UPDATE ON PHLOEM NECROSIS OF ELMS[1]

by W.A. Sinclair, E.J. Braun, and A.O. Larsen

Abstract. Phloem necrosis (PN), caused by a submicroscopic mycoplasmalike organism, is lethal in five elm (*Ulmus*) species native to North America. All American elms, including those resistant to Dutch elm disease, are highly susceptible. The causal organism, which has never been cultured apart from plant or insect hosts, spreads within infected trees only within phloem sieve tubes. Infected red (slippery) elm, less susceptible than American elm, develops witches' brooms before dying. Elms of European and Asiatic origin seem resistant. The causal agent was graft transmitted to clones of two European species, *U. carpinifolia* and *U. hollandica*, in which only witches' brooms developed, while no symptoms have yet been induced in Scotch (*U. glabra*) or Siberian (*U. pumila*) elms. The PN agent is naturally transmitted by adults of the white banded elm leafhopper, *Scaphoideus luteolus*, and possibly by other leafhoppers or planthoppers that feed on elm. PN epidemics are usually localized. In northern areas the disease is unknown where average minimum winter temperature is lower than -15 deg. F (-26 deg. C). Remissions of PN symptoms have occurred after trunk injections with tetracycline solutions in Mississippi and New York; but in northern areas death occurs so soon after first display of symptoms that therapy of infected trees may prove impractical on account of the low success rate. Protective antibiotic treatments have not been tested. No elm population has yet been preserved in the simultaneous presence of PN and Dutch elm disease.

Phloem necrosis (PN) of elms is one of several dozen plant diseases caused by mycoplasmalike organisms (MLO), transmitted by homopteran insects (leafhoppers, planthoppers, psyllids), and collectively called "yellows-type" diseases (12, 38). The name phloem necrosis (34) calls attention to a prominent internal symptom — death of the current season's phloem. This precedes death of the entire tree in highly susceptible species such as American elm. Epidemics of PN in many midwestern (8, 9, 10, 35), some southern (11, 14, 16, 22) and a few northeastern localities (24, 30, 37) have contributed to disappearance of elm shade trees. A 1974 report of PN in west-central New Jersey (37) extends the known range of the disease as reported previously (30). PN is unknown where average minimum winter temperatures drop below -26 deg. C (-15 deg. F) (31); the reason for this limitation is unknown.

Although information about PN is still scanty in relation to what is necessary for control, several developments of the past 5 years deserve the notice of arborists and others concerned with shade trees. This review is a mix of original and previously published information, plus speculation, intended to supplement the recent descriptions of gross symptoms (31, 32) and summarize current knowledge of the disease.

Association of MLO With Symptoms

Wilson et al. (40) reported MLO in secondary phloem of PN-infected American elms and the absence of such cells in healthy trees. Braun and Sinclair (7) confirmed this association and noted that MLO were apparently restricted to phloem sieve tubes, in which movement throughout infected plants is possible (Fig. 2A). The MLO were found erratically distributed in otherwise normal as well as discolored phloem of infected trees. Premature collapse of sieve tubes and companion cells is characteristic of PN (7, 23), but the presence of MLO in a sieve cell is not a prerequisite for collapse (7). Similarly, hyperactivity of the vascular cambium, resulting in abnormal production of phloem cells, is apparently stimulated at a distance from cells containing MLO.

Relationship of PN to Other Plant Diseases Caused by MLO

A few well-known MLO diseases are lethal. These include spike disease of sandal, lethal yellowing of palms, pear decline, and X-disease of peach (13, 18, 26, 28). A larger group of MLO diseases is characterized by initially sublethal effects; stunting, chlorosis, formation of witches' brooms, slow debilitation. Ash witches' broom (19) and *Salix* yellows (21) are examples.

Whether the PN agent is a rapid killer or just a broom-producing pathogen depends upon the host infected. American elms are usually dead within a year, often within a month, after first

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display of foliar symptoms. Brooming rarely occurs in this species. Red elm (slippery elm) is somewhat less susceptible, ordinarily displaying foliar symptoms in at least two growing seasons before death. Witches' brooms and a characteristic odor like that of maple syrup (31) usually develop during the final year of life. The relationships of brooming and odor to the better known symptoms of PN in red elm were made clear in a survey in central New York State. About 63% of PN-infected red elms developed brooms and 44% produced the maple syrup odor before death (Table 1).

