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TREE DECLINES: FOUR CONCEPTS OF CAUSALITY[1]

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Abstract. The term *decline* refers to premature progressive loss of vigor and health, not necessarily to any specific disease or disorder. Any case of decline can be explained on one of four bases. 1. A tree may decline primarily as the result of chronic irritation by a single agent. 2. A tree may decline because of damage by secondary agents after an injurious event such as defoliation or major wounding. The same agents would not cause decline in an uninjured tree, and the injury alone would not cause decline. 3. Chronic irritation by one or more agents may diminish the tolerance or resistance of a tree to another agent that then incites decline. Various factors including those that predisposed the tree and incited decline may then contribute to further decline. 4. Trees of similar age growing in groups tend to display group behaviour including premature senescence (synchronous cohort senescence) in response to stress.

Résumé. Le terme "dépérissement" réfère à une perte de vigueur et de santé prématurée et progressive, non associée nécessairement à un insecte ou à un pathogène spécifique. Tous les cas de dépérissements peuvent être expliqués par une des quatre raisons suivantes. 1. Un arbre peut dépérir suite à une irritation chronique par un agent unique. 2. Un arbre peut dépérir dû aux dommages causés par un agent secondaire après un événement préjudiciable tel qu'une défoliation ou une blessure majeure. Les mêmes agents ne causeraient pas de dépérissement à un arbre en santé et la blessure seule ne causerait pas de dépérissement. 3. L'irritation chronique par un ou plusieurs agents peut diminuer la tolérance ou la résistance d'un arbre à un autre agent, qui lui, entraîne un dépérissement. Plusieurs facteurs, incluant ceux qui ont prédisposé l'arbre et entraîné le dépérissement peuvent ainsi contribuer à accentuer le dépérissement. 4. Les arbres d'un même âge, croissant en groupes, tendent à présenter un comportement de groupe, incluant un affaiblissement prématuré en réponse à un stress.

Concern about decline of shade and forest trees waxes and wanes in relation to the visibility of damage caused by pests, pathogens, and environmental insults. Awareness of decline heightened in recent years as people became aware of the widescale adverse effects—both real and hypothetical—of polluted air and atmospheric deposition (4, 24, 31, 37), repeated wounding (33), cryptic pathogens (23), defoliating insects (14, 15, 38), and stressful urban environments (2, 20, 30, 40). Popular reports about tree decline, however, often promote confusion by: a) presenting as fact a hypothesis about the cause of decline, b) oversimplifying the interaction of biotic and abiotic causal factors, c) tying the concept of decline to one cause (such as acidic deposition), or d) disregarding the fact that decline in the sense of failing health is an inevitable phase of a tree's life. The purpose of this paper is to review the application of the term *decline* and to present a set of four alternative concepts that apply in different circumstances to the causation of premature decline. For additional discussion of decline concepts, readers should consult the review by Manion (22).

Most trees, in common with other life forms, pass through a period of decline—senescence—before death. Thus decline in one sense is a normal phenomenon. The only trees that escape decline are the minority that die quickly after an injury or as the result of infection by a virulent pathogen. As commonly used and understood, however *decline* connotes premature progressive loss of health. It is the concept of premature debilitation that leads us to classify declines as a major category of tree diseases (12, 13, 21, 22).

The term *decline* does not necessarily connote disease of any specific nature or cause, however. When we mention maple decline, oak decline, or

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ash decline, readers should understand that we are merely referring to diseases that are characterized by progressive debilitation, regardless of cause.

In order to understand decline, whatever its causes, one must recognize that normal trees continually interact with many biotic and abiotic environmental factors that are both favorable and unfavorable for growth and development. Tree growth and behaviour may vary from site to site or fluctuate widely on a given site and still be judged normal for the conditions present. When because of stress from injury, infection, or environmental insult, the tree loses most of its ability to respond to favorable conditions, it also loses the ability to tolerate or resist the unfavorable ones, and it declines. The stress may be caused by pathogens, by excess or insufficiency of abiotic environmental factors, or by repetitive injuries.

