Forest Ecology and Management 257 (2009) 1271–1280

Contents lists available at ScienceDirect

Forest Ecology and Management

journal homepage: www.elsevier.com/locate/foreco

Forest stand dynamics and sudden oak death: Mortality in mixed-evergreen forests dominated by coast live oak

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ARTICLE INFO

Article history:
Received 14 May 2008
Received in revised form 19 November 2008
Accepted 22 November 2008

Keywords:
Phytophthora ramorum
Quercus agrifolia
Oak mortality
Stand reconstruction
Forest response
Forest disease
Invasive

ABSTRACT

Sudden oak death (SOD), caused by the recently discovered non-native invasive pathogen, *Phytophthora ramorum*, has already killed tens of thousands of native coast live oak and tanoak trees in California. Little is known of potential short and long term impacts of this novel plant–pathogen interaction on forest structure and composition. Coast live oak (*Quercus agrifolia*) and bay laurel (*Umbellularia californica*) form mixed-evergreen forests along the northern California coast. This study measured tree mortality over a gradient of disease in three time periods. Direct measurements of current mortality were taken during 2004, representing a point-in-time estimate of present and ongoing mortality. Past stand conditions, c. 1994, were estimated using a stand reconstruction technique. Future stand conditions, c. 2014, were calculated by assuming that, given a lack of host resistance, live trees showing signs of the disease in 2004 would die. Results indicate that coast live oaks died at a rate of 4.4–5.5% year^{-1} between 1994 and 2004 in highly impacted sites, compared with a background rate of 0.49% year^{-1}, a ten-fold increase in mortality. From 2004 to 2014, mortality rates in the same sites were 0.8–2.6% year^{-1}. Over the entire period, in highly impacted sites, a 59–70% loss of coast live oak basal area was predicted, and coast live oak decreased from 60% to 40% of total stand basal area, while bay laurel increased from 22% to 37%. Future stand structures will likely have greater proportions of bay laurel relative to coast live oak.

1. Introduction

Forest invasions by non-native pests and pathogens, and the resulting decline in native species and communities, constitute important ecological, economic, and evolutionary processes in the 21st century (Castello et al., 1995; Everett, 2000; Ellison et al., 2005). Introduced forest pests and pathogens have become increasingly common in the past century and include such well-known examples as chestnut blight (caused by *Cryphonectria parasitica*), Dutch elm disease (caused by *Ophiostoma* spp.), and the introduced hemlock woolly adelgid (caused by *Adelges tsugae*). Current patterns of globalization suggest this trend will continue worldwide (Liebhold et al., 1995). In addition, today's outbreaks occur at a time when forests face a suite of novel threats, including nitrogen deposition (Vitousek et al., 1997), altered fire regimes (Franklin and Dyrness, 1973; Agee, 1993), abundant supplies of non-native invasive plants (Liebhold et al., 1995; Vitousek et al., 1997), habitat loss and fragmentation (Noss, 1987; Bennett, 1999), and a changing climate.

Loss of the dominant tree can have variable impacts on a forest stand, depending on factors related to host, pathogen, and the environment in which they interact. Despite historic and ongoing introductions of pathogens into forested ecosystems, there remains a critical lack of information regarding the impact of these invasions on forest composition (Orwig, 2002), and the response of temperate forest ecosystems to the selective removal of a dominant tree (Erlich and Mooney, 1983; Castello et al., 1995; Orwig and Foster, 1998; Ellison et al., 2005). A central reason for this is the limited pre-disease information in many forested ecosystems. Pre-disease stand structure, background mortality and early-stage mortality rates are critical to assessing impacts of non-native pathogen invasions, and to highlighting issues of management concern.

Stand reconstruction methods have been used for diverse purposes. They have been employed to compare the benefits of even- and uneven-aged management in Douglas-fir stands (Miller and Emmingham, 2001), and to test hypotheses about stand development theory including questions of size, frequency, and type of disturbance (Johnson et al., 1994). Reconstructions have been used to study past forest structure and dynamics and to establish reference conditions for managing present forest ecosystems (Pedersen and McCune, 2002). Several studies have used reconstruction to investigate past conditions in stands

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doi:10.1016/j.foreco.2008.11.030

undergoing outbreaks of non-native invasive pathogens (Busing and Clebsch, 1988; Fajvan and Wood, 1996; Waring and O'Hara, 2008).

Sudden oak death (SOD), caused by the non-native invasive pathogen *Phytophthora ramorum*, is an emerging forest disease that has killed tens of thousands of trees in 12 coastal counties of central and northern California (Rizzo and Garbelotto, 2003). Its range continues to spread and vulnerable forests exist state and nation-wide (Rizzo et al., 2002; Meentemeyer et al., 2004). At present, SOD's impact has been greatest in two mixed-evergreen ecosystems, the redwood–tanoak forests—due to mortality of tanoak (*Lithocarpus densiflorus*), and the coast live oak-bay laurel (*Umbellularia californica*) forests—due to mortality of coast live oak (*Quercus agrifolia*).

Functioning and intact oak-dominated ecosystems are critical resources in California. Important functions of these communities range from water filtration and nutrient cycling (Dahlgren et al., 1997; Querejeta et al., 2007), carbon storage (Gaman, 2008), and soil formation and erosion prevention (Ridolfi et al., 2000), to recreational, aesthetic, and symbolic values. Oak woodlands also have among the highest levels of biodiversity of terrestrial ecosystems in California, with at least 300 vertebrate terrestrial species, 5000 arthropod species, and 1100 native vascular plant species associates (Garrison, 1996; Swiecki and Bernhardt, 2001). Oaks span many of California's diverse climatic zones and define the landscape for many of its residents. Coast live oak-dominated woodlands and forests cover 1,939,770 acres (784,997 ha) of California; of these 1,277,630 acres (517,039 ha) are in the Central Coast region and 217,650 acres (88,080 ha) are in the North Coast region (FRAP, 2003). Both regions include counties known to have SOD.

