Phytophthora ramorum in North America

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Two apparently unlinked events marked the beginning of **one of the most devastating forest diseases known to man in recent times**: Sudden Oak Death (SOD). In the mid 1990s, while a novel species of *Phytophthora* was being isolated from mildly diseased *Rhododendron* and *Viburnum* plants in German and Belgian nurseries, the first reports of inexplicable death of tanoaks (*Notholithocarpus densiflorus*) started to come in from concerned citizens on the coast of California. In 2000, red oaks such as Coast Live oak and black oak were also reported to be dying inexplicably, and in June of that year an undescribed *Phytophthora* species was isolated and quickly identified as the causal agent of the disease known as sudden oak death. As it turns out, the species from European nurseries and the one infecting California oaks and tanoaks turned out to be the same. This novel species was named *Phytophthora ramorum* a name that refers to the branch dieback it causes in rhododendron and other ornamental plants, but on oaks and tanoaks, the pathogenic activity of the organism is mostly concentrated on the cambial layer of the main trunk, which gets rapidly destroyed by the growth of the pathogen. *P. ramorum* was maybe the first aerial *Phytophthora* species described from temperate forests: the deciduous large sporangia become airborne and are responsible for the apparently aerial dispersal of the pathogen. The exotic nature of the pathogen was immediately postulated based on its limited geographic distribution around the San Francisco Bay Area, on the fact that both the disease and the causal agent had never been described before, and on the very high susceptibility of tanoaks and red oaks.

Pathways of spreading into USA

Because of its known association with ornamental plants in Europe, the presence of *P. ramourm* in California nurseries was rapidly ascertained, thus providing a potential pathway of introduction into California. Although it was later found out that populations of *P. ramorum* in Californian forests and European nurseries belong to evolutionary distinct lineages of the pathogen (thus negating a European source for the California forest infestation), both lineages and an additional third one were found to be **present in US nurseries**. A series of papers confirmed the genetic diversity of the pathogen in California forest is extremely limited, and convincingly showed that **nursery genotypes were ancestral to the entire pathogen population in the wild**, providing solid evidence that the SOD pathogen had been introduced from an unknown location through the nursery trade. These studies also showed that multiple independent introductions had occurred in California and southern Oregon. The repeated introductions from infected nursery stock at different locations explained more convincingly what on surface appeared to be an incredibly rapid spread of the pathogen.

SOD infects almost all native plant species in the coast California

By 2004 it was determined that 1 Oregon and 14 California counties were infested and that several hundreds of kms of coastal forests were being severely affected by the spread of the pathogen: **up to 100% of adult tanoaks and 60% of oaks are currently (2011) locally reported as dead as a result of infection by this pathogen**. The die-off of these keystone trees has been shown to alter the ecology of coastal forests (including the unique redwood forest) in more than one way, with long lasting proven implications for productivity, regeneration, fungal symbionts, and wildlife. *P. ramorum*, not unlike many other *Phytophthora spp*. is a generalist, and it was found to be able to infect almost all the native plant species on the coast California, including herbaceous and woody plants, monocots and dicots, ferns and trees. Not all infected plants respond to infection in the same way: while oaks and tanoaks are killed by girdling cambial lesions, many plants develop a disease that is similar to that of rhododendrons and is better described as a branch die-back, that may progressively kill the plant. Finally, some plants only develop foliar lesions with rather minimal impact on infected individuals. These two types of disease (die-back and foliar symptoms) are referred to as Ramorum blight and better describe the disease in the understory and in ornamental nurseries.

Spread of SOD by aerial spores

A further layer of complexity is added by the fact that lesions on infected oak and tanoak trunks rarely produce infectious sporangia, while large number of these infectious structures are produced on leaves of trees including California bay laurels, tanoaks, and redwoods. In particular bay laurel and tanoak leaves have both been demonstrated to be key drivers of the disease. Because oaks are mostly sympatric with bay laurels, this species is responsible for the vast majority of oak infections, thus creating a complex system in which density of one species (bay laurel) will affect disease incidence of the other (oaks).

Foliar infections progress rapidly and in the presence of rainfall and moderate temperatures sporulation can be attained within 48-64 hours from infection: it is this incredibly **rapid reproductive cycle** that allows the pathogen to spread even during short wet spells or after long droughts. Although the large sporangia were shown to be airborne mostly within 10 m from a source, evidence from field observations and from spatial autocorrelation studies based on genetic data, also indicate the occasional ability to move up to 5 km from a source, thus allowing the organism to cover the gaps frequently present in forest cover. Notwithstanding the occasional natural ability of the pathogen to move at distances of a few kms, **long distance dispersal is most likely linked to the movement of infected substrates by humans**. While infected plants are an obvious and proven pathways for long-distance dispersal of the pathogen, wood, soil and contaminated water may also act as infectious vectors of the disease. At present, **the disease is still spreading** and when rains occur between mid-April and mid-June, outbreaks are reported throughout the range of the pathogen.

Possibility to prevent further spread of SOD in USA

The scale of the problem in California is such that no single institution, whether the State and Federal Governments, or the US Forest Service has the means to tackle it. UC scientists are currently engaging private landowners as well as managers of

county open spaces and regional parks to take charge and attempt to manage the disease, at least locally. Proven management tools to curtail the disease include preventive phosphonate treatments and selective thinning of infectious hosts such as bays and tanoaks.

Although a significant variability in susceptibility has been reported for bays, oaks, and tanoaks, it is unclear how much of this variation is environmentally driven vs. genetically acquired. A large common garden experiment is under way only for tanoaks, and preliminary results indicate an **absence of qualitative resistance**, while some quantitative tolerance to the disease has been detected both in laboratory tests and in plantings of seedlings n naturally infested sites.

Although nursery plants are highly regulated and periodically inspected for Ramorum blight, the fact that the disease **may remain asymptomatic for a significant amount of time** poses obvious challenges. The discovery of an oak recently infected by a nursery pathogen genotype in the Presidio National Park in San Francisco, and the infestation of rivers in Washington State and in the Southeastern USA, where only nursery infestations have been reported, show that, **in spite of regulations, a lot remains to be learned about this pathogen** in order to effectively prevent its movement and introduction. Because three evolutionary distinct lineages characterized by distinct genetic and phenotypic traits are present in US nurseries, while a single lineage is present in forests of California and Oregon, the potential escape of new genotypes is worrisome not only in other parts of the country but also in areas currently infested. The worry is that the release of genotypes belonging to the lineages currently absent in California and Oregon may intensify the outbreak.

Currently only about 10% of the habitat that could be colonized by *P. ramorum* has already been invaded, while the remaining 90% is obviously at risk. If indeed the pathogen will colonize its entire potential habitat, the damages it would cause would be orders of magnitude higher than the damages caused by the current infestation. SOD represents yet an additional current example of how human activities and natural plant communities are inextricably intertwined. One interesting fact: ***P. ramorum* was the first quarantined pathogen whose lack of detection had to be confirmed by DNA-based techniques in the USA**.

Bleeding tanoak (Photo by Matteo Garbelotto)

Tanoaks killed by *P. ramorum* (among green alive redwoods and Douglas firs) in California. (Photo by Janet Klein)