Bark Beetle, Fungus, and Host Interactions Involved in the Death of Pines in California

Founders Award Address by David L. Wood

Sacramento, California, March 1993

Following a reconnaissance trip through California, Oregon. Washington and Idaho in the spring and early summer of 1899, A. D. Hopkins (1899) concluded the following: ". ..It is well known that forest trees weakened by disease contribute to the multiplication of their insect enemies. It is also known that insects will attack healthy trees, and that diseases of the bark and wood follow as a result of such injuries. Therefore, in the investigations of unhealthy conditions of forests it is often exceedingly difficult, without some previous knowledge of the habits of the diseases and insects found associated with them, to decide which is to blame for the primary injury. Our present knowledge of the subject, however, indicates that as a rule unhealthy forest trees, like unhealthy animals, present characteristic symptoms, which indicate quite clearly the primary cause of the trouble. The evidence I have been able to gather in the forests of the East and Northwest makes it plain to me that of the two causes, while many small trees are killed by root diseases. the unhealthy condition of the larger trees is more often due to primary attacks by insects." Hopkins' clearly stated entomological bias sets the stage for

my collaborative research with forest pathologists at the University of California. Berkeley.

In 1965, Professor Dick Parmeter, a forest pathologist, was describing his and Paul Miller's pioneering research on a new disease of ponderosa pine stands, variously called x-disease, chlorotic decline, and ozone needle mottle. This disease had been observed since the early 1950's in the San Bernardino Mountains of southern California. They established the relationship between needle symptoms and photochemical air pollutants, in particular, ozone (Parmeter et al., 1963). He and his colleagues hypothesized that insects may be associated with these declining pines. This motivated a major collaborative research effort by entomologists (Stark and Wood) and pathologists (Cobb, Miller, and Parmeter). Upon extensive investigation we found that, as the severity of oxidant injury increased, the incidence of bark beetle infestation by the western pine beetle, *Dendroctonus brevicomis*, and the mountain pine beetle, *D. ponderosae*, increased (Stark et al., 1968). We also found that oleoresin exudation pressure, quantity and rate of flow, and phloem and sapwood moisture content were reduced, while the propensity of oleoresin to crystallize was increased in trees exhibiting advanced symptoms of oxidant injury (Cobb et al., 1968a). We postulated that these conditions favored the successful establishment of these*Dendroctonus* spp. in ponderosa pines injured by photochemical atmospheric pollution (Cobb et al., 1968b).

While investigating the relationship between oleoresin exudation pressure and tree mortality caused by *D. brevicomis* and *D. ponderosae* in the central Sierra Nevada, Professor Fields Cobb and colleagues discovered a root pathogen (*Leptographium wageneri* var.*ponderosum* (Harrington, 1988) infecting ponderosa pines and they hypothesized that this pathogen may predispose ponderosa pine to infestation by bark beetles. In early studies of risk factors associated with the mortality of old-growth ponderosa and Jeffrey pine caused by *D. brevicomis* in Lassen Co., California, Wygant (1942), a USDA entomologist, had observed a black staining fungus (identified as *Leptographium lundbergii*) in the roots of some trees. Dr. W. W. Wagener, a USDA forest pathologist, examined these pines, but the importance of the fungus in weakening trees was not determined. Our later collaborative studies in second-growth stands showed a strong association between the occurrence of root pathogens (primarily *L. wageneri* and *Heterobasidion annosum*) and bark beetle infestation of both ponderosa pine (primarily by *D. brevicomis* and *D. ponderosae*) and white fir (by *Scolytus ventralis*), (Cobb et al., 1974). This association was strongest at low bark beetle population levels, i.e., 1-3 trees per mortality group. At higher population densities, symptomless trees are killed as a result of higher levels of bark beetle aggregation pheromones. Thus, where large groups of dead trees occur over a short period of time, the association with root diseases is not as strong.

