Phytophthora ramorum canker (sudden oak death) in coast live oak and tanoak, 2000-2004: factors affecting disease risk, disease progression, and failure potential

2004-2005 Contract Year Annual Report

Prepared for:
Garland Mason, Ph.D.
USDA Forest Service
Pacific Southwest Research Station
P.O. Box 245
Berkeley CA 94701-0245
510-559-6313

Prepared by:
Tedmund J. Swiecki, Ph.D.
Elizabeth Bernhardt, Ph.D.
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PHYTOSPHERE RESEARCH
1027 Davis Street, Vacaville, CA 95687-5495
707-452-8735
email: phytosphere@phytosphere.com URL: http://phytosphere.com

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Cover photo: *Phytophthora ramorum* canker on small diameter *Lithocarpus densiflorus* at Jack London State Park, in September 2000 (left) and September 2004 (right). Margin of canker where the pathogen was originally isolated has been exposed in the 2000 photo. By 2004, callus had developed around the margins of the canker, which had ceased bleeding and did not appear to have increased in size since the 2000 photo. The tree was still alive in 2004, but canopy dieback was rated at 50-80%.

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SUMMARY

Key words: stem water potential, water stress, resistance, bark thickness, bark morphology, tree failure

This report discusses findings after five 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 2004, 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.

Between September 2000 and September 2004, the percentage of symptomatic coast live oak trees in the plots increased slightly, from 23% to 24%. Over the same period, the percentage of symptomatic tanoaks increased from 31% to 43%. Between 2000 and 2004, mortality due to *P. ramorum* increased from 4% to 9% in coast live oak and from 12% to 23% in tanoak. About 58% of coast live oak and 47% of tanoak study trees with disease symptoms in 2000 progressed to a more advanced disease severity class by 2004. Diffuse canopy dieback developed in many coast live oaks with advanced *P. ramorum* canker symptoms that survived for at least several years. This pattern of slow decline in infected trees is distinct from the "sudden oak death" pattern that was originally described in trees with *P. ramorum* canker.

Among trees for which we have the most detailed disease ratings, 3 of 17 (18%) symptomatic tanoaks and 16 of 60 (27%) coast live oaks showed no apparent disease progress between 2000 and 2004 based on either canker girdling rating or symptom class. In both species, some symptomatic trees developed callus tissue along at least part of the canker margin where canker expansion was apparently inhibited. Also, in some infected trees, cankers have not changed in size for several years and appear inactive. These resistant reactions represent another symptom development pattern distinct from the rapid decline and slow progressive decline patterns seen in other trees affected by *P. ramorum* canker.

Between September 2000 and September 2004, coast live oaks with *P. ramorum* canker symptoms failed at a significantly higher rate than trees without *P. ramorum* canker. With only one exception, failures in *P. ramorum*-affected trees occurred in dead trees or in live trees with evidence of *Hypoxylon thouarsianum* and/or bark or ambrosia beetle colonization. The failed part was dead at the time of failure in 77% of all scored failures. However, in living trees with *P. ramorum* canker symptoms, more than half of the failures occurred in live branches or stems.

Most coast live oaks and tanoaks with *P. ramorum* canker symptoms maintained relatively high stem water potential (SWP) levels and did not show progressive increases in water stress as disease progressed. For coast live oak, trees with low water stress (high SWP) were more likely to develop *P. ramorum* canker than were more water-stressed (low SWP) trees. Subsequent disease progress in symptomatic trees was not related to SWP.

Only two of numerous bark characteristics assessed were associated with *P. ramorum* canker in coast live oak. The presence of unweathered brown bark in bark furrows was the only bark surface characteristic that was positively correlated with disease. Unweathered brown bark in bark furrows appears to be associated with faster rates of bole radial growth. The correlation between unweathered bark and disease is consistent with other analyses indicating that faster-

growing coast live oaks may have a greater risk of developing *P. ramorum* canker than slow-growing trees. Bark thickness was positively correlated with both the risk of developing *P. ramorum* canker and the likelihood of disease progress among infected trees. Because bark thickness also increases with stem diameter, it is possible that the lack of cankers on small coast live oak stems and branches could be related to their relatively thin bark.

INTRODUCTION

Phytophthora ramorum, the causal agent of sudden oak death, causes bleeding bark cankers on the main stems of tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), California black oak (*Q. kelloggii*), and several other oak species in California (Garbelotto and others 2001, Rizzo and others 2002). The bark cankers can expand over time and eventually girdle susceptible trees. 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 sudden oak death. 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 predictors of disease risk in coast live oak include canopy dominance (canopy sky exposure), plot canopy cover, stem water potential (SWP, a measure of water stress), tree decline associated with other disease agents, stem diameter, and multiple main stems. 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.

By collecting data on disease status over time in these plots, we have been able to determine how the disease status of study trees has changed over time, improving our overall concepts of disease risk and disease progress in this population. In addition, between 2000 and 2004 we collected data on additional variables that were not considered in the original study design. In particular, bark morphology characteristics, including bark thickness, were tested as possible predictors of disease risk and progress in analyses reported herein.

Repeated observations on the trees in these study plots over the past 5 years has also provided detailed information on long-term symptom development and disease progress in this cohort. These observations include ratings of canker expansion; the abundance of *H. thouarsianum* stromata and beetle boring; changes in canopy health; branch and main stem failures; and September water stress levels, as indicated by SWP. From these observations, we have been able to document patterns of disease progress in trees affected by *P. ramorum* that were not apparent when sudden oak death was originally described in California (Garbelotto and others 2001, Rizzo and others 2002).

METHODS

Study site selection

During September 2000, we established plots at 12 study locations (Table 1, Figure 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 25 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.

The distribution of plots across the landscape varied by location. 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.

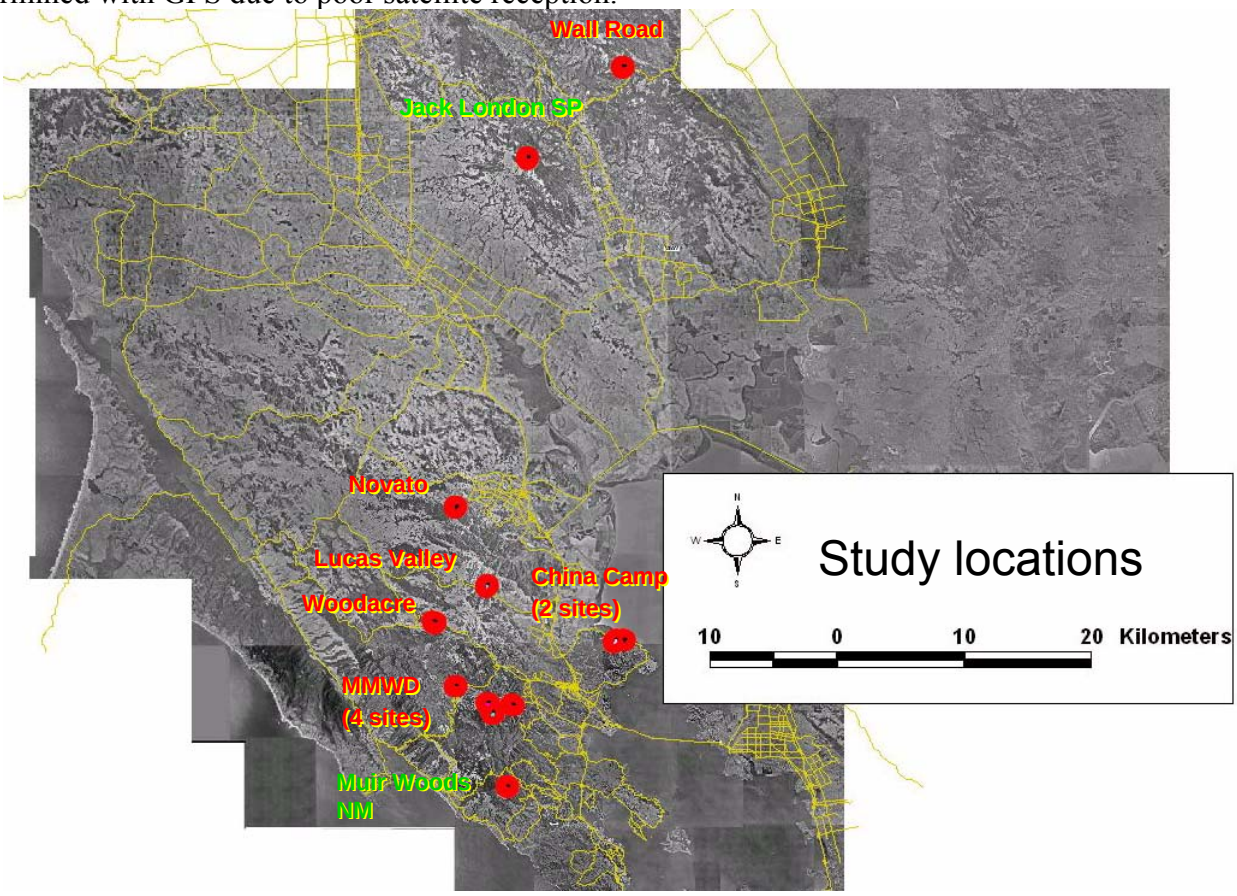

Figure 1. Map showing locations of study areas in Marin, Napa, and Sonoma counties. Background image is a mosaic of USGS digital aerial orthophotos.

Stem water potential measurements

In September of each year (2000-2004), 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) following methods outlined by Shackel (2000). In addition, starting in 2001, readings were made on additional trees in 45 of the plots (one additional tree per plot except one plot with two additional trees) for comparative purposes. 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 2 or more hours 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.

Stem water potential readings can vary from day to day due to differences in daily vapor pressure deficits (VPD). To estimate VPD during the period that SWP readings were made, we recorded minimum and maximum temperature and relative humidity values during this period using a portable electronic thermohygrometer (Mannix TH Pen, model PTH8708). The relative humidity readings of the thermohygrometers were calibrated using a sling psychrometer. In all years, one thermohygrometer was placed in a vented shelter mounted on a mast and was positioned near the upper portion of the tree canopy layer during the observation period. Starting in 2001, we used a second shaded thermohygrometer mounted about 1.5 - 2 m above the ground to measure conditions below the canopy and determine whether VPD varied with position in the canopy during the measurement period. VPD was calculated from the average of the recorded minimum and maximum temperature values using the following formula:

$$VPD\ (KPa) = [0.6108 \times e^{(17.27T/(T+237.3))}] \times (1-RH/100) \qquad \text{(Equation 1)}$$

where:
T = average temperature (degrees Celsius)
RH = average relative humidity.

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 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 paper.

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). We used a variety of descriptors to rate bark morphology of the lower bole. Bark characteristics rated included the abundance (none, trace, low-moderate, moderate-dense) and location of epiphytic lichens and mosses; various surface bark morphologies (striate, checkered, smooth, furrowed, irregular); the presence of shallow, medium, or deep bark fissures; the presence of deep bark cracks; and the presence of unweathered, brown bark in the center of furrows or fissures. In the 2004 evaluations, we rated the relative abundance of this last characteristic using the 0-6 scale described below.

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%.

Bark thickness

Bark thickness was first measured in 2003. Bark thickness was measured using a needle-type bark probe, which functioned in the same way as the one described by Gill and others (1982). The bark probe measured the distance between the outer bark surface and the wood using a blunt-ended 1.9 mm diameter steel probe that was pushed by hand pressure through the bark layers. The probe tip does not penetrate the wood, so the depth of penetration, measured to the nearest millimeter, indicated total bark thickness at the point of insertion. All bark depth measurements were made in "average" bark depth areas, avoiding atypically high or low spots.

To minimize possible effects associated with bark probing on study trees, bark thickness data in 2003 was measured only on dead trees in the plots and extra SWP trees (Swiecki and Bernhardt 2004). After determining that using the bark probe on live trees did not initiate disease or cause significant damage, we expanded bark probe measurements to include all remaining plot center trees in fall 2004. Bark thickness was measured at each of the cardinal compass directions at 1 m above the ground surface. The four readings at each height were averaged to give a mean bark thickness reading for each tree at each height. The status of the bark at each point of measurement (live, dead but moist, dead and dry) was recorded. For dead trees and dead areas of living trees, bark was chipped open so that its thickness could be measured while viewing the bark in a radial or cross section. This prevented errors associated with gaps between wood and dead bark or penetration of the probe into decayed wood. On live trees, the bark area to be probed was sprayed with 70% isopropanol prior to insertion of the bark probe, which was cleaned with 70% isopropanol before each probe.

