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Armillaria Root Rot: Th

The root disease fungus *Armillaria* is one of the most prominent killers and decayers of deciduous and coniferous trees and shrubs in natural forest stands, plantations, orchards, and gardens throughout the world. The pathogenic role of *Armillaria* has been controversial historically. The fungus has been described variously as an aggressive killer of healthy trees, a secondary pathogen of stressed trees, and a saprophyte decayer of dead trees.

The gamut of pathogenic relationships of *Armillaria* occurs throughout the United States. There is a major contrast, however, between eastern deciduous forests, where *Armillaria* is predominantly a secondary pathogen on stressed trees (21), and western coniferous forests, where the fungus is often an aggressive primary pathogen (18,19). In this paper, we explain these relationships with recent developments in fungal taxonomy, genetics, and physiology; we summarize disease expression, damage, and control strategies in coniferous forests of the western United States and deciduous forests of the eastern United States; and we indicate current needs and directions in disease research and management.

Identity of the Fungus

Historically, specimens of *Armillaria* associated with dead and dying trees and shrubs have been identified as *A. mellea* (Vahl: Fr.) Kummer (= *Armillariella mellea* (Vahl: Fr.) Karst.) (23). Behavior of this fungus has been an enigma; sometimes it is damaging only when the host is already suffering from a known and recognizable stress and sometimes it is an aggressive primary pathogen.

Recent taxonomic and genetic studies have delineated several new species of *Armillaria* in Europe, North America, and Australasia (Table 1). This more detailed classification has explained some of the variations in symptom expression, pathogenicity, sporophore features, and cultural characteristics common to material traditionally classified as *A. mellea*. For example, the use of sporophore morphology and genetic analysis has to date provided specific names for five species of *Armillaria* in Europe. All these species occur in southern England, where Rishbeth (14) found that *A. mellea*, sensu stricto, was pathogenic on hardwoods and conifers, *A. ostoyae* was pathogenic on conifers, *A. bulbosa* was pathogenic only on stressed trees, and *A. tabescens* was virtually nonpathogenic. An unknown "Species B" (*A. cepaestipes* [or *cepistipes*] subsp. *pseudobulbosa*, Table 1) occurred only rarely and its pathogenicity was not determined experimentally, although it is not considered aggressive (J. Rishbeth, *personal communication*). Limited tests from elsewhere in Europe indicate similar results. Thus, the major pathogenic relationships—aggressive primary pathogen, secondary pathogen of stressed trees, and nonpathogenic saprophyte—previously ascribed to the single species *A. mellea* can now be variously attributed to several closely related but reproductively distinct species.

Fig. 1. (A) Aerial view and (B) ground view of expanding Armillaria root disease center in a conifer stand in Montana.

There are at least 55 named species of *Armillaria*, many of which were previously "lumped" into *A. mellea*, sensu lato. Although the present taxonomic status and pathogenicity of all these "species" is unclear, Table 1 provides information on several of the more common ones. Table 1 also emphasizes the distinct lack of and need for taxonomic work in the United States. The coupling of our knowledge of biological species in North America (2) with recognition of morphological taxa and pathogenicity testing lags behind similar work in Europe and Australia. Nomenclature has not yet been developed for the 10 biological species identified in North America. Research over the next few years, however, should clarify which of these species are common in the chiefly deciduous forests of the eastern United States and which predominate in the coniferous forests of the western United States, where contrasting pathogenic relationships exist.

Behavior of *Armillaria* in Western Coniferous Forests

In western coniferous forests, *Armillaria* behavior, and thus expression of

Puzzle Is Being Solved

root disease, differs markedly between the dry interior forests and the wetter coastal forests (11). Although not yet shown definitively, the differences most likely relate to the species and genotypes within species of *Armillaria* present as well as to host species and environmental conditions.

In coastal forests, the fungus occurs commonly as a butt rotter in old trees and a decayer of dead and down trees. Its occurrence as a root disease organism generally is limited, however, and disease occurs primarily in plantations and natural stands less than 25 years old. An infection center involves only a few trees; on a stand basis, the proportion of trees affected rarely exceeds 5% (11). In gross appearance, damage from the disease resembles that caused by *Phellinus weirii* (Murr.) Gilb., a root disease of major consequence in coastal forests (20). The two fungi can occur together in the same tree or stand, but losses to *P. weirii* are greater.

