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# Responses of oaks and tanoaks to the sudden oak death pathogen after 8 y of monitoring in two coastal California forests

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ABSTRACT

Sudden oak death, caused by *Phytophthora ramorum*, is widely established in mesic forests of coastal central and northern California. In 2000, we placed 18 plots in two Marin County sites to monitor disease progression in coast live oaks (*Quercus agrifolia*), California black oaks (*Q. kelloggii*), and tanoaks (*Lithocarpus densiflorus*), the species that are most consistently killed by the pathogen in these areas. Through early 2008, the numbers of newly infected trees increased for all species. The infection rate for trees that were asymptomatic in 2000 was 5.0% $y^{-1}$ for coast live oaks, 4.1% $y^{-1}$ for black oaks and 10.0% $y^{-1}$ for tanoaks. Mortality rates were 3.1% $y^{-1}$ for coast live oaks, 2.4% $y^{-1}$ for black oaks, and 5.4% $y^{-1}$ for tanoaks. Mortality not attributed to *P. ramorum* was 0.54% $y^{-1}$ for coast live oaks, and 0.75% $y^{-1}$ for tanoaks. Weibull survival models of trees that were asymptomatic in 2000 provided overall median survival times of 13.7 y for coast live oaks, 13.8 y for black oaks, and 8.8 y for tanoaks. Survival of infected (bleeding) trees declined to 9.7 y for coast live oaks, 6.2 y for black oaks, and 5.8 y for tanoaks. Ambrosia beetle attacks on bleeding trees further reduced modeled survival times by 65–80%, reaffirming the earlier finding that beetle attacks on bleeding cankers considerably reduce survival. Across all plots, the modeled time for 90% of trees that were asymptomatic in 2000 to become infected is 36.5 y for coast live oaks and 15.4 y for tanoaks. There was a trend toward higher infection rates as tree diameter increased. Greater than 90% of living coast live oaks that failed during the study had extensive beetle tunneling at the site of the break. Disease intensity in coast live oaks at the plot level was positively associated with bay laurel (*Umbellularia californica*) basal area and negatively associated with Pacific madrone (*Arbutus menziesii*) basal area. This study demonstrates the use of survival modeling to characterize the effects of epidemic disease on different species and to project the future of forests infected with tree pathogens.



## 1. Introduction

Sudden oak death (SOD) continues to infect and kill oaks and tanoaks in coastal central and northern California. The first reports of dying tanoaks (*Lithocarpus densiflorus* Hook. & Arn.) in 1994 and coast live oaks (*Quercus agrifolia* Nee) in 1995, were from Marin County (Svihra, 1999a,b). Within several years, SOD was found within the Coast Ranges, from Sonoma County, north of San Francisco, and south to Santa Cruz and Monterey Counties. The known geographic range of infested forests and woodlands has not increased much since the start of intensive research on SOD in 2000. What has increased is infilling, in which increasing disease severity has been observed in numerous forest stands within infested areas that had little or no previously reported infections. Local patchiness among infected stands is characteristic of SOD, although its causes are not well understood (Kelly and Meentemeyer, 2002; Meentemeyer et al., 2004; Condeso and Meentemeyer, 2007). Recognized constraints on the spread of the pathogen include habitat type (Condeso and Meentemeyer, 2007), host distribution (Maloney et al., 2005; Rizzo et al., 2005), and climatic conditions that appear to prevent further expansion beyond mesic forests and woodlands (Davidson et al., 2005).

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Despite the wide host range of *Phytophthora ramorum*, presently understood to include most of the native woody plants found within coastal California forests (Rizzo and Garbelotto, 2003), only four tree species are consistently killed by this pathogen. Coast live oak, California black oak (*Q. kelloggii* Newberry), and Shreve oak (*Q. parvula* Greene var. shrevei (C.H. Muller)), all in the red oak group (Sect. *Lobatae*), and tanoak, are frequently the dominant overstory trees in these forests. Other non-host oaks found in these forests, including valley oak (*Q. lobata* Nee), blue oak (*Q. douglasii* Hook. & Arn.), and Oregon oak (*Q. garryana* Dougl. ex Hook.), generally constitute lower proportions of the overstory trees. As the *P. ramorum* epidemic continues, the effects are likely to ramify throughout the impacted forests and to have consequences for the numerous biota with which they are associated. This is particularly significant because mixed evergreen forests and oak woodlands provide some of the most ecologically diverse habitats in California (Pavlik et al., 1991).

We established disease progression plots in Marin County in March 2000 to study the impact of *P. ramorum* on coast live oaks, California black oaks, and tanoaks (McPherson et al., 2000). In a previous paper, we used both Weibull survival regression models and Cox proportional hazards (PH) models to estimate survival of same-symptom cohorts of coast live oaks and tanoaks (McPherson et al., 2005). These analyses produced disease stage-specific median survival estimates for a 3-y period for cohorts of coast live oaks and tanoaks that were categorized by their disease stage in March 2000. The modeled median survival for infected coast live oaks and tanoaks was 7–8 y, but was reduced to less than 3 y if ambrosia and bark beetles had attacked the cankers on these trees. The accuracy of these model predictions was constrained by uncertainty about the age of the trees, the length of time that trees had been symptomatic, and the limited time frame of the study period. There were too few black oaks for analyses after 3 y. This was the first use of disease-stage-specific models to evaluate the effects of an introduced tree pathogen in a wildland setting.

This paper reports on 5 additional years of semiannual observations on 18 disease progression plots. We compare survival models based on 3 and 8 y observations on the same coast live oak, California black oak, and tanoak populations and develop more robust survival estimates by analyzing only those trees that were asymptomatic in March 2000. For coast live oaks and tanoaks, we project the impact of the disease on host populations into future years. We evaluate the relationship between coast live oak infection levels in 2000 and 2008 and the stand basal area of two abundant co-occurring woody species that may serve as reservoirs for *P. ramorum*, bay laurel (*Umbellularia californica* Hook & Arn. Nutt.) and madrone (*Arbutus menziesii* Pursh). We also analyze the effect of *P. ramorum* infection on the structural failure of living coast live oaks.