Table 1. Relationship of witches' brooms and "maple" odor to other symptoms of phloem necrosis (PN) in red (slippery) elm in New York State.

Sample Group	Number of trees	Percent of trees with		
		Witches' brooms	Maple odor	Brooms and/or odor
Elms with foliar and phloem symptoms of PN	53	57	45	75
Elms with only foliar symptoms of PN	32	72	44	84
Recently killed elms near PN-infected trees and lacking xylem symptoms of Dutch elm disease	25	64	44	80
Total and averages	110	63	44	79

Witches' broom development one year after onset of foliar symptoms	Trees with PN	
	Without brooms in 1973	Broom development in 1974
Locality 1	14	10
2	13	7
Totals	27	17 (63%)

Asian and European species of elm may prove to be moderately or highly resistant to the PN agent. In experiments at Ithaca, N.Y., bark patches from PN-infected red and American elms, grafted into Siberian and Scotch elms, caused no external symptoms. Grafting of similar patches into Buisman elm (*Ulmus carpinifolia* cv. 'Christine Buisman') and a clone of *U. hollandica* in 1973 caused development of witches' brooms in 1974, and broom development continued in 1975. Non-grafted trees remained free of brooms. Foliar symptoms typical of PN in American or red elm did not appear in the European species, but MLO's in phloem sieve tubes of the brooms were confirmed by electron microscopy. When bark patches from brooms on a 'Buisman' elm were grafted into two American elm seedlings in a greenhouse, typical PN symptoms developed in one seedling after 8 weeks. It is not known whether natural transmission of the PN agent to these "exotic" elm species could occur.

Field observations also indicated resistance of European and Asiatic elms. Simultaneous epidemics of PN and Dutch Elm Disease (DED) killed nearly all large American and red elms on the Cornell University campus and in adjacent Ithaca, but Scotch, European smoothleaved, and Siberian elms have remained.

The only non-elm suscept to which the PN agent has been experimentally transmitted is periwinkle (*Vinca rosea*). This transfer was accomplished through the parasitic seed plant, dodder (*Cuscuta epithymum*). After strands of dodder were trained simultaneously on a PN-infected elm seedling and several periwinkle plants, two of the latter became chlorotic and stunted, developed witches' brooms, and failed to flower. The presence of MLO in these plants was confirmed by electron microscopy. To date, however, the reciprocal transfer, from periwinkle to elm, has not been accomplished. When trained on infected periwinkle, in which the titer of MLO is much higher than in elm, the dodder soon becomes feeble and cannot easily be reestablished on elm.

The ability of the PN agent to cause only witches' brooms in some elms and in periwinkle provides a basis for speculation about the origin of the disease. Since PN is lethal in 5 of the 6 native elm species that occur within the known range of the disease (susceptibility of rock elm has not been tested), the pathogen has presumably been introduced to North American elms from some other place and (or) some other type of plant. If it were an indigenous elm pathogen, natural selection would have favored native elms resistant enough to tolerate infection. Perhaps the PN agent is a common yellows-type MLO that causes only stunting and witches' brooms in its

usual host, and is occasionally transmitted to elm by a vector that usually feeds upon other plants. Or perhaps the PN agent is an elm pathogen native to, and unimportant in Europe or Asia, introduced to the USA during the 1800's in infected seedlings before they developed symptoms. Witches' brooms of elm are known in Europe (2). In any case, once introduced into susceptible North American elms, the PN agent was able to survive and multiply because of acquisition and transmission by indigenous vector insects.

Vector Relationships

W.L. Baker (3, 4, 5) showed efficient transmission of the PN agent between American elms by the white banded elm leafhopper, *Scaphoideus luteolus* (Fig. 1). This insect occurs throughout the known range of PN and has also been collected in more northern areas from Minnesota to Maine (25). Knowledge of its biology and habits is based upon Baker's observations (4) in Ohio. There *S. luteolus* overwinters as eggs in corky bark of small elm branches. The eggs begin to hatch soon after new foliage appears, and the hatching period is extended so that nymphs of two or three different instars (stages of development) may be found together. The nymphs pass through five instars within 36 to 42 days before reaching adulthood. The adults are present from early July until frost. Both nymphs and adults may be found in July and August. The nymphs, readily distinguished from other leafhoppers on elm by the white band across the middle of the back (Fig. 1B), prefer the inner and lower portions of elm crowns. Adults, which lack the distinctive white band, shows the same preference initially, but disperse throughout elm crowns late in the season. Only the adults are winged. Both stages feed via tubular sucking mouthparts in the phloem of leaf veins and succulent shoots. Thus they become infected with the MLO that causes PN.