Little is known of the potential short and long term impacts of SOD on forest structure and composition (Rizzo and Garbelotto, 2003), particularly in the early stages of the disease. In coast live oak-bay laurel forests, substantial changes may be expected for several reasons. First, in these forests, SOD causes mortality of coast live oak, the dominant tree (defined by highest proportions of basal area and canopy cover compared to co-occurring species), as well as of black oak (*Quercus kelloggii*), a smaller component of the canopy. Second, research indicates that SOD disproportionately kills larger trees (McPherson et al., 2005). Third, the co-dominant tree, bay laurel, is a critical driver of the disease, especially in mixed-evergreen forests dominated by coast live oak (Davidson et al., 2005). On it, *P. ramorum* causes a non-lethal foliar infection and sporangia production is higher than found in other species (Davidson et al., 2005; Garbelotto and Rizzo, 2005). Fourth, the majority of other co-occurring tree species, including Pacific madrone (*Arbutus menziesii*), bigleaf maple (*Acer macrophyllum*), California buckeye (*Aesculus californica*), Douglas-fir (*Pseudotsuga menziesii* var. *menziesii*), toyon (*Heteromeles arbutifolia*), and California manzanita (*Arctostaphylus californica*), are also foliar hosts capable of contributing *P. ramorum* inoculum to the environment. Only two co-occurring trees, foothill pine (*Pinus sabiniana*) and Oregon oak (*Quercus garryana*), are not foliar hosts. Evidence for variation in resistance of coast live oak to *P. ramorum* has been found, suggesting that less susceptible genotypes may be discovered in the future to provide the gene pool for regeneration (Dodd et al., 2005).

Sudden oak death is a relatively new disease in California forests and is still spreading. The first symptoms – only later associated with SOD – were not officially reported until 1994 in tanoak and 1995 in coast live oak (Svihra, 1999; McPherson et al., 2005). Anecdotal evidence suggests that the first infections may have occurred as early as 1989. Several heavy rain events during the mid-1990s, including the El Niño years of 1993 and 1998, may have resulted in widespread SOD infection and mortality 2 or 3 years later (Rizzo and Garbelotto, 2003). However, the first round of funding for SOD research was not made available until 1999 (Garbelotto and Rizzo, 2005), with the earliest monitoring efforts initiated in coast live oak stands in 2000 (Swiecki and Bernhardt, 2002; Kelly and Meentemeyer, 2002; McPherson et al., 2005). These studies did not account for downed logs in mortality estimates. Subsequent evidence presented by Swiecki et al. (2005) suggests that trees infected by *P. ramorum* have a greater likelihood of becoming colonized by secondary fungi and insects, causing these trees to fall.

The present study employs a stand reconstruction to measure pre-disease forest structure and capture early mortality in the form of fallen trees in SOD-impacted coast live oak-dominated forests. Such information serves as a reference, and will improve assessment of disease effects and allow for prediction of stand conditions following loss of coast live oak, ultimately leading to better management decisions. Our main objectives in the study were to (1) investigate stand composition and stand density over a gradient of SOD infection across a period from 1994 to 2014, (2) quantify species-specific mortality during this period, and (3) compare calculated mortality rates with background rates from the literature. These findings are used to predict how SOD will influence future stand dynamics and succession in coast live oak-bay laurel mixed-evergreen forests.

2. Methods

2.1. Plot establishment

This study was conducted at eight sites in the greater San Francisco Bay Area in California (Marin, Sonoma, and Contra Costa counties) on a mix of public and private lands (Fig. 1). Sites were chosen to represent a gradient of visible manifested signs of SOD infection (described below), and to monitor vegetation change over this gradient. Additional criteria were at least 30% and 10% relative cover by coast live oak and bay laurel trees, respectively, as well as accessibility to a road. Because of the patchiness of the disease in known infected areas, an additional criterion in infected sites was some known mortality. Skywalker (SKY) and both China Camp State Park sites (CC1, CC2) showed the most signs of SOD infection while both Briones East Bay Regional Parks sites (EB1, EB2) showed none. Olompoli State Park (OLO), Fairfield Osborn Preserve (FOP), and Bouverie Preserve (BVR) were intermediate.

At the eight 1 hectare (ha) sites, five 0.08 ha plots were randomly located. Slope, aspect, and elevation were measured at plot center in each plot. Throughout each plot, all live stems, dead standing stems (snags), and logs equal to or greater than 10 cm diameter at breast height (dbh) were tagged. On live stems and snags, dbh was measured at 1.37 m (4.5 ft) above the ground. If a live stem or snag forked below 1.37 m, each resulting stem (greater than 10 cm dbh) was tagged. Stem dbh was not re-measured each year so any increase (or decrease) was not factored into basal area estimates. Basal area of each log was taken at a combined distance of 1.37 m, subtracting the height of the remaining stump.

2.2. Time period overview

Stand conditions were tabulated for three different time periods. First, to investigate SOD mortality in the present period, direct measurements representing a point-in-time estimate of current mortality were taken during 2004. Second, a stand reconstruction was employed to quantify oak mortality attributed to SOD that occurred before monitoring efforts began in 2000–2002 and to estimate past stand conditions. The past period extends from approximately 1994, with absent or very early levels of SOD infection, through 2004. Third, stand conditions 10 years in

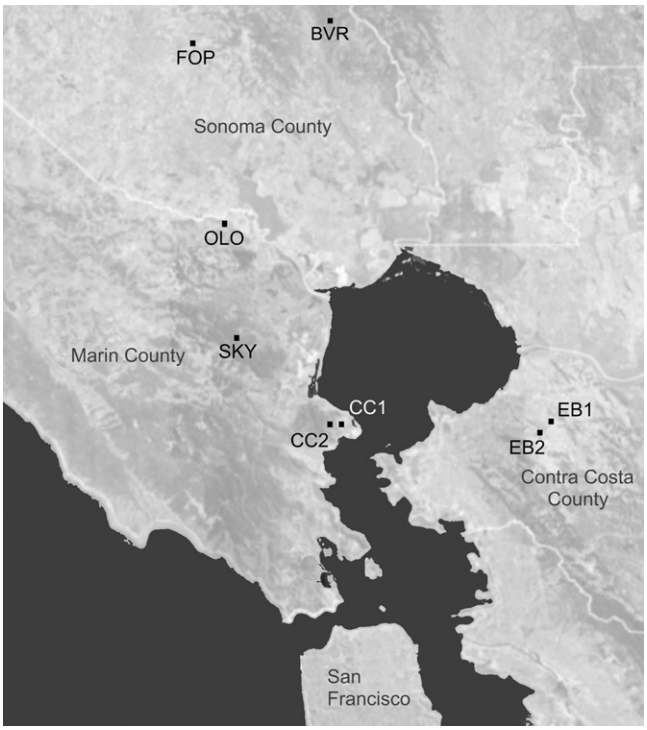

Fig. 1. Map of study sites, including Skywalker (SKY), China Camp State Park Miwok Meadows (CC1), China Camp State Park Back Meadows (CC2), Olompoli State Park (OLO), Fairfield Osborn Preserve (FOP), Bouverie Preserve (BVR), Briones East Bay Regional Park Alhambra (EB1), and Briones East Bay Regional Park Bear Creek (EB2). Study sites are in the Marin, Sonoma, and Contra Costa counties in California.

the future, 2014, were estimated by assuming that, given a lack of host resistance, live trees showing signs of the disease in 2004 would die, and that current infection rates would continue.

