Management of Sudden Oak Death in Wildland Settings across California

Max Ritson

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Introduction:

In 1995 a rapid die-off in tanoak (*Lithocarpus densiflora*) was noted in Marin County, California. At first, there was little concern over the loss of tanoak due to the species' lack of commercial value. It was not until 1998 when coast live oak (Quercus *agrifolia*) - a species prized as a symbol of California's natural beauty - started to die off, that scientists, land managers, and the public became alarmed (Frankel, 2008). It was deduced that an unknown pathogen was killing tanoak alongside certain species of true oaks (*Quercus* spp.). By 2000 the pathogen, *Phytophthora ramorum (P. ramorum),* had been isolated and was attributed to the die-off (Frankel, 2008). The disease caused by *P. ramorum* has become known as Sudden Oak Death.

Within a few years of discovery, Sudden Oak Death reached epidemic proportions in parts of California. Despite the efforts of scientists and land managers, the pathogen had rapidly spread across the state killing over one-million trees (Lee et al., 2011). The loss of these trees has resulted in incalculable damage to California's environment, economy, and cultural heritage. This essay will examine how the characteristics of *P. ramorum* and the Sudden Oak Death epidemic in California present challenges and opportunities for land managers trying to protect California's forests. Additionally, management options will be discussed, and recommendations made based on the current state of the epidemic.

Range of Sudden Oak Death Epidemic:

To understand the state of the Sudden Oak Death Epidemic and the challenges involved with the management of this pathogen, it is important to understand the range of the epidemic. *P. ramorum* has been found on the West Coast of the United States as well as in Europe. In the States, Sudden Oak Death has been identified in a 650 km strip from the Big Sur coastline in Monterey County, California, to Curry County in Southern Oregon. In California, fifteen counties along the Pacific coast have been identified as infected with Sudden Oak Death

(Alexander and Lee, 2010). Although generally occurring within 30 km of the Pacific coastline, Sudden Oak Death has also been reported as far inland as Solano County, which is 70 km off the coast (Davidson et al., 2003). In California, *P. ramorum* has been reported at elevations from as low as sea level to as high as 800 meters (Davidson et al., 2003). If land managers do not understand where Sudden Oak Death infections have been taking place, they have little chance of understanding where the pathogen may go, and what needs to be protected. Indeed, the United States is not the only country threatened by Sudden Oak Death.

In Europe, *P. ramorum* has been found in Germany, the Netherlands, Spain, Belgium, Poland, Sweden, France, and the United Kingdom (Davidson et al., 2003). Originally, little concern was placed on the pathogen in Europe, as it seemed to only occur in rhododendron species. However, after 2009 this changed as the pathogen was found to be killing japanese larch in the UK (Alexander 2012). Since 2009, increased scrutiny has led to the pathogen being located on sitka spruce (*Picea sitchensis*) and noble fir (*Abies procera*) throughout Europe (Alexander, 2012). *P. ramorum* has caused death in trees both in Europe and in the United States. The continuing discovery of the pathogen in a variety of tree species is cause for concern. Still, the rapid spread of sudden Oak Death across California over the past twenty years has presented the most daunting problem and challenge to both scientists and land managers.

The 650 km range of Sudden Oak Death in the United States makes management of the pathogen difficult. Intensive management actions in a single area may be able to significantly lower infection levels within a select range, but if these actions are not carried out across the extent of the pathogen's course, it is likely that *P. ramorum* will re-enter previously treated areas from adjacent infection areas. Coordination of management practices across the range of the pathogen in the United States is essential in order to ensure the long-term eradication of the pathogen in California. Coordination of management activities presents very real challenges. Coordination requires cooperation between private landowners and government bodies, spanning both county and state lines. Due to the expansive and varying values of private landowners across California, rallying the cooperation of private landowners may prove to be one of the largest challenges in the management of Sudden Oak Death. The fast moving and dynamic nature of the epidemic has further complicated the coordination of activities. The rapid spread of

Sudden Oak Death across the state caught land managers off guard, and can be attributed to the ability of *P*. *ramorum* to move rapidly across landscapes and infecting hosts with seemingly no resistance to the pathogen.

