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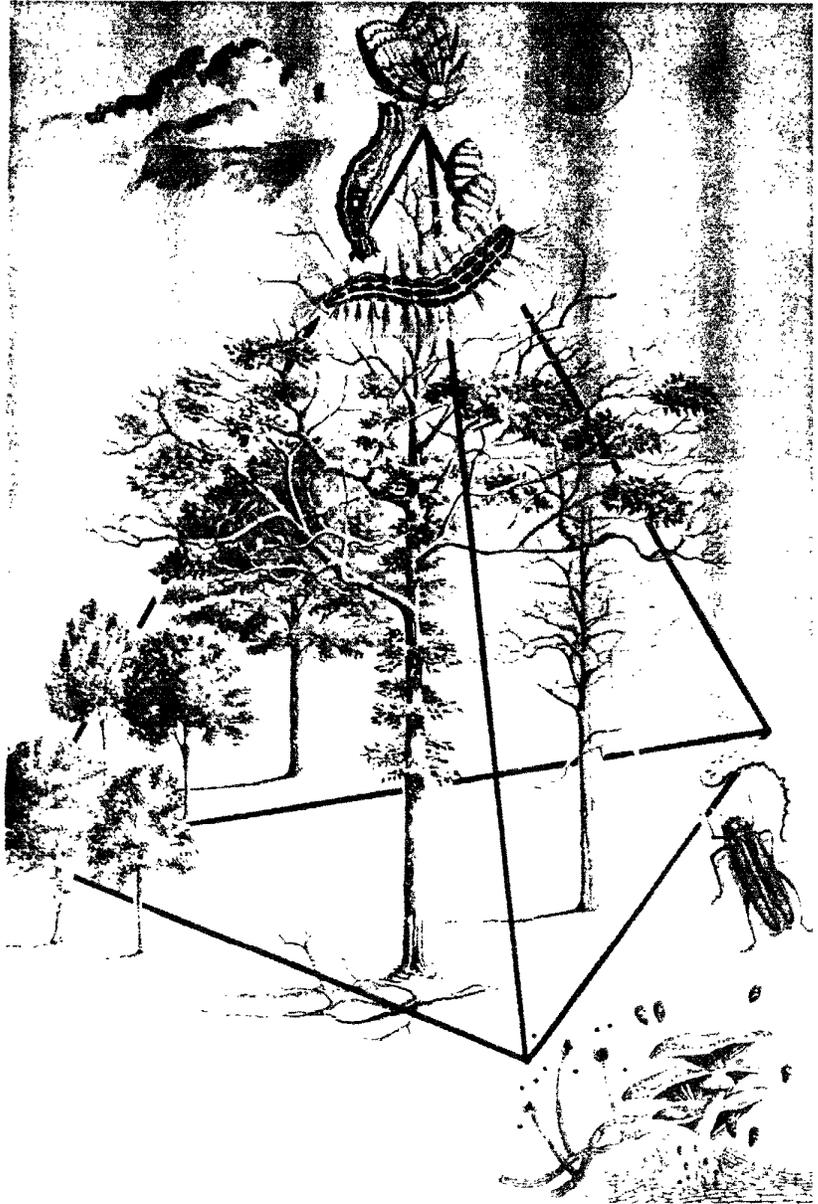


United States
Environmental
Protection Agency

and Cooperators

History of Hardwood Decline in the Eastern United States

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Abstract

Tree mortality events that have occurred in the eastern hardwood forest during the last century were examined to determine whether there is a relationship between patterns of mortality over time and current patterns of atmospheric pollution. A review of the literature indicates that the apparent increase in the decline and mortality of many hardwood species during the last few decades may be due to intensification of reporting and to the maturation of the forest itself. Most of the mortality is attributed to abiotic and biotic stress factors such as weather, silviculture, and damage by insects and diseases. There is evidence of damage to hardwoods by atmospheric pollutants from point sources such as smelters, and to eastern white pine by ozone. There is no conclusive evidence of an association between patterns of hardwood mortality and regional atmospheric pollution. An extensive list of references covering the major recorded mortality events in the eastern hardwood forest is presented. Also included is a discussion of the distribution of the major eastern hardwoods, changes in the forest that have occurred since the European settlement, and possible effects of these changes on the eastern hardwood forest.

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INTRODUCTION

The Problem

Forest decline results from a reduction in growth and vigor and/or widespread mortality of trees over large geographic regions. During the past decade, there have been numerous reports of unexplained growth decline and mortality of hardwood trees in the Eastern United States (Bernabo 1985). Recent reports have suggested that pollution might play a role in the decline of the eastern hardwood forest through the deposition of chemicals from the atmosphere to natural surfaces. Atmospheric deposition can be wet or dry, acidic or nonacidic.

Because of large uncertainties associated with historical events in unmanaged, remote forested areas, there has been no assessment of a possible spatial relationship between atmospheric deposition and patterns of forest decline. In this report we evaluate the evidence for such a relationship in the Eastern United States (Shriner 1986) by reviewing the available literature.

Authority and Justification

An Interagency Task Force on Acid Precipitation was formed by the Acid Precipitation Act of 1980 (Title VII of the Energy Security Act of 1980, P.L. 96.294). This resulted in the National Acid Precipitation Assessment Program, the objective of which is to develop and refine the scientific understanding of the causes and effects of the complex problem of atmospheric pollution. Several task groups were created, including a Terrestrial Effects Task Group administered by U.S. Department of Agriculture (NAPAP 1985). Within this Task Group, a Forest Response Program (FRP) was created to resolve longstanding questions about the role of atmospheric deposition and associated air pollutants in the decline of trees. The Eastern Hardwood Research Cooperative of the FRP is focused on answering the following policy question: Is there a significant problem of forest damage in North America which might be caused by acid rain, its precursors, and associated pollutants, e.g., SO_x, NO_x, H⁺, metals, O₂, H₂O, or hydrocarbons?

In addressing the policy question, two scientific questions were formulated (Shriner 1986):

1. Are changes in forest condition greater than can be attributed to typical trends and levels of natural variability?
2. What spatial patterns, if any, exist in the forest condition, and how do these patterns relate to spatial patterns of pollutant exposure?

Objectives

To answer the scientific questions, the Eastern Hardwood Research Cooperative sought to provide the following (USDA For. Serv. 1987):

1. A set of case studies of declines of major eastern hardwood forest species. By species, document the distribution of the species and its importance across that geographic distribution. The case history also documents climate and deposition patterns across the species distributions for the relevant components of acid rain, its precursors, and associated pollutants.

2. For each species, document the location, temporal history, severity, and causes of known declines. The measure of forest decline was defined as a mortality event covering an area larger than 1,000 acres, with tree mortality *exceeding that which is expected* during normal forest growth. The 1,000-acre limit was used because reliable records of mortality on smaller areas frequently are lacking, especially in the older literature.

This report attempts to determine whether present location, extent, duration, severity, and frequency of mortality events of the eastern hardwood forests differ significantly from those of the past; and if differences exist, to determine possible reasons for those differences, including spread of introduced insects and diseases (e.g., gypsy moth, beech bark disease), maturation of the eastern hardwood forest with associated changes in susceptibility to damage agents, and change in atmospheric deposition level.

Methods

The literature of forest declines was reviewed to determine: location; time period; duration; extent of the area; severity of damage (such as number of trees or volume killed); stated causes of decline; and contributing factors. The initial search covered major books and reviews on forest declines to find references to mortality events. Biological Abstracts and Forestry Abstracts were searched, and unpublished literature including Federal, state, and private reports were reviewed. A complete bibliography including references not cited in this report is available from the Forest Pest Management, USDA Forest Service, Northeastern Area, P.O. Box 640, Durham, NH 03824.

Limitations

Hardwoods

The literature search included the following tree species: American beech, black cherry, eastern white pine, northern red oak, shagbark hickory, sugar maple, white ash, white oak, yellow birch, and yellow-poplar. Eastern white pine was included because it is a component of hardwood forest in the Eastern United States. It has been considered one of the more sensitive conifer species to atmospheric pollutants, and is distributed widely in eastern forests. Several species were added during the literature review. Black and scarlet oak were added because many of the reports of decline included these species. Likewise, paper birch was added because in reports of birch decline this species frequently was combined with yellow birch. Red maple usually is included in reports of maple decline. Aspen

and cottonwood were added because they are two of the more important species in the North; and sweetgum was added because of its importance in the South. American chestnut and American elm are discussed briefly because of the major impact of introduced pathogens on the mortality and distribution of these two species over the past century.

Forest Decline

The primary emphasis was to document declines resulting in the death of trees. The search was limited to mortality events covering an area in excess of 1,000 acres, though the report documents significant mortality events on smaller acreages. All causal agents except man and fire are included.

Mortality

Whenever authors reported abnormal frequency of dead trees, these were considered as mortality events. Most of the reports of mortality events include poorly quantified data on the number or volume of trees lost. The most precise available estimates were used in documenting losses.

Location

Many authors have used general terms such as "oak mortality in the southern Appalachians" or "central New England." Attempts were made to interpret and map these locations. As a result, the maps in this report may give the appearance that an entire region incurred uniform tree mortality. The correct interpretation is that many stands within mapped areas contained a greater than normal amount of dying and dead trees. Also, an area with no mortality may reflect lack of reporting rather than a lack of mortality. For example, elm mortality from Dutch elm disease is common in New England, but it is rarely reported. By contrast, a great interest by an investigator in a specific mortality event may have generated many reports based on only several acres of dead trees.

Many mortality events, particularly those cited in older reports on minor hardwoods, were not investigated thoroughly. Causality may have been attributed to weather or other factors that could not be easily confirmed. In this report, the causal agents cited by the author are taken at face value.

THE EARLY FOREST

Before the European settlers arrived on the North American continent, the forests were managed by natural events and the American Indians. Great changes occurred during the settlement period as the Europeans became established on the Atlantic Coast and later moved west. The forest before 1900 can be best described in three phases: presettlement,

destruction during settlement, and regrowth.

Presettlement Forest

The history of the eastern hardwood forest should begin with the forest that was here when the first European settlers arrived. The only human factor at that time was the American Indian, who lived in the forest and survived on what it provided. The forest suffered minimal disturbance from the Indian (Devens 1983). Not all the tribes were nomadic. Some were sedentary horticulturists, such as the Huron and the Iroquois, who opened small areas in the forest. The extent to which the Indians manipulated the forest is not clear. It is possible that they altered the composition of the forest through food-gathering activities. Perhaps the high abundance of nut-bearing trees at the time of the European settlement was the result of these activities (Devens 1983). Some evidence suggests that the Indians used fire frequently where it was physically and ecologically feasible (Cronon 1983; Devens 1983). Cleland (1983) pointed out that the Indian learned to live within the forest ecosystem. Minor environmental changes were made, but these probably were negligible compared with those caused by weather, fire, insects, and diseases. At worst, the Indians directly influenced only a small proportion of the total forest (Davis 1983).

The eastern half of the United States was predominantly forested before the arrival of the first Europeans (Davis 1983). Within this region, forests generally covered more than 80 percent of the land. The exception was Illinois, of which only about 40 percent was forested (Kohlmeyer 1983). According to Davis (1983), the cooler and more moist regions of the Northeast had an abundance of conifers interspersed with hardwoods. Conifers also dominated many of the southern and eastern sandy areas where fire was frequent. The western boundary was limited by prairie where grass fires prevented the growth of trees. Most of the central region was pure hardwood forest. Clawson (1983) estimated that the average volume of wood per acre prior to the European settlement was twice that of the present volume. Except for the areas recovering from recent natural disasters and insect and disease epidemics, most of the forests were near maturity with near peak volumes of timber. However, Loucks (1983) presented evidence that the forests were continuously changing due to weather factors. These changes occurred in patches, sometimes of several hundred square miles, as a result of windstorms and other natural disasters. Loucks concluded that the forest, at least in the Lake States before the arrival of European settlers, was a landscape of patches at different ages and different stages of succession rather than a uniform climax forest.

Destruction During Settlement

The large-scale destruction of the old forest began with the arrival of the European settlers. Davis (1983) provided a compilation of forest histories by various authors for each state. A typical, detailed description for Connecticut was provided by Krug and Frink (1983), who described how the forest was first cleared for agriculture and construction

timber, or burned to protect the settlers from the enemies in the forest. Later, as the cities began to grow, vast areas of pine and spruce were logged to meet the increasing demand for construction timber. Fires, which often accompanied logging, became even more common with the advent of the wood-burning locomotive. As a result, much of the previously forested area was logged or burned. By the late 18th century, many of the eastern forests were less than half forested. Many conifer stands were converted to hardwoods as conifer seeds were destroyed by fire, and the hardwoods regenerated from stump sprouts. In many areas, only a few remnant spruce and white pine remained.

During the early logging, wasteful practices were the rule. The lumbermen set high standards for marketable lumber so the less than perfect stems were destroyed (Cronon 1983). Demands for forest products were not restricted to lumber. The forest also provided wood for fuel, potash, barrels, charcoal, naval stores, mine props, tannin, shingles, furniture, and pulp. The prevailing attitude was that the timber resource was inexhaustible.

Regrowth of the Forest

In many areas, erosion and poor agricultural practices caused a decline in crop production. With the discovery of the rich lands in the Midwest, eastern farms were neglected and abandoned. These lands were slowly invaded by pioneer tree species and then through successional stages the forest that we see today was established. The impact of long-term land use changes in eastern forests is not known. The following are some of the changes associated with land use that may contribute to a less vigorous forest.

- Pioneer species invading old farm lands are shorter lived than climax species, and usually are more susceptible to pests.
- Soils depleted by agriculture may require several generations of forest cover to restore nutrient status and water-holding capacity.
- Highgrading, past and present, removes the stronger trees, leaving those that are more susceptible to damage.
- Selective logging may increase sprout-origin trees, which are more prone to stem diseases than seed-origin trees.
- Introduced pests have eliminated certain well-adapted tree species in favor of less well-adapted species.
- Although controversial, it has been suggested that farming and logging practices have increased the alkalinity of soils, which are now undergoing natural acidification. These natural changes also may bring about changes in forest composition (Krug and Frink 1983).

Smith (1976) summarized the changes that have occurred in eastern hardwood forests since the European settlement:

Most of eastern North America was covered with forests when the European settlement started

almost 4 centuries ago. The most important thing that has happened since is that virtually all of this forest has been cut over at least once, or if not cut, as in some remote parts of Canada, heavily burned at least once. The result is that the average age of present forests is younger, by an indeterminable amount, than that of the so called "original" forest. Presumably we have, in general, reduced the biotic pests of the old forests and increased those associated with young forests.

However, human influences have changed forests and with them the interaction with pests. During the first three centuries after 1600, most of what man did to the forests had the dismal effects of increasing difficulties with pests. It is even more clear that because the forests all grow older uniformly, the problems and solutions in the forest management of a locality change from decade to decade. In most places, the forests of 1975 have changed since those of 1955 and, by the time we learn to accommodate to the damage, it will be 1995 and more changes will have taken place.

EASTERN HARDWOOD FORESTS TODAY

Eastern hardwood forests cover approximately 240 million acres (Fig. 1) and can be divided into six major forest types (Table 1). Within these broad forest types there are

Table 1.—Area of commercial hardwood timberland in the Eastern United States, by forest type^a

Forest type	Acres	Percent of total
	<i>(Millions)</i>	
Oak—hickory	108.9	26.8
Maple—beech—birch	36.2	9.0
Oak—pine	34.6	8.5
Oak—gum—cypress	26.7	6.6
Elm—ash—cottonwood	22.3	5.5
Aspen—birch	19.2	4.7
Eastern hardwoods	247.9	61.0
Other forests	158.2	39.0
Total	406.1	100.0

^a Adapted from USDA Forest Service 1982a.

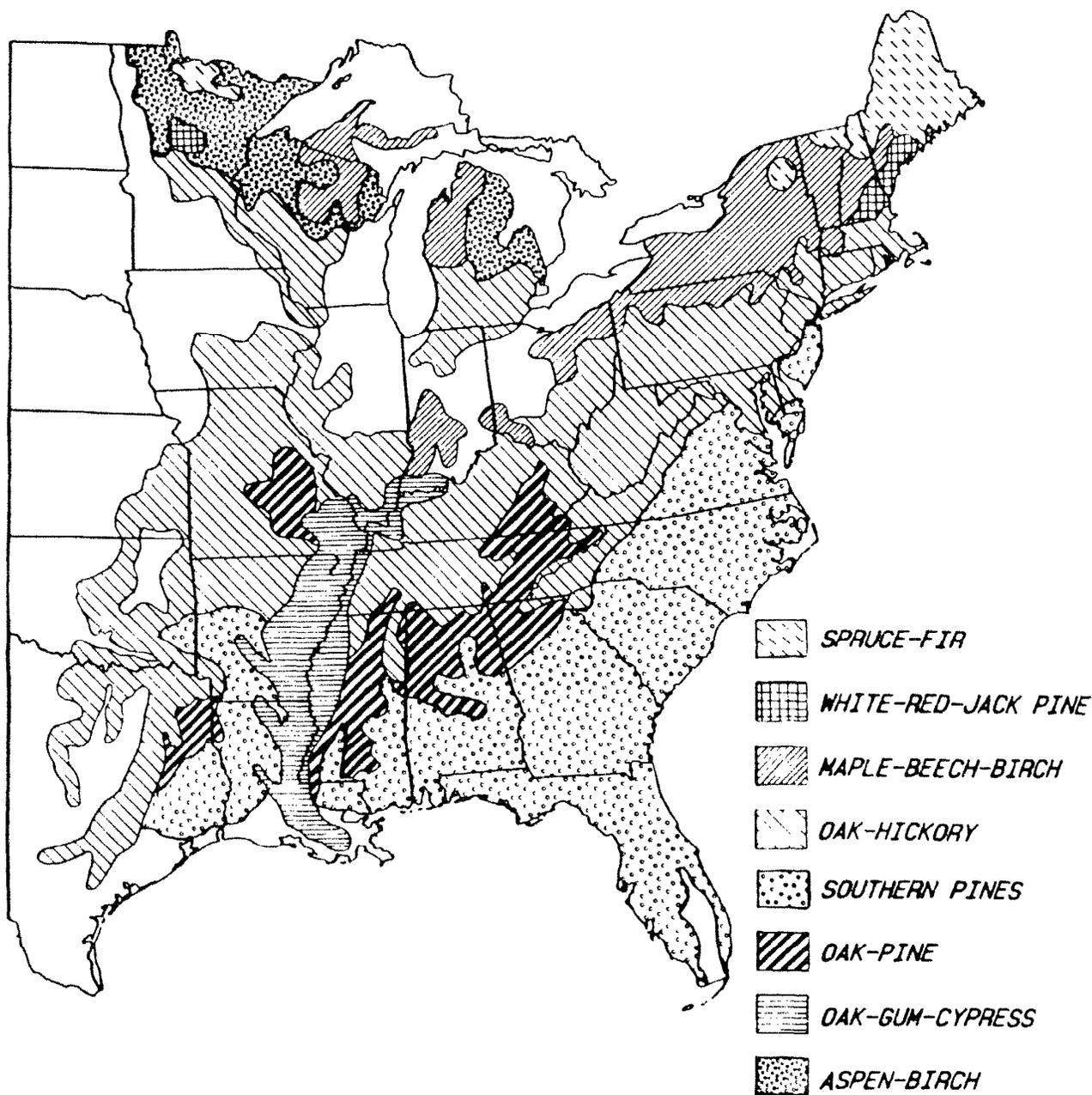


Figure 1.—Major forest cover types in the Eastern United States (from Eyre 1980).

numerous local species associations or cover types (Eyre 1980; Burns 1983). When reference is made to associations with other species or forest cover types, the numbers in parentheses refer to the Society of American Foresters forest cover types (Eyre 1980). For detailed discussions on the silviculture of the species, see Eyre (1980), Fowells (1965), and Harlow and Harrar (1969). Wood characteristics and uses are summarized from Panshin and De Zeeuw (1980).

Forest inventory surveys are made about every 10 years in most of the states. These surveys are conducted cooperatively between the USDA Forest Service and state

agencies. Reports are published by Forest Service Experiment Stations and frequently are summarized in national reports (USDA For. Serv. 1982a, b). For example, the most recent survey reports published in 1985 for New Hampshire (Frieswyk and Malley 1985a) and Vermont (Frieswyk and Malley 1985b) show forest inventory changes and provide data on forest health by species. On the basis of similar reports, Gansner et al. (1987) concluded that inventories of sugar maple in the Northeast have increased in the last decade and that there are more trees, and of higher quality, than before. Similarly, Hornbeck (1987) concluded from tree-ring studies that the hardwood species in the Northeast have not shown a steady decline in the last

few decades, but do show short periods of poor growth in response to below normal precipitation and insect defoliation.

The state of the forest condition in relation to reported declines for the United States was reported by Weiss and Rizzo (1987). Similar reviews are available for Canada (Auclair 1987; Linzon 1987; McIlveen et al. 1986; Rennie 1987). In their review of man's influence on Lake States forests, Ahlgren and Ahlgren (1983) concluded that major changes have been brought about by fire protection in that a lack of fires may be allowing successional development in the forest that had not occurred before. Stephens and Hill (1971) showed that successional changes occurring in the forests of Connecticut have led to a decrease of oak and increase of sugar maple and conifers. Thus, one can conclude that there is evidence that forests are changing in species composition, and that the decline of a tree species in one area cannot be used independently as evidence of an abnormal forest condition.

Oak and Declines

Several species of oak frequently are found in the same stands. Adverse weather stresses are likely to affect all species in a stand, although not to the same extent. Insects and diseases tend to attack several species within the major groups, such as red oaks or white oaks. Many of the reports of decline identify only the major oak components, or identify only the major oak groups. Here, we discuss briefly the major oak species; the declines and their causes are discussed for oaks as a whole.

Species, Range, and Use

White oak

White oak is found throughout the Eastern United States except in northern Maine. It also is found in southern Quebec and Ontario. It extends south to northern Florida and west into eastern Texas, extreme eastern Oklahoma and Kansas, Iowa, and southeastern Minnesota (Fig. 2). White oak is rarely found in the coniferous types of the high Appalachians, the Delta region of the lower Mississippi, and the coastal regions of Texas and Louisiana.

White oak is a major species in three forest cover types: white oak—red oak—hickory (52); white oak (53); and yellow-poplar—white oak—northern red oak (59). Some of the major associates of white oak are other upland oaks, hickories, yellow-poplar, American beech, sugar maple, shortleaf pine, loblolly pine, eastern white pine, and eastern hemlock. Hickories and oaks are the most frequent associates.

White oak is used for many purposes including railroad ties, fenceposts, barrel staves, and mine timbers. Its veneer is used widely for furniture and interior paneling. It is the principal species used for flooring because of its hardness and resistance to abrasion. Other uses are for railroad cars, ships and boats, caskets, and pulpwood. Because of its

high fuel value, white oak is an important species for firewood.

Chestnut oak

The distribution of chestnut oak is limited compared to that of other eastern oaks. It is found in the Appalachian region, Ohio River Valley, southeastern Michigan, and southern Ontario (Fig. 2). Chestnut oak is a major species of the chestnut oak type (51). Its common associates include eastern white and pitch pines, sweetgum, red maple, white, scarlet, and black oaks, and eastern redcedar. The uses are similar to the red and white oaks, but preferred where durability is required.

Northern red oak

Northern red oak is commonly found east of the Mississippi River except in Florida, southern Georgia, southern Alabama, and the eastern part of the Carolinas (Fig. 2). In the North, it is found in southern Ontario and Quebec, New Brunswick, Nova Scotia, and Cape Breton Island. In the West, it grows in most of Minnesota, all of Iowa except in the northwestern part of the state, eastern Nebraska, eastern Kansas, and eastern Oklahoma. Northern red oak also is found in scattered areas in southern Mississippi and Louisiana.

Red oak is associated with five major forest cover types: northern red oak (55); white pine—northern red oak—white ash (20); northern red oak—basswood—white ash (54); northern red oak—mockernut hickory—sweetgum (56); and yellow-poplar—white oak—northern red oak (59). Associated species of red oak vary with locality and site. Yellow-poplar, sugar maple, black cherry, white oak, white ash, American beech, and buckeye are among those found with red oak on moist sites in the eastern part of its range. In the western extremities, black walnut occasionally is found with oak. Associates on drier sites are upland oaks, hickories, blackgum, and red maple.

Northern red oak is used for similar purposes as white oak—railroad ties, fenceposts, veneer (furniture and interior paneling), flooring, kitchen cabinets, caskets, and pulpwood. Red oak is an important species for firewood because of its high fuel value.

Black oak

Black oak is found in nearly all upland hardwood forest types and in some pine types east of the Great Plains. Its range is from southern Maine to Iowa, south to eastern Texas and northern Florida (Fig. 2). Black oak is a major species of the post oak—black oak type (40) and the white oak—red oak—hickory type (52). It is found in pure stands and also with hickories and scarlet, southern red, post, blackjack, and chestnut oaks. It is found less frequently with white and northern red oaks. In the South, black oak grows with shortleaf pine and occasionally with loblolly pine, southern red oak, and sweetgum, where it occupies the highest and best drained areas. Many of its uses are similar to those of red oak.

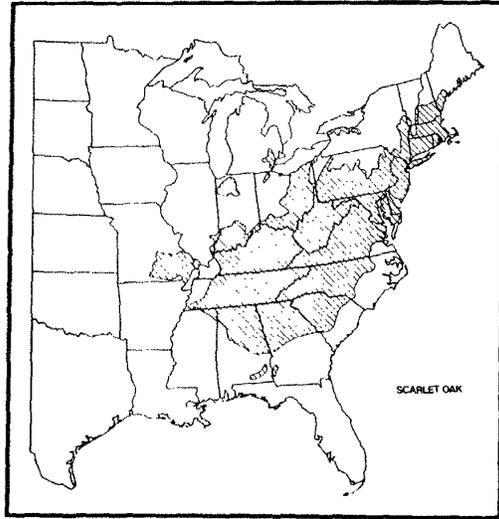
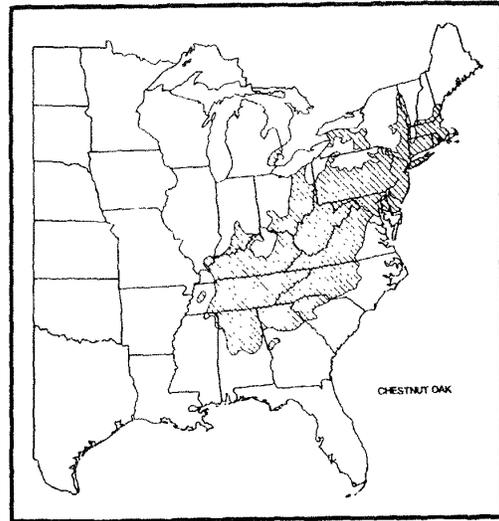
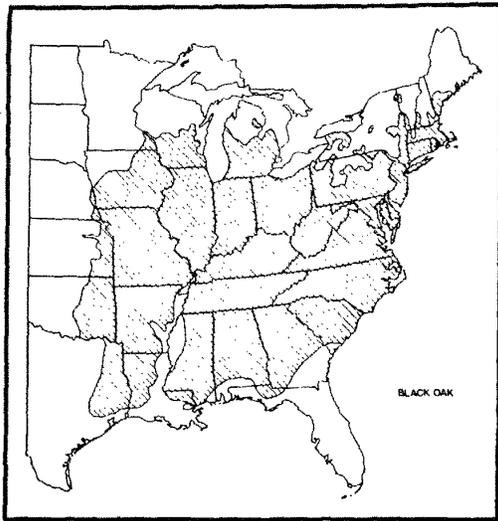


Figure 2. Natural range of oaks.

