The Origin of *Ceratocystis fagacearum*, the Oak Wilt Fungus*

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Key Words

exotic pathogens, invasive species, *Quercus*

Abstract

The oak wilt pathogen, *Ceratocystis fagacearum*, may be another example
of a damaging, exotic species in forest ecosystems in the United States.
Though *C. fagacearum* has received much research attention, the origin
of the fungus is unknown. The pathogen may have been endemic at a low
incidence until increased disturbances, changes in land use, and forest
management created conditions favorable for disease epidemics. The
host genus *Quercus* contains some relatively resistant species native to
the United States, further supporting the hypothesis that the pathogen is
native in origin. However, there are also many common, highly suscep-
tible *Quercus* species—a characteristic typical of introduced pathogens.
Most convincingly, studies have shown that the known populations of
C. fagacearum have experienced a severe genetic bottleneck that can only
be explained by a single introduction. The weight of evidence indicates
that *C. fagacearum* is an introduced pathogen, with possible origins in
Central or South America, or Mexico.

INTRODUCTION

Significance

The discovery in 1942 of *Ceratocystis fagacearum* as the cause of dramatic and rapid death of red oaks (*Quercus* Section *Lobatae*) in Wisconsin (32) followed the devastating epidemic of chestnut blight in Appalachian forest ecosystems and the emerging epidemic of Dutch elm disease in the eastern United States. These tree diseases were some of the first examples of how introduced exotic organisms can dramatically impact natural ecosystems (47). *Ceratocystis fagacearum* threatened a group of North America's foremost forest species, i.e., *Quercus* spp., and particularly the dominant oak-hickory forests of the central and eastern United States. Oak wilt was therefore the object of a great deal of attention and anxiety by the forest pathology community as reports of *C. fagacearum* steadily increased (13).

In spite of the destructive potential of the oak wilt pathogen, predicted losses (e.g., 52) failed to materialize. As knowledge of oak wilt etiology increased, it became evident that functional root connections between diseased and healthy trees and dissemination by certain sap-feeding and bark-inhabiting beetles were important regulators of disease incidence. Fortunately, for several regions of North America where oaks represent a major forest component, the pathogen either has not been introduced or has failed to become established and spread if it has been introduced. However, the pathogen is currently known to cause disease in 860 counties of 23 eastern and Midwestern states and Texas (70). *Ceratocystis fagacearum* continues to cause devastating losses on a local to subregional scale and is considered the primary forest disease of concern in five of these states (9).

Determining Origin of an Invasive Pathogen

There is currently a high level of concern about exotic invasive species and their potential for introduction to geographical regions of the United States or other countries where the species are not presently established (17). The origin of *C. fagacearum* is unknown, but the topic is intriguing and of interest to many, including regulatory agencies.

There are many benefits to knowing the geographic origin of any particular invasive pathogen. When the geographic origin is known, it is possible to identify and theoretically eliminate pathways of further spread and introduction of the species. Comparisons of host species in potential target areas to hosts in the area of origin aid epidemiological modelers in predicting regions at greatest risk of successful invasion by the pathogen. Estimates of potential impact are gained through comparative studies of the ecology and behavior of the invading species in its native environment and that in its new environment. Furthermore, with knowledge of the pathogen's origin, it is possible to evaluate the potential for pathogen introgression or hybridization with other related species present in the region at risk. Biocontrol organisms present in the pathogen's native ecosystem are potential candidates for deployment as biocontrol agents in the newly invaded ecosystem. Knowledge of a pathogen's origin is also valuable to identify resistant or tolerant hosts to plant in disease-ravaged areas or within areas of recent pathogen establishment. Further, knowledge of the genetic variability of the pathogen is important to develop disease resistance.