Origin of Sudden Oak Death:

Sudden Oak Death's rapid spread across the landscape and the apparent lack of resistance to the pathogen in many of its hosts in California strongly suggests the pathogen is an invasive species to the region. The origin of *P*. *ramorum* is currently unknown, (Garbelotto and Rizo, 2003), but the fact that *P*. *ramorum* behaves similarly to an invasive pathogen partially explains why land managers have had so much difficulty mitigating the Sudden Oak Death epidemic in California. No decisive conclusion has been drawn, but it has been hypothesized that P. *ramorum* originated in Europe or South America. One theory suggests *P*. *ramorum* arrived in California through the importation and improper introduction of rhododendron species (Ivors et al, 2006). Whatever its origin, Sudden Oak Death, as suggested by its name, has wreaked havoc on its most susceptible hosts.

Species affected by Sudden Oak Death:

In order to mitigate the Sudden Oak Death Epidemic, it is critical for land managers to understand which species the pathogen is able to infect and how these species are affected. Knowledge of susceptible hosts helps land managers understand where the disease has been, how it spreads, and what species and areas are at risk. Sudden Oak Death is generally associated with mortality in true oak species, as well as tanoak. But the pathogen affects a wide range of species in a variety of different ways.

Oaks and tanoak:

While Sudden Oak Death has killed countless oaks and tanoaks across California, not all oak species in California are affected by P. *ramorum.* Sudden Oak Death has been observed in oak species including; coast live oak, california black Oak (Quercus *kelloggii*), shreve's Oak (*Quercus parvula* var. *shrevei*) and canyon Live Oak *(Quercus chrysolepis*) (Alexander, 2014). Inoculation trials performed in a greenhouse setting, demonstrated that P. *ramorum* was able to infect all california red oak species, including interior live Oak (*Q. wislizenii*), northern red oak (*Quercus rubra*), and pin oak (*Q. palustris*) (Davidson et al, 2003).

Regardless of species, only mature oak trees are mortally threatened by *P*. *ramorum.* Oaks with a diameter at breast height (DBH) of less than 10 cm are generally not affected (California Oak Mortality Task Force). As diameter increases, so does the likelihood of the tree experiencing mortality as a result of infection (Cobb et al., 2013). Infection in wild seedlings has not been reported and infection is highly uncommon in saplings (Davidson et al, 2003). Unlike true oak species, all sizes of tanoak can be killed by *P*. *ramorum* (Alexander, 2014). Though not generally concerned about tanoak, Californians have taken notice of the mass die off of mature oak trees.

The name Sudden Oak Death was dubbed as trees infected with *P*. ramorum can survive for years without any obvious symptoms visible in the trees canopy (Cobb et al, 2013). The tree is eventually girdled and in a matter of weeks the canopy browns and the tree dies (Garbelotto and Rizo, 2003). In oak species, the time from the initial infection to tree mortality can range from several months to a few years, depending on environmental conditions, the physical resilience, and the genetics of the host (Alexander and Swain, 2010). In susceptible oaks species, tanoaks, and madrone *(Arbutus menziesii),* Sudden Oak Death generally manifests as a lethal stem infection. The stem infection results in the formation of a canker that will eventually encompass the bowl of the tree. The canker will serve to sever the tree's cambial system, cutting off the flow of nutrients and water, eventually killing the tree. The disease kills the tree's phloem which transports nutrients, and the tree's cambium, which divides into phloem and xylem (Davidson et al, 2003). The cankers generally occur between from ground level to four meters above the ground (Davidson et al, 2003). Cankers may have a reddish brown to black coloration. The cankers will often bleed with a thick and sticky sap that varies in color from black to amber

and that smells like the inside of a wine barrel (Alexander and Swain, 2010). The sap can cause large brown stains on trees. Understanding the symptoms of Sudden Oak Death will help land managers identify infections; a fundamental step in Sudden Oak Death management. It is important that land managers are able to identify infection, not just in commonly associated trees, but throughout *P. ramorum*'s wide range of hosts.

