SEVERITY AND CAUSES OF ASH DIEBACK

by Craig R. Hibben and Savel B. Silverborg

Abstract. Ash dieback is a disease that causes progressive death of branches and tree mortality in white ash and to a lesser extent in green ash *(Fraxinus americana* L., *F. pennsylvanica* March.). It affects all-aged trees in woodland, hedgerow, streetside, and home sites. Above-average tree mortality occurred in several northeastern states in the late 1950's and early 1960's. Recent surveys in New York show that the disease generally has become static, except in the southeastern Hudson Valley region. Here, for unknown reasons, ash dieback continues to increase in severity. Water stress in the trees and invasion of the bark by canker fungi are the primary causal factors. Air pollution, leaf spotting fungi, viruses, and mycoplasma-like organisms are additional stresses which may be involved in the etiology of ash dieback. Control recommendations are based on the maintenance of high tree vigor.

Ash dieback is a disease of white ash and less often of green ash (*Fraxinus americana* L., *F. pennsylvanica* March.). Since the late 1950's it has been one of the more important tree problems in the Northeast. There has been progress in learning the etiology of ash dieback. The apparent complexity of several interacting climatic and biological causal factors, however, suggest that more research is needed before we fully understand this disease.

An unexplained branch dieback was first reported in white ash during 1925-1930 in southeastern Quebec (13) and in several northeastern states of the United States (12). Ash again were reported dying back along roadsides and in hedgerows in southeastern New York in the late 1930's and the 1940's. (Pers. comm. D.S. Welch, Cornell Univ.) During the 1950's, white ash, and some green ash, were showing abnormal dieback in valuable forest stands (17, 19). Since then, ash dieback has continued to be a problem in woodlands, hedgerows, home plantings and along roadsides throughout New York, New Jersey, Pennsylvania, and parts of New England.

Dieback is a serious problem because ash is widely distributed in the eastern states and is an important component of hardwood forests. The wood has considerable commercial value. Partly because of their rapid growth, white and green ash also have been prominent trees in the nursery trade.

The following report will define ash dieback, describe its severity and known causes, and suggest control measures.

What is Ash Dieback?

The earliest symptoms are reduced radial stem increment, shortened internodal length of twigs, reduced leaf size, and leaves pale green to chlorotic in hue. The foliage sometimes shows premature fall coloration in the form of purplish flecks. Diseased trees often shed their leaves sooner than healthy ones. Reddish brown or orange-yellow, slightly sunken cankers appear on smooth bark of the main stem and on all sizes of branches.

Figure 1 illustrates some of the dieback symptoms. Branch tips fail to leaf out in the spring and terminal twig dieback commences. The numerous small dead twigs and the clumped foliage on shortened internodes result in a sparse leaf canopy. The stem and branch cankers become cracked and the overlying dead bark sloughs off.

In succeeding years dieback progresses toward the main stem(s), until most side branches are dead. At this stage epicormic sprouts, and occasionally witches' brooms, form along the trunk and towards the axils of large limbs. Trees in this advanced stage of dieback rarely recover. Ash can be salvaged for merchantable timber if harvested while the main stem is still alive (15). Dead trees are rapidly invaded by insects and by fungi that stain and decay the wood. Roots excavated from ash in advanced stages of dieback have been reported to be well developed and comparable in appearance to roots of healthy trees (1).

Branch mortality occurs primarily during the dormant season, i.e., buds fail to produce new shoots in the spring. Wilted or dead foliage is not a common symptom of this disease. Dieback is gradual; tree mortality has occurred from two years after the onset of symptoms for young trees to about ten years for ash 16 inches dbh and

