

The Impact and Control of Major Southern Forest Diseases

A. Dan Wilson, Theodor D. Leininger, William J. Otrosina, L. David Dwinell, and Nathan M. Schiff¹

Abstract—A variety of forest health issues, concerns, and events have rapidly changed southern forests and plantations in the past two decades. These factors have strongly impacted the ways we manage forest pests in the Southern United States. This trend will no doubt continue to shape forest pest management in the future. The major issues and events of concern include changing forest conditions, urbanization, multiresource issues, increased harvesting, forest fragmentation, expanding human populations, pesticide bans, expansions of native and nonnative pests into new regions, emergence of new damaging insect-disease complexes, and reduced resources to manage these problems. The effects of some of these factors on forest health priorities and specific pest-suppression practices are discussed in relation to some major hardwood and conifer diseases in southern forests. The ways in which these pests are influencing southern forest management priorities and practices and the progress that past and present pest-suppression research has made toward solving some of these pest-suppression problems also are discussed.

INTRODUCTION

The preceding two decades have brought a barrage of new developments that are shaping the evolution of forest management practices with regard to forest health issues and disease suppression in southern forests. Some of the more important developments impacting forest health management in the South include: (1) legislative bans on the use of many pesticides and chemical controls formerly used to manage forest pests; (2) continued introductions of nonnative pests to which many of our endemic tree species have little resistance; (3) expansion in distributions and outbreaks of important native pests into previously unaffected areas; (4) occurrence of new synergistic forest pest complexes previously unrecognized as important to forest management decisions; (5) nationwide reductions in the research work force (forest pathologists) available to study and develop new pest suppression technologies; (6) drastic reductions in forest management and pest-suppression activities on Federal lands; (7) inadequate approaches to regional pest problems as a result of overemphasis of theoretical research approaches, e.g., modeling systems and disease forecasting, instead of improvements in direct, applied approaches to disease suppression; and (8) the existence of new emerging endemic diseases such as bacterial leaf scorch (*Xylella fastidiosa* Wells and others) that are causing widespread damage previously unrecorded in commercially important fiber- and lumber-producing tree species (Billings 2000, Britton and others 1998). The impact of these issues and events on disease suppression and forest management decisions in general will be treated in the following discussions relevant to individual major hardwood and conifer diseases that occur in the southern region.

¹ Principal Research Pathologist, Research Plant Pathologist, and Principal Research Entomologist, U.S. Department of Agriculture Forest Service, Southern Research Station, Stoneville, MS, 38776; and Supervisory Research Pathologist and Principal Research Pathologist, U.S. Department of Agriculture Forest Service, Southern Research Station, Athens, GA 30602, respectively.

MAJOR HARDWOOD DISEASES

Oak Wilt in Urban Forests

Urban forests are becoming increasingly important components to be considered in the development of forest management objectives as cities and municipalities continue to encroach on natural forest stands. Protection of tree resources in urban areas is becoming more important, not only because urban trees have commercial lumber value or provide habitat and food for wildlife, but because their aesthetic value contributes significantly to property values. A good example of this trend has been demonstrated by the impact of oak wilt, caused by *Ceratocystis fagacearum* (T.W. Bretz) J. Hunt, on urban forestry. Within the last 20 years, oak wilt has caused increasingly devastating losses to valuable urban and suburban trees within and near metropolitan areas of Texas in the South and within major cities in Minnesota, Wisconsin, Iowa, and Illinois in the Midwest (Wilson 2001).

Tree mortality in urban areas causes economic losses in several ways. Reductions in landscape aesthetics resulting from tree mortality can significantly lower property values. The death of a single large urban live oak in Texas can result in a loss of as much as \$20,000 in property value (Dewers 1971). It is not uncommon for landowners

in Austin, TX, to sell their property once oak wilt has been diagnosed on their land in order to avoid the investment loss associated with the reduction in property value. Losing valuable shade trees can substantially increase utility bills (cooling costs) for homeowners. Tree removal costs also can be significant when they involve large trees. Finally, replacement costs associated with replanting trees adds to the final expense of losing valuable landscape trees. The consequences of increases in oak wilt incidence in valuable urban trees have resulted in accelerated economic losses now estimated to have exceeded \$1 billion over an area of at least 61 of 254 counties in Texas alone (Wilson 2001).

The rise in oak wilt incidence in urban areas has been attributed in part to increases in home construction and landscape improvement activities associated with urban development. Austin, TX, with over 10,000 live oaks (*Quercus fusiformis* Small and *Q. virginiana* Miller) killed by oak wilt in the last 20 years, may be the most heavily affected city in the United States. Residual trees often sustain considerable damage during initial tree clearing of land prior to home construction. Heavy equipment frequently scrapes and removes bark from trees, creating infection courts for the introduction of oak wilt inoculum by insect vectors (fig. 16.1). Tree wounding also occurs when trees are pruned by landowners during times when insect vector activity is high. When such trees become infected, they initiate infection foci from which new oak wilt infection centers develop and spread by root-graft transmission. The storage of oak wilt and bark beetle-infested firewood in piles near residences provides both inoculum and insect vectors by which wounded trees may become infected. An increase in incidence of oak wilt in natural stands has also contributed to a higher incidence in urban areas. Oak wilt incidence increased in many natural oak stands during the first half of the 20th century in the Eastern United States when Dutch elm disease, caused by *Ophiostoma ulmi* (Buisman) Nannf. and *O. novo-ulmi* Brasier, and chestnut blight [*Cryphonectria parasitica* (Murrill) Barr [formerly *Endothia parasitica* (Murrill) Anderson & Anderson]] caused changes in stand composition by removing dominant species that were largely replaced by red oak species susceptible to oak wilt (Wilson 2001). The increased incidence of oak wilt in natural stands has since been closely linked to changes in forest management practices such as high-grade harvesting, preferential thinning,



Figure 16.1—Live oak injured by heavy tree-clearing equipment at a residential building site in Austin, TX, providing entry points (infection courts) for introduction of the oak wilt fungus into the living sapwood by insect vectors. Photo by A. Dan Wilson.

overgrazing, and fire suppression that favor reduced species diversity and increase the number of susceptible red oaks in stands.

The Texas Forest Service administers the Texas Oak Wilt Suppression Project (TOWSP) with funding and technical assistance provided by a combination of U.S. Department of Agriculture Forest Service (Forest Service) funds and matching funds provided by the State of Texas. This has been among the very few cooperative (State) disease suppression programs in the country that have received Federal assistance since 1995 (Wilson 2001). A recent addition occurred when the sudden oak death pathogen, *Phytophthora ramorum* Werres, was first discovered in the United States (California) in 1995, and Federal funds were appropriated for research and suppression beginning in 2003. TOWSP personnel coordinate the efforts of local governments and private citizens to detect and control oak wilt. The project's goals are to educate the public, locate disease centers, provide technical and cost-sharing assistance in suppressing the fungus, and monitor suppression treatments to control spread.

Recent improvements in oak wilt management have resulted from modifications of existing control strategies, empirical advances arising from experiences gained during implementation of suppression programs, and research developments of new suppression technologies. Trenching, the practice of mechanically cutting root connections between healthy trees in advance of the visible expanding edge of infection centers to control root transmission of the oak wilt fungus, has been recommended for many years (Himelick and Fox 1961) and has long been the cornerstone and primary means of suppressing the spread of oak wilt in the United States (Wilson 2001). In Texas, the fungus spreads primarily through interconnected root systems, creating infection foci with radial expansion rates that sometimes exceed 31 m/year. In attempts to stop the advance of infection, cooperators in the TOWSP began cutting barrier trenches in 1988. The TOWSP installed over 762,000 linear m of trench around almost 4,000 infection centers detected in central Texas by 2000 (Wilson 2001). This represented treatment of about 44 percent of confirmed centers detected, but < 10 percent of infection centers likely to exist statewide. Trenching was about 67 percent successful in stopping the spread of encircled infection centers by 1994 (Billings and others 2001). Since 1994, 76 percent of trenches

have had no breakouts. This improvement was attributed to installation of deeper trenches (up to 1.8 m) and improved trench placement. The majority of trench breakouts that occurred within the first 3 years after trench installation were due to improper trench placement or insufficient trench depth that failed to sever preexisting root grafts. Breakouts that occurred 3 or more years after trench installation were more likely to result from the formation of new root grafts across the trench by fusions of new adventitious roots arising in the loose, trench-backfill soil from roots previously severed by trenching (Wilson and Lester 2002). During the first several years following trench installation, an abundance of small adventitious roots commonly formed from roots severed in the loose backfill soil by trenching. These roots provided opportunities for initiation of new root-graft connections across trenches in subsequent years.

A recent oak wilt suppression research study, conducted by a Forest Service scientist, investigated the effectiveness of trench insert materials in preventing trench breakouts initiated by root grafting across the trench. These results indicated that trench inserts did not significantly reduce or stop root transmission during the first 3 years following trench installation, but that the use of water-permeable inserts effectively improved the performance of trenches beyond the third post-trenching year, when trenches are still normally effective, and extended trench longevity indefinitely (Wilson and Lester 2002). Water-impermeable materials, however, sometimes promoted trench breakouts by their tendency to redirect root growth around these barriers, leading to the development of new root-graft connections and associated oak wilt root transmission across the trench. Water-permeable inserts were more effective root barriers because they did not direct root growth from the point of root contact. The additional minimal cost of trench inserts above trenching costs is justified in urban and rural homestead sites where valuable landscape trees require more protection, and additional retrenching costs are avoided. Assuming that trench depth and placement problems are now solved through experiences gained by the TOWSP, this improved method of oak wilt suppression should greatly increase trench effectiveness, and could potentially save Texas landowners (alone) hundreds of millions of dollars in tree removal costs and property value depreciations if this control is vigorously implemented by the TOWSP.

