Monitoring Disease Progression of *Phytophthora ramorum* on *Quercus agrifolia* in East Bay Regional Parks over a 4-year Period.

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ABSTRACT

Sudden oak death describes the invasive water mold *Phytophthora ramorum* which has caused mortality of oaks across coastal California and southern Oregon forests. This study investigates the progression of sudden oak death across Anthony Chabot, Redwood, and Wildcat Canyon Regional Parks between 2011 and 2015. This study reevaluated 53% of the randomized plots across oak-bay woodlands on these three East Bay Regional Parks established in 2011 and used visual symptomology to assess stages of coast live oak (*Quercus agrifolia*) morbidity and mortality. General descriptive statistics and bivariate regression were used to profile characteristics and progression of the disease. Since the establishment of the plots, 16.36% of coast live oaks that were asymptomatic during the summer of 2011 became visually symptomatic by 2015 and 13.31% were killed by *Phytophthora ramorum*. In 2011, 81.30% of coast live oaks were asymptomatic which decreased to 63.94% in 2015. The mean DBH (diameter at breast height) of an oak with symptomatic mortality in 2015 was 32.78 cm whereas the mean DBH of asymptomatic dead trees was 14.17, indicating that larger trees are more likely to become infected and die from the disease. Regeneration of coast live oaks was not correlated with percentage of symptomatic stems per plot. The results from this study will aid in understanding management concerns for East Bay Regional Park District. This study emphasizes the need for continued large scale monitoring for *Phytophthora ramorum* over an extended time period.

KEYWORDS

Invasive species, sudden oak death, coast live oaks, land management

INTRODUCTION

Sudden oak death (SOD) is a colloquial term used to describe the disease caused by an exotic pathogen responsible for the quick and unexpected mortality of oaks and infection of other woody species (Davidson et al. 2003). Although the name implies a rapid death of the trees, the process of infection can remain unnoticed until the tree is substantially weakened or already dead (Garbelotto and Rizzo 2003). Symptoms of sudden oak death were first observed in Marin County in 1994 (Garbelotto et al. 2001). The origin of the pathogen remains unknown, but genetic analysis suggests that there may have been multiple introduction events (Brasier 2003). The invasive disease continues to cause extensive tree mortality.

Phytophthora ramorum is the causal agent behind the phenomenon of sudden oak death (Garbelotto et al. 2001). *P. ramorum* is a water mold (oomycete) with a wide host range in native California plant species (Grünwald 2012). Among the major tree species affected are coast live oak, *Quercus agrifolia*, California black oak, *Quercus kelloggii* and California tanoak, *Notholithocarpus densiflorus* (Garbelotto et. al 2003). Millions of oaks and tanoaks have been infected and killed by this pathogen. The two primary symptoms of sudden oak death in coast live oaks are large visible main stem cankers (necrosis of the cambium and phloem) as well as red oozing resinous exudate (Grünwald 2012). Trees affected by these infections are commonly infested with bark beetles and ambrosia beetles (McPherson et al. 2005). Once colonized by beetles, fruiting bodies of an endophytic ascomycete fungus, *Annulohypoxylon thouarsianum* become visible in most infected trees indicating death of the cambium (McPherson et. al 2005). The exact means of dispersal of *P. ramorum* remains unknown, which emphasizes the need to understand disease dynamics.

Due to the devastating impact of this invasive pathogen, many studies have attempted to better understand its establishment and spread, with the ultimate goal of slowing the spread of *P. ramorum.* Previous oak inoculation studies have identified phenolic biomarkers that are associated with resistance to the pathogen in artificially inoculated trees, but this relationship has not been shown in naturally-infected trees (Bonello et al. 2011; McPherson et al. 2014). While application of phosphites to coast live oaks has been shown to prevent infection, this costly treatment is limited to individual trees (Garbelotto et al. 2007). A study of sudden oak death in China Camp State Park and Marin Municipal Water District that evaluated the progression of the

disease over an 8-year period (McPherson et al. 2010) found that increasing stem diameter was associated with higher infection rates and that disease intensity and the basal area of California bay laurel were positively correlated (McPherson et al. 2010). Potential contributors to *P. ramorum* dispersal are thought to be through rain splash, moving bodies of water and anthropogenic actions, indicating that spread of the disease might not be controllable (Grünwald 2012). While small-scale experimental studies have attempted to understand the spread of the disease, an observational understanding of how *P. ramorum* spreads in nature on a large scale has yet to be studied.

