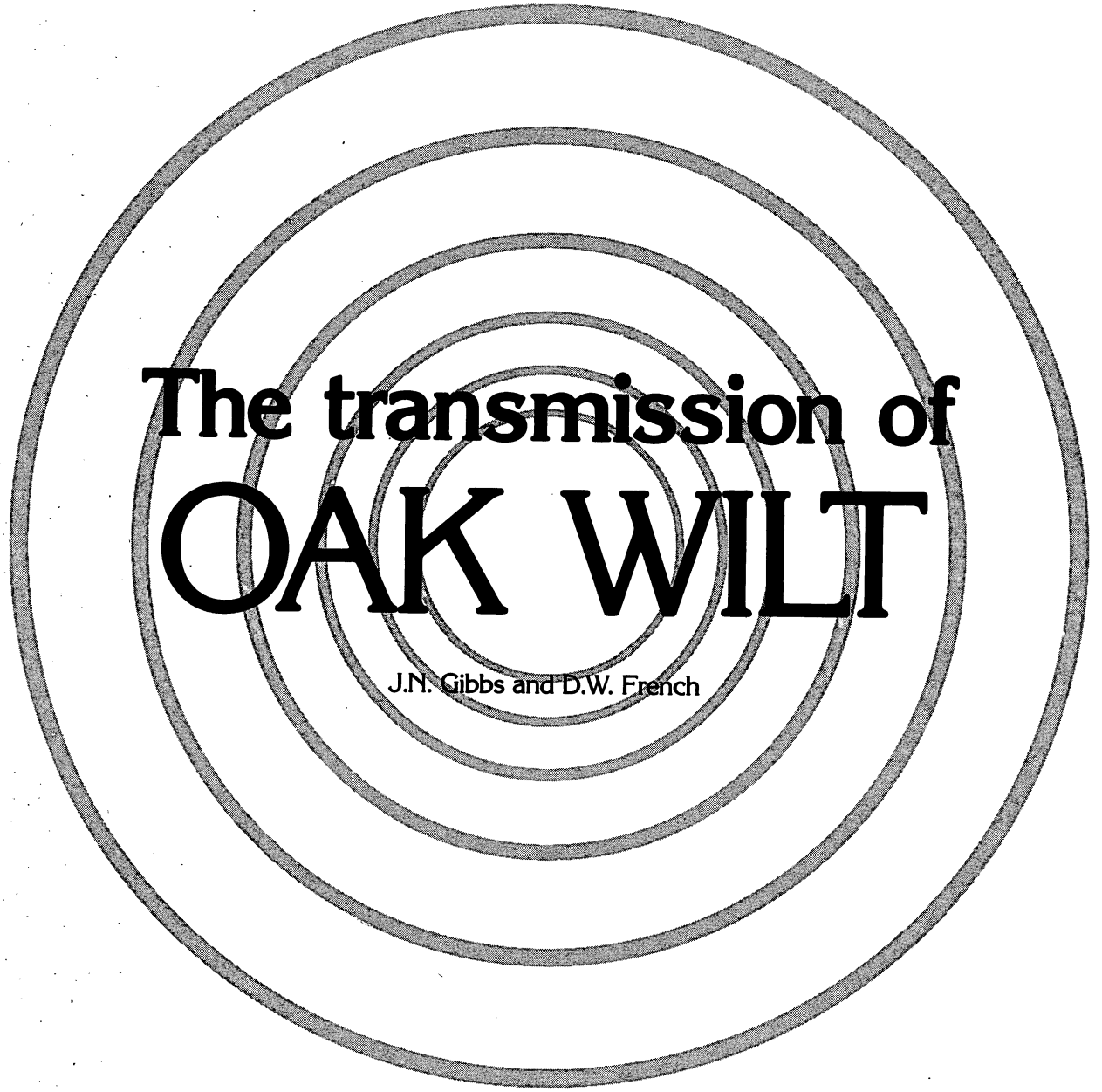
USDA FOREST SERVICE
RESEARCH PAPER **NC-185**

1980



The transmission of OAK WILT

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Manuscript approved for publication April 3, 1979
1980

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THE TRANSMISSION OF OAK WILT

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Ceratocystis fagacearum (Bretz) Hunt, the cause of oak wilt, is a fungus with the potential to be one of the most destructive of all tree pathogens. Red oaks (subgenus *Erythrobalanus*) usually die within a few weeks of infection, and although white oaks (subgenus *Lepidobalanus*) are more resistant they are not immune. Trees that are diseased 1 year may recover the next but alternatively symptoms may recur. The host populations are enormous within the known range of the disease. In the eastern half of the United States the growing stock of oak amounts to 60,000 million cu. ft., or about 35 percent of the total hardwood volume. In the 20 States in which oak wilt is now known to exist, the red oak growing stock amounts to 22,000 million cu. ft., and the white oak growing stock is almost as large (U.S. Department of Agriculture, Forest Service 1965).

C. fagacearum has not caused the devastation once so greatly feared because its spread from diseased to healthy trees is slow and erratic. Three chief methods of transmission have been recognized: (1) through root grafts; (2) via sap-feeding insects such as Nitidulid beetles; and (3) via tree-wounding insects such as oak bark beetles. There is general agreement that the importance of these means of transmission varies in different parts of the oak wilt range, but in recent years little attempt has been made to evaluate all the data that have accumulated. The objective of this paper is to provide such an evaluation.

HISTORY AND SEVERITY OF THE DISEASE

Oak wilt was first described in Wisconsin in the early 1940's (Henry *et al.* 1944) but disease survey records suggest that it was present at least as early as 1912 (French and Stienstra 1975). By 1947 it was known to be the major disease of oak in the Upper Mississippi Valley. In 1950 it was recorded in Pennsylvania and by 1951 was reported from 18 States. However, in many places it was undoubtedly present for a number of years before it was recognized and identified. Thus, in Cumberland County, Pennsylvania, one disease center was probably in existence by 1935 (Craighead and Nelson 1960), and in West Virginia, True *et al.* (1951) established that it had been present for at least 5 to 10 years before it was discovered. Although the disease was subsequently recorded in Oklahoma (1958) and South Carolina (1969), its known distribution has changed little since 1951, despite the presence of apparently suitable oak popu-lations in adjacent areas (fig. 1). In some places, for example in Minnesota, it is not now known in several counties where it was present earlier (French and Bergdahl 1973).

Disease severity varies greatly and is worst within the northwest part of its range. During a 10-year period prior to 1953, the disease destroyed 4.4 percent of the oak woodland in four counties of central Wisconsin. But in four adjacent counties the loss amounted to only 0.2 percent (Anderson and Skilling 1955). Even in the worst affected parts of States such as Missouri and West Virginia, less than one tree dies of oak wilt per square mile of forest each year (Lautz and Saufley 1970, Rexrode 1977).

It should be noted that the dramatic and continuing development of the disease in parts of Minnesota and Wisconsin during the last 40 years can at least partly be explained by the presence of a relatively new, highly susceptible, host population. The dense stands of northern pin oak (*Quercus ellipsoidalis* E. J. Hill) through which infection is spreading have resulted from the vigorous coppicing habit of this species, which has enabled it to establish itself as the dominant tree after logging operations and fire destroyed the original, more diverse woodland communities.

