

History of Sugar Maple Decline

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Abstract

Only a few episodes of sugar maple dieback or decline were recorded during the first half of the 20th Century. In contrast, the last 50 years have provided numerous reports of both urban and forest dieback/decline. In the late 1950s, a defoliation-triggered decline, termed maple blight, that occurred in Wisconsin prompted the first comprehensive, multidisciplinary study of a sugar maple decline. That research, and other investigations since, provided the conceptual framework for a model of sequential, stress-initiated cause and effect for dieback/decline disease. Many cases of urban maple dieback/decline have been attributed to soil compaction, drought, impeded soil water availability, or toxic effects of road deicing salt. Most cases of forest or sugarbush decline have been associated with the initiating stresses of insect defoliation or drought, singly or in concert. Mortality of stressed trees is often caused or hastened when roots or twigs are invaded by opportunistic, secondary organisms, especially the root rot fungi *Armillaria* spp. (and probably *Xylaria* sp.). In the past two decades, freezing of roots associated with periods of thaw-freeze and of deep cold, especially when snow cover was minimal or lacking, have been correlated with major decline episodes in eastern Canada and northern New England and New York. An hypothesis that dieback results when death of roots leads to transpiration-stress and vessel cavitation is supported by observations that dieback/decline episodes attributed to droughts appear correlated temporally with prior root-freeze events. Such events are now believed responsible for the serious maple dieback/decline problems in southern Quebec in the 1980-1990s that at first were hypothesized to result from atmospheric deposition. While atmospheric deposition has been discounted as a direct cause of maple declines, the long-term and perhaps complex effects on tree health of deposition-hastened changes in soil chemistry, especially in areas with soils susceptible to acidification, are the primary subjects of current investigations.

Introduction

Sugar maple (*Acer saccharum* Marsh.) has many highly valued qualities. Its long life, pleasing form, and brilliant fall color have made it a favored tree for gracing dooryards and roadsides from New England to the Lake States. Its hard, but easily-worked, light-colored wood is widely used for flooring, furniture, and many specialty products. Wood with uniquely figured patterns, including "bird's eye", "curley", or "fiddleback" is highly prized for fine woodworking. Further, the romanticism and economic values associated with spring sapflows and maple sugaring are as strong today as in colonial times.

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Certain ecophysiological characteristics of sugar maple have made it easy to exploit these values. Sugar maples fruit prolifically; seeds, which mature in the fall, are readily dispersed by wind and germinate the following spring. Well over 5,000,000 seeds per acre are common in good seed years and, establishment is often highly successful. Carpets of young seedlings are common, and the ability of shade-suppressed seedlings and saplings to respond when released has enabled the species to become a predominant component of many forest types. Sugar maple does best on deep soils that are moderately coarse-textured, moist and well-drained (Godman et al. 1990).

Sugar Maple Declines

Although records are lacking, it is highly probable that sugar maple has long experienced serious episodes of dieback/decline. Many of the environmental stresses affecting today's forests occurred in pre-European settlement times; droughts, insect defoliation, fire, damaging winds, and ice storms were no strangers to those early forests. Although opinions vary, the effects of these disturbances, especially, perhaps, fire and windstorms, created mosaics of both uneven-aged and even-aged forests of differing successional stages (Clawson 1983, Loucks 1983). Old, uneven-aged stands contained a high proportion of mature and overmature trees—those considered most susceptible to many of the stress factors that trigger declines. As colonization ensued, activities such as logging, clearing, burning, pasturing, and sugaring intensified dramatically. Areas best suited for tree growth were often those most desired for agricultural uses. Much of the old growth forest was removed, especially during the latter half of the 19th century. Reestablishment of forests on land withdrawn from agriculture has resulted in large areas of relatively even-aged forests that, during the latter half of the 20th century purportedly began reaching an age of increased susceptibility to stress events.

