

Sudden Oak Death: Disease Trends in Marin County Plots after One Year¹

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Abstract

Sudden oak death has emerged as a major threat to the oak forests of California. In oaks and tanoak, this disease complex consists of a previously unreported fungus-like pathogen, *Phytophthora ramorum*, insects (bark and ambrosia beetles), and a secondary fungus, *Hypoxyton thouarsianum*. Species monitored in this study were coast live oak (*Quercus agrifolia*), California black oak (*Q. kelloggii*), and tanoak (*Lithocarpus densiflorus*). Disease progression plots were initiated in March 2000 to determine infection and mortality levels, symptomology, and changes in disease status. Plots were placed in two ecologically different sites in Marin County, China Camp State Park and a protected watershed of the Marin Municipal Water District (MMWD). Ten plots of 0.1 to 0.2 ha, selected to encompass the range of habitat types and species compositions found within these forests, were placed in each site. For all oaks and tanoaks, the following data were recorded quarterly for one year: diameter (dbh); presence/absence and abundance of seeps; presence/absence of *H. thouarsianum* fruiting bodies; presence/absence of bark and ambrosia beetles; and condition of the foliage. More than 750 oaks and tanoaks in these plots have been permanently tagged and geolocated using a global positioning system (GPS). Across China Camp State Park, independent and unbiased estimates of infection and mortality levels were acquired in summer 2001 using point-centered quarter sampling.

Apparent infection and mortality levels increased for the three species between March 2000 and March 2001. For coast live oak in China Camp, apparent infection levels increased from 35 percent in 2000 to 38 percent in 2001. In MMWD, these values were 16 percent in 2000 and 19 percent in 2001. Mortality of coast live oak rose from 8 to 15 percent in China Camp and 6 to 8 percent in MMWD during this period. California black oak in both sites exhibited apparent infection levels of 19 percent in 2000 and 27 percent in 2001. Apparent infection levels in tanoak rose from 40 to 55 percent in one year. Mortality also rose, from 12 to 15 percent. The point-centered quarter method yielded estimates of 30 percent for infection and 14 percent for mortality, for coast live oak. For California black oak, estimates were 21

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percent for infection and 16 percent for mortality. The role of bark and ambrosia beetles in this disease complex is unclear. Every symptomatic tree that died had been colonized by beetles in the plots prior to death. These insects have consistently been found in association with seeping areas on infected trees. Normally these bark and ambrosia beetles are associated with dying, dead, and broken trees. These interactions between an apparently novel pathogen, its host plants, and native insects represent new ecological associations, with unknown future consequences for these forests.

Introduction

A disease that appears to be caused primarily by a newly described pathogen, *Phytophthora ramorum* (Werres and others 2001), has infected many woody plant species in central and northern coastal California. This disease is referred to as sudden oak death syndrome, in reference to the rapid color change of foliage from green to brown over a period of weeks to months, in its final stages in oaks and tanoaks (Svihra 1999a). Prior to the isolation and description of this fungus-like pathogen as a species of *Phytophthora* (Garbelotto and others 2001), the causal agent of elevated tree mortality was unknown. This organism was subsequently shown to be the same species as a previously described pathogen infecting rhododendrons in Western European nurseries in the early 1990s (Werres and Marwitz 1997).

Tanoaks, *Lithocarpus densiflorus*, were first reported to be dying in California in 1995 (Svihra 1999b). Although a number of other species are now known to be hosts, the effects of the disease appear to be most severe for tanoaks and for several native oak species. *Phytophthora ramorum* is pathogenic on a number of species in the Fagaceae, including coast live oak, *Quercus agrifolia*, California black oak, *Q. kelloggii*, Shreve oak, *Q. parvula* var. *shrevei*, and tanoak. In addition, pinoak, *Q. palustris*, and northern red oak, *Q. rubra*, are also susceptible in laboratory tests (Rizzo and others 2002). Native species in the families Ericaceae, Aceraceae, Hippocastanaceae, Rosaceae, Rhamnaceae, and Lauraceae have recently been shown to be hosts of the pathogen.

In California, the presence of the disease has been confirmed in forested areas from Big Sur, in Monterey County to southern Mendocino County, with major infestations in Marin, Sonoma, Monterey, and Santa Cruz Counties. An additional infestation in tanoak has been confirmed recently in Curry County, southwestern Oregon. The reasons for the apparently disjunct range of its geographic distribution are unknown.