Unfortunately, determining the geographic origin of a suspected, nonnative pathogen is often difficult, and such studies may not lead to definitive conclusions. Anecdotal and/or early surveys of geographic distribution and rapidity of spread of a suspected nonnative pathogen may support conclusions that it is exotic. The initial outbreaks of Dutch elm disease in North America led to clues about where the pathogens, *Ophiostoma ulmi* and *O. novo-ulmi*, originated (11). Relative levels of susceptibility or resistance of hosts in both newly invaded regions as well as the

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Invasive species:
species nonnative to the ecosystem under consideration and its introduction causes economic or environmental harm or harm to human health

Geographic origin:
implies the site of speciation and generally an area where individuals express the greatest genetic diversity

putative region of origin also is considered valid evidence. The relative resistance of Chinese chestnuts compared to the American chestnut has supported the opinion that the pathogen was of Asian origin (64). Genetic and phylogenetic analyses of a pathogen also can support or disprove hypotheses of a nonnative origin.

Clues about the geographic origin of a species may also be found by examining the biology of related species in their native habitats. Unfortunately, phylogenetic analyses have failed to identify a close relative of *C. fagacearum* (26). There are several unique aspects to the biology of *C. fagacearum*, but in most ways it is a typical species of *Ceratocystis* (26). The genus is comprised mostly of plant pathogens, many of which are wound colonizers like *C. fagacearum*, but no other species in the genus causes a true vascular wilt disease, and none of the native pathogens are as potentially devastating on native hosts (21, 42). Most members of the genus produce fruity volatiles thought to be attractive to insect vectors, and some have been associated with fungus and sap beetles in the family Nitidulidae (42). *Ceratocystis fagacearum* also forms sporulation mats that emanate fruity odors, as do many other species in the genus. However, sporulation mats of other *Ceratocystis* species usually form on the exposed wood. Only *C. fagacearum* is known to form pressure cushions or pads that push the bark away from the wood in order to crack the bark and form the cavities for mat formation between the inner phloem and outer xylem (23, 24, 68). The ecologically specialized relationship between the fungus and one sap beetle species in particular, *Colopterus truncatus*, is unusual and suggests that *C. fagacearum* may have evolved with this vector (31).

Chapter Objectives

In this chapter, we present evidence for the geographic origin of *C. fagacearum*. Following a brief summary of historical accounts of the pathogen's presence in the United States, we examine the evidence for two general scenarios concerning the pathogen's origin and the subsequent expression of oak wilt in the United States. Two alternative hypotheses are considered under the scenario that *C. fagacearum* is a native organism, i.e., a long-established, but unnoted pathogen or a recently evolved or arisen species in eastern states. The second scenario considers the hypothesis that *C. fagacearum* is a nonnative, invasive pathogen introduced to the United States. The chapter concludes with our opinion on the native vs nonnative issue and our speculation on the most likely center of origin for the pathogen and possible pathway for its introduction.

HISTORICAL ACCOUNTS OF *C. FAGACEARUM*

C. fagacearum was first described in the Upper Mississippi River Valley, and historically was thought to have migrated from this region eastward to the Appalachians (32, 68). Oak wilt was likely killing trees in the Upper Midwest as early as the 1890s based on historical accounts (24). The emergence of the disease in this area may, in part, have been the result of prior forest cutting practices and widespread fires that favored significant regeneration of oak species (34). After the discovery of the cause of oak wilt in Wisconsin, recognition of the disease in other regions of the United States came rapidly. During the subsequent two decades, the presence of oak wilt was confirmed throughout the north-central, midwestern, and mid-Atlantic states (61). By some accounts, the apparent pattern of spread reflected the successive recognition of an existing disease rather than an expansion in the range of an emerging pathogen (48). A similar story has emerged with oak wilt in Texas, the most southwestern extent of its known range. First discovered in Dallas in 1961, 20 years later oak wilt could be found in more than 30 counties and is currently found in 65 counties in Texas (6, 70). However, many reports of widespread oak mortality resembling oak wilt in Texas date back to the 1930s (6).

True vascular wilt: a disease in which the pathogen moves systemically throughout the host in the nonliving vessels without directly attacking parenchyma cells during the early stages of disease

Insect vector: an insect that transmits a disease-inducing organism or agent

Forest type: defined by the predominant forest species associated with a geographic area or region, e.g., oak-hickory; spruce-fir

Difficulty in isolating the pathogen from oaks during hot summer months is partly responsible for failure to recognize the presence of *C. fagacearum* even following its first report in the state.