The trenching research also confirmed that applications of the systemic fungicide propiconazole (Alamo®) by high-volume bole injections, used by the TOWSP until 1997, are ineffective in preventing root transmission of the oak wilt pathogen in individual trees, despite the high sensitivity of the fungus to this fungicide (Wilson and Forse 1997). The sporadic and undependable effectiveness of propiconazole was attributed to the predominately upward mobility of the fungicide, which precluded root treatment when the fungicide was injected into the lower bole. As previously applied, only a small fixed proportion of the injected active ingredient moved down into the root system by vapor phase activity. City arborists partially compensated for this by increasing the dosage of the fungicide from 3 to 10 ml/L or higher, thus increasing the amount of active ingredient moving down into the roots by vapor phase activity. However, previous research indicated that soil-drench applications of the fungicide at the tree dripline immediately prior to challenge inoculations provided more effective treatment (better coverage) and more protection of root systems, because the fungicide is applied and taken up at the distal ends of roots near root apices, thus allowing more complete and thorough distribution throughout the entire root system (Wilson and Lester 1995).

The combined effect of using the improved trenching methods (cultural control) with trench inserts to prevent root transmission of oak wilt, together with the increased effectiveness of soil-applied fungicide treatments, should significantly advance efforts to suppress oak wilt disease in semievergreen live oaks in Texas and in deciduous oak species affected by this malady in other States. If these controls are implemented, they could potentially save landowners hundreds of millions of dollars in tree removal costs and property value depreciations in Texas, and substantially greater savings in other areas of the United States affected by this disease.

Hardwood Plantation Diseases

Hardwood tree species have been grown in plantations throughout the Southeastern United States for more than 50 years, although the total acreage in hardwood plantations is much less than that in softwood species. Since the early 1990s, market conditions and new approaches to environmental issues have led to changes in cultural methods for growing hardwoods and the planting of many more acres of hardwoods. Fiber-farming technology has allowed industrial

growers to plant bottomland hardwood species on upland sites where rapid growth is fostered by irrigation and liquid fertilization, a method referred to as fertigation. This cultural method allows year-round harvesting, whereas wintertime harvesting in natural bottomland stands is limited by wet soil conditions and associated environmental concerns. In the Lower Mississippi River Alluvial Valley (LMRAV) and other areas in the Southeast, agricultural land is being afforested in response to changing agricultural markets and increasing interest in ecosystem restoration (Stanturf and others 2000). Fiber farming and large-scale afforestation present unique challenges and opportunities to growers and pest management professionals.

American sycamore (*Platanus occidentalis* L.) has good commercial value because of its rapid growth and excellent pulping qualities for the production of paper products. Sycamore is commonly used in afforestation efforts. During the early and mid-1970s, sycamore decline was the main problem of concern to sycamore producers in the Southeastern United States. Surveys conducted in the 1970s, focusing on leaf scorch, dieback, and cankers, found a complex of diseases associated with sycamore decline. These included canker stain, caused by *Ceratocystis fimbriata* (Ellis & Halst.) F. *platani* J. M. Walter; Botryosphaeria canker, caused by *Botryosphaeria rhodina* (Cooke) Arx; and anthracnose, attributed to two conidial stages of *Apiognomonium veneta* (Sacc. & Speg.) Höhn (Filer and others 1975). Leaf scorching occurred in all locations surveyed. Leaves were described as scorched, eventually turning completely brown, but not shedding prematurely. These symptoms are common for bacterial leaf scorch, a disease caused by *Xylella fastidiosa* Wells and others (Leininger and others 1999, Sherald and Kostka 1992), but which was attributed in the 1970s to late-summer symptoms of anthracnose caused by fungi, particularly species of *Colletotrichum* (McGarity 1976). Tree diseases caused by *X. fastidiosa* were considered hard to diagnose in the past because diagnosticians were unfamiliar with the pathogen and no diagnostic tools were available to detect it. Symptoms were easily confused with those of other biotic and abiotic factors such as moisture stress and herbicide damage. The presence of the bacterium in trees previously was difficult to confirm using routine laboratory techniques because of its fastidious nature (Sherald and Kostka 1992). The advent of enzyme-linked immunosorbent assays (ELISA) has made

diagnosis of *X. fastidiosa* infections in plants routine, and has facilitated the detection of bacterial leaf scorch in sycamore throughout the Southeast. Polymerase chain reaction is also being used to detect this bacterium in plants. Recent visual surveys and ELISA testing of sycamore plantations across the Southeast showed that bacterial leaf scorch caused severe dieback, decline, and mortality to sycamore saplings growing on sites with or without irrigation (Britton and others 1998). Initially, necrotic zones appear along the midrib and main veins of leaves by late July of the second growing season. Severe marginal leaf scorching in foliage throughout individual crowns and the entire stand is common by the third year. Branch and top dieback occurs in 50 percent or more of a stand and some mortality may occur by the fifth year. In severe cases, premature salvage harvests are justified because of concerns that stands will not contain sufficient volume at the normal pulpwood rotation age to pay for the additional carrying cost. Research is currently underway to identify sycamore genotypes that are tolerant to bacterial leaf scorch disease (Chang and others 2002).

Many of the same hardwood species used in fiber farming also are used for afforesting former agricultural fields. These include several oak species, eastern cottonwood (*Populus deltoides* Bartr. ex Marsh.), American sycamore, and green ash (*Fraxinus pennsylvanica* Marsh.). Forest restoration through afforestation is just beginning on a large scale in the LMRAV, and many successful planting and cultural methods are in use (Stanturf and others 2000). Development of new management methods for controlling insect and disease pests in these monocultural plantation settings is badly needed. Cherrybark oak (*Q. falcata* var. *pagodifolia* Ell.) seedlings growing in nursery beds are susceptible to leaf injury and stunting from *Cylindrocladium scoparium* Morg. (Smyly and Filer 1977). Newly emerged hardwood seedlings of many species are susceptible to damage from soil-borne fungi such as species of *Fusarium*, *Rhizoctonia*, and *Pythium* that cause damping-off (Filer and Cordell 1983). Insect and disease management guides for oaks (Solomon and others 1997), sycamore (Leininger and others 1999), cottonwood (Morris and others 1975), and ash (Solomon and others 1993) will aid in diagnosing many problems, especially in older stands. However, disease problems in nurseries and on stored and newly planted seedlings will require research and development of new control methods, especially since traditional controls such

as methyl bromide have been eliminated. Concern for the surrounding environment is likely to lead to the development of biological and chemical controls that minimize long-term effects on ecosystems adjacent to plantations.

Declines of Oaks and Other Hardwoods

Decline disease syndromes, commonly called declines, have been described by Manion and Lachance (1992) as a progressive interaction of abiotic events and biological factors or agents that eventually can lead to individual tree death and widespread forest mortality, depending on the severity of the decline event. Declining trees typically have been predisposed by abiotic factors such as site index, soil type, and climate change, although biotic factors such as old age or genotype also can be predisposing factors. Actual decline is triggered or incited by biotic or abiotic factors such as drought, flooding, insect defoliation, or air pollution. Trees that are already in a weakened physiological state are weakened further, and in this condition may die. They may recover, perhaps to succumb later to other stresses. Biological agents, for example, wood-boring insects, phytophagous insects, wood decay fungi, and bacteria can quickly colonize a physiologically weakened tree and contribute to its final demise. Declines occur periodically and are often triggered by climatic extremes. For example, a report by Ammon and others (1989) summarized 26 decline events during the previous 140 years, and many of these were brought on by periods of drought.

The most recent Forest Service report of forest insect and disease conditions in the United States (U.S. Department of Agriculture, Forest Service 2002) lists several Southeastern States in which forests are experiencing oak decline as a result of severe summer drought from 1998 to 2000. A drought-induced decline of red oaks in the Ozark and Ouachita Mountains of central Arkansas reportedly covers hundreds of thousands of acres and is associated with extraordinarily high numbers of red oak borers [*Enaphalodes rufulus* (Haldeman)] as a contributing factor in the decline (U.S. Department of Agriculture, Forest Service 2002). The Forest Service report also describes oak declines in the Appalachian Mountains of Virginia, North Carolina, Georgia, and in Tennessee, where white oaks were especially affected. During the summer of 2000, many Nuttall (*Q. nuttallii* Palmer), willow (*Q. phellos* L.), and water (*Q. nigra* L.) oaks began declining in the Dewey-Wills Wildlife Management Area in east-central Louisiana because of the 1998

to 2000 drought. To date, nearly 6,000 acres of red oaks have been affected, and the decline includes attacks by the red oak borer and bacterial wetwood infections. In west-central Mississippi during the late spring of 2001, about 2,000 acres of plantation-grown eastern cottonwood trees were severely defoliated by a notodontid moth [*Gluphisia septentrionis* (Walker)]; again, the 1998 to 2000 drought was believed to be a predisposing factor in this decline. Defoliation by the common *Gluphisia* was followed by cottonwood leaf beetle (*Chrysomela scripta* F.) defoliations on the second flush of leaves in 2001, further weakening the trees. This pest of *Populus* species, which is common in the Northeastern United States and Southeastern Canada, defoliated the same cottonwood trees in late spring 2002; lower boles were also infected with bacterial wetwood.

Decline diseases involving climate may be of particular concern for future southern forests if predictions of extremes in atmospheric temperature and precipitation resulting from increased greenhouse gases hold true. Anthropogenic inputs of gases such as carbon dioxide, methane, and oxides of nitrogen into the atmosphere have been increasing for some time above apparently normal historic levels (Ning and Abdollahi 1999). Some research suggests that these increased gas concentrations are affecting global surface temperatures by altering the amount of solar energy reflected off the Earth's surface, resulting in the greenhouse effect (Ning and Abdollahi 1999). Various hypotheses and process models attempt to explain possible climate changes and the subsequent effects to natural and man-made ecosystems (National Assessment Synthesis Team 2000). If there are major systematic changes occurring in the climate, they will likely give rise to more numerous decline-related insect and disease problems.

