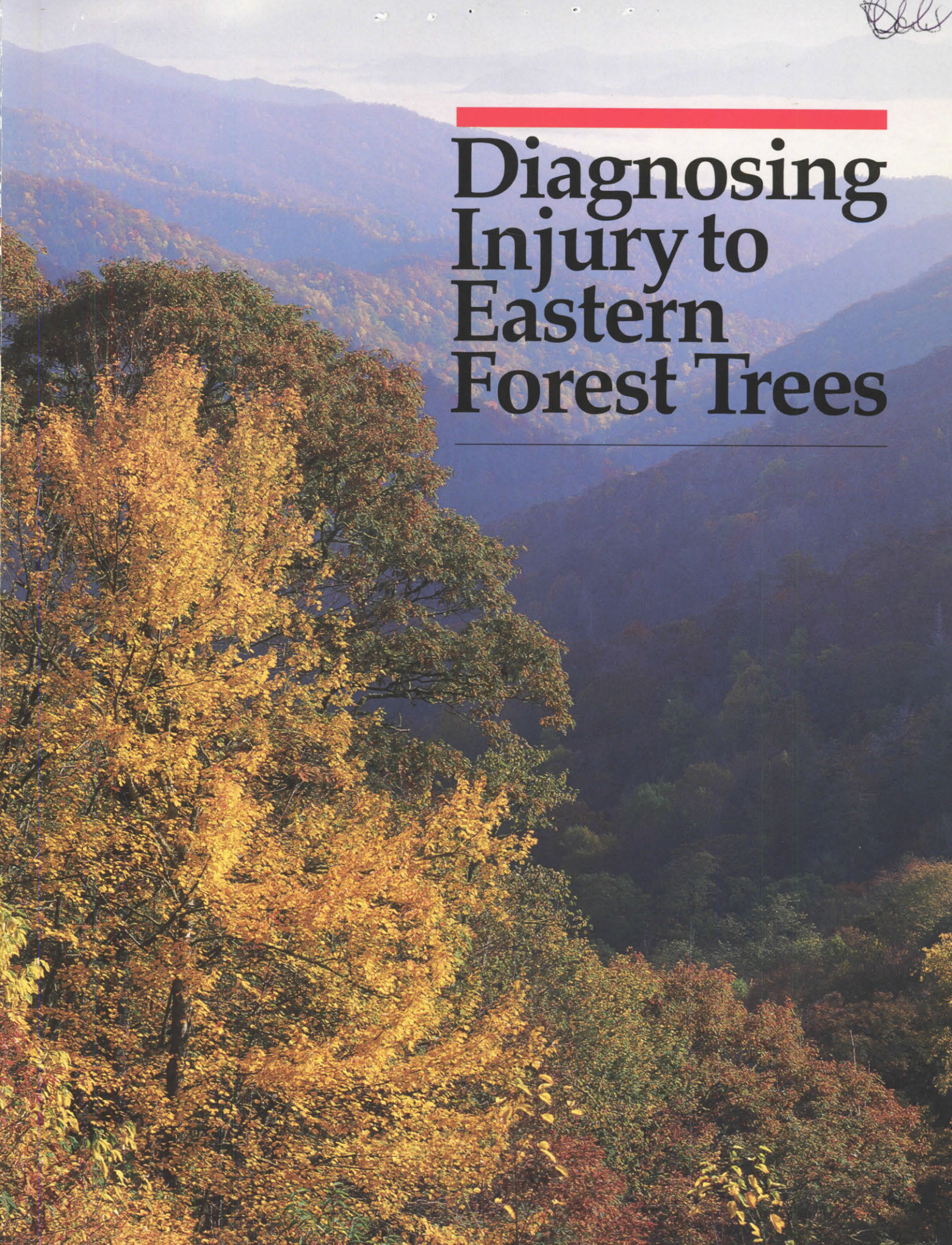

Diagnosing Injury to Eastern Forest Trees

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A manual for identifying damage caused by air pollution, pathogens, insects, and abiotic stresses

National Acid Precipitation Assessment Program
Forest Responses Program
Vegetation Survey Research Cooperative

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Preface

Awareness of and concern for air pollution injury to forest vegetation have increased over the past several decades. Our knowledge of expected air pollutant doses and their injurious effects on the foliage of forest trees and associated species has increased concurrently. Until 1985, however, critical, extensive field evaluation of air pollutant-induced injury to forest species had not been considered, either regionally or nationally.

In early 1985, under the auspices of the National Acid Precipitation Assessment Program, the U.S. Environmental Protection Agency and the U.S. Department of Agriculture Forest Service formally agreed to carry out a coordinated program of research on the effects of acid rain and its associated pollutants. Task Group F (Terrestrial Effects) of this effort includes a Forest Response Program consisting of six research cooperatives with specific responsibilities and appropriate deposition monitoring support to investigate air pollutant effects on various major timber types. The National Vegetation Survey, one of these cooperatives, has a major role in answering questions on the baseline and current condition of our nation's forest resources. Knowledge of the natural variation in forest resources is also needed in order to better understand potential atmospheric deposition effects and impacts. The USDA Forest Service chartered the National Vegetation Survey to accomplish a systematic, comprehensive inventory and analysis of those forests in the eastern United States that are exposed to air pollutants.

The National Vegetation Survey will develop and apply appropriate statistical procedures to the identification of variation in condition parameters for specified forests, stands, or tree species. At a minimum, these evaluations must have the potential to segregate natural variation from a second, unexplained category, which may be correlated with spatial patterns of various atmospheric pollutants. The spatial correlation step will require the recognition of spatial and/or temporal patterns in the existing condition of our forests.

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Purpose

The purpose of this manual is to assist members of the National Vegetation Survey in recognizing air pollutant-induced injury and in identifying disease and insect damage that may be confused with air pollutant-induced injury to forest vegetation in the eastern United States. Ozone, sulfur dioxide, and, to a limited geographic extent, hydrogen fluoride, are all pollutants of concern in relation to forest vegetation; detailed descriptions of the injuries caused by these pollutants are presented herein.

A second section of this manual has been prepared to assist the Survey in recognizing other major diseases, insects, and abiotic stresses that may be found at the survey plots. These disorders were chosen because their symptoms may be similar to those caused by air pollutants. Basic information and the most typical symptoms for each of these causes of stress are also presented photographically.

It is important that both sections of this manual be consulted thoroughly to relate field symptoms accurately to the most likely cause of the observed symptoms. In all cases, the presence of biotic pathogens, insects, and other indicators of plant stress should be investigated and recorded.

Finally, this manual has been designed to minimize errors in identifying the actual causes of symptoms evident in each plot. All potential causes must first be eliminated before the injury is ascribed to an unknown cause.

Survey crews should follow carefully the prescribed sampling and/or searching techniques for causal agents as outlined in this manual. Individuals should also consult other references to identify disease and insect problems that may not be covered in this manual.

Air Pollutants

Many gaseous pollutants exist in our atmosphere. Under widely variable forest conditions, however, only three major pollutants have been demonstrated to cause significant foliar injury to forest trees: ozone, sulfur dioxide, and fluoride. Recently, sulfates and nitrates—acid depositions—have also attracted interest, but direct foliar injury to vegetation has been observed under natural conditions only where the vegetation is very close to the pollutant sources, i.e., in a direct plume dispersion path.

Other minor pollutants, such as hydrogen chloride, bromine, particulates, and nitrogen oxides, have been shown to have local effects and may be of local importance. Survey crews must be aware of potential sources of these pollutants if they occur in areas close to established plots. If such sources are suspected of being involved in an observed symptom pattern, further checking should be done by experts.

Several available references show color illustrations of injury symptoms caused by these and other pollutants. If any one of these pollutants is suspected of causing injury observed during the survey, these references should be consulted.

VEGETATION SURVEYS FOR AIR POLLUTION

Surveying and identifying air pollutant impacts to forest ecosystems can be a complex and intensive task, depending upon the plant processes affected. Visible foliar injury resulting from exposure to air pollution commonly occurs on sensitive forest species. Symptoms of injury to sensitive species can be used to effectively survey the extent and severity of pollution injury to forest trees. The following is a discussion of survey techniques using foliar symptomatology. Refer to Skelly et al. (1979) for a more in-depth discussion of this subject.

Types of Field Surveys

The design of a field survey is generally dictated by the type of source from which the pollutant originates. Five different types of sources have been designated: single-event point sources (chemical spills); line sources (heavily utilized highways); continuous point sources (smokestacks); area sources (large urban communities); and regional sources adjoining urban or industrial areas.

Phytotoxic air pollutants can be characterized as to source type. Sulfur dioxide, hydrogen fluoride, coarse particulates, and most heavy metals except lead are emitted primarily from point sources. The principal source of atmospheric lead is the combustion of leaded gasoline; high concentrations of this metal can be found in vegetation adjacent to major highways. Ozone, acid sulfate aerosols, peroxyacetyl nitrate, oxides of nitrogen, and nitric acid vapor are considered to originate from area or regional sources, since most of these pollutants are formed secondarily by chemical reactions in the atmosphere. Acid sulfates and nitrates are currently being studied for possible direct and indirect effects.

Typical patterns of pollutant concentration are associated with a specific source type. A well-defined concentration gradient can exist in the prevailing downwind direction from a point source. Vegetation in this downwind region is often exposed to the highest pollutant doses, and a gradient of foliar injury may occur in conjunction with this concentration gradient. Injury is most severe near the pollution source and gradually diminishes in severity as the distance from the source increases. By contrast, concentrations of atmospheric pollutants resulting from area or regional sources are fairly uniform over large geographic areas, especially where terrain is relatively flat across extensive land areas. Foliar symptoms may be more intense directly downwind of urban or industrial sites but may not be limited to these regions.

A variety of survey designs have been used successfully to determine the extent and severity of air pollution effects on terrestrial vegetation. For point sources, radial transects are typically established from the center of the source. These transects are usually longer in the downwind direction and shorter in the upwind direction to delineate the impact zone most efficiently. Survey plots are located along these transects at geometric or logarithmic intervals and at distances from the source sufficient to encompass areas containing nonaffected vegetation. Survey designs for area or regional sources typically consist of some type of grid system. The grid spacing is determined by the size of the region to be surveyed and by the available resources. Plot sites are tentatively selected along grid lines on county, state, and regional maps. In practice, plot locations are determined by

accessibility and presence of selected species. Grid density can be varied to sample more intensively the vegetation surrounding urban and industrial pollution sources.

Plot Selection Considerations

Numerous physical factors can affect pollutant transport and dispersion and alter plant sensitivity to pollutant exposure. The effects of these factors on ambient-pollutant concentrations depend upon the type of emission source.

Regional scale meteorology and topography modify atmospheric conditions, thereby influencing pollutant concentrations regionally. During the summer months, large, slow-moving, high-pressure systems reduce surface air mixing and limit pollutant dispersion. Pollutant episodes may occur during these weather patterns, and high concentrations of regionally generated pollutants such as ozone and acid sulfates may persist for several hours daily until the high-pressure system passes.

In contrast, the direction of prevailing surface winds is a primary factor influencing pollutant transport from local point sources. In flat to gently rolling terrain, sources of pollution may dominate patterns of pollutant concentration on a local scale. Mountainous terrain, especially the ridge-and-valley topography typical of the central and southern Appalachian Mountains, substantially affects surface wind direction, resulting in air flow parallel to major terrain features. Pollutant dispersion is complex in these regions. Inversions are common in deep, steep-walled valleys, and high concentrations of pollutants can be expected near sources within these valleys.

Environmental and soil conditions affect the growth and physiological status of a plant and modify its sensitivity to air pollutant-caused stress. The availability of soil moisture influences plant response to gaseous air contaminants. In drought situations, plant stomata will close, limiting foliar gas exchange and reducing both the entry of pollutants into leaves and subsequent cell damage.

Soil nutrient status also can affect how a plant responds to air pollutants. Vegetation growing with limited essential nutrients is usually more sensitive to gaseous pollutants, such as ozone, than is vegetation growing with an adequate nutrient supply. Where soil sulfur is limited, however, low concentrations of sulfur dioxide or sulfate in wet deposition may enhance plant growth by increasing sulfur availability.

Species, Specimen Selection, and Timing Considerations

Air pollutants often induce a characteristic type of foliar symptom on plant species. In selecting species for a field survey, it is helpful to know what pollutants might be present at phytotoxic concentrations in the survey area. Those species that exhibit the highest sensitivity to a particular pollutant should be chosen for surveying. Ideally, two or three commonly occurring pollutant-sensitive species should be available in the survey area for a comprehensive assessment of air pollution injury.

The accessibility of foliar material for visual inspection should be considered in specimen selection. This approach limits size of specimens to those trees with foliage that can be sampled by hand or by some device such as a pole pruner. Since the canopy of a forest stand intercepts atmospheric pollutants, the concentration of pollutants decreases from the top of the canopy to the forest floor. Therefore, specimen trees selected for surveying should be dominant or co-dominant or on the edge of a stand where the foliage is openly exposed to atmospheric depositions.

The best time of year to conduct a vegetation survey in the eastern United States, particularly when ozone is the pollutant of interest, is in late summer, since the cumulative effects of ozone over a growing season may not appear until that time. Once fall coloration has begun, the foliar effects of air pollution are difficult to assess accurately.

OZONE

Ozone (O_3) is the most widespread air pollutant in the United States, causing more plant damage than any other. Injury has been observed throughout the United States in both rural and urban areas and on numerous plant species.

Sources

Ozone is known as a secondary pollutant and is formed in the air by the action of sunlight on hydrocarbons and oxides of nitrogen, the primary pollutants. Ozone is also generated naturally by electrical discharge during thunderstorms and by the action of sunlight on oxygen in the upper atmosphere. These natural sources do not contribute significantly to the ground-level concentrations of O_3, with the possible exception of those found at higher elevation sites. High O_3 concentrations usually occur many miles from the sources of the primary pollutant emissions; such concentrations can injure and retard the growth of many plant species.

Symptoms

The visible symptoms resulting from O_3 exposure are generally recognized as either acute or chronic responses. Acute injury

normally involves the death of cells and develops within a few hours or days following exposure. Expressed as stippling, flecking, bleaching, and bifacial necrosis, acute injury is usually associated with exposure to high O_3 concentrations. Chronic injury typically develops more slowly, within days or weeks following exposure, and may be manifested as chlorosis, pigmentation (stippling), premature senescence, and necrosis. Chronic injury usually appears in response to long-term, low-concentration exposure. Both symptom types, however, may occur in response to either high or low concentration exposures, depending on the environmental conditions and the genetic and physiological condition of the plant. Both types of symptoms, particularly those of chronic injury, may be confused with symptoms of other conditions, such as normal senescence, nutritional disorders, other environmental stresses, biotic pathogens, or insect infestation.

Broadleaf species: Ozone causes several general symptoms on broadleaf species in the field, the most common of which is stipple, or pigmentation.

The upper leaf surface of plants may have a tan, red, brown, purple, or black coloration that appears uniformly over the leaf surface, is restricted to certain areas of the leaf, or appears as discrete, dot-like lesions (Figures 1-8). The

Figure 1

Figure 2

Figure 3

Figure 1. Dark pigmented stipple on upper surface of green ash leaflets exposed to O_3.

Figure 2. Dark pigmented stipple on upper surface and general chlorosis of yellow-poplar leaves exposed to O_3.

