

SUDDEN OAK DEATH: A TALE OF TWO CONTINENTS

Matteo Garbelotto, Extension Specialist and Adjunct Professor, Department of Environmental Science, Policy and Management, University of California, Berkeley, CA, USA describes this new disease of oaks and related trees and compares the infection potential in the US and Europe

An emergent plant disease is uniting the European and the North American continents, its name, Sudden Oak Death (SOD) may be puzzling to most Europeans, as indeed, mortality of native oaks has not been widely reported in Europe. Historically, this disease owes its name to the rapid crown decline of SOD-affected trees observed in California in the early 1990s. Ironically, the most susceptible California trees are not oaks, but tanoaks a related species belonging to the genus *Lithocarpus*. California red oaks and intermediate oaks were subsequently identified as highly susceptible to SOD, displaying similar symptoms, e.g. bleeding trunks and rapid browning of the entire crown. In 2000, two significant events marked the history of SOD research: the discovery of the causal agent behind SOD and the unexpected finding that the same agent had been previously isolated not from bleeding trees, but from blighted ornamental rhododendrons in Germany. The microbe in question was at that time still undescribed, but it was subsequently named *Phytophthora ramorum*.

The genus *Phytophthora* is certainly a familiar one for pathologists and tree care specialists as it includes well-known plant pathogens, causing diseases in trees defined by descriptive names such as ink disease or gummosis.

Why then, is there so much concern in both continents, about this emergent disease? The answer lies in the levels of destruction this microbe has caused locally in California. In some sites, 100% of the mature tanoaks and 45% of California coast live oaks have been killed by *P. ramorum*, with obvious ecological and socio-economical consequences. To compound the seriousness of the SOD issue, *P. ramorum* is inevitably little understood since it has been recently discovered. Unfortunately, it appears to be an oddball in the world of forest Phytophthoras. While most forest Phytophthoras in the temperate world are root-infecting soil-borne and water-borne-organisms, *P. ramorum* spreads mostly aerially and generally infects trees and plants above the soil line, while maintaining the soil-borne and water-borne features of its close relatives. In light of the actual devastation caused by SOD in California, and because of the high levels of uncertainty, due to our limited knowledge of the biology of the causal agent, there is great concern surrounding the presence of *P. ramorum* in nurseries. Infestations in nurseries have been reported in most European Union countries, in Washington State, in British Columbia (Canada) and in Oregon State, where natural infections in a limited portion of a single county are actively being curtailed by a proactive eradication program. The discovery in 2003

of individual trees killed by SOD in Europe in correlation with the previous planting of infected rhododendrons has reaffirmed the need for active management of this disease.

...“Same but different”...

While irrefutable evidence pointing to a putative exotic nature of *P. ramorum* in both continents is still lacking, most scientists agree SOD seems to have the features of an emergent, i.e. ‘new’, disease. The high levels of mortality recorded in California are reminiscent of high mortality levels caused by exotic diseases such as chestnut blight, Dutch elm disease, and white pine blister rust. The causal organism and the disease have never been described before, and a more in depth genetic analysis of North American and European populations, revealed that there is limited genetic variability in each continent, another typical trait of introduced diseases. *P. ramorum* requires individuals bearing two different mating types (A1 and A2) in order to complete its sexual cycle. In nature in North America, only A2 individuals have been reported until now, while – with a single exception – only A1 have been reported from nursery or nursery-linked infections in Europe. DNA analysis has shown that the European and the North American populations are genetically very distinct and represent two lineages that are not intermixing with each other. This genetic distinction is also matched by different traits such as growth rate, morphology and differential pathogenicity on a range of hosts. Presumably, both mating types and representatives of both lineages would be present in the area where *P. ramorum* originated: the location of this area still eludes the world scientific community.

In nurseries of the Pacific Northwest, individuals belonging to both mating types and lineages were discovered in 2003; in plants adjacent to one another. The A1 and A2 isolates were shown to be interfertile in the laboratory. These findings are troubling for two reasons: a) The European lineage is, as stated above, genetically quite different from the North American one and could cause a second different wave of SOD; and b) The two lineages could mate and generate progeny with new traits, thanks to sexual recombination events.

