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Challenges and Successes in Managing Oak Wilt in the United States

Oak wilt, caused by the fungus *Ceratocystis fagacearum* (Bretz) J. Hunt, is an important disease of oaks (*Quercus* spp.) in the eastern United States. It has been particularly destructive in the North Central states and Texas. Oak wilt is one of several significant oak diseases that threaten oak health worldwide. The significant gains made in our knowledge of the biology and epidemiology of this vascular wilt disease during the past six decades has led to development of various management strategies.

Interest in oak wilt research and management has "waxed and waned" since the pathogen was initially discovered in the early 1940s (61). This ambivalence, accompanied by emphasis on newly emerging oak diseases such as sudden oak death (107) and *Raffaelea*-caused wilt of oaks in Japan and Korea (82,83), could have very costly consequences. Today, *C. fagacearum* remains a deadly pathogen, particularly of red and live oak species, and the fungus has demonstrated the capacity to impact a variety of ecosystems with disastrous results (6). The spread of the disease into new regions has been rather slow, but periodic and localized outbreaks frequently occur within the established range. Disease intensification is currently of particular concern in Michigan (J. O'Brien, *personal communication*). Oak wilt has proven to be a manageable disease in the relatively few areas where consistent and long-term disease suppression programs occur. Successful disease control depends on early diagnosis of the problem followed by creative use of the available management tools under variable circumstances. These seemingly simple steps have proven challenging to implement and will require continual educational efforts. The goal of this article is to illustrate the complex relationship between *Quercus* and *C. fagacearum* as well as to describe how these relationships influence our ability, or inability, to use various management tools to minimize future losses of the very important oak resource.

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Taxonomy, Occurrence, and Significance of Oaks

Quercus (Family Fagaceae), commonly referred to as oaks, is a large genus of trees and shrubs, containing over 400 species worldwide (67). Relative to the expansive worldwide distribution of oaks, oak wilt is known to occur only in part of its potential range in the United States. Further, *C. fagacearum* is pathogenic only to certain groups within the large variety of oak species. With the exception of *Lithocarpus*, differences in the fruit (acorns) of *Quercus* spp. serve to distinguish the oaks from other taxa in the Fagaceae (the beech family) (67). Taxonomically, *Quercus* currently is divided into four sections: Section *Cerris* with species in Asia, Europe, and the Mediterranean; Section *Lobatae*, or red oaks, found only in the Americas; Section *Quercus*, or white oaks, with species in the Americas, Asia, and Europe; and Section *Protobalanus*, or intermediate oaks, with only five species that are limited to the southwestern United States and northwestern Mexico (98). As will be seen in the discussion on hosts, there is variability in susceptibility of species among these taxonomic sections with important consequences for the epidemiology, impact, and management of the disease. The largest numbers of species occur in Mexico and Central America and the smallest in Canada. Within the United States, 58 native *Quercus* spp. plus 9 varieties are found (88). Ecologically, oaks occur largely in naturally regenerated forests in mesic (extremely wet and humid to moist upland forests) to xeric (dry) regions. Oaks are major components of three forest cover types in the eastern United States (oak-hickory, oak-pine, oak-gum-cypress) that collectively occupy 83.8 million ha (207 million acres) (116).

Economically, oaks are the most important group in the family Fagaceae in the Americas, with more than 50 species having commercial value in North America (67). Oaks provide a variety of timber and nontimber products and ecosystem services. They include high-quality lumber for furniture and flooring, tannins and dyes for leather and clothing, high-value urban and community landscape trees, and habitat and mast (food) for wildlife species (67). The potential impact of *C. fagacearum* and great economic value of the oaks have motivated respect for the potential, destructive impact of the oak wilt and the perceived need for better understanding the pathogen.

Geographic Distribution

Oak wilt is only known to exist in the eastern and midwestern states and Texas of the United States (Fig. 1). This range is limited, considering the presence of susceptible hosts and apparently suitable climate conditions in parts of the western United States, especially in California (5), and as yet nonaffected areas of the northeastern and the southeastern regions. It is possible that *C. fagacearum* is exotic to the United States, with possible origins in Central or South America, or Mexico (76). Surveys conducted during the two decades following the pathogen's discovery found numbers of affected counties and states steadily increasing over time east-wide (24,106). This trend was likely a result of increasing recognition of existing oak wilt rather than an expansion in the range of *C. fagacearum* (89). The historical records of the pathogen and the disease in Texas support the view that oak wilt may have been present throughout much of the current range in the United States at the time it was discovered (76). The identification of the disease was delayed in Texas because symptoms on live oak (*Q. virginiana* Mill. and *Q. fusiformis* Small) deviated from those known at the time for other oaks, but there are reliable indicators that oak wilt was present long before it was originally described there in 1961 (6). The presence of *C. fagacearum* was first documented in Wisconsin in 1942 (4); however, shortly thereafter, oak wilt was confirmed in 23 counties of Wisconsin, 5 in Minnesota, 2 in Iowa, and 1 in Illinois (61). The oak wilt fungus is currently considered to be present in 829 counties of 24 states (Fig. 1) based on a U.S. Forest Service database (Q. Chavez and J. Pokorny, *personal communication*). However, records of oak wilt occurrence in some counties are very old, and the lack of more recent detections suggests that the disease may no longer be present in some previously "positive" counties in Arkansas and Oklahoma (D. Starkey, *personal communication*), Minnesota (S. Burks, *unpublished data*), Missouri (B. Moltzan, *personal communication*), and North Carolina (R. Blaedow, *personal communication*).

Nonetheless, new reports of *C. fagacearum* in counties or states outside the previously known range sporadically occur. The first report of oak wilt in New York (Schenectady Co.) in 2008 represents a northeastern extension of the previously known range of *C. fagacearum* by approximately 300 km (187 mi) (66). Since 2006, two additional counties in South Carolina have been reported with oak wilt, which represents expansion of the southeastern portion of the disease range (USDA Forest Service, Pest Event Database, as provided by D. Starkey, Region 8). Similarly, at the southwestern edge of the oak wilt range, three new county records were recorded in 2007 through 2009 for Texas, bringing the total number of affected counties to 73.

Hosts

A diverse population of oak species exists in the many states where oak wilt occurs. The susceptibility of many of these species (greater than 33) is known as a result of artificial inoculation studies or through natural infection (113). A broad generalization is that members of the red oak group (e.g., *Q. rubra*, *Q. velutina*, *Q. falcata*, *Q. shumardii*) are highly susceptible compared to members

