

Introduction and Background

First Reports

An unusual die-off of tanoaks (*Lithocarpus densiflorus*) in the Mill Valley/Mount Tamalpais area of Marin County, California was first documented in April 1995 (Svihra 1999a, 1999c, 2001), when homeowners reported scattered patches of dying tanoaks with entire crowns dead and with bleeding basal cankers to Pavel Svihra, University of California Cooperative Extension (Figure 1). The symptoms were also being reported by arborists and others on nearby tanoaks, including those at Muir Woods National Monument, and further south in coastal Santa Cruz County and around Big Sur (Monterey County). Heavy ambrosia beetle attacks and black fruiting bodies of a *Hypoxyton* fungus, later identified as *Hypoxyton thouarsianum* (Swiecki 2001), were associated with the dying trees.



Figure 1. Dead tanoaks in Marin County. Image: Keith Parker, Marin County Fire Department.

Undetermined Cause

Attempts by arborists, pathologists and horticulturists to determine the cause of the die-off yielded only several saprophytic organisms – including *Armillaria* sp., *Hypoxyton* sp., *Pseudomonas tolaensis* and *Diplodia quercina* (Svihra 1999b) – and identification of two ambrosia beetles (*Monarthrum scutellare* and *M. dentiger*) and one bark beetle (the western oak bark beetle, *Pseudopityophthorus pubipennis*) from affected tanoaks. The micro-organisms identified and the beetles are considered secondary or opportunistic, attacking only stressed trees. It was postulated by some that several years of drought from 1990 to 1992 followed by excessively wet years (“El Niño”) in 1993 and 1994 resulted in tree stress, allowing the secondary fungi and beetles to attack the weakened trees, with bark beetle and ambrosia beetle populations subsequently building up to unprecedented levels (Hagen 1999, Svihra 1999b, Svihra 2001). Other stress factors – including overstocking, fire exclusion, construction, incompatible landscaping, and others – were also suggested as contributing to the die-off (Hagen 1999).

Epidemic Spread

By May 1997, not only tanoaks, but also coast live oaks (*Quercus agrifolia*) were dying in the Marin Municipal Water District lands and in China Camp State Park on San Francisco Bay (Svihra 2001). The symptoms on coast live oaks (seeping or bleeding areas on the trunk followed by fading of the foliage, beetle attack and appearance of *Hypoxylon* fruiting bodies) were similar in appearance to those of dying tanoaks.

By the spring of 1998, the beetle attacks on coast live oaks were resulting in a rapid increase of dying coast live oaks (Svihra 1999a, 1999c) and several California black oak (*Q. kelloggii*) in the Novato area (Marin County) were reported with similar symptoms. Although the dominant thinking was that the primary cause of the widening mortality was bark and ambrosia beetles attacking stressed trees, some scientists noted that young tanoaks without oozing trunk cankers or beetle attacks were also dying, suggesting that an unknown pathogen was involved as the primary stress agent rather than drought followed by high rainfall.

Public Concern

The rapid spread of the disease in 1998, in an urban-wildland interface area inhabited by millions of people, heightened public concern (Figure 2). The highly visible disease was killing trees on public and private lands, threatening residential and forest landowners, parks, industries, water



supply, soil retention, and wildlife. Weakened and dead trees were a hazard because they could fall, destroying life and property. They also posed a severe fire hazard. The unabated mortality, now being described as epidemic, increased through 1999 (McPherson and others 2000). Concerned homeowners pressed county supervisors, administrators and politicians for investigation and treatment.

Because the causal agent was unknown at the time, the syndrome was commonly referred to after the most obvious symptoms—the trees dying in a somewhat sudden fashion. A

University of California Cooperative Extension publication (Svihra 1999a) described the crown symptoms as appearing rather suddenly, resulting in rapid death. The mortality was referred to as

“sudden oak death” in a second Cooperative Extension publication (Svihra 1999b) and the term was picked up by others, notably the press, to describe the phenomenon.

