

# Long-Term Trends in Coast Live Oak and Tanoak Stands Affected by *Phytophthora ramorum* Canker (Sudden Oak Death)<sup>1</sup>

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## Abstract

Permanent plots were established in 2000 to examine how tree and site factors affect risk of *Phytophthora ramorum* stem canker (sudden oak death [SOD]) and determine how affected stands change over time due to disease. *P. ramorum* canker was prevalent in the sampled coast live oak (*Quercus agrifolia*) or tanoak (*Lithocarpus densiflorus*) stands in 2000. In September of each year from 2000 through 2008, we collected data on *P. ramorum* symptoms, tree condition, tree failures, regeneration, and various other factors in 150 circular plots (8 m radius) at 12 locations in Marin, Sonoma, and Napa Counties.

Disease development patterns over time differed between tanoak and coast live oak populations in the study (fig. 1). The increase in disease incidence between 2000 and 2008 was greater for tanoaks (31 to 49 percent) than for coast live oak (23 to 33 percent). Disease incidence in coast live oaks increased strongly from 2005 through 2007. This increase was associated with abundant late season rains that provided favorable conditions for disease spread in spring 2005 and spring 2006. Tanoak showed a less much pronounced increase in disease incidence for these two years. Based on time lags between favorable conditions for infection and the appearance of visible symptoms, most coast live oaks had latent periods from 0.5 to 1 year, whereas the minimum latent period in tanoaks was commonly about a year.

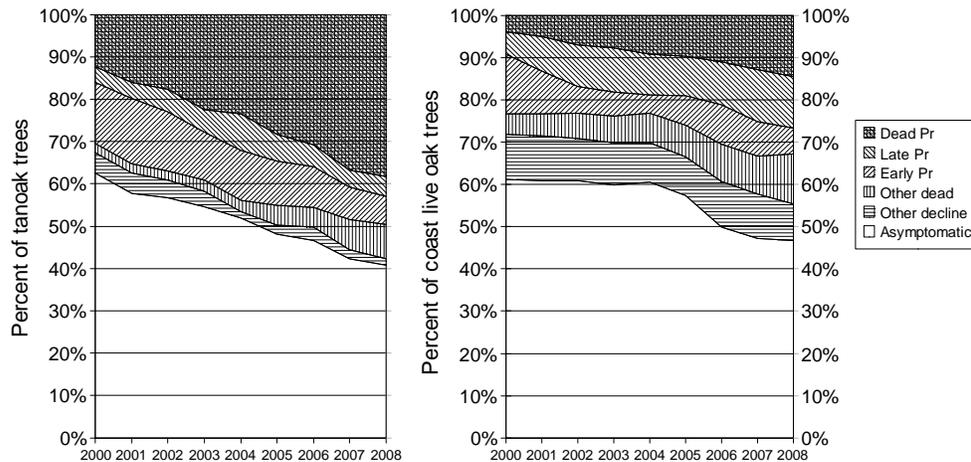


Figure 1—Changes in health of all tanoak (n = 187) and coast live oak (n = 655) study trees from September 2000 to September 2008. Dead Pr = tree dead as a result of *P. ramorum*; Late Pr = live trees with *P. ramorum* cankers plus beetle boring and/or *Hypoxylon thouarsianum* fruiting bodies; Early Pr = live trees with *P. ramorum*

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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.

The increase in SOD mortality over time has followed a linear trend between 2000 and 2008 ( $R^2 = 0.98$ ) for both oaks and tanoak. Extrapolating the regression lines back in time suggests that mortality would have initially appeared in the tanoak plots around 1996 and in the coast live oak plots around 1997. These estimated dates for onset of mortality are close to the first reported deaths of tanoaks in Marin County in April 1995 (Svihra 2001). Extrapolation of the regression line obtained for total tanoak infection ( $R^2 = 0.96$ ) suggests that the first canker symptoms in these stands could have developed in the mid 1980s.