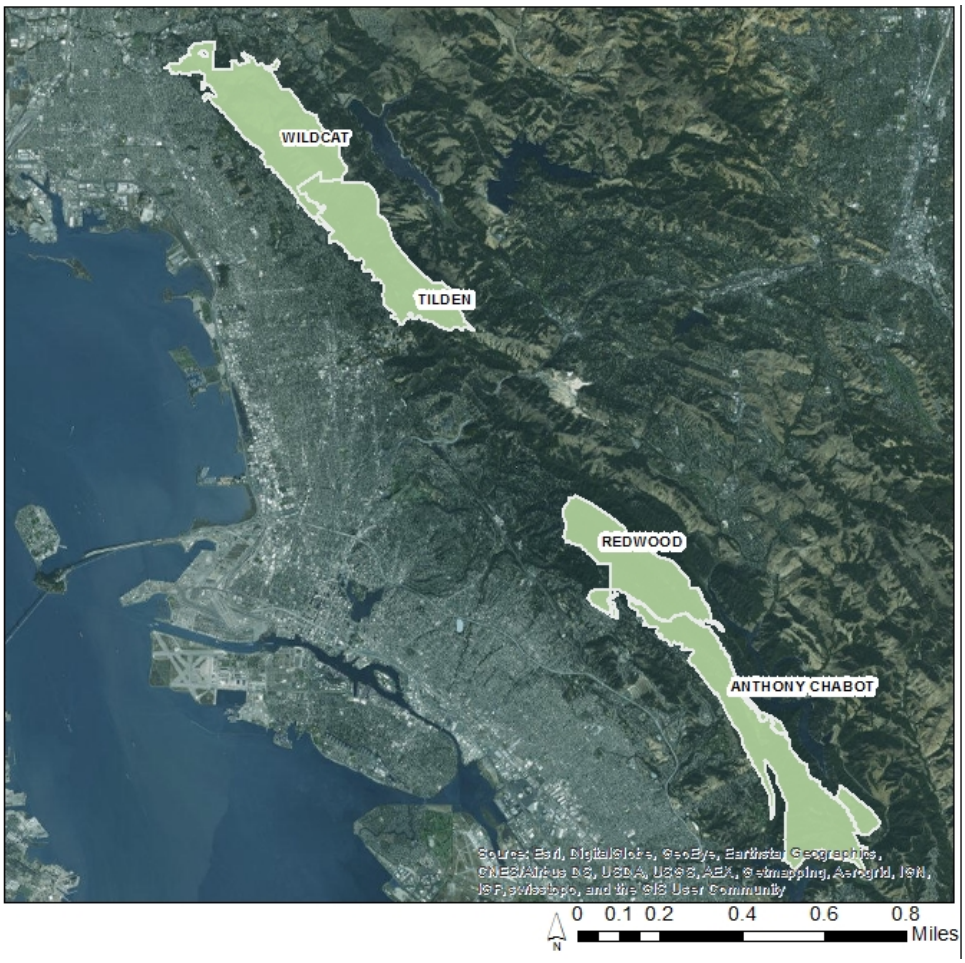
This study attempts to understand how *P. ramorum* morbidity and mortality has progressed throughout Anthony Chabot, Redwood, and Wildcat Canyon Regional Parks between 2011 and 2015. One goal of this project is to estimate rates of change associated with coast live oak infection to project future stand structure. I compared the changes in infection (morbidity), mortality and resistance over the 4-year period for each park. I then evaluated diameter at breast height (DBH) to see if there was a relationship between size and probability of mortality from sudden oak death. I also analyzed regeneration counts in each plot to understand if a correlation exists between disease severity and coast live oak regeneration. I predicted that plots will increase in disease intensity and mortality during the 4-year period. I expected that mortality in the plots would be higher where disease intensity exceeded a certain threshold. Understanding characteristics of *P. ramorum* spread may help identify best management practices for East Bay Regional Parks.

METHODS

Study system description

To monitor the progression of *Phytophthora ramorum* in a natural system, this study evaluated changes in tree morbidity in the three East Bay Regional Parks in 2011 and 2015. The East Bay Regional Park District encompasses 120,000 acres of coastal forest and chaparral across the East San Francisco Bay which is characterized by a Mediterranean climate. Wildcat Canyon Regional Park (37.939241, -122.290588) contains 2,427 acres spanning from El Cerrito, CA to Richmond, CA. Redwood Regional Park (37.813849, -122.166036) contains 1,830 acres

of chaparral, oak bay and evergreen habitat located in the hills east of Oakland, CA. Anthony Chabot Regional Park (37.735295, -122.095967) contains 3,314 acres stretching from San Leandro, CA to Castro Valley, CA characterized by chaparral, grasslands and oak-bay habitat. The decimal degree associated with each park is an approximate of the center of the plot boundary. Wildcat Canyon Regional Park is the north most park, while Redwood and Anthony Chabot Regional Parks are adjacent and south of this park.



Picture 1: Location of East Bay Regional Park Study Sites. The parks evaluated are Anthony Chabot Regional Park (Anthony Chabot), Redwood Regional Park (Redwood) and Wildcat Canyon Regional Park (Wildcat). Tilden Regional Park was not surveyed.

