District land.

Plot selection

At each study location, we established 10 to 16 circular 8 m radius (0.02 ha) fixed-area plots, each of which was centered at a subject tree. The number of plots per location was limited by the time constraints associated with making stem water potential measurements. After determining that symptomatic trees (cases) were present in adequate numbers in the stand, we established a random starting point and searched for the nearest case or control tree starting from that point. This tree became the first subject tree and the center of the first plot. Subsequent tree-centered plots were spaced approximately 20-30 m apart. Actual interplot spacing varied with vegetation and terrain, but to avoid overlap between plots, no two adjacent plots were

located closer than 16 m apart. We attempted to alternate case and control plots, but if the designated subject tree type (e.g., control) did not exist within a 4-8 m search radius of the target point, the other subject tree type was selected. Potential cases and controls were rejected if they did not have foliage low enough to be accessed for water potential measurements. In general, we attempted to distribute the plots across a range of topographic positions, slopes, and aspects.

We marked the center subject tree in each plot with a numbered aluminum tree tag. Tags were placed at varying heights, but generally point toward the next successive plot. To help relocate plot center trees within each study site, we recorded the distance and azimuth readings between plots. We subsequently determined the coordinates of the plots at each location using a GPS receiver with an external, mast-mounted antenna, although the position of some plots at location 9 could not be determined with GPS due to poor satellite reception.

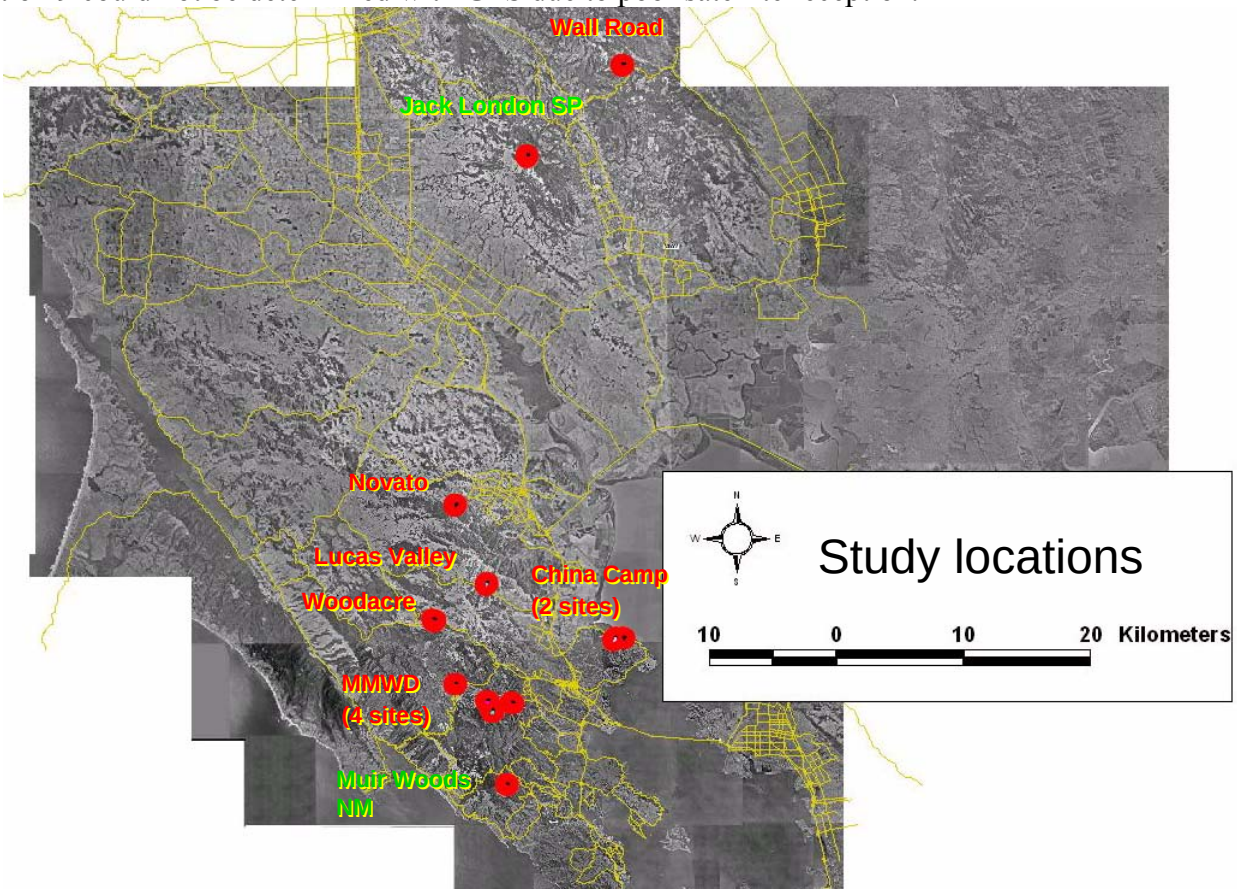

Figure 1. Map showing locations of study areas in Marin, Napa, and Sonoma counties. Background image is a mosaic of USGS digital aerial orthophotos.

Stem water potential measurements

In September of each year from 2000 through 2004, we collected midday stem water potential (SWP) readings on the center subject tree in each plot during the peak midday period (about 1300-1530 PDT). In addition, from 2001 though 2004, we also took SWP readings on additional trees in 45 of the plots (one additional tree per plot except one plot with two additional trees) for comparative purposes. Starting with September 2005, SWP monitoring was reduced to include only three plot center trees at each of the ten coast live oak locations. The selected trees were spread out within each location area. To allow for continued annual measurements on the

same trees, we selected trees that had an adequate supply of leaves within reach which could be used for future SWP measurements and avoided trees that appeared likely to die within the next few years.

SWP measurements were made following methods outlined by Shackel (2000). On each tree, we selected a minimum of two shoot tips with several leaves for measurement. We selected shoots and leaves that branched directly off the trunk or from main branches near the trunk, or from basal sprouts (primarily for tanoak). Each shoot tip was sealed in a clear plastic bag and overbagged with a larger opaque reflective plastic bag. These bags prevent the leaves from transpiring and overheating. Bags were left in place for at least 1 hour to allow leaf water potential to equilibrate to that of the subtending stem. At the time of the reading, the outer opaque bag was removed and the shoot tip was excised and placed into the pressure chamber while still sealed in the inner plastic bag. Two SWP readings were made on most trees. In general, two valid SWP measurements from a single tree were within 0.05 to 0.1 MPa of each other. SWP measurements were made with a pump-up pressure chamber (PMS Instrument Co., Corvallis OR) fitted with a 10.2 cm diameter 40 bar (0.4 MPa) gauge with 1% accuracy full scale. Other methods associated with SWP readings have been described previously (Swiecki and Bernhardt 2005).

Additional tree and plot variables

Plot center trees and the 47 extra SWP trees were rated for origin class (seed or coppice); stem count; DBH; and amount of crown exposure to overhead sunlight, and other factors (*table 2*). Plot variables recorded (*table 3*) included plot slope and aspect; total basal area; tree counts and condition by species; plot canopy cover; woody understory cover; cover of selected tree species and poison oak (*Toxicodendron diversilobum*). We also recorded the disease status of all other coast live oak, California black oak, and tanoak trees in the plot with respect to ***P. ramorum*** and other pathogens, and counts of regeneration of these three species. Coast live oak, black oak, and tanoak trees other than the plot center tree are collectively referred to as plot trees in this report.

In 2003, we collected data on physical characteristics of the bark of coast live oak plot center and extra SWP trees at all locations except 9 and 12, which did not include coast live oaks (Swiecki and Bernhardt 2004). One of these variables, the presence of unweathered, brown bark in the center of bark furrows or fissures, was shown to be correlated with disease risk (Swiecki and Bernhardt 2004) and starting in 2004, we rated the relative abundance of this last characteristic using the 0-6 scale described below. This variable was reassessed in 2005 through 2007 for all plot trees.

We used the following arcsine-transformed percentage scale for most ocular estimates of percentages: 0 = not seen, 1= less than 2.5%, 2 = 2.5% to 19%, 3 = 20% to 49%, 4 = 50% to 79%, 5 = 80% to 97.4%, 6 = more than 97.5%.

Bay leaf symptom counts

We assessed foliar infection levels in California bay by performing 45-second timed counts of symptomatic leaves within the canopy of previously defined zones of bay canopy (Swiecki and Bernhardt 2007a). A total of 106 mapped bay zones had been mapped around 37 coast live oaks in September and October 2005. The zones contained varying numbers of bay trees: 47 zones had one bay tree, 37 zones had clumps of two to three bay trees, and 22 zones had greater numbers (4 to 19) of bay trees. Initial counts were made in September and October 2005. A

subsample of the zones was recounted in January 2006. Counts of foliar symptoms in the all bay zones were made using the same methods between late May and early August 2006, in September 2006, June 2007 and September 2007. The bay zones at location 5 were also recounted in April 2008. All counts were made by the same observer for all trees and all sampling dates.

Statistical analyses

We used JMP® statistical software (SAS Inc., Cary NC) for data analysis. Unless otherwise indicated, effects or differences are referred to as significant if $p \leq 0.05$. Repeated measures multivariate analysis of variance (MANOVA) was used to examine the effects of factors on outcomes that have been repeatedly assessed in plots. The likelihood ratio chi square statistic was used to test the significance of difference of proportions in 2×2 contingency tables. We also used the Tukey-Kramer HSD for mean separation following a significant F level in a one-way analysis of variance.

Table 2. Tree variables measured for plot center trees, other plot trees, and selected out of plot trees.

Variable	Trees rated[1]	Year(s) evaluated[2]	Method	Scale/units and notes
General tree descriptors				
Tree species	C,A,P: O:	2000 2002		*Q. agrifolia, L. densiflorus or Q. kelloggii* (plot trees only)
Origin class	C,A: O:	2000 2002	visual assessment	seed (0) or sprout (1)
Distance to plot center	A: P,O:	2001 2002	laser rangefinder	m; recorded for plot trees in 2002
Azimuth to plot center	A: P,O:	2001 2002	compass	degrees; recorded for plot trees in 2002
DBH	C: A: P,O:	2000 2001 2002	flat tape measure	cm
Sky-exposed canopy	C: A: P,O:	2000 2001 2002	visual estimate	pretransformed 0-6 scale[3]; percent of canopy projection area with unobstructed access to direct overhead sunlight
Number of stems from ground	C: A: P,O:	2000 2001 2002	count	stems/tree
***P. ramorum* canker-related symptoms**				
Phytophthora-related symptoms	C,A,P: O:	2000-on 2002-on	visually assess symptoms present	(0) No symptoms (1) Early - bleeding cankers only (2) Late - cankers plus beetle boring and/or *H. thouarsianum* (3) Dead as result of *Phytophthora* infection; evidence of bark cankers present
Recent bleeding from cankers	C: A: P:	2000-on 2001-on 2002-on	visual assessment of exudate	Present (1) scored if bleeding appeared to have occurred within the previous 4-6 months / otherwise absent (0)
Phytophthora canker count	C: A:	2000-on 2001-on	count	Estimated on basis of external bleeding spots and limited inspection of canker margins. In 2000, only an overall count for all stems was made. In 2001, counts per stem for multistemmed trees were also made.
Percent girdling due to *Phytophthora* cankers	C,A:	2000-on	visual estimate	pretransformed 0-6 scale[2] 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 in some trees. In general, girdling ratings are difficult and less reliable on completely dead trees. In 2000, a single overall rating was made for all stems. Starting in 2001, individual ratings were also made for each stem of multistemmed trees.
Height of upper and lower *P. ramorum* canker margins above grade	C,A:	2003	tape measure	Height (cm) above soil level was noted for the upper edge of the highest canker and lower edge of the lowest canker on symptomatic trees.
Stems with Phytophthora symptoms	C,A,P,O:	2000-on	count	infected stems/tree
Dead stems	C,A,O:	2000-on	count	dead main stems/tree and likely cause of stem death (Phytophthora canker or other)

Table 2. Tree variables measured for subject trees, other plot trees, and selected out of plot trees. (continued)

Variable	Trees rated[1]	Year(s) evaluated[2]	Method	Scale/units and notes
***P. ramorum* canker-related symptoms (continued)**				
Tree dead / cause	C,A,P,O:	2000-on	visual assessment	Causes: (0) not dead (1) Phytophthora canker; (2) other agent(s); (3) unable to determine (4) Phytophthora canker plus other agent(s) Tree scored as dead if all main stems are dead, even if small live basal sprouts are present.
Hypoxylon thouarsianum Percent girdling	C: A: P,O:	2000-on 2001-on 2002-on	Visual estimate based on presence of fruiting bodies	pretransformed 0-6 scale[2] Percent of circumference affected estimated based on projection of cankered areas as if all were viewed on same cross section;
Hypoxylon thouarsianum Highest density in 0.1 x 1 m vertical strip	C,A,O:	2002	count	Count of fruiting bodies. Individual lobes counted separately.
Wood boring beetles in main stem	C,A,P,O:	2000-on	Shape and size of exit holes	Type of beetle based on shape of exit holes
Abundance of bark and/or ambrosia beetles in main stem	C: A: P,O:	2000-on 2001-on 2002-on	presence of boring dust and/or holes	(0) none seen (1) low (2) moderate (3) high
Other tree condition variables				
Canopy thinning	C: A: O:	2000-on 2001-on 2002	visual estimate	0-2 Scale: (0) none; (1) slight; (2) pronounced
Canopy dieback	C: A: P: O:	2000-on 2001-on 2002-on 2002	visual estimate	pretransformed 0-6 scale[3] Based on percent dead crown volume
Severe tree decline due to other agents	C,A,P: O:	2000-on 2002	visual assessment	yes (1)/ no (0) Trees scored in decline if overall condition was poor enough that death within 10 years was judged to be likely.
Decay impact	C: A: O:	2000-on 2001-on 2002	visual assessment	0-3 Scale: (0) none; (1) low; (2) moderate: (3) high 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 was based on the type(s) of decay present, location of decay within the tree, and the estimated extent of decay as rated by a trained observer. Levels were recoded to three classes as follows for some analyses: (1) none; (2) low or moderate; (3) high
Status change	C,A,P:	2000-on	comparison of tree data from 2 successive years	Evaluation based primarily on canker extent, colonization by secondary organisms, and dieback. (0) no change; (1) improved condition; (-1) degraded condition
Epicormics	C: A: O:	2000-on 2001-on 2002	visual assessment	0-2 Scale: (0) none; (1) few; (2) numerous