Root and Butt Rots

Root and butt rots are the most serious cause of lumber cull and degrade in southern forests. All southern hardwood species are affected, and the loss in terms of hardwood timber volume amounts to millions of board feet annually. The lower bole has always been of most concern to hardwood forest managers because these are the most valuable logs in the tree and the logs most likely to be wounded by harvest equipment, by logs pulled on skidder tracks, and by falling trees. During most of the 20th century, forest managers have tried to suppress root and butt rots in southern hardwood stands by preventing the

creation of wound scars by which most decay fungi gain entry into the tree. During the first half of the last century, much effort went into controlling wounds caused by fires. At least 80 percent of lower bole decays in bottomland hardwoods were attributed to fire scars during that period (Toole 1960). Protection of the lower bole is still of prime concern in avoiding wounding. However, because fires are rarely a problem in hardwood forests today, this concern has largely shifted from fire wound management to management of logging wounds in residual trees caused by heavy harvesting equipment during precommercial thinning and partial commercial cuts. As demand for hardwood lumber volume increases in the future, management of root and butt rots in hardwoods will slowly begin to move away from the tolerance approach, or a willingness to live with and allow for a certain amount of cull losses by increasing cut volume, to a more preemptive approach based on detecting these microbes in standing trees and adjusting harvest schedules to reduce losses. This approach will require the capability of detecting incipient decay in standing trees and determining the specific causes of decay. However, new technology and decay models will have to be developed to provide the necessary knowledge and detection capabilities before this approach becomes feasible.

At least 30 fungi are known to contribute to root and butt rots in southern hardwoods, but only a relatively few species cause most of the damage. The root and butt rot fungi most frequently encountered in most southern hardwood stands include *Pleurotus ostreatus* (Jacq.:Fr.); *Ganoderma lucidum* (W. Curt.:Fr.); *Hericium erinaceus* (Bull.:Fr.) P.; *Armillaria tabescens* (Scop.) Den.; *Inonotus dryadeus* (Pers.:Fr.) Mu.; and *Laetiporus sulphureus* (Bull.:Fr.) Mu. Other species that are important to a lesser extent in individual hardwood species include *Inonotus hispidus* (Bull.:Fr.) P. and *Tyromyces fissilis* (Berk. & Curt.) Donk, *Lentinus tigrinus* (Bull.:Fr.) Fr., *Phellinus igniarius* (L.:Fr.) Quél., *Trametes versicolor* (L.:Fr.) Pil., *Rigidoporus lineatus* (Pers.) Ryv., *R. ulmarius* (Sowerby:Fr.) Imazeki in Ito, *Tomentella* spp. (Pat.), and the ascomycete *Kretzschmaria deusta* (Hoffm.:Fr.) P. Martin (= *Hypoxylon deustum* (Hoffm.:Fr.) Grev.). The rate of decay development within hardwoods varies with the specific wood decay fungus present and the host species involved (Toole 1959). Thus, decay volume models must account for host species, decay fungi, and log taper equations of individual

hardwood species when predicting future lumber volume losses.² This information would be necessary for making stand harvesting decisions. Also, a portable, inexpensive, easily used detection device would be necessary to identify the presence and extent of damage by specific decay fungi in standing trees during routine stand evaluations by timber cruisers for the purpose of planning future harvest schedules.

The development of new technologies and methodologies for mitigating losses by wood decay fungi and other microbes causing defect losses in standing timber has been an active field of interest in recent years (Wilson and Lester 1997). Forest managers and cruisers responsible for monitoring forest stands are primarily interested in methods and criteria for minimizing losses in lumber volume and optimizing production in commercial forests. A major challenge facing forest managers is that of establishing policies and procedures for making management decisions to deal with defect losses including decay, discoloration, and structural alterations in the properties of wood caused by microorganisms in the sapwood and heartwood of standing timber. Most estimates indicate that at least 30 percent of the total lumber volume available in many southern hardwood stands is degraded or rendered unmerchantable by lumber defects caused by these pests. Defects in logs of standing trees can lead to significant economic losses ranging from reduced lumber production volume per acre to reduced lumber value (grade), degrade to pulpwood status with no merchantable lumber, and ultimately total loss with no commercial value available for salvage. The most significant challenges to be addressed in relation to defect volume losses in lumber production are to find ways of detecting defect in logs of standing trees and to determine when to cut individual trees that have log defects in order to optimize production on an individual tree basis. The methods used over the past 50 years to detect the presence of log defects in standing trees by cruisers of most commercial lumber producers have involved “sounding” the wood (butt log) by striking it with a hard object to locate hollows in the lower bole. This archaic method is useful only to detect advanced defect in standing trees because trees with incipient or even intermediate stages of defect usually cannot be distinguished from healthy trees. Unfortunately, detecting

advanced defect is of little value, because it only serves to identify unmerchantable trees. Also, it occurs long after the decision should have been made to harvest the tree and avoid the high level of cull losses associated with the development of defect to advanced stages.

Previous strategies for managing defects in southern hardwoods involved simply accepting the defect losses caused by microbes and insects by removing the cull volume as the logs were processed at the mill. With the growing demand for quality lumber volume in the United States, new technologies are now needed with the capability of detecting defects in logs of standing trees at incipient stages before significant damage reduces the resulting lumber value in individual trees. New methods and technologies under development, such as electronic aroma detection by conductive polymer analysis (CPA) of volatile metabolites released from microbial log-degrading pests, will allow preharvest field detection of log defects using a portable detector (Wilson and Lester 1997). This will be much more effective than older methods in optimizing lumber yields because it will prevent cull losses by allowing detection and control of the problem long before significant damage occurs. Early detection of these defect-causing microbes in standing trees is useful for predicting future potential damage because the damage potential is species-specific and thus the future depreciated value of individual trees can be estimated by using decay models coupled with fungi-specific decay expansion constants in different hosts. An integral part of this early detection system is the identification of the specific microbe(s) present, because the rate of development, type of damage, and location of defect volume depends on the particular pest present. Several applications of this technology are being developed. For example, CPA recently was used to distinguish the aroma signatures of sapwood cores (host woods) from southern hardwood species (table 16.1). Technology also has been developed to identify forest pathogens and wood decay fungi *in vitro* and in wood samples, and to distinguish between different *Armillaria* species for disease diagnosis (table 16.2). Host- and fungi-specific decay-volume models based on log-taper equations of individual hardwood species also are under development with the objective of predicting future lumber volume losses for planning and establishing future harvest schedules for individual hardwood stands (see footnote 2).

² Wilson, A. Dan. 2002. Wood decay volume models. [Not paged]. Unpublished data. On file with: Southern Research Station, P.O. Box 227, Stoneville, MS 38776.

Table 16.1—Global class membership (identity) of aroma profiles for sapwood cores of selected southern hardwoods based on electrical aroma signatures obtained from the 32-sensor array of the Aromascan A32S

Sapwood cores	Global class membership ^a					
	<i>A. rubrum</i> ^b	<i>C. caroliniana</i>	<i>C. laevigata</i>	<i>L. styraciflua</i>	<i>P. deltooides</i>	<i>P. occidentalis</i>
	----- percent -----					
<i>Acer rubrum</i>	98.0	0.0	0.0	0.0	0.0	0.0
<i>Carpinus caroliniana</i>	0.0	99.2	0.0	0.0	0.0	0.0
<i>Celtis laevigata</i>	0.0	0.0	91.3	3.8	0.0	0.0
<i>Liquidambar styraciflua</i>	0.0	0.0	3.7	96.7	0.0	0.0
<i>Populus deltooides</i>	0.0	0.0	0.0	0.0	98.8	0.0
<i>Platanus occidentalis</i>	0.0	0.0	0.0	0.0	1.5	99.3

^a Percentage global class membership (relatedness) of sapwood core aroma profiles based on comparison against reference database for southern hardwoods. Data only list comparisons between aroma signatures of sapwood cores from these six hardwood species.

^b Mean global class membership for 10 replications per treatment.

Table 16.2—Determinations of global class memberships (identity) of aroma profiles for four *Armillaria* spp. based on electronic aroma signature comparisons with an *Armillaria* reference library database obtained from the 32-sensor array of the Aromascan A32S

<i>Armillaria</i> spp.	Global class membership ^a			
	<i>A. gallica</i> ^b	<i>A. mellea</i>	<i>A. ostoyae</i>	<i>A. tabescens</i>
	----- percent -----			
<i>A. gallica</i>	100.0	0.0	0.0	0.0
<i>A. mellea</i>	0.0	99.2	8.7	0.0
<i>A. ostoyae</i>	0.0	0.0	91.3	0.0
<i>A. tabescens</i>	0.0	0.8	0.0	100.0

^a Percentage global class membership (relatedness) of *Armillaria* aroma profiles based on comparison against reference database for these four *Armillaria* species. Data only list comparisons between aroma signatures of these four *Armillaria* species.

^b Mean global class membership for 10 replications per treatment.