Data collection methods

Plot establishment

In 2011, 375 plots across the three parks were established and evaluated for signs and symptoms of sudden oak death (McPherson et al. 2014). Each park was stratified by vegetation type using maps provided by the East Bay Regional Park District. Plots were randomly placed in the oak-bay laurel vegetation type, restricted to sites with slope < 30%. We navigated to each 10-meter radius geotagged plot using a Garmin 60CSx GPS. In each plot a north, center and south tree was tagged with a unique identifier to order and approximate measurement within the plot boundary.

Plot measurements

In 2011, every coast live oak greater than 1.5 cm diameter at breast height (DBH: 1.37 meters above ground) was evaluated for disease symptoms and DBH was recorded (McPherson et al 2015). To estimate change in *P. ramorum* disease progression, in 2015 we reassessed tree morbidity and mortality by visual symptomology. Due to logistic limitations, the reassessment in 2015 was restricted to 199 of 375 plots with a goal of surveying approximately 50% of each park with the added constraint that the sampled plots were largely contiguous within each park.

Symptom Evaluation

Under the symptomology assessment designed by McPherson et al 2005, we diagnosed and scored trees infected by *P. ramorum* based on the visible presence of four chronic symptoms: bleeding, bark beetles, fruiting bodies of *Annulohypoxylon thouarsianum* and loss of foliage (McPherson et al. 2015). Bleeding was typically characterized by red resinous drips from the bark. The bleeding associated with the *P.* infection attracts up to six species of ambrosia and bark beetles, which was indicated by boring dust (McPherson et al. 2008). Charcoal-like pimpled sporocarps on the bole of a living tree indicated the presence of *A. thouarsianum.* Trees were assigned a score of 0 (asymptomatic) or 1 (symptomatic) for the following symptoms: bleeding, beetle attacks, and the presence of *A. thouarsianum* sporocarps. The values were summed so that the cumulative score was indicative of the general symptom succession (Table 1). Foliage was rated 1 for healthy, 2 for declining (thin or pale foliage or bare branch tips), and 3 for brown foliage (dead).

Table 1: Scoring of disease status based on visual symptomology. Scores of 7 and 8 were assigned to stems that showed apparent resistance to the disease in 2015.

Disease Status	Symptomology
1	Asymptomatic
2	Bleeding
3	Bleeding + bark beetles
4	Bleeding + bark beetles + *Annulohypoxylon thousarsianum*
5	Sudden oak death killed (all listed symptoms + dead foliage)
6	Dead- unknown
7	In remission from early stages of disease (showed previous bleeding or old canker)
8	In remission from late stages of disease (showed previous bleeding with beetles and/or *Annulohypoxylon thousarsianum*)

Regeneration Evaluation

At each plot, two 10x2 meter transects were installed to count understory growth of woody species. Species were differentiated by seedling (less than 6 inches tall) and sapling (greater than 6 inches tall) and tallied by presence. A transect was walked in the North (0 degrees) and South (180 degrees) from the tagged center tree of the plot. Actual azimuths were altered and recorded for deviations or obstructions in path. Vegetation counts were extrapolated to approximate understory growth for entire plot area.

Data analysis methods

We collected data on over 3205 coast live oaks between June 2015 and September 2015. The data collected from each tree was transcribed from field notes, categorized by plot number and exported into JMP to perform statistical analysis (JMP 2015). Diameter at breast height was adjusted for shrinking trees, living trees that were larger in 2011 than in 2015. The 2015 DBH measurement was used for both 2011 and 2015 size so that there would not be negative growth. Shrinking trees suggest limitations and errors in field data due to variation in bole structure.

Trees that were classified as SOD or non-SOD kills in 2015 were also adjusted based on 2015 measurements. Descriptive statistics were used to search for associations and distribution among the data including mean tree DBH of symptomatic versus asymptomatic trees, percentage infection and percentage mortality. I compared relationship between DBH and mortality using a one-way bivariate regression and t-test. Lastly, I used a bivariate regression to evaluate the relationship between disease severity levels and oak regeneration in plots.

RESULTS

Status since 2011

Summary of parks

Since the establishment of the plots across the three East Bay Regional Parks, 16.36% of coast live oaks that were alive during 2011 became visually symptomatic by 2015 and 13.31% of died from the late stages of *P. ramorum* infection. In 2011, 81.30% were asymptomatic whereas in 2015 63.94% were asymptomatic. There was an increase in the number of asymptomatic trees transitioning into the bleeding disease category; however, the number of trees in the bleeding plus beetles category remained few. Of the trees classified as sudden oak death kills in 2015, 33.57% of these dead trees were previously asymptomatic trees in 2011. Thus the mortality is approximately 8.39% per year. While the three parks showed similar levels of disease, there were still small variations between infection rates and mortality across the geographical boundaries. Overall, sudden oak death severity increased in each park during the 4 years of the survey (Table 1).

Table 1: Summary of stems in each disease severity level. Comparison among counts of trees in each sudden oak death disease in each severity class during summer of 2011 and 2015 surveys across the three East Bay Regional Parks.