Scarlet oak

Scarlet oak is found in all or parts of states east of the Mississippi River, and southeast Missouri (Fig. 2). Scarlet oak is a major species of the scarlet oak type (41). It is found with many other oaks, and with hickories and shortleaf pine, Virginia pine, loblolly pine, pitch pine, and eastern white pine. Frequently, scarlet oak is marketed as red oak, but it is not considered as durable.

Mortality and Decline

Major oak mortality events in the Eastern United States are summarized by year of occurrence in Table 2. For each event, the location, extent, and probable causes are given and references provided. Oak mortality events during this century also are summarized in Figure 3.

Oak decline and oak wilt are the two major regional problems that affect this species. Oaks also are susceptible to attack by numerous insects and pathogens, many of which are discussed in this report. For information on other insects and diseases that may be important in localized areas, see Hepting (1971); Solomon et al. (1980); Sinclair et al. (1987); Starkey and Brown (1986); and USDA For. Serv. (1985). For information on recent oak declines in adjoining Provinces of Canada, see Can. Dep. Fish. and For. (1971), Can. Dep. Environ. (1982), Embree (1967), and Kondo and Taylor (1984).

Distribution

Large areas of oak mortality occurred in New England and the Appalachian Mountains during the early part of this century (Fig. 3). During the 1920's, drought and frost were thought to be responsible for heavy mortality in both the red and white oak groups in North Carolina and Virginia (Table 2). More than 3 million board feet of white oak were lost in Bland County, Virginia, in 1925 (Beal 1926). In Pennsylvania, drought and insect attacks were implicated in oak mortality during the 1930's (Table 2). Between 1950 and 1980 there were numerous reports of extensive oak mortality, usually associated with insect defoliation, from the Mid-Atlantic States and New England (Table 2). Outbreaks of oak decline have affected thousands of acres, with mortality of up to 100 percent reported for some stands. For example, between 1967 and 1972, 2.4 billion board feet of oak were lost over 900,000 acres in Pennsylvania (Allison 1975). Widespread oak mortality also has occurred often throughout the Southeast (Table 2). Oak wilt is not included in Table 2 since many of the early studies report the initial detection of the disease rather than the extent of mortality.

Causes

In general, oak decline is thought to be initiated by a stress that predisposes trees to attack by secondary pathogens and insects. These secondary organisms often cause the death of the tree.

Silvicultural. Site and stand conditions such as soil type, slope, aspect, stand species composition, and stand age

may be important in determining the susceptibility of trees to these stress factors (Bess et al. 1947; Houston and Valentine 1977; Nichols 1968; Skelly 1974; Wargo et al. 1983).

Abiotic. Drought often has been reported as an initiating stress or as the direct cause of oak decline. Most of these reports are from the Southeast (Balch 1927; Bassett et al. 1982; Flake 1984a,b; Houston 1971; McCracken 1985; Mistretta et al. 1981; Rauschenberger and Ciesla 1966; Tainter 1985; Tainter et al. 1983, 1984; True and Tryon 1956; Tryon and True 1958; Yeiser and Burnett 1982). There also have been reports of drought-initiated decline from the Northeast (Abrahamson 1977; Houston 1971; Hursh and Haasis 1931; Karnig and Lyford 1968; McIntyre and Schnur 1936; Parker et al. 1964) and the Lake States (Walters and Munson 1980). Other abiotic stress factors include frost (Abrahamson 1977; Beal 1926; Houston 1981b; Hursh and Haasis 1931), ice storms (Dance and Lynn 1963), and high water tables (Dugar and Weiss 1976; Wargo et al. 1983).

Insects. In the Northeast, insect defoliation probably has been the most common initiating stress (Nichols 1968; Staley 1965). With reports dating to the early 1900's, defoliation by the gypsy moth has received the most attention for its role in oak mortality (Baker 1941; Bitzer 1971; Brown et al. 1979; Campbell and Valentine 1972; Campbell and Sloan 1977; Houston 1981a; Kegg 1971, 1973; McManus 1980; Quimby 1985; 1986; Stephens 1971, 1981).

Other defoliators that have been important at one time include the oak leafroller, oak leaf-tier, fall cankerworm, forest tent caterpillar, fruit tree leafroller, winter moth, and elm spanworm (Embree 1967; Fedde 1964; Knull 1932; Nichols 1968; Rauschenberger and Ciesla 1966; Staley 1965). Defoliation that occurs for several consecutive years can result in tree mortality (McManus 1980; Staley 1965; Trefry 1984).

Diseases. Leaf diseases such as anthracnoses may serve the same purpose as insect defoliators (McCracken 1985; Wargo et al. 1983). Root diseases frequently are associated with declines, but these are thought to be primarily secondary invaders on weakened trees (Wargo 1977).

Oak wilt. Reports of oak mortality from Wisconsin and Minnesota during the late 1800's and early 1900's are now considered the initial reports of oak wilt (Carter 1975; Tainter 1985). Oak wilt was first recognized as an important disease in Wisconsin in 1943 (Henry and Moses 1943) and now is found in 21 states. The disease has killed thousands of trees in localized areas, and has been especially destructive in the Midwest (Rexrode and Brown 1983). However, oak wilt never reached major epidemic proportions as originally feared. Oak wilt is a vascular disease caused by the fungus *Ceratocystis fagacearum* (Bretz) Hunt. The disease is transmitted by sap-feeding beetles, bark beetles, and natural root grafts. All oak species are susceptible to the disease, though red oaks are attacked more often and die sooner than white oaks (Rexrode and Brown 1983). A decline of live oak has been reported in central Texas since



Figure 3.—Distribution of oak mortality during this century.

Table 2.—Chronological summary of oak mortality in the Eastern United States

Year	Location	Severity/extent	Cause	Reference
1856	VA	Bland Co.; white oak.	Late spring freeze.	Beal 1926
Late 1890's to early 1900's	Appalachian Mountain region	Great amounts of mortality annually. Trees scattered throughout forest stands.	Attack by <i>Agrilus</i> .	Hopkins 1903
1911–21	MA, NH and ME (coastal regions)	37% of red oaks, 46% of black and scarlet oaks, 65% of white oaks died.	Gypsy moth defoliation and drought followed by <i>Agrilus</i> and <i>Armillaria</i> .	Long 1914
1913–14	NY	Chenango Co.; chestnut oak.	Spring frost and <i>Armillaria</i> .	Long 1914
1914–16	Southern Appalachian region	Great numbers of oaks died during this period.	Drought associated with <i>Agrilus</i> and <i>Armillaria</i> .	Balch 1927
1915	Southeastern MN	Great numbers of trees in the red and white oak groups died over several years.	Attack by <i>Agrilus</i> and <i>Armillaria</i> with drought as contributing factor.	Chapman 1915
1925	VA	3 million board feet of white oak killed in valleys and hollows between May 1925 and summer 1926 in Bland Co.	Late freeze in May 1925.	Beal 1926
1925–28	Southern Appalachian region	Great numbers of trees in red oak group died on ridges and upper slopes.	Severe drought in 1925 followed late freeze in 1927 and by <i>Agrilus</i> , <i>Prionus</i> , and <i>Armillaria</i> .	Balch 1927; Hursh and Haasis 1931
Early 1930's	Eastern and central PA	Severe mortality of oaks. Almost 100% mortality on 3,000-acre area in Pike Co.	Defoliation by fruittree leafroller and elm spanwork followed by <i>Agrilus</i> attack, diseases, and overmaturity.	Knull 1932; McIntyre and Schnur 1936
1933–52	New England	18% mortality of oaks in ME, NH, VT, and MA; 9% mortality in CT and RI. Mortality up to 50% in many stands.	Gypsy moth defoliation. Also associated were drought, frost, <i>Agrilus</i> attack, diseases, and overmaturity.	Crossman 1948; House 1952; USDA Agric. Res. Admin. 1948, 1953, 1954
1947	Central New England, eastern PA, southern Appalachians	Excessive mortality of oak.	Variety of stresses, insects, and diseases.	Hansbrough 1948
1953–59	Pocahontas Co., WV and other scattered sites in WV and VA	Area of 400 square miles with losses up to 50 scarlet oaks per acre. Red and black oak also affected.	Drought combined with poor site conditions in some areas.	Gillespie 1956; Lowe and Mook 1960; Tryon and True 1958
	Southwestern NY and north-central PA; NJ, MD, and DE	Significant mortality in the red oak group over 8,000 square miles in NY and PA.	Defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Zabel et al. 1958; Lowe and Mook 1960; Staley 1965

Table 2 (Continued)

Year	Location	Severity/extent	Cause	Reference
1953-68	Eastern and central PA	Severe mortality (3,000 to 7,000 board feet/acre) of red oak group species on 200,000 acres; serious dieback on 1 million acres. Mortality in white oak group over 50,000 acres; dieback on 2 million acres.	Leaf-tiers and other defoliators followed by <i>Agrilus</i> and <i>Armillaria</i> . Poor stand conditions also contributed.	Nichols 1968
1956-64	Hudson River Valley, NY	Extensive mortality of red oak group on ridgetops.	Drought, late spring frosts, and leafroller defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	SAF 1964
1956-65	Southeastern U.S. (particularly NC, GA, and TN)	Extensive areas of oak mortality across region. Losses included 25 million board feet (GA) and 2 million board feet (TN)	Drought and defoliation by elm spanworm and cankerworms followed by <i>Agrilus</i> and <i>Armillaria</i> .	Fedde 1964; Houston 1971
1957-67	CT	40% mortality, mostly in white oak group.	Defoliation by gypsy moth.	Stephens 1971
1958-59	MI	Loss of 20 million board feet in red oak group over 29,000 acres in central part of state.	Combination of drought in 1955, defoliation by fruit-tree leafroller in 1956-1957, frost in 1958.	Michigan Dep. Conserv. 1960; Gibbons and Butcher 1961
1959	Ozark Mountains, AR	Many dead and dying white oaks in 7 counties.	Poor site conditions and attack by <i>Scytinostroma galactinum</i> (= <i>Corticium galactinum</i>).	Toole 1960
1964-66	WI	N. pin oak mortality on 1,700 acres in Menominee Co. N. pin oak declining in central Wisconsin sandy area.	Defoliation by walkingstick. Drought, post-oak locust defoliation, hailstorm	Renlund 1966 Renlund 1966
1965	VA	104,000 acres on George Washington N.F. and 5,900 acres on Jefferson N.F.	Spring frost followed by <i>Agrilus</i> and <i>Armillaria</i> .	Raushchenberger and Ciesla 1966
1965-66	ME	Dieback and moderate mortality of red oak in Canton area.	Leafminers, water stress, and stand disturbance followed by <i>Armillaria</i> .	Stark 1967
1966-67	NY	Severe mortality of scarlet oak on hilly areas in the Hudson Valley.	Drought and defoliation by cankerworms and loopers.	Karnig and Lyford 1968; SAF 1968
1966-69	MI	Patchy oak mortality on the Huron and Manistee N.F.	Drought, late spring frosts, defoliation by leafrollers, and post-oak locust, followed by <i>Agrilus</i> .	Millers 1966, 1969

Table 2 (Continued)

Year	Location	Severity/extent	Cause	Reference
1967-70	NJ	28% mortality of oaks over 1,350 acres in the Morristown National Historical Park.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Kegg 1971
1967-72	PA	2.4 billion board feet lost on 900,000 acres. Estimated 5.5 million trees killed in 1971.	Defoliation by leaf-tiers and leaf-rollers (probably followed by <i>Agrilus</i> and <i>Armillaria</i>).	Allison 1975; USDA For. Serv. 1971a, 1972
1968	WI	Pockets of oak dying in Marinette Co.	Defoliation by post-oak locust and walkingstick.	Renlund 1968
1968-72	NJ	Severe mortality over 18,000 acres—white oak: 84% mortality; chestnut oak: 66%; red oak: 41%; black oak: 48%; scarlet oak: 27%. Estimated 1.2 million oaks died in Newark Watershed.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Kegg 1973
1969-73	CT	Average oak mortality of 43% on 13 study plots statewide. Chestnut oak: 80% mortality; white oak: 42%; scarlet oak: 33%; red oak: 32%; black oak: 27%.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Dunbar and Stephens 1975; Stephens 1981
1970-80	North, central, and eastern PA	Yearly losses averaged 21% in defoliated stands. In Pocono Mtns., an average of 43 trees/acre were lost.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Quimby 1986; Wargo 1977
1970-84	Southern States	Localized mortality (up to 25%) and dieback throughout region. Especially severe in Appalachian Mtn. region.	Variety of stresses and secondary organisms.	McCracken 1985; USDA For. Serv. 1981
1972-75	RI	Loss of 17% of basal area of oak over 3 years.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Brown et al. 1979
1973	East-central WI	Dieback and mortality on 4,500 acres of red oak and 8,000 acres of pin oak.	Defoliation by oak leaf-rollers and leaf-tiers.	Doerner 1974; Renlund 1974
1974-76	AL, AR, LA, MS, OK, TX	Large numbers of dead and dying oaks (mostly red oak group). Impact greatest in southern AR and eastern TX.	Wet springs and site disturbance followed by secondary organisms.	Dugar and Weiss 1976
1975-77	Southern NY	Mortality and dieback of red oak on 65,000 acres in Putnam, Westchester, and Sullivan Counties.	Defoliation by gypsy moth.	SAF 1976

Table 2 (Continued)

Year	Location	Severity/extent	Cause	Reference
Late 1970's	Western NY	Mortality and dieback of white oak in Allegany State Park and surrounding counties.	Defoliating insects, late spring frost, and damage from hurricane Agnes followed by <i>Agrilus</i> and <i>Armillaria</i> .	Abrahamson 1977; SAF 1976
	MA	English oak dying on Cape Cod. Red oak in Erwin and Wendell State Forests dying. Up to 25% of trees dead (more than 3,000 board feet per acre).	<i>Agrilus</i> and unknown fungus suspected. Repeated defoliation by oak leaf-tier.	Feder et al. 1980 Mass. Div. For. Parks 1980a, 1980b, 1983, 1984, 1985a,b
1976-78	MO, IL, IN, OH, WV, NJ, MI, IA	Oak mortality and decline. In Indiana, an estimated 321,778 board feet lost.	Probably drought and/or defoliation followed by secondary organisms.	Hanson et al. 1976
1976-80	WI	Several species of oak dying in southern half of state.	Drought, ice storms, defoliators, <i>Agrilus</i> sp., manmade disturbances.	Haack and Benjamin 1982; Renlund 1976, 1977, 1978, 1979, 1980
1977	South-central PA	Dieback and mortality of chestnut oak on 14,000 acres, mostly ridgetops, in Bedford, Franklin, Perry, and Juniata Counties. 40% of volume on 7,600 acres in Bedford Co. dead or dying.	Possibly drought. Not associated with defoliated areas.	Pa. Dep. Environ. Resour. 1978
1977-80	Central MN	Extensive mortality and dieback of red and pin oak on 30,552 acres in Sherburne National Wildlife Refuge.	Drought followed by <i>Agrilus</i> and <i>Armillaria</i> .	Minn. Dep. Nat. Resour. 1978, 1979, 1981; Walters and Munson 1980
1977-85	Central MA	Mortality up to 17% of red oak in eastern Franklin Co. Loss of 3,278 board feet per acre.	Defoliation by oak leaf-tier complex.	Mass. Div. For. Parks 1981, 1985a
1978-82	MI	Heavy mortality and dieback of red, black, and white oaks in northern areas of Lower Peninsula and eastern parts of Upper Peninsula.	Drought and leafroller defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Mosher et al. 1979, 1980, 1981, 1983
1978-80	AR, MS, TX, FL	Southeastern water oaks.	<i>Hypoxyton</i> spp., <i>Ganoderma lucidum</i> , <i>Agrilus</i> , and drought.	Lewis 1981
1979	NC	Thousands of red oaks died in patches up to 100 acres in Nantahala, N.F.	Prolonged drought. Decline did not continue in residual stand.	Tainter and Benson 1983; Tainter et al. 1984

Table 2 (Continued)

Year	Location	Severity/extent	Cause	Reference
1980-81	Northwestern AR	Up to 50% mortality in some stands on Ozark N.F. Average of 10% mortality of red and black oaks in northwestern part of state.	Drought followed by <i>Hypoxyton</i> spp. Drought in 1980 worst in 30 years.	Bassett et al. 1982; Mistretta et al. 1981
1980-83	Eastern and central PA	33% mortality of oak sawtimber on 350,000 acres and 51% of sawtimber on 341,000 acres. About 68 million trees died during this period.	Drought and gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Quimby 1985
1980-85	MA	Mortality up to 30% in plots in Franklin Co.	Defoliation by oak leaf-tier.	Rush 1986
1980-86	MO	Between 1980 and 1983, 22% of scarlet oak sawtimber was dead and 31% was dying. For black oak, 15% dead and 20% dying. By 1986, 35 million board feet had been salvaged over 180,000 acres on Mark Twain N.F.	Drought followed by <i>Agrilus</i> , <i>Hypoxyton</i> spp., and <i>Armillaria</i> .	Law and Gott 1987; Rush 1986
1981	Coastal SC	Severe mortality of red oaks (southern red, willow, laurel, water) from Georgetown to Myrtle Beach covering an area of approximately 190 square miles. Urban and forest trees affected.	Severe drought in 1980. Damaged trees attacked by <i>Hypoxyton</i> spp.	Tainter et al. 1983
1981-83	WV	In the eastern panhandle, average mortality of 20% in areas of 2 successive years of defoliation.	Defoliation by half-wing geometer and loopers.	USDA For. Serv. 1984
1982-84	VA	Red, black, and chestnut oak dying on the George Washington N.F. on 1,700 acres.	Drought and defoliation by fall cankerworm and linden looper, followed by <i>Agrilus</i> , root rots, and canker fungi.	Dull 1982; Sites 1982; USDA For. Serv. 1983
1982-85	VA	At Thunder Ridge, Jefferson N.F., 42% black and red oak declining and 18% dead. Mortality increasing throughout forest.	Overmaturity, drought, and defoliation followed by <i>Armillaria</i> and wood borers.	Flake 1982, 1984a, 1985
1983-85	IA	Scattered mortality and dieback statewide.	Drought followed by <i>Agrilus</i> , <i>Hypoxyton</i> , and <i>Armillaria</i> .	Rush 1986

Table 2 (Continued)

Year	Location	Severity/extent	Cause	Reference
1983-85	IA	Scattered mortality and dieback statewide.	Drought followed by <i>Agrilus</i> , <i>Hypoxylon</i> , and <i>Armillaria</i> .	Rush 1986
1984-85	Central MN	Mortality and dieback; mortality increasing.	Drought and defoliation (variable oakleaf caterpillar, redhumped oakworm, and walkingstick) followed by <i>Agrilus</i> and <i>Armillaria</i> .	Minn. Dep. Nat. Resour. 1985, 1986
	IL	16,500 acres mortality and dieback on Shawnee N.F.	Drought, defoliation, then <i>Agrilus</i> and root rot.	Miller-Weeks 1985
	MD and DE	Mortality and dieback scattered across states.	Drought and other stress factors.	Miller-Weeks 1985; Rush 1986
1984-86	TN	Mortality and dieback throughout state. Most severe in center of state. Some stands in Cherokee N.F. with 25 to 30% of the oaks declining.	Stress (cause unknown, possibly drought or defoliation) followed by root rots, wood borers, and canker fungi. Mortality in Kentucky.	Lewis 1984; Sites 1984
1985	NY	Mortality common in southeast, up to 90% mortality.	Defoliation by gypsy moth.	Miller-Weeks 1985
	VA, NC, SC, TN, GA, AR, AL	Survey of 38 plots. Scarlet oak: 55% dead or dying; black oak: 53%; unidentified red oaks: 48%; southern red oak: 41%; northern red oak: 40%; white oak: 32%; chestnut oak: 28%; post oak: 18%.	Not determined.	Starkey and Brown 1986
1985-86	Southeastern and western NY	Mortality up to 90% in some stands. Red oak primarily in Orange, Sullivan, Ulster, Albany, Chemung, and Genesee Counties. White oak decline in Steuben Co.	Gypsy moth defoliation followed by <i>Agrilus</i> and <i>Armillaria</i> .	Birmingham 1987 (pers. comm.); Rush 1986

the early 1930's (Lewis and Oliveria 1979). Work in the 1960's implicated species of *Cephalosporium*, but subsequent research has identified *C. fagacearum* as the causal agent (Appel and Maggio 1984; Lewis and Oliveria 1979). For detailed information on oak wilt see Mistretta et al. (1984).

Secondary organisms. The secondary organisms most commonly associated with oak decline are the twolined chestnut borer and the fungus *Armillaria* sp. (Abrahamson 1977; Baker 1941; Chapman 1915; Dunbar and Stephens 1975; Kegg 1973; Nichols 1968; Parker et al. 1964; Staley 1965; Walters and Munson 1980; Wargo 1977). These organisms usually are the final cause of death of most trees with oak decline (Dunbar and Stephens 1975; Wargo 1977). Other wood borers, such as those in the genus *Prionus*, have been reported as attacking stressed oak trees (Staley 1965). Other root rots associated with oak mortality are *Scytinostroma galactinum* (Fr.) Dank; *Ganoderma lucidum* (Leyss.:Fr.) Karst.; and *Clitocybe tabescens* Bres. (Filer and McCracken 1969; Lewis 1981; Toole 1960). Twig mortality and crown dieback often are caused by canker fungi, including *Hypoxyylon* spp.; *Dothiorella quercina* (Cke. & Ell.) Sacc.; *Endothia* spp.; *Fusarium* sp.; and *Botryodiplodia* sp. (Bassett et al. 1982; Boyce and Speers 1960; Gruenhagen 1965; Schmidt and Fergus 1965; Stipes and Phipps 1971; True and Tryon 1956).

Trends

In general, oak mortality events tend to occur frequently in the same areas (Fig. 3). Oak mortality has occurred throughout the Appalachians before 1950 and during every decade thereafter. There have been several episodes of mortality in the Lake States, Southern States, and in New England and New York.

Maple and Declines

Species, Range, and Use

Sugar maple

Sugar maple extends from Nova Scotia and Quebec west into Ontario, southeastern Manitoba, and western Minnesota, south to central Iowa, eastern Kansas, and Missouri, and east to Tennessee and northern Georgia (Fig. 4). The most important stands are found in the Lake States, Ohio, Pennsylvania, New York, New England, the southern Appalachians, and Canada.

Sugar maple is a major species of four cover types: sugar maple—beech—yellow birch (25); black cherry—sugar maple (28); red spruce—sugar maple—beech (31); and beech—sugar maple (60). White ash, American basswood, American beech, yellow birch, red maple, and red spruce are common associates in the northern range of sugar maple. Found less frequently with sugar maple are northern red oak, ironwood, American elm, balsam fir, black ash, black cherry, paper birch, sweet birch, eastern hemlock, eastern white pine, and white spruce. Typical associates in the central hardwood zone and the Appalachian Highlands are yellow-poplar, yellow buckeye, cucumbertree, several oak species, and black walnut.

Some uses of sugar maple include charcoal, railroad ties, and veneer used in the manufacture of panels for furniture and musical instruments. Sugar maple is one of the leading native woods because of its hardness, strength, and good working and finishing qualities. It is valuable for flooring because of its uniform texture and hardness. The maple syrup industry is important in some Northeastern and North-Central States. Sugar maple is the standard wood in the

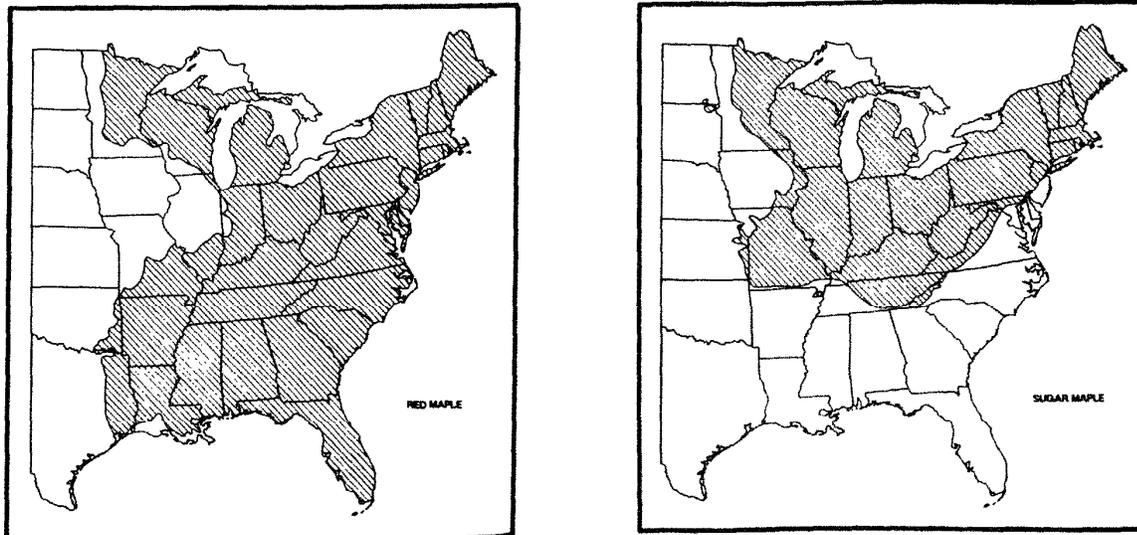


Figure 4.—Natural range of sugar and red maple (from Little 1971).

United States for shear test block.

Red maple

Red maple grows throughout the Eastern United States from the Atlantic Ocean to the prairies (Fig. 4). In Canada, it is scattered throughout the Maritime Provinces, southern Quebec, and Ontario. Red maple is a major species in three forest cover types: gray birch—red maple (19); black ash—American elm—red maple (29); and sweetbay—swamp tupelo—red maple (104). Red maple is associated with more than 70 commercial tree species including gray birch, yellow birch, paper birch, white pine, sugar maple, black cherry, northern red oak, pin oak, chestnut oak, black oak, and scarlet oak. Uses are similar to those of sugar maple except where strength and hardness are required.

Mortality and Decline

Major maple mortality events in the Eastern United States are summarized in Table 3. For each event, the location, extent, and probable causes are given and references provided. Maple mortality events during this century also are summarized in Figure 5. For information on more recent maple declines in Canada see Can. Dep. Environ. For. Serv. (1981, 1982); Can. Dep. For. Rural Develop. (1968); Lachance (1985); and Kondo and Taylor (1984).