When the field plots described in this report were established in March 2000, very little was known about the etiology of the disease in oaks and tanoaks. The most notable characteristics of sudden oak death syndrome were the apparently rapid death of large, mature trees, seeping (“bleeding”) of a dark, viscous exudate, typically on the lower trunk, and the association of large numbers of bark and ambrosia beetles (Coleoptera: Scolytidae) and fruiting bodies of *Hypoxylon thouarsianum* with the seeping areas of declining but still green trees.

These observations appeared to support a primary role for bark and ambrosia beetles (Svihra 1999c) and/or *H. thouarsianum* in tree death, in the absence of an identified pathogenic agent. There are two species of ambrosia beetles, *Monarthrum scutellare* and *M. dentiger*, that have been observed to tunnel into the sapwood of host trees. The western oak bark beetle, *Pseudopityophthorus pubipennis*, produces

galleries in the phloem and outer bark of many oak species. These insects are opportunists, utilizing weakened, broken, and uprooted host trees (Furniss and Carolin 1977), and in natural forests, have only rarely been reported to attack living trees (Solomon 1995).

Speculations concerning anthropogenic causes, e.g., acid precipitation and air pollution, as well as climatic fluctuations associated with the El Niño and La Niña phenomena were also advanced. The absence of information on the basic etiology of sudden oak death syndrome motivated the research reported here. The goals of this study were to: 1) describe the symptoms and the order of their appearance, 2) determine a time course for symptoms of the disease and signs of associated organisms, 3) investigate the association of beetles and of *H. thouarsianum* in trees with bleeding cankers, 4) determine whether beetles were attacking apparently healthy trees, and 5) establish the time of first appearance of seeping cankers on symptom-free trees and the subsequent response of the trees.

Methods

Research plots to monitor symptom progression were established in March and April 2000 in two sites in Marin County; China Camp State Park (10 plots) near San Rafael and Marin Municipal Water District (10 plots), a protected watershed on the lower elevations of Mt. Tamalpais (*fig. 1*). Both sites exhibit heavy levels of sudden oak death syndrome in some areas and light to no apparent infection in others. These two sites are approximately 12 km apart and differ somewhat in microclimate, elevation, and woody species composition. China Camp State Park (605 ha) is bordered by the city of San Rafael on the south and west and San Pablo Bay on the north and east. Study plots range from near sea level to slightly more than 200 m in elevation. The Marin Municipal Water District (MMWD) site is approximately 6 km from San Pablo Bay and 8 km from the Pacific Ocean. This site is bordered by Mt. Tamalpais State Park, Golden Gate National Recreation Area, and other forested land. Study plots were placed at elevations between 180 and 250 m. *Lithocarpus densiflorus* is present only in MMWD, although both *Q. agrifolia* and *Q. kelloggii* are found in both sites. Smaller numbers of two apparently resistant species, valley oak, *Q. lobata*, and blue oak, *Q. douglasii*, occur in China Camp State Park.

Symptom progression plots were not located at random, but were selected to encompass a range of habitat types, species compositions, and moisture regimes. Limiting consideration to the Fagaceae, the plots included coast live oak alone; coast live oak plus black oak; coast live oak, black oak, and valley oak; coast live oak, black oak, and tanoak; coast live oak and tanoak; and tanoak alone. Although these plots include a number of other woody species that have recently been shown to be either hosts for *P. ramorum* or to be susceptible to this pathogen in laboratory tests, the oaks and tanoak are the focus of this study.