We adjusted bark thickness measurements to account for shrinkage due to drying as reported in Swiecki and Bernhardt 2004. The average percent shrinkage in thickness for samples measured in 2003 was 22.5% for fresh to dry bark and 12% for fresh to dead but moist bark. These shrinkage values were used to adjust individual bark thickness measurements of dead/dry or dead/moist bark samples to the estimated fresh bark thickness prior to data analysis.

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 $p \leq 0.05$. The likelihood ratio chi square statistic was used to test the significance of difference of proportions in 2×2 contingency tables. Effects of year and tree variables on SWP in 2000-2004 were tested using repeated measures analysis of variance. We used linear regression to test for associations between continuous outcomes (e.g., SWP) and continuous or categorical predictor variables. The nonparametric Spearman test was used to test for correlations between pairs of categorical variables (e.g., those using the 0-6 rating scale). We also used analysis of variance (F-tests) or t-tests to test whether mean levels of continuous variables differed between groups such as cases and controls.

We developed logistic regression models to examine the effects of factors on the binary disease outcome (plot center tree is diseased, i.e., a case) or a binary disease progress outcome (disease progress observed). We screened possible predictor variables using univariate logistic regressions, examined correlations between predictor variables, and tested whether models were

strongly influenced by outlying observations by comparing models with and without extreme outliers. We also used recursive partitioning (also known as regression trees, CART™, etc.) to develop preliminary models, investigate interactions between predictors, and determine optimum thresholds for creating binary variables. The recursive partitioning procedure splits data in a dichotomous fashion according to a relationship between the predictor and outcome values, creating a tree of partitions. Each partition is chosen to maximize the difference in the responses between the two branches of the split.

We developed multivariate models using a stepwise procedure. Factors were generally considered for entry into the multivariate models if odds ratios from univariate models were significant at $p \leq 0.10$. The significance level of each factor reported in the final models should be interpreted as if it were the last factor added to the model. We also calculated Akaike's information criterion (AIC) to compare the fit of alternative models. For models constructed for a given data set, smaller AIC values indicate better model fit.

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 o/ verhead 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 2000 and 2001 data	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: C (live):	2003 2004	bark probe	mm
Brown bark from recent bark expansion in fissures	C,A:	2003 2004	visual assessment	2003: present/absent 2004: 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
Tree density / species composition	2000	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
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	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	visual estimate	pretransformed 0-6 scale[2]; includes both shrubs and small (<3 cm DBH) tree regeneration
Plot shrub cover	2001	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.
Count by general tree health class (trees other than SOD hosts[3])	2000, 2001	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	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	note presence	list shrubs and woody vines present within plot; herbaceous species and grasses were not scored
Disturbance	2000	Note type of disturbance	roads, trails, logging, etc. within plot or near edge of plot were noted
Oak/tanoak failure in plot	2001	count	Bole and large limb failures (>20 cm diam) observed in the 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, 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 2000-2004

Overall disease incidence and mortality

Among 122 live tanoaks that were asymptomatic in 2000, 21.3% showed disease symptoms by September 2004. These probably represent both new infections and trees with latent infections that developed visible symptoms during the observation interval. Among 473 live coast live oaks that were asymptomatic in 2000, 23 (4.9%) showed disease symptoms by 2004. This increase in disease incidence was significantly less than that seen in tanoak (likelihood ratio $p<0.0001$). More than 50% of the newly symptomatic coast live oaks first exhibited symptoms in 2003. In contrast, the largest yearly increases in the number of newly symptomatic tanoaks were observed in the 2001 and 2004 ratings.

Although newly symptomatic trees have been observed among both tanoak and coast live oak between 2000 and 2004, only tanoak shows a steady net increase in disease incidence over this period (Figure 2). Including recently-killed trees present at the start of the study in the base tree population, the overall *P. ramorum* canker incidence in tanoak has increased from 31% in 2000 to 43% in 2004 (Figure 2). For coast live oak, overall *P. ramorum* canker incidence increased only from 23% in 2000 to 24% in 2004 (Figure 2). In coast live oak, 16 of the 124 live coast live oak trees that had *P. ramorum* canker symptoms for at least the first two years of the study have been reclassified as asymptomatic due to a lack of apparent cankers in later ratings. Losses in symptomatic coast live oaks largely offset the gains due to new symptom development, leading to almost no net change of *P. ramorum* canker incidence in coast live oak over the period. In contrast, none of the tanoaks with *P. ramorum* symptoms in 2000 had become asymptomatic by 2004.

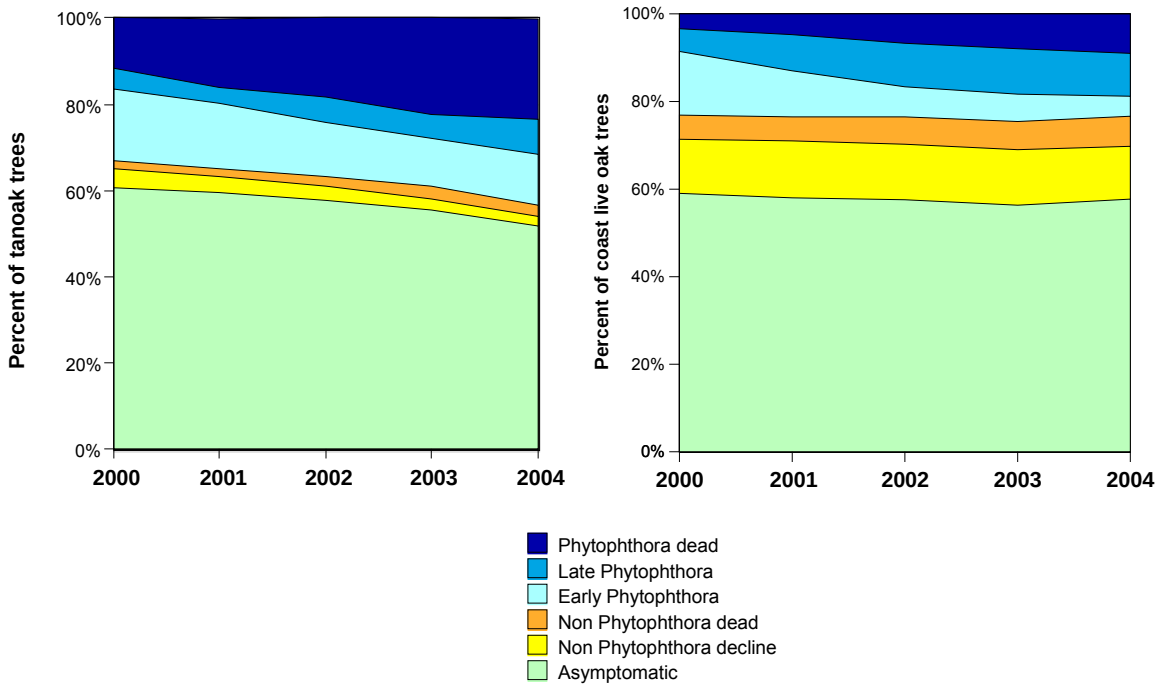

Figure 2. Changes in health of all tanoak (n=187) and coast live oak (n=655) study trees from September 2000 to September 2004. **Dead Pr** = tree dead as a result of *P. ramorum;* **Late Pr** = live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **Early 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.

Figure 3 shows the changes in *P. ramorum* symptom status between 2000 and 2004 for the 12 study locations. The two tanoak locations (9 and 12) had the highest percentage of trees that developed new *P. ramorum* canker symptoms over this period. All coast live oak locations except locations 2 and 6 also had some trees that developed *P. ramorum* canker symptoms between 2000 and 2004.

The percentage of newly symptomatic trees at a location was not significantly correlated with the percentage of trees that were symptomatic in 2000. For example, only locations 2 and 6 had no newly-symptomatic trees over this interval. These two locations had the lowest (location 2) and highest (location 6) initial levels of disease incidence in 2000 among the 10 coast live oak locations (Figure 3). Seven of the coast live oak locations also had some trees that were rated as symptomatic in 2000 and at least one additional year but were rated as asymptomatic by 2004. Because *P. ramorum* was not confirmed by isolations in all of these trees, it is possible that symptoms on some of these trees were due to causes other than *P. ramorum* and may not actually represent *P. ramorum* symptom remission.

Mortality due to *P. ramorum* has occurred at significantly higher rates in tanoak than in coast live oak (likelihood ratio $p<0.0001$) (Figure 2). In addition, most mortality occurred among trees that were already symptomatic in 2000. Among live trees that had *P. ramorum* canker symptoms in 2000, 35% of tanoaks and 26% of coast live oaks had died by 2004. Among trees that were asymptomatic in 2000, 6% of tanoaks but only 0.4% of coast live oaks developed *P. ramorum* canker symptoms and died by 2004.

Furthermore, symptomatic tanoaks typically died more rapidly than coast live oaks. Among tanoaks with *P. ramorum* canker symptoms that were live in 2000 and had died by 2004, 57% died within 1 year and 81% died within two years after symptoms were initially observed. In contrast, for coast live oaks, 26% died within 1 year and 62% died within two years after *P. ramorum* symptoms were initially observed. These totals include trees that developed symptoms after 2000 as well as trees that were symptomatic in 2000, so some trees may have been symptomatic longer than the periods noted.

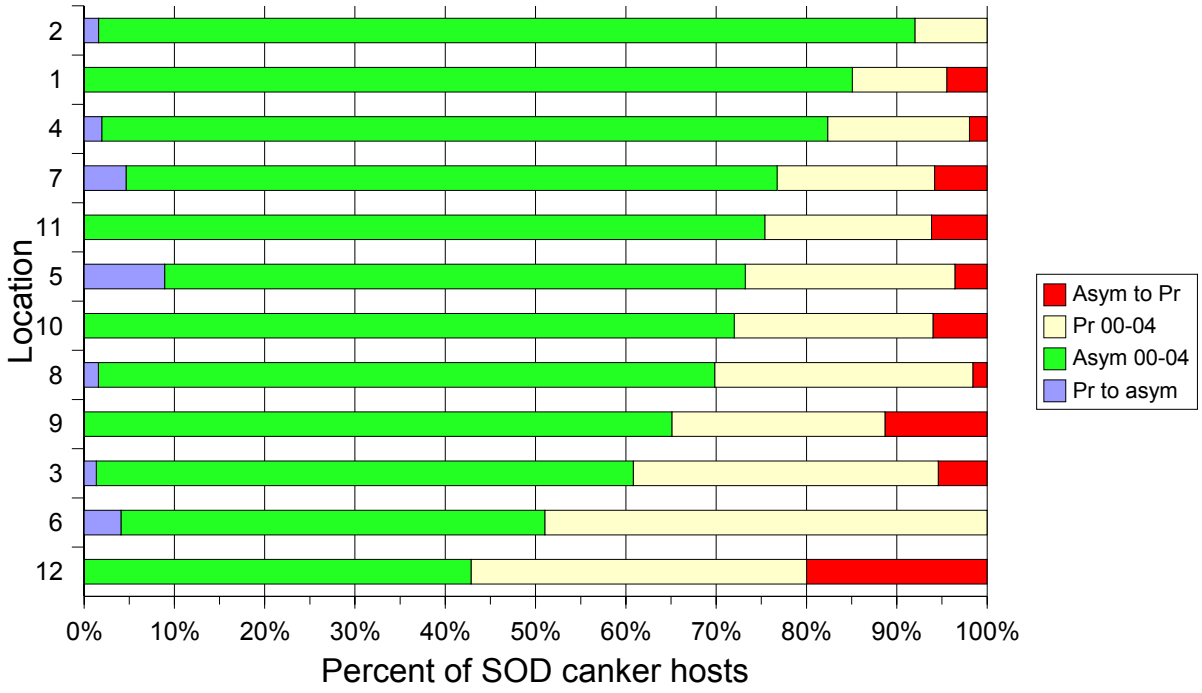

Figure 3. Percent of SOD canker hosts (coast live oak, California black oak, and tanoak) showing changes in overall *P. ramorum* canker symptom status between 2000 and 2004 ratings. For this graph, are pooled into the asymptomatic 2000-2004 category. Percentages include trees that were dead in 2000 or died between 2000 and 2004. **Asym to Pr=** trees which have developed *P. ramorum* canker symptoms since 2000; **Pr 00-04 =** tree with *P. ramorum* canker symptoms in 2000 through 2004; **Asym 00-04=** trees without *P. ramorum* canker symptoms in 2000 through 2004 (includes trees scored as having *P. ramorum* canker symptoms only in 2000); **Pr to asym=** trees scored with SOD symptoms in 2000 and at least one additional year but asymptomatic in 2004. Location numbers are shown in Table 1 and are sorted in order of increasing *P. ramorum* canker incidence in 2004.

