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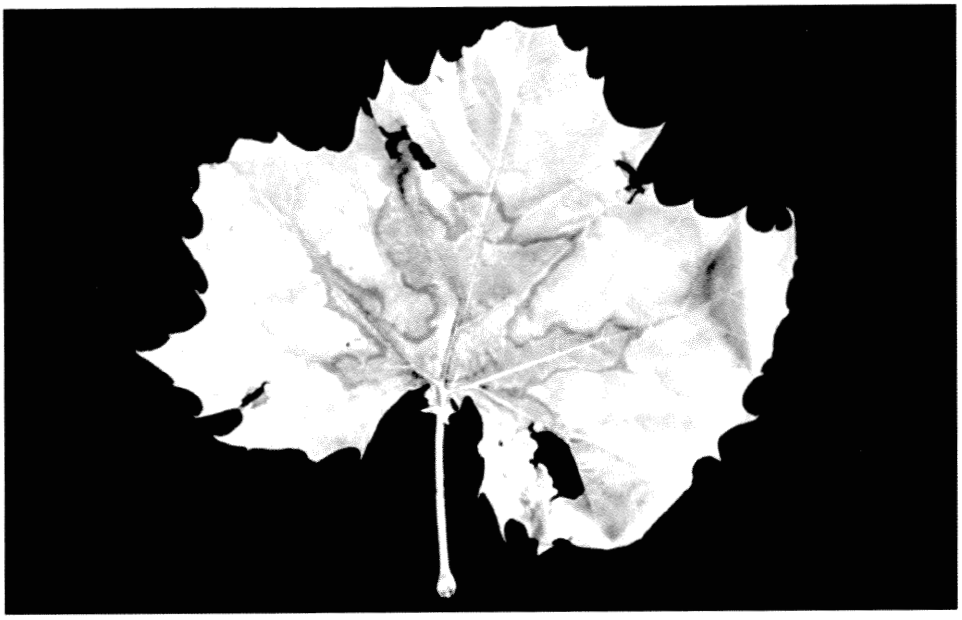

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Recent Advances in the Control of Oak Wilt in the United States

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Abstract: Oak wilt, caused by *Ceratocystis fagacearum* (T.W. Bretz) J. Hunt, is probably the most destructive disease of oak trees (*Quercus* species) in the United States, and is currently causing high mortality at epiphytotic proportions in central Texas. The serious potential for damage prompted an increase in federal funding within the past fifteen years for new oak wilt research. New research developments have included adaptive utilizations of industrial technologies such as Electronic Aroma Detection (EAD) and aerial infrared remote sensing surveys for early disease detection and diagnosis, geotextile polymeric landscape fabrics etc. All of these areas of increased knowledge have contributed to and provided means for improving oak wilt suppression in a more effective and integrated way. The development of these improved methods for oak wilt control are reviewed here and discussed in relation to current state forestry pest-control programs that have begun to implement these new methods in their oak wilt suppression operations.

Key words: *Ceratocystis fagacearum*, disease suppression, microinjectors, trench inserts, triazole fungicides

INTRODUCTION

Oak wilt, caused by *Ceratocystis fagacearum* (T.W. Bretz) J. Hunt, is an important infectious vascular wilt disease that plays an major role in shaping the health, structure and composition of hardwood forest stands in the eastern half of the United States. It is a disease that has plagued oak forests in this region for the majority of the twentieth century. Since oak wilt disease was first recognized in Wisconsin in 1942[1], the disease had a major impact on stand composition and mortality of hardwood forests in localized areas throughout the eastern states. For this reason, it is considered by many to be the most serious disease of oaks (*Quercus* SPP.) in North America[2-4]. The oak wilt fungus is potentially the most destructive of all forest pathogens because of its ability to kill its hosts within a short time after symptoms first appear[3-6]. The serious impact of the disease on eastern oak forests has been exacerbated by previous changes in forest stand composition and forest management practices that resulted in oak stands with greater proportions of susceptible red oak species[3,7]. Oak wilt has been particularly devastating in the state of Texas largely due to the existence of many pure stands of live oaks[8].

Oak wilt probably was first observed in the state of Texas in the 1930s within the Hill Country or Edwards Plateau region. Although the cause was not known at that time, unusually high live oak mortality was reported during this period in the Austin area[9,10]. The semievergreen live oaks, *Quercus fusiformis* Small (plateau live oak) and *Quercus virginiana* Miller (coastal live oak), are considered the most valuable woodland and urban tree species in central Texas[11]. Populations of the oak wilt fungus gradually increased over the next 40 years until they reached observable levels in the 1970s[7]. An oak wilt epiphytotic ensued that continues to cause increasingly devastating losses to oak resources in that state. Oak wilt disease has cause well over $1 billion (US dollars) in economic damage to oak trees in Texas alone[7,12]. High economic losses occur due to affects on property values in urban areas with high-value trees which can be worth up to US $20,000 for a single tree in metropolitan areas[13]. Tree removal costs also can be substantial when they involve large trees. Replacement costs associated with replanting trees adds to the final expense of losing valuable landscape trees. The cause of oak wilt in Texas was officially diagnosed in Dallas, Texas in 1961[14] and the widespread mortality of oaks in central Texas finally was attributed to the same causal agent in 1977[15]. By 2004, oak wilt had been confirmed by laboratory diagnoses in infected oaks from at least 65 of 254 Texas counties.

A number of eastern states have previously developed oak wilt suppression programs to combat the disease as it continued to cause increasing oak mortality and developed into a major economic problem within each state. Oak wilt suppression programs were first organized (chronologically by indicated year) in the following states: West Virginia (1952)[16], Pennsylvania (1952)[17], Texas (1982)[18], Minnesota (1988)[19], Michigan (1991)[20].

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However, the cooperative oak wilt suppression programs in Texas and Minnesota are the only ones that have received federal assistance from the Forest Health Protection (FHP) branch of the USDA Forest Service. The cooperative suppression program in Texas, the Texas Oak Wilt Suppression Project (TOWSP), is administered by the Texas Forest Service (TFS). The history of operations, activities and expenditures of the TOWSP were reviewed previously[7].

The objective of this study was to provide a technical review of new oak wilt disease suppression methods that have resulted from expanded federal funding for oak wilt research and suppression operations by state forestry agencies in the United States within the past decade. Some key aspects of the biology of oak wilt and the causal agent of the disease are discussed first to provide useful references and background information that serves as a basis for understanding these latest suppression alternatives now available for controlling this devastating disease.