The term *decline* is often used when the cause of a progressive disorder is unknown. When a single agent or circumstance is found to cause decline consistently, a name for the disorder is usually coined and the disorder is considered thereafter to be a discrete disease. Thus by default, *decline* appears in the names of disorders that are poorly understood or that are caused by multiple factors. For example, *maple decline* in popular use connotes deterioration that is at least partially unexplained. Maples in landscapes often decline because of girdling roots or infection by *Verticillium,* and those in woodlots because of root damage by cattle or exposure of residual trees after logging, but these disorders are usually held separate in our thinking.

Symptoms

Symptoms of decline include slow growth; sparse and/or undersized or distorted, often chlorotic leaves; browning of leaf margins; premature display of autumn color; premature leaf drop; abnormally large crops of fruit ("distress crops"); diminished storage of food reserves, especially starch; and progressive or intermittent dieback of twigs and branches and eventually the entire tree. Adventitious sprouts often develop for a time along the trunks of trees that have sustained branch dieback.

Two general sequences of symptoms are recognized. If decline is incited by a damaging event such as root cutting or severe defoliation, buds and twigs may die as a shock response to the injury, and this dieback may precede foliar symptoms. If decline results from chronic stress, as by salt, water shortage, or systemic infection, foliar symptoms and slow growth are likely to precede dieback. The symptoms may progress steadily or intermittently until the tree dies or its condition becomes static. If symptoms become static for a long time, decline has ceased.

The potential reversibility of decline depends on its cause and on the condition of the tree. Decline caused by systemic infection is usually not reversible, but that caused by abiotic stressing factors may be reversible if the stress is removed while the tree still has some resiliency.

Single Versus Multiple Causes of Decline

The list of causal factors includes insects, especially defoliators and borers; fungi that attack roots, bark, and sapwood; bacteria such as those associated with bacterial wetwood and bacterial leaf scorch; mollicutes such as the mycoplasmalike organisms involved in decline of ash trees; nematodes; viruses; water supply (too much or too little); asphyxiation of roots (as during flooding or around a gas leak); deicing salt; air pollutants (especially ozone); and site alteration.

Even when a tree declines primarily as the result of a single disease or environmental factor, there are always secondary, or contributing, causal factors. Often, however, decline is caused by several environmental and biotic factors acting in concert or in sequence. The key idea, whether we deal with one stressing factor or many, is that over periods of years these factors prevent normal growth and defensive processes, accelerate senescence, and hasten death.

Conceptual Explanations

Different conceptual schemes have been put forward to explain various declines, and each scheme seems applicable in particular circumstances. In the simplest scheme, decline is a slowly progressing syndrome caused primarily by one factor. In a second scheme, a tree sustains a major shock, such as defoliation, that makes it abnormally sensitive to adverse environmental factors and abnormally susceptible to opportunistic pathogens and other pests. The secondary factors and opportunistic organisms may be the immediate causes of decline. In a third scheme, decline is explained as a three-phase process caused by the chronic effects of multiple adverse factors. One or more of these factors first weakens or predisposes the tree. Then other factors incite decline, and still others contribute to the impact of the inciting factor. The predisposing, inciting and contributing factors are interchangeable. A fourth scheme, advanced in recent years by vegetation ecologists, is applicable to the decline of trees growing in groups, usually in forests. In this scheme, trees develop and age together until new or preexisting site factors constrain their growth and eventually cause such stress that the trees senesce and decline together. We will present examples that fit each scheme.

Decline caused primarily by perennial or continual irritation by one factor. The factor may be a pathogen or a component of the abiotic environment. We will consider 4 examples. The first is decline of pin oaks due to inadequate uptake of iron. The principal symptoms are chlorosis and progressive dieback. If untreated, the tree slowly declines and dies (27). This disease is caused not by a pathogen but by an environmental insufficiency.