Table 2. Tree variables measured for subject trees, other plot trees, and selected out of plot trees. (continued)

Variable	Trees rated[1]	Year(s) evaluated[2]	Method	Scale/units and notes
Other tree condition variables (continued)				
Live basal sprouts	C,A,P:	2000-on	visual observation	presence (1) / absence (0) scored for dead trees only Trees are scored as dead if all main stems are dead even if some live basal sprouts are present.
Other agents and symptoms	C,A,P,O:	2000-on	visual observation	Presence of wood decay fungi fruiting bodies and canker rot or root rot symptoms were noted.
Defect codes	C,A: P: (if failed)	2002-on 2002-on	visual observation	The presence of various structural defects that may contribute to the risk of tree failure were coded. (1) Dead branch or branch stubs (2) Multiple trunks/ codominant stems (3) Hollow branch stubs (4) Dense crown (5) Heavy lateral limbs/ excessive branch end weight (6) Uneven branch distribution: one sided (7) Uneven branch distribution: top heavy (8) Multiple branches from same point (9) Embedded bark in crotch (10) Crook or sweep (11) Leaning trunk (12) Cracks or splits (13) Kinked or girdling roots (14) Cavity (15) Decay column
Tree failure	C,A,P:	2000-on		Failures of bole or branches >20 cm diam noted if present
Failure type	C,A,P:	2001-on		(1) Root (2) Root crown (lower edge of fracture was near soil surface) (3) Bole (main stem) (4) Scaffold (lowest first order branches arising from bole) (5) Branch (all other branches)
Tree condition at time of failure	C,A,P:	2001-on	based on condition of twigs and foliage	(1) Live (2) Dead (3) Uncertain
Estimated failure date	C,A,P:	2001-on	based on weathering of failed surface, degradation of failed part, previous observations, etc.	(1) within previous 6 months (2) 6-12 months prior to rating More precise dates were estimated if supportable by observations (e.g., green foliage on failed part)

Table 2. Tree variables measured for subject trees, other plot trees, and selected out of plot trees. (continued)

Variable	Trees rated[1]	Year(s) evaluated[2]	Method	Scale/units and notes
Bark thickness	C (dead), A,O: 2003 C (live): 2004		bark probe	mm
Brown bark from recent bark expansion in fissures	C,A,P:	2003-on	visual assessment	2003: present/absent 2004-on: pretransformed 0-6 scale[3] – Percent of cumulative fissure length in lower 2 m of bole showing brown color
Lichen abundance (lower 2m of bole)	C,A:	2003	visual ranking of lichen cover	(0) none; (0.5) trace; (1) low; (2) moderate to high
Moss abundance (lower 2m of bole)	C,A:	2003	visual estimate of moss cover	(0) none; (0.5) trace; (1) low; (2) moderate to high
Moss location	C,A:	2003	visual assessment	(1) basal only (lower 1-2 m of bole) (2) extending up bole into upper bole and/or canopy
Type of bark fissures present	C,A:	2003	visual assessment	(1)shallow; (2) medium; (3) deep
Deep bark cracks	C,A:	2003	visual assessment	present/absent (Unlike fissures, cracks are abrupt discontinuities that extend deep into the bark or to the cambium that are not associated with normal growth patterns.)
Bark texture	C,A:	2003	visual description	bark texture was described using one or more of the following characteristics: smooth, irregular, striate, checkered, corky, furrowed

[1]Tree types: **C**=plot center tree; **A**=additional trees used for stem water potential readings starting in 2001; **P**=other plot trees; **O**= trees located beyond plot edges used for coring in 2002 (Swiecki and Bernhardt 2003a) and bark probe measurements in 2003. Only asymptomatic trees beyond plots were chosen for coring in 2002.

[2]Variables scored in a single year were reevaluated only for trees which showed a change from the original values.

[3]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. Except as noted, all variables were measured in the 8 m radius fixed-area plots.

Variable	Year(s) evaluated[1]	Method	Scale/units and notes
Plot slope	2000	clinometer	percent
Plot aspect	2000	compass	degrees
Plot drainage	2000	visual observation	none; creek/drainage with surface water; dry creek or drainage
Plot drainage proximity	2000	visual observation	0 if in plot; otherwise estimate meters from plot edge
Plot tree canopy cover	2000, 2006, 2007	visual estimate	pretransformed 0-6 scale[2]; overall tree cover in plot
California bay cover	2002	visual estimate	pretransformed 0-6 scale[2]; bay cover in plot, including regeneration
Madrone cover	2002	visual estimate	pretransformed 0-6 scale[2]; madrone cover in plot, including regeneration
Woody understory cover	2000, 2006, 2007	visual estimate	pretransformed 0-6 scale[2]; includes both shrubs and small (<3 cm DBH) tree regeneration
Plot shrub cover	2001, 2006, 2007	visual estimate	pretransformed 0-6 scale[2]
Poison oak cover	2002	visual estimate	pretransformed 0-6 scale[2]
Overstory canopy trees species in plot	2001	visual assessment	list of species; overstory canopy trees do not have to be rooted within the plot.
Tree density / species composition	2000, 2006, 2007	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 at least 1 m count as separate trees
Count by general tree health class (trees other than SOD hosts[3])	2001, 2006, 2007	tree count by species, subcategorized by symptom class and canopy position (overstory/understory)	Symptom classes: (1) live (2) decline (3) dead
SOD host[3] regeneration	2000-on	count or estimate if >100	regeneration = seedlings and saplings <3 cm dbh
Disease incidence in SOD host[3] regeneration	2000-on	count or estimate percent if count > 100	Disease may be due to *P. ramorum* and/or other agents or factors
Dead SOD host[3] regeneration	2000-on	count	Cause of mortality in regeneration was not determined
Regeneration of trees other than SOD hosts[3]	2000, 2006, 2007	presence noted by species	regeneration: seedlings and saplings <3 cm dbh
Other pathogens/agents	2000-on	note presence	listing of agents and symptoms observed, including various decay fungi, canker rot, root disease, *H. thouarsianum*, and beetles
Woody understory species	2001, 2006, 2007	note presence	list shrubs and woody vines present within plot; herbaceous species and grasses were not scored
Increase in weedy herbaceous cover	2006, 2007	visual assessment	noted whether weedy herbaceous understory cover in plot had increased substantially since 2000
Disturbance	2000-on	Note type of disturbance	roads, trails, logging, etc. within plot or near edge of plot were noted
Basal area[4]	2000	survey laser reticle	reticle BAF = 5 m^2/ha

[1]Variables scored in a single year were reevaluated only for trees which showed a change from the original values.

[2]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%

[3]SOD hosts = hosts of *P. ramorum* stem canker, i.e., coast live oak, California black oak, and tanoak

[4]Basal area measurements were made on a variable-radius plot centered at the plot center tree.

RESULTS

Symptom development and disease progress 2000-2007

Overall disease incidence and mortality

Baseline levels of disease in plots were established when the plots were originally evaluated in September 2000. Additional newly symptomatic tanoak and coast live oak trees have been observed at each annual September evaluation since then. Figure 2 shows the percent of each year's asymptomatic trees which became symptomatic in each September survey. Both 2005 and 2006 had relatively high rainfall (*fig. 3*) including substantial periods of rainfall in the spring. Coast live oak showed the greatest increases in newly symptomatic trees in 2005, 2006, and 2007 (*fig. 2*), associated with relatively high amounts of spring rainfall in 2005 and 2006 (*fig. 3*).

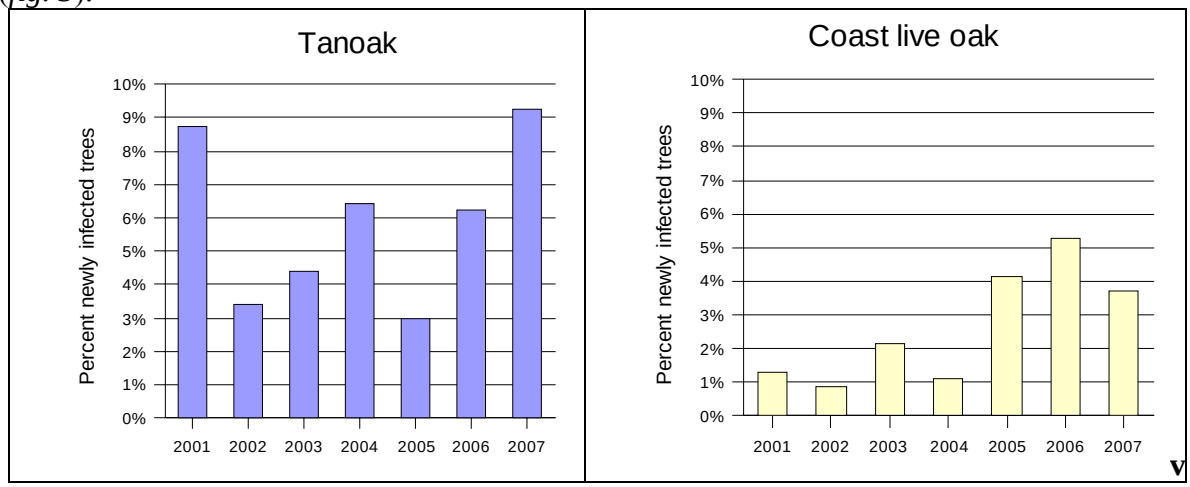

Figure 2. Newly symptomatic tanoak and coast live oak trees observed in September of each year as a percent of the number of asymptomatic trees present in the previous year. Total number of live asymptomatic trees present in 2000 was 126 for tanoak, 470 for coast live oak.

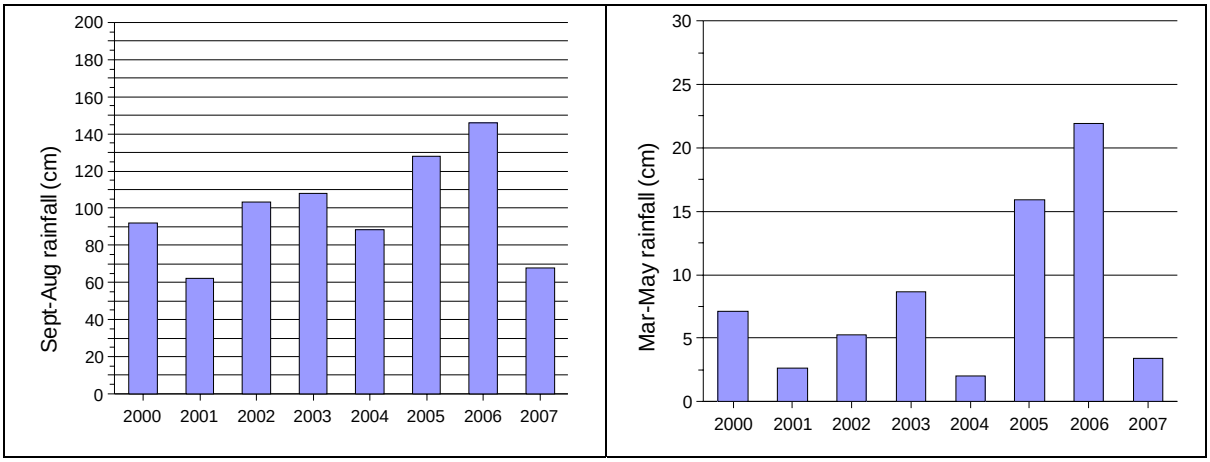

Figure 3. Average rainfall totals for weather stations nearest to the study locations for the previous September to August period of the years shown (left) and for March to May of the years shown (right).

Overall, *P. ramorum* canker incidence among tanoaks has increased from 31% in 2000 to 48% in 2007 (*fig. 4*). *P. ramorum* canker incidence among coast live oaks has increased from 23% in 2000 to 32% in 2007 (*fig. 4*). A logistic model of the disease incidence data for both species for 2000 and 2007 showed significant differences between species (likelihood ratio $p<0.0001$) and years (likelihood ratio $p<0.0001$).

The percentage of trees killed by *P. ramorum* was much higher for tanoak than for coast live oak at the start of the study (*fig. 4*). This trend has continued over the 2000-2007 interval. The absolute change in percent mortality due to *P. ramorum* (*fig. 4*) is much greater for tanoak (25%) than for coast live oak (9%). However, the relative increase in SOD-related mortality over the 2000 to 2007 interval was similar for both species (3× increase for tanoak, 3.25× increase for coast live oak).