Disease Status	Summer 2011			Summer 2015		
	Anthony Chabot (n=1039)	**Redwood Regional (n= 1014)**	**Wildcat Canyon (n=1060)**	**Anthony Chabot (n=1099)**	**Redwood Regional (n=1046)**	**Wildcat Canyon (n=1060)**

Asymptomatic	873	782	876	752	618	676
Bleeding	56	38	101	100	106	84
Beetles	0	4	8	0	4	21
Annulohypoxylon thousarsianum	23	67	11	20	26	37
SOD Kill	57	97	36	109	176	141
Non SOD Kill	30	26	28	64	44	65
In Remission	----	----	----	49	72	36

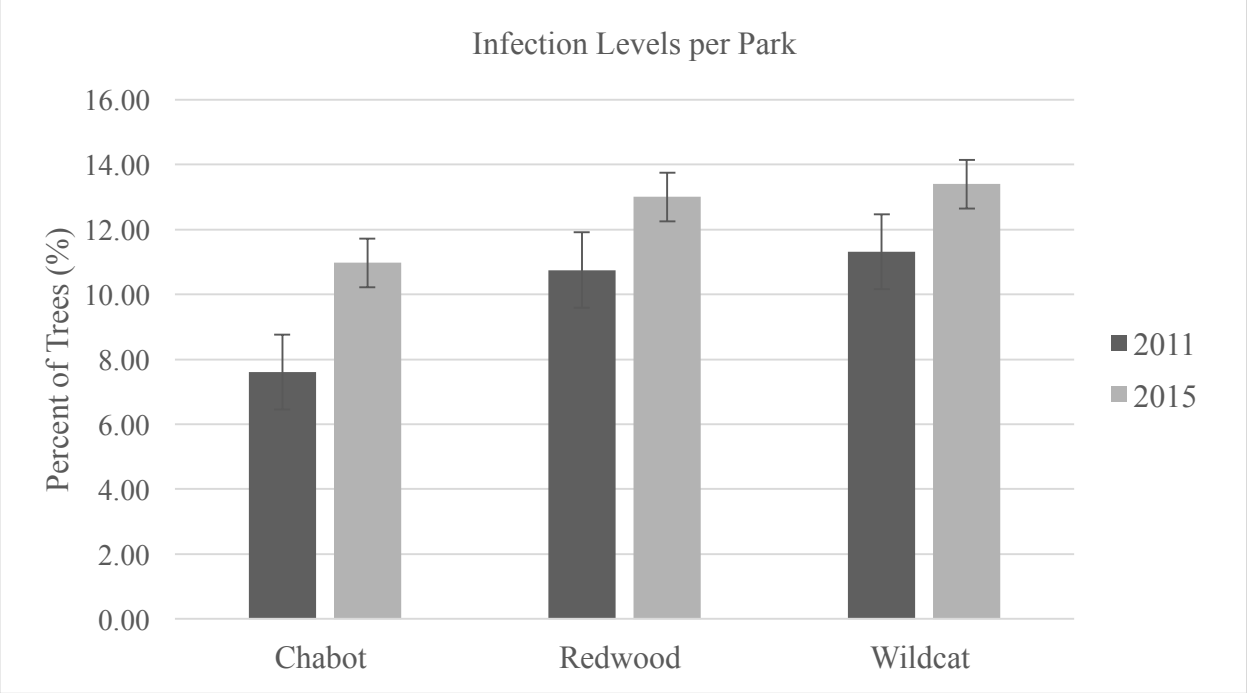


Figure 1: Infection Levels per East Bay Regional Park between 2011 and 2015. Percentage of coast live oaks showing symptoms of sudden oak death over a 4-year period.