The discovery of the association between root diseases and photochemical air pollution and elevated levels of tree mortality caused by bark beetles, led to investigations of the host selection behavior of *D. brevicomis*, *D. ponderosae* and the red turpentine beetle, *D. valens*(reviewed in Wood, 1982). Because disease severity could be

objectively classified by foliar symptoms (needles injured by oxidants are shortened, chlorotic mottled, and fewer in number) and sapwood blackstain caused by *L. wageneri*, differential landing rates of bark beetles could be recorded. Moeck et al. (1981) showed that the landing rates of *D. brevicomis* did not differ between severely diseased and symptomless trees. The key to this study was the prevention of bark beetle pheromone production by screening the bole into the live crown.

Doane et al. (1936) state that *D. valens* "...is not considered an aggressive pine killer, although it may do considerable damage to trees and weaken them so that they are more susceptible to other bark beetle attack. .." Salmon and Bongberg (1942), Keen (1952), and Smith (1971), considered the presence of pitch tubes caused by *D. valens* to be one of many indicators of increased risk of mortality of ponderosa pine and Jeffrey pine caused by*D. brevicomis* and *D. jeffreyi*, respectively. At an early conference on risk rating old-growth ponderosa pines (Whiteside, 1948), Bongberg stated "...that the work of the red turpentine beetles (*Dendroctonus valens*) should be given an important rating as indicating high risk trees and trees to be marked for cutting." These early observations of *D. valens* were generally made in old growth stands. However, Goheen and Cobb (1980), Moeck et al. (1981), and later Goheen et al. (1985) observed more attacks by *D. valens* on trees severely infected by *L. wageneri* than on symptomless trees. These trees were in second-growth stands, and were not screened to prevent *D. valens* attacks at the root collar.

To explore the interrelationships among *D. valens, D. brevicomis, D. ponderosae* and *L. wageneri,* Owen (1985) established research plots in the central Sierra Nevada. Between June 1981 and November 1983, a significantly higher proportion of ponderosa pines exhibiting *D. valens* pitch tubes were killed by *D. brevicomis* and *D. ponderosae* than trees without such pitch tubes. Also he found that living trees with *D. valens* pitch tubes were more likely to be infected with *L. wageneri* than trees without these pitch tubes. Because *D. valens* rarely kills trees, its unsuccessful attacks may be an early indication of stress caused by root pathogens, and perhaps mistletoe and other agents. Furthermore, *D. valens* and its associated microorganisms may play an important role in the succession of organisms that contribute to tree mortality. As discussed later, *D. valens* vectors *L. terebrantis* which was shown by Owen et al. (1987) to be the most pathogenic fungus carried by the three*Dendroctonus* spp. investigated.

Owen (1985) wounded trees in the same manner as Goheen and Cobb (1980), Goheen et al. (1985), and Moeck et al. (1981) (holes punched to the sapwood surface to assess the presence of blackstain), but excluded *D. valens*-associated attacks by screening the lower trunk and root collar. However, Owen (1985) found no difference in the distribution of trapped *D. valens* between diseased and symptomless trees. These results indicated that the preferences exhibited by *D. valens* for diseased trees likely occurs after landing on the tree. Differential attraction at close range, or differential feeding stimulation, or both behaviors may account for the observations of Goheen and Cobb (1980), Goheen et al. (1985), and Moeck et al. (1981).

At this time in our collaborative research, we turned our attention from agents that predispose or weaken trees, i.e., photochemical air pollutants, blackstain root disease, and*D. va/ens* tunneling activity, to the fungi vectored by tree-killing (*D. brevicomis* and *D. ponderosae*) and tree-debilitating (*D. valens*) bark beetles. Owen et al. (1987) wound-inoculated two-year-old ponderosa pine seedlings with fungi isolated from the above*Dendroctonus* species. *Leptographium terebrantis* from *D. valens, Ophiostoma (= Ceratocystis) minus* from *D. brevicomis*, and *O. clavigerum* from *D. ponderosae*, each killed a high proportion of the seedlings. Seedling mortallty caused by *L. terebrantis* was greater than that caused by the other two fungi. This was a surprising result in light of the well-known tree-klling habit of *D. brevlcomis* and *D. ponderosae* and the non-tree-killing behavior of *D. valens*. *Ophiostoma ips* from *D. valens* and *D. ponderosae* reduced seedling mortality caused by *L. terebrantis* and *O. clavigerum*, respectively. *Ophiostoma nigrocarpum* from *D. brevicomis* produced the same result when inoculated together with the pathogenic *O. minus*. Recently, Harrington (personal communication and see Harring!on, 1993, p. 43) has identified the mycangial hyphomycete (previously identified as *O. nigrocarpum*) from *D. brevicomis* as *Ceratocystiopsis ranaculosus*. Perhaps, Owen et al. (1987) have unknowingly shown that both *C. ranaculosus* and *O. nigrocarpum* inhibit the pathogenic activity of *O. minus*. However, further studies with known isolates of these fungi are required before such a conclusion can be made. Thus, each of these three *Dendroctonus* spp. that infest ponderosa pine carries one pathogenic fungus and one fungus that inhibits the effects of the pathogenic fungus in seedling inoculation studies.