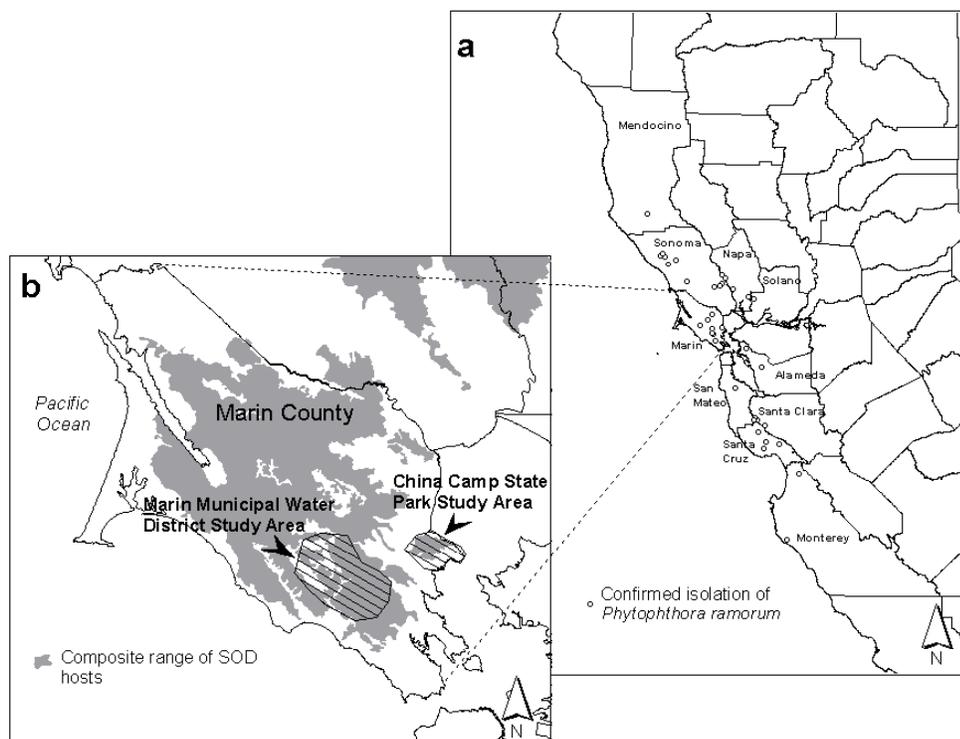


Figure 1—a) Distribution of confirmed cases of *P. ramorum* infection in coast live oak and tanoak in California, through August 2001. b) Locations of the permanent monitoring study sites in Marin County. Shaded areas indicate the composite range of hosts of *P. ramorum*.

In each of the 0.1 to 0.2 ha plots, the following variables were recorded for all oaks and tanoaks:

- Species
- Diameter at breast height (dbh)
- Presence/absence and abundance of seeps
- Presence/absence and abundance of fruiting bodies of *H. thouarsianum*
- Presence/absence, and age of bark and ambrosia beetle infestations
- Distribution of beetles in relation to seeping areas
- Condition of the foliage.

A tree was determined to be dead when its leaves had turned brown. Only trees that showed unambiguous evidence of bleeding cankers were counted in the assessment of mortality. *Phytophthora ramorum* has been repeatedly isolated from *Q. agrifolia*, *Q. kelloggii*, and *L. densiflorus* in the forests where these plots are located (see <http://camfer.cnr.berkeley.edu/oaks>). Individual trees in the plots were not tested for the presence of *P. ramorum*, but were instead characterized by the symptoms of

sudden oak death described above (McPherson and others 2000). The only consistently reliable diagnostic methods presently available require that samples from an active canker be removed for culturing. Since cutting into trees is an invasive technique, trees in these plots were not sampled for the presence of *P. ramorum*. For the assessment of mortality at the initiation of the study in March 2000, trees were recorded as dead only if they showed unambiguous signs of the seeping associated with sudden oak death. Data from trees have been collected four times per year, since March 2000, giving five observation points by March 2001. A total of 760 oaks and tanoaks in these plots have been permanently tagged and geolocated using a global positioning system (GPS).

An independent assessment of infection and mortality levels for *Q. agrifolia* and *Q. kelloggii* was conducted in China Camp State Park in June to September 2001. The point-centered quarter plotless density estimation method (Cottam and Curtis 1956, Engeman and others 1994) was used to provide unbiased estimates of these variables using transect-based sampling across broad areas of this forest. The anchor point for each transect was located near the San Pablo Bay side of the park on a North-South axis, from which sampling points (nodes) were placed at 100 m intervals. The parallel transects were spaced approximately 500 m apart. A GPS reading was taken at the anchor point and at each node to allow for follow-up monitoring. The nearest *Q. agrifolia* or *Q. kelloggii* that fell within each 90° quadrant (N, S, E, and W) around every node was permanently labeled and the same variables used for the permanent plots were recorded. The azimuth and distance from the node were recorded for use in GIS analysis. This provided a maximum of four host trees per sampling node. If a tree was more than 30 m from a node, that quadrant was considered empty.