## 2. Materials and methods

### 2.1. Study sites

In March 2000, disease progression plots in two protected watersheds were selected to include a range of tree species and habitat types that represented the local flora (McPherson et al., 2005). Plot area varied between 320 $m^2$ and 3600 $m^2$, with a mean of 1234 $m^2$ (SE = 199 $m^2$). Ten plots were placed in each of two sites, China Camp State Park (CCSP), latitude = 37.5721, longitude = −122.3636, and Marin Municipal Water District land (MMWD), latitude = 38.00059, longitude = −122.48514, in Marin County, California. The host species of interest in CCSP were coast live oaks and black oaks. Seven of the MMWD plots included these oak species and four included tanoaks. Two tanoak plots in MMWD that burned in 2003 were excluded from the analyses.

### 2.2. Symptom evaluation

Each host tree stem ≥5 cm dbh (diameter at breast height, approximately 1.3-m above ground), including those on multiple stem trees, was treated as a separate tree. Bleeding cankers appear to develop independently on each stem of such trees. Host tree symptoms were determined using the protocols of McPherson et al. (2000, 2005). Trees were assigned ordinal ratings of asymptomatic, bleeding only, bleeding + beetle attacks, and dead (stem breakage was noted for these trees). Once a tree was categorized as beetle-attacked, it was no longer classified as bleeding only. However, bleeding trees may shift to asymptomatic, and back to bleeding (Swiecki and Bernhardt, 2006). Analyses based on sequential evaluation of disease stages are reported either in biannual or annual increments.

### 2.3. Trends in tree health, 2000–2008

The status of the trees in all study plots was evaluated biannually from March 2000 to March 2008. Live trees were categorized as asymptomatic or bleeding, and dead trees were further categorized either as symptomatic or asymptomatic (without any evidence of *P. ramorum* infection) at time of death. With the exception of those that were crushed by falling trees, the cause of death was not known for non-bleeding trees.

The infection rate was determined for each species by dividing the total number of initially asymptomatic trees that developed bleeding by March 2008 by the number of asymptomatic trees in March 2000. Disease intensity, the combined total of trees of a species per plot that were symptomatic and those that died of *P. ramorum* infection, divided by the total number of standing trees in 2000, was calculated for coast live oaks and tanoaks for 2000 and 2008.

For coast live oaks and tanoaks, we modeled the decreasing numbers of asymptomatic trees as a result of the disease, using the over-dispersed Poisson regression for the family of Generalized Linear Models (McCulloch and Searle, 2005). The parameters were estimated using the SAS GENMOD procedure (SAS 9.1.3, Cary, NC, USA). A regression line was fitted using the log-transformed number of asymptomatic trees from each plot with time as the independent variable, in 6-month increments from March 2000 to March 2008, with plot nested in site as a random effect to account for the overdispersion due to the repeated measurements. Coast live oak populations from the two sites were regressed separately. The maximum likelihood estimation method was applied to the fitted count regressions to project the time to 50% and 90% decreases in the total mean numbers of asymptomatic trees.

### 2.4. Relationship between stem diameter (dbh), infection, and mortality

The relationship between dbh, recorded in March 2000, and cumulative new infections, through March 2008, was tested for the three species by regressing dbh, categorized by size class quantiles, against the average percentage of newly symptomatic trees in a repeated measures analysis. The relationship between dbh and cumulative mortality in coast live oaks and tanoaks was analyzed by regressing the survival status of trees in these quantiles against percentage mortality in 2000, 2004, and 2008.

### 2.5. Survival analysis

The response of the three species to *P. ramorum* infections was modeled using survival analysis. For each species, we (1) modeled the overall survival of all trees in the plots that were asymptomatic in March 2000, and (2) assessed changes in cohorts of

initially asymptomatic trees defined by the discrete disease stages that develop following infection with *P. ramorum*. The goal of this approach was to estimate expected survival for each species based on the disease stages described earlier and to determine if 3 y (McPherson et al., 2005) is sufficient to provide useful survival estimates.

Both the semi-parametric Cox proportional hazards (Cox PH) and parametric Weibull models (Lee and Wang, 2003) were used to generate survival curves, from which we estimated time to median survival for each disease stage, the point at which 50% of the initial population remained. We estimated model parameters using the program LIFEREG for the Weibull models and PH REG for the Cox PH models (SAS 9.1.3, Cary, NC, USA). Possible explanatory variables that were included in the regression models were disease stage, dbh in 2000 (as a proxy for tree age), site (CCSP or MMWD), and plot. First, we analyzed the survival of the trees that were asymptomatic in March 2000 to get the overall survival for each species. Once a tree was characterized as bleeding, it was included in the bleeding cohort. If beetles subsequently attacked the same tree, it entered the bleeding plus beetles cohort. Over time, the same tree could therefore be analyzed separately in three cohorts, asymptomatic, bleeding, and bleeding plus beetles. Because coast live oaks and black oaks are found in both CCSP and MMWD, the variable SITE was used as a fixed factor in these analyses. Where plot or dbh were found to be significant, they were included in analyses as random variables.

### 2.6. Influence of co-occurring tree species on coast live oak disease intensity

We examined the possible influence of stand basal area of bay laurel and madrone on coast live oak disease intensity, by plot. In summer 2001, a 0.04 ha circular subplot was placed in the approximate center of each disease progression plot. In each subplot, we recorded the species, dbh, and status (alive or dead) of every woody stem ≥5 cm dbh. The evaluation was repeated in summer 2007. Data were not analyzed for one subplot that was erroneously sited. Both sites were pooled for this analysis. The relationship between bay laurel and madrone stand basal area and log-transformed coast live oak disease intensity was analyzed for 2001 and 2007 by repeated measures ANOVA.

