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OAK WILT IN THE APPALACHIANS

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ABSTRACT

Oak wilt was discovered a few decades after blight nearly eradicated the American chestnut, heightening concern that the oak resource in North America might be threatened similarly. Fortunately, in most Appalachian areas, the disease has spread slowly and erratically among a susceptible population of red oak species. This has occurred in spite of the existence of disease components that are common to areas of the United States where oak wilt is devastating; namely, a highly virulent causal pathogen, *Ceratocyctis fagacearum*, the existence of insects that have been identified as vectors, and the presence of root graft unions among susceptible oak species. A variety of hypotheses have been forwarded as to why the spread of oak wilt has been slow in the Appalachians. Certainly the diversity of hardwood species has limited tree-to-tree spread that is typical of areas in the upper Midwest and southcentral United States where *C. fagacearum* spreads freely through interconnected oak root systems. Likewise, many Appalachian sites possess rocky soils which have been speculated to restrict the development of functional root grafts, thereby further limiting tree-to-tree spread. Although known insect vectors are present in the Appalachians, their effectiveness is highly dependent on a variety of temporal conditions including the availability of inoculum as well as fresh wounds to serve as inoculation sites. All evidence suggests that the vectors are highly inefficient and proof of their relative importance in establishing new infections is circumstantial. In spite of the limited spread of oak wilt in the Appalachians, the disease can have very consequential influences in localized areas where it may smolder for decades, killing hundreds of oaks over time. The future of this disease could change rapidly if a more efficient vector were to emerge in this oak-rich region.

Key words: *Ceratocystis fagacearum*, disease management, insect vectors

Discovery of oak wilt (caused by *Ceratocystis fagacearum* (Bretz) Hunt) in Wisconsin in the 1940s and its subsequent diagnosis in the mid-Atlantic region resulted in immediate concern about the long-term effects of the disease on highly valued eastern oak resources. Extensive early surveys detected the disease from Pennsylvania southward to the Carolinas (True et al. 1960). Early assumptions were that this represented disease spread into new areas of the Appalachians. Obviously, the then relatively recent demise of the American chestnut from chestnut blight weighed heavily on the minds of conservationists and foresters of the day. This concern seemed

warranted as even today no forest pathogen is known to be as capable of killing members of the red oak group as efficiently as *Ceratocystis fagacearum*. However, with time, what initially was deemed spread into new forested areas appeared to reflect a more complete recognition of the disease rather than the expansion of the range of a recently emerging pathogen (MacDonald 1995).

Today, the incidence of oak wilt in much of the Appalachians can best be characterized as sporadic. In some locales, the disease is significant but in most regions it is rare or absent. For the Appalachians, the disease may have gone unnoted for decades had the symptomatology and causal agent not been described in the upper Midwest. Many trees die annually in any forest ecosystem and the relative few that contracted oak wilt in the Appalachians easily could have been discounted by anyone not familiar with the disease or not intentionally surveying for it.

Although the range of the disease frequently is depicted by county-by-county maps, this is not indicative of where the disease has its greatest impact. Data from West Virginia in the 1960s when annual surveys were conducted indicated that on average about 3,200 trees died statewide each year (Haynes 1995). However, the majority of infected trees were detected in the eastern panhandle of the state. Further, the disease has never been detected in several counties that are rich in susceptible oak populations. Likewise, oak wilt has never been detected to the northeast of the Susquehanna River in central Pennsylvania, yet susceptible oak populations and recognized vectors occur to the northeast of this area. One must ask why the disease has never spread into oak wilt-free areas in spite of the existence of all the necessary prerequisites for the disease, with the exception of the causal fungus.

Regions of the United States where *C. fagacearum* has been a successful pathogen possess significant populations of susceptible oak hosts, vectors capable of transmitting the pathogen, networks of inter-connected root systems among susceptible species, and appropriate environmental conditions to promote disease. Many of these factors differ vastly from region-to-region and the influence each factor exerts undoubtedly has resulted in the varied disease outcomes that are witnessed in different areas where oak wilt persists.

The two dominant components of oak wilt are the oak host and the fungus. Significant oak populations reside in the Appalachians with some areas comprised of more than 60% oaks (DiGiovanni 1990). Most prominent among the susceptible species are northern red oak (*Quercus rubra*), scarlet oak (*Q. coccinea*), and black oak (*Q. velutina*). Chestnut oak (*Q. montana*), a white oak considered intermediate in susceptibility, and several resistant white oak species, principally *Q. alba* (True et al. 1960) also are common. Although the forest ecosystems of the Appalachians have been altered considerably by a history of previous cuttings and fire, most hardwood species that existed 250 years ago remain today (Hicks 1997). Even though oak dominates many of the forests, it is joined by over 40 other species that make up the diverse Appalachian forests.