The presence of sexual activity may turn *P. ramorum* into a formidable adversary; even without sex, this organism has been able to colonize entire forests. *P. ramorum* in fact produces large amounts of asexual infectious propagules. Genetic analyses have shown that a single clone has



Figure 1. Sudden oak death lesion on a bay laurel leaf (*Laurus nobilis*) (Photo courtesy of Matteo Garbelotto)

colonized most of California, while a larger variety of clones is present in European nurseries. At times, nurseries in different countries share the same clone: clearly indicating *P. ramorum* is traded with its hosts among nurseries. The finding that some North American nurseries are infested by both lineages is strongly suggestive of the role played by the industry in unknowingly aiding the movement and maybe even the introduction of this pathogen in the continent.

The biology of *P. ramorum* and the symptoms of SOD

Infestations in nurseries and in the wild (with the exception of the vast spread of California coast already colonized by the pathogen) have been treated very aggressively with a slash/burn/spray approach. Despite these strong efforts, true eradication of the pathogen has not always been possible both in the wild and in nurseries. One of the reasons for the limited success of these operations may be due to the limited understanding of the life cycle of the pathogen. Here is a summary of our knowledge of the pathogen's biology, based mostly on studies of diseased forest areas in California. The life cycle of the pathogen in forests elsewhere, or in artificial environments such as commercial nurseries, may differ



Figure 2. An exposed canker under the bleeding bark on a tan oak in the Big Sur (Photo courtesy of Matteo Garbelotto)

significantly from what is described in this section. Weather patterns and host composition will necessarily have a huge impact on the epidemiology of the disease.

Early symptoms

In order to fully understand SOD, we need to remove ourselves from the implicit preconception that the disease is



Figure 3. Characteristic bleeding on a coast live oak (Photo courtesy of Matteo Garbelotto)

“sudden”, that it affects mostly “oaks” and that it is always, or almost, “deadly”. It is estimated that a few years (two at a minimum) may be required for the disease to cause mortality at any infested site. In nature, infestations are driven by plant hosts that can be infected easily and support sporulation of infectious and/or resting propagules. Epidemiologically important hosts in California include in order of importance, bay laurel leaves (*Umbellularia californica*, Lauraceae), tanoak twigs and leaves (*Lithocarpus densiflora*, Fagaceae) and redwood needles (*Sequoia sempervirens*, Taxodiaceae). The importance of these hosts is exemplified by the fact that, with a single exception, all infested California sites, are characterized by the presence of bay laurel and/or tanoaks. Circumstantial evidence points to rhododendrons and camellias as potential important artificial hosts for the disease, possibly linking the ornamental industry and the wild land components of SOD. Leaves and twigs of these and other susceptible hosts are readily colonized by swimming zoospores carried in a microscopic oval-shaped structure called sporangium. The sporangium can be airborne and once it lands on a suitable host surface it will release the zoospores. The presence of a film of water on the plant surface for several hours seems to greatly facilitate the infection process. Warm temperatures

(around 18–20°C) also seem to be ideal for infection. Once infected, leaves and twigs develop black lesions often marked by a darker line, called a reaction zone. Reaction zones are more marked in hosts that, at least temporarily, can block off the growth of the pathogen. Species that are extremely susceptible (e.g. some *Rhododendron* species) do not normally display reaction zones, but the margin of the lesion is rather diffuse. In conditions of high humidity (close to 100%) and moderate temperature, new sporangia and resting round structures called chlamydozoospores will be produced on the lesions in approximately 48 hours. Chlamydozoospores produced by other *Phytophthora* species have been shown to be resting structures that allow for the survival of the pathogen during harsh weather conditions, but their role for *P. ramorum* is still unclear. These events mark the end of the initial stage of disease and the beginning of the second phase.

Second phase of the disease

Infestations will most likely be driven by the epidemiologically important hosts, but once in a site, the pathogen is capable of infecting a very broad range of hosts including trees and shrubs, woody and herbaceous perennials. In



Figure 4. A tan oak killed by sudden oak death (Photo courtesy of Matteo Garbelotto)



Figure 5. Dead coast live oaks at China Camp in Marin County (Photo courtesy of Matteo Garbelotto)

addition, re-infection of individual plants is a most likely occurrence. In some hosts leaf infection is accompanied by branch infections causing typical branch die-back symptoms: in the second phase of the disease leaf lesions may proceed into the stems and vice versa leading to obvious die-back symptoms such as those observed for instance in tanoaks, rhododendrons, Pacific madrones (*Arbutus menziesii*, Ericaceae), Toyons (*Heteromeles arbutifolia*, Rosaceae), Pacific huckleberry (*Vaccinium ovatum*, Ericaceae). In some hosts, including bay laurel, maple spp. (*Acer*), buckeye (*Aesculus californica*, Hippocastanaceae) only leaves are affected. Infectious propagules will also accumulate in the soil and water streams: soil and water are bound to play an important epidemiological role, especially when factoring in human fruition and management of woodlands. It has already been shown that leaves of susceptible hosts will become infected when splashed with infected soil. On the other hand, what happens at the interface between roots and infested soil awaits further research. Although dead and fallen leaves appear to lose infectiousness quite rapidly, they also contribute to increase the inoculum potential of the soil and water.