In some coast live oaks, symptoms became less obvious over time and appeared to go into remission to the point that the original canker could no longer be distinguished. Therefore, some coast live oaks classified as showing early symptoms of infection (bleeding cankers only) in one or more years were reclassified as asymptomatic because the cankers had become inactive and indistinct in succeeding years. During the favorable infection years of 2005 and 2006, some trees with inactive infections again became symptomatic. In most of these cases, new cankers were distinct from the older, inactive cankers, suggesting that they represented new infections rather than reactivation of inactive cankers.

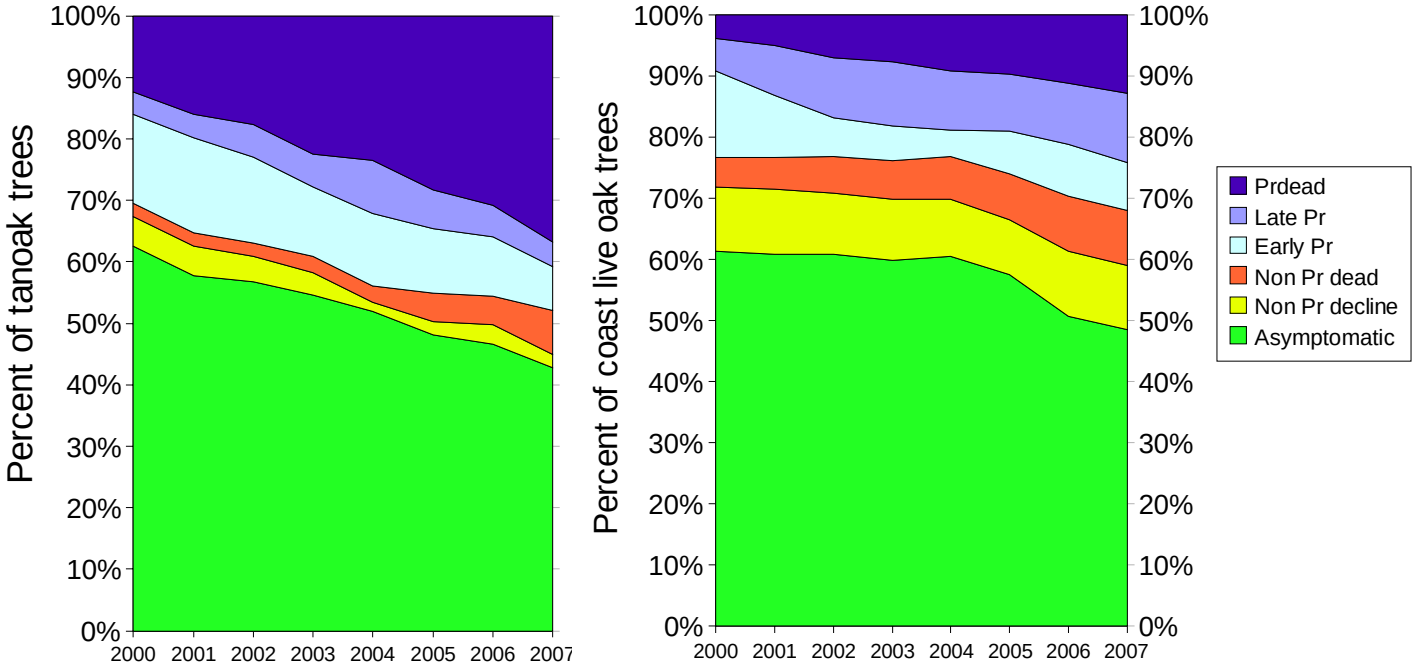

Figure 4. Changes in health of all tanoak (n=187) and coast live oak (n=655) study trees from September 2000 to September 2007. **Dead Pr** = tree dead as a result of *P. ramorum;* **Late Pr** = live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **Early Pr** = live trees with *P. ramorum* cankers only; **Other dead** = tree dead due to agents other than *P. ramorum;* **Other decline**=tree in severe decline due to agents other than *P. ramorum;* **Asymptomatic**= no evident symptoms of *P. ramorum* infection or decline due to other agents.

Time from symptom onset to mortality

Most of the *P. ramorum*-related mortality that has occurred since 2000 was seen in trees that had *P. ramorum* canker symptoms in 2000; 52% of the tanoaks and 72% of the coast live oaks killed by *P. ramorum* between 2001 and 2007 were symptomatic in 2000. Most of the difference in mortality between tanoaks and coast live oaks among this cohort of initially symptomatic trees developed in the first year (*fig. 5*). Cumulative mortality for oaks and tanoaks from the cohort that was symptomatic in 2000 were similar between years 2 and 4. However, tanoaks from this cohort showed a sharper increase in mortality rates in years 5 and 6 (2005 and 2006, respectively) compared to coast live oak. Both of these years had higher levels of rainfall that were more favorable for inoculum production and new infections, so it is possible that the elevated mortality among these tanoaks in years 5 and 6 was associated with additional bole infections that developed in these years. Among live trees that had *P. ramorum* canker symptoms in 2000, 35% of coast live oaks and 74% of tanoaks had died by 2007 (*fig. 5*).

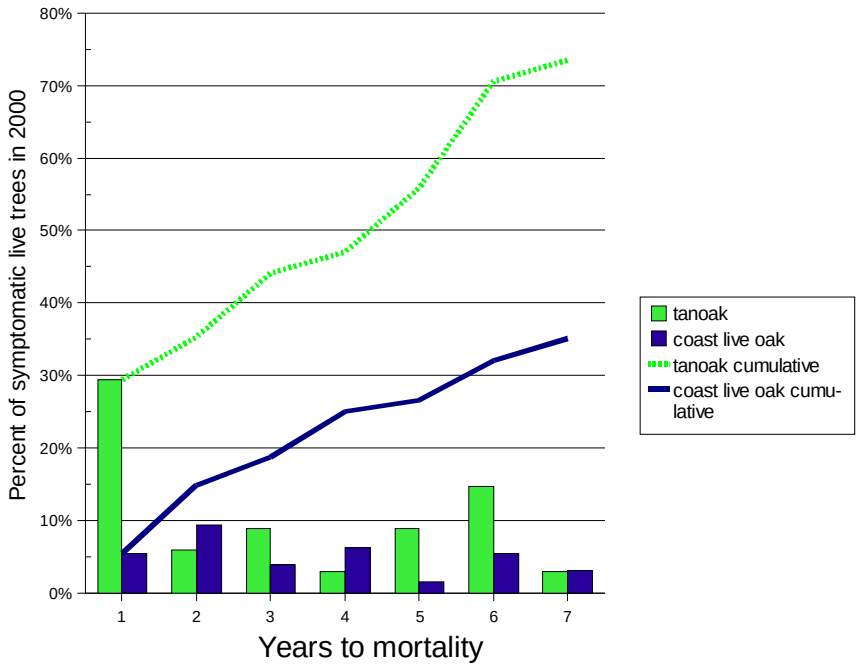

Figure 5. Distribution of the number of years from the start of the study to tree mortality (bars) and cumulative mortality from 2001 through 2007 (lines) for trees that were alive and showed symptoms of *P. ramorum* infection in September 2000. The date of initial symptom development for this cohort of trees is unknown (tanoak n=34, coast live oak n=128).

The trend in mortality rates over time among trees that first developed visible symptoms after 2000 (*fig. 6*) was similar to that seen in the cohort discussed above. For tanoaks that first became symptomatic during the period from 2001-2007 (n=45), 60% have died, many of these within a year or two of becoming symptomatic (*fig. 6*, top). Trees listed in the "less than one year category" were dead in the first year that symptoms were observed (i.e., status changed from asymptomatic in year *n* to dead due to *P. ramorum* in year *n*+1). Among coast live oaks that first became symptomatic between 2001 and 2007, only 12% have died (*fig. 6*). Because trees became symptomatic in different years, the minimum survival times for the time-censored

trees are at least one to six years (*fig. 6*, bottom). A high proportion of the surviving coast live oaks are shown in the "at least 2 year" category because they initially developed symptoms in 2006 (*fig. 2*).

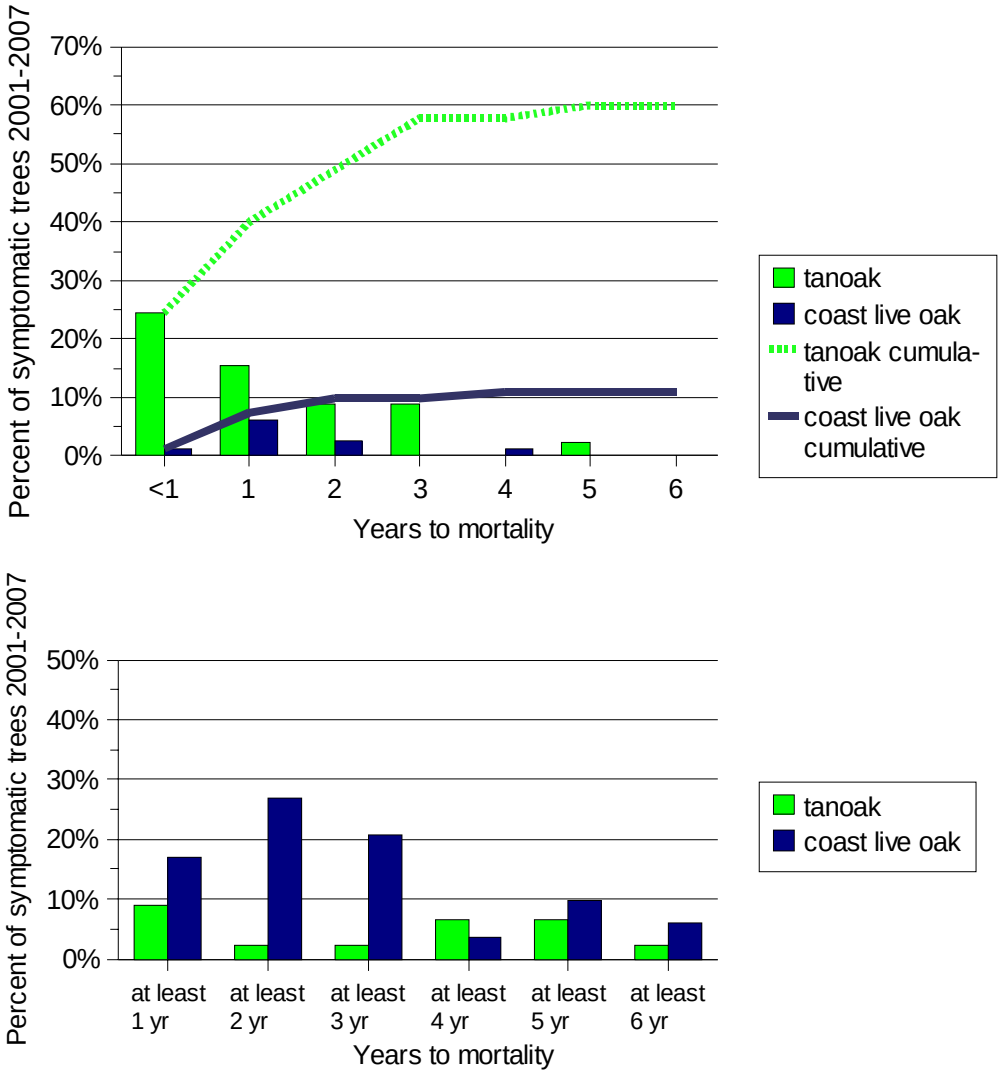

Figure 6. Years elapsed between symptom onset and mortality for trees that initially became symptomatic between 2001 and 2007 (tanoak n=45, coast live oak n=82). Top graph shows year by year (bars) and cumulative data (lines) for trees that died by September 2007; lower graph shows the minimum possible survival period for trees that were still live in September 2007

Disease incidence and mortality by study location

Figures 7 and 8 show changes in *P. ramorum* symptom status between 2000 and 2007 for trees at each of the 12 study locations. At tanoak locations 9 and 12 and coast live oak locations 3, 7, 8, and 11, more than 20% of the trees without symptoms in 2000 developed *P. ramorum* canker symptoms by 2007 (*fig. 7*, right). Most other locations showed increases in disease

incidence that fell between 10 and 20%. Coast live oak location 2 was notable for the low number of new infections observed across the entire 2000-2007 period (*fig. 7*).

Among the coast live oak locations, variation in the amount of new disease is likely to be related to factors other than weather. For example, locations 2 and 3, which differed substantially with respect to both overall disease incidence and disease increase between 2000 and 2007 (*fig. 7*, *8*) are only about 0.8 km apart. However, average bay cover in the plots at location 2 was significantly less (Tukey HSD $p<0.05$) than that at location 3 (1.1 versus 2.4, respectively using 0-6 pretransformed scale). Furthermore, all plots at location 3 had some bay cover whereas half of the plots at location 2 had no bay cover. Average plot bay cover at location 4, about 2 km from locations 2 and 3, did not differ significantly from that at location 3, but trees at location 4 have consistently had the lowest average SWP readings, suggesting that this factor interacts with other factors to influence disease risk.