Figure 3. Dark pigmented stipple on upper surface of sweetgum leaves exposed to O_3. This can become almost solid purple or maroon, similar to normal fall coloration.

upper leaf surface may be uninjured, with only the underlying palisade cells affected. Veins and veinlets are usually not involved, and veinlets often bound the injured areas, producing angular sections of affected tissue. Sometimes stippling is best observed by holding the leaf toward the sun (Figure 9); the symptom is often more intense on leaves exposed to direct light. In the absence of further stress, the stippling may fade and become difficult to discern one to four weeks following exposure.

Stippling has been described as the classic symptom of O_3 injury on broadleaf trees. The coloration of stippling is usually characteristic for a species but can vary with environmental or physiological conditions. The youngest fully expanded leaves are normally the most sensitive, although all but the very youngest leaves may be affected. On young leaves, symptoms tend to develop at the tips; on older leaves, toward the base. The entire surface of older leaves may exhibit symptoms when exposed to ozone periodically during growth. On plants with pinnately compound leaves, such as ash, hickory, and tree-of-heaven, only some leaflets at certain positions along the petiole may be af-

Figure 4

Figure 5

Figure 6

Figure 4. Dark pigmented stipple and slight chlorosis on upper surface of leaves on a black cherry seedling exposed to O_3.

Figure 5. Dark pigmented stipple on sassafras leaves exposed to O_3.

Figure 6. Dark pigmented stipple and senescence on a poplar leaf exposed to O_3.

Figure 7. Very mild stipple on upper leaf surface of sycamore seedling exposed to O_3.

Figure 8. Dark purple stipple on upper leaf surface of wild grapevine exposed to O_3.

Figure 9. Dark pigmented stipple on ash leaflet; observed when symptomatic leaflet is held up to the light.

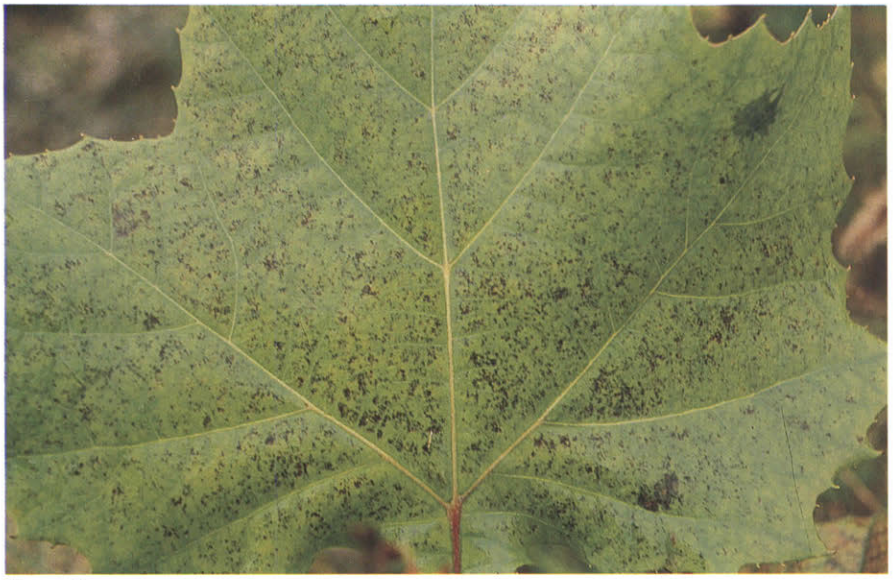

Figure 7

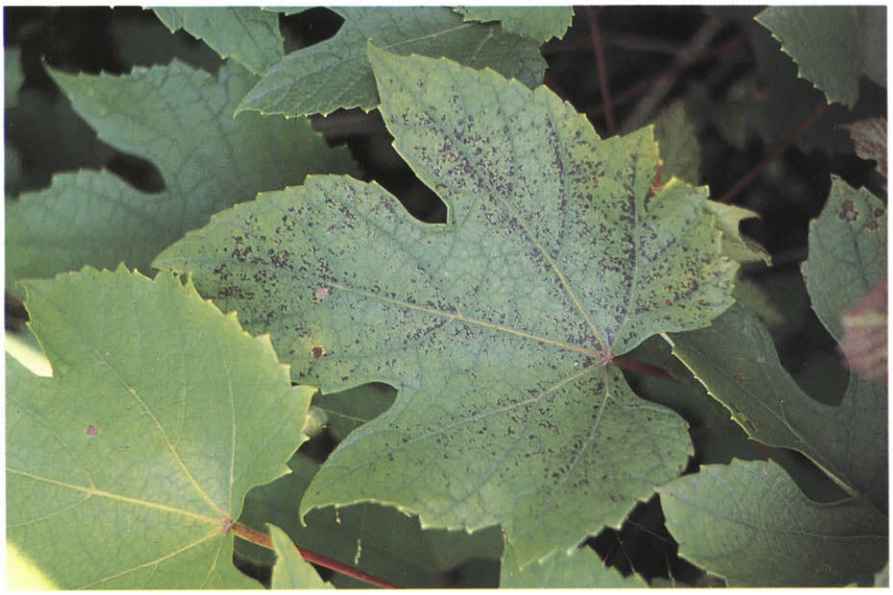

Figure 8

Figure 9

fected. Because stippling is a photosensitive response, overlapping leaflets or leaves may create a sharp line of demarcation between injured and uninjured, or shaded, tissue (Figure 10).

Chlorosis, or loss of chlorophyll, may occur as a generalized condition similar to senescence, in discrete patches called mottle, or in patterns similar to stippling. Chlorosis is often more prevalent on the upper leaf surface of species that have palisade cells (Figure 11). Fleck injury is characterized by small, discrete areas of dead cells—usually palisade and sometimes associated epidermal tissues—leading to the formation of irregular lesions on only one leaf surface, usually the upper (Figure 12). The lesions are often bleached (unpigmented) but can be colored as in stippling, and the affected areas may be slightly sunken.

Bifacial necrosis results when the tissues connecting the upper and lower leaf surfaces are killed. The tissue coloration ranges from white to red-orange to black and is often characteristic of a specific tree species. Small veins are usually included in the necrotic tissue, but larger veins often remain alive. The area of dead cells collapses, and the upper and lower leaf surfaces draw together to form a papery lesion.

Although stippling is usually interveinal, extensive injury can also affect many of the veins themselves, as in generalized chlorosis. In some species, especially sycamore, stippling tends to

Figure 10

Figure 11

Figure 12

Figure 10. Dark pigmented stipple on upper leaf surface of hickory exposed to O_3. Note protected area of leaf overlap.

Figure 11. American sycamore seedling exposed to O_3, showing tan stippling that gives an overall chlorotic and bronzed appearance to the upper leaf surface.

Figure 12. Fleck on poplar leaf exposed to O_3.

appear adjacent to the larger veins. In bifacial necrosis, the leaf margins are sometimes the most severely injured. Prolonged low dose exposures may cause coalescence of chlorotic tissues, light stippling, and/or production of a bronzed appearance. Premature defoliation may follow.

Conifer species: On conifer species, the two most common needle symptoms are chlorotic mottle and tipburn. In general, mottle of young and older needles is induced by low dose exposures, while tipburn of young needles is induced by high dose exposures. There is, however, considerable overlap depending on species, within-species variation, timing of exposures, and environmental conditions. Chlorotic mottle develops as small patches of yellow tissue surrounded by apparently healthy tissue (Figures 13-15). The tissue, especially in young needles, may also become tan. Necrotic tips result from tip dieback or from necrotic banding, which may develop when medium-aged tissue along an individual needle is most sensitive (Figure 16). Eventually, the rest of the tip portion of the needle may also die, producing necrotic tips with a reddish brown coloration that fades with age. Toward the end of the growing season, these necrotic tips may break off, making affected needles appear much shorter. The tipburn symptom, especially in white pine, usually affects all needles in a fascicle equally.

Figure 13

Figure 14

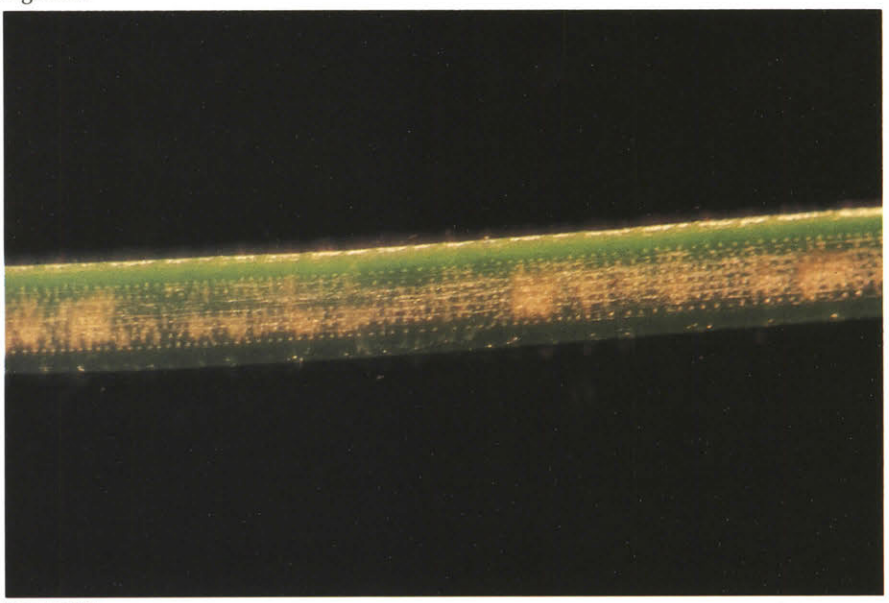

Figure 15

Figure 13. Chlorotic mottle on needles of eastern white pine exposed to O_3.

Figure 14. Chlorotic mottle, developed over a period of days or weeks, on older needles of eastern white pine exposed to O_3.

Figure 15. Close-up of chlorotic mottle on Virginia pine exposed to O_3.

Young, rapidly growing needles that are directly exposed to sunlight are the most sensitive, but older needles can exhibit mottle and premature senescence as a result of prolonged low dose exposures. As is true of broadleaf species, symptoms on plants within a single conifer species can vary considerably.

Ozone-sensitive plants exhibit premature defoliation of older needles, general needle chlorosis, and needle shortening. Such trees, known as chlorotic dwarfs, have thin chlorotic crowns and tufted foliage (Figure 17).

Grass and herbaceous species: On most grass species, tissue injury can be prominent on either leaf surface. However, as in the broadleaf and coniferous species, the leaf surface exposed to direct sunlight generally exhibits greater injury. Symptoms are often most pronounced at points of bending of leaves. Most of the grass species are less sensitive to ozone than are broadleaf and coniferous species like black cherry, yellow-poplar, and white pine.

Bioindicator species: Some particularly sensitive tree species, including white ash and clonal lines of eastern white pine, have been used as bioindicators of ozone air pollution. Black cherry appears to be a good indicator of ozone. Other forest species that may serve as bioindicators of ozone pollution are blackberry, milkweed, and poison-ivy. Blackberry

Figure 16

Figure 17

Figure 16. Tipburn on eastern white pine exposed to O_3. Length of necrotic tips may indicate pollutant concentration. Note evenness of symptoms on all needles in one fascicle.

Figure 17. Eastern white pine clones showing variable sensitivity to O_3 in trees of same age. Sensitive clone in foreground (chlorotic dwarf) and tolerant clone in background.

(Figure 18) and poison-ivy exhibit a dark purple-red stippling of the upper leaf surface that often coalesces over most of the leaf surface. The symptom on milkweed (Figure 19) is similar to that on blackberry, except that the coloration is purple-black. Milkweed has been reported as a common bioindicator of ozone.

Concerns in Diagnosis

Plants often respond to different stresses in a similar fashion. Several incitants other than air pollutants are known to cause symptoms similar to those induced by O_3. On broadleaf plants, leafhoppers and spider mites often cause a chlorotic stippling that may become pigmented with time. Close examination usually reveals evidence of the insect pest; these insects injure both the lower and upper leaf surfaces. Sunburn, sunscald, or severe late-season droughts may cause bronzing or general purpling of the upper leaf surface. Viruses can also induce chlorotic mottling. Marginal necrosis produced by O_3 on an expanding leaf may cause leaf crinkling and be misdiagnosed as a virus. Herbicide injury may also be confused with air pollution injury.

Individual plants within the same species may differ in pollution sensitivity. Genetic and microclimatic differences may produce apparently sensitive and tolerant individuals side by side, but multiple species are often found to exhibit injury at the same time. Symptoms generally appear over a whole tree rather

Figure 18

Figure 19

Figure 18. Purple-red coloration on upper leaf surface of blackberry, one of the first species to exhibit O_3 symptoms.

Figure 19. Dark stipple on upper leaf surface of milkweed and general chlorosis suggestive of senescence following exposure to O_3.

than a single stem or branch and may be more severe in plants growing on open sites. Leaf age, especially in broadleaf species, is an important factor in conditioning sensitivity; injury to very young tissue can also be caused by agents such as frost or herbicides. An appreciation of species sensitivity, classic symptom patterns, and other disease and insect-induced problems is important for accurate diagnosis.

SULFUR DIOXIDE

Sulfur dioxide (SO_2), a well-known air pollutant that is toxic to vegetation, has been recognized for causing damage to plants since the late 1800s. More studies have been conducted on the effects of sulfur dioxide than on any other pollutant.

Sources

Sulfur dioxide (SO_2) is emitted into the atmosphere when materials containing sulfur are burned. Industrial sources of SO_2 include those involved in smelting ores, manufacturing steel, and refining petroleum. Nationwide, the generation of electrical power at power stations that consume fossil fuel emits as much SO_2 as all other industrial sources combined. In many instances, major industrial sites have self-contained power plants, and, even if the primary manufacturing process does not emit SO_2, the pollutant is present downwind of such industries. Important local sources of SO_2 include fossil-fuel-fired furnaces used for space heating, refuse burning, and combustion of coal refuse piles.

The transportation industry emits some SO_2 through exhaust systems, but concentrations are rarely high enough to cause damage as a single pollutant. Sulfur dioxide is also emitted from natural sources such as fumaroles and volcanoes.

Symptoms

Sulfur dioxide injury to forest trees is restricted to localized areas immediately downwind from point sources. Foliar injury, typically most severe near the source of sulfur dioxide, decreases with distance from the industry. However, if the industry has tall smokestacks, as do many power plants, then injury to forests may occur 5 to 10 miles (8 to 16 km) downwind, where the plume touches down. Emissions from industries located in valleys usually cause more sulfur dioxide injury to adjacent forests than do emissions from sources located in flat or rolling, well-ventilated terrain. Forests on the sides of the valleys or on adjacent ridgetops may show severe sulfur dioxide injury.

Broadleaf species: Acute sulfur dioxide injury to broadleaf trees is usually observed as ivory to brown, bifacial, interveinal necrosis. The injured areas may appear initially as water-soaked, dull green areas between the veins, but this symptom is seldom observed in the field. As the injured tissue dies and dries out, the more typical symptoms occur. Leaves or leaflets with pinnate veination respond to sulfur dioxide with the classic herringbone response; i.e., injured interveinal tissue turns ivory or brown, while the tissues of and immediately adjacent to the veins remain uninjured and green. Examples of this type of acute injury pattern are shown on a single leaf of sweet birch and American chestnut and on a pinnately compound leaf of ash (Figures 20-22). In each case, the pinnate veination pattern has determined the final injury pattern induced by sulfur dioxide. Although beech trees also have pinnate veination, acute sulfur dioxide injury may be more marginal than interveinal (Figure 23).