Insect-Wood Decay Pest Complexes

Of all the pests that reduce hardwood lumber production, none are more important than the wood decay fungi and the hardwood borers. Those capable of acting together in symbiotic complexes are even more damaging. Recent Forest Service research at the Southern Hardwoods Laboratory in Stoneville, MS, has been aimed at identifying and quantifying losses caused by important insect and disease pests that are causing substantial reductions in hardwood lumber production and value. This work has revealed new, previously unknown woodwasp-wood decay fungi complexes capable, in some cases, of causing considerable

damage to logs in standing trees, ultimately reducing hardwood lumber value. These wood decay fungi are mycosymbionts of a peculiar group of insects, the woodwasps (Hymenoptera: Siricoidea), with larval stages that bore through the wood of stressed and weakened hardwood trees and cause significant damage by forming galleries and vectoring (transmitting) wood decay fungi in the process (Gilbertson 1984, Smith 1979). Like most wood-feeding insects, woodwasps must live in symbiotic relationships with wood decaying microbes because they are incapable of digesting cellulose. The decay fungi are carried in special glands (mycangia) at the base of the abdomen near

the ovipositor in female woodwasps. The adult female woodwasp stores inoculum of the wood decay fungus in these mycangial glands, which are connected directly to the oviduct that passes through the ovipositor. The decay fungus is injected into the wood with the eggs during oviposition. The fungus then grows rapidly and produces extracellular cellulases, which digest the wood for larval consumption (Kukor and Martin 1983). When the eggs hatch, the larvae begin boring through the decayed wood, consuming nutrients both from the decayed wood and the mycelium of the fungus itself. The larvae cannot consume and digest the wood until it is decayed by the enzymes of the fungus. The larvae produce extensive galleries throughout the rotting wood, eventually pupate in the wood, and emerge as adults making round exit holes. The wood is decayed far beyond these borer galleries in all directions. Most of these fungi grow very rapidly through the wood, and the wood is decayed almost completely over several years as both the cellulose (wood fibers) and lignin are digested by extracellular enzymes (Wilson and Schiff 2003). Thus, all of these fungi are physiological white rotters. The actions of these two pests together result in synergistic damage to and economic loss of merchantable hardwood lumber volume. The decay fungi also produce discoloration in the wood (a type of stain called zone lines) that further degrade lumber value. The zone lines, produced within decaying wood in association with these wood decay fungi complexes, are a result of somatic antagonism (SA) between different strains of the wood decay fungi competing for the same wood substrate (Wilson and Schiff 2000a). Zone lines that form in wood as SA interactions between xylariaceous fungi represent areas delimiting their territory around decay zones (fig. 16.2). The wood becomes riddled with all three types of damage (borer galleries, decay, and discoloration of wood) until the entire branch or bole becomes unmerchantable. This is a perennial process in which the damage may be compounded by repeated infestations of branches and boles by subsequent generations of the woodwasp.

Two major groups of woodwasps can affect hardwood lumber production. The large, siricid woodwasps (Siricidae: subfamily Tremicinae) attack predominantly oaks, sugarberry (*Celtis laevigata* Willd.), beech (*Fagus grandifolia* Ehrh.), and other bottomland hardwood species. Smith and Schiff (2002) provide a review and keys to the siricid woodwasps of the Eastern United States. These tremecine woodwasps vector predominantly

basidiomycetous wood decay fungi. The two most common species are *Tremex columba* (Linnaeus) and *Eriotremex formosanus* (Matsumura). The smaller xiphydriid woodwasps attack mostly maples, elms, and upland hardwood species. They carry ascomycetous wood decay fungi that form spores in microscopic sacks (asci) inside of perithecia embedded in black stromatic tissues that develop on the surface of the wood. The woodwasp family (Xiphydriidae) has 22 described genera, approximately 100 species, and a worldwide distribution (Smith 1978). The family is represented in the United States by a single genus, *Xiphydria*, with 10 described native species. Hitherto, we have isolated the mycosymbionts from 6 of the 10 native xiphydriid

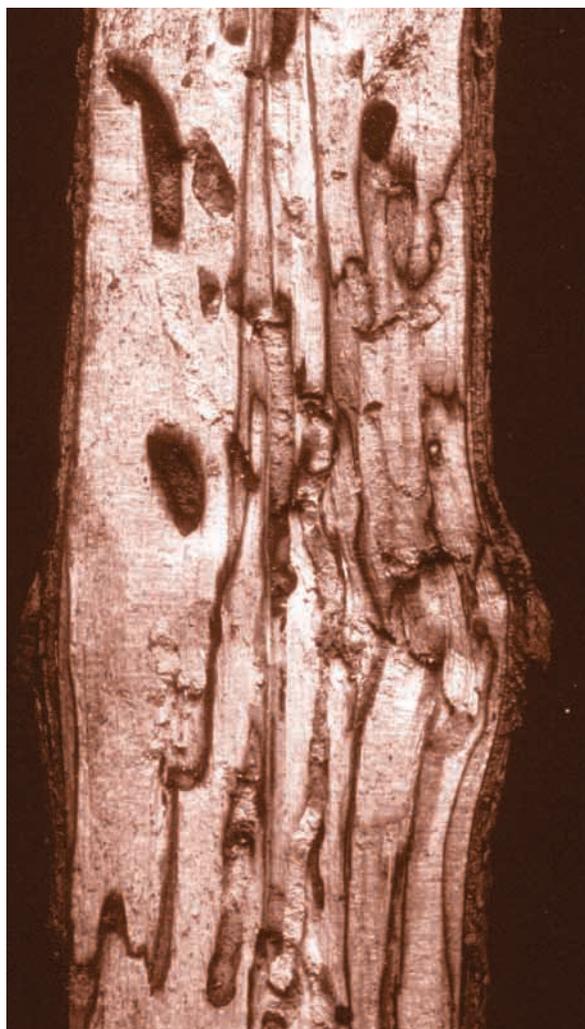


Figure 16.2—Zone lines observed in decayed wood of sugar maple colonized by *Daldinia concentrica*, mycosymbiont of *Xiphydria maculata* woodwasp larvae. These antagonistic interactions form between the decay zones of xylariaceous wood decay fungi around woodwasp galleries, and represent areas delimiting their territory defended by the production of dark inhibitory compounds. Photo by A. Dan Wilson.

Table 16.3—Symbiotic insect-wood decay fungi pest complexes that cause synergistic defect losses of hardwood lumber volume in southern and eastern hardwood species

Woodwasp	Fungal symbiont	Major tree hosts	Common names	References ^a
Siricidae				
<i>Eriotremex formosanus</i>	Basidiomycete	<i>Quercus phellos</i>	Willow oak	Unpublished data
<i>Tremex columba</i>	<i>Cerrena unicolor</i>	<i>Fagus grandifolia</i> <i>Celtis laevigata</i>	American beech Sugarberry	Stillwell 1964, 1965 Unpublished data
Xiphidriidae				
<i>Xiphydria abdominalis</i>	<i>Xylaria</i> sp.	<i>Tilia americana</i>	Basswood	Wilson and Schiff 2000b
<i>X. decem</i> ^b	<i>Xylaria</i> sp.	<i>Betula nigra</i>	River birch	Wilson and Schiff 2000b
<i>X. hicoriae</i>	<i>Daldinia</i> sp. ^c	<i>Carya ovata</i>	Shagbark hickory	Wilson and Schiff 2000b
<i>X. maculata</i>	<i>D. concentrica</i>	<i>Acer saccharum</i> <i>T. americana</i>	Sugar maple Basswood	Wilson and Schiff 2000b Wilson and Schiff 2000b
<i>X. mellipes</i>	<i>Daldinia</i> sp.	<i>B. papyrifera</i>	Paper birch	Unpublished data
<i>X. scafa</i>	<i>Xylaria</i> sp.	<i>Carpinus caroliniana</i>	American hornbeam	Wilson and Schiff 2000b
<i>X. tibialis</i>	<i>Xylaria</i> sp.	<i>A. saccharum</i> <i>C. caroliniana</i>	Sugar maple American hornbeam	Wilson and Schiff 2000b Wilson and Schiff 2000b

^a References refer to source(s) that reported the symbiotic association and/or new hosts of the woodwasp-wood decay fungus complex.

^b A new species (Smith and Schiff 2001).

^c The mycosymbiont of *Xiphydria hicoriae* was tentatively identified as a *Daldinia* species based on superficial observations of culture morphology of a single mycangial strain isolated from only one female woodwasp.

woodwasps known in North America, including *X. abdominalis* Say, *X. decem* Smith & Schiff, *X. hicoriae* Rohwer, *X. maculata* Say, *X. scafa* Smith, and *X. tibialis* Say (Smith 1976, 1979). The known wood decaying fungal symbionts of tremicine and xiphidriid woodwasps in southern and eastern hardwoods of the United States are summarized in table 16.3. All of these mycosymbionts are xylariaceous fungi (Ascomycotina: Xylariaceae), which are known for their ability to cause white rots in hardwood species. They are closely related to *Hypoxyylon* species that commonly attack and rapidly decay weakened hardwood trees. It was recently discovered that most of these mycosymbionts are *Xylaria* species not previously known to be symbionts with woodwasps (Wilson and Schiff 2000b). These fungi do not usually form the sexual stage in culture, which has hindered identification to species.

Xiphidriid woodwasps oviposit primarily into the axils of living hardwood branches, causing extensive decay and galleries in this area. This eventually weakens the limb which may then be broken by wind or ice accumulation. The decay can extend into the sapwood of the bole through the remaining branch stub after the limb falls off. Xiphidriid larvae continue to bore and develop in the fallen limb on the ground. Larvae produce galleries throughout the wood until the following

spring, pupate, and emerge as adults (Solomon 1995). Adults mate (optionally) and oviposit their eggs once again into the axils of living branches or into dead limbs on the ground to complete the cycle. Most woodwasp species seem to be fairly host-specific, often attacking only one or two hardwood species, although a few species such as *X. tibialis* have a number of hardwood hosts. There also appears to be high fidelity in the symbiotic association between woodwasp species and their fungal symbiont. All woodwasp species examined hitherto apparently depend only on a single mycosymbiont for food and cellulose decomposition.³

Woodwasp-wood decay fungi complexes have been found in every major hardwood species. Thus, these pest complexes are potentially significant sources of log defects in all hardwood stands. The occurrence of a new, nonnative siricid woodwasp [*Eriotremex formosanus* (Matsumura)] in hardwood forests of the Southern United States is of considerable concern because this pest has spread from Georgia to Texas since its introduction into the United States in infested wooden shipping crates brought back by the

³ Personal communication. 2002. A. Dan Wilson, Principal Research Pathologist, and Nathan Mark Schiff, Research Entomologist, Southern Research Station, P.O. Box 227, Stoneville, MS 38776.

military from Southeast Asia after the Vietnam War in the early 1970s (Smith 1996). Recent decay tests *in vitro* have demonstrated that the wood decay fungus vectored by this woodwasp has the potential to rapidly decay sapwood in many eastern hardwoods (Wilson and Schiff 2003). This fungus does not fruit readily on its oak hosts or *in vitro*. This makes identification difficult because the teleomorph or sexual stage contains key taxonomic characters required for identification. Perhaps these symbiotic fungi do not normally produce sexual fruiting bodies because they are regularly carried to appropriate tree hosts by their woodwasp vector and, therefore, do not have to expend energy to produce a metabolically costly fruiting body for sporulation and wind dispersal in order to survive. This is why the extensive damage caused by these pests often goes unnoticed until the tree is cut. These wood decay fungi are rarely visible on the outer surface of trees, and adult emergence holes of the woodwasps look similar to those of other hardwood borers. Consequently, the extent to which these pests are damaging southern oak forests is not known, although preliminary results with wood decay studies *in vitro* indicate that the mycosymbiont of *E. formosanus* and those of xiphydriid woodwasps can cause substantial white rots in eastern hardwoods after only 1 year (Wilson and Schiff 2003). We do not yet understand the importance of the role woodwasps play in the dispersal of wood decay fungi, the impact they have on forest health, or the effects that nonnative pests such as *E. formosanus* and its symbiont will have on lumber defect losses, hardwood timber salvage, and forest decomposition cycles. Further research is needed to elucidate the roles played by these new pest complexes that are invading our southern forests so that appropriate control strategies can be developed. These insect-disease pest complexes will likely receive increasing attention in the future as forest managers become more aware of their existence, their potential to cause damage, and their long-term impact on lumber production in hardwood forests.

