4. Forest Declines in Response to Environmental Change

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Decline diseases are intimately linked to stress and environmental change. There is strong evidence that, as a category, decline diseases have increased significantly in response to the climate, air chemistry, and other changes documented in the northeastern United States over the past century, and particularly the last two decades. No other forest response to environmental change and stress is expected to be as dramatic. Decline diseases occur in response to multiple, often overlapping and interacting, stressors that typify the ongoing and future environmental changes expected in the Northeast.

Forest declines have a special significance in the Northeast since, more than in any other region, they form the conceptual and experimental basis on which the theories and models of decline diseases have evolved. New significant scientific advances from recent research in the Northeast hold considerable promise for better understanding and effective treatment of these complicated diseases. Several studies are in progress on the development of an early warning of the risk to dieback and on innovative experiments on managing and adapting to future declines (Auclair, 1997).

Decline Disease: A Unique and Complex Response to Environmental Stress

Definition of Decline Disease

Decline denotes a deterioration of tree health, often leading to mortality. This may be a normal and inevitable phase of a tree's life before death (Castello et al., 1995a). The term decline also has been used to characterize reductions in radial growth observed on tree species (Hornbeck and Smith, 1985; LeBlanc, 1990; Van Deusen et al., 1991). However, decline disease is not always associated with decreases in growth (Van Deusen et al., 1991; Wargo et al., 1993). In this chapter, decline refers to the disease condition, while growth reduction is considered part of the syndrome of the decline disease (Wargo et al., 1993).

In forest pathology, decline is a major category of tree diseases characterized by premature and progressive deterioration in tree health related to stress and secondary-organism attack (Manion, 1987). The interaction of stressors with "secondary organisms" (Houston, 1981) results in dieback, decline, and mortality of trees. Forest trees typically "decline" in response to a plethora of stressors.

Stressors are any adverse environmental factors that induce damage or injurious strain to the living organism (Levitt, 1972). Stressors may be biotic or abiotic, may occur separately or in concert, and affect plants individually, as populations, or as communities. Stress may occur chronically over a significant period or it may be of short duration and acute. The strain caused by these stressors may be physical or chemical, or both, and sometimes is irreversible, that is, a portion or all of a plant is killed (Levitt, 1972).

Declines have occurred in the past and will continue to occur in the future. Because of the multiple factors involved in their development, declines are especially difficult to diagnose and to attribute cause and effect, and thus, to reproduce experimentally. The term decline was commonly assigned to diseases whose primary cause was unknown or "unexplained." Sinclair (1967) was one of the first to recognize that these diseases have a unique etiology and proposed the concept of complex causality.

Symptoms of Decline Diseases

Symptoms of decline diseases are general and similar among the various species and regions experiencing decline diseases. Manion (1991) lists 10 of the most common symptoms of decline, including reduced growth rate, dieback, and impaired energy relationships. The rate at which symptoms appear and their magnitude reflect primarily the condition of the host population, including its age (Auclair et al., 1997) and vigor and vitality (Wargo, 1978). Other factors include the genetic uniformity or heteroge-

neity of the tree populations, the intensity and frequency of stress, and the numbers and aggressiveness of secondary organisms attacking the weakened trees. Subsequent mortality can occur on scattered individual trees, in small clusters or groups of trees, or over extensive areas depending on the interaction of these factors (Houston, 1981; Wargo, 1981b).

Crown, Stem, and Root Characteristics

Crown. Anomalous leaf condition and dieback of the crown are among the first and most conspicuous features of trees that are stressed and undergoing decline. Leaf anomalies include reduced leaf size, scorch of leaf margins, veinal necrosis, seasonally premature leaf coloration, and leafdrop. Some pathologists have argued that these leaf anomalies can result from specific events, such as spring frost, severe drought, or heat stress, and do not necessarily lead to progressive dieback or decline. For this reason, Auclair et al. (1996, 1997) omitted leaf anomalies in their reconstruction of dieback episodes in northern hardwoods.

Progressive crown dieback over one or several consecutive years is a common feature of decline disease. Typically, the crowns of deciduous trees dieback from the tips inward toward the trunk. Clusters of epicormic shoots may distinguish the crown in later stages. In conifers, the older needles often succumb first, giving the appearance of clusters of young, live foliage at the outer ends of the branches. The rate at which these features develop may be related to the level and persistence of evapotranspiration stress on the crown. Hence, rapid dieback could occur in affected trees during periods of exceptional drought and/or heat stress. Conversely, cool wet weather could result in renewed vigor and health, as was dramatically evidenced on species of birch (*Betula* spp.) in the early 1950s (Auclair et al., 1996).

In their studies on sugar maple (*Acer saccharum* Marsh.) blight, Houston and Kuntz (1964) were the first to relate bud mortality and dieback to defoliation. Defoliation of sugar maple by a number of insects was associated with dieback and decline of sugar maple (Giese et al., 1964; Giese and Benjamin, 1964). Wargo (1981b) showed similar relationships in studies on oak (*Quercus* spp.) decline and mortality and defoliation by the gypsy moth (*Lymantria dispar* L.). In detailed studies on dieback in defoliated sugar maple, Gregory and Wargo (1986) showed that dieback was related to survival of terminal and axillary buds on defoliated branches. After early season defoliations, the developing buds in the axils of the removed leaves abscised, but axillary and terminal buds on the refoliated terminal shoots survived through winter. In late season defoliation, most buds of refoliated shoots did not survive and the next year's growth depended on axillary buds formed prior to defoliation. Thus, when progressing from early to late defoliations, the next year's shoot growth depended decreasingly on the last-formed and increasingly on the

first-formed portions of the previous year's shoot. Bud survival, and hence terminal bud and twig dieback, was related to the scale and leaf primordia production in buds formed after defoliation. Insufficient scale primordia increased bud susceptibility to winter injury and dieback the following spring.

Stem. A common feature of trees showing dieback is a reduction in radial and terminal growth. Reductions in radial growth have been most commonly reported and sometimes precede crown symptoms by several to many years (Staley, 1965—oak decline; Bauce and Allen, 1991; Kolb and McCormick, 1993—sugar maple decline; Wargo et al., 1993—red spruce decline). Reductions in terminal elongation on declining red spruce (*Picea rubens* Sarg.) trees also have been observed (Tobi et al., 1995; Wargo et al., 1993).

Another feature observed on trees showing dieback is the inability to rapidly transmit water to the crown. Greenidge (1951) used dye treatments to demonstrate air blockages or embolisms in the stem cross sections of yellow birch (*Betula alleghaniensis* Britton). The degree of blockage was roughly proportional to the severity of dieback in the crown. In trees with severe dieback, the dye dumped at the base of the tree, barely reaching the bottom of the crown.