By contrast, *Armillaria* frequently acts aggressively on pines (*Pinus* spp.), true firs (*Abies* spp.), and Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) in the drier interior region. It attacks, colonizes, and kills apparently healthy trees of all ages in enlarging disease centers (Fig. 1). These centers can cover several hectares and affect 25% or more of the trees in a stand.

Initiation of disease centers is frequently associated with harvesting operations (Fig. 2), although such centers are also common in unmanaged virgin forests. The fungus colonizes stumps and roots of cut trees from quiescent lesions that were on roots before harvest. These root systems serve as both an inoculum source and an avenue of fungus spread to roots of adjacent healthy trees. When the healthy host root is colonized by mycelium from a diseased root or by rhizomorphs, the root responds with a flow of resin that may occlude the fungus, confining it to a lesion at the infection site. The fungus either does not advance proximally within the root from such lesions or advances slowly, as long as the tree remains alive.

Small trees die relatively quickly, but large trees may remain alive despite numerous lesions on lateral roots. Eventually, an infection reaches or develops high on the taproot or at the root collar. The host responds with the usual flow of resin. The fungus progressively kills the cambium and adjacent tissues, causing a girdling, pitch-impregnated lesion that stresses and eventually kills the tree. Death is hastened by summer drought and high temperatures.

In dying trees, the fungus continues to develop in the root collar area, forming distinctive mycelial felts under the bark. The fungus continues to grow through the cambium of the roots and advances proximally from lesions on lateral roots. Roots of seedlings and smaller saplings may dry appreciably so tissues no longer support growth of the fungus or they may decay rapidly and not provide a food base for long-term survival. Root systems of larger trees, however, stay moist enough and are large enough for the fungus to decay root wood and survive for many years.

Rotting lateral roots of killed trees serve as inoculum sources and avenues

Fig. 2. Expanding disease center in a ponderosa pine stand associated with a large pine stump (foreground), a relic of previous harvesting operations in the stand.

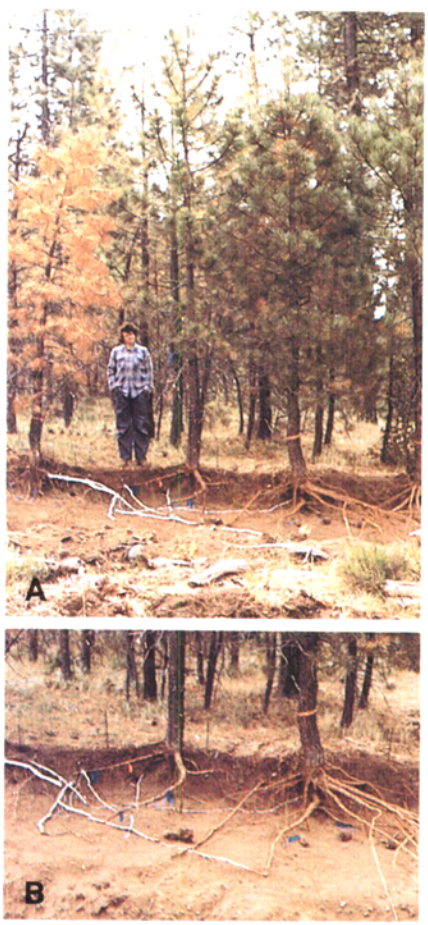

Fig. 3. (A) Spread of *Armillaria* from dead tree to living tree via root contacts. (B) Infected roots are painted white, and blue ribbons mark infection points. Metal stakes are about 1 m apart.

for spread of the fungus into adjacent healthy trees, and the infection process is repeated, enlarging the center by continued spread from tree to tree (Fig. 3). In central Washington State, Shaw and Roth (18) estimated that where stocking density and other environmental features are favorable, centers in ponderosa pine expand in diameter about 2 m per year.

The fungus spreads either by rhizomorphs, which grow through the soil from roots of infected trees to roots of healthy trees, or by direct transfer of mycelium at points of root contact. Spread by rhizomorphs is common in mesic coastal forests; in the drier interior forests where the fungus is most damaging, however, spread by rhizomorphs is of limited importance, even though rhizomorphs may occur frequently on root systems.