Sugar maple is susceptible to a large number of diseases (Hepting 1971; Ohman 1969). Discoloration and decay are the greatest causes of decreases in volume and growth of sugar maple, though they rarely result in the death of trees (Ohman 1968). Other diseases of sugar maple are

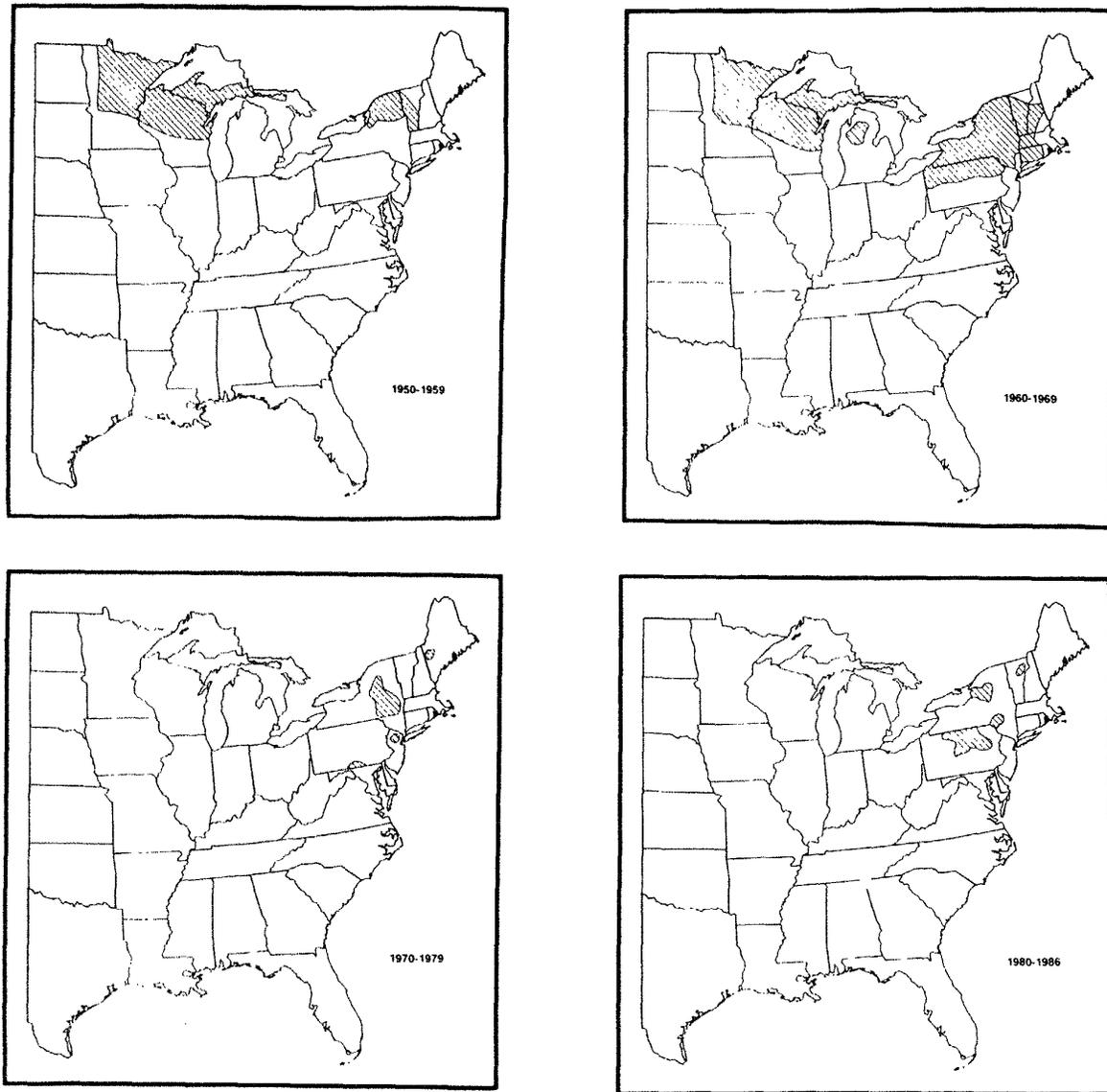


Figure 5.—Distribution of maple mortality during this century.

Table 3.—Chronological summary of map mortality in the Eastern United States

Year	Location	Severity/extent	Cause	Reference
1912–13	Washington, DC north through New England	Leaf scorch and browning on most Norway and sugar maples. Norway maple appeared severely affected.	Summer drought.	Hartley and Merrill 1915
1939–49	MA	Scattered mortality, dieback, and leaf scorch.	Drought, <i>Verticillium</i> wilt, and canker fungi.	Marsden 1950; McKenzie 1943
1951–54	NY and VT	Dieback over large areas of northern NY and VT.	Defoliation by forest tent caterpillar.	Waters 1955; Welch 1963
1956–58	ME, NH, VT, MA, CT, NY, PA	General distribution of dieback and some mortality. Worst areas were VT, NH, ME, and southern NY.	Bleeding canker, drought, <i>Armillaria</i> , and <i>Verticillium</i> wilt. Reports do not specify forest or roadside trees, but probably concern mostly latter.	Waters and Waterman 1957; Waters and Mook 1958
		Mortality in Florence Co. of sugar maple of all sizes on about 10,000 acres. More than 1,500,000 board feet of sawtimber dead. Small patches of severe mortality in Iron and Ashland Counties.	Defoliation by leafrollers and a webwork followed by <i>Armillaria</i> . Past cutting practices may also have contributed.	Anderson 1959; Lake States For. Exp. Stn. 1964
1958–62	Lake States	Dieback of approximately 28% of larger sugar maples and 38% of red maple in Upper Peninsula, MI. Dieback scattered throughout MN, WI, and MI. No extensive mortality.	High water tables and past cutting practices may have contributed.	Anderson and Schmiege 1959; Kessler 1963, 1965
1950's to 1960's	Northeastern U.S.	Widespread mortality and dieback. Most damage to overstory trees along roads and to ornamentals.	Salt damage, drought, defoliation, overexposure, and overmaturity	Lowe and Mook 1960; SAF 1960; Maine For. Serv. 1957, 1959; Stark 1965; Vermont Dep. For. Parks 1959, 1961, 1963; Westing 1966
		88% of sugar maple along state roads in NH in some state of decline.	Salt damage.	Westing 1966
		Survey of forest stands in 1963 found 18% of sugar maples with dieback and 3% dead in PA, 11% dieback and 5% dead in NH, 10% dieback and 3% dead in VT, and 8% dieback and 5% dead in MA.	Not specified.	Tegethoff and Brandt 1964
		Scattered mortality and dieback in forest stands and sugarbushes in NY in early 1960's. Heavy mortality reported from the southeastern part of NY.	Drought and other unfavorable environmental conditions. Overcutting and <i>Armillaria</i> may have contributed.	Hibben 1961, 1964; SAF 1964; New York Conserv. Dep. 1962

Table 3 (Continued)

Year	Location	Severity/extent	Cause	Reference
1952-67	CT	15-19% annual mortality rates for sugar and red maple in general forest stands scattered around state.	Heavy defoliation by gypsy moth, elm spanworm, and cankerworms.	Stephens 1971
Mid-1960's	MI	Dieback of roadside maples.	Drought and salt damage.	Michigan Dep. Conserv. 1966
1968-70	New England	Mortality and dieback of roadside trees. Problem is chronic.	Salt damage and possibly localized air pollution.	Mass. Div. For. Parks 1968, 1971; USDA For. Serv. 1970a,b
1968-71	Central NY and ME	Severe crown dieback of sugar maple on poor sites. Some mortality in intermediate and suppressed classes in ME.	Defoliation by saddled prominent.	Grimble and Newell 1973
1971	NJ	17% of red maple dying or dead over 17,855 acres in Newark Watershed.	2 years of defoliation by gypsy moth	USDA 1972
1973	PA	Mortality evident on approximately 2,800 acres in southern part of state.	Defoliation by forest tent caterpillar.	Doerner 1974
1975-78	ME	Dieback of urban maples throughout state. Problem is chronic.	Mechanical damage, soil compaction, air pollution.	Maine Dep. Conserv. 1977, 1979
1976	MI, WI, IN, VT, NH, NY	Increase in reports of maple decline. Most reports are of damage to urban and roadside trees. Some reports of declines in forest stands.	Drought, salt, and soil compaction probably involved.	Hanson 1977
1977	MI	10% sugar maple mortality over 1,800 acres on Drummond Island.	Defoliation by forest tent caterpillar and saddled prominent in consecutive years.	Mosher and Simmons 1978
1977-82	WI	Dieback and mortality of urban sugar maples. Problem is chronic.	Basal canker caused by <i>Fusarium</i> spp. and collar rot caused by <i>Phytophthora</i> sp.	Drilias et al. 1982
1978-81	Southern MN	Severe dieback and mortality of roadside, ornamental, and urban trees.	Drought. Some trees with Verticillium wilt. Damage noted in forest trees.	Minn. Dep. Nat. Resour. 1979, 1981
1980-82	Southeastern NY	100,000 acres in Delaware Co., up to 100% mortality in some stands.	Defoliation by forest tent caterpillar.	USDA For. Serv. 1982b, 1983
1980-85	PA	Dieback and mortality of red maple in 14 northern counties. 75,000 acres affected in McKean Co. and 23,000 in Potter Co.	Possibly gypsy moth defoliation, drought, and heavy seed crops.	Pa. Dep. Environ. Resour. 1985a

Table 3 (Continued)

Year	Location	Severity/extent	Cause	Reference
1981-85	NH	Dieback and mortality on 2.5 million acres of northern hardwoods.	Insect defoliators.	Rush 1986
1982	MO	Mortality and dieback of sugar maple in urban area. Problem is chronic.	Not specified	Missouri Dep. Conserv. 1982
1984	MN, MI	Mortality and dieback scattered.	Drought; <i>Agrilus</i> spp.	Miller-Weeks 1985
	NY	Moderate to heavy damage of sugar maple in 60,000 acres west of the Adirondacks. 100% mortality on 6,000 acres.	Dieback following suspected insect defoliation.	Miller-Weeks 1985
1985	NY	Red maple dieback noted on 130,000 acres in Herkimer and Lewis Counties.	Not reported.	Rush 1986
1984-85	North-central PA	40-60% mortality on 1,600 acres and more than 60% mortality on 1,700 acres in Potter. Most mortality above 2,000 feet elevation. Scattered dieback and mortality in other counties.	Related to past defoliation (cankerworms) and poor site conditions. Thrips and anthracnose damage on many trees.	Pa. Dep. Environ. Resour. 1985a,b; Rush 1986
1985	MA	Dieback and some mortality of sugar maple at Mt. Greylock.	Not determined.	MacConnell et al. 1986
1984-86	VT	Light to moderate mortality of sugar maple over approximately 8,400 acres in northern part of state.	Mortality on 3,900 acres possibly related to defoliation by forest tent caterpillar. Causes unknown for other mortality. Surveys are ongoing.	Rush 1986
	IA	Dieback and mortality of sugar maple in urban situations statewide. Problem is chronic.	Unknown; possibly a combination of stress factors including drought, cankers, leaf diseases, salt damage, and mechanical damage.	Sweets 1984a,b, 1986

discussed in Hepting (1971) and Ohman (1969). Insects also damage sugar maple in localized areas (USDA For. Serv. 1985). Declines of sugar maple have been a common occurrence during the past 100 years. These are referred to as maple blight, maple dieback, and maple decline. For this report, maple declines are discussed as they relate to forest stands, sugarbushes, and roadside and urban trees.

Decline in forest stands

Distribution. Reports of maple mortality in the Northeast date to the early part of this century (Hartley and Merrill 1915; Westing 1966). During the 1930's and early 1940's, maple mortality and dieback were noted in the Midwest (Aikman and Smelser 1938), the Northeast (McKenzie 1943; Westing 1966), North Carolina (Hepting 1944; Kessler 1972), and Quebec (Pomerleau 1953a). Dieback of maples in forest stands was reported during the early 1950's in New York, Pennsylvania, New England, and Ontario (Griffin 1965; McLaughlin et al. 1985; Waters and Mook 1958; Welch 1963). Unfortunately, most of these reports do not distinguish between forest maples and roadside or ornamental maples.

During 1956 and 1957, mortality of sugar maple in all age classes was severe on commercial timberland in Florence County, Wisconsin (Anderson 1959; Skilling 1959; USDA For. Serv. 1964). Pockets of mortality were scattered over 10,000 acres, with timber losses estimated at more than 1.5 million board feet (Anderson 1959). This mortality was known as maple blight because of the rapid death of affected trees of all ages, and was distinguished from maple dieback (Lake States For. Exp. Stn. 1964). Maple dieback affected mostly overstory trees and resulted in the slow deterioration of trees over time (Lake States For. Exp. Stn. 1964). Maple blight began to subside after 1958 (Skilling 1959; Lake States For. Exp. Stn. 1964).

Maple dieback was reported in the Lake States, the Northeast, and Canada during the late 1950's and early 1960's (Table 3). Reports from the Lake States indicate that dieback was scattered in forest stands across the region, and was most obvious in areas that had been cut (Anderson and Schmiede 1959; Kessler 1963, 1965). Extensive mortality was not associated with dieback (Kessler 1965). Westing (1966) noted that most of the maple decline in the Northeast concerned roadside or ornamental trees, though there were some reports of decline of forest maple. Reports from New York (Hibben 1961, 1964; SAF 1964; New York Conserv. Dep. 1962) indicate scattered dieback and some mortality in forest stands across the state (particularly in southeastern New York). A survey of ash dieback in six Northeastern States in 1963 noted dead and dying sugar maple trees in forest stands (Tegethoff and Brandt 1964). Dieback was observed on 10 to 20 percent of the trees, but mortality was not severe (less than 5 percent of observed trees).

Mortality of up to 100 percent has been reported for some stands (Norton 1983; USDA For. Serv. 1983). Most comprehensive surveys have reported mostly dieback with little associated mortality. For example, an aerial survey in Quebec (Paradis et al. 1985) found that 83 percent of the

maple stands within an 8,000 square-mile area had some dieback. However, only 6 percent of the stands were rated as having moderate (26 to 50 percent of foliage missing) or severe (more than 50 percent of foliage missing) dieback. The decline apparently began in one small area and since has spread over most of the maple region (Carrier 1986). However, in adjoining Vermont, the general condition of sugar maple is much better except for areas previously defoliated by the forest tent caterpillar (Kelley and Eav 1987). Periods of decline usually are followed by periods of recovery (Griffin 1965; Hibben 1964; Kessler 1963; Ohman 1969).

Causes. Drought often has been reported as important in maple decline (Griffin 1965; Hartley and Merrill 1915; Hibben 1961, 1964; Marsden 1950; McLaughlin et al. 1985; Ohman 1969; Skelly and Wood 1966; Westing 1966). High water tables and frost occasionally are involved (Kessler 1963, 1965; Westing 1966). Stands subjected to overcutting often show decline, probably due to overexposure (Griffin 1965; Hibben 1964; Kessler 1963, 1965; Skilling 1964). This is similar to postlogging decadence described for yellow birch (Hall 1933; Spaulding and MacAloney 1931). Skilling (1964) found that stands with more than 50 percent volume in sugar maple were susceptible to maple blight. Soil nutrient deficiencies, related to drought and poor site conditions, also have been suggested as contributing factors (Allen 1987; Kielbaso and Ottman 1976; Mader and Thompson 1969). Currently, air pollution is being considered as a possible stress factor, though evidence remains circumstantial (McLaughlin et al. 1985; Tomlinson 1983).

Maple decline in forest stands generally is considered to be stress initiated, much like oak decline (Houston 1981b). Insect defoliation probably is the most common biotic stress factor. The Wisconsin maple blight of 1956-57 (Giese et al. 1964; Lake States For. Exp. Stn. 1964) was caused by a complex of three defoliators occurring during different periods of the growing season. The defoliators were two leafrollers, *Sparganothis acerivorana* MacKay and *Acleris chalybeana* Fernald, and the maple webworm (Giese and Benjamin 1964). Other insects that have been associated with maple decline elsewhere include the forest tent caterpillar, saddled prominent, greenstriped mapleworm, cankerworms, and aphids (Grimble and Newell 1973; Knight 1969; McLaughlin et al. 1985; Miller-Weeks 1985; Teillon et al. 1984, 1986).

The fungus *Armillaria mellea* has been commonly associated with sugar maple mortality as a secondary invader (Hibben 1964; Houston 1981b; Houston and Kuntz 1964; McLaughlin et al. 1985; Ohman 1969; Wargo and Houston 1974). The canker fungus *Steganosporum ovatum* Pers.: Merat may cause twig dieback on stressed trees (Hibben 1964; Houston 1981b; Wargo and Houston 1974). Hibben (1964) also isolated numerous other canker fungi from twigs, but none was thought to be consistently important. Stem cankers caused by *Fusarium solani* Mart. have been found throughout the Northeast on trees stressed by drought or other environmental conditions (Skelly and Wood 1966; Weidensaul and Wood 1974). Root-feeding nematodes have been associated with maple trees

with dieback symptoms (Di Sanzo and Rohde 1969). Sapstreak disease has killed sugar maple in North Carolina (Hepting 1944; Southeast For. Exp. Stn. 1959) and the Lake States (Kessler 1972; Kessler and Anderson 1960; Ohman and Kessler 1963). Sapstreak recently was found in the Northeast, though it has not been associated with significant mortality (Beil and Kessler 1979; Houston and Fisher 1964).

Decline in sugarbushes

Distribution. In the Northeastern United States and Canada, reports of decline in sugarbushes generally have coincided with reports of forest maple decline. Because of economic concerns, dieback and mortality in sugarbushes receive much attention. Over the years, however, casual observations of sugarbush decline have not always been confirmed by more intensive follow-up evaluations (Houston 1963; Millers et al. 1977; Westing 1966). Extensive surveys and research are in progress in Vermont and Quebec to determine the extent and severity of recently reported maple declines.

Causes. In many respects, maple decline in a sugarbush is similar to that in forest stands. Insect defoliation and/or adverse environmental conditions stress trees, allowing secondary organisms to invade and kill them (Houston 1981b). Mismanagement often is cited as a major reason for declines in sugarbushes (Houston 1963; Norton 1983; Ohman 1969; Westing 1966). Overtapping, animal grazing, damage by farm machinery, overmature and poor-quality trees with root and trunk decay, and a lack of species diversity within stands can contribute to dieback and mortality (Griffin 1965; Houston 1963, 1981b; Norton 1983; Ohman 1969).

Decline in roadside and urban trees

Distribution. Dieback and mortality of roadside and urban maple trees has been a chronic problem for many years throughout the range of sugar maple (Table 3). The loss of large numbers of elms to Dutch elm disease and elm

yellow has led to sugar maple being planted extensively along roads and in urban environments. Dieback in these situations is obvious and is the most frequently reported type of maple decline.

Causes. The roadside and urban environment is especially harsh. Salt damage is considered the most common cause of dieback and mortality along roads (Houston 1981b; LaCasse and Rich 1964; Ohman 1969; Westing 1966). However, there are numerous other agents that contribute to the problem. Root damage due to road paving and soil compaction may lead to drought symptoms and serve as infection courts for decay fungi (Houston 1981b). Mechanical damage to stems, such as wounds from lawn mowers, also allows entry for decay fungi. Trees along roadsides and in urban areas are subjected to heat from pavements, herbicide use, and pollution from automobile exhaust (Houston 1981b; Ohman 1969; Welch 1963; Westing 1966). Many urban maples are old and overmature (Houston 1981b; Welch 1963; Westing 1966). Canker fungi such as *Fusarium* spp. and *Phytophthora* spp. also contribute to dieback symptoms (Drilias et al. 1982; Ohman 1969).

Trends. Maple decline was common in the Northern United States in the 1950's and 1960's (Fig. 5). Since that time it has been sporadic in the Northeastern United States and in Ontario and Quebec.

Birch and Declines

Species, Range, and Use

Yellow birch

Yellow birch extends from Newfoundland and the St. Lawrence area west through southern Quebec and Ontario to the north shore of Lake Superior and western Minnesota. It also is found throughout the Great Lakes region, extending southward into northeastern Iowa and the mountains of eastern Tennessee and northern Georgia (Fig. 6).

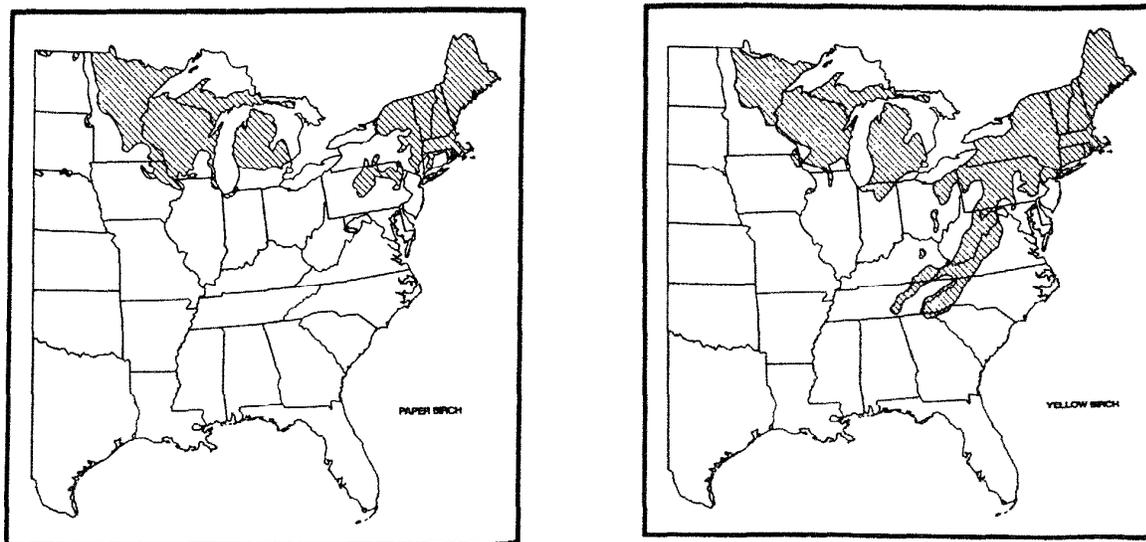


Figure 6.—Natural range of yellow and paper birch (from Little 1971).

Yellow birch is an important component of three cover types: hemlock—yellow birch (24); sugar maple—birch—yellow birch (25); and red spruce—yellow birch (30). Among the common tree associates of yellow birch are American beech, balsam fir, basswood, bigtooth aspen, black cherry, eastern hemlock, gray birch, paper birch, pin cherry, quaking aspen, red maple, sugar maple, red spruce, sweet birch, white ash, and eastern white pine.

The largest concentrations of yellow birch timber are in Eastern Canada, northern New York, New England, and the Upper Peninsula of Michigan. Yellow birch is one of the most important hardwoods used in the manufacture of furniture. It also is used in stereo and kitchen cabinets, planing mill products (particularly interior trim, flooring, sashes, and doors), and as pulpwood. Wintergreen oil is found in the inner bark of the stem and roots, but not in the large quantities found in black birch.

Paper birch

Paper birch is common in Alaska and throughout most of Canada, extending nearly to the limit of tree growth. In the United States, it is common in New England, New York, and the Lake States. It also is found in scattered localities in the other Northern States (Fig. 6).

Paper birch is an essential member of five forest cover types: jack pine—paper birch (3); aspen—paper birch (11); paper birch (18); paper birch—red spruce—balsam fir (35); and white spruce—balsam fir—paper birch (36). Additional associates include northern red oak, yellow birch, gray birch, white ash, sugar maple, pin cherry, eastern white pine, and hemlock. Paper birch is used frequently for turned products and specialty items.

Mortality and Decline

Major birch mortality events in the Eastern United States are summarized by year of occurrence in Table 4. For each event, the location, extent, and probable causes are given and references provided. Birch mortality events during this century also are summarized in Figure 7. Birch declines in Canada were reported by Balch and Prebble (1940); Barter (1953 a,b); Can. Dep. Fish. For. (1967, 1968); Pomerleau (1953b); and Sinclair and Hill (1953).

Distribution

Birch declines have been reported throughout its range in the Northern States. The declines usually are reported over

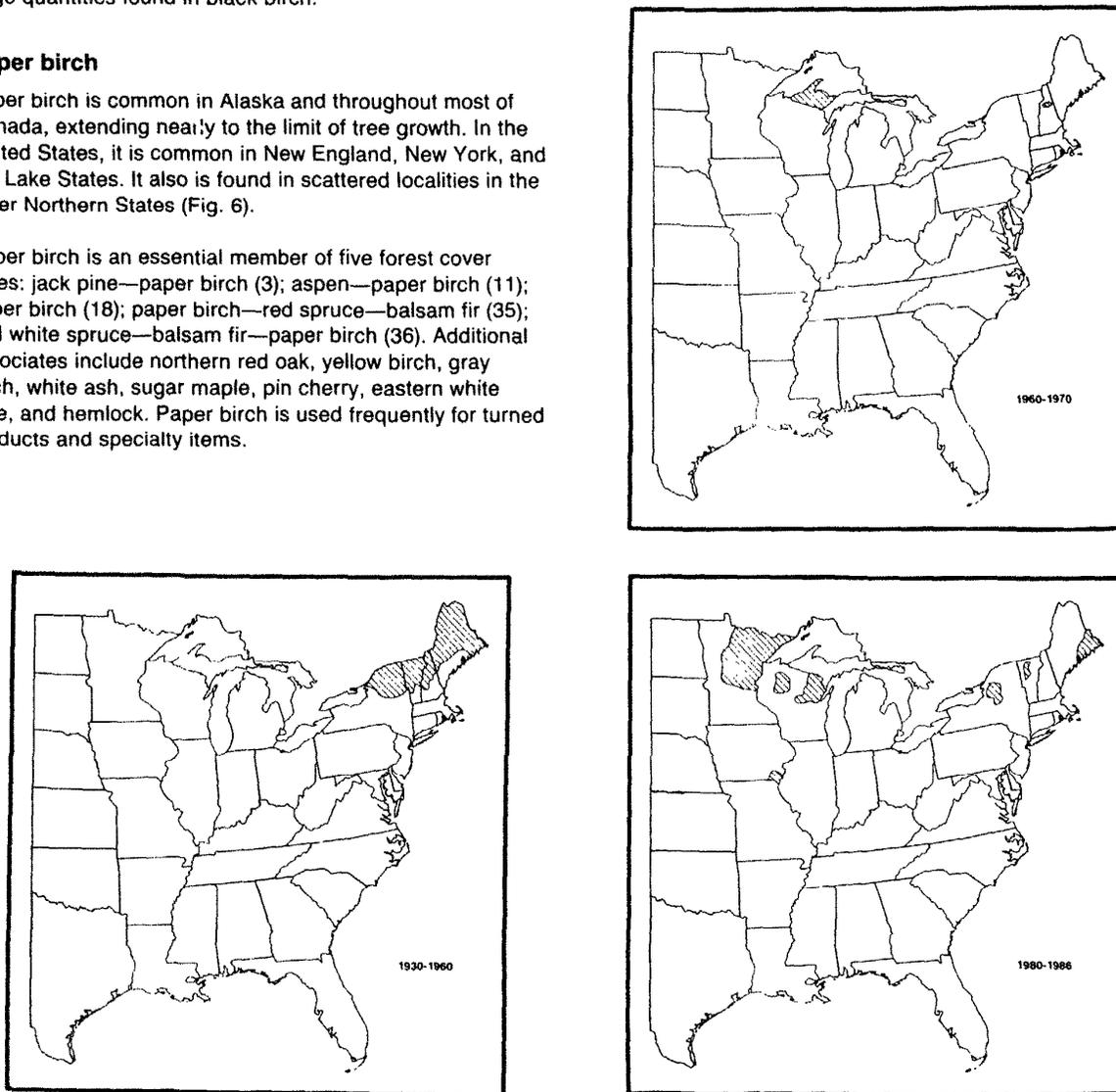


Figure 7.—Distribution of birch mortality during this century.

large areas. Redmond (1957) provided a summary of birch decline in Canada from 1940 to 1950. Local diebacks usually are associated with logging disturbance. Teillon et al. (1983, 1986) found birch decline more common on mountain slopes and dry sites.