### 2.7. Structural failure of coast live oaks

The disease stage at the time of failure was determined for each coast live oak that failed on the main stem. Trees were characterized as symptomatic alive, symptomatic dead, asymptomatic alive, asymptomatic dead, and symptomatic alive, where failure resulted from failure of other trees.

## 3. Results

### 3.1. Status since 2000

#### 3.1.1. Coast live oaks

Half of the coast live oaks that were alive in March 2000 had become symptomatic by 2008 and 32% had died with symptoms of infection. Asymptomatic coast live oaks steadily decreased as a percentage of the total population in both CCSP and MMWD, as the cumulative percentage of symptomatic trees increased (Fig. 1a). Through March 2008, 5.3% $y^{-1}$ (CCSP) and 4.5% $y^{-1}$ (MMWD) of the initially asymptomatic cohort became newly symptomatic, a combined overall rate of 5.0% $y^{-1}$. During this period, 40% of these trees became symptomatic. The mortality rate attributed to *P. ramorum* was 3.4% $y^{-1}$ and 2.6% $y^{-1}$ in CCSP and MMWD, respectively, for a combined rate of 3.1% $y^{-1}$. In contrast, dead trees without symptoms of *P. ramorum* infection in CCSP increased from 3.2% to 5.6%, a rate of 0.33% $y^{-1}$, and from <0.1% to 9.2% in MMWD, a rate of 1.1% $y^{-1}$, for a combined rate of 0.54% $y^{-1}$.

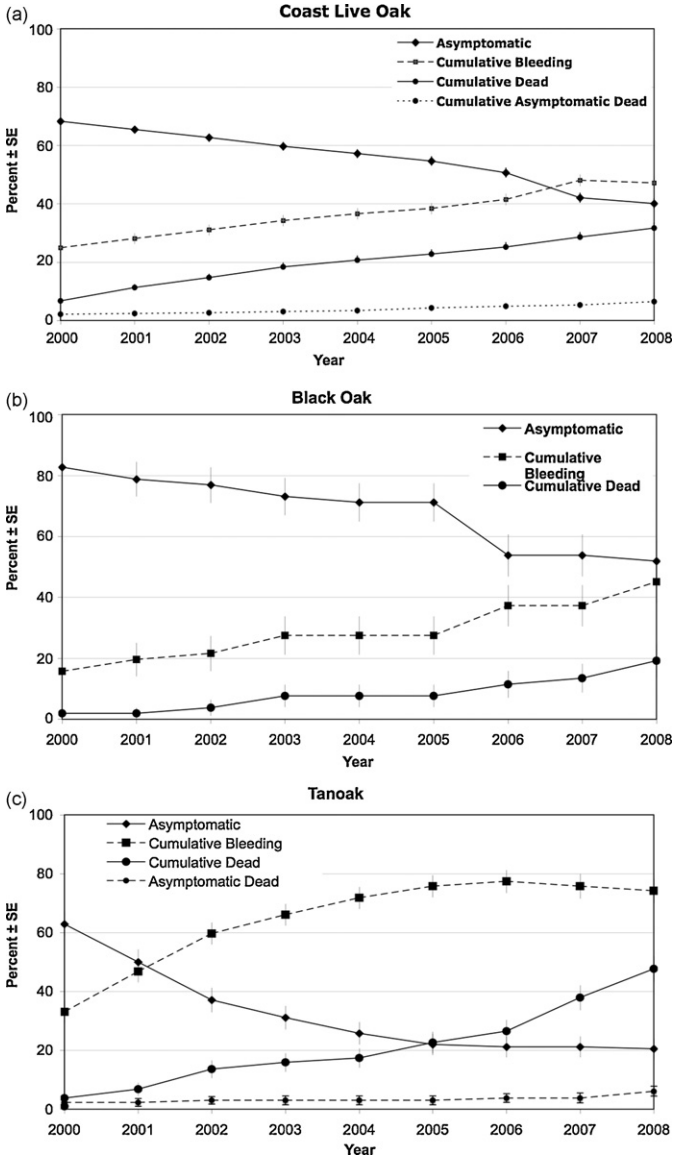


**Fig. 1.** Changes in the percentages (±SE) of (a) coast live oaks, (b) black oaks, and (c) tanoaks that were asymptomatic (♦), and the cumulative changes in trees that were bleeding (■), dead with symptoms of *P. ramorum* infection (□), and dead without symptoms of infection (●, dashed line).

Infected trees were present in every coast live oak plot by 2008, with disease intensity increases in some plots up to 25 times the levels recorded in 2000. Maximum disease intensity was 88%, averaging greater than 50%. Plots with greater than 40% disease intensity increased from five to ten. In four of the plots with less than 5% initial disease intensity, at least 33% of the trees were symptomatic or dead by 2008.

#### 3.1.2. Black oaks

The cumulative percentages of symptomatic and dead black oaks increased steadily over 8 y, to 47% and 20%, respectively (Fig. 1b). Through March 2008, 33% of the initially asymptomatic trees became symptomatic and 16% of these trees had died. The infection rate was 4.1% $y^{-1}$, with a mortality rate of 2.4% $y^{-1}$.

