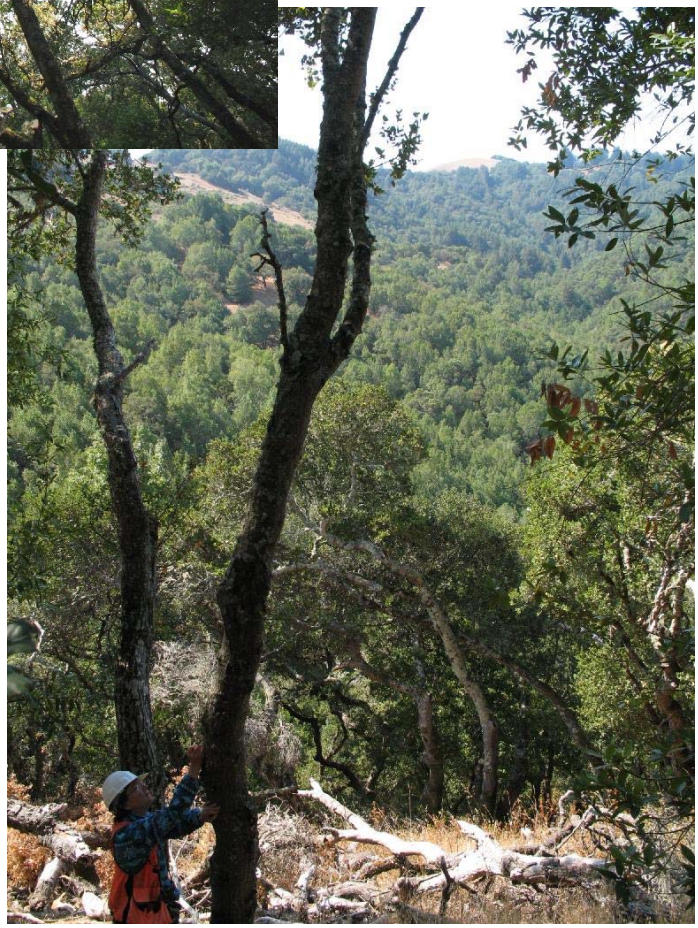
Phytophthora ramorum canker (sudden oak death) in coast live oak and tanoak, 2000-2006: factors affecting disease risk, disease progression, and failure potential

2006-2007 Contract Year Annual Report



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ACKNOWLEDGEMENTS

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SUMMARY

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

This report discusses findings after seven 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 2006, 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 2006. The overall percentage of coast live oaks with *P. ramorum* canker symptoms increased from 23% in 2000 to 30% in 2006. Most of the increase in infection occurred in 2005 and 2006 and was associated with heavy rainfall in the springs of 2005 and 2006. Between 2000 and 2006, tanoaks showed a significantly larger increase in disease incidence, from 31% to 46%. The percentage of newly symptomatic trees was greater for tanoak than coast live oak in all years except 2005. Disease incidence in 2006 varied widely between the 12 locations included in the study, and ranged from 8% to 56%. In 2006, mortality due to *P. ramorum* ranged from 4% to 24% at locations with coast live oak and from 27% to 39% at the two tanoak locations. Relatively stable differences in disease incidence between nearby locations appeared to be mainly due to factors other than weather and climate variables.

Overall, symptomatic tanoaks tended to survive for a shorter time after the onset of symptoms than did coast live oaks. Among live trees that had *P. ramorum* canker symptoms in 2000, 63% of tanoaks but only 32% of coast live oaks had died by 2006. For tanoaks that first became symptomatic between 2001 and 2006, 33% died within 2 years from the appearance of symptoms and 45% had died by 2006. In contrast, only 7% of coast live oaks that developed symptoms after 2000 had died by 2006; however, many of these new infections developed in 2005 and 2006. Disease progress and SOD mortality in coast live oak lagged behind that seen in tanoak because fewer new infections developed in coast live oak and infected coast live oaks tended to survive longer than infected tanoaks.

Because many plots had overlapping tree canopies, and contained species which are not killed by *P. ramorum*, tree mortality did not always result in a decrease in plot canopy cover. Among plots with *P. ramorum*-related coast live oak mortality, 41% showed reductions in plot canopy cover between 2001 and 2006. In contrast, 14% of plots without *P. ramorum*-related mortality had reductions in canopy cover over this period. Canopy cover in most tanoak plots had not changed substantially between 2000 and 2006, mainly because redwood overstory was present in many of the tanoak plots.

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 population numbers increased by 17%, and madrone population decreased 7% overall. Two locations had madrone mortality levels greater than 20%. *P. cinnamomi* has been associated with declining madrone and bay at one of these locations.

Nearly three quarters of the initial substantial failures in SOD-affected coast live oaks occurred in dead trees or dead stems of live trees. However, in live coast live oak trees with *P. ramorum* canker, more than half of the observed initial failures occurred in live stems or branches. Between 2000 and 2006, the failure rate of trees that had SOD symptoms in 2006 (34%) was about 10 times that of trees that remained free of *P. ramorum* canker symptoms (3.5%). The failure rate among tanoaks that developed *P. ramorum* canker symptoms by 2006 was 26%, compared with a 3% failure rate in tanoaks that were asymptomatic in 2006.

Data for the period from September 2000 to September 2006 suggest *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 2006 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 2006. In 2006, all tanoak plots that had *P. ramorum*-related mortality had tanoak seedlings present. Among plots with *P. ramorum*-related coast live oak mortality, only 6% lacked coast live oak seedlings.

In 2006, stem water potentials (SWP) of coast live oak trees deviated from the pattern seen in the previous 6 years. Between 2000 and 2005, SWP readings were been closely tied to the total rainfall for the preceding rainfall year (September to August). However, even though total rainfall for 2005-2006 rainfall year was the highest recorded since the start of the study, average SWP levels measured at all locations in September 2006 were lower than seen in September 2005, indicating higher levels of water stress in 2006 than in 2005. We hypothesize that the extreme heat event that affected the area in July 2006 was responsible for the greater levels of water stress seen in late 2006.

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 2005b), 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.

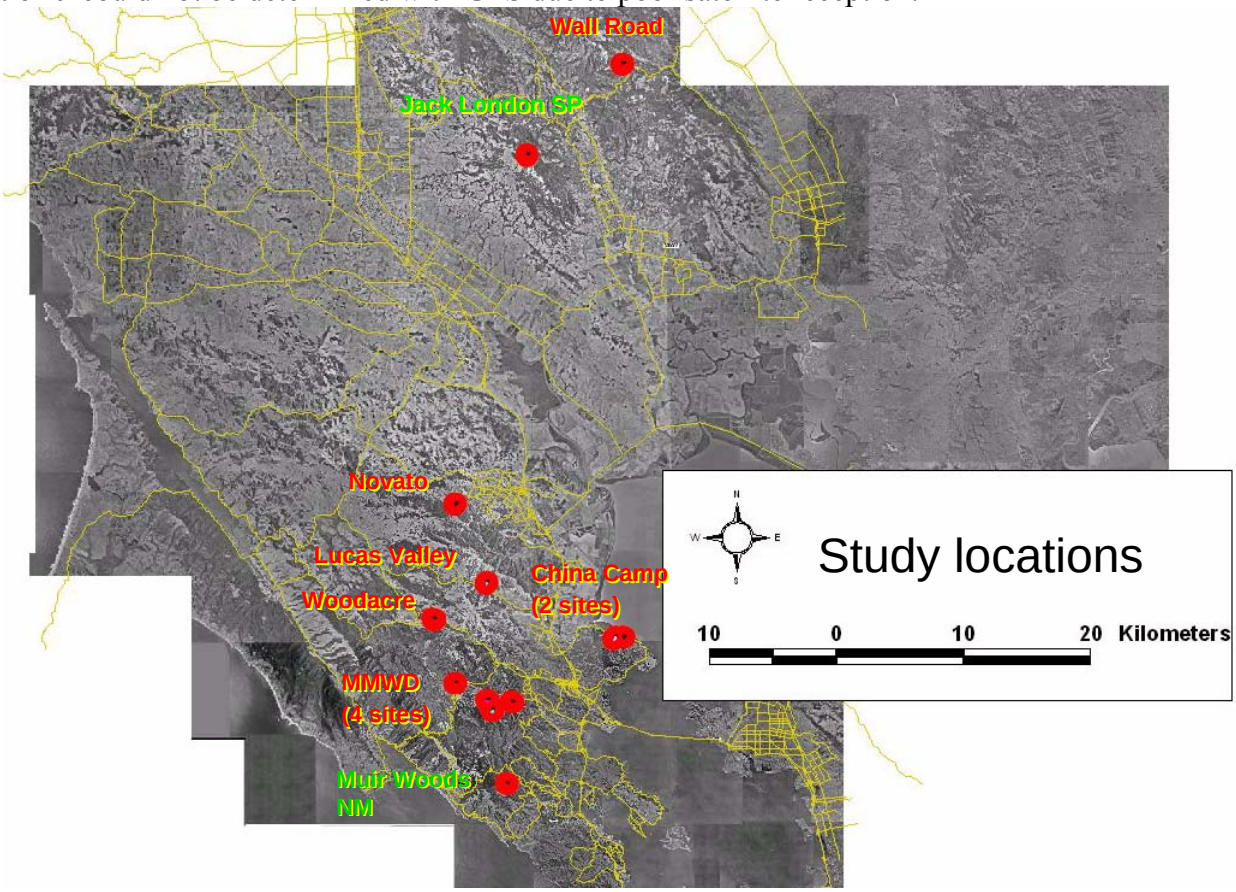

Figure 1. Map showing locations of study areas in Marin, Napa, and Sonoma counties. Background image is a mosaic of USGS digital aerial orthophotos.