The results obtained by Owen et al. (1987) with beetle-vectored fungi in ponderosa pine seedlings stimulated our evaluation of pathogenic effects of these fungi in small trees (diameter at breast height = 10-25 cm). Parmeter et al. (1989) inoculated four species of fungi vectored by *D. brevicomis* and *D. ponderosae* into *P. ponderosa*. Radial depth of sapwood occlusion at the inoculation site was assayed by standing stem sections in solutions of Fast Green dye. The depth of radial sapwood occlusion was greater for *L. terebrantis* from *D. valens* than for isolates of *O. ips* from *D. valens*, and *O. nigrocarpum*and *O. minus* from *D. brevicomis*. The average sapwood penetration was greater for *L. terebrantis* inoculated alone than when inoculated with *O. ips*. A similar trend (but not statistically significant) was observed when *O. minus* was inoculated with *O. nigrocarpum*from *D. brevicomis* and compared to *O. minus* inoculated alone.

Parmeter et al. (1992) proposed that "... the rate at which sapwood occlusion extends radially into the xylem should be adequate to account for lethal restriction of flow and for symptom development in trees under beetle attack." They showed that the length of sapwood lesions, depth of sapwood occlusion, and proportion of sapwood occluded (all determined by immersion of log sections in Fast Green dye) increased through the first 9 weeks following inoculation of small ponderosa pines with *O. minus* and *L. terebrantis*. They concluded that the rate of occlusion observed in these experiments was inadequate to account for the early (3-4 weeks in summer) development of crown symptoms in trees following mass attack by bark beetles that vector these fungi.

In another study utilizing the Fast Green dye assessment of sapwood occlusion, ponderosa pines attacked by *D. brevicomis* were periodically sampled over 7 weeks (Hobson et al., 1994). Neither *O. nigrocarpum* nor other bluestain fungi were commonly isolated at the interface of the occluded and functioning xylem. Bluestain fungi appeared to colonize already occluded xylem. In these studies, the second instar larvae of *D. brevicomis* had already turned out into the outer bark before any occlusion of the xylem was apparent.

These recent studies by Parmeter et al. (1992) and Hobson et al. (1994) indicate either that bluestain fungi are not causing sapwood occlusion or, if they are playing such a role, they may be exporting metabolites at some distance from where the fungi can be isolated. Furthermore, incubation of freshly cut disks eight weeks following inoculation showed almost no discoloration due to pigmented hyphae in occluded areas (Parmeter et al., 1989).