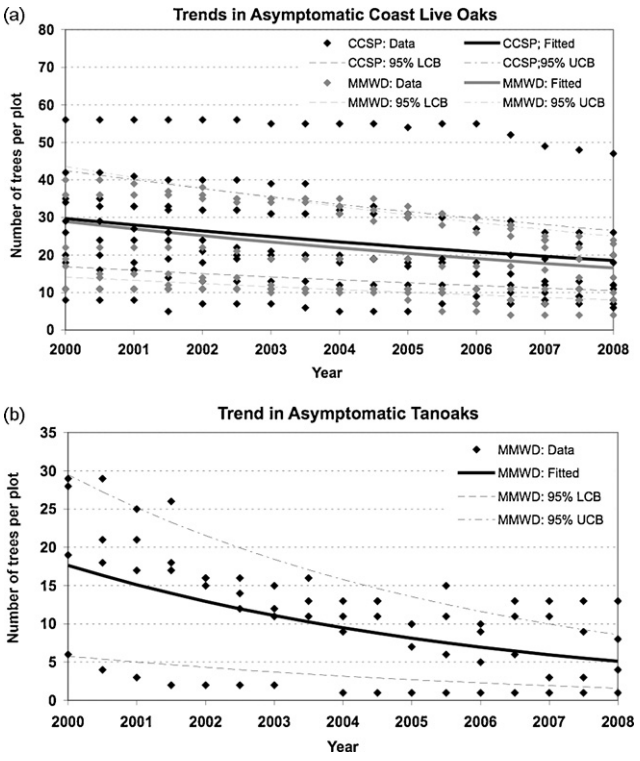


**Fig. 2.** Fitted Poisson-modeled regressions, with 95% upper and lower confidence bounds, for counts of asymptomatic trees in Marin County plots, in 6-month increments, from March 2000 to March 2008. (a) Coast live oaks in CCSP and in MMWD; (b) tanoaks.

### 3.1.3. Tanoaks

The cumulative percentages of symptomatic and dead tanoaks were 78% and 51% by 2008 (Fig. 1c). Of those that were asymptomatic in 2000, 80% had become symptomatic, a rate of 10% $y^{-1}$. Mortality of initially asymptomatic trees was 43% by 2008, a rate of 5.4% $y^{-1}$, compared with a mortality rate of 0.75% $y^{-1}$ for asymptomatic tanoaks. Disease intensity increased in tanoaks in every plot by a factor of at least 1.5, to a maximum level of 86%.

### 3.1.4. Tanoak vs. coast live oak infection rates

The infection rate for coast live oaks appeared to be relatively constant and did not differ between sites (Fig. 2a). The curves best fit a negative exponential function for both coast live oaks, CCSP fitted counts = $e^{3.3959 - 0.0296 \times \text{time}}$, MMWD fitted counts = $e^{3.3314 - 0.0347 \times \text{time}}$, and tanoaks, fitted count = $e^{2.9476 - 0.0775 \times \text{time}}$, although only the tanoak curve was statistically different from a straight line. The slope for tanoaks differed from those of both coast live oak populations ($P < 0.0001$), confirming that the infection rate was higher for tanoaks than for coast live oaks (Fig. 2b). The shape of the tanoak curve is suggestive of a slowing infection rate.

The modeled time for 50% of the initially asymptomatic coast live oak populations to become infected (with 95% CI) was 12.2 (4.7, 19.7) y ($t_{20} = 3.41$, $P = 0.0028$) in CCSP and 10.5 (3.0, 17.9) y ($t_{20} = 2.93$, $P = 0.008$) in MMWD, for an average of 11.3 (5.9, 16.7) y. The projected time to 90% infection of coast live oaks was 39.4 (28.5, 50.3) y ($t_{20} = 7.54$, $P < 0.001$) and 33.6 (23.3, 44.0) y ($t_{20} = 6.78$, $P < 0.001$), respectively, in CCSP and MMWD, for an average of 36.5 (29, 44) y. These estimates do not significantly differ between sites. The modeled time to 50% reduction in the population of asymptomatic tanoaks was 5.0 (0.7, 9.3) y ($t_{20} = 2.42$, $P = 0.025$). The projected time to 90% loss of tanoaks was 15.4 (10.4, 20.3) y ($t_{20} = 6.46$, $P < 0.001$).

## 3.2. Relationship between stem dbh, infection, and mortality

### 3.2.1. Coast live oaks

Coast live oaks with larger dbh were more likely to become infected than smaller trees. The mean dbh of trees that became symptomatic after March 2000 was 32.9 (1.3) cm; the mean dbh of asymptomatic trees was 28.1 (0.9) cm ($P = 0.0012$). The percentage of trees that became symptomatic increased from 5.7% for the smallest diameter class to 8.6% for the largest size class ($P = 0.0041$, Table 1). The mean dbh of the initially asymptomatic trees that died after developing *P. ramorum* symptoms, 32.8 (2.5) cm, was marginally greater than those that died without showing these symptoms, 25.3 (3.2) cm ($P = 0.061$).

### 3.2.2. Black oaks

There was a nonsignificant trend for bleeding and dead black oaks to have larger mean dbh values, 39.5 (5.7) cm, than asymptomatic trees, 35.9 (2.3) cm, by 2008.

### 3.2.3. Tanoaks

There was a trend for a greater percentage of newly symptomatic tanoaks to have larger stem diameters (Table 1). The mean dbh for asymptomatic and bleeding tanoaks did not differ in 2000 (McPherson et al., 2005) but were marginally different in 2008 ($P < 0.082$). The mean dbh of bleeding tanoaks decreased by 35%, from 17.8 (1.1) cm in 2000 to 11.5 (1.5) cm in 2008 ($P < 0.05$). Tanoaks also showed a trend of increasing cumulative mortality with increasing dbh.