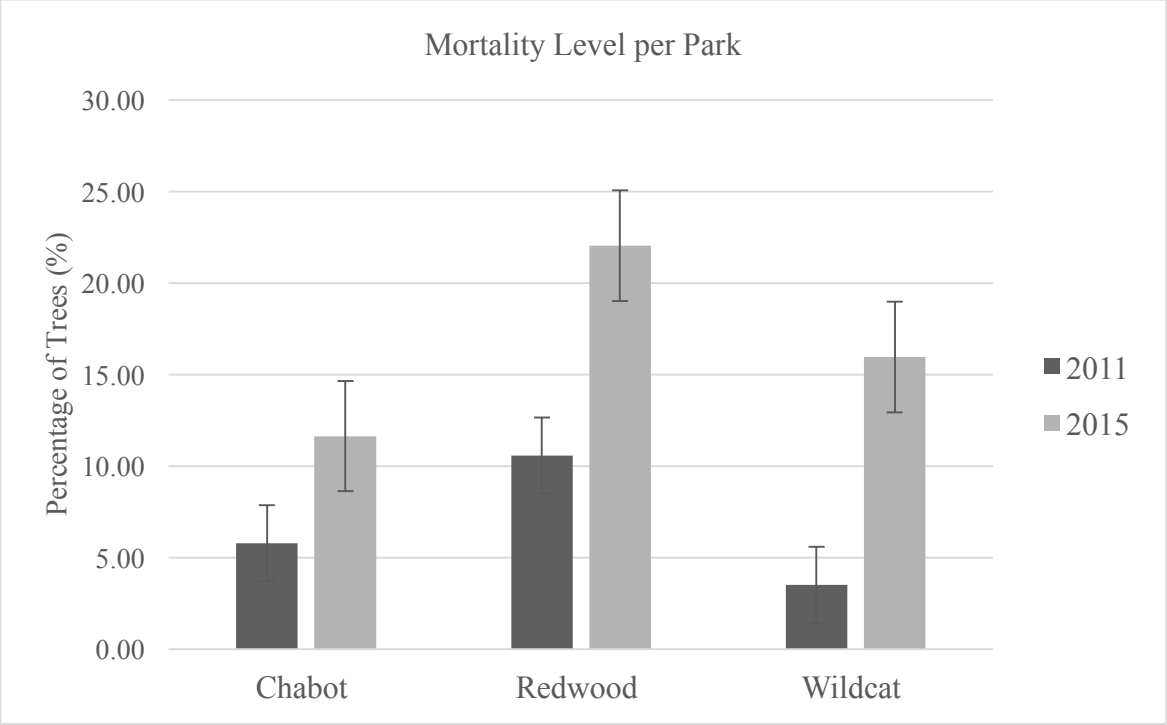


Figure 2: Mortality Levels per East Bay Regional Park between 2011 and 2015. Percentage of coast live oaks showing symptoms of sudden oak death over a 4-year period.

Park Summaries

Anthony Chabot Regional Park

Disease incidence in Anthony Chabot Regional Park increased from 40 of 64 (62.50%) plots in 2011 to 51 of 64 (79.69%) plots in 2015. Of the coast live oaks that were recorded as asymptomatic during 2011, 13.76% showed signs of infection in 2015. The mean DBH of infected oaks in 2011 was 31.12 (±14.96) cm while the mean DBH increased to 31.95 (± 14.60) cm in 2015. Remission was observed in 4.77% of oaks surveyed in 2015. Of the coast live oaks that died by 2015, 35 of 109 (31.11%) were surveyed as asymptomatic in 2011. Anthony Chabot Regional Park did not have any trees classified in the bleeding plus beetles category in either 2011 or 2015. The park had lower percentages of infection and mortality rates than the other two parks (Figures 1 and 2).

Redwood Regional Regional Park

In Redwood Regional Park in 2011, 44 of the 54 plots (81.48%) of the plots had symptomatic trees which increased to 52 (96.30%) in 2015. Of the coast live oaks that were asymptomatic during 2011, 18.05% showed signs of infection in 2015. The average DBH of infected tree at Redwood Regional Park in 2011 was 30.84 (±11.93) cm whereas the average DBH in 2015 was 31.13 (±12.07) cm, which suggests a higher probability of a larger tree being infected. 37 of the 176 trees that died from sudden oak death had been asymptomatic in 2011. Most notably, Redwood Regional Park had the greatest number of trees (6.88%) illustrating remission.

Wildcat Canyon Regional Park

In Wildcat Canyon, 18.26% of coast live oaks that were asymptomatic during summer of 2011 showed signs of infection in 2015. In 2011, 20 of the 67 plots (29.85%) had symptomatic trees which decreased to 12 (17.90%) in 2015. Of the 141 trees that died from sudden oak death, 64 had been asymptomatic in 2011. The average DBH of infected tree at Wildcat Canyon Regional Park in 2011 was 40.94 (±17.77) cm whereas the average DBH in 2015 was 41.22 (±17.46) cm. In 2011, 11.32% of all trees surveyed showed visible signs of infection which increased in 13.39 in 2015 (Figure 1). While the infection level remained fairly constant, the mortality level increased by 12.45%, almost quadrupling in severity (Figure 2).

Relationship between stem diameter and mortality

Coast live oak stem DBH was compared to mortality due to sudden oak death and unknown causes in each park using a one-way analysis of variance across the three parks. The one-way analysis allows comparison of the continuous diameter at breast height to the binary symptomatic ranking. Mortality due to natural or unknown causes was given a score of 0 (n=177), while mortality due to sudden oak death was given a score of 1 (n=422). Of the 599 cumulative total of dead trees across the parks in 2015, there was a strong trend ($P>0.0001$) between diameter at breast height of the stem (cm) and probability of mortality due to sudden oak death (Figure 3). The mean size for a tree dying of sudden oak death in 2011 was 33.98 cm

which decreased to 32.78 (±15.33) cm in 2015. The mean size of a dead asymptomatic tree in 2011 was 20.95 which decreased to 14.17 (±10.34) cm in 2015.