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 2005b).

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 and 2006 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%.

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 2001a) 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	bark probe	mm
	C (live):	2004		
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	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	visual estimate	pretransformed 0-6 scale[2]; includes both shrubs and small (<3 cm DBH) tree regeneration
Plot shrub cover	2001, 2006	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	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	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 >10	regeneration = seedlings and saplings <3 cm dbh
Disease incidence in SOD host[3] regeneration	2000-on	count or estimate percent if count > 10	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	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	note presence	list shrubs and woody vines present within plot; herbaceous species and grasses were not scored
Increase in weedy herbaceous cover	2006	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 2000-2006

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. For tanoak, highest numbers of trees with apparently new infections were seen in 2001 and 2004. When expressed as a percent of asymptomatic trees from the previous year (*fig. 2*), 2002 and 2005 had the lowest rates of newly symptomatic trees whereas 2001 had the highest. In contrast, coast live oak showed the greatest increases in newly symptomatic trees in 2005 and 2006 (*fig. 2*). Both 2005 and 2006 had relatively high rainfall (*fig. 17*) including substantial periods of rainfall in the spring.

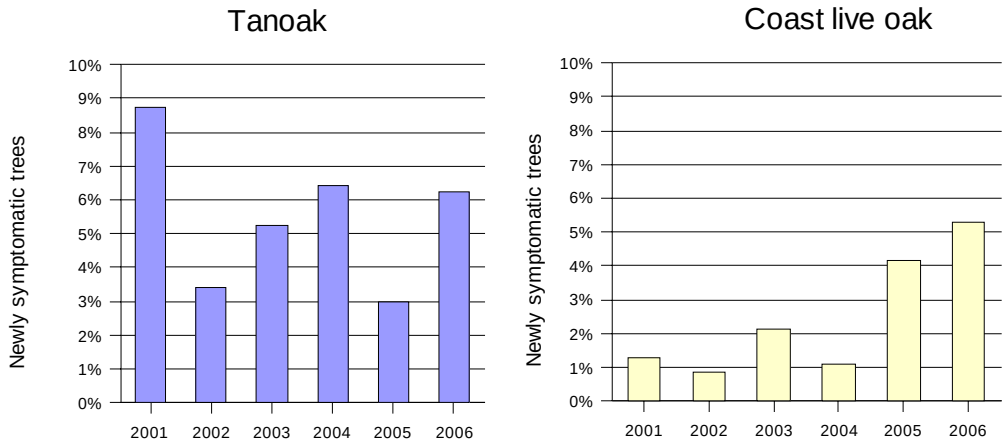

Figure 2. Newly symptomatic tanoak and coast live oak trees observed in each year shown as a percent of the number of asymptomatic trees present in the previous year. Total number of live asymptomatic trees present in 2000 was 126 for tanoak, 470 for coast live oak.

Overall, *P. ramorum* canker incidence among tanoaks has increased from 31% in 2000 to 46% in 2006 (*fig. 3*). *P. ramorum* canker incidence among coast live oaks has increased from 23% in 2000 to 30% in 2006 (*fig. 3*). A logistic model of the disease incidence data for both species for 2000 and 2006 showed significant differences between species (likelihood ratio $p<0.0001$) and years (likelihood ratio $p<0.0001$).

The overall percentage of the study tree population killed by *P. ramorum* was also significantly higher for tanoak than coast live oak (likelihood ratio $p<0.0001$) and has also increased significantly between 2000 and 2006 (likelihood ratio $p<0.0001$). The absolute change in percent mortality due to *P. ramorum* (*fig. 3*) over the 2000-2006 interval is much greater for tanoak (18%) than for coast live oak (7%). However, the percentage of trees killed by *P. ramorum* was much higher for tanoak than for coast live oak at the start of the study. The relative increase in SOD-related mortality over the 2000 to 2006 interval was been similar for both species (2.5× increase for tanoak, 2.8× 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 had again become 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. A total of 64 coast live oak trees became newly symptomatic between 2000 and 2006. Reclassification of 20 previously symptomatic trees to asymptomatic has resulted in a net increase of 44 symptomatic trees between 2000 and 2006.

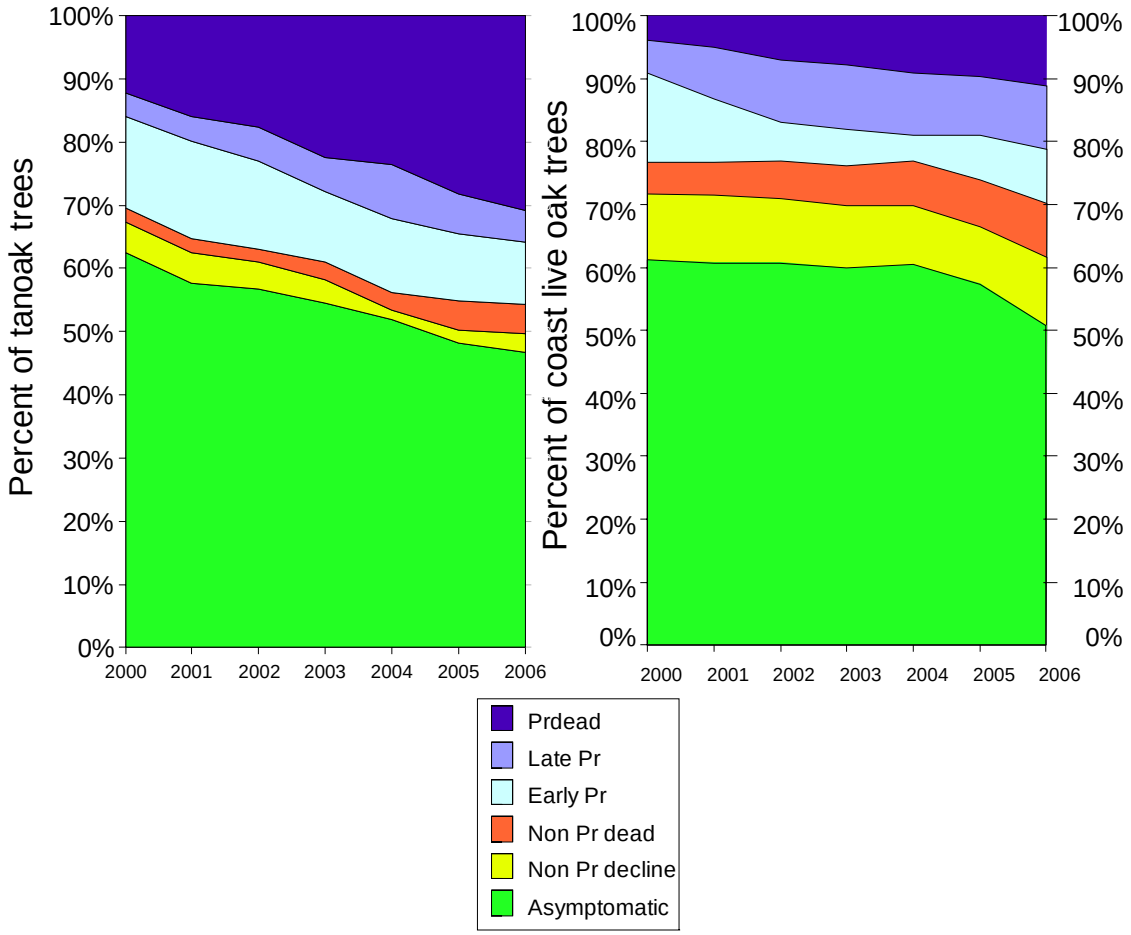

Figure 3. Changes in health of all tanoak (n=187) and coast live oak (n=655) study trees from September 2000 to September 2006. **Dead Pr** = tree dead as a result of *P. ramorum;* **Late Pr** = live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **Early 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; 54% of the tanoaks and 89% of the coast live oaks killed by *P. ramorum* between 2001 and 2006 were symptomatic in 2000. Much of the difference in mortality between tanoaks and coast live oaks among these trees developed in the

first year (*fig. 4*). 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. Since both of these years had higher levels of rainfall that were more favorable for inoculum production and new infections, it is possible that the elevated mortality among these tanoaks in years 5 and 6 was associated with additional infections that developed in these years. Among live trees that had *P. ramorum* canker symptoms in 2000, 32% of coast live oaks and 63% of tanoaks had died by 2006 (*fig. 4*).

