Intensification of Ash Decline in New York State from 1962 Through 1980

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ABSTRACT

Incidence and severity of decline of white ash (Fraxinus americana) were monitored periodically in New York State from 1962 through 1980. From 1962 through 1970 in one set of plots, mortality increased linearly at about 6.3% per year. From 1968 through 1980 in a second set of plots established in 1968, mortality increased linearly at about 3.3% per year. The relationship between the incidence, severity, and rate of ash decline development and spring drought conditions as represented by the Palmer Drought Index was not distinct.

White ash (Fraxinus americana L.), a species of significant commercial value, is widely distributed in the eastern United States. It is an important component of the hardwood forests and hedgerows of the Northeast (3). Ash decline (ash dieback) is a progressive disease characterized by reduced radial and apical growth, stem and branch cankers, epicormic sprouts, witches' brooms, sparse and chlorotic foliage often tufted at the ends of branches and at the nodes, and a gradual dieback of branches. Death usually occurs within 2–10 yr of symptom onset (6).

Although the date when ash decline first appeared is unknown, white ash were reported dying from unknown causes in the early 1900s (9,13). Since then, decline has been observed frequently on white ash and occasionally on green ash (F. pennsylvanica var. lanceolata (Borkh.) Sarg.) in forest and hedgerow sites. Silverborg and Brandt (18) reported extensive dying of white ash in New York State between 1942 and 1957. Considerable research was conducted on ash decline in the 1950s and early 1960s (12,13,15,16,18,19). The disease became important when white ash on home grounds, in hedgerows, along roadways, and in valuable forest stands in New York State were affected (6). A number of abiotic and biotic factors have been associated with decline of white ash. These include drought stress (6,16), low temperature as a cause of root injury (12), sensitivity to injury by ozone (22), canker fungi (15), mycoplasmalike organisms (7,10,17), and viruses (2,4,5,8). To date, no single causal agent has been demonstrated to cause the decline syndrome.

The objectives of this report are to document quantitatively the increase of ash decline in New York State over an 18-yr period and to reevaluate the role of drought as a causal factor.

MATERIALS AND METHODS
Four observation plots established in 1962 in Onondaga, Dutchess, Monroe, and Fulton counties (Fig. 1) have been described previously (19). Plots were selected to represent forest and hedgerow stands of white ash. Ten to 15 canopy trees were monitored in each plot at 2-yr intervals from 1962 through 1974. The mean diameter at breast height (dbh) of trees in these plots was 3.0 ± 1.1 in. Trees selected in these plots were primarily saplings. Each tree was rated for decline severity according to the system of Silverborg and Ross (19): class 1 = apparently healthy trees, class 2 = trees

Plant Disease/March 1985 243
with sparse and chlorotic foliage and a few dead branches, class 3 = trees with foliar symptoms but with less than one-half of the foliar crown dead, class 4 = trees with foliar symptoms and more than one-half of the foliar crown dead (classes 2, 3, and 4 = diseased trees), and class 5 = dead trees. In 1968, nine additional plots were established in Clinton, Essex, Columbia, Ulster, Onondaga, Cayuga, Steuben, Erie, and Cattaraugus counties (Fig. 1). Plots were selected to represent forest and hedgerow stands of white ash. Ten to 15 canopy trees were monitored periodically in each plot from 1968 through 1980 and rated for decline severity as mentioned before. The mean diameter of trees in these plots was 6.4 ± 2.2 in. dbh. A total of 175 trees in 13 plots throughout New York State were monitored periodically from 1962 through 1980. Calculations of rates of disease increase were attempted using a linear model and Vanderplank's simple- and compound-interest models (21). Regression analysis (least sums of squares method) using the NLIN procedure of SAS (SAS Institute Inc., Cary, NC) was used to test the fit of the models.

