



Review

A review of oak wilt management: A summary of treatment options and their efficacy

Karrie A. Koch^{a,*}, Gina L. Quiram^b, Robert C. Venette^c

^a Department of Entomology, 219 Hodson Hall, 1980 Folwell Ave., Saint Paul, MN 55108, USA

^b Department of Ecology, Evolution, and Behavior, 100 Ecology Building, 1987 Upper Buford Circle, Saint Paul, MN 55108, USA

^c USDA Forest Service, Northern Research Station, 1561 Lindig Ave., Saint Paul, MN 55108, USA

ARTICLE INFO

Keywords:

Ceratocystis fagacearum
Forest pathogen
Invasive species
Pest management
Quercus

ABSTRACT

Oak wilt, caused by the invasive fungal pathogen *Ceratocystis fagacearum* (Bretz) Hunt, is a serious and fatal disease of oaks, *Quercus* spp., with red oaks (section *Lobatae*) generally being more susceptible than white oaks (section *Quercus*). Oak wilt was first recognized in North America in 1944 and has since been confirmed in 24 eastern, midwestern, and southern states. The purpose of this paper is to review relevant literature on the efficacy of oak wilt treatment options. Root disruption, sanitation, and chemical control methods have been used most often to manage the disease. Root disruption has primarily focused on severing root grafts between oaks. Sanitation has focused on removal and proper disposal of potential spore-producing trees. Chemical control has focused on the use of systemic triazole fungicides. Efficacy of treatments can vary significantly, for example from 54% to 100% for root graft barriers. Educational programs can increase prevention efforts, detection, compliance with recommended management methods, and overall efficacy. Our review confirms that management programs should address underground and overland spread and include an educational component.

Published by Elsevier GmbH.

Contents

Introduction	1
Life history of <i>Ceratocystis fagacearum</i>	2
Biology and symptoms	2
History and distribution	2
Oak wilt management	2
Root disruption	3
Trench inserts	4
Sanitation	4
Chemical control	4
Preventive propiconazole treatments	5
Therapeutic propiconazole treatments	6
Other factors impacting efficacy of propiconazole	6
Propiconazole and fungal mat production	6
Prevention and education	6
Summary	7
Acknowledgements	7
References	7

Introduction

Oak wilt, caused by the invasive fungal pathogen *Ceratocystis fagacearum* (Bretz) Hunt, is a serious disease of oaks (*Quercus* spp.) across portions of the eastern and central US. In August 2008, oak wilt was first detected in New York State (Jensen-Tracy et al.,

* Corresponding author.

E-mail address: kochx141@umn.edu (K.A. Koch).

2009). As oak wilt spreads, forestry professionals unfamiliar with the disease will need information regarding its biology and management. We intend for this review to be an initial resource providing an overview of this information. This review does not provide specific management recommendations, as these vary by region. We have included a brief review of the life history of *C. fagacearum* and a summary of control options and the role of prevention and education in oak wilt management. Previous reviews of oak wilt biology and management have been published by Appel (1995a), Juzwik (2000), and Wilson (2005).

Life history of *Ceratocystis fagacearum*

Biology and symptoms

Oak wilt is a vascular disease infecting 33 oak species (Juzwik, 2000). Generally, infected red oaks (section *Lobatae*) have more severe symptoms than white oaks (section *Quercus*) and experience rapid and frequent mortality (Juzwik, 2000). However, white oak species native to Europe (*Quercus petraea* (Matt.) Liebl., *Q. robur* L., and *Q. pubescens* Willd.) are as susceptible to oak wilt as North American red oaks (MacDonald et al., 2001). Live oaks (section *Quercus*) are more susceptible than white oaks but less so than red oaks (Appel, 1995a).

Ceratocystis fagacearum invades the vascular system of infected oaks producing hyphae, spores, and metabolic by-products which clog xylem vessels. In addition, tyloses, defensive structures produced by oaks in response to various stressors including *C. fagacearum*, also contribute to xylem blockage and the development of characteristic wilt symptoms (Juzwik, 2000). Wilt begins at the crown and manifests differently in red, white, and live oaks which complicates detection of infected trees (Juzwik, 2000). Upper-most leaves of infected red oaks begin to wilt at the tips of leaf lobes causing discoloration (i.e., bronze, brown, or black) and leaf deformation. Discoloration moves down from the leaf edges toward the midrib (Fowler, 1953). In infected red oaks, defoliation occurs rapidly and trees usually die within a year of showing symptoms (Fowler, 1953). White oak foliar symptoms are similar to those in red oaks but develop slowly, often on only a few branches annually (Fowler, 1953; Juzwik, 2000). In live oaks, symptoms begin with vein discoloration (Juzwik, 2000). Vascular discoloration of sapwood can occur in infected trees (Juzwik, 2000).

After an infected red oak dies, *C. fagacearum* produces fungal mats under the bark which produce infective spores. These mats are usually formed on the trunk and large branches of recently killed red oaks. Pressure pads formed inside these mats force open the bark and provide access for insect vectors to contact spores (Juzwik, 2000). Spore mats are formed only once, producing spores for about 2–3 weeks depending on temperature, and only on red oaks (Appel, 2001; Wilson, 2005). Mats are commonly found in the spring and fall on trees killed by oak wilt the previous year (Wilson, 2005). The timing of mat formation depends on geographic location, environmental conditions and time of symptom development and tree death (Appel, 2001). They are more common on standing trees but can also be found on felled trees and firewood (Wilson, 2005). Human transport of firewood can be responsible for oak wilt outbreaks in once disease-free areas (Wilson, 2001; Haugen et al., 2008). Trees capable of producing spores have been called potential spore-producing trees (PSPT), a term used commonly when describing oak wilt management strategies.