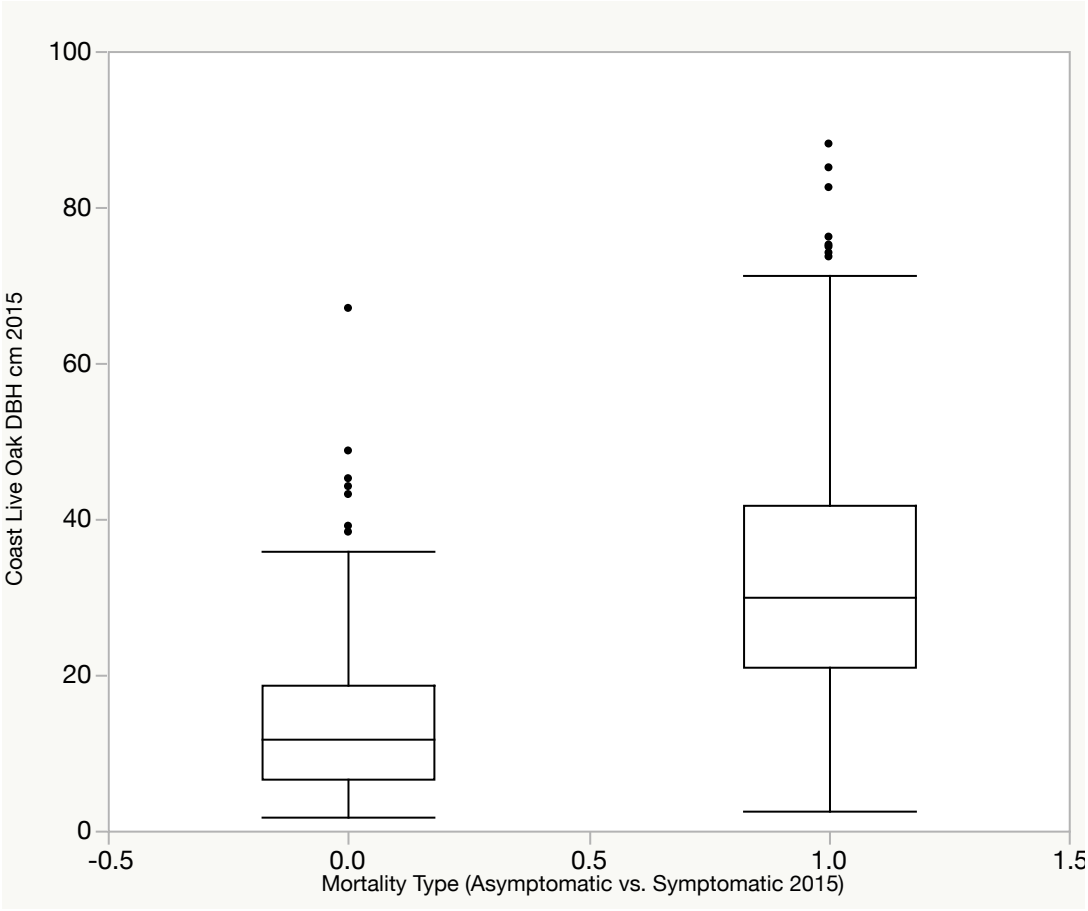
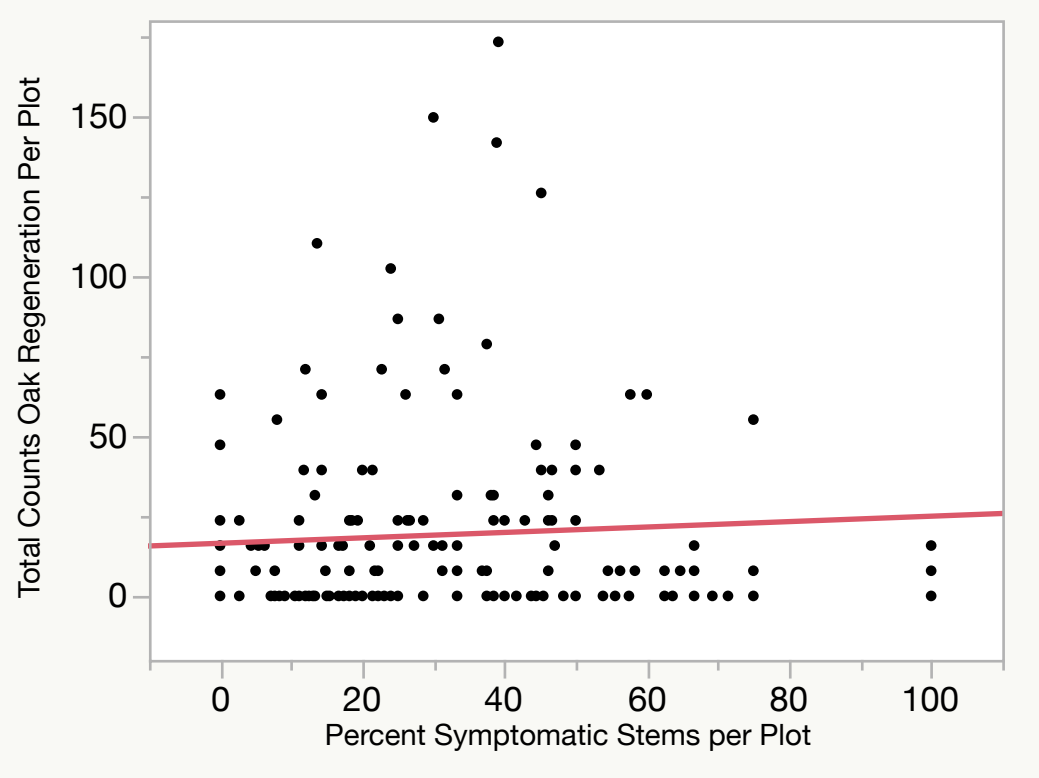


