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Dutch Elm Disease and Elm Yellows in Central New York

Out of the Frying Pan into the Fire

The American elm, *Ulmus americana* L., faces the specter of two lethal epidemic diseases, Dutch elm disease (DED) and elm yellows (EY). DED is unquestionably one of the most infamous and intensively studied of all tree maladies. EY, also known as elm phloem necrosis, is little known outside the mideastern United States where scattered outbreaks have occurred. Remnant mature specimens and many juvenile American elms usually remain in the wake of a DED epidemic. In central New York, an EY outbreak has virtually eliminated a population of mature and juvenile American elms within which DED had been controlled through sanitation and mass trapping of DED vectors. The explosiveness and intensity of this EY epidemic suggest that a new pathogen-vector relationship may have occurred.

Biology of the Diseases

Both DED and EY pathogens are transmitted by insects, but the organisms involved are quite different. DED is caused by a fungus, *Ceratocystis ulmi* (Buism.) C. Moreau, that is carried by bark beetles (Scolytidae) when new adults emerge from diseased elm material to feed in the twigs and branches of healthy trees. EY is believed to be caused by a mycoplasmalike organism (MLO) that is transmitted by sap-sucking insects (Homoptera). Many kinds of insects inhabit the bark of diseased elms, but in North America, only the European elm bark beetle, *Scolytus multistriatus* (Marsh.), and the native elm bark beetle, *Hylurgopinus rufipes* (Eichh.), normally complete the DED infection cycle by visiting and inflicting wounds in healthy elms. Similarly, dozens of insect species may suck sap from elms (5), but the MLO and the insect must be compatible in order for the microorganism to multiply within the insect and invade its salivary glands. To date, only the whitebanded elm leafhopper, *Scaphoideus luteolus* Van Duzee (1,9), has been demonstrated to be capable of regular transmission of the MLO associated with EY; single instances of EY induction were recorded in tests with two other insects, *Allygus atomarius* (Fab.) and the meadow spittlebug, *Philaenus spumarius* (L.) (9).

History and Origin

DED was first reported in Belgium, France, and the Netherlands in 1918 and in North America (Ohio) in 1930 (23). EY has been traced back to about 1880 when many elms began to yellow and die in Kentucky, Indiana, and Illinois (4). The origin of neither pathogen is known (23). DED and EY outbreaks occurred together in Ohio during the 1940s (20), in Champaign-Urbana, Illinois, in the 1950s and 1960s (3), and in the region of Ithaca, New York, in the 1970s (18). In these outbreaks, EY exacerbated DED by providing additional breeding material for bark beetles.

Sinclair (20) stated that an epidemic of EY proceeds at a slower pace than an epidemic of DED and that where both diseases occur, DED overshadows EY. We found the opposite to be true in central New York.

Intensive Study of an Elm Population for Ten Years

Identification of the aggregation pheromone of the European elm bark beetle (15) provided the impetus for developing a system for mass trapping this DED vector. Trials to assess the impact of trapping on beetle populations and incidence of DED were conducted in California, the Lake States, and the Northeast. The elm population in one of the northeastern areas, eastern Syracuse and adjacent townships (Fig. 1), provided the basis for intensive analysis of the impact of control measures on DED and documentation of a dramatic epidemic of EY.

An initial elm population delineated in eastern Syracuse (Fig. 1, area A) during the fall of 1974 and spring of 1975 consisted of 118 healthy "amenity value" American elms on street sides and in private yards. In 1976, 57 elms in DeWitt (Fig. 1, area B) were added, and in 1979, the study area was expanded to include an aggregate of 312 elms on 3,000 ha (Fig. 1, area C). About 5% of the elms in area A were slippery (*U. rubra* Muhl.) or European (*U. glabra* Huds., *U. carpinifolia* Gleditsch) elms; Asian species (*U. pumila* L., *U. parvifolia* Jacq.) were excluded because they are resistant to DED and therefore unsuitable for the original purpose of our study.

Elms were never the dominant shade tree in these areas. The scattered

distribution was probably the major reason a significant population had survived a DED epidemic that destroyed almost all of the elms in central Syracuse after an effective sanitation program was abandoned (for political reasons) in 1964 (11). Syracuse had no DED control program from 1965 until 1975, when we initiated our study.

