Effects of *Phytophthora ramorum* the Stomatal Conductance of Coast Live Oak, *Quercus agrifolia.*

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Abstract: *P. ramorum,* the fungus-like pathogen causing Sudden Oak Death (SOD), threatens several species of trees including coast live oak (*Quercus afrifolia*), tanoak (*Lithocarpus densiflorus*) and black oak (*Quercus kelloggii*) in Northern California forest. Little work has been done to understand how infectious agents like *P. ramorum* affect tree physiology prior to death. In this study I used carbon-13 isotope analysis to understand how *P. ramorum* infection influences the stomatal conductance of *Q. agrifolia* at China Camp State Park and Pacheco Valley. Using an existing sample of 36 randomly selected and cored oaks, I found carbon-13 discrimination ($\delta^{13}C$) using mass spectrometry. $\delta^{13}C$ was used as a proxy for understanding how the stomatal conductance of both symptomatic and asymptomatic oaks changed after the introduction of *P. ramorum* in the mid-1990s. Symptomatic and asymptomatic oaks were also broken up into two groups before (1992-94) and after (2004-06) *P. ramorum* introduction. Asymptomatic oaks in this study served as a baseline for changes in stomatal conductance due to other variables including environmental variations. Examining both symptomatic and asymptomatic *Q. agrifolia* before and after the introduction of *P. ramorum* there was an overall increase in $\delta^{13}C$, meaning an increase in stomatal conductance; however, this change was only significant in symptomatic individuals. I believe that infection with *P. ramorum* is causing tighter stomatal regulation in these oaks as water transport between the roots and the leaves is being cut off. In this way, symptomatic oaks did not parallel the significant increase in $\delta^{13}C$ and therefore stomatal conductance found in asymptomatic oaks after *P. ramorum's* introduction. This work demonstrates that trees will respond to stress such as an infection by altering physiology, however much more work needs to be done in order to understand the full magnitude of such changes.

Introduction

The introduction of *Phytophthora ramorum*, the causal agent of sudden oak death (SOD), to California and Oregon forests in the mid-1990s has become an epidemic killing tens of thousands of trees and leaving many more infected (Rizzo 2003). The geographic origin of *P. ramorum*, most closely related to brown algae, is unknown and debates about whether or not it is an exotic pathogen from outside the United States continues (Rizzo 2003, Rizzo and Garbelotto 2003). Prior to its introduction to the United States the pathogen was identified only in the Netherlands and Germany on *Rhododendron* sp. and *Viburnum* sp. (Rizzo *et al.* 2002). Scientists have not ruled out the hypothesis that *P. ramorum* is native to California and that changes in the local environment, such as land use patterns, fire suppression or climate change, have been conducive to *P. ramorum's* growth and spread (Rizzo 2003). Still others suggest a hybrid origin for *P. ramorum* is possible considering many similar species are common in agricultural and ornamental settings throughout California (Martin 2004).

P. ramorum was identified in January of 2001 as the fungus-like pathogen causing widespread deaths to many trees endemic to coastal California, Oregon, and Baja Mexico including coast live oaks and tanoaks (Rizzo and Garbelotto 2003). This fungus-like pathogen girdles an infected tree by penetrating, infecting, and killing the phloem and xylem tissues that

transport water and nutrients throughout the tree (Davidson 2003). The pathogen works its way through the phloem to the cambium and eventually into the xylem; however discoloration and disease are far more extensive in the phloem than in the xylem (Garbelotto 2001). The mechanism of disease progression and how trees such a coast live oaks respond to infection is poorly understood. Secondary signs of infection includes cankers on the bark, oozing of sap, *Hypoxylon,* and leaves that have faded to a dead brown (Rizzo 2003). The disease is known as "Sudden Oak Death" because of a rapid decline in the trees outward appearance as its entire crown quickly (two-four weeks) turns brown indicating the death of the tree (Davidson 2003). However, a tree may be infected with *P. ramorum* for several years and most oaks infected with *P. ramorum* do not die from the pathogen. In fact, a study by Rizzo *et al.* (2002) found mortality rates of tanoaks and coast live oaks to be 22% and 15% respectively. *P. ramorum's* impact is not merely limited to oaks, as it has been found to infect at least 26 other plant and tree species including Douglas fir and coastal redwoods (Goheen *et al.* 2002). Most non-oak species such as bay laurels do not die from infection, but there is concern that such trees may serve as a reservoir for the pathogen (Rizzo 2003).