While a few reports of maple dieback/declines appeared in the first half of the 20th century (Hartley and Merrill 1915; Marsden 1950; McKenzie 1943), it was not until after 1950 that accounts of such problems become numerous. Several reviews present the chronologies and presumed causes of sugar maple dieback/decline episodes (e.g. Allen et al. 1992; Houston 1985, 1987; McIlveen et al. 1986; Millers et al. 1989; Sinclair 1964; Westing 1966). The purpose of this brief paper is not to restate what was presented in those reviews, but, rather, to discuss the main themes that seem most pertinent to the present situation.

The Nature of Sugar Maple Dieback/decline

One conceptual model of sugar maple dieback/declines in forest situations was structured around the premise that disease manifestation (progressive crown dieback sometimes leading to continued tree decline and death) results when one or more predisposing (*sensu stricto*) stress

factors reduces resistance to invasion by opportunistic, secondary-action organisms that result in death of tissues—sometimes of trees (e.g. Houston 1981, 1992). This model evolved initially from research on “maple blight”, a dieback/decline of sugar maple in northeastern Wisconsin, triggered by insect defoliation (Anonymous 1964, Giese et al. 1964). A complex of insects including several species of leafrollers and the maple webworm, *Tetralopha asperatella* (Clem.) caused severe defoliation on about 10,000 acres in the mid-late 1950s (Giese and Benjamin 1964). Dead and dying trees and saplings usually were attacked aggressively by *Armillaria* sp. (Houston and Kuntz 1964). Severely damaged stands, prior to being defoliated, had low basal area and density, and a high proportion (> 50%) of sugar maple. During the 10 months prior to the onset of mortality, the region had below-normal precipitation (-8.3 inches) (Skilling 1964).

Later studies elsewhere confirmed the defoliation stress/*Armillaria* association and clarified the biochemical basis for the lowered resistance of defoliation-affected tissues to attack by this opportunist (Parker and Houston 1971; Wargo 1972; Wargo et al. 1972; Wargo and Houston 1974). Another opportunist, *Steganosporium ovatum* (Pers.) S.J. Hughes, appeared to hasten the death of defoliation or drought-stressed twigs and branches (Hibben 1959, Wargo and Houston 1974). Research on several other stress-initiated problems (e.g., Appel and Stipes 1984, Ehrlich 1934, Houston 1994a, Schoeneweiss 1981a, b, Wargo 1977, 1983) has validated the chronological and spatial premises of the following simple, general model for dieback/decline diseases:

1. Healthy trees + stress → Altered trees (tissues) (dieback begins)
2. Altered trees + more stress → Trees (tissues) altered further (dieback continues)
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- n. Severely altered trees (tissues) + organisms of secondary action → Trees (tissues) invaded. (Trees lose ability to respond to improved conditions, decline, and perhaps die.)

For the defoliation-triggered sugar maple dieback/declines just described, the model would be:

1. Healthy sugar maple trees + defoliation → Sugar maples altered (dieback begins)
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- n. Altered trees + *Steganosporium ovatum* → Twig dieback accelerated + *Armillaria* sp. → Roots, root collars invaded, trees decline, die.

In these statements, the numbers refer to sequential episodes of stress events and host response; “n” indicates that at some point or degree of host change, organisms of secondary

action are able to invade altered tissues successfully. The model above indicates that although host changes sufficient to allow organism attack can occur after a single severe stress event, such changes usually follow multiple or repeated events. Arrows are to be read as “leads to”.

The statements of this model can be construed as summaries of several important relationships:

- i) Dieback of trees or tissues often results from the effects of the stress factor(s) alone. With abatement of stress, and in the absence of significant colonization by saproogens or secondary insects, dieback often ceases and trees recover. The dieback phase can be viewed as a survival mechanism whereby the tree adjusts to its recently encountered adverse environment.
- ii) Stress alone, if sufficiently severe, prolonged, or repeated, can cause continued or repeated dieback and even death. Numerous reports exist of tree mortality following either unusually severe and prolonged drought or episodes of severe defoliation, especially if repeated, perhaps even in the same growing season. Even one severe defoliation occurring concomitantly or sequentially with drought can result in high tree mortality.
- iii) Usually, however, the decline phase, wherein vitality lessens and trees succumb, is the consequence of organism invasion of stress-altered tissues. Recovery from this phase depends on many factors including the vitality of the tree, the particular tissues invaded, the relative aggressiveness of the organism(s), and the degree of invasion.
- iv) Where and when the dieback phase occurs is closely related to where and when the triggering stress(es) occurs. The decline and mortality phase is related, in addition, to the temporal and spatial distributions of the organisms of secondary action.

