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THE OAK WILT ENIGMA: Perspectives from the Texas Epidemic

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ABSTRACT

Ceratocystis fagacearum (Bretz) Hunt, the oak wilt pathogen, is currently causing massive losses of semievergreen live oaks (*Quercus fusiformis* Small and *Q. virginiana* Mill.) in central Texas. Given the relatively limited oak mortality caused by *C. fagacearum* in the deciduous forests of the North Central, Midwestern, and Mid-Atlantic United States, this Texas epidemic was not anticipated. The intensity of oak wilt in Texas is attributed to a number of factors related to host characteristics and the ability of the pathogen to adapt to limiting environmental conditions. Oak wilt management in semievergreen oaks requires considerable revision of the control techniques previously designed for deciduous oaks. The Texas oak wilt epidemic provides a new perspective from which to evaluate questions concerning oak wilt, including the origins of the pathogen as well as the potential for future losses in unaffected oak forests.

INTRODUCTION

The vascular pathogen *Ceratocystis fagacearum* Bretz Hunt, causal agent of oak wilt, is potentially the most destructive of all tree pathogens (34, 94). This potential is expressed most dramatically when infected red oaks (genus *Quercus*, subgenus *Erythrobalanus*), common constituents of midwestern forests, invariably die within a few weeks after symptoms appear. The destructive

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nature of oak wilt in the U.S. throughout the Upper Mississippi River Valley during the decade following World War II raised considerable alarm for the threat to the nation's valuable oak resources (17, 25, 41). The concern at the time was particularly acute due to the relatively recent devastation left in the wake of the epidemics of chestnut blight and Dutch elm disease. Numerous states subsequently devoted considerable resources to prevent another catastrophe. However, despite the widespread existence of susceptible oaks in the U.S., *C. fagacearum* has still caused only limited losses when compared to those more widely recognized pathogens, *Cryphonectria parasitica* (Murr.) Barr and *Ophiostoma ulmi* (Buisman) Nannf., respectively.

Texas is the most recent state to undertake extensive oak wilt research and control programs. These efforts were stimulated by massive losses of live oaks (*Q. fusiformis* Small and *Q. virginiana* Mill.) throughout a 40–50 county region over the past 30 years (10, 52). The losses are largely focused on the geographically unique Balcones Escarpment of central Texas, where semievergreen live oaks are the most valuable woodland and urban tree species (57). Oak wilt research and the implementation of statewide control programs in Texas have followed a pattern common to other states previously affected by the disease. Following confirmation of the causal agent, systematic detection and survey of affected areas has been the usual first response. In the case of oak wilt, strong public education programs and widespread demonstration projects for disease control were often implemented prematurely, with questionable results. Fortunately, throughout most of the oak wilt range in the U.S., localized epidemics have abated (56). In many cases they never developed at all and interest in the disease declined. This is not the case in Texas, where annual losses are occurring at immeasurable rates and significant resources are spent on control (7, 9, 11).

There exist some excellent reviews of the extensive research on oak wilt (34, 56). Each is written under the premise that the damage that *C. fagacearum* could potentially cause has not been realized because of some limitation in the life history of the pathogen. These previous reviews also were prepared from the perspective of oak wilt as it occurs on deciduous oaks in the forests of the Mid-Atlantic, Midwestern, and North Central U.S. Observations of oak wilt in the semideciduous live oaks of Texas indicate that the concerns about the destructive potential of oak wilt were warranted. Many natural resource managers and landowners in Texas are convinced that *C. fagacearum* is causing a detrimental, permanent impact on the fragile, oak savannah ecosystem of central Texas. These observations should elicit concern for the well-being of our extremely valuable resources of *Quercus* wordwide. Although some details of past research on the biology of oak wilt are pertinent and are discussed here (64), this review covers the more recent literature focusing on comparative epidemiology of oak wilt in Texas and the occurrence of the

disease elsewhere. This analysis allows some of the more puzzling aspects of oak wilt to be addressed: Why has the pathogen incited an epidemic in a forest ecosystem previously considered as safe from the disease; what is the capacity for *C. fagacearum* to cause losses in presently unaffected oak forests; and what is the origin of *C. fagacearum?*