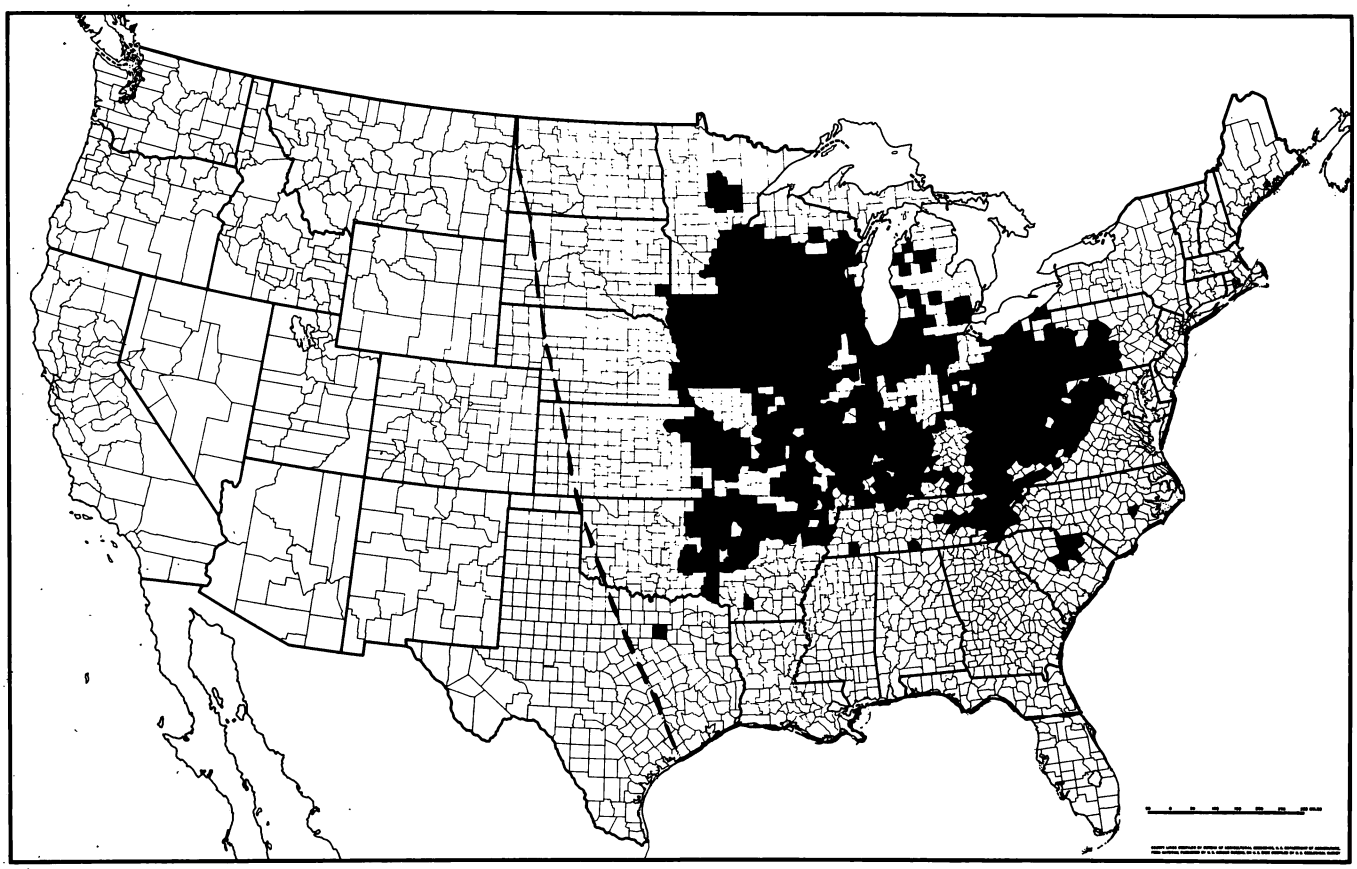

Figure 1.—*The known distribution of oak wilt as of 1978.*

All oaks and related species in the family Fagaceae that have been subjected to artificial inoculation with *C. fagacearum* have shown some susceptibility (Bretz 1955). However, as indicated earlier, the white oaks are much less susceptible than the red oaks. In one study area in northern Iowa, Young (1949) found that 55 percent of *Q. ellipsoidalis* (northern pin oak) and 53 percent of *Q. rubra* L. (red oak) had died but only 28 percent of *Q. alba* L. (white oak) and 20 percent of *Q. macrocarpa* Michx. (bur oak) had died. As might be expected, this difference in susceptibility has resulted in a change in the proportion of the two species in some areas. French and Bergdahl (1973) described an area in Sherburne County, Minnesota, in which 83 percent of the red oaks but only 11 percent of the bur oaks died from oak wilt between 1960 and 1971. This increased the percentage of bur oaks in the stand from 48 to 83.

SAPROPHYTIC EXISTENCE OF C. FAGACEARUM IN THE DYING TREE

Before discussing the various means of disease transmission it is necessary to consider the status of the pathogen within the diseased tree—the infection source. Working with naturally infected red oaks, Henry and Riker (1947) in Wisconsin and Young (1949) in northern Iowa readily isolated C. fagacearum from the xylem of roots, trunk, branches, twigs, and even leaf petioles. In the trunk Young (1949) found that the fungus was present only in the outermost sapwood layer. This pattern of distribution is not surprising. It is typical of vascular wilts of trees for the pathogen to be confined within the xylem vessels of the current annual ring until the host

becomes moribund. Following the death of the tree the mycelium of *C. fagacearum* grows extensively in the xylem vessels, and the fungus penetrates the ray cells and begins to grow out toward the cambium (Struckmeyer *et al.* 1958) and also inward. In one tree studied in Missouri about 8 months after its death, Jones and Bretz (1955) recovered the fungus from the tenth annual ring. Similarly Englerth *et al.* (1956) recorded inward colonization of the sapwood in logs from diseased trees. At the cambium the fungus may form a sporulating mat characterized by a layer of mycelium and conidiophores surrounding a raised pressure "pad" or "cushion". These mats are a famous feature of oak wilt, but it is not always realized that their formation requires the pathogen to be present simultaneously in the inner bark and adjacent xylem. Inner bark colonization was nicely demonstrated by Curl (1955) who found that when bark pieces from wilted trees were placed on the forest floor, 25 percent of them produced some kind of mat. He also found several mats that had developed entirely within the bark on standing trees. Such mats were also reported by Barnett *et al.* (1952) and Fergus and Stambaugh (1957).

Because the formation of mats requires that *C. fagacearum* be present in the inner bark as well as the xylem, it follows that data on the distribution of mats on diseased trees give some information on bark colonization by the pathogen. Curl (1955) reported that in northern Illinois, mats were produced only on the trunk and large branches, and this is the common situation in other places also. However, Morris and Fergus (1952) in Pennsylvania reported that mats sometimes occurred on branches as small as 3 cm in diameter, and Engelhard (1955) in northern Iowa recorded them on 2.5 cm diameter branches. During the summer of 1977 in Minnesota, mats developed on 2-to 4-cm diameter branches on young *Q. ellipsoidalis* that died in June, following artificial inoculation with *C. fagacearum* in May. No published accounts are available from more southerly States on the presence of mats on small branches, although C. O. Rexrode[1] has observed them in West Virginia.