2.3. Stand reconstruction methods

A variety of techniques are available to perform stand reconstructions, including remeasurement of permanent plots, tree ring series, and decay-class analysis (Maser et al., 1979); in this study, the log decay-class system, as first outlined by Triska and Cromack (1979), was used. The system assigns downed logs to one of several decay classes based on visual indicators, such as bole shape, degree of fragmentation, fungal colonization, and retention of fine twigs and leaves. The system then ties each stage to a number of years since death. This requires an index of log decomposition rates as these vary across species and environment (Pyle and Brown, 1998). However, while a considerable number of studies of log decomposition exist for eastern hardwood forests (Harmon, 1982; Hale and Pastor, 1998; Pyle and Brown, 1998; Wilson and McComb, 2005) and coniferous forests of the Pacific Northwest (Maser et al., 1979; Franklin et al., 1981; Graham and Cromack, 1982; Sollins, 1982), no such studies were found to exist in western hardwood forests, nor in California coastal hardwood forests in particular.

All logs were inspected and assigned a class based on visual indicators (Table 1). The 'recently fallen' designation was given to logs that still retained their foliage or their fine twigs. This is commensurate with a decay class of 1 or 2 using the Hofgaard decay classes for logs (Hofgaard, 1993), which corresponds with approximately 14 years since death (Storaunet and Rolstad, 2002). Modifying the Hofgaard system based on other studies in northern hardwood ecosystems (e.g. Hale and Pastor, 1998), it was conservatively estimated that such stems had died within the last 10 years. All recently fallen logs and dead standing stems were added to the current number of live stems for a determination of past stand density and species composition.

2.4. Future predictions

Future stand estimates were calculated as a sum of predicted mortality caused by SOD and predicted background mortality. The SOD component was determined by subtracting all presently live but seeping stems from live, non-seeping stems. The background component was based on Barrett's (2006) calculated natural (not including trees harvested, girdled or sprayed with herbicide) mortality rates for 9 SOD host tree species over the period from approximately 1984 to 1994. Using data from the statewide inventory of forestland in California USDA Forest Service Forest Inventory and Analysis (FIA) program, rates were calculated as percentages of initially live trees lost over the period of 10 years. Annual mortality was calculated as a simple arithmetical average over the remeasurement interval, rather than an annually compounded value. Annual mortality rates were 0.49%, 0.69%, 0.56%, and 0.58% for coast live oak, bay laurel, madrone and black oak, respectively. For the small component of trees defined in this study as 'other species', we used the average mortality rate calculated in Barrett (2006) for all California tree species over the time period, which was 0.5%. These mortality rates were not used for the past to present period estimates as our methods captured both background and SOD-related mortality during this time.

The choice of the 10-year range (2004–2014) for future projections is commensurate with the literature on survival time following seeps. McPherson et al. (2005) estimated 2.6 ± 0.32 (S.E.) to 7.5 ± 1.6 (S.E.) years survival time following initiation of seeps in coast live oak. Using the upper range of the survival period for seeping trees, we conservatively inferred that trees seeping in 2004 would be dead in 2014.

2.5. Epidemiological methods

To verify coast live oak infection with *P. ramorum*, two live coast live oaks were selected from each plot for sampling. Wood samples were removed from the base, breast height, and any visible seeps on the bole, ranging from two to six samples per tree. With support of the UC Berkeley Forest Pathology and Mycology lab, samples were collected and stored for PCR and culture analysis to test for presence of *P. ramorum* DNA. PCR analysis was conducted by the Forest Pathology and Mycology lab, UC Berkeley, and culture analysis was conducted by the Allen-Diaz lab with support of the Forest Pathology and Mycology lab, UC Berkeley

Table 1
Indicators for stand reconstruction classes are shown. Shown are visual indicators, decay class, and approximate number of years since falling used in this study, based on Hofgaard (1993) decay classes from *Picea abies* boreal forests, modified by northern hardwood studies (e.g. Hale and Pastor, 1998). Stems with decay greater than class 2 were considered not to have been killed by *P. ramorum*.

Decay class	Description of logs	Approx. # years since death
1 or 2: Recently fallen	Leaves and/or small twigs still present. Bark present. Solid wood. Species easily identifiable.	~Up to 10
3 or more: Older fallen	Any of above not true.	~10 or more

Table 2
Study site summary characteristics including elevation (average in meters), aspect (range in degree and major cardinal direction), and slope (range and average in percent) were measured at plot center in each plot. The final column indicates change in live coast live oak basal area from the past to present period, divided by 2004 total live tree basal area. Both measures were taken in $m^2\ ha^{-1}$. This metric was used to order sites along a SOD-impact gradient (from low to high SOD impact).

Site	Elevation (average) (m)	Aspect (degree)	Aspect (direction)	Range slope (%)	Average slope (%)	SOD-impact gradient
EB2	239.8	260–300	W–NW	20.5–61.0	47.7	0.03
EB1	370	358–85	N–E	33.0–62.5	47.2	0.04
OLO	183.7	310–350	NW	10.3–25.0	21	0.05
BVR	191	268–300	W	25.0–40.0	33.4	0.08
FOP	440.3	220–320	SW–NW	21.0–28.5	23.5	0.09
CC2	49.4	82–165	E–S	22.0–43.5	33.8	0.35
CC1	74.4	200–5	SW–N	30.5–41.0	37.3	0.49
SKY	115	155–184	S	22.0–43.5	34.6	0.5

and the Rizzo lab, UC Davis. These data were taken in the late spring of 2003 and 2004. *P. ramorum* was confirmed from live coast live oak stems in all but the Briones sites. Due to the low rate of success in isolating *P. ramorum* from dead woody tissue of coast live oak, dead logs and dead standing stems were not tested for *P. ramorum*.

Because not all terminal host trees could be tested using molecular diagnostic tools, visual symptoms were used as proxies. For all live coast live and black oak stems, a suite of epidemiological information was collected including foliar characteristics (live, failing or dead leaves) and presence of *Hypoxylon thouarsianum* (a secondary fungus associated with *P. ramorum*), presence of frass, a boring dust resulting from bark beetle tunneling and associated with *P. ramorum* infection (see McPherson et al., 2005 for additional detail), and visual presence of seeps. These data were taken each spring in 2002, 2003 and 2004 for five of the sites (Skywalker, China Camp sites, and Briones sites) and in 2003 and 2004 for the other three sites (Bouverie Preserve, Olompoli and Fairfield Osborn Preserve).