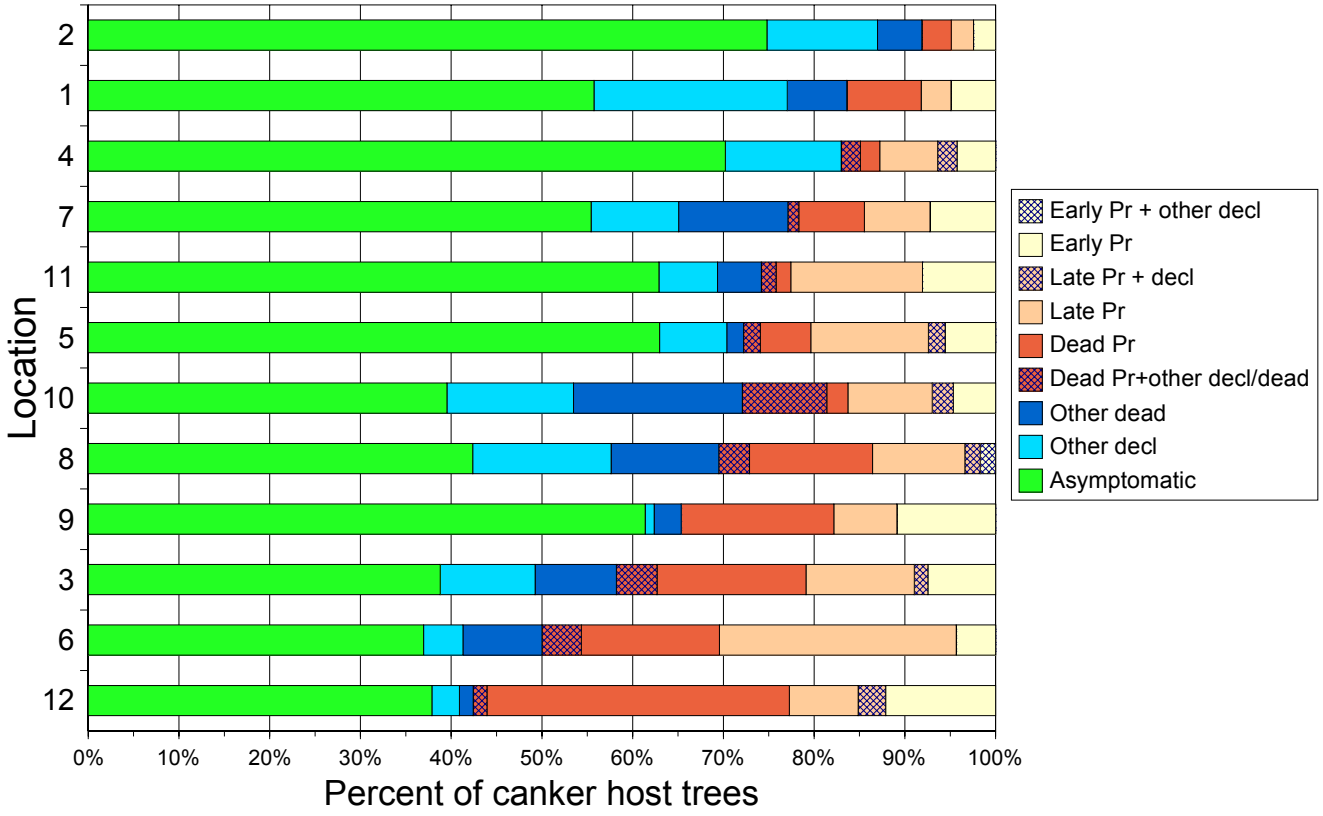

Figure 4. Overall disease status of SOD canker hosts (coast live oak, California black oak, and tanoak) in 2004. Pr = tree with *P. ramorum* canker symptoms; early = bleeding cankers only, late = bleeding cankers with beetles and/or *H. thouarsianum;* other decl/dead = tree declining or dead due to agents other than *P. ramorum*. Hatched bars indicate trees that have *P. ramorum* canker symptoms and are also declining due to other agents other than secondary agents typically associated with *P. ramorum* canker. Location numbers are shown in Table 1 and are sorted in order of increasing *P. ramorum* canker incidence.

P. ramorum canker has been the main cause of study tree mortality at both tanoak locations (locations 9 and 12, Figures 2, 4). Because most tanoak trees in the two tanoak study locations are relatively young and vigorous, only one tanoak has died due to agents other than *P. ramorum* since 2000. This was a 4 cm DBH root sprout originating from a tree which died and failed before the study started. The sprout died as the result of root disease associated with the failed stem. Mortality was also attributed to causes other than *P. ramorum* for three other tanoaks that were dead in 2000.

In contrast, canker rots and other wood decay fungi are relatively common in the coast live oak study locations, and these agents were associated with decline and death of numerous coast live oaks in the study (Figures 2, 4). At locations 2, 7, 10, and 11, most of the recent mortality (dating from approximately 1990) within plots was associated with agents other than *P. ramorum* (Figure 4). Due to high incidences of both *P. ramorum* canker and decline associated with other agents at locations 3, 6, 8, and 10, less than half of the SOD canker hosts at these locations were rated as asymptomatic.

Symptom development

In coast live oak and tanoak trees that were killed by *P. ramorum* canker, early symptoms typically consisted of bleeding bark cankers, but cankers did not necessarily bleed in all years. Recent bleeding (within the previous 6-12 months) was seen in 26% of the live tanoaks with *P. ramorum* symptoms each year from 2002 through 2004. In comparison, recent bleeding was seen in 39% of the live, symptomatic coast live oaks in 2002 and 2004, and in 54% of the live, symptomatic coast live oaks in 2003.

Over time, most *P. ramorum* cankers expanded in both radial and longitudinal directions. The number of apparently separate cankers tended to decrease over time, either because adjacent cankers merged or because the true extent of cankers became more obvious over time. We rated the percent of the bole circumference girdled by *P. ramorum* cankers in the plot center and extra SWP trees at each annual evaluation. For both tanoak (n=17) and coast live oak (n=52) trees with *P. ramorum* canker symptoms, the mean 2004 *P. ramorum* girdling rank of trees with only early disease symptoms was significantly less than the girdling rank of trees that had progressed to late disease symptoms or died (Table 4). All of the trees that had died were at least 50% girdled by *P. ramorum* cankers.

Table 4. Percent of total main stem circumference affected by *P. ramorum* cankers in tanoaks (n=17) and coast live oaks (n=52) by disease stage in 2004

	Mean (sd) *P. ramorum* canker girdling rating[1]		
Species	Early symptoms	Late symptoms	Trees killed by *P. ramorum*
tanoak	2.60 (0.55) a[2]	4.44 (0.73) b	5.00 (0) b
coast live oak	2.36 (1.36) a	3.97 (1.17) b	4.67 (1.07) b

[1] Percent of main stem circumference girdled by *P. ramorum* cankers estimated using 0-6 scale (see methods).
[2] Means followed by different letters are significantly different ($p<0.05$) according to Tukey-Kramer HSD test.

Late symptoms of *P. ramorum*-related decline include ambrosia beetle boring and/or stromata of *H. thouarsianum* that develop on larger cankers and may extend well beyond the cankers around and up the stem. All of the coast live oak study trees with *P. ramorum* symptoms that died between 2000 and 2004 had beetle boring and/or stromata of *H. thouarsianum* in the year prior to mortality and almost all trees had both of these agents visible in the year that death was recorded (Table 5). However, some small diameter (4.5 to 17 cm DBH) tanoaks did not exhibit late symptoms (beetle boring and/or *H. thouarsianum* stromata) before being killed by *P. ramorum* cankers (Table 5). Hence, although many smaller tanoaks were apparently killed by *P. ramorum* cankers alone, secondary agents were consistently associated with mortality in coast live oaks with *P. ramorum* canker.

Table 5. Number of tanoaks and coast live oaks with *P. ramorum* cankers that also had beetle boring or *H. thouarsianum* stromata in the year that tree mortality was recorded

Species	beetle boring	*H. thouarsianum* stromata	no beetles or *H. thouarsianum*	Total trees[1]
tanoak	7	4	7	15
coast live oak	28	29	0	29

[1] Data set includes plot center trees and extra SWP trees that were live in 2000 and died by 2004 and other plot trees that were live in 2001 and died by 2004.

Multiple stems and basal sprouts

In trees with multiple stems, each stem generally functions independently with respect to *P. ramorum* canker. Death of main stems generally occured independently and was related to canker severity on each stem. We recorded symptom data for individual stems in plot center and extra SWP trees for coast live oaks and tanoaks. Of the 70 multistemmed trees in this subset, 14 of 56 coast live oaks and 10 of 14 tanoaks had symptoms of *P. ramorum* canker in at least one main stem (Figure 5, left). However, in a substantial percentage of these symptomatic trees (7 coast live oaks and 7 tanoaks), main stems with and without *P. ramorum* symptoms were present in the same tree. By 2004, multistemmed trees with a mixture of both live stems and stems killed by *P. ramorum* were more common than multistemmed trees in which all stems had been killed by *P. ramorum* (Figure 5, right).

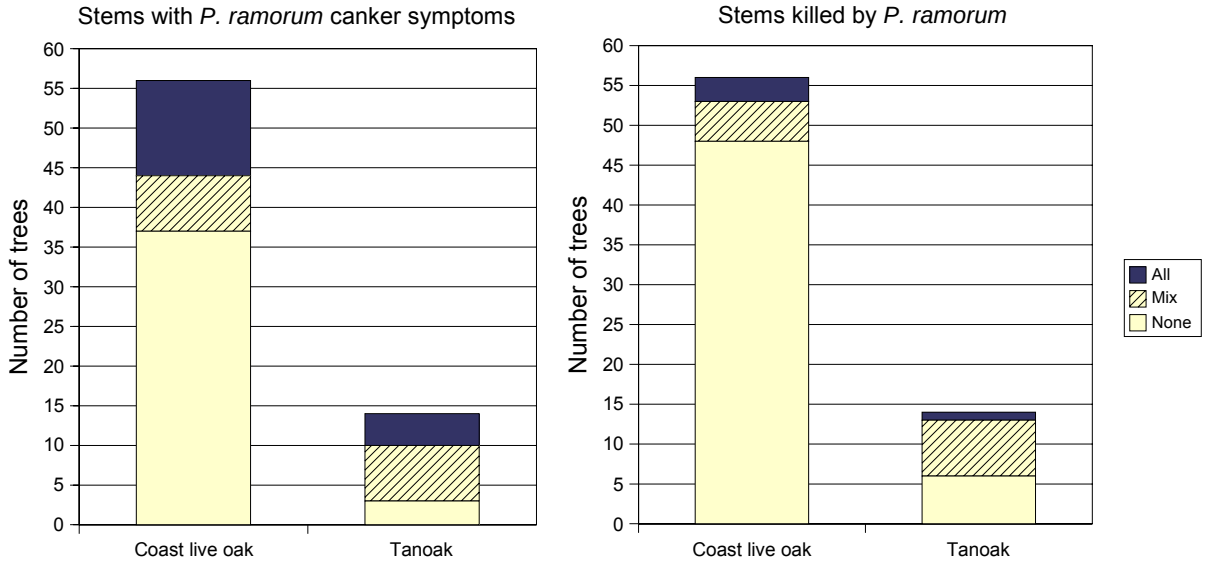

Figure 5. Occurrence of *P. ramorum* canker symptoms (left) or *P. ramorum*-related mortality (right) among individual main stems of multistemmed coast live oak (n=56) and tanoak (n=14) in September 2004. **All** = all main stems symptomatic or dead; **Mix** = mixture of asymptomatic and symptomatic (left graph) or live and dead (right graph) stems; **None** = no stems symptomatic or dead.