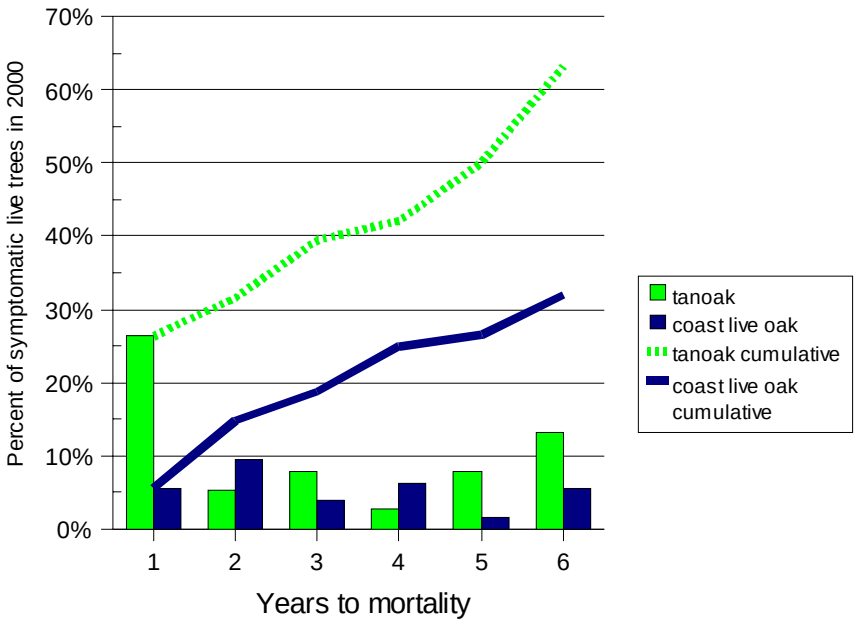

Figure 4. Years to mortality and cumulative mortality from 2001 through 2006 for living trees showing symptoms of *P. ramorum* infection in September 2000. The date of initial symptom development for this cohort of trees is unknown. (tanoak n=38, coast live oak n=128).

The trend in mortality rates over time among trees that first developed visible symptoms after 2000 (*fig. 5*) was similar to that seen in the cohort discussed above. For tanoaks that first became symptomatic during the period from 2001-2006 (n=38), 45% have died, many of these within a year or two of becoming symptomatic (*fig. 5*, 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 2006, only 7% have died, and these deaths happened within the first two years after symptoms were first observed (*fig. 5*). Because trees became symptomatic in a number of different years, the minimum survival times for the time-censored trees is at least one to five years (*fig. 5*, bottom). A high proportion of the surviving coast live oaks are shown in the "at least 1 year" category because they initially developed symptoms in 2006 (*fig. 2*).

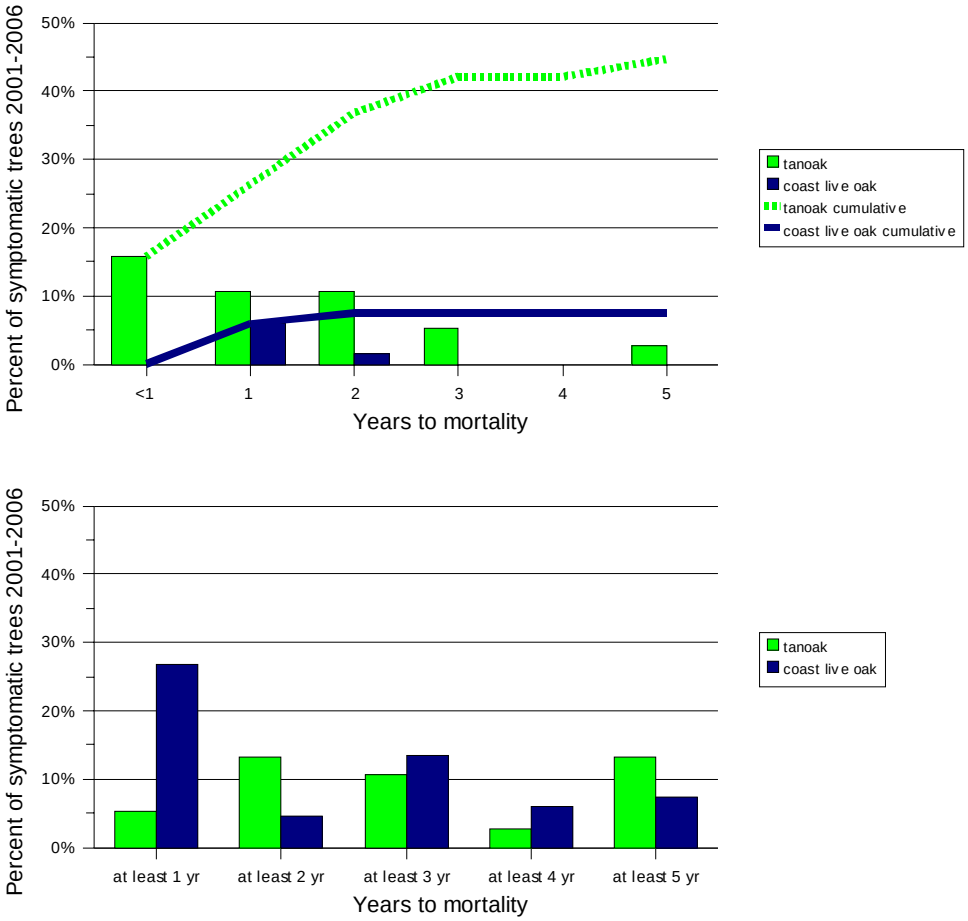

Figure 5. Years from symptom onset to mortality for trees that became symptomatic in the period 2001-2006 (tanoak n=38, coast live oak n=67). Top graph shows year by year and cumulative data for trees that died by 2006; lower graph shows the minimum possible survival period for trees that were still live in 2006

Disease incidence and mortality by study location

Figures 6 and 7 show changes in *P. ramorum* symptom status between 2000 and 2006 for trees at each of the 12 study locations. At tanoak locations 9 and 12 and coast live oak locations 3 and 7, more than 20% of the trees without symptoms in 2000 developed *P. ramorum* canker symptoms by 2006 (*fig. 6*, right). Most other locations showed relative disease increases of between 10 and 20%. Coast live oak locations 2 and 4 were notable for the low numbers of new infections observed across the entire 2000-2006 period (*fig. 6*). These locations also showed almost no increase in disease in 2005 and 2006 (*fig. 7*). Disease incidence increased at most other coast live oak locations in one or both of these years (*fig. 7*); location 7 showed an especially large increase in disease incidence between 2004 and 2005.

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 2006 (*fig. 6*, *7*) 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.

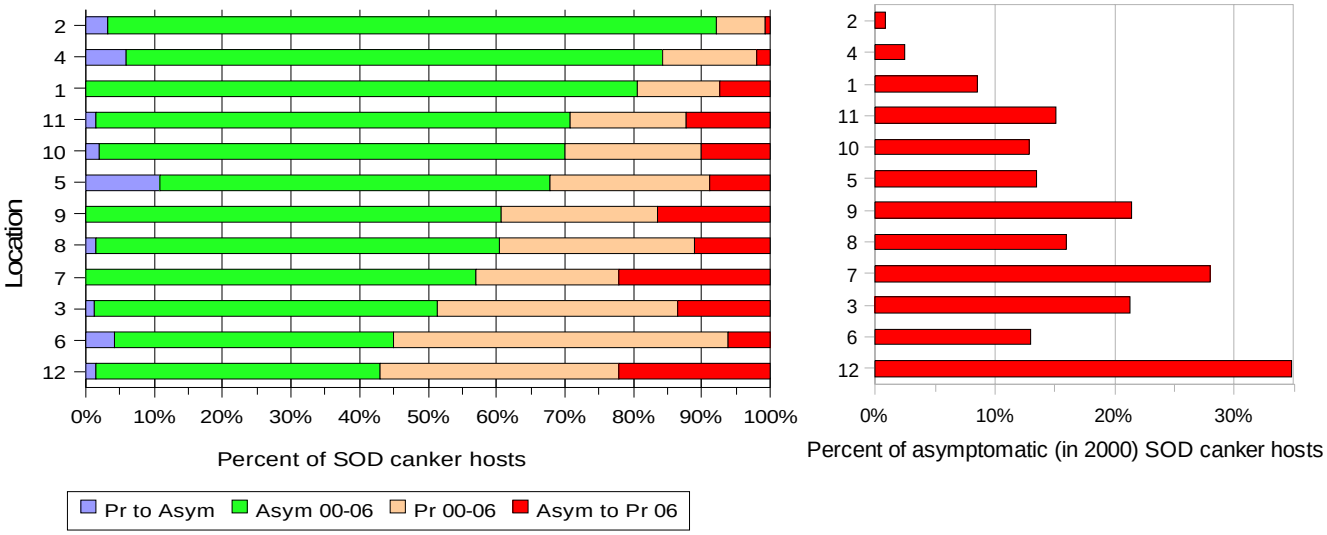

Figure 6. Percent of SOD canker hosts (coast live oak, California black oak, and tanoak) showing changes in overall *P. ramorum* canker symptom status between 2000 and 2006 by study location. **Left:** disease status changes as percent based on the total tree population present in 2000, including trees that were dead in 2000 or died between 2000 and 2006. **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 2006. **Pr 00-06** = tree with *P. ramorum* canker symptoms in 2000 through 2006; **Asym 00-06**= trees without *P. ramorum* canker symptoms in 2000 through 2006 (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 2006.