Leaves or leaflets with palmate veination, such as maple leaves, also show acute sulfur dioxide in-

Figure 20. Bifacial interveinal necrosis on sweet birch leaf exposed to SO_2.

Figure 21. Bifacial interveinal necrosis on leaf of American chestnut sprout exposed to SO_2.

Figure 22. Bifacial interveinal necrosis on leaflets of white ash exposed to SO_2.

Figure 23. Marginal necrosis with some interveinal necrosis on American beech leaves exposed to SO_2.

Figure 20

Figure 21

Figure 22

Figure 23

jury between the veins (Figure 24). Since this vein pattern is more fan-shaped, however, the resultant pattern is not a herringbone.

Chronic sulfur dioxide injury or injury to a more tolerant species appears as chlorotic areas between the leaf veins (Figure 25). This injury pattern is more difficult to diagnose in the field, since other stresses cause similar patterns. Note that the injury pattern is similar to that of acute injury but is slightly more diffuse.

Oak foliage exposed to sulfur dioxide shows an even less classic response. The veination pattern of red oak leaves, as well as some inherent mechanism that governs response, produces irregularly shaped patches of tan, dead tissue across the leaf surface. This blotchiness can be seen on red or black oak that has been exposed to sulfur dioxide (Figure 26).

Figure 24

Figure 25

Figure 26

Figure 24. Interveinal necrosis on red maple leaves exposed to SO_2.

Figure 25. Interveinal chlorosis on elm leaves exposed to SO_2.

Figure 26. Irregular pattern of bifacial necrosis on black oak leaves exposed to SO_2.

White oak and chestnut oak respond to SO_2 with more typical symptoms (Figure 27).

Interveinal tissue injured by sulfur dioxide is not always brown or tan. Leaves with high levels of anthocyanin pigments, such as dogwood or black gum, produce a reddish interveinal response to sulfur dioxide (Figure 28). Injured hickory leaves show a reddish brown discoloration, whereas aspen or poplar leaves form reddish brown to black injury patterns. As the injured tissue on broadleaf species weathers, it often darkens with age (Figure 29).

Leaves that have recently matured appear to be most sensitive to sulfur dioxide. Therefore, species with determinant terminal growth, such as oak and ash, are more sensitive to sulfur dioxide early in the summer. Species with indeterminant growth patterns have leaves in a sensitive growth stage throughout the summer.

Conifer species: Sulfur dioxide injury to conifers is very difficult to diagnose. During the summer, diagnostic efforts should always be complemented with observations of broadleaf plants that produce more typical symptoms. Sensitive pine trees, such as eastern white, Scots, and Virginia, appear to be most sensitive to sulfur dioxide early in the summer while the needles are still elongating. Injury appears as a reddish brown discoloration of the needle tips, or, in severe cases, of the entire needle, except for the tissue adjacent

Figure 27

Figure 28

Figure 29

Figure 27. Bifacial necrosis on white oak leaves exposed to SO_2.

Figure 28. Dark, reddish interveinal pigmentation on leaves of a dogwood shrub exposed to SO_2.

Figure 29. Old bifacial necrosis on quaking aspen leaves exposed to SO_2.

to the fascicular sheath (Figures 30-32). Elongation of the needles from their meristematic base extends the area of injured tissue farther along the length of the needles. Later in the summer, the most sensitive tissue is located in a narrow band; sulfur dioxide exposure at this time results in a banding pattern of injury (Figure 33). Current-year pine needles showing severe injury seldom drop off. In contrast, spruce trees damaged by sulfur dioxide during the spring or summer usually lose their current-year needles (Figure 34). Injured larch needles tend to remain on a tree longer than do damaged spruce needles but not as long as injured pine needles.

Figure 30

Figure 31

Figure 32

Figure 30. Tip necrosis on Scots pine needles exposed to SO_2.

Figure 31. Tip necrosis on immature needles of eastern white pine exposed to SO_2 in June.

Figure 32. Tip necrosis on recently mature needles of eastern white pine exposed to SO_2 in late July.

Grass and herbaceous species: At low concentrations of sulfur dioxide, the tips of several monocotyledonous species become yellow, as do tissue areas parallel to the longitudinal veins. Higher concentrations cause leaf tip and interveinal necrosis, which is characteristically ivory or light tan. Sensitive species include annual bluegrass, oats, barley, wheat, gladiolus, and lily.

Bioindicator species: When sulfur dioxide injury is suspected, associated vegetation of the woodlands or adjacent open areas should be examined. Within the forest, tree seedlings show symptoms similar to those of mature trees. Sensitive bioindicator species, such as sarsaparilla, which occurs in the shade of more closed canopies, may be useful indicators (Figure 35). In nearby

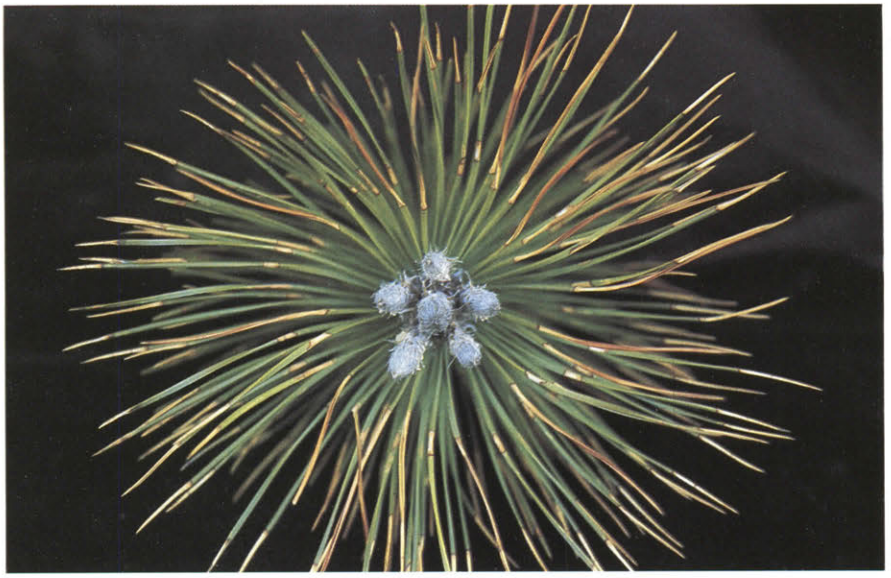

Figure 33

Figure 34

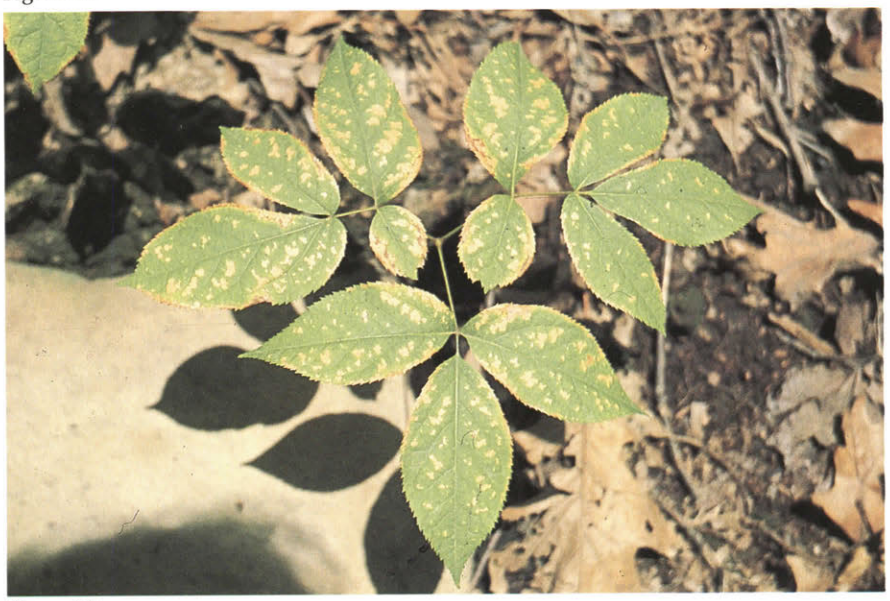

Figure 35

Figure 33. Tip necrosis with banding pattern, common on Austrian pine exposed to SO_2.

Figure 34. Current-year needle abscission on Norway spruce exposed to SO_2.

Figure 35. Bifacial necrosis on sarsaparilla leaves exposed to SO_2.

fence rows, one can usually find blackberry, which is one of the most sensitive species (Figure 36), or raspberry, which is only slightly less sensitive (Figure 37). Bracken fern, found in open areas, is sensitive to SO_2 until normal foliage browning begins in late summer (Figure 38). Disturbed areas with bare soil may have giant ragweed, another excellent indicator species (Figure 39).

Concerns in Diagnosis

Sulfur dioxide injury is usually associated with point sources, and injury patterns resemble plume dispersion patterns; therefore, distance and direction from known SO_2 sources must be noted. Several other agents may induce symptoms similar to SO_2 injury, including leaf diseases incited by fungi, insects, and nutrient deficiencies. Other stresses that induce similar symptoms include drought, frost, and pesticide exposure.

HYDROGEN FLUORIDE

Hydrogen fluoride (HF) pollution is most often a localized problem for two reasons: (1) the sources generally release the pollutant close to the ground, thereby limiting the dispersal of fluoride in the air, and (2) HF is very reactive and is quickly absorbed or adsorbed by buildings, soil, or vegetation. When HF is released from tall stacks above the ground, it may be scrubbed from the plume by other pollutants. Hence, fluoride problems usually occur within a few miles of a source of the pollutant. Under certain mountain and valley conditions where

Figure 36

Figure 37

Figure 38

Figure 36. Interveinal bifacial necrosis on blackberry leaves exposed to SO_2.

Figure 37. Interveinal bifacial necrosis on raspberry leaves exposed to SO_2.

Figure 38. Tip necrosis on fronds of bracken fern exposed to SO_2.

an industrial plume is not readily dispersed, effects of the pollutant can be observed at distances up to 12 miles (19 km).

Sources

Fluoride may occur in many forms, but the two most common are HF and silicon tetrafluoride (SiF_4). Both gases are emitted during heating or chemical treatment of fluoride-containing rocks and minerals.

Fluoride may reach the plant as a gas, a particle, or a gas adsorbed onto particles. The most important phytotoxic form of fluoride is HF gas, but particulate forms may be toxic if they exist as highly soluble salts. Natural sources of HF include volcanoes and fumaroles, which can emit large quantities of the gas into the atmosphere and cause localized problems for both plants and animals. The most important sources of fluoride are industrial processes such as the manufacture of aluminum, phosphate fertilizer, glass, bricks, steel, and chemicals, and the combustion of coal for heat or steam.

Symptoms

Symptoms can result from an exposure to high concentrations of HF for short periods of time or to very low concentrations for days, weeks, or months. Symptoms generally appear as chlorotic or necrotic areas near the margins of leaves (Figures 40-44). In such cases, measurable and elevated concentrations of fluoride are found in affected leaf tissue and are diagnostic. In general, conditions that favor rapid growth of

Figure 39

Figure 40

Figure 41

Figure 39. Ivory-colored, bifacial necrosis on giant ragweed exposed to SO_2.

Figure 40. Marginal chlorosis on a sweetgum leaf exposed to hydrogen fluoride.

Figure 41. Marginal chlorosis on sugar maple leaves exposed to hydrogen fluoride.

the plant—and therefore open stomata—also favor injury from fluoride exposures. Genetic variation has been reported to affect symptom expression in forest trees, resulting in severely injured trees being adjacent to healthy trees in the same field.

Trees on the edges of forest stands near sources of fluoride are the most likely to be injured, since the HF concentration diminishes rapidly inside a stand. In open stands, injury may be uniformly distributed within the population, and plants growing alongside openings within the stand may also be injured. Injury usually occurs at the tops of trees where foliage is exposed. Severe exposures to fluoride may produce a visible gradient or distinct area of vegetation injury close to the source.

Broadleaf species: Symptoms of fluoride on broadleaf plants consist of chlorosis of varying severity, necrosis, or both. Symptoms appear first at the margins or tips of leaves, where fluoride accumulates; with increasing severity, the symptoms extend toward the middle of the leaf. Se-

Figure 42

Figure 43

Figure 44

Figure 42. Marginal chlorosis on hickory leaves exposed to hydrogen fluoride.

Figure 43. Marginal necrosis on sumac leaves exposed to hydrogen fluoride.

Figure 44. Marginal chlorosis on an ash leaf exposed to high concentrations of hydrogen fluoride.

vere exposure can cause necrosis of tissues between veins similar to that caused by sulfur dioxide, except that fluoride does not bleach the necrotic area. Chlorosis is not usually present on leaves exposed to high concentrations of HF. In many species, the necrotic tissue is reddish brown. Expanding leaves are generally most susceptible.

Less severe exposures may cause a mild marginal chlorosis or anthocyanosis. In addition, leaves may become cupped, indicating retarded growth at the tips and margins of the leaves (Figures 45, 46). In many instances, the cupping is related to a notched tip or small necrotic area on the leaf tip which may have broken off and is, therefore, no longer visible.

Conifer species: Symptoms of fluoride injury on conifer needles may easily be confused with symptoms caused by other pollutants. The most common symptom observed on conifers exposed to HF is tipburn, or necrosis of the needle tips (Figures 47-50). The injury pattern is uni-

Figure 45

Figure 46

Figure 47

Figure 45. Anthocyanosis and cupping of blueberry leaves exposed to hydrogen fluoride.

Figure 46. Chlorosis and cupping of sassafras leaf exposed to hydrogen fluoride. Note uninjured leaf on left.

Figure 47. Tip necrosis on needles of eastern white pine exposed to hydrogen fluoride.

form over the exposed portion of the tree (Figure 51). The more severe the exposure or the more fluoride accumulated, the greater the extent of the necrosis. The boundary between living and dead tissue is distinct and even. Necrotic tissue directly adjacent to the living tissue may be a shade darker than the remaining dead tissue, and sometimes a reddish band develops (Figure 52). Subsequent exposures may produce additional bands of necrotic tissue, each with a ring of darker tissue demarcating the extent of the previous injury. Exposures that cause necrosis are of three types: (1) a single severe exposure that causes death of the entire length of needle, (2) a series of severe exposures that cause sequential areas of necrosis, or (3) a series of long-term exposures at lower concentations that cause elevated fluoride concentration high enough to cause tip necrosis in needle tissues.

Mild exposure to HF may cause chlorosis of the needle tips, but this symptom generally appears on no more than a few millimeters of the needle length.

Figure 48

Figure 49

Figure 50

Figure 48. Tip necrosis on needles of slash pine exposed to hydrogen fluoride.

Figure 49. Tip necrosis on needles of Scots pine exposed to hydrogen fluoride.

Figure 50. Tip necrosis on needles of spruce exposed to hydrogen fluoride.

Grass and herbaceous species: Symptoms on monocots fall into two categories: stippling and flecking, and tip necrosis. In grain plants such as wheat, barley, and corn, chlorotic stipples first appear at the tips and margins of the leaves. As the severity of exposure increases, stipples form down the margins from the tips and inward toward the midrib. The stipples may coalesce to form chlorotic bands. Necrosis follows the same general pattern, beginning at the leaf tip and extending inward and down the margins of affected leaves.