Data on mat formation can also provide information on the length of time during which *C. fagacearum* can survive in the dead host. The formation of a sporulating mat represents the climax of the saprophytic growth of the pathogen in that part of the tree and its replacement by other microorganisms soon follows. Shigo (1958) in West Virginia was only able to isolate *C. fagacearum* from the xylem below 2 out of 58 sporulating mats, while other fungi such as *Ceratocystis piceae* (syn. *Graphium rigidum), Gliocladium roseum,* and *Trichoderma lignorum* (syn. *T. viride*) were very common. In the northern part of the oak wilt range where mats are formed frequently, trees that wilt in the first half of the summer (up to mid-July) produce mats in late August or September, while those that wilt later in the summer produce mats the following spring (Curl (1955) in Illinois; Morris and Fergus (1952) in Pennsylvania; and Campbell and French (1955a) in Minnesota). In Minnesota mat formation begins on the upper part of the bole on the south side of the tree. The inner bark on the lower south side and on the north side is normally still fresh at this time although the fungus can readily be isolated from the xylem.

In the southwest of the oak wilt range, the saprophytic survival of *C. fagacearum* is probably even more limited. Mats are rarely produced on diseased trees in Missouri, Arkansas, and Ohio. This is not due to genetic differences in the *C. fagacearum* isolates involved, because isolates from these States were just as capable of producing mats on inoculated trees as isolates from Wisconsin, Illinois, and Pennsylvania (Cobb and Fergus 1964). Some light is cast on the problem by experiments in which logs cut in the summer from diseased trees in Pennsylvania and Missouri and piled in Pennsylvania produced some mats, while similar logs piled in Missouri produced none (U.S. Department of Agriculture, Forest Service 1967). It was noted that the higher air temperatures and lower relative humidities in Missouri led to a lower wood moisture content, and also that colonization by *Hypoxylon punctulatum* occurred earlier. Similarly in Arkansas, Tainter and Gubler (1973) have noticed that *Hypoxylon atropunctatum* rapidly establishes itself in the sapwood of artificially inoculated oaks, and have suggested that this fungus, together with the drying out of the branches and the high summer temperatures, greatly reduces saprophytic survival of the pathogen.

Some data on the effect of high temperatures on survival are provided by Houston *et al.* (1965). They recorded temperatures approaching 50C in the cambial region of infected northern pin oak logs exposed to the July sun for 3 days. The pathogen could not be isolated from the exposed side of the log but remained viable on the shaded side. Working with the same tree species in Minnesota, we[2] found that between

[1] *Rexrode, C. O. Personal communication.*

[2] *Unpublished report on file at Stakman Hall, University of Minnesota, Department of Plant Pathology, St. Paul, Minnesota.*

June 21 and July 1, 1977, the percentage of xylem chips yielding *C. fagacearum* dropped from 33 to 8 on the exposed side of 2-to 5-cm diameter branches, while it remained unchanged on the shaded side.

Data on the survival of *C. fagacearum* in the xylem of standing trees come from the work of Turk (1955) who took samples at breast height from 52 northern pin oaks in Minnesota at various times after they had wilted. Within 6 months, recovery of the pathogen had dropped to 40 percent, by 9 months it was 20 percent, and by 10 months it was 0. Competition from other fungi including the sapwood colonizer *Nummularia bulliardi* and antagonism from *Trichothecium roseum* were considered important in limiting the survival of *C. fagacearum*. Merek and Fergus (1954) in Pennsylvania found that they could isolate the fungus from the trunk of diseased trees that were felled and left lying in the forest for up to 44 weeks, while in twigs it could only be isolated for 3 weeks. Recently Gibbs (1979a) examined the survival of *C. fagacearum* in small branches (1 to 10 cm in diameter) of northern pin oak in Minnesota. *C. fagacearum* survived in the xylem for only 1 to 2 months in trees that died in May or June but survived longer in trees that wilted later in the summer. *Dothiorella quercina* and *Coryneum kunzei* were the two chief antagonists of *C. fagacearum*.

Survival of the pathogen in sawn lumber has received some attention, principally because of the fear that the disease might thereby be introduced to other parts of North America or to other continents such as Europe (Gibbs 1978). Englerth *et al.* (1956) found that the frequency with which *C. fagacearum* was isolated from the wood dropped rapidly after sawing, but that the fungus could be recovered for up to 24 weeks (6 weeks as roundwood, 18 weeks as bulk-piled boards). Steaming or kiln-drying killed the pathogen. Little evidence exists linking the spread of infection with the movement of wood from diseased trees, although French and Bergdahl (1973) found an isolated outbreak of the disease in central Minnesota that might have begun from transported firewood.

From the above it would seem that within a few months in trees that die in early summer and well within a year in trees that die later, the oak wilt fungus has disappeared from the above ground parts of the tree. It is likely to survive longest on the lowest part of the north side of the trunk. As indicated above, antogonism by other fungi is one of the chief factors influencing survival of the pathogen. Additional information on this comes from the work of Shigo (1958) on trees girdled as part of the oak wilt control program in West Virginia. This treatment reduces mat formation due at least in part, to rapid colonization of the sapwood by *Hypoxylon punctulatum*.

In the roots of a diseased tree, survival of *C. fagacearum* may be more prolonged, although this seems to depend on whether the roots are grafted to those of a neighboring tree. In central Wisconsin, where root grafting is common, survival of up to 3 years has been recorded[3] and in Pennsylvania, with roots that were probably grafted, the fungus was isolated from one out of three trees that had been dead for 3 years and five out of eight trees that had been dead for 2 years (Yount 1955). Skelly (1967), also in Pennsylvania, obtained similar results. He recovered the fungus from the root systems of 52 percent of red oaks that had been dead for 1 year, 18 percent from trees dead for 2 years, and 3 percent from trees dead for 4 years. By contrast, Amos and True (1967) working with girdled trees not grafted to living trees in West Virginia, found that although the fungus could readily be isolated from roots after 1 year, it was rarely found after 2. *Armillaria mellea* rhizomorphs were commonly present on the roots after 1 year. *Umbelopsis versiformis, Trichoderma viride,* and *Pencillium* spp. were the fungi most commonly obtained from the bark, although *Gliocladium roseum* and *Dothiorella* spp. were also present. *T. viride* and *Pencillium* spp. were also commonly isolated from the wood.

Survival of the pathogen has hitherto been discussed only in relation to red oaks. In diseased white oaks the distribution of the fungus in the xylem of the current annual ring is much more restricted. And if the tree recovers, the infected ring will be buried under new wood (Parmeter *et al.* 1956). Isolation studies indicate that the pathogen survives in these buried rings for only a few years and in any event it is unlikely to constitute a significant source of inoculum. This is borne out by studies on infected but asymptomatic chestnut oak (Cobb *et al.* 1965a). If a white oak becomes infected and dies within a single season, the same events as described for red oaks could probably occur and an inoculum source could thus be created.

SEASONAL SUSCEPTIBILITY OF TREES TO INFECTION

One factor that may have some significance for all the possible means of disease transmission is the effect of season on host susceptibility. Engelhard

[3]*Kuntz, J. E. Personal communication.*

(1956) in Iowa inoculated fresh stem wounds on red oaks at 4- to 5-week intervals throughout the year. Disease only occurred on trees inoculated between April 25 and August 28. Drake *et al.* (1956) in Wisconsin inoculated northern pin oak from April to September. The trees were most susceptible in early June. Many of the trees inoculated in April and May also became diseased, but symptoms did not appear as quickly. Few of the trees inoculated after summerwood had begun to form became diseased. Nair and Kuntz (1963b) found that some northern pin oak trees wilted following branch inoculations in every month from February to late November, with a peak between May and August. All wilting trees died. Trees became diseased following stem inoculations in all months of the year. With both methods trees inoculated in the dormant season showed symptoms the following June. Skelly and Merrill (1968) in Pennsylvania also found that some red oaks became diseased following stem inoculations during each month from November to March. They suggested that temperatures above freezing at the time of inoculation might be important. Nair and Kuntz (1963b) also inoculated bur oaks. The highest disease incidence following branch inoculations was from mid-May to early August. Some wilt occurred following stem inoculations in every month except December, January, and February. The highest incidence (40 to 80 percent) was from early May to early August. Cobb *et al.* (1965a) in Pennsylvania found that Chestnut oak was also most susceptible to stem inoculations in May and June.