These summary statements point out the inherent difficulty in separating “dieback” from “decline”. Such a separation seems difficult and arbitrary—for most dieback/declines reflect complex continuums of host responses to successive and/or multiple events.

Temporal-spatial Patterns of Sugar Maple Dieback/declines

Comparisons of reported major episodes of sugar maple dieback/decline provide a picture of when and where trees have been affected and the stress factors or events that triggered them. For example, sorting the reports listed by Millers et al. (1989) by place, i.e., by urban or roadside (Table 1) vs. forest or sugarbush (Table 2), reveals that in eastern U.S., the few early (pre-1950) episodes were limited to urban/roadside problems that were triggered (as have subsequent problems in these arenas) by the effects of such stresses as drought, road deicing salt, soil compaction, and occasionally by *Verticillium* wilt, *Phytophthora* and *Fusarium* cankers, and *Armillaria* root disease (Table 1).

Table 1.—Chronology of dieback, decline, mortality problems of sugar maple reported for roadside or urban situations (adapted from Millers et al. 1989).

Dates	Location	Cause
1912-13	Washington DC - New England	drought
1939-49	Massachusetts	drought, defoliation
1956-58	Lake States	drought, <i>Verticillium</i> , <i>Phytophthora</i> , <i>Armillaria</i>
1950-60	Northeast	drought, salt, defoliation
mid-60's	Michigan	drought, salt
1968-70	Northeast	salt, pollution (?)
1975-78	Maine	mech., soil comp., pollution (?)
1976	Lake States, Northeast	drought, salt, compaction
1977-82	Wisconsin	<i>Fusarium</i> , <i>Phytophthora</i>
1978-81	Minnesota	drought, <i>Verticillium</i> wilt
1982	Missouri	?
1984-86	Iowa	?

Table 2.—Chronology of dieback, decline, mortality problems of sugar maple reported for forest trees and sugarbushes (adapted from Millers et al. 1989).

Dates	Location	Cause
1951-54	NY, VT	defoliation (Forest tent)
1956-58	MA	drought
1958-62	WI	defoliation/ <i>Armillaria</i> (leaf rollers, webworm)
1958-62	Lake States	high water tables
1950-60's	Northeastern US	drought, etc.
1957-67	CT	defoliation, (gypsy moth, spanworm)
1968-71	NY, ME	defoliation (saddled prominent)
1973	PA	defoliation (forest tent caterpillar)
1977	MI	defoliation (forest tent caterpillar, saddled prominent)
1978-81	MN	drought
1980-82	NY	defoliation (forest tent caterpillar)
1981-85	NH	defoliators
1984	MN, MI	drought, <i>Agrilus</i>
1984	NY	defoliation
1984-85	PA	defoliation, poor sites, thrips, anthracnose
1985	MA	?
1984-86	VT	defoliation (forest tent caterpillar)
1988-89	WI, MN	drought
1980-90	Quebec, NY	thaw-freeze, root-freeze

In contrast, the principal stress factors reported as triggers of major dieback/decline problems of forest and sugarbush, (noted only since 1951 in U.S.), are insect defoliation and drought, singly or in concert. Saprogens involved in forest decline situations have included *Armillaria* spp., *Agilus* spp. and anthracnose fungi (Table 2).

Accounts of maple dieback/decline from Canada generally have paralleled those from the U.S. Thus, except for episodes of dieback in the Beauce region of southern Quebec in 1932 (Pomerleau 1944) and elsewhere from 1937 to 1949 (especially from 1946 to 1949) (Pomerleau 1953), and in Ontario in 1947 (McIlveen et al. 1986), few accounts appeared prior to 1950. Most of the early episodes were triggered by insect defoliation, but a few were associated with the drought periods of the 1930s and later.