Many trees killed by *P. ramorum* cankers produce basal sprouts that can persist for at least several years after the death of the top. In addition, some trees with *P. ramorum* cankers that have experienced bole failures while still alive have subsequently produced vigorous epicormic sprouts on the remaining standing portion of the bole. As of 2004, 81% of tanoaks (33/39) and

43% of coast live oaks (22/51) with tops killed by or failed due to *P. ramorum* cankers had live epicormic sprouts (arising after bole failure) or basal sprouts. The data on sprouting and disease in multistemmed trees support our general field observations that, in most trees with *P. ramorum* canker, the decline of the top was directly related to the presence of stem cankers and was not associated with decline due to pre-existing root disease.

Limited symptom development

Overall, 47% of the tanoaks and 58% of the coast live oaks with early or late *P. ramorum* canker symptoms in 2000 progressed to a more advanced symptom class (late or dead, respectively) by 2004. In many of the remaining trees, symptoms progress has been limited and symptoms have not advanced beyond the symptom stage (early or late) observed in 2000.

Disease severity can increase without a change in symptom class if existing cankers expand and/or new cankers are initiated. For plot center trees and SWP trees, we monitored increases in *P. ramorum* canker severity by recording both symptom class (early, late, or dead) and the proportion of the bole girdled by cankers. Using both of these criteria, 3 of 17 (18%) tanoaks and 16 of 60 (27%) coast live oaks showed no apparent increase in disease severity between 2000 and 2004. These counts include all trees that were rated as symptomatic at least 3 of the 5 years. Most trees that showed no increase in disease severity over the five years initially had only a few small cankers that subsequently exhibited little or no bleeding and appeared to be inactive.

Over the past several years, we have also noted the production of apparently healthy callus at the edge of *P. ramorum* cankers in coast live oak and tanoak trees (Figure 6), especially in trees that have shown little or no symptom progress from year to year. Callus may either partially or completely encircle cankers. Callus development was noted in the detailed disease ratings for 3 of 17 live tanoaks and 22 of 52 live coast live oaks with *P. ramorum* cankers in 2004.

Figure 6. Callus development around apparently inactive *P. ramorum* cankers in coast live oak (left) and tanoak (right). Arrows indicate callus edge.

Many trees with late *P. ramorum* canker symptoms developed elevated levels of diffuse canopy dieback during the study (Figure 7). This gradual dieback and canopy thinning occured in trees with extensive cankers that survived for at least several years. It was distinct from the rapid dieback of the entire canopy ("sudden death") seen in trees that died within one to two years from the appearance of stem cankers. Among *P. ramorum*-infected trees that were still alive in 2004, canopy dieback rating was positively correlated with the *P. ramorum* stem girdling rating (Spearman R=0.555, p=0.034, n=14 for tanoak; Spearman R= 0.367, p=0.020, n=40 for coast live oak). Diffuse canopy dieback also increased over time in trees that were in severe decline due to other mortality agents, such as canker rot fungi (Figure 7).

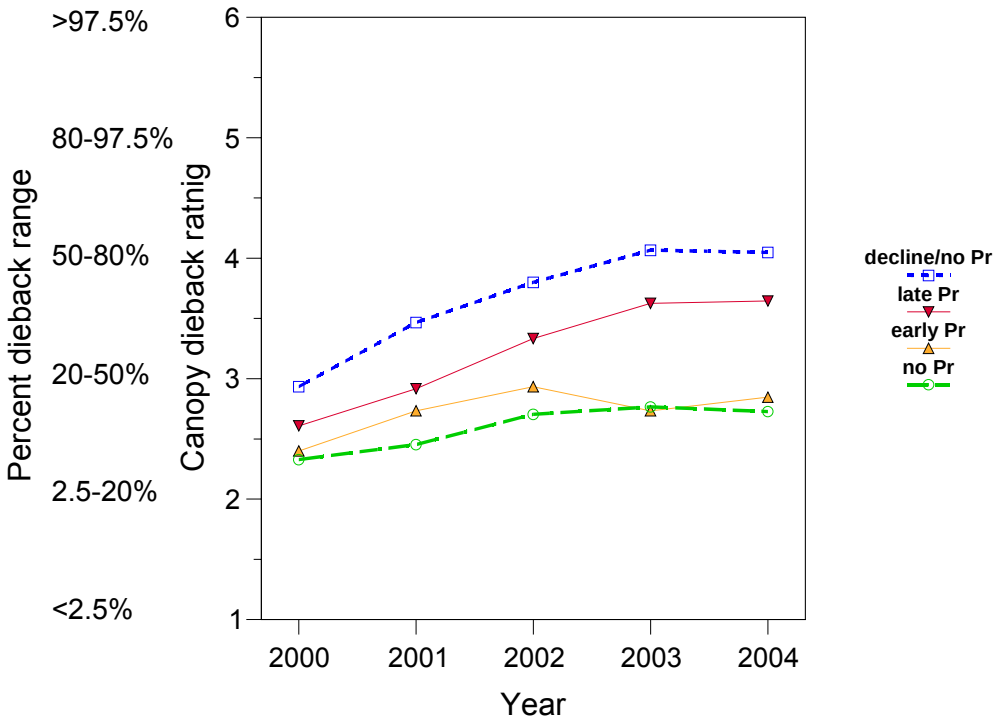

Figure 7. Canopy dieback ratings from September 2000 to September 2004 in coast live oak that were still living in 2004; **decline/no Pr** =trees in severe decline due to agents other than *P. ramorum*, **late Pr**=live trees with *P. ramorum* cankers plus beetle boring and /or *H. thouarsianum* fruiting bodies; **early Pr**=trees with *P. ramorum* cankers only, **no Pr**=non-declining trees with no evident symptoms of *P. ramorum* infection.

Tree failure

Between 2000 and 2004, we observed 56 failures above the minimum size threshold (branch failures ≥20 cm diameter; and bole, root or root crown failures of trees greater than ≥ 3cm DBH) among 153 coast live oaks with *P. ramorum* symptoms. This 36% failure rate in *P. ramorum*-infected trees was significantly greater (likelihood ratio $p<0.0001$) than the 4% failure rate seen over this period among 484 coast live oaks without *P. ramorum* symptoms. Bole and branch failures were the most common type of failures in trees with *P. ramorum* canker symptoms (Figure 8). The lack of root failures among trees with *P. ramorum* was consistent with other observations noted above that suggest that such trees werre not commonly affected by root disease.

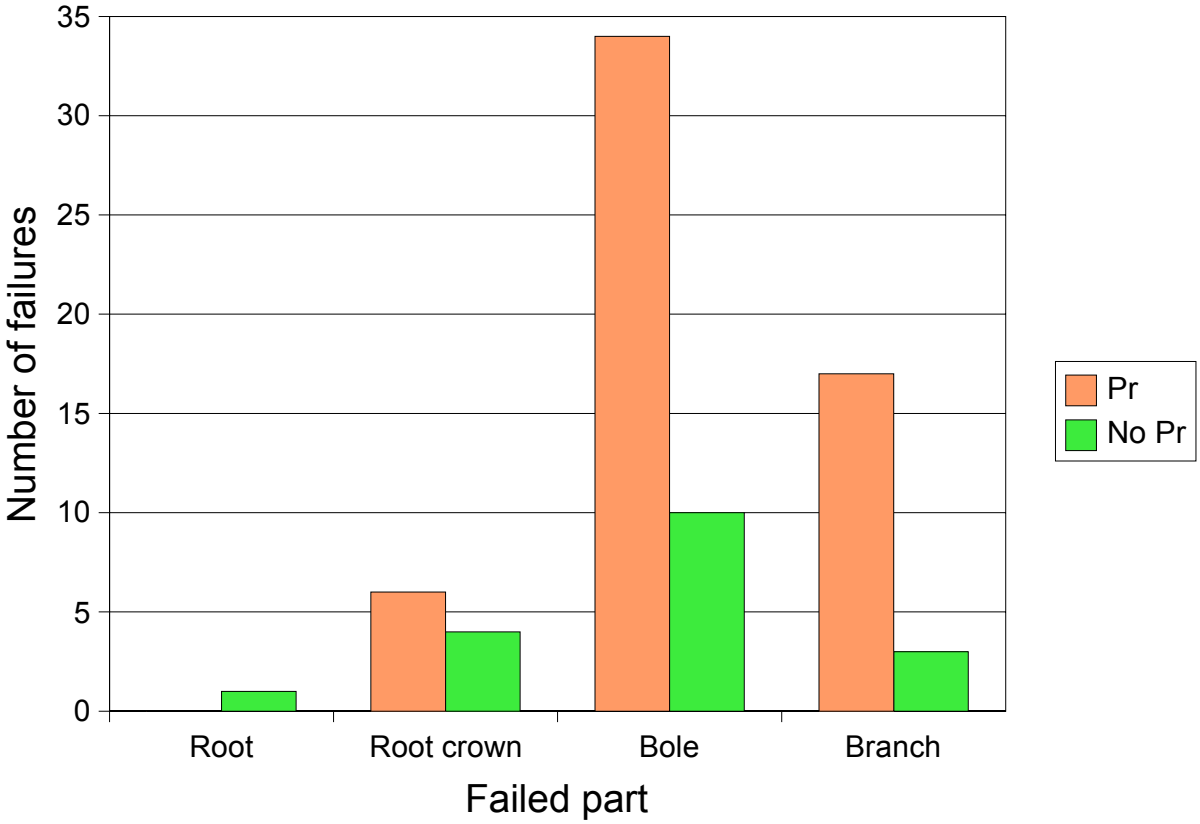

Figure 8. Number of initial failures above threshold size (20 cm diameter at break for branch failures; any main stem ≥ 3 cm DBH for bole, root, and root crown failures) occurring between September 2000 and September 2004 among coast live oaks with (**Pr**) and without (**No Pr**) symptoms of *P. ramorum* canker at the time of failure.

Although many *P. ramorum*-infected coast live oaks experienced major failures before the top had died, most of the observed failures (60%) occurred in trees that were dead prior to failure. This percentage was based on 73 initial failures for which the trees' status prior to failure could be determined. Furthermore, the part that failed was dead prior to failure in 77% of these trees (Figure 9). The highest number of live stem failures occurred among trees with late *P. ramorum* canker symptoms, i.e., symptomatic trees colonized by secondary organisms including *H. thouarsianum* and/or bark or ambrosia beetles. Overall, among living trees with *P. ramorum* canker, more than half of the failures (11/20) occurred in live branches and stems (Figure 9). All of the failures in trees declining due to other factors and most of the failures in asymptomatic trees also occurred in live branches and stems (Figure 9).