An attempt was made to determine if the incidence and severity of white ash decline could be correlated with drought. The 13 observation plots were located within six climatic zones in New York State. Monthly values of the Palmer Drought Index (PDI) (11) for a central location within each zone from 1931 through 1980 were obtained from the National Meteorological Center, Washington, DC. The PDI is used to evaluate the scope, severity, and frequency of prolonged periods of abnormally wet or dry weather and is an index of the relative wetness or dryness of soils. Calculation of the PDI is based on current precipitation and temperature, climatic zone constants, previous PDI values, and soil conditions. PDI values of 0.49 to −0.49 = normal moisture conditions, −0.5 to −0.99 = incipient drought, −1.0 to −1.99 = mild drought, −2.0 to −2.99 = moderate drought, −3.0 to −3.99 = severe drought, and ≤−4.0 = extreme drought. Conversely, values of 0.5 to 0.99 = incipient wet spell, 1.0 to 1.99 = mild wet spell, 2.0 to 2.99 = moderate wet spell, 3.0 to 3.99 = severe wet spell, and ≥4.0 = extreme wet spell (11).

Two procedures, using the PDI, were used to identify periods of drought. First, the means and standard deviations of PDI values for April through June for all six climatic zones for each year (1931–1980) were determined. Since white ash is ring porous (1) and has a determinate growth habit, drought that occurred in the spring (April, May, and June) was considered most likely to contribute significantly to suppressed annual growth and the decline syndrome. On the basis of this reasoning, we have illustrated spring drought (Fig. 2) for comparison with disease progress in the observation plots and ash decline development as described in the literature. Second, the number of months per year with PDI values ≤−0.5 (incipient drought) for each climatic zone, as a general indication of annual drought, was tabulated for all years from 1931 to 1980.

RESULTS

In 1962, about 37% of the 55 trees examined in plots A–D were healthy, but this decreased to about 8% in 1968 through 1974 (Fig. 3). The decrease in percentage of healthy trees coincided with an increase in the percentage of dead trees during this time (from 0 to about 48%) (Fig. 3). In 1968, about 33% of the 120 trees examined in plots 1–9 were healthy, but this decreased to 0% in 1980. Similarly, this decrease coincided with an increase in the percentage of dead trees observed during the period (from zero to about 38%) (Fig. 4).

Regression analysis of changes in condition class status vs. time based on linear and simple- and compound-interest models for healthy (class 1) or declining (classes 2–4) trees were not acceptable (probability of F > 0.05). This could be attributed in part to fluctuating moisture conditions but is probably more closely related to the subjectivity of assigning trees to specific condition classes. From 1962 to 1970 in the first set of years, 13 plots were distributed through five different climatic zones. Two procedures, using the PDI, were used to identify periods of drought. First, the means and standard deviations of PDI values for April through June for all six climatic zones for each year (1931–1980) were determined. Since white ash is ring porous (1) and has a determinate growth habit, drought that occurred in the spring (April, May, and June) was considered most likely to contribute significantly to suppressed annual growth and the decline syndrome. On the basis of this reasoning, we have illustrated spring drought (Fig. 2) for comparison with disease progress in the observation plots and ash decline development as described in the literature. Second, the number of months per year with PDI values ≤−0.5 (incipient drought) for each climatic zone, as a general indication of annual drought, was tabulated for all years from 1931 to 1980.

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of plots (Fig. 3) and from 1968 to 1980 in the second set of plots (Fig. 4), however, mortality increased linearly. The linear model was the simplest model tested and provided the best fit with probabilities of $F = 0.0052$ and $0.0003$ and $r = 0.973$ and 0.996 for 1962–1970 and 1968–1980, respectively. The slope of the regression line calculated for 1962–1970 was about twice that of the line calculated for 1968–1980 (6.3 vs. 3.3%, respectively). Therefore, mortality progressed at an annual rate of 6.3 vs. 3.3% during these two periods, respectively. In the first set of plots, an asymptote occurred at about 50% (Fig. 3), whereas in the second set of plots, mortality was still increasing in a linear fashion in 1980 (Fig. 4).

**DISCUSSION**

Ash decline has been widely attributed to drought, which predisposes trees to attack by canker fungi that in turn accelerate the death of the tree (6,16,19). The early 1930s, 1941–1942, 1949, 1955, and the early to mid-1960s were characterized by spring drought conditions (Fig. 2). In addition, calculation of the number of drought months per year identified the early to mid-1930s and the 1960s as unusually dry years. Conversely, the 1970s were unusually wet (Fig. 2). Although drought years occurred locally and sporadically throughout the 50-yr period, no other drought period resembles the extended drought conditions of the 1960s. If the 1960s are characterized as drought years, then the period 1942–1957, when white ash were dying in increasing numbers (18), is better characterized as a fluctuating period with generally normal to above-normal moisture conditions (Fig. 2). It is therefore difficult to reason that extensive dying of white ash during this period (1942–1957) was due primarily to drought stress.