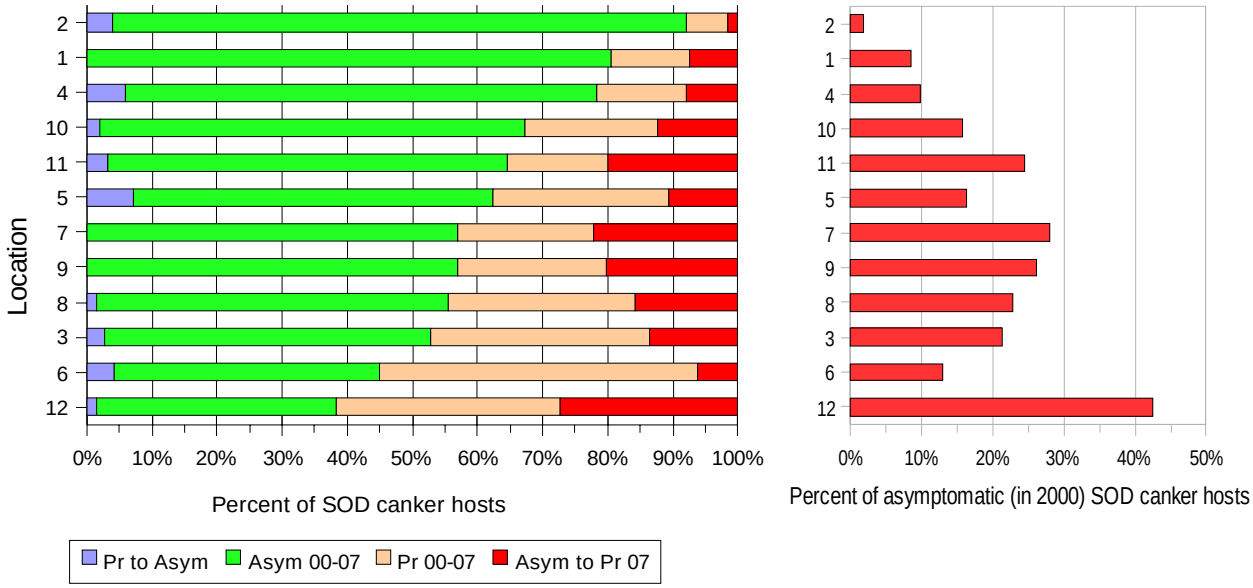

Figure 7. Percent of SOD canker hosts (coast live oak, California black oak, and tanoak) showing changes in overall *P. ramorum* canker symptom status between 2000 and 2007 by study location. **Left:** disease status changes as percent based on the total tree population present in 2000, including trees that were dead in 2000 or died between 2000 and 2007. **Right:** trees that developed *P. ramorum* canker symptoms after 2000 (**Asym to Pr**) expressed as the percentage of asymptomatic trees present in 2000. Location numbers are shown in *table 1* and are sorted in order of increasing *P. ramorum* canker incidence in 2007. **Pr 00-07** = tree with *P. ramorum* canker symptoms in 2000 through 2007; **Asym 00-07**= trees without *P. ramorum* canker symptoms in 2000 through 2007 (includes trees scored as having *P. ramorum* canker symptoms only in 2000); **Pr to asym**= trees scored with *P. ramorum* canker symptoms in 2000 and at least one additional year but asymptomatic in 2007.

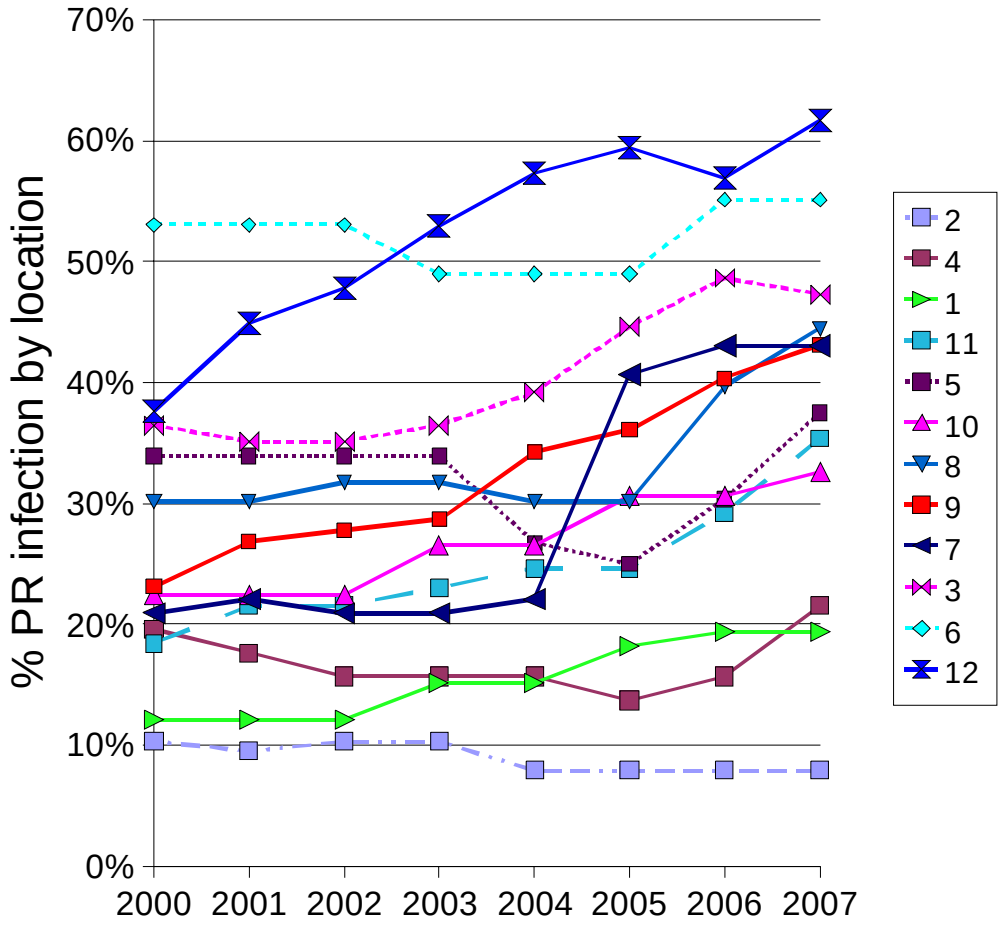

Figure 8. Percent of SOD canker hosts (coast live oak, California black oak, and tanoak) at each of the study locations with *P. ramorum* (PR) symptoms from 2000 through 2007. Graph shows decreases in the disease incidence at some locations because trees were reclassified as asymptomatic if symptoms went into remission, even if *P. ramorum* infection had been confirmed by culturing canker tissue.

Figure 9 shows the overall contribution of *P. ramorum* canker to tree disease and mortality at each of the 12 study locations. Tree mortality includes all observed mortality occurring after 2000 plus trees that were dead in 2000 but were estimated to have died within the previous 10 years (i.e., mortality dating from 1990 or later). Levels of mortality from all causes varied greatly across the study locations. Mortality rates at the coast live oak study locations ranged from 8% of the study trees at location 4, to 39% at location 6. Overall mortality rates among tanoak at the two tanoak study locations were 44 and 48%. By a large margin, *P. ramorum* has been the leading cause of mortality among study trees at both tanoak locations. Among coast live oak locations, *P. ramorum* canker was the primary or a contributing factor in the majority of tree deaths at six of the ten locations (*fig. 9*). Wood decay fungi, particularly species of *Inonotus* and *Ganoderma*, have been important causes of tree mortality at several of the coast live oak locations.

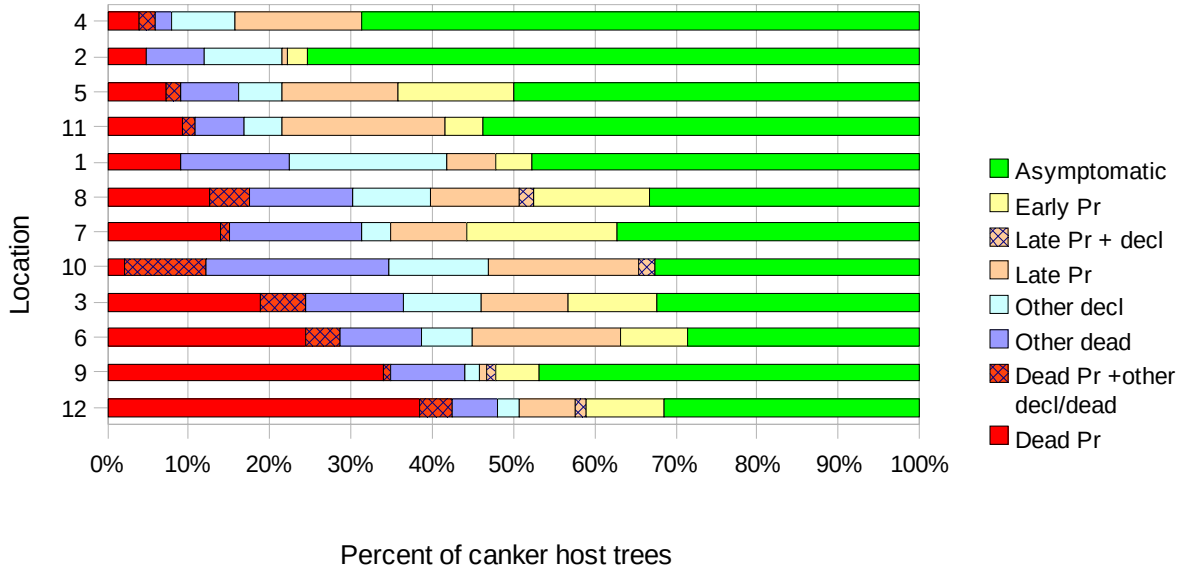

Figure 9. Overall disease status of SOD canker hosts (coast live oak, California black oak, and tanoak) in 2007 by study location. **Pr** = tree with *P. ramorum* canker symptoms; **early** = bleeding cankers only, **late** = bleeding cankers with beetles and/or *H. thouarsianum*; **other decl/dead** = tree declining or dead due to agents other than *P. ramorum*. Hatched bars (Late Pr + decline, Dead Pr + other dead) indicate trees with *P. ramorum* canker symptoms that were also declining due to agents other than the secondary agents typically associated with *P. ramorum* canker. Location numbers are shown in *table 1* and are sorted in order of increasing incidence of recent tree mortality from all causes. Trees with *P. ramorum* symptoms that were killed when nearby trees fell on them have been grouped into the 'other dead' category for the purposes of this graph.

SOD-related changes in stand structure

In many of the study plots, tree canopies overlap and some trees are partially to completely overtopped. Hence, tree mortality in these plots did not necessarily result in a decrease in plot canopy cover. Among the 22 tanoak plots, six (27%) showed decreases in canopy cover between 2001 and 2007, while two (9%) had increased canopy cover. All six plots with decreased canopy cover had *P. ramorum* induced tree mortality. However, 15 other plots with *P. ramorum*-related mortality (68% of all plots) did not show decreases in plot canopy cover due to the presence of other overstory species, such as coast redwood, in the plots.

Among the 128 coast live oak plots, 65 percent of the plots had no change in canopy cover rating between 2001 and 2007. Plot canopy cover decreased in 31 percent of the plots and increased somewhat in 3.9 percent of the plots. Among plots with *P. ramorum*-related coast live oak mortality, 45 percent were rated as having decreased plot canopy cover in 2007 compared with 2001. In comparison, 21% of plots without *P. ramorum*-related mortality showed a decrease in canopy over this same interval. However, some of these latter plots had *P. ramorum*-related decreases in canopy cover caused by limb death or failure in infected trees which were not yet killed.

For both tanoak and coast live oak, the average maximum DBH of trees (i.e., DBH of the largest stem) with *P. ramorum* canker symptoms by 2007 was significantly higher than that of asymptomatic trees (one way ANOVA $p<0.0001$ for both species). In 2007, the average

maximum DBH of coast live oaks (live or dead) with *P. ramorum* symptoms was 36 cm, compared to an average DBH of 26 cm for asymptomatic trees. For tanoak, trees with *P. ramorum* symptoms had an average maximum DBH of 14.4 cm, compared with 8.4 cm in uninfected trees. This effect, which has been noted previously, suggests that larger diameter trees are more susceptible to infection than are smaller diameter trees. For coast live oak, bark thickness is correlated with DBH, and bark thickness was a somewhat better predictor of disease risk than DBH (Swiecki and Bernhardt 2004, 2005).

Given that larger-diameter trees are more likely to be infected by *P. ramorum*, we would expect that the average DBH of the surviving trees in the stand will decline over time. However, over the interval from 2000 to 2007, the mean diameter of the largest stem of live trees in study plots did not change significantly for either coast live oak or tanoak. Two factors contribute to the lack of change over this period. First, the diameter distributions of symptomatic and asymptomatic trees overlap quite extensively: trees killed by *P. ramorum* are not exclusively the largest in the stands. Second, many trees with *P. ramorum* symptoms have survived for extended periods and had not died by 2007. Although mortality due to *P. ramorum* has reduced the mean stem diameter in individual plots and portions of the study stands, this trend had not become generalized across the study areas by 2007.

We counted and assessed the condition of all trees other than SOD canker host trees in the plots in 2001 and again in 2007. California bay was the most common plot tree after coast live oak, with a total of 450 live trees in all plots as of 2007. California bay tree populations remained relatively constant over the study period, decreasing by only 1% since 2001. Among the non-SOD canker host species, Douglas-fir was the only species that showed a population increase between 2001 and 2007. Douglas-fir was the third most common tree other than coast live oak, with 243 live trees in the plots in 2007. The Douglas-fir population increased by 31 percent between 2001 and 2007, due to sapling recruitment into the >3 cm DBH size class. Only 4 Douglas-fir trees died over this interval.