Spread by basidiospores also appears to be limited in interior western forests. The occurrence of the same genotype throughout a 600-ha infection center (3,18) indicates spread predominantly by vegetative extension. One would expect much more genetic diversity with spread by spores (3). Since sporophore production is moisture-dependent, the dryness of these interior forests may limit the frequency and amount of fruiting by *Armillaria* and hence its genetic diversity. The survival and infectivity of basidiospores in various environments need further study, however (16). The paucity of sporophore formation also causes problems in taxonomic classification based on fruiting body characteristics. The development of a reliable method for inducing formation of sporophores of *Armillaria* in vitro would considerably enhance taxonomic and pathological studies.

Behavior of *Armillaria* in Eastern Deciduous Forests

The dominant role of *Armillaria* in the chiefly deciduous forests of the eastern United States is as a secondary pathogen attacking trees weakened by biotic or abiotic stresses. The fungus colonizes and kills deciduous and coniferous trees that have been weakened by such stresses as defoliation by insects or frost, foliage diseases, stem cankers, bark-sucking insects, drought, waterlogging, soil compaction, and air pollution.

Weakened trees are colonized by rhizomorphs growing from roots of dead colonized trees or by mycelium from quiescent lesions reactivated by stress. The rhizomorphs are usually already on the surface of lateral roots of healthy and weakened trees. Although quite common and numerous in forested soils, rhizomorphs occur less frequently in new-growth forests on recently reforested arable land where tree mortality is low and the amount of substrate in the soil for colonization and rhizomorph development is limited and where inoculum from old-growth trees is absent. Rhizomorph occurrence on the root surface of healthy trees places the fungus in a position to take advantage of chemical changes induced by stress that may stimulate vigorous hyphal growth (21).

Armillaria can quickly colonize an entire root system after a severe stress such as defoliation or drought, or it may colonize portions of the root system beneath points of localized stem stress, such as cankers caused by beech bark disease (21). Several root lesions may coalesce and the mycelial fans advance toward and colonize the root collar. Attacked root tissues are decayed by the fungus, and rhizomorphs become abundant in the soil around killed trees. The rhizomorphs can spread from a diseased tree to a nearby tree. If that tree has also been stressed and weakened, it may be colonized and killed by the fungus. If the stress has abated and tree health is restored, however, colonization does not occur and spread of mortality ceases. No disease centers are formed by progressive colonization and mortality of adjacent trees. The fungus thus depends heavily on host stress for fulfilling its pathogenic role.

Even though individual trees of the same deciduous species may receive similar stress and have root system rhizomorphs, some may not be attacked by *Armillaria*. Differences in site, soil factors, and tree vigor are mitigating influences. The distribution of different species of *Armillaria* in the forest also may explain variations in attack and subsequent patterns of mortality (Fig. 4).

The genetic diversity of *Armillaria* may be greater in the eastern forests than in the western forests (3,18). There are at least eight biological species in eastern deciduous stands (2), compared with six in western coniferous stands (12). Within a biological species, clones (multiple isolates of the same genotype [8]) are smaller and occur more frequently in eastern forests, where as many as six clones may occur near (no more than 50 m apart) one another (3). The distribution in the same stand of multiple species with multiple genotypes of *Armillaria* having different pathogenic capabilities may explain differences in disease expression. The pathogenicity of different biological species and genotypes within species needs to be tested to verify this hypothesis. The genetic diversity in the East probably results from the diversity of host species and frequency and

Table 1. Better-known *Armillaria* species and their geographic distribution and pathogenicity[a]

Species	Location	Pathogenicity
A. mellea (Vahl: Fr.) Kummer, sensu stricto (Species D of Korhonen)	Europe, possibly northeastern United States	High
A. ostoyae (Romagn.) Herink[b] (Species C of Korhonen)	Europe, western North America	High
A. bulbosa (Barla) Kile et Watling	Europe, western North America	Low
A. tabescens (Scop.: Fr.) Emel.[c] (= *A. socialis* (D.C.: Fr.) Herink)	Europe	Nonpathogenic
A. cepaestipes Vel. subsp. *cepaestipes*[d]	Europe	Unknown
A. cepaestipes Vel. subsp. *pseudobulbosa* Romagn. et Marxmuller[d] (Species B of Korhonen)	Europe	Low
A. borealis Marxmuller et Korhonen (Species A of Korhonen)	Europe	Low
A. luteobubalina Watling et Kile	Australia	Moderate
A. hinnulea Kile et Watling	Australia, New Zealand	Unknown
A. fumosa Kile et Watling	Australia	Unknown
A. novae-zelandiae (Stevenson) Herink	Australia, New Zealand, South America	Moderate[e]
A. limonea (Stevenson) Boesewinkel	New Zealand, South America	Moderate