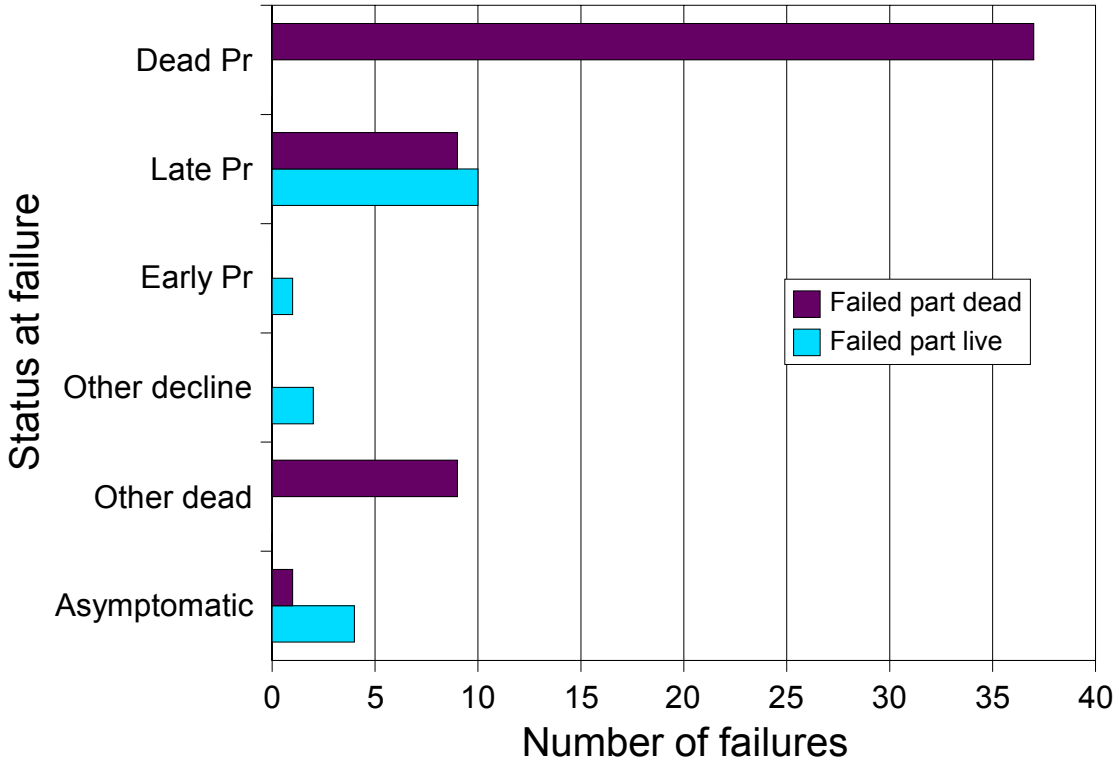

Figure 9. Status at the time of failure with respect to disease status and condition of the failed part (live or dead) for initial failures above threshold size occurring between September 2000 and September 2004 in coast live oak study trees.

For all trees in the study, we recorded data on the initial failure as well as subsequent failures above the size threshold that have occurred since September 2000. We did not score subsequent failures for a given main stem once it had failed at the lower bole, root crown, or roots. Trees that experienced multiple rated failures over this period mostly followed one of two general patterns:

(1) trees that initially had branch failures and subsequently had either additional branch failures or failed at the bole or root crown; this pattern was commonly seen in trees killed by *P. ramorum* canker; or

(2) multistemmed trees in which different main stems failed at different times. In multistemmed trees with a mixture of live and dead stems, the dead stems commonly failed first, but in some trees, live stems failed before dead stems.

Based on characteristics of the failed part (weathering and accumulation of detritus on broken surfaces, etc.), failure dates were estimated to the nearest six-month interval of the preceding year at each evaluation. As shown in Figure 10, the number of failures among coast live oaks increased dramatically over each 6 month interval in the first two years of the study. Almost all of the failures that occurred before September 2002 were initial failures. In the 6-month intervals after September 2002, decreasing numbers of failures, especially initial failures, have been observed (Figure 10).

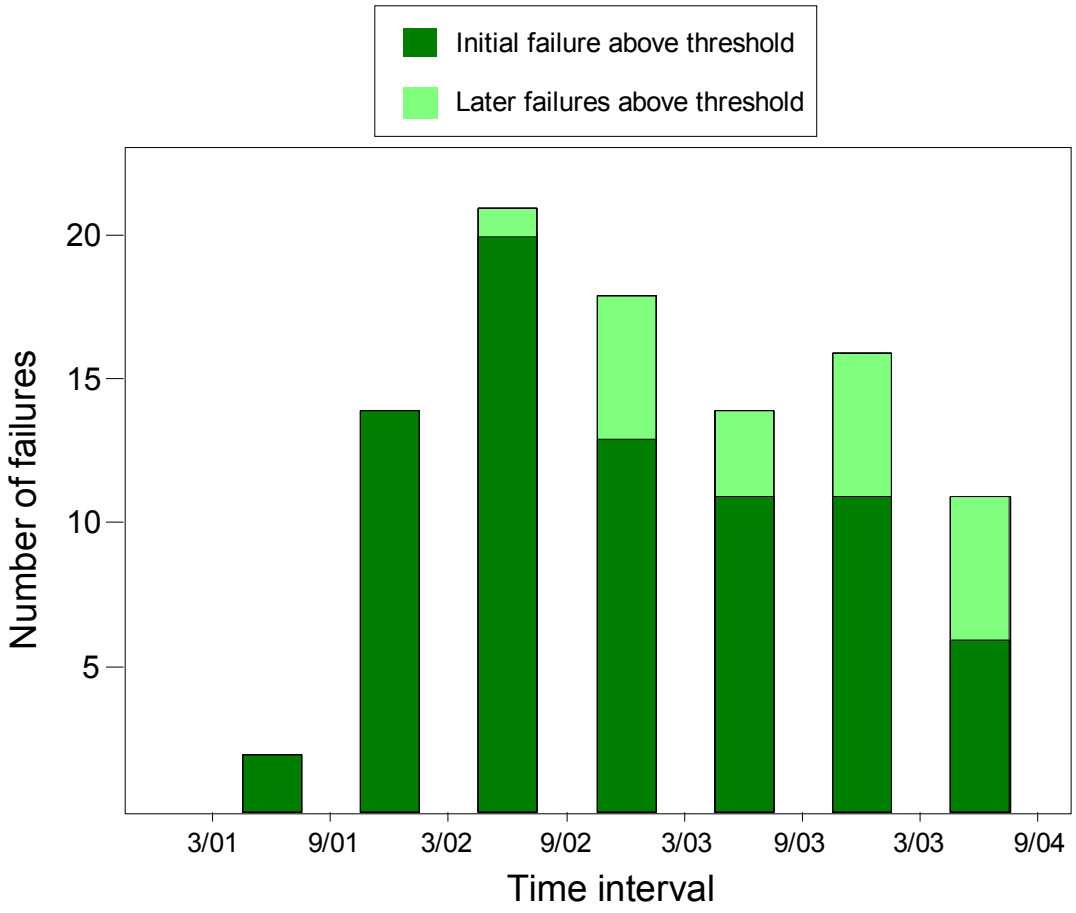

Figure 10. Number of initial (dark green) and subsequent (light green) failures above threshold size occurring between September 2000 and September 2004 among 629 coast live oaks that had not failed prior to September 2000.

Calculation of failure rates for trees in different disease classes was complicated by the fact that the disease status of many trees changed over the course of the study. In Figure 11, cumulative failure rates over time of coast live oak study trees are plotted based on the disease symptom class observed in 2000. In addition, the disease status of the failed trees at the time of failure is shown. The data exclude dead trees that had already failed by 2000.

Coast live oaks that had late *P. ramorum* symptoms or were dead due to *P. ramorum* canker in 2000 have failed at similarly high rates over the course of the study (Figure 11). However, about half of the trees that had late *P. ramorum* symptoms in 2000 were dead at the time they failed. Hence, failure rates among live trees with late *P. ramorum* symptoms were lower than among trees killed by *P. ramorum*.

When plots were established in 2000, we counted trees within plots that were estimated to have died within the previous 10 years. By 2004, the overall failure rate of trees killed by factors other than *P. ramorum* canker did not differ from the failure rate seen in trees killed by *P. ramorum* (Figure 11).

Trees that were asymptomatic in 2000 showed very low rates of failure over the study period (Figure 11). Compared with trees that were asymptomatic in 2000, failure rates were significantly higher among trees with only early *P. ramorum* canker symptoms in 2000

(likelihood ratio test p<0.0001) and trees rated as being in decline in 2000 (likelihood ratio test p=0.0005). Most of the trees from these two groups that failed had developed more severe symptoms (late *P. ramorum* canker or dead) prior to failure (Figure 11). Only one tree had early *P. ramorum* canker symptoms at the time of failure. In this tree, the *P. ramorum* canker was inactive and not associated with significant decay. However, drying of the outer sapwood in the area of the canker, combined with internal decay unrelated to *P. ramorum* canker and poor branch structure contributed to the failure of a large scaffold branch in this tree.

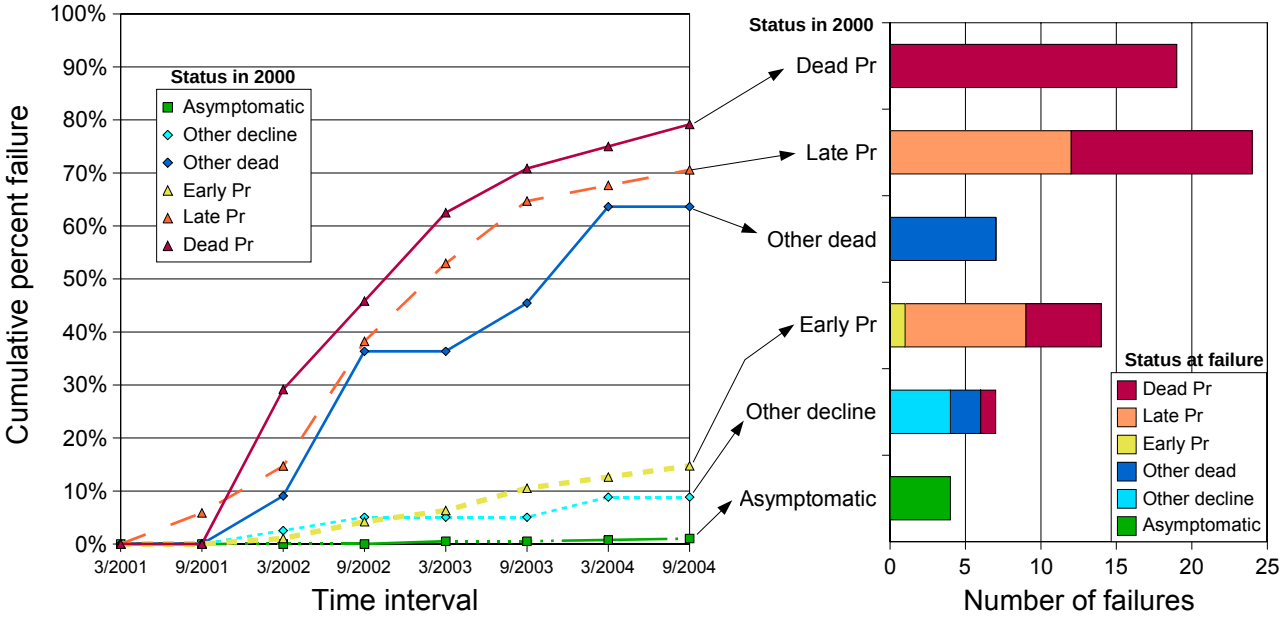

Figure 11. Cumulative failure rates (left) and tree disease status at failure (right) for coast live oaks by initial disease status in 2000. Only initial failures above the size threshold are shown. Failure dates were estimated to the nearest 6 month interval and failure percentages are plotted at the 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. Dead trees that had failed prior to September 2000 are excluded.

Stem water potentials of coast live oaks and tanoaks

In a repeated measures analysis of variance of stem water potential (SWP), the effects of year and species were highly significant (p<0.0001), but the interaction between these factors was not (p=0.071). Tanoaks consistently had higher average SWP readings than coast live oaks (Figure 12). Overall mean SWP fluctuated from year to year, apparently in response to the level of precipitation in the previous year (Figure 12). For coast live oak, the study location average SWP was positively, but not strongly, correlated with the rainfall for the location and year (adjusted R^2=0.080, p=0.026, n=10). Our rainfall data were from nearby weather stations rather than from the actual study sites, which may have weakened the apparent correlation. As seen in previous years (Swiecki and Bernhardt 2004), there was no significant effect of vapor pressure deficit on SWP.

Stem water potentials (SWP) measured in September of 2000, 2001, 2002, 2003, and 2004 were highly correlated from year to year for individual coast live oak and tanoak trees. A correlation matrix analysis showed that SWP readings from individual trees in different years were significantly correlated (p<0.0001) in all combinations, with adjusted R^2 values ranging

from 0.71 to 0.85. Although mean SWP varied from year to year, we found that the SWP of most trees shifted up or down by an amount that approximated the overall mean year to year difference. After removing the overall year to year differences, the SWP of individual trees were quite consistent from year to year. This suggests that relative SWP levels measured over the study period were likely to be similar to those that existed prior to the start of the study, i.e., during the period when most trees were initially infected.