For about 3 weeks after feeding on PN-infected elms, the leafhoppers are incapable of transmitting the MLO to healthy elms. Thereafter, the adult hoppers remain infective until death. Based upon knowledge of other leafhopper-MLO relationships, it is assumed that the PN agent parasitizes and multiplies within the leafhopper vector, and becomes available for transmission to new elms after spreading from the gut into the salivary glands. Presumably MLO infection adversely affects *S. luteolus,* but the literature contains no specific information. The causal agent of PN has not yet been cultivated apart from its insect or plant hosts.

Although *S. luteolus* is the only confirmed vector, there may be others. Gibson (17) listed 87 species of Cicadellidae (leafhoppers) and 13 species of Fulgoridae (planthoppers) that have been found on and probably feed on elm to some extent. Included were 13 known vectors of plant viruses or MLO and representatives of 19 genera that contain vector species.

Fig. 1

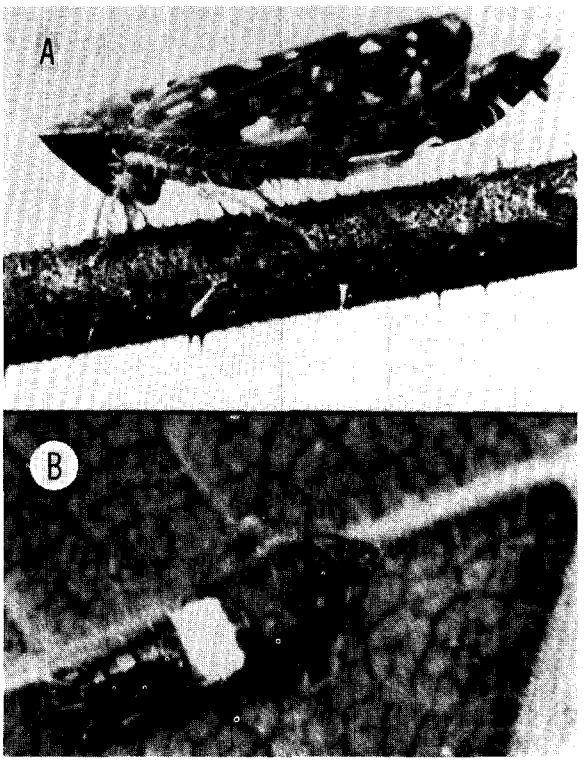

Figure 1. Adult (A) and nymph (B) of white banded elm leafhopper. Photo courtesy of U.S. Forest Service.

Pathological Histology

Some characteristics of healthy elm phloem must be appreciated as a basis for understanding pathological changes. In American elm, the normal conducting secondary phloem consists of sieve tube elements, companion cells, phloem parenchyma, fibers, and ray parenchyma (Fig.

