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DIEBACK AND DECLINES OF URBAN TREES 1

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Abstract. Dieback-decline diseases occur when trees, stressed and altered by abiotic or biotic agents, are attacked by organisms of secondary action. The primary stress factors in forests are insect defoliation and extremes of moisture and temperature. In urban situations, drought is probably the most important stress factor. Most organisms of secondary action are native opportunists which often are more successful in urban than in forest situations — perhaps because urban trees are more frequently subjected to prolonged or intense stress. Attempts to control these diseases usually should be focused on preventing or reducing stress effects rather than on direct actions against the secondary organisms.

Contrary to popular opinion, trees are mortal, and, at some point in time, will die. And it is highly probable that as trees die they will die back and decline. If dieback followed by decline and death is a natural sequence of events, why do we consider the diebacks and declines as a specific group of diseases? This question is addressed in this paper. The answers are important because dieback and decline diseases are especially significant in urban environments.

If moisture, nutrients, and light are adequate, and external disturbances and stresses from abiotic or biotic agents are not severe, trees will, within their genetic limitations, grow larger. Eventually, however, every tree reaches the point when increasing physiological demands for essential materials can barely be satisfied. At this point, growth is very slow, and even relatively minor stress factors may initiate dieback. Trees that can do so may die back to some degree as they adjust to this increasingly adverse environment — giving up some tissues in order to support

the remainder. Trees not able to do this are apt to be short-lived. When stresses are prolonged or intense, the dieback mechanism may not be sufficient to maintain this balance and tissues altered by the stress may fall prey to pathogenic organisms ordinarily of little importance. Under conditions of prolonged or intense stress, even trees not yet physiologically mature may be affected. When this occurs, the condition is known as dieback-decline disease.

Diebacks and Declines — A definition of cause and effect

Most dieback-decline diseases of forest trees share a common type of cause-effect relationship that serves to differentiate them from other types of disease.

Cause. Diebacks-declines occur when trees are stressed by adverse abiotic or biotic factors that render them susceptible to attacks, often lethal, by organisms of secondary action. Stress adversely alters tree condition. Sometimes stress triggers processes that lead to dieback directly; sometimes stress also produces anatomical or physiological changes that permit organisms of secondary action to attack and kill tissues. These secondary organisms include a wide variety of fungi and insects that can kill fine roots, buds and fine twigs, or bark and cambium of branches, stems and roots. Invasion by any of a number of these organisms can deliver the coup de grace in a given host/stress episode.

Effects. The syndrome shared, in part or in whole, by many of these diseases includes the death of buds and the dieback of twigs that begins

at the margin of the crown and progresses basipetally — inward and downward. Leaves of declining trees are often small, sparse, and off-color. Frequently, foliage in successive years is borne on sprouts and may appear clumped or tufted. Sometimes leaves show fall colors and drop prematurely, and sometimes terminal and radial growth is reduced even before external symptoms appear.

This common cause-effect relationship can be expressed as a series of stages:

- 1. Healthy trees + stress => Altered trees (dieback and decline begin)
- 2. Altered trees + more stress =>Trees altered further (dieback and decline continues)
- 3. Severely altered trees + organisms of secondary action => Trees invaded and (perhaps) killed.

Understanding these general stages helps in understanding how and why dieback-decline diseases occur and how to approach their control. It is also useful to compare forest and urban situations with respect to the conditions that lead to the development of dieback-declines.

Concepts. (1) Dieback often results from the effects of stress alone, and when stress abates, the dieback process often ceases and trees recover. (2) The decline phase, wherein vitality lessens and trees succumb, is usually the consequence of organisms attacking the stress-altered tissues. Recovery from this phase, which is less likely, depends on such factors as tree vigor, the location and severity of tissue invasion, and the relative aggressiveness of the organisms.

Comparisons — Natural forests vs. urban situations

1. When is dieback, decline, and death not dieback-decline disease? In natural forests, shaded and crowded trees often die. This attrition, the result of competition for light, moisture, and nutrients, is accepted, indeed even expected, as a natural consequence of forest development. Examination of such trees frequently reveals that their demise was hastened by attacks of the same secondary-action organisms that attack and kill vigorous trees altered by stress. In the forest, therefore, dieback-decline diseases only occur when trees are affected by

extraordinary stress.