[a] References for these fungi are available from the authors.
[b] Same species as *A. obscura* (Schaeff.: Secr.).
[c] Relationship with *Clitocybe tabescens* (Scop.: Fr.) Bres. in southeastern United States is unclear.
[d] Species should be spelled *cepistipes*, according to F. Roll-Hansen (Eur. J. For. Pathol. 15:22-31, 1985).
[e] Considered low in Australia.

abundance of sporophores (3). In the East, fruiting occurs annually in the fall and sporophore abundance, not occurrence, is influenced by the amount of moisture.

The low pathogenicity on healthy trees and high pathogenicity and virulence on stressed trees in the eastern deciduous forest seems to be linked with physiological requirements of the fungus for growth and with its metabolic capabilities (21). Stresses such as drought and defoliation can induce chemical changes in roots that enhance fungal growth. Sugars, alcohols, and nitrogen compounds are some of the constituents whose concentrations change in root tissues with tree stress. These same compounds are associated with vigorous growth of the fungus (21). They not only stimulate rapid growth of *Armillaria* but also enable the fungus to grow in the presence of inhibitory substances such as phenolics (22).

Oxidation of phenolics may be especially important to the successful colonization by *Armillaria* of root tissues on stressed trees. In vigorous trees, *Armillaria* is confined to wounded and necrotic tissues; contiguous healthy tissues are not colonized. In trees weakened by stress, contiguous living tissues are "browned" by oxidative enzymes of the fungus, then colonized. Advance of the fungus into healthy tissues may be prevented because these tissues are in a highly reductive state that prevents oxidation of phenols by fungal enzymes. In stressed tissues, this ability to maintain the reductive state and confine the oxidative processes may be lost, and necrosis occurs as fungal oxidative enzymes are secreted and act on host phenolics.

Phenol metabolism may also influence major pathogenic relationships observed among isolates of *Armillaria*, as their abilities to oxidize and metabolize phenolics differ markedly (17,21). For example, gallic acid inhibits growth of western conifer isolates of *A. ostoyae* but highly stimulates growth of eastern oak isolates of *Armillaria* sp. (Fig. 5). Coniferous and deciduous tree groups and species within these groups have different kinds and quantities of phenolics in their root bark. Interactions of bark chemistry and fungal metabolic abilities may determine pathogenic relationships and therefore need to be investigated further and defined for both hardwoods and conifers.

In some conifer stands in the Northeast, *Armillaria* has acted aggressively. Stands of red spruce (*Picea rubens* Sarg.) at low elevations in the Green Mountains of Vermont have been severely affected by Armillaria root rot (Table 2). These stands had stagnated from overstocking and were thinned in the mid-1960s after one of the most severe droughts experienced in the Northeast. Some root disease centers have continued to develop since these initial infections were noticed.

Other stands of managed red spruce in Vermont have experienced similar problems after stagnation. The fungus is of little consequence, however, if early management operations maintain the stands in a healthy condition. *Armillaria* also was of little consequence in higher elevation spruce and spruce/fir stands where extensive mortality of red spruce has occurred recently (6). The scarceness of the fungus on dead and dying trees in these highly stressed upper elevation

Table 2. Examples of damage caused by Armillaria root rot in western and eastern coniferous forests