The second example is decline of sugar maple caused primarily by uptake of deicing salt. Sugar maple along roadsides in northeastern states and in Ontario began to decline during the 1950's and 1960's as the use of deicing salt increased. Evidence accumulated that the salt, although not the only stress-inducing factor, was primarily responsible for the decline (10, 42). In recent years, however, we don't hear much about salt as a cause of decline in sugar maple. One reason is that where once there were many sugar maples along the roadsides, now there are few. Those that were in position to take up large quantities of chloride and sodium ions from deicing salts are gone.

Air pollutants may also cause continual stress resulting in decline, provided that the plant is intrinsically sensitive to the pollutants. Some eastern white pines, for example, are highly sensitive to ozone and also to sulfur dioxide. If chronically exposed to these pollutants, the trees lose vigor, bear only one age class of needles, and either turn yellow or show tip burn of needles. The yellowing and decline of white pines along the Blue Ridge Parkway in Virginia has been related to their sensitivity to ozone (1).

As a final example in this category, consider decline of white ash and red/green ash in central and eastern states. Although in some localities these ash species and other trees apparently decline because of stressful environment, much damage to the ash also occurs on sites where other trees appear normal and where ash formerly grew rapidly and to large sizes. Circumstantial evidence and some experimental data support the hypothesis that a microbial pathogen is primarily responsible. Declining ash in a region extending from the Great Plains to the Atlantic may be found infected by mycoplasmalike organisms (MLOs). These are submicroscopic prokaryotic organisms, lacking cell walls, that infect the phloem of trees systemically and are transmitted by certain leafhoppers and other insects that feed by sucking ploem sap. Some ash trees tolerate MLO infection for many years, but others decline and die. Matteoni and Sinclair (23) have coined the name *ash yellows* for the mycoplasmal disease that debilitates ash trees.

For each of the examples just cited, although one factor is primarily responsible for decline, additional factors undoubtedly contribute. Some pin oaks are genetically predisposed to damage from iron deficiency (3), and this disorder is most significant where the trees grow in neutral to alkaline soils in which iron is bound in insoluble forms (27). Salt stress causes nutrient imbalances and impairs the winter hardiness of woody plants (39). Plants weakened by air pollutant injury become abnormally susceptible to opportunistic fungi and insects that cause further damage (16, 17). Plants infected with MLOs do not become normally cold hardy, and they may sustain severe winter damage, such as split bark at the base of the trunk. Also, opportunistic fungi cause cankers and dieback in ash trees weakened by MLOs (23).

Decline caused by drastic injury plus secondary stress. This scheme was proposed and verified by plant pathologists and entomologists studying the role of defoliating insects in tree declines. David Houston and Philip Wargo of the U.S. Forest Service have made significant contributions to our understanding of declines that fit this conceptual scheme (11-14, 41). Consider, for example, the decline of oaks after defoliation by insects. In central and eastern states, oaks in both wild and urban forests are defoliated during sporadic outbreaks of oak leaf rollers, cankerworms, or the gypsy moth. Severe defoliation—the removal of three fourths or more of the foliar surface—may cause dieback and death, or it may predispose trees to decline caused by other agents. Defoliation is most damaging if the foliage is removed just as leaves become fully expanded. This loss triggers a second flush of growth during the same season, and the replacement growth depletes the stored carbohydrate reserves of the tree and leaves it abnormally susceptible to attack by secondary insects and opportunistic fungal pathogens. If the tree is defoliated in two successive years, food reserves are reduced to essentially nil, branch dieback begins, and water sprouts develop along the trunk and major limbs. The opportunistic organisms that most often cause further damage to defoliated oaks are root-rotting fungi, especially *Armillaria* species, and secondary insects, particularly *Agrilus bilineatus*, the two-lined chestnut borer. Larvae of this insect tunnel in the cambial region of weakened trees and girdle and kill limbs or entire trees (12, 14, 41).