Bioindicator species: Bioindicators of fluoride pollution may be either plants that are extremely sensitive to the pollutant and show visible symptoms, even from mild exposure, or plants that accumulate fluoride from the atmosphere in a predictable fashion. Among the best indicators —those that respond by producing visible symptoms—are redbud, catbrier, St. John's-wort, wild grape, birch, and young expanding needles of most pines and spruces. Nonforest species that may serve as bioindicators include gladiolus and field or sweet corn. In plants such as gladiolus, symptoms begin with tip necrosis

Figure 51

Figure 52

Figure 51. General needle necrosis on eastern white pine exposed to high doses of hydrogen fluoride.

Figure 52. Tip necrosis and chlorosis on loblolly pine needles exposed to hydrogen fluoride.

(Figure 53). Similar symptoms are observed in tulip, iris, lily, and some other monocots.

A second type of bioindicator program requires chemical analysis of plant tissue to determine fluoride concentrations. In such a program, it is desirable to use a plant that is a good accumulator of fluoride but does not have a high natural level in leaves. Species such as grasses and pines are good choices; dogwood and hickories are not, because they accumulate fluoride from the soil.

Concerns in Diagnosis

In the field, symptoms may be infrequent or atypical of those normally associated with fluoride pollution; it is important, therefore, to establish the suspected sources of the pollutant. Fluoride problems are generally localized to an area within a few miles of the source, except under special circumstances. If the area of impact is far removed from a source, alternative causes should be considered.

After the source has been located, a survey should be conducted to establish the pattern of injury around it; the incidence and severity of injury usually increase as distance from the source decreases. The appearance of species known to be sensitive should be noted. Symptoms may or may not be uniformly distributed, depending on the exposure of the plant. As part of the survey, the same species of plants should be observed at sites that are physically similar but far removed from the source of pollution in order to establish that symptoms are absent under clean-air conditions.

Figure 53

Figure 53. Tip and marginal necrosis on *Gladiolus* exposed to low concentrations of hydrogen fluoride.

Forest Tree Diseases

Numerous forest tree diseases occur in the temperate regions of the eastern United States. In many instances, these diseases have symptom complexes similar to those induced by air pollutants. Causal agents range from simple abiotic stress, such as prolonged drought or spring frost, to complexes of fungi, insects, and abiotic stresses. In this section, the examples of leaf diseases, twig and stem cankers, root diseases, and slowly occurring diebacks and declines reveal similarities between symptoms of these diseases and symptoms caused by air pollutants. In many instances, the biotic pathogen being reviewed has not been associated with specific sites; however, its general range has been presented.

This section of the manual is divided into two parts: Hardwood Diseases and Conifer Diseases. Both parts are organized by disease types, based on the part of the tree affected and on symptom expressions. The causal agent for each disease is also presented.

HARDWOOD DISEASES

Diebacks and Declines

In recent decades, dieback and decline diseases have killed or damaged millions of trees in the eastern United States. Most hardwood tree species are affected by these disorders at some point, but relatively few species have suffered greatly. Most dieback and decline diseases of forest trees share a common type of cause-and-effect relationship that serves to define and differentiate them from other types of disease.

The syndrome shared, in part or in whole, by many of these diseases frequently has two phases—a dieback phase and a decline phase. The term *dieback-decline* is a descriptive label that implies a progressive development of symptoms, beginning with the dying back of buds, twigs, and branches, progressing inward and downward, and culminating in the decline and death of the tree. Leaves of declining trees are often small, sparse, and off-color; foliage in successive years is borne on sprouts and may appear clumped or tufted; leaves show premature fall colors and drop; and terminal and radial growth is often reduced even before external symptoms appear.

Diebacks and declines are complex diseases initiated by adverse environmental factors that create biotic and abiotic stress and often culminate in lethal attacks by organisms that are otherwise not harmful. Thus, these diseases differ from those caused by single primary pathogens in that trees suffer from a multiplicity of abiotic and biotic stress factors. In the context of these diseases, predispositional stress refers to environmental pressure sufficient to trigger changes in the physiology, form, or structure of a tree. The stress factors can be abiotic (e.g., extremes of moisture or heat) or biotic (e.g., insect defoliation, scale or aphid attack, infection by fungi, or combinations of these). In the absence of such stresses, the organisms of secondary action, often ubiquitous in the ecosystem, occupy various niches ranging from saprophyte to weak pathogen. Without these organisms, trees would most likely recover when the stress abates. This common cause-effect definition can be expressed as a series of stages:

1. Healthy trees + stress = altered trees (dieback begins)

2. Altered trees + more stress = trees altered further (dieback continues)

3. Significantly altered trees + organisms of secondary action = trees invaded, decline, and perhaps die.

These statements imply that the dieback stage often results from stress alone and that when stress abates without lethal attacks by secondary organisms, dieback often ceases and trees recover. The decline stage, in which vitality lessens and trees succumb, usually occurs when organisms attack the stress-altered tissues. Recovery from this phase, which is less likely than recovery from the dieback stage, depends on such factors as tree vigor, location and severity of tissue invasion, and aggressiveness of the secondary organisms involved.

These statements do *not* imply that severe stress, if repeated or prolonged, cannot by itself cause a decline of tree vigor and even death.

Because diebacks and declines are frequently initiated by broad environmental changes, they may suddenly emerge over a wide area, and the types of sites where they develop may appear to be closely related.

Oak decline and mortality (Figure 54) over large areas of Pennsylvania, West Virginia, New York, and many other states is related in time and place to drought and severe insect defoliation. Similarly, a dieback of white and green ash (Figure 55), triggered by defoliation caused by the ash rust fungus, is limited to coastal regions by the restricted range of the alternate hosts, the salt marsh grasses. In Massachusetts, an extraordinarily high number of maple disorders not attributable to primary pathogens occurred regularly two years after periods of severe water shortages. A decline in the roadside sugar maple population (Figure 56) and beech bark disease (Figure 57), triggered by applications of winter road salt and infestations of beech scale insects, respectively, is obviously related in time and place to these initiating stresses.

The diagnosis of dieback and decline disease involves the following procedures:

1. Recognizing symptoms, including progressive dieback and deterioration of crowns; thin, sparse, tufted foliage produced on main stem sprouts; early fall coloration; and reduced height and radial growth rates.

2. Identifying which events may have triggered the problem. Records of disturbances such as weather extremes, insect defoliation, fire, and logging activity are extremely valuable. Identifying when disturbances happened by examining growth ring patterns or age of sprout tissues may help to pinpoint the disturbances and when they occurred.

Figure 54

Figure 55

Figure 54. Oak decline and mortality in a mixed oak/hickory stand. Branch dieback and whole tree mortality are present.

Figure 55. Ash dieback with typical symptoms of reduced viable crown and abundant sprouting along main stem and lower branches.

3. Determining the presence and developmental stage of secondary organisms, such as root-invading and twig-cankering fungi and bark borers. This determination also helps to confirm the diagnosis and date of the events that triggered these attacks.

Knowing when and where significant disturbances occur today will help predict when and where they may occur tomorrow. It is also important to realize that a multitude of stress-host interactions can trigger dieback and decline diseases and that only diligent observation can determine the underlying factors. Several examples of important diebacks and declines are discussed in more detail in the following sections.

MAPLE DECLINE

Disease description: Early symptoms of maple decline include late foliage flush in the spring, smaller, slightly chlorotic leaves, twig dieback, reduced growth, and premature leaf coloration in mid- to late summer. Lateral buds on dying twigs sprout, producing tufts or clumps of foliage that may be off-color in the spring (Figure

Figure 56

Figure 57

Figure 56. Decline of roadside sugar maple; in this case, most likely due to deicing salt application and subsequent uptake through roots.

Figure 57. Beech bark disease on an isolated American beech, leading to mortality and a hole in the forest canopy.

58). Twig dieback appears initially in the upper branches; the progressive death of branches inward and downward yields the characteristic stagheaded tree (Figure 59).

Death of weakened twigs and branches can be hastened by attacks of weakly pathogenic fungi, such as *Stegonosporium ovatum*. Roots of weakened trees are often rapidly invaded and killed by the shoestring root rot fungus, *Armillaria*. (See oak decline for details on *Armillaria*.)

Cause: Maple decline in the forest often occurs after defoliation by a number of insects, including leafrollers and webworms, the saddled prominent, and the forest tent caterpillar. More recently, drought and severe winters with little snow cover have been linked to maple decline.

Suscepts: Maple decline is primarily a disease of sugar maple and occurs throughout its range. Crown deterioration of red maple has been observed in forests after gypsy moth defoliation as well as during and immediately after years of heavy seed crop.

Range and site description: Decline has been observed throughout much of the range of the two suscepts. Pockets of decline may be found across many sites, depending upon the agents involved, e.g., droughty soils or those recently attacked by defoliating insects. Many sugar maple trees have declined and died in the Northeast. While the reasons for this are not thoroughly understood, the decline has been most severe in sugar maple stands subjected to drought, heavy grazing, or

Figure 58

Figure 59

Figure 58. Foliage discoloration on sugar maple suffering from maple decline.

Figure 59. Top dieback or "staghead" symptom of maple decline on sugar maple.

overtapping and heavy traffic by farm machinery used in sap gathering. Many of the seriously affected trees are overmature and have been heavily tapped for many years. These trees are usually riddled with stain and decay associated with tapholes and root wounds; insect defoliation often accelerates decline in these stands.

Diagnostics: Look for current evidence of defoliating insects, fungal fruiting structures on leaves, rhizomorphs of *Armillaria* under the bark at the base of the tree or along excavated roots, and/or sporophores of *Armillaria* at the base of trees or stumps. Look at site specificity, i.e., droughty soils and frost pockets. Compile site history from previous records of insect-caused defoliation, sugarbush operations, fire, logging, and so on.

SAPSTREAK DISEASE OF SUGAR MAPLE

Disease description: The first noticeable symptom of sapstreak of sugar maple is a dwarfing of the foliage on all or a portion of the crown; dwarfing becomes more intense and may spread to previously unaffected parts of the crown in following years (Figure 60). In addition, some branches may begin dying back. Trees without crown symptoms one year may fail to leaf out the next, while others may die a year after the first foliar symptoms. Some trees linger for several years, exhibiting repeated sequences of crown dieback and recovery, until they finally die.

Inside the tree, the wood of buttress roots and lower stems exhibits a stain of distinctive color and pattern (Figure 61). When

Figure 60

Figure 61

Figure 60. Poorly developed small leaves on sugar maple due to sapstreak disease, resulting in thin and ragged crowns.

Figure 61. The wood of a buttress root of sugar maple, showing streaking and discoloration due to sapstreak disease.

freshly exposed, the stained tissue is yellowish green, is bordered by a thin, dark green margin, and appears water-soaked. Reddish flecks are visible in freshly exposed stain areas. Within a few minutes of exposure to air, the stain darkens dramatically and the red flecks become less discernible. On drying, the discolored tissues fade to a light brown. In cross section, the stain appears to radiate outward toward the bark in fingerlike or starlike projections. Cambium infiltrated by the stain dies and cankers develop. In severely diseased trees, this radiating pattern disappears as the entire cross section of roots becomes stained.

Cause: Sapstreak disease is caused by the fungus *Ceratocystis coerulescens*. This fungus is a common stainer of hardwood logs and lumber, but it sometimes can enter and kill living trees as well.

Suscepts: Sugar maple is the most common suscept.

Range and site description: Sapstreak has been reported in North Carolina, Michigan, Wisconsin, Vermont, and New York. It is very likely, however, that the disease occurs throughout the range of sugar maple wherever the conditions for infection occur in the presence of the causal fungus. The fungus appears to enter the tree primarily through logging wounds, either on roots or near the base of the trunk (Figure 62). Because such wounding may occur many years before symptoms develop, callus sometimes nearly closes the wounds by the time the disease is recognized. Disease development seems to be related to wounding

Figure 62

Figure 63

Figure 62. Logging wounds at the base of a sugar maple, which may serve as entrance courts for the sapstreak fungus.

Figure 63. Waxy secretions of beech scale insect evident on the bark of an American beech following a heavy infestation.

involved in sugaring operations or in road construction and related mechanical injuries.

Diagnostics: Look for small leaves, thin crowns, dieback, and stain of the wood in the buttress roots and root-collar zones when exposed to air. Look for cankers near the groundline and note site conditions, including recent logging or sugarbush operations.

BEECH BARK DISEASE

Disease description: Beech bark disease results in significant mortality and defect in American beech. The white wax secreted by the beech scale insect is the first sign of invasion. Isolated dots of white "wool" appear on the bole of the tree on roughened areas of bark, beneath mosses and lichens, and below large branches. Eventually, as the insect population increases, the waxy scales may cover large areas of the bole (Figure 63).

Serious damage results only after the fungus *Nectria* invades scale-infested bark. *Nectria* produces two types of spores: asexual and sexual. Asexual spores are produced in the summer in small, white cushions that resemble the woolly wax produced by the scale. More obvious are the tiny, bright red, lemon-shaped perithecia, which produce the sexual spores (Figure 64). Perithecia are often produced in clusters on dead bark; they mature in the fall.

On some trees, a red-brown exudate called a slime flux or tarry spot oozes from dead spots. Tarry spots are often the first symptom of Nectria infection. If the outer bark of an infected tree is cut away, a distinct orange coloration

Figure 64

Figure 65

Figure 64. Red fruiting bodies (perithecia) of *Nectria* on the trunk of an American beech formerly infested by the beech scale insect.

Figure 65. Sunken and callused-over cankered areas on American beech tree severely affected with beech bark disease.

may be seen where *Nectria* is actively invading the bark. The fungi may infect large areas on some trees, completely girdling them. On such trees, large numbers of perithecia often form, giving a reddish cast to extensive areas of the bark. On dying trees, leaves that emerge in the spring do not fully enlarge, giving the crowns a thin, open appearance. Later, the leaves turn yellow and usually remain on the tree during the summer. Although severely affected trees exhibit progressive crown deterioration symptoms, they may live for several seasons. The rate of crown decline depends primarily on the severity of scale and fungus attack.

The fungus frequently infects only narrow strips or patches on the bole, and the resultant symptoms differ from those of girdled trees. Callus tissue forms around these areas, and the bark becomes roughened (Figure 65). Callus tissue may wall off small Nectria cankers from the remainder of the living sapwood.

Cause: Beech bark disease occurs when bark that has been attacked and altered by the beech scale insect, *Cryptococcus fagisuga*, is invaded and killed by one of two fungi, *Nectria coccinea* var. *faginata* or *N. galligena*.

Suscepts: American beech is susceptible. Although small beech trees can be infested by beech scale and infected by *Nectria*, heavy attacks are rare. Consequently, mortality is significant only in trees with a diameter larger than about 8 inches (20 cm).

Range and site description: Beech scale has been found in Quebec and throughout New England, New York, New Jersey, and northern and eastern Pennsylvania. Recently it has been found infesting stands in northeastern West Virginia, in adjacent areas of Virginia, and in several counties in northeastern Ohio. It seems likely that the disease will continue to spread throughout the range of beech.

Diagnostics: The pattern of insect spread and the subsequent occurrence of Nectria infection and tree death have led to the following arbitrary classification of disease development over time and space:

- The advancing front—areas recently invaded by beech scale and characterized by forests with many large, old trees supporting scattered, sparse, but increasing populations of beech scale.
- The killing front—areas characterized by high populations of beech scale, severe *Nectria* attacks, and heavy mortality of trees.
- The aftermath zone—areas that at some time suffered heavy mortality and that now have some residual large trees and many stands of small trees, often of root-sprout origin. In the aftermath zone, young stems are often rendered highly defective by the interactions of beech scale, *Nectria*, and another scale insect, *Xylococculus betulae*.