Causes

Several abiotic stresses have been proposed as possible causes of birch dieback. Soil type and rooting depth were correlated with rootlet mortality and crown injury (Greenidge 1953; Pomerleau and Lortie 1962; Redmond 1955). Proposed causes of rootlet mortality were excessive soil temperatures (Braathe 1957; Redmond 1955) or decreased soil temperatures (Hepting 1971). Drought and a general climatic warming were implicated by some researchers (Hawboldt 1947, 1952; Nash and Duda 1951) as initiating stresses in birch dieback, but these were discounted by others (Clark 1961; Clark and Barter 1958; Redmond 1957).

Listed as contributing factors to top dieback in the Lake States are high water tables (Godman 1956, 1958; Jacobs 1960; Kessler 1963), heavy seed crops (Gross and Harnden 1968; Kessler 1969; Miller-Weeks 1985; Prey et al. 1984), frost (Miller-Weeks 1985), stand disturbance from logging, and *Nectria* cankers (Kessler 1967). Insect defoliation has been implicated in birch dieback in Maine, New Jersey, Wisconsin, Minnesota, and Newfoundland (Can. Dep. Fish. For. 1970; Miller-Weeks 1985; USDA 1972). La Bonte (1978) reported that mortality of yellow birch following insect defoliation in logged areas of Maine was much higher than in nonlogged areas. The bronze birch borer frequently is reported as associated with birch declines, but it does not appear to be an initiating factor (Conklin 1969; Quirke 1953). Hawbolt and Skolko (1948) suggested that the patterns of birch decline indicate biological agents rather than abiotic factors. Gottlieb and Berbee (1973) identified the apple mosaic virus in birch and suggested that at least some of the decline might be caused by the virus weakening the trees prior to attack by the bronze birch borer.

Trends

The most extensive dieback occurred from the 1930's through 1959 in the Northeast (Fig. 7). Since then, birch dieback has been sporadic in the northern part of the Lake States, Ontario, and Quebec and in small areas in the Northeastern United States.

Ash and Declines

Species, Range, and Use

White ash

White ash extends from Nova Scotia to eastern Minnesota, and southward to eastern Texas and northern Florida (Fig. 8). White ash is a major species in two cover types: white pine—northern red oak—white ash (20) and northern red oak—basswood—white ash (54). Major associates are

eastern white pine, northern red oak, white oak, sugar maple, red maple, yellow birch, American beech, black cherry, American basswood, eastern hemlock, American elm, and yellow-poplar.

The wood of white ash is used for many purposes, especially handles, where it is second only to hickory. It also is used for furniture, vehicle parts, railroad cars, sporting and athletic goods (nearly all wood baseball bats), paddles, snowshoes, and tennis racket frames.

Mortality and Decline

Major ash mortality events in the Eastern United States are summarized by year of occurrence in Table 5. For each event, the location, extent, and probable causes are given and references provided. Ash mortality events also are summarized by decade in Figure 9.

Distribution

The first reports of ash dieback and mortality were from Quebec in 1925 (Pomerleau 1953a) and the Northeastern United States in 1930 (Marshall 1930). Ross (1966) cited records of dieback on white and green ash from Iowa during this period. Silverborg and Brandt (1957) reported dieback and mortality of white ash in New York and New England beginning about 1940. Initial concerns were for roadside and hedgerow trees, but later reports noted extensive dieback in forest stands (Hibben and Silverborg 1978). The disease became increasingly severe during the 1950's (Table 5). Trees affected ranged from saplings to trees 24 inches in d.b.h. (Silverborg and Brandt 1957). A survey in New York during 1960-61 indicated that 31 percent of the ash trees were dying and that 6 percent were dead (Silverborg et al. 1963). A 1963 survey covering New Hampshire, Vermont, Massachusetts, Connecticut, New Jersey, and Pennsylvania found that 27.2 percent of the ash examined were dying and that 8.9 percent were dead (Table 5). Conditions were worse in Pennsylvania, where 33.2 percent of the trees were dying and 10.2 percent were dead (Tegethoff and Brandt 1964).

Since the mid-1960's, the disease has stabilized throughout the Northeast (Hibben and Silverborg 1978; Silverborg and Ross 1968), though severe mortality has continued in southern New York (Hibben and Silverborg 1978). Current reports of ash dieback and mortality have been from the Northeast and the Midwest.

Causes

The species is attacked by insects and diseases that may cause damage in localized areas. Of the insects, the oystershell scale, forest tent caterpillar, and the green fruitworm are considered the most important in forest stands (Fowells 1965; USDA For. Serv. 1985). Defoliation and twig mortality may be caused by leafspots (*Mycosphaerella effigurata* and *M. Fraxinicola*), anthracnose (*Gloeosporium areidum*), and a rust (*Puccinia peridermiospora*) (Hepting 1971).

Table 4.—Chronological summary of birch mortality in the Eastern United States

Year	Location	Severity/extent	Cause	Reference
1896	NY, MI, IL	Hundreds of ornamental white birch killed in many cities.	Attack by bronze birch borer.	Hall 1933
1939 to Mid-1950's	ME	70% of the yellow and white birch in the state died during this period. More than 3 billion board feet lost.	Not determined.	Nash and Duda 1951
1945 to Mid-1950's	NY, VT, and NH	Severe mortality and dieback, particularly at higher elevations. In the Adirondacks of NY, approximately 25% of paper birch died. Most of the paper birch at high elevations in NH died.	Not determined.	Baldwin 1949; Hansbrogh et al. 1950; New York Conserv. Dep. 1949
1954 to early 1960's	MI	Severe crown dieback on 68% of yellow birch in Upper Peninsula in 1959. Mortality up to 20% in stands previously thinned heavily.	High water tables, stand disturbance, possibly secondary organisms.	Godman 1958; Jacobs 1960; Kessler 1965
1970–71	NH	Mortality and dieback of yellow birch.	Defoliation by saddled prominent.	USDA For. Serv. 1982b
1979–80	WI	Approximately 100,000 cords of dead and dying white birch in west-central and northern parts of state.	Probably due to 1976–77 drought and defoliation by birch leafminer and birch skeletonizer. Attack by bronze birch borer may follow.	Renlund 1982a,b
1979–86	MN	Dieback widespread in central and northeastern areas. Over 1/3 of paper birch expected to be lost in central areas.	Drought, exposure, bronze birch borer, and defoliation by birch leafminer and birch skeletonizer.	Minn. Dep. Nat. Resour. 1981, 1982, 1985
1981	NH	Severe dieback of yellow birch.	Defoliation by saddled prominent.	USDA For. Serv. 1982b
1984–86	WI	Top dieback on approximately 56,000 acres in Florence, Forest, Rusk, and Sawyer Counties.	Heavy seed crops. Frost and insect defoliation.	Prey et al. 1985, 1986
1982–86	IA	Top dieback and some mortality of river birch on the Shimek State Forest and in the north-central part of the state.	Not determined. Most damage in areas with high water tables.	Sweets 1986
1983–84	ME	Mortality of paper birch in Hancock and Washington Counties.	Overmaturity, drought, and defoliation followed by attack by birch borer.	Maine For. Serv. 1983
1984–86	NY and VT	Dieback and mortality of yellow birch.	Not specified.	Miller-Weeks 1985; Rush 1986
1985	MI, MN, WI	Mortality of paper birch scattered in northern part of states.	Drought, defoliation, heavy seed crops.	Rush 1986

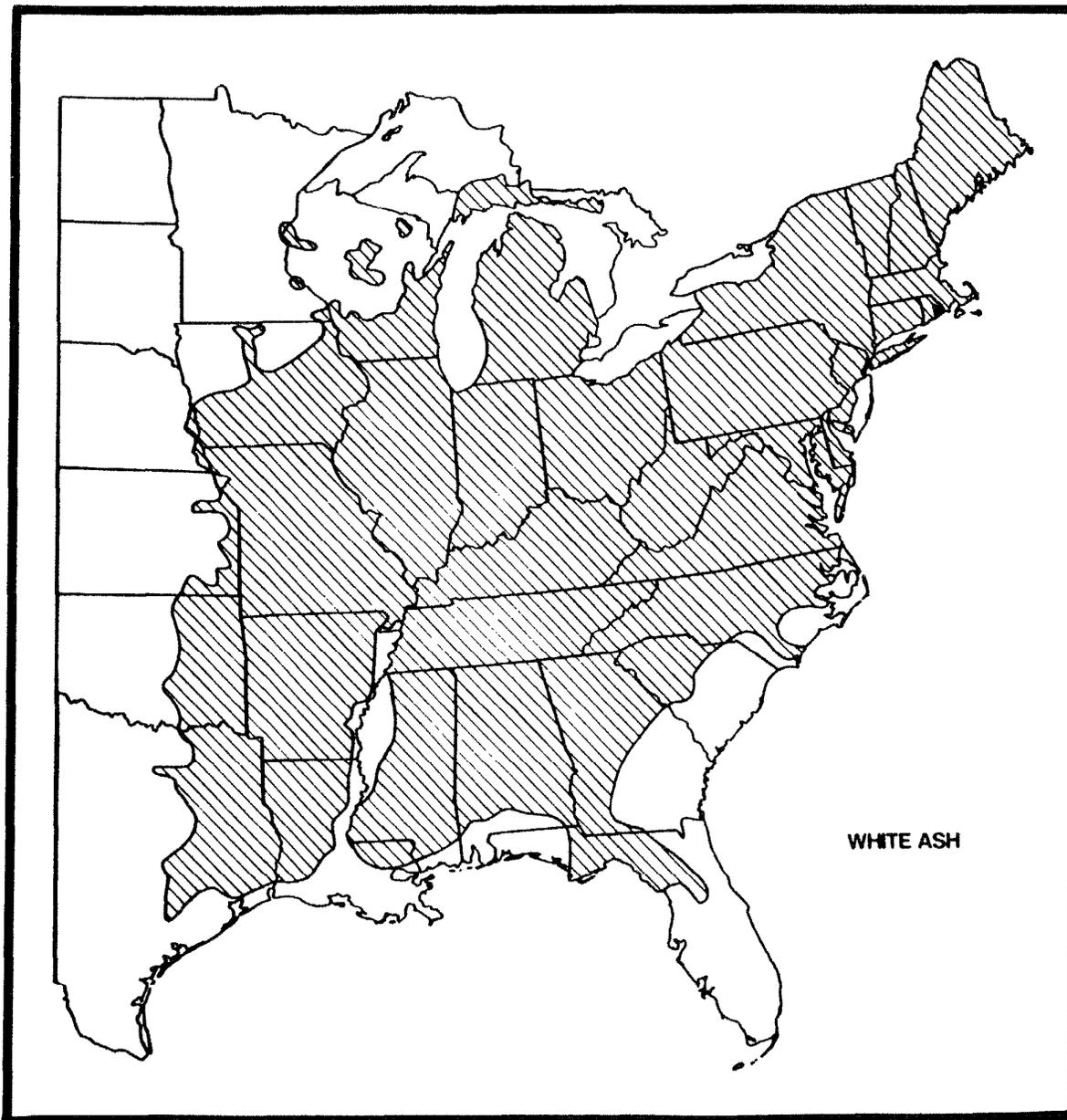


Figure 8.—Natural range of white ash (from Little 1971).

Ash dieback is the most important regional problem of white ash. Numerous agents, acting individually or in combination, have been proposed as possible causes of ash dieback. Drought has been the stress factor most consistently associated with ash dieback (Hibben and Silverberg 1978; Marshall 1930; Ross 1966; Tobiessen and Buchsbaum 1976). Periods of severe dieback between 1950 and the early 1960's coincided with periods of abnormally low rainfall (Ross 1966; Tobiessen and Buchsbaum 1976). However, disease incidence increased in New York's Hudson Valley throughout the 1970's even with adequate rainfall. Castello et al. (1985) reported other permanent-plot data from New York that indicate that regional drought may not be involved in all cases of ash dieback. Other abiotic

stresses that might play a role in ash dieback include climatic changes, edaphic factors, cambial damage by freezing, and localized air pollution (Hibben and Silverberg 1978; Houston 1981b; Matteoni and Sinclair 1985; Pomerleau 1953a; Ross 1966).

Canker fungi may contribute to crown dieback of stressed trees (Ross 1966; Silverberg and Brandt 1957). Numerous fungi have been isolated from cankers on trees with dieback, including *Cytophoma pruinosa* (Fr.) Hohn. *Fusicoccum* sp., *Coniothyrium* sp., and *Alternaria* sp. (Ross 1964; Silverberg and Brandt 1957). None of these fungi is considered a primary pathogen (Ross 1964, 1966).

Table 5.—Chronological summary of ash mortality in the Eastern United States

Year	Location	Severity/extent	Cause	Reference
1930	Northeastern U.S.	Dieback and mortality of numerous medium and large ash trees.	Possibly drought followed by canker fungi in the crown.	Marshall 1930
1930's	IA	Decline of green ash.	Causal agent not identified. Canker fungi found as secondary.	Rush 1966
Late 1930's to early 1940's	Southeastern NY	Dieback of white ash along roadsides and in hedgerows.	Not determined.	Hibben and Silverborg 1978
Early 1940's to Mid-1960's	Northeastern U.S. west of MI	Intensity of disease began rising in early 1940's, affecting forest trees.	Possibly drought followed by canker fungi. Mycoplasma-like organisms (ash yellows) possibly involved.	Ross 1966; Sinclair 1986
		64% of ash in 18 eastern NY counties dying or dead; 37% dead or dying throughout state.		Brandt 1961, 1963
		27% of white ash in NH, VT, MA, CT, NJ, and PA dying or dead.		Tegethoff and Brandt 1964
		Up to 50% mortality in some stands in southern MI.		State of Mich. 1964
1970	Northeastern U.S.	Chronic dieback and mortality at low levels during 1970's.	Drought followed by canker fungi. Ash yellows probably involved.	Hibben and Silverborg 1978; Sinclair 1986
1974-78	NY	Mortality and dieback increased in the Finger Lakes region.	Not specified (probably ash yellows).	Allison 1975; Hibben and Silverborg 1978
1978-86	OH	50% of ash volume in decline. Disease scattered statewide but concentrated in northern and western counties.	Not specified.	USDA For. Serv. 1980
1980-86	IA	Mortality and dieback increasing over past 5 years.	Probably ash yellows.	Rush 1986; Sinclair et al. 1987; Sweets 1984a, 1986
1982-86	IN	Dieback on approximately 50% of trees in northern third of state.	Drought and probably ash yellows.	Miller-Weeks 1985; Rush 1986; Sinclair et al. 1987
1983	PA	31-60% branch mortality in Sullivan Co. Symptoms recognized in eastern half of state.	Not determined but possibly ash yellows. Drought may contribute.	Pa. Dep. Environ. Resour. 1986; USDA For. Serv. 1984

Table 5 (Continued)

Year	Location	Severity/extent	Cause	Reference
1983–86	VT	Dieback and mortality of mature trees scattered throughout the state. Roadside trees are more severely affected than forest trees.	Possibly defoliation by forest tent caterpillar in some northern areas, unspecified for other areas.	Miller-Weeks 1985; Rush 1986
1983–86	NY	Up to 90% mortality in some stands. 218,000 acres with moderate to heavy mortality in Clinton, Jefferson, and St. Lawrence Counties. Ash dieback in 32 other counties.	Ash yellows.	M. Birmingham 1987 (pers. comm.); Rush 1986

Recent work has implicated a mycoplasma-like organism as a possible causal agent of ash dieback (Hibben and Wolanski 1971; Matteoni and Sinclair 1985). The disease, known as ash yellows, has been identified throughout the Northeast and recently in the Midwest (Reddy et al. 1934; Sinclair 1986; Sinclair et al. 1987). Sinclair et al. (1987) speculated that ash yellows has been involved in the ash declines reported over the past 40 years. Viruses also have been associated with ash dieback, though their role has not been determined (Castello et al. 1984; Hibben 1966; Lana and Agrios 1974).

Trends

Ash decline has been reported in approximately the same areas since the 1930's (Fig. 9).

Other Hardwoods and Declines

Included in the other hardwoods group are several species considered common or economically important for which extensive records of mortality are lacking. These include American beech, the poplars (quaking aspen, bigtooth aspen, and eastern cottonwood), black cherry, shagbark hickory, sweetgum, and yellow-poplar.

American Beech

Distribution and use

American beech extends from Cape Breton Island, Nova Scotia, west through Quebec, Ontario, and northern Michigan to eastern Wisconsin, south through southern Illinois, southeastern Missouri, northwestern Arkansas, and southeastern Oklahoma to eastern Texas, and east to northern Florida (Fig. 10). American beech is a major species in four forest cover types: sugar maple—beech—yellow birch (25); red spruce—sugar maple—beech (31); beech—sugar maple (60); and beech—southern magnolia (90).

It is associated with many other trees in eastern North America, including sugar maple, yellow birch, American basswood, black cherry, southern magnolia, eastern white pine, red spruce, several hickories, and oaks. Beech wood is used for charcoal, railroad ties, pulp, slack cooperage, veneer (mostly in the manufacture of crates, baskets, etc.), boxes, furniture, and planing mill products (especially flooring). Beech wood also ranks high for its fuel value.

Mortality and decline

It has been reported that drought, severe winters, and late frosts have caused mortality and dieback of beech in New York (Spaulding and Hansbrough 1935) and West Virginia (Tryon and True 1968). Dieback of beech was noted in southwestern New York beginning in the early 1950's (Buzzard and Risley 1970; SAF 1968). Initial reports considered drought and late frost as the primary causes (Buzzard and Risley 1970). Dieback was later shown to be caused by infestations of the saddled prominent. Outbreaks of the oystershell scale have caused extensive mortality of beech in Pennsylvania—more than 30 percent of the trees on 5,000 acres—and in Maine—10 to 15 percent of the trees on 60,000 acres (LaBonte 1978; Maine Dep. Conserv. 1979; USDA For. Serv. 1971).

Beech bark disease. Beech bark disease (Fig. 11) was first reported in Nova Scotia in 1911, though it probably was present there at least 20 years earlier (Hawboldt 1944). From Nova Scotia, the disease spread to Prince Edward Island and New Brunswick. In the Maritime Provinces, approximately 50 percent of the beech trees were lost during the initial outbreak of the disease in the 1930's (Ehrlich 1934; Magasi and Newell 1983). From there, the disease moved into New England and was well established in New Hampshire by 1950 and Vermont by 1960 (Shigo 1972). Losses of beech trees greater than 8 inches d.b.h. in New England from 1940 to 1977 ranged from 24 percent in New Hampshire to 50 percent in Maine (Miller-Weeks 1983). Beech scale (Fig. 11) was found in eastern New York in the mid-1930's, though the fungus was not discovered until



Figure 9.—Distribution of ash mortality during this century.

1950 (Buzzard and Risley 1970). Heavy *Nectria* infections are found on 30 percent of the trees in central New York (Rush 1986). In Pennsylvania, the scale was first recorded in 1958 in the northeastern part of the State, and the fungus was noted in 1969 (Towers et al. 1971). Mortality is reported to range from 10 to 15 percent in areas where the fungus is present (Rush 1986). Beech scale was first reported in West Virginia in 1981 on about 71,000 acres on the Monongahela National Forest. However, that infestation was thought to be at least 20 years old (Mielke et al. 1982). The infestation has expanded to 125,000 acres (Rush 1986). Scale infestations were first reported in Ohio in 1984 (Rush 1986).

The primary canker fungus is *Nectria coccinea* var. *faginata*

Lohman, Watson, and Ayers, but in West Virginia, *N. galligena* Bres. is associated with the disease (Houston and O'Brien 1983; Mielke and Houston 1983). The cankers damage the bark, cause dieback, and eventually kill the tree. Bark damaged by beech bark disease often is invaded by other organisms including *Hypoxylon* spp., wood-boring insects, and decay fungi (Houston and O'Brien 1983).

Abiotic factors, especially moisture, may influence infection by the fungus (Ehrlich 1934; Shigo 1972). Low temperatures may restrict the establishment of the scale insect (Hawboldt 1944), but the scale infests beech so readily that no predisposing environmental factors may be necessary (Houston et al. 1979).



Figure 10.—Natural range of American beech (from Little 1971).

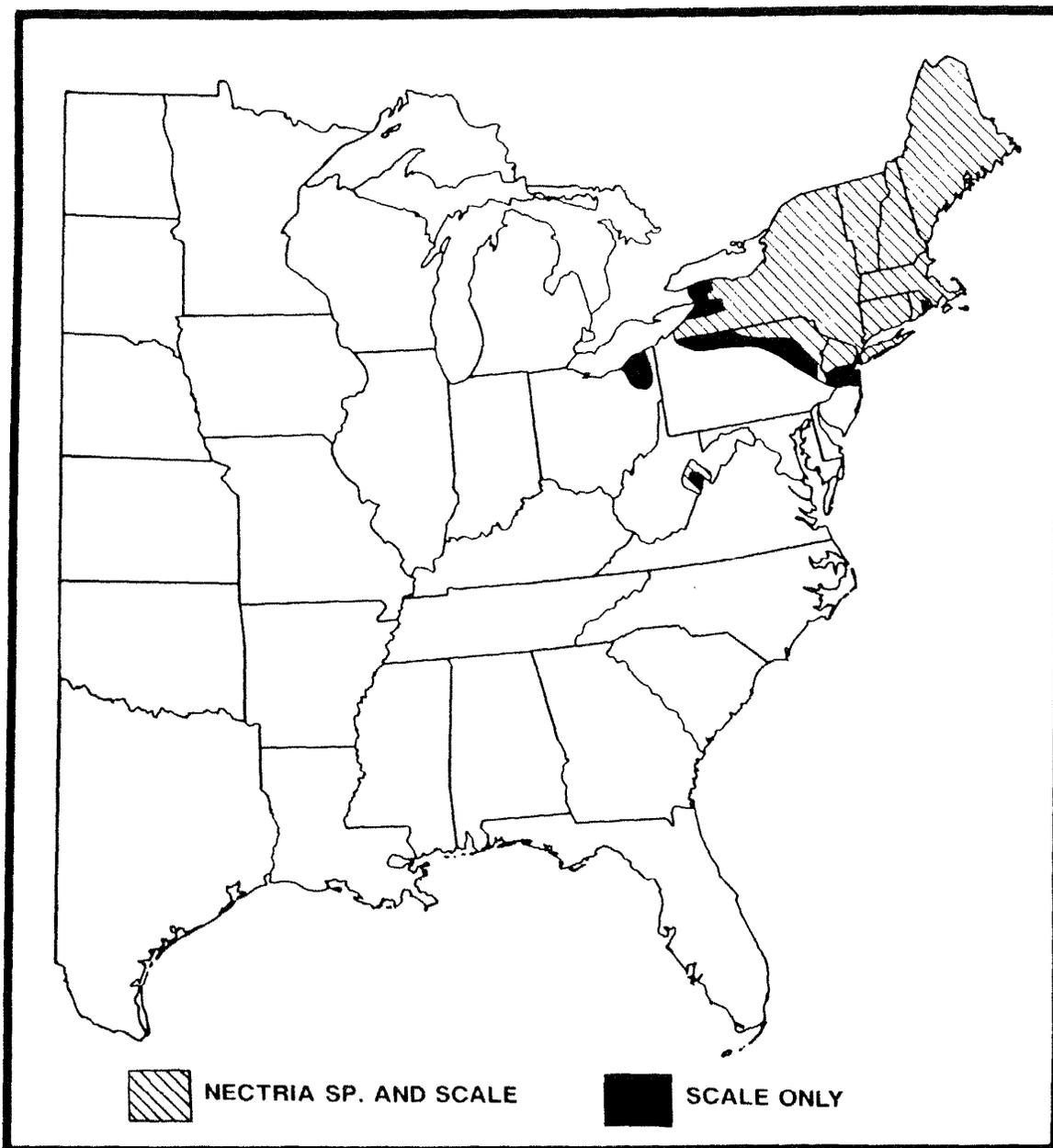


Figure 11.—Distribution of beech bark disease during this century (Houston and O'Brien 1983; Rush 1986).

Quaking Aspen

Distribution and use

The three major poplars of the 35 species of *Populus* found in the United States are quaking aspen, bigtooth aspen, and eastern cottonwood. All of these species are fast growing and short lived. Quaking aspen is the most widely distributed tree species in North America. It is found in the north from Labrador to Alaska, south from New Jersey to Nebraska, along the Rockies to Mexico, and in the Sierra Nevada (Fig. 12). It also grows in scattered areas in the Appalachian Mountains as far south as Kentucky. Quaking aspen is a major component of four cover types: jack pine—aspens (8); white spruce—aspens (10); aspen—paper birch (11); and aspen (16 and 217). Associated species in the East include balsam poplar, yellow birch, eastern white pine, red pine, white spruce, sugar maple, red maple, northern red oak, pin oak, bur oak, pin cherry, American elm, basswood, black ash, tamarack, and northern white-cedar. Aspen is used for high-grade book and magazine paper, veneer, furniture wood, and particle board.

Mortality and decline

Quaking aspen is a short-lived intolerant species (Brinkman and Roe 1975). It usually invades open areas after fires and clearcutting. Large stands are found in the Lake States and Canada. Aspen stands tend to break up within 30 years in the southern range, but may live to 100 years in northern Canada (Graham et al. 1963; Zehngraff 1949). The stands may break up within a 4-year period from the beginning of decline (Fralish 1972).

Weather may affect the growth of aspen, cause wounding, and predispose trees to other destructive organisms. Wind

breakage is most common in mature and overmature stands (Brinkman and Roe 1975).