Figure 3: One-way bivariate analysis of oak DBH and sudden oak death mortality. The relationship between mortality and DBH in 2015 was compared for trees that were asymptomatic or symptomatic. The regression illustrates best fit ($P<0.0001$).

Regeneration

Plot infection severity based on percentage symptomatic stems per plot was compared to coast live oak regeneration in each plot using a bivariate analysis. The number of coast live oak seedlings and saplings per plot were extrapolated based upon the two 10x2 meter transects surveyed. The percentage of symptomatic stems was used to approximate plot infection severity levels. There was no relationship ($R^2= 0.00405$) between the symptomatic stem counts and regeneration based on a linear regression. Of the 184 plots with regeneration data, 104 showed regeneration of oaks and 157 showed symptomatic basal area.

Figure 4: One-way bivariate analysis of percentage symptomatic stems and total oak regeneration per plot. The percentage of symptomatic stems per plot was used as a metric to describe plot infection and was compared against total coast live oak regeneration per plot. The regeneration was approximated using a north and south transect per plot. This regression illustrates no correlation between level of sudden oak death and oak recruitment (R^2= 0.00405).



DISCUSSION

Sudden oak death syndrome has caused the unexpected mortality of coast live oaks since its introduction in 1994 (Garbelotto et al. 2001). In recent years, the disease has continued to spread throughout the coastal forests of the San Francisco Bay area at a substantial rate. This study illustrates that the disease increased rapidly in three East Bay Regional Parks over the 4-year period of this study. While 3.62% of trees showed late stages of the disease by exhibiting bark beetles and *A. thouarsianum* in 2011, 33.57% of trees classified as dead in 2015 were initially asymptomatic in 2011 suggesting that the infection leads to mortality in less than 4 years. Diameter at breast height was strongly associated with the probability of coast live oak mortality from sudden oak death ($P<0.0001$). Regeneration of coast live oak was not associated

with percentage disease severity suggesting that canopy opening and exposure to full sunlight does not positively affect counts of oak regeneration over the 4-year period of the study. To better assess the ultimate impact of sudden oak death on coast live oaks, it will be necessary to continually monitor the established plots into the future to best determine the elapsed course of the disease.

Changes in levels of infection and mortality

Based on a survey of the disease incidence of *P. ramorum* in Anthony Chabot, Redwood and Wildcat Canyon Regional Parks, we have seen increases of 13.76%, 18.05%, 18.26% in the percentages of asymptomatic trees showing infection, respectively. While the level of infection has remained fairly constant from the initial establishment of plots in 2011, Redwood Regional Park showed the greatest increase of sudden oak death induced mortality. Surprisingly, Redwood Regional Park also showed the highest percentage of oaks showing remission. The reason behind infection stabilization rate is unknown, however, the low increase in disease suggests that morbidity will continue in the following years, Further research should evaluate the trends of the disease across a stratified landscape (Anacker et al. 2008).

Remission

Nearly 5% **(**4.91%) of coast live oaks that were symptomatic in 2011 showed apparent remission in 2015. The remission categorization was based upon evidence of infection in 2011 surveys, with the presence of healed and callused tissue in 2015. Trees in remission represented 4.48%, 6.88% and 3.40% of those surveyed in Anthony Chabot, Redwood and Wildcat Canyon Regional Parks, respectively. Redwood Regional Park showed the highest percentage of remission but also the highest mortality at 22.06% in 2015 (Figure 2). Conversely, Wildcat Canyon had the lowest level of remission but the highest infection level at 13.40% (Figure 1). While 157 coast live oaks showed apparent remission to sudden oak death, only 3 trees showed late stage resistance, resistance after symptoms of beetles and *A. thouarsianum*. The aggregation of bark and ambrosia beetles on *P. ramorum* infected oaks has been shown to strongly decrease median lifespan based on a survival analysis (McPherson et al. 2005). The limited number of

oaks showing late stage resistance suggests that beetle and *A. thouarsianum* succession are detrimental to infected oak survival.