Madrone was the second most common non-SOD canker host tree species in the plots, with 315 live trees in 2007. This species also had the highest level of mortality among the non-SOD canker host species. Between 2001 and 2007, 50 madrone trees died, 15% of the live madrone population present in 2001. Because some saplings were recruited over the same period, the number of live madrones in plots decreased by only 7% overall between 2001 and 2007. Locations 5, 6, and 7 showed the highest overall incidence of madrone mortality (26, 17 and 31%, respectively) and the greatest change in mortality between 2001 and 2007 (percent madrone mortality in 2001 for these locations were 14, 8, and 14%, respectively). These levels of mortality are similar to total mortality due to all causes of coast live oak at most of the study locations (*fig. 9*). We have confirmed the presence of *P. cinnamomi* associated with declining and dead madrone and bay at location 5 (Swiecki and Bernhardt 2006), but have not tested soil at locations 6 and 7 for the presence of *P. cinnamomi* or other *Phytophthora* species.

Other trees are present in much lower numbers than bay, madrone and Douglas-fir, and populations of these other species showed little or no change between 2001 and 2007.

Canopy dieback in coast live oak

We scored canopy dieback in the plot center trees in all plots starting in 2000. In 2002, we began evaluating dieback on all other coast live oak and tanoak trees in the plots as well. Although the observation interval is shorter for the full data set (plot trees plus center trees), the

sample size is much greater than the center tree data set, which was used in our previous analyses of canopy dieback (Swiecki and Bernhardt 2005).

In the full data set, trees that were either healthy or had only early symptoms of *P. ramorum* canker (bleeding cankers only) in September 2007 showed no overall change in their average levels of canopy dieback since September 2002 (*fig. 10*). Trees that had late symptoms of *P. ramorum* infection (cankers plus beetle boring and or wood decay fungi) or were in decline due to other factors in 2007 showed increasing levels of canopy dieback between 2002 and 2007 (*fig. 10*). Effects of year, 2007 disease status, and the interaction between year and 2007 disease status were all highly significant (P<0.0005) in a repeated measures analysis of variance for the canopy dieback ratings of trees that were alive in 2007.

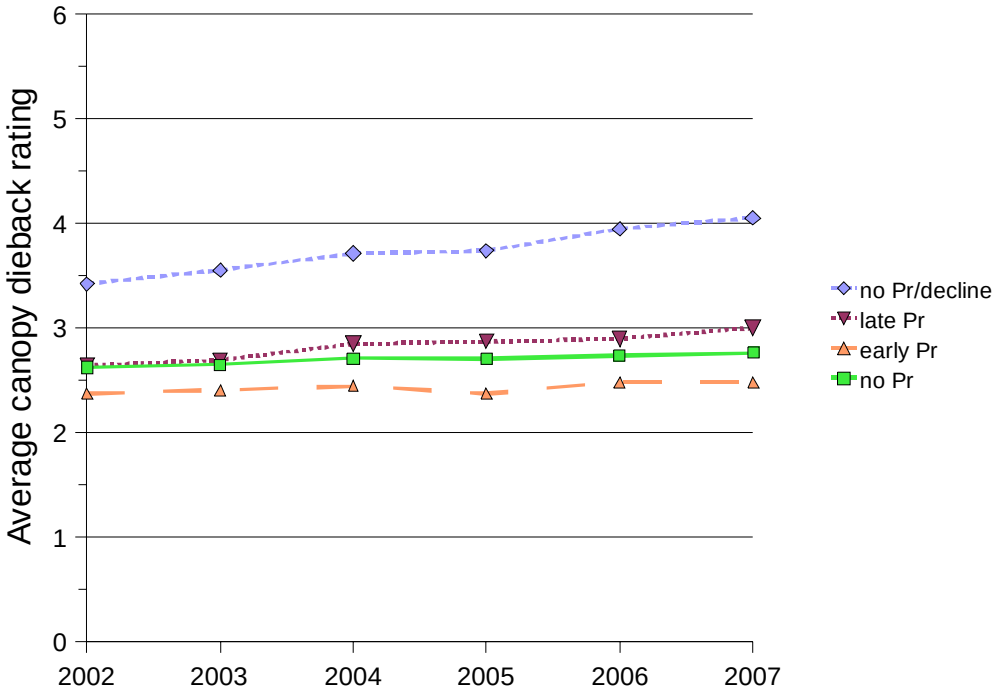

Figure 10. Canopy dieback ratings in September for coast live oak trees in each disease class as of Sept 2007; **decline/no Pr** =trees in severe decline due to agents other than *P. ramorum*, **late Pr**=live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **early Pr**=trees with *P. ramorum* cankers only, **no Pr**=non-declining trees with no evident symptoms of *P. ramorum* infection.

Tree failure

Throughout the study, we have recorded data on branch, stem, and root failures that exceeded the following size thresholds: branch failures ≥20 cm diameter, and bole, root crown, or root failures of main stems greater than ≥ 3 cm DBH. We recorded 164 failures in coast live oak, four in California black oak, and 33 in tanoak that exceeded these thresholds. Most (56%) of the initial coast live oak failures (i.e., the first failure above the threshold size) occurred in trees that were completely dead at the time of failure. Considering both live and dead trees, 76% of the initial recorded failures in coast live oak occurred in stems or branches that were dead. However, if we consider only living trees with *P. ramorum* canker, more than half of the failures

occurred in live branches and stems (*fig. 11*). To date, most of the failures observed in asymptomatic trees also occurred in live branches and stems.

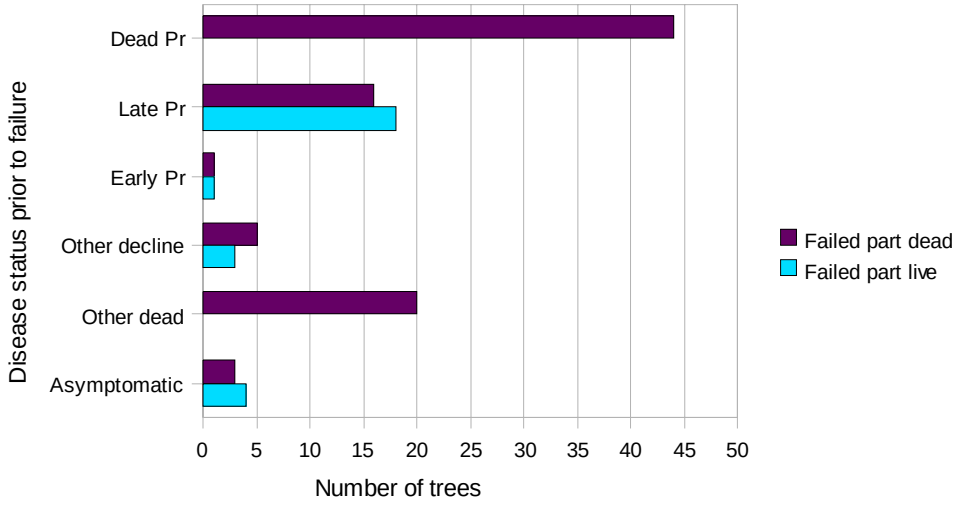

Figure 11. Tree disease status and condition of the failed part (live or dead) at the time of failure for initial failures above threshold size occurring between September 2000 and September 2007 in coast live oak study trees.

The vast majority of failures among coast live oaks have occurred in trees that were already infected with *P. ramorum* at the start of the study in September 2000 (Table 4). These trees were likely infected at some point between the estimated start of the epidemic around 1990 (Swiecki and Bernhardt 2006) and 2000. Four failures have occurred among the 75 coast live oak study trees that developed *P. ramorum* canker symptoms after 2000 and were still symptomatic in 2007. These four failures occurred in trees that had late *P. ramorum* canker symptoms or were dead prior to failure.

Table 4. Year failed (12 months previous to date shown) for trees infected by *P. ramorum* in various years.

		Year failed								
Year infected	Number of trees	Sept 2000	Sept 2001	Sept 2002	Sept 2003	Sept 2004	Sept 2005	Sept 2006	Sept 2007	Nonfailed
1990?-Sept 2000	154	0	2	26	21	8	8	7	7	76
Sept 2001	6		0	0	0	1	0	0	0	5
Sept 2002	4			0	0	0	0	0	0	4
Sept 2003	10				0	0	0	0	1	9
Sept 2004	5					0	0	1	0	4
Sept 2005	34						0	0	1	33
Sept 2006	8							0	0	8
Sept 2007	15								0	15
Noninfected[1]	419	0	0	8	4	7	4	5	7	384

[1] Includes trees which were dead at the start of the study due to causes other than P. ramorum, trees that were in decline due to wood decay fungi, and healthy trees.

Because the disease status of many study trees has changed between 2000 and 2007, various failure rates can be calculated from the data. In *figure 12*, cumulative failure rates over time for coast live oak study trees are plotted according to the disease symptom class observed in 2000. The disease status of the trees at the time of failure is also shown. These data exclude dead trees that had already failed by 2000.

All of the trees that were dead due to causes other than *P. ramorum* in 2000 had failures above the threshold size by March 2004 (*fig. 12*). For trees that were killed by *P. ramorum* prior to September 2000, it was not until September 2007 that the last tree had failed. Both of these groups of trees had a 50% failure rate by September 2002.

Among coast live oaks trees that were alive in 2000, trees with late *P. ramorum* canker symptoms (with *H. thouarsianum* sporulation and/or wood boring beetles) experienced the highest failure rates (*fig. 12*). Among failed trees with *P. ramorum* canker, almost all had died or progressed to the late disease stage prior to failure (*fig. 12*).

Trees that were asymptomatic in 2000 showed very low rates of failure over the study period, and most of those failures occurred in trees that had either developed *P. ramorum* canker or were declining due to other diseases (*fig. 12*).

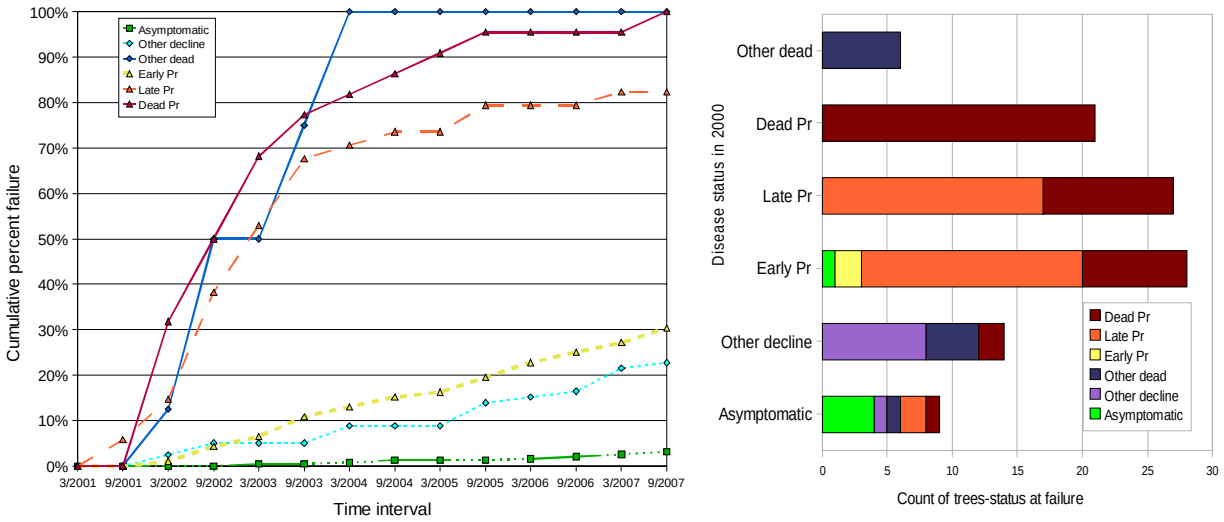

Figure 12. Cumulative failure rates (left) and tree disease status at failure (right) for coast live oaks by initial disease status in 2000. Only initial failures above the size threshold are shown. Failure dates were estimated to the nearest 6 month interval and failure percentages are plotted at the end date of each interval. Trees that had symptoms of both *P. ramorum* (**Pr**) and decline due to other agents (**other decline / dead**) are pooled with the respective *P. ramorum* symptom class. Trees that had failed prior to September 2000 are excluded.

Based on characteristics of the failed part (weathering and accumulation of detritus on broken surfaces, etc.), we estimated the date that each failure occurred to the nearest six-month interval. As shown in *figure 13*, the number of initial failures (first failure above threshold size for a given tree) among coast live oaks increased dramatically over time in the first two years of the study but has been decreasing since 2002 (*fig. 13*). For trees with branch or high bole failures or multi-stemmed trees, additional failures over the threshold size are possible after the initial failure. The number of such later failures occurring each year has increased substantially between 2002 and 2005 and has decreased somewhat thereafter (*fig. 13*).