Results

Trees exhibiting symptoms of sudden oak death, as well as recently killed trees, were widespread within the areas where the plots were located. Although the symptom progression plots were selected without regard for the presence of trees with symptoms, none was free of symptomatic trees. No trees recorded as having seeping cankers were observed to be free of this symptom on subsequent sampling dates, although seeps were difficult to detect during the dry months.

The percentage of symptomatic trees increased for all species monitored during the period of March 2000 to March 2001 (*table 1*). For *Q. agrifolia* in China Camp State Park, the overall level of symptomatic trees was 35.4 percent in March 2000 and 38.6 percent in March 2001 (*table 1*). Between-plot variation in the percentage of symptomatic coast live oaks ranged from 7 to 95 percent in 2000 and from 13 to 95 percent in 2001. In the MMWD plots, the overall percentage of symptomatic coast live oaks was 16.3 percent in 2000 and 18.9 percent in 2001. The between-plot range was 0 to 43 percent in 2000 and 0 to 48 percent in 2001.

Table 1—Percentages of coast live oak, California black oak, and tanoak exhibiting the seeping symptom of sudden oak death syndrome in Marin Co. plots, March 2000 and March 2001.

Site	Species	n	Trees alive		Trees alive and seeping	
			2000	2001	2000	2001
China camp	Coast Live Oak	293	92.5	86.7	35.4	38.6
MMWD	Coast Live Oak	214	94.4	91.6	16.3	18.9
MMWD	Tanoak	157	89.2	87.3	40.4	55.5
China Camp and MMWD (both sites)	California black oak	32	96.9	87.3	19.3	26.7
China Camp and MMWD (both sites)	Coast Live Oak	507	93.3	88.8	27.3	30.0

Every *Q. agrifolia* that died between March 2000 and March 2001 had symptoms of sudden oak death syndrome. The overall mortality for *Q. agrifolia* in China Camp State Park rose from 7.5 percent (22 trees) in 2000 to 14.7 percent (43 trees) in 2001 (table 2). Variation in mortality by plot was 0 to 14.3 percent in 2000 and 0 to 28.6 percent in 2001. In MMWD, overall mortality of *Q. agrifolia* was 5.6 percent (12) in 2000 and 8.4 percent (18) in 2001. Mortality varied among plots from 0 to 23 percent in 2000 and from 0 to 35.9 percent in 2001. For the China Camp State Park plots, 7.8 percent (21) of the trees that were alive in March 2000 were dead by March 2001. In MMWD, 3 percent (6) of *Q. agrifolia* died after one year. For *Q. agrifolia* summed across both sites, 5.7 percent (27) of the trees died during this period.

Table 2—Mortality of coast live oak, California black oak, and tanoak in Marin Co. plots, March 2000 and March 2001. Only trees with seeping cankers were included in these figures.

Species	Site	Dead trees pct	
		2000	2001
Coast live oak	China Camp	7.5	14.7
	MMWD	5.6	8.4
Tanoak	MMWD	12.1	14.6
California black oak	China Camp plus MMWD	3.1	6.3
Coast live oak	China Camp plus MMWD	6.9	12.0

The percentage of symptomatic *L. densiflorus* trees in MMWD increased from 40.4 percent in 2000 to 55.5 percent in 2001. Every tree recorded as dead in March 2000 exhibited the seeping symptom. The percentage of symptomatic trees among plots varied from 20 percent to 63.6 percent in 2000 and from 23.3 percent to 77.8 percent in 2001. Mortality among *L. densiflorus* rose from 12.1 percent (19) in 2000 to 14.6 percent (23) in 2001. The variation in mortality among plots was 0 to 23 percent in 2000 and 0 to 25.6 percent in 2001. Between March 2000 and March 2001, 2.5 percent (4) of the *L. densiflorus* in the plots died.

Quercus kelloggii, although not uncommon in both study sites, is under-represented in the plots (32 out of 760 trees). For this reason, data from both sites

were pooled. The percentage of symptomatic trees rose from 19.3 percent in 2000 to 26.7 percent in 2001. One tree died during the year, doubling the mortality level to 6 percent. The relative scarcity of this species in the plots precludes meaningful site-level discussion.