Location	Forest type	Synopsis[a]
Central Washington	Ponderosa pine	Mortality from Armillaria root rot increased from 9 m^3/ha in 1957 to 24 m^3 in 1971. If infection centers continue to enlarge at the current rate, this forest cannot be expected to persist as a commercial one.
Southern Oregon	Mixed conifer	Several large (1–4 ha) root disease centers have developed; 23% (103 m^3/ha) of the stand volume (448 m^3/ha) has already been killed by Armillaria root rot, and at least 9% of the live trees are infected.
Northern Idaho and western Montana	Mixed conifer	*Armillaria* is the most common root pathogen encountered. Annual losses of 2.3 million m^3 of wood occur from all root rots; 16% of commercial forest land has scattered root rot mortality, and 1% (3,100 ha) has severe damage in expanding disease centers ranging from less than 0.1 to over 40 ha.
Southern Idaho, Utah, and Wyoming	Mixed conifer	Conservative estimates place annual losses to *Armillaria* at 1,400 m^3 on 2,223 affected hectares.
Colorado	Subalpine fir	Root disease, caused primarily by *Armillaria*, occurs on 10.6% of the subalpine fir type on the San Juan National Forest, with an annual volume loss of 841 m^3. Estimates for the entire area conservatively place losses for the subalpine fir type at 3,500 m^3.
Arizona and New Mexico	Mixed conifer	Root diseases and associated pests were responsible for 34% of mortality and 30% of volume loss on six national forests. *Armillaria* occurred in 78% of diseased trees containing 80% of the volume.
Northcentral Minnesota	Balsam fir	*Armillaria* caused a 9% loss of stems in a 10-year-old balsam fir plantation established on an old hardwood site. Drought was suspected as a predisposing factor.
Wisconsin	Red pine	Outbreaks of Armillaria root rot in red pine stands caused losses of 12, 18, and 37% of the stems in three widely separated plantations. The disease was most common where plantations were established under oak and released by aerial sprays of chlorophenoxy acid herbicides.
New York	White pine	*Armillaria* had infected about 38% of the trees in a 32-year-old plantation. Mortality was just beginning and was scattered.
Central Vermont	Spruce and spruce/fir	Total mortality based on permanent plots in the Green Mountain National Forest is calculated at 81,000 m^3 per year for 7,200 ha of spruce and spruce/fir. About 75% of this mortality is associated with Armillaria root rot, or about 8.5 m^3/ha per year.

[a] References for these cases are available from the authors.

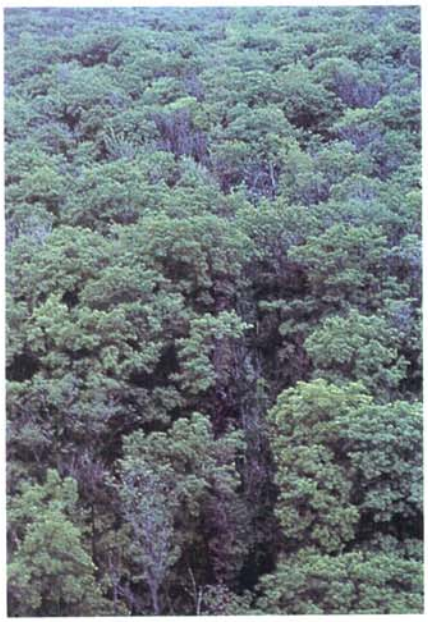

Fig. 4. Random patterns of clumped and individual dead oak trees colonized by *Armillaria* after gypsy moth defoliation in an eastern deciduous forest.

forests was unexpected, and factors responsible for its infrequent occurrence need to be clarified.

Calculating the Damage

Damage resulting from Armillaria root rot varies substantially from stand to stand and cannot be generalized for even one forest. The disease, however, is of some concern in most forested states. The selected examples of losses presented in Table 2 indicate a concern with the disease in forests throughout the country. In some stands, numerous, scattered disease centers may represent only 8% of the area. In other stands, several expanding and coalescing disease centers may account for as much as 32% of the area, with up to 95% of the commercial conifer species infected or killed by *Armillaria* (11). Commercial forests certainly would not persist without disease control. Losses in these areas occur not only because timber is destroyed but also because regeneration is killed by the fungus that remains viable in the roots of the previously killed trees. The stand is rendered to a commercially nonproductive status for decades. In stands where mortality occurs primarily through deaths of scattered individuals or small groups, volume losses may be minimal. This type of mortality occurs when the stands are young and the trees small.

Accurate estimates of losses to Armillaria root rot are not available for the more than 35 million hectares of timberland administered by the USDA Forest Service from the crest of the Sierra Nevada and Cascade mountains to the Great Plains. Most major forest holdings in the East are on private lands (many in small parcels), and data on losses are limited. In the East, most losses are associated with some stress factor and such secondary agents as bark beetles (21); in the West, losses are commonly associated with other root pathogens and bark beetles (7). Assigning or separating losses by cause becomes difficult, if not impossible. Reexamining these complex interactions after determining which *Armillaria* species are present should further clarify the roles of both organisms in tree decline and death—particularly as these roles relate to the long-debated topic of predisposition.