Potentially damaging or fatal additional pathogens were likely to have been present in all sites. A recently isolated pathogen, *P. nemorosa*, has been found to cause bleeding cankers in coast live oak, although it appears to be much less consistently fatal in oaks than *P. ramorum* (Hansen et al., 2003). Although *P. nemorosa* was found on stems at the Briones sites, these stems were not included in estimates of future mortality. Other pathogens (e.g. *Pythium* spp.) can cause seeping on coast live oak stems. Primary wood decay fungi such as *Phellinus* spp. and *Inonotus andersonii* are found in or near some or all of our sites and can cause damage to coast live oak trees. Notably, Swiecki et al. (2005) found that fruiting bodies of various decay organisms were significantly more common among *P. ramorum*-infected trees (based on visual signs of *P. ramorum* cankers) than controls. Such additional pathogens were treated as components of background mortality.

2.6. Methods for establishment of the SOD-impact gradient

An index of SOD impact was developed to create a ranking of SOD severity across study sites. To do this, the basal area of coast live oak snags and recently dead logs was divided by the total live stem basal area ($m^2\ ha^{-1}$) for each site. The resulting order of sites (Table 2), from low- to high SOD impact, was used in all tables and figures in this paper.

2.7. Statistical methods

One-way ANOVA was used to test for species-specific differences among sites for the following metrics: (1) past, present and future basal areas, (2) present stem counts, (3) recently downed log basal areas, and (4) mortality rates. When ANOVA identified significant differences in rates of change, the Tukey Honestly Significant Differences test, which assesses all pair-wise comparisons of sites, was employed to determine where significant differences among sites occurred. All statistical tests were completed using S Plus 6.1 (SAS, 2002).

3. Results

3.1. Environmental variables

Slope, aspect and elevation varied among sites (Table 2). Slopes (for individual plots) ranged from 10% to 61%, with site averages ranging from 21.0% to 47.7%. Elevation ranged from 49.4 to 440.3 m, with an average of 208 m. Aspects varied widely, and all major cardinal directions were represented.

Table 3
Present (2004) counts for all live stems were measured in stems ha^{-1} (average of 5 plots per site ±1 standard error). Standard errors are denoted in parentheses. ANOVA results (p values) are displayed in column headings, with significant differences (if present) shown in the subsequent column. "Other species" include Oregon white oak, Douglas-fir, toyon, foothill pine, California buckeye, big leaf maple, and California manzanita.

Site	Coast live oak (stems ha^{-1}) $p = 0.0035$	Significant differences	Bay laurel (stems ha^{-1}) $p = 0.0669$	Madrone (stems ha^{-1}) $p = 0.4261$	Black oak (stems ha^{-1}) $p = 0.1212$	Other spp. (stems ha^{-1}) $p = 0.0821$	Total (stems ha^{-1})
EB2	267.5 (37.0)	SKY, CC1	210 (28.6)	0	7.5 (7.5)	55 (25.9)	540
EB1	330 (37.3)	SKY, CC1	160 (24.8)	25	0	2.5 (1.2)	517.5
OLO	300 (37.0)	SKY, CC1	52.5 (28.3)	77.5 (34.3)	32.5 (13.5)	7.5 (12.1)	470
BVR	277.5 (48.6)	SKY, CC1	252.5 (51.8)	206.3 (81.3)	15 (10)	20 (13.8)	771.3
FOP	225 (38.1)	SKY, CC1	460 (163.7)	0	55 (20.4)	117.5 (49.2)	857.5
CC2	165 (37.6)	SKY, CC1	225 (71)	209.4 (99.7)	17.5 (12.2)	40 (21.1)	656.9
CC1	112.5 (43.5)	All but SKY	245 (96.8)	0	40 (17.4)	42.5 (30.2)	440
SKY	117.5 (28.4)	All but CC1	242.5 (46.4)	0	7.5 (3.1)	0	367.5
Total	1795		1847.5	518.2	175	285	4620.7
% of total	38.85%		39.98%	11.21%	3.79%	6.17%	100%

Table 4
Present (2004) basal area values of all live stems, shown in $m^2\ ha^{-1}$ (average of 5 plots per site ±1 standard error). Standard errors are denoted in parentheses. ANOVA results (p values) are displayed in column headings, with significant differences (if present) shown in the subsequent column.

Site	Coast live oak ($m^2\ ha^{-1}$)	Significant differences	Bay laurel ($m^2\ ha^{-1}$)	Madrone ($m^2\ ha^{-1}$)	Significant differences	Black oak ($m^2\ ha^{-1}$)	Other species ($m^2\ ha^{-1}$)	Total live tree BA ($m^2\ ha^{-1}$)
	p value: 0.0010		p value: 0.4775	p value: 0.0001		p value: 0.1055	p value: 0.1244	
EB2	24.76 (2.5)	CC1	10.38 (2.63)	0	OLO	1.5 (1.5)	0.77 (0.36)	37.27
EB1	29.61 (3.23)	CC1, CC2	7.72 (0.81)	0.88 (0.88)	OLO	0	0.03 (0.02)	38.28
OLO	22.47 (3.58)	–	2.81 (1.74)	8.75 (2.75)	All but CC2	6.38 (3)	0.03 (0.01)	40.53
BVR	23.82 (3.43)	CC1	8.82 (5.35)	1 (0.08)	OLO	0.63 (0.38)	2.95 (0.98)	37.33
FOP	14.49 (3.33)	–	13.01 (6.25)	0	OLO	2.50 (1)	3.77 (2.13)	33.77
CC2	12.25 (3.44)	EB1	6.09 (2.15)	5.75 (2.13)	–	2.88 (2)	0.67 (0.12)	27.54
CC1	8.2 (2.93)	EB1, EB2, BVR	7.04 (3.01)	0	OLO	4.5 (1.88)	0.90 (0.2)	20.65
SKY	17.07 (4.01)	–	12.63 (2.33)	0	OLO	0.63 (0.25)	0	30.38
Total	152.63	–	68.5	16.38	–	19	9.12	265
% of total	57.5%	–	25.8%	6.0%	–	7.1%	3.5%	100%

3.2. Forest stand structure in 2004

A total of 2306 stems were tagged and measured in this study. Coast live oak stems represented 39% of live stems, averaged across sites, in 2004 (Table 3). Bay laurel accounted for the majority with 40% of live stems. Pacific madrone accounted for 11% and black oak 4%. Other species included Oregon white oak, Douglas-fir, toyon, foothill pine, California buckeye, bigleaf maple, and California manzanita (hereafter referred to as 'other species') and these combined formed 6% of the total stem count across sites. ANOVA results indicated that the number of coast live oak stems at Skywalker and China Camp Miwok were significantly different than at other sites ($p = 0.0035$). Bay laurel, madrone, black oak, and 'other species' did not exhibit significant differences across sites (Table 3).