In 2008, disease incidence at coast live oak locations ranged from 8 to 57 percent. The increase in disease incidence since 2000 has varied widely by location. Relatively stable differences in disease incidence between nearby coast live oak locations were mainly associated with differences in California bay laurel (*Umbellularia californica*) cover rather than weather and climate variables. Disease risk of individual coast live oaks increased with increasing proximity and density of bay laurel foliage.

We also reexamined other factors previously associated with SOD risk in individual coast live oaks to account for changes in disease status due to new infections that developed in 2005 and 2006. Previously significant relationships were unchanged. Increased SOD risk was associated with greater bark thickness, which is positively correlated with stem diameter; higher amounts of active bark expansion, as expressed by unweathered bark in fissures; and lower than average water stress, as indicated by higher (less negative) stem water potential readings.

After the onset of symptoms, tanoaks showed high initial rates of mortality: 40 percent were killed within a year after the onset of symptoms and 63 percent were dead by 2008. In contrast, among coast live oaks first showing symptoms in 2001 or later, only 13 percent died within one year and total mortality had only reached 21 percent by 2008. Symptom remission, in which cankers stopped bleeding and showed no evidence of expansion for multiple years, was common in coast live oak. Among all trees observed since 2000, cankers became inactive in about 25 percent of the trees. In contrast, cankers became inactive in 46 percent of the trees that developed symptoms in the 2005 to 2006 disease pulse. Many of the cankers developing in 2005 or later were first observed while they were relatively small and may have been more likely to become inactive than larger cankers detected at a later stage. In addition, trees that were infected by *P. ramorum* in 2005 and 2006 represent trees that survived an earlier round of disease and could represent the more resistant trees in the population. Among all trees in which *P. ramorum* symptoms were verified by isolation of *P. ramorum* on selective media, 22 percent of the cankers became inactive.

The overwhelming majority of all host tree failures through 2008 (69 percent) occurred in trees that had *P. ramorum* canker symptoms in 2000. Early *P. ramorum* canker symptoms (bleeding only) were not associated with tree failure; only SOD-affected trees colonized by secondary organisms had elevated failure rates. Over two thirds of the initial failures in SOD-affected coast live oaks occurred in dead trees or dead stems of live trees. Relatively few trees that have become symptomatic since 2000 had failed by 2008. Trees rated as in decline or dead due to factors other than SOD also failed at elevated rates.

A survival analysis for all coast live oaks indicated that time to failure was most strongly affected by whether the tree was already dead (table 1). *P. ramorum* symptoms, *Hypoxylon thouarsianum* sporulation, and severe tree decline associated with other agents (typically decay fungi) also had highly significant effects on time to failure. Among live trees, time to failure was most strongly related to *H. thouarsianum*. Time to failure decreased as the proportion of the stem circumference with *H. thouarsianum* fruiting bodies present increased.

**Table 1—Significance of parameters in a survival analysis (exponential model fit) for years to failure in coast live oak; predictor variables reflect conditions that existed in the year prior to failure**

Source	DF	Prob>ChiSq
Overall model		<0.0001
Tree dead	1	<0.0001
<i>P. ramorum</i> symptoms	1	0.0025
<i>H. thouarsianum</i> sporulation	1	0.0030
Tree in decline	1	0.0077
Beetle boring	1	0.0414

Among plots with SOD-related mortality, 27 percent of the tanoak plots and 45 percent of the coast live oak plots showed decreases in plot canopy cover. Since 2001, coast live oak plots have shown an increase in Douglas-fir (*Pseudotsuga menziesii*) density (+31 percent), no change in California bay laurel density, and decreased madrone (*Arbutus menziesii*) density (−7 percent). Three locations had relatively high levels of madrone mortality, ranging from 17 to 31 percent. We have determined that *P. cinnamomi* is associated with declining madrone and bay laurel at one of these locations.

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## Literature Cited

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