

Diagnosing Sudden Death of Oaks in California: A Historical Overview

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"The death of a small twig may be an insignificant event, or it may be the first sign of a fatal disease" (Strouts and Winter, 1994).

Isolation and identification of an unknown pathogen in urban forest trees is not only a professional, but also an intellectual, challenge. Whether planted or preserved from the preexisting flora, urban trees commonly undergo physical, chemical, biological, and environmental stresses that produce intense strain and may confound early identification of underlying pathogens when a disease becomes evident. This article describes the effort to diagnose a new oak disease that was first noticed on a few trees in Mill Valley, Marin County, California, but has now become a widespread epidemic.

Progression of the Disease

In April 1995, an unusual dieback of tanbark oak (also known as tanoak), *Lithocarpus densiflorus*, was first noticed by homeowners in Mill Valley. The University of California Cooperative Extension office in Marin County received a call to investigate more than a dozen dead trees along a creek near residences adjacent to the urban-forest interface (the area where wild lands and urban areas meet). Along the valley beneath a canopy of large redwoods, tanbark oaks of various sizes displayed brown foliage. Beneath the bark in the root crown area of the largest tanbark oak were mats and strands of "shoe-strings" which are typical of the oak root fungus, *Armillaria mellea*. Tanbark oaks are usually tolerant of this pathogen and can withstand some drought. However, prolonged drought from 1990 to 1992, followed by very wet years in 1993 to 1994, may have reduced the vigor of tanbark oaks to such an extent that *A. mellea* was able to infect and kill them.



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Figure 1. Only tanbark oaks show wilting/drooping of the new shoots.

In June 1996 the Mill Valley Superintendent of Parks requested reexamination of the dying tanbark oaks. It was shocking to see the extent of dying and dead trees, not only along the creek, but also on the slope and along the crest of a hill. Disease symptoms could no longer be attributed to *A. mellea*. Some trees had wilted new shoots (Fig. 1), suggesting occlusion of the vascular system. The current-year foliage of other trees had turned yellow-green and new shoots drooped, which is typical of trees with root disease. Trees 12 to 20 inches in diameter were dead and had bleeding cankers and heavy infestations of ambrosia beetles, indicated by piles of fine white-to-tan sawdust on the lower trunks (Fig. 2a & b). Dead trees also had charcoal-black spherical fungal fruiting bodies on bark surfaces. With the cooperation of a num-

ber of arborists, horticulturists, and pathologists, intensive sampling of affected trees was conducted in June, August, and October 1996. Samples of discolored tissues and wilted twigs were sent to University of California and State laboratories. Most reports failed to identify a probable cause of the problem.

Suited to a wide variety of soils, healthy tanbark oaks grow to great size in coastal regions from southern Oregon to Santa Barbara, California. They reproduce naturally, but few attain a large size in commercial forests, where tanbark oaks are considered weed species and are often killed with herbicides. The leaves of tanbark oaks are similar to those of the chestnut, *Castanea dentata*. Flowers appear in May-June, producing a spectacular display of creamy-yellow blossoms. The fruit takes the form of an acorn similar to that of true oaks (*Quercus* spp.). The question arose "Could the mysterious dieback and death be the dreadful chestnut blight

caused by *Cryphonectria parasitica*, or perhaps oak wilt caused by *Ceratocystis fagacearum*?" However, neither fungus was detected in laboratory tests.

In May 1997, the first coast live oaks, *Quercus agrifolia*, began to die in Marin County gardens. The timing and geographical location of this dieback raised the possibility of a causal link to the tanbark oak malady. The most noticeable symptom of the live oak disease was heavy attack by ambrosia beetles near the tree base. It also was very clear that each tree that had died was under stressful environmental conditions, such as improper irrigation, landscape changes, and soil compaction. The more coast

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live oaks that were examined, the more striking was the similarity of their symptoms to those of dying tanbark oaks, especially the presence of heavy infestations by oak bark beetles and ambrosia beetles and growth of a charcoal-colored hemispheric fungus on the bark. There were also, however, differences in symptoms. Some young tanbark oaks died in the vicinity of mature, dead tanbark oaks without noticeable bleeding or beetle attacks, but young coast live oaks were not being killed. A viscous ooze near the base of the tanbark oak trees appeared as large patches whereas only scattered droplets of tarry ooze were found on coast live oak trees (Fig. 3 a & b).