INFLUENCE OF HOST COMPOSITION ON PATHOGEN BEHAVIOR

Subgeneric Classification and Host Response

RED VS WHITE OAKS One approach to describing the biology of oak wilt is to analyze how various oaks respond to infection by *C. fagacearum*. The genus *Quercus* is an important, complex group of trees and shrubs representing at least 500 species worldwide. In the U.S., there are approximately 50 oak species (45). The majority are evenly distributed between two subgenera: *Quercus* subg. *Erythrobalanus*, the red or black oaks, and *Quercus* subg. *Quercus* (synonym *Lepidobalanus*), the white oaks. Members of these two subgenera are reproductively isolated, display distinctively different anatomical characteristics, but usually will be found growing in close association with one another (65). Differences between the groups, combined with the propensity to cohabitate, have an important influence on the potential for a developing oak wilt epidemic.

No *Quercus* species is immune to infection by *C. fagacearum*. However, species within the two subgenera are not equally susceptible (31, 41, 42). As mentioned above, red oaks are extremely susceptible, whereas white oaks typically respond with a tolerant reaction by limiting symptom development to a few branches. White oaks rarely die from oak wilt (56). This contrast in response to infection between the two subgenera was noted in the definitive description of oak wilt (41). In that report, the species most seriously affected were *Quercus velutina* Lam. (black oak), *Q. borealis* Michx. f. (northern red oak), and *Q. coccinea* Muench. (scarlet oak). The white oaks originally noted as tolerant were *Q. alba* L. (white oak) and *Q. macrocarpa* Michx. (bur oak). Since then, the outcomes of natural and artificial inoculations of red and white oaks have proven to be consistent for all deciduous oaks tested (74).

There is another, important response to infection by *C. fagacearum* that contrasts the two oak subgenera. Under conducive environmental conditions, the only known inoculum source for pathogen transmission by insects is produced on diseased red oaks. Reproductive structures of *C. fagacearum*, called fungal mats, form below the inner bark on the surface of the sapwood (24, 66). These structures appear to represent a brief proliferation of saprophytic growth that occurs just as the tree undergoes the last stages of dying

(34). No similar structures are found on diseased white oaks. As a result of a two-allele, heterothallic mating system, each mat may be one of two mating types (designated *a* or *b*). Depending on the state of maturity and potential for crossing with an opposite mating type, mats will be covered with conidia and/or perithecia that contain ascospores. The relative importance of the two spore types or the influence of sexual recombination on a developing oak wilt epidemic have not been determined (27, 56). The teleomorph is formed when insects contaminated with spermatia, in the form of endoconidia, fly from a mat growing on one tree to a mat of the opposite mating type on another tree. Naturally infected trees are rarely found to be colonized by both mating types (6, 14). Insects that serve to fertilize fungal mats are not necessarily the same as those implicated in long-distance spread of the pathogen (34). Any types of insects inclined to repeatedly visit different mats will aid in fertilization. Fruit flies, for example, are ideal spermitizing agents because they are abundantly attracted to the sweet-smelling mats. Fungal mats form on only a small proportion of diseased red oaks and last only a few weeks before they deteriorate (24). Mat formation occurs mainly on moribund trees during the spring following the season of infection and is the only known saprophytic phase of the pathogen. The reliance of *C. fagacearum* on mat formation for long-distance transmission is given as one factor involved in the limited impact of oak wilt (8, 56).

An additional limiting factor for pathogen transmission is an inefficient vector relationship. Those types of insects involved in recombination of opposite mating types may not have the ability to transmit *C. fagacearum* to new, healthy trees. Contaminated fruit flies are not attracted to healthy oaks, nor do they have the means to inoculate the fungus into vascular systems. Of the dozens of insects known to visit fungal mats, only sap-feeding nitidulid beetles (Coleoptera: nitidulidae) fulfill the requirements to act as long-distance vectors for *C. fagacearum*. Nitidulids are attracted to the sweet-smelling mats to feed and breed (86). As the mats deteriorate, the contaminated nitidulids emerge and may then be attracted to fresh wounds for further feeding (48, 49). Wounds remain receptive to infection for only a few days, and are considered to be one of the most important factors in determining whether successful transmission will occur (77). During these activities, the vector occasionally introduces the pathogen into new trees (46, 69). Because of these requirements, very few trees probably die from insect transmission of *C. fagacearum* (34). This contrasts with the more efficient vector system of Dutch elm disease (DED), where every diseased elm is a potential source of inoculum for elm bark beetles (*Scolytus multistriatus* and *Hylurgopinus rufipes*) (51). Elm bark beetles are also better vectors because they make their own infection courts on healthy trees during feeding, whereas the nitidulids only feed on fresh wounds. An oak bark beetle (*Pseudopityophthorus* spp.) with some similarities to elm bark

beetles has been implicated in spread of *C. fagacearum* (73). As with the nitidulids, the oak bark beetles have limiting characteristics that decrease the efficiency with which they may transmit the pathogen and are considered inconsequential in some regions where oak wilt occurs (34).