BIOLOGY OF THE OAK WILT FUNGUS

The biology of *C. fagacearum*, its interaction with numerous hosts in the eastern United States and its host-colonization characteristics has largely determined the ability of this pathogen to spread to its current range, cause widespread mortality, and affect the health of host trees over such a large region. The optimum temperature range for growth of the oak wilt fungus (approximately 22-27°C) was once considered a limiting factor that would prevent the fungus from expanding its range into oak resources in the southern United States[21,22], where ambient temperatures often exceed 35°C. The assumption that the fungus could not survive in higher-temperature environments was proven incorrect when it was found that *C. fagacearum* was responsible for causing an epiphytotic in central Texas[23] and that the biological habits of the fungus protected it from the hot, arid habitats of central Texas. The fungus builds up most of its inoculum in the roots of its hosts as a result of its movement and infection of new plants primarily through root transmission via root grafts and interconnected roots systems[8,24]. Root-borne inoculum is protected and insulated underground from relatively high ambient temperatures during hot summer months. *Ceratocystis fagacearum* is an ascomycetous fungus that also produces an asexual stage (*Chalara quercina* B.W. Henry)[25] capable of sporulating inside of host roots to produce small single-celled conidia that move easily and rapidly within the xylem transpiration stream, allowing colonization of the above-ground parts of the tree. The fungus also produces toxins from root inoculum that translocate to and accumulate in the leaves, causing veinal necrosis, marginal necrosis, and eventually defoliation[24]. Tree bark provides effective insulation from heat for xylem-borne inoculum in the boles of infected trees, but not in smaller branches[26,27].

Geographical distribution: The oak wilt fungus is not known to occur outside of North America. It is only known to be endemic to the United States where it continues to slowly expand its range within 22 eastern states (Fig. 1). In most cases, the expansion involves the addition of new counties within states in which the disease is already endemic. However, there are several examples that suggest the oak wilt fungus is slowly encroaching into the southern tier of states along the Gulf Coast. These include reports of new infection centers in a live oak population in Houston, Texas in 1993 and in Aiken County, South Carolina in 1998[28]. This probable expansion into the valuable coastal live oak (*Q. virginiana*) populations, extending along the Gulf Coast eastward to the lower Atlantic coast and north to Virginia, could eventually facilitate the colonization of interior forests of southern states.

Epidemiology and future expansion: The geographical expansion of oak wilt has been slow (often less than 17 m/year) in mixed interior hardwood stands because highly diverse forests tend to create space barriers due to the intermixing of nonhosts that hinder root transmission of the pathogen[7]. This may also be attributed to the poor localized distribution of the pathogen by inefficient nitidulid beetle vectors and the low incidence of random root grafting within mixed hardwood stands. By contrast, movement can be quite rapid (up to 45 m/year) in pure stands containing a single oak species, such as is common in the large stands of live oaks in central Texas[29].

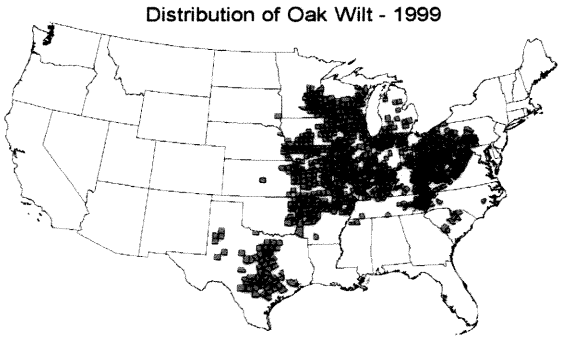

Fig. 1: Distribution of oak wilt incidence by county in the United States as of December 1999. Shaded counties have confirmed cases of oak wilt

The southern states also have a number of important red oak species that are highly susceptible to oak wilt, making this area vulnerable to future attacks[7]. Susceptible stands may be divided into coastal, upland and bottomland forest types. The large coastal populations of live oak (*Q. virginiana* Mill.) that are often planted as landscape trees with water oaks (*Q. nigra* L.) and other red oaks in coastal communities are at considerable risk. Within interior upland stands, black oak (*Q. velutina* Lam.), northern red oak (*Q. rubra* L.), scarlet oak (*Q. coccinea* Münchh.), southern red oak (*Q. falcata* Michx.), blackjack oak (*Q. marilandica* Münchh.), laurel oak (*Q. laurifolia* Michx.) and turkey oak (*Q. laevis* Walter) are susceptible species. Inland bottomland species such as cherrybark oak (*Q. falcata* Michx. var. *pagodifolia* Elliott), Nuttall oak (Q.), shumard oak (*Q. shumardii* Buckley), water oak and willow oak (*Q. phellos* L.) are equally susceptible. Any incidental introduction of *C. fagacearum* into hardwood stands containing high densities of these species or having high proportions of total basal area consisting of susceptible red oaks will likely sustain heavy damage. The creation of new infection centers puts adjacent stands in jeopardy of mortality for many years.

Host range and symptomology: The oak wilt fungus attacks fagaceous hosts including oaks (*Quercus* spp.), chestnuts (*Castanea* spp.), chinkapins (*Castanopsis* spp.) and tanoak (*Lithocarpus* spp.)[30,31]. Oaks are the most seriously affected and common hosts. All oaks are susceptible to oak wilt to some degree, but some species are affected more than others. For example, red oaks such as Texas red oak or Spanish oak (*Q. texana* Buckley = *Q. buckleyi* Dorr and Nixon), blackjack oak (*Q. marilandica* Münchh.) and Shumard oak (*Q. schumardii* Buckley) are the most susceptible species to oak wilt in Texas, but disease incidence is greater in live oaks due to their growth-form predispositions and shallow, extensive root systems[8]. Most live oaks defoliate and die over a 1-6 month period following initial appearance of symptoms. Suckers often develop on the bole and on larger limbs in the same year following defoliation as apical dominance is lost due to death of growing tips. However, some live oaks take longer to die and a few untreated trees may survive many years in various stages of decline. Other live oaks in oak wilt infection centers (perhaps 20%) escape infection or may be resistant to the fungus and apparently remain unaffected by the disease[24]. Red oaks seldom survive oak wilt and often die within 3 to 4 weeks following the initial appearance of symptoms. White oaks, such as post oak (*Q. stellata*), bur oak (*Q. macrocarpa*) and chinkapin oak (*Q. muhlenbergii*), are resistant to the fungus and rarely die from oak wilt. This difference in susceptibility between oak species in the white-oak group (subgenus *Quercus* section *Quercus*) from those in the red/black-oak group (subgenus *Quercus* section *Lobatae* Loudon), as defined by Nixon[32], has been attributed primarily to the formation of tyloses within the xylem vessels of white oaks[31]. Members of the red oak group generally lack tyloses, are much more susceptible to oak wilt and are killed more rapidly and more frequently than are members of the white oak group.