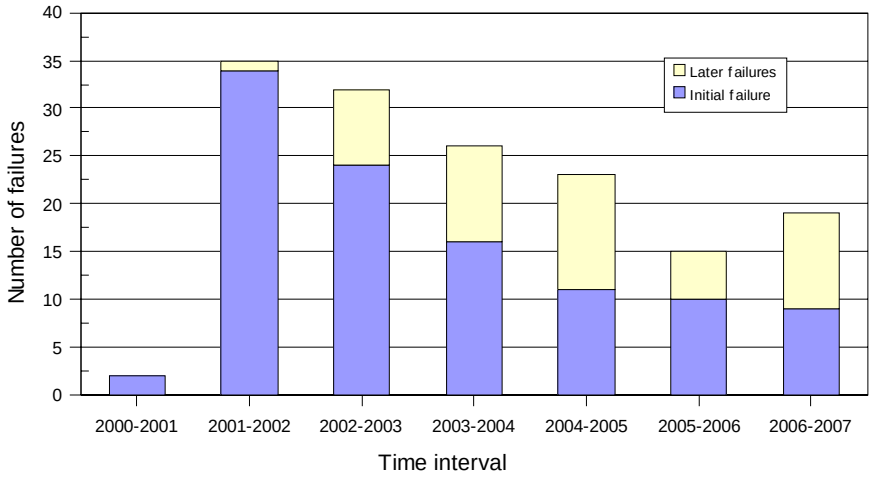

Figure 13. Number of initial (blue bars) and subsequent (yellow bars) failures above threshold size occurring in annual observation intervals between September 2000 and September 2007 among 629 coast live oaks that had not failed prior to September 2000. Annual observation intervals are from October of the first year through September of the following year.

Cumulative mortality and failure rates for *P. ramorum*-infected coast live oaks are compared in *figure 14*. Since plots were first observed in September 2000, tree failure data is not available for the first time interval in the graph. However, for the six locations included in our tree failure study (Swiecki and Bernhardt 2003b, Swiecki and others 2006), the failure rate in *P. ramorum*-affected trees prior to 2000 was 0.5%. Figure 14 shows that although mortality had increased in a relatively linear fashion over the interval from September 2000 to September 2007, failure rates showed an initial lag followed by a steep increase through 2003. From 2003 through 2007, the percentage of *P. ramorum*-infected coast live oaks with initial failures has approximated the mortality rate due to *P. ramorum.*

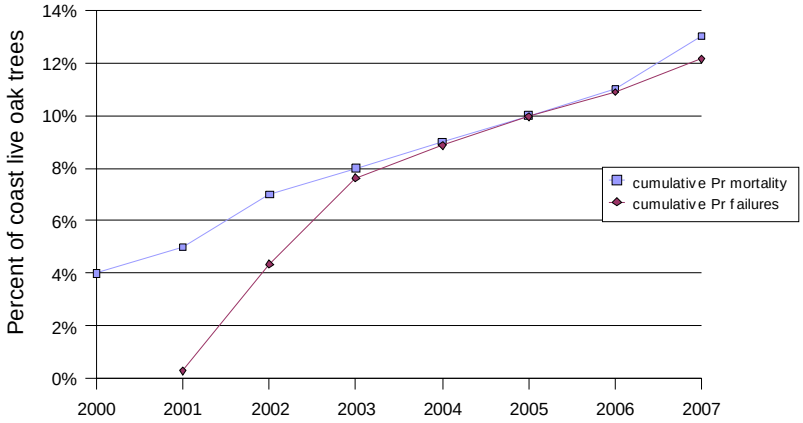

Figure 14. Cumulative initial failures in *P. ramorum*-infected coast live oak trees compared to the cumulative mortality rate due to *P. ramorum*. Mortality and failure percentages shown are cumulative to September of the year shown on the x axis.

Bole failures were the most common type of failure in coast live oaks with and without *P. ramorum* canker symptoms (*fig. 15*, bottom), but most of the bole failures observed were in trees with *P. ramorum* canker symptoms (*fig. 15*, top). Bole failures were common in trees that were

declining due to infections by canker rot and other wood decay fungi. Branch failures were somewhat more common than root crown failures in trees with *P. ramorum* canker, whereas the frequencies of these two failure types were similar for trees without *P. ramorum* canker. Only one root failure has occurred among the coast live oaks in the study, and it affected a tree that did not have *P. ramorum* symptoms.

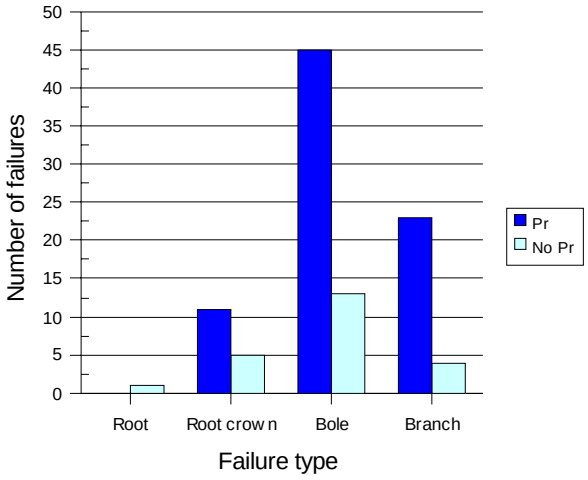

Figure 15. Frequency of initial failures above threshold size by failed part for failures occurring between September 2000 and September 2007 among coast live oaks with (**Pr**) and without (**No Pr**) symptoms of *P. ramorum* canker at the time of failure.

Our data set for failures in tanoak is much smaller than the coast live oak data set. Among the 195 tanoaks in the study, 32 failures have been recorded since 2000, including two root failures, 22 bole failures, and eight root crown failures. Twenty-eight of the failures occurred in tanoaks killed by *P. ramorum* canker and one (a root failure) occurred in a tree with early *P. ramorum* canker symptoms. Five of the failures occurred among the 44 tanoaks that first developed *P. ramorum* canker after 2000. The incidence of failure among tanoaks with *P. ramorum* canker in 2007 (33%) was significantly greater than that observed in asymptomatic trees (3%).

Regeneration

To determine the potential for killed SOD canker hosts to be replaced by seedlings of the same species, we have tracked the number of seedlings (plants with DBH<3 cm) of coast live oak, California black oak, and tanoak in the study plots during each September evaluation.

Coast live oak

Almost all of the 128 plots with coast live oak overstory had coast live oak seedlings at some point between 2000 and 2007. Only two of these plots had no coast live oak seedlings in any of the seven years of the study. Fifty one percent of the plots had no seedlings in at least one year. In 2006, 23% of coast live oak plots had no live seedlings in the understory. In 2007, 30% of coast live oak plots had no live seedlings in the understory. This decrease in seedling populations may have been due in part to reduced rainfall in 2007.

Seedling numbers fluctuated widely, both within plots at the same location, and in the same plots from year to year (*fig. 16*). In half of the plots, no more than ten coast live oak seedlings were observed in any year.

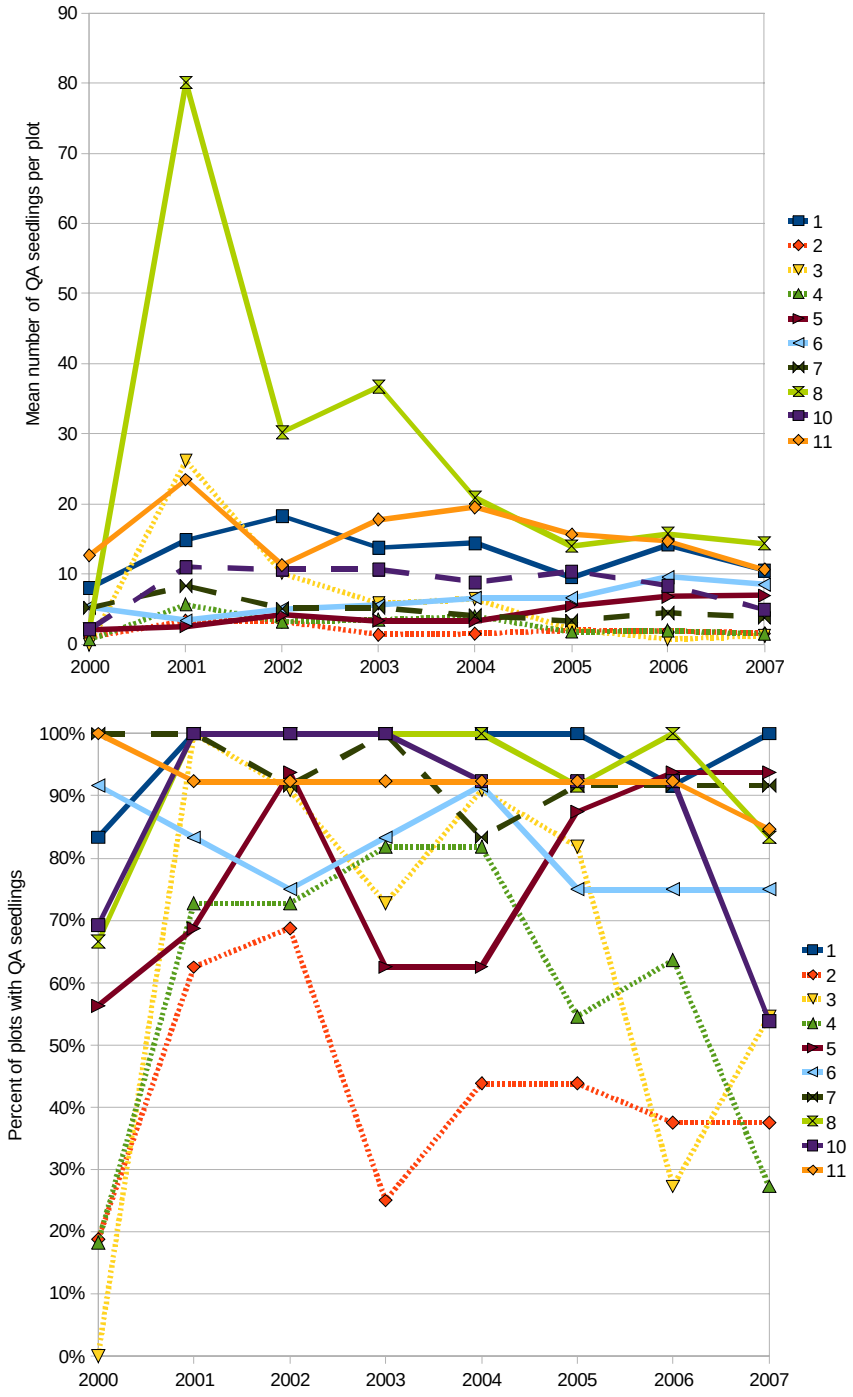

Figure 16. Mean numbers of coast live oak seedlings per plot (top) and percent of plots with coast live oak seedlings (bottom) at each of 10 locations with coast live oak overstory from 2000 through 2007. Location numbers shown in the legend correspond to locations in *table 1*.

Seven locations showed peak seedling populations in 2001 (*fig. 16*, top), the only year in which average seedling counts per plot differed from other years (*table 5*). Location 8 was unique in having exceptionally high seedling counts (up to about 200) in some plots in 2001. This location had the highest observed average seedling counts per plot (80) in 2001 (*fig. 16*). In addition, the percentage of plots with coast live oak seedlings also peaked in 2001 at many but not all locations (*fig. 16*, bottom).

Table 5. Mean number of coast live oak seedlings or tanoak seedlings per plot by year. Means are calculated from plots with overstory of coast live oak (128 plots) or 39 plots with tanoak seedlings. Standard deviations are shown in parentheses.

Year	2000	2001	2002	2003	2004	2005	2006	2007
Coast live oak	3.9 (5.8)	17 (32)*	9.8 (15.8)	10 (20.5)	8.7 (11.3)	7 (9.1)	7.6 (9.75)	6.3 (8.6)
Tanoak	11.8 (13.4)	11.5 (9.5)	13.8 (12.9)	13.1 (13.3)	12.4 (10.3)	13.8 (12.3)	14.2 (11.7)	15.4 (13)

* significantly different from all other means within species according to Tukey-Kramer HSD.

Tanoak

Tanoak seedlings were present in all 25 plots with tanoak overstory between 2000 and 2007. At some coast live oak locations, tanoak seedlings were also found in plots lacking overstory tanoak. Tanoak seedlings most commonly occurred as shrubby seedling-sprouts with multiple stems, and were commonly at least 50 cm tall, although smaller seedlings were also present in some areas. Consequently, tanoak seedling counts in plots were more stable from year to year than coast live oak seedlings counts (*fig. 16, 17, table 5*). At least seven tanoak seedlings in the study plots have attained a DBH of 3 cm or more over the study period and are now classified as trees.

Unlike coast live oak twigs, tanoak twigs are susceptible to *P. ramorum.* Understory tanoak seedlings commonly showed tip dieback and/or mortality of individual stems typical of that caused by *P. ramorum*. However, over the period of the study, this damage has not resulted in a reduction in tanoak seedling populations (*fig. 17*).

California black oak

No California black oak seedlings were observed in 2007. Only five plots had California black oak seedlings present (1 to 3 seedlings) in 2001. By 2005, only two plots had California black oak seedlings (one seedling in each plot). In 2006, California black oak seedlings were found in three plots, one of which had five seedlings. The seedlings observed in both 2005 and 2006 occurred in plots without overstory California black oak trees.