The oak wilt fungus spreads to susceptible trees in two ways: via dissemination of infectious spores by insect vectors (Juzwik, 2000) and via underground root grafts. Transport of infected firewood can move the pathogen long-distances (Wilson, 2001), but insects are still needed to move the pathogen from infected

wood to uninfected trees. In the upper Midwest and Texas spread via insect vectors has been mainly attributed to nitidulid beetles which are attracted to fungal mats produced by *C. fagacearum* and fresh tree wounds. Disease transmission can occur if a beetle acquires spores prior to visiting a fresh wound on an uninfected tree (Juzwik and French, 1983). Fungal transmission by insect vectors in the Midwest and Texas occurs in spring to early summer and late winter, respectively (Ambourn et al., 2005; Hayslett et al., 2008a). Studies in Missouri, Texas, and Minnesota have shown that *Colopterus truncatus* Randall is the most common nitidulid associated with fresh oak wounds and is the species most frequently carrying *C. fagacearum* spores, suggesting that it may be an important vector (Juzwik et al., 2004b; Hayslett et al., 2008a, 2008b).

Underground spread of oak wilt occurs via common root systems in live oaks and root grafts, which occur predominantly between trees within the same section (*Quercus* or *Lobatae*) and are especially common between trees in the red oak group. Root grafts are estimated to be responsible for 90% of tree infections in Minnesota (Cook, 2001; Wilson, 2005). The roots of infected red oaks can harbor and transmit the fungus through root grafts for several years after tree death (Gleason and Mueller, 2005). Because the infection spreads from tree to tree through root grafts and common root systems of live oaks (Appel, 1995a; Appel et al., 1989), infection is often observed in groups of trees called infection centers (O'Brien et al., 2000). The spread of oak wilt can be quantified by changes in the area of existing centers or by the number of new infection centers. Underground spread generally leads to expansion of existing infection centers, and overland spread is responsible for new infection centers (Juzwik, 2000). The relative importance of the two means of spread can change with distance between infected and susceptible trees, root graft frequency, frequency of fungal mat formation, oak species composition, and environmental conditions, especially soil type (Appel et al., 1987; MacDonald and Double, 2005).

History and distribution

Oak wilt has been detected in 861 counties of 24 US states (USDA FS, 2005), including the recent find in New York (Jensen-Tracy et al., 2009). Dying oaks with wilt symptoms were reported as early as 1881 in Wisconsin (Warder, 1881). In 1944, when the disease was first formally described, oak wilt had only been reported from Wisconsin, Minnesota, Illinois and Iowa (Henry et al., 1944); however, it is impossible to determine when oak wilt first appeared in the US (Juzwik et al., 2008). Today the disease is particularly problematic in Texas, Illinois, Iowa, Wisconsin, Michigan, and Minnesota (Billings, 2000). Range expansion to the west is possible as several oak species in California are susceptible (Appel, 1994).

Although the pathogen is generally thought to be a non-native, newly evolved strain, or newly emerged species, the origin of *C. fagacearum* is not known. Several lines of evidence indicate that *C. fagacearum* may be an invasive alien species to the US, including limited genetic variation, a characteristic of non-native populations arising from a single introduction (Juzwik et al., 2008). Evidence points to Mexico, Central America, and northern South America as the potential geographic origin of the fungus (Juzwik et al., 2008).

Oak wilt management

Oak wilt management is most often an integration of root disruption, sanitation, and chemical application. The goal of root disruption is to sever root connections, which can be root grafts in any oak species or common root systems in live oaks, between

Table 1
Efficacy* of treatment methods to achieve various management goals for oak wilt.

Management goal by species group	Efficacy of treatment method			
	Root graft barriers/ trench inserts ^a	PSPT removal/ sanitation	Preventive fungicide injections	Therapeutic fungicide injections
Live oaks				
Eradicate ^b <i>C. fagacearum</i> from a tree	–	–	NA	–
Preserve ^c infected oaks	–	–	NA	+
Prevent ^d infection of individual tree	–	–	–	NA
Reduce ^e underground spread	++	–	–	–
Deciduous red oaks				
Eradicate <i>C. fagacearum</i> from a tree	–	–	NA	–
Preserve infected oaks	–	–	NA	+
Prevent infection of individual tree	–	NA	–	NA
Reduce underground spread	++	–	–	–
Reduce inoculum availability for overland spread ^f	–	++	++	++
Deciduous white oaks				
Eradicate <i>C. fagacearum</i> from a tree	–	–	NA	–
Preserve infected oaks	–	–	NA	++
Prevent infection of individual tree	–	NA	+	NA
Reduce underground spread	++	–	+	–

* ‘++’=management goals met $\geq 50\%$ of the time; ‘+’=management goals met $< 50\%$ of the time; ‘–’=treatment method does not address or is not effective in meeting the management goal; all classifications are according to published literature and assuming that treatment method is properly executed.

^a Trench insert efficacy data was collected in Texas live oak systems and should only be applied to that system (live oaks).

^b Eradicate the fungus from an infected tree. The tree would survive, not require further treatment, and not be a source of oak wilt infection.

^c Significant proportion ($\geq 75\%$) of trees can survive indefinitely with consistent monitoring and treatment (i.e., for high value trees).

^d Significantly reduces infection of healthy oaks.

^e Significantly reduces underground transmission of an infected tree to a healthy tree.

^f Significantly fewer spore mats produced.

infected and healthy trees. Sanitation measures focus on eliminating potential inoculum sources. Chemical applications most commonly consist of intravascular (at the stem or root flare) injection of propiconazole.

Assessments of efficacy of oak wilt treatments depend on management goals. Complete eradication of the pathogen from a tree to preserve it and avoid the need for future treatments is often a desired goal. Unfortunately, no management option exists to achieve it. Other management goals might include: survival of infected oaks, protection of individual trees, stopping or slowing the expansion of an infection center, or preventing the formation of new infection centers (Table 1). Deciding on a management goal will depend on location and how the trees are used and valued.

Once the management goal is identified, managers will want to select effective treatments. We have summarized published efficacy data for common treatments. We consider a treatment within an individual study “effective” if the treatment confers a statistically significant advantage compared with an untreated control. If results for untreated controls are not reported, we still consider the treatment potentially effective if it achieves the desired outcome in $\geq 75\%$ of applications, often measured at the scale of an individual tree. When conflicting results about a treatment are reported, we consider it effective, if it meets the above standards more often than it does not (Table 1). In specific reference to chemical treatments, we interpreted the efficacy of single applications only. We note that sequential applications may increase effectiveness.