The importance of studying SOD stems from the numerous threats it poses to a forests' ecological function. Oaks are a keystone species that provide resources such as food and shelter for hundreds of invertebrate and vertebrate species (Pavlik *et al.* 1991). Disrupting its ability to provide even just one resources has the potential to compromise entire ecosystems. For example, the acorns from oaks are an essential source of food for several herbivores including black bears, grey squirrels, and deer (Pavlik *et al.* 1991). Moreover, *Q. agrifolia* and other oak species provide shelter for insects that bird species rely on for survival. Recent studies have shown that the abundance of avian species including the acorn woodpecker could drop 52-68% as a result of SOD induced tree loss (Monahan and Koenig 2006). Another ecosystem-level impact due to tree mortality includes changes in soil water absorption due to increased exposure of soil surfaces (Rizzo 2003). Rizzo and Garbelotto (2003) further contend that microbial community dynamics may be altered as fungi that rely on the oak's root system shift to another host species for survival. Prevention of further loss of this keystone species is essential for maintaining ecosystem functions such as providing resources to many species in California's oak woodlands. In an attempt to prevent further destruction of forests and the spread of *P. ramorum* eastward it is

vital that we gain a firm understanding of how the pathogen survives, spreads, and influences tree physiology.

The fairly recent discovery of *P. ramorum* as the causal agent for SOD has created an emerging area of study focused on the mechanisms of dispersal, how fast the disease spreads from region to region in addition to the long-term and short-term impacts on forest biodiversity. We know that *P. ramorum* infiltrates and kills the phloem and xylem in trees, but how the tree's water and nutrient distribution systems change has not been identified. This paper will focus on how infected *Q. agrifolia* alter their stomatal conductance in response to damage to their xylem, thereby limiting water access to parts the tree.

Stomatal conductance is the rate per unit area at which gases diffuse through the stomates (Hale and Orcutt 1987). The primary reason a tree regulates its stoma is to control water loss. When a tree experiences an increase or decrease in stomatal conductance it is effectively decreasing and increasing its water-use efficency (WUE) respectively. I will reference this relationship between WUE and stomatal conductance throughout my paper. WUE is the amount of carbon gain (photosynthetic activity) per unit of water loss via transpiration (Bacon 2004). By measuring the stomatal conductance of several *Q. agrifolia* trees we can get an idea about how water stressed each tree is at different points in time. There are many factors that influence WUE and stomatal conductance including nutrient availability, light, and carbon dioxide concentration (Aranda 2007). A study by Aranda (2007) detailed the effects of light variation and water stress on the WUE of cork oak. Aranda (2007) described that WUE increases or stomatal conductance decreases in response to drought and that shaded plants had lower water-use efficiencies. The relationship between nutrient availability and WUE was compared in pine and oak species and it was found that WUE decreased or stomatal conductance increased as nutrients decreased (Guehl et al. 2005). Several studies have detailed how variables like light and nutrients influence WUE, and even more analyze instantaneous WUE in oak and other tree species (Aranda 2007, Guehl et al. 2005). However, little work has been done to understand how the introduction of SOD directly affects the stomatal conductance of infected *Q. agrifolia* trees and how surrounding, non-infected, oaks may be indirectly influenced.