Since 1950, episodes of maple dieback/decline associated with insect defoliation, drought, logging, and more recently, with root freezing have increased in number. The relationship between root freezing (associated with deep soil freezing during times of low or absent snow cover) and the onset of dieback has received increasing attention in Canada and northeastern U.S. (e.g. Lachance 1985, Bauce and Allen 1991), has been replicated experimentally (Pomerleau 1991, Robitaille et al. 1995), and has been proposed as a major factor triggering maple decline in eastern Canada (e.g. Auclair et al. 1992). Drought and root freezing have been proposed as causes of irreversible cavitation in sapwood vessels that, in turn, prevents water movement (Auclair 1993, Auclair et al. 1992, Sperry et al. 1988, Tyree and Sperry 1989). Impairment of water conduction is believed to be responsible for crown dieback.

Usually, close examination of dieback/decline problems, even in remote areas, has revealed evidence for the prior occurrence of stress factors that singly, or in combination, are known to initiate dieback and also to render tissues susceptible to opportunists whose attacks can prevent recovery and hasten tree decline. Prior management practices (e.g., thinning) (Kelley 1988) and climatic episodes (e.g. drought) (Allen 1987, Bauce and Allen 1991) have been correlated with increased mortality following defoliation. It is probable that variations in trees' response to stress can occur locally due to differences in genotypes, local differences in tree vigor, patterns of stress occurrence, presence or absence or vigor of opportunistic organisms, or variations in site quality.

Sometimes imbedded within the maple decline complexes are two diseases caused by primary pathogens: *Verticillium* wilt, a vascular disease caused by *Verticillium dahliae* Kleb. which has been noted only in urban situations, and sapstreak, also a vascular disease, caused by *Ceratocystis virescens* (Davidson) C. Moreau, which rarely has been observed other than in forests and sugarbushes. Although caused by a primary pathogen, sapstreak should be considered part of the decline complex based on symptomatology and the facts that (1) injuries to roots or root collars are necessary as infection courts for *C. virescens*, and (2) tree mortality is almost always associated with

attacks by *Armillaria* sp. or *Xylaria* sp. (Houston 1993, 1994b). The importance of injuries for sapstreak infection creates a close temporal-spatial relationship between human activities and disease development. Thus, most diseased trees are located adjacent to skid trails or woods roads, and they develop symptoms within 1 to 4 years after infection. Recognized in North Carolina and Tennessee in the late 1930s and early 1940s (Hepting 1944), sapstreak was not reported from the Lake States until 1960 (Kessler and Anderson 1960) or from the northeast until 1964 (Houston and Fisher 1964). Whether recent increases in reported cases of sapstreak represent increases in disease incidence or in disease recognition is not known.

The ability of sugar maple to dominate favorable forest sites was noted earlier. Sometimes, however, this species colonizes sites unfavorable for later growth and development. For example, stands of sugar maple have developed on many abandoned fields in New England and New York. It is on these sites, often too wet, too dry, or nutrient impoverished, and along roadsides, that many sugar maple decline problems have occurred. Favoring sugar maple on wet, cool, bottom lands can create an unstable situation, as the species is neither long-lived nor vigorous on wet or dry soils and is extremely sensitive to abiotic or biotic stresses under such conditions. Widening and paving roads certainly have affected roadside maples adversely, and the added insult of road salt has created an intolerable environment for this mesically adapted, nutrient demanding species (LaCasse and Rich 1964). Trees along roadsides are prone to damage from drought events not sufficiently severe to affect forest trees.

Ironically, it was the dieback and deterioration of roadside trees that prompted a major research program on maple decline in Massachusetts in the early-mid 1960s (Westing 1966). Forest researchers were put in the position of trying to characterize a problem that at that time did not exist in the forests of Massachusetts. From this effort, however, came the initial thrust to understand soil nutrient - maple tree condition relationships (Mader and Thompson 1969).