Root disruption

The goal of root graft disruption is to stop the underground expansion of existing infection centers. Root graft disruption can be accomplished by installing a trench or plow line that severs and separates the roots of healthy and infected trees. Trenches or plow lines can be primary or secondary (Fig. 1). Placement and

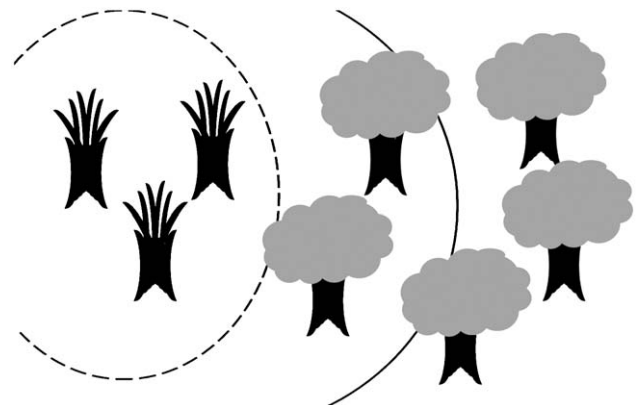


Fig. 1. An example of primary and secondary line placement (figure adapted from O'Brien et al., 2000). The primary line (solid) separates trees outside of the potential root grafting distance from the infection center and asymptomatic trees within root grafting distance. A secondary line (dashed) can be placed inside the primary line between symptomatic trees and apparently healthy trees inside root grafting distance.

depth of the primary line will vary but in any case the primary line should contain all symptomatic trees and often contains all asymptomatic trees within root grafting distance of infected trees. Trench inserts in the primary line, or a secondary line inside the primary line between infected and healthy trees, can be installed to increase the efficacy of root graft barriers (O'Brien et al., 2000; Wilson and Lester, 2002). Vibratory plows, bulldozers with ripper blades, or backhoes are used to disrupt grafted or common root systems (Appel, 2001). Equipment used varies regionally with its availability and cost (Shelstad, 1988).

The placement of the primary line is critical in root graft barrier installation (Fig. 1). If the primary line fails to extend beyond the radius of currently infected trees, containment of oak wilt is unlikely. The currently infected radius includes the roots and root grafts of all wilting trees and asymptomatic but infected

trees. The size of the infected radius is variable and depends on the rate of spread via root grafts and whether common root systems are present (Appel et al., 1989). For example, when common root systems occur, as in live oaks in Texas, *C. fagacearum* can spread up to 40 m per year (Appel et al., 1989); however, when common root systems are lacking, as in deciduous oaks in the Midwestern US, spread is typically less than 15.2 m per year (Wilson, 2001). Some authors suggest that the recommended primary line placement, to contain the currently infected radius, is a function of soil type and combined tree size (Bruhn and Heyd, 1992). Others suggest an absolute distance be used, for example between 15.2 and 18.3 m ahead of active symptoms in the Midwestern US to account for annual rates of underground advance (Gleason and Mueller, 2005; Wilson, 2005).

Early reports suggested that vibratory plowing is effective 54% of the time, with the infection spreading beyond the line once in every 46.6 m of line installed with a 122 cm plow blade (Shelstad, 1988). As personnel installing root graft barriers gained experience and equipment was improved, treatment efficacy increased. In Texas, barriers installed between 1991 and 1994 were effective 67% of the time while 76% of barriers installed after 1994 were successful (Billings et al., 2001). In other reports between 88% and 100% of root graft barriers were successful at preventing local spread (Gehring, 1995; Cook, 2001). Eventually, re-grafting can occur and retreatment may be necessary.

Trench inserts

Because roots can form grafts across root graft barriers, some management programs have utilized trench inserts as an additional means of separating roots. The majority of trench insert efficacy data was collected in Texas in live oaks with common root systems. Water-impermeable inserts tend to direct root growth above or below the insert facilitating the formation of root grafts and the spread of oak wilt (Wilson and Lester, 2001). Water permeable inserts, on the other hand, extend the effective life of root graft barriers by several years, perhaps indefinitely (Wilson and Lester, 2002). In field tests in Texas, trenches with Biobarrier, Typar, and Geomembrane liners (30 mil.) were 100% effective at stopping disease progression beyond the trench line 6 years after installation (Wilson and Lester, 2002). The inserts are recommended in areas containing high value oaks in Texas as an alternative to secondary trenches (Wilson and Lester, 1997).

Evidence suggests that root graft barriers can slow or eliminate underground spread of oak wilt. Because of the potential for overland spread, root disruption alone will not prevent disease transmission. Management programs including PSPT removal and sanitation can address the risk of overland spread. It is important that root graft barriers are installed prior to PSPT removal as below ground fungal spread can be accelerated by above ground cutting (Wilson, 2005).

Sanitation

Removal of PSPT and sanitation of infected wood is used to reduce available infective inoculum to prevent pathogen transmission by insect vectors. Two primary options exist in the removal of PSPT (O'Brien et al., 2000). The first option is the removal of all currently infected red oaks and subsequent annual removal of all red oaks that develop symptoms. The second option is a cut-to-the-line treatment which consists of removal of all oak trees (or just those in the same section as infected trees) that fall within an infection center, frequently delimited by the placement of a root graft barrier. Often the removal of only visibly infected oaks leaves some infected trees and may not stop spread. For example, removal of visibly infected trees did not reduce the

formation of new infection centers; however, removal of all oak trees within 15.2 m of a diseased tree did (Jones and Bretz, 1958). Similar results were found in Iowa where removal of all diseased and healthy oaks suppressed the spread of oak wilt whereas removal of only symptomatic trees did not (Young, 1949). In Minnesota, it is estimated that at least 1/3 of infected red oak trees have the potential to produce fungal mats the spring following infection, demonstrating the importance of PSPT removal (Cook, 2001).

Once a PSPT removal strategy has been implemented, further sanitation of infected wood is necessary. Simply felling infected trees is not sufficient. In Pennsylvania, 31% of trees felled 6 weeks after complete defoliation were able to form fungal mats (Morris, 1955). In an effort to prevent fungal mat formation, managers have used several sanitation methods including burning, debarking, girdling, drying, chipping, covering wood with plastic, and chemical treatment (Gillespie et al., 1957; Jones and Bretz, 1958; Boyce, 1959; French and Juzwik, 1999; Cook, 2001; Wilson, 2001, 2005; Gleason and Mueller, 2005; Greene et al., 2008). Sanitation measures are recommended for all material 7.6 cm in diameter or larger (Bruhn, 1995).