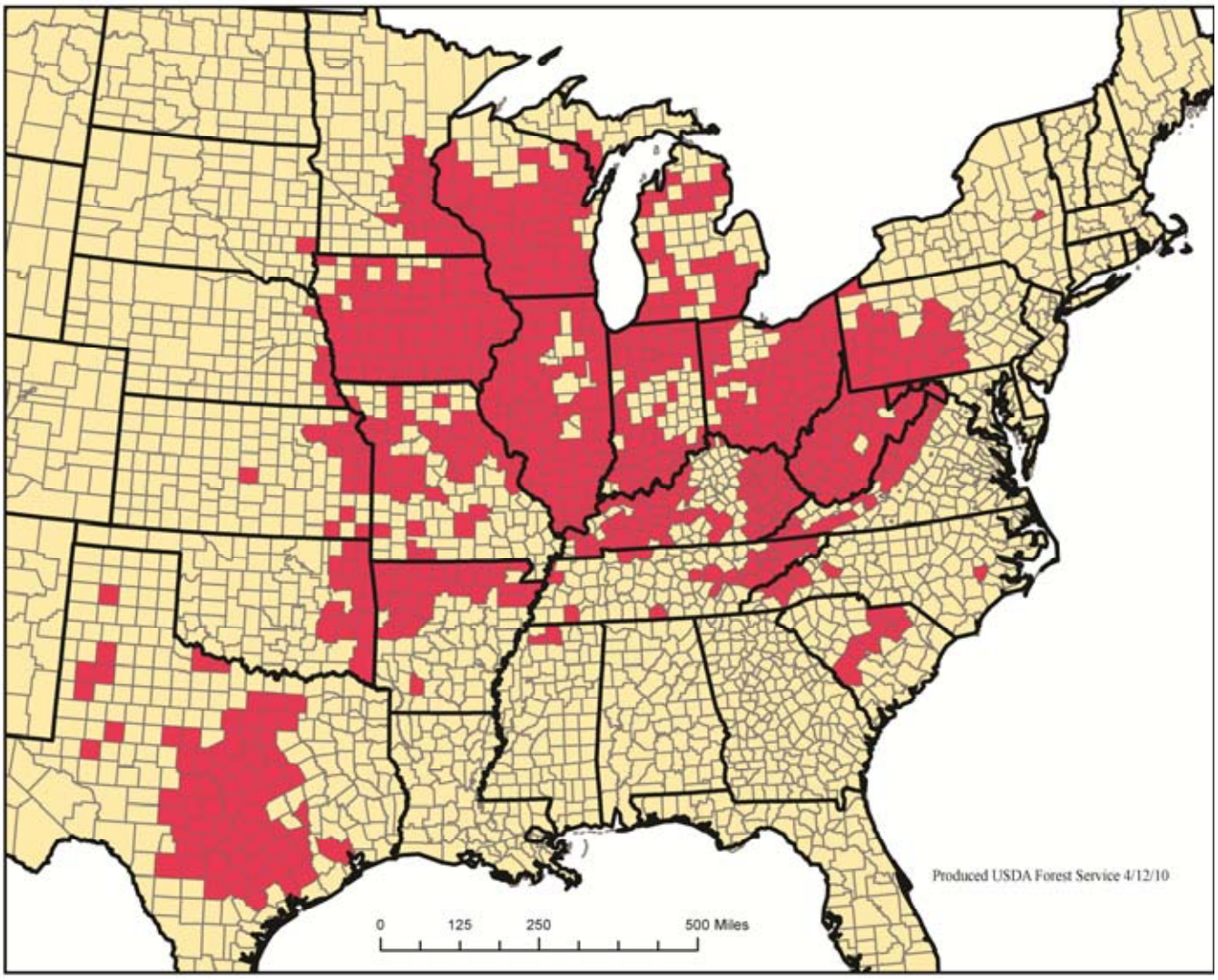

Fig. 1. Oak wilt distribution in the United States, 2009. Source: Q. Chavez and J. Pokorny, Northeastern Area State and Private Forestry, U.S. Forest Service.

of the white oak section, which displays moderate-to-high levels of resistance. Natural infection of the "highly resistant" white oak (*Q. alba*) by *C. fagacearum* commonly results in dieback of one or more branches, but such trees may not die from the disease for decades if at all (73). Other white oak species (e.g., *Q. macrocarpa*, *Q. fusiformis*, and *Q. virginiana*) may exhibit moderate resistance, with tree mortality occurring several years following infection (100). The experience with oak wilt in Texas has demonstrated all too well the susceptibility of live oaks to infection. Although symptoms develop more slowly in Texas live oaks than in species of red oak, the outcome generally is mortality or severe crown loss. The susceptibility of other live oak species growing in areas of North America where oak wilt does not occur is unknown. Interestingly, the resistance of white oaks to oak wilt did not prove to be the case when European species were artificially inoculated with *C. fagacearum* (91). European white oaks (*Q. robur* and *Q. petraea*) were found to be as susceptible as North American red oak species in field trials conducted in South Carolina and West Virginia. As a result, regulations governing the export of oak logs and lumber are in place to minimize the risk of international movement of *C. fagacearum* (92).

The differences in susceptibility of species between the red and white oak groups has generally been attributed to differences in anatomical features (e.g., diameter of xylem vessels, tylose formation) (65,108,112) and physiological responses of the host (e.g., embolisms, reduced transpiration) to infection by the pathogen (14,110).

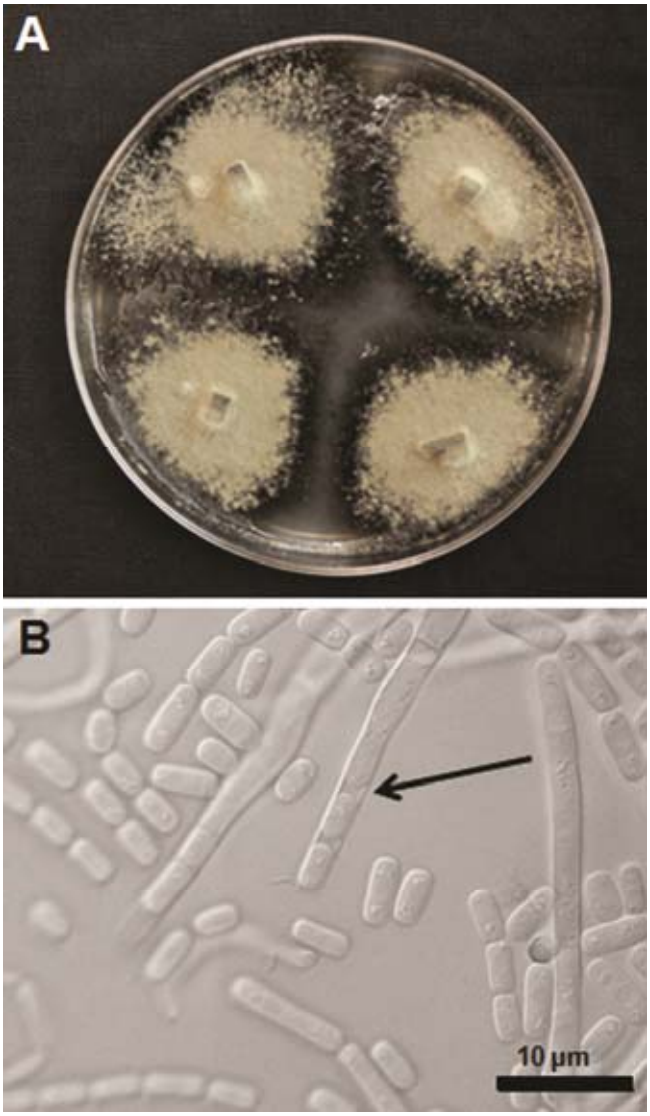

Fig. 2. Vegetative and asexual reproductive states of *Ceratocystis fagacearum*. **A,** Mycelial colony appearance on potato dextrose agar, and **B,** enteroblastic conidiophores (arrow) and conidia. Photo source: Mark Double, West Virginia University.