Bark beetles colonizing second-growth ponderosa pines generally occupy specific areas on the main stem in relation to one another, i.e., *I. latidens* and *I. paraconfusus* in the tops or upper crown, and larger branches; *D. valens* in the roots, root collar and lower 3m of the bole; and *D. brevicomis* and *D. ponderosae* in between these species in the upper and lower portion of the tree. Fox and colleagues (1993) reared the California 5-spined ips, *Ips paraconfusus*, from egg (after surface sterilization) to adult in the presence of its vectored fungi (*O. ips* and yeasts) and the pathogenic fungi carried by the above *Dendroctonus* spp. that colonize ponderosa pines. Survival in intact ponderosa pine phloem was reduced by all of the bluestaining fungi, i.e., *L. terebrantis, O. minus* and *O. clavigerum*. The lowest survival was observed in the *L. terebrantis* treatment and the highest survival occurred with untreated eggs. Thus, should *I. paraconfusus* become associated with the pathogenic fungi carried by cohabiting *Dendroctonus* spp., its survival may be reduced in the new host. Nevertheless, some progeny will survive in the presence of the newly associated pathogen, indicating the potential, at least, for a new fungal association with this bark beetle. In this regard, *L. terebrantis* has been isolated occasionally from *I. paraconfusus* (Fox et al., 1993). These investigators also showed that females reared free of any fungi (readily cultured) did not oviposit in surface- sterilized logs, whereas females reared from naturally-infested trees laid eggs in these logs. Perhaps these vectored fungi produce cues that identify the suitability of the phloem for oviposition.

Stephen and Dahlsten (1976a, b) determined the arrival pattern of associated insects on ponderosa pines during the concentration phase of host colonization (Wood, 1982) by *D. brevicomis*. Generally, predators arrive during this phase in response to pheromones, e.g. exo-brevicomin attracts *Temnochila chlorodia* (reviewed in Wood, 1982). However, parasites, such as *Roptrocerus xylophagorum* (Hymenoptera: Torymidae) and *Dinotiscus burkei* (Hymenoptera: Pteromalidae), arrive during the establishment phase, some 4-6 weeks after initial attacks have occurred. Parasitoids attack their bark beetle hosts, usually during the late larval stage. Dahlsten and colleagues (unpublished data) initiated field studies to test the hypothesis that beetle-

vectored fungi may be producing attractants that natural enemies use to locate beetle-infested trees. Logs were inoculated with *D. brevicomis*females, *O. ips, O. minus, O. nigrocarpum,* an unidentified basidiomycete and a combination of all four fungi. In one test, three species of hymenopterous parasitoids (*Coeloides* spp. and *Rhopalicus*) (families Braconidae and Pteromalidae) which parasitize bark beetles were trapped in significantly larger numbers at logs colonized by a combination of all fungi than at controls. These results are encouraging, because they indicate that the critical timing observed between these natural enemies and their prey may be a result of cues emanating from trees inoculated with fungi carried by bark beetles during host colonization.

Returning to a familiar theme, our present research addresses agents that predispose pines grown primarily in coastal, urban areas to infestation by bark beetles. In 1986 pitch canker disease, caused by *Fusarium subglutinans*, was discovered in Santa Cruz Co., CA, infecting Monterey, bishop and Aleppo pines (McCain et al., 1987). Studies by Correll et al. (1991, 1992) concluded that the *F. subglutinans* pathogenic to pines is a specific form and designated it *F. subglutinans* f. sp. *pini* (hereafter *F. s. pini*). They also showed that the California population included only five vegetative compatibility types (VCG) compared to 45 distinct VCGs isolated from a Florida population. Their studies suggested that this pathogen may be a recent introduction to California.

The sequence of symptoms of pitch canker disease in Monterey pines appears to begin with tip dieback which is followed by the occurrence of large bole cankers that produce copious amounts of resin. *Ips paraconfusus* generally kills the tops of large diseased trees, and soon thereafter, the entire tree is killed by *I. paraconfusus* and *I. mexicanus*. In native stands of Monterey pine, *I. plastographus maritimus* replaces *I. paraconfusus*. Thus, pitch canker appears to weaken trees and/or makes them more attractive to native *Ips* spp.

Although this pathogen appears to be endemic to the southeastern U.S., it is potentially much more destructive to forests of western North America than prior introductions, i.e., white pine blister rust, Dutch elm disease and chestnut blight. The host range of these pathogens is limited to a few species. In California *F. s. pini* has been isolated from twelve pine species and *Pseudotsuga menziesii* (Douglas fir) (Storer et al., in manuscript). The native pine species infected in California are *Pinus attenuata* (knobcone), *P. contorta contorta* (shore), *P. coulteri* (Coulter), *P. muricata* (bishop), *P. ponderosa* (ponderosa),*Pinus radiata* (Monterey), *P. radiata* x *attenuata* (Monterey x knobcone), *P. sabiniana*(Digger), and *P. torreyana* (Torrey). The non-native pine species infected in California are:*P. canariensis* (Canary Island), *P. halepensis* (Aleppo), and *P. pinea* (Italian stone) (McCain et al., 1987; Storer and Dallara, 1992).