In addition to the amenity elm population, naturally seeded juvenile American elms were very abundant in lots and green spaces. A random plot survey of 10% of areas A and B projected an aggregate of 4,500 young elms (2.5–15 m tall) within these areas. The parts added in 1979 were never sampled in the same manner, but we estimate that area C contained in excess of 100,000 elms (mostly young) in 1979.

Elms in the three base populations were individually listed and the state of health of each tree was recorded at least twice annually, in early July and early September. The DED infection rate assigned for any year was provisional until cryptic infections had been detected during the first inspection of the following year. If a tree was suspected to have been infected one or more years before the onset of foliar symptoms, year of infection was documented by dissecting the symptomatic portions of infected elms and observing the oldest annual rings in which discoloration occurred. Samples from about 10 trees that showed decline without typical DED symptoms were cultured in potato-dextrose agar for *C. ulmi*, but trees showing typical DED foliar symptoms and sapwood streaking were not routinely cultured.

Elm yellows was diagnosed on the basis of yellowing foliage in late summer, precocious exfoliation, flecking and yellowing of the phloem surface, and the odor of wintergreen in the discolored phloem. Unlike DED, symptoms of EY usually appeared throughout the entire tree (Fig. 2). Because the incubation period between inoculation of the infective agent and symptom expression is uncertain, incidents of EY were always scored in the year that symptoms were first observed.

The study areas were established to monitor the impact of recently developed DED management techniques. These were mass trapping of beetles on pheromone-baited sticky traps (1975–1984) (8), treatment of diseased elms by injecting the fungicides thiabendazole (Arbotect) and carbendazim (Lignasan) (22) with or without pruning of infected limbs (2), and the cacodylic acid trap tree technique (1979–1983) (8,12). The municipalities made no deliberate changes in sanitation (removal of diseased trees) during our study, and the elm population was never routinely sprayed with insecticides, although some private individuals contracted with arborists to spray methoxychlor on elms on or adjacent to their properties.

In 1975, we initiated mass trapping in area A by placing 104 traps in clusters of four or five in areas devoid of elms. The trapping was expanded into area B in 1976 with the placement of 20 additional traps. Trap positions remained fixed until 1979, when a total of 172 traps were scattered in 43 clusters in area C. In 1979, we began using the cacodylic acid trap tree technique to eliminate diseased elms and further reduce the beetle population.