To determine whether the SWP of the plot center tree (case or control) was representative of the water status of the plot as a whole, we compared SWP readings for 2001 through 2004 from plot center trees with the extra SWP trees present in 45 of the 150 plots. Readings between pairs of trees from the same plot (n=45) were significantly correlated in all four years (adjusted R^2 varied between 0.519 and 0.784 in the different years). This suggests that much of the variation in SWP was related to the available soil moisture level within the plot, which was influenced by local factors including soil type and depth, slope, aspect, and vegetative cover. Hence, SWP of any tree in a plot (center tree or a different tree) providee an indication of tree water stress levels within the plot as a whole.

Because year to year variations in SWP tend to obscure directional changes in SWP over time, we used repeated measures analysis of variance to test whether SWP was influenced by disease progress. For this analysis, we compared SWP of coast live oaks that were asymptomatic in 2000 through 2004 (n=107) to trees that had progressed from early to late *P. ramorum* symptoms between 2000 and 2004 (n=22). In this analysis, only the effect of time was significant ($p<0.0001$); neither disease status (asymptomatic vs. early to late) nor the interaction between disease status and time were significant. Similar results were obtained when all live trees with *P. ramorum* canker in 2004 (n=36) were compared with asymptomatic trees. These results indicate that year to year changes in SWP were not affected by disease status, i.e., SWP in trees with *P. ramorum* did not change directionally over time.

Although the year-adjusted SWP of most trees with *P. ramorum* canker did not change over time, a few trees did show SWP changes. In several coast live oaks with late *P. ramorum* symptoms that were in the process of turning brown due to stem girdling, SWP readings were generally at least 1 MPa lower than previous readings, a difference well in excess of the overall annual change. In contrast, SWP readings on root sprouts of trees killed by *P. ramorum* were typically similar to or higher than readings made on the same trees before death of the top. This indicates that at least some portion of the root system continued to function after the death of the canopy in these trees.

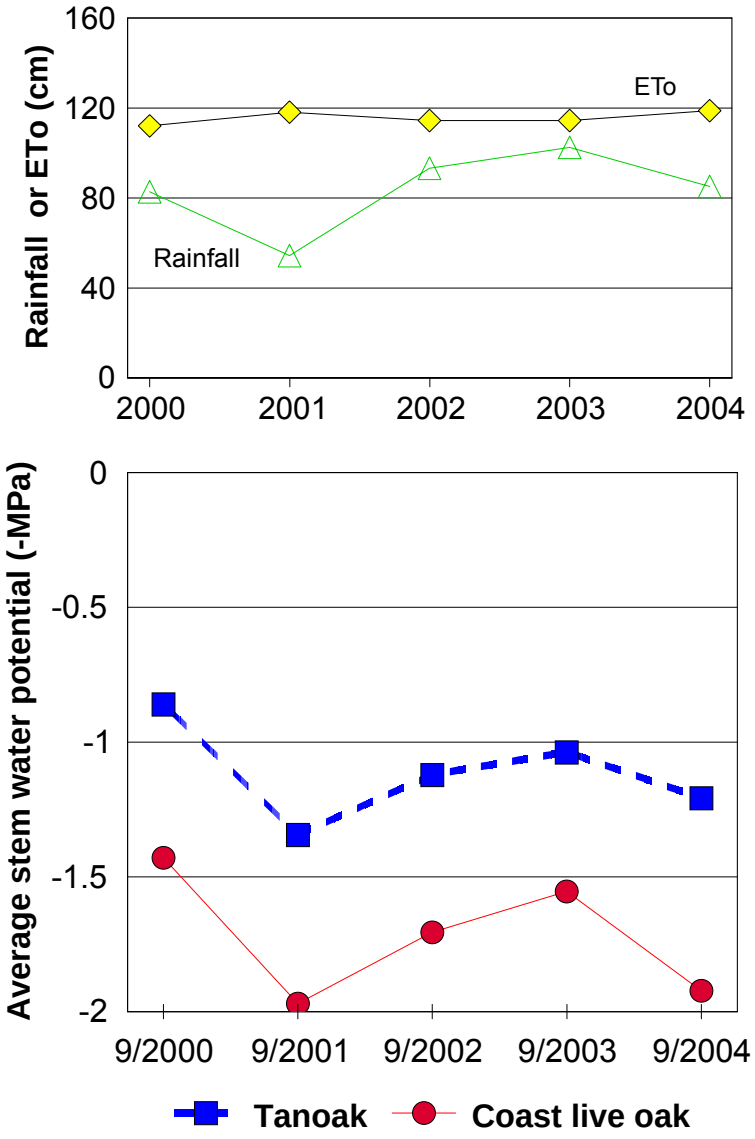

Figure 12. Average stem water potential for coast live oaks and tanoaks (bottom graph) across all locations and years compared with cumulative potential evapotranspiration (ETo) and average seasonal rainfall (top graph). ETo is for September of the previous year through August of the listed year; data are from the California Irrigation Management Information System (CIMIS) station in Santa Rosa. Rainfall is the mean for all study locations and was calculated from the nearest weather station data for each location.

Bark characteristics and disease

Bark thickness

Overall, the mean adjusted bark thickness of trees with *P. ramorum* canker symptoms in 2004 was significantly greater than that of the asymptomatic trees (t-test $p<0.0001$, Figure 13). Furthermore, mean bark thickness was greatest among trees that had already died and lowest among trees that still had only early symptoms of disease in 2004. Trees with late disease

symptoms in 2004 had intermediate bark thickness levels (Figure 13). Given that most of these trees were symptomatic in 2000, this suggests that symptoms progressed more rapidly in trees with thicker bark. Bark thickness was also positively correlated with other indicators of disease severity, including the number of bleeding cankers in 2001 (adjusted R^2=0.101, p=0.008) and the rating of the percentage of the bole girdled by *P. ramorum* cankers in 2001 (adjusted R^2=0.098, p=0.009).

Among all (symptomatic and asymptomatic) coast live oaks, bark thickness increased in a nonlinear fashion with increasing stem diameter (Figure 14), with an upper plateau near 5.5 cm. As seen in Figure 14, bark thickness can vary by 2 cm or more for trees with a given stem DBH. Bark thickness increased as the tree's sky exposed canopy rating increased (adjusted R^2=0.17, p<0.0001 for quadratic regression line), suggesting that bark thickness was generally greater in more dominant trees, which also tended to have large stem diameters. In addition, among coast live oaks without *P. ramorum* canker symptoms, trees that were severely declining due to other agents or factors had significantly thinner bark (mean 2.2 cm, n=23) than non-declining trees (mean 2.7 cm, n=113; t-test p<0.0001). Overall, these data suggest that bark thickness was generally greater in more vigorous, faster-growing coast live oaks. These dominant, fast-growing coast live oaks also had a higher risk of developing *P. ramorum* canker symptoms than suppressed, slow-growing oaks in the same stands.

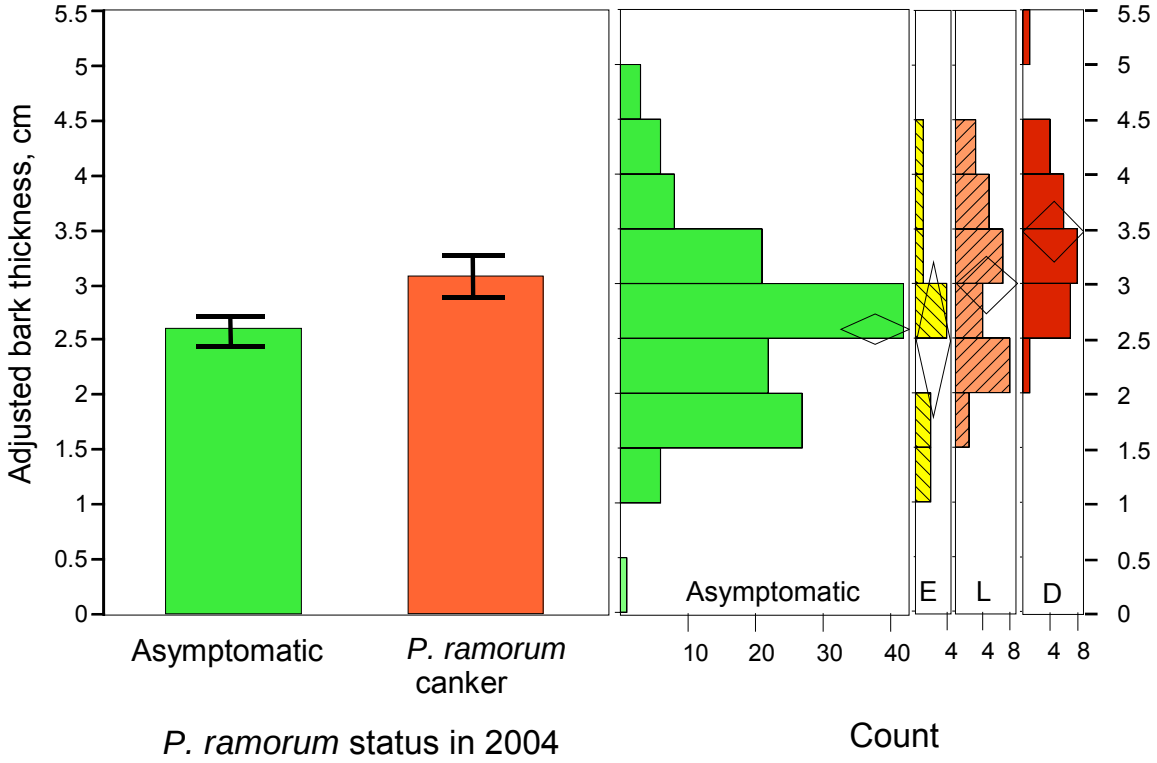

Figure 13. Relationship between coast live oak bark thickness and *P. ramorum* disease status in 2004. Bark thickness readings in dead bark samples were adjusted to account for shrinkage due to drying. Error bars (left graph) and outer points of means diamonds (histograms, right) denote 95% confidence intervals of the means. *P. ramorum* symptoms in histograms are **E**=early (bleeding cankers only), **L**=late (cankers + beetles and/or *H. thouarsianum*), **D**=dead; n=203.

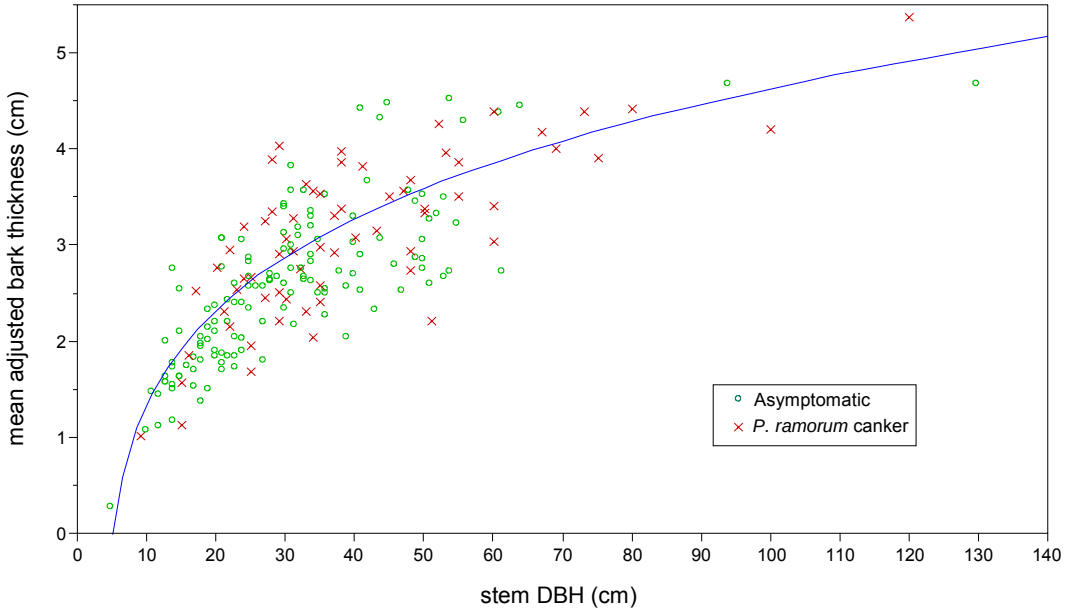

Figure 14. Relationship between bark thickness at 1 m height and stem diameter (at 1.37 m height, DBH) for coast live oaks with or without symptoms of *P. ramorum* canker. Fitted curve is $e^{(\text{adjusted bark thickness})} = -1.52 + 0.471(\text{DBH}) + 0.00573(\text{DBH})^2$; adjusted R^2=0.62, p<0.0001, n=202.