Decline of street trees following construction damage to roots is another example that is best explained by a concept of two-stage causation. The massive removal of roots leads to water stress and to invasion of the wounds by opportunistic fungi, such as *Ganoderma lucidum*, that cause root decay. The limbs and trunks of such trees become abnormally susceptible to secondary insects and to fungi that cause cankers and decay of sapwood. By the time symptoms of decline become noticeable, the original root damage may have been forgotten.

Interchangeable predisposing, inciting, and contributing factors. In 1965-1967, Sinclair (34, 35) proposed a scheme in which multiple factors acting interchangeably may first weaken a tree, then trigger decline, and finally exacerbate the problem by their continual influence. He referred to these interacting causes as predisposing, inciting and contributing factors. The key thought in this scheme was that the introduction of a new biotic or abiotic factor or a change in the supply of a factor in an already stressful environment may trigger decline.

Manion refined Sinclair's concept in the textbook, *Tree Disease Concepts* (21). Manion proposed that we think of decline as a spiral of diminishing health. The tree is first predisposed by adverse factors, and its health and vigor diminish somewhat. Then another factor incites decline, and in due course various contributing factors perpetuate decline until, at the center of the spiral, the tree dies.

The concept of predisposing, inciting and contributing factors is applicable to decline of sugar maple trees in forests and sugarbushes (maple stands managed for syrup and sugar production), since multiple factors usually seem to be involved. Sugar maple grows best on moist well-drained soils where its root zone is shaded and remains undisturbed. In forests and sugarbushes, trees of this species may be weakened (thus predisposed to decline) by any of the following alone or in combination: grazing livestock, overzealous tapping and sap extraction, timber harvesting, possibly acidic deposition, and certainly defoliation by insects. If these factors stress the tree over several years, its growth will slow, and it may lose the capacity to respond to favorable factors. That is, it may begin to decline. Timber harvesting promotes decline in the residual stand because tree trunks and roots are subject to wounding during logging and to abnormal heating and drying by sunlight thereafter. These changes not only increase the possibility of drought stress, but alter the interactions of organisms in the root zone. Grazing cattle cause or contribute to decline because their hoofs break feeder roots. Sap harvesting may promote decline if too many tap wounds are made or chemicals are used that thwart compartmentalization of tap holes (causing death of a large volume of sapwood) (32) or too much sap is extracted (depleting sugar that would be used as the energy source for growth). Acidic deposition possibly also plays a role, but direct evidence for this is scant. Opportunistic organisms, especially fungal pathogens, however, always contribute to the damage. *Armillaria* species kill roots, and fungi such as *Cerrena unicolor; Valsa ambiens,* and *Steganosporium* species kill twigs and branches of weakened trees. These organisms are often the immediate causes of dieback (8, 12).

All maple species that are commonly planted in landscapes are also subject to decline for which the concept of predisposing, inciting, and contributing factors seems applicable. Common predisposing and inciting factors include girdling roots, restricted rooting space leading to water stress, cankers and collar rots caused by fungi (especially *Phytophthora* species), soil compaction leading to water shortage and rootlet mortality, deicing salt, chronic effects of *Verticillium dahliae,* severe trunk wounds, and root cutting during excavation. The same opportunistic fungal pathogens found in forests contribute to decline of maples in urban plantings, and they are joined there by such fungi as *Ganoderma lucidum,* which causes root rot, and *Nectria cinnabarina* and *Botryosphaeria obtusa* which cause cankers and dieback (36).

As a further example, consider birch dieback. This disorder caused great damage to paper and yellow birches in the forests of eastern Canada in the 1930s to early 1950s and then subsided. Birch dieback was never fully explained (5), but both circumstantial and some experimental evidence pointed to the involvement of a climatic warming trend that occurred between 1920 and 1950 (7). Mean annual temperatures at various places in the Maritime provinces rose between 1.0 and 1.4°C during that period. It was tempting to explain birch dieback as simply a response to long-term climatic change, but this explanation was not intellectually satisfying because it did not account for the sudden onset of the dieback syndrome in a given tree (5), and it did not take into account the possible involvement of pathogens. Moreover, the magnitude of the temperature increase was insufficient to cause visible damage under experimental conditions.