UNDERGROUND SPREAD

Oak wilt spreads underground by the passive movement of spores of the fungus from a diseased tree to an adjacent healthy tree via the continuous xylem system that exists between root-grafted trees (fig. 2). Kuntz, Riker, and their associates in Wisconsin (Kuntz and Riker 1950, Beckman and Kuntz 1951) showed that almost all the northern pin oak within 15 m of each other in the central part of the State were grafted together. Under these circumstances root graft transmission is, not surprisingly, the main means of disease spread. In Wisconsin and parts of southern Minnesota the disease characteristically develops as a number of clearly defined disease centers which expand at the rate of about 7.5 m per year in each direction (fig. 3) (French and Bergdahl 1973).

From similar studies conducted in other States in the oak wilt range it was concluded that root grafting

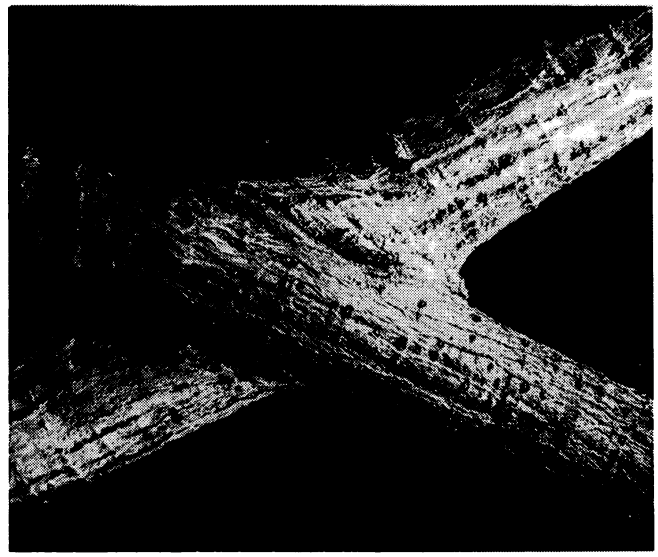

Figure 2.—*Grafted roots of northern pin oak.*

Figure 3.—*Aerial view of an infection center in northern pin oak in Minnesota. The disease is spreading through root grafts.*

was less common and consequently root transmission of the disease less important. In Pennsylvania it was suggested that 20 percent of the oaks were grafted together (Craighead and Nelson 1960), in North Carolina 15 percent (Boyce 1957a), and in West Virginia 10 percent (True *et al.* 1960). In Missouri 14 percent of 175 oaks were root grafted and it was considered from inoculation studies that many of these grafts were not functional as far as transmission of the pathogen was concerned (Jones and Partridge 1961). In these four States the maximum distance between grafted trees was 10 m.

A number of factors influence grafting frequency. Site factors seem to play a role because grafting frequency in northern pin oak is high in parts of southern Minnesota but is apparently low in north-central Minnesota. In Crow Wing County no root transmission occurred from inoculated to uninoculated trees although many were as close as 1.5 m (French and Schroeder 1969). Genetically controlled differences between species are also important. In that part of central Wisconsin in which virtually all the northern pin oak are grafted together, Parmeter *et al.* (1956) found that only 6 percent of the bur oaks were grafted. Jones and Partridge (1961) found that out of 29 grafts detected in Missouri, only one was between two different species (*Q. velutina* and *Q. marilandica*) so grafting between species is generally not important. However, in a situation where grafting is uncommon, interspecific grafting may comprise a significant portion of the total. Boyce (1957a) in North Carolina found that out of a total of seven grafts, three were between black oak and white oak. Similarly, Parmeter *et al.* (1956) in Wisconsin found that two of seven bur oak grafts were with northern pin oak.

Recently, work in West Virginia has placed renewed emphasis on the role of root transmission in that State. First, injections of wilting red oak trees with cacodylic acid showed that 31 percent were connected to healthy trees (Rexrode and Frame 1977), substantially more than the 10 percent suggested from earlier work in the State. Interestingly, 3 of the 12 grafts were between red and white oaks. Second, and more important, Rexrode (1978) showed that transmission via the root system might take several years to occur—4 out of 10 trees that wilted through root transmission of the fungus did not develop symptoms until 3 years after the adjacent inoculated trees had died. As Rexrode points out, such a method of disease transmission would agree with the observed distribution of the disease in northeast West Virginia where 50 percent of the wilting trees are within 15 m of previously infected trees, but where several years may elapse between the death of one tree and the appearance of symptoms in another. Such a distribution pattern is hard to explain in terms of transmission by insect vectors. Delayed root transmission may also be important in other States. In the Sinissippi Forest in Illinois, Himelick and Fox (1961) noted that 91 percent of infected trees were within 9 m of trees killed by the disease, but that the infection centers might remain dormant for several years before breaking out again. In 25 percent of the infection centers 4 or more years elapsed between the appearance of the disease in one tree and its appearance in another. Similar situations have been described in Pennsylvania (Craighead and Nelson 1960) and southern Michigan[4]. In Pennsylvania, where it was the practice to fell all healthy oaks within 15 m of a diseased tree, Yount (1955) suggested that slow movement of the fungus through grafted roots of these felled trees might explain the sporadic appearance of disease at the periphery of the cleared area several years later.

OVERLAND SPREAD

The Detection of *C. fagacearum* on Insects and Other Material

C. fagacearum is well adapted to spread by animals, and it has long been accepted that overland spread involves a vector. Yount *et al.* (1955) detected *C. fagacearum* on the bodies of insects collected from sporulating mats by plating out washings from these insects onto agar. However, *C. fagacearum* is normally overrun by other microorganisms when isolated on conventional agar media and no adequate selective medium has been devised. Therefore, most workers use the spermatization technique as described by Jewell (1956), which relies on the fact that *C. fagacearum* possesses two mating types—A and B. In this technique insects are macerated in water and then aliquots of the macerate are spread over cultures of the A and B mating types. The production of perithecia in the cultures then demonstrates the presence of viable propagules of the opposite mating type. Both ascospores and conidia can be detected in this way (Stambaugh *et al.* 1955).

Fergus *et al.* (1961) found that as few as 20 conidia could be detected by this technique. However, a number of factors must be optimum for this degree of sensitivity to be realized: (1) the composition of the medium (Bell and Fergus 1967), (2) the receptivity of the female isolates (Fergus *et al.* 1961), (3) the age of the cultures (Rexrode and Jones 1971), and (4) the temperature of incubation (Cobb *et al.* 1962). Care must be taken that sterile perithecia, which will form even in unspermatized cultures (Bell and Fergus 1967), are not regarded as evidence of fertilization. Recently Peplinski and Merrill (1974) suggested that pycnidia of *Pyrenochaeta* sp. and even synnemata of *Graphium rigidum* (syn. *Ceratocystis piceae*) developing on *C. fagacearum* cultures might have been confused with fertile perithecia. Despite these complications the technique has provided valuable data.