In June 1997 two adjacent tanbark oaks that were 6 inches in diameter and showed initial

ambrosia beetle attacks in Mill Valley were examined. The first tree had died during the previous winter; the second had yellow foliage with typical wilted shoots. Both trees were uprooted with a backhoe. Samples of necrotic or discolored roots, inner bark, and cambium tissues under the patches of ooze, and branches and shoots that showed discoloration, streaks, or staining were collected. Eight tissue samples of different symptomatic areas from each tree were sent to University of California and State laboratories. Three fungi, *Daldinia concentrica*, *Hypoxyylon thouarsianum*, and *Diplodia quercina*, and one bacterium, *Pseudomonas tolaensis*, were identified. None of these organisms was considered the primary agent of the tree mortality.



Figure 2a. The white-to-tan sawdust produced by ambrosia beetles is visible on the lower stem for several weeks or months while reddish paprika-like sawdust produced by oak bark beetles lasts only a few days.

Figure 2b (inset). The oak ambrosia beetles' egg galleries penetrate deep into the sapwood.

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Figure 3a (right) Sticky liquid has seeped out and dried and small, scattered tarry globules appear on the bark of the lower stem of coast live oak.



Figure 3b (above) On tanbark oaks the ooze is patchy.

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Also in June 1997, a large dying live oak that was heavily infested by ambrosia beetles and showed extensive bleeding (scattered globules) near the base of the stem was examined. Its roots, beginning at the root crown, were traced out to over three feet. They were yellow and from them emanated a pungent vinegary-to-alcoholic odor. Root samples for isolations, delivered to the plant pathology laboratories at UC Berkeley and Davis revealed no plant pathogens.

In June 1997, beetle-infested trunk sections were removed from both tanbark oaks and coast live oaks and placed in separate rearing chambers to collect emerging beetles. In September, samples were collected from both oak species. The California Department of Food and Agriculture laboratory identified three species of beetles, *Monarthrum scutellare*, *M. dentiger*, and *Pseudopityophthorus pubipennis*.

By the fall and winter of 1997 many trees

had died, and the Marin County UC Cooperative Extension office was receiving numerous telephone calls from concerned homeowners and professionals.

Disease Characterization and Control

Because no causal pathogen could be implicated in the dieback, which continued to expand, a control strategy was formulated in the spring of 1998. It seemed probable that drought followed by excessive rains had caused stress in the affected trees, resulting in the buildup of bark beetle and ambrosia beetle populations to an epidemic stage in dying tanbark oaks. It was thought that the new beetle generations not only attacked tanbark oaks, but also shifted their attacks to coast live oaks. A program of preventive insecticidal sprays, combined with tree

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health care (irrigation, cleaning of root crown areas, pruning, fertilization), was initiated. This control strategy was communicated to arborists through regional and local meetings and workshops.

In June 1998, soon after the treatment program had begun, two major observations were made: (a) a substantial increase in dead coast live oaks from Mill Valley to Novato, and (b) death of two black oaks, *Quercus kelloggii*, in Novato. The symptoms on the black oaks were almost identical to those on dying tanbark oaks and coast live oaks.

Homeowners were delivering hundreds of symptomatic wood and branch samples to the UC Cooperative Extension office in Novato. The problem became more complex and had the potential to cause great economic and emotional concern.

The word "epidemic" began to be used widely and the media were informed about the problem. However, there was little agreement on the definition or diagnosis of the problem because new inconsistencies continued to emerge. For example, bark beetle and ambrosia beetle infestations were considered the primary cause of death of mature coast live oak and black oak, but only because these trees were predisposed by environmental stresses. However, the death of very young tanbark oaks that had no ooze on their trunks and were not attacked by beetles seemed to contradict the involvement of insects as the primary cause. It appeared that an unknown pathogen was traveling from mature dead trees to kill these young trees.

In the winter of 1999, El Niño's wicked sister La Niña was in retreat, but the number of dying trees continued to rise. A homeowner on

Mountain View Avenue in San Rafael counted 316 dead trees, and asked bluntly, "What are we going to do?" At the same time, an opinion circulated that the oak dieback was a temporary phenomenon (a "natural cycle") related to climatic extremes. In June 1999 the California Forest Pest Council met to assess the massive death of oaks. Cooperating plant pathologists uprooted a symptomatic tanbark oak tree and collected samples of twigs, bark and roots. No plant pathogens were recovered. Three "Pest Alerts" (Švihra, 1999, 1999a and 1999b) were printed. One was titled "Sudden Death of Tanoak," and the name to describe the syndrome became "sudden oak death." Plant pathologists protested such a terminological "inaccuracy", while the media embraced not only the descriptive name, but also its acronym, "SOD." The Marin County Cooperative Extension office continued to be flooded with samples and requests for "Pest Alerts" (15,000 were disseminated to professionals and homeowners).