The abundance of symptomatic *Q. agrifolia* in China Camp State Park varied through the monitoring year (fig. 2). The decline in the total number of asymptomatic trees reflects the increasing number of symptomatic trees that died, plus those newly identified as seeping. For *Q. agrifolia* in MMWD (fig. 3), the trend is similar. When symptomatic and dead *L. densiflorus* are compared, the numbers of dead trees increased slightly, while the numbers of symptomatic trees increased more dramatically (fig. 4).

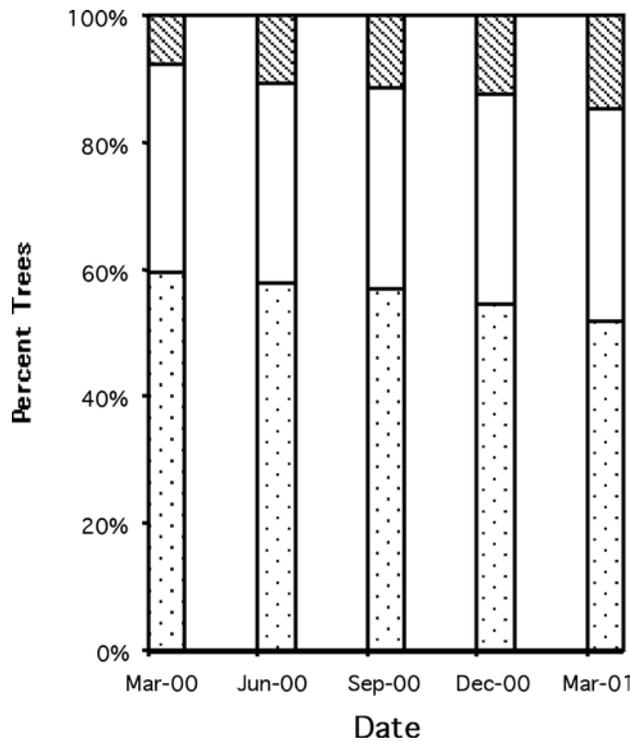


Figure 2—Percentages of coast live oaks in China Camp State Park study plots that are asymptomatic (stippled bars), have seeping cankers (clear), and are dead (cross hatched) at five sampling dates.

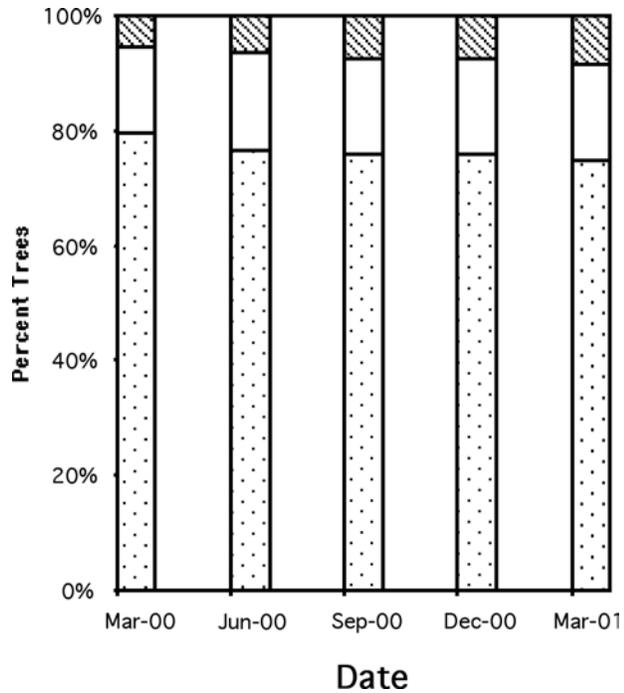


Figure 3—Percentages of coast live oaks in Marin Municipal Water District study plots that are asymptomatic (stippled bars), have seeing cankers (clear), and are dead (cross hatched) at five sampling dates.