[4]*Hart, J. Personal communication.*

Transmission by sap-feeding insects

The discovery of sporulating mats of *C. fagacearum* excited an immediate interest in their function. It was quickly realized that the fruity odor attracted insects and that these gained access to the mats at points where the central pressure pad had cracked the bark. Particularly abundant were Nitidulid beetles (fig. 4). Leach *et al.* (1952) showed that these insects could act as agents of fertilization for *C. fagacearum,* transporting conidia of type A to mats of type B, and vice versa. Perithecia then developed on the mat. In rare cases, if a mixed thallus of A and B mating types was present in a single tree (Hepting *et al.* 1952), perithecia might be formed before the bark cracked open to expose the mat. Perithecia are not produced on all mats—Curl (1955) found perithecia on 90 out of 393 mats in Illinois, Morris and Fergus (1952) found perithecia on only 1 tree out of many examined in Pennsylvania, and Shigo (1958) found them on only 19 out of 164 mats collected in West Virginia.

Clearly the mats could serve as abundant reservoirs of inoculum, initially in the form of conidia and later as ascospores. Knowledge that the Nitidulids (sap-feeding insects) were attracted to the mats indicated that they might act as vectors if they migrated from mats to wounds on healthy trees. In May 1953 Dorsey *et al.* in West Virginia placed Nitidulids that had been artificially contaminated with *C. fagacearum* in wounds on healthy oaks. Five out of six of the trees developed symptoms. In the same year in Iowa, Norris (1953) caged Nitidulids, freshly collected from mats, on wounds on red oaks and obtained infection in every case.

Figure 4.—*Nitidulids feeding on a mat of* Ceratocystis fagacearum *on a dead tree.*

In 1954 infection of wounds via Nitidulids was also reported from Illinois, Pennsylvania, and Wisconsin, but success rates were much lower. In Illinois Himelick *et al.* (1954) used naturally contaminated insects and obtained infection in 1 out of 36 trees wounded between April 22 and May 22. In Pennsylvania Thompson *et al.* (1955) working with field-collected insects subsequently placed on mats producing ascospores, only obtained infection in 1 out of 175 trees wounded between April 19 and June 15. In the work of McMullen *et al.* (1955a) in Wisconsin, field-collected beetles were exposed overnight to mats or laboratory cultures. Infection was obtained in 15 out of 52 trees wounded between May 1 and June 19.

Many of these experiments were highly artificial. Indeed it has been suggested that glass marbles rolled on a sporulating mat and then placed in fresh wounds might also act as "vectors". Consequently, subsequent research was devoted to a more detailed examination of the inoculum source, behavior of the Nitidulids, and the infection court.

Inoculum source.—Following the discovery of mats in Illinois, reports of their occurrence on wilt-killed red oaks quickly came in from many States. By contrast, there have been few reports for white oaks. Parmeter *et al.* (1956) did not find any mats on hundreds of inoculated bur-oaks in Wisconsin. However, in May 1954 Engelhard (1955) found 42 matlike structures on an inoculated bur oak in northern Iowa. Some consisted of both mycelium and pad; others of pad only. They were difficult to locate because of the furrowed or flaky nature of the bark. Nair and Kuntz (1963a) also described minute mats on the trunks and branches of inoculated bur oaks in Wisconsin. The mats sporulated profusely and sap-feeding beetles were found on them. In November 1965 numerous oak wilt mats were observed on a recently killed 12-inch d.b.h. white oak in West Virginia. Thirty of these had apparently formed pads that had cracked the bark (Cones 1967).

Data on the proportion of wilted red oaks producing mats are available from several workers. In northeastern West Virginia about 25 percent of the trees produced mats, whereas in the southern part of the State the percentage was around 80, (Rexrode and Frame 1973[1], Rexrode 1977). By contrast, mats are rarely formed in Missouri and Ohio (Donley 1959). T. W. Jones[2] indicated a figure of only 5percent for Missouri. Particularly important are data on mat production during the crucial period of peak host susceptibility between April and July or late June. In northern Illinois, Curl (1955) found that 22 out of 28 trees wilting between June and August produced mats the following spring. In North Carolina, Boyce (1954) reported that 12 out of 22 trees infected in the summer of 1952 produced mats the following spring. In Minnesota, Campbell and French (1955a) established that spring mat production occurred principally on trees wilting in August. Jeresek (1976) found that in 1972, 19 percent of oaks wilting in August and early September produced mats the following spring and in 1973 the figure was 26 percent. In Pennsylvania, Cobb *et al.* (1965a) found that spring mat production occurred principally on trees that had been inoculated in July and that had developed symptoms a few weeks later.

The seasonal pattern of mat formation, with peaks in spring and autumn, has been related to temperature—both high and low temperatures are unfavorable. In Minnesota mean weekly temperatures of 50°F (10C) are optimum for mat formation although they survive longer at cooler temperatures (Campbell and French 1955a). A high sapwood moisture content is also considered important (Campbell and French 1955b). Boyce (1957b) thought that high rainfall during autumn might influence the production of spring mats on felled trees.

There is no evidence that the nature of the inoculum is critical. Himelick *et al.* (1954) wondered if the low level of infection in their experiments might be because they were using an endoconidia inoculum while Dorsey *et al.* (1953) and Norris (1953) had used beetles that could also have been carrying ascospores. However, in other experiments a low degree of infection has been obtained with ascospores (Thompson *et al.* 1955) and a higher level with endoconidia (McMullen *et al.* 1955a).

It is possible that the Nitidulids are contaminated with inoculum from sources other than the sporulating mats. In Pennsylvania, Craighead and Nelson (1960) suggested that sporulation might occur in infected but symptomless chestnut oak but Cobb *et al.* (1965b) found no evidence for this. However, they did show that sporulation could occur in wounds on diseased red oak that were close to the point of mat production.

Vector behavior.—The biology of the Nitidulids was studied first in West Virginia (Dorsey and Leach 1956) and in Iowa (Norris 1956). Subsequent studies included those of McMullen *et al.* (1960) in Wisconsin and Skalbeck (1976) in Minnesota. The time of peak abundance varies with each species but is usually in the spring. Dorsey and Leach (1956) considered mean weekly temperatures of about 50°F (10C) to be optimum for Nitidulid activity. The circumstances under which Nitidulids leave the sporulating mats and fly to wounds on healthy trees have long been the subject of discussion. In Pennsylvania Morris *et al.* (1955) found that Nitidulids remained on the mats until the latter deteriorated or dried up. When migration did occur it was more often from mat to mat than from mat to wound. They concluded that "unless mats at the right stage of decline are available at the time the wounds are made or shortly thereafter, infection through these wounds is improbable". McMullen *et al.* (1960) concurred with this. They discovered that the largest numbers of insects were found on wounds made in mid-June and that this coincided with cessation of mat production and the deterioration of the existing mats in the area. The highest percentage of wilting (about 20 percent) also occurred in trees wounded at this time.

Little information is available on other sap-feeding animals that might be vectors but a number of possible candidates are discussed by Craighead and Nelson (1960).