White oaks usually die slowly, one branch at a time, over a period of years[7]. Wilting and death of leaves on individual branches occurs in a fashion similar to that in red oaks, but usually progresses much more slowly. Affected leaves exhibit a pattern of discoloration similar to that seen in red oaks, proceeding from the margins to the base and sometimes interrupted by the leaf veins. Brown streaking in the outer growth rings is often readily apparent in infected white oaks.

The leaves on diseased Texas live oaks often develop foliar symptoms rapidly[24]. Leaf symptoms are produced primarily in the spring and fall. Leaves develop chlorotic (yellow) veins that eventually turn necrotic (brown), a diagnostic symptom known as veinal necrosis. Dead and dying trees are often seen near the expanding edge of oak wilt infections centers (Fig. 2). Defoliation may be rapid and dead leaves with brown veins often can be found under the tree for months after defoliation. The tree crown progressively thins out until the entire tree is defoliated. Leaves also may exhibit other patterns of chlorosis and necrosis, such as interveinal chlorosis, marginal scorch, or tip burn, but these symptoms are less common and unreliable for recognizing oak wilt in live oaks.

Fig. 2: Plateau live oak (*Quercus fusiformis*) trees in central Texas along the advancing edge of an oak wilt infection center. Healthy green trees (left side) are compared with symptomatic dying trees (right side)

Foliar symptoms of oak wilt in red oaks are less distinct than in Texas live oaks[24,31]. In early spring, young leaves simply wilt, turning pale green or brown. Mature leaves develop dark green water soaking symptoms or turn pale green or bronze, starting at the leaf margins and progressing inward. Diseased red oaks often can be spotted from a distance during summer months because of their bright autumn-like coloration in contrast to the surrounding greenery. Red oaks usually show rapid leaf discoloration and wilting. Often the initial symptom is a subtle off-green color shift that may be visible in the upper portion of the tree crown. This symptom appears in the northern part of the disease range from late June to early July. Shortly after this initial color shift, the leaves begin to wilt from the top of the crown downward. As the disease progresses, individual leaves become discolored, taking on a bronzed appearance. The discoloration progresses around the margins of the leaf from the tip to the base. Progressive discoloration may be interrupted by the leaf veins or may affect the entire upper portion of the leaf. Leaves are cast rapidly as the infection progresses and infected trees become almost entirely defoliated within a few weeks after symptom onset. Fallen leaves usually are brown at the tips and margins and sometimes green at the base and along the lower veins. Occasionally, the outer ring of vessels in diseased trees will be plugged with a brown substance that may be visible in cross sections as a ring or series of dark spots in the outer sapwood. In tangential cuts, a longitudinal streaking may be observed in wood exposed by removing the bark. Discoloration is most readily observed in tangential cuts of branches.

Signs of oak wilt: Fungal mats are special spore-producing structures that form on red oaks with advanced symptoms of oak wilt. The fungus sometimes forms fungal mats or pressure pads under the bark of these species, causing splits in the bark during the moist fall and spring months. Fungal mats usually form following infections that occurred the previous late summer or fall. Individual fungal mats produce spores for only a few weeks. Red oak infections in late spring and summer usually do not give rise to fungal mats due to high temperatures and low soil moisture conditions. The presence of fungal mats are reliable indicators for diagnosis and can be found by looking for inconspicuous narrow cracks in the bark of dying red oaks, leading to hollow areas between the bark and sapwood. They often have a distinctive odor similar to fermenting fruit. The grayish felty mats can be exposed for inspection by chopping away the loose bark. These structures are believed to be a major source of primary inoculum acquired by insect vectors in areas where red oaks are found[2], but fungal mats are likely to be of little importance as sources of inoculum in stands that lack red oaks. Fungal mats are not known to form on native white oaks or in live oaks. Although fungal mats are most commonly formed on standing trees, they also can develop on logs, stumps and fresh firewood cut from diseased red oaks.

Vector transmission: New primary oak wilt infection centers (foci) in nature probably form as a result of inoculum being carried by contaminated insect vectors, by air-borne spores that come in contact with exposed sapwood wounds, or by direct penetrations by insects that can bore directly through bark. The oak wilt fungus may be spread overland by insect vectors and by man through the movement of wood with fungal mats from infected red oaks to other locations. The fruity odor of fungal mats attracts many kinds of insects, the most important of which are believed to be sap-feeding beetles. The oak wilt fungus may be carried and transmitted by these small beetles when they emerge from mats and visit fresh wounds on healthy trees[33,34], but this has not been conclusively demonstrated. Dissemination of the oak wilt fungus also may occur over long distances by other possible insect vectors carrying spores from fungal mats of unharvested dead trees. Although oak sap beetles (Nitidulidae) and bark beetles (Scolytidae) have been most implicated in insect transmission, these insects generally are considered inefficient vectors. Other potential contributors to vector transmission could include animals such as root-feeding and bark-feeding insects, rodents and woodpeckers that cut into and expose sapwood to potential inoculum sources[7].

Root transmission: Subsequent spread of the fungus, once introduced by insect vectors, can occur vegetatively through root transmission, repeated cycles of sporulation on dying red oaks, and dispersal by insect vectors and human activity within the new area. The rate of root transmission is largely dependent on environmental conditions, stand density, species composition, soil depth and the tendency of oak species to form root grafts[8,35]. The fungus may spread from infected trees through root grafts into healthy adjacent trees to create expanding infection centers. Root grafts mostly form between oaks of the same species and red oaks form roots grafts more commonly than do white oaks. Root grafts are very rare between red oaks and white oaks. The rate of root transmission is slower in red oaks and occurs over shorter distances than in live oaks.

Live oaks are highly susceptible to root transmission because they tend to grow in large, dense clonal groups

(called motts) with often highly interconnected roots due to abundant root grafts. As a result, the entire mott can die. Live oaks that are killed by oak wilt often maintain viable root systems for many years. This is possible because the living roots give rise to root sprouts or suckers that supports the surviving root tissue following the death of the aerial parts of the tree. The foliage on this coppice growth often becomes symptomatic, but is able to keep the root system alive. These surviving roots systems may serve as effective reservoirs of the oak wilt fungus.