Studies have shown that proper disposal of infected wood can reduce fungal mat formation. While most published efficacy information pertains to girdling, girdling alone may not be the best sanitation method. Girdling infected trees in West Virginia before August in the initial year of symptom occurrence reduced fungal mat formation (Gillespie et al., 1957). Girdling standing Texas red oak (*Q. buckleyi* Nixon and Dorr) significantly reduced the number of branches forming fungal mats and those producing mats produced 50% fewer (Greene et al., 2008). Girdling treatments when combined with chemical debarking has been shown to be 100% effective at suppressing mat formation in red oaks (Morris, 1955). However, fungal mat suppression does not necessarily translate into landscape control of oak wilt. A study in Texas demonstrated that reducing the number of fungal mats does not reduce infections in treated areas (Greene et al., 2008).

Proper installation of root graft barriers has been shown to reduce the underground spread of oak wilt when taking into account local soil conditions and rates of local spread. Removal of PSPT followed by sanitation may reduce overland spread of oak wilt and the formation of new infection centers. Once a tree is infected, the key is to complete PSPT removal and sanitation quickly to prevent fungal mat formation. Infected wood should never be stored near susceptible oak trees (Bruhn, 1995; Wilson 2001, 2005). Root graft barriers and sanitation address infection on a landscape scale. In some cases it may be appropriate to manage for individual tree survival. The integration of chemical control into management programs is a way to protect individual trees.

Chemical control

Fungicide treatments can be preventive (i.e., applied to an apparently healthy tree) or therapeutic (i.e., applied to an apparently infected tree). The desired outcomes for preventive treatments might include preventing infection, preventing symptom development or mortality and for therapeutic treatments might include preventing disease progression or mortality in a symptomatic tree or reducing spore mat formation. Because of the biological differences among live oaks, deciduous white oaks, and deciduous red oaks, the efficacy of fungicide treatments for one group should not be used to inform treatment decisions for any other. Study-specific application methods and concentrations are excluded from the text but are available in Table 2. Table 2 is not intended to directly inform treatment decisions, rather its

Table 2
Summary of fungicide treatment^a details for included studies.

Oak species treated	A.I. applied ^b	Volume ^c	Application details	Citation
Live oaks (section <i>Quercus</i>)	0.25–1.25 mg/tree	1.0–5.0 ml/tree	Injection of immature trees	Appel (1990)
	100–500 mg	1.0 l	Injection of mature trees	Appel (1990)
	0.131 mg/tree	1.0 ml/tree	Injection of immature trees 2 cm above soil line (organic solvent)	Appel and Kurdyla (1992)
	0.121 mg/tree	1.0 ml/tree	Injection of immature trees 2 cm above soil line (water-based)	Appel and Kurdyla (1992)
	50–900 mg	1.0 l	Injection of mature trees at root flares	Appel and Kurdyla (1992)
	467 mg	Not provided	Injection as part of Texas Oak Wilt Suppression Project	Billings et al. (2001) ^d
	115–435 mg	1.0 l	Injection of mature trees	Appel (1995b)
<i>Quercus virginiana</i>	10,400 mg/tree	1.0 l/tree	Soil drench with Tilt [®] at dripline	Wilson and Lester (1995)
	10,400 mg/tree	1.0 l/tree	Injection with Tilt [®] at bole	Wilson and Lester (1995)
	11,700 mg/tree	1.0 l/tree	Soil drench with Banner [®] at dripline	Wilson and Lester (1995)
	11,700 mg/tree	1.0 l/tree	Injection with Banner [®] at bole	Wilson and Lester (1995)
	23,400 mg/tree	1.0 l/tree	Injection with Banner [®] at bole	Wilson and Lester (1995)
	168 mg	0.39 l	High volume injection at bole	Wilson and Lester (1996) ^d
	421 mg	5.9 ml	Microinjection (ARBORx) at bole	Wilson and Lester (1996) ^d
	421 mg	0 ml	Microinjection (Mauget) at bole	Wilson and Lester (1996) ^d
	168 mg	2.0 l/tree	Intermediate volume soil drench at dripline	Wilson and Lester (1996) ^d
Deciduous red and white oaks	1400 mg	1.0 l	Injection of mature trees	Eggers et al. (2005)
<i>Q. macrocarpa</i> , <i>Q. alba</i> (section <i>Quercus</i>)	420 mg	Not provided	Injection of mature trees at root flares	Osterbauer et al. (1994)
<i>Q. ellipsoidalis</i> , <i>Q. rubra</i> (section <i>Lobatae</i>)	420 mg	Not provided	Injection of mature trees at root flares	Osterbauer and French (1992)
	2859 mg	1.0 l	Injection of mature trees at root flares	Ward et al. (2005a)
<i>Q. rubra</i>	2859 mg	1.0 l	Injection of mature trees at root flares	Peacock and Fulbright (2008)
<i>Q. velutina</i> (section <i>Lobatae</i>)	858 mg	1.0 l	Injection at high rate	Johnson (2001) ^d
	572–715 mg	1.0 l	Injection at medium rate	Johnson (2001) ^d
	429 mg	1.0 l	Injection at low rate	Johnson (2001) ^d

^a All applications made with Alamo[®] formulation (Syngenta Crop Protection, Greensboro, NC) of propiconazole unless otherwise noted.

^b Active ingredient (A.I.) applied per 2.5 cm of diameter at breast height unless otherwise noted.

^c Volume of water in which product is diluted per 2.5 cm of diameter at breast height unless otherwise noted.

^d Product label consulted to estimate A.I. applied.

purpose is to illustrate the range of chemical treatments studied and utilized for oak wilt management.