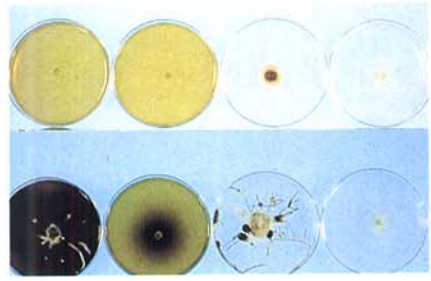

Fig. 5. Isolates probably representing different species of *Armillaria* with different abilities to metabolize gallic acid in vitro. Top row, western conifer isolate; bottom row, eastern oak isolate. Left to right: Gallic acid + ethanol, gallic acid alone, basal medium + ethanol, basal medium alone.

Fig. 6. Stump and root removal as part of site preparation before replanting in a forest affected by Armillaria root disease.

Considerations in Selecting Methods for Control

Even where mortality is noticeable, management implications of the disease depend on objectives for land use. For example, scattered mortality in dense, young stands may serve as a natural thinning agent and actually improve stand quality. Small disease centers in certain forest types may create openings that improve forage production and thus contribute to wildlife habitat. By contrast, where land is being used for recreation, such as campgrounds, the presence of even a few diseased individuals can be hazardous to people and property.

Table 2 indicates the type of losses in wood volume that can occur in affected stands. The volumes lost in many of these situations make commercial forestry on such sites economically unattractive. On the other hand, the desirability of disease control is somewhat limited on these same sites because of low crop values—an obvious dilemma for the land manager.

The extensive control operations affordable in orchards (13) are not applicable in forestry. Any control method must be compatible with other forestry values and be financially justifiable. Shaw and Roth (19) reviewed the options for controlling *Armillaria* in forests managed for timber production, and with some modification their summary follows:

1. Critically evaluate disease impact to ensure that the level of loss justifies control. Determine if *Armillaria* is the primary pathogen or is involved in a complex with other agents or pathogens.
2. Determine what information is needed—biological and economic—to develop a control program. Because of the general low value of forest crops, low-cost control through silvicultural modifications should be given first priority.
3. Exercise care in selecting a site for a new plantation. If the site is found to have a high disease hazard, one must be prepared for costly preestablishment treatments (probably through inoculum removals by more thorough site preparation), postponement of plantation establishment for an unknown period, or elimination of the site from the forest land base. Small-scale planting trials to evaluate disease potential can be established before large-scale land clearing and plantation development.
4. In planting, thinning, or harvesting operations, try to favor resistant species or varieties, if known, that are compatible with other forestry values.
5. Maintain stands in a vigorous condition by preventing damage to trees by other agents and by avoiding adverse sites. This recommendation is especially applicable in eastern forests, where stresses from biotic agents are involved in triggering Armillaria root disease.

Biological or chemical control measures may be desirable but require further development for practical application in forestry.

Roth and Rolph (15) have conducted successful control operations in commercial pine forests through removing stumps and roots from the margins of expanding disease centers (Fig. 6). Such operations, however, require detailed information on disease behavior and damage levels, accessible terrain, proper soil conditions, and a site of high enough quality to produce a reasonable timber volume after disease effects have been minimized. Even if all these needs have been met, disease management will not be successful unless the timber manager is knowledgeable of the disease and is willing to commit the resources needed for control.

Genetic Classification of *Armillaria* Species

All species of *Armillaria* tested have been found to be bifactorial heterothallic fungi with multiple alleles for the mating-type loci (2,10). The nuclear condition of somatic cells of most, if not all, naturally occurring isolates of *Armillaria* is uninucleate and diploid; yet, isolates developing from single spores can be maintained in the laboratory as haploids. Since clamp connections in *Armillaria* are confined to brief portions of the life cycle, compatibility between monosporous isolates paired in culture is determined by appearance of the mycelium. Single-spore isolates are haploid colonies with primarily white and fluffy aerial mycelium. In incompatible pairings, haploid isolates remain haploid and colonies remain white and fluffy (Fig. 7); compatible pairings yield diploid colonies with darkish crustose mycelium (Fig. 7) that are similar to those isolated from fruiting body stipes, rotten wood, or rhizomorphs (Fig. 8). Both single-spore haploid isolates and diploid isolates produce rhizomorphs when cultured on enriched malt media, although they appear more frequently and in greater quantities in diploid isolates.