Sperry et al. (1988) ascribed the development of embolized branches in sugar maple to winter insolation at the time of extreme cold, and to drought in the late summer. Tree stresses most likely to cause irreversible embolisms include freezing and drought stresses (Tyree and Sperry, 1988, 1989). There is great individuality in the susceptibility within and between tree species to emboli formation; differences noted do not necessarily or systematically relate to vessel and tracheid structure (Zimmerman, 1983). Contrary to early concepts on embolism formation, conifers, as with deciduous species, can embolize and show as severe water transport dysfunction.

Deciduous trees are particularly susceptible to embolisms due to freezing stress (Cochard and Tyree, 1990). Auclair et al. (1996) noted that freezing (prolonged winter thaw followed by sudden freezing and/or root freeze and root kill) was correlated to the onset of major dieback episodes in northern hardwoods. Once putatively injured, the affected trees seem to have been especially sensitive to drought which, when it followed a freezing event, determined the rate and severity of dieback in the crown. Auclair et al. (1996) demonstrated that each major episode of dieback during the century (1910 to 1995) coincided with occurrences of potential winter freezing events followed by drought during the growing season. This suggests a direct link between changing levels of climatic stresses in the region and the extent of decline disease.

Root. Reduction in carbon reserves, especially obvious in the roots, is a major symptom of declining trees. Below normal levels of starch have been

observed in declining oak (Staley, 1965; Wargo, 1981c), sugar maple (Wargo et al., 1972), and red spruce (Wargo et al., 1993). Carbohydrate dynamics seems to integrate the interaction of stressors, tree vitality, and subsequent decline (Wargo, 1999).

Another common feature in declining trees is a high level of rootlet mortality. Greenidge (1951) excavated 74 declining yellow birch trees and quantified the levels of rootlet mortality. Levels of fine root loss ranged from 10 to 40% in slightly affected trees, and up to 40 to 90% in severely declining trees. Similar relationships of fine root loss were also observed in declining red spruce (Wargo et al., 1993) and oak species (Staley, 1965).

This observation has particular significance to forests in the Northeast undergoing climatic change. Root tissues are by far the least frost hardened of any tree tissue (Larcher and Bauer, 1981; Sakai and Larcher, 1987), are relatively shallow, and usually are covered by snowpack from December through early March. "Open winters" (i.e., low snowpack accumulation or prolonged winter thaws resulting in the complete subsidence of snowpack) create risk for root kill in the event of cold air temperatures (Pomerleau, 1991; Robitaille et al., 1995). Auclair et al. (1996) quantified the incidence and magnitude of potential root freezing stress in southern Quebec (Lennoxville) and in northern Vermont (Burlington). Throughout the last century, at intervals of 8 to 12 years, episodes of conditions that could induce severe root freezing occurred at both stations. Since the mid-1970s (especially in southern Quebec) there has been a strong trend for winters without snowpack. The incidence of dieback has followed these trends.

Mortality, Recovery, and Regeneration Patterns

Mortality. The common outcome in the event of mounting stress is progressive deterioration in tree health. In the event of persistent stress and, in most instances, attack by secondary organisms, mortality is likely. Tree death can occur within one growing season but has been known to occur as long as 6 to 10 years after the first symptoms are apparent. This extended dying period is significant ecologically because the declining tree occupies the site for an extended period and reduces the levels of resources (light, water, and nutrients) otherwise available for growth of associated healthy individuals.

A common observation has been that decline diseases, including mortality, most affect older or larger trees in the population. Possible reasons for this include change in root/shoot ratio, the sapwood/canopy area ratio, and physiological or metabolic senescence (Mueller-Dombois, 1992). Another possible explanation is the much greater risk of emboli formation in larger and older trees (Zimmerman, 1983).

There are marked differences among species of northern hardwoods in susceptibility to mortality. Using periodic plot inventories of forest growth

and mortality in Vermont from 1960 to 1995, Auclair (1997) estimated that decline accounted for 73% of the total volume loss (reduced growth plus mortality) in white (*Betula papyrifera* Marsh.) and yellow birch compared with only 26% in sugar maple. White ash (*Fraxinus americana* L.) (53%) and red spruce (58%) were intermediate. This is consistent with the high levels of tree death (70 to 100%) in episodes of birch dieback in the Northeast in the 1940s and 1950s. Although the overall observed tree death rates in sugar maple (2 to 7%) have been notably less in the 1980s and 1990s, mortality can be much higher in this species in the event of persistent multiple or interacting stresses (see Case Study 1 later in this chapter).

Recovery. Decline diseases in the Northeast have been highly episodic. Onset of a major episode can be surprisingly rapid, as can the subsidence of the episode and resumption of partial or full health of the affected trees. Trees showing decline have been known to recover, especially if favorable growth conditions resume at an early point in the decline spiral and secondary-organism attack is absent or minimal. Sugar maples that had as much as 40% branch mortality survived and had good crowns after three years of recovery (Gross, 1991). Oak species defoliated by the gypsy moth continued to deteriorate for five years after the last defoliation and then recovered to their original healthy condition within five years (Campbell and Sloan, 1977).

Regeneration. Seedlings and saplings are often unaffected and remain healthy, even at the time of severe dieback and decline of the canopy trees. Vigorous regeneration often can be of the same species as the declining trees, though exceptions do occur. Dieback and decline is widespread in eucalyptus (*Eucalyptus* spp.) forests across Australia and many, if not most, declining stands are not regenerating (Heatwole and Lowman, 1986). Case Study 1, presented later in this chapter, shows that regeneration in declining sugar maple forests in northwestern Pennsylvania is inadequate to sustain closed forest.

Regeneration during and after a tree decline is one of the least studied aspects of decline disease. For example, relatively little is known about the pattern of regeneration and succession that followed the massive mortality of birch throughout the 1940s and 1950s. Regeneration in declining red spruce forests in the Northeast requires better data on replacement species before a full understanding of the changes effected by decline is possible.

Two Theories on Causal Mechanisms of Decline Diseases

It is generally accepted that decline diseases are characterized by the widespread premature senescence and mortality of canopy trees over

a relatively short period, and by the absence of evidence of a single causal factor (Ciesla and Donaubauer, 1994). Also, it is generally accepted that environmental stressors, both abiotic and biotic, play an important role in the etiology of declines (Sinclair, 1967; Sinclair and Hudler, 1988).

The feature that distinguishes declines from other forest diseases is a chain of stress–response events that results in progressive deterioration in tree health and mortality in at least part of the population. The sequence begins with the increased vulnerability of the host (for reasons not always clear), followed by inciting or triggering stress (usually first evident in the form of one or more obvious decline symptoms), and by the additional stress of attacking disease organisms and insects.