In 2004, coast live oak and bay laurel accounted for 58% and 26% of total live basal area of stems in the sites, respectively (Table 4). Black oak accounted for 7%, and Pacific madrone for 6%. 'Other species' accounted for 4% of the total basal area of the sites. Coast live oak basal area was significantly different ($p = 0.00001$) at China Camp Miwok compared to the Bouverie and Briones sites, and at China Camp Back Meadows compared to the Briones Alhambra site. Significant differences in 2004 madrone basal area were found between Olompoli and all but both China Camp sites ($p = 0.0001$). Other species did not exhibit significant differences among sites.

Many recently downed logs at the time of plot establishment indicated a recent flush of tree mortality. This recent mortality was comprised almost exclusively of coast live oak. As Fig. 2 indicates, out of a total basal area of 31.13 $m^2\ ha^{-1}$ of recently fallen logs in all sites, 27.85 $m^2\ ha^{-1}$ was coast live oak, accounting for 89%. Of this, 91% of the coast live oak logs were in three heavily impacted sites. ANOVA results indicate that basal area of logs was significantly different (p value = 0.0036) at Skywalker compared to all other sites but the China Camp sites. Other species did not exhibit significant differences among sites.

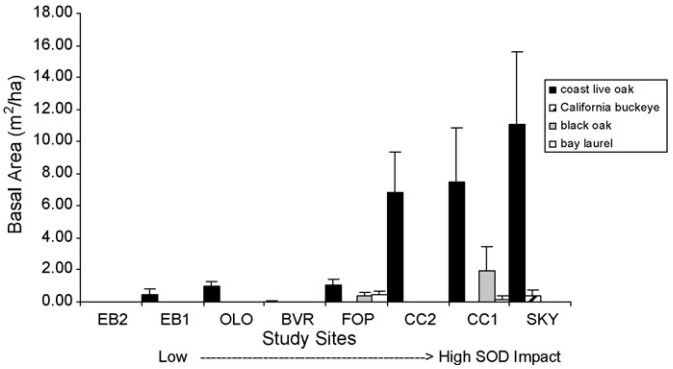

Fig. 2. Basal area of all recently downed logs, measured in $m^2\ ha^{-1}$ (average of 5 plots per site ±1 standard error), are shown. Species included coast live oak, California buckeye, black oak, and bay laurel. Significant differences in coast live oak basal area were found between Skywalker and all but the China Camp sites (p value = 0.0036). Other species did not exhibit significant differences among sites.

3.3. Past forest stand structure

In the past period, coast live oak and bay laurel accounted for 61% and 22% of the basal area across sites, respectively (Table 5).

Table 5
Past period (approximately 1994) basal area values are shown in $m^2\ ha^{-1}$ (averages ± 1 standard error), with standard errors in parentheses. ANOVA results (p values) are displayed in column headings, with significant differences (if present) shown in the subsequent column.

Site	Coast live oak ($m^2\ ha^{-1}$)	Bay laurel ($m^2\ ha^{-1}$)	Madrone ($m^2\ ha^{-1}$)	Significant differences	Black oak ($m^2\ ha^{-1}$)	Other species ($m^2\ ha^{-1}$)	Total live tree BA ($m^2\ ha^{-1}$)
	p value: 0.0707	p value: 0.4723	p value: 0.0007		p value: 0.1055	p value: 0.1021	
EB2	25.73 (2.43)	10.50 (2.6)	0	OLO	1.45 (1.45)	0.93 (0.21)	38.6
EB1	31.23 (3.63)	7.72 (0.81)	1.08 (1.08)	OLO	0	0.03 (0.01)	40.05
OLO	24.45 (3.42)	2.81(1.74)	9.48 (3.13)	All sites but CC2	6.83 (2.91)	1.7 (1.0)	45.28
BVR	26.83 (3.78)	8.82 (5.35)	1.34 (0.83)	OLO	0.62 (0.41)	3.05 (0.98)	40.65
FOP	17.6 (3.88)	14.0 (6.68)	0	OLO	3.77 (1.87)	4.00 (1.2)	39.37
CC2	21.78 (4.20)	6.21 (2.2)	6.21 (2.39)	–	3.07 (2.3)	0.9 (0.3)	38.16
CC1	18.25 (4.16)	7.92 (3.42)	2.01 (1.53)	OLO	6.37 (2.89)	0.96 (0.29)	35.51
SKY	32.13 (3.89)	12.66 (2.31)	0	OLO	0.92 (0.47)	0.38 (0.16)	46.08
Total	197.98	70.64	20.13	–	23.03	11.94	323.7
% of total	61.16%	21.82%	6.22%	–	7.11%	3.69%	100%

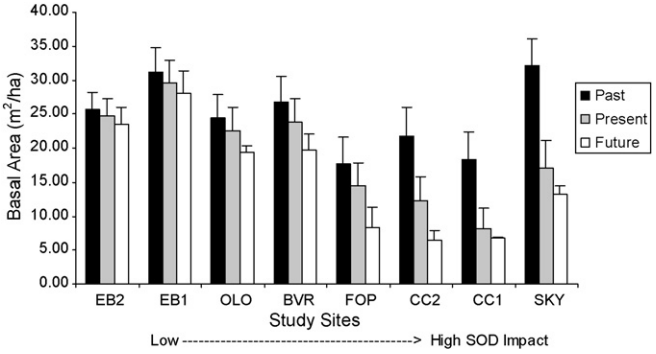

Fig. 3. Basal areas of coast live oak in the past, present, and future periods, measured in m^2 ha^{-1} (average for 5 plots per site ±1 standard error), are shown. Tables 5 and 4 present ANOVA results for the past and present periods. Future basal area was significantly different among sites ($p = 0.000013$); the Briones sites had higher values compared to Fairfield Osborn Preserve and the China Camp sites, and Briones Alhambra had higher values than the Skywalker site (also see text).

Black oak comprised 7%, madrone 6%, and 'other' species 4% of the total basal area of the sites. Madrone basal area was significantly different ($p = 0.0007$) between Olompoli and all other sites but China Camp Back Meadows. Coast live oak basal area was not significantly different ($p = 0.0707$) among sites, nor was that of any other species (Table 5).

Between the past and present time periods, sites lost variable amounts of coast live oak basal area (Fig. 3). Uninfected sites Briones Alhambra and Briones Bear Creek lost 1.6 and 1.0 m^2 ha^{-1}, or 5% and 4% of their past period coast live oak basal area respectively. Infected sites China Camp Back Meadows, China Camp Miwok, and Skywalker lost 10.0, 10.0, and 15.1 m^2 ha^{-1}, or 44%, 55% and 47%, respectively, and intermediate sites Olompoli, Fairfield Osborn Preserve, and Bouverie lost 2.0, 3.1, and 3.0 m^2 ha^{-1} or 9%, 20% and 11%, respectively. Over the same period, averaging over all sites, the mortality of bay laurel was 3% (ranging from 0% to 18%) compared with 24% (ranging from 4% to 55%) in coast live oak (Table 6).