In this study I will also consider how the introduction of *P. ramorum* has impacted healthy oaks in close proximity to infected oaks. The death of oaks within the forest will undoubtedly alter tree-to-tree relations and their microclimates. A microclimate is a localized region where

the climate is different than surrounding areas and conditions within this region strongly influence that ecosystem's structure and function (Davies-Colley et al. 2000). Death of some oaks will result in newly created edges that can alter microclimate conditions including light, temperature, and moisture (Asbjornsen et al. 2004). As infected oaks die off we expect neighboring, uninfected oaks to receive more sunlight and therefore decreased soil moisture as a result of newly created edges, decreased competition, and reduction in the canopy. Studies by Aranda (2007) and Dawson et al. (2002) found that with greater irradiance and decreases in soil moisture trees responded by increasing their WUE or decreasing stomatal conductance. A study by Dawson (1993a) showed that neighboring trees might actually have positive rather than negative associations because of a process known as hydraulic lifting. Hydraulic lifting brings water and nutrients from deep roots to shallow soil via shallow roots during the night providing benefit to all plants in close proximity (Dawson 1993a). Ishikawa and Bledsoe (2000) found there to be evidence of hydraulic lifting in blue oaks, *Quercus douglasii*, and I believe that coast live oaks may carry out a similar process. We would expect that the loss of hydraulic lifting as neighboring trees die from infection would result in less access to water for surviving oaks. Generally, trees respond to less water availability with greater stomatal regulation and decreased transpiration and thus higher rates of WUE.

I will be using $^{13}C/^{12}C$ isotope analysis to measure WUE patterns pre and post-*P. ramorum's* introduction. Several studies by Farquhar (1989) have found that carbon discrimination and WUE are inversely correlated to each other because both variables are linked to stomatal conductance (Ebdon 1998). More specifically, WUE and stomatal conductance are inversely related to one another. WUE is a dynamic property that is often measured instantaneously, but with the use of stable isotope analysis I can look at historic patterns. Because *Q. agrifolia* actively discriminates against ^{13}C during diffusion and fixation of carbon dioxide in photosynthesis, one can effectively use isotopic analysis to understand WUE and stomatal conductance over this entire timetable (Farquhar et al. 1989). Stomatal conductance influences the rate of carbon dioxide entering and leaving the stomata (Hale and Orcutt 1987). As stomatal conductance decreases we would expect less intercellular concentrations of carbon dioxide and less water loss to the environment. Biochemical discrimination of ^{13}C by Rubisco and throughout the photosynthetic biochemical pathway in addition to the long-term carbon dioxide concentrations in a leaf's intercellular space are negatively correlated to WUE (Aranda 2007).

Rubisco is an essential enzyme for photosynthesis that catalyzes the first major step of carbon fixation into organic molecules including *Q. agrifolia* (Schneider 1992). This enzymatic process, which actively selects against ^{13}C, will make isotope and stomatal conductance analysis possible. More specifically, by comparing the molar ratio of carbon isotopes (^{13}C to ^{12}C) in the tree to that of the environment at the time, isotopic discrimination can be found (Dawson et al. 2002). Calculating carbon isotope discrimination values requires subtracting the concentration of $^{13}C/^{12}C$ in your sample from the Peedee belemnite carbonate formation standard, measured by the mass spectrometer. The value of discrimination must be compared to the levels of atmospheric carbon at the time of the sample. Changes in atmospheric amounts of carbon, mostly anthropogenic, will be scaled over this study to limit background variation in $^{13}C/^{12}C$ (Farquhar 1989). Atmospheric carbon is well documented and a corrected value for discrimination was used for data analysis.

After carbon-13 isotope discrimination ($\delta^{13}C$) was adjusted to changing background atmospheric levels in the environment I analyzed how weather would influence these oaks carbon-13 discrimination. Temperature and precipitation have been found to be the most important factors that alter $\delta^{13}C$ and may contribute to the changes in $\delta^{13}C$ of the oaks in this study (Leavitt and Long 1982, Warren *et al.* 2001). Studies attempting to describe trends between precipitation and $\delta^{13}C$ have found a positive correlation between carbon-13 isotope discrimination and rainfall (Read and Farquhar 1991). Because changes in temperature and precipitation play an important role in how a tree regulates its water it is important to find how these factors differed before and after the introduction of *P. ramorum*. Using weather data I was able to determine if changes in $\delta^{13}C$ are due to likely infection or due to changes in weather in the two groups.