Other hosts:

In addition to true oaks and tanoaks, Sudden Oak Death is known to be able to affect more than 125 different hosts including woody, brushy, and herbaceous species (Alexander, 2012). An up-to-date list of hosts is upheld by the US Department of Agriculture. Sudden Oak Death affects its many hosts in different ways. While most hosts only suffer from non-lethal foliar infections, Sudden Oak Death is able to kill oaks and tanoaks as well as certain other species including Madrone trees (Garbelotto and Rizo, 2003). Many of the plants and trees that *P. ramorum* can infect are common horticultural plants regularly raised and sold in California nurseries.

While it is well known that Sudden Oak Death kills oaks and tanoaks, in the majority of hosts it manifests as a non-lethal foliar infection, or the infection of small twigs (Alexander and Swain, 2010). Symptoms of infection include brown tipped leaves, brown or yellow spotting on leaves, and possible shoot dieback (Alexander and Swain, 2010). Though foliar infections and small twig infections are both generally non-lethal they can cause mortality in small plants (California Oak Mortality Task Force).

The majority of hosts are not killed by *P. ramorum.* Many hosts do not even support large enough populations of spores to facilitate spread of the pathogen to new hosts (Alexander, 2014). For management purposes, it is extremely important to note that most host species do not support large enough populations of *P. ramorum* spores to facilitate spread to new hosts. It is also critical for land managers to be familiar with the host species that facilitate the spread of Sudden Oak

Death. A few species serve as bases, from which spores can be spread through the movement of water, wind, plant material and/or soil. Foliar infections in non-oak species serve as the main source of the spores that spread Sudden Oak Death (Alexander, 2014) Species that support large enough populations to facilitate spread include; Tanoak, California bay laurel, Rhododendron species and Camellia species (Garbelotto et al., 2014).

Nursery hosts:

Some of the species that support large enough spore populations to spread Sudden Oak Death, are commonly found in California's nurseries. It is important for land managers to be aware that *P. ramorum* may be spread through the trade of horticultural plants as the nursery trade has the potential to spread the pathogen across county, state, and national lines. *P. ramorum* may well have been brought to California by nursery plants. Regardless of its unknown origin, P. *ramorum* was first isolated a nursery in the United States in 2001.(Frankel, 2008). The pathogen was located on rhododendrons in a commercial nursery in Santa Cruz County (Frankel, 2008). The pathogen was identified after the owner of the facility reported a die-back of tanoak, and coast live oak on forest land adjacent to the facility. By 2003, P. *ramorum* had been located in twenty different nurseries in California, Washington, Oregon, and British Columbia (Frankel, 2008). In addition, the pathogen was identified in fifteen nurseries and three public gardens in the UK (Beales et al. 2004). Millions of potentially infected plants were shipped across the world from these nurseries.

Life cycle of P. *ramorum:*

As well as being able to identify potential hosts, a basic understanding of the lifecycle of *P. ramorum* provides essential insight to the management of Sudden Oak Death. The pathogen P. *ramorum* is an oomycete in the Straminipile group. They are related to diatoms and algae (Frankel, 2008). *P. ramorum* exhibits characteristics similar to a fungus or water mold

(Alexander, 2014). Phytophthora species, including *P*. ramorum, thrive in humid conditions, such as the pacific coast of Northern California and Southern Oregon, and produce huge numbers of reproductive spores under the right conditions (Cobb et al., 2013). Much of the spread of *P*. *ramorum* has been attributed to the movement of spores by raindrops and wind (Alexander and Swain, 2010). Drier conditions, such as those observed in the current drought in California, are less suitable for *P*. *ramorum*, and fewer viable spores are available to spread infection (Frankel, 2008).