In many urban situations, stresses of extraordinary duration or intensity occur more than occasionally. Indeed, relatively more trees in urban areas than in forests die from the direct effects of stress. (Perhaps, if no organisms of secondary nature are involved, these problems should not be called dieback-decline diseases?) Usually, however, attacks by secondary organisms are even more important in the dieback of urban trees than in forests. Because growing conditions are usually less favorable and stresses more frequent and intense in urban situations, trees there often reach their physiological limits sooner than in the forest. Although the stress/decline relationships of suppressed understory forest trees are somewhat analogous to those of city street trees. the early demise of the latter is neither expected nor accepted.

2. **Important stresses.** In the forest, the most significant stress factors that predispose trees to dieback-decline are insect defoliation, extremes of moisture (drought, winter desiccation, flooding), heat (freezing, frosts, sunscald), and attacks by sucking insects. Although the direct and

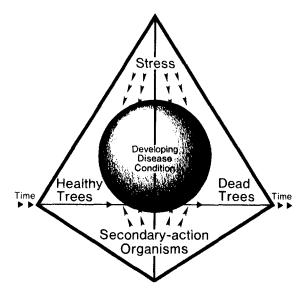


Figure 1. A conceptual framework for the dieback-decline diseases. Healthy trees are affected by environmental stress; over time, trees altered by that stress are invaded at some point by secondary-action organisms. The disease condition develops and trees dieback, decline, and ultimately may die.

acute effects of air pollutants such as ozone and oxides of sulfur and nitrogen are recognized, their long-term and indirect effects and those of other pollutants, including acid deposition and heavy metals, as predisposing factors are not yet known.

These stress factors are also important in urban situations. Drought, exacerbated by the effects of heat, is probably the most important. Urban trees are often grown where spaces for their roots are restricted, or where they are subjected to sudden changes in water tables and drainage patterns, and to root and stem injuries. And, in contrast to forest trees, they are often subject to the effects of herbicides and salt. Trees in urban environments must often compete with dense grass sod covers for moisture, nutrients, and oxygen. Urban trees also may be uniquely harmed by too much light. Daily and seasonal photoperiods lengthened by artificial lighting may prevent normal hardening-off and result in frost or winter injury and attack by twig-blighting organisms.

3. The organisms of secondary action. In



Figure 2. Defoliation-triggered ash dieback. The tree on the right was completely defoliated by ash rust in two successive years. The tree on left was not defoliated.

natural forest situations, the secondary-action pathogens are usually opportunistic saprogens — saprophytic fungi with the ability to attack weakened plants. Most of these organisms are common inhabitants of natural forests, and harmless to healthy, non-stressed trees. Many even can be considered beneficial as decomposers or destroyers of weak, defective plants. There are some exceptions to this generalization. Occasionally stress may render tissues of one tree species exceptionally susceptible to organisms that are primary pathogens on other hosts.

In the urban environment, many organisms assume much more importance than in natural forests — a reflection, perhaps, of the greater stress encountered by urban trees and perhaps, also, of the greater number of exotic (and therefore less resistant) plants grown there.

4. Diagnosis and control of diebacks and declines. Diagnosis and control of diebacks-declines are governed by their peculiar cause-effect relationships. Regardless of whether they occur in the forest or downtown, their diagnosis

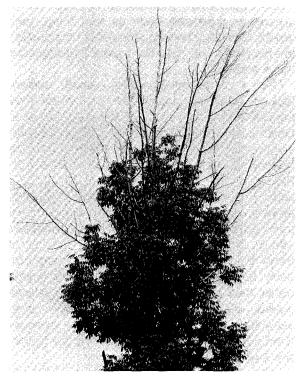


Figure 3. A closeup of the dieback tree in Figure 2 showing 'recovery' as a new crown of sprout-origin foliage develops.

entails the following steps: a) recognizing symptoms, b) associating in time and place the stress events or factors that triggered the problem and c) identifying the secondary-action organisms.