## 3.3. Modeled survival

### 3.3.1. Coast live oaks

The median overall survival time estimate for the initially asymptomatic coast live oaks was marginally greater in CCSP, 15.8 (1.5) y, than in MMWD 11.7 (0.8) y ($P < 0.1$) (Fig. 3a and Table 2). The average median survival for both sites was 13.7 (0.9) y. The estimates for the bleeding only cohort, 11.7 (2.7) y and 7.5 (1.6) y for CCSP and MMWD, respectively, showed a similar trend for divergence with time ($P > 0.1$) (Fig. 3b). Averaged across sites, median survival of bleeding trees was 9.7 (1.6) y. Median survival of bleeding trees that were subsequently attacked by beetles also showed a trend for longer survival in CCSP, 3.3 (0.4) y than in MMWD, 2.0 (0.2) y ($P > 0.1$). Increasing stem diameter was associated with a trend toward increased survival for beetle-attacked trees. Survival

**Table 1**
Percentage of initially asymptomatic trees that developed bleeding in relation to stem dbh (cm). The size distribution of each species was categorized into quantiles. Percent symptomatic is presented with the 95% confidence interval. Means for coast live oak categories are different ($P < 0.01$) where followed by different letters.

| Coast live oak | | Black oak | | Tanoak | |
|---|---|---|---|---|---|
| Stem dbh category | Percent symptomatic | Stem dbh category | Percent symptomatic | Stem dbh category | Percent symptomatic |
| <20.3 | 5.7 (4.4–7.2) a | <30 | 6.3 (3.2–12.1) | <7.5 | 17.1 (12.3–23.4) |
| 20.4–27.9 | 7.6 (5.5–10.3) ab | 30–40 | 7.1 (3.4–14.1) | 7.6–12.5 | 19.2 (11.7–19.9) |
| 28–36.8 | 6.8 (5.2–8.7) ab | >40 | 8.2 (3.4–18.3) | 12.6–20 | 25.7 (13.9–42.6) |
| >36.8 | 8.6 (6.7–10.8) b | | | >20 | 34.8 (22.5–49.4) |

**Table 2**
Weibull model estimates of median survival times for coast live oaks, California black oaks, and tanoaks, in years (±SE). In the 2000–2003 model, tree disease stages were defined by their status in 2000. The first estimate in each bleeding + beetles cell for coast live oaks is for bleeding + beetles; the second estimate is for bleeding + beetles + *Hypoxylon*.

| Cohort status | Coast live oak: CCSP | Coast live oak: MMWD | Black oak | Tanoak |
|---|---|---|---|---|
| 2000–2003 Model | | | | |
| Overall | 29.5 (8.4) | 31.8 (9.3) | NA | 12.6 (3.8) |
| Bleeding | 7.0 (1.2) | 7.6 (1.6) | NA | 6.2 (1.3) |
| Bleeding + beetles | 2.6 (0.3)<br>3.0 (0.3) | 2.8 (0.5)<br>3.2 (0.6) | NA | 1.9 (0.9) |
| 2000–2008 Model | | | | |
| Overall | 15.8 (1.5) a | 11.7 (0.8) b | 13.8 (3.0) | 8.8 (0.7) |
| Bleeding | 11.7 (2.7) a | 7.5 (1.6) a | 6.2 (1.3) | 5.8 (0.7) |
| Bleeding + beetles | 3.3 (0.4) a | 2.0 (0.2) a | 1.9 (0.9) | 1.7 (0.4) |

In the 2000–2008 model, all trees were asymptomatic at the start of the evaluation period. For coast live oaks in the bleeding + beetles category, the estimate is for site-averaged dbh (CCSP: average dbh = 29.3 (0.9) cm, MMWD: average dbh = 31.1 (1.1) cm). Using the normal approximation, median survival estimates for coast live oaks are different ($P < 0.1$) where followed by different letters in a row.

of trees in the smaller quantile, mean dbh = 20 cm, was 2.4 (0.6) y and 1.9 (0.5) y in CCSP and MMWD, respectively. Because the relative times for survival by dbh show similar patterns, only the graph for the larger quantile is shown (Fig. 3c). The median survival times of trees in the larger quartile, with a mean dbh of 40 cm, were 3.4 (0.8) y and 2.6 (0.5) y in CCSP and MMWD, respectively, 35–40% greater than smaller trees.

#### 3.3.2. Black oaks

The estimated median overall survival time from the Weibull model for black oaks that were asymptomatic in March 2000 was 13.8 (3.0) y (Fig. 4a and Table 2). All of the symptomatic trees were in MMWD. Median survival time for the black oaks that developed bleeding after March 2000 declined to 6.2 (1.3) y (Fig. 4b). Beetle attacks reduced survival to 1.9 (0.9) y (Fig. 4c).

#### 3.3.3. Tanoaks

Averaged across all plots, the Weibull modeled median overall survival time of tanoaks in the March 2000 asymptomatic cohort was 8.8 (0.7) y (Fig. 5a and Table 2). Estimated median survival varied among plots (Bonferroni adjustment, $P = 0.05$), from 6.0 (1.3) y to 11.5 (1.8) y. The two plots with the greater median estimated survival were on a relatively dry ridge top and those with the lower median survival were in more mesic sites associated with coast redwoods (*Sequoia sempervirens* Lamb. ex D. Don Endl.).

The median survival time estimated from the Weibull model for tanoaks that developed bleeding after 2000 was 5.9 (0.7) y (Fig. 5b). Trees that were attacked by beetles had much lower estimated survival, 1.7 (0.4) y (Fig. 5c). Tree dbh was not found to affect survival.

### 3.4. Association of bay laurel and madrone with coast live oak disease intensity

The mean bay laurel basal area in all plots increased from 8.4 (2.0) $m^2$ in 2001 to 9.1 (2.3) $m^2$ in 2007 ($P = 0.029$, Wilcoxon signed-rank test). In 2001, there was a strong positive relationship between basal area of living bay laurels and coast live oak disease intensity at the plot level ($F_{1,14} = 13.096$, $P < 0.0028$). In 2007, the relationship between bay laurel basal area and 2007 coast live oak disease intensity was still positive ($F_{1,14} = 4.99$, $P < 0.042$), but considerably weaker.

Mean madrone basal area did not differ between 2001, 0.24 (0.1) $m^2$ and 2007, 0.21 (0.1) $m^2$. In 2001 the basal area of living madrones was weakly and positively associated with coast live oak disease intensity ($F_{1,14} = 3.41$, $P > 0.086$). However, by 2007 the relationship was negative ($F_{1,14} = 5.16$, $P < 0.039$), indicating that the plots with lower madrone basal area were those with higher disease intensity.