Fig. 2

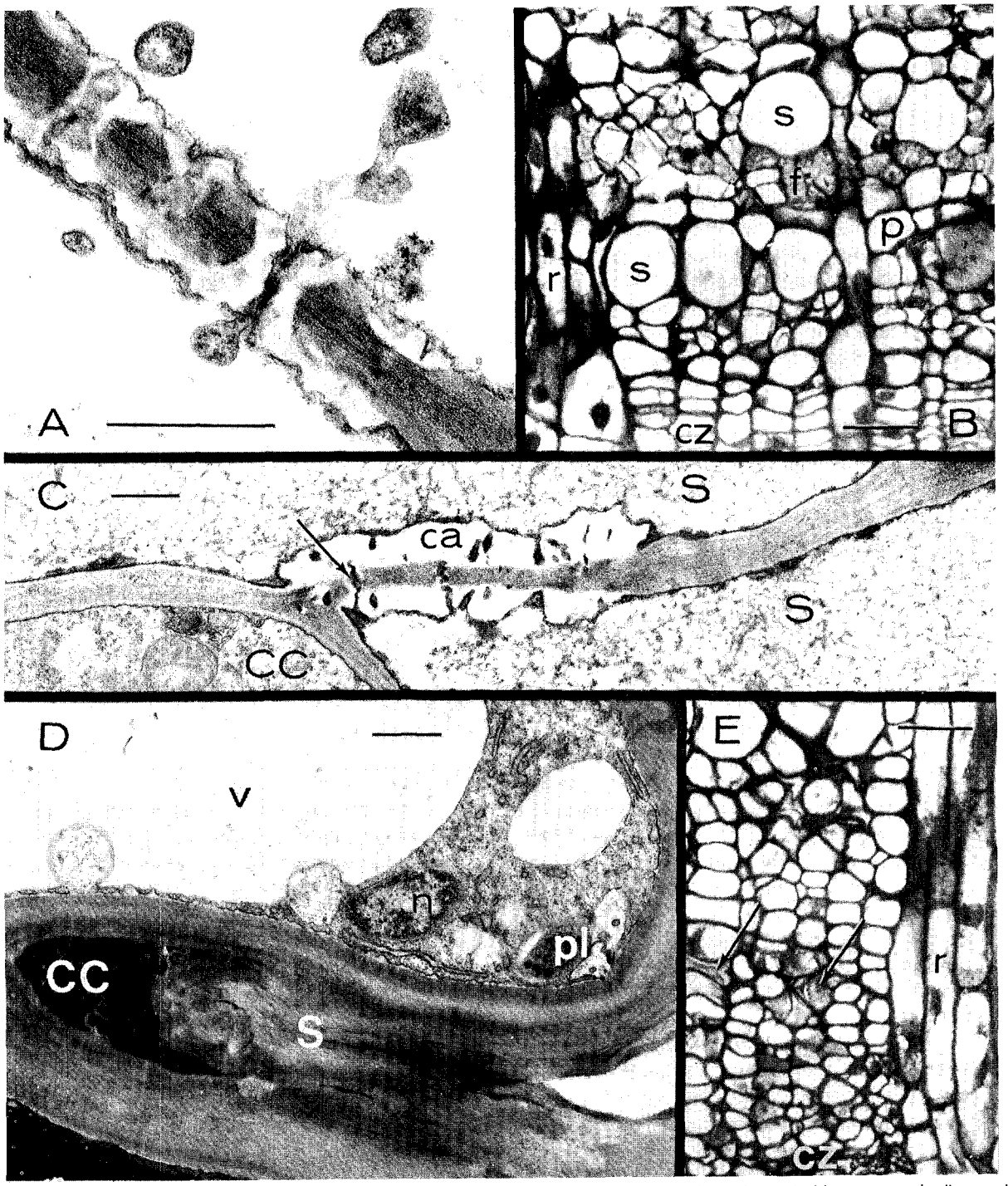

Figure 2. Light (LM) and electron micrographs (EM) of transverse sections from trunks of healthy and phloem necrosis-diseased American elms. A—Mycoplasmalike organism in pore of lateral sieve area between sieve elements in sample from diseased tree; EM, bar = 1 μm. B—General view of healthy phloem; cz = cambial zone, p = phloem parenchyma, r = ray parenchyma, f = fibers, S = sieve elemtns; LM, bar = 25μm. C—Callose (ca) on lateral sieve area in infected phloem; arrow indicates a pore in the sieve area; cc = companion cells, s = sieve element; EM, bar = 1 μm. D—Companion cell (cc) and sieve element (s) crushed by adjacent parenchyma cells in infected phloem; v = vacuole, n = nucleus, pl = plastid in a parenchyma cell; EM, bar = 1 μm. E—General view of diseased phloem before death; arrows indicate sieve elements for comparison to those in Fig. 2-B; cz = cambial zone, r = ray parenchyma; LM, bar = 25μm.