Pathogen

The causal fungus of oak wilt was first identified when the asexual stage was isolated from dying oaks in Wisconsin (61). In culture, colonies typically are gray-to-olive green, often with patches of tan mycelium (Fig. 2A). Cylindrical one-celled, truncated conidia arise endogenously from slightly tapered conidiophores (Fig. 2B). This feature served as the original basis for naming the fungus *Chalara quercina* B.W. Henry (61). This anamorph recently has been placed in the amended genus *Thielaviopsis* as *T. quercina* (B.W. Henry) A.E. Paulin, T.C. Harr. & McNew (102). Several years after the description of the asexual stage, perithecia were discovered when strains were paired in culture, providing the first evidence for mating types (63). Perithecia typically are black, flask-shaped (globose at the base), and embedded in a mycelial matrix (stroma) and form following spermatization by conidia of the opposite mating type. In culture, long, black perithecial necks protrude through the mycelium to liberate a sticky matrix of hyaline, elongate-ellipsoid, one-cell ascospores (119). Following the discovery of the sexual stage, the asexual name of the fungus was changed to *Endoconidiophora fagacearum* Bretz (25). After reexamination of the *Endoconidiophora–Ophiostoma* taxa, the organism was renamed *Ceratocystis fagacearum* (64). The taxonomic status and phylogenetic relationship of the oak wilt pathogen to other *Ceratocystis* spp. has been reviewed recently by Harrington (55).

Ceratocystis species typically inhabit wood, but one truly unique feature of host colonization by *C. fagacearum* is the formation of

Fig. 3. Sporulation of *Ceratocystis fagacearum* on northern red oak with **A,** associated vertical crack (arrow) in outer bark, and **B,** exposed mirror image of fungal mat with pressure cushions.

mats between the bark and wood (Fig. 2) (55,96). Mats occur on infected red oaks but seldom or not at all on white oak species (35,43). They develop in the cambial region of the tree, forming after colonization of the vascular system is complete and the tree has wilted. The mats arise from the mycelium that proliferates from the infected xylem.

Depending on host and environmental factors, mats may range in size from 4 cm^2 to over 208 cm^2 on red oaks (86; J. Juzwik, *personal observation*), but are smaller on white oaks. Fresh mats are gray-to-tan in color, similar to the pigmentation displayed by *C. fagacearum* in culture (115). Opposing elongated cushion-like structures called pressure pads form back-to-back, one associated with the sapwood and the other with the inner bark (Fig. 3). As the pads increase in size and thickness, they exert enough pressure to separate the bark from the wood, often rupturing the bark (Fig. 3). The cushions actually are comprised of modified fungal cells that internally form a net-like, chambered, daedaloid pattern. Another remarkable aspect of the fungal mats is the fragrant fruit-like odor associated with their occurrence. During periods of peak mat formation, the volatiles produced by the fungus (87) often can be detected meters away from an infected tree. This same fragrance attracts a myriad of insects, including some well-documented vectors of the disease such as nitidulid or sap beetles (Coleoptera: Nitidulidae) (75,99). As with other ascomycetous fungi, conidia of the opposite mating type, acting as spermatia, presumably fuse with trichogynes at the tip of the ascogonia to initiate perithecial development on the mats. Various insects are responsible for carrying conidia from mat-to-mat, resulting in cross-fertilization (115). The ascospores that eventually result are considered the dominant spore type for overland dissemination as their sticky nature, typical of many species of *Ceratocystis*, makes them ideally suited for insect spread. *C. fagacearum*–infected trees generally are killed by a single strain of the fungus resulting in mats of a single mating type (23,80). In areas of high disease incidence, perithecia are common on mats. An almost equal distribution of mating types occurs (11,80), supporting the assertion that sexual reproduction, in the absence of selfing, is occurring in the populations throughout the pathogen range where mating types have been examined.

Recent studies have provided some evidence as to the origin of the fungus. The organism appears to exhibit little genetic variation; a feature *C. fagacearum* appears to have in common with other exotic pathogens. This became clear when restriction fragment length polymorphism (RFLP) analyses of mitochondrial and nuclear DNA showed few differences among isolates from Texas, West Virginia, and Wisconsin (85). Likewise, additional study of isolates from the Upper Midwest that were subjected to mitochondrial RFLP analysis demonstrated almost identical banding patterns using *Hai*III digestion of genomic DNA (T. Harrington, *unpublished, as presented in 55*). There is little evidence for variability in pathogenicity for isolates from widely separated geographic regions (57). Admittedly, tests of pathogenicity have been limited in scope, but most isolates have proven to be highly pathogenic to species of red oak as well as causing symptoms to other members of the Fagaceae (26,34). These results have led to a hypothesis that *C. fagacearum* is not native to the United States (76).

Disease Symptoms and Signs

The tree crown symptoms of oak wilt are most dramatic and notable among members of the red oak group. Red oaks commonly develop symptoms within weeks of infection, and rapid wilting of foliage throughout the crown generally follows (Fig. 4A) (100). The pattern of crown wilt may vary according to the manner of pathogen entry. For example, the side of the crown closest to an adjacent diseased tree may wilt first, suggesting that the fungus is invading via connected roots (see next section). Mature leaves can appear water-soaked. Bronzing and necrosis of the leaf tips and margins also are common. Leaf abscission of completely green leaves as well as those that are symptomatic is a unique feature of the disease. Often, an oak wilt–infected tree is discovered when the ground beneath the tree is littered with symptomatic leaves during the peak of the growing season. Sucker shoots can develop on the main stem of an infected red oak but are short-lived and rapidly become symptomatic. In contrast, disease progress and ultimate outcome is less predictable among various members of the white oak group in North America as previously noted (Hosts section). Branch dieback (single or several) in highly resistant species may or may not lead to tree death and may be misdiagnosed as oak decline. Moderately resistant species may express foliar symptoms on scattered branches in the crown that then progresses to death of the entire tree (Fig. 4B) (73,100). Live oaks in Texas develop symptoms more slowly than red oaks but usually die within 3 to 8 months of infection (Fig. 4C) (100). Veinal chlorosis and necrosis are unique leaf symptoms in live oaks that can be diagnostic for the disease. Leaf abscission of the semi-evergreen live oaks, as with the red oaks, also is typical.

Less reliable symptoms of oak wilt include vascular staining in the xylem of branches and main stem of moribund hosts (105). In at least some white oak species, discoloration occurs as dark brown to blackish streaks in the outer xylem when the bark is removed from an infected branch. A ring of discolored xylem tissue also may be evident in cross sections of such a branch. Bluish gray to dark brown vascular discoloration also occurs in red oak species, but is often harder to detect.

The definitive sign of oak wilt is the presence of the sporulating mats of *C. fagacearum* on recently killed trees, particularly red oak species (previously discussed). However, the hidden nature of these