In 1999 a multidisciplinary research team of University of California scientists was formed to address the cause and management options for control of sudden oak death. A 23-person team was formed, including forest pathologists, forest entomologists, ecologists, silviculturists, wildlife biologists, remote sensing/GIS experts, and urban forestry/arbiculture experts from UC Davis and UC Berkeley, and advisors from Cooperative Extension county offices. Funds were provided by both UC and the USDA Forest Service. The University of California system provided \$70,000 in emergency funding, and the USDA Forest Service, Forest Health Protection provided \$85,000 for research and monitoring. Susan Frankel, then with the Pacific Southwest Region of the USDA Forest Service, was instrumental in focusing and coordinating early efforts.

Causal Agent Found

The availability of funding and similarity of the bleeding canker symptoms to those caused by species of *Phytophthora* elsewhere (Erwin and Ribeiro 1996) renewed efforts to find a causal agent (Garbelotto and Rizzo 2005). In June 2000, David Rizzo, U.C. Davis, consistently isolated a *Phytophthora* species from bleeding cankers on oaks and tanoaks at different locations. By early fall, Koch’s postulates had been completed on seedlings through artificial inoculations, and by the late fall of 2000, inoculation experiments on adult oaks and tanoaks were successfully completed (Garbelotto and others 2001, Rizzo and others 2002). Inoculated trees developed large girdling stem cankers that started bleeding approximately a month after inoculation. *Hypoxylon* and beetles became visible on the trees only after the *Phytophthora*-caused lesions developed in the bark. Now that a cause was known, forces were focused on obtaining information on the causal organism.

In June 2000, the Marin County Board of Supervisors passed a resolution declaring that sudden oak death had created a state of emergency. “We are in the midst of a catastrophe,” Supervisor Cynthia Murray stated at the time (San Francisco Chronicle Nov. 5 2000). In August 2000, under the leadership of the California Forest Pest Council and the California Department of Forestry and Fire Protection, local, state, and federal agencies joined to form what would become the statewide California Oak Mortality Task Force (COMTF). Involved early on were the California Department of Forestry and Fire Protection, the California Forest Pest Council, the University of California and the USDA Forest Service. The advisory group, chaired by Susan Frankel, undertook the challenge of establishing a cooperative, unified approach and offering guidance to funders, lawmakers, management agencies, research institutions, and others concerned with the disease. In November of that year, the state of California appropriated \$100,000 to address

sudden oak death and federal officials announced that an additional \$1 million would become available for research, monitoring and outreach (San Francisco Chronicle Nov. 2, 2000).

In Oregon, pathologists from the Oregon Departments of Agriculture (ODA) and Forestry (ODF), Oregon State University, and the USDA Forest Service formed a less formal coordinating group, and initiated SOD detection surveys in the southern part of the state.

In October 2000, British plant pathologist Clive Brasier, UK Forestry Commission, was visiting with Dr. Everett Hansen at Oregon State University. At the conclusion of that visit Dr. Brasier traveled to the San Francisco area to observe the impacted forests and the newly isolated *Phytophthora*. While there, he was shown laboratory cultures of the unidentified *Phytophthora* isolated from cankers. Several weeks after his return to the UK, Dr. Brasier heard a description of a new and unnamed *Phytophthora* species from rhododendron in Germany and the Netherlands, isolated in 1993 (Werres and Marwitz 1997), and noted a striking similarity with the cultures he had been shown in California.

In Europe the organism was at that time known only as a pathogen causing leaf blight and branch dieback of *Rhododendron* spp. and root collar cankers on *Viburnum* spp. This knowledge led California researchers to isolate a *Phytophthora* from rhododendron with leaf symptoms in a Santa Cruz container nursery surrounded by dying oak woodlands, in December 2000. The cultural morphology was identical to that of the sudden oak death *Phytophthora*, and by January 2001 Matteo Garbelotto, U.C. Berkeley, confirmed that the ITS (internal transcribed spacer) DNA sequence of isolates obtained from rhododendrons was identical to that of isolates obtained from oaks.

When researchers from California and Europe exchanged pathogen ITS sequences, a perfect match of the sequences from isolates collected in Germany and the Netherlands and from isolates collected in California was found (Garbelotto and Rizzo 2005). In April 2001, the pathogen was formally named *Phytophthora ramorum* Werres, de Cock, & In't Veld sp. nov. (Werres and others 2001).