Caution must be exercised in diagnosis, since thin crowns and small chlorotic leaves are general responses to other stresses. Chlorosis of beech, for example, can result from an iron deficiency.

OAK DECLINE

Disease description: Trees affected by oak decline show a general and progressive dying back from the tips of the branches (Figure 66). Other symptoms include chlorotic, dwarfed, and sparse foliage; sprouts on main branches and stem; and premature autumn leaf color and leaf drop. Growth decline is also evident.

As dieback and reduced growth continue, larger branches die and form the characteristic stagheaded crown. Foliage is mainly limited to sprouts on the larger branches and main stem. Trees eventually succumb, sometimes suddenly, to the root-killing and girdling actions of secondary insects and diseases.

The two major pests associated with oak decline are *Armillaria mellea*, the cause of shoestring root rot, and *Agrilus bilineatus*, the two-lined chestnut borer.

The common forest fungus, *Armillaria*, attacks the roots of stressed oaks from rootlike structures called rhizomorphs that grow through the soil and over the surface of tree roots (Figure 67). Infection eventually girdles the large buttress roots and root collar and kills the tree. White mycelial fans may form under the

Figure 66. Early decline and crown dieback symptoms on an unidentified oak species with typical twig and small branch dieback evident.

Figure 67. Shoestring-like rhizomorphs of *Armillaria* covering a piece of fallen wood. Such structures are evident under the bark of roots and lower stems.

Figure 66

Figure 67

bark (Figure 68), and, in autumn, clusters of honey-colored mushrooms may form at the base of invaded trees (Figure 69). *Agrilus bilineatus* attacks the branches and stems of weakened trees; the larvae begin feeding and form meandering galleries in the inner bark and outer wood (Figure 70). These borers first infest branches in the upper crown; later infestations are lower, often reaching the base of the tree. The combined actions of the borer in the stem and the fungus in the roots can bring about rapid decline and death. Final symptoms of oak decline reflect the root-killing and girdling effects of these organisms. Leaves sometimes fail to develop in spring, wilt shortly after budbreak, or suddenly wilt or turn brown late in the growing season.

In some instances, decline and death may occur over a short period of one to two years or even within one growing season. Such declines are usually seen on ridges or other drought-prone areas (Figure 71). Symptoms develop rapidly and usually involve the entire crown; all leaves turn brown but remain attached throughout the growing season. Short-term declines are most common in suppressed, overtopped, and/or otherwise stressed trees.

Figure 68

Figure 69

Figure 68. White mycelial fans of *Armillaria* under the bark of trees suffering from crown dieback and mortality.

Figure 69. Cluster of honey-colored *Armillaria* mushrooms at the base of an infected tree. Clusters may be formed along roots or slightly higher up the stem.

Figure 70. Feeding galleries of the two-lined chestnut borer, which attacks oaks in various stages of dieback and mortality.

Figure 71. Seasonal decline, dieback, and mortality of oak on ridges, following defoliation by insects or persisting drought conditions.

Figure 70

Figure 71

Another characteristic of short-term decline throughout the Southeast is the almost universal occurrence of the sap-rotting fungus, *Hypoxylon atropunctatum*, on boles and larger branches of affected trees. This fungus, a bark inhabitant, is an early, rapid colonizer of the sapwood of stressed or recently dead red oaks, white oaks, and hickories. Presence of the solid, black-fruiting bodies of this fungus on tree stems can reliably indicate an immediate past history of biotic or abiotic stress (Figure 72).

Cause: Oak decline is stress-initiated, the most frequent stress factors being drought, frost injury, and insect defoliation. Trees on dry ridge tops and in wet areas suffer the most from major fluctuations in moisture conditions that lead to severe drought injury. Frost often affects trees growing in valleys and in associated frost pockets. Defoliated trees that refoliate the same season may exhibit dieback symptoms the next year. Other factors such as leaf diseases and waterlogged, compacted, or shallow soils have been implicated in oak decline. Waterlogging is especially important in the heavy clay soils of the Midwest. These stress factors weaken trees so that they succumb, sometimes suddenly, to pathogenic organisms and insects.

Suscepts: Oak decline has occurred throughout the range of oak in both forests and in urban environments. The most frequent and severe outbreaks have been in southern New England, the Middle Atlantic states, and the Southeast. Red, scarlet, pin, and black oak in the red oak group and white and chestnut oak in the

Figure 72

Figure 72. Fruiting bodies of *Hypoxylon*, which has invaded and colonized the bark and sapwood of a dying oak as part of the oak decline complex.

white oak group are the most significantly affected species.

Range and site description: This disease may be found on specific sites throughout the range of oak. A characteristic feature of oak decline is its sudden development on many trees over large areas affected by the initiating stress factor. Within the affected areas, decline and mortality may be in patterns, reflecting the intensity and severity of the stress, distribution of hosts, aggressiveness of *Armillaria mellea,* abundance of two-lined chestnut borers, and other site features such as frost pockets and dry, shaly soils.

Diagnostics: Look for current evidence of defoliating insects, fungal fruiting structures on leaves, rhizomorphs of *Armillaria* under the bark at the base of trees or along excavated roots, and sporophores of *Armillaria* at the base of trees or stumps. Look at site specificity, i.e., droughty soils or frost pockets. Compile site history from previous records of insect-caused defoliation, fire, logging, and so on.

Wilt Diseases

OAK WILT

Disease description: Oak wilt is a vascular wilt disease characterized by a sudden wilt of foliage in infected trees. Observable symptoms appear from early June until leaf coloration is completed in the fall. There are two distinctly different symptom patterns, depending upon the group of oaks affected.

■ Red oak group: Symptoms are characterized by a sudden wilting of the leaves at the top of the tree; wilting progresses downward through the crown very rapidly (Figures 73, 74). Wilting and

Figure 73

Figure 73. Rapid oak wilt development of red oak in Pennsylvania, ten days following an inoculation by *Ceratocystis fagacearum.*

water-soaking (Figures 75, 76) of the leaves are most often accompanied by leaf fall before the leaves have turned brown. Defoliation of the entire tree is usually complete in three to six weeks after infection. The petioles of wilted leaves are black at the base, and the outer sapwood of diseased branches and main stems may have a dark-streak discoloration. Identifying signs of the causal fungus are mycelial, oval-shaped pads, which are produced under the bark of infected trees three to nine months after wilting and defoliation (Figure 77). Appearing in late spring and early fall, these black and gray pads force open the bark; their fruitlike odor is very attractive to insects. Lightly tapping the bark for a hollow sound will assist in locating these pads.

■ White oak group: Symptoms are characterized by a slow wilt of a single branch. Leaves usually remain on the tree, and only the terminal portion of the affected leaves turns brown. Death of the entire tree is rare but may occur one to four years after infection. The killing of individual branches over a period of years, which causes stagheading of the crown, may be confused with oak decline. Fungus pads are only rarely produced on infected white oaks.

Cause: Oak wilt is caused by the fungus *Ceratocystis fagacearum*. After the fungus invades the vascular system of the tree, it induces formation of tyloses, which further hinder water transport. No other stress agents are involved.

Figure 74

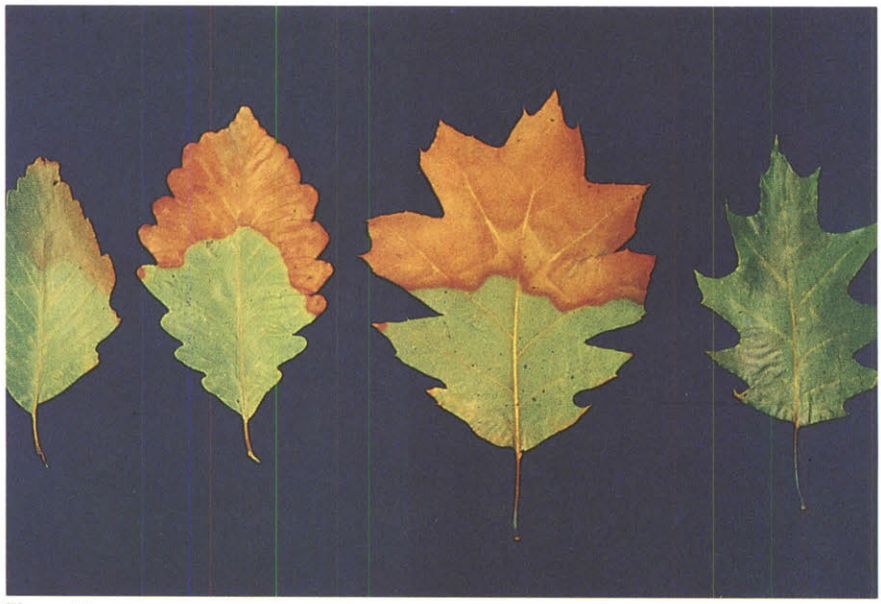

Figure 75

Figure 74. Crown of red oak, showing typical oak wilt symptoms consisting of severely wilted leaves and rapid defoliation, leaving a thin, dying crown.

Figure 75. Oak wilt symptoms of chestnut and red oak leaves, showing the line of demarcation between green and dying tissues. Water-soaking symptoms are evident on the leaf to the right.

Suscepts: There are between 200 and 300 species and hybrids of oaks. To date, not one has proven immune to the attack of the causal fungus. Species belonging to the red oak group (black, scarlet, pin, southern red, and northern red oak) are more susceptible to infection than are species of the white oak group (white, chestnut, swamp white, and post oak). American and oriental chestnut, chinkapin, tanoak, and apple are also susceptible.

Range and site description: The range of oak wilt extends from Minnesota to Pennsylvania to North Carolina to central Texas; however, it exists primarily in the mountainous section of the East, particularly on west-facing slopes and ridgetops. The disease has not been found in New England, New York, New Jersey, or east of the Susquehanna River in Pennsylvania. The disease may affect individual trees, but there may also be centers of infection with numerous dead trees adjacent to currently wilting trees. Spread by local root graft accounts for these infection centers. The rapidity of wilt and defoliation — leading to death — in red oak is quite distinctive to this disease.

Diagnostics: Oak wilt, unlike oak decline, occurs in isolated trees or small pockets of trees with no history of abiotic stress. Particularly in white oaks and sometimes in red oaks, laboratory diagnosis is usually necessary to conclusively identify trees dying from infection by the oak wilt fungus.

Figure 76

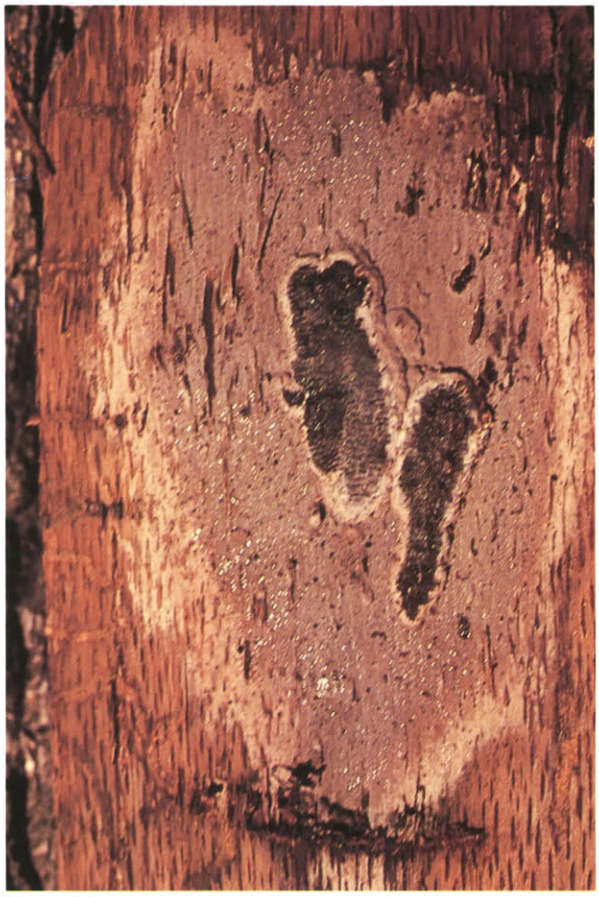

Figure 77

Figure 76. Oak wilt symptoms of live oak leaves following infection by *Ceratocystis fagacearum*.

Figure 77. Oval mycelial mat of the oak wilt fungus found under the bark of dying or dead oaks. The mat has a fruitlike odor.

Leaf Diseases

Most tree species produce an abundance of leaves far exceeding the numbers required for normal growth. Therefore, unless severe defoliation takes place, enough leaf surface is usually left to produce normal annual growth. When a tree loses all or most of its leaves to disease or other injurious agents, poor vigor, growth loss, and even death may follow. Early summer refoliation, during which food reserves are depleted as a second flush of leaves is produced, may eventually cause decline and death of affected trees.

Most organisms that cause leaf disease are favored by cool, wet weather at the time of leaf development in early spring. Only a few diseases develop late in the summer months when the weather is hot and dry.

Numerous leaf diseases occur on eastern forest trees. Those of a fungal nature usually show evidence of some type of fruiting structure, growth (zone) lines of increasing lesion size, or a distinctive symptom pattern associated with veins or veinlets. Careful diagnosis is required to distinguish the representative diseases presented below, as well as other disorders, from air pollutant-incited injury.

ANTHRACNOSE

Disease description: Anthracnose is characterized by sharply defined areas of dead tissue on affected leaves. These areas usually are found in association with veins on the leaf, but, in severe cases, entire leaves may die. Infected young leaves may become distorted due to the continued growth of noninfected tissues on the periphery of infected, dying areas (Figure 78).

Sycamores are particularly susceptible to infection by the anthracnose fungus, which may advance into small twigs, where it causes cankers. Such infections may remain active for several years and may eventually kill the terminal portions of twigs and branches.

Anthracnose of other species may be evidenced by smaller spots on the leaves that are black with irregular borders. Small, dark fruiting bodies of the causal fungi may form on the infected tissues. With the aid of a hand lens, one may see these fruiting bodies embedded in the dead tissues.

Severe infection of leaves causes defoliation during late spring and early summer. In some instances, only the top of the crown remains unaffected, giving the tree a tufted appearance (Figure 79). Refoliation of affected trees is usually poor and results in a very thin crown.

Cause: Anthracnose is caused by several species of fungi in the genus *Gnomonia*.

Suscepts: Most hardwood trees are susceptible to infection by the causal fungi. Sycamore, oak, maple, horsechestnut, and black walnut are most severely affected. Hickory, ash, and several other minor species are affected less severely.

Range and site description: While anthracnose is found throughout the United States, it is primarily a concern in the eastern, southern, and central hardwood forests. Disease development is favored by cool, wet weather in early spring.

Diagnostics: Sharply defined areas of dead tissue on leaves characterize anthracnose. This disease may be confused with acute SO_2 injury. Extent of disease development and patterns of injury in the stand should be noted. Examination of dead tissues for *embedded* fruiting structures will assist diagnosis.

LEAF BLISTER OF OAK AND OTHER SPECIES

Disease description: Leaf blister is characterized by raised, wrinkled, and brown blisters on affected leaves during late summer or early fall (Figure 80). Initial symptoms involve a slight yellowing of the infected tissues. Blisters form when the fungus stimulates cell growth after the surrounding infected cells become rigid. These blisters are usually less than 1 inch (2 cm) in diameter, and they remain light green in color for several weeks before tissue death turns them brown. Severe infection results in numerous blisters on a single leaf and thus leaf distortion also occurs. Premature leaf fall may occur in early autumn. Such defoliations stunt growth and may weaken the tree's resistance to attack by other agents. This disease is favored by cool, wet weather in spring.