### 3.5. Structural failure in coast live oaks

Of the 44 living coast live oaks that failed on the main stem, 41 were bleeding with both beetle attacks and *Hypoxylon thouarsianum* sporophores, and only one was asymptomatic. Breakage in beetle-attacked trees only occurred where beetles had tunneled into the stem. The mean survival following initial beetle attacks was 2.8 (0.7) y when the date of first beetle attack could be determined ($n = 10$). Only two dead trees failed without evidence of *P. ramorum* infection.

## 4. Discussion

The *P. ramorum* epidemic in California forests is still in its early stages. Although dying coast live oaks and tanoaks were first reported in 1994 and 1995, it was not until 2000 that the causal agent was isolated (Rizzo et al., 2002) and shown to be a recently described species (Werres et al., 2001). Among the many hosts, both woody and herbaceous (Rizzo and Garbelotto, 2003), the three trees studied here support diverse communities of organisms, often define the forest type, and can dominate stand structure in these forests (Miles and Goudey, 1997). This study confirms that more than a decade into the SOD epidemic in California, infection levels in these species remain elevated, though annually variable, and mortality has steadily increased.

The mortality rates we report here are the result of repeated observations between 2000 and 2008. For the period 1994–2004, Brown and Allen-Diaz (2009) used retrospective examination of dead trees to estimate rates of 4.5% $y^{-1}$ and 5.5% $y^{-1}$ for two highly impacted sites in CCSP, compared with rates of 3.4% $y^{-1}$ for CCSP and 3.1% $y^{-1}$ overall in the present study. The mortality rate reported here for asymptomatic coast live oaks, 0.33% $y^{-1}$, is also lower than their calculated rate of 0.45% $y^{-1}$ or the background mortality rate of 0.49% $y^{-1}$ estimated by Barrrett (2006) for the period prior to the likely introduction of *P. ramorum* into California. The more intensive repeated evaluation of the same trees over an 8-y period in this study allowed us to determine the causes of mortality, in many cases beginning with the first observation of bleeding.

The mortality rate of *P. ramorum*-infected California black oaks, 2.4% $y^{-1}$, is consistent with the maximum rate of 2.4% $y^{-1}$ reported by Brown and Allen-Diaz (2009). Barrrett (2006) reported a baseline mortality rate of 0.36% $y^{-1}$, but there was an insufficient number of trees in our plots to estimate a rate. Coast live oaks and black oaks, which hybridize readily, appear to respond similarly to *P. ramorum* infections.

The close agreement between the mortality rate for *P. ramorum*-infected tanoaks that we found in this study, 5.5% $y^{-1}$, with the

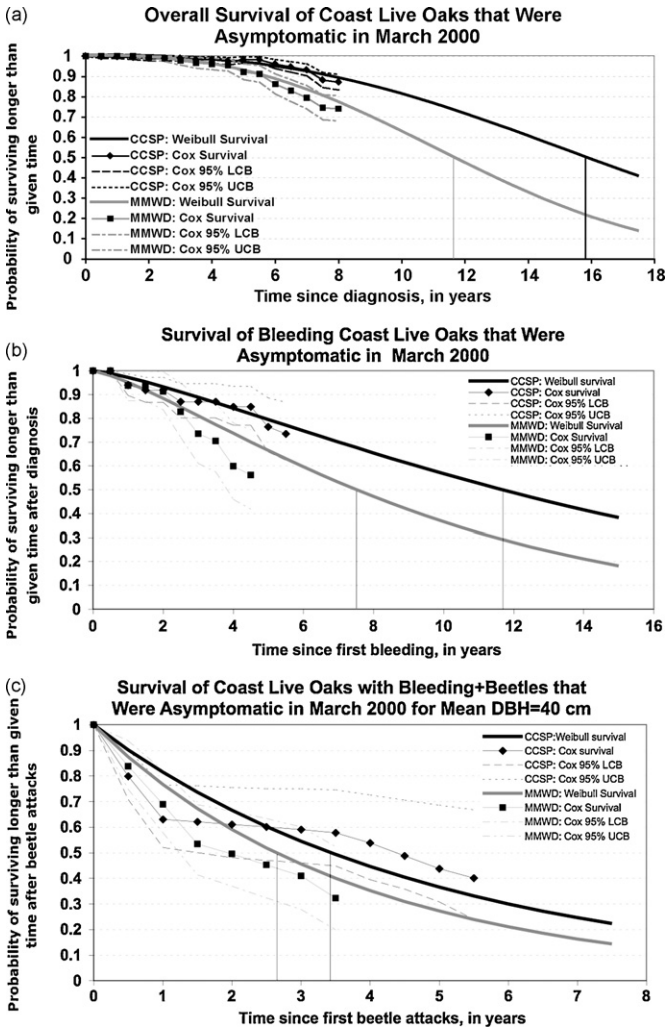


**Fig. 3.** (a) Overall survival probabilities for coast live oaks that were asymptomatic in March 2000 estimated using Weibull (solid lines) and Cox PH (dashed lines) regression models. Regression curves are shown for CCSP (solid line) and MMWD (grey line). Data points are shown for the Cox PH regressions. In this and following survival curve graphs, 95% lower and upper confidence bounds are included for the Cox PH regressions. The vertical axis defines the probability of survival beyond the corresponding time on the horizontal axis. In all survival curves shown, vertical lines denote the time to 50% survival probability (median survival). The same symbols are used for the two sites in each of the following graphs. (b) Survival probabilities for coast live oaks that were asymptomatic in March 2000 and that subsequently developed bleeding. The horizontal axis provides the survival time after the diagnosis of a tree as bleeding. (c) Survival probabilities for coast live oaks, mean dbh = 40 cm, that were asymptomatic in March 2000 and that subsequently developed bleeding and were attacked by beetles. The horizontal axis provides the survival time after a tree was attacked by beetles.