By pairing monosporous isolates and observing the compatibility/incompatibility reactions, 10 reproductively isolated groups have been identified in North America (2), five in Europe (10), and four in Australia (G. Kile, *personal communication*). Limited compatibility exists between some groups from Europe and North America (1), although this work needs to be expanded using isolates from Siberia and Alaska where a land connection once existed.

These groupings are currently referred to as biological species, only some of which have been recognized as taxonomic species. Biological species are defined as groups that are reproductively isolated and the intersterility between groups is absolute (3). Epithets, based on morphological characteristics of sporophores, have been assigned to five European (14) and four Australasian (9) groupings (Table 1). None of the North American groupings has been recognized as a distinct taxonomic species. However, *A. ostoyae* and *A. bulbosa* are known to occur in certain forests of western North America (12,17), and Morrison et al (12) have proposed the species names of *A. ostoyae* and *A. bulbosa* for North American groups I and VII, respectively.

The biological species of a diploid field isolate can be determined by pairing it with haploid isolates of each biological species group and observing a change corresponding to the Buller phenomenon (10). In a compatible pairing of a diploid isolate with a haploid tester, the appearance of the cottony haploid changes to the appearance of the crustose diploid; in an incompatible pairing, the two isolates maintain their distinct appearances (8,10). Genotypes within biological species can be determined by pairing diploid field isolates (3,8); a distinct demarcation line forms between dissimilar genotypes but not between similar ones. The occurrence of different biological species and of different genotypes within biological species can be determined using these pairing techniques, and their distribution can be mapped within specific geographic locations. Such studies of localized distribution have been conducted in certain forest stands in Europe (10), Australia (8), New Zealand (4), and North America (3). Similar studies in orchards where Armillaria root rot is troublesome would seem appropriate.

Such work has shown that the age and area occupied by individual clones can vary considerably. For example, some clones in western coniferous forests are 400 m or more in diameter (3,18), whereas those in eastern forests rarely exceed 50 m in diameter (3). Those large, expanding clonal disease centers in western forests could be at least several hundred years old (18).

The ability to determine the biological species of field isolates, along with our increasing knowledge of variation in pathogenicity and virulence among and within species, provides diagnostic laboratories with the opportunity to evaluate the role of *Armillaria* in causing disease. This method also enables researchers in North America to place field isolates into a taxonomic species, or at least into a biological species. We strongly recommend that scientists working with *Armillaria* use these species names or the Roman numeral biological species classification of Anderson and Ullrich (2) when reporting their results. Thus, researchers and practicing pathologists can assist in solving the puzzle of why *Armillaria* is sometimes troublesome only when the host is suffering from a known and recognizable stress and other times behaves as an aggressive primary pathogen.

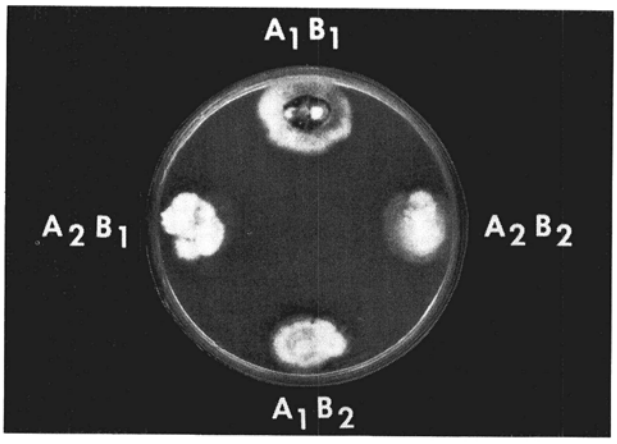

Fig. 7. Mycelial morphology developed from pairings of an unknown isolate with four haploid testers. Compatibility of the unknown isolate with the A_1B_1 tester is indicated by development of pseudosclerotia and oppressed mycelial growth.