Cause: The fungus *Taphrina coerulescens* causes leaf blister.

Suscepts: While all oaks are susceptible to leaf blister, black and red oaks are the most susceptible to attack. Other species occasionally affected include poplar, birch, and cherry.

Range and site description: Leaf blister occurs throughout the United States but is important on oaks only in the East. A major leaf disease of oaks in the southern United States, leaf blister occasionally causes 50 to 85 percent defoliation of affected trees by midsummer.

Diagnostics: Initial yellowing of foliage, usually in a circular pattern, is quickly followed by formation of a raised blister. Examination later in the season will detect raised dead areas and considerable leaf distortion.

Figure 78

Figure 79

Figure 80

Figure 78. Anthracnose of ash leaves showing distortion due to necrosis of infected tissues.

Figure 79. Anthracnose of sycamore; infection of leaves and twigs will result in thin crowns and a tufting appearance of the top crown; both dead and green leaves may be found under the tree.

Figure 80. Leaf blister disease on red oak leaves, showing ragged, raised blisters in circular and coalesced patterns.

Disease description: This rust disease alternates between poplars and larches. The most significant damage occurs on poplars, where up to a 20 percent reduction in radial growth has been observed following repeated defoliations. Symptoms on poplar include yellow to orange bifacial spots, which contain pustules of the rust fungus in the uredial, telial, and basidial stages (Figure 81). Spots later turn purple-brown, and severely infected leaves fall prematurely (Figure 82). Dieback and mortality follow repeated defoliations; defoliated trees have been shown to be more sensitive to winter injury. Symptoms on larch include initial minute yellow spots, followed by the fungus producing more easily seen fruiting structures on needle surfaces, e.g., the pycnial and aecial stages (Figure 83).

Cause: Leaf rust of poplar and larch is caused by the fungus *Melampsora medusae*.

Suscepts: This rust disease occurs most commonly on hybrid poplars and eastern larch. It also occurs on quaking aspen, eastern cottonwood, Douglas-fir, and several pines, spruces, and hemlocks.

Range and site description: The range of this disease includes much of the central and northeastern portions of the eastern United States. It can occur wherever the alternate hosts exist in proximity to each other but also may occur outside the range of larch or other alternate hosts due

Figure 81

Figure 82

Figure 81. The uredial and telial spore stages of *Melampsora medusae* on poplar leaves. Note chlorotic spotting on upper leaf surface.

Figure 82. Purple-brown spots on fallen leaves of poplar infected with the rust fungus, *Melampsora medusae*.

to wind dissemination of the repeating spores (uredospores) from poplar to poplar.

Diagnostics: The disease is best identified by looking for the fungal signs present in the form of fruiting structures on leaves of either alternate host. Fallen poplar leaves should be observed for these structures. Purple spotting could be confused with chronic ozone injury.

POWDERY MILDEW

Powdery mildew affects many forest tree species but is not usually considered to be of any significance. It is recognizable by the powdery white mycelium of the causal fungus and by numerous black fruiting bodies on the lower leaf surface (Figure 84). This disease is prominent during dry weather from spring until leaf drop in the fall. Infected leaves may appear chlorotic. Oaks are particularly susceptible.

PHYLLOSTICTA LEAF SPOT

Disease description: This disease is characterized by small, circular to somewhat irregularly shaped brown lesions on leaf surfaces (Figure 85). Lesions may remain separate or may coalesce to cover almost the entire leaf surface. Individual spots may have a very distinct purple border. Black fungal fruiting bodies are embedded in the lesions on upper leaf surfaces. Infections in wet seasons may cause severe defoliation.

Figure 83

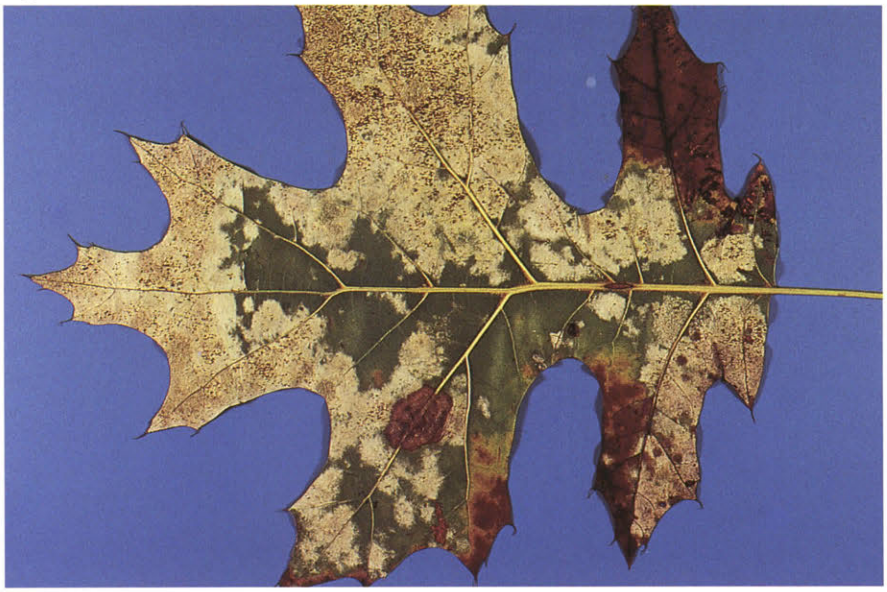

Figure 84

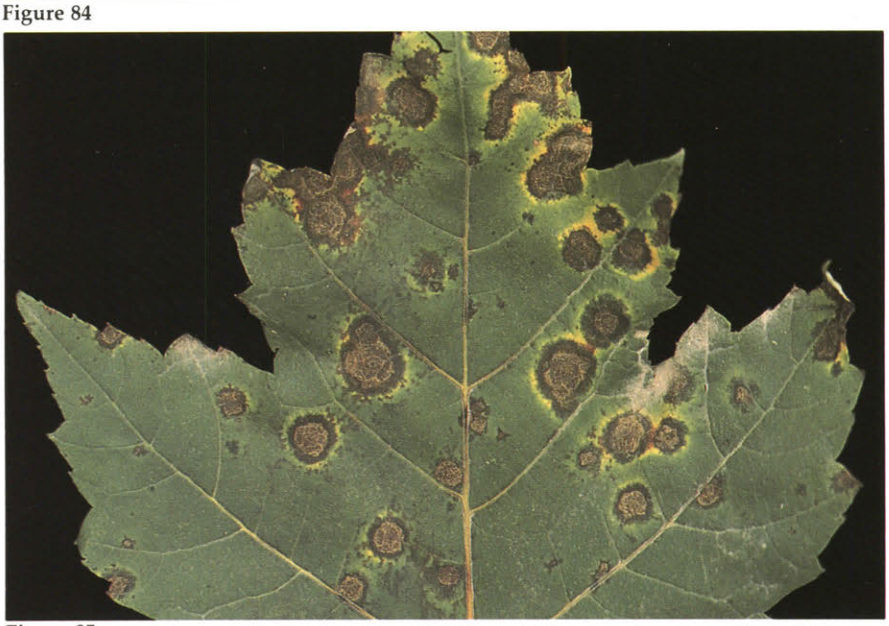

Figure 85

Figure 83. The pycnial and aecial stages of *Melampsora medusae* on needle of larch; chlorotic spotting precedes fruiting structure emergence.

Figure 84. Powdery mildew on lower leaf surface of an infected red oak. Small, black fruiting bodies of the causal fungus are evident.

Figure 85. Phyllosticta leaf spot on red maple, showing the zonate pattern of fungal colonization and yellow halo around each infection spot.

Cause: The fungus *Phyllosticta* spp. causes this leaf spot.

Suscepts: Many hardwood species, especially maples, are susceptible to this fungus.

Range and site description: This disease is found throughout the range of its suscepts and is favored on sites with cool, wet weather early in the growing season.

Diagnostics: The halo of purple tissue around individual lesions and the presence of fruiting bodies embedded in the tissues assist in diagnosis. Zone lines may also be present within the lesion.

TOBACCO RINGSPOT VIRUS OF ASH

Disease description: Following infection early in the growing season, faint chlorotic spots develop on ash leaves; the spots may fade as the season progresses. Spots that develop further are normally less than 1/10 inch (1 mm) in diameter and appear to be diffused without a distinct margin between yellow and green tissue. Chlorotic areas include the veins (Figure 86). Spots later coalesce to form diffuse complete or partial rings. When numerous, rings may merge into a general chlorotic mottle. Infected leaflets sometimes are crinkled and more stunted than noninfected leaves. Late in the growing season, spots may turn reddish brown or may remain yellow-green, while the rest of the leaflet turns yellow or reddish purple in the fall.

The development of foliar symptoms on individual leaflets, twigs, or branches varies greatly. Sometimes only a few scattered leaves on an entire tree are involved.

Cause: An ash isolate of the tobacco ringspot virus is the cause of this disease. The only known natural vector is the dagger nematode, *Xiphinema americanum*.

Suscepts: All ages of white and green ash appear to be susceptible. Tobacco ringspot virus becomes systemic in ash, as in other hosts, but symptom expression varies considerably during the growing season and on a year-to-year basis.

Range and site description: From only limited survey work, virus-infected ash have been confirmed in several sites in New York. From observations of field symptoms elsewhere, it is likely, but unproven, that the disease extends over much of the range of ash in the northeastern states. Symptoms indicative of virus infection have been observed on a variety of different woodland, hedgerow, and homeowner sites, regardless of soil quality or type.

Diagnostics: Distinctive patterns of foliar symptoms, e.g., chlorotic spots or ringspots, are scattered on leaves but rarely over the entire crown. When the chlorotic spots turn brown to reddish brown late in the growing season, the symptoms may resemble the flecking caused by oxidant air pollutants.

TOBACCO MOSAIC VIRUS OF ASH

Disease description: In May and June, infected foliage shows chlorotic spots and ringspots, a pale green coloration, and sometimes interveinal chlorosis. In June and July, symptoms consist of chlorotic spots, rings, and mottle; occasionally, chlorotic, light green, or whitish line patterns may appear along the main veins (Figure 87).

Cause: Tobacco mosaic virus causes this disease.

Suscepts: White and green ash are susceptible.

Range and site description: This disease has been identified in ash in New York and Massachusetts and is widespread in ash in all probability because of the stability and ease of transmission of tobacco mosaic virus. No site-specific relationships have been reported.

Diagnostics: Some of the foliar symptoms of tobacco mosaic virus infection in ash resemble those caused by tobacco ringspot virus and could, therefore, be confused with oxidant-induced injury. Patterns and locations of symptoms should be noted.

Figure 86

Figure 86. Chlorotic spot on ash leaflet; caused by tobacco ringspot virus.

Disease description: Trees infected by the ash yellows organism are characterized by chlorotic to pale green, undersized leaves (Figure 88). Infected trees have thin crowns with twig and branch dieback and premature purplish autumn coloration. Small witches' brooms may appear at the ends of branches, and abundant epicormic branches and brooms appear along the main stem in advanced stages of disease development (Figures 89, 90).

Cause: Ash yellows is caused by a mycoplasma-like organism which infects the phloem.

Suscepts: White ash is the primary host of the causal organism. In addition, green ash and several other ash species are susceptible.

Range and site description: This disease has been observed throughout the New England states and in New York, New Jersey, Wisconsin, and Indiana. The exact range has not been established. The disease does not appear to be site specific and has been found in forest, hedgerow, park, and home landscape trees. Infection centers are evident under forest conditions.

Diagnostics: Small yellow leaves, thin crowns, and witches' brooms are evidence of the presence of this disease. The occurrence of the disease in infection centers within forest stands is also diagnostic. The causal agent is visible only with the aid of light or transmission electron microscopes.

Figure 87

Figure 88

Figure 87. Line or ring pattern on ash leaflet; caused by tobacco mosaic virus.

Figure 88. Ash yellows disease of American ash, showing chlorotic and small leaves, thin crowns, and dieback of the crown.

Disease description: The most predominant symptom of this disease is a pattern of chlorotic lines forming a jagged-edge design, irregular concentric rings, and/or chlorotic flecks (Figure 91). Symptoms occur as the leaf enlarges and are complete once leaves are fully expanded. Later growth shows milder symptoms.

Symptoms appear on large trees as well as on small, one- to two-year-old seedlings. Leaf symptoms can appear anywhere within the crown; the most obvious signs appear in spring. The virus is biologically capable of being transmitted in seeds. Small trees in the understory often show symptoms of the disease.

Cause: Apple mosaic virus causes this disease.

Suscepts: White and yellow birch are both susceptible.

Range and site description: Line pattern symptoms on birch have been seen in the northeastern United States, in eastern Canada, and in Wisconsin. This disease might be expected to occur wherever birch grows. There are no site-specific relationships known.

Diagnostics: Distinctive line symptom patterns are evident on infected leaves; most often, only a small number of leaves show symptoms.

Figure 89

Figure 90

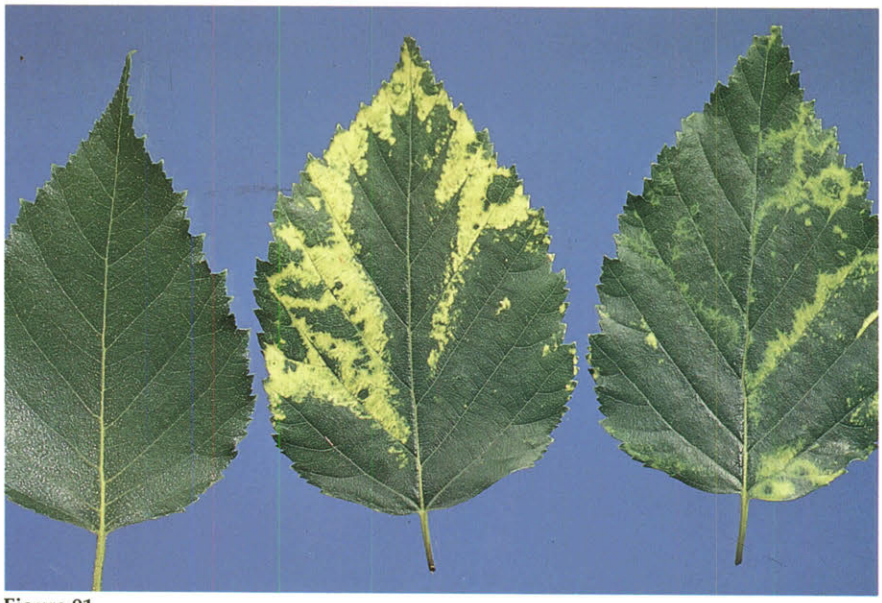

Figure 91

Figure 89. Ash yellows mycoplasma-infected leaves and induced witches' broom on American ash.

Figure 90. American ash twigs showing greatly reduced internode length and deliquescent branching patterns following infection by the ash yellows mycoplasma.

Figure 91. Line pattern on birch; caused by apple mosaic virus.

CONIFER DISEASES

Root Diseases

ANNOSUM ROOT ROT

Disease description. Annosum root rot is a major disease of pines and other conifers, causing growth loss and mortality in plantations and natural stands. The disease is most severe in thinned stands where stump surfaces and root wounds are points of entry for the fungus. Severely affected trees grow more slowly and are more susceptible to attack by bark beetles. Tree mortality tends to be in pockets, but it may be uniformly distributed throughout severely affected stands.