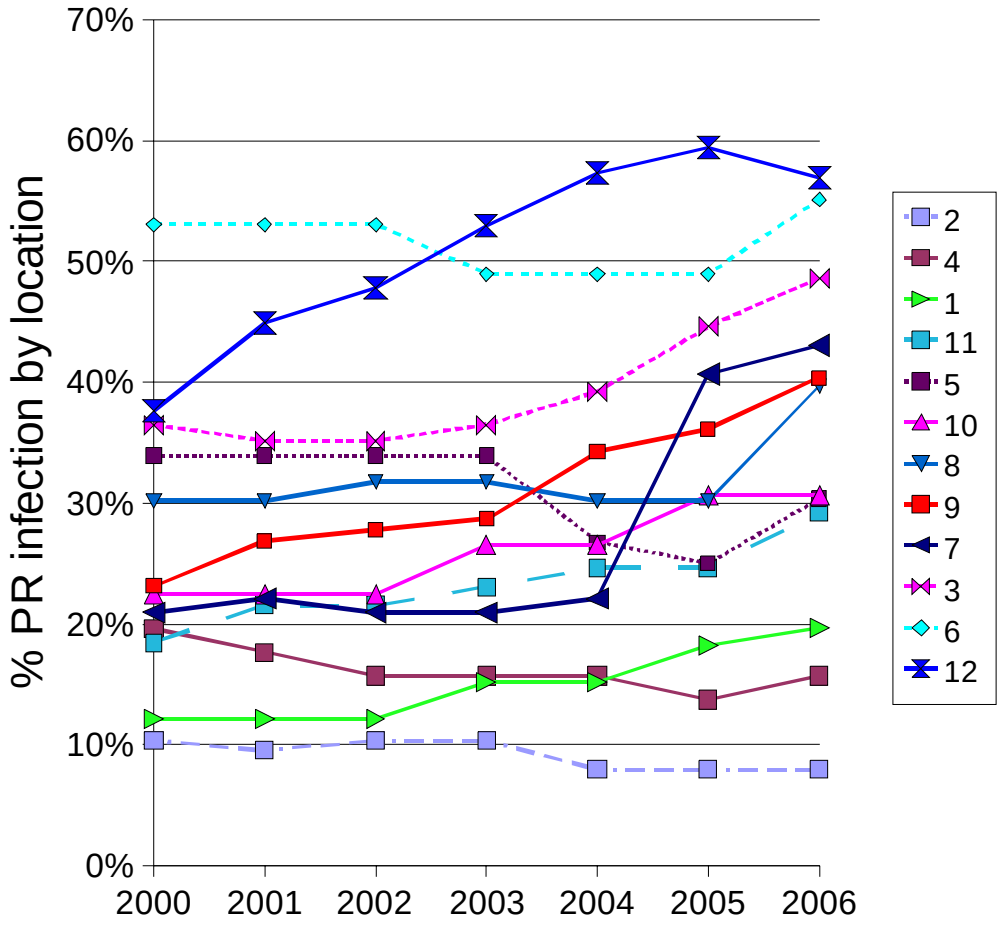

Figure 7. Percent of SOD canker hosts (coast live oak, California black oak, and tanoak) with *P. ramorum* (PR) infection in 2000 through 2006.

Figure 8 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 36% at location 3. Overall mortality rates among tanoak at the two tanoak study locations were 33 and 44%. *P. ramorum* has been the leading cause of mortality among study trees at both tanoak locations and at 6 of the 10 coast live oak locations (locations 3, 4, 5, 6, 8, and 11). Wood decay fungi, particularly species of *Inonotus* and *Ganoderma*, have been important causes of tree mortality at several of the coast live oak locations.

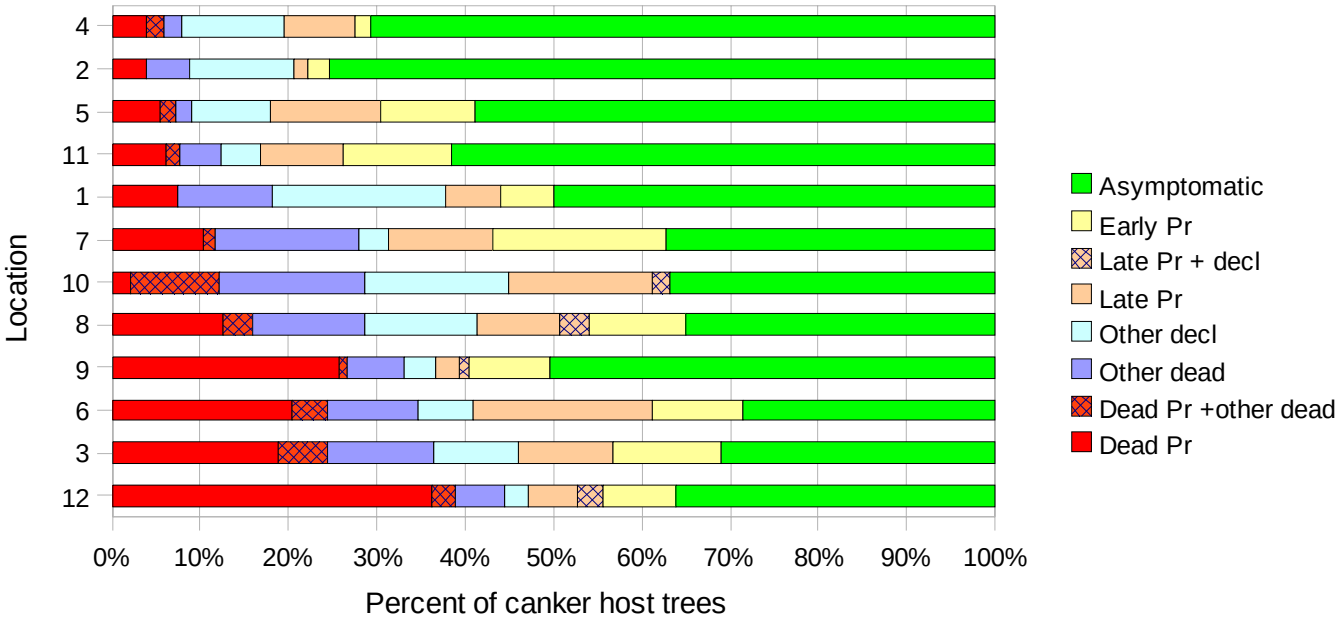

Figure 8. Overall disease status of SOD canker hosts (coast live oak, California black oak, and tanoak) in 2006 by study location. **Pr** = tree with *P. ramorum* canker symptoms; **early** = bleeding cankers only, **late** = bleeding cankers with beetles and/or *H. thouarsianum*; **other decl/dead** = 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.

Changes in stand structure

Because most plots had overlapping tree canopies, tree mortality did not always result in a decrease in plot canopy cover. Among the 22 tanoak plots, four showed decreases in canopy cover between 2001 and 2006, while two had increased canopy cover. All four plots with decreased canopy cover had *P. ramorum* induced tree mortality. However, 12 other plots with *P. ramorum*-related mortality did not show decreases in plot canopy cover. Among the 128 coast live oak plots, canopy cover levels between 2001 and 2006 did not change in 75% of the plots, increased somewhat in 2.3% of the plots and decreased in 22.7% of the plots. Forty-one percent of plots with *P. ramorum*-related coast live oak mortality showed decreased plot canopy cover between 2001 and 2006. In comparison, 14% of plots without *P. ramorum* induced mortality showed a decrease in canopy over this same interval.