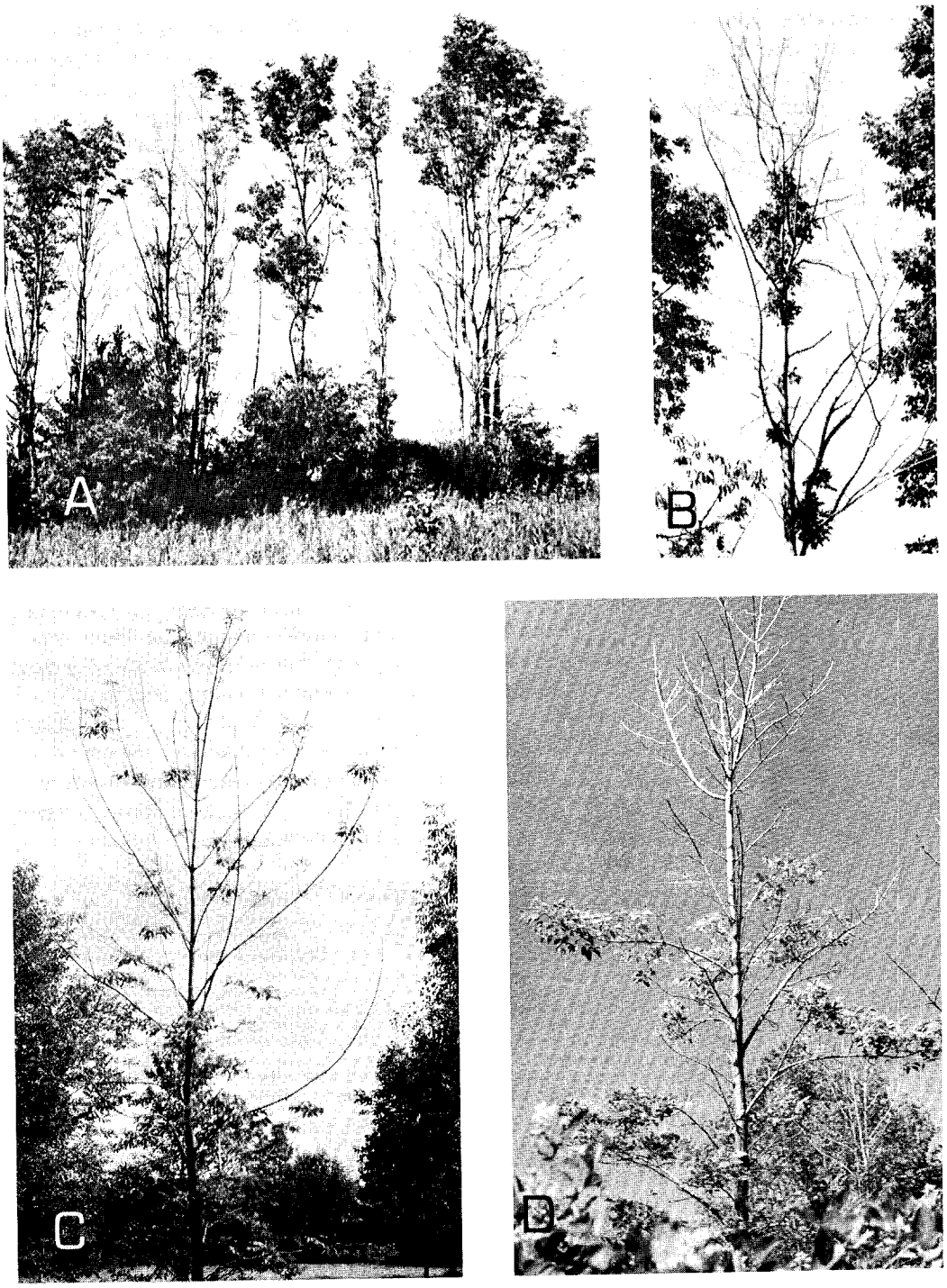

Figure 1. Symptoms of ash dieback. A) In hedgerow trees, B) Clumped leaves and epicormic sprouts, C) Premature defoliation and clumped leaves, D) Dieback in top portion of tree.

larger. Most reports (1, 19, 20) state that ash in natural sites rarely recover once afflicted with the disease. The senior author in 1975 noted ash in roadside and hedgerow sites in Westchester County, N.Y. that had fully recovered and had no dieback. Surveys in 1960 had rated these same trees in the early stages of dieback.

Tree resistance to ash dieback is clearly evident in stands where the disease is severe. Healthy ash are scattered among dead and dying trees.

Severity of Ash Dieback

A survey of ash in New York in 1960-61 revealed that 37% of the trees in the sample plots were in various stages of decline, and 6% were dead (18). Another survey in 1963 (20) showed that in the six-state region of New Hampshire, Vermont, Massachusetts, Connecticut, New Jersey, and Pennsylvania, 27.2% of sampled ash were either dead or dying, and of these 8.9% were dead. From 1965 through 1967 ash dieback remained static in New York (19). The one exception was in Dutchess County, where the disease increased in severity. From a later survey in New York in the early 1970's and from observations of ash throughout the Northeast in 1975-76 by the junior author, the same stabilized condition prevailed. Ash dieback, for unknown reasons, continues to increase in the Hudson Valley region, generally from Albany south to Westchester County.

Primary Causes of Ash Dieback

Research in ash dieback in New York has shown that water stress in the trees followed by invasion of the bark by canker fungi are the primary causal factors of this disease.