Scientists studying birch dieback in Quebec and the Maritime provinces implicated heat, root-infecting fungi, viruses, and secondary insects in the decline. For example, when the soil temperature in the rooting zone of birch seedlings was raised 2°C throughout a growing season by means of heating cables installed underneath, rootlet mortality rose from 6% in control seedlings to near 60% in the treated ones. It appeared that the altered root environment was unfavorable for mycorrhizal fungi but conducive to damage by root infecting fungi that would normally be innocuous (29). In Quebec, birch dieback was most severe where birch was most shallowly rooted. Significant damage to roots was thought to occur during open winters when roots in exposed soil were subject to abnormal freezing and drying (28). This damage would be most likely to occur during abnormally warm winters when the insulating blanket of snow is temporarily absent. As for contributing factors, the bronze birch borer (*Agrilus anxius*) attacked the weakened trees and in many cases was directly responsible for dieback and death. This same insect attacks and kills stressed birches in landscapes. Assorted viruslike symptoms were part of the dieback syndrome, and a strain of apple mosaic virus was eventually found in birch (6), but this virus probably had at most a contributory causal role. It was found in a much larger region than that where birch dieback occurred. Thus the concept of predisposing, inciting, and contributing factors conveniently integrates what is known about the causes of birch dieback.

Synchronous cohort senescence. The fourth concept, which I regard as a variation of the third, was elaborated by Mueller-Dombois and coworkers during the early 1980s, first as an explanation for the decline of ohia *(Metrosideros collina)* trees in Hawaiian forests (25), and later as a general explanation for assorted decline problems in North American Forests (26). The key thought in this concept is that trees of similar age, growing together, display group behaviour. As they become older and larger, they are increasingly likely to come under stress, especially as the result of seasonal water shortage. Thus they naturally become predisposed to damage that could incite decline. When a new adverse factor such as drought or a climatic warming trend causes increased stress, the trees may senesce and decline together. This phenomenon is called synchronous cohort senescence. Contributing factors, such as opportunistic fungi and insects, then hasten the decline. Mueller-Dombois et al. (26) adopted a narrow concept of disease in their discussions of synchronous cohort senescence, but neither this shortcoming nor the dissenting opinions of forest pathologists about the cause of ohia decline (9) diminishes the intellectual attractiveness of the central idea in the cohort-senescence concept.

Mueller-Dombois et al. (26) cited birch dieback and pole blight of western white pine as widescale declines for which their concept is appropriate. Pole blight, like birch dieback, was among the several tree declines that came to prominence during the 1930s and then subsided in the 1950s. Pole blight occurred, and still occurs in some localities, in scattered parts of the intermountain region of the northwestern United States and adjacent British Columbia. The name *pole blight* indicates that this disorder affects dominant and codominant trees that have grown to pole size (15-30 cm diameter). The symptoms include death of rootlets, slow trunk and twig growth, tufted foliage at branch tips, resinous cankers on the lower parts of the trunk, dieback, and death. The cankers are caused by opportunistic fungi, such as *Ophiostoma trinacriforme,* that are innocuous to trees of normal vigor. Pole blight was eventually explained as primarily a response to a temporary climatic trend of increasing temperature and diminishing rainfall (18). Young white pines growing in soils of low water holding capacity could develop vigorously during periods of normal rainfall, but during prolonged dry periods, the roots could not supply the transpirational demands of the tops, and decline resulted (19). The trees declined synchronously in groups of similar age because the members of a group were all subject to a similar level of stress. Thus the concept of synchronous cohort senescence seems highly applicable to pole blight.

Conclusion

Many different disorders of trees can be grouped under the general heading *decline.* Up to now, four general concepts have been advanced to explain decline, and three of these concepts deal with the roles of multiple causal factors. Each concept is a variation on the theme that decline is caused by chronic stress or sequential insults to the tree. Each concept seems applicable to one or more decline syndromes, but no single concept of causality is applicable to all decline syndromes.