Preventive propiconazole treatments

Propiconazole is a fungistatic compound which inhibits *C. fagacearum* growth *in vitro* (Wilson and Forse, 1997) and is the primary fungicide used for oak wilt control. Preventive propiconazole treatments are not effective at preventing infection of live oaks or deciduous red oaks. Appel and Kurdyla (1992) found that although symptom development was delayed or arrested, 50–60% of live oaks treated with propiconazole prior to inoculation with *C. fagacearum* became infected. In Minnesota, 18% (Eggers et al., 2005) and 40% (Osterbauer and French, 1992) of red oaks treated preventively became infected within 2–5 years of treatment. When applied 2 weeks prior to tree inoculation with *C. fagacearum*, preventive propiconazole treatments did not prevent infection via root grafts in northern red oaks in Minnesota; however, symptom development was delayed for at least 24 months in all treated trees (Blaedow, 2009). Limited evidence seems to demonstrate that preventive treatments of white oaks can prevent infection as only 4% of white oaks preventively treated showed wilt symptoms 5 years post-treatment (Eggers et al., 2005). However, *C. fagacearum* is capable of infecting preventively treated white oaks (Eggers et al., 2005).

Preventive propiconazole treatments are effective at preventing or delaying symptom development and decreasing mortality of all oak groups studied. Live oaks of all ages showed 100%

protection from oak wilt symptoms when treated with propiconazole prior to infection (Appel, 1990). All treated live oaks showed significantly less crown loss and only 9% mortality when compared to untreated controls (70% mortality) (Appel and Kurdyla, 1992). In a check of 383 live oaks preventively treated with propiconazole in Texas 1–4 years earlier, 74% of trees still had < 30% defoliation (Billings et al., 2001).

From the available data, preventive treatments of white oaks seem to be nearly always effective. Five years after preventive treatment with propiconazole, only 1 of 26 white (*Q. alba* L.) and bur (*Q. macrocarpa* Michx.) oaks showed symptoms and all trees survived the natural advancement of the infection front (Eggers et al., 2005).

Red oaks are the most susceptible to oak wilt infection and preventive fungicide applications have been frequently studied to find consistent, effective ways to protect these trees. The efficacy of preventive fungicide treatments of red oaks is variable, most studies show significant improvement with treatment (Osterbauer and French, 1992; Johnson, 2001; Ward et al., 2005a, 2005b; Peacock and Fulbright, 2008) but one other demonstrated relatively high mortality (Eggers et al., 2005). After propiconazole treatment, 82% of red oaks (*Q. rubra* L. and *Q. ellipsoidalis* E.J. Hill) remained asymptomatic, while only 54% of untreated trees remained healthy (Osterbauer and French, 1992) and < 2% of red oaks had wilted within 2 years of treatment while 19% of untreated trees had wilted (Ward et al., 2005a). On the other hand, 39% of red oaks were killed by oak wilt even when receiving preventive propiconazole treatments (Eggers

et al., 2005). Efficacy of preventive treatments of red oaks may be increased with retreatment of individuals at risk of infection every 2–3 years (Osterbauer and French, 1992). However, efficacy of repeated, sequential treatments is not explicitly addressed in this review.

Therapeutic propiconazole treatments

Therapeutic propiconazole treatments cannot eradicate the fungus from the tree, but are effective at delaying symptom development and decreasing mortality of white oaks. Too little information on therapeutic treatments of live oaks is available to determine efficacy, and therapeutic propiconazole treatment offers little to no protection to red oaks in advanced stages of crown wilt (Ward et al., 2005a). For live oaks, Appel (1995b) found that asymptomatic trees treated with propiconazole sustained 19% crown loss, and the symptomatic trees treated with propiconazole sustained nearly twice that level of damage with 36% crown loss.

White oaks infected with oak wilt respond well to therapeutic treatments and such treatments are usually effective at delaying symptom development and extending the life of a tree. Bur and white oaks with 5–45% crown wilt were treated with propiconazole and 87% of bur and 71% of white oaks showed no new symptoms 5 years after treatment (Eggers et al., 2005). Osterbauer et al. (1994) treated bur and white oaks therapeutically with propiconazole and found that 1 year later, 81% of treated trees showed no increase in wilt symptoms and exhibited significantly less crown wilt than untreated trees, of which 86% wilted completely. Therapeutic treatments of white oaks are so effective that professional arborists rarely treat white oaks with propiconazole until symptoms develop (Eggers et al., 2005).

Therapeutic treatments of red oaks can be effective at protecting the tree from further symptom development or death if applications are made before 25% of crown is wilting. When red oaks exhibited >25% crown wilt, therapeutic treatments were ineffective at slowing disease progression, although treated trees with <25% crown wilt survived the following growing season (Ward et al., 2005a). Northern red oaks treated with propiconazole 2 weeks after inoculation with the oak wilt fungus showed no wilt for at least 24 months after treatment (Blaedow, 2009). However, these results are inconsistent with a similar study which found that 79% of northern pin oaks (*Q. ellipsoidalis*) inoculated with *C. fagacearum* 2 weeks prior to propiconazole treatment wilted and died (Ward et al., 2005b). Therapeutic propiconazole treatments can be effective at delaying symptom development in red oaks if treatments are made soon after infection occurs. However, propiconazole treatments should be repeated every 2 years for continued protection (Blaedow, 2009).

Other factors impacting efficacy of propiconazole

Tree size, fungicide formulation and application method, treatment timing, and proximity to infected trees have also been studied for impacts on treatment efficacy. More frequent mortality and more severe symptom development are consistently reported for larger live oaks (Billings et al., 2001), white oaks (Eggers et al., 2005), and red oaks (Osterbauer and French, 1992; Eggers et al., 2005). Propiconazole delivered in organic and water-based solvents were equally effective in live oak treatment (Appel and Kurdyla, 1992). Wilson and Lester (1996) determined that three application methods (low-concentration high volume injection, high-concentration low volume microinjection, and low-concentration medium volume soil drench) exhibited similar levels of efficacy in live oak when compared to an untreated control. Black oaks (*Q. velutina* Lam., section *Lobatae*) at risk of oak wilt infection were treated with one of three volumes of

propiconazole (Table 2), mortality rates were inversely correlated with amount of propiconazole applied, with the highest rate exhibiting the lowest level of mortality, 5% (Johnson, 2001). Injections were most effective when made in spring and least effective in fall and winter (Billings et al., 2001).