This study will present new and important information on how *P. ramorum* has impacted local *Q. agrifolia* by answering the following questions: How did *P. ramorum's* introduction in the mid-1990s influence the stomatal conductance of asymptomatic oaks? In other words, how did the stomatal conductance of asymptomatic oaks change over this time? Did infection with *P. ramorum* alter stomatal conductance of symptomatic oaks? I hypothesize that by altering microclimate and tree-to-tree interactions *P. ramorum's* introduction will result in a decrease in stomatal conductance for asymptomatic *Q. agrifolia*. In addition, I believe that for those *Q.*

agrifolia that become infected with *P. ramorum* that we will see a decrease in stomatal conductance as the tree's xylem is cut off forcing them to conserve water.

More specifically, I will analyze and compare how *Q. agrifolia* have interacted with their environment prior to and after *P. ramorum's* introduction by sampling oaks in regions with documented cases of SOD: China Camp State Park and Pacheco Valle. Two distinct sites were selected to get a more representative sample of oaks. The cores from these oaks are naturally divided into rings, which give a rich timetable and history of plant-environment interaction before and after the introduction of *P. ramorum*.

Methods

This was an observational study comparing asymptomatic and symptomatic Coast Live Oaks, *Quercus agrifolia,* at two sites, China Camp State Park and Pacheco Valley. The two sites are 28 and 22 kilometers north of San Francisco respectively, and oaks in each group were compared before and after the introduction of *P. ramorum*. Using aerial photos, a total of 36 trees at these two sites were randomly selected within regions of similar slope and aspect to limit variation in temperature and light. Once points were selected, the site was visited and the tree nearest to the randomly selected point on the map was chosen for the study. Each of the 36 trees was cored three times around the circumference of the trunk, starting perpendicular to the terrain's slope for a total of 36 independent samples. This process was done to limit the microclimate variation that exists around a tree. Each core was labeled and placed in a long, cardboard tube for future carbon-13 isotope analysis.

Carbon-13 isotope preparation and analysis was completed in the Dawson lab. First, each oak core was cut into smaller pieces using the tree rings as a natural timeline. The smaller fragments were labeled and placed into centrifuge tubes based on age: before *P. ramorum*, 1992-1994, and after *P. ramorum*, 2004-2006. Next, each fragment was sent through a Wiley Mill approximately three times or until fragmented into smaller chips. After, each of the tubes was prepared for homogenization by adding three metal beads, and then placing them is a micro-beater for two intervals of three minutes. If a sample was not completely ground to a fine powder, additional time was added in one-minute intervals until each was completely homogenized. Upon completion, the samples were run through the micro-beater again as described above. Once each sample was homogenized to fine sawdust, they were then prepared for the mass spectrometer. Approximately 3-5 micrograms of each sample was weighed out and wrapped in small foil

capsules. Once all the samples were completed they were sent through the mass spectrometer for isotopic composition analysis. Ultimately, samples were prepared for carbon isotopic analysis because it serves as an indicator for stomatal conductance. However, this is only the first step needed to analyze the relationship between stomatal conductance and the introduction of *P. ramorum* in the mid-1990s.

To determine which oak trees in my sample were likely infected with *P. ramorum*, I went into the field and individually assessed each tree for symptoms related to infection. Bleeding cankers, *Hypoxylon*, beetle dust, and browning of the crown were all taken into consideration to decide which trees were likely infected (Rizzo 2003). This procedure is essential to my study as it defines which trees in my sample population are likely to be infected with *P. ramorum* or symptomatic. Asymptomatic oaks, those most likely to be free of infection, offer a baseline with which I can compare and rule out possible environmental factors.