By the mid-1960s, a growing appreciation that oak wilt was relatively widespread soon invalidated an epidemiological model that predicted further disease spread and intensification (52). Thus, it may be argued that the current disease range reflects a distribution pattern that has existed for more than 100 years (48).

The reason for the relatively static distribution of the fungus is difficult to explain Currently, oak wilt is largely associated with the upland oaks of the Oak-Hickory Forest type (Eastern Type 6 of the U.S. Forest Service Renewable Resources Evaluation Group) (22), which is one of the most prominent forest types in the United States. Oak species comprise at least 20% of the total basal area in this type. Susceptible oaks exist in forest types adjacent to much of the current disease range, yet generally oak wilt has not spread into these forests. Therefore, the mere presence of oaks does not favor spread of the disease. There appears to be some sort of ecological barrier to significant advancement of the pathogen. The Susquehanna River in Pennsylvania has historically been noted as the northeastern boundary of oak wilt in that state and other U.S. states (61, 70). Within the disease range, the incidence of oak wilt occurrence and severity vary among and within regions (39). Forest stand composition and structure, in conjunction with population densities and behavior of insect vectors, probably hold the key to the vulnerability of particular forest types.

Several notable extensions to the otherwise static disease range have occurred in recent decades. The "jump" in oak wilt occurrence from Central Texas to the largely treeless plains of West Texas, e.g., to the oak-dominated, urban forests of Midland and Odessa (67), has been attributed to insect transmission via mat-laden firewood moved from oak wilt

areas to the east. Similarly, the introduction of *C. fagacearum* to the south-central portion of the Upper Peninsula of Michigan (70) in the 1970s has been attributed to firewood movement from oak wilt counties in Wisconsin. Thus, human activities are important considerations in the large-scale epidemiology of oak wilt.

Insect transmission of *C. fagacearum* is also aided by human activities that result in attractive and suitable infection courts, i.e., fresh wounds on susceptible oaks. Without the aid of human transport via contaminated logs, the distances over which insect transmission naturally occurs is generally limited to several kilometers or less (2, 55, 65). Given the current understanding of the purported limits to long-distance spread of the pathogen, it is difficult to envision that the initial discoveries in Wisconsin or in Texas were single-point introductions leading to widespread epidemics within a few decades. In conclusion, it has not been possible to confidently date or place the emergence of *C. fagacearum* in the United States.

NATIVE PATHOGEN SCENARIO

First Hypothesis

One hypothesis is that *C. fagacearum* is a longstanding, endemic North American pathogen that simply went unnoticed. This hypothesis assumes that the pathogen has been at its current geographic extent for centuries or millennia but was not noticed because it failed to cause much damage. Even today, the incidence of infection in many of our eastern forests is extremely low, particularly when compared to the previous epidemics of chestnut blight and Dutch elm disease. Inoculum production is erratic and susceptible oaks are unevenly distributed in many places, resulting in poor pathogen dissemination, especially in diverse forests like those found in the Appalachian Mountains. Many trees naturally die in any forest ecosystem annually, and the relative few that contract oak wilt in such forests could

easily be discounted by anyone not familiar with the disease or intentionally surveying for it. Indeed, the confusion over the causal organism of live oak mortality in central Texas in the mid-twentieth century supports this point (19, 25, 46).

If *C. fagacearum* is a recently introduced pathogen, it is difficult to explain how it could have spread so rapidly when its vectors are so inefficient in many areas where the disease occurs and when incidence has remained relatively static over much of its current range. Further, a number of North American oak species in the white oak group (Section *Quercus*) are relatively resistant or tolerant to *C. fagacearum*, a situation often equated with long-standing host-pathogen associations. Resistance has not been demonstrated in the limited number of oak species from other continents that have been tested (14, 49).