There are several different theories or "models" of decline disease. A general point of agreement in all models is that stress has a major and unique role in the etiology of decline diseases. Another point of agreement is that declines are by nature complicated. Typically, multiple abiotic and biotic stresses act individually or in concert to amplify their effect. It is this multiplicity that marks forests as especially vulnerable to decline disease because of the plethora of simultaneous environmental changes now occurring in eastern North America.

Sinclair–Manion–Houston Models

Sinclair (1967) proposed three categories of "causal factors" [sic]: (1) predisposing factors that weaken trees and reduce their ability to tolerate adverse conditions, (2) inciting factors that trigger the decline event, and (3) contributing factors that intensify and perpetuate the disorder. In a recent version of their theory, Sinclair and Hudler (1988) listed four factors in decline causality: (1) perennial or continual irritation by one factor, (2) drastic injury plus secondary stress, (3) interchangeable predisposing and contributing factors, and (4) synchronous cohort senescence. They regarded the fourth as a variation of their third causality mechanism.

Manion (1991) proposed that the three factors in Sinclair's (1967) model interact. The result is a spiraling decrease in tree health, and frequently culmination in tree death. Houston (1967, 1981, 1992) proposed a stress-altered tree/secondary pathogen model for decline diseases. Stressors effect physical, physiological, and/or biochemical changes in healthy tissues and predispose trees to attack by secondary organisms (Houston, 1992). These changes result directly in disease or dysfunction if severe enough, or render tissues susceptible to pathogenic organisms (insects and microbes) that usually are resisted by the tree (Crist and Schoeneweiss, 1975; Schoeneweiss, 1978, 1981a,b; Wargo, 1972, 1975, 1988, 1996; Wargo and Houston, 1974). Mortality usually can be ascribed to attacks by one or more of these naturally occurring opportunistic organisms (Houston, 1992; Wargo, 1977, 1980, 1981a).

Houston's (1992) "predisposing factors" are equivalent to Sinclair's (1967) and Manion's (1991) "inciting factors" and many of their suggested "predisposing factors." Houston's (1992) "predisposing factors" are related to a narrower interpretation of the definition of predisposition, which means "to bring about susceptibility to infection." The "predisposing factors" in the Sinclair–Manion model influence the response of trees to the "inciting factor," but they do not necessarily predispose trees to infection.

Are the levels of abiotic and biotic stresses increasing in the Northeast? Do these changes indicate a greater or lessor risk of decline than historically, and into the future? There is the strong impression and considerable documentation on increasing levels of extreme climatic stress, lower air quality (especially oxides of nitrogen [NO_x] and ozone [O_3]), and pest outbreaks (including explosive deer population) in parts of the Northeast. These models imply that the increased levels of stress are interacting and adversely impacting the forests in the region. A higher level of decline disease is a likely outcome.

Mueller-Dombois Model

Mueller-Dombois proposed a natural dieback phenomenon due to synchronous cohort senescence as an alternative to the decline disease theory (Mueller-Dombois, 1983a,b, 1992; Mueller-Dombois et al., 1983). This theory focused on decline as a response to aging of cohort populations and to changes in stand demographics. The four additional components of the Mueller-Dombois (1992) theory are: (1) simplified forest structure, (2) edaphically extreme sites, (3) periodically recurring perturbations, and (4) biotic agents.

The synchronous cohort senescence model is compatible with the Sinclair–Manion–Houston "disease models" of decline. In the Sinclair and Manion models, synchronous cohort senescence would be a predisposing factor. In the Houston model, cohort aging is considered part of the forest relationships that influence the vulnerability of trees to stress-triggering events.

Many, if not most, forests in the Northeast are at or nearing biological maturity. Effective fire suppression, pest management and control measures, forest conservation, and a limited rate of harvesting since about 1930 have favored survival and increasing forest age. Auclair et al. (1997) noted that the timing of the first major successive episodes of dieback on ash, birch, sugar maple, and red spruce in the Northeast during this century corresponded closely with the species' age to maturity. Species populations that were young and vigorous early in the century did not show dieback despite high levels of freezing and drought stress (Auclair, 1997; Auclair et al., 1996). This observation is consistent with the concept of cohort senescence. It implies that as an increasing number of tree

species in the region reach biological maturity simultaneously, dieback will become extensive. The pattern of significant forest loss to dieback in the Northeast since the mid 1970s is believed to be due to multiple species having reached maturity (Powell et al., 1993) at a time of unusually high climate stress in the region (Auclair et al., 1996, 1997; Auclair, 1997). In addition, there have been expanded invasions by exotic insect pests as well as recent increases in epizootics of native insect pests. The increase in forest susceptibility to both exotic and native pests could reflect the aging of the tree species along with changes in species composition throughout much of the Northeast.

The cohort senescence theory implies that the high level of risk is likely to persist or even increase as forests age. Increasing levels of climatic stress, lower air quality (especially NO_x and O_3), and pest outbreaks in the region do not bode well for sustaining forest health of aging tree populations in the absence of strong countermeasures. A possible management strategy is to selectively increase the harvesting of mature stands and ensure their replacement with young forests of stress-resistant tree species.

History and Distribution of Decline Diseases in the Northeast

Decline diseases have occurred in most major tree species in the United States. Some examples include ash, beech (*Fagus grandifolia* Ehrh.), birch, maple, oak, pine (*Pinus* spp.), red spruce, and sweet gum (*Liquidambar styraciflua* L.) in the eastern United States (Houston, 1987). Episodes have been reported as early as the 1870s and probably occurred much earlier than that. The history of many of these decline disease episodes is reviewed in Houston (1987), Millers et al. (1989), Walker et al. (1990), and Auclair et al. (1997).

Relationship to Previous Disturbances

The development of decline diseases also may reflect the effect of previous disturbance. Widespread forest harvesting for timber, chemical wood, and firewood in the Northeast began about 1860 and continued until about 1930. In the 1860s, extensive areas in marginal cropland were abandoned to regrowth. At the same time, the use of railways was expanded, including the use of narrow-gauge rail lines to harvest hardwoods in terrain not easily accessed by rivers or lakes used for transport of timber (Bormann and Likens, 1979). Extensive disturbance in the 1860s appears to have sharply "synchronized" forest establishment, and hence the timing of maturation and decline of different tree species (Auclair et al., 1997).

In addition, the effects of previous disturbance history can be a significant factor affecting disease development. The extent and

magnitude of decline and mortality on oak (*Quercus* spp.) often concurs with areas formerly occupied by American chestnut (*Castanea dentata* [Marsh.] Borkh.), which was essentially removed from the forests by the chestnut blight fungus (*Cryphonectria parasitica* [Murrill] Barr). Both the dominance of oak and its occurrence on sites where it was excluded by chestnut increase the susceptibility of oak stands to persistent gypsy moth defoliation and decline (Healy et al., 1997). The majority of sugar maple stands experiencing decline in northwestern Pennsylvania may occupy "off-sites" or occur in much higher densities than before on sites colonized aggressively by maple after extensive cutting and burning early in the century (see Case Study 1 later in this chapter).