Foliage of infected trees may appear healthy or may be colored from light green to yellow-brown. Branches may have only current-year needles that may be shorter than normal, giving the crown a thin, tufted appearance (Figure 92). These symptoms may be easily confused with pollutant-induced injury. Windthrown or leaning trees may also be present in severely affected stands, especially on sandy soils. Roots of infected trees are initially resin-soaked and brownish red (Figure 93), later becoming white, stringy masses of decayed tissue (Figure 94).

Figure 92

Figure 93

Figure 92. Mortality yellowing and thin crowns of loblolly pine; caused by root disease incited by *Heterobasidion annosum*.

Figure 93. Resin-soaked roots indicative of infection by *Heterobasidion annosum*; roots often break at this point during excavation.

Basidiocarps, or conks, of the causal fungus may appear on the bark surface at the base of infected trees or stumps (Figure 95) or may be hidden within the duff layer. Conks may be small and difficult to locate unless the duff layer at the base of infected trees or stumps is removed. The upper surface of conks is light reddish brown, the margin is white, and the lower surface has numerous pores that are visible without the aid of a hand lens. Released spores form a white cast that may be evident on needles in the duff surrounding a spore-producing conk. Most infected trees do not produce conks.

Cause: Annosum root rot is caused by the fungus *Heterobasidion annosum (= Fomes annosus).*

Suscepts: Annosum root rot has been found on numerous conifers and hardwoods. Of the widely occurring species, loblolly, slash, shortleaf, eastern white, red, and Scots pine are the most severely affected. Other species are only slightly less susceptible.

Range and site description: This disease occurs throughout the northern temperate regions of the world and in some subtropical and tropical areas.

Annosum root rot tends to be more severe on soils that are well drained, sandy to sandy-loam, at least 12 inches (30 cm) deep, and that have a low seasonal water table.

Figure 94

Figure 95

Figure 94. Advanced, stringy decay of root tissue of tree infected with *Heterobasidion annosum*. Portions of roots closer to the main stem would be resin-soaked.

Figure 95. Basidiocarp (conk) of *Heterobasidion annosum* on an infected loblolly pine. Note location in relation to duff layer.

In most plantations and stands, this disease becomes a serious problem following some type of disturbance, especially thinning. Bark beetles may indicate the presence of root rot in pine trees.

Diagnostics: Like many root diseases, the symptoms of this disease are often overlooked during the initial stages of decline, and, when death occurs, other types of damage are usually thought to be responsible. This disease is often confused with the effects of littleleaf disease, other root diseases, or bark beetle infestations.

In many instances, no symptoms are evident until windthrow of living trees occurs.

LITTLELEAF DISEASE

Disease description: In its early stages, this disease is characterized by symptoms of nutrient deficiency; the needles become chlorotic and are slightly shorter than normal. Length of new needles may be only 0.5 inch (1 cm), although normal, healthy needles may be 3 to 5 inches (7 to 13 cm) long. Shoot growth is also reduced.

In later stages of the disease, the symptoms become progressively more distinctive. The crown of an infected tree appears thin and tufted (Figure 96). New needles are discolored and shorter than normal, and the tree loses all but the new needles near the tips of the branches. Branches in the lower crown die first, and death progresses upward through the crown. During this time, radial growth is markedly reduced.

About three years before death, diseased trees commonly produce abundant crops of small cones. These stress-crop cones generally contain sterile seed. Trees killed by littleleaf disease can often be recognized by their persistent crops of undersized cones.

Figure 96

Figure 96. Thin crown of shortleaf pine; caused by invasion of root fungi leading to littleleaf disease.

Close examination of the fine feeder-root system reveals dead root tips, lack of mycorrhizal associations, and cortical root rot.

Stands affected by this disease tend to break up and decline. Affected trees rarely recover from the effects of the causal complex.

Cause: Littleleaf is caused by the fungus *Phytophthora cinnamomi* acting in concert with species of the fungus *Pythium* and nematodes. The primary fungus has a mobile spore that requires moisture for dissemination.

Suscepts: Littleleaf disease affects shortleaf pine, and, to a lesser degree, loblolly pine. Other pines, including Virginia, pitch, slash, and longleaf, are susceptible when site conditions favor attack by the causal fungi.

Range and site description: Littleleaf disease of shortleaf and other pines is found throughout the Southeast. Severely affected stands are in Virginia, North Carolina, South Carolina, Georgia, Alabama, Mississippi, and Tennessee. The disease seldom affects stands younger than twenty years and is most intense in stands older than forty years.

Littleleaf is severe in stands on clay soils but is seldom a problem in sandy or other well-drained soils. Disease occurrence is directly related to soil and site characteristics. Wet, eroded, heavy clay soils that are nitrogen deficient provide the ideal environment for an epidemic of this disease.

Diagnostics: Early symptom expression is difficult to discern from nutrient or water deficiencies. As the disease progresses, however, the foliage becomes sparse and only tufts of needles remain. Needles from trees affected with littleleaf disease have a general chlorosis; the yellowing is seldom concentrated in a specific area. Abundant cone production characterizes later stages of disease development.

POLYPORUS TOMENTOSUS ROOT ROT

Disease description: Symptoms of root rot caused by *Polyporus tomentosus* include basal bark resinosis, excessive mortality of lower branches, crown thinning with a tufting of foliage at branch ends, reduced height and diameter increments, and eventual mortality (Figure 97). In addition, infected trees are more susceptible to windthrow. Infection is most common in trees older than about fifty years. The disease occurs in both natural stands and plantations.

Buff-colored sporophores of the fungus are produced in autumn around the base of a tree and/or on the ground directly above infected roots (Figure 98). Infected root wood is usually stained reddish brown; white pocket rot may be evident.

Cause: *Polyporus tomentosus* is one of the most widespread root-inhabiting fungi in the temperate region of the northern hemisphere.

Suscepts: In North America, this fungus has been reported on all native species of spruce and on most species of fir, larch, pine, hemlock, Douglas-fir, and juniper.

Range and site description: *Polyporus tomentosus* is common in coniferous regions of the eastern United States. It appears to be most prevalent on well-drained, upland glacial tills in the Northeast and on sandy soils of the Southeast, where soils are typically shallow, thereby restricting rooting depth, and very acidic, i.e., have a pH of 4 to 5. These sites usually have low nutrient levels and poor moisture-holding capacities. Sites with a thick, moist duff layer also encourage the disease by favoring root weevils in the *Hylobius* complex; these weevils inflict feeding wounds that serve as infection courts for the fungus.

Diagnostics: Early stages of root rot appear as a gradual reduction of height and diameter increments caused by the progressive decline of roots over a period of years. Infected trees tend to decline slowly, a condition that could be confused with symptoms induced by other biotic or abiotic agents. Positive identification of *P. tomentosus* can be made only by finding sporophores on diseased trees or by culturing the fungus from infected roots on artificial nutrient media.

PROCERA ROOT DISEASE

Disease description: Procera root disease, also known as white pine root decline, has been a problem, primarily in Christmas tree plantations. Recent information indicates, however, that this disease may be widespread in southern pine plantations and may be associated with declining eastern white pine stands in the southern Appalachian Mountains.

Symptoms are extremely subtle until the final stages of decline and occur within roots, root collars, and lower boles. Initial symptoms appear in spring with delayed bud break followed by re-

Figure 97. Yellow needles and thin, dying crowns of sand pine; caused by *Polyporus tomentosus*.

Figure 98. *Polyporus tomentosus* fruiting bodies on the duff layer at the base of a sand pine. These conks retain their yellowish color for several months, then darken with age.

Figure 97

Figure 98

duced candle elongation (Figure 99). After a few weeks or months, the needles of eastern white pine wilt and become pendant. On other pine species, especially those with short needles, the drooping effect is not evident. Needles change uniformly from deep green to light green to yellow-green to reddish brown and remain on the tree (Figure 100).

During the final year, the foliage may appear tufted, all but the most recent needles drop, or the most recent needles are shorter than normal. Quite often there is little or no decrease in height until the year the tree dies.

The bark of limbs and stems becomes spongy to the touch. Resin pockets develop within this tissue, and resin flows freely if the tissue is punctured. Stem cankering has frequently been noted at the groundline, as evidenced by a flat area or by sunken bark on one side of the stem.

Infected roots and lower stems are usually resin-soaked, and occasional black streaks run vertically through the sapwood (Figure 101). Resin may also ooze through the bark and crystallize on the surface, appearing as white or gray deposits (Figure 102). Weevils and bark beetles may inhabit these tissues and be mistaken as the primary cause of the problem.

Cause: This disease is incited by the fungus *Verticicladiella procera*. The wet soils and basal resin flow associated with white pine maladies were described by early investigators as "resinosis disease" and "wet foot."

Figure 99

Figure 100

Figure 99. Delayed budbreak and candle elongation (left) due to procera root disease in eastern white pine.

Figure 100. Reddish brown and wilting needles retained on young eastern white pine infected with *Verticicladiella procera*.

Suscepts: Eastern white, Scots, loblolly, red, Virginia, slash, and Austrian pines are the primary hosts of the causal organism.

Range and site description: This disease is found primarily in the eastern United States. The majority of affected trees have been observed growing on heavy soils that exhibit poor internal drainage. Extremely wet weather in the spring may initiate decline.

Procera root disease has been associated with ozone-sensitive white pine in the Blue Ridge Mountains of Virginia. This disease is also associated with annosum root rot and blue stain diseases.

Diagnostics: Initial symptoms of procera root disease include sudden decline and wilt, uniform fading and discoloration of the foliage, resin oozing through the bark, and weevil or bark beetle activity. Initial foliage discoloration (slight chlorosis) may be confused with ozone injury. The final above-ground symptoms are dry twigs and uniform, rapid browning of the foliage and branches.

Needle Diseases

Symptoms of several foliage diseases of conifers can be easily confused with symptoms of injury caused by air pollutants. These diseases are foliage blights that primarily affect drought-stressed trees. Other needle diseases are incited by more aggressive needle-invading fungi. The initial symptoms of these are easy to confuse, but a careful observer

Figure 101

Figure 102

Figure 101. Black stain in wood of eastern white pine infected with *Verticicladiella procera*. Such stain may be evident in wood of roots or lower stems.

Figure 102. Gray, crystallized resin deposits on the bark of an eastern white pine; symptomatic of *Verticicladiella procera* infection.

can locate and identify fruiting structures of the biotic causal agents and recognize the respective symptom patterns to determine the exact cause.

There are three types of needle diseases: needlecasts, needle rusts, and needle blights. In addition, several canker diseases of young twigs are best evidenced by foliage symptoms, even though the actual causal agent is present on twigs and stems. A brief description that includes representative examples of each type of disease follows.

NEEDLECASTS

These diseases are caused by fungi in the class Ascomycetes. With few exceptions, as noted in the specific disease descriptions, these fungi infect only the needles on the young elongating shoots, but no symptoms appear until the following winter or early spring. At that time, the infected needles develop chlorotic spots that rapidly turn tan to reddish brown. Characteristic fruiting structures form on the needles from midspring to early summer. These structures are usually football-shaped to elongate brown to black hysterothecia that open with a longitudinal slit. They are easily visible to the naked eye, especially when the needles are wet. Affected trees shed, or cast, the previous year's needles, leaving the tree with only a single year's complement of needles.

LOPHODERMIUM NEEDLECAST

Disease description: The previous year's needles redden in late winter or early spring; they brown and fall from the tree in late spring and early summer (Figure 103). In midsummer, black, raised, football-shaped fruiting bodies of the fungus, visible to the naked eye, develop on cast needles. During wet weather, these fruiting bodies open with a distinct longitudinal split that is easily seen with a hand lens (Figure 104). Since most spores are released from needles lying on the ground, branches close to the ground are more severely infected than branches higher in the tree. Severely infected trees retain only one year's complement of needles. Repeated infection leads to mortality of buds and dieback of lower branches. Spores are disseminated by wind from midsummer to early fall and infect current-year needles. The fungus overwinters in these needles, usually causing no visible symptoms until the following spring, when a yellow spot appears at each point of infection.

Cause: The fungus *Lophodermium seditiosum (= Lophodermium pinastri)* causes this disease.

Suscepts.: Although most pines may be infected, Scots and Austrian pines are particularly susceptible.

Range and site description: Lophodermium needlecast is found worldwide wherever two- and three-needled pines grow. The causal fungus requires abundant moisture, so the disease frequently develops first in stands with poor or no weed control, in low-lying areas, or in plantings adjacent to taller trees that reduce wind-drying of the foliage.

Diagnostics: This disease causes severe defoliation; affected trees hold only current-year needles over winter. Needles under trees should be examined for the presence of characteristic fruiting bodies. Infected trees are found most often in areas with poor air drainage.

HYPODERMA NEEDLECAST

Disease description: The previous year's needles may redden in late winter and early spring and be cast in late spring and early summer. More often, however, only scattered red to brownish spots appear on infected needles. These spots may girdle the needle and kill portions beyond the original point of infection, while the bases of the needles remain green and healthy. Fruiting bodies may develop in late spring and early summer within the spots and dead needle tissues. These fruiting bodies are small, black, football-shaped structures barely visible to the naked eye but readily apparent under a 10x hand lens. When wet, fruiting structures open with a longitudinal slit. Although the fruiting bodies look exactly like those of *Lophodermium* (see Figure 104), the two diseases are distinguishable in the field. Needles infected by *Lophodermium* turn brown and fall, whereas many needles infected by *Hypoderma* still have healthy green bases and remain attached to the tree. From a distance, Hypoderma needlecast looks like red band or brown spot needleblight, but it can be distinguished from the latter two diseases by the fruiting body of the causal fungus. Severely affected trees may lose most of the needles on the bottom whorls of their branches. The fungus overwinters in new needles, usually inducing no symptoms until the following spring, when yellow spots appear at each point of infection.

Cause: The fungus *Ploioderma lethale (= Hypoderma lethale)* causes this disease.

Suscepts: Most hard pines, such as pitch, shortleaf, sand, slash, Table-Mountain, red, pond, loblolly, Virginia, and Austrian, are susceptible. In Christmas tree plantations, Austrian pine is most commonly infected.

Range and site description: *Hypoderma* is found throughout the eastern United States, from New England to Florida and Louisiana. Like other needlecasts, this fungus requires abundant moisture, and, therefore, frequently appears first in stands with poor or no weed control or in low-lying areas that are naturally wet or have poor air drainage. It is also more severe in plantations adjacent to taller trees, which restrict air flow, or in older trees that are crowded together, thus restricting drying of the foliage.

Diagnostics: Needles remain attached to infected trees. The fungus produces fruiting structures on infected needles attached to and under the tree. Infected trees are most often found in areas with poor air drainage.