The third and the final phases of SOD

In the next disease phase, the main stems of tanoaks and oaks may be infected. Although infection appears to occur in the absence of any visible wound, *P. ramorum* may choose the path of least resistance, whether lenticels or other openings in the bark. The pathogen colonizes preferentially the sugar-rich phloem of the host, while only marginally colonizing the outer bark and the xylem. As a result of phloem colonization, a canker develops under the bark, and sappy exudates will flow outside the bark. Aerial seeps not connected to the root collar are good indications that a tree has been infected by *P. ramorum*. Other *Phytophthora* species, in fact, can cause identical seeps, normally though linked to root or root collar infection. It is noteworthy to mention that multiple cankers, at different heights, are often visible on tanoaks. These cankers are thought to be started by sporangia produced on leaves and twigs of the same or adjoining plants. On the more resilient coast live oak trees, cankers are normally found in the lower part of the bole, presumably where sporangia accumulate, or in areas adjacent to sources of sporangia, e.g. infected bay leaves. Coast live oak leaves and twigs, in fact, are never or rarely colonized by *P. ramorum*. Although the production of toxins by *P. ramorum* has not been excluded yet, it is believed that the main effect of the disease on oaks and tanoaks is girdling of the cambium. Upon girdling, the irreversible death process is started, but it may take a year or more, before the browning of the crown becomes apparent.

Significant girdling of the tree circumference marks the beginning of the last phase of the disease, characterized by the presence of secondary organisms and opportunistic pathogens. Bark and ambrosia beetles are attracted by these dying trees, and a variety of wood decay fungi and canker rots can develop freely on the moribund specimens. At the landscape level, this phase is characterized by the removal of

the most susceptible species including not only oaks and tanoaks, but also other plant hosts.

Epidemiological notes

Areas with wet climates and constant mild temperatures, resembling the central California coast are optimal to disease development. Most of the sporangia production in California occurs during the rainy season (December to June). In the dry season, sporangia production is reduced dramatically, and as the soil dries, *P. ramorum* in the soil becomes non-viable, whether permanently or temporarily is still unclear. The pathogen can be recovered all year round in streams, indicating that sporulation is also occurring all year round and the pathogen is viable when it is in favorable environments.

The disease spreads almost exclusively where epidemiologically important, or foliar hosts, are available. The levels of mortality appear to be correlated with incidence of foliar hosts: maximum levels of mortality in tanoaks are observed where both tanoaks and bay laurels are present, while coast live oak mortality is positively correlated with abundance of bay laurel trees. There is no record of SOD from areas where coast live oak grows without bay laurel. It has been recently shown that some populations of bay laurels are less susceptible to infection by *P. ramorum* than others. In the absence of other foliar hosts, areas characterized by these more resistant bay trees should be less prone to epidemic-levels of SOD. Additionally, in each one of seven coast live oak populations tested, some individual oaks consistently appeared to be "slow-cankering" when challenged with the pathogen in artificial inoculation studies on cuttings. This trait has been also observed in nature, where a few trees have survived for several years despite being infected.

Sporangia will be effectively airborne for 5 metres; thus, increased spacing among trees may slow down the spread of the disease. *P. ramorum* will sporulate on the epidemiologically important hosts listed above, but also on many of its foliar hosts: two native California roses were just found to be infected and able of supporting sporulation. Sporulation does not seem to be important on the bark of oaks and tanoaks, but it is triggered in debarked wood. The abundance of foliar hosts in California and the ability to be spread aerially for short distances can easily explain local infestation, but rarer long distance events, or movement aided by animals and humans have to be invoked to explain the overall range of the pathogen, which stretches for over 300 miles along the California coast. Predictions of SOD in Europe are hinged on the understanding of which hosts may be significant sources of infection, and which hosts may be deadly affected by the disease. As in California, white oaks have been shown to be relatively resistant to *P. ramorum*, while Mediterranean live oaks and European beech were highly sensitive to the pathogen when tested in the laboratory. North American red oaks are also widely planted in the old continent; most species belonging to this group of oaks are highly susceptible to the disease. In the case of Europe, interregional transport of the disease appears to have already occurred through the trading and sales of ornamental plants.