LIVE OAKS Live oaks do not conform to the distinct pattern of resistance and susceptibility exhibited by white and red oaks. Although most live oaks die within 3–6 months following infection, a small proportion (5–20%) survive indefinitely in various stages of crown loss (11, 19). The cause for this intermediate response between the total susceptibility of red oaks and tolerance of white oaks is unknown. Semievergreen live oaks are difficult to classify according to the two traditional subgenera and the reasons for these difficulties may relate to the variable reaction to infection. Resistance to oak wilt is a poorly understood phenomenon in white oaks, but is suspected to relate to anatomical characteristics and host response that serve to localize colonization within the vascular system and allow the host to produce healthy, unaffected xylem (56). The deciduous red and white oaks are ring porous species, meaning there is an abrupt decrease in size from large, "springwood" vessels formed at the initiation of the growing season to the smaller "summerwood" pores formed as the season progresses. Deciduous, white oak summerwood vessels contrast with those of red oaks by being smaller, thinner-walled, and more angular in shape (85, 91, 92). Also, tyloses, the bubble-like structures believed to be useful in limiting the spread of vascular parasites, form more readily in the vessels of white oak xylem. Although live oaks have numerous similarities to white oaks with regard to acorn maturity, floral anatomy, and leaf characters, the vessels resemble those of the red oaks. Live oaks are further distinguished as being semidiffuse porous, an intermediate pattern between the two subgenera. Due to these characteristics, live oaks are classified by some as subgenus Quercus (68) and by others as Erythrobalanus (92). These anatomical distinctions may also be responsible for the variable response exhibited by live oaks to infection by *C. fagacearum*.

Stand Composition

In Texas, fungal mats have been found on two deciduous red oak species, Spanish oak (*Q. texana* Small) and blackjack oak (*Q. marilandica*) (12). These species comprise only a minor component of the central Texas oak savannahs when compared to live oak. Even though live oak is clearly the most seriously affected species, no fungal mat has ever been found on a diseased live oak. This disparity in the availability of inoculum for insect transmission has a distinct influence on the spatial pattern of disease incidence. As in other states, the majority of losses result from transmission of the pathogen through root connections between diseased and healthy trees rather than through insect

transmission (34). Root graft transmission is a well-documented phenomenon in deciduous oaks and is recognized as an important factor in spread of *C. fagacearum*. Root grafts are particularly evident among live oaks with crowded roots growing in the thin, rocky soils of central Texas. In addition to root grafts, live oaks form root connections through the ability to propagate vegetatively by means of root suckering (67). This habit is considered advantageous to the trees during long periods of deficient soil moisture and contributes to their ability to rapidly colonize disturbed sites. Rhizomatous live oaks appear to maintain many of their juvenile root connections through maturity. As a result, live oak stands become highly interconnected systems of clonal mixtures with root:shoot ratios approaching 10:1 (16). Introduction of a vascular parasite into this massive system initiates rapidly expanding infection foci that have steep disease gradients, typical of a disease caused by a pathogen with a poor long-distance dispersal mechanism (22). In live oak, however, a typical focus is several hectares in size, expands at rates of up to 45 m/year, and results in the death of hundreds of trees annually (11). This contrasts with the slower growth and smaller sizes of foci in deciduous oaks (1, 34).