At least 300 insects have been found on aspen (Davidson and Prentice 1968). However, only a few have caused mortality over large areas. One of the most common defoliators is the forest tent caterpillar (Batzer 1972). Outbreaks occur at intervals of about 10 years, defoliating areas sometimes in excess of 100,000 square miles. Nearly complete defoliation of aspen occurs for 2 to 3 years before the outbreak collapses. Defoliated trees refoliate within several weeks. Growth reduction is common but tree mortality is negligible (Duncan et al. 1956; Duncan and Hodson 1958; Kulman 1971). Occasionally, defoliation

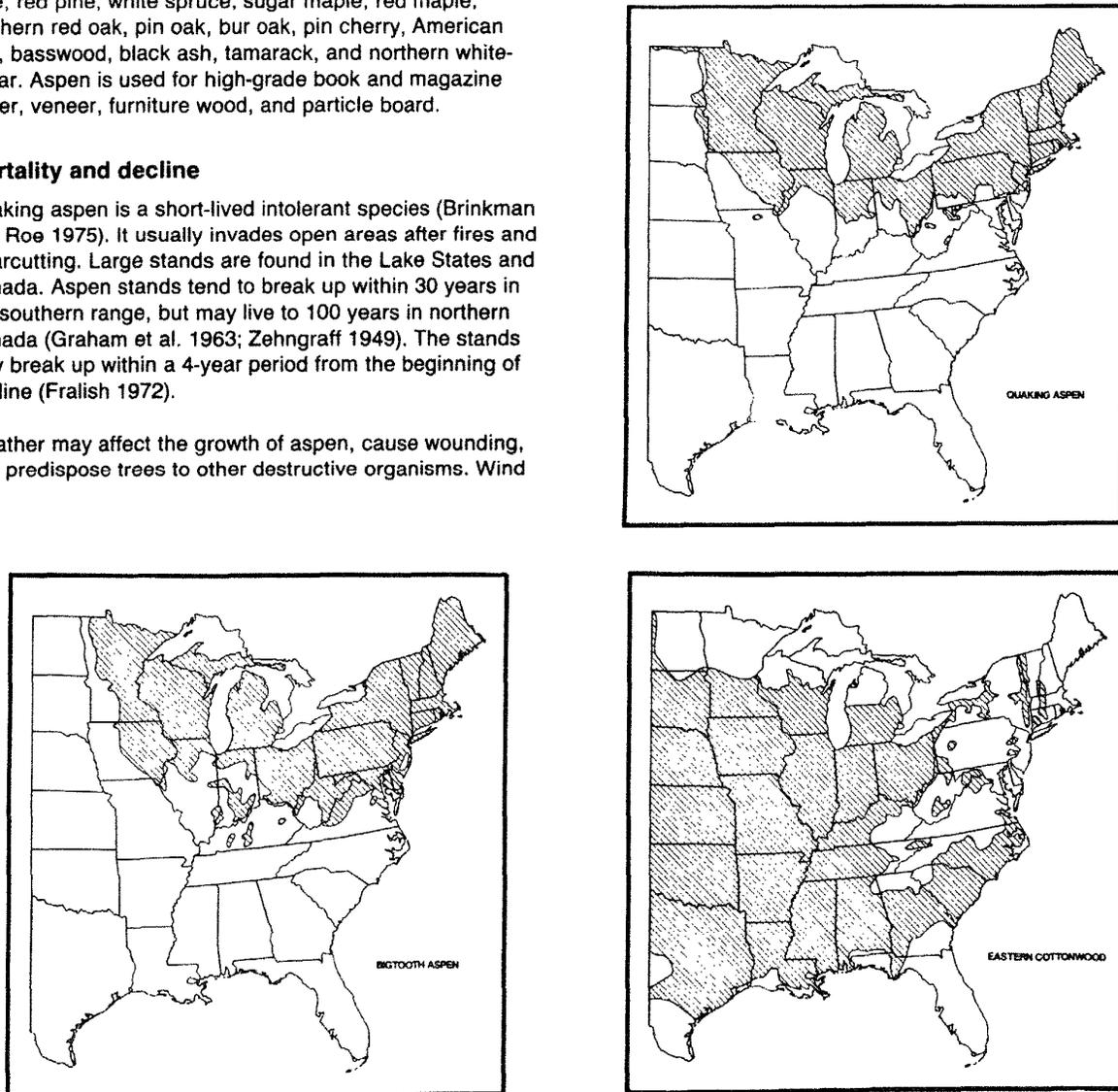


Figure 12.—Natural range of quaking aspen, bigtooth aspen, and eastern cottonwood (from Little 1971).

persists longer and mortality of up to 80 percent of the stand occurs (Duncan and Hodson 1958; Barter and Cameron 1955; Ghent 1958; Witter et al. 1975). Currently, about 15,000 acres in Minnesota's Cloquet Valley have been defoliated for more than 10 successive years. In several stands, more than half of the trees have died (personal communications, 1987; H. Kulman, Department of Entomology, Univ. of Minnesota; S. Munson, USDA Forest Service, St. Paul, MN).

Another major defoliator is the large aspen tortrix. Infestations covering more than 1 million acres have occurred in the Lake States and Canada, though tree mortality is not common (Batzer 1972).

Several woodborers attack aspen. The major economic loss in mature trees occurs from the poplar borer. In some areas, up to 64 percent of the trees were attacked (Graham et al. 1963). The overmature trees usually are the most severely attacked and succumb to subsequent wind damage.

There are many diseases that damage various parts of the tree in the United States (Anderson 1972). About 150 fungi have been recorded on aspen in Canada (Davidson and Prentice 1968). Among the more common diseases that weaken trees and may lead to their death are heart rot caused by *Fomes ignarius* (L. ex Fr.) Kickx, and hypoxylon canker caused by *Hypoxylon mammatum* (Wahl.) Miller. Although diseases are killing an estimated 300 million cubic feet of aspen each year, the dead trees are scattered several per acre throughout the stands and are not considered as part of a decline.

Bigtooth Aspen

Distribution and use

Bigtooth aspen is found from northeastern North Dakota through the Lake States, the Northeast, and along the Appalachians to Tennessee (Fig. 12). It is one of the major species in the aspen cover type (16). Common tree associates are quaking aspen, gray and paper birch, and red maple. Like quaking aspen, bigtooth aspen is used for high-grade paper, veneer, furniture wood, and particle board.

Mortality and decline

Reports of decline and mortality of bigtooth aspen are included with those of quaking aspen as aspen. Thus, the problems affecting bigtooth are similar to those for quaking aspen.

Eastern Cottonwood

Distribution and use

Eastern cottonwood grows along streams and bottomlands from southern Quebec to Montana, south to northern

Florida to west-central Texas (Fig. 12). It is rarely found in New England and the higher Appalachians. Eastern cottonwood is found mostly in the cottonwood cover type (63) in association with black ash, green ash, red maple, sweetgum, and many other lowland hardwoods. The uses of cottonwood are similar to those of quaking aspen.

Mortality and decline

Eastern cottonwood is highly susceptible to fire damage (Hepting 1971). Although the species is resistant to damage from flooding, annual major fluctuations in water levels appear to cause decline. Starkey et al. (1981) reported decline over 150 miles along the Beaver River and its tributaries in Oklahoma. More than 20 percent of trees were dead, mostly in the smaller size classes (less than 10 inches d.b.h.). The suspected cause was moisture stress by lowered water tables from irrigation wells. Drought-caused mortality of cottonwood was reported from the lower Mississippi Valley for the period 1952-56 (Johnson and Krinard 1965). Tree mortality was highest in stands where crown dieback was most severe.

Although many insects and diseases are found on eastern cottonwood, they rarely cause extensive stand decline (Morris et al. 1975; Hepting 1971).

Black Cherry

Distribution and use

Black cherry is found from Nova Scotia west to Minnesota, south to central Florida (Fig. 13). It also grows in central Arizona and extends southward through Mexico into Guatemala. Black cherry is a major species in two cover types: black cherry—sugar maple (28) and black cherry (29). It also is found with many other species in various parts of its range. Best growth is on deep, rich, and moist soils where it can be found in pure stands. More often it is mixed with species such as northern red oak, white ash, sugar maple, basswood, eastern white pine, and hemlock. Black cherry's hard wood, beautiful natural color, and finishing quality make it valuable for use as furniture and interior finish. Other uses include printer's blocks, piano actions, handles, woodenware, and scientific instruments.

Mortality and decline

Several insect defoliators are found on black cherry, but only the cherry scallop shell moth occurs in major outbreaks (Allison 1975; SAF 1976). The trees may be weakened, though extensive mortality has not been reported. Schultz and Allen (1977) reported tree mortality on poorly drained soils from the peach bark beetle after defoliation by the cherry scallop shell moth. Several diseases are associated with dieback of black cherry (Hepting 1971).

A cytospora canker caused by *Cytospora leucostoma* Fr. was associated with widespread dieback in Pennsylvania, West Virginia, and Maryland (Gross 1967; Pa. Dep. Environ. Resour. 1986).

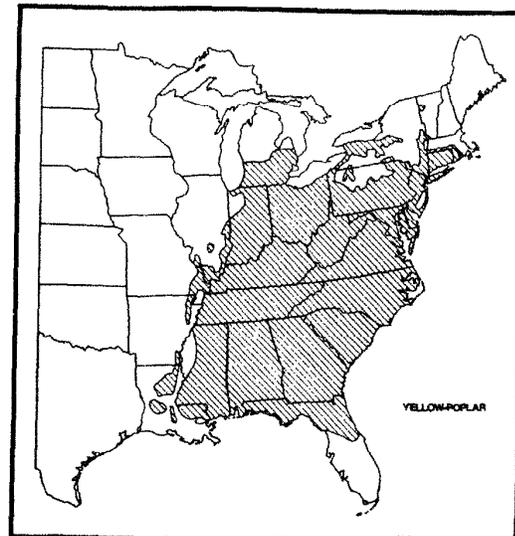


Figure 13.—Natural range of black cherry, shagbark hickory, sweetgum, and yellow-poplar (from Little 1971).

Shagbark Hickory

Distribution and use

Shagbark hickory extends from southern Maine westward through the southern tip of Quebec, southern Ontario, Michigan, Wisconsin, and southeastern Minnesota, southward through most of Iowa and southeastern Nebraska to eastern Texas, east to Georgia and the eastern Coastal States (Fig. 13). It is not a major component of any forest cover type, though it has many associates throughout its range. Some of the more common of these species are white oak, black oak, northern red oak, southern red oak, bur oak, chinkapin oak, chestnut oak, Shumard oak,

mockernut hickory, bitternut hickory, yellow-poplar, pitch pine, American elm, black cherry, sweetgum, sugar maple, and black walnut.

Shagbark hickory is one of the best commercial hickories. Its wood is extremely hard and strong which is important in the manufacture of tool handles (especially impact tools). Other uses are for ladders, furniture, sporting goods (skills, gymnastic bars), agricultural implements, and fuelwood for the smoking of meats.

Mortality and decline

Most reports of large-scale mortality in hickories have

implicated the hickory bark beetle. Reports of serious outbreaks began around 1900 and continued for about 15 years (Hopkins 1903, 1910, 1912; New York State Mus. 1915). Much of the natural range of hickory was affected from Wisconsin to Vermont and south to central Georgia (Hopkins 1912). Thousands of hickories were killed in southeastern and central New York in an outbreak that began in 1908 and peaked in 1912 (New York State Museum 1910, 1915). Beetle attacks were thought to be associated with a prolonged drought. Outbreaks of the beetle in the Southeast also have been associated with drought (USDA For. Serv. 1985). Mortality of hickories was considerable in western North Carolina in the mid-1950's after a beetle outbreak (USDA For. Serv. 1956). Gypsy moth defoliation on 17,000 acres in northern New Jersey resulted in a decline in 15 percent of the hickory trees and mortality in another 5 percent (USDA For. Serv. 1972). Quimby (1985) reported that mortality of hickory in Pennsylvania was 40 percent in scattered stands over 600,000 acres.

Starkey and Brown (1986) reported 13-percent mortality of hickory on plots originally established for monitoring oak decline in eight Southern States. No causes of the mortality were determined. Although the two-lined chestnut borer was commonly observed, recent drought was the suspected predisposing factor. Crown mortality of up to 80 percent was noted on hickories in central Pennsylvania following the 1983 drought (Pa. Dep. Environ. Resour. 1983). Dieback and mortality of hickories was reported in northwestern Indiana (USDA For. Serv. 1980) and in eastern Iowa (Sweets 1986). Secondary organisms such as *Armillaria* sp. and wood borers were found on the dying trees; however, the actual cause of the dieback is unknown (Sweets 1986; USDA For. Serv. 1980).

Gypsy moth defoliation in northern New Jersey resulted in 15 percent of the hickories rated as declining and 5-percent mortality on 17,000 acres (USDA For. Serv. 1972). In Pennsylvania, Quimby (1985) reported 40-percent hickory mortality in scattered stands after gypsy moth defoliation on 600,000 acres.

Sweetgum

Distribution and use

Sweetgum grows from southwestern Connecticut south to southern Illinois (Fig. 13). It also is found in sections of Mexico, Guatemala, Salvador, Honduras, and Nicaragua. Sweetgum is an important component of four forest cover types: northern red oak—mockernut hickory—sweetgum (56); pin oak—sweetgum (65); sweetgum—yellow-poplar (87); and sweetgum—Nuttall oak—willow oak (92). Associates include many of the oaks in the northern, central, and southern forest regions, southern yellow pines, and the southern bottomland species. Sweetgum is the most important species in the production of veneer, which is used extensively in furniture, panels, and a variety of containers. Other uses include railroad ties, mine props, and pulp.

Mortality and decline

Sweetgum blight is the most important cause of tree mortality. Dieback and decline of sweetgum was first noted at Fort Benning, Georgia, and on more than 100 square miles in east-central Alabama in 1948 (Garren 1949), and at University Park, Maryland, in 1949 (Miller and O'Brien 1951). Subsequent reports documented the spread of sweetgum blight (Garren 1954; Miller and Gravatt 1952; Young 1955; Young et al. 1954). Hepting (1955) found that 36 percent of sweetgum in the South had some form of dieback or decline. Toole and Broadfoot (1959) reported annual mortality of up to 13 percent in the Mississippi River floodplain. Other areas with heavy localized mortality included University Park, Maryland, Gainesville, Florida, and Middleboro, Kentucky (Hepting 1955).

The cause of sweetgum blight is not known. Numerous isolations from roots and shoots of blighted sweetgum failed to reveal a primary causal pathogen (Garren 1949, 1954; Miller and O'Brien 1951; Berry 1955). Toole and Broadfoot (1959) concluded that sweetgum blight in the Mississippi Delta region was a reaction to low soil-moisture content. Studies in Maryland (Fowler 1963) failed to confirm this hypothesis. However, it is generally considered that sweetgum blight was the result of prolonged drought in the Southern States during the early 1950's (Hepting 1971; Toole 1959).

Yellow-Poplar

Distribution and use

Yellow-poplar is found throughout the Eastern United States from southern New England west to Michigan, south to Louisiana and central Florida (Fig. 13). Yellow-poplar reaches its largest size in the lower Ohio River Valley and on the mountain slopes of North Carolina, Tennessee, Kentucky, and West Virginia.

Yellow-poplar is an important species in four forest cover types: yellow-poplar (57); yellow-poplar—hemlock (58); yellow-poplar—white oak—northern red oak (59); and sweetgum—yellow-poplar (87). Tree species associated with yellow-poplar vary according to locality and site. Oaks, red maple, baldcypress, the gums, and occasionally loblolly pine are found with yellow-poplar on bottomlands and better drained soils of the Coastal Plains. In the Piedmont, associated species include those found on bottomlands and American elm, shortleaf pine, and hickories. Yellow-poplar grows with black locust, white oak, black oak, eastern white pine, eastern hemlock, and black walnut at the lower elevations of the Appalachians. At higher elevations, associates include northern red oak, white ash, black cherry, cucumbertree, buckeye, American beech, sugar maple and yellow birch. Yellow birch may be found along with black oak, sugar maple, blackgum, and dogwood.

Yellow-poplar timber is one of the most valuable. It is used for many purposes including furniture, cabinet work, piano

cases, musical instruments, and interior finish veneer. Other uses are television consoles, radio and phonograph cabinets, fixtures, and berry boxes.

Mortality and decline

There are no reports of serious regionwide declines of yellow-poplar. The species is considered to be unusually pest free (Burns 1970; Hepting 1971). The yellow-poplar weevil is the most important defoliator of yellow-poplar (Burns 1971). Large outbreaks of this insect caused various amounts of damage in the Appalachian Mountains during the 1960's (Burns 1971). The tuliptree scale occasionally kills small trees and may limit natural regeneration of yellow-poplar in some areas (Donley and Burns 1971). It has been reported that cankers caused by *Fusarium solani* (Mart.) Appel & Wr., *Botryoshaeria ribis*, and *Myxosporium* spp. have resulted in dieback and occasional mortality in several areas in the South (Arnett and Witcher 1972; Hepting 1971; Johnson et al. 1957; Toole and Huckenpahler 1954). Attack by the yellow-poplar weevil and canker fungi may be initiated or exacerbated by drought stress (Burns 1970; Hepting 1971; Toole and Huckenpahler 1954).

Eastern White Pine and Declines

Eastern white pine is included here because it is common throughout the Eastern United States and frequently is associated with various hardwoods. If regional pollution were likely to cause significant mortality among hardwoods, eastern white pine also would show damage at the same time. This species is one of the few for which damage from atmospheric pollution has been documented.

Distribution and Use

White pine is found across southern Canada from southeastern Manitoba to Newfoundland, and in the United States from Minnesota and northeastern Iowa to the Atlantic Coast, southward along the Appalachians into northern Georgia (Fig. 14).

Eastern white pine is a major component of four cover types: white pine—northern red oak—white ash (20); white pine (21); white pine—hemlock (22); and white pine—chestnut oak (51). White pine also is found with pitch pine, jack pine, shortleaf pine, sweet birch, bigtooth aspen, quaking aspen, black cherry, black oak, white oak, hickories, red maple, sugar maple, beech, paper birch, red spruce, balsam fir, and northern white-cedar. Eastern white pine has been the most important conifer in the Northeast and Lake States since colonial times. It is one of the principal species used for boxes and crates because of its light weight, good color, and lack of objectionable odor and taste. Other uses include millwork (sash and doors), signs, caskets, and matches, and in building construction.

Mortality and Decline

There are many insects and diseases associated with eastern white pine (Garrett 1986; Hepting 1971). Not all of these pests are important in terms of mortality, so only the major insects and diseases are discussed here. There are no reports of tree mortality over large areas of naturally generated forest. Foliage discoloration is reported frequently, but regional mortality or even growth declines have not occurred.

Insects

The white pine weevil is the major nemesis of eastern white pine (Houseweart and Knight 1986; USDA For. Serv. 1985). This insect rarely kills trees but causes severe losses in volume and lumber quality, and loss in rotation time (Houseweart and Knight 1986). Mortality of white pine due to gypsy moth defoliation has been documented in the Northeast, but merchantable stands of timber usually are not affected (House 1960). For information on other insects of white pine see USDA For. Serv. (1985).

Diseases

White pine blister rust, caused by *Cronartium ribicola* Fisch., is an introduced disease that has caused severe losses of five-needled pines in some areas, particularly in the Western United States (Hodges 1986). Losses of eastern white pine generally have been less serious than losses of western pines because many trees grew on in low-rust hazard areas (Garrett 1986). White pine root decline has received increasing attention over the past few years. The disease is caused by *Verticicladiella procera* and currently is found in nine states (Anderson and Alexander 1979). It is most damaging in plantations, but there are reports of damage to forest stands (Anderson and Alexander 1979). Root rot by *Heterobasidion (Fomes) annosum* and trunk rot by *Phellinus (Fomes) pini* also cause losses of white pine (Hepting 1971). For information on other diseases of white pine see Hepting (1971) and Hirt (1959).

White pine generally is considered highly sensitive to and easily damaged by air pollution (Gerhold 1977; Skelly and Johnston 1979; Smith 1981). During the first half of this century, there were many reports on tip blights or chlorosis not associated with known biotic agents of white pine needles on trees removed from localized pollution sources (Baldwin 1954; Clinton 1907; Rane 1908; Deuber 1931; Faul 1919, 1922a,b; Pierson 1955; Swingle 1944; Toole 1949; Walker 1946). In the 1960's, ozone was shown in laboratory studies to induce the symptoms of white pine blight (Berry and Ripperton 1963; Costonis and Sinclair 1969; Dochinger and Seliskar 1970). Suspected ozone injury covering large forest areas is reported annually from various parts of the country, particularly the Southeast. Mosher (1977) reported chlorosis and death of about 10 percent of white pine near Marquette, Michigan. However, there has been no association of ozone injury with widespread tree mortality. Severe mortality of white pine due to localized air pollution has been reported. Mortality resulting from point-source pollution is discussed elsewhere in this report.



Figure 14.—Natural range of eastern white pine (from Little 1971).

CAUSES OF FOREST DECLINE AND MORTALITY

Sinclair (1964, 1966) concluded that many factors generally operate in concert to cause tree mortality. He indicated that adverse weather usually provides the initial stress; this is followed by attacks by insects or diseases that eventually kill the tree. The decline sequence usually is a reduction of tree growth followed by dieback and death. Recovery depends on when the stresses are removed and how far the damage has progressed. Generally, some predisposing factors occur that allow biological stresses to develop. These factors can include the maturation of trees or a silvicultural factor that favors the development or intensifies the consequences of stress. Often ignored is the fact that the mere presence or abundance of a susceptible host is sufficient to predispose the tree to stress. When stress is exerted, the tree reacts by first slowing its growth and then shedding parts. The tree dies if the stress is sufficiently severe or prolonged. A tree may recover when the stresses are removed provided that the primary damage is not too severe, or the tree is not killed by secondary agents.

Normally, most tree mortality occurs during the seedling or sapling stages from site and competition factors. Tens of thousands of seedlings may germinate per acre, but only a few hundred reach 5 inches d.b.h. Thereafter, mortality rates typically decline and are low—barring a natural disaster—until the trees approach overmaturity. The primary concern is the premature death of trees from other than natural causes. Our primary focus in this review is the mortality of trees during the maturation period, that is, after they have reached 5 inches d.b.h.

Predisposing Stress Factors

Overmaturity

Aging of trees is considered a predisposing factor for forest growth decline and mortality. Rarely can the death of a tree be attributed directly to old age, yet many insects and fungi favor older trees, and eventually kill them. Toumey and Korstian (1947) wrote:

The tissues are continually undergoing change and are continually being repaired; they are for the greater part renewed annually. Although living and dead tissues form in a tree a sort of unity which often endures for centuries, in the course of time the living cells become reduced in number and finally die; the tree decays and disappears.

Spurr and Barnes (1980) explained that senescence occurs because of increasing distance between the terminal feeding roots and the top of the tree. As the distance increases, growth slows and the tree eventually reaches a point where it is barely able to stay alive without further growth. Toumey and Korstian (1947) concluded that the death of the tree results from damage from insects, diseases, weather, or other forms of stress that occurs at a

point when the tree is unable to repair the damage.

Data on maximum age for most tree species are based on the oldest tree found in the forest (Table 6). However, one should be aware that the survival of a tree depends on the statistical probability of a killing factor finding it. The maximum age of an individual tree is not likely to be the same as when a stand disintegrates or when one half of the trees in a stand die. Sinclair (1966) observed that the useful lifespan of trees is much shorter than the total or potential lifespan. For most species we do not have a good measure of when a tree reaches overmaturity and when normal mortality should be expected. Without knowing normal life expectancy, it is difficult to determine when tree mortality is premature. Toumey and Korstian (1947) listed the following factors that affect the longevity of a tree:

1. Species: Some tree species live longer than others. For example, oaks and sugar maple generally live longer than most other species in the Eastern United States.

Table 6.—Estimated maximum longevity and suggested rotation age of eastern hardwoods

Tree species	Maximum age	Rotation age
		Years
Oak		
White	600	130–160
Northern red		80–100 ^a
Chestnut		100
Scarlet		
Black	200	100
Bur	300	
Maple		
Sugar	400	150
Red	150	80
Birch		
Yellow	200 +	
Paper	200	75
Poplar		
Eastern cottonwood	120(?)	45
Bigtooth aspen		45
Quaking aspen		30–100
Other		
American beech	366	150 ^a
White ash		60 ^a
Shagbark hickory	300	100–150 ^a
Yellow-poplar	200 +	
Black cherry	250	100
Sweetgum	150	75
E. white pine	450	200

^a Height growth slowed significantly.

2. **Durability:** Trees with more durable wood, such as oak, live longer.
3. **Strength:** Brittle trees are more readily damaged by wind and ice.
4. **Regeneration:** Rapidly growing trees recover from injury faster.
5. **Site:** Trees growing on a good site survive longer.
6. **Protection:** Trees in protected environments live longer. Protection may be provided by other trees, topographic relief, or other vegetation.
7. **Origin:** Trees that originate from seed tend to live longer than trees that arise from coppice.

Stand Senescence

A forest stand may be thought of as having a stand age, that is, a period of time when it exists as a stand, and then mortality of trees becomes sufficiently high to change the stand characteristic to a different type of stand. The term "pathological rotation" often is used to denote the age of the stand when growth has slowed to the point where it falls behind mortality in the stand. All trees reach a point during their life at which growth is maximized. In other words, all attributes of tree and stand growth exhibit both increasing and decreasing growth rates as a part of normal development. Thus, while it is true that all declines are characterized by reductions in growth, not all reductions in growth are evidence of decline (Hyink and Zedaker 1987).

Hardwoods frequently tend to grow in a mixture of several species. For example, northern hardwood stands have sugar maple, American beech, yellow birch, paper birch, white ash, and red maple as major components (Leak et al. 1969; Tubbs 1977). Stand decline may become apparent when the shorter lived species reach maturity and begin to die. Stress factors such as drought and defoliation may hasten the decline of a tree species in a stand without affecting other species (McGee 1984).

The causes of pathological rotation in stands probably are similar to those for individual trees. Silvicultural factors may hasten the mortality rate of trees in the stand. The nearness of one tree to another of the same species increases the chances of a pest finding them. As the trees begin to die, openings are created that expose edge trees to new stresses such as wind, sun, and increasing soil temperature. The mass destruction of one species may favor the development of another. Logging that favors stump regeneration may weaken a stand over time by preserving a disease organism in the stump and passing it along to the new tree.

The more common measure of stand maturity is that used by most foresters: when the value of growth rate or volume in the stand drops below protection and management costs, the stand is considered ready for harvest. Here, tree condition, insect damage, and presence of disease are considered together.

Fire

The damage immediately after fire is obvious. Less pronounced is fire damage decades later when many of the wounds are overgrown. These trees may have shortened lives because of the secondary organisms that entered through fire scars. Little is known of the effects of fire prevention. Some forest types are managed by recurring fires. The exclusion of fire may result in the conversion of stands from dominance by one species over another less adapted to the site or to damage from insects and diseases. As a result, changes in fire protection over the last century may have created conditions more favorable to the development and/or detection of tree decline.

Seed Production

Heavy seed production in some hardwood species may result in branch dieback. For example, heavy seed production of birch was associated with birch dieback in the late 1960's (Gross 1972).

Stress and Mortality Factors

Many insects and diseases can kill young and vigorous trees. For example, repeated severe defoliation of oak or maple was mentioned frequently as a cause of tree decline and mortality. One insect species may cause damage that favors another insect species' development, which then causes the death of trees. An example is gypsy moth defoliation that favors later development of the twolined chestnut borer (which then girdles the oak) and/or root diseases that eventually lead to the demise of the trees (Houston 1981a). There are introduced diseases that now are eliminating the host species from commercial consideration. Examples are the chestnut blight that has nearly eliminated the American chestnut and Dutch elm disease, which is eliminating the American elm. Weather extremes seldom kill trees outright but frequently damage trees to the extent that tree mortality continues for years following the stress event.