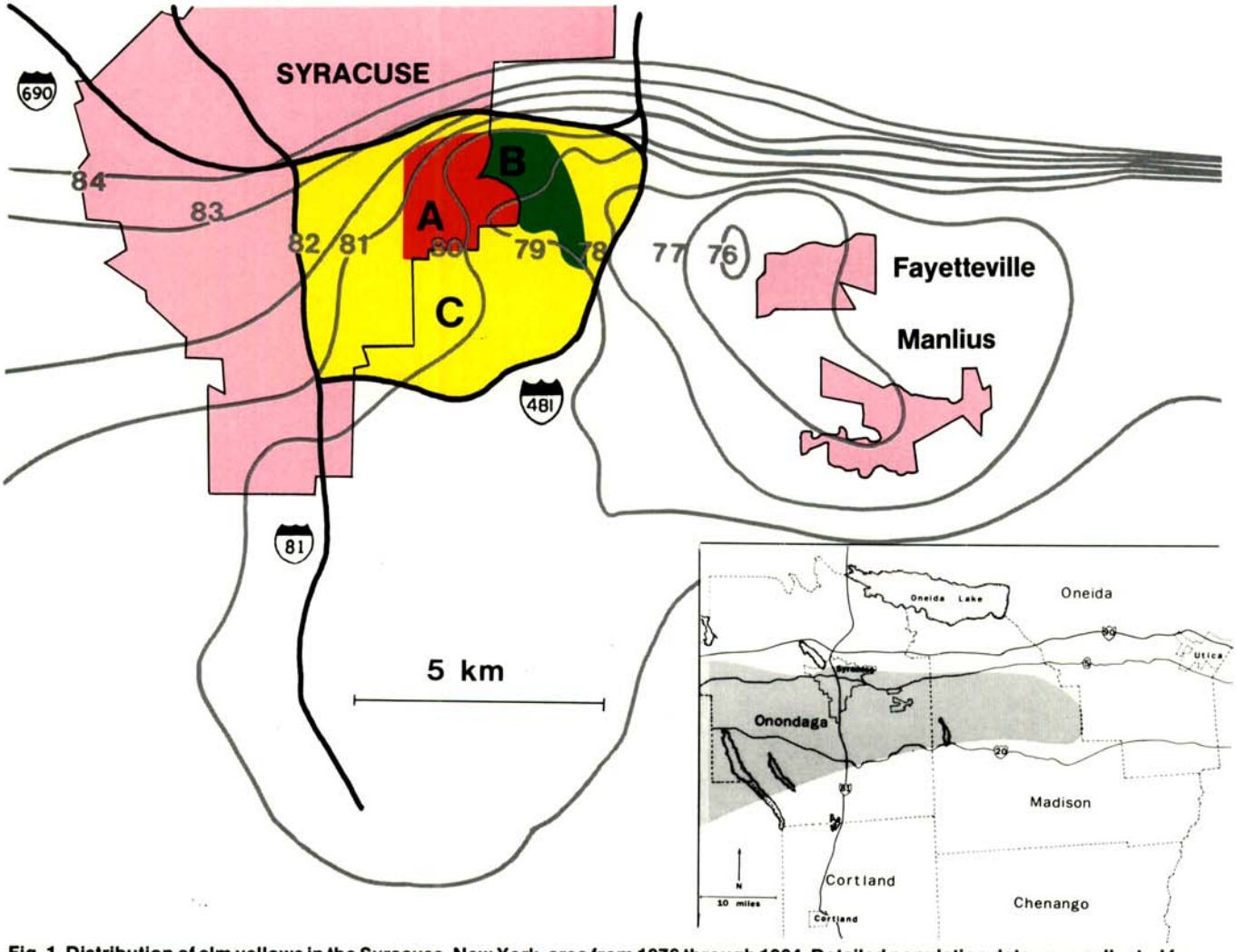


Fig. 1. Distribution of elm yellows in the Syracuse, New York, area from 1976 through 1984. Detailed population data were collected from 1975 to 1985 in eastern Syracuse (A), from 1976 to 1985 in an adjacent section in the town of De Witt (B), and from 1979 to 1985 in the expanded study area (C). In 1981, the Syracuse outbreak coalesced with elm yellows moving from the west. (Inset) Distribution of elm yellows in Onondaga and Madison counties in 1985.

This technique appeared to reduce DED by 50% in both 1978 and 1979 when it was the subject of a controlled test in southwestern Syracuse (11).

DED Control and EY Insurgence

Decline of DED has been reported to coincide with mass trapping of European elm bark beetles (8,14) and application of the cacodylic acid trap tree technique (8,12). Furthermore, when an index of beetle twig feeding injuries was correlated with DED infection rates, twig feeding indices were found to be consistently lower inside than outside areas A and B where beetles were mass-trapped (16). These results infer that mass trapping and trap tree operations caused the observed decreases in DED rates. In another study, using a different trapping strategy, no reduction of DED rates could be attributed to mass trapping, even when the beetle population was severely depleted (13).

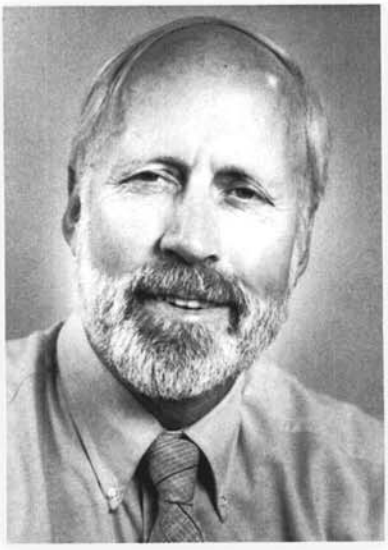
During 1975–1985, an aggregate of 33% of the 118 listed elms in area A were infected by DED. Because of successful therapy, however, only 21% of the elm population was lost to DED over the 11-year period (Fig. 3). The EY outbreak was much more severe: 58% of the elm population was infected and lost during 1981–1984. Ten percent of the trees were lost to other causes. At the end of the 1985 growing season, only 11% of the original population remained.