Propiconazole and fungal mat production

Propiconazole injection is effective at preventing the production of fungal mats. No fungal mats were formed on oak wilt-killed red oaks treated with propiconazole but were observed on untreated trees (Osterbauer and French, 1992). No infected black oak treated with propiconazole produced fungal mats, while all infected but untreated trees produced at least one fungal mat (Johnson, 2001). As discussed for root disruption and sanitation methods, in Texas, reducing fungal mat formation does not necessarily slow local spread (Greene et al., 2008).

Propiconazole injection does not eliminate the oak wilt fungus from an infected tree (Wilson, 2005; Blaedow, 2009). However, propiconazole is effective against oak wilt in other ways. Preventive propiconazole treatments are effective at preventing symptom development and mortality in live, white, and red oaks. Therapeutic propiconazole treatments are effective at arresting symptom development in white oaks and can delay wilt in red oaks if applied prior to extensive crown wilt. Integrating root disruption, sanitation, and chemical application have the potential to increase the efficacy of oak wilt management and adding an educational component may increase efficacy even more (Juzwik et al., 2004a).

Prevention and education

New oak wilt infection centers are formed via overland spread, through the action of insect vectors or human mediated transport. Movement of infected firewood can introduce fungal mats into previously uninfected areas (Wilson, 2001; Haugen et al., 2008). Management programs to minimize overland spread have attempted to reduce fungal mat formation via sanitation and to avoid wounding of healthy oak trees. Pruning of oaks should be avoided between April and June and between February and June in the Midwest and Texas, respectively (O'Brien et al., 2000).

When pruning or other damage to oak trees is unavoidable, many authors recommend immediately applying wound paints or wound dressings (French and Juzwik, 1999; Wilson, 2001, 2005; Camilli et al., 2007). Wound paints and dressings can be effective by either reducing the attractiveness of fresh wounds to nitidulid beetles or preventing the entry of *Ceratocystis* spores into the vascular system of wounded trees (Camilli et al., 2007). Studies have demonstrated repeatedly that wound paint and wound dressings in Minnesota reduce or prevent infection and death of wounded oaks (Juzwik et al., 1985). More recently, painting wounds from pruning seemed to reduce infection levels by 40% in Texas, but this reduction was not statistically significant (Camilli et al., 2007). The consensus of published oak wilt management recommendations includes avoiding wounding during critical infection periods, utilizing recommended pruning methods and applying wound paints or dressings (French and Juzwik, 1999; Wilson, 2001, 2005; Camilli et al., 2007).

Education can build community support of management programs, increase capacity for detection of invaders, and train individuals and practitioners on best management practices; all of which can increase the capacity for controlling epidemics. In the context of oak wilt management, educational programs have been used to inform the public to avoid pruning during critical periods and to avoid moving firewood (Giedraitis et al., 1995). Stressing the role of pruning and firewood transportation in the spread of

oak wilt may limit the establishment of new infection centers thereby increasing the effectiveness of large scale oak wilt management programs (Davies, 1992; O'Brien et al., 2000; Wilson, 2001).

Educational efforts have successfully been incorporated into oak wilt management programs including the Texas Oak Wilt Suppression Project (TOWSP). After oak wilt was discovered in Texas, involvement of stakeholders in developing the TOWSP increased understanding and willingness to participate in the program (Davies, 1992; Cameron and Billings, 1995; Johnson, 1995). Because of this success, after 20 years the TOWSP is continuing education efforts (Billings, 2008).

Despite the difficulty in recognizing new infection centers (Juzwik, 2000), detection remains critical for the development of an effective management plan (Appel, 2001). Education efforts may help the public detect and identify new infection centers more quickly and accurately. Educating individuals impacted by oak wilt on the best management practices can be challenging as the details of best management practices vary across affected areas. However, locally tailored programs are useful to disseminate relevant updates on oak wilt management to landowners, nurseries, and tree care professionals (French, 1995). When such programs are combined with readily available extension publications (i.e., Gleason and Mueller, 2005; French and Juzwik, 1999), the capacity for education, even with limited funding, can be extensive.

Summary

This review continues to support the USDA Forest Service recommendation that an effective oak wilt management program should address underground and overland spread of *C. fagacearum*. No single management tactic is capable of protecting susceptible oaks from both overland and underground spread in a consistent and effective manner (Table 1). However, by integrating root disruption, sanitation, and chemical application, effective best management practices can be tailored to address the specific management needs of any given oak wilt infection center. Because multiple tactics are available, modifications of recommendations can be made on a site-by-site basis. Such tailoring of treatment is central to the concept of integrated pest management and has the potential to increase both treatment compliance and efficacy (Juzwik et al., 2004a).

Public education can aid in detecting infection centers, encouraging landowners to participate in management programs, and promoting best management practices. Reflections on the role of education in oak wilt management in Texas indicate that public education increased the community's capacity to deal with the epidemic (Cameron and Billings, 1995; Johnson, 1995). As the range of oak wilt continues to expand educational programs can increase awareness and understanding of prevention strategies and best management practices. For example, following the recent discovery of oak wilt in New York, a video featuring a Cornell University professor was created and uploaded to YouTube (Hudler, 2009). Utilizing popular technologies such as YouTube can reach segments of the public not normally targeted by traditional educational programs.

In a retrospective examination of the response to oak wilt epidemics in Texas, Appel (2008) concluded that the reaction was warranted and the substantial investment in research resulted in more efficacious management tools. Most of that research has been presented here; however, there are still gaps in our understanding and further research is necessary. Future research efforts might focus on developing an effective biological control program, determining the role of fungal mat suppression in

reduction of overland spread, and continuing the clarification of regional best management practices.

Acknowledgements

Partial funding for KAK and GLQ provided by the NSF IGERT: Risk Analysis for Introduced Species and Genotypes (NSF DGE-0653827). We thank J. Pokorny, USDA Forest Service, State and Private Forestry, for useful information on recommended oak wilt treatments. We also thank F. Homans, J. Juzwik, J. Pokorny, K. Smith, and two anonymous reviewers for helpful comments on previous drafts.

