# Control of branch, leaf, and twig dieback of oak

*John R. Parmeter*
*Eva I. Hecht-Poinar*

Oak dieback is common and sometimes severe in the coastal counties of California. Reports of dieback in the Central Valley have been less frequent, possibly because fewer oaks occur there, while varying amounts of the disorder have been observed in the Sierra foothills. The disease occurs in a variety of sites, topographies, and exposures. Individual trees also vary widely in susceptibility--unaffected trees can often be found next to trees with severe dieback. Observations suggest that repeated heavy infection may lead to the death of trees.

Our study was conducted to determine (1) the disease cycle of the fungi involved in oak dieback, (2) environmental factors, if any, that might be associated, (3) the possible association of the disease with insects, and (4) chemical control measures that might be feasible for use in California.

Three fungi have been implicated in the dieback of oaks in California. *Diplodia quercina*, associated mainly with the dieback of branches up to 4 inches in diameter, was prevalent in 1979-82, declined during the wetter winters but became more serious again in 1985, following a dry winter. Dieback of current year's twigs is caused by two anthracnose fungi, *Cryptocline cinerescens* and *Discula quercina*. All three fungi occur commonly on coast live oak (*Quercus agrifolia*) and valley oak (*Q. lobata*).

Field inoculations with *Diplodia* always resulted in infections, but repeated attempts to inoculate oaks with *C. cinerescens* or *D. quercina* in the field were unsuccessful, regardless of the season, making it necessary to undertake greenhouse studies. We obtained 250 potted one-year-old seedlings of coast live oak, removed twigs that appeared to be naturally infected, and held the plants in the greenhouse to force production of new, uninfected shoots. The plants were then

California oak trees are subject to periodic epidemics of dieback disease. The fungi causing the disease can be chemically controlled, as on this tree, of which only the left half was treated, but natural selection for tolerance may be the best answer. Studies have linked dieback to oak pit scale (above), which is common in California.

inoculated with atomized spore suspensions of anthracnose fungi. Usually, leaf spots appeared within a week after inoculation, and dieback of twigs was obvious by six weeks. Infection by *C. cinerescens* or *D. quercina* in nature occurs during the period when oaks are producing succulent new shoots in the early spring, depending upon the availability of inoculum. Symptoms of dieback, however, do not become visible until July or August when the trees have been stressed by high temperatures and lack of water.

On greenhouse seedlings, fruiting structures (acervuli) with mature spores were produced about two months following inoculation. In the field, mature fruiting structures apparently are not produced on current-year infections until late fall or winter, a lapse of eight months or more. On older dead twigs, mature acervuli were present throughout the year. When we wetted mature acervuli in our trials, openings enlarged and, within six hours, large numbers of viable spores were produced. It is therefore likely that dieback infection can occur on trees during any prolonged rainy period when new shoots are being produced (usually twice a year). Our observation of oaks showed that leaf and petiole symptoms first appeared in March, probably from infection during the rains of January and February. By July, dead brown leaves on dead twigs were obvious. Fruiting occurred on these newly infected twigs in late fall, thus completing the disease cycle.

### Bud and seed infestation

Isolations from surface-sterilized buds indicated that both anthracnose fungi and *D. quercina* commonly contaminated buds and could be found throughout the year. Bud infestation was highest in late spring, after winter sporulation, and lowest in the late fall. In our trials with acorns collected from healthy, moderately infected, or severely infected oaks, we germinated and grew seedlings but could not observe infection from seed nor isolate pathogenic fungi. Inoculation of seedlings likewise did not suggest that subsequent infection was related to the condition of the parent tree. Acorn production, however, has been shown to be severely reduced on heavily infected trees.

These limited studies of epidemiology provide general information on conditions for infection. They do not, however, explain the rather sudden and previously unrecorded appearance of dieback at elevated levels. Association of the *Diplodia* dieback with severe drought here and in Europe suggests strongly that the stress caused by drought may lead to increased activity by the fungus.

The current outbreak may also be related to oak pit scale (*Asterolecanium minus*) which is common in California. Studies have shown that the amount of twig dieback is correlated with the amount of pit scale.

We designed two experiments to investigate scale-disease interactions. In one, diseased branches with heavy scale infestation were placed near healthy oak seedlings so that any spread by the scales could be detected. In the other, scale insects and young crawlers from diseased twigs were placed on healthy seedlings. We were unable to show that the diseases could be spread in this way.

### Chemical control

We evaluated a number of fungicides in laboratory, greenhouse, and field tests for activity against the dieback fungi. Benlate (benomyl), Banner (ectaconazole), and Rubigan (fenarimol) proved to be most promising.

An experiment in 1985-86 at the Stanford University Arboretum combined careful pruning of infected twigs in the fall, followed by a fall application of Benlate, a second application of Benlate at budbreak, and a late spring application of oil and Sevin (carbaryl) for scale control. The experiment showed very good control of dieback and will be repeated in the future.

### Resistance

We tested 143 oak seedlings in the greenhouse for resistance to dieback organisms. While 16 died, 19 were selected

as highly resistant; upon repeated inoculations, no infection took place. If we could test large numbers of seedlings, we might be able to acquire a stock of live oaks or valley oaks that are resistant to dieback, from which we could propagate in the future.

However, oaks have survived successfully for millennia in California. It is likely that the present epidemic will subside and that natural selection for tolerance will ensure the continued success of oak species.

*John R. Parmeter is Professor and Plant Pathologist, and Eva I. Hecht-Poinar is Associate Specialist, Department of Plant Pathology, University of California, Berkeley.*