For those areas of North America where oak wilt is significant (i.e., the Upper Midwest and Texas), root grafting or the common root systems arising from root suckers appear to be responsible for significant perpetuation of the disease. As mentioned above, the substantial increase in recent times of the regeneration of significant populations of root-grafted or common root system oaks may offer a partial explanation for the ongoing oak wilt epidemics and the recent recognition of a long-standing pathogen.

The increase in oak wilt incidence and the subsequent recognition of the disease may be attributed to altered ecosystems that have allowed for remarkable levels of mortality. The savannas of large, scattered oak trees that once occupied millions of hectares from Texas to Manitoba have been reduced to approximately 0.02% of their pre-1850 area because of land use change and fire suppression. Part of this reduction is the result of tree and shrub encroachment due to protection from normal ecosystem disturbances such as fire, floods and grazing. For example, in the sand plains of east central Minnesota, fire suppression has produced homogeneous, oak-hickory forests. The highly interconnected roots systems of northern pin oak (*Q. ellipsoidalis*) in these areas have allowed for epidemics of oak wilt (36). The dramatic emergence of oak wilt in Texas may also be due to the dense oak regeneration that followed the abuse and abandonment of large acreages of rangelands (7, 62). Approximately one third of Missouri's land base is covered with oak-hickory forests (45). These extensive oak forests are the result of past logging and land conversions that have greatly affected their present-day health (56). Since pre-European settlement, shortleaf pine forests in Missouri have been reduced from ∼1.6 million hectares to ∼219 thousand hectares, and these pine forests are being replaced largely by oaks. This increase in extent of oak forests also has allowed for the widespread expression of oak wilt in the state (48).

Certainly, changes in land-use and forest management on lighter textured soils have promoted development of oak wilt and subsequent epidemics (39, 48). The red oak component of oak forests, in particular, is a key factor in building and maintaining an oak wilt epidemic. The overland spread of the fungus depends on the availability of oak wilt sporulation mats that commonly form on moribund red oaks infected with *C. fagacearum*. The percent red oak in a stand is also positively correlated with the frequency of below-ground, root-graft spread in the Upper Midwest (51). As more red oaks succumb to oak wilt via below-ground spread, an increase in the mortality of red oaks leads to an increase in mat production (i.e., available inoculum), sap beetle acquisition of pathogen spores, and transmission rate of *C. fagacearum* to wounds on healthy trees. Mats commonly form on red oaks species but very infrequently to never on white oak species (6, 18, 24, 57). In general, the severity of oak wilt in a state is positively correlated with red to white oak ratios (on a wood volume basis) in oak forests (39). The calculated ratios for states sustaining epidemics range from 1.32 to 2.58 for Michigan, Minnesota, and Wisconsin. Thus, forest management and land-use activities that favored

development of dense stands of red oak species have also promoted oak wilt.

Second Hypothesis

The second hypothesis considered under the native pathogen scenario is that *C. fagacearum* is a newly evolved species within the United States. If *C. fagacearum* arose from a relatively recent speciation event in eastern North America, where the mycoflora on oaks is fairly well studied, then one should be able to identify its most recent ancestor or a sibling species, as has been done with most of the new *Ceratocystis* species recently recognized in eastern North America (20, 28, 30, 38). However, phylogenetic analyses have failed to identify a closely related *Ceratocystis* species, even among the well-studied mycoflora of the United States and Canada (26). The closest relatives may be *Ambrosiella* species, which are asexual symbionts with ambrosia beetles (Coleoptera: Scolytidae). In general, ambrosia beetle symbionts are not well known, so a sibling or progenitor species may have been overlooked. However, it is not likely that *C. fagacearum* is derived from a highly specialized *Ambrosiella* species even though they are close relatives to the asexual *Chalara* relatives of *Ceratocystis*. *Ambrosiella* species have no known teleomorph in nature and are obligate associates of ambrosia beetles throughout their life cycle. Some *Ambrosiella* species and *C. fagacearum* may share a recent *Ceratocystis* ancestor, however. The lack of a sibling species in the United States may suggest that *C. fagacearum* evolved elsewhere.