Bark surface characteristics

In the 2003 survey we found that most bark characteristics, including the abundance and location of epiphytic lichens and mosses, various bark morphologies (striate, checkered, smooth, furrowed, irregular), and the presence of bark fissures of various depths occurred at nearly equal frequencies in symptomatic and asymptomatic trees and were thus not predictors of disease status (Swiecki and Bernhardt 2004). However, one bark characteristic was a significant predictor of the case (disease) outcome in both single variable and multivariate models. This was the presence of non-weathered, brown bark in the center of furrows or fissures that resulted from recent bark expansion (Figure 15).

To explore this factor further, in the 2004 survey we estimated the proportion of bark fissures on the lower bole of plot center trees that contained unweathered brown bark using the 0-6 scale. This quantitative brown fissures variable was a significant predictor of the case outcome in both single and multivariate models, as noted below. However, brown fissures were not significantly correlated with variables related to disease progress, including the amount of *P. ramorum* girdling and progress in disease symptoms between 2000 and 2004. This suggests that the presence of brown fissures may be related to risk of infection, but not to later canker expansion.

Average brown fissure ratings were significantly lower among severely declining trees without *P. ramorum* symptoms (mean 0.30, n=20) than among relatively healthy (non-declining) asymptomatic trees (mean 2.2, n=96; t-test p<0.0001). Brown fissure ratings also declined as decay impact ratings increased. Furthermore, brown fissure rating was positively correlated with sky exposed canopy rating (adjusted R^2=0.31, p<0.0001, n=123), and negatively correlated with plot canopy cover (adjusted R^2=0.12, p<0.0001, n=123). These correlations suggest that brown fissures are more common in trees that are likely to have at least moderate growth rates. However, brown fissure ratings were not correlated with either stem diameter or bark thickness.

Hence, although brown fissures and bark thickness may both be related to tree vigor or growth rate, they appear to reflect different ways that bark morphology can be affected by growing conditions.

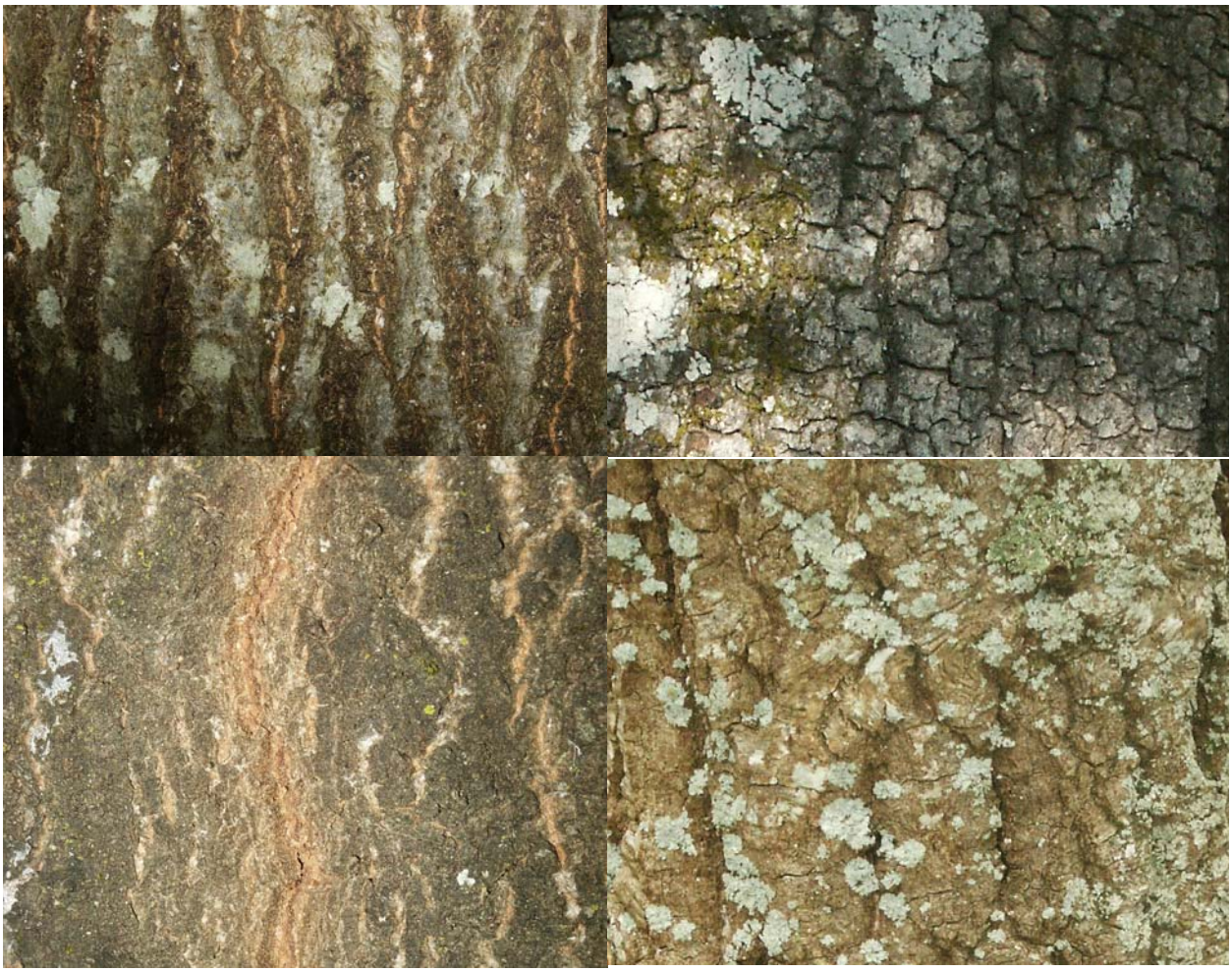

Figure 15. Images of coast live oak bark on the left have unweathered brown bark within bark fissures, presumably resulting from recent bark expansion. Unweathered brown areas are lacking in bark fissures shown on the right.

Disease risk models for coast live oak

We used recursive partition models and logistic regression to model the risk of *P. ramorum* infection in plot center trees. The outcome variable for these analyses included all plot center trees that had expressed likely *P. ramorum* canker symptoms through September 2004, including some initially symptomatic trees in which disease had apparently become inactive. Including these non-progressing trees maximized our ability to detect factors that may be related to infection but not later disease progress. For these models, tree and plot variables that could be considered outcomes related to disease were not tested as explanatory variables. For instance, diffuse canopy dieback was not included as a predictor because our data (above) suggest that it was a consequence of *P. ramorum* canker.

We constructed recursive partition disease risk models using both plot center trees only (n=128) and a larger data set that included extra SWP trees (n=168). The two initial splits were the same for both data sets, but subsequent splits varied between the data sets. In both models, the best initial split of the data was based on the brown fissure rating (0 and 1 vs. ≥2). About 90% of the trees with brown fissure ratings of 1 or less were controls. Within the group with brown fissure ratings of 2 or more, the next split for both models was based on 2002 SWP. About 80% of the trees with 2002 SWP<-2 MPa (relatively high water stress) were controls. Other splits in the models were based on further division by 2002 SWP (disease risk higher with high SWP/low water stress), the number of overstory and understory California bay in the plot (disease risk higher with more bay), sky exposed canopy (disease risk higher with greater canopy exposure), and bark thickness (disease risk higher with thicker bark).

We also developed logistic regression models for the case outcome using only the plot center tree data set. Two optimized models with similar fit based on AIC are shown in Table 6. The two models differ only in that the SWP variable was replaced by a binary bark thickness variable (bark thickness>3.3 cm). The optimum split for the binary bark thickness variable was determined from recursive partition models. Bark thickness was not significantly correlated with SWP overall. However, SWP was somewhat higher (t-test p=0.08) in trees with bark thickness>3.3 cm than in trees with thinner bark. SWP readings from all years could be substituted for each other in the model, with 2002 SWP being the most significant. We used 2001 SWP in the model because it was the earliest year that SWP was measured in both plot center and extra SWP trees.

Table 6. Logistic regression models for *P. ramorum* canker disease risk in coast live oak

	Model 1[1]		Model 2[1]	
Predictor variables	P level[2]	Odds ratio (CI)[3]	P level[2]	Odds ratio (CI)[3]
Brown bark fissures rating (0-6 scale)	<0.0001	53.28 (10.01-363.68)	<0.0001	64.27 (10.85-531.30)
California bay cover in plot (0-6 scale)	0.0285	8.21 (1.25-61.11)	0.0009	21.06 (3.35-162.43)
More than 2 stems	0.0029	8.27 (1.98-46.72)	0.0028	9.11 (2.06-53.46)
2001 SWP (MPa)	0.0372	19.28 (1.88-396.59)		
Bark thickness>3.3 cm			0.0128	3.87 (1.33-12.33)

[1] The Akaike Information Criterion (AIC) for models 1 and 2 are 123.56 and 127.76 respectively. Overall significance levels of for both models were p<0.0001

[2] Likelihood ratio test significance level

[3] Odds ratios and 95% confidence intervals; odds ratios greater than 1 indicate that a factor is positively associated with the case (disease) outcome

Predictors in the model included only one plot variable, California bay cover, which is related to local inoculum density. Disease risk increased as bay cover in the plot increased, presumably because the amount of inoculum present in the plot also increased with bay cover. Other predictors are tree variables that may be related to host susceptibility. SWP can be considered both a tree and plot factor because SWP levels within a plot were correlated as noted above. Wetter conditions within plots (indicated by higher SWP levels) may favor host susceptibility, inoculum production, and the duration of suitable infection periods.

To validate the model, we used the model to predict the disease outcome in the extra SWP trees, which were not used to develop the models. For this smaller data set (8 cases, 32 controls),

model 1 correctly predicted 72% of the outcomes and model 2 correctly predicted 69% of the outcomes. The case outcome was strongly underpredicted. However, the bay cover variable was based on cover in the 8 m plot around the plot center tree. Actual bay cover in an 8 m radius plot around the extra SWP trees could differ somewhat from this value, possibly contributing to poorer prediction of the case outcome.

Disease progress models for coast live oak

We also developed models for coast live oak to determine whether any of the factors we rated were related to disease progress or resistance in trees that were initially infected (Table 7). This data set includes 58 coast live oaks (plot center and extra SWP trees) which were alive and had *P. ramorum* canker symptoms in 2000. Of these, 42 showed disease progress, as shown by canker expansion and/or progression to a later disease stage (e.g., early to late or dead).

Indicators of initial disease severity, including canker count in 2001 and *P. ramorum* canker girdling rating in 2000 or 2001 are highly significant predictors of disease progress (likelihood ratio $p<0.0001$; AIC 37.4, 46.5, 44.7, respectively). Canker count was a somewhat better predictor of disease progress than canker girdling rating. Trees with greater numbers of cankers and/or higher girdling rating in 2000 or 2001 were more likely to show disease progress than trees that initially had few and/or small cankers. The mean canker rating in 2000 was significantly higher (t-test $p=0.031$) for those that showed disease progress than for those that did not (mean ratings 3.0 and 2.1, respectively). However, because almost all of these trees were symptomatic at the start of the study, the presence of larger and more cankers in 2000 and 2001 can also be interpreted to be the result of disease progress up to that point. Thus, although canker severity at a given point in time may be a good indicator of future disease progress, these variables tell us little about factors that might predispose trees to greater disease progress.

The best multivariate model that excludes the initial disease severity variables is shown in Table 7. When initial disease severity variables are excluded, bark thickness is the best single predictor of the disease progress outcome (likelihood ratio $p<0.0075$, AIC 64.5). As shown in Figure 13, this factor was positively correlated with disease risk and the disease stage in 2004, which was a component of the disease progress outcome.