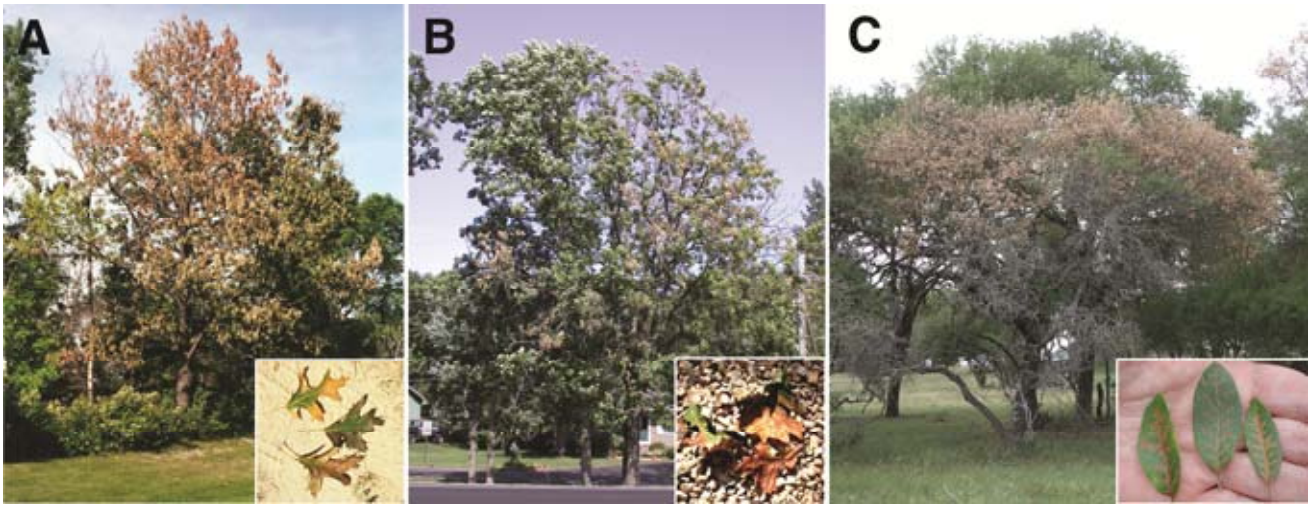

Fig. 4. Foliar and crown symptoms of oak wilt in selected species: **A,** northern red oak; **B,** bur oak; and **C,** Texas live oak.

mats and the deep furrowed bark of large diameter oaks makes their detection challenging. The vertical cracks in the tree bark resulting from growth of these mats and associated sweet odor are clues to their presence. Mats are generally formed on branches and stems >7.6 cm (3 in.) in diameter. Mat formation is influenced by amount of precipitation (21), moisture content of the sapwood (32), temperature (43), and other organisms that colonize the wilted oak (114). The frequency of mat formation can vary greatly among different seasons and regions within areas where oak wilt occurs (72). In Minnesota, for example, peak mat formation occurs during spring and fall months (70). Onset of mat production is related to how early or late wilting occurred during the growing season. For some red oaks, mats may start to form in the fall on the upper main stem and branches and then resume lower on the main stem the following spring. In Texas, mats commonly form in late winter and spring but not during fall (6).

An outwardly expanding circle of dying oaks occurring over years within a forest stand or in a managed landscape also suggests the presence of oak wilt. This pattern is the result of belowground spread of the pathogen as discussed in the next section. Viewed aerially, large disease centers or multiple disease centers can be seen across a landscape in areas experiencing oak wilt epidemics (Fig. 5).

Epidemiology

Aboveground pathogen spread. New oak wilt infection centers are established when *C. fagacearum* is transmitted successfully aboveground from diseased to healthy oaks. There is no evidence that dissemination occurs by wind, wind-driven rain, or via smoke of burning infected firewood. Spread via contaminated tree pruning or cutting tools has never been demonstrated; rather, contaminated insects are commonly regarded as the means by which this type of spread occurs (50). Although squirrels, birds, and several insect taxa have been cited as vectors, there is little published data to support the assertions made for many of these animals (71,95). The preponderance of evidence points to species of nitidulid beetles (Coleoptera: Nitidulidae: *Carpophilus* spp. and *Colopterus* spp.) as primary vectors and oak bark beetles (Coleoptera: Curculionidae: *Pseudopityophthorus* spp.) as lesser or minor vectors in the Upper Midwest and Texas. A recent study specifically addressed the relative importance of vectors within the Nitidulidae (3).

Various species of nitidulid beetles are attracted to oak wilt mats formed on recently killed oaks (75,86,99). The beetles acquire pathogen spores during their feeding and oviposition-related activity in and on the mats. Only a small subset of these insect species, however, is known to visit "fresh" xylem-penetrating wounds that are required for successful infection by nitidulid beetles (59,79,99). One species, *Colopterus truncatus*, is known to arrive on wound surfaces within 10 minutes of their creation (99; J. Juzwik, *personal observation*)! Wounds generally less than 72 hours old are required for successful transmission (84,124). Several species of *Colopterus* and *Carpophilus sayi* are considered the primary vectors in the central states (Minnesota to Texas) (Fig. 6) (59,60,79). Although *C. fagacearum*–laden adult beetles of these species may be found dispersing in oak stands between early spring and early fall, they are only found visiting fresh wounds during spring and early summer in Minnesota (3,79). Surprisingly high percentages (≤84%) of such beetles collected from fresh wounds have yielded the fungus upon isolation (79).

Unlike the nitidulid beetles, the oak bark beetles are capable of creating fresh wounds that allow for natural *C. fagacearum* inoculation during the beetles' maturation and reproductive feeding activity on healthy and stressed oaks (2,40). Bark beetles likely acquire conidia produced in larval galleries and pupal chambers prior to adult emergence. During spring in Minnesota, the frequencies of *C. fagacearum*–laden oak bark beetles dispersing in oak stands are quite low (0.04 to 0.13%), especially when compared with that of *Co. truncatus* (4 to 32%) and *Ca. sayi* (14 to 79%) (2,3). For this and other reasons, oak bark beetles are considered minor vectors in the Upper Midwest and Texas (2,60). Although oak bark beetles and nitidulid beetles are found in the Appalachians, species within both groups are viewed as very inefficient dis-

Fig. 5. Oak wilt across the landscape showing **A,** live and red oak mortality (arrows) in central Hill Country of Texas, and **B,** red oak mortality on the Anoka Sand Plains in east central Minnesota. Photo source **(B)**: S. Cook.

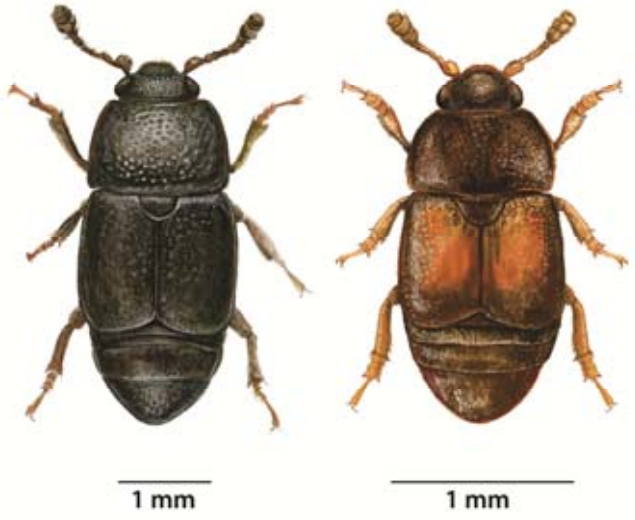

Fig. 6. Important nitidulid beetle species (*Carpophilus sayi*, left; *Colopterus truncatus*, right) involved in transmission of *Ceratocystis fagacearum*. Photo source: Juliette Watts.

seminators of *C. fagacearum* in the region (90). Concern does exist, however, for the potential establishment of a non-native insect that is better suited for transmission, especially in the diverse hardwood forests of the Appalachians. *Scolytus intricatus* (Coleoptera: Curculionidae: Scolytinae), an oak bark beetle native to Europe, is one such insect worthy of inclusion in exotic beetle detection surveys (123).

Human-aided insect transmission also occurs. Because oak wilt mats may form on logs and firewood sawn from recently killed oaks, transport of such material can result in spread of the pathogen to previously oak wilt–free areas, such as most likely happened in infected counties in west Texas (6) and in New York (66). However, there is no evidence that *C. fagacearum* is transmitted via nursery stock or seed (27).