2B). Fibers and sometimes tannin-containing parenchyma cells are found in tangential bands; sieve elements, companion cells and parenchyma cells occupy the intervening spaces. The cells of greatest diameter are the sieve elements. Most sieve elements become fully mature when about 60 to 80 μm from the vascular cambium and are 25 to 30 μm in diameter. Contiguous sieve elements are connected via pores 0.5 to 5.0 μm in diameter in sieve plates and lateral sieve areas. These pores, through which MLO can pass (Fig. 2A) are lined with a carbohydrate substance called callose. The pores connecting older sieve tubes gradually become occluded with this callose. The conducting phloem is regenerated annually. At the end of each growing season all or nearly all of the sieve tubes and companion cells produced in that year collapse and are crushed.

In trees with PN the first observable symptom is abnormally rapid, heavy deposition of callose, plugging the pores that connect sieve elements (Fig. 2C). Next noted is premature collapse and crushing of sieve elements and companion cells (Fig. 2D). The collapse seems to occur very rapidly and with no observable changes in the cells beforehand. Abnormal deposition of starch grains in phloem parenchyma also occurs at about this time. These changes occur progressively closer to the vascular cambium, which becomes hyperactive, producing many new phloem cells (Fig. 2E) but few or no new xylem cells. This "replacement phloem" (29) contains no fibers; the frequency of sieve elements is more than twice the normal while their diameters are less than half normal (compare Fig. 2B and 2E). Sieve and companion cells in replacement phloem, before general necrosis of that tissue, undergo the same changes seen in normally formed infected phloem (7).

In infected trees that survive the winte phloem of normal color is formed as the next se son's growth begins. Discolored necrotic phloe of the previous season is pushed outward, a pearing as a dark tangential band in light colored tissue. By early to mid summer, howev phloem of the current season begins to darke The amount of wintergreen odor from th phloem, characteristic of PN in all native elm sp cies except red elm, is more or less proportion to the intensity of discoloration until the affected tissue begins to dry out. Then, or at summer's end, the odor disappears.

Epidemiology

PN epidemics, although spectacularly destructive, are usually localized. The spread of a PN epidemic into contiguous localities is neither quick nor certain. Observations in New York State suggest a rate of spread of 3 to 5 miles (5 to 8 km) per year in some localities, no spread or disappearance of PN in others. In a given locale, PN may exist endemically for many years between epidemic flareups. These observations are explainable if elm, vector, and MLO populations are considered as discontinuous in both time and space in a geographic region. Only where all three come together and are sustained for at least a few years can an epidemic develop. Theoretically, a collapse of any of the three populations will halt an epidemic. In actuality, epidemics often continue until few susceptible elms remain in affected localities.

Except in southern areas (16), Dutch elm disease occurs wherever PN does. The presence of DED tends to slow PN epidemics by reducing the availability of healthy elms (Fig. 3), but the overall rate of elm loss where both diseases occur is rapid until nearly all susceptible elms are dead.

Control

At present there is no proven method for prevention or cure of PN. For a time DDT sprays for vector suppression were recommended (1, 6) on the basis of tests to which USDA scientists alluded (5, 39) but did not report in detail. Current suggestions for vector control by insecticidal sprays (32) are not backed by experimental evidence of effectiveness.

Early reports of discovery and propagation of PN-resistant American elms gave promise of a new generation of elms not subject to PN, but these trees proved susceptible to DED (39). Recent experience is similar. All DED-resistant American elms selected by Sinclair et al. (33) died after graft-inoculation with bark patches from PN-infected trees. Two DED-resistant American elm clones selected by Ouellet and Pomerleau (27), including 'L'Assomption', were also tested at Ithaca, N.Y. and found susceptible to PN.

