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Susceptibility of western conifers to laminated root rot

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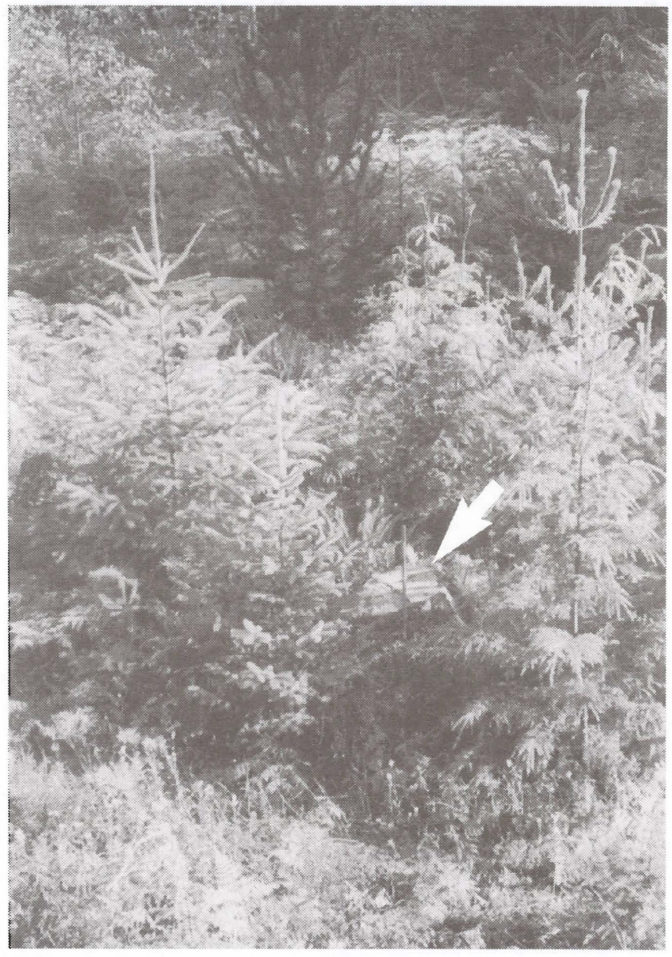
Laminated root rot caused by the fungus *Phellinus weirii* is one of the most important root diseases of conifers in western North America. Wood volume losses begin with reduced growth rates of attacked trees and increase as infected trees are killed by the fungus or windthrown because their structural roots are weakened by decay. Much of this loss is not economically salvageable because the disease occurs in small, scattered pockets in which mortality is sporadic. On some sites, disease pockets remain unproductive for the duration of the established stand. On others, fiber production from ingrowth of shade-tolerant (but often less valuable) trees may temper volume losses by the time the stand is harvested. Reduced site productivity persists long after harvest because the pathogen may live saprophytically in stump roots for as long as 50 years (Hansen 1979), and it often infects the roots of conifers in the succeeding stand.

Management of laminated root rot has focused on reducing inoculum by removing stumps and roots, or planting commercially desirable immune or resistant species in areas where infected stumps remain. Resistant conifers have been identified through years of observation, comparisons made from root excavation studies (Wallis and Reynolds 1965, Wallis 1976), and surveys of mortality in infected, mixed conifer stands (Filip and Schmitt 1979).

This study presents mortality data for several species of western conifers as a measure of susceptibility to laminated root rot, which will aid foresters in managing *P. weirii*-infested sites. It was carried out on five sites, four in Oregon (Coast Range) and one in B.C. (Cowichan Valley) over a period of 17-20 years. A standard method was used to set up the sites, with test species being planted on a fixed radius around infected stumps.

Results

Phellinus weirii was the most common pathogen on the five sites combined; in addition to recorded mortality, infection of living trees continues. Grand fir (*Abies grandis*), followed by Douglas-fir (*Pseudotsuga menziesii*), appeared most susceptible to *P. weirii* on sites in both Oregon and British Columbia. Sitka spruce (*Picea sitchensis*) and western hemlock (*Tsuga heterophylla*) appeared moderately susceptible to *P. weirii*, as did noble fir (*Abies procera*), giant sequoia (*Sequoiadendron giganteum*), and



Infected stump (arrow) surrounded by test species

ponderosa pine (*Pinus ponderosa* var. *ponderosa*), planted only on the Oregon sites. *Phellinus weirii* killed one or none of the remaining four test species: western redcedar (*Thuja plicata*), western white pine (*Pinus monticola*), redwood (*Sequoia sempervirens*), and lodgepole pine (*Pinus contorta* var. *latifolia*).

In British Columbia (and to some extent in Oregon), some trees, mostly Douglas-firs, appeared to have recovered from the disease. These trees, which had returned to normal color and rate of growth, had been previously noted as having crown symptoms and ectotrophic mycelium in past, nondisruptive examinations of root crowns.

Discussion

Orders of susceptibility reported here are similar to those reported by Hadfield (1985), who lists grand fir and Douglas-fir among the most susceptible of Pacific Northwest conifers. He lists ponderosa pine on Oregon sites as resistant, but we found it to be of intermediate susceptibility on Oregon sites, even though it grew well under Coast Range conditions. Some other species did not grow well, as evidenced by the considerable differences in their average diameters. In choosing species to reforest sites where *P. weirii* inoculum remains, forest managers must consider both their susceptibility to the disease and their suitability to the site.

Redwood and sequoia are not normally considered to fall within the geographic range of *P. weirii* and, except for the redwood in extreme southwestern Oregon, would not be considered indigenous to the Pacific Northwest; however, both are planted there as ornamentals and, to some extent, as forest trees. At least one of the two, giant sequoia, may not be a good risk on sites where *P. weirii* is present.

Mortality percentage figures given here are conservative because they are based on established seedlings; they do not take into account those trees missing or killed from other causes and therefore no longer candidates for infection by *P. weirii*. The design of this study should have otherwise allowed equal opportunity for infection from all species on a site because seedlings were planted equidistant from infected stumps. Opportunity for infection is, however, influenced by initial planting location (randomly selected for each seedling on our sites) and by rate of root growth. Roots of faster growing individuals might be expected to contact inoculum earlier and more frequently than roots of slower growing trees. These faster growing individuals can perhaps compensate for losses suffered through infection by more rapidly increasing their root systems, so that symptoms of infection would be slow to develop. Neither of these factors are accounted for in this study.

Although recovery of several symptomatic trees in this study may be only temporary, we will continue to monitor these trees and, where possible, investigate factors associated with this phenomenon.

Our results, based on some 20 years of observations, are reasonably consistent and support general observations made over many years on the susceptibility of coniferous species to *P. weirii*. Although our trees are still relatively young, we do not expect that trends in susceptibility among species tested will change greatly before they mature.

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