Mechanism of disease: *Ceratocystis fagacearum* initiates pathogenesis by several different mechanisms that occur simultaneously and result in the characteristic symptoms associated with oak wilt disease. The fungus tends to remain localized primarily in xylem vessels within the active (water-conductive) outer sapwood. Most of the inoculum potential for colonization is accumulated in the root system after root transmission[24]. The fungus becomes most active during the spring and early summer months when increasing ambient temperatures and abundant soil moisture allow the host tree to initiate transpiration, resulting in the production and expansion of new leaves. The fungus begins to accumulate mycelium and produce asexual endoconidia and toxins in root xylem that are released into the transpiration stream within xylem vessels. The asexual spores are carried up into the bole by transpiration and begin colonizing the lumen of vessel elements. The conidia germinate and the growth of hyphal inoculum in the vessels, in combination with mucilaginous gummy materials, clogs up and disables the water-conducting system, effectively reducing transpiration and water flow to the leaves. The reduction in water flow slows down leaf expansion and often results in smaller leaves that lose turgor pressure and begin to wilt. If transpiration is totally shut down early in the season, new leaves will not expand at all and the tree will not leaf out. If not, the toxins produced by the fungus eventually move into the leaves causing the rupture of cell membranes and electrolyte leakage, resulting in water soaking symptoms, chlorosis and eventually necrosis of leaf tissue. These mycotoxins eventually accumulate at the margins of leaves, at the end of the vascular system and cause marginal leaf chlorosis and finally necrosis. A host-wound response occurs as a consequence of the leaf tissue necrosis, causing ethylene to be produced that initiates leaf abscission and eventually defoliation.

EARLY DETECTION AND DIAGNOSIS

The ability to rapidly detect and locate new oak wilt infection centers within hardwood stands has been a key factor in applying timely and effective suppression treatments for new locations affected by the disease. The early application of disease suppression treatments to small infection centers makes containment easier, less costly and limits the amount of damage before the infection center expands to unmanageable sizes resulting in prohibitive costs.

Aerial infrared remote sensing: One of the best tools developed for detecting new oak wilt infection centers over large areas in Texas has been the use of color infrared remote sensing technology (Fig. 3). This method of surveying for oak wilt infected areas has been used extensively by the TFS in the TOWSP to identify new infection centers for closer inspection by ground confirmation surveys. The TOWSP is a large, well organized program developed over a relatively short time by a team of dedicated individuals. The program maintains a computerized database (the Texas Oak Wilt Information System) that keeps track of suppression activities and results using ground survey data taken from confirmed disease centers primarily in central Texas. Aerial surveys using infrared photography were conducted over more than 13 million acres from 1996-2000. These surveys recorded more than 7,000 mortality centers throughout the state. The mortality centers identified so far probably represent less that 10% of the disease present statewide. Aerial surveys are used to plan suppression operations and to study the rate of movement and changes in distribution of the disease over time[27,29]. The successful management of oak wilt depends on the ability to understand how the pathogen spreads in different oak species.

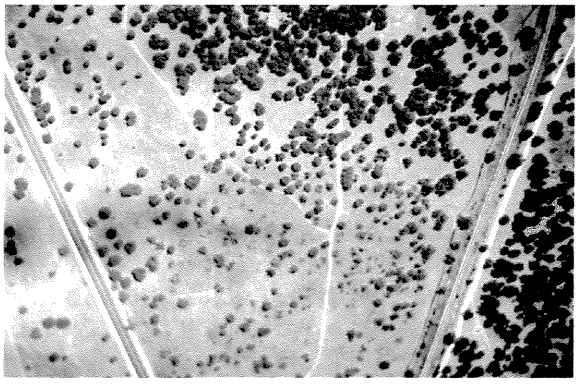

Fig. 3: Image of oak wilt infection center in central Texas taken by aerial infrared photography. Bright red and dark red trees are healthy; pink trees are declining due to oak wilt and grey trees are dead and totally lack foliage

Laboratory diagnosis: A correct diagnosis of oak wilt is essential for proper treatment. Foliar symptoms, patterns of tree mortality and the presence of fungal mats can be used as indicators of oak wilt. However, laboratory isolation of the fungus is usually required to confirm the diagnosis. A trained expert should be consulted when in doubt. The traditional method for laboratory diagnoses involves isolating the fungus from diseased tissues in pure culture. Sapwood samples taken from boles of symptomatic trees can be submitted to a plant disease diagnostic laboratory which are usually located at state universities. County extension agents, regional foresters and trained arborist should be consulted for proper collection and submission of samples.

A new more rapid and relatively nondestructive method was recently developed to diagnose oak wilt from wood increment cores taken from symptomatic, infected trees. This new technology called Conductive Polymer Analysis (CPA), a type of Electronic Aroma Detection (EAD), provides accurate and effective diagnoses without having to isolate *C. fagacearum* in pure culture[36]. Diagnoses using CPA are based on the recognition of aroma signatures of unique mixtures of volatiles (secondary metabolites) produced by the oak wilt fungus that are released into sampled headspace from infected sapwood cores.

CULTURAL AND SANITATION CONTROLS

Several sanitation measures have been useful for reducing the formation of new oak wilt infection centers in certain states. Sanitation measures include avoiding the wounding of trees when insect vectors are most active, management of firewood to avoid risks from inoculum sources, and killing infected red oak trees with fungal mats as inoculum sources. Cultural controls include trenching, installation of trench inserts, and pushing down or roguing buffer trees outside of infection centers in rural areas. Each of these control measures are covered in more detail in the following discussion.

Avoiding injury to healthy trees: Tree pruning in urban areas creates wounds that increase the susceptibility of trees to insect transmission particularly during spring months when insect activity is highest. Evidence of this activity is seen where new outbreaks of the disease occur around homesteads that are often traced to insect-infested firewood piles with active fungal mats. Thus, it is important to avoid injury to oaks during favorable conditions for infection. In the north, infections may occur in spring and early summer when fungal mats are present and potential insect vectors are flying. Favorable conditions usually occur between April 15 and July 1 in the Great Lake States and occur over a correspondingly longer period of time in the south[7]. In Texas, damage to oak trees should be avoided from February through June[37]. If construction activity or pruning are unavoidable, or if storms injure oak trees during the critical period, wounds should be treated immediately with a commercial tree paint or wound dressing. If trees are wounded during the dormant season in the North, tree paints are not necessary. However, judicious use during the rest of the year is recommended. Tree paint should be used immediately after trees are wounded, regardless of the time of the year, in the southern areas from Missouri to Texas.

Firewood management: Since the harvest and transport of oak firewood from oak wilt-endemic areas across county and state lines is not controlled by quarantine regulations, *C. fagacearum* may be spread freely in this manner[24]. Human activity, particularly movement of firewood, is a common and important means by which the oak wilt fungus has been introduced into new areas[7]. The harvesting of firewood from dead trees killed by oak wilt is common. The risk of spread occurs when infested unseasoned firewood is transported to other areas. Firewood often contains fungal mats that form under the bark of diseased red oak wood in wood piles. Insect vectors that often breed and form galleries in the wood are attracted to the sweet aroma of these mats and pick up spores produced on these structures and may be carried to wounds on healthy trees initiating new infections.