Belowground pathogen spread. Although individual trees do become infected with *C. fagacearum* as a result of vector transmission, the disease is most dramatic in settings where oaks of like species grow together and form root grafts (Fig. 7). To be significant in the disease cycle, the root grafts must be functionally "organic," meaning they allow for the movement of xylem contents from one tree to another when the roots are joined (44). Early evidence for the role of root grafting in the epidemiology of oak wilt came from injecting trees with dyes and poisons and subsequently observing phytotoxicity in adjacent trees (13). Another form of root unions arises when a species is clonally propagated by root sprouting and the ramets share a common root system (97). When these root unions occur, the oak wilt pathogen can spread rapidly, with many trees developing symptoms in a single season. Such transmission results in patches of dying oaks typical of the disease in a population of susceptible hosts and usually is attributed to killing far more trees than insect vector transmission. The rates of disease center expansion over time are highly dependent on the rooting habits and root grafting frequencies in different regions of the disease range (72). In the Upper Midwest, large stands of oaks often share connected root systems, particularly on deep sand soils that promote root graft formation. The average radial expansions on such soils in Minnesota and Michigan are 7.6 m/year to 12 m/year, respectively. The same can be said for rural and urban areas in Texas, where the proliferation of sprouts from live oak root systems has resulted in significant oak populations that share a common root system. The estimated rate of disease center expansion in Texas live oak is up to 40 m per year in this ecosystem (10). In contrast, root grafts appear to play a less significant role in disease development in the Appalachians, where diverse hardwood stands are comprised of numerous species, thereby limiting tree-to-tree spread because of the infrequent occurrence of interspecific root grafts (44,50,89). No rates of disease center expansion have been published for this region. The rocky soils and the steep slopes in the Appalachians and the Ozarks also have been suggested to act as a further deterrent to root grafting and thus belowground spread of the fungus.

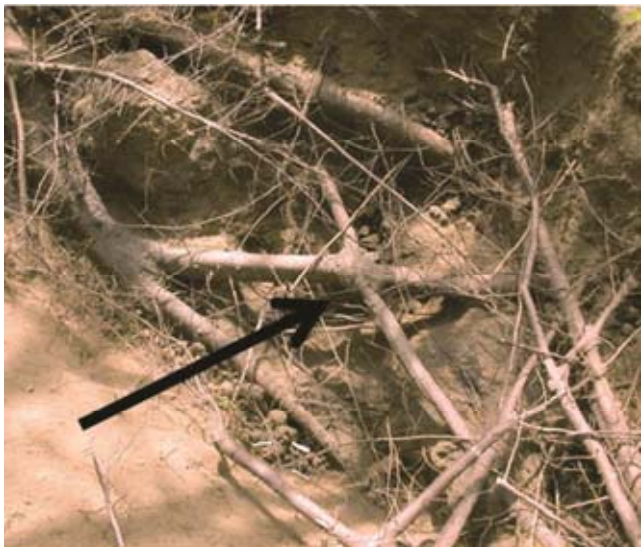

Fig. 7. In situ view of grafted roots of northern red oaks in an oak wilt infection center in east central Minnesota. Arrow points to graft union.

Disease Impacts

Oak wilt is a devastating disease in portions of its range where epidemics are on-going. It is difficult to quantify with any accuracy the number of trees killed by *C. fagacearum*. Several efforts have been made to map and quantify oak wilt in Minnesota and Texas (39,54), but they are too narrow in scope to obtain statewide, let alone national, statistics on mortality. In an ongoing economic assessment in 734 counties in the eastern and midwestern United States, U.S. Forest Service researchers are predicting the total number of trees expected to become infected with the oak wilt fungus over the next decade and the cost of removing those trees when they occur in developed areas (R. Haight, *personal communication*).

Numbers of infection centers at landscape levels have been obtained by extrapolation from satellite imagery and aerial photography coupled with results of ground-checking. This approach was used to estimate that ≥800 actively expanding centers exist in the Fort Hood Military Installation lands (87,900 ha) in central Texas (39). In a recent modeling effort associated with the U.S. Forest Service economic assessment, researchers estimated that 5.92 million oaks are present in Anoka County (110,000 ha), Minnesota, based on Forest Inventory and Analysis data of the same agency, and that 885 actively expanding disease centers occupying 547 ha (=5.47 km^2) were present in 2010 (54).

Conservative estimates of numbers of infection centers in a state also can be obtained from statistics compiled on sites treated for oak wilt under natural resource agency administered control programs (see next section). In central Texas, 2,654 active centers were treated with federal cost shares or technical assistance from Texas Forest Service field foresters between 1988 and 2010 (R. Billings, *unpublished data*). Many other centers remain to be treated or were treated by private landowners without stage agency supervision.

In contrast to central Texas and the Upper Midwest, oak wilt occurs sporadically in much of the Appalachian region (89). As a result, small isolated disease centers often do not perpetuate the disease. The reduced level of disease is further restricted especially in years when the conditions for vector spread are not met. The sporadic nature of the disease is demonstrated by a 2010 survey in North Carolina where oak wilt was found to be active in only three of five counties where disease incidence had been historically high (R. Blaedow, *personal communication*; 22). Interestingly, only 1 of 83 "historical" centers revisited was found to be active; 6 new and active oak wilt centers were identified. The number of oaks killed per infection center varies greatly, particularly in relation to soil type and red oak abundance in affected stands. Light-textured soils (e.g., sand or loamy sand) with red oak accounting for the majority of the trees in the stand have been positively correlated with high mortality (93,94). In heavier-textured soils with a diversity of oak species and variable topography, such as occur in West Virginia and in the Ozarks of Missouri, oak wilt centers are comprised of only a few diseased trees (72). In contrast, tens of thousands of oaks die annually from oak wilt in the Lake States (anecdotal estimate) and millions of oaks have died in Texas since discovery of the disease there (6,72).

The huge losses in number of trees due to oak wilt are associated with economic losses such as timber and decreased property values (6). Tree removal and tree replacement costs are substantial, particularly in urban and community forests affected by the disease (15). Other impacts are more difficult to measure, such as aesthetic losses of historic trees and featured landscape specimens. Tree mortality caused by *C. fagacearum* indirectly affects ecosystem services (e.g., carbon sequestration, storm water run-off mitigation, cooling during summer) and ecological functions of urban and

rural forests (6,38). For example, oak wilt in juniper-oak woodlands in central Texas leads to critical losses of suitable habitat for the golden-cheeked warbler, an endangered species (8).

Disease Management

Overview. As with any plant disease, successful oak wilt management is achievable when the proper control methods are applied according to proven, recommended protocols (7,45,100,120). However, difficulties in implementing controls may arise due to the wide variety of circumstances encountered throughout the range of the host. Methods appropriate to minimize losses in urban environments, for example, may not be appropriate in rural forests (56). Given the variety of responses exhibited by different *Quercus* species toward infection by *C. fagacearum*, the relative proportions of red oaks, white oaks, or live oaks must be considered to control oak wilt. Many disease control methods are expensive and may be environmentally disruptive. Given these challenges, prevention is the preferred approach. Preventive measures range from stopping the aboveground ("local" and long distance) spread by insects, to preventing "local" tree-to-tree transmission through connected roots. Once a tree is infected, or in imminent danger of being infected, direct control measures aimed at debilitating the fungus are available. Mitigating measures, such as planting resistant and tolerant *Quercus* spp. or nonhost species, also are recommended. The success of each protocol depends on interrupting specific stages in the disease cycle of the pathogen. These approaches are discussed below.