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 2006 was significantly higher than that of asymptomatic trees (one way ANOVA $p<0.0001$ for both species). In 2006, 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 15 cm, compared with 8.4 cm in uninfected trees. This effect, which has been noted previously (Swiecki and Bernhardt, 2004),

suggests that larger diameter trees are more susceptible to infection than are smaller diameter trees.

Over time, we would expect that this would reduce the average DBH of the remaining trees in the stand. However, the mean diameter of the largest stem of trees that were live in 2000 and 2006 did not differ 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 2006. 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 2006.

We counted and assessed the condition of all trees other than SOD canker host trees in the plots in 2001 and again in 2006. California bay was the most common plot tree after coast live oak, with a total of 451 live trees in all plots as of 2006. 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 2006. Douglas-fir was the third most common tree in this group, with 217 trees in 2006. The Douglas-fir population increased by 31 between 2001 and 2006, an increase of 17%, 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 313 live trees in 2006. This species also had the highest level of mortality among the non-SOD canker host species. Between 2001 and 2006, 33 madrone trees died, 9.8% 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 2006. Locations 5, 6, and 7 showed both the highest overall levels of madrone mortality (22, 17 and 27%, respectively) and the greatest increase in mortality between 2001 and 2006 (9, 10, and 17% respectively). These levels of mortality exceed the levels *P. ramorum*-related mortality seen in coast live oak at most of the study locations (*fig. 8*). 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 2006.

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 2005b).

In the full data set, trees that were either healthy or had only early symptoms of *P. ramorum* canker (bleeding cankers only) in September 2006 showed no overall change in their average levels of canopy dieback since September 2002 (*fig. 9*). 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 2006 showed increasing levels of canopy dieback between 2002 and 2006 (*fig.*

9). Effects of year, 2006 disease status, and the interaction between year and 2006 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 2006,.

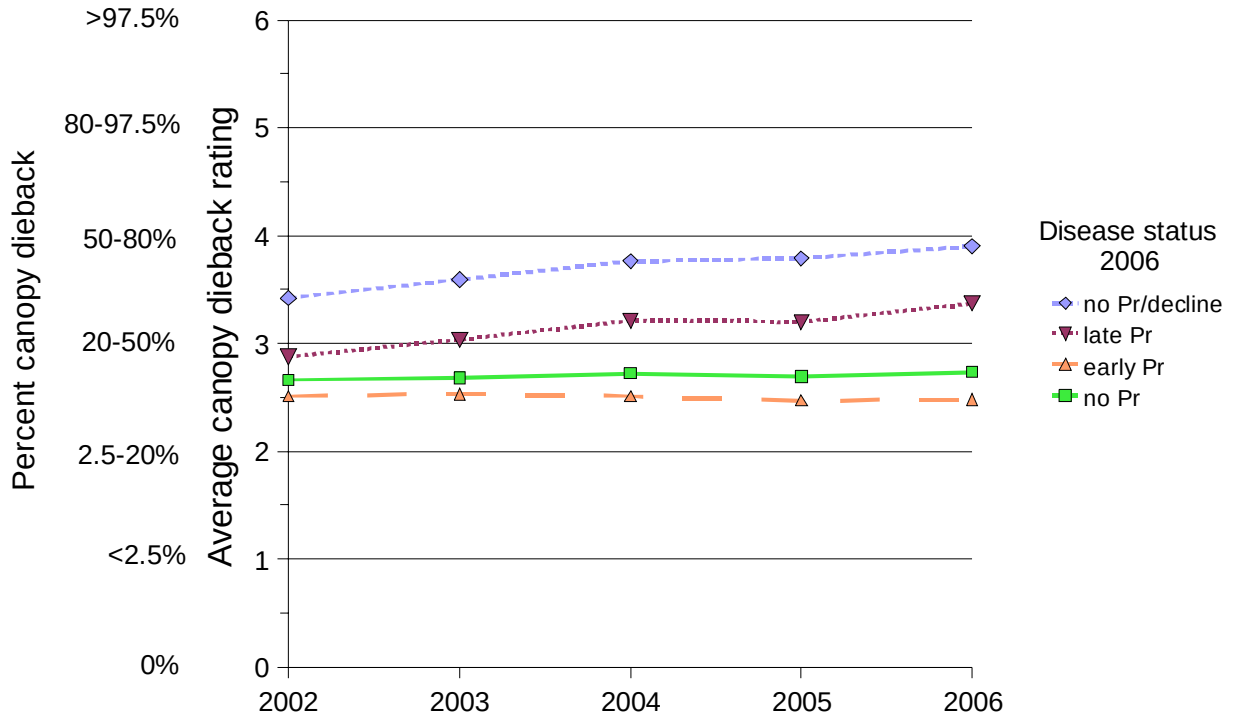

Figure 9. Canopy dieback ratings in September for coast live oak trees in each disease class as of Sept 2006; **decline/no Pr** =trees in severe decline due to agents other than *P. ramorum*, **late Pr**=live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **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 141 failures in coast live oak, 4 in California black oak and 26 in tanoak that exceeded these thresholds. For coast live oak, the majority of the failures affected the main stem (n=104); the average stem diameter at the failure was 32 cm. The average diameter at the point of failure for the coast live oak branch failures was 30 cm. All of the above-threshold failures in tanoaks occurred in main stems (average diameter at break 17 cm).

Most (54%) 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, 74% 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. 10*). To date, most of the failures observed in asymptomatic trees also occurred in live branches and stems.

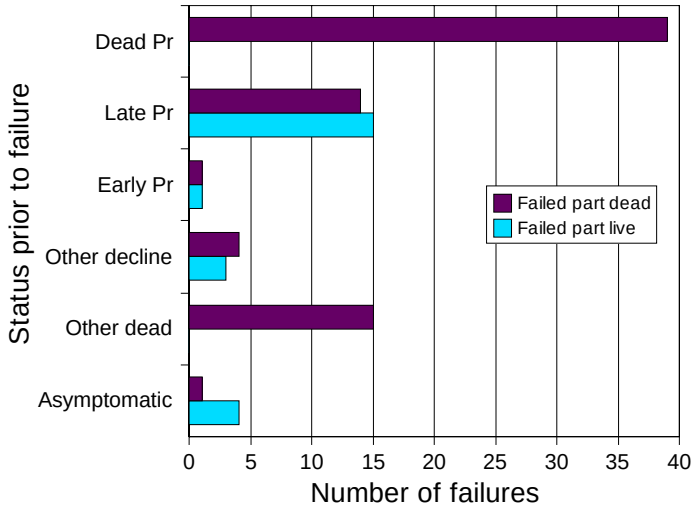

Figure 10. Tree disease status and condition of the failed part (live or dead) at the time of failure for initial failures above threshold size occurring between September 2000 and September 2006 in coast live oak study trees.

Because the disease status of many study trees has changed between 2000 and 2006, various failure rates can be calculated from the data. In *figure 11*, 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. Graphs shown in previous reports (e.g., Swiecki and Bernhardt 2006) included some trees which had failed prior to 2000 as nonfailed. Additional field data collected in 2006 allowed us to verify the status of these trees so they could be excluded from the analyses.

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. 11*). Trees that were dead due to *P. ramorum* in 2000 have remained at a 95% failure rate since September 2006; one of the trees in this group has not yet had a major failure. Both of these groups of dead trees attained 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. 11*). Among failed trees with *P. ramorum* canker, all but one tree had either died or progressed to the late disease stage prior to failure (*fig. 11*). Thus, the elevated failure rate among trees with early *P. ramorum* canker symptoms in 2000 is almost entirely related to the progression of disease to the late stage or tree death prior to failure.

As of 2006, only one tree has failed that was rated as having early *P. ramorum* canker symptoms at the time of failure. The canker was inactive in this tree, but the wood in the cankered area was exposed and dry, though not obviously decayed. The defect associated with this inactive canker and pre-existing structural problems (included bark and excessive branch end weight) contributed to the failure of a large (52 cm diameter at failure) branch.

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. 11*). If we consider only trees that were live in 2000, the failure rate among trees that were asymptomatic from 2000 to 2006 (18/513 = 3.5%) was significantly lower ($p<0.0001$) than among trees with symptoms of *P. ramorum* canker from 2000 to 2006 (49/144 = 34%).