- a. Progressive dieback and deterioration of crowns; thin, sparse "tufted" foliage produced on sprouts; early fall coloration; and reduced height and radial growth rates are all tree responses to adverse environmental factors.
- b. Determining which event(s) triggered the prolem is often difficult, perhaps more so in remote forest areas than in closely observed urban locations. Records of such disturbances as weather extremes, insect defoliation, air pollution episodes, and construction events are extremely valuable. Determining when disturbances occurred perhaps by growth ring patterns or age of sprout tissues, may help to pinpoint what disturbance occurred. It is also clear that knowing when and where significant disturbances occur today will help predict, or at least explain, when and where diebacks-declines may occur tomorrow.
- c. The presence and state of development of secondary organisms such as root fungi, bark borers, and twig cankering fungi also help confirm the diagnosis and date the events that triggered these attacks.

Control of diebacks-declines usually centers first on measures to prevent or reduce their predisposing stress factors. When the stress is biotic, a direct approach, such as spraying to prevent insect defoliation or scale infestation, may be feasible in both forest and urban situations. Preventing abiotic stresses is more difficult. For urban situations, watering, fertilizing, mulching, pruning, applying antidesiccants, and planting trees away from road edges can help alleviate the effects of drought. Development or selection of trees resistant to diebacks-declines should probably focus on resistance to the stress agents rather than to the often numerous and variable agents of secondary action. However, when secondary organisms are limited in numbers (kinds) and are specific, it may be feasible to control them either directly or indirectly.

Table 1 lists a number of selected forest and urban dieback-decline diseases. These are chosen to represent a variety of hosts, predispos-

ing stresses, organisms of secondary action, and approaches to control.

Bibliography

General

- Houston, D.R. 1967. The dieback and decine of northeastern hardwoods. Trees 28: 12-14.
- Houston, D.R. 1973. *Diebacks and declines: Diseases initiated by stress, including defoliation.* Proc. Int. Shade Tree Conf. 49: 73-76.
- Houston, D.R. 1974. Diagnosing and preventing diebacks and declines. Morton Arbor. Q. 10(4): 55-59.
- Houston, D.R., J. Parker, and P.M. Wargo. 1981. Effects of defoliation on trees and stands. Chapter 5. In: The gypsy moth: Research toward integrated pest management. U.S. Dept. Agric. Tech. Bull. 1584.
- Houston, D.R. 1981. Stress triggered tree diseases: The diebacks and declines. U.S. Dept. Agric. For. Serv. NE-INF-41-81. 36 p.
- Houston, D.R. 1981. Some dieback and decline diseases of northeastern forest trees: Forest management considerations. In: Proc., Annu. Silvic. Workshop p. 248-265.
- Houston, D.R. 1982. Basic concepts of diebacks-declines. In: Proc., Urban and Suburban Trees: Pest Problems, Needs, Prospects, and Solutions Conf. p. 57-60.
- Houston, D.R. 1984. Diebacks and declines of urban trees. In: Proc., Int. Symp. Urban Hortic. p. 235-249.
- Messenger, A.S. 1976. Root competition: Grass effects on trees. J. Arboric. 2:228-230.
- Roberts, B.R. 1977. The response of urban trees to abiotic stress. J. Arboric. 3: 75-78.
- Schoeneweiss, D.F. 1975. Predisposition, stress, and plant disease. Ann. Rev. Phytopathol. 13: 193-211.
- Schoeneweiss, D.F. 1978. The influence of stress on diseases of nursery and landscape plants. J. Arboric. 4: 217-225.
- Schoeneweiss, D.F. 1981. Infectious diseases of trees associated with water and freezing stress. J. Arboric. 7: 13-18.
- Sinclair, W.A. 1964. Comparisons of recent declines of white ash, oaks, and sugar maple in northeastern woodlands. Cornell Plant. 20: 62-67.