References

- Ambourn, A., Juzwik, J., Moon, R.D., 2005. Use of force of infection to determine relative importance of two sap beetle species in the overland transmission of *Ceratocystis fagacearum*. *Phytopathology* 95, S161.
- Appel, D.N., 1990. The use of propiconazole for control of oak wilt in live oak. *Phytopathology* 80, 976.
- Appel, D.N., 1994. The potential for a California oak wilt epidemic. *Journal of Arboriculture* 20, 79–86.
- Appel, D.N., 1995a. The oak wilt enigma: perspectives from the Texas epidemic. *Annual Review of Phytopathology* 33, 103–118.
- Appel, D.N., 1995b. Chemical control of oak wilt. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 81–88.
- Appel, D.N., 2001. The basics of oak wilt biology and factors influencing disease incidence and severity. In: Ash, C.L. (Ed.), *Shade Tree Wilt Diseases*. APS Press, St. Paul, MN, pp. 71–81.
- Appel, D.N., 2008. Oak wilt; did our response match the threat? *Phytopathology* 98, S187.
- Appel, D.N., Kurdyla, T., 1992. Intravascular injection with propiconazole in live oak for oak wilt control. *Plant Disease* 76, 1120–1124.
- Appel, D.N., Maggio, R.C., Nelson, E.L., Jeger, M.J., 1989. Measurement of expanding oak wilt centers in live oak. *Phytopathology* 79, 1318–1322.
- Appel, D.N., Peters, R., Lewis Jr., R., 1987. Tree susceptibility, inoculum availability, and potential vectors in a Texas oak wilt center. *Journal of Arboriculture* 13, 169–173.
- Billings, R.F., 2000. State forest health programs: a survey of state foresters. *Journal of Forestry* 98, 20–25.
- Billings, R.F., 2008. The Texas cooperative oak wilt suppression project: lessons learned in the first twenty years. In: Billings, R.F., Appel, D.N. (Eds.), *Proceedings of the Second National Oak Wilt Symposium*, Austin, TX, pp. 225–236.
- Billings, R.F., Gehring, E.H., Cameron, R.S., Gunter, J.T., 2001. Current practices in managing oak wilt: federal cost share programs, trenching, chemical injection, and the Texas suppression program. In: Ash, C.L. (Ed.), *Shade Tree Wilt Diseases*. APS Press, St. Paul, MN, pp. 117–129.
- Blaedow, R.A., 2009. Use of the systemic fungicide propiconazole for oak wilt management: an assessment of uncharacterized host–pathogen–fungicide interactions. Ph.D. Dissertation, University of Minnesota, Saint Paul, MN.
- Boyce Jr., J.S., 1959. Oak wilt spread in control-treated and untreated counties in the southern Appalachians. *Journal of Forestry* 57, 660–661.
- Bruhn, J.N., 1995. Oak wilt management in Michigan. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 109–116.
- Bruhn, J.N., Heyd, R.L., 1992. Biology and control of oak wilt in Michigan red oak stands. *Northern Journal of Applied Forestry* 9, 47–51.
- Cameron, R.S., Billings, R.F., 1995. The Texas oak wilt suppression project: development, implementation, and progress to date. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 139–146.
- Camilli, K., Appel, D.N., Watson, W.T., 2007. Studies on pruning cuts and wound dressing for oak wilt control. *Arboriculture & Urban Forestry* 33, 132–139.
- Cook, S.J., 2001. Current practices and suppression methods for managing oak wilt disease. In: Ash, C.L. (Ed.), *Shade Tree Wilt Diseases*. APS Press, St. Paul, MN, pp. 93–100.
- Davies, C.S., 1992. Environmental management of oak wilt disease in central Texas. *Environmental Management* 16, 323–333.
- Eggers, J., Juzwik, J., Bernick, S., Mordaunt, L., 2005. Evaluation of propiconazole operational treatments of oaks for wilt control. Research Note NC-390 of the USDA Forest Service. North Central Research Station, St. Paul, MN.
- Fowler, M.E., 1953. Oak wilt: its distribution and control. *Plant Disease Reporter* 37, 104–109.
- French, D.W., 1995. Oak wilt management in Minnesota. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 117–120.
- French, D.W., Juzwik, J., 1999. Oak wilt in Minnesota. Report MI-03174 of the University of Minnesota Extension Service. University of Minnesota, St. Paul, MN.