Management of the Sudden Oak Death epidemic in California wildland is made difficult by many factors. Sudden Oak Death has acted similarly to, and possibly is an invasive pathogen. The pathogen appeared suddenly, and moved rapidly through large areas. Sudden Oak Death has a broad range, crossing county, state, and national borders. Coordination of management practices would require cooperation between different government bodies as well as private landowners. Sudden Oak Death has a wide range of hosts, many of which have seemingly no resistance to the pathogen. SOD can be easily spread through the movement of water, soil, or plant material, and can be difficult to identify.

Detection of Sudden Oak Death:

To further management challenges, currently there is no accepted method for treating hosts that have already been infected by *P*. *ramorum*. As such, disease management practices revolve around limiting the spread of the pathogen, and protecting high value trees (Garbelotto and Rizo, 2003). Any management activity requires knowledge of the presence and location of Sudden Oak Death. The first step in management is the detection of the pathogen (Alexander, 2014).

Laboratory testing:

Early detection has been the first step in protecting trees from Sudden Oak Death. When determining if certain trees have been affected, it first it needs to be ascertained whether the trees in question are a known host species. If the trees belong to a susceptible species, the trees would need to be examined for symptoms of Sudden Oak Death. Visual analysis has been a useful tool for determining the presence of *P. ramorum.* To confirm the infection with certainty, samples must be sent to a lab for testing. Lab testing is necessary as many other pathogens cause similar symptoms. (Alexander and Swain, 2010). Samples can consist of foliar samples or bark samples. Foliar samples from known foliar hosts, such as bay laurel, may be submitted in the form of approximately ten symptomatic leaves (Alexander and Swain, 2010). Bark samples are the only way to confirm infection in a specific tree with certainty. However, bark sampling involves an invasive process that requires special equipment (Alexander and Swain, 2010). Improper sampling technique may greatly bias the results of laboratory testing (Lee et. al, 2011). Samples may be submitted through County agriculture departments, or the University of California Cooperative Extension Office.

SODMAP:

When determining if samples should be sent to the lab, the risk of Sudden Oak Death in the area should be considered. The risk of it affecting an area can be evaluated based on the proximity of the area or trees in question to known infection sites (Cobb et al., 2013). The proximity of the nearest infection may be impossible to determine due to a lack of accurate up-to-date mapping at the appropriate scale (Alexander, 2014). Accurate mapping of infection sites is difficult due to the rapid spread of the disease, and the tendency of P. *ramorum* to affect isolated wild land stands.

One program that has attempted to map Sudden Oak Death across California is the UC Berkeley Forest Pathology and Mycology Lab's SODMAP program. The SODMAP program has mapped the locations of lab-confirmed Sudden Oak Death infections across California from 2005 through 2014. The program uses data from samples submitted by scientists and private citizens.

Proximity of bay laurel:

Another factor that puts trees at risk is the proximity of hosts known to support large enough spore populations to spread the pathogen. Bay laurel is one of the species most susceptible to P. *ramorum.* Often these trees become the first hosts infected in the area. Infection in Bay Laurel trees has been a strong indication that nearby oaks and tanoaks may be infected, or are at a high risk of infection. Symptoms of infection in bay laurel only serve as an indicator that nearby trees may be infected with P. *ramorum.* Similar to oaks, the symptoms found in Bay Laurel caused by P. *ramorum* can be very similar to the symptoms caused by other pathogens (Alexander and Swain, 2010).

Aerial Detection:

An indication of the possible threat of Sudden Oak Death affecting an area is the presence of large patches of dead tanoak or oaks within a one to three mile radius (Lee et. al, 2011). These large patches have been located using aerial photography (Alexander 2012). Many pathogens, which are a crucial part of forest ecosystems, can cause mortality in single or small patches of trees in particular suppressed trees, but wide scale mortality in canopy trees is much rarer. Large patches of mortality in dominant canopy oaks and tanoaks may indicate the presence of P. *ramorum* (Lee et. al, 2011). Aerial photography along with waterway detection have proven to be some of the best methods for detecting P. *ramorum* in wildland settings.

Waterway Detection:

P. *ramorum,* being a water loving microorganism, often lives in waterways as well as plant material and soil. Through the sampling of waterways, it can be determined if P. *ramorum* is present in a particular watershed (Alexander, 2012). This method of detection has proved

effective, though sometimes ambiguous. Detection in watersheds has led to the detection of Sudden Oak Death outbreaks in certain cases, and in other cases no subsequent infection has been found.