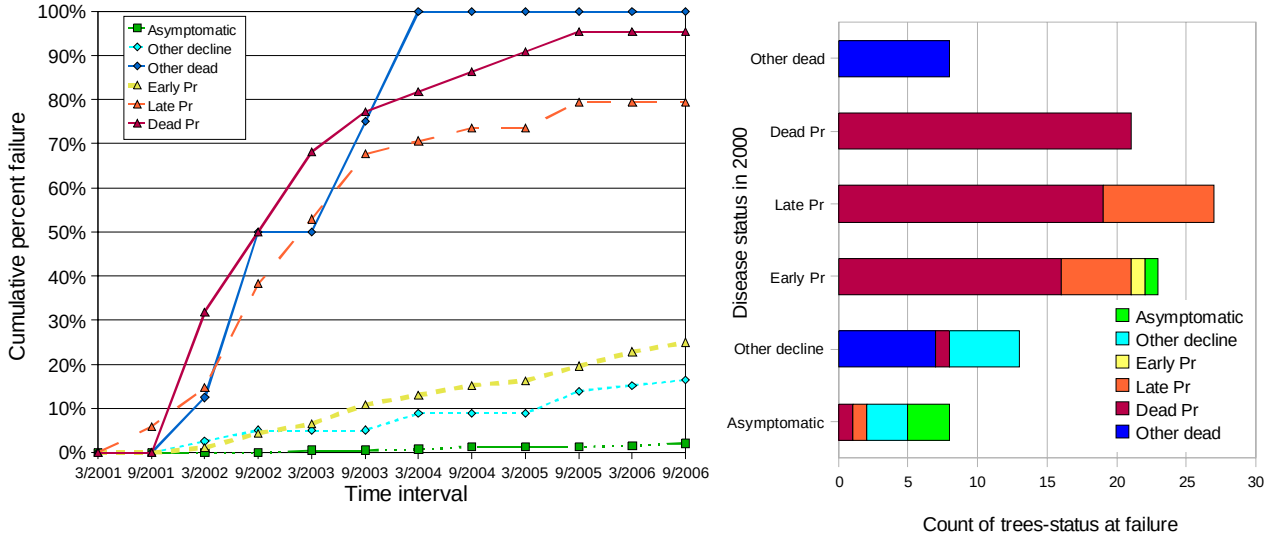

Figure 11. Cumulative failure rates (left) and tree disease status at failure (right) for coast live oaks by initial disease status in 2000. Only initial failures above the size threshold are shown. Failure dates were estimated to the nearest 6 month interval and failure percentages are plotted at the 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 12*, 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. 12*). 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 in 2006 (*fig. 12*).

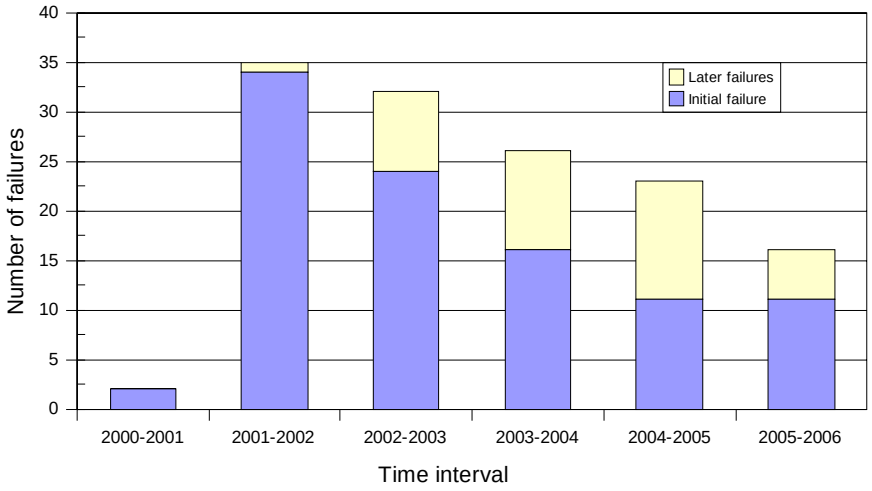

Figure 12. Number of initial (blue bars) and subsequent (yellow bars) failures above threshold size occurring in annual observation intervals between September 2000 and September 2006 among 629 coast live oaks that had not failed prior to September 2000. Annual observation intervals are from October 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 13*. 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 13 shows that although mortality had increased in a relatively linear fashion over the interval from September 2000 to September 2006, failure rates showed an initial lag followed by a steep increase in the first half of this period. For the past several years, the percentage of *P. ramorum*-infected coast live oaks with initial failures has approximated the mortality rate due to *P. ramorum.* Because failures are also common in living trees with late *P. ramorum* canker symptoms (*fig. 11*), the failure rate among *P. ramorum*-infected coast live oaks could eventually exceed the mortality rate.

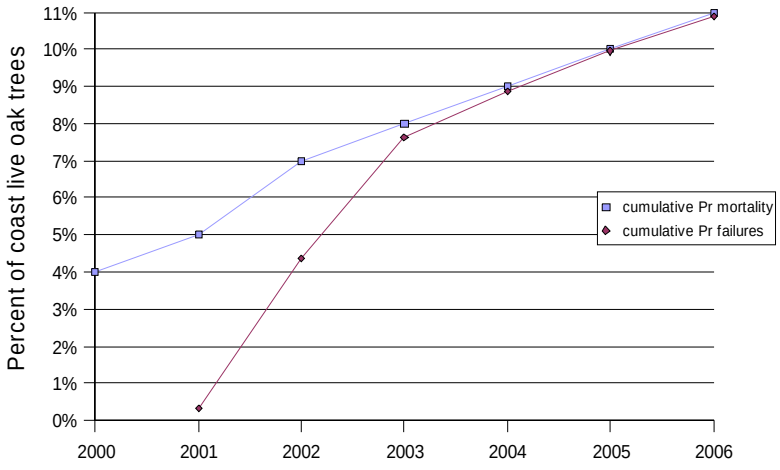

Figure 13. Cumulative initial failures in *P. ramorum*-infected coast live oak trees compared to the cumulative mortality rate due to *P. ramorum*. Mortality and failure percentages shown are cumulative to September of the year shown on the x axis.

Bole failures were the most common type of failure in coast live oaks with and without *P. ramorum* canker symptoms (*fig. 14*, bottom), but most of the bole failures observed were in trees with *P. ramorum* canker symptoms (*fig. 14*, top). Bole failures were common in trees in decline due to various wood decaying 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 occurred among the coast live oaks in the study, and it affected a tree that did not have *P. ramorum* symptoms.

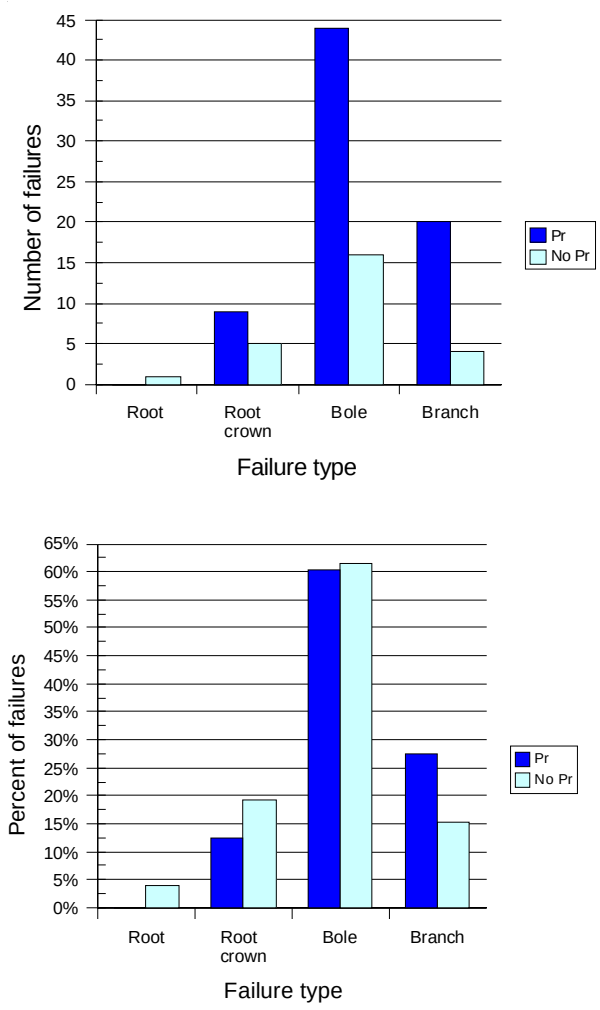

Figure 14. Frequency of initial failures above threshold size by failed part for failures occurring between September 2000 and September 2006 among coast live oaks with (**Pr**) and without (**No Pr**) symptoms of *P. ramorum* canker at the time of failure.

Our data set for failures in tanoak is much smaller than the coast live oak data set. Among the 195 tanoaks in the study, 26 failures have been recorded since 2000, including two root failures, 18 bole failures, and six root crown failures. Twenty-two 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. The incidence of failure among tanoaks with *P. ramorum* canker in 2006 (23/89=26%) was significantly greater than that observed in asymptomatic trees (3/106=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 2006. Only two of these plots had no coast live oak seedlings in any of the seven years of the study. Forty six percent of the plots had no seedlings in at least one year. In 2006, 23% of coast live oak plots lacked live seedlings in the understory.

Seedling numbers fluctuated widely, both within plots at the same location, and in the same plots from year to year (*fig. 15*). Average seedling counts per plot reached a maximum of 80 at location 8 in 2001 (*fig. 15*). In half of the plots, no more than ten coast live oak seedlings were observed in any year.

