Dieback of Oaks in California

EVA I. HECHT-POINAR, Assistant Specialist III, Department of Plant Pathology, University of California, Berkeley 94720; J. C. BRITTON, J. Britton Tree Service, St. Helena, CA 94574; and J. R. PARMETER, J.R., Professor, Department of Plant Pathology, University of California, Berkeley 94720

ABSTRACT

Extensive dieback, and in some cases death, of live oaks (Quercus agrifolia Née) in the Napa Valley, California, was caused by a fungus tentatively assigned to Diplodia quercina West.

In 1978, dieback of live oak (Quercus agrifolia Née) was widespread in the Napa Valley of California. The disease has increased through 1980. Diseased oaks have been found on hillsides and on the valley floor, in native stands and in yards and parks. Severity has varied from scattered twig dieback to death of entire trees. Because oaks are important and valued in the Napa Valley and other areas of California and because such widespread damage to oaks has not been reported previously in the state, investigation of the disease was undertaken.

Initial symptoms were wilting and browning leaves, usually distal to branch girdling characterized by dead bark and cambium, and sapwood in which the wood was stained brown and the bark was usually roughened by the emergence of black pycnidial stromata. Sometimes single leaves died without twig dieback or girdling. In such cases, the base of the petiole was black.

Isolations from branches and petioles or from single pycnidiospores regularly yielded grey-black colonies on potato-dextrose agar (PDA). Five isolates (four tissue and one single spore) were used to test pathogenicity. Inoculations were made by placing small blocks (2–3 mm³) of PDA mycelial cultures at the bases of 10 petioles on a branch and enclosing the branch in a polyethylene bag for 24–48 hr. Branches 1–2 cm in diameter were inoculated by placing similar inoculum blocks in notches approximately 5 mm long and cut transversely through the bark and into the sapwood. Inoculated notches were wrapped with masking tape. For controls, petioles or branches were treated with sterile agar blocks. All inoculated oaks were mature, open grown trees with diameter at breast height of 40 cm or more.

Of 400 petioles inoculated (five isolates, 10 petioles per isolate on each of four live oak trees inoculated on 8 April 1980 and repeated on 29 April 1980), 21% developed black bases and the leaves died within 2 wk. Differences among isolates, trees, or dates were negligible. The fungus was reisolated from all eight inoculated petioles sampled. Three of 40 control petioles (7.5%) also died and yielded the fungus.

Of 40 branches (five isolates, two branches per isolate on each of four live oak trees) inoculated on 8 April 1980, 93% were killed within 2 wk. Symptoms were identical to those found on naturally infected branches. Pycnidia were found at most sites of inoculation within a month. The pathogen was reisolated from all six inoculated branches sampled. One of eight (12.5%) control branches also died and yielded the fungus.

On 29 April 1980, a single isolate was used to inoculate five branches on each of two trees of Q. kelloggii Newb., Q. douglasii H. and A., Q. agrifolia Née, Q. lobata Née, and one tree of Q. robur L. Of these, 96% (all but two branches on one Q. kelloggii tree) became infected and died. The pathogen was reisolated from all 13 branches sampled. None of the control branches (two per tree) became infected.

The fungus, tentatively assigned to Diplodia quercina West., regularly produces pycnidia clustered in erumpent stromata on branches with bark or singly and superficially on decorticated branchwood. Pycnidiospores are uniformly large (generally longer than 30 μm and about half as wide), beginning as hyaline, single-celled spores and, if incubated for sufficient time, ending as dark, two or more celled spores.

The regularity with which oak diebacks and declines are reported in the United States (1–5, 7–11) suggests that the total impact on forest and landscape resources and the investments of research time have been considerable. Roland (6) found that this type of damage following drought led to devastating losses over a period of years in European oak forests. Since this current outbreak followed a severe drought and appears to be increasing, it is very possible that extensive losses will also occur in California.

ACKNOWLEDGMENTS
We thank W. Otkowski and H. Otkowski of the John Muir Institute, Berkeley, CA, and R. Raabe and J. Hurlimann for encouragement in the pursuit of this project.

LITERATURE CITED