One of the most effective ways of monitoring the spread of Sudden Oak Death and detecting outbreaks has been waterway detection (Alexander 2012). Waterway detection has served as an important management tool on many occasions. In central Humboldt County in 2009, P. *ramorum* was detected in a watershed in a part of the county that had previously not contained the pathogen. Within a year, a small patch of infected bay laurel and tanoak was found. The early detection of the small infection area allowed for eradication of the pathogen in the area before it could spread further (Alexander 2012). In other cases the use of waterway detection has proven frustrating, In particular, in areas near horticultural nurseries, P. *ramorum* has been detected in waterways where no local Sudden Oak Death outbreak can be located (Alexander 2012). In these cases, outbreaks in the wildland may be too difficult to locate, or P. *ramorum* may have entered the waterway from a different source, such as a nursery. For this reason, amongst others, it is important for land managers to be aware that P. *ramorum* can be spread from common nursery plants.

Detection in nurseries:

Many common horticultural plants, including rhododendron species, serve as hosts for P. *ramorum.* To prevent the unintended spread of P. *ramorum,* the leaves of all known host species should be visually inspected for signs of infection before being purchased or transported. All host plants shipped from the known infested areas should be inspected. As of May 17, 2001 The California Department of Food and Agriculture initiated restrictions on the export of infected oak and rhododendrons products from the “zones of infestation” (Alexander and Swain, 2010). By law, all host species being shipped from the fifteen known infected counties in California must be inspected and approved before being shipped. If a known host species is purchased, it is advisable to quarantine the plant or tree for around two months to see if symptoms manifest

before planting. It is not advisable to plant host species in close proximity to susceptible oak trees, tanoak, or arbutus (Lee et. al, 2011).

Management practices:

After it has been determined that Sudden Oak Death is present in a particular landscape, or that the landscape is likely to be threatened by Sudden Oak Death, management activities fall into two categories; preventing the spread of the pathogen, and the protection of high value trees. The following methods have been utilized to keep Sudden Oak Death out of areas that have not yet been infected; removal of infected oak, removal of host species, implementation of barrier zones, sanitation practices, fostering forest health, and the use of fungicides.

Removal of infected oaks:

Oaks are one of the relatively few hosts in which P. *ramorum* infection causes mortality. While removal of infected oak trees has been attempted to prevent the spread of Sudden Oak Death, oak trees do not support large enough populations of P. *ramorum* spores to facilitate the spread of Sudden Oak Death to new hosts. As such, removal of infected oaks is not an effective strategy to mitigate the spread of P. *ramorum* (Alexander and Swain, 2010). Whether an infected oak should be removed or not, is the decision of the land owner. In wildland areas, where human access is rare, infected oaks should be left standing in order to maintain stand structure and wildlife habitat. In areas where human access is more common, such as parks or residential areas, a hazard tree assessment should be performed by a qualified professional, and if the tree is found to be a significant hazard to life or property it should be removed (Alexander and Swain, 2010).

Removal of host species:

While removal of oak is not an effective management strategy, one of the most effective methods of preventing the spread of Sudden Oak Death is the removal of specific host species from the landscape. P. *ramorum* only spreads from foliar infections, so removal of certain non-oak host species, if feasible, may prevent spread to neighboring oak trees. In particular, the removal of Bay Laurel and Tanoak may be effective. It has been found that P. *ramorum* produces more spores on Bay Laurel than any other host (Alexander and Swain, 2010). Due to the number of spores produced on Bay Laurel, the greatest correlation in oaks for a high risk of infection, is the proximity of bay laurel to the tree (Lee et al., 2011).Removal of host species can be performed manually or, if conditions and regulations allow, through the application of herbicides or the use of prescribed fire.