MAJOR CONIFER DISEASES

The total land area in pine plantations now exceeds 25 million acres in the Southern United States (Belanger and others 2000). The area is expected to more than double by the year 2030. This valuable resource continues to expand primarily on private lands, which furnish the vast majority of timber products obtained from southern forests (U.S. Department of Agriculture,

Forest Service 1988). The majority of these plantations are more than 10 years of age. Loblolly pine (*Pinus taeda* L.) and slash pine (*P. elliottii* Engelm. var. *elliottii*) are the two most planted and economically important pine species in the South. The importance of southern forests and plantations as the major suppliers of renewable wood products in the United States continues to increase as production in Western States declines because of changing public land management policies which are placing less emphasis on forest commodity production in that region. The South is well suited to take on this role because of the rapid tree growth and production possible in southern forests, the wide diversity of wood products that can be produced, the responsiveness of southern pines to intensive culture and even-aged silvicultural systems, and the abundance of low-relief sites allowing fully mechanized harvesting (Blakeslee 1997).

Although relatively few pathogens have had major impacts on pine production in southern plantations and forests, the diseases caused by these pathogens have caused very significant losses in pulpwood and sawtimber production. Fortunately, there also have been significant advances within the past two decades in the development of management strategies to reduce losses to most major pine diseases. These advances have often taken into consideration changes in pathogen adaptations to suppression strategies, environmental conditions, host genetics, and legislative constraints on management alternatives.

Fusiform Rust

Fusiform rust continues to be recognized as the most damaging disease of southern pine forests and plantations. The causal agent, *Cronartium quercuum* (Berk.) Miyabe ex Shirai f. sp. *fusiforme* (Hedge. & N. Hunt) Burdsall & G. Snow, occurs in a broad band across the Southern States and is prevalent in the most productive high-quality loblolly and slash pine sites in this region (Anderson and others 1986). Fusiform rust incidence has increased dramatically within the last 30 years, especially in intensively cultured stands and in afforestation areas, where 47.9 million acres of former agricultural lands have been converted into pine stands and plantations (Starkey and others 1997). Annual losses to the disease have been estimated at \$35 million in five Southeastern States (Schmidt 1998). Forest managers throughout the South are concerned about this disease because it affects stocking,

product quantity, and product quality. Fusiform rust management in many areas is highly integrated into land management activities. For example, pine fertilization is frequently delayed by managers until after the trees are 5 years of age to reduce infection during the most vulnerable years (Blakeslee 1997).

The development of genetic resistance in planting stock has been the major disease management strategy used to reduce the incidence and severity of fusiform rust. The efforts of many forest pathologists over the past 40 years have brought genetic resistance to the forefront as an effective routine tool for managing this disease. The absence of a genetic linkage between rust resistance and tree growth rate has allowed the simultaneous development of genetically superior fast-growing trees with enhanced fusiform rust resistance. The genetic resistance approach generally has reduced pine mortality and disease severity in many sites, but some problems have been encountered as a consequence of the wide geographical variation in the genetics of the fungus, which has apparently given rise to strain-specific resistance, variations in pathogen virulence, and perhaps pathogen adaptations to host-resistance genes (Powers and Matthews 1979). Consequently, fungal strains in some areas eventually overcome resistance. Previously, pine breeders have attempted to stay ahead of the rust fungus by constantly producing and rotating new resistant pine growing stock to avoid genetic changes in the fungus that occur when pine selections are grown for too many rotations in the field. However, a new strategy involves the production of breeding lines that minimize rust damage, not prevent infection entirely, to avoid putting selection pressure on the fungus to produce new virulent strains, but maintain low-virulent strains to which pines are tolerant (Walkinshaw and Barnett 1995). Nevertheless, development of fusiform rust resistance has translated directly to increased economic value because the disease affects both the quantity and quality of timber produced (Cubbage and Wagner 2000).

Alternative approaches to fusiform rust suppression have been helpful in shaping efforts to develop integrated programs to manage this disease. The development of predictive models has been useful for identifying the relative hazard or susceptibility of sites to rust damage based on site and stand characteristics (Anderson and others 1986, Borders and Bailey 1986, Froelich and Snow 1986, Starkey and others 1997); and for predicting

preharvest rust-associated mortality (Devine and Clutter 1985, Geron and Hafley 1988). Other models have emphasized the importance of preventing rust during stand establishment (during the first 5 years) when the potential impact of rust infection is the greatest (Nance and others 1985). Triadimefon (Bayleton) seed treatments followed by protective foliar sprays have helped reduce the incidence of rust in the early stages of stand development (Hare and Snow 1983). The selective thinning of trees with moderate-to-severe stem girdling caused by rust galls is an effective means of reducing losses to fusiform rust and greatly improves the quality of trees in residual stands (Belanger and others 2000).

Recent research has utilized molecular techniques to study population structure, cellular, and biological aspects of the pathogen to determine genetic variation, identify the genetic mechanism of fungus-induced gall formation in pine hosts, locate rust-resistance genes in pine host genomes, and define cellular resistance responses (Covert and others 1977, Roberds and others 1997, Wilcox and others 1996). This information will ultimately be useful in developing new genetic engineering strategies for creating more resistant pines by taking advantage of new knowledge of host-pathogen interactions at the molecular level.

Pitch Canker

Pitch canker is a disease of pines caused by *Fusarium circinatum* Nirenberg et O'Donnell [= *F. subglutinans* (Wollenweber & Rienking) P. E. Nelson, Toussoun & Marasas f. sp. *pini* Correll and others]. The disease derives its name from the induction of copious pitch flow associated with cankers of pines. The classic symptom is a bleeding, resinous canker of the main stem or trunk, terminals, large branches, shoots, and exposed roots. The canker is usually sunken and the bark is retained, while the wood beneath the canker is deeply resin-soaked. Dieback in the crown results from cankers forming on the branches or shoots. As the branches or shoots are girdled by the fungus, the needles turn yellow to reddish brown; later they turn grayish brown to dark gray. It may take several years, however, for a canker to girdle the main stem. The pitch-soaked wood is a diagnostic character useful in separating pitch cankers from most other maladies of pines (Dwinell and others 1985). The symptoms of pitch canker frequently vary by pine host and management practices. In southern pines, trunk cankers are common on Virginia (*P. virginiana*

Mill.), longleaf (*P. palustris* Mill.), and eastern white (*P. strobus* L.) pines. Dieback is common on slash, loblolly, shortleaf (*P. echinata* Mill.), sand [*P. clausa* (Chapm. ex Engelm.) Vasey ex Sarg.], and pond (*P. serotina* Michx.) pines. Trunk cankers on slash pine are common in seed orchards and are usually associated with the use of tree shakers for cone removal. Cankers on exposed roots can be found on slash pine in seed orchards and other pines in landscape plantings (Dwinell and others 1985).

Pitch canker is an incomplete descriptive name for the range of damage caused by *F. circinatum*. The pathogen infects a variety of vegetative and reproductive pine tissues at different stages of maturity and produces a diversity of symptoms. Damage to pines by this fungus includes growth suppression, stem deformation, and tree mortality. The pitch canker fungus also causes mortality of female flowers and mature cones, and deteriorates seeds of several pine species. Dwinell and Fraedrich (1997) isolated *F. circinatum* from the surface and interior of immature shortleaf pine cones from a North Carolina seed orchard. They concluded that interior contamination by *F. circinatum* was not correlated with necrotic regions, caused primarily by insects, on the cone surface. The mode of entry of the pitch canker fungus into cones is unknown. Entire slash pine seedlots and entire longleaf pine seed-crops have been lost as a consequence of contamination by *F. circinatum*, which resulted in low seed viability and germination (Dwinell and others 1985). Current research is aimed at determining whether the pathogen is primarily on the seed surface or infects the embryo. Contamination of seed in longleaf and shortleaf pines is mostly on the seed surface (Dwinell and Fraedrich 1997, Fraedrich and Dwinell 1997). The fungus appears to be primarily external (Dwinell 1999). There is little empirical data linking seed contamination by *F. circinatum* with seedling cankers that occur in nursery beds and on outplanted sites. The major result of seed contamination by the pitch canker fungus is preemergence and postemergence damping-off (Dwinell 1999, Dwinell and Fraedrich 2000). In addition, pitch canker occurs in bare-root and container nurseries. Diseased pine seedlings show chlorotic or reddish brown needles and wilting. Pitch-soaked lesions usually occur at or near the soil line, but occasionally are found in the region of the cotyledonary node (Barnard and Blakeslee 1980). The pitch canker fungus has been associated with late-season mortality in longleaf pine nurseries (Carey and Kelley 1994).