Detection and monitoring. Early diagnosis of oak wilt is essential to successfully control the disease and justify the expense of treatment. In areas with a history of oak wilt, foliar symptoms may be sufficient for identification, particularly when the pathogen is spreading through stands in characteristic patterns. The veinal necrosis symptom on live oak in Texas is often used as the sole diagnostic criteria, although even that distinctive symptom may be mimicked by other causes. Bronze-colored leaves progressing from margins to midrib plus cast leaves commonly are used to visually diagnose the disease in red oaks on sites in the Upper Midwest. Diagnosis in white oak species such as bur (*Q. macrocarpa*) and white (*Q. alba*), however, requires sampling and lab confirmation. The presence of fungal mats is considered a definitive sign of the disease, but they are not always present. Therefore, isolation of the pathogen has been a standard practice when laboratory confirmation is needed. Standard protocols for sampling and isolating the pathogen are available (105).

Fig. 8. False color infrared satellite (IKONOS 1-meter pan sharpened) imagery of an oak wilt infection center (circled) in Central Texas.

There are challenges in successfully isolating *C. fagacearum* from the xylem tissues of diseased oaks. Branches from a symptomatic tree, or sapwood excised from the trunk, should be obtained and cultured before the tree has desiccated following death. Secondary microorganisms rapidly invade and replace the oak wilt fungus in moribund trees. Successful isolation is more common with samples from red oaks than live oaks. The fungus can easily be identified in culture based on conidiophores and conidial morphology as well as the sweet odor produced when growing on acidified potato dextrose agar (12). Further confirmation may be obtained through sequencing of the internal transcribed spacer (ITS) and large subunit regions of the nuclear ribosomal DNA (66). Presence of *C. fagacearum* in artificially inoculated wood sticks has been detected using a standard DNA extraction protocol followed by PCR using a nested protocol (standard ITS1 and ITS4 primers and species-specific primers) (122).

On a broader scale, the widespread occurrence and severity of oak wilt in some states have resulted in establishing long-range policies by state and federal natural resource or urban forestry agencies that have committed significant resources to deal with the disease. Survey and detection were mainstays for formulating these regional oak wilt management programs. Numerous states adopted regular, low-level aerial monitoring for oak wilt in the past, but these are not as widely practiced now due to cost, danger, and the development of improved technologies (58). Satellite imagery (Fig. 8), combined with geographic information systems (GIS), have emerged as an alternative for measuring the incidence and severity of oak wilt (39). Currently, however, spatial data commonly is obtained from ground-based surveys, aerial photo imagery, and traditional aerial sketch map surveys. These data are entered into a GIS to map disease incidence and are used to coordinate disease suppression efforts. These tools also aid in the decision-making process for developing the appropriate control recommendations.

Preventing sources of inoculum. Once located, wilted red oaks can be cut and treated or destroyed prior to development of the fungal spore mats and subsequent spread of spores by nitidulid beetles. Historically, in Minnesota, wilted red oaks have been reinspected between November and March following the growing season in which wilt occurred (36). If the vascular cambium is found to be white or have vascular streaking characteristic of pathogen presence, the trees are considered potential spore-producing trees and are marked for removal prior to 1 April. Acceptable treatment methods for dealing with diseased wood include burning, chipping, or any other process used to encourage drying of the wood or to facilitate rapid colonization by competing fungi (7,100,117). Removal and proper disposal should occur before mat production occurs in the late winter or spring of the year following tree wilt. Varying success has been achieved by deep girdling and stripping the bark (debarking) from the lower trunks of diseased red oaks to encourage drying and thus prevent mat production (53,68). This is most effective when treatment occurs during the incipient wilt stage. Logs from diseased oaks also may be cut into firewood log lengths, split, and stored on-site under clear plastic that is sealed at the ground line through the field season following tree death (7,73,100). Where such treatment is not applied, it is important to strongly discourage the use of infected red oaks as firewood, since mats may form on trees even after cutting, transporting, and sale of small logs for home use (7).

Preventing vector transmission. The topic of vector transmission via the sap-feeding nitidulid beetles has a long history of re-

search and control technology developed to prevent initiation of new infection centers (115). The vector can be interrupted at a number of stages in the disease cycle. Nitidulid beetles are highly effective in locating mats and subsequently becoming contaminated with *C. fagacearum* spores. Thus, it is very important to prevent inoculum formation as described above. Infection of healthy trees can be prevented most easily by avoiding wounding of uninfected oaks. Suitable infection courts (e.g., cut branch ends, fresh stump surfaces, and stem wounds) can result from activities such as tree removal, utility line clearing, pruning, and construction of buildings and roads, particularly during times of the year when contaminated beetles are attracted to fresh wounds and the oak species are most susceptible to infection. In the Lake States, this critical time period is between early April and mid-July (3,46), while in more southerly states, such as Missouri and Texas, the critical winter–spring period begins and ends earlier (7,59,60). Utility companies, commercial arborists, landowners, and others are advised to avoid such activities in known oak wilt areas or protect oaks from wounding during construction, particularly during the critical time period. Unfortunately, broken branches and stems caused by high winds associated with spring storms are also attractive infection courts and may result in new infection centers (78). When cutting or construction activity cannot be avoided, wounds should be immediately painted (e.g., latex paint or a wound dressing) to prevent nitidulid beetle access to the exposed wood (31,46). This recommendation contradicts the commonly held view by many arborists that wound paints have no use in tree health practices, as their use promotes wood discoloration and decay (109). Recent evidence confirms that wound paints are beneficial in the case of this insect-transmitted vascular wilt pathogen (31). Although insecticide treatment of diseased logs and firewood was recommended in the 1950s (20), sanitation and proper disposal of diseased trees (described previously) are recommended currently for preventing dispersal of fungus-contaminated oak bark beetles or nitidulid beetles.

Preventing spread through connected roots. Whether through root grafts between healthy oaks and diseased ones in close proximity or the common root systems arising from vegetative propagation of live oaks, prevention of "local" belowground spread of *C. fagacearum* among neighboring trees is a major focus of most oak wilt control efforts. Control actions designed to stop belowground spread attempt to either sever roots between adjacent trees along a treatment line (29,37,46) or create a wide zone of dead oak roots outside the perimeter of an active infection center (68). Repeated efforts at managing pathogen spread by removing all oaks within 15 to 30 m, with or without herbicide treatment, have been largely unsuccessful (6,29).

Successful treatment results from digging a deep trench, slicing through soil and roots with a long blade, or using a circular rock-saw blade to sever root connections between neighboring oaks (Fig. 9). Soil conditions as well as land use (urban versus rural) obviously dictate the type of machinery necessary to accomplish the task. For example, in deep sand soils a vibratory plow with a 1.5 m (60 in.) blade is sufficient to sever most roots and the width of the soil disturbance is minimal. In contrast, in shallow, rocky soils, backhoes, belt trenchers, or rock-saws are used to get to the required depth. Again, soil type dictates the necessary depth of cutting, with 1.2 m (48 in.) being the most commonly recommended, minimal depth in Texas. Most treatment failures are considered to be a result of insufficient depth or poor placement of root cutting lines or "trenches" as they are referred to in Texas. Due to the common occurrence of undetected root infections in asymptomatic trees, the challenge is to sever tree roots beyond the last asymptomatic infected oaks bordering an infection center.