3.4. Future forest stand structure

From the present to the future time period, the three most infected sites, China Camp Back Meadows, China Camp Miwok, and Skywalker, were projected to lose an additional 28%, 8%, and 12% or 4.7, 0.6, 2.2 m^2 ha^{-1} more coast live oak basal area, respectively. At Olompoli, Fairfield Osborn Preserve, and Bouverie the projected losses were 11%, 26%, and 14%, or 1.9, 5.2, and 2.8 m^2 ha^{-1} respectively. The Briones sites were projected to experience only background mortality levels, or a 4.9% loss of coast live oak basal area over the 10-year period (Table 6).

Projecting loss of coast live oak from the past time period (approximately 1994) to the future (approximately 2014) provides a broad picture of the potential effects of *P. ramorum* on coast live oak-bay laurel forest composition in the Bay Area. Infected sites, China Camp Back Meadows, China Camp Miwok, and Skywalker were projected to lose a total of 14.3, 10.6, and 17.3 m^2 ha^{-1} of live coast live oak basal area, or 70%, 63%, and 59% of their past period coast live oak basal area, respectively, and Olompoli, Fairfield Osborn Preserve, and Bouverie were projected to lose 3.9, 8.3, and 5.8 m^2 ha^{-1} or 20%, 46%, and 25%, respectively (Table 6, Fig. 4). The uninfected sites, Briones Alhambra and Briones Bear Creek, were projected to experience background levels of mortality only, losing 1.6 and 1.0 m^2 ha^{-1} of live coast live oak basal area, or 10% and 9% of their past total coast live oak basal area. ANOVA results identified significant differences in coast live oak basal area at the China Camp and Fairfield Osborn Preserve sites compared to the

Table 6
Mortality rates (fraction of initial live trees lost over the given period) are shown for the past to present (~1994–2004), and past to future (~1994–2014) periods based on per hectare basal areas (m^2 ha^{-1}). Standard errors are shown in parentheses. NA indicates that the particular species is missing from the site. Barrett's (2006) species-specific mortality rates (shown here as 10-year rates) are displayed in parentheses in column headers for the 20-year rates. ANOVA results (p values) are shown in column headings, with significant differences (if present) displayed in the subsequent column.

Site	10-year mortality rate (past to present)						20-year mortality rate (past to future)				
	Coast live oak $p = <0.0001$	Significant differences	Bay laurel $p = 0.0787$	Madrone $p = <0.0001$	Significant differences	Black oak (0.069) $p = 0.1055$	Coast live oak (0.049) $p = <0.0001$	Significant differences	Bay laurel (0.069) $p = 0.0787$	Madrone (0.056) $p = 0.3134$	Black oak (0.058) $p = 0.1055$
EB2	0.04 (0.01)	SKY, CC1, CC2	0.01 (0.01)	NA	NA	0	0.09 (0.01)	SKY, CC1, CC2, FOP	0.08 (0.01)	NA	0.06
EB1	0.05 (0.02)	SKY, CC1, CC2	0	0.24	CC2	NA	0.10 (0.02)	SKY, CC1, CC2, FOP	0.07 (0.00)	0.3	NA
OLO	0.09 (0.05)	SKY, CC1, CC2	0	0.05 (0.03)	CC2	0.10 (0.10)	0.20 (0.05)	SKY, CC1, CC2	0.07 (0.00)	0.11 (0.03)	0.16 (0.1)
BVR	0.11 (0.03)	SKY, CC1, CC2	0	0.23 (0.15)	CC2	0	0.25 (0.06)	CC1, CC2	0.07 (0.00)	0.29 (0.15)	0.06
FOP	0.20 (0.04)	CC1	0.06 (0.02)	NA	NA	0.19 (0.12)	0.46 (0.10)	EB1, EB2	0.13 (0.02)	NA	0.25 (0.12)
CC2	0.44 (0.07)	EB2, EB1, OLO, BVR	0.01 (0.01)	0.06 (0.04)	CC1	0.06 (0.06)	0.70 (0.05)	EB1, EB2, OLO, BVR	0.08 (0.01)	0.11 (0.04)	0.11 (0.06)
CC1	0.55 (0.13)	EB2, EB1, OLO, BVR, FOP	0.18 (0.12)	1.0	EB1, OLO, BVR, CC2	0.24 (0.15)	0.63 (0.14)	EB1, EB2, OLO, BVR	0.25 (0.12)	NA	0.3 (0.15)
SKY	0.47 (0.11)	EB2, EB1, OLO, BVR	0	NA	NA	0.15 (0.15)	0.59 (0.09)	EB1, EB2, OLO	0.07 (0.00)	NA	0.21 (0.15)
Average	0.24		0.03	0.32		0.11	0.38		0.10	0.16	0.16

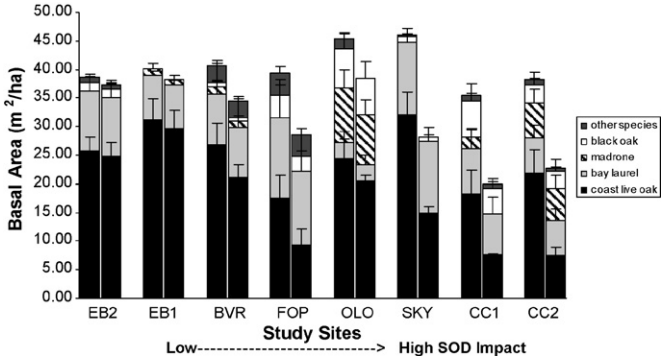

Fig. 4. Side-by-side comparisons of past and future stand basal areas (left and right columns, respectively), measured in $m^2\ ha^{-1}$ (±1 S.E.), are shown. Among sites, significant differences in change between past and future basal areas were found for coast live oak ($p = 0.0006$) (see text for differences) but not for other species, including madrone ($p = 0.3077$), black oak ($p = 0.3872$), bay laurel ($p = 0.0802$) and 'other species' ($p = 0.2099$).

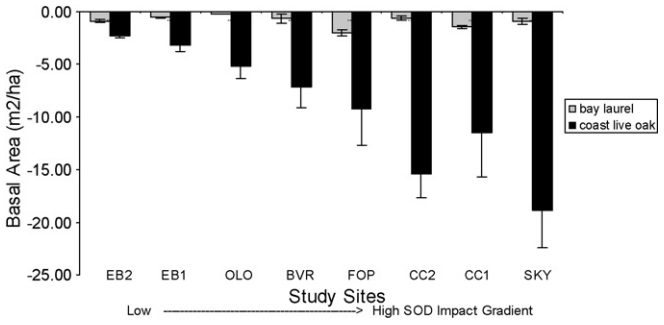

Fig. 5. Relative change in coast live oak and bay laurel basal areas ($m^2\ ha^{-1}$) (±1 S.E.) is shown between the past and future time period.