The most severe oak wilt epidemics occur in those areas where the *Quercus* composition and stand densities have been altered by land-management practices. The relatively high incidence of oak wilt in Wisconsin and Minnesota has been attributed to the availability of a recently developed highly susceptible host population (34). In these regions, dense stands of a red oak species, *Q. ellipsoidalis* E.J. Hill (northern pin oak), have become established owing to a reduction in forest diversity from logging operations and fire. Northern pin oak has a vigorous habit for coppicing, or reproduction through stump sprouts following disturbance. A similar pattern has been repeated in central Texas, where widespread, homogenous stands of the rhizomatous live oak have developed as a result of fire control, overgrazing, and selective thinning practices (10, 21). The ability to produce root sprouts is probably responsible, in part, for the widespread homogenous stands of live oak in the region. Prior to widespread settlement in the early 1830s, central Texas was part of the southernmost extension of the Great Plains where live oaks grew on isolated, selected sites. In both Minnesota and Texas, widespread site disturbance and subsequent vegetative reproductive habits of the oak hosts have predisposed generations of individuals to destruction by *C. fagacearum*.

Large numbers of red oaks will provide abundant primary inoculum for random dispersal of *C. fagacearum* over long distances. This type of transmission has been termed overland spread and has been viewed to be any infection occurring at distances greater than 15 m from a known inoculum source (34). Presumably, any species of oak is liable to become infected by feeding of

contaminated nitidulids, as long as it has suitable infection courts. However, only the infection of more red oaks will result in further production of primary infections over long distances. White oak infections result in little or no additional disease development. Live oak infections, in contrast, will allow for rapid development of secondary infections through root connections to adjacent trees, but will not support long-distance transmission. The stand density and species composition of oaks will have a strong impact on the character of a localized epidemic.

ENVIRONMENTAL CONSIDERATIONS

Temperature Constraints

The oak wilt epidemic in Texas was unexpected in that it contradicted many assumptions concerning the influence of environmental conditions on pathogen behavior. In natural forest ecosystems, various factors are believed to effectively suppress the development of pathogen populations and epidemic losses. This attribute is called functional diversity (76). In the case of oak wilt, high temperatures and biological controls, combined with inefficient vectors, were believed to be largely responsible for a lack of spread into southern forest ecosystems (34, 76). Temperatures in the range of 30–35°C significantly limit growth of the fungus in vitro. Higher temperatures are lethal (54, 80). Similar temperatures also retard symptom development in artificially inoculated, containerized trees (43). In apparent support of these conclusions, high summertime temperatures are believed to be partly responsible for survival of susceptible turkey oaks (*Q. laevis* Walt. subg. *Erythrobalanus*) in South Carolina (82). Inoculum production, as well as disease development, was also thought to be suppressed by conditions found in the southwestern portion of the oak wilt range. Mat formation occurs in moribund trees with a moisture content of 14% or greater (78). High air temperatures and low relative humidities were assumed to be instrumental in accelerating drying in dying trees and account for the low incidence of mat production in Missouri (34). A canker-causing facultative saprophyte, *Hypoxylon atropunctatum*, is common in dying oaks in the southwestern U.S., and was believed to directly compete with the saprophytic phase of *C. fagacearum* and prevent mat formation (79, 81). In an experiment to demonstrate the consequence of those factors on mat formation, colonized logs of diseased red oaks were transported from Missouri to Pennsylvania and a similar load of trees was brought from Pennsylvania to Missouri (34). Mats formed on the Missouri trees in Pennsylvania, but none formed in Missouri. The environment was considered to be too extreme for pathogen growth and reproduction, contributing to the apparent southern limit to expansion by the oak wilt pathogen.

Survival in Texas

Temperatures throughout the range of the disease in Texas often exceed 35°C, yet the epidemic seems to be little affected. The pathogen is apparently able to survive in the boles of live oaks, where temperatures do not exceed those considered to be limiting to the growth of the pathogen (53). It is also reasonable to assume that the pathogen can survive below ground in the extensive root systems of live oaks during periods of temperature extremes. Even though high temperatures are not sufficient to prevent oak wilt epidemics, heat may contribute to the variable survival rates observed in live oak. Heat is believed to eliminate *C. fagacearum* from the smaller limbs and branches, explaining why the pathogen is difficult to isolate from those tissues during high summertime temperatures (10, 18, 52). At 32°C, hyphal growth, conidial formation, and production of mucilage are inhibited in vitro (81). If heat operates in a similar manner in vivo, high temperatures would eradicate the pathogen from smaller limbs and branches and serve to limit the extent of colonization in live oaks.