Available weather data from the National Climatic Data was used to see how changes in precipitation and temperature would influence carbon-13 isotope discrimination. Weather data was compared for the three years before (1992-94) and after (2002-04) the introduction of *P. ramorum* to rule out climate as a possible driver of the results in carbon-13 isotope discrimination.

Results

Examining both symptomatic and asymptomatic *Quercus agrifolia* between 1992-94 and 2004-06 there is an overall increase in carbon-13 isotope discrimination ($\delta^{13}C$), meaning an increase in stomatal conductance (Table 1, Fig. 1); however, this change was only significant in asymptomatic individuals (Wilcoxon Signed-Rank Test W=24, $p<0.0019$). More specifically, $\delta^{13}C$ for asymptomatic oaks decreased 0.49‰ from a mean value of -24.67‰ ± 0.02 in the 1992-94 group to -25.16‰ ± 0.19 in the 2004-06 group (Table 1). Analysis of $\delta^{13}C$ in symptomatic oaks revealed a 0.20‰ overall decrease between the two groups, however there was no statistical difference between the means (Wilcoxon Signed-Rank Test W=25, $p<0.461$) of $\delta^{13}C$ (Table 1). More specifically, symptomatic oaks between 1992-94 (-25.09‰, ± 0.297) and 2004-06 (-25.29‰, ± 0.3548) tended toward an increase in $\delta^{13}C$ indicative of an increase in stomatal conductance (Table 1).

Table 1: Carbon Isotope Discrimination ($\delta^{13}C$) of symptomatic and asymptomatic *Q. agrifolia* trees. Statistical Results of Wilcoxon Signed-Rank Test. Mean values of isotope discrimination before and after the introduction of sudden oak death

	Mean $\delta^{13}C$					
Sample	1992-94	2002-04	$\Delta\ \delta^{13}C$	N	W	*P*
Symptomatic	-25.09‰ ± 0.297	-25.29‰ ± 0.355	0.20‰	8	25	<0.4661
Asymptomatic	-24.67‰ ± 0.021	-25.16‰ ± 0.193	0.50‰	25	236	<0.0019