Briones sites, and at Briones Alhambra compared to Skywalker ($p = 0.000013$) (Fig. 3).

Changes to total stand basal area are shown in Fig. 4. Factoring in all species, results indicated that from past to future China Camp Back Meadows, China Camp Miwok, and Skywalker will lose 17.5, 17.3 and 20.4 $m^2\ ha^{-1}$, or 46%, 49%, and 44% of their past total tree basal area, respectively. Olompoli, Bouverie, and Fairfield Osborn Preserve will lose 9.2, 8.4, and 13.1 $m^2\ ha^{-1}$ or 20%, 21%, 33%, respectively, and Briones Alhambra and Briones Bear Creek will lose 4.0 and 3.4 $m^2\ ha^{-1}$, or 10% and 9%, respectively. Relative changes in coast live oak and bay laurel basal areas are shown in Fig. 5.

3.5. Mortality rates across periods

ANOVA results indicated coast live oak mortality rates were significantly different across sites over the 10-year period ($p = <0.0001$) (Table 6). Measured mortality rates were higher at Skywalker and the China Camp sites than at Olompoli, Bouverie, and the Briones sites. China Camp Miwok also had a higher rate than Fairfield Osborn Preserve. Additionally, over the 20-year period, coast live mortality rates were significantly different ($p = <0.0001$). Skywalker, Fairfield Osborn Preserve and the China Camp sites had higher rates than the Briones sites, and the Skywalker and China Camp sites had higher rates than Olompoli. The China Camp sites also had higher mortality rates than Bouverie. Measured mortality rates in Pacific madrone were significantly different in the 10-year period ($p = <0.0001$) but not in the 20-year period ($p = 0.3134$); one site (China Camp Miwok) with two stems that died between past and present seemed to account for this. Mortality rates in bay laurel ($p = 0.0787$) and in black oak ($p = 0.1055$) were not significantly different.

4. Discussion

Introduced pests and pathogens are among the most serious threats to the integrity of forest ecosystems. The frequency of exotic pest and pathogen outbreaks has continued to increase throughout the past century, resulting in widespread and dramatic shifts in forest structure and function (Castello et al., 1995; Liebhold et al., 1995; Ellison et al., 2005). Still, there remains a critical lack of information regarding the impact of these invasions on forest composition. In many cases, and with sudden oak death in particular, pre-disease stand conditions are poorly known and the lag between the first waves of infection and monitoring efforts raises the possibility that disease impacts have been underestimated. Reference stand structure and composition and tree mortality rates are critical to both assessing disease impacts and predicting future stand composition.

At the time of study initiation, large numbers of recently fallen logs were found in sites known to be infected with *P. ramorum*, and most of the fallen logs were coast live oak. Such pulses of downed wood have been documented in other forest types hit by non-native pathogens such as in former chestnut (*Castanea dentata*)-dominated communities due to chestnut blight fungus (McCarthy and Bailey, 1994), eastern hemlock (*Tsuga canadensis*)-dominated forests due to the hemlock woody adelgid (Benzinger, 1994), and in American beech (*Fagus grandifolia*)-dominated forests due to beech bark disease (caused by an interaction between the *Nectria* spp fungus and the scale insect, *Cryptococcus fagisuga*) (McGee, 2000). Using stand reconstruction, we found that infected study sites had already lost from 44% to 55% of coast live oak basal area when our monitoring efforts began. In contrast, counts based only on standing dead stems underestimated the degree of coast live oak mortality on our plots by one-half to two-thirds.

Stand reconstruction indicates that infected study sites began with similar coast live oak basal area, diverged in the following decade, and will continue to diverge in the future. In the three most impacted sites, coast live oak accounted for 60% of stand basal area in 1994; it decreased to 40% by 2014. Results indicate that severely infected sites will lose up to 70% of their coast live oak basal area over this period, a seven-fold increase over uninfected sites, which will lose only 9–10% over the same period. Because coast live oak is the dominant tree, this mortality accounts for almost half of the total forest stand, constituting a substantial change to forest structure.

Tree mortality rates are a fundamental characteristic of forests (Franklin et al., 1987) in that they influence forest species composition, size and age-structure of tree communities (Oliver and Larson, 1990), and are therefore critical to projecting future forest conditions (Pacala et al., 1996; Pedersen and McCune, 2002). Barrett (2006), employing data from the statewide inventory of forestland in California, conducted by the USDA Forest Service Forest Inventory and Analysis program, calculated pre-epidemic mortality rates for 9 SOD host species during the 10-year period between 1984 and 1994. Comparing these rates to the rates found in this study allows for an assessment of elevated mortality due to SOD.

Barrett (2006) reported an average rate of natural mortality for all California tree species of 0.5% $year^{-1}$, and for coast live oak of 0.49% $year^{-1}$. These rates are similar to those found in our uninfected sites, where an average rate of coast live oak mortality of 0.45% $year^{-1}$ was calculated. Our results indicate that, between 1994 and 2004, the three most impacted sites exhibited coast live oak mortality rates of 4.4–5.5% $year^{-1}$, representing a ten-fold increase over the expected coast live oak background rate. From 2004 to 2014, mortality rates in the same sites remained elevated over baseline rates but were reduced from the previous decade to

0.8–2.6% year^{-1}. This reduction is potentially attributable to genetic resistance in remaining coast live oak trees (Dodd et al., 2005), or abiotic factors like variation in yearly rainfall (Davidson et al., 2005) impacting numbers of seeps inventoried in 2004. An increase in mortality in our intermediate sites, from 0.09 to 0.20% year^{-1} in 1994–2004, to 1.1 to 2.6% year^{-1} in 2004–2014, may suggest that sites further from initial *P. ramorum* introductions in Marin County (Mascheretti et al., 2008) are experiencing waves of mortality later than our most impacted sites.

Elevated mortality rates have been found in a number of systems impacted by invasive forest pathogens. Orwig (2002a) reports eastern hemlock mortality due to hemlock wooly adelgid of 5–15% year^{-1}, causing overall mortality of 50–99%. Maloney et al. (2005) reports a 6% year^{-1} mortality rate in a *P. ramorum*-infected Douglas-fir–tanoak forest in northern California, an increase from 2.1% year^{-1}, the rate calculated in an uninfected Douglas-fir–tanoak forest in northern California (Hunter, 1997). Our study design would benefit from background rates derived from a longer period of study and from pre-1989 for better confidence of capturing pre-SOD levels. Such numbers were not available in the literature, perhaps reflecting the lack of commercial importance of coast live oak. In addition, tree mortality can be highly variable at local and regional scales, and SOD mortality in particular appears to be very patchy across the landscape (Kelly and Meentemeyer, 2002). The high mortality rates reported here may be representative of such severely infected patches. Further study is needed to establish an "average" of *P. ramorum*-induced mortality across affected portions of California.