Diseased wood from any oak species should never be stored near healthy oak trees unless precautions are taken. It is best to purchase wood that has been thoroughly dried for at least one full year. If firewood from diseased trees is stored near healthy oak trees, it should be covered with clear plastic with the edges buried to prevent insects from leaving the pile[37].

Remove infected trees: Trees that are infected or have died from oak wilt should be removed and properly treated to prevent development of spore mats[37]. These treatments include debarking, chipping or splitting and drying the wood. Trees that die in summer should be removed and treated before the following spring, when new fungal mats can develop. If the wood is sufficiently dried, however, fungal mats will not develop. Removing a diseased tree that is still living may actually facilitate the spread of oak wilt by accelerating the movement of the fungus into adjacent trees that are grafted to it by the roots. To avoid this problem, disrupt interconnected roots before removing diseased living trees.

Infected red oaks that die in late summer or fall should be cut down and burned or buried soon after discovery to prevent insects from transmitting spores from fungal mats that may form on these trees in the fall or following spring[37]. If this is not possible, the trees should be injected with herbicide or deeply girdled with an ax and stripped of all bark 2 to 3 ft above the soil line. Drying of the wood before fall discourages formation of fungal mats. Regardless of season, all pruning cuts or other wounds to oak trees, including freshly-cut stumps and damaged surface roots, should be treated immediately with a wound or latex paint to prevent exposure to contaminated insect vectors.

Roguing trees in buffer zones: The pushing and removing of trees, often referred to as roguing, in advance of the fungus outside of infection centers is a practice that is used primarily in rural environments where lower value trees can be sacrificed to protect against the expansion of existing infection centers[2,37]. The removal of buffer trees between the advancing front of the infection center and healthy trees creates a distance barrier within which root transmission cannot occur via root grafts. This practice, which is usually done with a bulldozer, involves removing the roots as well so that inoculum cannot pass through possible grafted roots remaining in the ground that may provide a route for root transmission to healthy trees outside of the buffer zone. The size of the buffer zone necessary for control is determined by the rate at which the fungus is known to spread through the grafted root systems of a given oak species[7]. For example, in Texas live oaks, the oak wilt fungus may move 20-30 m or more per year in advance of symptomatic trees. Thus, a buffer zone of at least 30 m is required to minimize the risk of root transmission. In oaks of the upper Midwest, the fungus usually moves at less than 15 m per year, requiring a proportionally smaller buffer zone. Unfortunately, this method does not protect against insects vectors that may carry inoculum across the buffer zone.

Trenching to prevent root transmission: The practice of mechanically cutting root connections to control root transmission of the oak wilt fungus has been recommended for many years as the primary means of suppressing the spread of oak wilt in the United States[38]. Trenching to sever root connections in advance of the visible expanding edge of infection centers is the corner stone of oak wilt suppression efforts both in Texas and in Midwestern states with active control programs[7]. Since 1988, the Texas Forest Service which administers the Texas Oak Wilt Suppression Project (TOWSP) has installed over 650,000 m of trench to combat this disease[39]. Trenching has been a particularly important tool for dealing with the disease in highly valued live oak stands because root grafts result in extensively interconnected root systems. This tendency is further compounded by the growth habit of live oaks in forming root sprouts from mother trees that often give rise to large clusters of clonal trees with common root systems[24,40,41]. These natural growth tendencies increase the predisposition of live oaks to root transmission and have often resulted in dramatic mortality over very large areas.

Disrupting root connections between infected and healthy trees through trenching limits the spread of oak wilt and is an effective control measure. Trenches are usually cut completely around infection centers. Because the oak wilt fungus moves through roots in advance of visible symptoms, a buffer zone is required between the visible edge of the infection center and the trench just as was described above for roguing. In either case, unless infected and healthy trees are already spaced at a distance wide enough to serve as a buffer zone, a trench is usually cut between healthy trees at the outer edge of the buffer zone to sever root grafts between them before the fungus gets there. Interconnected root systems can be disrupted with a trencher, vibratory plow, backhoe, rock saw, or other equipment, depending on location and soil type[7].

The most common technique used in Texas is to cut trenches at least 1.2 m deep with trenching machines, rock saws, or ripper bars to sever roots. Trenches more than 2 m deep may be needed to assure control in deeper soils. Commercially-available root barriers may be inserted in the trench to reduce the potential for trench breakouts (see trench insert installations). Correct placement of the trench is critical for successful protection of uninfected trees because there is a delay between colonization of the root system by the fungus and appearance of symptoms in the crown. Therefore, all trees with symptoms should be carefully identified first. Then, the trench is placed a minimum of 31 m beyond these symptomatic trees, even though there may be "healthy" trees at high risk of infection inside the trench. Trees within the 31 m barrier, including those without symptoms, may be uprooted or cut down and removed to improve the barrier to root transmission. Tree removal is initiated after trenching, starting with healthy trees adjacent to the trench and gradually working inward to include symptomatic trees. The untreated trees immediately outside the treated area are closely monitored for several years. If the pathogen appears to have crossed a barrier, the same measures (new trenching and treatment of trees within the barrier) should be repeated while the diseased site is still small[37].

Breakouts caused by insufficient depth are due to the passage of root inoculum through deep root connections

or grafts across the trench zone that failed to be severed by trenching. Breakouts caused by improper trench placement occur when inoculum of the fungus has already passed through root connections across the trench before the roots were severed. Consequently, these two types of breakouts tend to occur soon after trench installation because root transmission is not impeded. However, breakouts that occur during and beyond the third year after trench installation are probably due to inoculum passing through new root connections forming across the trench from anastomoses of newly formed roots. The reason for this delay in root grafting across trenches is that the process of root regeneration within trench backfill soil takes at least 3-4 years after roots are cut by trenching before it is sufficient for root grafting. Small adventitious roots <1 cm in diameter commonly form from the ends of lateral live oak root branches severed by trenching. These small roots grow and accumulate within the trench in the loose backfill soil, which favors root growth relative to the hard, compact undisturbed soil.

Trench insert installations: Recent oak wilt suppression research conducted by USDA Forest Service scientists examined the effectiveness of trench inserts in providing long-term protection against root transmission of oak wilt[8]. In this 7-year study, trench inserts of two major types were tested, including water permeable and water impermeable landscape materials. These inserts were installed onto the inside wall of the trench on the side closest to the infection center. Water-impermeable materials, Geomembrane, 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. Root grafting can occur either because the insert was buried too deeply in the trench (soil above insert) or because soil depth to bedrock is greater than the width of the insert (soil below insert). By contrast, water-permeable inserts, such as Typar and Biobarrier, were more effective root barriers because they did not direct root growth from the point of root contact[8]. The additional, the minimal cost of trench inserts above trenching costs justified the expense in urban and rural homestead sites where valuable landscape trees require more protection and additional retrenching costs are avoided. Water permeable inserts also extended the effectiveness of trenches indefinitely, saving landowners the costs of installing expensive backup trenches when original trenches fail.