The number of dead seedlings tallied in plots each year was almost always much smaller than the drop observed in live seedling numbers from one year to the next, presumably because small dead seedlings do not persist in the plots. This is certainly the case for seedlings destroyed by herbivores. Hence, counts of dead coast live oak seedlings within plots did not provide an accurate picture of seedling mortality from year to year.

Seven locations showed peak seedling populations in 2001 (*fig. 15*, top), the only year in which average seedling counts per plot differed from other years (*table 4*). Location 8 was unique in having exceptionally high seedling counts (up to about 200) in some plots in 2001. In 2001, coast live oak seedlings also established in many plots where they had previously been absent (*fig. 15*, bottom). In locations that showed strong increases in seedling counts in 2001, the mean number of seedlings per plot has decreased sharply in subsequent years. In some of these locations, for example 2 and 11, the percentage of plots with seedlings also declined after 2001 (*fig. 15*, bottom). It appears that these decreases were due to mortality of many of the seedlings established in 2001.

Table 4. 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
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)
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)

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

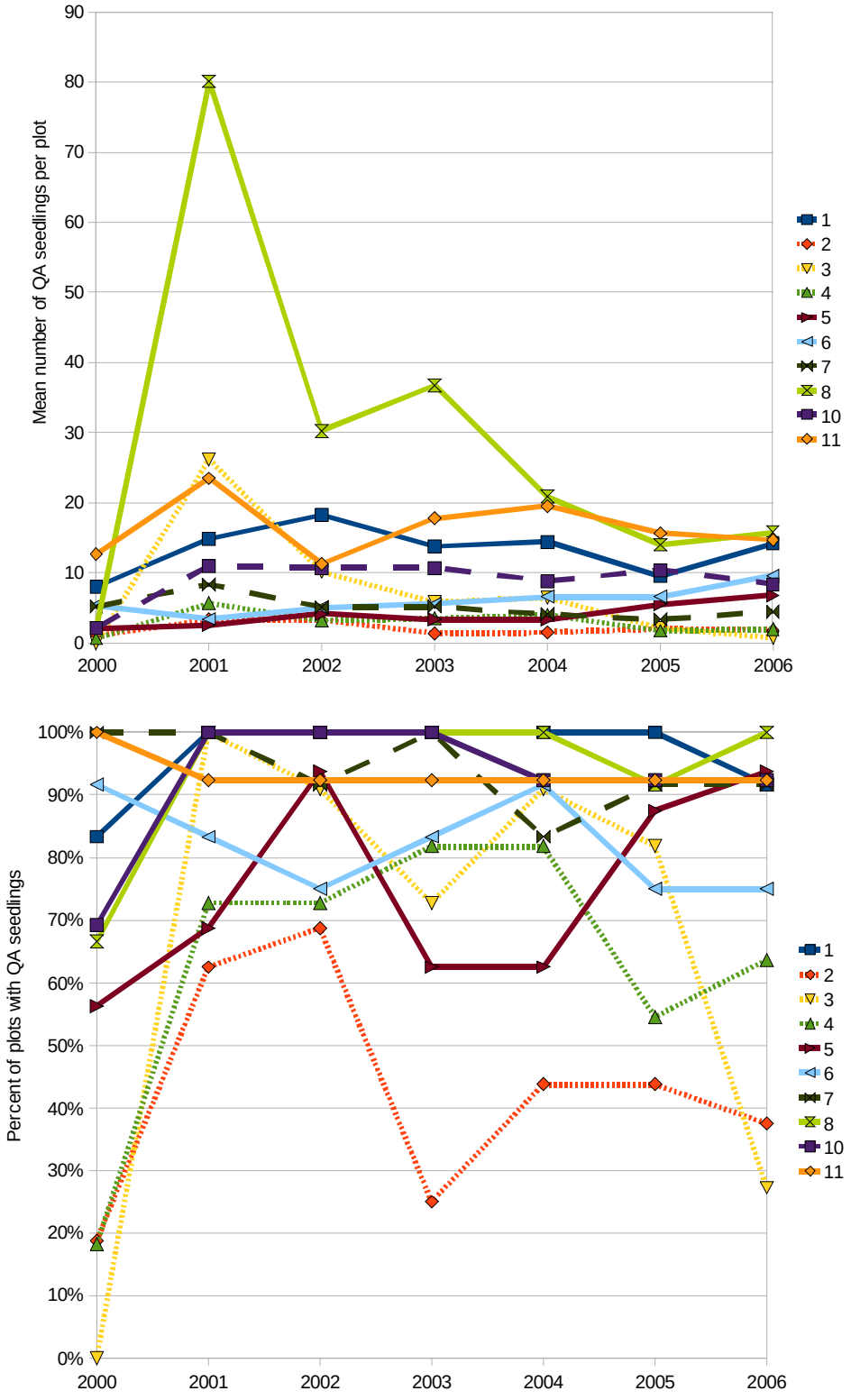

Figure 15. Mean numbers of coast live oak seedlings per plot at each of 10 locations with coast live oak overstory from 2000 through 2006, (top) and percent of plots with coast live oak seedlings (bottom). Location numbers shown in the legend correspond to locations in *table 1*.

Tanoak

Tanoak seedlings were present in all 25 plots with tanoak overstory between 2000 and 2006. 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. 15, 16, table 4*). 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. 16*). According to a repeated measures analysis of variance, seedling counts per plot have not changed significantly between 2000 and 2006 even though the incidence of disease and mortality due to *P. ramorum* has continued to increase among trees over this period (*fig. 3*).

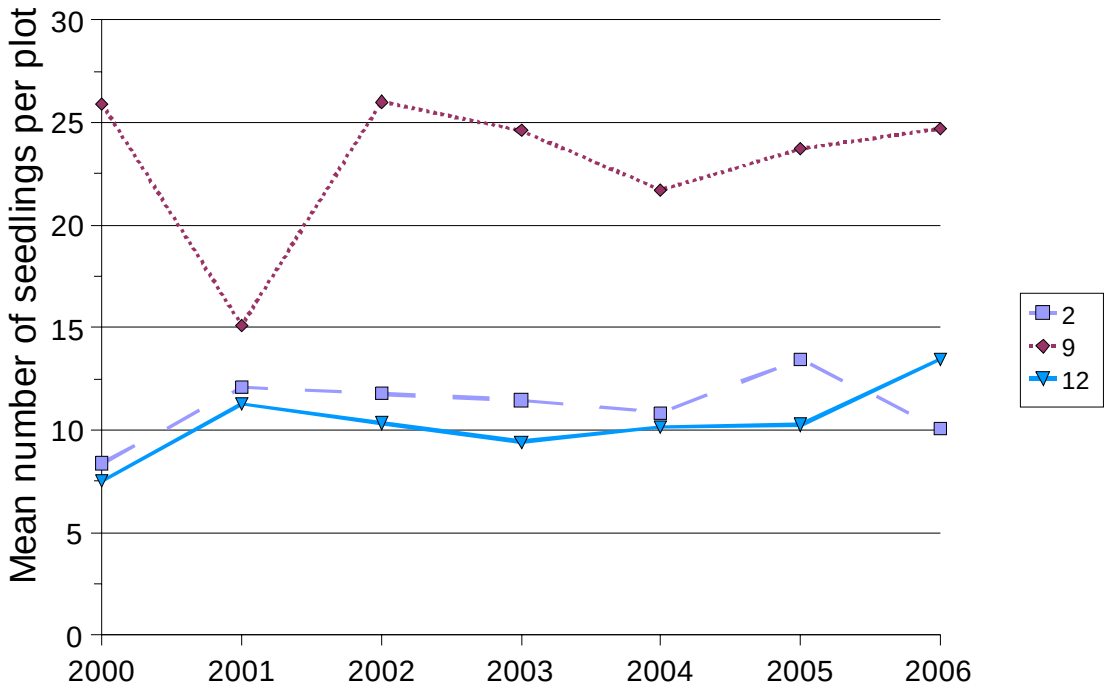

Figure 16. Mean number of tanoak seedlings and seedling sprouts per plot at each of three locations with tanoak overstory. Location numbers shown in the legend correspond to locations in *table 1*.

California black oak seedlings were extremely scarce in the plots. 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.

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, 2005b, 2006). 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 SWP for the three monitored trees at all locations is shown in *figure 17*. The pattern of annual changes in SWP is very similar among the locations, and correlates well with the annual changes in rainfall for years up until 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. However, the high temperatures did not result in unusually high reference evaportranspiration (ETo) readings, based on data from the nearest CIMIS weather station in Santa Rosa (*fig. 17*).