Ash Dieback and Decline

- Hibben, C.R., and S.B. Silverborg. 1978. Severity and causes of ash dieback. J. Arboric. 4: 274-279.
- Hibben, C.R., and J.A. Reese. 1983. Identification of tomato ringspot virus and mycoplasma-like organisms in stump sprouts of ash. (Abstr.) Phytopathology 73: 367.
- Lana, A.O., and G.N. Agrios. 1974. Properties of a strain of tobacco mosaic virus isolated from white ash trees. Phytopathology 64: 1490-1495.
- Marshall, R.P. 1930. A canker of ash. Proc., Sixth Natl. Shade Tree Conf. p. 128-130.
- Matteoni, J.A. 1983. Yellows diseases of elm and ash: water relations, vectors, and role of yellows in ash decline. Dissertation, Cornell Univ., Ithaca, NY. 152 pp.
- Ross, E.W. 1964. Cankers associated with ash dieback. Phytopathology 54: 272-275.
- Ross, E.W. 1966. Ash dieback: etiological and developmental studies. N.Y. State Coll. For., Tech. Pub. 88. 80 pp.
- Silverborg, S.B., and R.W. Brandt. 1957. Association of

Cytophoma pruinosa with dying ash. For. Sci. 3: 75-78. Silverborg, S.B., and E.W. Ross. 1968. Ash dieback disease development in New York State. Plant Dis. Rep. 52: 105-107.

Beech Bark Disease

- Ehrlich, J. 1934. The beech bark disease, a Nectria disease of Fagus following Cryptococcus fagi (Baer.). Can. J. Res. 10: 593-692.
- Houston, D.R. 1983. Developments in biological control of beech bark disease. **In:** Proc., 10th Intl. Congress of Plant Protection. p. 1035-1041.
- Houston, D.R. 1984. What is happening to the American beech? The Conservationist, May-June p. 22-25.
- Houston, D.R., E.J. Parker, R. Perrin, and K.J. Lang. 1979. Beech bark disease: A comparison of the disease in North America, Great Britain, France and Germany. Eur. J. For. Path. 9: 199-211.
- Shigo, A.L. 1964. Organism interactions in the beech bark disease. Phytopathology 54: 263-269.

Dogwood

Daughtrey, M.L. 1983. Lower branch dieback, a new disease of northeastern dogwoods. (Abstr.) Phytopathology 73: 365.

Maple Declines

- Drilias, M.J., J.E. Kuntz, and G.L. Worf. 1982. The role of Phytophthora and Fusarium in Wisconsin maple decline. In: Urban and Suburban Trees: Pest Problems, Needs, Prospects and Solutions Conf. p. 65-67.
- Giese, R.L., D.R. Houston, D.M. Benjamin, J.E. Kuntz, J.E. Kapler and D.D. Skilling. 1964. Studies of maple blight. Univ. Wis. Res. Bull. 250, 128 p.
- Hibben, C.R. 1962. Investigations of sugar maple decline in the Northeast. Ph.D. Thesis, Cornell Univ., Ithaca, NY. 301 p.
- Lacasse, N.L., and A.E. Rich. 1964. Maple decline in New Hampshire. Phytopathology 54: 1071-1075.
- Parker, J., and D.R. Houston. 1971. Effects of repeated defoliation on root and root collar extractives of sugar maple trees. For. Sci. 17: 91-95.
- Wargo, P.M. 1972. Defoliation-induced chemical changes in sugar maple roots stimulate growth of Armillaria mellea. Phytopathology 62: 1278-1283.
- Wargo, P.M. and D.R. Houston. 1974. Infection of defoliated sugar maple trees by Armillaria mellea. Phytopathology 64: 817-822.
- Wargo, P.M., J. Parker, and D.R. Houston. 1972. Starch contents of roots of defoliated sugar maple. For. Sci. 18: 203-204.