C. fagacearum obviously overcomes the detrimental effects of high temperatures on fungal growth and survival to cause widespread, epidemic losses. The temperatures proven to be limiting to northern isolates of *C. fagacearum* in vitro are also limiting to Texas isolates (53). Selection in the pathogen population toward environmental races with heat tolerance is not responsible for the extent of oak wilt in Texas. Another explanation is provided by the Hypotheses of Compensation, advanced to explain the occurrence of epidemics of some pathogens in a variety of climatic regions (75). According to one of those hypotheses, a pathogen may compensate for marginal environmental conditions in a phase of its life cycle by vigorous growth and reproduction in another phase where conditions are favorable. Although high temperatures, low humidity, and *H. atropuctatum* may suppress oak wilt development during the summer, the pathogen can survive in boles and roots of infected trees. Climatic conditions during the remainder of the year in Texas are conducive to *C. fagacearum*. Limitations in inoculum production and inefficient insect vectors are overcome by means of the highly interconnected live oak root systems and large, dense host stands.

IMPLICATIONS FOR OAK WILT MANAGEMENT

Traditional Control Principles

A reasonably complete understanding of the oak wilt disease cycle led to various strategies to prevent losses. The development of recommendations for oak wilt management was believed to be a successful chapter in the history of North American forest pathology (30). The management system has tradi-

tionally relied on a mixture of preventive measures aimed at reducing potential sources of inoculum for long-distance spread, disrupting root connections for local spread, and eliminating potential infection courts (31). Throughout much of the range of oak wilt, inoculum has been reduced through some means of preventing fungal mat formation. Trees may be felled and burned or buried. In some cases, spraying felled trees with diesel oil has proven effective in discouraging nitidulid beetles (86). A laborious, but simple, technique to prevent fungal mats is to deep-girdle the tree at two feet above the soil surface and then strip the bark from the trunk below the girdle (87). This promotes drying of the tree and invasion of saprophytes that prevent the mats from forming.

Felling of red oaks has also been proposed to prevent transmission of *C. fagacearum* through root grafts among deciduous trees. Establishment of a 15-m zone of dead trees around the perimeter of a disease focus is intended to create a barrier of dead and dying roots. Trunk injection of poisons, such as cacodylic acid, was also recommended to kill roots and further contribute to reduction of inoculum formation on red oaks (72). When these disease-management programs were implemented statewide in Pennsylvania and West Virginia, the results proved to be only marginally successful (47). Subsequent analyses of oak wilt epidemiology indicated that rates in disease progress, as measured by losses of trees, did not justify major expenditures in disease management (56). Many states ceased to implement region-wide disease control efforts, and since the late 1960s few advances have been made in the technology for oak wilt control in the deciduous forests of the eastern and midwestern U.S. One exception has been in devising models to predict rates of local spread of the pathogen through root grafts (20, 59, 60). From these studies have developed decision keys for establishing trenches around the perimeters of disease centers to prevent root transmission. The keys are based on the probability of annual pathogen spread between healthy and diseased trees of certain sizes and at specific distances from one another.

Modifications for Oak Wilt Control in Live Oak

Upon recognition of oak wilt as the major cause of widespread live oak mortality in central Texas, large-scale control and demonstration programs for disease management were implemented, based on the technology developed for oak wilt control in deciduous trees. Although their basic principles were sound, the control programs often failed to contain the pathogen. For example, placement of a trench at 15 m beyond the perimeter of the focus—based on the distance considered to be typical for root graft transmission among deciduous oaks—was insufficient to prevent root transmission of *C. fagacearum* (9). The extensive, complex root connections and relatively longer latent period for symptom development obscure the limits of colonization by *C. fagacearum*

in a stand of live oaks. The 15-m barrier was thus inadequate for judging the potential distance for spread of the pathogen through the common root systems of live oaks (11). The decision keys developed outside of Texas (20, 59, 60) are not effective and have been replaced by acceptance of a 30-m barrier for trenching as a suitable convention for stopping underground pathogen spread. Trenches supplemented with roguing of trees within the 30-m barrier are the most reliable technique for disrupting root systems and subsequent pathogen spread (11).