Fox et al. (1990) have found that pitch canker probably increases the abundance of *Ips* spp. in Monterey pine in Santa Cruz Co., CA. *F. s. pini* has been isolated from *Ips* spp. emerging from *F. s. pini*-infected trees. *Ips* spp. also inoculated this

pathogen into wind-broken trees and thus produced reservoirs of the fungus. Experimentally contaminated *I. paraconfusus*transmitted this pathogen to seedlings and mature pines, and pheromone-induced attacks resulted in cankers on large trees (Fox et al., 1991). Also, transmission of this pathogen from adults to progeny was demonstrated by Fox et al. (1991).

Early observations of the disease in Monterey pine in the Santa Cruz area showed that new infections were often associated with cones (M. E. Schultz and T. R. Gordon, unpublished). Because the Monterey pine cone beetle, *Conophthorus radiatae* (Coleoptera: Scolytidae), attacks living cones, we initiated studies of the association of the pitch canker fungus with this beetle (Hoover, et al., 1994). In samples of cones taken from June, 1990 through September, 1991, we found that 25% of *C. radiatae* and 30% of *Ernobius punctulatus*(Coleoptera: Anobiidae) adults were carrying propagules of *F. s. pini. E. punctulatus* is a common associate of *C. radiatae* that oviposits on or in cones infested with *C. radiatae*. Furthermore, the percentage of cones containing contaminated *C. radiatae* larvae and adults was greater when *E. punctulatus* progeny were also contaminated than when *E. punctulatus* was not. Interspecific transmission may be significant in the epidemiology of this pathogen because we found both species together in 26% of the cones sampled. The parasitoid, *Cephalonomia utahensis* (Hymenoptera: Bethylidae), was observed parasitizing late instar larvae of *E. punctulatus* and thus may be another source of interspecific transmission.

In December, 1992 we isolated *F. s. pini* from bishop pine in southern Mendocino Co., about 150 km north of San Francisco (Dallara and Storer, in manuscript). In 1993, we confirmed the presence of the pathogen in two of the three native stands (Monterey peninsula and Ano Nuevo point) of Monterey pine found on the central coast of California (Storer et al., 1994). These new infections appear as dead tips in the crowns of the trees. *Pityophthorus* spp. are usually found infesting *F. s. pini*-infected tips in landscape plantings of these pine species. In early studies, Fox (in Wood et al. 1990) found greater than 10% of trapped *Pityophthorus carmeli* carrying propagules of *F. s. pini.* Hoover et al. (1994) have found up to 12% of*Pityophthorus* spp. trapped at cone whorls were carrying propagules of *F. s. pini*.*Pityophthorus carmeli, P. nitidulus*, and *P. setosus* have been found to carry this pathogen when emerged from symptomless and infected Monterey pine branches (Dallara, unpublished). Also these species infest other conifers in central, coastal California (Dallara, unpublished).

During the course of our six-year investigations, we have documented significant range expansion of *F. s. pini*. In Mendocino Co. infected bishop pines are intermixed with shore pine and Douglas-fir. These coastal conifers are all native stands. Infections reported from Monterey pine Christmas tree plantations in San Diego and Los Angeles Counties have increased since the pathogen was first discovered there in 1988 (Correll et al., 1991).

Our concerns about range expansion of the pitch canker fungus in California are twofold:

1) Endemic populations of some California pine species are very small. Monterey pine is limited to three coastal populations on the central coast. Torrey pine is limited to one mainland population in San Diego Co., and the northern distribution of Coulter pine occurs in small isolated stands in the San Francisco Bay Area.