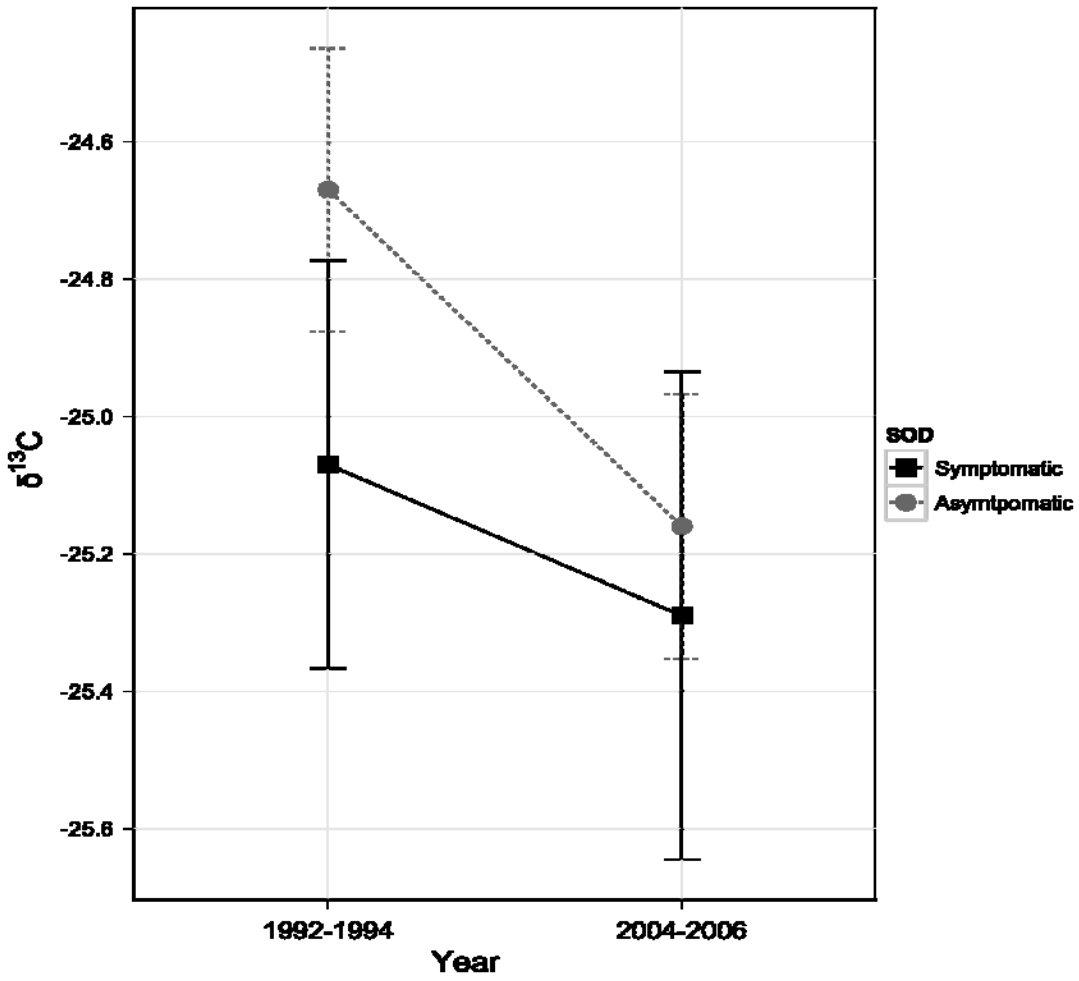

Figure 1: Carbon Isotope Discrimination ($\delta^{13}C$) of symptomatic and asymptomatic *Q. agrifolia* trees. Mean values of isotope discrimination before and after the introduction of sudden oak death. Symptomatic oaks (Wilcoxon signed-rank test: N=8, W=24, p<0.46) versus asymptomatic (Wilcoxon signed-rank test: N=25, W=236, p<0.0019).

Weather data for the calendar years between 1992 and 1994 show that the monthly mean temperature (MNTM) was 14.67 Cº and that total monthly precipitation (TPCP) averaged 973.33 mm (Table 2). Available weather data between 2002 and 2004 show a monthly mean temperature of 14.94 Cº and a total monthly precipitation value of 704.76 mm (Table 2). Overall, there is little difference in the mean monthly temperature data between the two groups before (1992-1994) and after (2002-2004) the introduction of *P. ramorum*. However, looking at the two groups the total monthly precipitation reveals a trend toward less rainfall.

Table 2: Mean monthly temperature (MNTM) and total monthly precipitation (TPCP) in San Rafael, CA. MNTM and TPCP before (1992-94) and after (2004-06) the introduction of *P. ramorum.*

Year	**MNTM (C^0)**	**TPCP (mm)**
1992-94	**14.67**	**973.33**
2004-06	**14.94**	**704.76**

Discussion

Upon examination of asymptomatic oaks before and after the introduction of *P. ramorum* $\delta^{13}C$ decreased 0.49‰ from -24.67‰ to -25.16‰ indicating a significant increase in these oaks stomatal conductance. This finding contradicts my initial hypothesis that asymptomatic oaks would tend towards decreased $\delta^{13}C$ and stomatal conductance as neighboring trees infected with *P. ramorum* die, increasing light availability and decreasing soil moisture in living, asymptomatic oaks. Analysis of the data for symptomatic oaks revealed a small increase in $\delta^{13}C$, however this change was not statistically significant (Fig 1, Table 1). However, the data does suggest that symptomatic trees are responding to possible infection be decreasing stomatal conductance supporting my initial hypothesis.