Logit analysis (24) shows that the rate of DED in area A decreased substantially during 1977–1980 (Fig. 4). We attribute this to beetle trapping commencing in 1975 and to the trap tree technique initiated in 1979. The apparent infection rate (r) decreased from 0.50 for 1975–1977 to 0.12 for 1977–1980. If the 1977–1980 rate had been maintained, loss of 50% of the initial 1975 population would not be reached until 1990. When trees that were eventually lost to EY were subtracted from the base population, an r value of 0.06 for 1977–1985 was calculated; at this rate, 50% of the original population would endure through 2003 and 10% would remain through 2038. This is comparable to the lowest rates of DED losses reported by Sinclair (19). Unfortunately, the eruption of EY brought the population to the brink of extinction in just 4 years. The 1980–1982 rate for EY ($r = 0.93$) was similar to that reported for chestnut blight (10). As the population was decimated (1982–1985), the r value declined to 0.38. Although the base population was small, the r values calculated reflect the general features of the disease progression within the region.

The domination of EY in the base population of 57 amenity value elms in area B was spectacular. Between 1976 and 1985, five of these trees were lost to DED, four to "other" causes, and 46 to EY. The trees that remained in 1985 were European elms; we found no surviving American elms, even among the saplings that had been abundant along property boundaries and in green spaces.

Progression of the EY Epidemic

In an attempt to stem a possible epidemic of EY in our study area, we killed 3,000 green space elm saplings with cacodylic acid during 1978 and 1979.



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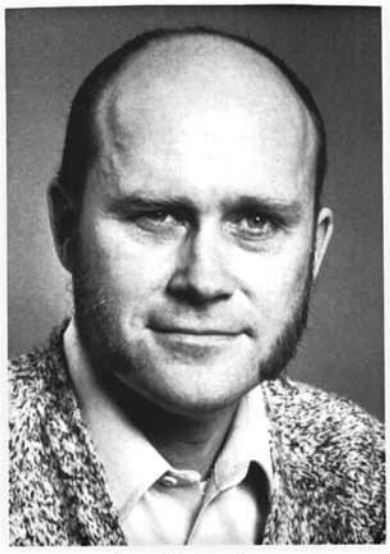

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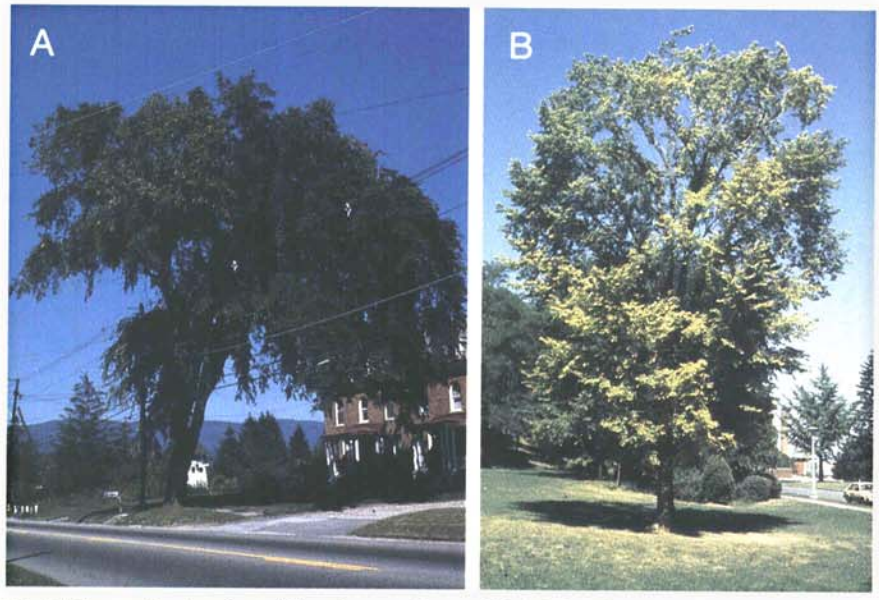

Fig. 2. Elms yellowing from (A) early symptoms of Dutch elm disease and (B) elm yellows. The sectional yellowing sometimes caused by Dutch elm disease is distinct from the uniform yellowing associated with elm yellows.