A case of “do or die”? Emerging management options for SOD

Preventing the introduction of *P. ramorum* into new areas should be the first priority when managing SOD. This entails identifying the most likely sources of infection: in order of importance these sources may be live plants, green waste, soil and water, debarked untreated wood, entire logs or firewood. Plants supporting sporulation are obviously more likely to start new infestations than ones that do not do so. For non-live substrates, moisture content is indeed a decisive factor in determining potential risk associated with the substrate. The drier the substrate, the less likely it is to be contagious; it should be noted, however, that if the substrate supports sporulation, it may become highly infectious upon being re-hydrated. Wood chip from coast live oak for instance can be easily sanitized by air-drying at room temperature, while bay leaves – which support abundant sporulation – require heat treatment in order to be sanitized.

Green waste is normally composed of both woody and leaf debris and should be considered infectious if originating from an infested area. Prolonged exposure (> than 7 days) to temperature of 55°C, or composting have been shown to sanitize these substrates effectively; the jury is still out regarding mulching as a sanitation tool. Some specific protocols used by the spice industry combine in a 22 hour treatment progressive heating to 55°C and a moderate vacuum, again effectively killing the pathogen in bay leaves. As mentioned above, soil can be infectious, and the elimination of soil particles from vehicles, shoe soles, paws, or arborist tools is essential and potentially more important than the actual surface sterilization of tools or tires using bleach or lysoform solutions. The use of infested water may be an issue in forests where water is drawn from creeks for dust abatement. When *P. ramorum* gets into the water in recycling systems of nurseries, it can be recovered with ease from the water, providing a convenient source of infection.

Once infection is established, an obvious strategy is that of decreasing the amount of infection at a site. This may be achieved by elimination of infected plants or plant parts, by partial removal of top soil and litter, especially under plants known to be infected, and in the case of nurseries, by the use of contact fungicides like copper derivatives or metalaxyl. Fungicides though are bound to simply mask the presence of the pathogen. In our experience, even metalaxyl caused only a maximum of 50% reduction in viability. The second step consists in slowing down the epidemic: this can be achieved in a variety of ways: spacing between foliar hosts should be increased, combination of foliar and terminal hosts (e.g.

oaks and bay laurel) should be avoided, recycled water should be treated, movement of soil avoided, drip irrigation should be preferred to overhead irrigation, early morning irrigation and decreased shading levels should be implemented to avoid long hours of water accumulation on plant surfaces. Fertilization should be avoided, as *P. ramorum* favors succulent fast grown tissue. Management operations such as plant removal should be carried out during cold spells or dry periods, when the pathogen is least active. Wounds in tree areas exposed to inoculum may be colonized promptly by *P. ramorum*: small branch pruning of oaks for instance rarely increases infection levels, as they are not likely areas for sporangia accumulation, but larger wounds and pruning of major branches, may increase infection by *P. ramorum*.

Oaks and tanoaks at risk can also be treated using phosphites. Two registered treatments are available in California, and work optimally in a preventive way: injection in the outer xylem and topical application of phosphites combined with an organosilicate surfactant-penetrant. In both cases, the compound is systemically translocated and broken down into phosphonic acid, the active ingredient that triggers a defense response by the plant. Research has shown that these treatments will effectively prevent infection and slow down the growth of *P. ramorum* cankers. Other treatments aimed at coating the bark of trees with a contact fungicide were not shown to be effective. Phosphite treatments will not kill the pathogen, but only slow it down, and are designed to be administered on a yearly basis. Foliar treatments of oaks and tanoaks using phosphites only provided a short-lived enhanced resistance, and caused significant phytotoxicity. In Australia though, many plant species respond well to foliar treatments: this may be a further option available to Europeans.

For further readings I recommend to log on www.suddenoakdeath.org and check all the information and links available there.

Matteo Garbelotto is an Extension Specialist and Adjunct Professor of Forest Pathology and Mycology in the Department of Environmental Science, Policy and Management of the Ecosystem Sciences Division at the University of California at Berkeley, where he has studied sudden oak death following its outbreak in California.