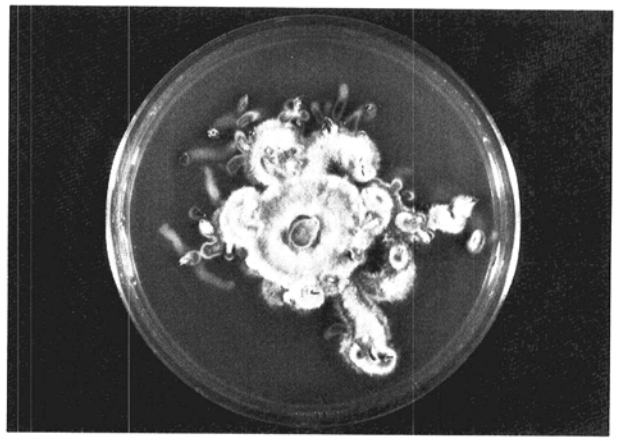

Fig. 8. Crustose mycelium of a diploid field isolate.

In the interior forests of western North America, *Armillaria* is widely dispersed, varies in behavior, and can cause lasting and debilitating effects on commercial forest land. Because of these concerns, the USDA Forest Service is sponsoring a series of workshops to develop a model of root rot behavior and the effects of various silvicultural practices on losses (5). When available, the proceedings should indicate current research needs and provide a method for evaluating options for disease management on any site. Practicing foresters and pathologists will be involved in developing the model. Perhaps this coinvolvement will provide the needed impetus for foresters to commit the necessary resources to control *Armillaria* and other root rots.

Acknowledgments

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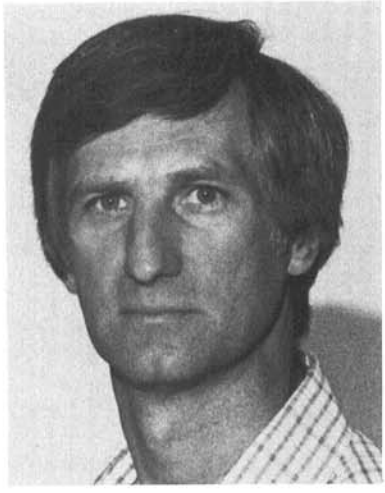

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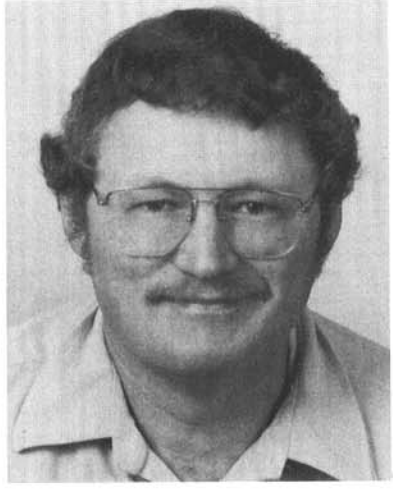
Dr. Wargo is a principal research plant pathologist with the USDA Forest Service's Center for Biological Control of Northeastern Forest Insects and Diseases in Hamden, Connecticut. He received a B.A. in biology from Gettysburg College in Pennsylvania in 1962 and an M.S. and Ph.D. in plant pathology from Iowa State University, Ames, in 1964 and 1966, respectively. His theses dealt with root and fungal physiology. From 1966 to 1968 he worked on phanerogamic root parasites while on active duty at Ft. Detrick, Frederick, Maryland. Since then he has been with the Dieback and Decline Diseases project of the Northeastern Forest Experiment Station at Hamden, determining the effects of stress on tree physiology and chemistry and evaluating the role these changes play in predisposing trees to secondary pathogens. His work with *Armillaria* has focused on the interaction of stress and fungal physiology on host susceptibility.



Charles G. Shaw III

Dr. Shaw is a principal research plant pathologist with the USDA Forest Service's Forestry Sciences Laboratory in Juneau, Alaska. He received a B.S. degree in forestry from Washington State University in 1970 and a Ph.D. in plant pathology from Oregon State University in 1974. His thesis dealt with an epiphytotic of Armillaria root rot in ponderosa pine, and he continued working on that disease from 1974 to 1977 while serving as a research scientist with the Forest Research Institute in Rotorua, New Zealand. Although his current research in Alaska deals with several forest diseases, he maintains an active research program on root rots, particularly those caused by *Armillaria*. During 1982–1983 he had a 6-month assignment with the Pathology Branch of the British Forestry Commission, where he was able to establish a worldwide culture collection of various species of *Armillaria* and test their in vitro responses to ethanol and phenolic compounds.