Insects

Primary insects attack apparently healthy trees, reduce tree growth or vigor, and set the stage for forest declines (Allen 1987). Usually, primary pests are not responsible for tree mortality; rather, they predispose trees to attack by lethal secondary insects and fungi. The most spectacular are the defoliators, which can change the appearance of the forest in a short time. Thousands of square miles of forest have been defoliated by the gypsy moth and forest tent caterpillar. Other major defoliators include the redhumped oakworm, orangehumped mapleworm, saddled prominent, cankerworms, post oak locust, walkingstick, Bruce spanworm, and large aspen tortrix. Forest declines have been attributed to these insects when defoliation occurs for several successive years.

Another group of defoliators attacks the buds and newly developing leaves. Among these major forest pests are the leafrollers and leaf tiers. Again, repeated damage has caused branch dieback and tree mortality.

A large number of insects mine and skeletonize leaves. Among the major insects are the birch leafminer, birch skeletonizer, oak skeletonizer, cherry scallop shell moth, and locust leafminer.

Insect defoliators are reported to be the major causal agents of many hardwood declines (Houston 1986; Wolfe 1985). Severe defoliation depletes food reserves, predisposing trees to secondary insects and diseases (Haack 1985; Parker 1981; Wargo and Houston 1974). Defoliation may cause changes in carbohydrate storage in a tree and consequently increase susceptibility to root diseases (Wargo 1972). Secondary organisms usually cause the death of defoliated trees. A single defoliation may cause growth reduction, but trees may die when several defoliations occur in a single year from different agents (Kulman 1971). Gypsy moth defoliation was the suspected cause of oak mortality in early outbreaks in Massachusetts during 1912-25 (Minott and Guild 1925).

Wood borers and bark beetles frequently are secondary invaders of weakened trees, causing their eventual death. Two borers associated with hardwood declines are the bronze birch borer and the twolined chestnut borer. During an outbreak, when population densities are high, these insects can kill apparently healthy trees.

Insects frequently are vectors of diseases. Bark beetles are vectors of Dutch elm disease and nitidulids of oak wilt. Elm yellows and probably ash yellows are vectored by leafhoppers. Beech bark disease involves an interaction between a scale insect and a canker fungus. In discussing the interaction of insects, trees, and air pollutants, Hain (1987) pointed out that the stress of a pollutant or even the presence of a pollutant may favor a build up of insect populations and consequent damage to the trees.

Diseases

Chestnut blight, Dutch elm disease, elm yellows, and oak wilt are examples of destructive diseases where the fungus is the primary causal agent. However, in most declines, pathogens are considered secondary agents that become destructive only after the trees are weakened by another stress agent.

One of the most common of these secondary agents is the root disease caused by *Armillaria sp.* The fungus rhizomorphs are common in forest soils and quickly colonize the roots of stressed trees (Wargo and Shaw 1985). Numerous canker fungi have been identified as contributors to crown dieback in declines, but most of these are nonaggressive bark saprophytes that have invaded stressed trees or previously killed tissue.

Difficulty in isolating or identifying pathogens has led to some confusion in determining the etiology of some

declines and diebacks. For example, ash yellows has only recently been considered a major factor in ash dieback. Matteoni and Sinclair (1985) presented evidence for a mycoplasma-like organism as the causal agent. For many years butternut canker was thought to be caused by *Melanconis juglandis* (Graves 1923). Recent isolation and inoculation studies show that *Sirococcus clavignenti-juglandacearum* is the actual cause of the disease (Nair et al. 1978). Anderson and Lamadéleine (1978, unpublished) reported that most of the butternuts in the Carolinas were eliminated by this disease. More recently, the forest inventory survey in that area found that 77 percent of the butternut in North Carolina and Virginia died from *Sirococcus* (personal communication, R. Anderson, USDA Forest Service, Asheville, N.C.).

Sapstreak disease. A wilt disease caused by *Geratocystis coerulescens* (Munch) Bakshi, sapstreak disease has caused mortality of sugar maple in North Carolina (Hepting 1944; Southeast For. Exp. Stn. 1959); and the Lake States (Kessler 1972; Kessler and Anderson 1960; Ohman and Kessler 1963). Sapstreak recently has been found in the Northeast, though it is not yet associated with mortality over extensive areas (Beil and Kessler 1979, Houston and Fisher 1964).

American chestnut blight

Caused by *Endothia parasitica* (Murr.) P.J. and H.W. Anderson, American chestnut blight is responsible for the nearly complete destruction of American chestnut (Griffin and Elkins 1986). This tree once was the most abundant hardwood in the Eastern United States, where it often was the dominant component in hardwood stands. The American chestnut also was considered of high economic value. Once the disease arrived in the United States, it spread rapidly and decimated the species to a degree that may not be paralleled in history. Today, large trees above 10 inches d.b.h. are rare. The long-term ecological consequences of losing a major component of forest stands and the resulting change to another dominant species are not known.

Dutch elm disease

Although the American elm is not considered a prime timber species, it does grow over a wide area in the Eastern United States and is considered one of the most valuable shade trees (Fowells 1965). Its role in the forest and in the landscape is diminishing rapidly because of the widespread mortality from Dutch elm disease (Stipes and Campana 1981; USDA For. Serv. 1977). This is an example of a disease complex where the host tree species is killed by a fungus *Ceratocystis ulmi* (*Ophiostoma ulmi* (Buism.) Nannf.) carried by two species of bark beetles.

Weather

Sinclair (1964) concluded that most of the declines in the 1950's were initiated by weather stresses, and then exacerbated by insects and diseases to the point of death. Many abiotic factors have been implicated in dieback and

decline. These include drought, moisture excesses, frost, exposure to excessive light, high temperatures, mechanical damage, edaphic factors, and changes in climate. Kim and Siccama (1987) found that the annual increment of beech, sugar maple, yellow birch and white ash was affected more by wide deviations in temperature and precipitation from normal seasonal averages than by overall annual deviations. Weather-related stress factors cause trees to become susceptible to primary and secondary organisms, but can kill them in extreme situations. Hepting (1971) discussed diebacks and declines of many hardwoods and related many of them to stress from weather and insect defoliation. Hepting (1963) suggested that climatic changes may effect the incidence and the severity of pests and pathogens. Normally weak pests or pathogens may become aggressive when climatic changes favor them while adversely affecting the tree.

Drought

Drought probably is the most frequently mentioned stress agent among tree declines. However, difficulties are encountered in quantifying drought stress. Diaz (1983) used the Palmer Hydrologic Index (PHI) (Palmer 1965) to determine drought conditions. The Index is calculated from average monthly temperatures and precipitation to balance available water against evapotranspiration. Comparison of monthly drought index charts suggests no relationship between Palmer Drought Index and mortality events. Several regional declines of ash, birch, and maple seem out of phase with the PHI. This might suggest a lag time between drought conditions and the onset of decline. Further evaluation of temporal and spatial patterns of the PHI/decline relationship seems warranted. However, the Palmer Index was designed primarily for agricultural crops, and may not be satisfactory for predicting effects on forest trees.

Wind

During hurricanes and tornadoes, damage to branches, boles, and roots may allow entry of disease-causing organisms. Teillon et al. (1984) reported red maple and beech mortality due to a windstorm in 1982. Strong gusts of wind during the passage of weather fronts are common and may cause similar damage. Wind damage typically is not considered a decline and was not included in the literature review. However, Loucks (1983) showed that wind can be a significant factor in the successional development of a forest. Further, decline leading to deteriorating root systems could result in trees that are more susceptible to windthrow.

Flood

Flooding following heavy rains can cause damage when roots are submerged during a critical period of tree growth. Rapidly fluctuating water tables may not allow sufficient time for many trees to adjust their root systems. Manmade impoundments have caused extensive tree mortality in some areas by flooding.

Snow and ice

Snow and ice damage is common in some areas. Breakage of branches and small trees may create wounds for the entry of pathogens.

Cold

Unusually low temperatures can damage and kill certain tree species. The most common damage occurs from frosts in late spring. On several occasions, large areas of oak mortality were reported from damage to buds and young foliage in the spring (Beal 1926). Unusually low winter temperatures may cause crown dieback and tree mortality. Cope (1935) reported that previously vigorous sugar maples and beeches failed to leaf out after a severe winter freeze in the western Adirondacks in 1934. Maple and white ash regeneration was killed to the snow line in clearcut areas. Many apples and cherries died along Lake Ontario. Spaulding (1946) and Nash (1943) reported similar declines in northern New Hampshire, Vermont, and adjoining Maine after temperatures dipped below -40°F . Teillon et al. (1981, 1985) reported sugar maple decline following severe winters.

Dieback, Decline, and Mortality

In the literature, the terms dieback, decline, and stand mortality often are used interchangeably. Sinclair et al. (1987) stated that the term decline refers to a progressive loss of vigor and health, not to a specific disease or disorder. Manion (1981) reserved the term decline for diseases caused by the interaction of a number of interchangeable, abiotic and biotic factors to produce a gradual, general deterioration, often ending in the death of the tree. Houston (1986) interpreted dieback, decline, and mortality as a sequence of tree responses increasing in severity with the severity of environmental stress. Mueller-Dombois et al. (1983) suggested that where old age of trees is the primary cause of tree mortality (that is, where only the older trees are affected without symptoms on young trees), use of the term decline to indicate a disease may be inappropriate.

Teskey et al. (1987) emphasized the importance of weather factors in tree declines and concluded that water and temperature stress alone can impose fundamental limits on forest productivity. They discussed examples where trees may respond less to pollutants when they are under temperature stress.

Atmospheric Deposition and the Forest

Forests act as filters to remove pollutants from the atmosphere, yet they also emit natural hydrocarbons to the atmosphere. These contribute to photochemical oxidant

levels on a regional scale (Smith 1981). Gas exchange and the assimilation of a variety of substances from the atmosphere are normal components of plant metabolism and growth. Chemical pollutants in the air may benefit or have no effect on the growth of trees. But an excessive amount of such compounds may interfere with physiological or biochemical processes, or cause physical damage to trees and plants.

Man has been aware of damage from atmospheric pollutants for several hundred years (Cowling 1982). Traditionally, atmospheric pollution has been separated into point-source (local) and regional (long-range transport) pollutants. Pollution damage associated with point sources such as smelters and with other industrial activities also have been studied for many years (Heck et al. 1986). Recent detailed reviews of atmospheric pollutant effects on North American forests (McLaughlin 1985; Linzon 1986) conclude that ozone currently is the most prevalent phytotoxic pollutant on a regional scale. Nitrogen oxides (NO_x) have caused damage to trees near point sources, though such occurrences are rare. Current interest in NO_x stems from their regional contribution as precursor pollutants in the formation of ozone and acid rain. Sulfur dioxide, which has damaged trees more than 100 kilometers from point sources, also is an important precursor of acid rain on a regional scale. Hydrogen fluoride is toxic to trees

locally (up to 12 kilometers downwind from point sources).

While there is no conclusive evidence that acid precipitation has a widespread impact on the productivity of forests in eastern North America (Woodman 1987), the adverse effects of ozone on the growth and productivity of crop vegetation have been demonstrated (Heck et al. 1986) on a regional level. This suggests that similar regional impacts on forest vegetation are likely.

It has been hypothesized that atmospheric deposition is involved in forest dieback, decline, and mortality by contributing to the gradual, general deterioration of trees and, therefore, stands. The mechanisms by which this process is thought to occur are summarized in Figure 15.

Although the historical data on tree mortality vary in quality and duration by species, there is sufficient information with which we can analyze the spatial pattern of mortality over time. If this pattern does change over time, we can then formulate hypotheses as to why this change occurs. Historical data on atmospheric deposition are not readily available to compare with historical data on mortality. However, we can compare the spatial patterns of mortality in the post-1980 period with data on pollutant deposition/concentration for the same period.

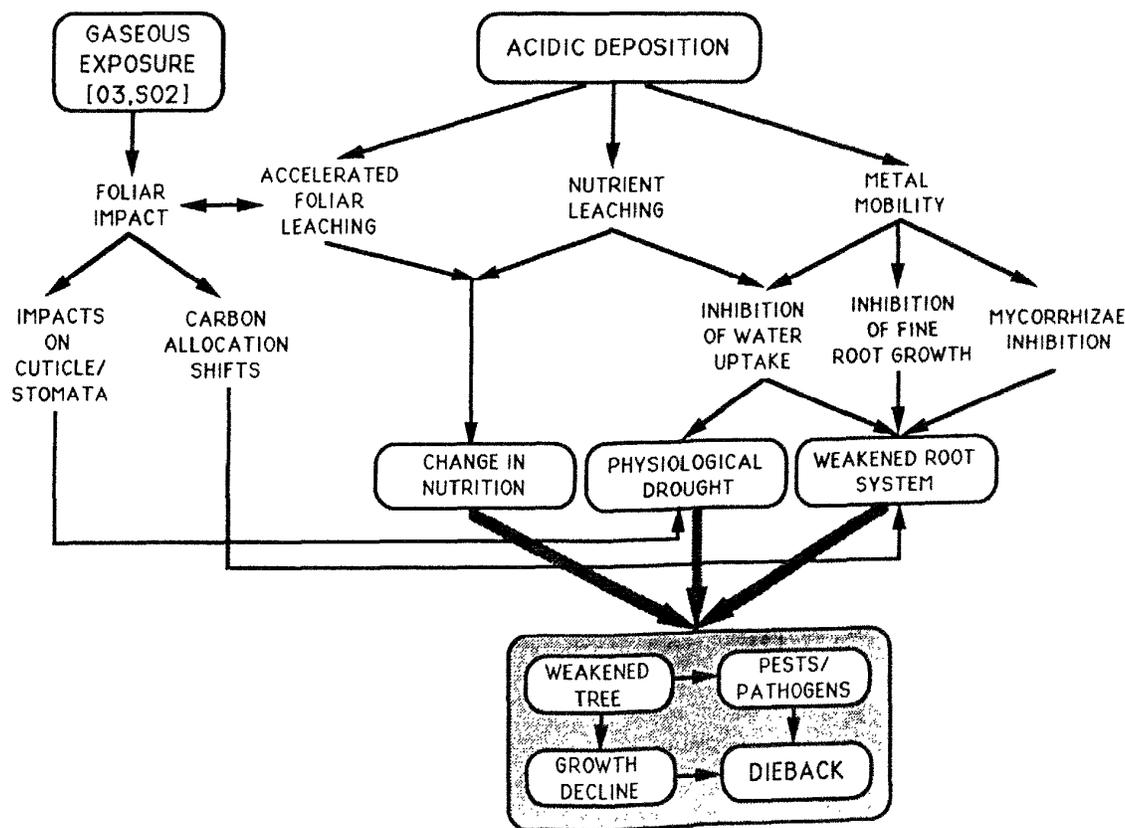


Figure 15.—Mechanisms by which atmospheric deposition is thought to contribute to forest dieback, decline, and mortality.

Point-Source Pollution

Chronological reports of damage from point-source pollution in the Eastern United States are listed in Table 7. For examples of damage from point-source pollution in Canada see Can. Dep. Environ. (1972, 1973), Linzon 1965, and Van Sickle (1973). The following major damage sites in Tennessee and Ontario have been described in detail.

Copper Basin, Tennessee

Mining operations in the Copper Basin began in 1850, with the greatest activity occurring between 1890 and 1895 (Hursh 1948). By 1910, the damage caused by sulfur dioxide fumes had resulted in the formation of three new vegetation zones in the vicinity of the smelter (Haywood 1910; Hedgecock 1914; Hursh 1948). Following initial timber harvesting to fuel the open-pit ore-roasting process, about 7,000 acres closest to the smelter were damaged by SO₂ and failed to revegetate. The second vegetation zone included about 17,000 acres of grassland. The third, a transition zone, consisted of about 30,000 acres of trees and grass (Hursh 1948). Trees found within this transition zone included sassafras, red maple, sourwood, and post oak (Smith 1981). The vegetation damage extended approximately 12 to 15 miles to the north and 12 miles to the west. Injury to white pine was observed up to 20 miles from the smelter (Hursh 1948).

Sudbury, Ontario

Since about 1885, the forests in the Sudbury area of Ontario have been subjected to sulfur dioxide fumes, sulfuric acid mist, acid precipitation, and heavy metal contamination from several nickel and copper smelters (Freedman and Hutchinson 1980; Gorham and Gordon 1960a,b; Watson and Richardson 1972). As with the Copper Basin, much of the nearby forest cover was removed to fuel the smelters. Forty square miles directly adjacent to the smelters are classified as severely barren, with an additional 140 square miles considered to have impoverished vegetation (Watson and Richardson 1972). Eastern white pine has been killed over a 700-square-mile area with damage observed more than 25 miles from the smelters (Linzon 1971; Smith 1981). Red oak, red maple, and redberry elder are more tolerant and exist in disturbed forest as close as 1 mile from the smelter (Smith 1981). With the construction of a 1,250-foot smokestack at the Copper Cliff Smelter and the closing of the Coniston Smelter in 1972, the spread of mortality and damage has been slowed (Freedman and Hutchinson 1980).

Wawa, Ontario

An iron-sintering plant began operation at Wawa, Ontario, in 1939 and was expanded in 1949. Damage from sulfur dioxide emissions was mostly restricted to a narrow strip of land northeast of the plant in the direction of the prevailing wind (Gordon and Gorham 1963). Four zones of vegetation were affected. All vegetation was killed within 5 miles of the smelter. From mile 5 to mile 12, only mountain maple and elder were present. Tree mortality was still high from mile 12 to mile 17 with only scattered white spruce and birch

present. Sulfur dioxide damage was evident on both conifers and hardwoods up to 23 miles away, though there was no white pine within 30 miles of the plant.

Regional Atmospheric Pollution

Measurements of regional atmospheric deposition or concentrations are sketchy before 1950 and subject to a variety of methods (McLaughlin 1985). See Husar (1986), Nat. Res. Council (1986), Cogbill (1976), and Likens et al. (1984) for discussions of atmospheric deposition in the United States. Much of the historical data on atmospheric deposition are estimates derived from records of coal consumption, oil and gas usage, and smelter production. The following briefly summarizes regional pollution in eastern hardwood forests with respect to acid precipitation, sulfur and nitrogen oxides, and ozone.

Acid precipitation

Pure water in condensation is neutral, that is, at pH 7.0. After reaching equilibrium with atmospheric concentrations of CO₂, the resulting carbonic acid solution would represent a theoretical "unpolluted" rain with an acidity of approximately pH 5.6. When acidity drops below pH 5.6, it is referred to as "acid rain." The acidifying substances are derived primarily from anthropogenic atmospheric pollutants (Smith 1981). Systematic pH measurements of rainfall are not available for the United States before 1962 (Cogbill and Likens 1974). Reliable continuous measurements of precipitation chemistry date only to 1978. The pattern of pH in rainfall in 1985 is shown in Figure 16.

Hydrogen ion deposition in summer is twice that in winter in the area of greatest deposition. Sulfate deposition in summer is 3 times higher than in winter over much of the Northeastern United States. Nitrate deposition over eastern North America is 1 to 2 times higher in summer than in winter (NAPAP 1987). Most of the major ions in precipitation decreased in concentration between 1978 and 1984 at more than half of 44 sites analyzed in eastern North America. Decreases in sulfate concentration were more frequent and of a larger magnitude than for any other ion. Changes in deposition of major ions in precipitation were less consistent than for concentration because of variations in the amount of precipitation. However, at more than half of the 44 sites evaluated, sulfate, calcium, magnesium, and hydrogen ion deposition decreased over that period (NAPAP 1987).

Sulfur oxides

Husar (1986) estimated that sulfur emissions in the United States increased from the 1890's to 1910, remained high until 1930, and have fluctuated since 1930. His estimates are based on data on coal and oil use and metal production. Regional patterns in recent years indicate a downward trend in the Northeast, generally stable levels in the Midwest, and rapid increases in the Southeast. In the Northern States west of Michigan, sulfur oxide (SO_x) emissions have remained low. Similar trends have been reported for the deposition of SO_x materials (Nat. Res. Council 1986; NAPAP 1987) (Fig. 17).

Table 7.—Summary of tree injury from point-source atmospheric pollution in the Eastern United States

Year	Location	Source	Affected species	Severity	Extent	Cause	Reference
1914	Naugatuck Valley, CT	Manufacturing plants.	All species. Coniferous species gradually being replaced by resistant broad-leaved species.	Growth of all species reduced by 25–50%.	Throughout Naugatuck Valley's entire length.	Sulphur dioxide and zinc sulphate.	Toumey 1921
1921	Eastern MA	Kiln burning high-sulphur content coal as part of its fuel.	White pine and other trees.	Reddening of foliage on white pine; foliage affected on others.	1/2 mile by 1/4 mile.	Acid fumes.	Snell and Howard 1922
1939	Radford, VA	U.S. Army Radford Ammunition Plant.	White pine and other conifers, and yellow-poplar.	Highly significant inverse relationship between production levels and annual ring widths.	Acute and chronic damage evident on white pine 1,000 yards downwind.	Sulphur dioxide and nitrogen oxides.	Skelly et al. 1972
1940	South-eastern OH: Monroe and Washington Counties (study areas)	Industrial sites.	All species of the mixed mesophytic type; overstory dominated by sugar maple, yellow buckeye, white ash, slippery elm, red oak, and basswood.	Stand density in overstory decreases; gradual shift in species composition.	30-mile section of Ohio River Valley (study area).	Chloride, sulphur dioxide, and flourides (all airborne)	McClenahan 1978
1950	Rockwood, Harriman, Kingston, Oak Ridge area of TN (Cumberland Plateau and Morgan Co.)	Coal-burning power plant, uranium refinery, pulp mill, ferroalloy reduction plant, and iron smelter.	White pine.	Postemergence chronic tip burn described; severity not specified.	Several hundred square miles.	Sulphur dioxide likely, possibly in combination with other stack gasses and ozone.	Berry and Hepting 1964
1970	South-eastern MO	Lead smelter.	Red and black oak, hickory, and shortleaf pine.	Not specified.	2,000 acres.	Oxidants and sulphur dioxide.	USDA For. Serv. 1971b

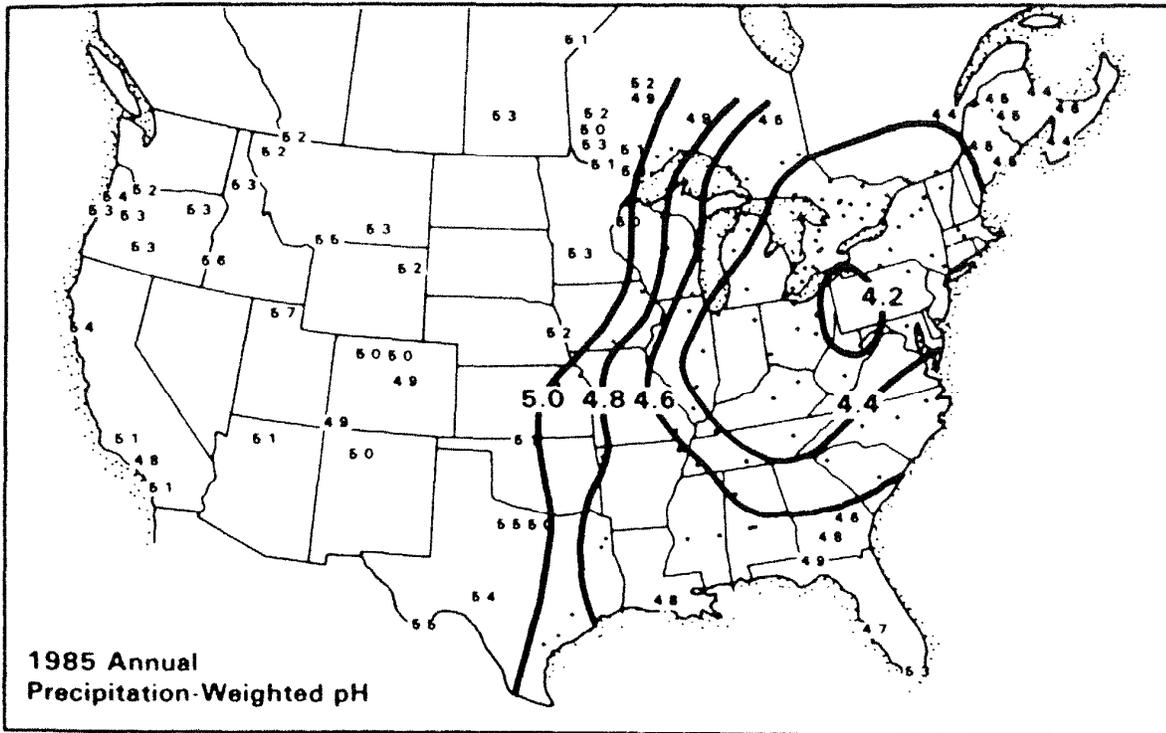
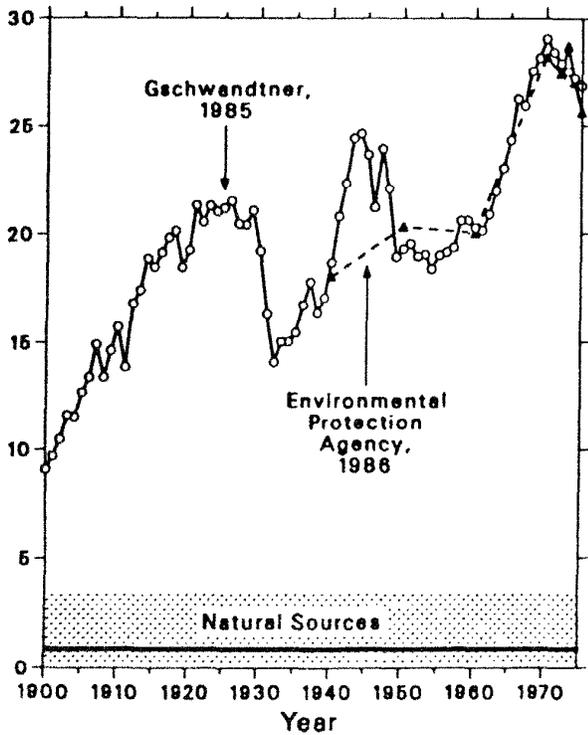


Figure 16.—Patterns of pH in rainfall in the United States in 1985 (NAPAP 1987).

Long-range Trends: 1900-1975
Emissions (10^6 metric tons/yr)



Recent Trends: 1975-1985
Emissions (10^6 metric tons/yr)

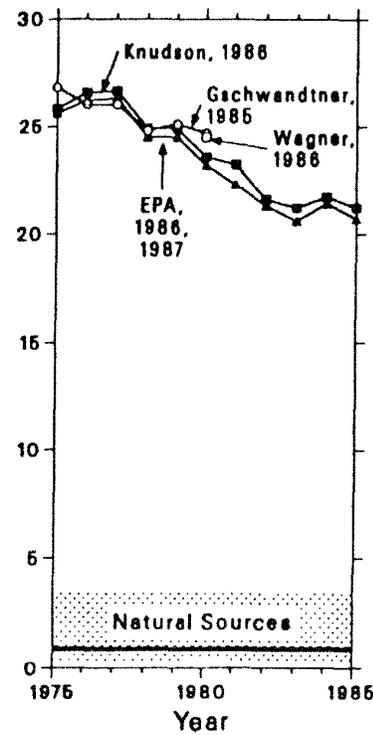


Figure 17.—Estimates of sulfur dioxide emissions in 1900–85 compared to SO_x emissions from natural sources in 1975–85 (NAPAP 1987).