Timing and Episodicity

The onset and subsidence of decline diseases is typically rapid. Major episodes over large regions can erupt within 1 to 2 years and disappear with equal rapidity. The incidence of successive episodes of dieback and decline in the Appalachian region illustrates this pattern (Fig. 4.1a). Several characteristic features are evident. First, early episodes involved one or several species in each episode. Since the mid-1970s, many hardwood species have been affected simultaneously. Second, the onset date corresponds within 5 years to the estimated age to maturity of each species. Third, the severity and extent of dieback episodes has not changed over the century (Fig. 4.1a); in the more recent episodes, the number of tree species in the vegetation affected simultaneously has increased, giving the appearance of an increase in decline (Fig. 4.1b).

Spatial Incidence and Heterogeneity

Decline diseases typically show a high level of spatial variability. The fact that different age classes and different species are affected in different degrees results in a complex or heterogeneous appearance of decline within a forest stand. Even within the same population cohort, trees rooted to different depths or that occupy different soils may show differences in the severity and rate of disease development. Superimposed on this are subtle interactions among numerous stressors, genetic variations, opportunistic organisms, and predisposing factors.

Regional differences between decline episodes have not been well studied. Auclair et al. (1997) noted a tendency for episodes of hardwood dieback to be distinctly more severe and abrupt in the more northern areas. This possibly reflects the magnitude of the inciting/predisposing stress, the simplified composition and age structure of the northern populations, and differences in a wealth of site legacies, such as soil nutrient status, structure, and drainage, soil microbial populations, and disturbance history (Vogt et al., 1996).

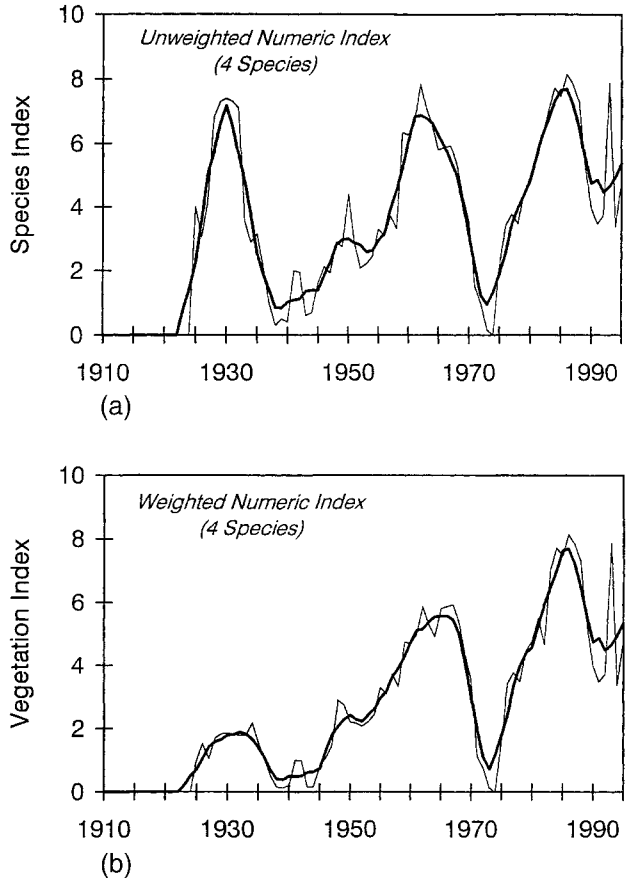

Figure 4.1. (a) Species index of forest dieback in Vermont from 1910 to 1995. This index is the sum of the numerical indices (Auclair et al., 1997) of four species (ash spp., birch spp., sugar maple, and red spruce) divided by the number of species showing dieback in any given year (i.e., unweighted by the number of species). (b) Vegetation index of forest dieback in Vermont from 1910 to 1995. This index is the sum of the numerical indices of the four species divided by four (i.e., weighted by the total number of species affected, 1910 to 1995).

Environmental Stress and Change in the Northeast

Primary Stressors

Lists of environmental factors that can act as primary stressors are found in Houston (1987), Manion (1991), and Millers et al. (1989). Drought and defoliation are the most common stressors associated with decline disease in the Northeast. Stress from sucking insects or defoliation from late spring frosts or fungal leaf pathogens also have been associated with

decline disease (Millers et al., 1989; Wargo, 1996). Defoliation by exotic insects has played a major role in oak decline (Wargo, 1981b, 1996). Defoliation by native defoliators, sometimes in combination with anthracnose fungi, has been related to decline episodes in sugar maple (Hall, 1995; Millers et al., 1989). Similar abiotic and biotic stressors have been associated with decline diseases in Europe (Ciesla and Donaubauer, 1994; Siwecki and Liese, 1991).

Primary Abiotic Stressors

The Northeast has experienced more change in climate, air chemistry, land use, site alterations, and other human impacts than any other region in the United States. The most significant of these include: (1) region-wide notable increases in mean annual temperature and precipitation over the century and a trend toward unseasonable and intense rainfall events since 1960 (Karl et al., 1996); (2) increased frequency and magnitude of winter thaw–freeze and root freeze events over the century (Auclair et al., 1996); (3) significant increases in tropospheric ozone (O_3), acidic deposition, and heavy metals from air pollution as well as increases in atmospheric CO_2 and NO_x concentrations; (4) widespread historic human disturbances, including forest harvesting, burning, cropland abandonment, rail and road construction, and suburbanization; extensive areas are off site following the reestablishment of aggressive tree species on sites not usually occupied by them under competition and gap replacement; (5) current extensive site and stand alteration through forest land use and management (including changes in species composition, such as the increased dominance by hardwoods, and changes in forest age structure), alteration of drainage patterns (e.g., by road construction), forest thinning, and forest harvest. Trees occupying borders of city and suburban streets and croplands and the introduction of exotic ornamentals represent a special but common variation in the Northeast.

Primary Biotic Stressors

Some insects and diseases can kill trees directly, but many cause damage that weakens trees and predisposes them to other insects or pathogenic organisms that ultimately kill the tree. Thus, their primary role is as a stressor of forest trees. Defoliating insects are the major members of this group, often causing spectacular, sudden damage over extensive forest areas in a diverse array of tree species, especially deciduous species (Millers et al., 1989; Houston, 1987). Sucking insects, such as adelgids, aphids, and scales, also have acted as primary biotic stressors and have triggered episodes of decline disease (Houston, 1987; Millers et al., 1989). Some foliar pathogens also can cause defoliation and predispose trees and trigger decline diseases (Hall, 1995; Wargo, 1996). These organisms rarely kill trees by themselves.