Fraedrich and Dwinell (1997) concluded that *F. circinatum* is a wound pathogen of longleaf pine seedlings. Any fresh wound, regardless of cause or location, provides an infection court for the pathogen. Insects can create wounds that can be infected by airborne spores of the pathogen or serve as vectors. In the Southeastern United States, the deodar weevil (*Pissodes nemorensis* Germar) creates wounds that may become infected by airborne spores of the pathogen (Blakeslee and others 1978). Recent unpublished research indicates that the Nantucket pine tip moth [*Rhyacionia frustrana* (Comstock)] may not be associated with pitch canker in loblolly pine.⁴ In slash pine seed orchards, main stem cankers often develop after injury caused by mechanical cone harvesters. Also, injuries caused by wind and hail may serve as entry points. Hurricanes and tornadoes, in particular, have contributed to the intensification of the disease in some seed orchards (Dwinell and others 1985). The involvement of insects, interactions with other pine diseases, and numerous biotic and abiotic factors can influence the incidence and severity of infections by *F. circinatum*.

Annual mortality due to pitch canker in the Southeastern United States has been low. Southern pines, particularly loblolly, pond, and shortleaf pines, usually recover from outbreaks of shoot dieback (Barrows-Broadus and Dwinell 1985, Kuhlman and others 1982). From 1945 to 1973, limited outbreaks of pitch canker were noted in the Southeastern United States, but the disease was not considered to be economically important. In 1974, a shoot dieback identified as pitch canker reached epidemic proportions on slash pine in Florida plantations and seed orchards, and on loblolly pine in North Carolina and Mississippi seed orchards (Dwinell and others 1985). These outbreaks spawned considerable research on pitch canker. Over the last three decades, pitch canker outbreaks in the South have occurred sporadically in time and place. Pitch canker has also evolved from a regional problem to one of national and international importance (Dwinell 1999). Because each outbreak has its own unique history, no specific management strategy has been developed to reduce or eliminate the threat of pitch canker disease. An integrated management approach, including chemical control, biocontrol, genetic selection for resistance, and altered cultural

⁴ Personal communication. 2002. L. David Dwinell, Principal Research Pathologist (retired), Southern Research Station, 320 Green Street, Athens, GA 30602.

practices should be considered for specific hosts and growing conditions (Dwinell and others 1985). External contamination of pine seeds can be reduced or eliminated by appropriate seed treatments (Dwinell 1999, Dwinell and Fraedrich 2000). Because wounds serve as infection courts for *F. circinatum*, understanding the cause or causes of the wounding is tantamount to managing pitch canker (Dwinell and others 1985). In cases where the wounding agent is an insect, chemical control may reduce disease intensification. However, regulations on the use of chemical pesticides have severely limited this option. Biocontrol organisms have been ineffective (Barrows-Broadus and others 1985). Variation in the incidence of pitch canker is common among clones within seed orchards, suggesting that genetic selection for resistance is possible (Barrows-Broadus and Dwinell 1985, Dwinell and others 1985).

Annosus Root Disease

The fungal root pathogen *Heterobasidion annosum* (Fr.) Bref. is an economically important pest of temperate conifers worldwide and a powerful ecological force that can affect stand structure and composition. Currently, *H. annosum* S and P biological species in North America are genetically distinct entities, but have not yet been elevated to species status (Niemi and Korhonen 1998). Virtually no gene flow occurs between the North American S and P groups, despite their close proximity and overlapping host niches (Otrosina and others 1992, 1993). At this time, only the P biological species of *H. annosum* is known to occur east of the Mississippi River in the United States.

The most crucial stage in the disease cycle of this pathogen is the entry of the fungus into the stand through freshly cut stump surfaces. These cut surfaces provide a suitable niche in which airborne basidiospores can germinate and subsequently bring about mycelial colonization of the stump and root wood. Direct infection of roots through root wounds or possibly unwounded roots can occur in southern pines such as slash pine (Hendrix and Kuhlman 1964) and in *Abies* species (Garbelotto and others 1999). Once present in a stand, infection spreads from stumps to healthy trees via root contacts or grafts, creating ever-widening mortality centers. The fungus derives its nutrition from the enzymatic decomposition of woody tissues, particularly lignin and to a lesser extent cellulose, resulting in a physiological white rot. Thus, wood rotted by *H. annosum* has a

characteristic delaminated appearance, and later becomes lighter to almost white as the lignin is removed from cellulosic wood fibers. Infected trees are subject to windthrow as a result of these structural changes in decayed wood.

Roots infected by the fungus in living trees become highly resinous in advance of the invasion front containing active mycelia. Resin production is a physiological, host-defense response of the tree to invasion and may slow and sometimes contain the advance of the infection. The production of resinous compounds in response to infection is metabolically very costly in terms of expended energy and may result in the weakening of the tree over time. The expense of energy for host defense in response to extensive root infection by *H. annosum* predisposes conifers to attack by bark beetles and other root diseases (Alexander and others 1981, Schowalter and Filip 1993). The fungus can persist saprotrophically in the highly resinous stumps and stump roots in longleaf pine for at least 7 years after thinning, providing inoculum potential to infect healthy residual trees via root grafts and contacts (Otrosina and others 2002). Mortality is a dramatic effect of *H. annosum* root disease, but growth reduction usually results from sublethal infections. Because root disease infection in trees is invisible until very advanced stages, considerable growth increment loss can occur in affected stands without significant mortality (Alexander 1989, Alexander and others 1981). On the other hand, slash and loblolly pines may be able to sustain considerable root infection before growth reduction occurs (Bradford and others 1978, Froelich and others 1977).

Considerable research has been done regarding risk assessment with respect to *H. annosum* root disease in the Southeastern United States. Edaphic factors are important elements associated with occurrence and hazard associated with this disease. Sites classified as high risk have well-drained soils containing sand, low organic matter, and low water table (Alexander 1989). These edaphic risk factors have been used to develop hazard-rating maps (Anon. 1999). While these maps provide correlations between certain soil types and *H. annosum* root disease, there is little information available to explain why or how soil factors affect disease development. Soil type affects factors such as water stress, microbial activity, aeration, and root habit, and root configuration can affect the root-infection processes.

Control of *H. annosum* root disease is achieved primarily through prevention. The most effective means to date is the prophylactic application of powdered borax formulations to freshly cut stump surfaces. Borax is toxic to basidiospores and conidia of *H. annosum* (Hodges 1970). Prevention and control is achieved only if borax applications are timely, ideally within a few hours after tree cutting. Technology that automates application of powdered borax by devices that attach to feller buncher equipment is now under development (Karsky 1999). Another avenue for *H. annosum* root disease control is through silvicultural management. Research by Ross (1973) revealed that thermal inactivation of basidiospores is achieved when stump surfaces reach $> 35^{\circ}\text{C}$, resulting in no stump colonization. These temperatures are common during the summer months south of 34°N . latitude and form the basis for the recommendation that southern pine stands south of this latitude be thinned in the summer. On the other hand, high temperature may not be the sole factor responsible for lowering rates of stump infection. Some research suggests that microbial synergy at the stump surface may be affected by high temperatures on stump surfaces, since the fungus could be reisolated from surface sterilized and inoculated wood bolts at temperatures up to 40°C (Gooding 1964).

Less emphasis has been given to *H. annosum* root disease control in recent years. Preventive measures such as application of borax after thinning are becoming less common. While some data suggest that mortality of trees planted in severely infested sites is minimal up to 22 years after planting (Kuhlman 1986), multiple stand entries and thinning without the proper preventive measures, combined with longer rotation lengths, will increase the importance of this disease in coniferous forests in the Southern United States. Such a scenario exists in certain longleaf pine stands where *H. annosum* root disease results in significant and steady mortality beginning when trees are about 40 years of age. Longleaf pine has been regarded as highly tolerant to this disease, but various factors such as degraded soils, root damage by equipment, and lengthened prescribed fire regimes have resulted in increased mortality due to *H. annosum* and other root pathogens in these stands. Thus, long-term goals of managing longleaf pine on a 75- to 120-year rotation for red-cockaded woodpecker [*Picoides borealis* (Vieillot)] habitat, stand restoration, and seed production can be thwarted if appropriate caution is not exercised

regarding root disease (Otrosina and others 1999, 2002). More comprehensive information on *H. annosum* root disease in North America and Europe is presented in Otrosina and Scharpf (1989) and Woodward and others (1998).

ACKNOWLEDGMENTS

The authors thank Evan Nebeker, Frank Tainter, and Steve Meadows for reviewing the manuscript.

LITERATURE CITED

- Anon. 1999. Southern forest health atlas of insects and diseases. R8-MR 35. Atlanta: U.S. Department of Agriculture, Forest Service, Southern Region, Forest Health Protection. 36 p.
- Alexander, S.A. 1989. Annosus root disease hazard rating, detection, and management strategies in the Southeastern United States. In: Otrosina, W.J.; Scharpf, R.F., tech. coords. Proceedings of the symposium on research and management of annosus root disease (*Heterobasidion annosum*) in Western North America. Gen. Tech. Rep. PSW-116. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwestern Research Station: 111-116.
- Alexander, S.A.; Skelly, J.M.; Webb, R.S. 1981. Effects of *Heterobasidion annosum* on radial growth in southern pine beetle-infested loblolly pine. *Phytopathology*. 71: 479-481.
- Ammon, V.; Nebeker, T.E.; Filer, T.H. [and others]. 1989. Oak decline. *Miss. Agric. For. Exper. Stn. Bull.* 161. Mississippi State, MS: Mississippi State University. [Not paged].
- Anderson, R.L.; Cost, N.D.; McClure, J.P.; Ryan, G. 1986. Predicting severity of fusiform rust in young loblolly and slash pine stands in Florida, Georgia, and the Carolinas. *Southern Journal of Applied Forestry*. 10: 38-41.
- Barnard, P.E.; Blakeslee, G.M. 1980. Pitch canker of slash pine seedlings: a new disease in forest nurseries. *Plant Disease*. 64: 695-696.
- Barrows-Broadus, J.B.; Dwinell, L.D. 1985. Branch dieback and cone and seed infection caused by *Fusarium moniliforme* var. *subglutinans* in a loblolly pine seed orchard in South Carolina. *Phytopathology*. 75: 1104-1108.
- Barrows-Broadus, J.B.; Dwinell, L.D.; Kerr, T.J. 1985. Evaluation of *Arthrobacter* strains: a biocontrol of the pitch canker fungus (*Fusarium moniliforme* var. *subglutinans*) on slash pines. *Canadian Journal of Microbiology*. 29: 1382-1389.
- Belanger, R.P.; Miller, T.; Zarnoch, S.J. [and others]. 2000. An integrated approach toward reducing losses from fusiform rust in merchantable slash and loblolly pine plantations. Res. Pap. SRS-23. Asheville, NC: U.S. Department of Agriculture, Forest Service, Southern Research Station. 14 p.
- Billings, R.F. 2000. State forest health programs: a survey of State foresters. *Journal of Forestry*. 98: 20-25.
- Billings, R.F.; Gehring, E.H.; Cameron, R.S.; Gunter, J.T. 2001. Current practices in managing oak wilt: Federal cost share programs, trenching, chemical injection, and the Texas suppression program. In: Ash, Cynthia L., ed. Shade tree wilt diseases: Proceedings from a national conference. St. Paul, MN: APS Press: 117-129.