These results indicate that trench inserts extend the life of trenches principally by preventing the formation of new root graft connections across the trench between new adventitious roots forming from severed root apices within trench backfill soil. However, trench inserts are of little use if problems of trench depth and placement are not first controlled. No physical barriers will protect against root transmission if improper trench placement allows root transmission to occur before the trench is installed or insufficient trench installation depth allows root transmission to occur under the trench.

CHEMICAL CONTROL

Propiconazole (Alamo) is the only fungicide used as a preventative treatment to protect live oaks against oak wilt infection. Limited success also may be achieved in trees treated with therapeutic injections during the earliest stages of infection. The fungicide is injected into the tree's water-conducting vascular system (in sapwood) through small holes drilled into the root flares at the base of the tree. Treatment success depends on the health of the candidate tree, application rate and injection technique. Injection should be done only by trained applicators. Currently, fungicide applications using high-volume injections under pressure, low-volume injection using Mauget miroinjectors and ArborX microinjectors have been tested[8]. High-volume injection is the only method used in the TOWSP.

Fungicide injections do not stop root transmission of the fungus. This treatment, therefore, is used best in conjunction with trenching or to protect individual high-value trees in situations where trenching is impractical. Healthy live oaks at high risk of infection in advance of an expanding infection center are preferred candidates for injection. Foliar symptoms can be used in selecting trees as candidates for preventative or therapeutic treatments. A tree with foliar symptoms of oak wilt, as well as any non-symptomatic tree immediately adjacent to a tree with symptoms, should receive a therapeutic treatment. If symptoms are observed in more than 30% of the crown, it is unlikely a fungicide injection will be effective. Injections of non-symptomatic trees at greater distances from symptomatic trees (i.e., 23 to 46 m) will yield the best results for preventative treatments[37]. However, fungicide applications are practical only for high value trees due to the high cost of treatments.

Fungicide application methods utilizing high-volume intravascular injections of propiconazole (Alamo) have been employed by the TOWSP for oak wilt suppression in live oaks since 1990. The suppression program applies propiconazole primarily as a preventative treatment against infection to protect asymptomatic trees remaining inside of containment trenches. This high-volume/low-concentration method, previously employed for Dutch elm

disease control with benomyl, commonly prescribes an application of approximately 1 L of fungicide solution (3-10 ml L^{-1}) per 2.5 to 6.3 cm of DBH to be evenly distributed into injection ports on root flares at or below ground level. TFS personnel have estimated that fungicide injections applied by their field foresters, trained by experienced pathologists, have averaged about 50% success in preventing infections by *C. fagacearum* (Texas Forest Service, personal communication). This relatively low success rate is hard to rationalize given the very high sensitivity of the fungus to this triazole[12]. The inconsistency is most likely explained by the very minimal movement of systemic fungicides down into the roots via the symplast[42]. This hypothesis is supported by the observation that propiconazole root-flare injections do not appear to act as a barrier to inhibit transmission of the pathogen through root connections between treated and untreated trees[8,43]. Since propiconazole treatments have yielded inconsistent and unpredictable results as currently applied by high-volume root flare injections and do not prevent oak wilt root transmission as confirmed here, this control method was discontinued in 1997 from the federal cost-shared program offered through the TOWSP. Previously, the primary justification for incorporating Alamo treatments in the TOWSP was to provide an incentive to landowners to cooperate in recommended trenching operations, not because it was effective[39].

The problem with propiconazole treatments is not the fungicide since the oak wilt fungus is highly sensitive to triazoles in the 10-200 ppb range[24]. The ineffectiveness of fungicide injections is largely explained by the predominantly upward movement of triazole fungicides such as propiconazole (Alamo) in the vascular system of injected plants[44]. Thus, fungicide injections are ineffective in preventing root transmission because the fungicide does not come in contact with fungal inoculum in the roots. Thus, insufficient quantities of fungicide are translocated down into the root system to prevent viable inoculum from being translocated through root grafts into adjacent noninfected trees. Applications of propiconazole as a chemical control to prevent root transmission could be modified so that the material is introduced at the distal ends of the root system, as with soil application treatments[8]. The most effective results with propiconazole are achieved when it is applied annually as a soil drench within the dripline of the tree and over several consecutive years, immediately prior to challenge by *C. fagacearum* through root transmission (Wilson, unpublished data).

Propiconazole injections should be effective in preventing root transmission if adequate application methods are used that provide thorough treatment of the root system. Efficacy tests of new fungicide application methods using tracker dye in the fungicide solution have suggested that some of the fungicide moves down into the root system[45,46]. Some basipetal movement of the fungicide into the root system seems necessary to explain any level of control since the bulk of the inoculum resides in the roots and lower bole[24]. However, any downward movement probably can be attributed primarily to vapor phase activity rather than symplastic movement in the phloem since triazoles with sufficient vapor pressure such as propiconazole, etaconazole and triadimefon are well known for exhibiting vapor phase activity that generates biologically effective concentrations beneath the area of application[44]. Consequently, limited basipetal movement could explain the 50% success rate since such very low concentrations of propiconazole are needed to inhibit growth of the oak wilt fungus.

The success of TFS field foresters and certified Texas arborists in protecting uninfected live oaks by propiconazole injections have improved somewhat in recent years[7]. The improvement is related to changes in the formulation used and in the application rates allowable on the Alamo® label. Previously, the xylene-based (yellow) formulation was used. This formulation was replaced by the water-based, microencapsulated (blue) emulsifiable concentrate formulation that does not separate when diluted with water and causes fewer phytotoxic effects on callus regeneration at injection wounds. The microencapsulated formulation is more miscible in water and appears to be more effectively distributed in the tree. The allowable rates of application specified on the label also was changed recently from 3-5 to 6-10 ml L^{-1}. The doubling of the legal application rate probably does not change the proportion of the total material that moves into the root system, but it may allow a greater amount of the fungicide to move down by vapor phase activity since more active ingredient is present and available within the tree at each injection port.