The Stresses

Defoliation.—Effects of defoliation are addressed elsewhere in this symposium. Comments here are limited to pointing out that the consequences of defoliation stress were intensively investigated in studies of maple blight in Wisconsin (Anonymous 1964, Giese et al. 1964). Research on this problem comprised the first truly multidisciplinary investigation of a sugar maple decline. Those investigations, together with several since (Bauce and Allen 1991, Parker and Houston 1971, Wargo 1972, Wargo and Houston 1974) revealed the complex nature of host response and secondary organism attacks that characterize a dieback/decline disease and as described earlier, provided the model used as a framework for study of dieback/decline diseases of many tree species (Houston 1981, 1992).

Defoliation can affect all age classes, and even young trees exhibit twig and branch dieback that can progress with

repeated defoliations. Death of young defoliated trees is usually the consequence of root invasions by opportunists, especially *Armillaria* sp. Such killing attacks of young trees may occur in forest situations, even after a single defoliation, where abundant and vigorous opportunist populations occur. Where such populations are absent, tree mortality may not occur, even after repeated defoliations (e.g., Gregory and Wargo 1986, Parker and Houston 1971).

Drought.—Throughout this century, drought has been cited as a cause or a possible contributing factor of maple dieback/decline (Bauce and Allen 1991, Griffin 1965, Hartley and Merrill 1915, Hibben 1962, 1964, Marsden 1950, Ohman 1969, Sinclair 1964, Skelly and Wood 1966, Skilling 1964, Westing 1966). Beginning in the 1950s, notable episodes of maple dieback/decline occurred during or following periods of severe water shortage (e.g., Table 2). Observations that defoliation episodes, that are coincident or closely followed by drought are especially devastating (e.g. Allen 1987), are paralleled by the recent analyses by Auclair et al. (1996) suggesting that the effects of root freezing are especially damaging if followed by drought. Dieback/decline appears to result when desiccation of branches and re-leafing tissues (following defoliation), or of the first spring flush of leaves (following winter root freeze), is enhanced when conducting tissues are injured or killed by cavitation. The biochemical changes in sugar maple caused by drought and defoliation are similar (Parker 1970), and these changes favor growth and invasion by *Armillaria* spp. (Wargo 1972, Wargo and Houston 1974). Thus, because of their effects on host-defense systems, combinations of stress factors render trees exceptionally vulnerable to lethal attacks by opportunistic organisms.

Extremes of Temperature.—In northern Wisconsin, fall frosts killed immature leaves and terminal buds that formed after a midsummer defoliation and thus contributed to branch and twig dieback (Houston and Kuntz 1964). Other cold events also have been associated with maple dieback and decline. Episodes of thaw-freeze and of deep cold during snow-free winter periods were associated with diebacks of sugar maple and other species (e.g. Pomerleau 1944, 1991). These events apparently occurred commonly in the first half of this century yet, major diebacks did not occur during that time, presumably because tree populations were relatively young (Auclair et al. 1992, Auclair et al. 1996). Thus, forest maturation is postulated as a key factor preconditioning trees to climatic injury and dieback (Auclair et al. 1996, 1997). The recent re-recognition that root freezing is an important factor in northern forests stems largely from the work by Auclair and coworkers (e.g., Auclair et al. 1992, 1996) in Quebec, and by Bauce and Allen (1991) in New York. Auclair et al.'s (1996) analysis of climate data suggests that episodes of forest dieback are correlated with heat and drought stress but only after forests have been affected by root-freezing events. According to Auclair et al. (1996), crown dieback reflects drought effects in trees injured previously by freezing.