The infection court.—Evidence that wounding leads to infection is a vital part of the case for Nitidulids as vectors. In Pennsylvania, Guyton (1952) noted an association between wilt and wounds blazed to mark a future logging road, and Jeffrey (1953) reported many instances of wilt associated with pruning, climbing-iron damage, and logging wounds. Infections only occurred when the wounds were made between late April and early June, the period of spring wood formation in Pennsylvania. In northern Iowa, Norris (1955) observed that when a tree some distance from other wilted trees became diseased, a conspicuous wound was almost always present on the trunk or major limbs. Frequently the wilt symptoms were restricted to that part of the tree distal to the wound. (This last may not be relevant because symptom expression usually begins in the distal part of a

branch.) In Wisconsin, McMullen *et al.* (1955a) reported that 26 trees wounded in June became diseased in July. Further evidence was provided by Kuntz and Drake (1957) who reported that 19 percent of 109 oaks pruned in mid-June became diseased, and that the removal of stump sprouts in May and June led to 27 percent of the remaining stems becoming infected. In Minnesota, D. W. French[2] has shown from evidence collected during a 20-year-period that overland infection is closely associated with tree wounding in late May or early June. He noted that trunk wounds such as those made by climbing irons, appeared to be more important infection courts than pruning wounds.

Experiments involving deliberate wounding have confirmed that wounds made in May and June are most likely to lead to infection. In Pennsylvania, Craighead *et al.* (1953) reported that eight trees wounded in May were diseased in July and that larvae of Nitidulids and Diptera were abundant in the wounds of these trees. In Iowa, Norris (1955) found that disease occurred in 31 of 122 trees wounded between April 24 and June 22. Only 2 of 217 control trees died and these had been wounded by rodents. Similar results were obtained in Wisconsin with 11 out of 60 trees wounded between May 15 and June 18 becoming diseased (McMullen *et al.* 1955a, 1960). Recently Jeresek (1976) provided similar data for Minnesota. In 1974, 19 out of 82 trees wounded between May 19 and June 9 became diseased and in 1975, 6 out of 30 trees wounded between May 26 and June 9 became diseased. Trees on which the wounds were painted immediately did not become diseased, nor did any unwounded control trees.

The rate of wound infection seems to vary from year to year. Nair and Kuntz (1963) wounded 15 bur oaks and 20 Northern pin oaks at weekly intervals from May to August each year from 1960 to 1962. In the first 2 years no trees were infected but in 1962 10 bur oaks and 28 pin oaks became diseased. These trees were all wounded between May 21 and June 11.

There is only one report of wound infection that does not fit the seasonal pattern. In Pennsylvania, 21 of 101 red oaks pruned during the winter of 1957, wilted the following summer (Craighead and Nelson 1960).

Only one experiment has been described on the effect of wounding on infection from outside the northern part of the oak wilt range. In Missouri Buchanan (1960) found that oak wilt developed only in trees wounded between April 19 and April 26—7 out of 100 trees wounded during this period became diseased.

The possible importance of the condition of the wound and its location have received some attention. Clean cut or "bruise" wounds seem to be equally attractive to Nitidulids (Morris *et al.* 1955) and, as long as they reach the xylem, equally suitable for infection. Cobb *et al.* (1965a) showed that wound location could be important for red and Chestnut oak; less infection occurred following inoculation of branch wounds than trunk wounds. Wound age also has an important effect. Kuntz and Drake (1957) in Wisconsin inoculated 10- to 30-cm diameter northern pin oaks at various intervals after wounding. They obtained 100 percent infection the first 4 days but no infection after 8 days. Natural infection occurred only within 24 hours of wounding. Morris *et al.* (1955) and Cobb *et al.* (1965a) found that wounds in red oak in Pennsylvania were not suitable as infection courts after 3 days. On bur oak, Nair and Kuntz (1963a) reported that they could only obtain infection for 24 hours after wounding, and on Chestnut oak, Cobb *et al.* (1965a) found a progressive reduction in susceptibility, with no infection after the fifth day. The reduction in wound susceptibility with time may be due in part to the formation of wound tyloses or the accumulation of phenolic compounds, as suggested by Cobb *et al.* (1965a). However, the colonization of the wound surface by other microorganisms is also important. Gibbs (1980b) showed that if *Ceratocystis piceae* (syn. *Graphium rigidum*), one of the fungi most commonly associated with wounds on healthy oak (Shigo 1958), was introduced to a fresh wound 24 hours prior to its inoculation with *C. fagacearum,* no infection resulted. However such a wound remained fully susceptible in the absence of *C. piceae.* In nature *C. piceae* is probably brought to the wounds by insects (Jewell 1956).

It has long been thought that moisture in the wound has an important influence on natural infection (Himelick *et al.* 1954). It could affect both the transfer of spores from the insect to the xylem surface and also the germination and development of the fungus. Such moisture is probably most readily available in early summer when the vascular cambium is at its most active stage and many cells are in the process of differentiation. Rain water may also be important. In the experiments of McMullen *et al.* (1955a, 1960) in which Nitidulids were caged over drill holes, there was some evidence for a correlation between rainfall during the first 3 days after wounding and infection. The "fermenting sap" sometimes present in older wounds and regarded as highly attractive to Nitidulids (Morris *et al.* 1955) is almost certainly not an aid to infection. Its formation is

associated with the activity of other microorganisms, the presence of which is likely to prevent the establishment of *C. fagacearum.*

As with other wilt pathogens the number of spores introduced to the wound is not of great importance. Cobb *et al.* (1965a) found no great difference in the incidence of infection in trees inoculated with spore doses of between 10^3 and 10^6.

Transmission by Tree-wounding Animals

Oak bark beetles

The oak bark beetles *Pseudopityophthorus* spp. were among the first insects suspected as vectors of the disease. These minute beetles breed in wilt-killed trees and feed on the twigs of healthy oak (fig. 5). Using artificially contaminated beetles Griswold and Bart (1954) obtained infection in 2 out of 6 oak seedlings and Donley (1959) obtained infection in 14 out of 135 seedlings. Attempts to achieve infection by caging field-collected beetles on young oaks were largely unsuccessful, however. In Pennsylvania Craighead and others[2] caged beetles from oak wilt trees on healthy saplings each year from 1954 to 1957. As many as 300 beetles were placed in some cages but no disease resulted. Buchanan (1958) in Missouri was a little more successful. He obtained oak wilt in 2 out of 204 red oak seedlings fed on by beetles that had emerged in spring from trees that had become diseased the previous year. (About 140,000 beetles were involved in these tests.) With these results it is not surprising that in their review of oak wilt, True *et al.* (1960) did not consider the bark beetles to be of great importance in the transmission of the disease. More recently, however, the oak bark beetles have received much more attention. The main reason for this renewed interest has been the opinion that mat production in the south and west of the oak wilt range, is too rare an event to explain the observed incidence of the disease. In view of this change of emphasis, the relevant data on the biology of the oak bark beetles, and of the evidence for their role as vectors, are reviewed in some detail.

Figure 5.—Pseudopityophthorus pruinosus *feeding on scarlet oak (photograph courtesy of C. O. Rexrode).*

The beetles and their life cycles.—*Pseudopityophthorus* spp. are present throughout and far beyond the range of oak wilt. The two key species are *P. minutissimus* and *P. pruinosus.* The former appears to have a more northerly distribution and is the only species recorded in Wisconsin. The two species are, however, similar in behavior and research workers often have not differentiated between them. There are at least two generations per year as far north as the Lake States (McMullen *et al.* 1955b). In Ohio, all the stages successfully overwinter except the pupae (Rexrode 1969). In Wisconsin, however, the larger larvae are the only winter-resistant stage, and emerge as adults in May (McMullen *et al.* 1955b).