2) Some of the most widely distributed conifers in North America have been found naturally infected in the Santa Cruz area, i.e., ponderosa pine, shore pine and, recently, Douglas-fir. Infected landscape plantings of Monterey pines are in close proximity to intermixed native stands of ponderosa pine, knobcone pine and Douglas-fir. Douglas-fir and ponderosa pine are the most abundant lumber species in western North America. We have recently found that several beetle species share many of these conifer hosts (Hoover et al., 1994; Dallara, unpublished). Furthermore, we have isolated *F. s. pini*from some of these species.

In summary, I have given a brief overview of our collaborative research on bark beetle, fungus, and host interactions involved in the death of pines. Photochemical air pollution, blackstain root disease, and tunneling by *D. valens* were shown to be associated with increased mortality of ponderosa pines caused by *D. brevicomis* and *D. ponderosae*. The role of fungi vectored by *Dendroctonus* spp. in causing the death of pines was described.*Ophiostoma minus,* carried by *D. brevicomis, O. clavigerum* carried by *D. ponderosae*, and*L. terebrantis* carried by *D. valens* kill seedlings. *O. minus* and *L. terebrantis* cause interruption of water conduction in the xylem of small trees. However, bluestain fungi (*Ophiostoma* spp.) are not frequently isolated from non-water conducting sapwood in *D. brevicomis*-killed ponderosa pines. Other fungi vectored by these *Dendroctonus* spp., when co-inoculated with pathogenic fungi, decreased the effect of the pathogenic fungi on seedling mortality and water conduction in small trees. The pitch canker fungus kills tree parts, i.e., limbs, branch tips, and cones and creates large cankers on the main stem. Together these effects increase the probability of tree mortality caused by Ips spp. This interaction is related to the bark beetle interaction with the blackstain root pathogen. A root-infesting bark beetle, *Hylastes macer,* is probably a vector of *L. wageneri* in ponderosa pine (Goheen and Cobb, 1978). This root pathogen predisposes ponderosa pines to infestation by *Dendroctonus* spp.
Similarly, *Pityophthorus* spp. and *C. radiatae* are likely vectors of a pathogen that predisposes Monterey pines to infestation by *Ips* spp.

Logging activity has undoubtedly increased the breeding material available for *H. macer*. This activity may have created a larger vector population for *L. wageneri*, with a concomitant increase in the incidence of blackstain root disease. Similarly, human activity is probably responsible for the recent introduction and spread of the pitch canker fungus in California. In this non-coevolved system, the increasing incidence of this disease is likely a result of increased abundance of the many potential bark beetle vectors identified to date (i.e.,*Conophthorus* spp., *Ips* spp., *Pityophthorus* spp.). This

increased abundance is probably a result of the new association with this pathogenic fungus that results in the death of cones, twigs, branches, tree tops and ultimately the death of the entire tree.

Since 1970, the above research was conducted under USDA Regional Research Project -W-110, "Interactions Among Bark Beetles, Pathogens, and Conifers in North American Forests." In 1993, this project was reorganized under RRP-W-187, with the same title. The knowledge generated from this long term cooperative research project was summarized by W-110 scientists in a recently published book entitled, "Beetle-Pathogen Interactions in Conifer Forests" (edited by T. D. Schowalter and G. M. Filip, 1993, Academic Press, N.Y. 252 pp.). These studies clearly demonstrate the tremendous complexity of the interactions among insects, fungi and conifers.

I acknowledge with deep appreciation my collaborators: university colleagues - Bill Bros (San Jose State University), Fields Cobb, Don Dahlsten, Tom Gordon, Dick Parmeter, Ron Stark; graduate students - Paul Dallara, Joe Fox, Ken Hobson, Kelli Hoover (San Jose State University), Ken Lindahl, Henry Moeck, Don Owen; post-doctoral researchers - Joe Fox, Andrew Storer. My wife, Caroline, encouraged and supported my efforts in these very enjoyable and facinating scientific explorations of forest biology. I also thank my nominators and the WFIWC Awards Committee for selecting me for the Founder's Award. Reviews by Fields Cobb, Don Dahlsten, Paul Dallara, Tom Gordon, Don Owen, Dick Parmeter, Steve Seybold, and Andrew Storer improved this manuscript.

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