Local Spread of Oak Wilt in Northeastern West Virginia During 1970–1982

MANFRED E. MIELKE, Plant Pathologist, Forest Pest Management, USDA Forest Service, Morgantown, WV 26505; CLARK HAYNES, Plant Pathologist, Plant Pest Control Division, West Virginia Department of Agriculture, Charleston 25305; and CHARLES O. REXRODE, Principal Entomologist, USDA Forest Service, Northeastern Forest Experiment Station, Delaware, OH 43015

ABSTRACT

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In 1982, 41 oak wilt centers originally found in 1970–1973 in northeastern West Virginia were revisited to determine the presence of oak wilt and the increase in oak mortality since the time of discovery. Fourteen centers had no dead or wilting trees. Ten centers had wilting trees and 27 had dead or dying trees within 15.2 m of the tree that died first on the site. Neither cacodylic acid injections nor deep girdling in 1970–1973 affected the subsequent incidence of mortality compared with no treatment. The average rate of increase in the 27 centers showing symptoms of oak wilt was 0.39 newly diseased trees per center per year. Total basal area per hectare was affected little by oak wilt.

The oak wilt fungus (*Ceratocystis fagacearum* (Bretz) Hunt) was first discovered in West Virginia in 1951 (8). At that time, it was found throughout the state and seemed to be spreading. Characteristics of the organism and the pattern of disease occurrence implicated insects as overland vectors and root grafts as important for local intensification (7). Overland spread of oak wilt was demonstrated in 1953, when sap beetles (Coleoptera: Nitidulidae) visited fungus mats on affected trees and vectored propagules from mats to wounds on healthy trees (1). Oak bark beetles, *Pseudopityophthorus* sp. (Coleoptera: Scolytidae), were later implicated as important vectors of the oak wilt fungus in the absence of fungus mats (6). Subsequent work indicated that root-graft incidence at oak wilt sites were more prevalent and potentially more important than previously reported (5). Cacodylic acid injected into infected trees reduced mat formation and appeared to prevent spread of the fungus through roots (4).

The first attempts to control oak wilt involved cutting and burning and were designed to check the overland spread from infection centers (7). Later attempts involved injecting aqueous copper sulfate into deep girdle wounds in diseased trees to facilitate their drying and enable competing fungi to enter, thereby reducing fungal mat formation. Copper sulfate had some effect in reducing mat formation; however, control trees girdled to the heartwood and debarked below the girdle produced even fewer mats (7). The deep girdle and bark peeling method became the primary method of control. An appraisal of oak wilt control programs conducted from 1958 through 1968 indicated this method reduced local spread of the fungus in Pennsylvania and southern West Virginia but not in northeastern West Virginia (2). No methods to stop spread through root grafts were adopted in West Virginia forests because chemical and mechanical severence proved unsatisfactory.

This study evaluates the incidence and rate of local spread of oak wilt during 1970–1982 in untreated centers and in centers treated by cacodylic acid injections and deep girdle control methods.

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MATERIALS AND METHODS

Records for about 100 centers where oak wilt first occurred during 1970–1973 were obtained from the West Virginia Department of Agriculture, Charleston. During July and August 1982, we visited these centers, located in Grant, Hampshire, Hardy, and Mineral counties in the eastern panhandle region of the state, and at each site, attempted to reestablish a plot of 15.2-m radius measured from the tree that had first become diseased. Forty-one plots were located by discovery of the stem or stump of the original infected tree. Each plot originally contained nine or more living oaks. Data taken on each plot included basal area by species, number of oaks killed since the original discovery, and number of actively wilting oaks.

RESULTS AND DISCUSSION

Between the time of initial detection and 1982, additional mortality occurred in 60% of (12 of 20) centers treated by deep girdle, 57% (4 of 7) treated by injection, and in 79% (11 of 14) untreated plots. Thus, the control treatments were not effective.

Total basal area per hectare did not differ significantly ($P<0.01$) between centers with subsequent mortality and/or active symptoms and centers without subsequent mortality. Basal area per hectare of individual tree species also did not differ significantly. Members of the red oak and white oak groups were similarly represented in both groups of plots. This corroborates previous data on natural regeneration in oak wilt centers (3). Basal area data are summarized in Table 1.

Annual aerial and ground surveys for oak wilt conducted since 1955 have shown Grant, Hampshire, Hardy, and Mineral counties to be among those with the highest incidence of oak wilt in the state (9). During 1955–1977, except for 1965 and 1966 (data unavailable), an average of 0.19 trees per center per year died from oak wilt in those counties (9). Data from the 27 centers where trees died after the original discovery show the rate of increase over the past 9–12 yr to be 0.39 trees per center per year. This difference may be insignificant due to the small sample size or may reflect the fact that we looked only at old established centers with nine or more oaks. Such sites would be favorable for spread by root grafts. In addition, old disease centers are more likely to have multiple tree mortality in any given year than new infection centers, which tend to have single affected trees.

C. fagacearum is still present and continues to kill oaks in West Virginia.

Table 1. Basal area (m^2/ha) of tree species in oak wilt centers established during 1970–1973 in relation to subsequent oak wilt in 1982[a]

Oak wilt status of plots	Red oak group[b]	White oak group[c]	Other[d]	Total
Active symptoms and/or trees killed since original discovery	5.02	7.89	4.74	17.65
No mortality since original discovery	3.94	8.20	5.90	18.04

[a] There were no significant differences ($P < 0.01$) between plots.
[b] *Quercus rubra* L., *Q. coccinea* Muenschh., and *Q. imbricaria* Michx.
[c] *Quercus alba* L., *Q. prinus* L., and *Q. stellata* Wang.
[d] *Pinus strobus* L., *P. virginiana* Mill., *Carya* spp., *Acer rubrum* L., *Fraxinus americana* L., *Sassafras albidum* Nutt., *Juglans nigra* L., *Robinia pseudoacacia* L., *Nyssa sylvatica* Marsh., and *Ulmus americana* L.

However, stand composition and stocking vary little between active and inactive oak wilt centers. Therefore, ecological effects seem minimal. Although oak wilt has not caused the devastation once feared, infected trees die rapidly and the potential for great damage exists. This damage possibly could still be realized with the introduction of a more efficient vector.

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