This effort probably dampened DED by absorbing great numbers of elm bark beetles, but it did not thwart EY. In 1979, we diagnosed EY in seven elms in our area B base population. The disease spread explosively, and by 1983 only two of the original population remained. We first found EY among young (non-inventoried) elms in area A in 1980, although none of the trees in our base population showed symptoms. In 1981, we diagnosed 21 new cases of EY in our area A 1975 base population. The disease intensified in 1982 but began to subside in 1983.

Regional Spread of EY

Biannual inventories of elm populations in the Syracuse region provided data that document the spread of EY from 1976 through 1985 (Fig. 1). The initial detection near Fayetteville, New York, included about seven trees, so it seems possible that EY was present a year or so earlier. Spring 1978 surveys showed that

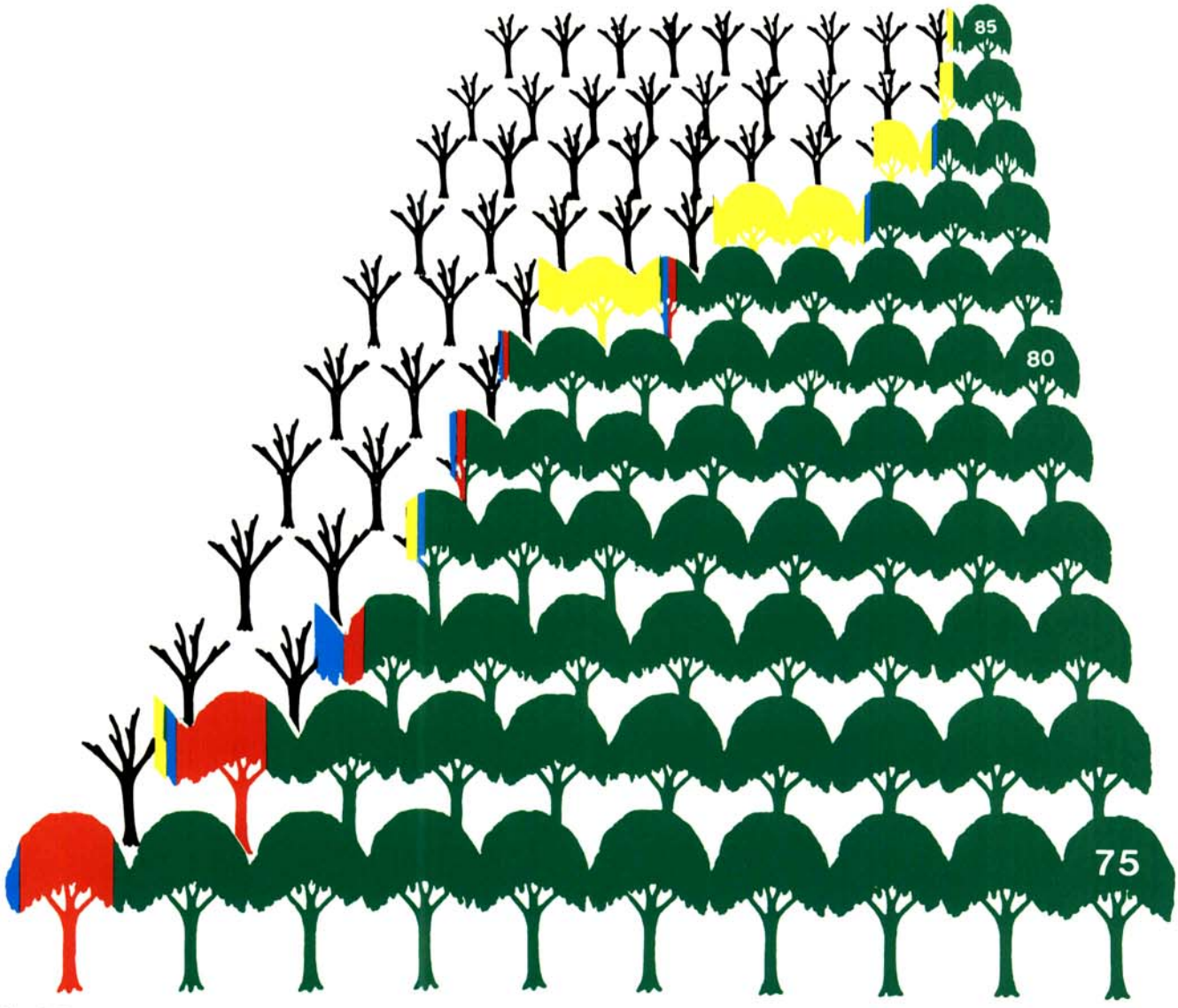

Fig. 3. Fate of 118 American elms in eastern Syracuse (area A in Figure 1) during 1975–1985. Annual losses to Dutch elm disease (red), elm yellows (yellow), and other causes (blue) are indicated on tree crowns. Each horizontal row represents a year, and each elm or elm carcass in a row represents 10% of the original population.

the range of EY had expanded throughout the Fayetteville-Manlius area and had reached the eastern boundary of study area B. Although we did not survey the Fayetteville-Manlius area in the late summer of 1977, elms that would have shown initial symptoms of EY during July–September 1977 were revealed in the spring of 1978 by their very weak foliation, chlorosis, dead roots, and dark yellow phloem. By July 1978, EY was so pervasive that a DED control study in this area was discontinued (11). EY spread eastward in a narrow band but moved westward about 1 km a year. During 1980–1981, the epidemic moved down the Onondaga Valley 6–8 km to Lafayette, New York. About 1980, the western front apparently merged with a separate outbreak moving from the west.