Breeding and feeding habits.—Pseudopityophthorus spp. most commonly breed in oak, although other hosts have been recorded. Interestingly, McMullen *et al.* (1955b) in Wisconsin found that *P. minutissimus* was common in trees of the red oak group but was not found in white or bur oak. Breeding normally takes place in stems or branches from 1- to 10-cm diameter, although it has been recorded in a 42-cm diameter tree in West Virginia (Rexrode *et al.* 1965). The proportion of oak-wilted trees that are successfully colonized ranges from near 0 to 50 percent. It seems to be at its highest in Missouri where most of the diseased black and scarlet oaks (*Q. velutina* and *Q. coccinea*) were attacked (Buchanan 1956) and where successful breeding occurred in 45 out of 87 wilted trees (Rexrode and Jones 1972). In West Virginia, attack in the main stem occurred in 8 of 27 trees in 1961 and 4 of 30 trees in 1962 (Rexrode *et al.* 1965). Studies of trees that had been dead for several years suggested that these figures were above average. Attack on small branches occurred in half the

trees in both years. In 1967 in Ohio Rexrode (1967) found only 3 out of 27 trees to be attacked, and colonization was light and restricted to the branches. In Minnesota, although small low branches on healthy trees almost invariably become colonized when they die from "shading", attack on the crowns of oak-wilted trees is usually light and concentrated on branches from 2 to 5 cm in diameter (Gibbs 1980a). From the observations in Minnesota, and earlier in Missouri (Buchanan 1956) and Wisconsin (McMullen *et al.* 1955b) it seems that *Pseudopityophthorus* spp. are best adapted for the colonization of slowly dying branches. Attacks on diseased trees before defoliation has occurred are normally abortive (Rexrode and Jones 1970) presumably because host resistance is too high. After defoliation, branches quickly become unsuitable for breeding, probably because the tissues dry out rapidly and the bark is colonized by fungi such as *Dothiorella quercina* and *Coryneum kunzei* (Gibbs 1980a).

Although it is clear that the bark beetles do not take full advantage of the death of trees through oak wilt, many can emerge from a single tree. In Ohio, Donley (1959) found that an average of 11,400 Scolytids (virtually all *Pseudopityophthorus* spp.) emerged in spring from a 15-cm diameter diseased black oak.

The feeding habits of the oak bark beetles on healthy trees were first investigated by Griswold and Neiswander (1953) and Griswold and Bart (1954). They concluded that *Pseudopityophthorus* spp. commonly made deep feeding wounds in the crotches, leaf axils, bud axils, and immature acorn axils of both red and white oaks. Rexrode and Jones (1970) reported that fresh feeding wounds could readily be found from mid-April onwards in Missouri, Ohio, and West Virginia. Feeding was primarily in the top branches of dominant trees and occurred mainly at the node between the previous year's and the current year's growth. The beetles bored through the bark, cambium, and xylem to the center of the twig. Rexrode and Jones found that such wounds acted as infection courts when they were inoculated with a spore suspension of *C. fagacearum*.

Bark beetles and *C. fagacearum*.—The first evidence that bark beetles could carry *C. fagacearum* was provided by Buchanan (1956) who obtained some infection when large numbers of bark beetles from oak wilt trees were macerated in water and placed on wounds of healthy trees. Stambaugh *et al.* (1955) reported that between 0.7 and 7 percent of the *Pseudopityophthorus* beetles emerging in June and July from trees that had wilted earlier that year were contaminated with *C. fagacearum*. In Missouri Berry and Bretz (1966), also working on a *Pseudopityophthorus* generation that emerged during midsummer, found that beetles from 9 out of 12 trees were carrying *C. fagacearum*. As many as 30 percent of the beetles were contaminated from some of the trees. In general the mating type of the fungus on the beetles was the same as that from the xylem of the trees from which they emerged. A few beetles were carrying the other mating type, however, and both mating types were found on beetles from five of the nine trees. Several studies have shown that both mating types are rarely found together in the xylem of an infected tree (Boyce and Garren 1953, Barnett and Staley 1953). Therefore, some of the *C. fagacearum* present on the emerging beetles was probably introduced to the branches by the parent beetles when they entered to breed. This is known to occur in elms infected with *Ceratocystis ulmi* (Lea 1977).

Beetles emerging during the latter half of the summer are not likely to act as vectors. Host susceptibility is low and late summer feeding is concentrated on the petioles of the leaves of the current shoots which provide a less favorable avenue for infection than spring feeding wounds[1]. With these considerations in mind Rexrode and Jones (1971) examined beetles that emerged in early spring from the small branches of trees that had wilted the previous July. They recorded *C. fagacearum* on beetles from 8 of 17 trees from Missouri and 7 of 15 trees from West Virginia. The percentage of contaminated beetles from all the trees together was between 0.4 and 2.5. They also reported that the fungus was present on three immature stages of the beetle—larvae, pupae, and tenerals —and also in the frass. No sporulating mats were found on any of the branches and attempts to isolate the fungus from the branches during the time of beetle emergence were unsuccessful.

Parent beetles might also be transmitters of the disease in the spring. They may make a gallery system in a diseased tree, emerge to feed on twigs of healthy trees, and then breed again in a healthy tree. Rexrode *et al.* (1965) found that between 0.5 and 5 percent of 440 parent adults re-emerging from the stems of wilted trees in West Virginia were carrying *C. fagacearum*. They suggested that the beetles ingested the fungus from the xylem vessels while making the galleries. It is also possible, however, that they were already contaminated when they entered the trees and retained the fungus on or in their bodies while there.

Extensive microscopic examination of beetle galleries by J. G. Leach and others (see Rexrode *et al.* 1965) have failed to reveal mycelium or spores of *C. fagacearum,* and the mechanism whereby the beetles become contaminated with the pathogen is not clear. Both inner bark and outer xylem are possible sources because early larval stages develop principally within the bark and later larval stages scar the xylem. If the inoculum comes principally from the strain of the fungus that kills the tree, the xylem is the more likely source because *C. fagacearum* rarely invades the inner bark of small branches (Gibbs 1980a). Nothing is known about situations in which the fungus might be introduced to gallery systems by beetles entering to breed. By analogy with Dutch elm disease, development of the pathogen in either bark or xylem seems possible.

Other tree-wounding insects

Although the oak bark beetles have received the most attention, they are not the only possible vectors among the tree-wounding insects. The other chief candidates include the flat-headed borers *(Buprestidae)* such as the two-lined chestnut borer *(Agrilus bilineatus)* (Rexrode 1968). Young adults emerge from the trunks of wilted trees and then feed on twigs and leaves of healthy trees. Although the necessary period of at least a year for larval development would be expected to reduce greatly the number of adults carrying *C. fagacearum,* Stambaugh *et al.* (1955) found that between 4 and 20 percent of a sample of 128 insects were contaminated with the fungus. However, Craighead *et al.*[2] did not achieve infection when 200 field-collected beetles were caged on small trees. Himelick and Curl (1958) also obtained negative results, even though beetles they used had been exposed to cultures of *C. fagacearum.* The flat-headed apple tree borer, *Chrysobothris femorata,* has similar habits and Himelick and Curl (1958) and Donley (1959) achieved some infection of seedlings with artificially contaminated insects of this species.

Donley (1959) found that artificially contaminated adults of the round-headed borer *Urographis fasciatus* infected 16 of 135 oak seedlings. It is interesting to note that these results are almost identical to those obtained by him for *P. minutisimus,* although the number of *U. fasciatus* adults used per cage was only one-third of the number of oak bark beetles. Whether *U. fasciatus* ever carries the pathogen in nature, however, has not been determined.