Acidic Deposition.—In the 1980s concern arose that atmospheric deposition, especially acidic deposition, was

causing sugar maple decline in Ontario, Quebec, and Vermont (Carrier 1986, McLaughlin et al. 1985, Vogelmann 1982, Vogelmann et al. 1985). Surveys and studies showed damage to sugar maple on a number of sites with soils deemed highly susceptible to acidification (e.g., the Muskoka area in southern Ontario, the Beauce region of southern Quebec and Camel's Hump in Vermont). Observers promoting acid-deposition hypotheses tended to dismiss such factors as defoliator outbreaks, climatic events such as early thaws or droughts, or disturbances caused by harvesting or tapping as the primary or sole cause. Attention was focused narrowly on atmospheric deposition. Subsequently, it was realized that the rather sudden appearance of dieback and decline in the Canadian forests was not the result of a direct effect of acid deposition, but rather a consequence of one or a combination of several factors previously associated with dieback/declines, especially the winter freeze-thaw events during periods of little snow cover (e.g., Pitelka and Raynal 1989, Auclair et al. 1992). Concern remains that acidic deposition may play a role in certain cases of sugar maple decline, especially as it may influence the chemistry of soils susceptible to acidification over the long term. That concern is strongly demonstrated by the emphasis placed on soil chemistry relationships at this symposium.

Synthesis

The fact that few major episodes of maple dieback/decline occurred during the first half of the 20th Century suggests that either there were fewer or less severe predisposing stresses, or that the forests then were more resistant or resilient to such stress. Arguments for the latter seem most plausible if, as Auclair et al. (1996, 1997) suggest, younger trees are less susceptible than mature ones to cavitation-inducing climatic events. Correlations of dieback episodes and a number of weather/climate indices, both local and global, support that hypothesis (Auclair et al. 1996, 1997). The paucity of reported sugar maple dieback episodes early in this century, even though there were frequent and severe winter-thaw-freeze and root-freeze events, as well as significant drought periods, presumably stems from the fact that in most northern hardwood forests, maple had not reached its susceptible age (= commercial maturity) of 100 years (Auclair et al. 1996) or 150 years (Millers et al. 1989) following the massive harvesting that occurred between 1860 and 1890. Under this scenario, forest (species) maturation would seem critical.

On the other hand, old-age may be less critical when stresses other than climate extremes are involved. Defoliation was the initiator of maple blight, but fall frosts, drought, and root pathogens, especially *Armillaria* sp. were all involved in the dieback, decline, and mortality of defoliated trees. Outbreaks of a unique suite of defoliators began in "young" stands soon after unusually heavy harvesting of older trees had opened up the forest and stimulated sapling growth. As the insect populations increased and spread, all ages were defoliated—and all ages suffered heavy mortality. Perhaps forest stand "maturation" is not only a matter of physiological

predisposition to cavitation of individual trees, but also of the abundance and condition of secondary-organism populations within "mature" stands.

Whether, or how, outbreaks of defoliating insects are associated with the climate measures utilized by Auclair et al. (1996) needs to be determined. Populations of some defoliators are favored by hot, dry summers, and the concomitant or successive occurrence of defoliation and drought has proven disastrous to sugar maple (Ailen 1987). Finally, it is clear that natural changes in soil chemistry, especially acidification, when augmented by acidic deposition, may significantly affect growth and tree resistance to stresses and opportunistic organisms. How such edaphic factors influence or are influenced by the stress factors known to severely affect sugar maple remains unclear.

Conclusion

Most of the maple dieback/declines that have been studied intensively were initiated by severe, acute stress factors, such as defoliation, drought, or winter root freezing. The concomitant or sequential occurrence of these in various combinations contributes strongly to the acuteness and severity of host responses.

Understanding of cause-effect relationships and underlying mechanisms is increasing. For example, the emerging hypothesis that dieback following certain stresses is a consequence of vessel column cavitation is balanced by climate models which suggest that cavitation may not be especially serious unless followed by severe drought. These concepts must be evaluated in light of current understanding of the role of secondary-action organisms as agents that impair recovery and cause mortality of stressed trees.

The focus now and for the immediate future, and which is emphasized in this symposium, is on soil relationships. It is clear, however, that knowledge gained from the past needs to be carefully interpreted in light of what already is known here about histories of management, drought, defoliation, and root pathogens. All of these factors are part of the picture. From the point-of-view of a forest dieback/decline researcher, it does not get any better than this!

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