Removal of Bay Laurel and Tanoak is one of the most effective strategies for preventing the spread of Sudden Oak Death available to land managers (Alexander and Swain, 2010). While this strategy may be effective on smaller landscapes such as private property or parks, application on a statewide scale presents many challenges. In order to manage the Sudden Oak Death Epidemic on a whole, removal of Bay Laurel and Tanoak across the range and potential range of Sudden Oak Death would be required. Removal can be performed manually, through the application of herbicides, or through the use of controlled burns (Alexander, 2012). The removal of host species across the 650 km range of P. *ramorum* would present extreme challenges as it would require huge financial and labour commitments. Cooperation across state and county lines as well as cooperation between government and private landowners would be required. One method to limit the challenges of widespread host removal is the implementation of barrier zones.

Implementation of barrier zones:

In order to mitigate the challenges of widespread host removal, the use of barrier zones is an effective. One strategy for limiting the spread of Sudden Oak Death on a landscape scale, involves the creation of barrier zones. Barrier zones can be created through the removal of host species throughout the zone. By removing all hosts between an infected area and an uninfected area, the likelihood of the spread of P. *ramorum* to the uninfected area is decreased.

An example of the creation of barrier zones occurred in 2007. The Pacific Lumber Co. was funded by the USDA Forest Service to create a 3.2 kilometer wide and several kilometer long barrier zone, by treating hosts with herbicide in order to prevent northward spread of the disease. The zone was created 80 kilometers north of the Redway/Garberville infection zone (Fankel, 2008).

While the removal of host species in barrier zones may be an effective way to mitigate the spread of Sudden Oak Death through natural processes, this strategy can be quickly undermined by careless land users. To effectively limit the spread of Sudden Oak Death, the introduction of infected material into disease free areas must be prevented. P. *ramorum* can be carried through the movement of water, infected plant material, and soil (Alexander, 2014). Though limiting the spread of the disease through the flow of waterways may be very difficult, there are steps that can be taken to limit spread through the movement of soil and infected plant material. On the February 14, 2002 the United States Department of Agriculture Animal and Plant Health Inspection Service created federal regulation restricting the transport of host material from the "zones of infestation". International restrictions were also implemented in coordination with the European Union and Canada, the UK, South Korea, New Zealand¸ Australia, the Czech Republic, Mexico, and Taiwan.

Sanitation practices:

Those working or recreating in areas affected by Sudden Oak Death should clean the tires of vehicles or bikes as well as shoes, clothing, tools, or equipment that were used in the area with

a bleach based compound before leaving the area. Signs should be posted in infected areas to alert those using the area of sanitation practices and their importance.

Likewise effort should be made to limit the transport of infected plant material. Under no circumstance should any plant or derived product (including firewood) be transported from an infected area. If pruning, thinning, removing Bay Laurel, or other similar activities, foliage, branches, and stem material should be disposed of onsite, to prevent the spread of P. *ramorum* to other areas. Material should either be burned, or chipped and then composted. Both of these methods will destroy the P. *ramorum* pathogen. If material is chipped, the chipper should be sanitized with a bleach-based product before being removed from the site.

Fostering forest health:

As well as attempting to limit the spread of Sudden Oak Death, land managers can try to protect specific high value trees that have not been infected from becoming infected. Fostering tree health is one method that has been tried in order to prevent trees from becoming infected with Sudden Oak Death. Despite efforts, native species have not evolved resistance to the likely invasive microorganism P. *ramorum.* Therefore, Sudden Oak Death is capable of killing trees in all states of health. Consequently, maintaining the health of trees is not a totally effective method of protecting them from Sudden Oak Death. Healthy, vigorous trees do have a greater potential to resist, or even recover from P. *ramorum* infection than the suppressed, unhealthy, or stressed trees. Therefore, maintaining and fostering overall forest health is recommended when managing against Sudden Oak Death (Lee et al., 2011).

Application of fungicide:

A final method of protecting high value trees is the application of a phosphonate based fungicide sold named Agri-Fos. The application of Agri-Fos on individual trees has been shown

to be an effective method of protecting them from P. *ramorum* infection (Lee et al., 2011). While Agri-Fos application is effective for protecting high value trees on a landscape scale, widespread application is not a feasible option due to the high cost of the chemical and the number of man-hours that would be required. (Alexander, 2012).