Literature Cited

1. Benoit, L.F., J.M. Skelly, L.D. Moore, and L.S. Dochinger. 1982. *Radial growth reductions of Pinus strobus L. correlated with foliar ozone sensitivity as an indicator of ozone-induced losses in eastern forests.* Can. J. For. Res. 12:673-678.
2. Berrang, P., D.F. Karnosky, and B.J. Stanton. 1985. *Environmental factors affecting tree health in New York City.* J. Arboric. 11:185-189.
3. Berrang, P., and K.C. Steiner. 1980. *Resistance of pin oak progenies to iron chlorosis.* J. Am. Soc. Hort. Sci. 105:519-522.
4. Bormann, F.H. 1982. *The effects of air pollution on the New England landscape.* Ambio 11:338-346.
5. Clark, J., and G.W. Barter. 1958. *Growth and climate in relation to dieback of yellow birch.* For. Sci. 4:343-364.
6. Gotlieb, A.R., and J.G. Berbee. 1973. *Line pattern of birch caused by apple mosaic virus.* Phytopathology 63:1470-1477.
7. Hepting, G.H. 1963. *Climate and forest diseases.* Annu. Rev. Phytopathol. 1:31-50.
8. Hibben, C.R. 1964. *Identity and significance of certain organisms associated with sugar maple decline in New York woodlands.* Phytopathology 54:1389-1392.
9. Hodges, C.S., K.T. Adee, J.D. Stein, H.B. Wood, and R.D. Doty. 1986. Decline of ohia *(Metrosideros polymorpha)* in Hawaii: a review. U.S. For Serv. Gen. Tech. Rep. PSW-86. 22 pp.
10. Hofstra, G., R. Hall, and G.P. Lumis. 1979. *Studies of salt-induced damage to roadside plants in Ontario.* J. Arboric. 5:25-31.
11. Houston, D.R. 1973. *Diebacks and declines: diseases initiated by stress, including defoliation.* Proc. Int. Shade Tree Conf. 49:73-76.
12. Houston, D.R. 1981. Stress triggered tree diseases. The diebacks and declines. U.S. For. Serv. NS-INF-41-81. 36 pp.
13. Houston, D.R. 1985. Diebacks and declines of urban trees. Pages 120-137 **In:** Improving the Quality of Urban Life with Plants. D.F. and S.L. Karnosky, eds. N.Y. Bot. Gard. Inst. Urban Hort. Pub. No. 2. 200 pp.
14. Houston, D.R. J. Parker, and P.M. Wargo. 1981. Effects of defoliation on trees and stands. Chapter 5, pp. 217-297 **In:** The Gypsy Moth: Research Toward Integrated Pest Management. C.C. Doane and M.L. McManus eds. U.S. Dep. Agric. Tech. Bull. 1584. 757 pp.
15. Kulman, H.M. 1971. *Effects of insect defoliation on growth and mortality of trees.* Annu. Rev. Entomol. 16:289-324.
16. Lackner, A.L., and S.A. Alexander. 1983. *Root disease and insect infestations on air-pollution-sensitive Pinus strobus and studies of pathogenicity of Verticicladiella procera.* Plant Dis. 67:679-681.
17. Laurence, J.A. 1981. *Effects of air pollutants on plant-pathogen interactions.* Pflanzenkrankh. Pflanzenschutz 88:156-172.