Interpreting the ecological role of these primary biotic stressors is complicated. Many of these organisms are nonindigenous and exotic to the ecosystem they are disturbing. These biotic stressors may be responding to abnormal abundance of certain tree species within a landscape or to species that are growing off site. Both situations can provide the trigger for pest outbreaks. Population dynamics of some of these organisms also are related to abiotic stressors, especially water deficits. Whether drought weakens trees and enables population growth of these organisms and subsequent attack, or whether drought enables population growth of these organisms that weaken the trees, is not clearly understood.

Secondary Organisms

Most of the pathogenic organisms (i.e., insect pests and traditional disease organisms) involved in decline disease syndromes are considered incapable of attacking and causing disease in healthy trees. Houston (1992) referred to these as "secondary-action organisms" to indicate their role as secondary in the time sequence of decline disease but not in importance to the syndrome. This group of organisms includes a variety of fungi and insects, and probably bacteria and viruses (not intensively studied) (Castello et al., 1995b) that can kill fine roots, buds, twigs, inner bark, cambium, and/or xylem on the major branches and boles of mature forest trees. Most of these organisms have narrow host ranges but some can infect a range of hosts.

The organisms that have been investigated and associated with important decline diseases of forest trees (1) are ubiquitous inhabitants of natural forest ecosystems whose evolved roles in the absence of major external stress events are ecologically beneficial, (2) are unable to succeed in living tissues not previously predisposed by stress, and (3) affect stressed trees principally by invading and killing meristematic regions of roots, stems, twigs, or buds (Houston, 1992). Secondary-action organisms act as ecosystem "roguers," killing individuals or groups of trees that have been stressed and are no longer fully functional members of the ecosystem (Wargo, 1995). Some secondary-action organisms also act in a dual role as scavengers. These decay the woody substrate they have killed, releasing additional resources for use by healthier existing or replacement trees (Wargo, 1995). The *Armillaria* root disease fungus is an excellent example of a roguer–scavenger organism (Wargo, 1980, 1995).

Have the abundance and/or aggressiveness of secondary-action organisms changed in the Northeast? The population levels of these organisms typically respond in proportion to the amount of tree mortality. Losses in forest volume to reduced growth and mortality due to recent dieback (1976 to 1995) increased 2.6-fold compared with the long-term historical (1910 to 1975) average (Fig. 4.2a). We can safely assume that the presence of secondary-action organisms has followed these upward trends. This trend of volume loss was close to the annual pest-related tree mortality

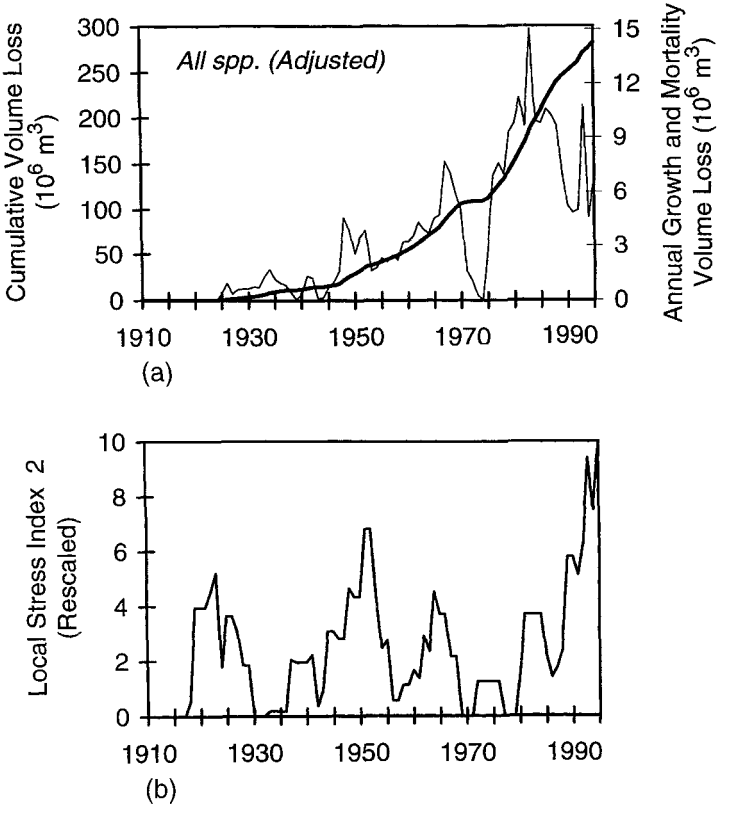

Figure 4.2. (a) Century trends in annual and cumulative loss to mortality and reduced growth due to dieback in the Appalachian region (Vermont, New York, Pennsylvania, West Virginia, Maryland) of the northeastern United States. (b) The local stress index based on extreme freezing and drought stresses at Burlington, VT, 1910 to 1995.

rates inventoried in northeastern hardwoods. The overall increase in tree mortality measured in permanent forest inventory plots from 1977 to 1991 was 2.2-fold compared with the previous 14-year period (1962 to 1976) (Powell et al., 1993).

Four Case Studies of Decline Diseases

Case Study 1. Biotic and Land Use Factors: Decline of Sugar Maple in Northwestern Pennsylvania

Multiple tree species are being affected by decline disease on the Allegheny National Forest in northwestern Pennsylvania. Sugar maple and red

maple (*Acer rubrum* L.) are in varying stages of decline, beech stands are declining in response to beech bark disease, butternut (*Juglans cinerea* L.) has declined sharply since the 1960s due to butternut canker, oaks are declining from persistent defoliation by the gypsy moth, and nearly pure stands of black cherry (*Prunus serotina* Ehrh.) are being defoliated by the cherry scallop shell moth (*Calocalpe undulata* L.), possibly setting the stage for a defoliation-induced decline of that species. Decline diseases reported in the past have been discrete episodes of "tree declines" or species declines involving one or several tree species (Millers et al., 1989). The current declines are exceptional in the large number of tree species being affected simultaneously.

In response to this mortality, except for beech and striped maple (*Acer pennsylvanicus* L.), stands are failing to regenerate back to forests because of heavy browsing by excessive populations of white-tail deer and competition from heavy forest-floor cover of ferns and grasses, also a result of excessive deer browsing. Closed-canopy forests are shifting to savanna-like vegetation with scattered trees and shrubs growing among ferns and grasses. This may be a unique situation or it may indicate what may happen in the future when stressors, exacerbated by global change, interact with biotic agents.