Drought. — Of all the etiologic agents investigated by Ross in his study of ash dieback in New York (15), periods of low rainfall constituted the only factor directly correlated with the initiation of the disease. Three extended periods of abnormally low rainfall during the growing season occurred in New York from 1950 to 1962. These droughts coincided with sudden decreases in both radial increment and shoot internodal length in ash. This association corroborates earlier observations of dieback in ash following droughts in Quebec (13) and in the Northeast (12). A recent study in New York (21) presented further evidence that the inception of ash dieback was correlated with local drought. The authors suggest that stomatal closure during periods of water stress in ash, with resulting decreases in carbon fixation, may be an additional mechanism linking drought to dieback.

Canker fungi. — The fungi *Fusicoccum sp.* and *Cytophoma pruinosa* (Fries) von Hoehnel were found to invade ash bark through wounds (14, 15). This infection causes annual cankers, which form during the dormant period between leaf fall in the autumn and leaf emergence in the spring. Canker enlargement is most rapid in early spring. The fungi are active in a canker usually only for one season. Numerous cankers on a branch or stem result in death of distal portions. These canker fungi, harmless to healthy ash, act as secondary invaders whereby they attack only those trees predisposed by moisture stress. Cankers develop usually at least one growing season after the initial evidence of growth reductions caused by drought conditions.

Ross (15) concluded, ". . . ash dieback is induced by periods of low rainfall with the fungi acting primarily as secondary invaders that accelerate death of the tree through severe stem and branch cankering."

Additional Stresses on Ash

Three observations raise the possibility that there are additional, as yet unknown, factors which should be included with the etiologic agents of ash dieback: a) From random observations of trees in the field, dieback and mortality can occur on ash with few or no branch and stem cankers, b) ash mortality continues in the Hudson Valley region of New York, where in recent years rainfall generally has been adequate for normal tree growth, and c) the pattern of diseased ash within a stand sometimes suggests an infectious agent because of the outward spreading of disease from individual infection centers. Although there is no explanation for these anomalies based on research, the following disease agents should be considered in our assessment of additional stresses detrimental to ash.

Air Pollution. — Ozone is produced by the photochemical reaction of sunlight on exhaust gases from motor vehicles and industrial sources. It probably causes more injury to vegetation than any other pollutant in this country. Ozone has damaged vegetation in the Northeast (7,8). Experimental fumigations have shown that white ash is one of the more sensitive deciduous trees to injury by ozone (22). It causes an upper surface purplish stippling on ash leaflets. Ozone injury may contribute to the premature fall coloration of ash foliage, which is part of the ash dieback syndrome. Ash, therefore, especially in or downwind of urban centers, is being subjected to another stress, air pollution.

Leaf Spots. — Ash is susceptible to several fungi that infect the leaves and cause necrotic lesions and scorch. Anthracnose, caused by *Gloeosporium aridum* Ell. and Holw., is especially injurious in wet springs. It causes scorching of large areas of the leaflets and premature leaf dehiscence. When environmental conditions are ideal for these foliar pathogens, they constitute an additional stress that significantly reduces the vigor of ash.

Viruses. — Viruses are infectious particles composed of a nucleic acid core within a protein coat. They multiply only within living cells. Virus particles become visible at extremely high magnification with an electron microscope. Relatively little is understood about how viruses affect forest and ornamental trees.

Two viruses have been isolated from white ash in the Northeast. A polyhedral virus was recovered from ash leaflets that had faint chlorotic spots and rings (2). These trees were located in a region of severe ash dieback in Dutchess County, New York. From the symptoms produced on a wide range of virus indicator plants, plus laboratory analyses of its physical, chemical, and serological properties, this isolate was identified (5) as tobacco ringspot virus, a known plant pathogen with a wide host range. When healthy ash seedlings were inoculated with the ringspot virus, infection sometimes resulted in significant reductions in leaf size, stem elongation, and root development (6). The dagger nematode, *Xiphinema americanum* Cobb, was shown to be a vector of the ash virus (4).

A rod-shaped virus was isolated from white ash in Massachusetts (9). Symptoms consisted of chlorotic rings, spots, line patterns, mosaic, and occasional reddening of the ash leaves. From similar laboratory procedures, it was identified as a strain of tobacco mosaic virus (10).