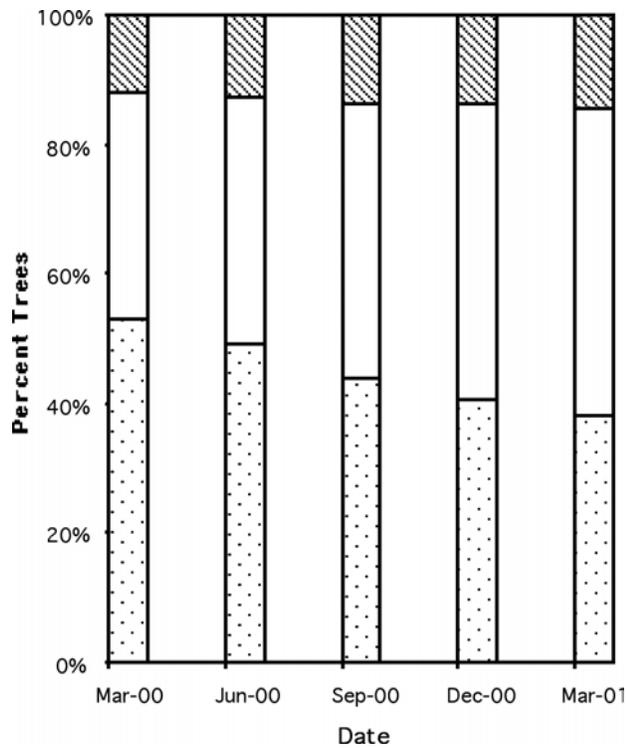


Figure 4—Percentages of tanoaks in Marin Municipal Water District study plots that are asymptomatic (stippled bars), have seeing cankers (clear), and are dead (cross hatched) at five sampling dates.

The association of bark and ambrosia beetles with symptomatic trees appears to be more consistent in *Q. agrifolia* than in *L. densiflorus*. For *Q. agrifolia* in China Camp State Park, the percentage of seeping trees that had been colonized by bark beetles alone was 19.8 percent (19) in March 2000 and 11.2 percent (11) in March 2001 (table 3). Among the symptomatic *Q. agrifolia*, those that exhibited both beetles and fruiting bodies of *H. thouarsianum* constituted 26.0 percent (25) in 2000 and 47.9 percent (47) in 2001. All 21 *Q. agrifolia* in China Camp State Park plots that died between March 2000 and March 2001 had been colonized by bark beetles while the foliage was green and apparently functional.

Table 3—The co-occurrence of bark beetles and *H. thouarsianum* with seeping trees, March 2000 and March 2001. Numbers in boldface denote the presence of bark and ambrosia beetles in seeping trees. Where percentages are reported in the text, these are in reference to the total number of seeping trees.

Signs and symptoms associated with sudden oak death syndrome	Coast live oak, China Camp		Coast live oak, MMWD		Tanoak, MMWD	
	2000	2001	2000	2001	2000	2001
Year	2000	2001	2000	2001	2000	2001
Number of living trees	271	254	202	196	140	137
Number of Seeping Trees	96	98	33	37	56	76
Seeping only	46	40	18	24	49	62
Beetles only	0	1	1	0	1	0
Hypoxylon only	5	3	1	0	0	0
Seeping + Beetles	19	11	10	2	5	7
Seeping + <i>Hypoxylon</i>	6	1	1	1	1	0
Beetles + <i>Hypoxylon</i>	1	0	0	0	0	0
Seeping + Beetles + <i>Hypoxylon</i>	25	47	4	10	1	6

In MMWD, the percentage of seeping *Q. agrifolia* that had been colonized by bark beetles but which did not exhibit signs of *H. thouarsianum* was 30.3 percent (10) in March 2000 and 5.4 percent (2) in March 2001. The fraction of seeping trees that exhibited both beetles and fruiting bodies of *H. thouarsianum* constituted 10.8 percent (4) in 2000 and 27.0 percent (10) in 2001. All six symptomatic *Q. agrifolia* that died during this period were colonized by bark beetles prior to dying. These data show that bark beetles and *H. thouarsianum* fruiting bodies are rare on living trees in the absence of the seeping cankers characteristic of *P. ramorum*. For all *Q. agrifolia* monitored (both sites), one tree was found colonized by beetles in the absence of the seeping symptom in March 2000 and in March 2001 (table 3). The reproductive structures of *H. thouarsianum* are commonly seen on dead stems and branches on living oaks, fallen logs, and standing dead oaks. However, in the absence of bleeding cankers, the number of living *Q. agrifolia* in all the plots exhibiting these fruiting bodies was six in March 2000 and three in March 2001. These trees were characterized by generally poor appearance and the fruiting bodies of other fungi were often present.