Oak Declines

- Dunbar, D.M., and G.R. Stephens. 1974. Association of twolined chestnut borer and shoestring fungus with mortality of defoliated oak in Connecticut. For. Sci. 21: 169-174.
- Houston, D.R., and H.T. Valentine. 1977. Comparing and predicting forest stand susceptibility to gypsy moth. Can. J. For. Res. 7: 447-461.
- Kegg, J.D. 1973. Oak mortality caused by repeated gypsy moth defoliation of oak in New Jersey. J. Econ. Entomol. 66: 639-641.
- McManus, M.L., D.R. Houston, and W.E. Wallner. 1979. The homeowner and the gypsy moth: Guidelines for control. U.S. Dept. Agric. Home and Gard. Bull. 227. 34 p.
- Nichols, J.O. 1968. Oak mortality in Pennsylvania. A 10-year study. J. For. 66: 681-694.
- Roane, M.K., R.J. Stípes, P.M. Phipps, and O.K. Miller, Jr. 1974. Endothia gyrosa, causal pathogen of pin oak blight. Mycologia 66: 1042-1047.
- Staley, J.M. 1965. Decline and mortality of red and scarlet oaks. For. Sci. 11: 2-17.
- Stipes, R.J., and P.M. Phipps. 1971. A species of Endothia associated with a canker disease of pin oak (Quercus palustris) in Virginia. Plant Dis. Rep. 55: 467-469.
- Ware, G.H. 1970. The destructible oak. Morton Arbor. Q. 6: 42-47.
- Ware, G.H. 1982. Decline in oaks associated with urbanization. In: Proc., Urban and Suburban Trees: Pest Problems, Needs, Prospects and Solutions Conf. p. 61-64.
- Wargo, P.M. 1977. Armillariella mellea and Agrilus bilineatus and mortality of defoliated oak trees. For. Sci. 23: 485-492.
- Wargo, P.M. 1978. Defoliation by the gypsy moth: How it hurts your tree. U.S. Dept. Agric. Home and Gard. Bull. 223, 15 p.
- Wargo, P.M. 1978. Insects have defoliated my tree now what's going to happen? J. Arboric. 4: 169-175.

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Table 1. A selected representative list of dieback-decline diseases chosen to represent a variety of hosts, predisposing stresses, organisms of secondary action and control.

HOSTS AND DISEASE		ORGANISMS OF SECONDARY ACTION	EFFECTS (SYMPTOMS AND SIGNS)	CONTROL
Ash Ash dieback, decline (general) ¹ Fraxinus americana F. pennsylvanica	Drought periods	Bark canker fungi: Cytophoma pruinosa Fusicoccum sp.	a) Reduced terminal and radial growth b) Death of terminal buds and branches c) Thin, "tufted" crowns d) Sometimes, early fall coloration e) Death of trees f) Reddish-brown to orange-yellow cankers on branches and stems	a) Forest: little can be done, salvage b) Urban: water when feasible, possibly reduce water loss by mulching
Dieback (general, near coastal salt marshes with Spartina grass hosts Fraxinus spp.	Defoliation by rust fungus, Puccinia peridermiospora	None	Dieback occurs the season following severe defoliation/ refoliation a) Death of terminal buds and branches b) Thin, "tufted" crowns c) Death of some trees	Most trees will recover unless defoliation is repeated or coincides with drought.
Beech Beech bark disease (general in east through NY and mid-PA; northeastern WV.) Fagus grandifolia F. sylvatica	Attack by the beech scale, Cryptococcus fagisuga	Bark canker fungi: Nec <i>tri</i> a coccinea var. faginata; N. galligena	a) Thinning chlorotic crowns b) Presence on bark, especially in roughened spots, of white "wool-like" wax of the beech scale c) Presence on bark previously infested by scale of small, red fruiting bodies of Nectria fungi d) Weeping exudates may precede or accompany the fruiting bodies e) Bark is killed, and trees die. Some trees snap off where hark dies and wood decays	a) Forest: salvage trees when scale populations become high and Nectria is abundant. Trees free from scale (resistant) should be favored b) Urban: treat individual trees with dormant sprays or chemical fungicides registered for use against the beech scale