Fig. 3

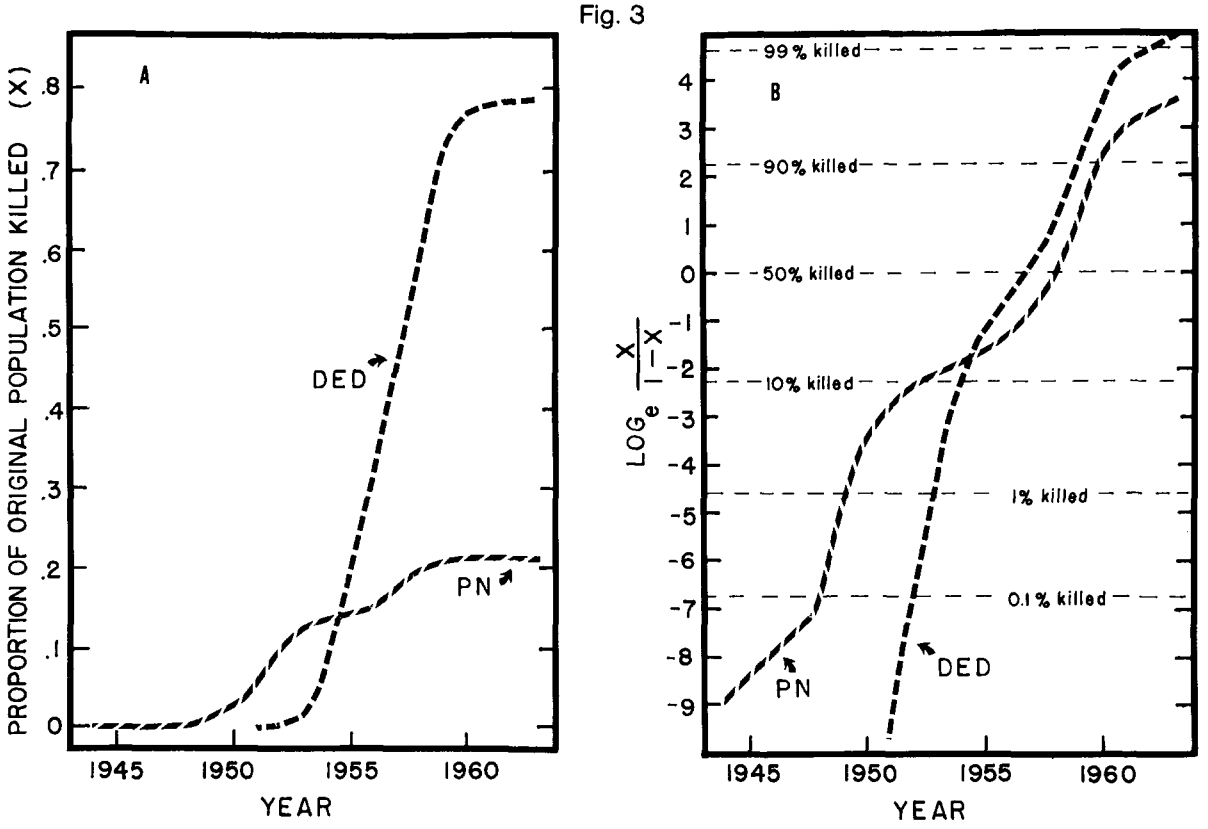

Figure 3. Disease processs curves for phloem necrosis (PN) and Dutch elm disease (DED) in Champaign-Urbana, Illinois; data of Carter and Carter (10). PN killed about 21% of the elms; DED killed the remainder. A—arithmetic plot. B—data converted to logits (logarithm of the proportion of the original elm population killed) and corrected for the diminishing number of healthy trees available for invection ($\log_e \frac{x}{1-x}$) (36). the logit transformation is used because tree losses in the early stages of these epidemics escalate exponentially. When losses are plotted on a logarithmic scale over time, the transformed data show that infection of 1% of the trees at an early stage is as important in epidemic development as infection of 10% of the trees at a later stage. Reasons for retardation of the PN infection rate in the mid-1950s are unknown.

Chemotherapy. In the field, PN-infected trees cannot be cured, but temporary remission of symptoms can be obtained by treatment with certain antibiotics at an early stage of disease. Filer (16) reported remission of PN symptoms in American and cedar elms in Mississippi after pressure injection (15) of solutions of tetracycline antibiotics into elm trunks. Trees found naturally infected in 1970-1972 were subjected to continuing tetracycline therapy, restored to apparent good health, and maintained in good health through 1975 (T.H. Filer, Jr.; personal communication).

At Ithaca, N.Y., tetracycline antibiotics applied in large volumes (e.g. 20 to 80 liters per mature elm) by high-pressure injection (20) have been less effective than Filer's treatments. Sustained remission of symptoms has not yet been obtained (Table 2). In the N.Y. tests a 10-minute injection period was arbitrarily set as standard, and stems were simultaneously injected at three to eight circumferential points near soil level. Best results to date have been obtained with two applications of 500 ppm active oxytetracycline (from Pfizer's experimental Terramycin Tree Injection Formula), one before and one at the end of the growing season (Table 2). Research on minimum effective doses and the necessary number of treatments per year is continuing.