BIOLOGICAL CONTROL

A type of natural biological control is known to sometimes occur after infection by *C. fagacearum* when the fungus causes the water content of sapwood to decline[5,47]. This lowering of sapwood moisture allows *Hypoxylon atropunctatum*, a common successional microbe, to rapidly colonize the sapwood and exclude the oak wilt fungus[48]. Unfortunately, Hypoxylon species are not useful as applied biocontrol agents because they cause white rots in oak sapwood. Endophytic bacteria isolated from the sapwood of live oak trees, including

species of *Bacillus pumulis*, *Pseudomonas denitrificans* and *Erwinia herbicola* also have been investigated as potential biocontrol agents[49-51]. These microbes produced either siderophore-like compounds or antibiotics that inhibited growth of the oak wilt fungus *in vitro* (fungistatic), but proved to be ineffective for oak wilt control because they failed to reproduce and colonize the xylem, particularly in the roots, and they were not persistent in sapwood at low water potentials[51,52]. Another problem was that the biologically active metabolites produce by these bacteria were not fungicidal. Similar studies indicated that *Pseudomonas cichorii* also was ineffective in protecting mature live oaks against development of oak wilt disease[53]. Endophytic microbes with the ability to systemically colonize sapwood would be particularly applicable to oak wilt control in live oaks because of the growth habit of these hosts with their highly interconnected root systems. The high incidence of root grafting and common root systems in these oak species make them readily suitable for facilitating the dispersal and colonization of all trees contained within a mott, resulting from a single inoculation with a microbial biocontrol agent[24].

HOST RESISTANCE

Oak wilt disease is generally known to affect about 80% of oak trees in a stand challenged with active oak wilt infection centers. The remaining trees either are escapes that are not exposed to infection by chance, or possess some level of resistance to infection. Relatively little research has been done to find sources of oak wilt disease resistance in oaks. Some initial work focused on the possibility of finding disease resistance in oak populations in Texas by the selection of progenitors from survivor trees within oak wilt infection centers[54]. Such progenitors tended to die at faster rates, but resprouted with healthy shoots more vigorously than seedlings from unaffected populations[55]. This apparently acquired hypersensitive response of post-epidemic plants relative to pre-epidemic live oak populations was unexpected because it is a resistance mechanism that apparently allows young seedlings to recover from infection, but never allows the tree to grow to maturity if subsequently challenged by the pathogen[2]. Problems associated with the screening process to find lines of live oak trees with truly durable genetic resistance and the long time period needed to generate mature oak trees from resistant seedling cultivars for subsequent field testing, has made the development of genetically superior trees with oak wilt resistance a very slow process. Thus, finding host resistance to oak wilt among oak species must be considered a potential long-term solution due to the slow growth rate of these hosts[24]. However, the large amount of variation in survival and implied resistance among families of live oaks indicates that selection for resistance is feasible[56]. Further work is needed to identify the genetic basis and determine the mechanisms involved in oak wilt disease resistance.

INTEGRATED PEST MANAGEMENT

The TOWSP provides a good example of a suppression program that utilizes an IPM approach to oak wilt suppression. A chronological summary of the incidence and impact of oak wilt in Texas since inception of the TOWSP demonstrates the dramatic progression and devastating potential of oak wilt (Table 1). Between 1988 and 2000, the number of confirmed infection centers increased almost 1100% and the area affected increased from about 450 ha to over 5800 ha. The estimated rate of damage accumulation within infection centers has approximately doubled every two years during that period. The damage estimates are based on the total expenditures for control, the percentages of centers treated, a cost:benefit ratio of 1:6 and the assumption that 80% of trees within centers will die of oak wilt.

The major control strategies used by the TOWSP have included trenching, removal of symptomatic red oaks, and tree injections with fungicide. The oak wilt fungus commonly moves from infected trees to adjacent healthy trees through root grafts or fused common root systems. Trenches are cut into the soil to a depth of 1.2-2 m completely around infection centers to sever root graft connections in advance of the fungus for prevention of root transmission. Trenching continues to be the most effective and predominant method used to control the disease. Over 760,000 m of trench were installed through 2000, treating about 45% of confirmed infection centers (Table 2). Trenching was about 67% successful in stopping the spread of encircled infection centers by 1994[39]. Since 1994, 76% 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 removal of over 3 thousand infected red oaks hitherto was done with hopes of slowing down the formation of new infection centers, although red oak removal has not yet been demonstrated to significantly affect disease epidemiology in Texas. However, red oak removal apparently has helped in some states. Injections of more than 7,000 high-valued trees with a systemic fungicide (Alamo) by 2000 saved some trees from further

Table 1: Incidence, severity and impact of oak wilt disease in Texas, 1988-2000

Year	Counties affected[1]	Oak wilt centers	Area affected (ha)[2]	Biannual damage (US $1,000s)[3]	Total damage (US $1,000s)[4]
1988	35	362	453	645	48653
1990	46	714	830	1225	49878
1992	51	1261	2415	4892	54770
1994	54	2084	3699	7337	62106
1996	55	2781	4639	8229	70335
1998	59	3285	5112	15746	86081
2000	60	3903	5811	16083	102165

[1]Includes only those counties with confirmed cases of oak wilt as determined by isolation of *C. fagacearum* in pure culture. Data were not made available for summarization after 2000.
[2]Cumulative area affected only within confirmed oak wilt infection centers. Tree values ranged from $750 per acre in rural areas up to $60,000 per acre in urban areas.
[3]Cumulative damage estimates based on total expenditures for oak wilt control, percentage of oak wilt centers treated, an average cost-benefit ratio of 1:6 and the assumption that an average of 80% of the trees within trenched infection centers died of oak wilt.
[4]Cumulative total takes into account only damage that occurred within confirmed oak wilt infection centers. Damage for 1988 includes losses accrued prior to and including that year.

Table 2: Summary and costs of oak wilt suppression efforts in Texas, 1988-2000

Year	Percentage of centers trenched	Trench installed (1,000s m)[1]	Infected red oaks removed	Oaks injected with Alamo[2]	Cost of control (US $1,000s)[3]
1988	3.3	4.8	74	0	336
1990	15.7	42.8	142	77	1630
1992	33.4	206.4	1336	2135	3092
1994	40.0	393.7	1939	5862	5178
1996	44.0	559.8	2157	7188	6444
1998	44.8	650.7	2796	7234	8036
2000	45.3	764.0	3042	7234	9636

[1]Cumulative total linear feet of trench installed to control root transmission from infected trees within infection centers. Data were not made available for summarization after 2000.
[2]Alamo® (propiconazole) was the only registered fungicide used by Texas Oak Wilt Suppression Project (TOWSP) personnel to control oak wilt.
[3]Cumulative total costs for oak wilt suppression by the Texas Forest Service are based on expended funds from a combination of sources including: 50% from the federal government (U.S. Forest Service), 28% from the State of Texas, 19% from private landowners and 3% from the City of Austin. Costs include direct suppression costs, operating expenses, administrative overhead, staff salaries and employee benefits.

decline due to the disease, but in many cases only delayed the disease process.