The current maple forests are the result primarily of production of charcoal and alcohol from massive clearcutting for chemical wood from about 1890 to 1930. The abundance of sugar maple is 3 to 5 times greater than its predisturbance composition of about 5%, and stands are of relatively similar age, that is, 70 to 90 years old (Whitney, 1999). These forests are experiencing significant decline disease in response to a variety of factors (Kolb and McCormick, 1993). Recently, stands have been defoliated by a number of native insects, including the elm spanworm (*Ennomos subsignarius* Hbn.) and the forest tent caterpillar (*Malacosoma disstria* Hubner), and one exotic insect, the pear thrips (*Taeniothrips inconsequens* Uzl.) (Long et al., 1997). Some stands have experienced up to four successive years of defoliation. Also, trees are being affected by the maple borer (*Glycobius speciosus* Say.), attacked very aggressively by several *Armillaria* spp. (Marcais and Wargo, 1998), and colonized by *Steganosporium*, a twig and branch canker fungus. In addition, the forest recently experienced significant drought in 1988, 1991, and 1995. Mortality can be as high as 65% in some stands, especially on dry and nutrient-poor sites.

Sugar maple decline is more severe in stands growing in unglaciated soils (>500,000 years ago) than in stands growing in recently glaciated soils (~12,000 years ago) (Horsley et al., 1999). Not only does the disease seem less severe on glaciated sites but also reproduction is greater and more vigorous. This has led to speculation that acidic deposition also might be a factor on unglaciated soils (Drohan and Sharpe, 1997; Sharpe and Sunderland, 1995).

Some stands are not regenerating back to sugar maple. This is primarily the result of excessive deer browsing but also from extreme competition for light from ferns and grasses in the understory. Millers et al. (1989) list a total of 65 different species of trees that have been affected by decline diseases in the eastern United States this century. In these cases, however, the affected forests, have characteristically and quickly regenerated back to forest tree species.

Case Study 2. Winter Injury and Acidic Deposition: Decline of Red Spruce in the Northeast

Midwinter thaws play a significant role in winter injury on red spruce (DeHayes et al., 1990; Friedland et al., 1984). Injury is related to needle freezing followed by drying rather than strictly to "acute frost desiccation" or "winter desiccation" (DeHayes, 1992; Hadley et al., 1991; Herrick and Friedland, 1990; Johnson, 1992; Johnson et al., 1992; Perkins et al., 1991; Vann et al., 1992).

DeHayes (1992) found that current-year foliage of red spruce exposed to experimentally induced winter thaws for five days dehardened by 5 to 14°C and did not attain full cold tolerance for at least five days. Cold tolerance of current-year foliage of red spruce was reduced by 3 to 14°C during a natural thaw in January (Strimbeck et al., 1995). Under a regime of subfreezing temperatures, pre-thaw cold tolerance levels were reestablished within 10 to 20 days.

Rapid drops in winter temperature after midwinter thaws (vs. extreme low temperatures) also have been implicated from field observations (Manion and Castello, 1993) of "winter reddening" of red spruce foliage. Perkins and Adams (1995) confirmed these observations experimentally. Curry and Church (1952) and more recently Tobi et al. (1995) reported that radial growth was reduced after winter injury. This reduction occurred in the growing season immediately after winter injury. The reduction of internode growth occurred in the third growing season after injury. This suggests that a series of winter injuries in succession or in close years can account for the reduced radial growth noted on red spruce since about 1960 (Cook and Zedaker, 1992). A lower resistance to other stresses also has been inferred (LeBlanc and Raynal, 1990).

The most recent research indicates that acidic deposition may enhance the probability of winter injury on red spruce (DeHayes, 1992; DeHayes et al., 1991; Fowler et al., 1989; Peart et al., 1991; Sheppard, 1994; Vann et al., 1992). DeHayes et al. (1991) demonstrated that cold tolerance was reduced from 3 to 5°C in seedlings treated with acidic mist. The role of acidic deposition in decline diseases is not completely understood. Aluminum-induced calcium depletion has been hypothesized as a major mechanism through which acid deposition causes decline in red spruce. This hypothesis is discussed in detail in Chapter 6.

Case Study 3. Extreme Climatic Fluctuations and Forest Age: Decline of Northern Hardwoods

Recent evidence indicates that forest maturity plays a pivotal role in the onset of decline diseases in northern hardwoods. Under high levels of climatic stress, it is apparent that tree populations will not die back unless they have reached or are nearing biological maturity or large size (Auclair et al., 1997). Once tree populations near maturity, they are vulnerable to extreme climate and other stressors. Auclair et al. presented convincing evidence that extreme climatic fluctuations have triggered major episodes of dieback in mature population cohorts of northern hardwood tree species as well as in other forest types and geographical regions (Auclair, 1987, 1993a,b, 1997; Auclair et al., 1992, 1996, 1997).

Forest Maturity

A comparative study of the actual onset dates of nine principal episodes of dieback across subregions of the northern hardwoods in the U.S. and Canada indicated a close relationship of maturity to the expected date of onset (i.e., within five years). The latter was based on the age to maturity (Millers et al., 1989), adjusted by a "lag interval" based on the tree's growth rate. This adjustment was necessary since the six subregions represented widely different temperature and precipitation regimes that affected growth and hence the rate at which the tree reached maturity (Auclair et al., 1997).

The implications of this finding are significant with respect to the question of decline diseases vs. environmental change in the Northeast. Prior to about 1975, trees did not decline in response to climatic and other environmental stressors until the populations of successive individual species had approached maturity. High levels of freezing and drought stresses early in this century (pre = 1925) failed to trigger major dieback events. Conversely, dieback and decline is now (post = 1975) widespread in response to both an aged forest structure and high levels of climatic stress (Auclair et al., 1996, 1997). The estimated levels of mortality since 1975 have been unprecedented; approximately 58% of all climate-related dieback this century has occurred since 1976 (see Fig. 4.2a). There is some question whether defoliator-induced diebacks have followed this pre = 1975/post = 1975 pattern (Millers et al., 1989).

Extreme Freezing and Drought Stress

Sudden freezing, such as prolonged winter thaw followed by a rapid return to subzero (°C) temperatures, and/or severe summer drought are likely to incite air emboli in sapwood and other irreversible damages to stem and root tissue (Tyree and Sperry, 1989; Cochard and Tyree, 1990). Root freezing can result in similar damages as demonstrated in field experiments

in which snow was removed or suspended above the soil surface over the winter months (Pomerleau, 1991; Robitaille et al., 1995). Root freezing as a result of deep soil frost triggered dieback symptoms on the test plots the following summer. The marked absence of snowpack over the 1930s, late 1940s to mid-1950s, the 1960s, and since the late 1970s (Fig. 4.3) concurs closely with the timing of severe and extensive dieback episodes in eastern Canada and the northeastern U.S.