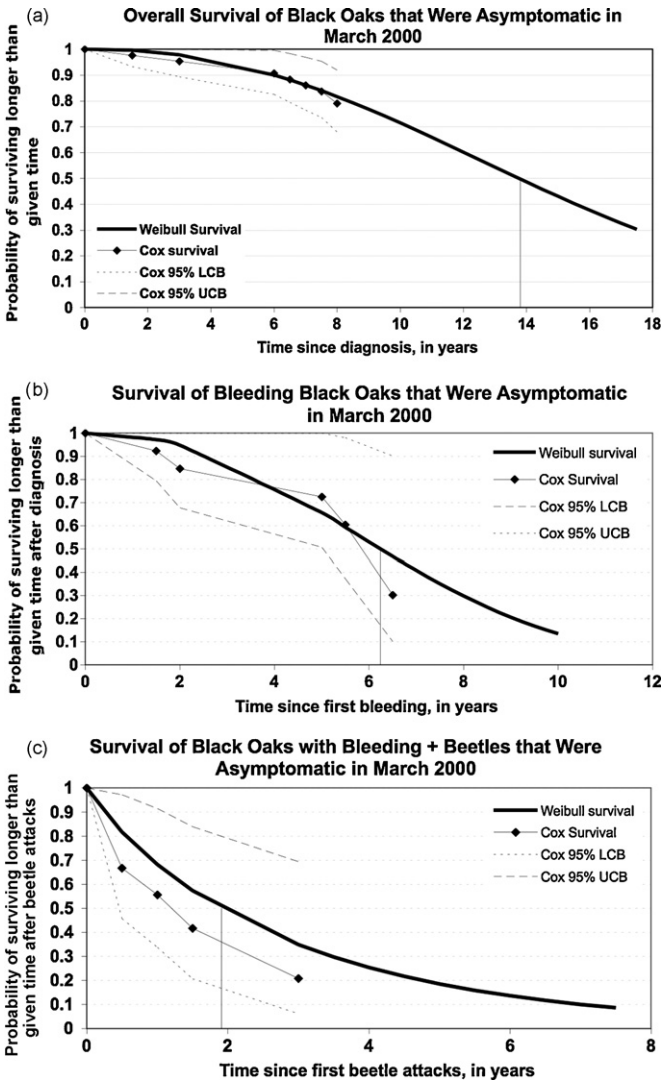


**Fig. 4.** (a) Overall survival probabilities for black oaks that were asymptomatic in March 2000 estimated using Weibull (solid line) and Cox PH (thin line) regression models. Data for CCSP and MMWD were pooled for all analyses. (b) Survival probabilities for black oaks that were asymptomatic in March 2000 and that subsequently developed bleeding. The horizontal axis provides the survival time after the diagnosis of a tree as bleeding. (c) Survival probabilities for black oaks, that were asymptomatic in March 2000 and that subsequently developed bleeding and were attacked by beetles. The horizontal axis provides the survival time after a tree was attacked by beetles.

6.0% $y^{-1}$ rate reported for a wide range of sites across California (Maloney et al., 2005), suggests that infection and mortality rates are similar throughout the range of infected tanoaks in California. The background mortality rate in the present study, 0.75% $y^{-1}$, is considerably greater than the estimated pre-epidemic rate of 0.36% $y^{-1}$ made by Barrett (2006). This difference may be due to the rapid local environmental changes resulting from the death of large numbers of overstory trees in the study plots, some of which have lost more than 60% of their tanoaks since 2000. Because infection and mortality rates are higher in tanoaks than in the true oaks, relatively few asymptomatic tanoak still remain in many infested stands.

As native forests worldwide are exposed to increasing invasions by nonnative organisms (Haack, 2001), there is a need to develop predictive models of tree responses to introduced pathogens for which there is little or no epidemiological information. A recent application of survival analysis to multiple oak species in Missouri forests used increase in dbh as a proxy variable for tree vigor, although the study did not involve invasive organisms (Woodall et al., 2005). Survival analysis can incorporate different variables into the regression, such as discrete disease stages, environmental factors, site, and the influence of secondary organisms, including insects or fungi. In this study we show that the symptoms and signs of *P. ramorum* infection can be used as predictive variables to model disease response in genetically diverse tree populations.

The value of Weibull survival models is shown by the relatively close agreement between estimated survival times for 3 and 8 y of observation. Overall survival estimates for asymptomatic coast live oaks and tanoaks based on 3 y of data were 30–50% greater than those derived from 8 y of data from the same population of trees. Median survival estimates from the Weibull model for trees that were asymptomatic in 2000 and subsequently became symp-