One pathway for speciation of *C. fagacearum* may have been that a subpopulation of a progenitor species became reproductively isolated, perhaps due to a geographic barrier or a pre- or post-meiotic barrier. However, such a speciation event should give rise to a new species with significant genetic variation as more than two individuals would parent the new species and some introgression from the parent species would be expected. As discussed below, the genetic data do not support a gradual evolution of *C. fagacearum* in the eastern United States.

Alternatively, the species *C. fagacearum* could have arisen more abruptly (29), perhaps in one haploid individual with a unique phenotype, as a single mutation or a few mutations, or through introgression from another species, as has been proposed in *Ophiostoma novo-ulmi* and *O. ulmi* (12). More than a single or a few mutations would be required as many unique genes are likely involved in such phenotypes as the ability to cause vascular wilt and the formation of pressure pads on fungal mats. The mutations or introgressions of such characters into a population of a mother species should have been accompanied by many other genetic polymorphisms, even if the speciation event were relatively recent. Again, the extremely limited genetic variation found in *C. fagacearum* argues against such a speciation event.

Finally, *C. fagacearum* may have arisen through hybridizaton of two *Ceratocystis* species. Although hybridizations are considered rare in ascomycetes, some asexual grass endophytes have been shown to be aneuploids derived from two *Epichloe* species (69). *Ceratocystis* species can form transient hybrids when closely related species are paired in the laboratory, but progeny from interspecific hybridizations lead to unstable aneuploids that grow poorly and do not mate well (20, 27, 28, 30, 38). The occurrence of normal mitotic or meiotic division in *C. fagacearum* (1), including the regular production of the sexual state in nature and laboratory culture, also undermines the hybridization hypothesis.

The most convincing argument against the recent evolution of the pathogen in eastern U.S. states is the extremely limited genetic variation in the pathogen. As a general rule, natural fungal populations generally show high incidence of genetic variation, whereas recently introduced populations show reduced variation associated with a genetic bottleneck. That is, the single or a few individuals that are successfully introduced bring only a tiny portion of the pathogen's inherent variation. The limited

Sibling species: closely related species that are similar in appearance and other characteristics but are not capable of interbreeding

Genetic bottleneck: reduction of a population/species and loss of genetic diversity associated with severe mortality or reproductive isolation from the main population(s)

genetic variation found in *C. fagacearum* populations is discussed in the following scenario, that the pathogen is a recent resident of the eastern United States.

NONNATIVE PATHOGEN SCENARIO

The mode of sexual reproduction in *C. fagacearum* predicts that natural populations should have substantial genetic variation. *Ceratocystis fagacearum* is heterothallic with two mating types (33). Developing ascogonia on fungal mats of one mating type are typically fertilized by conidia of the opposite type carried by insects from other mats (68). Ascospores resulting from sexual reproduction are exuded in a gelatinous matrix from which they are readily acquired and disseminated by mat-inhabiting insects (33, 40). The conidia are borne in an aqueous matrix that likely reduces the frequency of their acquisition by insects, a conjecture supported by scanning electron micrographs of exoskeletal surfaces of mat-inhabiting sap beetles (40). Furthermore, *C. fagacearum* perithecia were observed on more than 75% of mats collected from two east central Minnesota sites during spring of two successive years (J. Juzwik, unpublished). The two mating types occur in roughly equal proportions (8, 72), so sexual reproduction appears to be a common form of reproduction. Thus, one would expect substantial genetic variation in a heterothallic species with frequent sexual recombination. There has been some indication of variation in pathogenicity and other phenotypic characteristics among *C. fagacearum* isolates, but these characters have not been shown to be genetically determined (6).

Kurdyla et al. (43) found very limited variation among isolates of *C. fagacearum* that they studied using restriction fragment length polymorphisms (RFLPs) of mitochondrial (*mt*) and nuclear (*nu*) DNA. The 27 isolates that were obtained from throughout the known range of the species in the United States (mostly from Texas, but also from West Virginia and Wisconsin) showed no variation in the *mt* DNA markers. There was some minor RFLP variation among nine isolates using anonymous probes of *nu* DNA, but the variation was substantially less than that found with similar markers in other heterothallic ascomycetes, even in introduced populations such as North American populations of *Cryphonectria parasitica* (53).