- Gehring, E., 1995. Evaluation of suppression project treatments. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 147–154.
- Giedraitis, J., Drozda, E., Culver, J., 1995. Urban oak wilt management in Austin, Texas. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 167–174.
- Gillespie, W.H., Shigo, A.L., Tryon, E.H., 1957. The degree of mat-production control obtained by girdling oak wilt trees in West Virginia and some factors influencing mat formation in girdled trees. *Plant Disease Reporter* 41, 362–367.
- Gleason, M.L., Mueller, D., 2005. Oak wilt: identification and management. *Sustainable Urban Landscapes* 15. Iowa State University Extension, Ames, IA.
- Greene, T.A., Reemts, C.M., Appel, D.N., 2008. Efficacy of basal girdling to control oak wilt fungal mat production in Texas red oak (*Quercus buckleyi*) in central Texas. *Southern Journal of Applied Forestry* 32, 168–172.
- Haugen, L., O'Brien, J., Pokorny, J., Mielke, M., Juzwik, J., 2008. Oak wilt in the North Central region. In: Billings, R.F., Appel, D.N. (Eds.), *Proceedings of the Second National Oak Wilt Symposium*, Austin, TX, pp. 155–163.
- Hayslett, M., Juzwik, J., Moltzan, B., Appel, D., Camilli, K., 2008a. Insect vectors of the oak wilt fungus in Missouri and Texas. In: Billings, R.F., Appel, D.N. (Eds.), *Proceedings of the Second National Oak Wilt Symposium*, Austin, TX, pp. 109–120.
- Hayslett, M., Juzwik, J., Moltzan, B., 2008b. Three *Coleoptera* beetle species carry the oak wilt fungus to fresh wounds on red oak in Missouri. *Plant Disease* 92, 270–275.
- Henry, B.W., Moses, C.S., Richards, C.A., Riker, A.J., 1944. Oak wilt: its significance, symptoms, and cause. *Phytopathology* 34, 636–647.
- Hudler, G., 2009. Oak wilt in New York. Retrieved on 7 May 2009 from <<http://www.youtube.com/watch?v=XVUZsvyZlVE>>.
- Jensen-Tracy, S., Kenaley, S., Hudler, G., Harrington, T., Logue, C., 2009. First report of the oak wilt fungus, *Ceratocystis fagacearum*, in New York State. *Plant Disease* 93, 428.
- Johnson, J.D., 1995. The role of extension in oak wilt management. In: Appel, D.N., Billings, R.F. (Eds.), *Oak Wilt Perspectives: The Proceedings of the National Oak Wilt Symposium*. Information Development, Inc., Houston, TX, pp. 161–166.
- Johnson, J.R., 2001. Effects of propiconazole fungicide on fungal mat formation by *Ceratocystis fagacearum* and the long distance spread of oak wilt from infected black oaks. Unpublished M.Sc. Thesis, University of Wisconsin-Green Bay, Green Bay, WI.
- Jones, T.W., Bretz, T.W., 1958. Experimental oak wilt control in Missouri. *Research Bulletin 657 of the University of Missouri*, College of Agriculture, Agricultural Experiment Station, Columbia, MO.
- Juzwik, J., 2000. An oak wilt primer. *International Oaks* 11, 14–20.
- Juzwik, J., Cook, S., Haugen, L., Elwell, J. (Eds.), 2004a. Oak wilt: people and trees, a community approach to management. General Technical Report NC-240. USDA Forest Service, North Central Research Station, St. Paul, MN. CD-Rom version 2004 v 1.3.
- Juzwik, J., French, D.W., 1983. *Ceratocystis fagacearum* and *C. piceae* on the surfaces of free-flying and fungus-mat-inhabiting nitidulids. *Phytopathology* 73, 1164–1168.
- Juzwik, J., French, D.W., Jeresek, J., 1985. Overland spread of the oak wilt fungus in Minnesota. *Journal of Arboriculture* 11, 323–327.
- Juzwik, J., Harrington, T.C., MacDonald, W.L., Appel, D.N., 2008. The origin of *Ceratocystis fagacearum*, the oak wilt fungus. *Annual Review of Phytopathology* 46, 13–26.
- Juzwik, J., Skalbeck, T., Neuman, M., 2004b. Sap beetle species (Coleoptera: Nitidulidae) visiting fresh wounds in healthy oaks during spring in Minnesota. *Forest Science* 50, 757–764.
- MacDonald, W., Double, M., 2005. Continental and intercontinental spread of *Ceratocystis fagacearum*: Should we be concerned? *Phytopathology* 95, S127.
- MacDonald, W., Pinon, J., Tainter, F., Double, M., 2001. European oaks—susceptible to oak wilt?. In: Ash, C.L. (Ed.), *Shade Tree Wilt Diseases*. APS Press, St. Paul, MN, pp. 131–137.
- Morris, C.L., 1955. Control of mat formation by the oak wilt fungus by early felling of diseased trees. *Plant Disease Reporter* 39, 258–260.
- O'Brien, J., Mielke, M., Starkey, D., Juzwik, J., 2000. How to identify, prevent, and control oak wilt. NA-PR-03-00. USDA Forest Service, Northeastern Area State and Private Forestry, St. Paul, MN.
- Osterbauer, N.K., French, D.W., 1992. Propiconazole as a treatment for oak wilt in *Quercus rubra* and *Q. ellipsoidalis*. *Journal of Arboriculture* 18, 221–226.
- Osterbauer, N.K., Salisbury, T., French, D.W., 1994. Propiconazole as a treatment for oak wilt in *Quercus alba* and *Q. macrocarpa*. *Journal of Arboriculture* 20, 202–203.
- Peacock, K.L., Fulbright, D.W., 2008. Effective longevity of propiconazole following injection into *Quercus rubra*. In: Billings, R.F., Appel, D.N. (Eds.), *Proceedings of the Second National Oak Wilt Symposium*, Austin, TX, pp. 181–188.
- Shelstad, D., 1988. An evaluation of oak wilt root graft barriers. Report for FR 8100-Research Problems: Silviculture, University of Minnesota.
- USDA FS (United States Department of Agriculture Forest Service), 2005. Oak wilt distribution map. Retrieved on 22 April 2009 from <http://www.na.fs.fed.us/fhp/ow/maps/ow_dist_fs.shtm>.
- Ward, K., Eggers, J., Juzwik, J., 2005b. Efficacy of propiconazole for preventative and therapeutic control of oak wilt in Minnesota. *Phytopathology* 95, S166.
- Ward, K., Juzwik, J., Bernick, S., 2005a. Efficacy of Alamo for prophylactic and therapeutic treatment of oak wilt in red oaks, 2004. *Fungicide and Nematicide Tests* 60, OT018.
- Warder, J.A., 1881. Forests and forestry in Wisconsin. *Wisconsin State Horticultural Society Transactions* 11, 143.
- Wilson, A.D., 2001. Oak wilt: a potential threat to southern and western oak forests. *Journal of Forestry* 99, 4–11.
- Wilson, A.D., 2005. Recent advances in the control of oak wilt in the United States. *Plant Pathology Journal* 4, 177–191.
- Wilson, A.D., Forse, L.B., 1997. Sensitivity of Texas strains of *Ceratocystis fagacearum* to triazole fungicides. *Mycologia* 89, 468–480.
- Wilson, A.D., Lester, D.G., 2002. Trench inserts as long-term barriers to root transmission for control of oak wilt. *Plant Disease* 86, 1067–1074.
- Wilson, A.D., Lester, D.G., 2001. Trench inserts improve trenches for long-term control of oak wilt. *Phytopathology* 91, S95.
- Wilson, A.D., Lester, D.G., 1997. Subsequent tests of trench inserts as barriers to root transmission for control of oak wilt in Texas live oaks. *Biological and Cultural Tests* 12, 63.
- Wilson, A.D., Lester, D.G., 1996. Evaluation of propiconazole application methods for control of oak wilt in Texas live oaks. *Fungicide and Nematicide Tests* 51, 389.
- Wilson, A.D., Lester, D.G., 1995. Application of propiconazole and *Pseudomonas cichorii* for control of oak wilt in Texas live oaks. *Fungicide and Nematicide Tests* 50, 393.
- Young, R.A., 1949. Studies on oak wilt, caused by *Chalara quercina*. *Phytopathology* 39, 425–441.