One problem with chemotherapy under New York conditions is that after first appearance of foliar symptoms in large trees, decline and death

occur so rapidly that opportunities for chemotherapy are poor. Protective antibiotic treatments of valuable elms during PN epidemics would be a more logical approach to control, but no data for evaluation of this possibility are yet available.

Table 2. Influence of tetracycline antibiotics on elm mortality due to phloem necrosis (PN), Ithaca, NY.[a]

Test number and treatment	Dates of application	Typical total annual dose (g/cm dbh)	PN-infected trees alive after one growing season[b]	
			Treated	Control
1. Tetracycline HCl, 1000 ppm	May 1973	0.13	6 of 18	1 of 24
2. Tetracycline HCl, 1000 ppm	Sept. 1973	0.25	2 of 14	4 of 12
3. Oxytetracycline, 100 ppm	1 May 1974 7 June 1974 11 July 1974	0.22	5 of 13	—[c]
4. Oxytetracycline, 500 ppm	10 Oct 1974 25 May 1975	0.46	6 of 8[d]	—[c]

a Trees treated in spring were identified as infected during the previous September; those treated in autumn first showed foliar symptoms in July to September of the same year.

b Longer-term data could not be obtained because trees not killed by PN succumbed to Dutch elm disease.

c. All mature elms with PN in the test locality were treated.

d Observations 15 September 1975. This test originally included 12 mature elms, of which 4 were cut on account of Dutch elm disease. Of eight trees remaining, two were killed by PN; three showed yellowing of leaves indicating active PN, and three showed normal foliar color.

Prognosis for Elms

In areas where both PN and DED are epidemic, attempts to preserve elms are probably bad investments. Susceptible elms have never yet been protected against simultaneous epidemics of PN and DED.

Antibiotic treatments, still in experimental phases of development, offer promise for future protection against PN or therapy of infected trees in areas where DED is absent or under control.

Among elms native to North America, prospects for a tree resistant to both PN and DED are poor.

Elms of Asian and European origin are apparently resistant to PN. Selection and breeding for DED resistance and desired horticultural characters within these species may therefore yield PN resistance as a bonus.

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ABSTRACTS

Andresen, J.W. and B.M. Williams. 1975. **Urban forestry education in North America.** Journal of Forestry 73(12):786-790.

Urban forestry and related courses and curricula are now offered in at least 29 universities in Canada and the United States. This new educational emphasis is an outgrowth of the increasing interest in environmentally directed forestry education, a trend prompted by outdoor recreation pressures in the mid-1960's. Expansion into the urban arena is a further manifestation of a shift from production-cenetered to amenity-oriented forestry education. It is reflective of a growing concern by the forestry profession to respond to the needs of an urbanizing North America. This review and inventory of urban forestry education grew from a joint endeavor of the Urban Forestry Working Group of the Society of American Foresters and the Urban Forestry Committee of the International Shade Tree Conference. Its major aim was to assemble a listing of urban forestry educational offerings at undergraduate and graduate levels. The listing serves to provide inquiring students with descriptions of urban forestry courses and curricula, and it can advise potential employers of a source of graduate foresters possessing urban forestry expertise.

Peterson, G.W. 1975. **Dothistroma needle blight: a problem in production of landscape pines.** American Nurseryman 141(12):11,94-96.

Nurseries producing pines for landscapes are being confronted with a needle disease caused by the fungus *Dothistroma pini.* Twenty pine species and hybrids, including two, three and five-needle pines, are known hosts in North America. The fungus has been found in 23 states in the U.S. Damage has been particularly severe in some midwestern nurseries that produce such pines as *Pinus nigra, Pinus ponderosa* and *Pinus mugo,* which are highly susceptible. The fungus causes a blighting and early drop of needles. Some nurseries have discontinued growing highly susceptible pines. However, research in Nebraska has shown that this disease can be controlled inexpensively. Pines heavily infected over the entire crown have been saved for sale by methods described in this article.