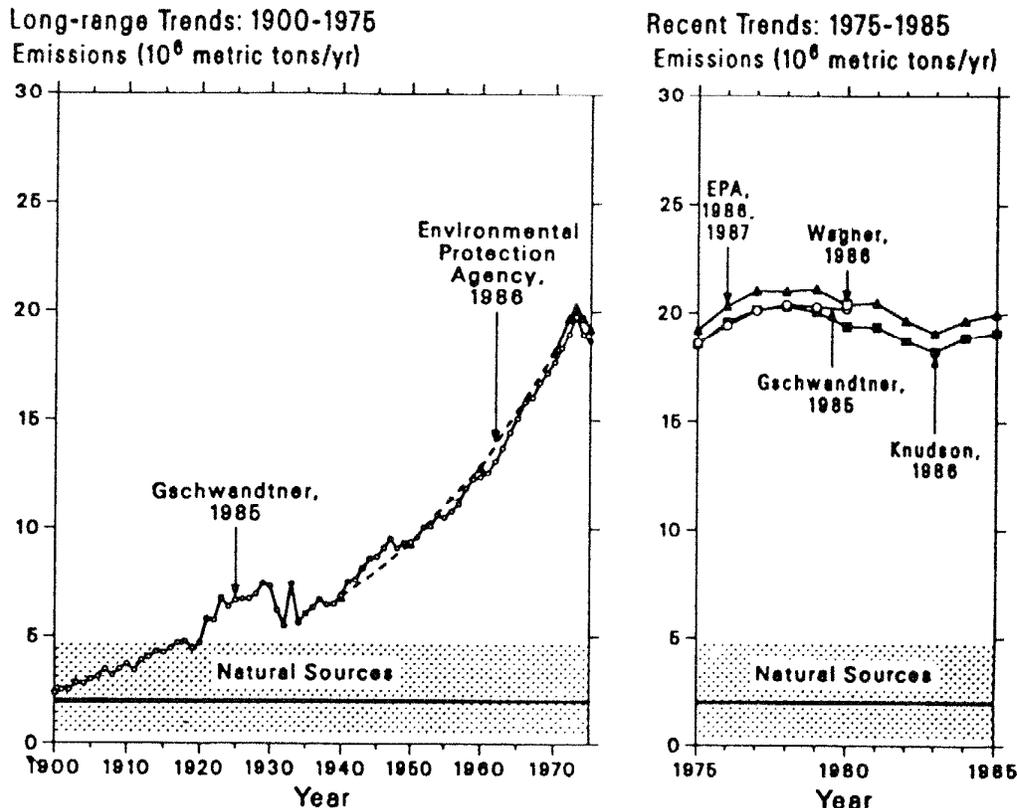


Figure 18.—Estimates of nitrogen dioxide emissions in 1900–85 compared to NO_x emissions from natural sources in 1975–85 (NAPAP 1987).

Nitrogen oxides

Like sulfur oxides, coal and oil combustion produces the major share of nitrogen oxides. Automobile combustion gases also contribute a significant share of NO_x (Husar 1985). Historical trends are difficult to determine since combustion processes rather than simple oxidation processes are responsible for NO_x production. These processes have changed continually. However, in general, NO_x emissions have increased since 1900 (NAPAP 1987), with the greatest increase in the Southeastern United States (Fig. 18).

Ozone

Long-term monitoring and, therefore, distribution maps of ozone (Fig. 19) are limited (NAPAP 1987; Husar 1985; Nat. Res. Council 1986), though increasing concentrations for the last 30 years have been suggested by McLaughlin (1985). Certainly, forestry-related concerns have increased greatly as suggested by reports of damage to ponderosa pine (Miller and McBride 1975) and eastern white pine (Linzon 1966; Berry and Ripperton 1963; Berry 1973; Skelly and Johnston 1979). In general, ambient levels of ozone can reduce photosynthesis, leading to an overall reduction in growth (Reich 1987). A number of conifer and hardwood

tree species are equally sensitive to ozone injury, as are agricultural crop species.

Mortality Associated with Patterns of Deposition

A simple spatial correlation between patterns of forest tree mortality and atmospheric deposition is insufficient as a basis for suggesting a cause-effect relationship between the two. Establishing such a relationship would require a consistent spatial relationship over time, a plausible mechanism, some form of response relationship, and the absence of other known causal agents. The presence or absence of a consistent spatial relationship would help define the boundaries of the problem and establish priorities for research. As part of the NAPAP Forest Response Program, other research is directed at evaluating growth loss through such mechanisms as the remeasurement of USDA Forest Service Forest Inventory and Analysis plots.

The following analysis of the spatial relationship between the distribution of mortality of oak, maple, birch, and ash and the patterns of atmospheric deposition in the Eastern United States is intended to identify regions where mortality

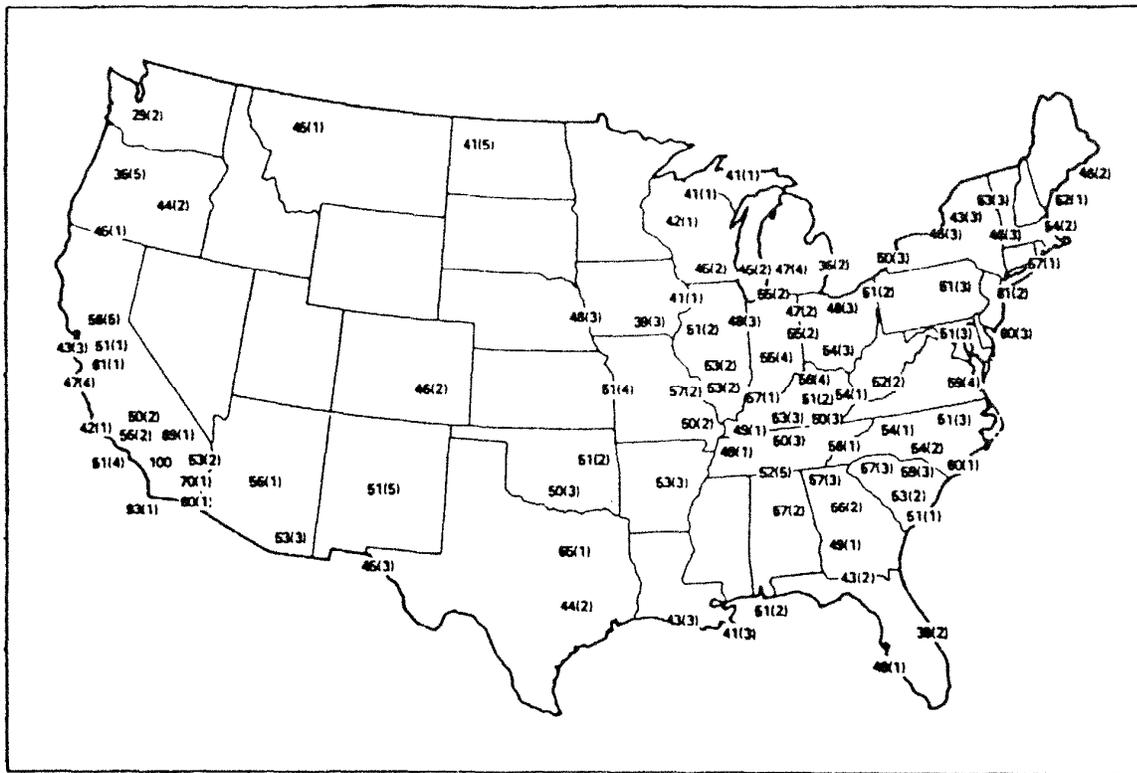


Figure 19.—Distribution of ozone concentrations (in parts per million) in the United States during the growing season, 1980–84, average maximum 7-hour daily mean. Number of observations are in parentheses (NAPAP 1987).

has occurred consistently under high levels of atmospheric deposition. Obviously, such areas should receive priority status for further evaluation over areas in which mortality has been low and/or has occurred under low-deposition regimes. Additional research should include an analysis of long-term patterns for climatic variables such as moisture and temperature extremes.

Oak Mortality

Patterns of oak mortality (Fig. 20) have varied from decade to decade, but from this analysis there does not appear to be evidence of a temporal trend in that variability. As stated previously, any analysis of the relationship between deposition variables and mortality is confined to the period 1980-86. For this period there is no evidence of a consistent relationship between areas of high mortality and high amounts of hydrogen ion deposition (Fig. 21). Most areas of moderate to high deposition have not experienced mortality during 1980-86, suggesting that hydrogen ion deposition was not a significant factor in the overall pattern of oak mortality.

There also was no evidence of a relationship between patterns of wet sulfate or nitrate deposition and patterns of oak mortality.

A significant portion of the southernmost distribution of oak mortality falls within the region of highest growing-season ozone concentrations. However, this region also

experienced periods of drought in 1980, 1981, 1985, and 1986, along with major insect defoliations, which seriously confound any spatial relationship with ozone patterns. Further investigation of the relationship among ozone, climate, and oak mortality would appear warranted in this southern region.

Maple Mortality

Pockets of maple mortality in the Northeastern United States and Southeastern Canada coincide with high levels of H^+ , SO_4 , and NO_3 – wet deposition (Figs. 22-23). As a result, maple mortality in those areas of high deposition should receive additional evaluation. As maple mortality was confined to portions of the Northeastern United States where ozone concentrations tend to be moderate to low, no relationship between patterns of maple mortality and ozone concentration is apparent.

Birch Mortality

A limited area of birch mortality in upstate New York coincides with high levels of H^+ , SO_4 , and NO_3 – deposition (Figs. 24-25). However, the remainder of birch mortality is in areas of low deposition. Ozone does not appear to be a factor in patterns of birch mortality. The mortality in upstate New York suggests a need for further evaluation in that area.

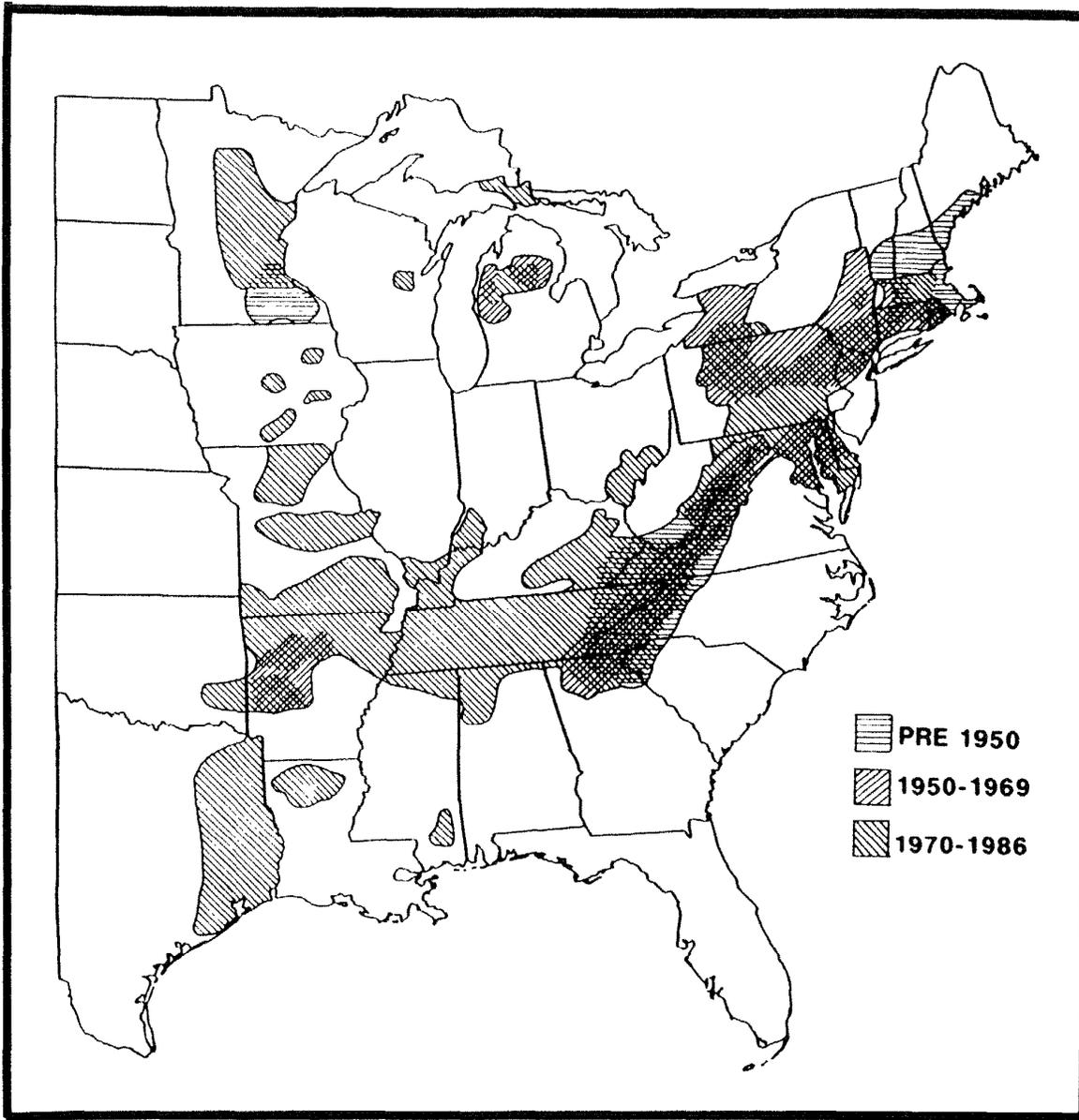


Figure 20.—Composite of oak mortality distribution shown in Figure 3.

Ash Mortality

Nearly all of the ash mortality between 1980 and 1986 occurred in areas of moderate to high levels of wet H^+ , SO_4^{2-} , or NO_3^- deposition (Figs. 26-27). As a result, these

factors cannot be dismissed as other variables such as climate and disease are evaluated. There is no evidence for a spatial relationship between ash mortality and ozone concentrations.

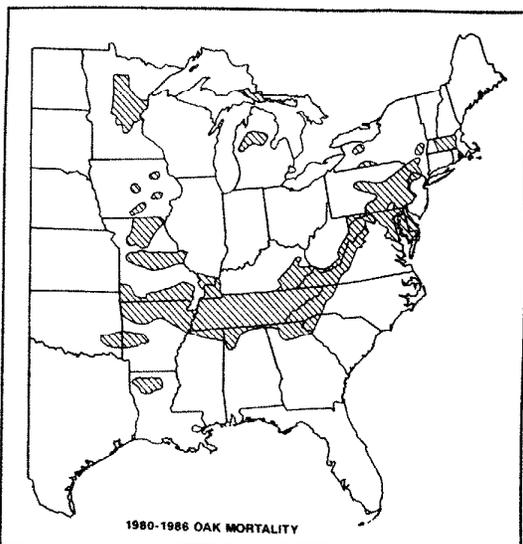
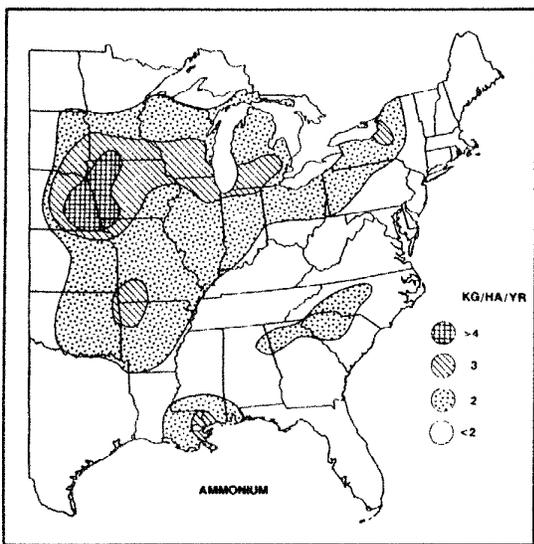
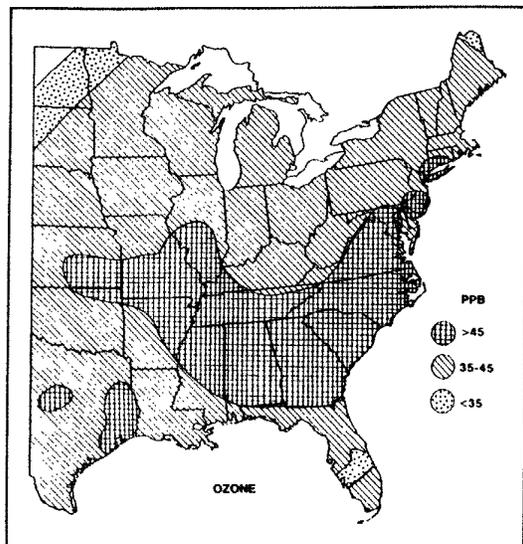
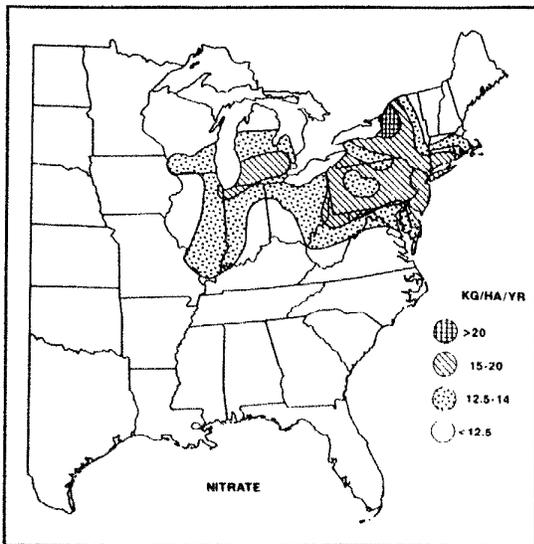
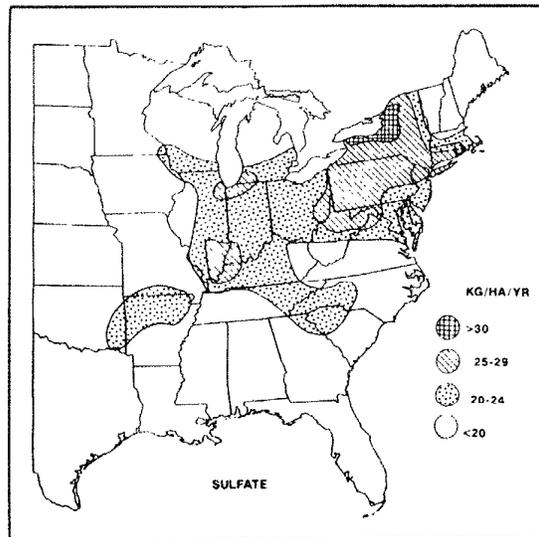
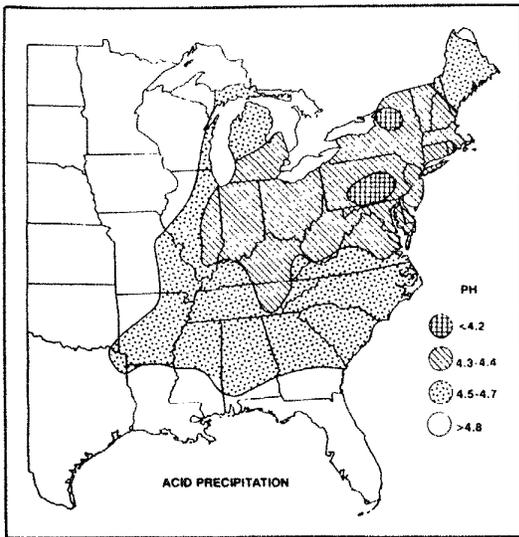


Figure 21.—Patterns of atmospheric deposition and oak mortality in the Eastern United States, 1980–86.

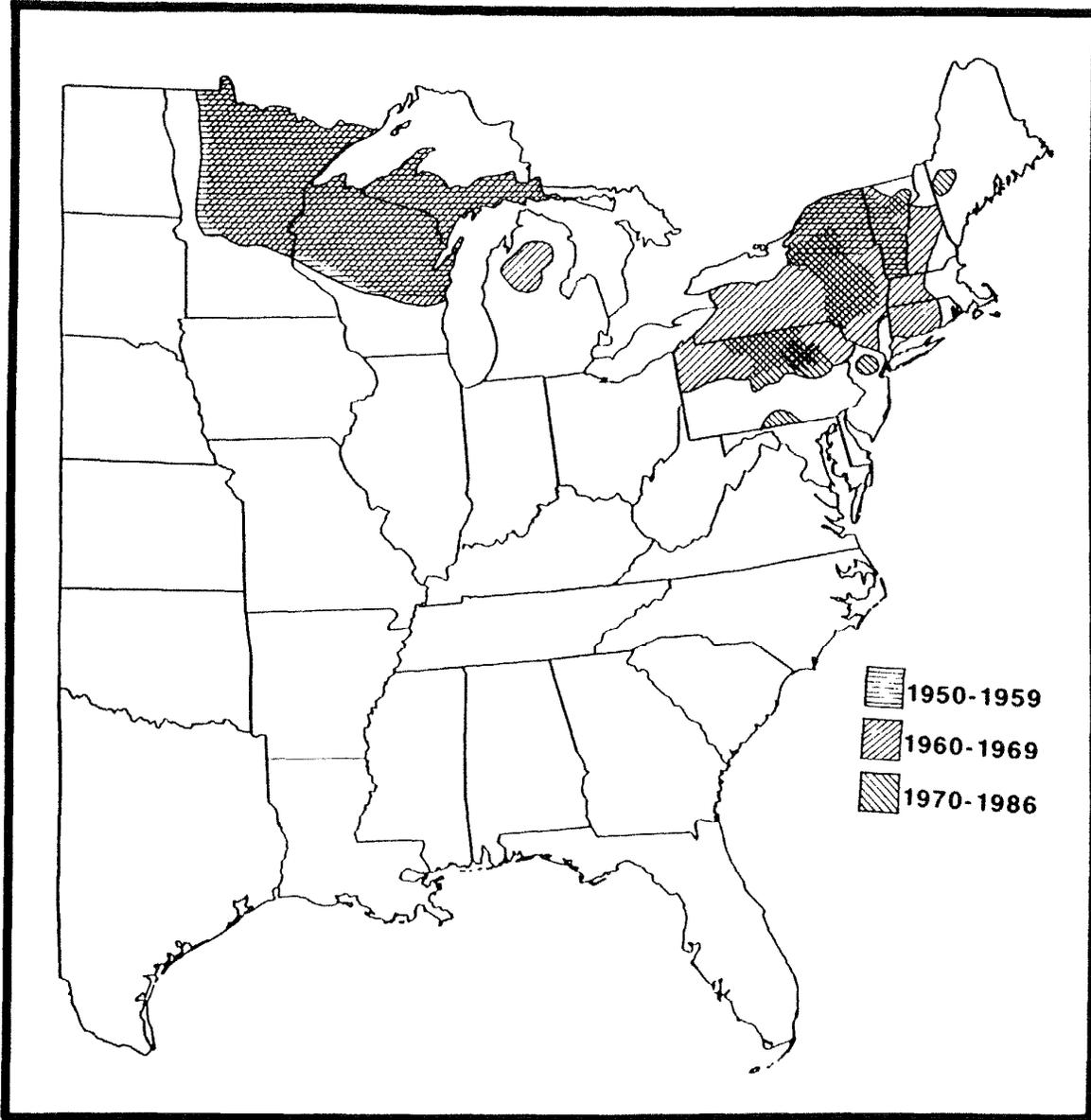


Figure 22.—Composite of maple mortality distribution shown in Figure 5.

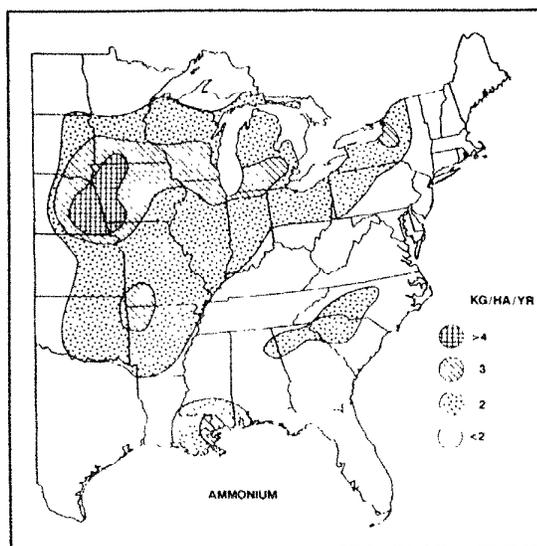
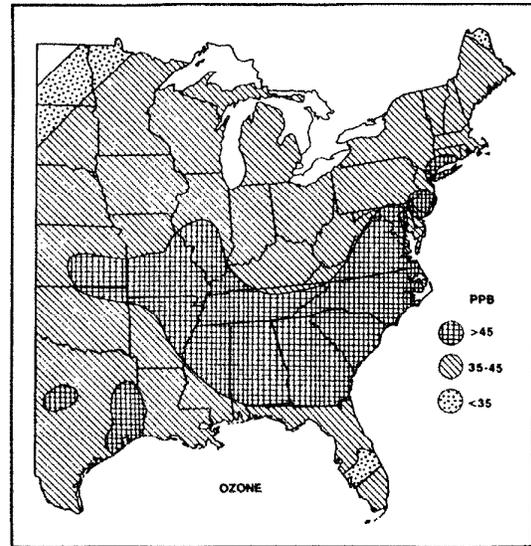
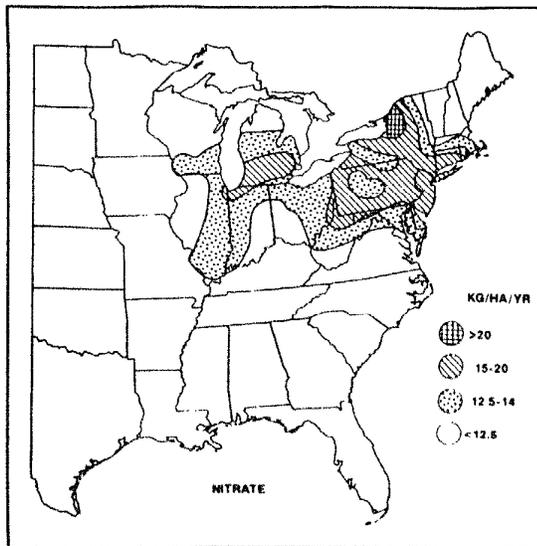
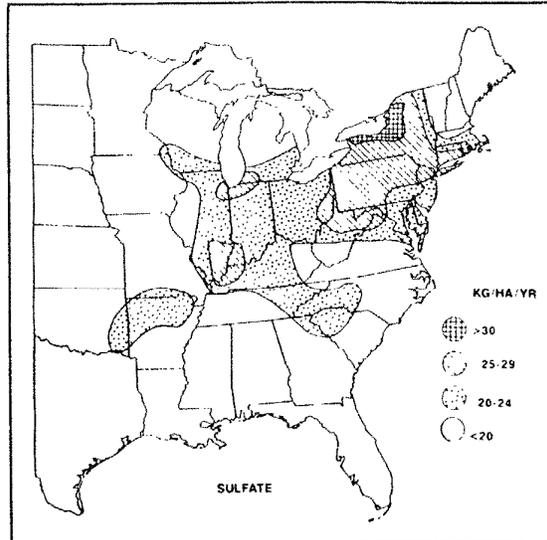
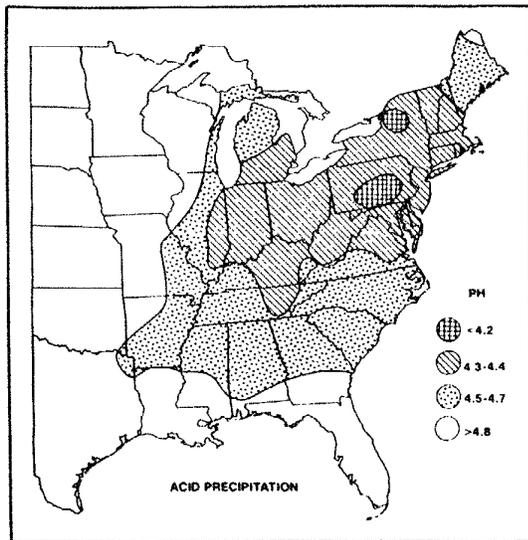


Figure 23.—Patterns of atmospheric deposition and maple mortality in the Eastern United States, 1980–86.