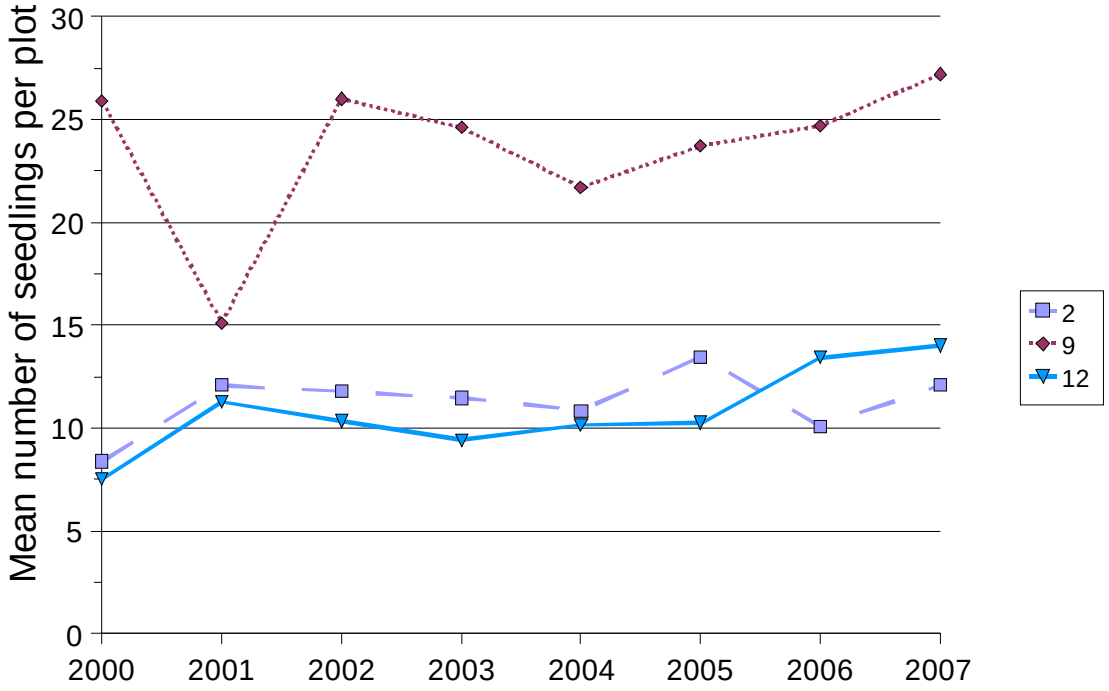

Figure 17. Mean number of tanoak seedlings and seedling sprouts per plot at each of three locations with tanoak overstory. Location numbers shown in the legend correspond to locations in *table 1*.

Stem water potentials of coast live oaks

We collected data on changes in stem water potentials (SWP) during the first six years of the study and have previously reported on this data (Swiecki and Bernhardt 2001, 2002ab, 2003a, 2004, 2005, 2006, 2007c). Beginning in 2005, we began to measure SWP on a subset of trees from each location, instead of on all plot center and additional SWP trees from each location. Retrospective analysis of SWP readings from 2000-2004 showed a high correlation between the full set and reduced subset of trees; according to repeated measures MANOVA, the mean SWP did not differ between these two sets over time.

The average SWPs for the trees monitored in all years at all locations are shown in *figure 18*. The pattern of annual changes in SWP is very similar among the locations, and correlates well with the annual changes in rainfall for all years except 2006. Although 2006 was the wettest year of the series, SWP readings were lower in 2006 than in 2005. July of 2006 featured a record-breaking heat wave which lasted from July 16 to July 26 (Kozlowski and Edwards 2007). One of the characteristics of the heat wave, in addition to the high daily temperatures, was unusually high night temperatures. The high temperatures did not result in unusually high reference evapotranspiration (ETo) readings, based on data from the nearest CIMIS weather station in Santa Rosa (*fig. 18*), but the high temperatures may have caused stress that influenced plant water relations for the remainder of the season

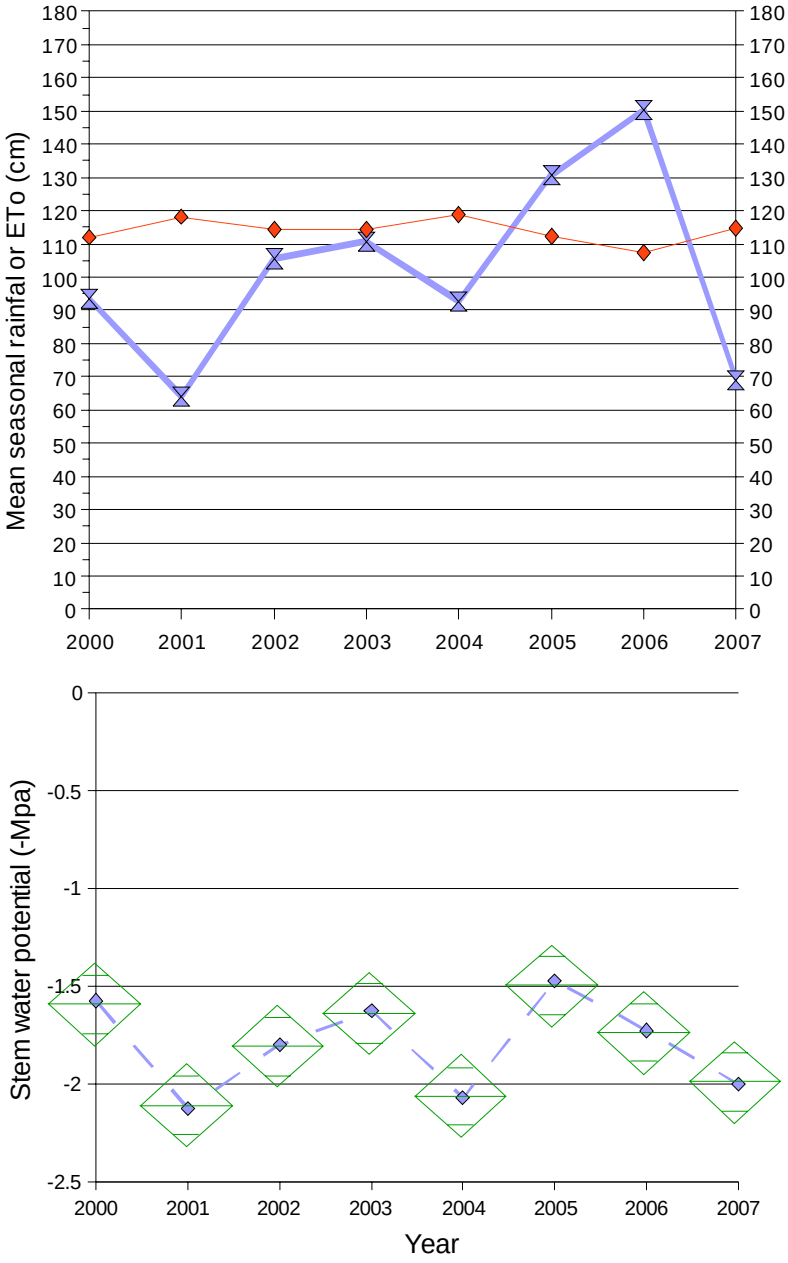

Figure 18. Average stem water potential for coast live oaks across all years and locations (bottom graph) compared with average seasonal rainfall and reference evapotranspiration (ETo) (top graph). ETo and rainfall are for September of the previous year through August of the listed year. ETo is from CIMIS station 83 in Santa Rosa. Rainfall total is the mean for all coast live oak study locations calculated from the nearest weather station data for each location. The vertical extent of each diamond represents the 95% confidence interval for each mean from a one-way analysis of variance of SWP across all years.

P. ramorum foliar symptoms on bay

We previously reported on repeated observations of bay foliar symptoms for the period from September 2005 to September 2006 (Swiecki and Bernhardt 2007a, 2008). These analyses showed no significant correlations between timed counts of bay foliar symptoms seen in fall 2005 with counts made in late spring 2006 or fall 2006. However, spring 2006 bay foliar symptom counts were positively correlated with counts from the same trees measured in fall 2006. To examine this relationship further, we collected additional foliar count symptom data in June and September 2007 for most of the bay zones and made additional counts in April 2008 of bay zones at location 5 only.

Counts for the bay zones at location 5 are shown in *figure 19*. All counts showed a strong peak in May 2006, following the wet spring of that year. Some bay zones also showed obvious peaks in symptomatic leaf counts in spring 2007 and 2008, but the peak foliar symptom counts in these two relatively dry springs were significantly lower than levels seen in 2006 (Tukey-Kramer HSD multiple comparison $p \leq 0.05$). Furthermore, the mean counts for the remaining dates did not differ significantly from each other. As seen in *figure 19*, numbers of infected leaves in two of the bay zones remained relatively high into early 2008. Both of these zones had timed counts that had reached the highest observed values in spring 2006. However, most other bay zones that reached similarly high levels of foliar disease in spring 2006 had low levels of infected leaves by fall of 2006 and foliar infection rates remained relatively low through spring 2008.

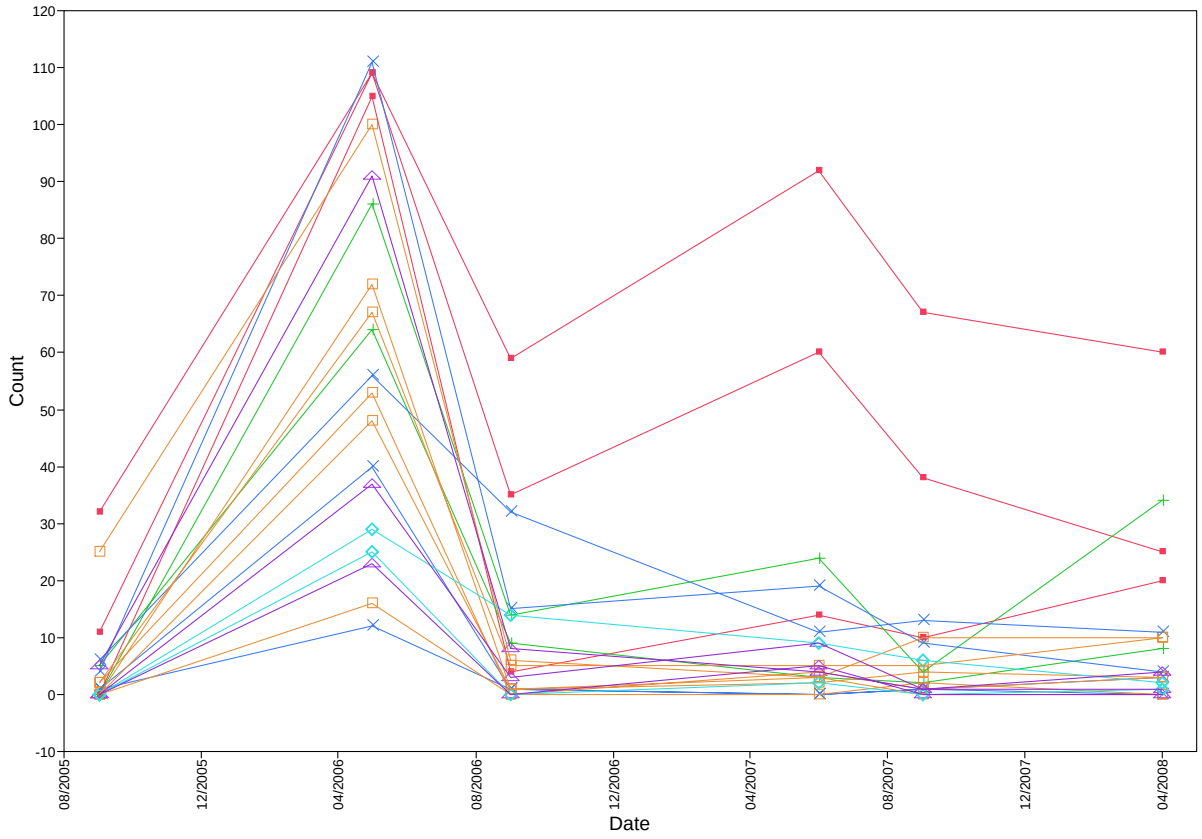

Figure 19. Counts of symptomatic bay leaves for bay zones at location 5 from September 2005 through April 2008.

Comparison of mean counts for all locations through fall 2007 shows the same overall trend. Peak foliar symptom counts were observed in spring 2006. Although notable outliers are visible for the other count periods, all other spring and fall count averages did not differ significantly. In addition, average counts did not differ significantly between bay zones located around trees with *P. ramorum* canker symptoms and those around asymptomatic trees.