One likely reason that I found a significant increase in stomatal conductance in asymptomatic oaks is that there may be more water available as neighboring oaks die. With greater water availability I would expect oaks that are less water efficient and have higher stomatal conductance. Comparing weather data in the two groups before (1992-94) and after

(2004-06) *P. ramorum* introduction at our study sites indicates an overall decrease in mean monthly precipitation. Analyzing the weather data alone one would expect these oaks to respond to a decrease in water availability via less precipitation by decreasing stomatal conductance. However, in this study we found that despite less rainfall in 2002-04 (when compared to 1992-94), which would drive oaks toward water conservation and decreased stomatal conductance, we actually found a significant increase in stomatal conductance. As a result, we can rule out changes in precipitation as a possible confounding factor driving our $\delta^{13}C$ results. Weather data of the monthly mean temperature values for the areas these two groups occupied did not significantly vary. Several studies have found that the $\delta^{13}C$ decreases in plants that are exposed to lower ambient temperatures, however we can conclude that temperature did not vary enough to explain the significant increase in stomatal conductance found in the oaks in this study (Aranda 2007).

Symptomatic oaks revealed a small increase in $\delta^{13}C$, however this change was not statistically significant (Fig 1, Table 1). Using asymptomatic oaks as a comparison we noted a significant increase of $\delta^{13}C$ and stomatal conductance between 1992-94 and 2004-06. Even though the data suggests little change in $\delta^{13}C$ for symptomatic oaks, it does suggest infection has a direct impact on their stomatal conductance. I believe that the presence of the infection is preventing symptomatic oaks from tending toward the prevailing increase in stomatal conductance that we see in the asymptomatic group of oaks. The stresses that a pathogen such as *P. ramorum* place on a trees' nutrient and water transport system may be causing a physiological response in these trees to become more efficient with water usage. Bradford and Hsiao (1982) mention that a tree's physiological response to a stress will attempt to increase the probability of survival most often via changes in water use and supply. These oaks may be responding to the pathogen by preventing the natural increase in stomatal conductance we saw in the group of asymptomatic oaks. In other words, these oaks are decreasing their stomatal conductance relative to the others because their xylem is being cut off, meaning leaves have lower access to water. To compensate for this stress the individuals infected with the disease are preventing water loss by lowering their stomatal conductance.

This study is the first step to understanding how *P. ramorum* infection impacts the stomatal conductance of coast live oaks. Carbon isotope discrimination tends to vary between species, however future studies might find similar trends in studying infection of different species. Also,

P. ramorum infects and impacts the physiology of various tree species differently and the results found in this study would be expected to vary from species to species. Furthermore, this study offers a glimpse of the physiological impacts that a pathogen such as *P. ramorum* has on coast live oaks, although much more can be done to gain further insight into the affects of such an infection. Very little if any research has been done concerning stomatal conductance and infection with *P. ramorum*. Future research might focus on why or how an infected tree alters its stomatal conductance in response to a pathogen. Moreover, one could study how such a pathogen impacts other physiological characteristics of a tree such as growth and carbon fixation. One might also consider analyzing the defense mechanisms that trees utilizes to prevent infection and how it tries to get rid of the infection while the pathogen is girdling the tree. Finally, a future study might use molecular diagnostics to definitively identify *P. ramorum* and sample a larger population of trees to offer a more concrete conclusion as to how *P. ramoum* infection impacts the stomatal conductance of coast live oaks or another infected tree species.

The results of this study indicate that *P. ramorum* infection results in a physiological response in the coast live oaks to prevent the natural increase in stomatal conductance we saw in the group of asymptomatic oaks. More work needs to be done to determine whether or not the findings of this study are relevant and apply to other species of trees. Future studies may focus on how *P. ramorum* infection impacts other characteristics of tree physiology and why such changes occur. With greater understanding of physiological responses to pathogens such as *P. ramorum* we can begin to look at ways to help infected trees survive and prevent further infection of California's forests.

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