Despite the spread of EY to the west and south of Syracuse, the total movement northward has been less than 3 km in 10 years (Fig. 1). The limits of the southern advance of the EY outbreak seem to be related to topography, i.e., a hilly area at the south end of Cayuga Lake in Cortland County. Another factor may be differences in the abundance of sucking insects (Homoptera) between areas within the range of EY and areas immediately north of the disease border (17). Most notably, *S. luteolus* appears to be replaced by another, yet unidentified *Scaphoideus* species at the northern limit of the disease. Even within the EY outbreak area, however, *S. luteolus* did not seem to be abundant enough to account for the intensity of the epidemic (Matteoni [9] made the same observation). Other leafhopper species probably were responsible for local intensification of EY. Laboratory tests, however, failed to document transmission of EY by any of the species (including *S. luteolus*) that we tested (17).

The Aftermath

American elms of all size classes have been virtually eliminated in the villages of Fayetteville and Manlius and in study area B. The few prominent elms that remain in eastern Syracuse have been injected annually since 1982 with oxytetracycline HCl. To the west of area A, the EY infection rate appears to have declined since 1982, probably because elms here are more scattered and few fields are densely populated with juvenile elms.

Projected Southeastern Spread

Elm yellows was discovered in New York, New Jersey, and Pennsylvania in the early 1970s. An isolated occurrence of EY in eastern Massachusetts represents the most easternly record of the disease (7). The New Jersey, Pennsylvania, and Massachusetts outbreaks destroyed low numbers of trees and apparently subsided; only in New York has EY reached expanding and damaging epidemic levels. The Syracuse area epidemic moved eastward at about 7 km a year until 1984, when it stalled at high elevation in Oneida County. Another outbreak south of Lake Ontario and west of Syracuse expanded eastward and coalesced with the Syracuse outbreak, but EY present at the south end of Cayuga Lake in the early 1970s (18,21) apparently did not traverse a hilly area in Cortland County and did not merge with the Syracuse epidemic. High elevation (about 300 m) is clearly associated with the slow spread of EY in central New York, and an unknown factor, possibly displacement of the vector(s) by closely related species, has limited northward occurrence of EY beyond 43° 5′ latitude.