Even though molecular studies of *C. fagacearum* isolates from North America have demonstrated limited genetic variability, morphological and pathological variation have been observed among Appalachian isolates (Haynes 1976, Kurdyla et al. 1995). However, to the susceptible oak populations *C. fagacearum* infects, this variability may be irrelevant, as the fungus rapidly colonizes its host resulting almost certainly in sudden death.

TWO MAJOR FACTORS REGULATE DISEASE SPREAD

If both susceptible populations of oak species and the causal fungus exist in the Appalachians, what has limited its spread through the entire region creating a disease that is so sporadic in its

occurrence? Two major factors appear to be most responsible for the lack of significant dissemination of *C. fagacearum* in the Appalachians. The first is the frequency of root system spread. In areas of the upper Midwest and in Texas, root grafts among susceptible oaks provide a conduit for the movement of *C. fagacearum* from tree to tree (Appel 1995). Functional root grafts allow this vascular pathogen to move freely from infected host to an adjacent healthy tree. The frequency of root grafting in these high incidence areas appears to be tied to soil depth and texture, with higher rates of grafting occurring in lighter, sandier soils (MacDonald 1995). Likewise, the density and age similarities of like species that occur together clearly enhance the possibility of interconnected, functional grafts.

For the Appalachians, root grafting undoubtedly plays a role in maintaining centers of infection where like species are in close proximity to one another; however, the importance of the role of root grafts to pathogen transmission in the Appalachians often has been played down. The hypothesis remains that the rugged, often rocky soils typical of many areas in this region are detrimental to the development of functional root grafts thus restricting tree-to-tree spread (True et al. 1960). Likewise, the pathogen often can be isolated within a single tree or small group of trees if adjacent trees are not susceptible oaks or are other species. Thus, the inherent diversity of the species in the Appalachians likely minimizes root graft transmission as an avenue of pathogen spread. Further, there are instances where healthy oaks, within root graft distance of an infected tree, persist for 2-3 years before symptoms are detected (Rexrode 1978). No explanation exists for this delayed transmission phenomenon or whether root grafting even is involved when disease develops in an adjacent, previously healthy tree.

A second disease regulating factor relates to transmission by insect vectors. *C. fagacearum* largely is a xylem-limited organism. Its only phase outside its host is when it is acquired by a variety of insect vectors and is spread overland by those vectors. The strongest case for such overland transmission of the pathogen can be made for insects in the family Nitidulidae. These sap-feeding insects routinely have satisfied the prerequisites as vectors, particularly so when fresh wounds oozing sap occur during spring months (Merrill and French 1995). Beetles are lured to the fresh wounds on healthy oaks from the fragrant inoculum-producing mats on infected oaks, thereby spreading *C. fagacearum* to the healthy, but wounded trees.

Even though the role of the sap-feeding beetles has been demonstrated, their effectiveness has been questioned especially when evidence of mat production is rare or when spring wounding events have not occurred. Likewise, oak bark beetles of the genus *Pseudopityophthorus* are common in the Appalachians and their biology qualifies them as vectors, but evidence that they contribute significantly to overland spread is not convincing (Merrill and French 1995). There is little doubt that insects can and do vector *C. fagacearum* but apparently only low percentages of the many insects that have been studied actually acquire the fungus through their activities and, for those that do, their dispersal within a diverse hardwood forest makes them very inefficient disseminators. Clearly, for most vectors the critical sequence of events among the pathogen, host, and the environment that promotes vector efficiency seldom is met or the incidence of oak wilt in the Appalachians would be significantly greater.

OTHER DISEASE INFLUENCES

The topographic patterns of oak wilt spread have been studied in Pennsylvania and West Virginia particularly with respect to elevation and aspect and some with implications about insect vectors. For most studies, relationships of new infections to old have been difficult to establish as results often conflicted (MacDonald 1995). In some instances, new oak wilt

infections were found to occur more commonly on ridges and upper slopes, particularly on hillsides facing the prevailing wind currents. Presumably, fungal-laden insect vectors were carried downwind until they impacted stand openings or dominant trees protruding above the forest canopy, thereby transmitting the pathogen.

C. fagacearum has proven to be a virulent, aggressive, lethal, and systemic fungal pathogen. As a vascular pathogen, it largely is restricted to an existence within its host or by its relationship with vectors outside the infected host. Fortunately, the incidence of this disease appears to be rather static in the Appalachians and most regions of North America where oaks are a major forest component. In these regions either the pathogen has not been introduced successfully or conditions for its dissemination have not been fulfilled. Therefore, it would appear that for there to be a substantial change in disease incidence in most oak forest regions, a dramatic shift in vector relationships would be necessary. This could readily occur as pathways for the introduction of potential vectors from other continents abound and is made ever more likely by the increasing rates of international trade of logs and lumber. Obviously exotic vectors, capable of infesting sapwood of infected oaks, could establish new vector relationships and enhance pathogen spread in North America. Clearly for many oak regions, *C. fagacearum* is a pathogen in search of a better vector!

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