Conclusion:

The Sudden Oak Death epidemic in California has proven to be an extraordinarily difficult challenge for land managers and scientists. The epidemic, caused by the pathogen P. *ramorum* has killed over one million trees across the state. Countless amounts of cost and damage to both private and public land. Despite efforts, there has been little success in mitigating the rapid spread of Sudden Oak Death or damage caused by its effects.

Severe damage has already been caused to coastal forests in central and northern California. While it is difficult to speculate, it can only be assumed that if the pathogen continues to spread unchecked, severe and possibly irreversible damage will be caused to California's forests. Damage may be in particularly severe in those forests where traditional oak species play significant roles in the ecosystem.

While the situation is grave, the fate of California's coastal forests is by no means sealed. Though vast amounts of damage have been done, the efforts of land managers have helped slow the spread of the pathogen, keep P. *ramorum* out of areas that otherwise would have infected, and protect high value trees. One source of hope has debatably caused greater harm to California than Sudden Oak Death.

The ongoing record-setting drought that has been plaguing California for the last four years has, according to some, been more successful in reducing P. *ramorum* infection across California than any technique so far implemented by land managers. *The San Francisco Chronicle* published an article in June of 2014, entitled *Sudden Oak Death Drying up with*

Drought. The article quoted Dr. Matteo Garbelotto, the head of UC Berkeley's Forest Pathology and Mycology Laboratory, and one of the lead scientists heading research on P. *ramorum*, stating: "The pathogen hates the drought. It's like an outbreak of influenza. When it is cold and wet, people get sick. We know that when it is dry, we do not have outbreaks."

*The San Francisco Chronicle'*s article went on to state that according to surveys done between April 4th and June 5th, 2014 infection levels on California Bay Laurel in the 15 infected counties ranged from 2-10% while pre-drought levels ranged from 20-80%. The survey was performed by using the SODMAP program.

Through the conclusions of the San Francisco Chronicle have not been put through the peer review process, they offer some insight into the results of the SODMAP survey. It is clear from viewing the map for 2014 that very few new infections were reported that year, possibly supporting the theory that the drought is limiting the spread of Sudden Oak Death. Despite this, 2013 was one of the highest years for reports of new infections. In 2012 there were also relatively high numbers of new infections reported compared to previous years, though this may be attributed to heavy rains in parts of California in 2011. Regardless of *The San Francisco Chronicle*'s claims, the results of the SODMAP program cannot be interpreted as clear confirmation that the drought in California has lowered infection results, as other factors may be at play. The SODMAP results may have been unintentionally biased by the reporting process due to changes in public awareness and perception of Sudden Oak Death over time. This may be a factor in the relatively low report numbers from the early years of the epidemic.

The theory that the spread of Sudden Oak Death has been limited by the ongoing drought in California has not been confirmed. The theory may prove to be false. But as it has been well documented in scientific literature that Sudden Oak Death thrives in moist and humid conditions, the theory may prove to be correct. If correct the drought may serve to end the Sudden Oak Death Epidemic by itself. If the drought alone is not enough to stop the spread of Sudden Oak Death, then the actions of land managers in combination with the drought may be. Considering the amount of damage the pathogen has already caused and its ongoing potential to cause harm, it is not unreasonable to act on the theory.

The current year, 2015, so far being an even drier year than 2014 in California means that, if the spread of Sudden Oak Death is being limited by the drought, aggressive action to be taken against the pathogen this year may be particularly effective. Dr. Matteo Garbelotto, referring to the drought, said: "This is a little bit of a positive because it gives us an opportunity to see and attack the weakest link of the disease - that is, when the populations really, really shrink." To take advantage of the shrinking populations a series of management activities, as highlighted in this essay should be decided upon and undertaken. If these actions are taken as soon as possible, in combination with the ongoing drought there may be a real chance of effectively combating the spread of the Sudden Oak Death in California.

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