Auclair (1997) developed an index of freezing stress (thaw freeze and root freeze) and one of drought stress (soil water deficit and heat stress) for the Northeast that extends over the century (1910 to 1995). The indices, based on extreme events evident in daily meteorological records at Burlington, Vermont, were combined to produce an overall annual estimate of the level of extreme climatic stress on forests of the region (see Fig. 4.2b). The combined "local stress index" indicated that climate stress during the 1990's has been significantly higher than historical levels, and that the onset of each principal episode of dieback occurred at a time of high climatic stress. Conversely, subsidence of dieback or "recovery" occurred when the levels of stress had decreased about one-tenth of the index maximum.

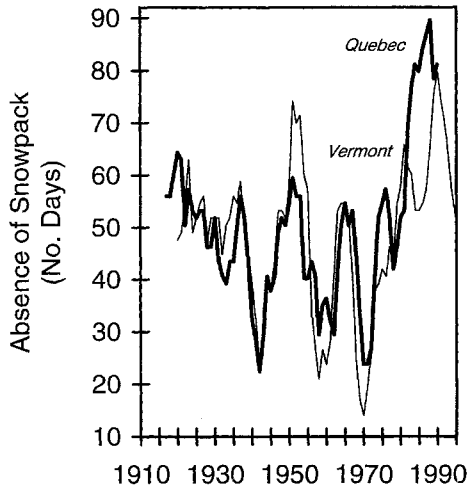

Figure 4.3. Five-year running mean of the number of days over the winter period (December to mid-March) with snowpack of 5 cm or less. The absence of snowpack in southern Quebec (Lennoxville) increased 57% and in northern Vermont (Burlington) 24% in the 1976 to 1995 period compared with pre-1976 levels. Peak levels (i.e., open winters) were intervals marked by significant dieback episodes in northern hardwoods.

Case Study 4. Global Climate Change: Simultaneous Declines in the Northeastern United States and Central Europe

In the mid-1970s, a general decline ("Waldsterben" and later "neuartige Waldschaden") occurred throughout central Europe, including Austria, Belgium, France, Germany, and Italy. Both conifers, such as Norway spruce (*Picea abies* [L.] Karsten), white fir (*Abies alba* Mill.), and Scots pine (*Pinus sylvestris* L.), and angiosperms, such as beech (*Fagus sylvatica* L.) and oaks (primarily *Quercus robur* L. and *Quercus petraea* Liebl.) were among the species reported to be affected (Schutt and Cowling, 1985).

In the eastern United States, red spruce was reported to be declining and dying throughout its range in the Appalachian Mountains in the late 1950s, and again in the mid-1970s (Bruck, 1984; Siccama et al., 1982; Vogelmann et al., 1985; Weiss et al., 1985). Surveys throughout the Appalachian range indicated that mortality was greater in high-elevation stands (>800 m) (Craig and Friedland, 1991; Johnson and Siccama, 1983; Miller-Weeks and Smoronk, 1993; Scott et al., 1984; Weiss et al., 1985).

The simultaneous events in Europe and North America in and after the mid-1970s led to much speculation about their cause. At the time, it was believed that an unprecedented decline of forests that occurred in the late 1970s and early 1980s was attributable to air pollution effects (Skelly and Innes, 1994). Although there was lack of clear evidence for traditional stressors and opportunistic pathogens to be involved in these declines (Carey et al., 1984; Wargo et al., 1987, 1993; Bruck, 1989; Weidensaul et al., 1989), there was also lack of clear evidence that air pollutants, particularly acid deposition, was involved (Skelly and Innes, 1994; Kandler, 1990, 1992a,b, 1993).

Working with global climate phenomena, including anomalies in the global mean annual air temperatures and El Niño–Southern Oscillation Index, Auclair proposed that global climate change was responsible for the simultaneous forest declines in geographically distant regions (Auclair, 1987; Auclair et al., 1992, 1996). Fig. 4.4 identifies the onset of major diebacks in northern hardwoods (northeastern United States, southeastern Canada) and in central Europe in relation to changes in global mean annual temperature. The onset of successive dieback episodes coincided with rapid temperature increases; recovery occurred as global temperatures relaxed or dropped from previous highs. It was assumed that periods of rapid change in global temperature were marked by intervals of exceptional climatic stress at the regional level, both in North America and in Europe.

In Fig. 4.5a, anomalies in global temperature (from the 1910 to 1995 mean annual temperature) and in the Southern Oscillation Index (i.e., 1000 mb pressure difference between Tahiti and Darwin, Australia) were combined to give an integrated estimate of the risk to dieback from

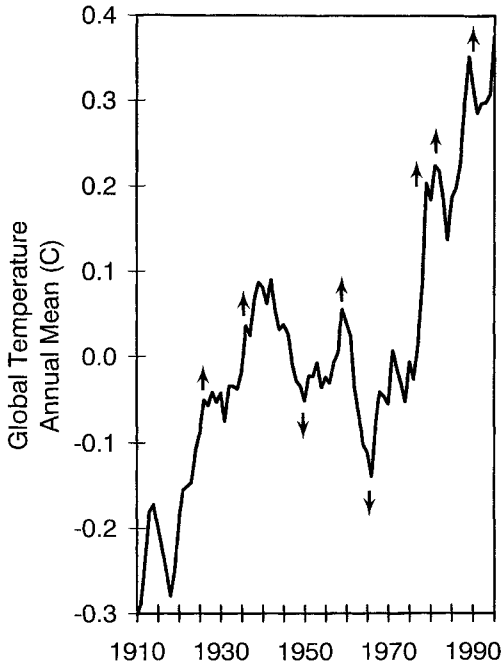

Figure 4.4. Relation of global warming (i.e., five-year running mean of the global mean annual temperature) to the onset (↑) and recovery (↓) of forest dieback episodes in eastern North America and central Europe.

climatic stress. Especially evident is the high level of current climatic stress; from 1976 to 1995, inclusive, the index was 6-fold higher than historically (i.e., from 1910 to 1975). Fig. 4.5b shows the close relationship between the global stress index and the incidence of local freezing events in the northeastern U.S. (Burlington, VT). As in the northeastern U.S., extreme fluctuations in winter temperatures have been implicated in triggering the extensive forest declines in Europe from the mid-1970s onward (Auclair, 1993a; Hartmann and Blank, 1993; Hartmann et al., 1991).

Future Research Needs

It remains important that forest agencies and managers in the Northeast have access to a rich array of accurate, timely information on forest condition and health. Potential benefits are improved capability to anticipate and manage effectively decline at a time of rapid change. Three areas in which research can facilitate this goal are:

1. Development of Geographical Information Systems (GIS) Regional Maps and Early Warning System (EWS) of Risk to Forest Health.