In a similar study, Harrington (26) applied nuclear and mitochondrial markers previously used to characterize other *Ceratocystis* species to examine a limited population of *C. fagacearum* from the Upper Midwest, including 37 isolates from Iowa, 6 from Minnesota, and 1 from Illinois. The mitochondrial genome was nearly uniform, and there was very limited variation in the nuclear genome, consistent with the results of Kurdyla et al. (43). This was substantially less variation than had been found in other heterothallic and homothallic *Ceratocystis* species (21, 30, 63), suggesting that the populations in the Upper Midwest had developed from a single introduction.

The uniform mitochondrial genome, which is maternally inherited, and limited variation in the nuclear genome of *C. fagacearum*, except for the two mating-type idiomorphs, are consistent with an introduction of a single ascospore mass or of ascospore masses resulting from a single cross. Such a singular introduction would limit the population to the nuclear alleles present in the male and female parents and later mutations. The precise origin of the pathogen cannot be convincingly demonstrated unless a population of *C. fagacearum* with a substantial amount of genetic diversity is identified.

Another argument supporting a relatively recent arrival of *C. fagacearum* to the eastern United States is that the fungus appears to have extended its range in the past century. New occurrences of oak wilt outside the previously reported boundaries of *C. fagacearum* range during the last three decades include western Texas (67), the south-central portion of the Upper Peninsula of Michigan (70), and South

Carolina (10). These range extensions are likely a result of human transport of logs harboring viable sporulation mats.

The occurrence of white oak species moderately to highly resistant to *C. fagacearum* within the pathogen's known range may not represent a situation where host species have coevolved with the pathogen or have resulted from selection pressure from oak wilt in an endemic scenario. Rather, the phenotypic adaptation of white oaks that allows them to exist in drought-prone regions may coincidentally confer tolerance to the pathogen (58).

The greater weight of current evidence supports the scenario that *C. fagacearum* was introduced to the United States and probably originated from some region where it is less aggressive on oaks or other species in the Fagaceae. The key lines of supporting evidence include the following: (*i*) limited genetic variation within the pathogen, suggesting that the U.S. population originated from a single mass of ascospores, (*ii*) phylogenetic analyses of DNA sequences fail to identify a close relative of *C. fagacearum* within the species' known range of existence, (*iii*) the pathogen continues to expand its range in some U.S. regions, and (*iv*) the high susceptibility of red oak species native to the United States suggests that the fungus did not evolve in the eastern states or Texas. The latter evidence, however, must be tempered with the fact that there are moderately to highly resistant white oak species within the known disease range. Anecdotally, few true vascular wilt diseases of forest trees have been recognized and few, or none, of these are thought to be endemic (66), lending further support to our conclusion that *C. fagacearum* is an exotic pathogen.

MOST PROBABLE REGION OF ORIGIN

Portions of the world with Fagaceae forests are potential native sources of *C. fagacearum*. There are estimates of 400 to 500 species in the genus *Quercus* (37). *Quercus* and other potential host genera in the Fagaceae are distributed widely throughout the Northern Hemisphere and the related Nothofagus (Nothofagaceae) are found in the Southern Hemisphere. The great majority of *Quercus* species occur below 35° north latitude, and many exist in the arid climates of Mexico, North Africa and Eurasia. The susceptibility of European oaks has been demonstrated (49); Chinese chestnut (*Castanea mollismima*) is also highly susceptible. Furthermore, no near relatives of the fungus have been identified in either Europe or eastern Asia, where the mycoflora of oaks has been extensively studied. Thus, we conclude that Europe, at least, is not the likely origin of the pathogen. Western USA and Canada have a number of *Quercus* species, but the *Ceratocystis* species on these hosts are reasonably well characterized, and none are morphologically or genetically close to *C. fagacearum* (26).