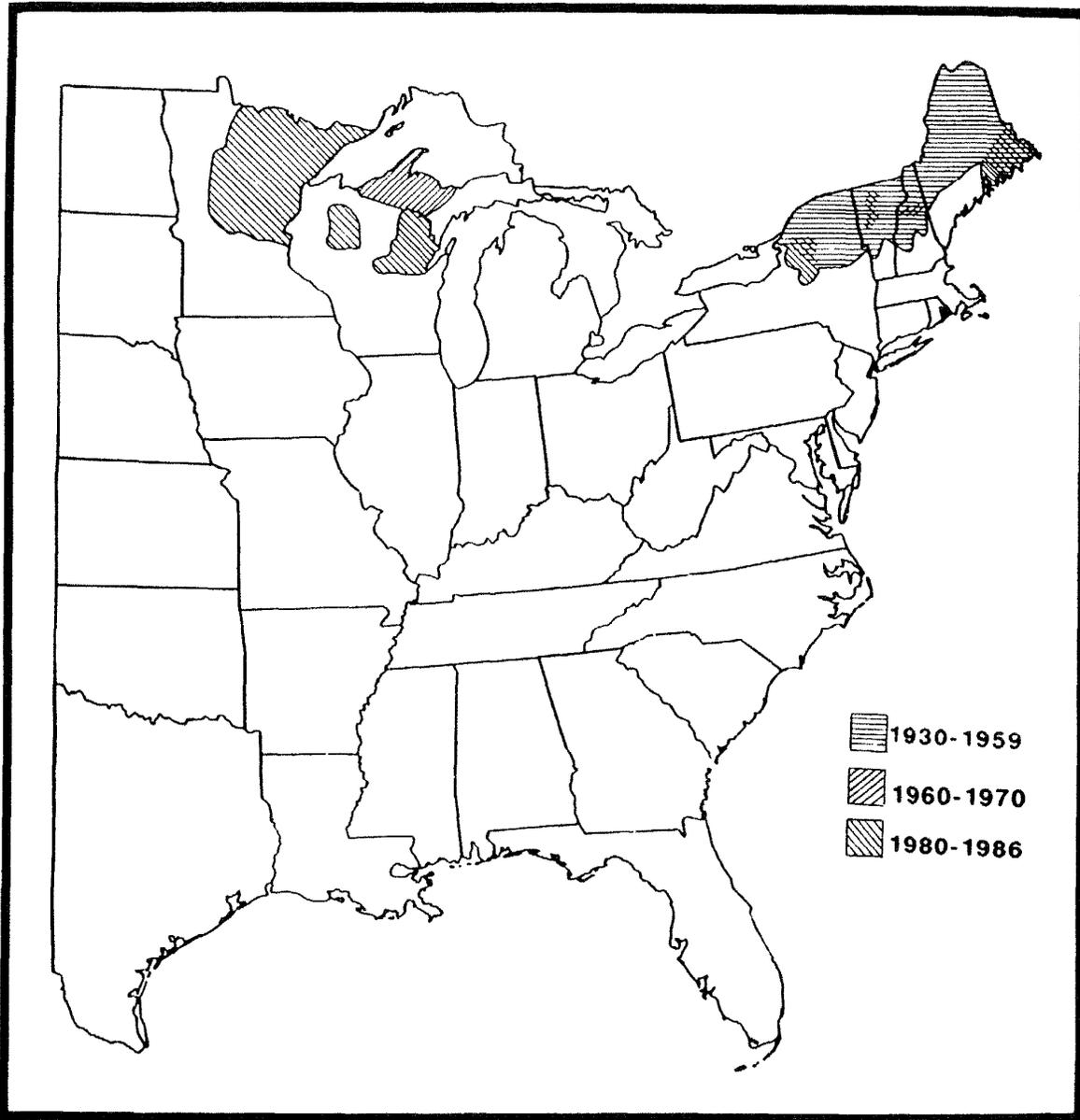


Figure 24.—Composite of birch mortality distribution shown in Figure 7.

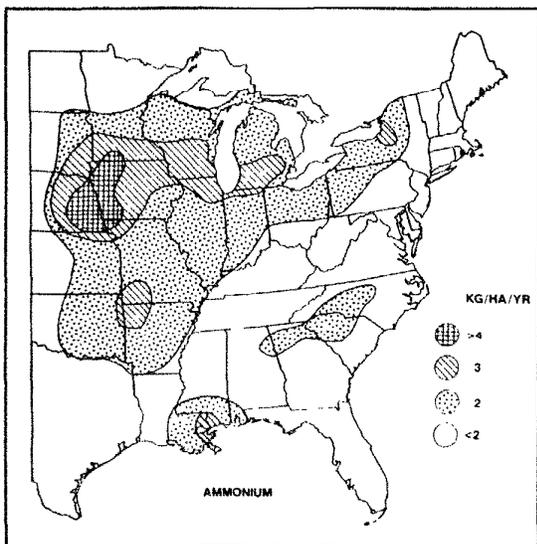
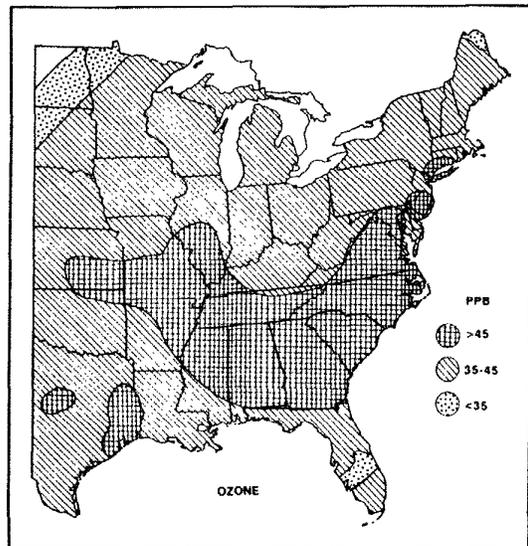
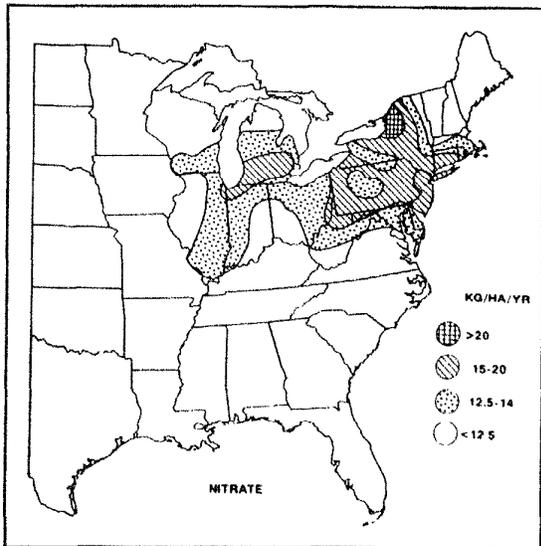
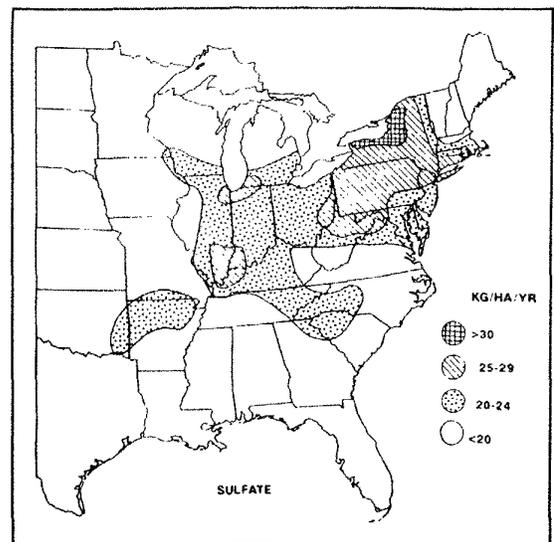
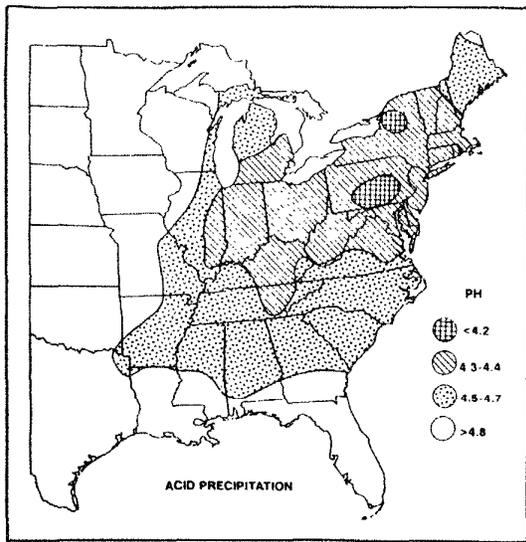


Figure 25.—Patterns of atmospheric deposition and birch mortality in the Eastern United States, 1980–86.

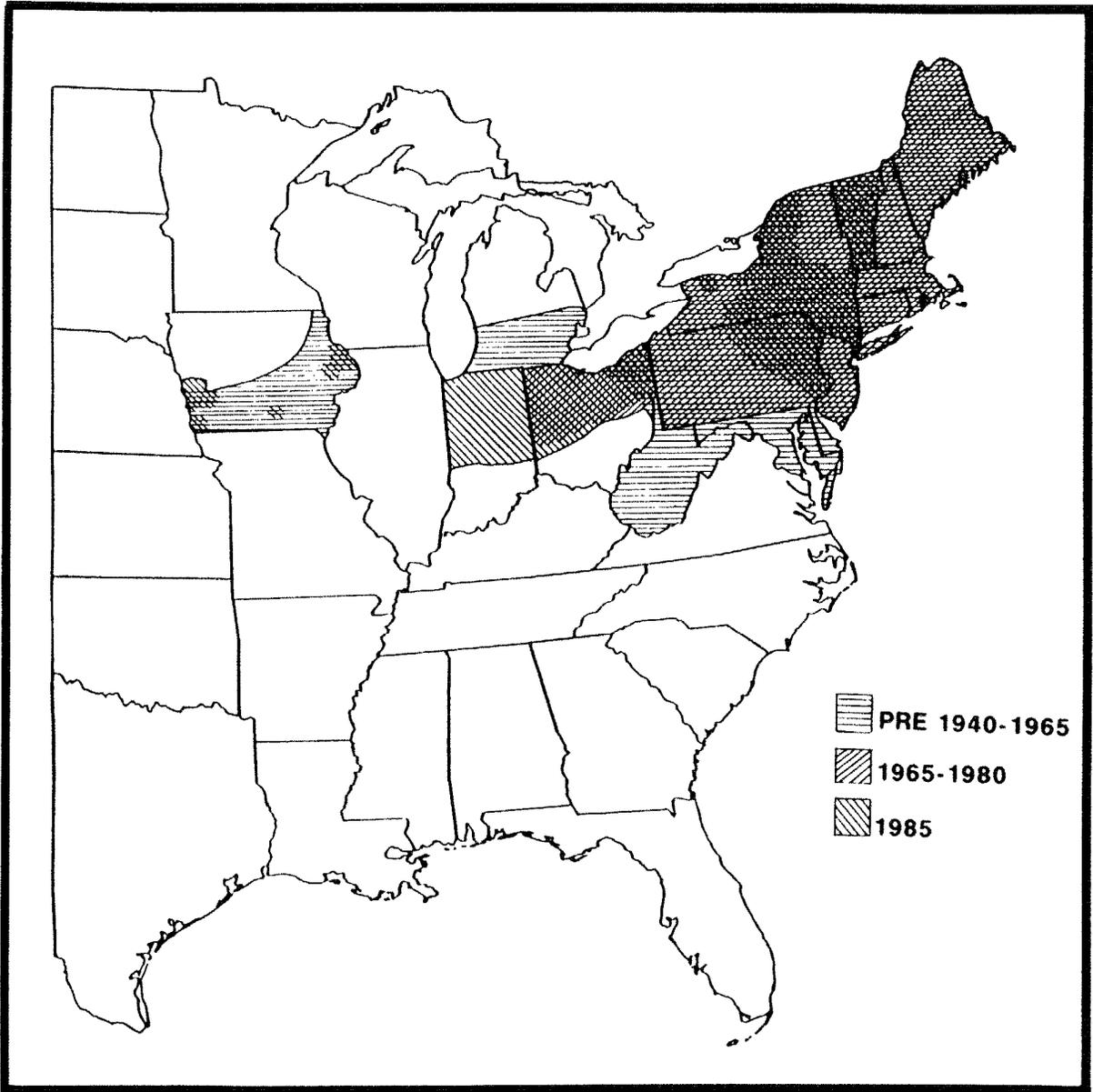


Figure 26.—Composite of ash mortality distribution shown in Figure 9.

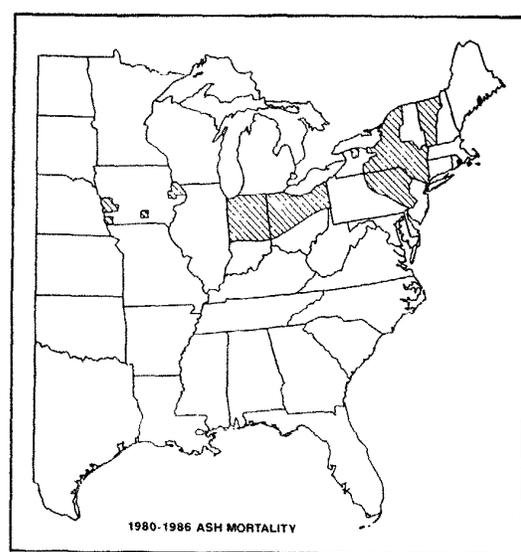
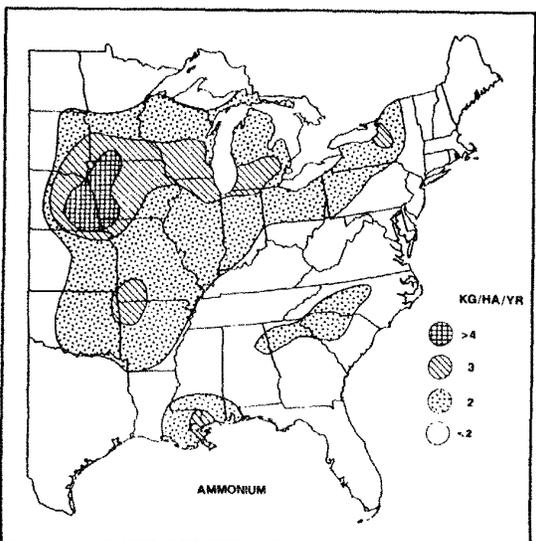
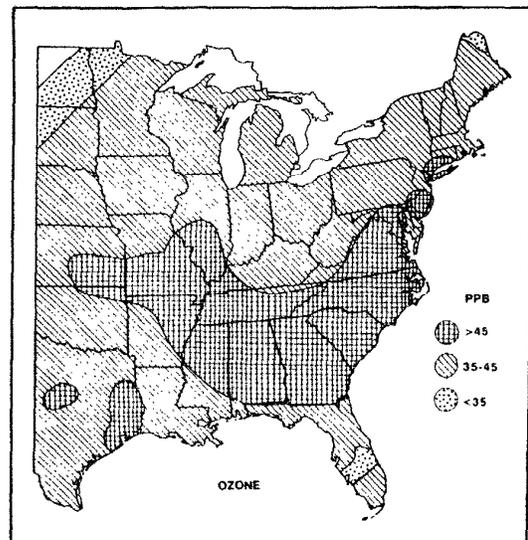
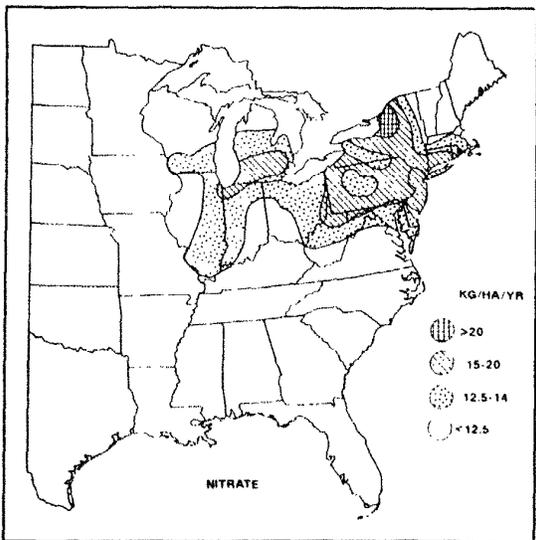
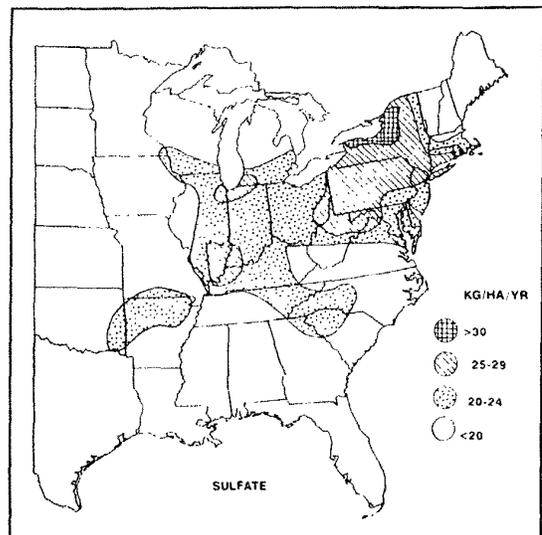
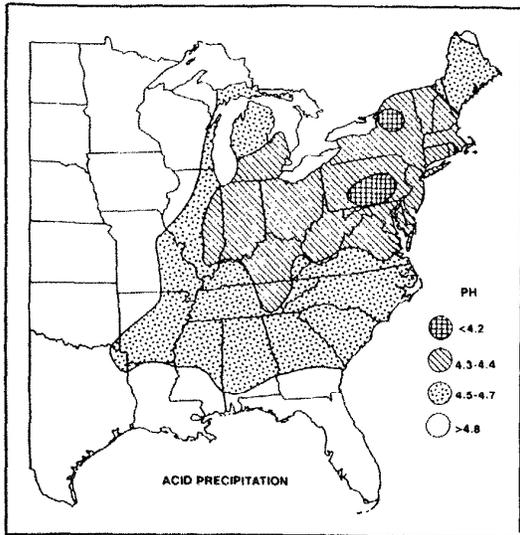


Figure 27.—Patterns of atmospheric deposition and ash mortality in the Eastern United States, 1980–86.

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APPENDIX

Common and Scientific Names

Tree Species

American basswood (*Tilia americana* L.)
American beech (*Quercus alba* L.)
American chestnut (*Castanea dentata* (Marsh.) Borkh.)
American elm (*Ulmus americana* L.)
Baldcypress (*Taxodium distichum* (L.) Rich.)
Balsam fir (*Abies balsamea* (L.) Mill.)
Balsam poplar (*Populus balsamifera* L.)
Bigtooth aspen (*Populus grandidentata* Michx.)
Bitternut hickory (*Carya cordiformis* (Wangenh.) K. Koch)
Black ash (*Fraxinus nigra* Marsh.)
Black cherry (*Prunus serotina* Ehrh.)
Black locust (*Robinia pseudoacacia* L.)
Black oak (*Quercus velutina* Lam.)
Black walnut (*Juglans nigra* L.)
Blackgum (*Nyssa sylvatica* Marsh.)
Blackjack oak (*Quercus marilandica* Muenchh.)
Buckeye (*Aesculus spp.*)
Bur oak (*Quercus macrocarpa* Michx.)
Chestnut oak (*Quercus prinus* L.)
Chinkapin oak (*Quercus muehlenbergii* Engelm.)
Cucumbertree (*Magnolia acuminata* L.)
Dogwood (*Gornus spp.*)
Eastern cottonwood (*Populus deltoides* Bartz. var. *deltoides*)
Eastern hemlock (*Tsuga canadensis* (L.) Carr.)
Eastern redcedar (*Juniperus virginiana* L.)
Eastern white pine (*Pinus strobus* L.)
Gray birch (*Betula populifolia* Marsh.)
Green ash (*Fraxinus pennsylvanica* Marsh.)
Ironwood (*Ostrya virginiana* (Mill.) K. Koch)
Jack pine (*Pinus banksiana* Lamb.)
Live oak (*Quercus virginiana* L.)
Loblolly pine (*Pinus taeda* L.)
Mockernut hickory (*Carya tomentosa* (Poir.) Nutt.)
Northern red oak (*Quercus rubra* L.)
Northern white-cedar (*Thuja occidentalis* L.)
Nuttall oak (*Quercus nuttallii* Palmer)
Paper birch (*Betula papyrifera* Marsh.)
Pin cherry (*Prunus pensylvanica* L.f.)
Pin oak (*Quercus palustris* Muenchh.)
Pitch pine (*Pinus rigida* Mill.)
Post oak (*Quercus stellata* Wangenh.)
Quaking aspen (*Populus tremuloides* Michx.)
Red maple (*Acer rubrum* L.)
Red pine (*Pinus resinosa* Ait.)
Red spruce (*Picea rubens* Sarg.)
Scarlet oak (*Quercus coccinea* Muenchh.)
Shagbark hickory (*Carya ovata* (Mill.) K. Koch.)
Shortleaf pine (*Pinus echinata* Mill.)
Shumard oak (*Quercus shumardii* Buckl.)
Southern magnolia (*Magnolia grandiflora* L.)
Southern red oak (*Quercus falcata* Michx.)
Southern yellow pine (*Pinus echinata* Mill.)
Sugar maple (*Acer saccharum* Marsh.)
Swamp tupelo (*Nyssa sylvatica* var. *biflora* (Walt.) Sarg.)
Sweet birch (*Betula lenta* L.)
Sweetgum (*Liquidambar styraciflua* L.)

Tamarack (*Larix laricina* (Du Roi) K. Koch)
Virginia pine (*Pinus virginiana* Mill.)
White ash (*Fraxinus americana* L.)
White oak (*Quercus alba* L.)
White spruce (*Picea glauca* (Moench) Voss)
Willow oak (*Quercus phellos* L.)
Yellow birch (*Betula alleghaniensis* Britton)
Yellow buckeye (*Aesculus octandra* Marsh.)
Yellow-poplar (*Liriodendron tulipifera* L.)

Insect Species

Beech scale (*Cryotococcus fagisuga* Lindinger)
Birch leafminer (*Fenusa pusilla* (Lepelletier))
Birch skeletonizer (*Bucculatrix canadensisella* Chambers)
Bronze birch borer (*Agrilus anxius* Gory)
Bruce spanworm (*Operophtera bruceata* (Hulst))
Cherry scallop-shell moth (*Hydria prunivora* Ferguson)
Elm spanworm (*Ennomos subsignarius* (Hubner))
Fall cankerworm (*Alsophila pometaria* (Harris))
Forest tent caterpillar (*Malacosoma disstria* Hubner)
Fruittree leafroller (*Archips argyrospilus* (Walker))
Green fruitworm (*Lithophane antennata* (Walker))
Greenstriped mapleworm (*Dryocampa rubicunda* (Fabricius))
Gypsy moth (*Lymantria dispar* (L.))
Half-wing geometer (*Phigalia titea*)
Hickory bark beetle (*Scolytus quadrispinosus* Say)
Large aspen tortrix (*Choristoneura conflictana* (Walker))
Linden looper (*Erannis tiliaria* (Harris))
Locust leafminer (*Odontota dorsalis* (Thunberg))
Maple webworm (*Tetralopha asperatella* Clemens)
Oak leafroller (*Archips semiferanus* (Walker))
Oak leaf-tier (*Croesia semipurpurana* (Kearf.))
Oak skeletonizer (*Bucculatrix ainsliella* Murtfeldt)
Orangehumped mapleworm (*Symmerista leucitys* Franclemont)
Oystershell scale (*Lepidosaphes ulmi* (L.))
Peach bark beetle (*Phloeotribus liminaris* (Harris))
Poplar borer (*Saperda calcarata* Say)
Post-oak locust (*Dendrotettix quercus* Packard)
Redhumped oakworm (*Symmerista canicosta* Franclemont)
Saddled prominent (*Heterocampa guttivitta* (Walker))
Tuliptree scale (*Toumeyella liriodendri* (Gmelin))
Twolined chestnut borer (*Agrilus bilineatus* (Weber))
Variable oakleaf caterpillar (*Heterocampa mateo* (Doubleday))
Walkingstick (*Diaperomera femorata* (Say))
White pine weevil (*Pissodes strobi* (Peck))
Winter moth (*Operophtera brumata* (L.))
Yellow-poplar weevil (*Odontopus calceatus* Say)

Diseases and Fungi

American chestnut blight (*Endothia parasitica* (Murr.) P.J. and H.W. Anderson)
Annosum root rot (*Heterobasidion (Fomes) annosum* (Fries) Berf.)
Armillaria or shoestring root rot (*Armillaria mellea* Vahl. ex Fr.)
Ash anthracnose (*Gloeosporium aridum*)
Ash rust (*Puccinia peridermiospora*)

Beech bark disease (complex) (*Nectria coccinea* var. *faginata* (Loh., Wats., & Ay.)
Bleeding canker (*Phytophthora cactorum*)
Butternut canker (*Sirococcus clavigignenti-juglandacearum*
sp. nov. *Melanconis juglandis* (E. & E.) Graves)
Cytospora canker (*Cytospora leucostoma* Fr.)
Dutch elm disease (*Ceratocystis ulmi* (Buism.) C. Mor.)
Fusarium canker (*Fusarium solani* (Mart.) App. & Wr.)
Heart rot (*Phellinus (Fomes) ignarius* (L. ex Fr.) Kickx.)
Hypoxyton canker (*Hypoxyton mammatum* (Wahl.) Miller)
Mushroom or Clitocybe root rot (*Clitocybe tabescens* Bres.)
Oak wilt (*Ceratocystis fagacearum* (Bretz.) Hunt)
Sapstreak (*Ceratocystis coerulea* (Munch) Bakshi)
White pine blister rust (*Cronartium ribicola* Fisch.)
White pine root decline (*Verticicladiella procera* Kendrick)