In the meantime, ambrosia beetles expanded high-density attacks from tree bases to the middle and high portions of stems of dying coast live oaks. Because of the epidemic, we had to act. The most effective way to draw attention to this emergency was by encouraging concerned citizens to contact newspapers and county supervisors. When tanoaks and coast live oaks were dying as far south as Big Sur in Monterey County, it was clear that the problem was no longer "local."

Eventually Marin County Supervisors realized that the economic and ecological impacts of the dead trees, as well as the fire hazards associated with them, had been greatly underestimated. In January 2000 they appropriated \$5,000 to enable UC Cooperative Extension in



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Figure 4. Cutting under the black "tarry globules" of coast live oaks reveals patches of dead or dying inner bark. The new *Phytophthora* fungus was found in tissues showing margin lines.

Marin County to appoint a Task Force to address SOD in the county. Under the leadership of UC Cooperative Extension and the Marin County Supervisors, the Task Force was staffed with professionals as well as members of the public. The Marin Community Foundation allocated \$20,000 to support a Public Relations Coordinator to establish mechanisms for direct professional/public/media communication of consistent and credible messages. Sudden oak death was put on state and national maps, even though the cause was not known. The Task Force also emphasized the importance of research in providing information to mount a timely response to the emerging and still unexplained disease. Both the University of California and USDA Forest Service led an accelerated research program to guide sudden oak death

control efforts. Plant pathologists and entomologists suspected that a still unidentified pathogen was the cause of the problem. Despite the elusive nature of the problem and the method of spread from infected to healthy trees, professional clientele and the public needed guidance. Information offered focused on insect control and implementation of plant health care (Palkovsky and Švihra, 2000). Through the spring and summer of 2000 more than 30 local and regional workshops were conducted to help arborists, horticulturists, landscape contractors, and homeowners combat sudden oak death. The professional community and the public viewed a newly prepared "Homeowner's Guide" favorably, and 6,000 copies were distributed. The guide's impact on the media was enormous: Sudden oak death was featured on major national TV networks and on the front pages of the Marin Independent Journal, New York Times, Los Angeles Times, USA Today, Chicago Tribune, San Francisco Chronicle, and numerous regional and local newspapers throughout the state and nation. Finally a scientific breakthrough came. University of California Assistant Professor of Plant Pathology, David Rizzo, sampled infected coast live oaks in China Camp Park, San Rafael, and on the same day returned to his laboratory to make isolations from the tissue samples. As it turned out, this rapid processing of tissue samples was essential for recovery of a new and previously unnamed species of *Phytophthora*. Previous samples (with a zone of necrotic tissue and dark resinous lines) that had been mailed or even hand-delivered had not revealed the cause of the problem (Fig. 4). Once this new *Phytophthora* was isolated from sample tissue it became clear that this pathogen was an underlying cause of sudden oak death (McPherson et al., 2000). The species of *Phytophthora* is still unknown, but seems to be

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of *Phytophthora* is still unknown, but seems to be related to *Phytophthora lateralis*, the cause of a deadly root disease of Port-Orford cedar, *Chamaecyparis lawsoniana* (David M. Rizzo and Matteo Garbelotto, personal communications). Now we know that this new disease has a broad host range and seems to have an extremely adaptive biology. The oak infections probably occur from the spores that are copiously produced on the leaves of alternate hosts, such as California laurel, *Umbellularia californica*, and madrone, *Arbutus menziesii*.

Conclusions

At present no control method is available that will alter the underlying disease process for this new "SOD" *Phytophthora*. Expansion of research will be crucial for identifying the inciting species of *Phytophthora* and providing information needed to develop diagnostic (sampling) procedures and treatment. The potential for fungicides, other management practices, and wood disposal to mitigate the impact of the disease is currently under investigation. *In vitro* studies have demonstrated that some fungicides have potential for slowing down or preventing development of the underlying disease. This finding needs to be confirmed in the field. Compounding the Extension Advisor's plight is general disagreement among researchers as to how to treat the magnificent oaks infected with this new *Phytophthora* fungus, given that very few well designed studies have been completed. Is it not a familiar story, pitting researchers (who must favor long term studies) against Advisors (who provide timely practical advice with their reputations on the line),

impatient homeowners and politicians (who favor short-term solutions)?

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