Mexico, Central America, or northern South America, however, could be potential ranges for *C. fagacearum* as many oak species occur in these regions (35), especially in cool, high-elevation, cloud forests. The *Ceratocystis* species that occur there, other than the agriculturally important species, are largely unknown. It is also possible that *C. fagacearum* is a wound colonizer of oaks there, and the hosts are suitably resistant to the extent that the fungus does not cause a severe vascular wilt, symptoms are cryptic or atypical of those on known hosts, and/or has gone unrecognized.

In eastern United States, *C. fagacearum* infects and kills red oak species within a relatively short period of time. In contrast, the susceptibility and mortality incidence of U.S. white oak species varies greatly by species, and the length of time for symptom development and mortality in moderately susceptible white oak species is much longer than in red oak species. A small test of some domestic and exotic oak seedlings and oak relatives concluded that most were susceptible (5); furthermore, European white oaks are also highly susceptible to

the pathogen (49). Thus, more extensive testing, particularly of Central and South American Fagaceae, is warranted because species of the white oak group are unpredictable in their susceptibility.

Perhaps more important than disease resistance or susceptibility, red and white oaks differ greatly in their propensity to support *C. fagacearum* sporulation mats (39). Mats are commonly produced on red oak species; as mentioned above, mats infrequently to never develop on diseased white oak species. The mycelial mat production with pressure pads is a unique feature of *C. fagacearum*, and it appears that the fungus evolved this characteristic on red oaks which clearly are the better substrata for sporulation. Red oak species are native to the Americas (58). If *C. fagacearum* is well adapted to form extensive sporulation mats only on the red oaks, and the well-studied red oak species of Canada and the United States are excluded as possible native hosts, then Mexico, Central America, and northern South America could be likely origins of this pathogen.

Lastly, many species of nitidulids have been found on sporulation mats of *C. fagacearum*, but *Colopterus truncatus* has proven to be particularly well-suited for transmission of the pathogen. *Co. truncatus* is considered a universal vector of *C. fagacearum* in the midwestern and the mid-Atlantic states (31) because it is a common visitor of fresh wounds on healthy oaks and a common resident of maturing oak wilt sporulation mats (16, 31, 41). Indeed, *Co. truncatus* appears to have an unusually close ecological association with *C. fagacearum*, whereas no other *Ceratocytsis* species is tied closely to a single nitidulid beetle species (26). This New World beetle species is found from Quebec to British Columbia in Canada; from Florida west to Southern California in the United States, and occurs south through Middle America to Brazil and Chile in South America (60). Thus, the principal vector of *C. fagacearum* occurs throughout the range of *Quercus* species in the Americas.

POSSIBLE PATHWAY OF INTRODUCTION TO THE UNITED STATES

If a non-U.S. origin of *C. fagacearum* is accepted, the pathway of its arrival is difficult to envision. Introduction from another continent via trade or other human activity is unlikely because mycelial mats form under the bark and only when the sapwood moisture content is between 37% and 45% (15). Trade movement of oak logs from Mexico or Latin America to Texas or further north in the late 1800s, if such shipments occurred, would likely have taken too long for the logs to arrive with fresh mats for sap beetle dispersal. However, *C. fagacearum*-laden *Co. truncatus* could have been introduced with such logs. Adults of this important insect vector can overwinter in bark furrows of red oaks recently killed by the pathogen and that possess disintegrated mats (44). Dispersal of *C. fagacearum*-contaminated oak bark beetles from log imports from such trees is highly improbable as these species most commonly colonize branches in the crowns of oaks, not main stems. Furthermore, pathogen transmission by oak bark beetles native to the United States is highly variable in importance within the known oak wilt range (4, 39). Trade movement of *C. fagacearum* within oak nursery stock (i.e., living oak seedlings or saplings) is highly unlikely as natural infections in bare-root or container nurseries have never been reported.