Eastward movement of EY from the Midwest appears to have occurred mainly through long-range dispersal of vectors aided by wind or perhaps by hitchhiking on motor vehicles (6). It seems probable that these modes of dispersal and the movement of infected growing stock will eventually expose elms throughout North America to the EY pathogen. The constraints of elevation and latitude that seem to limit EY in the Midwest and New York appear to be absent along the Atlantic seaboard and in the Deep South. Significant populations of American elms in cities such as Washington, D.C., Richmond, Virginia, and Atlanta, Georgia, that have

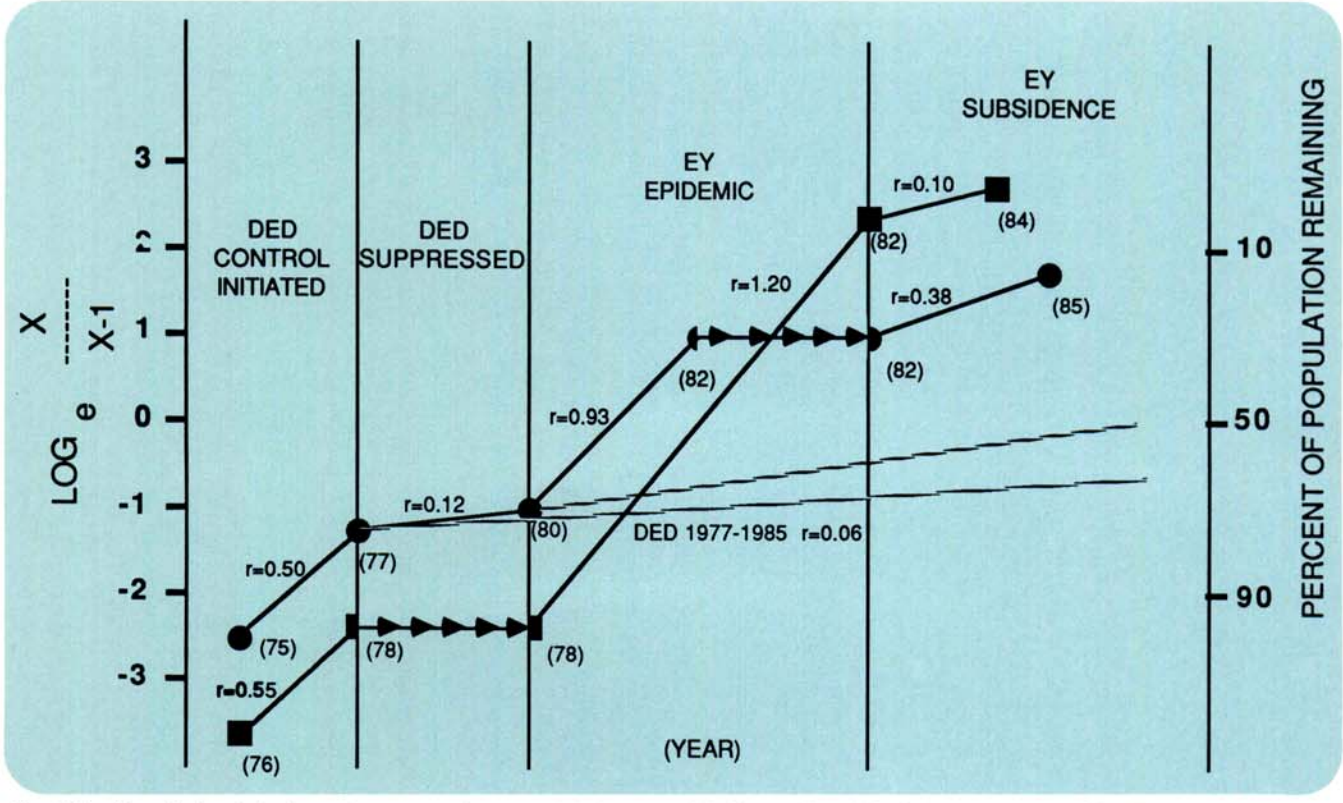

Fig. 4. Logit analysis of elm losses in eastern Syracuse (circles; area A in Figure 1) and De Witt (squares; area B in Figure 1) from initial suppression of Dutch elm disease through the elm yellows epidemic. In the absence of elm yellows, the 1977–1979 disease increase rate (r = 0.12) in eastern Syracuse indicates that 50% of the original population would be present through 1990. At the 1979–1985 Dutch elm disease loss rate (r = 0.06), one-half of the population would have persisted through 2003 and 10% would be alive in 2038, 63 years after control of Dutch elm disease was initiated.

persisted under the protection of DED management programs are in jeopardy of escaping the frying pan of Dutch elm disease only to be consumed in the fire of elm yellows.

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