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Mortality projections and treatment recommendations for sudden oak death

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Since its first published description by Pavel Svihra in 1999, sudden oak death is now estimated to have killed over one million trees (Garbelotto, 2007). The disease is caused by the fungus-like pathogen *Phytophthora ramorum* (Rizzo *et al,* 2002), and has had devastating effects on the California's central coast forests, as well as a small patch of forest in extreme southwestern Oregon (Goheen *et al,* 2002b).

Because this disease was unknown until the 1990s, much of its biology and control are new to science, and our understanding is still changing over time. Prior to its discovery, there were no known foliar *Phytophthora* species killing trees in the forests of the western United States. Since the discovery of *P. ramorum*, two new *Phytophthora* species have been discovered and described: *P. nemorosa* (Hansen *et al,* 2003) and *P. pseudosyringae* (Jung *et al,* 2003). While these other *Phytophthora* species produce symptoms identical to *P. ramorum*, they only kill trees individually or in small clusters, not on the landscape scale, so it's a good bet that if you see mortality on the landscape scale, it's *P. ramorum* that's responsible. However, because the symptoms of these *Phytophthora* spp. are so similar, testing is still generally required to tell which pathogen is causing the decline of any given tree.

Elevated mortality levels due to *P. ramorum* infection on oak (*Quercus spp.*) and tanoak (*Lithocarpus densiflorus*), typically follow particularly wet spring weather in areas with a high density of foliar hosts such as bay laurel (*Umbellularia californica*) (Rizzo *et al,* 2002). In California, the conditions in the spring of 2005 and 2006 were ideal for disease development, and consequently an upsurge in tanoak death is beginning to occur in infected landscapes. The disease takes longer to develop in true oaks such as coast live oak (*Quercus agrifolia*), Shreve's oak (*Quercus parvula* var. *shrevii*) and black oak (*Quercus kellogii*), so mortality rates for these species are expected to rise in the near future.

Sudden oak death has a 'patchy' distribution over the landscape (Kelly and Meentemeyer, 2002), so in areas with a comparatively long disease history like Marin and Santa Cruz Counties in California, we can expect the disease to begin to kill trees that escaped previous disease cycles. In areas where *P. ramorum* has been more recently introduced, such as Mendocino and Humboldt counties, the disease is likely to expand into previously uninfected areas (Meentemeyer *et al*, 2004, Valachovic, 2006). In California, these are the areas where most of the effort is being made to stop the disease from spreading, as they are currently at the greatest risk (Meentemeyer *et al,* 2004).

Risk maps and disease progress can be monitored by going to the maps section of the sudden oak death website: www.suddenoakdeath.org . When viewing these maps, it is important to remember that the new infection sites are typically found where people are looking for *P. ramorum*. Thus, while disease levels may be quite high where sudden oak death has been established for years, few new infection sites will be recorded because disease presence in these areas is well known. In areas where *P. ramorum* has only been recently introduced and disease levels are still quite low, monitoring efforts are comparatively intense, and new disease locations are likely to be discovered.

Geography appears to play another, more unusual, role in the disease cycle of *P. ramorum*, in that the disease cycle appears to depend to some degree on geographic location. For instance, in Oregon, the spread of *P. ramorum* is far less dependent on exclusively foliar hosts like bay laurel and instead appears to be driven by transmission from tanoak to tanoak, or perhaps *Rhododendron* to tanoak (Goheen *et al*, 2002a, Huberli *et al*, 2002). As always, oak-to-oak transmission is not known to occur.

Until recently, sudden oak death has previously described as a canker disease (e.g., Rizzo *et al*, 2002), but recent research indicates that *P. ramorum* frequently colonizes the xylem tissue of oaks and tanoaks, causing a wilt disease (Parke *et al*, 2006). As arborists, we are familiar with vascular wilts. Vascular wilts are diseases where the pathogen invades the xylem tissue, and the host plant typically responds by plugging the xylem vessels with tissue (tyloses) from adjoining parenchymal cells (Agrios, 1997). The large rays that characterize oak wood and make it so beautiful are composed mostly of parenchymal cells (Harris, 1992), which may help explain why oaks are particularly good at this form of self-defense. The ability of oaks to form tyloses is one reason that oak wood is favored for making wine barrels: it's water tight. None of the vessels in the wood conduct water once the tree is infected or cut. Unfortunately, when *P. ramorum* invades our native tanoak and oak species, the trees appear to be too slow at recognizing the threat. They fail to form tyloses in time to contain the infection and keep it from spreading.

One of the best-known wilt diseases is Dutch elm disease, caused by the fungus *Ophiostoma ulmi*. It's important not to confuse sudden oak death with oak wilt. Oak wilt, caused by the fungus *Ceratocystis fagacearum*, is a serious, beetle- and root-transmitted disease of oaks in the mid-west, and is not known to occur in California (see Appel, 1994). Nonetheless, the fact that *P. ramorum* can act as a wilt explains why a canker disease, which typically grows slowly and only kills trees after causing structural failures after many years, can kill oaks so quickly. Thus, *P. ramorum* acts as both a canker-causing and wilt-inducing pathogen.

While different treatments have been evaluated for efficacy against *P. ramorum*, only one - phosphonate, has proven realistically effective in scientific trials (Schmidt *et al*, 2006). Phosphonate was developed in Australia for use against *Phytophthora cinnamomi* (Barrett *et al*, 2003), which has killed large numbers of Jarrah (*Eucalyptus marginata)* trees (Davison, 1988). Phosphorous acid has both a direct and an indirect effect on *Phytophthora* species. In its direct mode of action, it interferes with proper metabolism of the pathogen (Grant, *et al*, 1990). In addition, evidence suggests that phosphorous acid

has an indirect effect by stimulating the plant's natural defense response against pathogen attack (Smillie *et al*, 1989). Because phosphonate is systemic, and is stable in plants (Smillie *et al*, 1989), it should not be applied frequently. Current recommendations are to re-apply no more frequently than every 18 months to two years (Garbelotto, pers. comm.).

Phosphonates are most effective if applied well before the tree is attacked by *P. ramorum* (Schmidt *et al*, 2006). Some trees, notably tanoaks, may not show symptoms of infection until after the disease is fairly well developed, so, if a tanoak is showing symptoms of infection, it is almost certainly too late for treatment. The infection process is longer in true oaks, and symptom development often seems to occur earlier in the process, so if a true oak appears to have the earliest stages of infection it might not be too late for treatment. As with any inoculation, disease prevention is never 100 percent under the best of conditions, so not all treated oaks will necessarily respond to inoculation even under ideal circumstances. Thus, the success rate of treating true oaks that are showing symptoms isn't really known – it depends upon many factors including how naturally resistant the oak already is, how far along in the infection process the disease is and how well the oak responds to the treatment.

Elimination of bay laurel from around specimen oaks may keep oaks from being exposed in the first place. Spores of *P. ramorum* rarely travel more than 100 feet from infected bay laurels (Davidson *et al*, 2005) and most infections of oaks occur at distances much closer to bay laurels (Swiecki and Bernhardt, 2007). While these findings don't suggest that all bay laurels should be removed, they do suggest that the removal of infected bay laurel trees in the immediate vicinity of specimen oaks may provide an additional level of protection, especially if the bay foliage is within about 10 feet of the oak trunk (Swiecki and Bernhardt, 2007). On the other hand, removal of bays is probably unwarranted where oaks are already infected, or where the relative landscape value of the bays exceeds that of the oaks.

Because sudden oak death is a new disease, it's reasonable to expect that recommendations will continue to change as new research findings are made. To stay up to date on the most recent findings, log on to www.suddenoakdeath.org.

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