In Texas, the patterns of pathogen spread observed on rangelands are also found in towns and cities. Because live oaks are very tolerant to site disturbances, urban expansion has encroached on the oak savannah while maintaining the stand structure of the original woodlands (3, 5). As a result, focal expansion in the urban forests of central Texas is rapid and extremely destructive. Conventional tools for trenching and roguing trees, such as bulldozers, backhoes, and ditching machines, are often impractical for disease control in parks and lawns. Therefore, direct control of individual high-value trees became a higher priority than previously experienced with oak wilt in deciduous forests (3, 5). Injection of a relatively new fungicide, propiconazole, was tested and found to be efficacious for preventing losses, although the fungicide does not prevent spread of the pathogen through the root systems of treated trees (7). Propiconazole is an ergosterol-biosynthesis-inhibitor (EBI) and has many desirable characteristics for successfully inhibiting colonization of a vascular pathogen in infected trees. This fungicide in combination with existing techniques now provides a successful strategy to reduce losses from oak wilt under most situations where the disease is found.

THE ORIGINS OF *CERATOCYSTIS FAGACEARUM*

A better understanding of the origins of *C. fagacearum* would greatly improve our ability to determine the probability of severe oak wilt epidemics in areas that are currently unaffected. In comparison to *O. ulmi* and *C. parasitica*, which have clearly demonstrated the capacity for intercontinental spread (32), the known geographic range of *C. fagacearum* is extremely limited. Although first described in 1944, there were earlier descriptions of extensive oak mortality in Wisconsin and Minnesota resembling oak wilt (41). Oak wilt was subsequently reported in adjacent states and eventually a range covering 20 states was established over a 20-year period (29, 74). This apparent spread, in all likelihood, consisted of verifying a long-established range rather than documenting a rapidly spreading pathogen. Although occurrences of oak wilt are occasionally reported outside the 22-state region (93), the fungus has never been positively identified in those areas.

By 1965, *C. fagacearum* was thought to be at the limit of its potential range

in the U.S. (74). This range included only one county in Texas. Ten years later a survey in the southwestern range of the disease found no further infections in Texas (70). However, this survey extended no further than 18 counties into the northeastern corner of the state, under the presumption that southward expansion would result from contiguous spread of the pathogen out of southern Arkansas. Meanwhile, widespread live oak mortality in central Texas was reported and attributed to *Cephalosporium diospyri*, the causal agent of persimmon wilt and live oak decline (89, 90).

Historical records of oak mortality in Texas are ambiguous, obscuring the origins of the disease in the state. Foresters from the Bureau of Plant Industry conducted the earliest recorded systematic disease survey in Kerrville during 1909 and 1910 (40), the area currently with the highest incidence and severity of oak wilt. No unusual oak mortality was reported. In 1934 and 1935, widespread live oak mortality with features similar to oak wilt was reported from Austin (83, 84). Over the next 35 years, live oak mortality received much attention and was attributed to a variety of causal agents other than *C. fagacearum* (26, 38). By the time the causal agent of oak wilt, *C. fagacearum*, was verified (52) and the extent of the epidemic in Texas fully appreciated (10), the disease had probably been long established within that range. Both mating types of the pathogen were distributed throughout the range in equal proportions, similar to mating type distributions in other states (6, 95). Likewise, most new reports of oak wilt in Texas are the result of locating well-established disease centers rather than recent introductions. One exception is identification of the pathogen in Houston, TX (DN Appel, unpublished); this represents the eastern-most expansion of the pathogen into the valuable live oak population that extends into the Gulf Coast States. There is also evidence that the pathogen is extending into planted live oak populations in communities located in the north Texas high plains.

Ceratocystis fagacearum may have been introduced into North America relatively recently, and the limited range is a result of insufficient time for the poorly vectored pathogen to distribute throughout the available host population. Alternatively, the pathogenic *C. fagacearum* may represent a recent speciation event from a fungus that previously was not a pathogen of oaks. In either case, some authors assume that the disease originated in the Upper Mississippi River Valley of Wisconsin and Minnesota and extended southward (1, 70, 74). However, the history and current status of the oak wilt epidemic in Texas cast doubt on this assumption.