18. Leaphart, C.D., and A.R. Stage. 1971. *Climate: a factor in the origin of the pole blight disease of Pinus monticola Dougl.* Ecology 52:229-239.
19. Leaphart, C.D., and E.F. Wicker. 1966. *Explanation of pole blight from responses of seedlings grown in modified environments.* Can. J. Bot. 44:121-137.
20. Manion, P.D. 1981. *Norway maple decline.* J. Arboric 7:38-42.
21. Manion, P.D. 1981. Decline diseases of complex biotic and abiotic origin. Pages 324-339 **In:** Tree Disease Concepts. Prentice Hall, Englewood Cliffs, NJ. 399 pp.
22. Manion, P.D. Decline as a phenomenon in forests: pathological and ecological considerations. Pages 267-276 **In:** Proc. NATO Advanced Research Workshop. Effects of Acidic Deposition on Forests, Wetlands, and Agricultural Ecosystems. T.C. Hutchinson and K.M. Meema, eds. Springer-Verlag, New York.
23. Matteoni, J.A., and W.A. Sinclair. 1985. *Role of the mycoplasmal disease, ash yellows, in decline of white ash in New York State.* Phytopathology 75:355-360.
24. Miller, P.R., tech. coord. 1980. Proceedings of Symposium on Effects of Air Pollutants on Mediterranean and Temperate Forest Ecosystems. U.S. For. Serv. Gen. Tech. Rep. PSW-43. 256 pp.
25. Mueller-Dombois, D. 1983. *Canopy dieback and successional processes in Pacific forests.* Pacific Sci. 37:317-325.
26. Mueller-Dombois, D., J.E. Canfield, R.A. Holt, and G.P. Buelow. 1983. *Tree group death in North American and Hawaiian forests: a pathological problem or a new problem for vegetation ecology?* Phytocoenologia 11:117-137.
27. Neely, D. 1976. *Iron deficiency chlorosis of shade trees.* J. Arboric. 2:128-130.
28. Pomerleau, R., and M. Lortie. 1962. *Relationships of dieback to the rooting depth of white birch.* For. Sci. 8:219-224.
29. Redmond, D.R. 1955. *Studies in forest pathology. XV. Rootlets, mycorrhiza, and soil temperature in relation to birch dieback.* Can. J. Bot. 33:595-627.
30. Rich, S., and G.S. Walton. 1979. *Decline of curbside sugar maples in Connecticut.* J. Arboric. 5:265-268.
31. Schutt, P., and E.B. Cowling. 1985. *Waldsterben, a general decline of forests in central Europe: symptoms, development, and possible causes.* Plant Dis. 69:548-558.
32. Shigo, A.L., and F.M. Laing. 1970. Some effects of paraformaldehyde on wood surrounding tapholes in sugar maple trees. U.S. For. Serv. Res. Pap. NE-161. 11 pp.
33. Shigo, A.L., and H.G. Marx. 1979. Tree decay. An expanded concept. U.S. Dep. Agric. Agric. Inf. Bull. 419. 73 pp.
34. Sinclair, W.A. 1965. *Comparisons of recent declines of white ash, oaks, and sugar maple in northeastern woodlands.* Cornell Plantations 20:62-67.
35. Sinclair, W.A. 1967. *Decline of hardwoods: possible causes.* Proc. Int. Shade Tree Conf. 42:17-32.
36. Sinclair, W.A., H.H. Lyon, and W.T. Johnson. 1987. Diseases of Trees and Shrubs. Cornell University Press, Ithaca, NY. 574 pp.
37. Smith, W.H. 1981. Air Pollution and Forests. Interactions Between Air Contaminants and Forest Ecosystems. Springer-Verlag, New York. 379 pp.
38. Stephens, G.R. 1981. Defoliation and mortality in Connecticut forests. Conn. Agric. Exp. Stn. Bull. 796. 13 pp.
39. Sucoff, E., and S.G. Hong. 1976. *Effect of NaCl on cold hardiness of Malus spp. and Syringa vulgaris.* Can. J. Bot. 54:2816-2819.
40. Ware, G.H. 1982. Decline in oaks associated with urbanization. Pages 61-64 **In:** Urban and Surburban Trees: Pest Problems, Needs, Prospects, and Solutions. B.O. Parks, F.A. Fear, M.T. Lambur, and G.A. Simmons, eds. Proc. of Conf., Michigan State Univ. 253 pp.
41. Wargo, P.M. 1978. *Insects have defoliated my tree—now whats going to happen?* J. Arboric. 4:169-175.
42. Westing, A.H. 1969. *Plants and salt in the roadside environment.* Phytopathology 59:1174-1179.

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