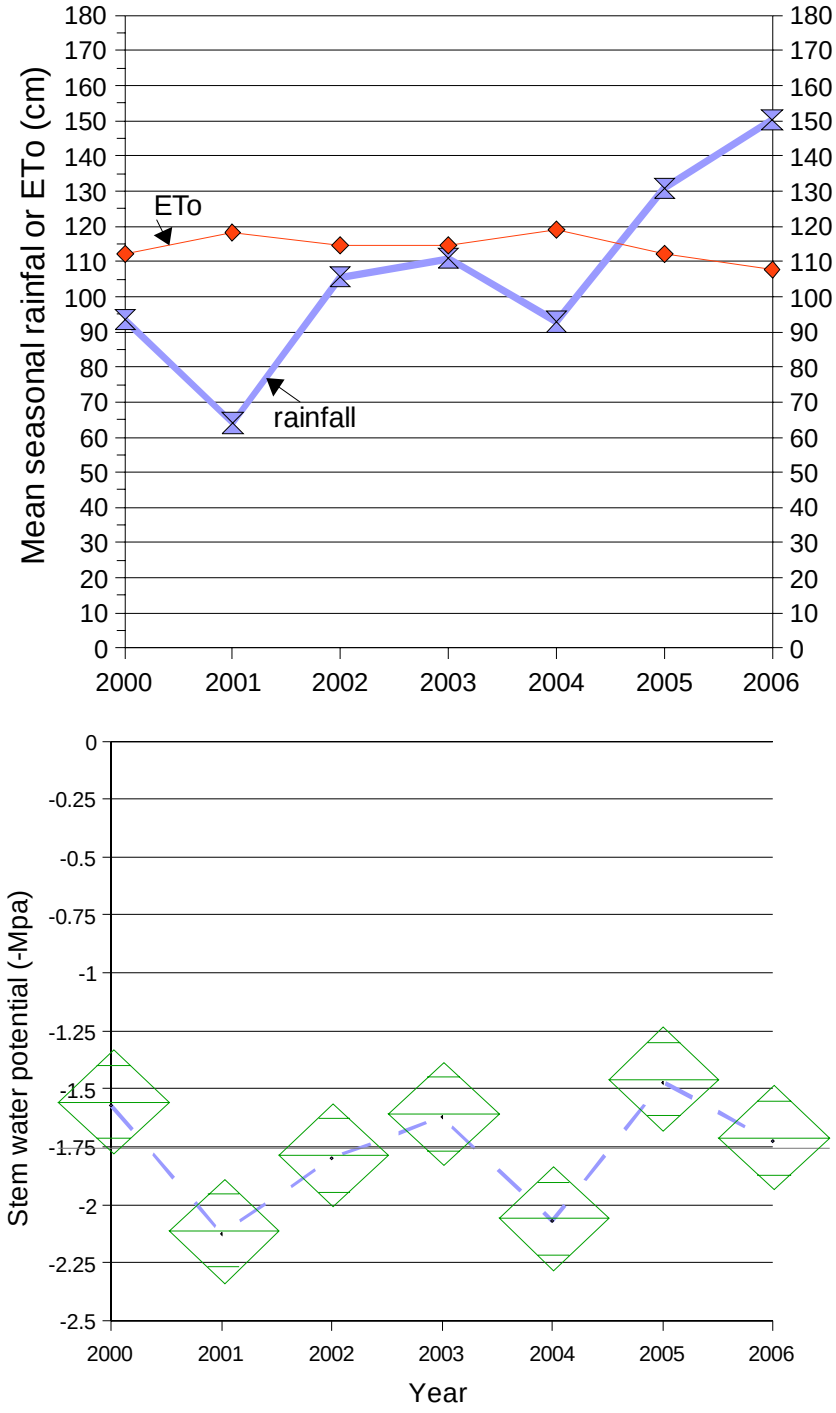

Figure 17. Average stem water potential for coast live oaks across all years and locations (bottom graph) compared with average seasonal rainfall and reference evapotranspiration (ETo) (top graph). ETo and rainfall are for September of the previous year through August of the listed year. ETo is from 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.

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, 3).* 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. 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 (*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. 17*). 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. High levels of new infection seen in 2001 after relatively low rainfall in 2000 may represent a carryover of latent infections from previous 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. If the most susceptible trees in the population tend to the killed at an earlier point in the epidemic, many of the most susceptible tanoaks may have already become infected by 2000. The favorable disease years of 2005 and 2006 may have had had less effect on new infections in tanoak than coast live oak (*fig. 2, 3*) if most susceptible tanoaks were already diseased by 2005 whereas many highly susceptible coast live oaks were not yet infected.

In addition, our data clearly show that coast live oak typically survives much longer than tanoak after symptoms of infection become apparent (*fig. 4, 5*). 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. 5*). 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 exist between the sites. Furthermore, most locations do not differ with respect to other climate and host factors that Meentemeyer and others (2004) used to create their model for risk of establishment and spread of SOD in California. All coast live oak locations with the exception of locations 4 and 10 are uniformly in areas predicted to be at high risk for establishment and spread of SOD based on the GIS layer from this model available at the Oakmapper website (http://giifserv.cnr.berkeley.edu/website/OakMapper/viewer.htm, accessed 31 January 2008). That model shows that the risk at location 10 is moderate and location 4 is shown to be at high risk but near the edge of a small moderate risk patch. Hence, the model does not differentiate well between coast live oak locations that have shown widely divergent disease progress curves (*fig. 7*).

The Meentemeyer and others (2004) model also did not predict relative disease levels for the two tanoak locations. Plots within location 12, which had the highest disease incidence of all study locations (*fig. 6*), are in a narrow band mapped as moderate risk within a larger high risk zone. Location 9, which has shown lower disease incidence over the entire study period compared to location 12, is mapped as a high risk zone.

We have previously constructed models that correlate disease risk with plot- and tree-level variables, including plot bay cover, variables describing the distribution of bay foliage around individual trees, SWP, and unweathered tissue in bark fissures (Swiecki and Bernhardt 2005b, 2007). These models can be used to predict the risk of disease at the level of individual trees, but models constructed to date require relatively detailed data for each tree. Because multiple factors operating at the level of the individual tree or plot can influence disease risk, broader models that use only landscape-level predictors (e.g., Meentemeyer and others 2004) do not readily account for differences in disease incidence seen within and between stands.

Failures

Most recent coast live oak failures in oak woodlands affected by *P. ramorum* have occurred in trees infected by *P. ramorum* (*fig. 11*; Swiecki and Bernhardt 2003b, 2004, 2005b, Swiecki and others 2006). Through 2006, live trees with SOD symptoms in 2000 had an overall failure rate that was about 10 times that of trees that have remained free of SOD symptoms over this period.

Data from this study confirm our previous observations (Swiecki and Bernhardt 2003b, 2004, 2005b, Swiecki and others 2006) that elevated failure potential in trees with *P. ramorum* canker is associated with colonization of trees by secondary organisms including *H. thouarsianum,* other decay fungi, and ambrosia beetles. 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. Given that small *P. ramorum* cankers in coast live oak can sometimes fail to progress to the late stage and may go into remission, 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. If tree hazard reduction is an issue, trees with only early *P. ramorum* canker symptoms (bleeding cankers only) should be monitored closely 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).

Although our data on failure rates in tanoak is much more limited than our coast live oak data, it is clear that failures are also more common in tanoak with *P. ramorum* cankers than in asymptomatic tanoaks. Due to structural differences between the tanoak and coast live oak trees included in the study, large branch failures have been less common in tanoaks than in coast live oaks. Both tree growth form (excurrent growth habit in tanoak compared to more spreading canopies in oak) and plot topography (48% average slope in tanoak plots compared to 35% in oak) probably contribute to the greater frequency of root failures in tanoak than in coast live oak.

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. 8*). 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 result in reduced 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. 16*) and most coast live oak locations (*fig. 15*). 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 2006. 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. 17*, 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.

One possible contributor to this unexpected result was associated with the record-breaking heat wave in July 2006, which lasted from July 16 to July 26 (Kozlowski and Edwards 2007). The heat wave had many days with consecutive record daily high temperatures but was also unusual in that night minimum temperatures were also unusually high. For 2000 through 2006, seasonal ETo did not appear to have a major effect on annual variation in SWP. However, it is possible that, during the extended period of high temperatures during the July 2006 heat wave,

soil moisture reserves were depleted much more than in a typical year, leading to reduced September SWP readings.

Another possibility is that prolonged wet soil conditions in 2006 adversely affected root health, leading to a reduced ability of the trees to exploit available soil water resources. Root health could have been directly impacted by prolonged periods of anoxia associated with soil saturation, and/or indirectly affected by opportunistic or weak root pathogens that were favored by wet soil conditions. We have documented that *P. cinnamomi,* a known pathogen of coast live oak is present in at least one of the study locations, and impacts to madrones in the infested area clearly showed that the activity of this pathogen had been favored by the wet soil conditions.

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