Several models have been developed to guide practitioners in this task; these include a "rule of thumb" model initially proposed by French and Stienstra (47) and mathematical models by Bruhn and others (30). In Minnesota, a vibratory plow commonly is used to sever roots in primary and often secondary lines beyond the perimeter of active disease centers; high levels of control have been achieved (36,77). The major drawback of this model is the need for an experienced oak wilt forester to "tailor" the model for the situation or site based on his or her experience with the disease (77). The mathematical models are based on soil type, tree diameters, inter-tree distances, and the estimated pathogen spread distance in one year via grafted roots (30). Use of these models to place treatments at 95 and 99% confidence levels is recommended in Michigan (1,29). The needless sacrifice of healthy red oaks is the biggest drawback in the use of the mathematical models, particularly when applied to sites with soils different from those for which the particular model used was developed (29). Trenches are placed at a much greater distance from the edge of the advancing disease front in Texas (minimum of 30 m or 100 ft) because the fungus has been found to spread up to 40 m per year through common root systems (7,10). The percentage of treatment success in Texas has increased from 67% for all trenches established between 1991 and 1994 to 76% or higher after 1994 (16,48). Re-grafting of roots

Fig. 9. Heavy equipment used to sever connected root systems around perimeter of oak wilt infection centers: **A,** vibratory plow commonly used in the Great Lakes States; **B,** rock-saw commonly used in Texas; and **C,** belt trencher machine occasionally used in northern states. Photo sources: **A and C,** J. Juzwik; **B,** B. Moltzan.

across the trenches likely occurs but has not been demonstrated (120); however, some sites may be re-treated as a precaution (77). Alternatively, water permeable fabric can be installed as "trench inserts" and extend the number of years over which the treatment is effective (121). The additional costs and labor associated with inserts have limited their use to only special circumstances where the resources are available.

A previously untested method for stopping pathogen transmission via roots recently has been used on an operational basis in the rocky soils of rolling terrain on national forest land in northern Wisconsin (56); however, formal documentation of effectiveness is not available yet but is forthcoming (J. O'Brien, *personal communication*). All infected oaks and their closest neighbors are cut and removed. An excavator then is used to rip apart the roots extending from the stumps and then push or pull the stump and root mass out of the soil.

Prevention through regulatory means. On a local and regional scale, the movement of potential mat-bearing firewood always has been a concern in the epidemiology of oak wilt. There are several communities in west Texas and the Upper Peninsula of Michigan, for example, where the oak wilt fungus was likely introduced on contaminated firewood. More direct evidence of firewood involvement was reported with the recent discovery of oak wilt in Schenectady Co., New York, where mat remnants were found on firewood (66). This same concern has been the focus of international attention where exportation of red oak timber from the eastern United States to Europe has been regulated (51,104). Treatment protocols such as log fumigation (e.g., methyl bromide) must be followed in order to allow the movement of timber from infected areas (92).

Direct control with fungicides. The only practical means of managing oak wilt with fungicides is through intravascular injection, or infusion, of trees at high risk of infection via root connections between healthy and infected trees (Fig. 10). Rarely are trees injected to prevent insect transmission of the pathogen. Currently registered fungicides are triazole type compounds (e.g., propiconazole). Injection has been demonstrated to achieve the greatest survival rates in live oaks when used in a prophylactic manner, just prior to spread of the pathogen into the root system of a healthy tree (9). In the Lake States where oak wilt occurs most commonly in red oaks, a single prophylactic treatment of asymptomatic *Q. rubra* and *Q. ellipsoidalis* suppresses disease development for two growing seasons following injection (18,41,103). Based on results of operational evaluations and experimental trials, crown wilt has occurred in up to 40% of red oaks 3 to 5 years after a single injection. Thus, arborists may re-treat high value red oaks that remain under high disease pressure. Re-treatment afforded at least one additional year of disease suppression in a recent, controlled trial (103). Expectations of success must be reduced when therapeutic treatments are administered to live oaks exhibiting some crown wilt symptoms. Therapeutic treatments of symptomatic red oaks generally are unsuccessful (118), while such treatment in two white oak species, *Q. alba* and *Q. macrocarpa*, generally prevents further disease development within 5 years (41).

Landowners and arborists alike need to recognize the capabilities and limitations of propiconazole when used as a tool in the integrated management of oak wilt. *C. fagacearum* is highly sensitive to small amounts (ppb range) of triazoles based on laboratory assays (9). Concentrations of propiconazole in the ppm range occur in roots and lower stems of red oaks up to 2 years after injection (18). The chemical moves well systemically through the stems and branches of injected trees, while basipetal movement into the roots at least 1 m from injection points also occurs (9,18). As a growth regulating chemical, propiconazole seems to be supplementing natural resistance mechanisms in treated trees. Even in highly susceptible red oaks, its use has been correlated with reduced vessel diameters and reduced width of early and latewood produced for several years post-treatment (17). Degradation of the chemical in oak tissues over time is a common explanation for breakdown of chemical control after 2 years (9,18,101). Furthermore, although the chemical may be detected in lower stems and roots at 2 years post-treatment, it may not be translocated laterally into newly produced woody tissues at sufficient concentrations to stop reinvasion by the fungus via grafted roots (18).

Lastly, injection is not considered to be a substitute for severing connected root systems to stop belowground spread. At sufficient levels, propiconazole prevents disease development in the aboveground portion of red oaks but does not eradicate the fungus from

Fig. 10. Intravascular injection of live oak with systemic fungicide.

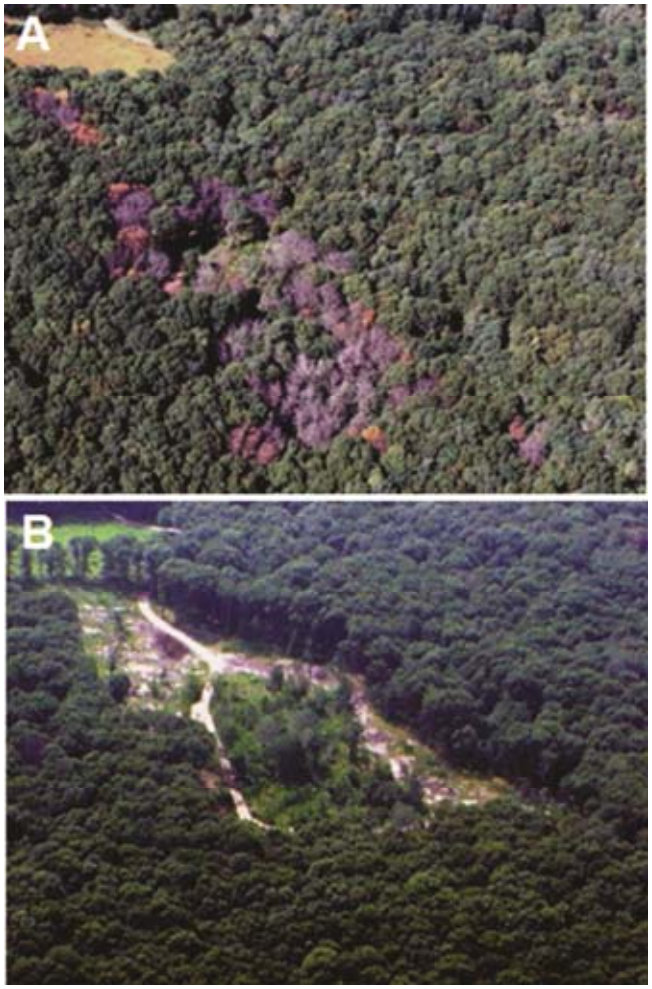

Fig. 11. Before **(A)** and after **(B)** root severing and red oak removal treatments in a rural forest setting in east central Minnesota. Photo sources: A. Marin and S. Nelson.

the roots (18). Furthermore, in live oaks, the fungus is known to pass through the roots of a treated live oak without affecting it and into those of a neighboring tree via interconnected roots (9).