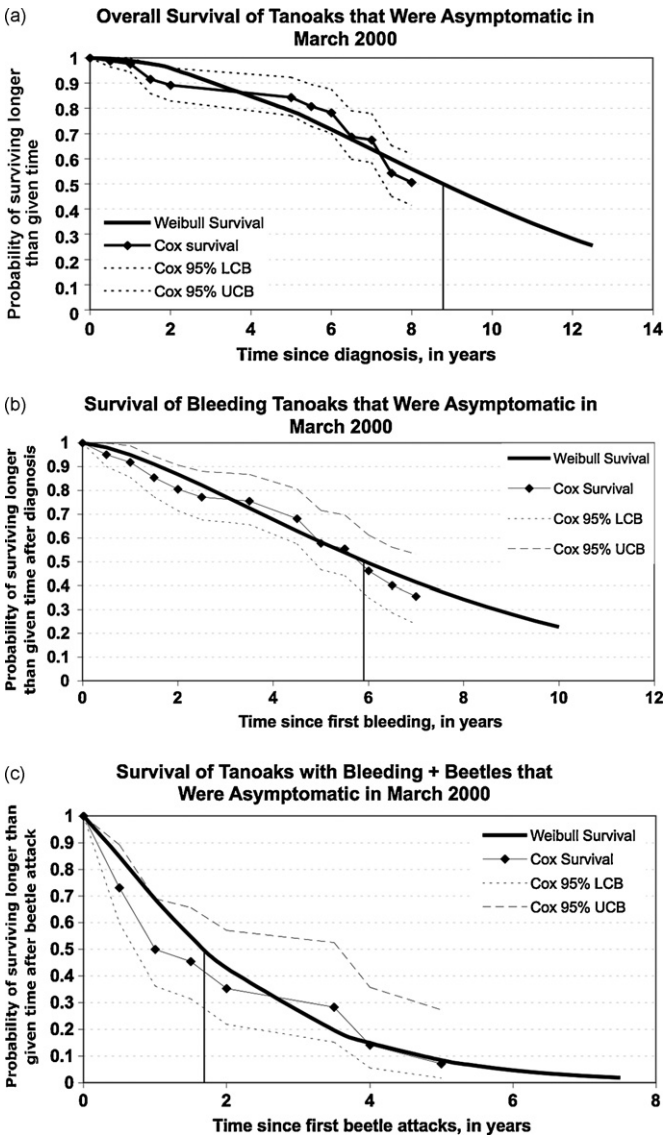


**Fig. 5.** (a) Overall survival probabilities for tanoaks that were asymptomatic in March 2000 estimated using Weibull (solid lines) and Cox PH (thin lines) regression models. (b) Survival probabilities for tanoaks that were asymptomatic in March 2000 and that subsequently developed bleeding, estimated using Weibull (solid lines) and Cox PH (broken lines) regression models. The horizontal axis provides the survival time after the diagnosis of a tree as bleeding. Symbols are the same as those used for overall survival. (c) Survival probabilities for tanoaks that were asymptomatic in March 2000 and that subsequently developed bleeding and were attacked by beetles. The horizontal axis provides the survival time after a tree was attacked by beetles.

tomatic (Table 2) were generally similar to those derived for the same-symptom cohorts that were defined by their March 2000 disease status (McPherson et al., 2005). The semi-parametric Cox PH model cannot be used to model the outcome of a process beyond the time scale of the input data. In the present study the survival probability regressions estimated using the Weibull model were always within the 95% confidence intervals of the Cox PH regressions. Considering the magnitude of the uncertainty that was introduced by the use of trees that had been symptomatic for unknown times prior to the start of the study, the similarity of the modeled survival estimates based on 3 y of observations with those derived from 8 y of observations supports the wider use of this modeling approach.

Increased risk of infection and mortality with increasing tree dbh was also reported from a study in the Big Sur region of California for tanoaks with dbh greater than 10-cm (Cobb et al., 2008). In contrast to coast live oaks, in which smaller diameter trees such as seedlings and saplings are much less susceptible than older trees to *P. ramorum*, it is likely that tanoak populations will decline more rapidly wherever the pathogen is introduced.

Beetle attacks on bleeding cankers affect the progress of sudden oak death. For the three species, median survival of bleeding trees varied from 5.8 to 11.7 y; following beetle attacks, median life expectancy decreased by 65–70%, to 1.7–3.4 y. The ambrosia beetle *Monarthrum scutellare* tunnels up to 15 cm into coast live oak sapwood and can introduce a variety of fungal taxa into this substrate (Erbilgin et al., 2008), in the process impairing water conduction through xylem tissue, and in many cases, leading to structural failure long before the foliage shows evidence of stress (McPherson et al., 2008). Beetles tunneling through infected phloem tissue circumvent tree defenses by vectoring pathogens and decay fungi deep into sapwood.

Sudden oak death continues to expand into previously unaffected stands in infested areas. It is not clear why *P. ramorum* infection levels vary so widely in coast live oaks, often in sites separated by only tens of meters. Maloney et al. (2005) proposed that the disease distribution in tanoaks is more reflective of the historical movement of propagules into certain sites than any particular site characteristics. In the present study, all coast live oak plots that initially had little or no infection showed increasing infections by 2008, in some plots exceeding 50% of the trees. It is likely that similar expansion at local scales will be observed throughout the range of *P. ramorum* infections.

Our results support the hypothesis that bay laurel is associated with the infection in coast live oaks (Davidson et al., 2005). However, plots with little or no bay laurel basal area had, by 2007, attained levels previously found in the plots with higher bay laurel basal area. Bay laurels may be locally important in the earliest stages of the epidemic, but become less important in disease propagation with the passage of time. The presence of numerous other foliar hosts in these forests may provide sufficient propagules for new infections. The negative association of madrone basal area with coast live oak disease intensity may reflect its presence in sites that are drier, on average, than those with more abundant bay laurels, which would be more conducive to *P. ramorum* sporulation.

In light of the high infection and mortality levels of all three species, evidence for resistance to *P. ramorum* is of considerable interest. The persistence of asymptomatic coast live oaks and tanoaks for 8 y in plots that initially had high disease intensity levels implies the presence of resistant trees. The relatively steady infection rates calculated in this study may mask the presence of resistant individuals, as suggested by the negative exponential regressions for counts of asymptomatic coast live oaks and tanoaks (Fig. 2a and b). If the most susceptible trees were infected early in the epidemic, infection rates would be expected to decrease as the proportion of resistant trees increases. Although decreasing stem density at the stand level might be expected to correlate with lower inoculum levels, Maloney et al. (2005) did not find a relationship between overall stem density by plot and disease incidence in tanoaks. Since coast live oak is reported to be a terminal host for the pathogen (Davidson et al., 2002), and therefore not a source for further infections, stand density of this species, per se, should not affect infection rate. In the present study, decreases in inoculum load cannot explain the apparently slowing infection rate, as bay laurel basal area increased during this time. Under the pressure of this aggressive pathogen, the presence and propagation of resistant genotypes among the host oaks and tanoaks may provide the best chance for sustainable wildland populations of these species and for management of these forests.

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