Diameter at breast height and probability of mortality

There was a strong relationship between diameter at breast height and mortality caused by sudden oak death. The average DBH of a tree dying with symptoms of sudden oak death in 2011 was 33.98 (±16.26) cm which decreased to 32.78 (±15.33) cm in 2015. The mean size of a dead asymptomatic tree in 2011 was 20.95 (±13.92) cm which decreased to 14.17 (±10.34) cm cm in 2015. These findings support the current research that bigger coast live oaks have increased susceptibility to sudden oak death infection (McPherson et al. 2010). While an exact understanding why larger trees have a higher probability of sudden oak death infection is lacking, there are a few possible explanatory variables. Larger trees often grow faster than their smaller counterparts increasing the probability of bark fissures (Swieck and Bernhardt 2015). The splitting of bark may allow greater area for landing of spores. Lastly, because DBH is a proxy variable for age, it may be possible that older trees lose immune response and become generally more susceptible to disease.

Regeneration

The results suggest that the level of disease severity in each plot is not associated with counts of coast live oak regeneration growing in the the understory. The results do not support the research that coast live oaks recruitment best in shade cover (Muick 1991). If so, there would be a correlation between increased levels of sudden oak death and greater canopy opening from thinning foliage and dead trees as indicated by symptomatic stems per plot. Because the counts of oak regeneration were extrapolated to plot level based on transect data, the regeneration counts are a mere approximation and may not fully describe oak recruitment. The extrapolated counts of oak regeneration ranged from 0 to 173 however, the majority of values lie in the 95% confidence interval between 14 and 23 oaks per plot. However, this study did not evaluate current research that shows a positive relationship between disease and basal area of bay laurel (McPherson et al. 2010). As noted above, increased DBH is associated with higher probability of

disease incidence (Figure 3). Previous research has not looked at the relationship of seedlings and saplings to disease severity, instead focusing on basal area of mature trees as an indicator (McPherson et al. 2005). Because the method of sporulation and transmission is still misunderstood, it is unclear if regenerating trees may be a factor in their ability to act as a host or produce viable spores. The average DBH size of a symptomatic dead tree decreased from 33.98 (±16.26) cm in 2011 to 32.78 (±15.33) cm in 2015 which suggests that the disease is infecting smaller trees (Figure 3). The smallest coast live oak to show infection across the three parks was 2.54 cm. If this trend follows increased infection rates, it is possible that regenerating vegetation may also be susceptible to this disease.

Future direction

Sampling of these plots is a part of a long-term study on disease progression in East Bay Regional Parks. The original goal of the study was to revisit each of the established plots in the three East Bay Regional Parks. However, due to lack of logistical support, only a subset (53.1%) of the plots were revisited. Efforts were made to make the sampling process as random as possible with plots evaluated by proximity and accessibility. The other plots that were not surveyed in 2015 will be surveyed during summer of 2016. Ideally all plots will be reevaluated every four years to collect data on each tree. Measuring the impact and progression of the disease is crucial to understand implications of fire hazard and park safety associated with structural failure of trees. Phloem and leaf samples were collected from trees illustrating remission for future genetic and chemical analysis.

Study Limitations

The limitations of this study are mostly attributed to the size and complexity of the survey process. Visual symptomology was used to diagnose the disease rather than enzyme-linked immunosorbent assay (ELISA) tests to validate the presence of the inoculum (Brown and Brasier 2007). Thus misdiagnoses may have occurred as symptomology was assumed with the naked eye, not the presence or absence of the water mold itself as small cankers can heal without visible scarring (Swiecki and Bernhardt 2015). A small percentage (4.91%) of previously

infected stems showed apparent remission to *P. ramorum* infections. Based on visual symptomology, trees in remission showed healed cankers around sites of previous bleeds. Data collected in 2011 also included trees that initially showed low levels of infection and were reclassified as asymptomatic (i.e., in remission) in 2015. It is important to note that the trees showing remission may not be fully healed from the disease and relapse forming new and reactivating old cankers (Swiecki and Bernhardt 2015).

Management Implications

During the 4-year period, the total morbidity and mortality from sudden oak death increased from 15.74% to 25.75% over the three East Bay Regional Parks observed. Based on these levels, it is evident that the pathogen will continue to persist unless there is a disturbance great enough to alter the virulence of the disease. Due to the ability for boles to "suddenly" snap from the disease, East Bay Regional Parks should evaluate the areas of high disease in close proximity to trails and recreational areas. Because there currently is not a wide scale treatment of *P. ramorum* infected trees, it is important to practice active management including pruning and removal of infected trees for park and visitor safety. It is also advised to educate park visitors of the symptoms of the disease for personal awareness. Lastly, diversified reforestation efforts should be considered for future vitality of the forest. East Bay Regional Parks should consider the potential climax communities that may dominant the future canopy and how factors such as host viability, drought and climate change will affect long term succession. Due to the trajectory and lack of wide scale treatment plans, it is likely that there will be significant change to the existing and historical oak bay woodlands.

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