Two viruses are now known to infect white ash, but we know little about their capacity to incite or contribute to the dieback, or to predispose ash to other causal agents. We know nothing about how widespread these pathogens are over the natural range of ash. More research is required, and justified. Viruses injure trees, as we have learned from a long history of research on fruit trees. The wide herbaceous and woody host range of many viruses, the prevalence of virus vectors (insects, nematodes, pollen), and the ease of virus spread through vegetative propagation of infected plants, all increase the likelihood that viruses are infecting our forest and ornamental trees.

Mycoplasma-like organisms. — Witches' brooms have been found on the trunks of ash in advanced stages of dieback in New York (3) and Massachusetts (16). These abnormal growths consist of congested groups of twigs resulting from shortened internodes and forcing of shoots from axillary buds that normally remain dormant. The leaves are small, often simple rather than compound, and chlorotic. This syndrome is typical of the yellows-type diseases, formerly thought to be caused by plant viruses.

An infectious agent was transmitted from ash with witches' brooms to healthy ash by grafting (16), and to Madagascar periwinkle (*Vinca rosea* L.) by dodder (3). The latter disease agent was identified (3) as a mycoplasma-like organism (MLO), also called a Mollicute-like organism (11). MLO's are newly discovered (in 1967) plant pathogens, heretofore associated only with animal and human diseases. The MLO's are single celled yeast-like bodies without a cell wall. They contain ribosome-like bodies and strands of DNA, and they are somewhat smaller in size than bacteria. MLO's are found primarily in the phloem sieve tubes of plants, and they are spread from plant to plant by leafhopper insects.

The importance of MLO's in the etiology of ash

dieback also remains unknown until there is further research. The abundance of potential leafhopper vectors in the Northeast, and the wide host range of MLO's in herbaceous and woody plants, raise the possibility that these newly discovered plant pathogens are, or could become, widespread in ash.

Control Recommendations

Ash with extensive dieback rarely recovers under field conditions. However, no work has been reported on attempts to encourage recovery by supplemental tree care, such as fertilization and watering. Although not based on research with ash, the following steps to increase tree vigor are recommended, both as a preventative maintenance program, and to attempt the arrest of early dieback. This program would apply to especially valuable trees in home, streetside, and park sites.

a) Water during dry periods, especially in May, June and July.
b) Fertilize at recommended rates for trees.
c) Apply a fungicide during wet springs to control foliage pathogens and destroy fallen diseased leaves.
d) Apply insecticides, especially to control periodic infestations by leaf-chewing caterpillars.
e) Prune out dead branches and cover wounds with a fungicide-augmented dressing.
f) Prevent injuries to the bark and cover wounds with a fungicide-augmented dressing.
g) Prevent compacting of soil in the root zone; aerate the soil if compacted.
h) Avoid planting white ash in sites exposed to high concentrations of air pollutants.

Summary

1) Ash dieback is a disease that causes a progressive death of branches and mortality in white ash of all ages.
2) It was especially severe in the Northeast in the late 1950's and early 1960's, but since then ash dieback generally has stabilized. For unknown reasons, ash continue to die in southeastern New York.
3) The primary causal factors are drought followed by invasion of the bark by canker fungi.
4) Additional stresses which may be part of the etiology of ash dieback include air pollution, leaf-infecting fungi, viruses, and mycoplasma-like organisms.
5) Control recommendations are based on the maintenance of high tree vigor.