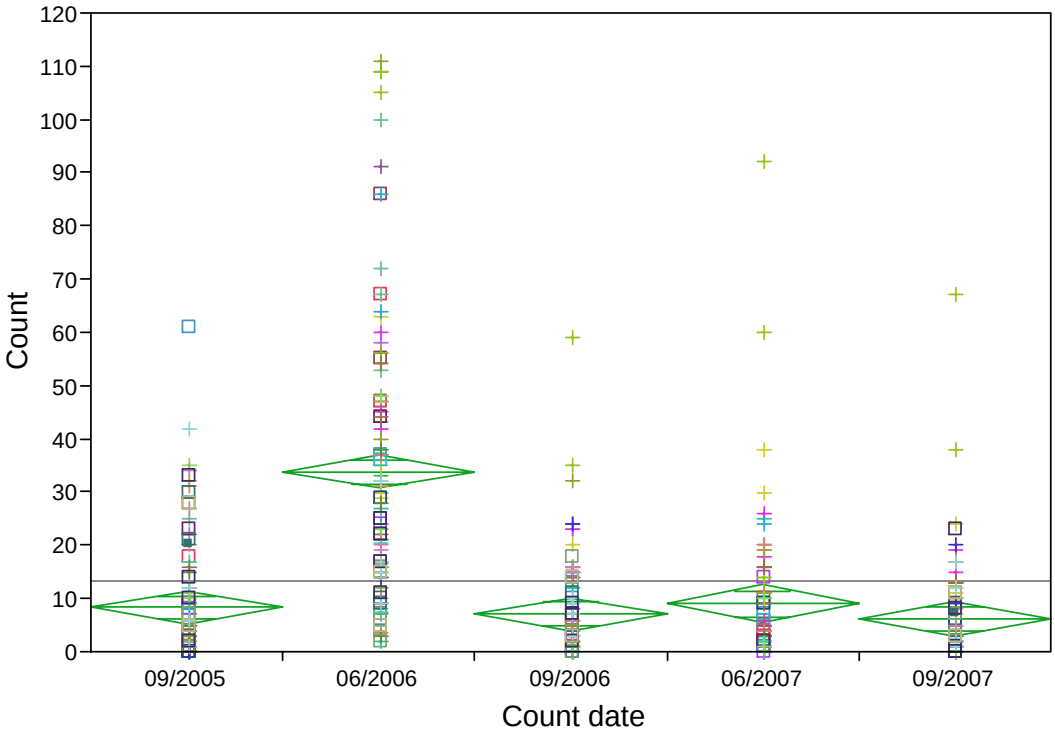

Figure 20. Means of timed counts of *P. ramorum* foliar symptoms on California bay in spring and fall from September 2005 through September 2007. Center line in each diamond is mean for that date; the vertical extent of each diamond represents the 95% confidence interval for each mean from a one-way analysis of variance. Cross (+) symbols indicate bay zones centered around trees with *P. ramorum* canker; boxes (□) indicate bay zones centered around asymptomatic trees. Each individual bay zone is denoted by a different symbol/color combination.

We previously reported (Swiecki and Bernhardt 2007a, 2008) that counts made in September 2005 were not correlated with counts from the same zones made in mid-2006 (May-July) or September 2006, but that May-July and September 2006 counts were correlated. As shown in *table 6*, counts made in June and September 2007 were even more highly correlated than the two sets of counts made in 2006. Furthermore, both sets of 2007 foliar symptom counts were also significantly correlated with both sets of counts made in 2006.

Table 6. Pairwise nonparametric correlations between arcsine transformed bay foliar symptoms counts made from the same zones at different times. One high outlier was omitted from the analysis (n=68 zones).

Variable 1	Variable 2	Spearman ρ	Prob>|ρ|
2005 Sep-Oct	2006 May-Jul	-0.0649	0.5233
2005 Sep-Oct	2006 Sep-Oct	0.1191	0.2401
2005 Sep-Oct	2007 Jun	-0.1263	0.2835
2005 Sep-Oct	2007 Sep	0.1609	0.1319
2006 May-Jul	**2006 Sep-Oct**	**0.3620**	**0.0002 ***
2006 May-Jul	**2007 Jun**	**0.5631**	**<.0001 ***
2006 May-Jul	**2007 Sep**	**0.3035**	**0.0040 ***
2006 Sep-Oct	**2007 Jun**	**0.7340**	**<.0001 ***
2006 Sep-Oct	**2007 Sep**	**0.7222**	**<.0001 ***
2007 Jun	**2007 Sep**	**0.7072**	**<.0001**

DISCUSSION

Disease incidence and progress

Differences between tanoak and coast live oak

Due to rainy periods that extended into late spring, conditions for inoculum production were highly favorable in the spring of both 2005 and 2006 (D. Rizzo, personal comm.). Disease incidence in coast live oak showed strong increases in 2005 and 2006 compared with 2001 through 2004 (*fig. 2)*. Under the assumption that the spike in new infections seen in September 2005 was associated with increased inoculum production in the spring of that year, we can conclude that the latent period between infection and symptom expression in many coast live oaks is less than one year. However, given the lack of *P. ramorum* inoculum detected in spring 2007 (Dave Rizzo, personal comm.., Swiecki and Bernhardt, 2007b.), the continued peak of new infections seen in September 2007 represents infections with longer latent periods. Given that disease levels in coast live oak were essentially flat between 2000 and 2004, it appears that disease outbreaks in coast live oak woodlands may require heavier than normal rainfall to generate inoculum levels sufficient to initiate disease in a large number of trees.

In contrast, the rates of new symptom development in tanoaks in 2005 and 2006 were similar to that seen in previous years and peaked in 2007, a year with relatively low rainfall (*fig. 2, 3*). Observed new infection frequencies in tanoak were lowest in 2002 and 2005 (*fig. 2*), years that followed the low rainfall years of 2001 and 2004 (*fig. 18*). The year lag between low rainfall and low new infection rate suggests that many tanoaks in these stands had a latent period of a year or more. A comparison of figures 2 and 3 suggests that new infections in a given year are correlated with rainfall in the previous one to two years. New infections in tanoak stands appeared to occur in virtually every year between 2000 and 2006, suggesting that infection in these tanoak stands is not overly dependent on high rainfall events. Artificial inoculations (Rizzo and others 2002) have shown that tanoak is more susceptible than coast live oak to *P. ramorum* canker development. Our data indicate that these differences in susceptibility may contribute to different patterns of infection over time.

Observed differences between the frequencies of new infections in coast live oak and tanoak may also be related in part to the difference in disease incidence that existed in 2000 between the two species. *P. ramorum* canker incidence in the coast live oak plots in 2006 was about the same as the incidence seen in tanoak plots in 2000, so the epidemic was clearly at different stages in these two tree populations. However, the elevated levels of newly symptomatic tanoaks seen in 2007 suggest that the supply of susceptible tanoak trees in the population is not limiting the epidemic in these stands at this point.

Our data clearly show that coast live oak typically survives much longer than tanoak after symptoms of infection become apparent (*fig. 5*, *6*). Some of this difference may be due to the fact that symptoms in tanoak are often cryptic; in many cases tree mortality was the first obvious symptom of disease in tanoak (*fig. 6*). In such cases, it is unclear when trees were initially infected, though the foregoing data suggests that many of these trees may have been infected at least a year before they died.

The net effect of these differences is that the tanoak and coast live oak populations represented in this study showed substantially different patterns with respect to the timing of symptom onset and disease progress over time. As a result, disease progress and SOD mortality in coast live oak lagged behind that seen in tanoak, a pattern that has been observed since the start of the epidemic in California (Garbelotto and others 2001).

Differences between study locations

We have observed persistent differences in infection rates between study locations. Several of the locations with widely different levels of disease (e.g., locations 2, 3) are so close to each other that no substantial differences in precipitation are likely to exist between the sites. As we have previously shown, the presence and abundance of California bay within plots and around individual oaks accounts for a large portion of the differences in disease risk within and between locations (Swiecki and Bernhardt 2005, 2007a, 2008). The persistence of the variation in *P. ramorum* canker incidence between these locations provides hope that management strategies involving reductions in bay cover within at-risk stands may be able to minimize disease impacts over extended time periods.

Failures

Most recent coast live oak failures in oak woodlands affected by *P. ramorum* have occurred in trees infected by *P. ramorum* (*fig. 12*; Swiecki and Bernhardt 2003b, 2004, 2005, Swiecki and others 2006). Almost all of the failures occurring in *P. ramorum*-infected oaks have involved trees that were already dead or were extensively colonized by secondary organisms including *H. thouarsianum,* other decay fungi, and ambrosia beetles (i.e., late stage disease status). These data are consistent with our previous observations (Swiecki and Bernhardt 2003b, 2004, 2005, Swiecki and others 2006).

Trees with only early symptoms of *P. ramorum* canker are no more likely to fail than asymptomatic trees with similar levels of decay or other defects. *P. ramorum* cankers in coast live oak can sometimes take several to many years to progress to the late stage and may sometimes go into remission. Hence, the appearance of early stage *P. ramorum* cankers in trees that lack other significant failure-related defects does not require an immediate response to mitigate failure potential. It is noteworthy that only four coast live oaks that have first developed symptoms after 2000 had failed by 2007, and all of these had progressed to the late stage or had died prior to failure.

For sites where SOD-affected trees have the potential to pose a hazard, dead trees and trees with late disease symptoms and extensive decay should be the primary focus for hazard reduction. Trees with only early *P. ramorum* canker symptoms (bleeding cankers only) should be monitored to determine if secondary wood decay organisms and or wood-boring beetle activity become evident. Once these secondary organisms are evident, their extent and the presence of other defects should be assessed to determine the likelihood of failure (Swiecki and Bernhardt 2003b).

P. ramorum cankers were also associated with most failures occurring in the SOD-affected tanoak stands. Failures occurred almost exclusively in dead trees, most commonly trees that had been dead for several years. Hence, as with coast live oak, tree hazard management in SOD-affected tanoak stands needs to focus primarily on dead trees, especially those that have been dead for multiple years.

Stand structure changes and regeneration

At both tanoak locations and at six of the ten coast live oak locations, mortality associated with *P. ramorum* canker was more common than mortality associated with other causes (*fig. 9*). However, because mortality was unevenly distributed between plots and locations, changes in stand structure associated with SOD tended to be patchy rather than generalized across the landscape. Tree mortality due to SOD did not consistently reduce canopy cover, so impacts of the disease on stand structure can vary even between sites with equal amounts of mortality. During the study period, madrone populations have declined, particularly at three locations, and populations of Douglas-fir have increased. Because the mix of species present and the population dynamics of these species differ between locations, there has been no single overall effect of SOD on stand composition across the study locations.

Both tanoak and coast live oak produce persistent seedlings that can survive in the understory for multiple years. These persistent seedlings contribute to the relative stability in seedling counts seen at tanoak locations (*fig. 17*) and most coast live oak locations (*fig. 16*). These persistent seedlings provide an opportunity for natural regeneration to occur in plots with tanoak and coast live oak mortality. A few tanoak seedlings in plots have grown to the point that they are now considered trees, but no coast live oak seedlings have been recruited to the tree size class between 2000 and 2007. Because seedlings can be suppressed or killed by various agents, including herbivores, fire, and competition from other overstory or understory species, active management may be needed to ensure that coast live oak or tanoak can be recruited in plots that have been affected by mortality.

Stem water potentials

Over the course of the study, year to year variation in stem water potential (SWP) readings for monitored coast live oaks has generally been correlated with rainfall levels from the preceding wet season (*fig. 18*, Swiecki and Bernhardt 2006). The 2004-05 and 2005-06 rainy seasons provided the first example of two successive high rainfall years over the course of the study. Although we had anticipated that SWP readings would be relatively high (indicating low water stress) following a second wet year, average SWP readings in 2006 were actually lower (showing greater water stress) in 2006 than in 2005. SWP readings were again lower in 2007, but not as low as we might have expected based on the especially low rainfall in that year.

The record-breaking heat wave in July 2006, which lasted from July 16 to July 26 (Kozlowski and Edwards 2007), may have contributed to the somewhat aberrant readings in that

year. However, the fact that 2007 SWP readings were similar to those seen in 2004 (a wetter year) suggests that carryover of deep soil moisture from the 2006 rainy season may have influenced the 2007 SWP readings. If so, this suggests that effects other than simply soil moisture depletion contributed to the unusual 2006 readings. It is possible that heat-related stress triggered changes in tree physiology and development (e.g., early onset of summer dormancy) to the degree that exploitation of available soil moisture was reduced. Another possibility is that prolonged wet soil conditions in early 2006, rather that the midsummer heat wave, adversely affected root health, leading to a reduced water use. Root health could have been directly impacted by prolonged periods of anoxia associated with soil saturation, and/or indirectly affected by other opportunistic or weak root pathogens that were favored by wet soil conditions. Dry spring conditions in 2007 may have allowed trees to produce new healthy roots, which were able to tap into some of the deeper soil moisture remaining after the 2006 season.

Bay foliar symptoms

Based on data from September 2005 through September 2006, we previously noted that correlations in bay foliar symptom counts were only seen within the same year (Swiecki and Bernhardt 2007a). With additional observations through 2007, we have now observed correlations across years, as have been noted by Rank and others (2008) for plots located in Sonoma County. Spring rainfall in 2007 was low, as were levels of *P. ramorum* inoculum (D.M. Rizzo, personal comm., Swiecki and Bernhardt, 2007b.), so it is likely that most of the infected bay leaves counted in 2007 were actually infected in 2006. In contrast, the interval from September 2005 to June 2006 included spring precipitation that was favorable for *P. ramorum* infection to occur (*fig.19, 20*). Levels of infection in September 2005 were not correlated with any of the subsequent counts. This suggests that fall foliar infection levels may be poor predictors of infection levels in the following spring when disease levels are increasing due to favorable rainfall conditions. In contrast, both spring and fall foliar infection levels in 2006 were correlated with foliar infection levels in 2007, which represented a period when infected leaves were gradually being lost after the peak infection levels were reached in spring 2006. Hence, the strength of year to year correlations between bay foliar infection levels may depend on whether disease levels are increasing or decreasing.

The bay foliar symptom count data also provide evidence that some zones of bay foliage tend to reach especially high levels of infection and may maintain elevated infection levels for long periods of time (*fig.19*). Local microclimate, canopy architecture, genetically-controlled susceptibility, and/or other factors may be interacting in these zones to produce conditions favorable to foliar disease development. Such zones may be important in inoculum carryover over unfavorable seasons and may serve as local disease foci when conditions favorable for disease spread return. This suggests the possible management strategy of targeting such areas for bay removal or other reductions of bay canopy density. It may be easier to identify such areas in years such as 2007 and 2008, which were generally unfavorable for *P. ramorum* inoculum production.

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