With a life cycle that may be as short as 6 weeks, the Ambrosia beetles might be expected to carry the pathogen frequently, and consequently they have been studied in some detail. *Xyleborus* spp. and *Xyloterinus politus* were found emerging through oak wilt mats, and, not surprisingly, many of them were contaminated with *C. fagacearum* (Stambaugh *et al.* 1955). Stambaugh *et al.* (1955) also found that between 2 and 10 percent of 100 adults of *X. politus* from the sapwood of wilted trees were carrying the pathogen, and Skelly (1966) found the pathogen on various Ambrosia beetles from the roots of wilted trees. However, it has recently been concluded that these insects do not act as vectors, some species because they do not attack healthy trees and others, in particular *X. politus,* because they are no longer carrying the fungus by the time their tunnels reach the xylem (Wertz *et al.* 1971).

The only quantitative data about the relative abundance of these insects are those obtained by Donley (1959) in Ohio from artificially inoculated 15-cm diameter black oak. He found that about 230 Buprestids and 150 Cerambycids (round-headed borers) emerged from each tree, but that these were out-numbered by the Scolytids (99 percent *Pseudopityophthorus* spp.) 50 to 1 and 75 to 1, respectively. Together with the other data presented here, this is enough to indicate that the oak bark beetles have good claim to be regarded as the most likely of the tree-wounding insects to spread infection.

Squirrels and other animals

In the north-central States, squirrels commonly feed on sporulating mats and consequently, have been suspected of transmitting the disease (Himelick *et al.* 1953). Transmission by squirrels has been reported under artificial conditions (Himelick and Curl 1955) but there is little reason to think that these animals are natural vectors (True *et al.* 1960). It has been postulated that birds might act as vectors, becoming contaminated while feeding on insects that inhabit the sporulating mats. There is no evidence for this (Tiffany *et al.* 1954, True *et al.* 1960), however, the downy and hairy woodpeckers (*Dendrocopus* spp.) might merit further investigation because they have been reported to make peck marks on healthy trees.

INFORMATION FROM RESEARCH ON CONTROL

Experiments on disease control are potentially a good source of information on the mechanisms of

disease spread. Early control work in central Wisconsin in which roots were severed by mechanical or chemical means provided strong supporting evidence for the importance of root graft transmission. Also, the prompt application of tree paints to wounds provided evidence for the importance of those wounds as infection courts (Kuntz and Drake 1957). Other work has given more equivocal results, particularly because any one treatment may influence several possible mechanisms of spread. In the large-scale experiments carried out in West Virginia between 1970 and 1972, Rexrode and Frame (1973) found that felling wilted trees reduced bark beetle breeding by half and the production of fall sporulating mats almost to zero. Deep girdling to the heartwood, as carried out in the West Virginia control program, had no effect on beetle breeding but did reduce fall mat production by about 75 percent. However, 2 years of these treatments had no effect on the incidence of new infection centers.

A later series of experiments involved the injection of cacodylic acid, which was more effective than felling both in reducing beetle breeding and mat production (Rexrode 1977). During the 4 years of the study, the number of new infection centers was 13 percent less in the treated than the untreated plots. In the last 2 years of the study, the reduction in new centers in the treated plots was 26 percent. There was a similar reduction in the number of 'breakover centers' (newly infected trees within 15 m of a previously diseased tree) but part of the effect here might have been due to a lower frequency of root transmission.

INFORMATION FROM THE DISTRIBUTION OF THE DISEASE

Until now, little consideration has been given to the geographical distribution of oak wilt in the United States and, in particular, to its static nature and clearly defined boundaries. Some of these boundaries can be readily explained. The western limits of the disease coincide with the original prairie/forest boundary beyond which few large populations of red oak exist. A reasonable explanation of the sharply defined southern boundary in Arkansas can be found in terms of the effects of high temperature and competing fungi on the saprophytic survival of the pathogen in diseased trees (Tainter and Gubler 1973). Other boundaries cannot be so readily explained. In Minnesota and Wisconsin the disease has a distinct northern limit, which is not marked by any obvious difference in host population, climate, or soil. The same is true of the distribution of the disease in the East. True *et al.* (1960) produced a map showing those parts of the Appalachians in which oak wilt was concentrated. The same woodland types, physiographic characteristics, and shallow soils are found to the northeast and the southwest of this affected area and True *et al.* expected the disease to spread into these areas. No such spread has occurred. This is demonstrated in a striking way in Pennsylvania where the disease has a particularly abrupt eastern boundary (Craighead and Nelson 1960). Theoretically studies conducted on either side of these transition zones should provide useful evidence on mechanisms of disease transmission.

CONCLUSIONS

Through much of the oak wilt range, transmission of the disease via root grafts is responsible for most of the mortality. This is particularly evident in parts of Minnesota and Wisconsin where infection centers enlarge steadily through the summer. It may also be true in Illinois, Pennsylvania, and West Virginia. The frequency of root grafts is lower in these States than it is further west but it seems that root transmission best explains the facts that (1) a high proportion of wilting trees are within a few meters of ones previously killed by the fungus and (2) several years may elapse between the death of one tree and the appearance of symtoms in another.

In these five States most overland spread can be explained in terms of transmission by Nitidulids. In Minnesota and Wisconsin, sporulating mats are commonly found on diseased trees in spring, and for both these States and for Pennsylvania there is a wealth of evidence to show that disease commonly appears in trees that were wounded in the spring (May-June). The fact that many wounded trees escape infection is explicable in terms of factors influencing the movement of Nitidulids, the presence of spores on Nitidulids and the condition of the wound surface.

It does not seem to be a coincidence that the part of the oak wilt range within which infection by Nitidulids is most important, is also the area in which root transmission is most conspicuous. The abundant root transmission leads to the wilting of many trees in July and August. These trees produce mats the following spring at the time when trees are most susceptible to infection.

In the southern part of the oak wilt range, in particular in southern Ohio and Missouri, workers are less persuaded of the importance of root infection than their colleagues further north; although here, as elsewhere, many of the trees that die are within 15 m of infected ones. Natural infection of wounds has been shown to occur, but in general there is little enthusiasm for Nitidulids as vectors, chiefly because sporulating mats are so rarely produced. The oak bark beetles appear to be better equipped to act as vectors in these States than further north, principally because all development stages can survive the winter. In addition it appears that the beetles may breed in larger diameter branch and stem material. Consequently, they have a greater chance of linking up with the pathogen. The relation between the diameter of the branch used for breeding and the occurrence of *C. fagacearum* on beetles emerging from that branch is a matter meriting further investigation.

It has been nearly 40 years since oak wilt was first described in Wisconsin, and, in many parts of the United States, the disease now arouses only limited interest. It must not be forgotten, however, that the present situation could change rapidly if transmission of the disease were to become more efficient. The European oak bark beetle *Scolytus intricatus* Ratzeburg would seem equipped to be a formidable vector of *C. fagacearum* (Gibbs 1978), and care should be taken that this and other similar exotic insects do not become established in North America.

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☆ U.S. Government Printing Office: 1980—669-244/54 Region No. 6

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1980. The transmission of oak wilt. U.S. Department of Agriculture Forest Service, Research Paper NC-185, 17 p. U.S. Department of Agriculture Forest Service, North Central Forest Experiment Station, St. Paul, Minnesota.

Provides an up-to-date review of factors affecting the transmission of oak wilt, *Ceratocystis fagacearum*. Discusses the history and severity of the disease, the saprophytic existence of the fungus in the dying tree, seasonal susceptibility of trees to infection, overland and underground spread, the role of animals and insects as vectors or tree wounders, and the distribution of the disease.

KEY WORDS: *Ceratocystis fagacearum, Quercus,* vectors, fungus, forest management.

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