The association of seeping *L. densiflorus* with bark beetles was weaker than for *Q. agrifolia*. In March 2000, 10.2 percent (5) and in March 2001, 11.3 percent (7) of seeping trees in the plots had been colonized by these insects (table 3). The percentage of seeping *L. densiflorus* with the combination of beetles and *H.*

thouarsianum fruiting bodies was 2.0 percent (1) in 2000 and 9.7 percent (6) in 2001. One *L. densiflorus* was identified with beetles in the absence of the seeping symptom.

The point-centered quarter analysis for the forest in China Camp State Park provided estimates for both alive symptomatic (30 percent) and dead (14 percent) *Q. agrifolia* (n=174) in August 2001 that are broadly consistent with those derived from the symptom progression plots, 38 percent and 13 percent, respectively, in March 2001. For *Q. kelloggii* (n=45), the point-centered quarter estimates for symptomatic (21 percent) and dead (16 percent) trees appear similar to the plot-based results of 27 percent and 6 percent, respectively.

Discussion

Sudden oak death is not as sudden as implied by both the name and general impressions. Once seeping cankers develop, trees may live for two years or more, although they may also die sooner than this. Observations are yet insufficient to fully address this issue. Trees may survive for several years or more once seeping cankers appear, but whether they can recover from an infection is unknown. There is a general sequence of the appearance of symptoms and associated organisms. For *Q. agrifolia* and *Q. kelloggii*, bleeding cankers precede foliage symptoms and the appearance of *H. thouarsianum* and bark beetles (McPherson and others 2000). Bark and ambrosia beetle colonization clearly follows the appearance of bleeding cankers. The longer a tree has seeping cankers, the greater is the likelihood of colonization by beetles. *Hypoxylon thouarsianum* is apparently present as an endophyte in a majority of *Q. agrifolia* surveyed in northern California (Chapela, personal communication). As the production of fruiting bodies is associated with drying out of wood infected by this fungus, their presence on a tree with green foliage indicates that its health has been impaired. For *L. densiflorus*, the initial symptom is often flagging of branch tips and scattered leaf death, which is then followed by a sequence of symptoms similar to those of *Q. agrifolia* (McPherson and others 2000). Because our estimates for numbers of symptomatic *L. densiflorus* are based on the presence of seeping cankers, this probably underestimates the true level of symptomatic trees. Changes in the foliage of the deciduous *Q. kelloggii* are less reliable indicators of sudden oak death syndrome, but the other symptoms also follow the pattern observed for *Q. agrifolia*.

The emergence of new diseases, or of newly recognized diseases, appears to be a feature of the modern global economy. Once a pathogenic agent becomes established in populations of susceptible host plants that lack evolved resistance, the effects can be catastrophic. Examples of introduced diseases in North American forests include chestnut blight, Dutch elm disease, white pine blister rust, beech bark disease, and pitch canker, to name the most obvious. The symptom progression plots described here were not started prior to the introduction of *P. ramorum* and development of sudden oak death. The mix of low to high infection levels they represent can illustrate different stages in the development of the epidemic. The increasing numbers of both symptomatic (seeping) and dead trees in these plots, even in forests where trees were observed dying more than five years ago, indicates that trees are still being infected. It is also possible that trees inoculated prior to the establishment of these plots developed cankers much later. Information on baseline, pre-epidemic mortality levels in these forests is lacking, and as a result, the true effects of this epidemic may be difficult to quantify. However, monitoring forest

stands at different stages in the course of this disease provides an opportunity to understand forest dynamics under the influence of this apparently novel pathogen.