In surveys conducted in eastern New York State (20) in the early 1960s, the disease intensified slowly or not at all, even though drought was very severe at that time (Fig. 2). From 1962 through 1970, mortality in our observation plots increased steadily at an average of 6.3% per year. Spring drought conditions did prevail from 1962 to 1966; however, an above-normal spring water supply prevailed from 1967 to 1980 (Fig. 2). A second set of plots established in 1968 and monitored periodically through 1980 likewise shows a linear increase in mortality of 3.3% per year—half the rate of the earlier period.

If drought is the principal cause of ash decline, a leveling off of the mortality rate would be expected when drought was alleviated, as predicted by Silverborg and Ross (19). Nevertheless, mortality continued to increase in the plots established in 1968 (Fig. 4) even though available moisture was normal to above normal in most parts of the state from 1967 to 1980 (Fig. 2). The percentage of healthy trees (class 1) in the earlier plots did remain constant after the drought ended in 1967 (Fig. 3), but this represents only five healthy trees out of an original population of 55 trees in these four plots. The percentage of healthy trees (class 1) in the nine plots established in 1968 decreased to zero in 1980 (Fig. 4), indicating that ash trees continue to deteriorate even during periods of above-normal moisture (Fig. 2).

Mortality increased linearly in both sets of plots. Biennial changes in mortality were not affected by changing moisture conditions. Since there was little deviation from the mortality line during fluctuating moisture conditions, we interpret this as no direct relationship between moisture and mortality. Because mortality continued at a linear rate, even in the presence of above-normal moisture during the 1970s, it is difficult to envision a major effect of moisture on disease progress.

The evidence for the role of drought in the decline of white ash is contradictory. There is evidence that drought is associated with mortality of white ash. First, the decrease in the number of healthy and declining trees did level off when the drought ended in 1967 (Fig. 3). Second, the mortality rate for the period 1962–1970 was twice that of the period 1968–1980; this coincides with the overall moisture levels for the two periods (Fig. 2). Third, linear disease increase is not common and therefore may be indicative of abiotic problems in general. In addition, white ash is a ring-porous species (1) and the wood has an unusually low moisture content (14). These characteristics make white ash particularly vulnerable to drought-induced stress.

There also is evidence that drought may not be associated with mortality. First, ash decline was prevalent in New York State between 1942 and 1957 (18), prior to the drought of the early 1960s, yet this was a period with generally normal to above-normal moisture conditions (Fig. 2). Second, mortality in the first set of plots continued to increase until 1970, 3 yr after the drought ended (Fig. 3). In addition, in the second set of plots (Fig. 4), mortality increased linearly from 1968 through 1980 and the number of healthy trees decreased to zero, even though the 1970s were unusually wet in New York State (Fig. 2). Third, the rather steady linear increase in mortality from 1962 through 1980, during a period of fluctuating moisture, indicates that drought may not be an important factor contributing to mortality. Fourth, another line of evidence against regional drought as a major factor in decline comes from Matteoni’s (10) analyses of radial growth rates. The year of onset of decline varied among trees at one site and among sites. Therefore, a common causal abiotic factor, such as drought, or rapid infection by a common pathogen, is unlikely.

We interpret linear disease increase and the apparent lack of a distinct relationship between mortality and moisture as an indication of the need to further evaluate the role of drought, and other factors acting alone or in concert, in producing ash decline syndrome. The possibility of multiple causal factors should not be ignored. Factors that should be investigated include the physiology of white ash, environmental factors other than drought, and possible pathogens. Since linear and simple-interest disease increase is independent of the number of affected individuals, secondary spread of disease among individuals in the stand does not occur (21). Consequently, if pathogens are involved in the decline of white ash, they must be introduced into ash trees from other sources with very slow or no tree-to-tree spread. Abiotic diseases, such as those induced by drought, also increase linearly. On the basis of our results, however, the role of drought in ash decline is uncertain.

**LITERATURE CITED**