The other factors in the model are more difficult to interpret. Disease was more likely to progress in trees located in plots with no or low levels of madrone cover. Disease progress occurred in 88% of the trees in plots without any madrone cover but only occurred in 52% of the trees in plots with madrone cover. Furthermore, the likelihood of disease progress decreased as madrone cover increased. However, plots with the highest levels of madrone cover (>20%) were only found at 3 of the 10 coast live oak study location (5, 6, and 7), so this variable may be partially confounded with other location-specific factors. Within the study areas, madrone tends to occur on soils that appear to be more shallow and are often more rocky than soils that are dominated by other tree species. This may indicate that coast live oaks growing in more unfavorable sites are less likely to show disease progress. However, SWP levels did not vary with madrone cover in these plots, and SWP was not a significant predictor of disease progress. The third factor in the model, poison oak cover at least 2.5%, was not significant in a single variable model. In the multivariate model, the higher level of poison oak cover was positively correlated with disease progress.

Table 7. Logistic regression model for *P. ramorum* disease progress in coast live oaks[1]

Predictor variables	P level[2]	Odds ratio (CI)[3]
Bark thickness	0.0457	17.97 (1.054-450.6)
Madrone cover	0.0236	0.051 (0.00304-0.671)
Poison oak cover >2.5%	0.0488	7.878 (1.010-184.9)

[1]Overall significance level for the models is p<0.0021, AIC=61

[2] Likelihood ratio test significance level

[3] Odds ratios and 95% confidence intervals; odds ratios greater than 1 indicate that a factor is positively associated with the case (disease progress) outcome

DISCUSSION

Over the course of the study, levels of disease and mortality due to *P. ramorum* have been consistently higher in tanoak than in coast live oak. Compared with tanoak, coast live oak had lower initial levels of disease observed in 2000, fewer trees that developed new symptoms between 2000 and 2004, and more trees that became asymptomatic after showing some early disease symptoms. These results support the overall consensus that tanoak is more susceptible to *P. ramorum* canker than coast live oak. In addition, our data suggest that conditions favorable for new bole infections occur more regularly in the tanoak study sites than in the coast live oak sites. Although apparent bole infection rates in tanoak have increased fairly steadily throughout the study period, most of the apparently new coast live oak bole cankers developed between the 2002 and 2003 ratings. This interval had the first relatively wet spring of the study period. Davidson and others (2005) found that *P. ramorum* inoculum levels in rainwater sampled in a coast live oak woodland in Sonoma County were greatly elevated in spring 2003 relative to the two previous years.

After 5 years of observation, we have been able to document several different symptom progression patterns in trees with *P. ramorum* canker. The "sudden oak death" pattern presented in the original descriptions of the disease (Garbelotto and others 2001, Rizzo and others 2002) involves fairly rapid canker expansion followed by a rapid dieback of the entire canopy of affected stems. Presumably many of the trees that were dead at the time of the initial evaluation in 2000 had followed this pattern. Since that time, up to about 81% of tanoaks and about 62% of coast live oaks that have died have followed this rapid decline pattern. However, especially in coast live oak, cankers caused by *P. ramorum* are frequently smaller and less aggressive. Trees with less aggressive cankers follow different disease patterns, including apparent symptom remission in some trees.

Most tanoaks (65%) and coast live oaks (74%) with *P. ramorum* canker symptoms in 2000 were still alive in 2004. In coast live oak, trees with extensive late-stage cankers commonly developed diffuse canopy dieback (Figure 7, Swiecki and Bernhardt 2002c, 2004) that can eventually lead to canopy thinning. Some slow-declining tanoaks and coast live oaks have developed healthy callus tissue around the margins of old *P. ramorum* cankers that did not appear to be expanding (e.g. cover photos, Figure 6). Callus development is evidence of a host resistance response, but it was not clear whether callus development observed actually limited canker expansion or whether it simply represented the normal host wound closure response acting in areas where pathogen activity had ceased.

In addition, cankers in some coast live oaks and tanoaks have not expanded or oozed in several successive years and appeared to be inactive. In the most extreme case of arrested disease progress, *P. ramorum* canker symptoms appeared to have gone into remission in at least

16 coast live oaks. Because *P. ramorum* canker was not confirmed by culturing all symptomatic trees at the start of the study, it is possible that some of these trees were not actually infected and had bleeding due to other causes. However, disease failed to progress in some trees in which *P. ramorum* had been confirmed. Furthermore, the strong correlation between disease progress and initial disease severity suggests that disease progress was commonly limited in trees that had only a few small cankers. In addition, *P. ramorum* was typically difficult to recover from cankers in the latter part of the dry season of the year, and from older, relatively inactive cankers (D. Rizzo, personal communication). This suggests that the activity of *P. ramorum* varies over time within infected hosts. If *P. ramorum* cankers in coast live oak are limited at an early stage of development due to host resistance and/or low initial levels of infection, affected trees may be able to recover more or less completely.

Trees that developed late *P. ramorum* canker symptoms or were killed by *P. ramorum* had a much greater risk of failure compared with asymptomatic trees, trees with only early *P. ramorum* canker symptoms, or trees declining due to other factors (Figure 11). Elevated risk of failure in trees with *P. ramorum* canker was associated with the degradation of wood by secondary colonizers such as *H. thouarsianum* and other decay fungi. Trees with only *P. ramorum* cankers (i.e., early symptoms) did not have an elevated risk of failure. These findings are consistent with those from a retrospective study of tree failures that we conducted in 2002 (Swiecki and Bernhardt 2003b, Swiecki and others in press).

The number of failures occurring per 6-month interval peaked in late 2002 (Figure 10), which corresponds to the time when the percentage of trees in the late and dead *P. ramorum* canker classes began to level off sharply among coast live oak study trees (Figure 2, right). The decline in the number of initial failures seen over time after September 2002 was largely due to fact that many trees that had a high risk of failing had already failed, which reduced the population of trees at risk of failing. In appears that the coast live oak study areas, a cohort of trees was infected by *P. ramorum* prior to 2000. Many of the trees in this cohort have failed as symptoms have progressed to the late disease stages, which involve secondary colonization of trees by wood-degrading organisms and tree mortality. However, unless an additional pulse of disease affects a large portion of the remaining trees, the number of initial failures occurring over time is likely to decrease. Given that many of the symptomatic trees in the study were probably infected prior to 2000, the data show that a lag of 2 years or more may exist between the initiation of a *P. ramorum* epidemic in coast live oak and a subsequent increase in the rate of tree failure. As this study continues, we plan to use survival models to estimate the time interval between the onset of specific symptoms and tree failure.

Given that *P. ramorum*-infected trees have consistently had higher average SWP levels (i.e., lower water stress) than asymptomatic trees, we conclude that water stress is not a significant predisposing factor for the development of *P. ramorum* canker in coast live oak. To the contrary, trees with chronically high levels of water stress in September have a lower risk of developing *P. ramorum* canker. *P. ramorum* canker occurred more commonly in trees that have both relatively low levels of water stress and high canopy exposure (Swiecki and Bernhardt 2001, 2002abc). Because high canopy exposure is associated with high levels of transpiration, the low levels of water stress seen in these trees indicates that they did not have any significant impairment of root function prior to infection.

Furthermore, in coast live oaks with *P. ramorum* canker, the decline of the tree's canopy appears to be driven primarily by the girdling of the bole rather than by loss of root function. Symptomatic trees show no decrease in SWP and sometimes develop higher SWP levels over

time despite increasing symptom severity. If decay or dysfunction of the root system was the primary cause of top dieback, lower SWP readings should have been common in symptomatic trees, rather than being limited to the few trees that are in the final stages of collapse due to extensive sapwood decay.

Many *P. ramorum*-killed coast live oaks and tanoaks produced vigorous basal sprouts or epicormic sprouts on the lower portions of failed stems that survived for several years after loss of the tree canopy. This indicates that at least some portion of the root system survived in these trees. In addition, disease progressed independently among different stems in multistemmed trees (Figure 5, Table 4). This provides further evidence that decline of *P. ramorum*-infected trees was directly related to the number and extent of stem cankers, and was not primarily related to root disease. Although root pathogens, e.g., *Armillaria mellea,* may eventually affect some trees with late *P. ramorum* canker symptoms, we saw very little evidence of root disease among any of the trees in this study. Hence, several independent lines of evidence indicated that decline and death of trees with *P. ramorum* canker symptoms was not associated with pre-existing root disease or overall tree decline related to root dysfunction, but was a direct consequence of girdling by *P. ramorum* cankers.

Despite its relatively large host range, *P. ramorum* causes bark cankers on the main stems of only a few species under field conditions. This suggests that tree species that develop main stem cankers have relatively unique bark characteristics that permit *P. ramorum* to infect and colonize living bark tissues on mature main stems. For coast live oak, bark thickness and unweathered brown tissue within bark fissures were the only bark characteristics we rated that were positively correlated with *P. ramorum* disease risk. The unweathered bark appears to represent relatively rapidly expanding regions of the outer bark. Slow growing trees, including highly suppressed understory trees and declining trees, had very few or no fissures with unweathered bark, and were also at low risk for developing *P. ramorum* canker (Swiecki and Bernhardt 2001, 2002abc).

Bark expansion zones may represent sites that are more easily breached by *P. ramorum* zoospores. It is also possible that the outer periderm in these areas may be thin enough that substances might diffuse from these areas when the bark surface is wet. If *P. ramorum* zoospores were chemotactically attracted to these zones, greater aggregation of zoospores cysts might occur in these areas, leading to a greater likelihood of successful infection. Bark fissures may also remain wet longer than other areas of the bark, increasing the chance of successful infection. These areas of the bark deserve further attention in studies of the infection process in coast live oak. The correspondence between brown fissures and high-moisture bark channels imaged by MRI (Florance, in press) should also be investigated.

Bark thickness was the first variable we have measured in coast live oaks that appeared to be correlated with both disease risk (Table 6) and disease progress (Table 7, Figure 13). Coast live oak bark is relatively thick and consists primarily of living tissue, with only a thin (a few mm) outer layer of dead periderm (rhytiderm) in most trees. Trees with thicker bark appeared to be more likely to become infected by *P. ramorum* and, once infected, disease appeared to progress faster in trees with thicker bark. These findings are consistent with field observations that *P. ramorum* cankers do not naturally occur in either small diameter coast live oaks or in the smaller-diameter upper stems of coast live oak.

Several lines of evidence from this study indicate that *P. ramorum* canker is most likely to affect relatively vigorous or fast-growing coast live oaks, and is not primarily a disease of stressed trees. As we have previously reported, coast live oaks with *P. ramorum* canker tended to have more dominant canopy positions (higher levels of sky-exposed canopy) and larger stem

diameter, and were less likely to be declining due to other agents or factors including canker rot and overtopping (Swiecki and Bernhardt 2001, 2002abc). In addition, *P. ramorum*-infected trees had consistently had higher average SWP levels (i.e., lower water stress) than asymptomatic trees. Increased bark thickness and the presence of unweathered bark in bark fissures were additional variables that correlated both with faster tree growth and greater risk of *P. ramorum* canker. We previously examined a sample of increment cores in trees with and without *P. ramorum* canker to investigate recent growth rates more directly, but several technical issues made this approach impractical (Swiecki and Bernhardt 2003a).

Because many of the factors that were related to faster growth were correlated with each other, it is difficult to determine which, if any, actually play important roles in affecting disease risk. For example, dominant and /or larger trees may intercept more inoculum, bark expansion zones may constitute favorable infection courts, and thicker bark may provide a better substrate for *P. ramorum* growth. However, additional investigations are necessary to determine whether these and/or other factors correlated with tree growth rate actually contribute to disease risk.

Although we have identified a number of variables that can serve as predictors of disease risk in this study (Table 6, Swiecki and Bernhardt 2001, 2002abc), we have found very few good predictors of disease progress in infected trees. This is in part due to the fact that the data set for disease progress analyses is much smaller than the disease risk data set. Overall, initial disease levels observed in 2000 or 2001 were the best predictors of later disease progress. Although these predictors are not completely distinct from the outcome, assessments of disease severity may be useful for predicting the likely survival of trees for management purposes.

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