Bark and ambrosia beetles are found in association with *Q. agrifolia*, *Q. kelloggii*, and *L. densiflorus* trees that have symptoms of sudden oak death. This association with only the seeping areas on trees that have asymptomatic foliage suggests a new relationship between these insects and their living host trees, mediated by a novel pathogen. Their normal substrate is recently killed trees, fallen or standing, or individual dead branches on living trees. These insects have not been previously reported to colonize the main stem of trees with apparently healthy, green foliage. Both species of ambrosia beetles are reported to be rare (Chamberlain 1960), yet large increases in their populations have occurred where the epidemic is present (Svihra 1999c). The scarcity of these organisms on *Q. agrifolia* trees that do not show symptoms of sudden oak death is in contrast to their abundance on infected trees (table 3). The ability of these beetle species to infest and kill disease-weakened trees when present in large numbers is unknown. However, in the case of other scolytids, large populations produced during epidemics enable the beetles to overcome tree defenses and infest apparently healthy trees (Wood 1982). It is well documented that bark beetles more readily kill trees infected with root pathogens than uninfected trees (Goheen and Hansen 1993). Our preliminary data show that during the one year of monitoring *Q. agrifolia* trees, 1) bark and ambrosia beetles were scarce in the absence of sudden oak death syndrome, as detected by the presence of seeping cankers, 2) the trees that became colonized by these beetles during the year of monitoring also exhibited seeping as the initial symptom of sudden oak death syndrome, and 3) all trees with sudden oak death syndrome that died were previously colonized by these beetles and also had fruiting bodies of *H. thouarsianum* on the main stem. Despite concerted efforts to find bark beetles in trees that lacked symptoms of sudden oak death syndrome, this remained a rare event.

Observations in MMWD plots indicate that the abundance of bark and ambrosia beetles on infected trees varied considerably among sites. This variation may be due to local population size and site factors such as local tree species composition. It is also likely that the variation in bark beetle population levels at different sampling times reflects seasonal variation in temperature and rainfall.

Data collected from the symptom progression plots will be coupled with multispectral reflectance data collected from remote imaging (Kelly and McPherson 2001). The goal is to correlate tree health status characterized by symptoms obtained from ground observations, such as seeping and beetles, with the remote imagery. Research continues to explore the possibility of detecting trees with sudden oak death syndrome prior to the appearance of visible foliage symptoms.

Conclusions

In forested areas where sudden oak death syndrome and the *P. ramorum* pathogen have been established for several or more years, we have identified a number of trees that were symptom-free at the beginning of the study and subsequently developed the initial seeping symptoms. The fact that these apparently healthy trees developed symptoms recently argues for caution in ascribing the presence of symptom-free trees in such stands to disease resistance.

Although the role of bark and ambrosia beetles in tree death is unclear, several lines of evidence suggest that these insects may hasten this process. The percentage of *Q. agrifolia* trees with symptoms of sudden oak death that were colonized by these beetles, singly or in combination with *H. thouarsianum*, while maintaining green foliage, varied from 42.4 percent to 59.2 percent in 2000 and 2001. This is in contrast to the scarcity of these beetles in non-symptomatic trees. Every *Q. agrifolia* that died with symptoms of sudden oak death syndrome had been colonized by beetles prior to dying. Since bark beetles are associated with the decline and death of numerous tree species following infection by pathogenic fungi, this association is consistent with other tree-pathogen interactions (Goheen and Hansen 1993).

The analysis of these plots confirms the impression that the distribution of this disease complex is patchy. This pattern may be a consequence of the relatively early stage of the epidemic in forests of coastal California. It is also possible that the presence of other host species influences the level of infection occurring in oaks and tanoak.

The symptomology in *Q. kelloggii* appears to be similar to that in *Q. agrifolia*,⁶ although the results presented here are not sufficient to document this. *Lithocarpus densiflorus* populations appear to be more susceptible to *P. ramorum* than the oak species studied. This conclusion is suggested by the higher level of infection found in these plots and by the high levels of sudden oak death symptoms observed in sapling *L. densiflorus* (McPherson, personal observation). On the other hand, the oak species are relatively scarce in the understory, and thus under-represented in the symptom progression plots.

These study plots were initiated when the known geographic range of the epidemic included Big Sur to the south and Sonoma County to the north. This distribution indicates that the pathogen had already been established for some unknown period of years. Thus, one year of data is insufficient for any trends to be detected. Yearly climatic variation may alter such variables as the mean response of trees to *P. ramorum*, beetle population size and species composition, and the transmission of disease propagules. Based on current apparent infection levels and observed mortality levels, significant mortality is likely for years to come, even in areas where the disease appears to have been established for five or more years.

The plots described here will be monitored quarterly for the next several years. These plots will also be used for additional ecological assessments of long-term vegetation changes that are likely to follow this epidemic. As eastern North American oak species appear to be highly susceptible to this pathogen, the response of the California forests may be predictive of forests in other regions if the pathogen becomes established elsewhere.

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