g Not yet known	a) Forest: prevent defoliation, salvage if significant portion of crown is killed b) Urban: prevent defoliation s	a) Avoid use of deicing salt when feasible b) Plant replacement trees where brine will not run over roots	s a) Improve soil drainage b) Insure planting is shallow <
 a) Blighted leaves in early spring b) Dieback of buds, twigs and branches beginning on lowermost parts of tree c) Development of branch cankers d) Water sprouts from near branch cankers - these in turn are killed, trees die 	a) Death of buds, terminal twigs and branches the year after severe defoliation b) Thin "tufted" crowns c) New foliage on sprouts d) Continued decline and death of some trees e) White fungus "fans" often present beneath bark of roots and bases of dead and dying trees	a) Crown dieback, especially on road side of tree b) Leaves show fall colors early and drop c) Leaves may also show interveinal necrosis (scorch) d) Dieback progresses over years, trees eventually die	a) Leaves small, show fall colors early and drop b) Twigs and branches die back c) Rootlets die d) Bark at tree base loosens to show discoloration and decay of underlying wood e) Trees die one to several years after first symptoms
Leaf and twig fungus (Discula sp. Gnomonia sp.) of anthracnose type	The shoestring root rot fungus, Armillaria mellea, and twig fungus, Stegonosporum sp.	Many twig, root and stem decay fungi	Soil borne canker fungi: Fusarium sp.; Phytophtho: citricola
Suspected: series of unusually wet springs, followed by summer dry periods	Defoliation by a number of insects including: a) saddled prominent b) forest tent caterpillar c) maple webworm d) green stripped mapleworm	Deicing safts, and/or construction damage to roots	Suspect stress of planting trees too deeply in heavy soils, or settling of trees after planting
Dogwood Lower branch dieback, anthracnose (southern New England, northern mid- Atlantic states; urban, woodlands) Cornus florida, native and cultivars	Maple Maple decline (general; forest) Acer saccharum	"Roadside" maple decline ² (general) A. saccharum	"Urban" maple decline (Wisconsin) A. saccharum

b) Death of some treesc) Orange-red to brown-black fruiting pustules on dead bark

CONTROL	a) Forest: prevent defoliation: salvage b) Urban: as above; supply water during refoliation if feasible. Possibly spray tree trunks to prevent attack by borers	Keep "wooded" areas as natural as possible; avoid watering with hard water, runoff from concrete surfaces, fertilizing with limebased fertilizers, mixing of soil by construction, grading, etc. Mulch and reduce grass competition	None known	a) Forest: not a problem b) Urban: water during drought periods. Avoid pruning during dry times.
EFFECTS (SYMPTOMS AND SIGNS)	a) Dieback of terminal buds and branches in next season b) New foliage produced on sprouts in the crown and on the bole — crowns "tufted" c) Progressive dieback; mortality d) White fungus "fans" beneath bark (see Maple decline forest) e) "D"-shaped holes in bark and meandering insect galleries on outer sapwood beneath bark	a) Deterioration of fine feeder root system b) Dieback and decline of upper crowns progressing sometimes until the tree dies c) Signs of A. mellea and A. bilineatus	a) Rapid dieback and decline of mature and overmature trees b) Signs of A. mellea and A. bilineatus	a) Dieback of lower branches, especially those from which smaller ones have been pruned
ORGANISMS OF SECONDARY ACTION	The shoestring root rot fungus, A. mellea; and the twolined chestnut borer, Agrilus bilineatus	The shoestring fungus, A. mellea, and the borer, A. bilineatus	The shoestring fungus, A. mellea, and the borer, A. bilineatus	Bark canker fungus: Endothia gyrosa
TRIGGERING STRESS	Defoliation by any of several insects including: a) gypsy moth b) leaf rollers and tiers c) canker worms d) webworms, etc., and sometimes exacerbated by periods of water shortage	Adverse soil changes, including alkalinization, caused by various urban practices and disturbance	Periods of unusual spring- time wetness in areas where soil overlies dense clays	Pruning wounds made during drought
HOSTS AND DISEASE	Oak Oak decline (general) Q <i>uercu</i> s spp.	Oak decline (midwest; urban) Q. rubra Q. alba Q. macrocarpa	Oak decline (midwest; general) <i>Q. rubra</i> Q. <i>alba</i>	Pin oak canker dieback ³ (mid-Atlantic states; urban) Q. palustris

^{1.} Ash trees are hosts also to several viruses and a mycoplasmalike organism (MLO). Evidence is mounting that the MLO can cause growth loss and mortality — and effects may be especially serious in times of drought. This disease is now known as ash yellows, or ash yellow decline.

^{2.} Both Na and Clions are cumulatively toxic to sugar maple. Heavy, repeated salt contamination can kill trees even in the absence of secondary organisms.

^{3.} Endothia gyrosa also causes cankers and dieback associated with pruning wounds in American and Formosan sweetgum and in American beech. In this respect it is similar to the fungus Nectria cinnabarina, a pruning wound pathogen of many urban tree species that is especially serious during severe