What impacts will elevated coast live oak mortality have on the future stand development and composition of infected forests? The answer will involve a complex mix of factors including growing space occupancy and compensatory growth by surviving mature trees (both coast live oaks and other species), seedling and sapling recruitment in the understory, and disease and genetic factors such as *P. ramorum* residence time in carrier species and degree of resistance found in coast live oak individuals. Our evidence suggests that major compositional changes are occurring quickly. As oak canopy was removed, bay laurel increased substantially in relative importance, from 22% of pre-infection total to over 37% of projected total stand basal area in one site. The average past to present mortality rate for bay laurel across all sites was 0.30% year^{-1}, compared to 2.4% year^{-1} in coast live oak, with more marked differences between infected sites. Besides bay laurel, other co-occurring tree species – Pacific madrone, toyon, bigleaf maple, California buckeye, Douglas-fir, California manzanita, foothill pine and Oregon oak – were small components pre-infection and were predicted to increase in importance. Research indicates that infill by bay laurel and other co-dominant trees into gaps created by the death of mature coast live oak trees is occurring rapidly in some SOD areas (De Chant et al., 2006). In other areas or microsites less suitable to bay laurel (e.g. more xeric) and other co-occurring tree species, such gaps may be captured by annual grasses or shrubs, leading to a more savanna-like structure.

The implications of such changes could extend beyond spatial replacement of oaks. Bay laurel, as the largest producer of *P. ramorum* inoculum in California coastal forests (Davidson et al., 2005), may increase ambient inoculum loads and potentially override defense systems in remaining oaks. Similar positive feedback cycles have been recorded for other novel plant–pathogen interactions. When white pine blister rust (*Cronartium ribicola*) kills lower elevation whitebark pine (*Pinus albicaulus*), it promotes the growth of lodgepole pine (*Pinus contorta*) which in turn supports high populations of the mountain bark beetle (*Dendroctonus ponderosae*). These disperse into adjacent whitebark pine stands, attacking and killing trees, especially ones already weakened by disease (Kendall and Keane, 2001). Further research is needed to elucidate the relationship between bay laurel prevalence and inoculum load dynamics in impacted coast live oak-bay laurel forests.

The degree of canopy change will be highly dependent on SOD mortality rates. In the present study, the SOD component of future projections was based on number of seeping trees. Using the upper range of the survival period for seeping trees calculated by McPherson et al. (2005), we inferred that trees seeping in 2004 would be dead in 2014. This assumption could prove erroneous in either direction. Dodd et al. (2005) found evidence for variation in resistance of coast live oak to *P. ramorum* in studies in which mechanical inoculations produced cankers that varied consistently in size and in frequency of disease development in trees. Conversely, increased mortality could occur if, for example, several wet years raise inoculum loads and cause new infections in the absence of sufficient genetic resistance. A complex set of factors seems to determine when spore loads increase and decrease and when new infections occur. If, as our model predicts, future mortality rates (while still higher than background levels) decrease in some stands from 1994 to 2004 rates, these may begin to stabilize with regard to species composition and density. Over the long term, much will depend on recruitment processes occurring in the understory.

While mature bay laurels may capture some gap openings left by dead oaks, seedling and sapling exploitation of such openings is a key determinant of future composition. In a separate study, the authors found that bay laurel seedlings were much more prevalent than coast live oak seedlings in all of the same eight study sites, but that presence of bay laurel seedlings was associated spatially with areas of lower light (Brown and Allen-Diaz, 2006). This finding may simply relate to lower light conditions under mature con-specifics, but it may suggest a limitation of bay laurel seedlings in their ability to establish in open gaps. Further research is needed. However, saplings can compete more rapidly for gap resources than seedlings; bay laurel saplings were found to be eight times more numerous than coast live oak saplings in our sites (Brown, 2007). The low number of coast live oak seedlings and saplings in our study is commensurate with findings that coast live oaks are not regenerating well in California, and that the sapling size class is particularly underrepresented in forest stands (Muick and Bartolome, 1987; Bolsinger, 1988).

The factors contributing to the observed variations in the rate of oak decline in our sites remain unclear. The recorded rate of decline at Briones and Olompoli may be lower because of site characteristics, climatic influence, genetic factors, rate of *P. ramorum* dispersal, or levels of pathogen exposure. Bay laurel has been positively linked to SOD induced oak mortality (Kelly and Meentemeyer, 2002; Swiecki and Bernhardt, 2002); however, we found no evidence of an association between bay laurel basal area or stem density and oak mortality on our sites. At the landscape scale, distance from the point of disease introduction, potentially in Marin County (Mascheretti et al., 2008), may also play a role. It is likely that much variation in oak mortality is due to unmeasured variables in our sites.

We attribute elevated rates of coast live oak mortality to SOD, but several additional factors may have exacerbated mortality during the period of study. Primary wood decay fungi such as *Phellinus* spp. and *I. andersonii* are found in some or all study sites and cause damage in coast live oak. Laboratory diagnostic techniques were used on a subset of stems in our study while visual indications (seeps) were used as proxies for diagnosis of *P. ramorum* infection for the majority of coast live oak stems. This introduces a potential error as other pathogens (e.g. *Pythium* spp.) can cause seeping in coast live oak. Future studies would benefit from a full accounting of such mortality agents.

Final consideration must be given to the possibility that elevated coast live oak mortality may lead to cascading effects in

SOD-impacted ecosystems. Wildlife communities may decline due to oak's importance as a mast-producing species. Two bird species were found to feed and nest in coast live oak and to avoid bay laurel, indicating a significant impact by SOD (Apigian and Allen-Diaz, 2006; Apigian et al., 2006); another study projected similar losses in other oak-dependent bird species (Monahan and Koenig, 2006). Future research should examine further impacts to biodiversity as SOD-infected forests move away from oak dominance.

Acknowledgements

This study was funded by the Pacific Southwest Research Station of the USDA-Forest Service, Agreement # 01-JV-11272164-165, and by an award from the California Native Plant Society. We thank the staff at our study sites for their cooperation and assistance. For lab and field support, the assistance of Rebecca Wenk, Ann Huber, Sarah Toas, Jenny Loda, Robert Spears, Ananda Case, and Zac Kaylor was invaluable. For help with pathology data in both field and lab, we thank the Garbelotto and Rizzo labs, in particular Daniel Huberli, Steve Swain, Katy Hayden, Shannon Murphy and LeAnn Douhan. For useful discussion of the manuscript (in early or late stages) we thank Anne Eschtruth, John Battles, Joe McBride, Cherie Briggs, Adam P. Simons, and two anonymous reviewers.

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