The population of *C. fagacearum* has proven to be fairly homogeneous. Although a few studies have noted variability in some cultural characters and pathogenicity (13, 23, 39), these traits are insufficient to complete the analyses needed to elucidate basic questions concerning population biology of the fungus. A series of experiments utilizing RFLPs (restriction fragment length

polymorphisms) as genetic markers for *C. fagacearum* was initiated to better understand oak wilt epidemiology. Depending on the sample of the pathogen population analyzed, these markers can also be used to study diversity in mitochondrial and nuclear genomes that will help determine the origin of the fungus (58). For example, RFLPs in nuDNA of US isolates of the chestnut blight pathogen, *C. parasitica*, are lower in diversity than isolates from China (62, 63). This finding supports the hypothesis that the North American epidemic originated from China. A remarkably low level of variation in mtDNA and nuDNA has been detected in isolates within Texas and among isolates from throughout the United States (50). Because oak wilt, as caused by *C. fagacearum*, is as yet unknown outside the United States, the possibility of an introduction as the source of the disease becomes increasingly unlikely. However, oak wilt may easily be overlooked in an area where a host population has coevolved. Even in Texas, where the epidemic was intense, foliar symptoms on live oak were sufficiently unusual when compared to those on deciduous trees to make the disease unrecognizable (2, 90). The search for *C. fagacearum* outside of the current range should continue, especially in Mexico and Central America, where there is a high diversity of deciduous and evergreen *Quercus* spp. (44). Alternatively, several close relatives of *C. fagacearum* that are saprophytes or weak parasites of trees could be considered as predecessors in evolution of the pathogen (50). A more exhaustive survey of genetic variation in the genus may provide further insights into the origins of the pathogen.

CONSIDERATIONS FOR THE FUTURE OF OAK WILT

Oak wilt was one of the most important pathogens affecting North American forests in the mid-20th century (28). The impact on developments in the forest pathology community probably far exceeded actual losses of trees. When expected losses did not occur, interest in oak wilt diminished (56, 61). However, the Texas epidemic has shown that the potential expansion of *C. fagacearum* into the oak forests of unaffected regions should be taken seriously.

The impact of oak wilt on currently infected deciduous oak forests is not entirely negative. Surveys of stand regeneration in small openings caused by *C. fagacearum* in West Virginia present promising results for the projected compositions of future stands (88). Disease-free regeneration in the form of seedlings and stump sprouts was similar to that expected of selection cuttings in unaffected stands and normal development was anticipated. Similar, disease-free regeneration from root sprouts is regularly observed for live oaks in Texas (DN Appel, unpublished). In addition, attempts have been made to

determine the possibility of selection for disease-resistant trees in Texas during the course of fungal transmission through the highly interconnected live oak stands. Isozyme analyses of pre-and post-epidemic live oak populations revealed significant differences in the genetic compositions of the two populations (15). The low proportion of surviving live oaks may therefore have potential as disease-resistant selections for improved live oak propagation. These survivors may be the progenitors of native live oak populations with increased resistance to the disease. Further evidence for these conclusions is provided by artificial inoculation of seedlings derived from acorns produced by live oak "survivors" (36). The responses of the "survivor" seedlings differed significantly from those observed in progeny from trees growing in pre-epidemic, unaffected populations. Unexpectedly, the "survivor" seedlings died at faster rates, but resprouted with healthy shoots more vigorously than did seedlings from the general population. Resistance and survival to *C. fagacearum* in live oak may relate to pathogen recognition and rapid host response in the form of growth initiation to aid in compartmentalizing the pathogen (36).

The impact of *C. fagacearum* on unaffected oak forests is difficult to predict. In California, there are several valuable oak populations consisting of varying proportions of red and white oaks and deciduous and evergreen oaks (37, 71). Seedlings or young trees of many of those species were susceptible to artificial inoculation by the oak wilt pathogen (4). The structures of California oak woodlands have similarities to those of Texas. For example, a deciduous red oak, *Q. kelloggii* Newb., is intermingled with coast live oak (*Q. agrifolia* Nee) in the Coast Ranges and interior live oak (*Q. wizlesnii*A. DC.) in the Sierra Nevada-Cascades (37). These and similar regions could thus be extremely vulnerable to an oak wilt epidemic (71).

The likelihood of transport of *C. fagacearum* over longer distances must also be considered. The risk of an oak wilt epidemic has been assessed by the European forest pathology community (33, 35) and precautions are being taken to insure the fungus is not introduced through importation of contaminated timber from the United States (55). Given the versatility exhibited by *C. fagacearum* in North American oak forests and the lessons learned specifically from the Texas epidemic, it is important that assumptions concerning the behavior of *C. fagacearum* be carefully analyzed and due recognition be given to the possibility that the fungus may yet exhibit unexpected abilities to incite further epidemic losses.

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