- Blakeslee, G.M. 1997. Diseases in the forest: Southern United States. In: Hansen, Everett M.; Lewis, Katherine J., eds. Compendium of conifer diseases. St. Paul, MN: APS Press: 82–83.
- Blakeslee, G.M.; Oak, S.W.; Gregory, W.; Moses, C.S. 1978. Natural association of *Fusarium moniliforme* var. *subglutinans* with *Pissodes nemorensis* [Abstract]. Phytopathology News. 12: 208.
- Borders, B.E.; Bailey, R.L. 1986. Fusiform rust prediction models for site-prepared slash and loblolly pine plantations in the Southeast. Southern Journal of Applied Forestry. 10: 145–151.
- Bradford, B.; Alexander, S.A.; Skelly, J.M. 1978. Determination of growth loss of *Pinus taeda* L. caused by *Heterobasidion annosum*. (Fr.) Bref. European Journal of Forest Pathology. 8: 129–134.
- Britton, K.O.; Leininger, T.; Chang, C.J.; Harrington, T.C. 1998. Association of *Xylella fastidiosa*, *Ceratocystis fimbriata platani*, and *Botryosphaeria rhodina* with declining sycamore plantations in the Southeastern United States. In: Proceedings of the seventh international congress of plant pathology. Edinburgh, Scotland: [Publisher unknown]. 1 p. Vol. 3.
- Carey, W.A.; Kelley, W.D. 1994. First report of *Fusarium subglutinans* as a cause of late-season mortality in longleaf pine nurseries. Plant Disease. 78: 754.
- Chang, C.J.; Leininger, T.D.; Britton, K.O. 2002. Screening for sycamores that may be tolerant to leaf scorch disease caused by *Xylella fastidiosa*. Phytopathology. 92: S13.
- Covert, S.F.; Warren, J.; Zwart, A. 1977. Differential gene transcription in galled and asymptomatic tissues of loblolly pine infected with *Cronartium quercuum* f. sp. *fusiforme*. In: TAPPI proceedings: biological sciences symposium. Norcross, GA: TAPPI Press: 93–98.
- Cubbage, F.W.; Wagner, J.E. 2000. An economic evaluation of fusiform rust protection research. Southern Journal of Applied Forestry. 24: 77–85.
- Devine, O.J.; Clutter, J.L. 1985. Prediction of survival in slash pine plantations infected with fusiform rust. Forest Science. 31: 88–94.
- Dewers, R.S. 1971. Shade tree evaluation. Fact Sheet L–958. [Place of publication unknown]: Texas Agricultural Extension Service. 1 p.
- Dwinell, L.D. 1999. Association of the pitch canker fungus with cones and seeds of pines. In: Devey, M.E.; Matheson, A.C.; Gordon, T.R., eds. Current and potential impacts of pitch canker in radiata pine: Proceedings of IMPACT Monterey workshop. Collingwood, Victoria, Australia: Commonwealth Scientific and Industrial Research Organization Publishing: 35–39.
- Dwinell, L.D.; Barrows-Broadus, J.; Kuhlman, E.G. 1985. Pitch canker: a disease complex of southern pines. Plant Disease. 69: 270–276.
- Dwinell, L.D.; Fraedrich, S.W. 1997. Recovery of *Fusarium subglutinans* f. sp. *pini* from shortleaf pine cones and seeds. In: James, R., ed. Proceedings of the diseases and insects in forest nurseries IUFRO working party conference. [Place of publication unknown]: [Publisher unknown]: 42–47.
- Dwinell, L.D.; Fraedrich, S.W. 2000. Contamination of pine seeds by *Fusarium circinatum*. In: Lilja, A.; Sutherland, J., eds. Diseases and insects in forest nurseries. Proceedings of 4th meeting of IUFRO Working Party 7.03.04. [Place of publication unknown]: [Publisher unknown]: 75–82.
- Filer, T.H., Jr.; Cooper, D.T.; Collins, R.J.; Wolfe, R. 1975. Survey of sycamore plantations for canker, leaf scorch, and dieback. Plant Disease Reporter. 59: 152–153.
- Filer, T.H., Jr.; Cordell, C.E. 1983. Nursery diseases of southern hardwoods. For. Insect and Dis. Leaflet. 137. New Orleans: U.S. Department of Agriculture, Forest Service, Southern Forest Experiment Station. 6 p.
- Fraedrich, S.W.; Dwinell, L.D. 1997. Mortality of longleaf pine seedlings caused by *Fusarium subglutinans* and an evaluation of potential inoculum sources. In: James, R., ed. Proceedings of the diseases and insects in forest nurseries IUFRO working party conference. [Place of publication unknown]: [Publisher unknown]: 48–54.
- Froelich, R.C.; Cowling, E.B.; Collicott, L.V.; Dell, T.R. 1977. *Fomes annosus* reduces height and diameter growth of planted slash pine. Forest Science. 23: 299–306.
- Froelich, R.C.; Snow, G.A. 1986. Predicting site hazard to fusiform rust. Forest Science. 32: 21–35.
- Garbelotto, M.; Cobb, F.W.; Bruns, T.D. [and others]. 1999. Genetic structure of *Heterobasidion annosum* in white fir mortality centers in California. Phytopathology. 89: 546–554.
- Geron, C.D.; Hafley, W.L. 1988. Impact of fusiform rust on product yield of loblolly pine. Southern Journal of Applied Forestry. 12: 226–231.
- Gilbertson, R.L. 1984. Relationships between insects and wood-rotting basidiomycetes. In: Wheeler, Q.; Blackwell, M., eds. Fungus-insect relationships. Perspectives in ecology and evolution. New York: Columbia University Press: 130–165. Chapter 6.
- Gooding, G.W. 1964. Effect of temperature on growth and survival of *Fomes annosus* in freshly cut pine bolts. Phytopathology. 54: 893–894.
- Hare, R.C.; Snow, G.A. 1983. Control of fusiform rust in slash pine with bayleton (triadimefon) seed treatment. Res. Note SO–288. New Orleans: U.S. Department of Agriculture, Forest Service, Southern Forest Experiment Station. 4 p.
- Hendrix, F.F.; Kuhlman, E.G. 1964. Root infection of *Pinus elliotii* by *Fomes annosus*. Nature. 201: 555–556.
- Himelick, E.B.; Fox, H.W. 1961. Experimental studies on control of oak wilt disease. Illinois Agric. Exper. Stn. Tech. Bull. 680. [Place of publication unknown]: [Publisher unknown]: [Number of pages unknown].
- Hodges, C.S. 1970. Evaluation of stump treatment chemicals for control of *Fomes annosus*. In: Hodges, C.S.; Rishbeth, J.; Yde-Andersen, A., eds. Proceedings of the third international conference on *Fomes annosus*. Washington, DC: U.S. Department of Agriculture, Forest Service: 43–53.
- Karsky, D. 1999. Dry borax applicator: operators manual. Tech Rep. 9934–2812–MTDC. Missoula, MT: U.S. Department of Agriculture, Forest Service, Missoula Technology and Development Center. 30 p.
- Kuhlman, E.G. 1986. Impact of annosus root rot minimal 22 years after planting pines on root rot infested sites. Southern Journal of Applied Forestry. 10: 96–98.