Integrated use of control tools. As with many other plant diseases, an integrated approach to control results in greatest success in managing a destructive disease like oak wilt. With high-value shade trees in an urban landscape, property owners may opt for integrated use of root cutting, removal of recently wilted trees, and fungicide injection of asymptomatic oaks. Less aggressive treatment, such as root cutting alone or root cutting plus sanitation also can be effective with relative success depending on the situation. In a rural forest setting with lower individual tree values, root cutting coupled with removal of all oaks inside the primary line is the most aggressive approach (Fig. 11). Sale of timber cut from asymptomatic trees removed to the primary cutting line may help offset some of the costs of the disease treatment. Of course, landowner preferences, management objectives, and site conditions may lead to a different prescription for a similar diseased stand. At the landscape level, disease management is most effective when regular monitoring and detection are conducted, control actions are coordinated among neighbors or landowners, infection centers are treated while still small, and post-treatment evaluations are faithfully carried out.

Education/outreach and suppression. The dramatic appearance of Dutch elm disease (49) and chestnut blight (62) in the United States during the early twentieth century led to a significant research response when oak wilt was discovered. Those efforts were followed by state and federal partnerships within the range of the disease to develop some unprecedented survey and control programs (58,93,111). Educational materials were promoted to advance public understanding of the disease and describe the methods needed to avoid losses (e.g., 20,47). The original emphasis of those efforts was on the impact of oak wilt on timber production (68). As our understanding of oak wilt improved and better technologies became available, successful oak wilt control in small woodlots and urban landscapes became possible (28,46). Urbanization into former woodlands leads to increases in oak wilt incidence in localized areas due to wounding associated with site development (28,37,52). These developments led to even greater educational activities, and made it feasible for some states to establish comprehensive suppression programs, some of which continue today (see next section) (15,16,56,114). A careful search of the internet will find oak wilt educational materials and management guidelines developed for a wide variety of audiences, including natural resource managers, commercial audiences, loggers, large landowners, homeowners, and policy makers (7,19,37,52,73,74) (see also http://www.texasoakwilt.org). From another perspective, the environmental impact of oak wilt management resulting from root cutting and removal of oaks requires focused educational efforts to convince landowners of their necessity (15).

Cost-share suppression programs. *Texas*. A long history of widespread and destructive oak mortality in central Texas culminated with the discovery of oak wilt as the cause in the late 1970s. The Texas Oak Wilt Suppression Project under the leadership of the Texas Forest Service was established with the support of the USDA Forest Service, Forest Health Protection in 1988 (15). This comprehensive program was designed to provide public education, survey and detection, federal assistance to private landowners for oak wilt management, and long-term monitoring of project outcomes (15,16,56,111). There have been considerable accomplishments. Through aerial survey, ground inspections, and reports from landowners, C. *fagacearum* has been detected in 73 counties. During the 22 years of the program, 2,654 oak wilt centers have been treated by digging 1.14 million m (3.72 million ft) of trenches. The success rate of the trenching has varied, being as high as 79% in the 2008 postsuppression evaluation of 121 trenches installed between 2002 and 2005 (15). The total federal cost shares used to reimburse participating landowners for trenching and tree removal is approximately $2.7 million. For a brief time, fungicide was provided free-of-charge to participating landowners. Success of the program can be measured in a number of other ways. For example, working partnerships were stimulated by the project among the Texas Forest Service, Texas Agrilife Extension Service, and various central Texas municipalities. Educational efforts have been developed to reach a broader audience through advanced Master Gardeners training and arborist certification programs, thus improving the level of disease management in urban environments. A website (www.texasoakwilt.org) also was developed to increase public awareness of oak wilt; it includes resources available to landowners, such as vendors for oak wilt control work and distribution maps of known disease centers in the state.

Minnesota. The Minnesota Oak Wilt Suppression Project was established in 1991. Funding for different time periods of the project came from two sources: the cost-share program of the USDA Forest Service, Northeastern Area State and Private Forestry, 1991–1997 and 2003–2007 (96); and the Minnesota ReLeaf Program, 1998–2002. The project ended in 2008 due to changes in funding, staffing, disease levels, and management priorities.

The Minnesota project addressed the need for disease survey and management at the landscape level. The stated goal of the project between 1991 and 1997 was to reduce the density of oak wilt across east central Minnesota to approximately 0.15 ha per 1 km^2 (1 acre per mi^2), a disease level which Minnesota Department of Natural Resources (DNR) staff believed most communities could manage on their own (42).

Much was accomplished over the life of the project. Over $4.2 million in federal funds were matched with an estimated $7.4 million in state and private landowner funds. Between 2003 and 2008, 187 km (117 mi) of root cutting treatment (vibratory plow) occurred and 9,107 potential spore producing and/or asymptomatic red oaks were removed from inside the primary root cutting line of treated disease centers that covered 2,876 ha (7,108 ac) (numbers based on 2006 Minnesota DNR unpublished data, S. Burks).

Success of Minnesota DNR coordinated oak wilt management efforts have been measured in several ways. First, the calculated density of oak forest affected by the disease in the Minnesota project's treated area was reduced from 0.5 ha per km^2 (3.0 acres per mi^2) to 0.2 ha per km^2 (1.3 acres per mi^2) (S. Burks, *unpublished data*). For 66 of 91 municipalities, the densities of disease centers per area of infected forest were less than the average endpoint stated above. More recently, the efficacy of applied treatments to stop further expansion of disease centers has been the primary measure. Disease centers treated in 2002, 2003, and 2004 were evaluated in 2005, 2006, and 2007, respectively. Lack of recently wilted oaks outside but within 20.1 m (66 ft) of the primary root cutting line was the metric for success. Preliminary results found no further expansion of the centers for 70 to 99% of the treated and evaluated sites 3 years after treatment (S. Burks, *unpublished data*). Finally, success has also been measured in terms of numbers of communities receiving assistance and increases in internal capacity (e.g., education and outreach, oak wilt trained staff) of local units of government to manage urban and community forests within their jurisdictions.

Conclusions and Future Outlook

C. fagacearum continues to be a deadly pathogen, particularly for red and live oaks in urban and rural forests in the central United States. In spite of this, oak wilt has proven to be a manageable disease in states experiencing on-going epidemics when consistent, integrated, and sustained suppression programs are employed. In such areas, oak wilt should be considered a "smoldering fire" that can flare up again if the commitments to its management are dropped. Control of oak wilt in urban areas is best achieved when neighborhoods work together in coordinating treatments for protecting trees of high amenity value. In states where the disease is expanding its local range, aggressive measures to eradicate the disease are undertaken (29). Creative and lower cost methods are continuing to be tested for controlling oak wilt in hard-to-access forested areas, including those with boulder-strewn landscapes (56). In areas where disease centers occur sporadically and result

in death of small numbers of trees, little effort is expended to control oak wilt although state forest health specialists generally monitor disease occurrence (R. Blaedow, *personal communication*).

Concern still exists over the potential for undetected spread into new locations within the United States, in neighboring countries, and possibly abroad (5,104). The unexpected symptomology of oak wilt in Texas misled investigators for several decades and delayed the identification of *C. fagacearum* as the causal agent for extensive live oak mortality in the state. Based on the surprising finding of European white oak susceptibility to *C. fagacearum,* continued concern is justified in Europe and perhaps other continents. Conversely, the possibility exists for introduction and establishment of a more efficient and exotic insect vector that could significantly increase the frequency of aboveground spread of the pathogen in the United States (123).

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