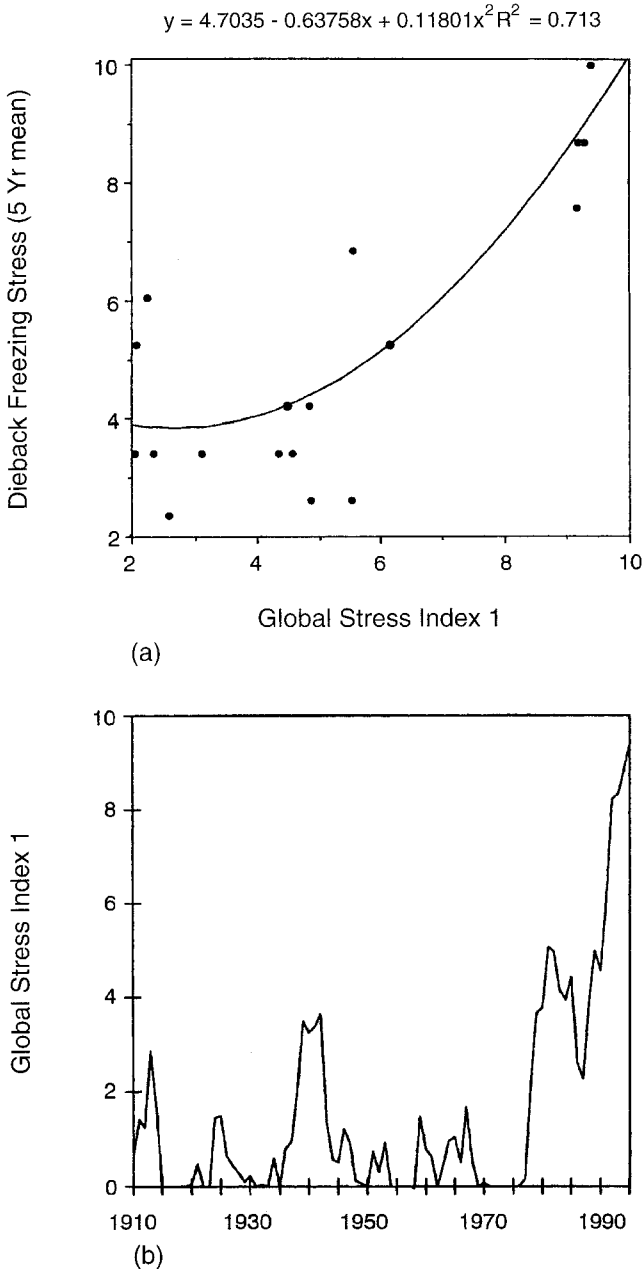

Figure 4.5. (a) Century trends in the global climatic stress index based on the average of the anomalies in global mean annual temperature and the Southern Oscillation Index. (b) The relation of the index of dieback freezing stresses (at Burlington, VT) to the global stress index. A strong relationship is evident, especially at values >4.0. Values are five-year running means of each index.

A GIS/EWS for risk of dieback and decline could identify "hot spots" in the Northeast that historically have been at risk to dieback injuries, the "real-time" situation now, and how the risk of dieback and decline has changed and is changing as global warming and other stresses continue. The EWS would alert managers and the public to expected events and impacts on health of the forests. Anticipatory planning is a decided advantage in adjusting both expectations and management actions to minimize the impacts.

2. Development of a suite of indicators of forest susceptibility to stress and vulnerability to disturbance from stressor–host–pathogen interactions. Several biochemical, physiological, dendrochemical, and microbial methods show promise as early warning indicators. However, further evaluation is needed before they can be developed into useful tools for land managers.
3. Evaluate the factors (site, species, stress, pathogen, etc.) that affect the spatial and temporal development of decline disease within forests and across landscapes. Tree–host genetic variability in relationship to susceptibility and vulnerability to stress and pathogens is poorly understood. Legacy factors (Vogt et al., 1996) such as previous disturbance from insects, fire, pathogens, forest management, and land use changes, and how they have influenced tree species abundance and distribution on the landscape, must be evaluated.
4. Development of management prescriptions to reduce damage and growth/volume loss to decline diseases. One option is to "weatherproof" and "pestproof" the forest by selectively identifying areas/locals at high risk and treating them in such a way as to minimize injury and mortality. For example, actions may include

- Increased monitoring of areas identified to be at high risk to ensure rapid response by managers and forest agencies. Links with the Forest Service's Forest Inventory and Analysis Program and the Forest Health Monitoring Program are needed.
- Specialized observations on the etiology of disease, dieback, and decline. This could include successive measurements of the inciting injury, moisture stress, infection by disease organisms, insect outbreaks, growth loss and mortality, and tree regrowth/recovery processes.
- Salvage harvesting in advance of or early in the dieback episode to ensure high quality and economic value of wood products (i.e., free of pests, fungi, decay).
- Harvesting of "stands at risk" and the replacement of these with trees that are younger, more vigorous, and more resistant to freezing, drought, and other stresses.
- Management practices that discriminate against tree species off site and favor those adapted to site and timber stand.

- Thinning or improvement cuts to reduce competition for moisture on drought-prone sites.

5. Process-level climate experiments (in the laboratory and in the field) designed to better identify the action and mechanism of stressors (e.g., role of freezing stresses in the development of irreversible emboli).

Conclusions

Forest scientists have made significant progress in understanding the role of natural and some anthropogenic stressors in decline disease. The major challenge will be to sort out declines due to natural factors from those due to anthropogenic factors, as well as those resulting from the interactions of both types of factors. Natural stressors, such as persistent defoliation, drought, and frost, have triggered episodes of decline. As global atmospheric chemistry and climate change, the potential for natural and anthropogenic stressors interacting to affect forest health is likely to increase. The extent and severity of forest declines has been high since the mid-1970s. During this period, much new information and insight into decline processes has opened the possibilities for predicting the incidence of decline and for better managing and adapting forests to future conditions.

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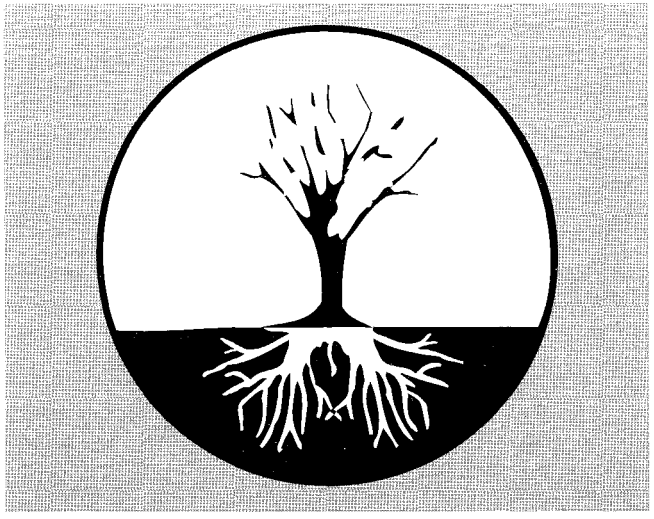

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