- Kuhlman, E.G.; Dianis, S.D.; Smith, T.K. 1982. Epidemiology of pitch canker disease in a loblolly pine seed orchard. *Phytopathology*. 72: 1212–1216.
- Kukor, J.J.; Martin, M.M. 1983. Acquisition of digestive enzymes by siricid woodwasps from their fungal symbiont. *Science*. 220: 1161–1163.
- Leininger, T.D.; Solomon, J.D.; Wilson, A.D.; Schiff, N.M. 1999. A guide to major insects, diseases, air pollution injury, and chemical injury of sycamore. Gen. Tech. Rep. SRS–28. Asheville, NC: U.S. Department of Agriculture, Forest Service, Southern Research Station. 44 p.
- Manion, P.D.; Lachance, D. 1992. Forest decline concepts: an overview. In: Manion, P.D.; Lachance, D., eds. *Forest decline concepts*. St. Paul, MN: American Phytopathological Society: 181–190.
- McGarity, R.W. 1976. Sycamore disease incidence survey. Unnumbered Intern. Rep. Asheville, NC: U.S. Department of Agriculture, Forest Service, Hardwood Management Research. 9 p.
- Morris, R.C.; Filer, T.H.; Solomon, J.D. [and others]. 1975. Insects and diseases of cottonwood. Gen. Tech. Rep. SO–8. New Orleans: U.S. Department of Agriculture, Forest Service, Southern Forest Experiment Station. 37 p.
- Nance, W.L.; Shoulders, E.; Dell, T.R. 1985. Predicting survival and yield of unthinned slash and loblolly pine plantations with different levels of fusiform rust. In: Branham, S.J.; Thatcher, R.C., eds. *Proceedings of the integrated pest management research symposium*. Gen. Tech. Rep. SO–56. New Orleans: U.S. Department of Agriculture, Forest Service, Southern Forest Experiment Station: 62–72.
- National Assessment Synthesis Team. 2000. *Climate change impacts on the United States: the potential consequences of climate variability and change*. Washington, DC: U.S. Global Change Research Program. 154 p.
- Niemla, T.; Korhonen, K. 1998. Taxonomy of the genus *Heterobasidion*. In: Woodward, S.; Stenlid, J.; Karjalainen, R.; Hüttermann, A., eds. *Heterobasidion annosum: biology, ecology, impact and control*. New York: CAB International: 27–33.
- Ning, Z.H.; Abdollahi, K.K. 1999. Global climate change and its consequences on the gulf coast region of the United States. Baton Rouge, LA: Gulf Coast Regional Climate Change Council. 98 p.
- Otrosina, W.J.; Bannwart, D.; Roncadori, R.W. 1999. Root-infecting fungi associated with a decline of longleaf pine in the Southeastern United States. *Plant & Soil*. 217: 145–150.
- Otrosina, W.J.; Chase, T.E.; Cobb, F.W. 1992. Allozyme differentiation of intersterility groups of *Heterobasidion annosum* isolated from conifers in the Western United States. *Phytopathology*. 82: 540–545.
- Otrosina, W.J.; Chase, T.E.; Cobb, F.W.; Korhonen, K. 1993. Population structure of *Heterobasidion annosum* from North America and Europe. *Canadian Journal of Botany*. 71: 1064–1071.
- Otrosina, W.J.; Scharpf, R.F. 1989. *Proceedings of the symposium on research and management of annosus root disease (Heterobasidion annosum) in Western North America*. Gen. Tech. Rep. PSW–16. Berkeley, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Forest and Range Experiment Station. 177 p.
- Otrosina, W.J.; Walkinshaw, C.H.; Zarnoch, S.J. [and others]. 2002. Root disease, longleaf pine mortality, and prescribed burning. In: Outcalt, Kenneth W., ed. *Proceedings of the eleventh biennial southern silvicultural research conference*. Gen. Tech. Rep. SRS–48. Asheville, NC: U.S. Department of Agriculture, Forest Service, Southern Research Station: 551–557.
- Powers, H.H., Jr.; Matthews, F.R. 1979. Interactions between virulent isolates of *Cronartium quercuum* f. sp. *fusiforme* and loblolly pine families of varying resistance. *Phytopathology*. 69: 720–722.
- Roberds, J.H.; Kubisiak, T.L.; Spaine, P.C. [and others]. 1997. Selection of RAPD markers for investigation of genetic population structure in fusiform rust fungus infecting loblolly pine. In: *Proceedings of the 24th biennial southern forest tree improvement conference*. [Place of publication unknown]: [Publisher unknown]: 293–298.
- Ross, E.W. 1973. *Fomes annosus* in Southeastern United States: relation of environmental and biotic factors to stump colonization and losses in the residual stand. *Tech. Bull.* 1459. Asheville, NC: U.S. Department of Agriculture, Forest Service, Southeastern Forest Experiment Station. 26 p.
- Schmidt, R.A. 1998. Fusiform rust disease of southern pines: biology, ecology, and management. [Place of publication unknown]: University of Florida, Agricultural Experiment Station. 14 p.
- Schowalter, T.D.; Filip, G.M. 1993. *Beetle-pathogen interactions in conifer forests*. London: Academic Press, Harcourt Brace & Co. 252 p.
- Sherald, J.L.; Kostka, S.J. 1992. Bacterial leaf scorch of landscape trees caused by *Xylella fastidiosa*. *Journal of Arboriculture*. 18: 57–63.
- Smith, D.R. 1976. The xiphydriid woodwasps of North America (Hymenoptera: Xiphydriidae). *Transactions of the American Entomological Society*. 102: 101–131.
- Smith, D.R. 1978. Suborder Symphyta (Xyelidae, Pararchxyelidae, Parapamphiliidae, Xyelydidae, Karatavidae, Gigasiricidae, Sepulcidae, Pseudosiricidae, Anaxyelidae, Siricidae, Xiphydriidae, Paroryssidae, Xyelotomidae, Blasticotomidae, Pergidae). In: van der Vecht, J.; Shenefelt, R.D., eds. *Hymenopterorum catalogus. Pars 12*. Holland: Dr. W. Junk B.V., The Hague. 193 p.
- Smith, D.R. 1979. Suborder Symphyta, In: Krombein, K.V. [and others], eds. *Catalog of Hymenoptera in America north of Mexico*. Washington, DC: Smithsonian Institution Press: 3–137. Vol. 1.
- Smith, D.R. 1996. Discovery and spread of the Asian horntail, *Eriotremex formosanus* (Matsumura) (Hymenoptera: Siricidae) in the United States. *Journal of Entomological Science*. 31: 166–171.
- Smith, D.R.; Schiff, N.M. 2001. A new species of *Xiphydria* Latreille (Hymenoptera: Xiphydriidae) reared from river birch, *Betula nigra* L., in North America. *Proceedings of the Entomological Society of Washington*. 103: 962–967.
- Smith, D.R.; Schiff, N.M. 2002. Review of the siricid woodwasps and their ibaliid parasitoids (Hymenoptera: Siricidae, Ibaliidae) in the Eastern United States, with emphasis on the mid-Atlantic region. *Proceedings of the Entomological Society of Washington*. 104: 174–194.
- Smyly, W.B.; Filer, T.H., Jr. 1977. *Cylindrocladium scoparium* associated with root rot and mortality of cherrybark oak seedlings. *Plant Disease Reporter*. 61: 577–579.

- Solomon, J.D. 1995. Guide to insect borers of North America broadleaf trees and shrubs. Agric. Handb. 706. Washington, DC: U.S. Department of Agriculture, Forest Service. 735 p.
- Solomon, J.D.; Leininger, T.D.; Wilson, A.D. [and others]. 1993. Ash pests: a guide to major insects, diseases, air pollution injury, and chemical injury. Gen. Tech. Rep. SO-96. New Orleans: U.S. Department of Agriculture, Forest Service, Southern Forest Experiment Station. 45 p.
- Solomon, J.D.; McCracken, F.I.; Anderson, R.L. [and others]. 1997. Oak pests: a guide to major insects, diseases, air pollution and chemical injury. Prot. Rep. R8-PR7. Atlanta: U.S. Department of Agriculture, Forest Service, Southern Region; Asheville, NC: U.S. Department of Agriculture, Forest Service, Southern Research Station. 69 p.
- Stanturf, J.A.; Gardiner, E.S.; Hamel, P.B. [and others]. 2000. Restoring bottomland hardwood ecosystems in the Lower Mississippi Alluvial Valley. *Journal of Forestry*. 98: 10-16.
- Starkey, D.A.; Anderson, R.L.; Young, C.H. [and others]. 1997. Monitoring incidence of fusiform rust in the South and change over time. Prot. Rep. R8-PR 30. Atlanta: U.S. Department of Agriculture, Forest Service, Southern Region. 29 p.
- Stillwell, M.A. 1964. The fungus associated with woodwasps occurring on beech in New Brunswick. *Canadian Journal of Botany*. 42: 495-496.
- Stillwell, M.A. 1965. Hypopleural organs of the woodwasp larva *Tremex columba* (L.) containing the fungus *Daedalea unicolor* Bull. ex Fries. *Canadian Entomology*. 97: 783-784.
- Toole, E.R. 1959. Decay after fire injury to southern bottomland hardwoods. Tech. Bull. 1189. Washington, DC: U.S. Government Printing Office. 25 p.
- Toole, E.R. 1960. Butt rot of southern hardwoods. For. Pest Leaflet 43. Washington, DC: U.S. Government Printing Office. 4 p.
- U.S. Department of Agriculture, Forest Service. 1988. The South's fourth forest: alternatives for the future. For. Res. Rep. 24. Washington, DC. 512 p.
- U.S. Department of Agriculture, Forest Service. 2002. Forest insect and disease conditions in the United States 2000. Washington, DC: Forest Health Protection. 100 p.
- Walkinshaw, C.H.; Barnett, J.P. 1995. Tolerance of loblolly pines to fusiform rust. *Southern Journal of Applied Forestry*. 19: 60-64.
- Wilcox, P.L.; Amerson, H.V.; Sederoff, R.R. [and others]. 1996. Detection of a major gene for resistance in fusiform rust disease in loblolly pine by genomic mapping. *Proceedings of the National Academy of Sciences*. 93: 3859-3864.
- Wilson, A.D. 2001. Oak wilt: a potential threat to southern and western oak forests. *Journal of Forestry*. 99: 4-11.
- Wilson, A.D.; Forse, L.B. 1997. Sensitivity of Texas strains of *Ceratocystis fagacearum* to triazole fungicides. *Mycologia*. 89: 468-480.
- Wilson, A.D.; Lester, D.G. 1995. Application of propiconazole and *Pseudomonas cichorii* for control of oak wilt in Texas live oaks. *Fungicide and Nematicide Tests*. 50: 393.
- Wilson, A.D.; Lester, D.G. 1997. Use of an electronic-nose device for profiling headspace volatile metabolites to rapidly identify phytopathogenic microbes. *Phytopathology*. 87: S116.
- Wilson, A.D.; Lester, D.G. 2002. Trench inserts provide long-term barriers to root transmission for control of oak wilt. *Plant Disease*. 86: 1067-1074.
- Wilson, A.D.; Schiff, N.M. 2000a. Somatic antagonism between fungal symbionts of xiphydriid woodwasps. *Phytopathology*. 90: S84.
- Wilson, A.D.; Schiff, N.M. 2000b. Xylariaceous wood decay fungi: mycosymbionts of xiphydriid woodwasps. *Phytopathology*. 90: S84.
- Wilson, A.D.; Schiff, N.M. 2003. Wood decay potential of mycosymbionts from xiphydriid and tremicine woodwasps. *Phytopathology*. 93: S90.
- Woodward, S.; Stenlid, J.; Karjalainen, R.; Hutterman, A. 1998. *Heterobasidion annosum*: biology, ecology, impact, and control. New York: CAB International. 589 p.