The discovery and naming of the pathogen was an essential step, but the problem continued to increase in intensity and concern. In early 2001 the State of Oregon issued an emergency rule banning host plants and other plant products coming from California unless they had been treated. A few months later the Oregon quarantine became permanent (March 27, 2001) and Canada issued a similar quarantine (March 9 2001), prohibiting the import of nursery stock and unmanufactured non-propagative material (such as logs and mulch) of all oaks (*Quercus*), tanoak (*Lithocarpus*), *Rhododendron* spp. and evergreen huckleberry (*Vaccinium ovatum*) and of soil from areas where sudden oak death occurs. At about the same time, the California Board of Forestry declared an

official Zone of Infestation for the seven counties (Santa Clara, Marin, Sonoma, Napa, Santa Cruz, San Mateo, Monterey) known to be infested with *P. ramorum*. The creation of the Zone allowed for use of California Department of Forestry and Fire Protection resources to combat the problem. On May 17, 2001 the California Department of Food and Agriculture issued emergency regulations that restricted the export of diseased oak products and rhododendrons from the seven infested counties.

New hosts for *P. ramorum* – including California bay laurel and coastal redwood – were being reported with increasing frequency, and the pathogen was discovered in July of 2001 in a forest location in Curry County, Oregon. The hosts and the spread of the pathogen will be covered in later chapters. A complete chronology of important events in the *P. ramorum* story can be found on the California Oak Mortality Task Force website at <http://nature.berkeley.edu/comtf/html/chronology.html>.

From the beginning, political leaders and their constituents in the densely populated Bay Area were involved in the process. Efforts to obtain funding were fruitful largely because of the public concern and media coverage that the new problem received and efforts of the COMTF (Figure 3). California's 2001- 2002 state budget contained \$3.6 million to address sudden oak death in the state, with the funds to be administered through the California Department of Forestry and Fire Protection, and the COMTF serving in an advisory capacity. At the federal level, \$2.4 million (\$1.4 million from a Federal Emergency Supplemental Appropriation, and \$1 million from the USDA Forest Service) was made available in 2002 for research, monitoring and management projects.

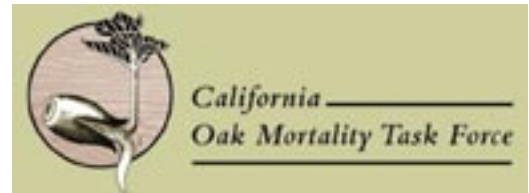


Figure 3. The California Oak Mortality Task Force logo. Image: COMTF.

***Phytophthora* as a Forest Pathogen**

Phytophthora ramorum was not the first member of this genus to be a concern in forest ecosystems. The closest related species genetically, the apparently introduced pathogen *P. lateralis*, has resulted in significant damage to native forest ecosystems in California and Oregon containing Port-Orford-cedar (Zobel and others 1985). The pathogen is significant because infection results in mortality of a host with a relatively limited distribution. The introduction of *Phytophthora cinnamomi* to the southeastern United States in the early 1800s resulted in mortality of the American chestnut and related *Castanea* species (Crandall and others 1945, Mistretta 1984) as well as being associated with littleleaf disease of shortleaf pine (*Pinus echinata*). The introduction of *P. cinnamomi* to Australia (Shearer and Smith 2000) and to oak forests of several Mexican states (Tainter and others 2000) has resulted in similar drastic changes to forest ecosystems.

Brasier (2003) noted that pathogens in the genus *Phytophthora* became a major threat to European forests during the 1990s. During the 1990s, twelve previously unknown *Phytophthora* species were discovered in European forests or nurseries (Brasier 2003). The ecological roles or places of origin of the species were unknown (Brasier 2003). One species, *P. alni*, has been demonstrated to be a hybrid (Brazier 1999, Brasier and others 2004) and is of significant concern in Europe as the cause of alder dieback (Gibbs and others 2003).

In addition to the above examples of the genus *Phytophthora* as an introduced pathogen or as a hybrid, recent investigations have found numerous indigenous species of the genus in forests of California and Oregon (Hansen 2003, Hansen and others 2005), and in Europe (Brasier 1999, Brasier and others 1999). Hansen and others (2005) discuss 15 distinct *Phytophthora* species that have been at least partially characterized from trees, soil or streams in oak forests in Oregon and California. Eleven are named species; four of these were described within the last five years; and four taxa are not formally described. The ecological role and significance of these newly discovered species remains largely unknown.

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