Literature Cited

1. Brandt, R.W. 1961. Ash dieback in the Northeast. U.S. Dept. Agr. Northeastern Forest Expt. Stat. Paper No. 163, 8 p.
2. Hibben, C.R. 1966. *Transmission of a ringspot-like virus from leaves of white ash.* Phytopathology 56:323-325.
3. Hibben, C.R. and B. Wolanski. 1971. *Dodder transmission of a mycoplasma from ash witches'-broom.* Phytopathology 61:151-156.
4. Hibben, C.R. and J.T. Walker. 1971. *Nematode transmission of the ash strain of tobacco ringspot virus.* Plant Dis. Reptr. 55:475-478.
5. Hibben, C.R. and R.F. Bozarth. 1972. *Identification of an ash strain of tobacco ringspot virus.* Phytopathology 62: 1023-1029.
6. Hibben, C.R. and S.S. Hagar. 1975. *Pathogenicity of an ash isolate of tobacco ringspot virus.* Plant Dis. Reptr. 59: 57-60.
7. Jacobsen, J.S. and W.A. Feder. 1974. A regional network for environmental monitoring: atmospheric oxidant concentrations and foliar injury to tobacco indicator plants in the eastern U.S. Univ. Mass. Expt. Sta. Bull. 604, 31 p.
8. Jacobsen, J.S. and W.A. Feder. 1975. *Photochemical oxidants in the New York-New Jersey metropolitan area.* Atmospheric Environment 9:321-332.
9. Lana, A.O. and G.N. Agrios. 1974. *Transmission of a mosaic disease of white ash to woody and herbaceous hosts.* Plant Dis. Reptr. 58:536-540.
10. Lana, A.O. and G.N. Agrios. 1974. *Properties of a strain of tobacco mosaic virus isolated from white ash trees.* Phytopathology 64:1490-1495.
11. Maramorosch, K. 1974. *Mycoplasmas and Rickettsiae in relation to plant diseases.* Annu. Rev. Microbiology 28:301-324.
12. Marshall, R.P. 1930. *A canker of ash.* Proc. Sixth Nat. Shade Tree Conf., p. 128-130.
13. Pomerleau, R. 1953. History of hardwood species dying in Quebec. Canada Dept. Agr. Rept. of Symposium on Birch Dieback, Part 1: 10-11.
14. Ross, E.W. 1964. *Cankers associated with ash dieback.* Phytopathology 54:272-275.
15. Ross, E.W. 1966. Ash dieback, etiological and developmental studies. State Univ. Coll. Forest. Syracuse, N.Y. Tech. Pub. 88. 80 p.
16. Schall, R.A. and G.N. Agrios. 1973. *Graft transmission of ash witches'-broom to ash.* Phytopathology 63: 206 (Abstr.)
17. Silverborg, S.B. and R.W. Brandt. 1957. *Association of Cytophoma pruinosa with dying ash.* Forest Sci.:75-78.
18. Silverborg, S.B., J.H. Risley, and E.W. Ross. 1963. *Ash dieback spreads.* The Conservationist 17(4): 28-29.

19. Silverborg, S.B. and E.W. Ross. 1968. *Ash dieback disease development in New York State.* Plant Dis. Reptr. 52:105-107.
20. Tegethoff, A.C. and R.W. Brandt. 1964. *Ash die-back in New Hampshire, Vermont, Massachusetts, Connecticut, New Jersey, and Pennsylvania, 1963.* Plant Dis. Reptr. 48:974-977.
21. Tobiessen, P. and S. Buchsbaum. 1976. *Ash dieback and drought.* Can J. Bot. 54:543-545.
22. Wilhour, R.G. 1970. The influence of ozone on white ash (*Fraxinus americana L.*). Ph.D. Thesis, Dept. Plant Pathology, Pennsylvania State Univ. 86 p.

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RESPONSE OF FOREST-GROWN TREES TO TOPPING[1]

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Abstract. Mortality of topped trees, four and five years after partial crown removal, was low. Several smaller trees died from exposure after the protection of overhead trees had been removed. Height growth of topped trees was rapid. After topping, dominant trees grew at an annual rate of 0.77 m (2.54 ft) and co-dominants at a rate of 0.68 m (2.22 ft). Height growth of smaller trees was much less. Unless topping removes a major portion of the crown, diameter growth is not seriously affected. Diameter growth of topped trees during the five years after topping was only slightly less than during the five years prior to topping.

Selective clearing of electric transmission line corridors has increased during the past decade. This practice softens the visual impact of corridor establishment and maintenance activities. Basically it entails removing only those trees or portions of trees which interfere now, or will interfere in the near future, with safe and uninterrupted electric current transmission, or with tower erection or inspection.

During selective clearing operations many trees are topped rather than completely removed. The amount of tree crown left after topping is determined primarily by distance from the electric transmission wires rather than by biological considerations of an individual tree's condition. Thus, some trees are cut back severely, even to the point of removing the major photosynthetic area of the crown.

Tree topping for transmission line construction is not comparable to the tree trimming commonly done in urban areas along distribution lines. Street shade trees are usually open-grown, and have large, deep crowns. During each periodic trimming of shade trees, a relatively small portion of the total crown area is removed. When transmission line corridors penetrate forest land, however, individual trees are closely spaced, and the crown area is restricted to a small percentage of their total height; usually 20 to 40 percent. Topping thus removes a significant percentage of the total photosynthetic surface. Topped trees respond in a number of ways: they may make a vigorous recovery through sprouting, they may remain in a static condition with little immediate change in crown area, or they may decline from the top and eventually die.

The objectives of this study were to determine (1) the growth response of selected tree species after topping, (2) what percentage of the live crown can be removed before decline occurs, and (3) whether such factors as relative crown position prior to topping, and total length of crown

[1]Presented at the annual meeting of the International Society of Arboriculture in Toronto, Ontario in August 1978.