There have been a number of problems associated with the development and implementation of an effective oak wilt control program in Texas. Some of the key problems include the large buildup of the disease before a control program was initiated, the late diagnosis of the disease (not recognized until 1977), the learning curve associated with implementation of suppression measures, and problems with specific control methods themselves. The most immediate problem now, however, is the insufficient resources to adequately address the problem. The TOWSP control program simply is not large enough to handle the problem. With the current approach, it is estimated that the program is treating only 150-200 oak wilt centers each year (TFS personal communication). However, 250-400 new confirmed centers are detected each year. At the current rate of treatment, the program will not be able to keep up with the growth of the epidemic.

Any successful control program for oak wilt control depends on an integrated program incorporating measures from several different approaches. To summarize, TOWSP operations currently involve four primary IPM approaches for oak wilt management in Texas[37]. The first approach attempts to prevent the formation of new oak wilt infection centers by eliminating diseased red oaks, handling firewood properly, and painting wounds on healthy oaks. The second approach involves trenching or other measures to disrupt root connections responsible for root transmission of the pathogen. The third approach consists of applications (injections) of the fungicide propiconazole (Alamo) into individual, high-value trees to help reduce crown loss. Finally, the fourth approach is to plant resistant or immune tree species to avoid the disease. These measures will not cure oak wilt, but will significantly reduce tree losses. However, early detection and prompt action is an essential first step before successful management of oak wilt can be undertaken using these suppression approaches. The specific measures taken depend on individual circumstances, but should include appropriate combinations of the following:

Prevention of new infections:

- Cut and dispose of diseased red oaks immediately
- Avoid wounding oak trees, including pruning, when vectors are active
- Restrict intercounty transport of *C. fagacearum*-infested firewood by state quarantines
- Handle oak firewood cautiously, burn before spring and never store unseasoned oak wood from infected trees near healthy oaks
- Cover unseasoned firewood with clear plastic and bury the edges of the plastic

Stop root transmission:

- Cut a trench around the entire perimeter of infection centers at least 1.2 m deep and 31 m beyond symptomatic trees to sever root grafts and interconnected root systems
- Install water permeable inserts into trenches to prevent trench breakouts
- Cut or uproot all trees within the 100-ft barrier (except those injected with fungicide)

Treat high-value oaks with fungicide:

- Identify susceptible, high-value oak trees near expanding oak wilt infection centers

- Consult a trained and licensed arborist for injections of propiconazole (Alamo)

Select resistant tree species:

- Plant tree species that are resistant or immune to oak wilt
- Choose a diversity of tree species adapted to the locality during landscape planning
- Avoid wounding susceptible oaks during planting

DISCUSSION

Recent improvements in oak wilt management in the United States have resulted from modifications of existing control strategies, empirical advances arising from experiences gained during implementation of suppression programs, new research developments utilizing new suppression technologies, and effective integration of control measures. For example, applications of new research developments such as installing trench inserts into primary trenches to preclude breakouts due to root grafting across trenches have significantly reduced incidences of oak wilt root transmission.

Postsuppression evaluations (PSEs) made by the TOWSP during the first 10 years of the suppression program indicated that of the 30% of trenches that failed, approximately 80% of the breakouts occurred within the first 2-3 years after trench installation (Texas Forest Service, unpublished report). The majority of these breakouts were likely due to insufficient trench depth or improper trench placement. Since 1994, PSEs have indicated that the primary trench failure rate has been only 24%. This improvement has been attributed to installation of deeper trenches and experience gained in proper trench placement[39]. This also was likely the result of a previous increase in the buffer zone in 1990[57].

Many problems associated with trench suppression in the past have been resolved with experience gained during implementation of the TOWSP. Problems such as improper placement of trenches or insufficient depth largely have been resolved with appropriate adjustments in these suppression parameters. For example, the buffer zone or barrier distance gap that is used between the visible edge of the infection center and the position for primary trench installation was increased from 15 to 23 m and then to 31 m since the program was initiated. This was necessary because the fungus was observed to move through grafted root systems in advance of crown symptoms at a rate up to 25 m or more per year. Trenching depth was initially set at about 1m but now the recommended depth has been extended to a 1.2 m minimum. The rates of trench failures have declined consistently since inception of the TOWSP. However, the law of diminishing returns has prevented further advancements in trenching success using current methods. Thus, there has been a need for new technology to improve on existing methods to allow for further progress in the refinement of this cultural control.

The 76% trenching success rate of TOWSP, however, does not appear to be stopping the progression of this disease in Texas at the state-wide level[7]. Some areas of Texas have large aggregates of infection centers so dense that it is impractical to install suppression trenches[39]. Thus, the unchecked progression of oak wilt in Texas has been due to the inability to treat many disease centers, the unchecked expansion of infection centers at failed trench sites and more importantly, the creation of new infection centers by currently unknown processes which might involve insect vector activity and intercounty transport of infested firewood. However, all known potential vectors to date, such as nitidulid beetles, are extremely inefficient and are unlikely suspects to explain the discovery of at least 250-400 new infection centers each year of which only 45% are treated annually[7]. Other as yet undiscovered sources of long-distance (overland) spread are likely involved that will ultimately explain this rapid appearance of new oak wilt disease centers.

The combined effect of using 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 as these methods are implemented. The improved trenching methods using trench inserts alone could potentially save hundreds of millions of dollars in tree removal costs and property value depreciations for Texas landowners. The technology is equally applicable in other areas of the United States affected by this disease, especially urban areas, and could provide substantial savings in these areas as well[58,59]. These water permeable trench inserts provide cost-effective insurance because they may be installed at a fraction of the cost (less than 10%) of installing expensive backup trenches when primary trenches fail[8].

Even though oak wilt currently occurs only in the United States, there is considerable concern abroad that it could spread to other countries. The destructive potential of this disease has been recognized internationally for many years due to the threat it poses to oak resources in Europe and Asia[60,61] and because of the ability of *C. fagacearum* to sometimes survive in air-dried lumber[62]. Quarantine regulations that restricted the export

of unfumigated oak lumber and logs to Europe and Asia were established soon after the discovery of oak wilt in America. Mistretta *et al.*[63] provides more references on all aspects of oak wilt biology and control both globally and in the United States. The current concerns that many foreign officials have for the potential importation of the oak wilt fungus into Europe and Asia should be shared by forest managers in other areas that do not yet have oak wilt. Since the oak wilt fungus is potentially one of the most destructive forest pathogens known, any threat of its expansion into oak forests in unaffected areas should be taken seriously, particularly outside of the United States.

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