Another pathway for rapid movement could have been a spore-laden insect blown into the United States by a hurricane or other storm event. Oak bark beetles would not be a likely vector in this scenario. Bark beetles carrying *C. fagacearum* would likely be contaminated with only conidia, and the genetic data suggest that the introduction of the pathogen was via an ascospore mass. Sap beetles are a more plausible vector group to consider. A sap beetle contaminated with a single ascospore mass of *C. fagacearum* could have been blown into the southern United States, but would have needed to visit a fresh wound on a susceptible oak in

order to establish the pathogen. The probability of any of these insect-mediated events happening is extremely low based on what is known about the frequency of contaminated insect dispersal within non–oak wilt stands within the range of oak wilt (3).

Birds have been considered as potential vectors of *C. fagacearum*, but they have never been proven to transmit the organism to healthy oaks, and their acquisition of spores may be unlikely or at very low frequencies within the known disease range (24, 68). Nonetheless, migratory birds from Latin America are a potential pathway for long-range dissemination of the pathogen to the United States in the hypothesized single event (26). Sapsuckers (*Sphyrapicus* spp.) forage for insects below the bark of oaks as well as other hardwoods (71), so they may occasionally feed on insects inhabiting oak wilt mats and thus acquire spores on their beak. Sapsuckers are also known for drilling through the bark of healthy oaks to produce sap that attracts insects and for foraging for insects. They also feed on inner bark and cambium tissues of healthy trees, an activity that produces wounds suitable for *C. fagacearum* infection. The yellow-bellied sapsucker (*S. varius*) overwinters in Central America and Mexico and migrates northward through Texas to the Upper Midwest in the spring when oaks are physiologically most susceptible to infection (26).

FUTURE ISSUES

We conclude that *C. fagacearum* is most likely an exotic pathogen in the United States, with unknown evolutionary and geographic origins. The weight of evidence for a nonnative origin is much less than the evidence for the introduction of other significant nonnative forest pathogens, such as *Ophiostoma novo-ulmi*, *Cronartium ribicola*, *Cryphonectria parasitica*, and *Ceratocystis platani* (12, 50, 54, 59). Further research is needed to convincingly demonstrate the origin of *C. fagacearum*. Discovery of a population of *C. fagacearum* with a substantial amount of genetic diversity would be a key finding. A search in the most probable region of origin and genetic analysis of representative isolates is a logical next step. Identification of close sibling species in the most probable region of origin would also support the assertion of *C. fagacearum* as an introduced species in the United States. Specifically, phylogenetic analysis of the relatedness of *C. fagacearum* to ambrosia beetle symbionts or other *Ceratocystis* species from wounds on Fagaceae in Mexico, Central America, or northern South America would be appropriate future work (26). *C. fagaearum* may be a relatively innocuous colonizer of wounds on relatively resistant oak species in the proposed region of origin (38). Alternatively, the fungus should be considered a possible causal agent during investigations of oak dieback or mortality in the region. Lastly, *Co. truncatus* may be operating as a vector of *C. fagacearum* in the suspect region, if the fungus is indeed present. Fresh wounds on healthy oaks represent an ecological niche where *Co. truncatus* could be expected and assays for *C. fagacearum* presence on such beetles could potentially yield the fungus.

SUMMARY POINTS

1. Following review of available evidence, we conclude that *Ceratocystis fagacearum* is not a U.S. native species, though the fungus has not been found elsewhere.

2. The occurrence of both highly susceptible and relatively resistant oak species within the known range of oak wilt, the vascular wilt disease caused by *C. fagacearum*, can be cited as evidence for both exotic and native scenarios of origin.

3. It is not possible to confidently date or place the emergence of *C. fagacearum* in the United States. Furthermore, expert opinion is divided on whether the disease range is still expanding or has already reached the limits of its potential distribution.

4. The limited genetic variation within *C. fagacearum* suggests that the species population in the United States originated from a single mass of ascospores.

5. Phylogenetic analyses of DNA sequences of *Ceratocystis* species and related asexual genera (i.e., *Ambrosiella* and *Thielaviopsis*) have failed to identify a close relative of *C. fagacearum* within the known range of the pathogen's occurrence.

6. Mexico, Central America, and northern South America are considered potential locations for the existence of natural populations of *C. fagacearum*, though the fungus has not been detected there.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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Contents

Errata

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