CYCLANEUSMA NEEDLECAST

Disease description:
Cyclaneusma needlecast can be readily diagnosed in the field because the symptoms differ significantly from other pine needlecasts. Symptoms develop primarily in the fall on one-year-old or older needles, which turn yellow with distinct brown bars

Figure 103

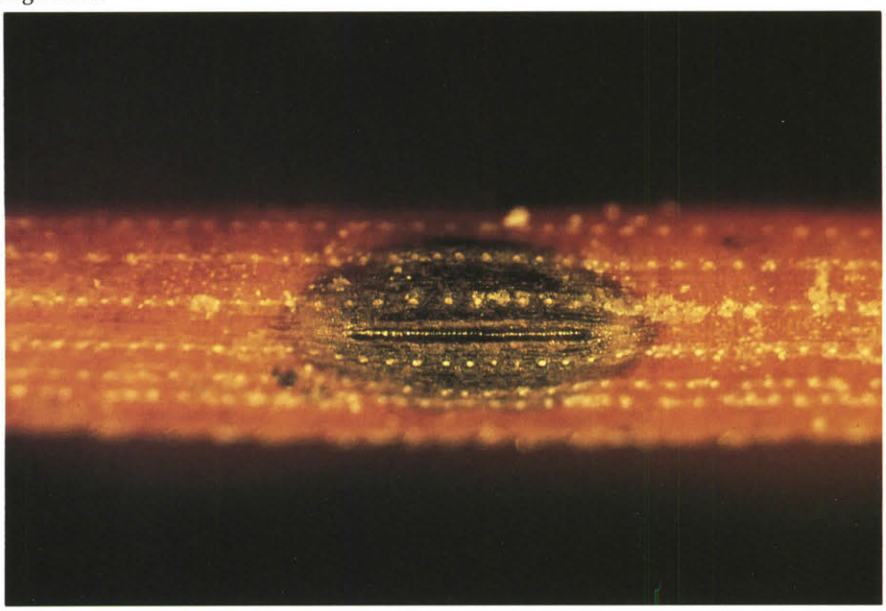

Figure 104

Figure 103. Browning and thinning of the bottom crown of Scots pine due to Lophodermium needlecast disease.

Figure 104. *Lophodermium* fruiting body embedded in Scots pine needle, showing elongate shape and slit down the middle.

across the needle surface (Figure 105). Fruiting bodies of the fungus are white to tan and develop primarily within the brown bars (Figure 106). Symptomatic needles do not drop immediately but remain hanging in or on the tree for several months. Symptoms are not concentrated on the lower branches but develop more or less uniformly throughout the tree.

These symptoms resemble those caused by feeding of the two-spotted pine aphid, *Eulachnus agilis*. Needles damaged by aphid feeding, however, do not develop the distinct brown bars and do not bear fruiting bodies of the fungus.

The fungus overwinters in infected needles, either in or on the tree or on the duff. Severe infection usually occurs from April to June on the previous year's needles. Infection may occur from July through December on first- and second-year needles. Infected needles do not develop symptoms until the end of the second or beginning of the third growing season. Symptoms are most apparent from late September to early December and from April through June. Severely affected trees retain only one year's complement of needles and appear thin and ragged.

Cause: The fungus *Cyclaneusma minus* causes Cyclaneusma needlecast.

Suscepts: Scots and Austrian pines. All trees of commonly grown Scots pine provenances are moderately to highly susceptible; trees of southern European provenances appear more susceptible than those of northern European origin.

Range and site description: Cyclaneusma needlecast is found in the northern United States and southern Canada.

Diagnostics: Cyclaneusma needlecast of hard pines differs from other needle diseases and air pollutant-induced injury in that the fungus affects needles of any age at any time of the year. Symptoms may develop from spring through late fall on second-year or older needles, and the characteristic pale, waxy, tan fruiting structures can be present year round. These structures may be more easily seen if suspect needles are wrapped in a wet towel for one hour and then viewed with a hand lens.

NEEDLECAST OF TRUE FIR

Disease description: Needlecasts of true firs typically have a two-year disease cycle. The tender first-year needles are infected in June and July. No symptoms appear until early in the second growing season, when infected needles turn various shades of red-brown, brown, or tan. During June or July of the second growing season, asexual fruiting bodies, or pycnidia, develop on the upper surfaces of infected needles. These pycnidia may be straw-colored to dark brown or black and are usually arranged in long, straight or sinuous stripes down the middle groove of the needle, or in two bands, one on either side of the central groove. The discolored needles remain attached to the tree throughout the summer and winter of the second season. By June or July of the third growing season, the needles are usually bleached to a light tannish brown or straw color and bear a different type of fruiting body on the undersides. These fruiting bodies typically are reddish brown to black, depending upon species, and are football-shaped to elongate. They may extend the entire length of the needle and are usually arranged along the midvein. The fruiting bodies are very distinct and easily visible to the naked eye. When wet, they open with a longitudinal slit that is easily seen with a hand lens. Infected needles are normally cast during the third growing season.

Cause: Several different species of closely related fungi cause needlecasts of the true firs. Some of these fungi are considered secondary, attacking older needles about to be shed or needles in dense shade; other species are considered aggressive parasites.

Suscepts: The species infected by the various causal fungi include balsam, Fraser, concolor, grand, subalpine, and noble fir.

Range and site description: These diseases may be found throughout the ranges of their suscepts. As is true of other needlecasts, sites that favor high-moisture conditions in the crowns and surrounding understory species are most conducive to outbreaks of the disease.

Diagnostics: Although infected needles turn brown just before the second growing season, sexual fruiting structures do not form and needles are not cast until the beginning of the third growing season. Thus, severely infected firs have one year's complement of healthy green needles and one year's complement of firmly attached symptomatic brown needles (Figure 107). Sites with poor air drainage, i.e., those with high air moisture, are conducive to the disease.

RHABDOCLINE NEEDLECAST OF DOUGLAS-FIR

Disease description: In late winter or early spring, the previous year's needles develop spots that initially are yellow and later reddish brown. There is usually a distinct border between healthy

Figure 105

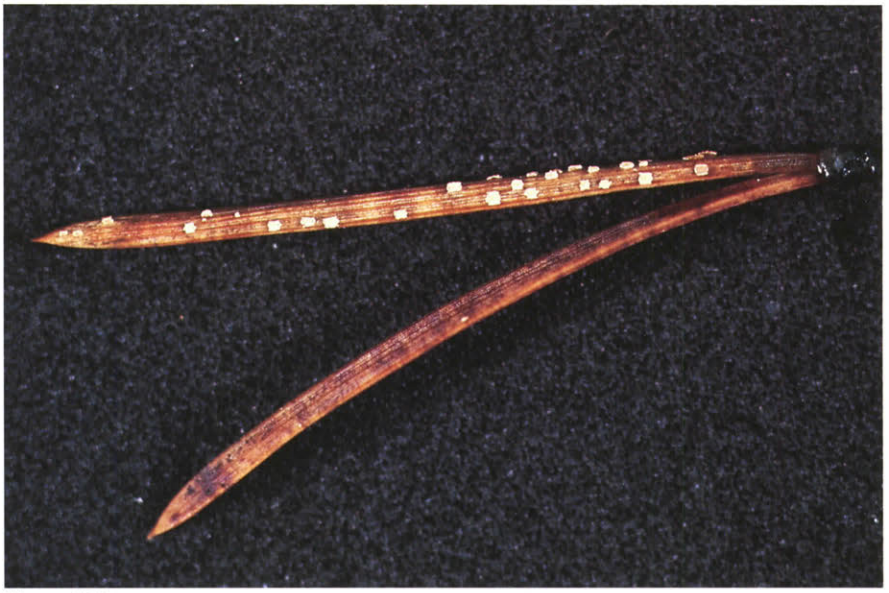

Figure 106

Figure 107

Figure 105. Casting and barring of older needles caused by *Cyclaneusma* on Scots pine. Such defoliation causes thin, see-through crowns.

Figure 106. White fruiting bodies of *Cyclaneusma* on infected needles of Scots pine. Infected needles may remain in trees or be found in the duff under the tree.

Figure 107. Needlecast of true fir with retention of infected needles through the third growing season.

and infected areas on a needle (Figure 108). In late spring, large brown to orange-red fruiting bodies develop in these spots. Fruiting bodies completely fill the discolored areas, and, when wet, rupture the epidermis, becoming readily visible to the naked eye. Infected older needles are cast in early summer, leaving trees with only one year's complement of needles. The disease is most severe on lower branches. Repeated severe infection may weaken and kill young trees.

Cause: The fungi *Rhabdocline pseudotsugae* and *R. weirii* cause Rhabdocline needlecast of Douglas-fir.

Suscepts: Douglas-fir is susceptible. Trees of southwestern provenances are more susceptible than those of the Pacific Northwest.

Range and site description: This disease is found in Douglas-fir stands throughout New England, New York, Pennsylvania, Minnesota, Wisconsin, Michigan, and western North America. The fungi require abundant moisture and low temperatures; the optimum temperature range is about 53°F to 59°F (12°C to 15°C). Thus, the disease develops first in low-lying areas with poor air drainage or on north-facing slopes. Close spacing also creates favorable microclimates for the fungi.

Diagnostics: From the time of bud break through shoot elongation, the fruiting structures are quite evident on fallen needles. Toward late season, only current-year needles remain on infected trees. This disease is present more frequently in low-lying areas with poor air drainage.

SWISS (PHAEOCRYPTOPUS) NEEDLECAST OF DOUGLAS-FIR

Disease description: In late winter and early spring, one- or two-year-old needles may turn yellow or appear mottled. These needles gradually turn brown and usually show no distinct margins between healthy, yellow, or brown areas. Examination of the undersides of these needles with a hand lens in late spring or early summer reveals two rows of minute, round, black fruiting bodies, one row on each side of the midrib of the needle (Figure 109). These rows may appear as two rows of "dirt" to the naked eye. Severely infected trees normally maintain only current-year needles on the lower branches. The disease progressively intensifies, and may kill, branches. Repeated severe infection may weaken and kill trees.

Cause: The fungus *Phaeocryptopus gaumannii (= Adelopus gaumanni)* causes Swiss needlecast of Douglas-fir.

Suscepts: Douglas-fir is the susceptible species.

Range and site description: This disease is found throughout New England, New York, Pennsylvania, Minnesota, Wisconsin, Michigan, and western North America. The fungus requires abundant moisture; it usually develops first in stands in low-lying areas that either are naturally moist or have poor air drainage.

Diagnostics: Trees should be examined for characteristic fruiting structures on the undersides of two- and three-year-old needles. Frequently, only current-year needles remain on the lower branches.

RHIZOSPHAERA NEEDLECAST

Disease description: Between April and early June, infected previous year's needles turn lavender or purple. Minute, stalked, black, cup-shaped fruiting bodies appear on the needles, each arising from a stomate. In late May through June the fruiting bodies can be seen with a hand lens as small rows of "dirt" running lengthwise along the needle (see Figure 109). Infected previous-year needles then drop, leaving the tree with only current-year needles at the branch tips.

In southeastern Pennsylvania, infection may enter a second cycle on Colorado blue spruce in late summer and early fall. In late August and September, current-year needles infected earlier that summer turn purple and bear fruiting bodies. The fruiting bodies release spores, further infecting the current-year needles. Needles infected in early summer then drop, frequently leaving bare-twigged holes in the branches of trees (Figure 110).

Cause: The fungus *Rhizosphaera kalkhoffii* causes Rhizosphaera needlecast.

Suscepts: In the United States, the fungus attacks spruces, especially Colorado blue spruce.

Range and site description: This disease is found throughout the range of its suscepts. The fungus requires abundant moisture and rather cool temperatures. Thus, it frequently develops first in stands within low-lying areas which are naturally moist or which have poor air drainage, or in stands adjacent to taller trees that reduce wind-drying of the foliage.

Diagnostics: Infected foliage appears to have a lavender or purple coloration. In addition, fruiting structures on lower needle surfaces and disease occurrence in low-lying sites with poor air drainage are also diagnostic. Damage by spruce needleminers may easily be confused with the symptoms of this disease, but the characteristic fruiting structures of *Rhizosphaera* will not be present.

NEEDLE RUSTS

Disease description: Most needle rust fungi cannot spread from conifer to conifer but involve a second, or alternate, host. Needle rusts are characterized by easily seen yellow, orange, red-brown, or white pustules (aecia) filled with powdery spores of the same

Figure 108

Figure 109

Figure 110

Figure 108. Rhabdocline needlecast symptoms on Douglas-fir needles, with orange-red fruiting bodies of the causal fungus evident.

Figure 109. Swiss needlecast on Douglas-fir needles, with rows of *Phaeocryptopus* fruiting bodies appearing as dirt particles to the naked eye.

Figure 110. Hole in a branch of Colorado blue spruce; caused by needlecast incited by *Rhizosphaera kalkhoffii*.

color (Figure 111). One known exception occurs on spruce with *Chrysomyxa weirii*, where the rust fungus appears to be autoecious, and telia are produced within the pustules (Figure 112).

Infected needles usually die and are cast by late summer. A few of the rust fungi may persist as perennial mycelia in infected conifer twigs, invading each successive crop of new needles. In these cases, the conifer host may develop conspicuous witches' brooms.

Cause: Numerous species of the rust fungi cause needle rust.

Suscepts: All species of pines, spruces, true firs, and Douglas-fir are susceptible to one or more species of needle rust fungi.

Over twenty species of needle rust fungi are known to attack true firs and alternate to various woody hosts such as willow and blueberry, herbaceous hosts such as mouse-ear chickweed, or ferns. Fir needle rusts may occur on current-year or older needles.

About a dozen known species of needle rust fungi that attack spruces alternate primarily to woody shrubs, such as Labrador tea, bearberry, or brambles. These spruce needle rusts infect only current-year needles (Figure 112).

About eighteen known species of needle rust fungi that attack pine alternate to woody shrubs such as gooseberry or to herbaceous plants, particularly species of composites such as goldenrod and wild aster. Fruiting structures may be found on current-year or older needles but are most frequently found on second- and third-year needles. The most prominent conifer species infected include loblolly, Virginia, shortleaf, red, and Scots pine.

Two known species of needle rust fungi that attack Douglas-fir alternate to species of *Populus*, especially aspen. They occur only on current-year needles.

Range and site description: In most cases, the rust fungi are distributed wherever the suscepts grow naturally. Moist, cool weather favors disease development in the spring. Several young loblolly pine plantations have been reported as severely affected. Needle rust fungi seldom attack large trees; when they do, they affect only the needles on lower limbs.

Diagnostics: The best feature for diagnosing needle rust on conifers is the presence of the causal fungus, signaled by a pustule on the needle surface. Infected needles are usually cast from the tree, but mildly infected needles may remain attached. When needles are handled, the spores of the fungus may be released from their delicate membrane coverings, producing a cloud of dust and turning one's fingers orange.

NEEDLE BLIGHTS

Needle blights are a diverse group of diseases. Some of their causal organisms can incite explosive epidemics when environmental conditions are suitable. Since needles of any age may be attacked, signs and symptoms may occur on first-year needles as well as on second-year or older needles.

BROWN SPOT NEEDLE BLIGHT

Disease description: Symptoms begin to show on current-year needles in July and continue to develop throughout the summer and fall. Symptoms initially appear on the lower branches but may develop on much of the tree after two or three years of infection. At first, infected needles appear mottled with yellow or pale green spots or bands that may have yellowish margins and appear to be resin-soaked (Figure 113). The centers of these spots then turn brown and the lesions enlarge to girdle the needle. An infected needle may initially have a spotted midsection, a dead tip, and a healthy green base. The entire needle eventually dies and turns brown before being cast. Severely affected needles may be cast in the fall; however, many needles remain hanging in the tree and are cast the following spring and summer. Repeated infections result in thin, ragged-looking trees, stunting, and even death.

In the spring, dark brown to black fungus fruiting bodies erupt through the epidermis in the dead portions of infected needles. These fruiting bodies are barely visible to the naked eye but can be readily seen with a hand lens. They vary in size and shape, ranging from round "pimples" to elliptical ridges several times longer than wide. Fruiting bodies of the causal fungus lack the longitudinal slit characteristic of needlecast fruiting bodies.

Cause: The fungus *Scirrhia acicola* causes brown spot needle blight.

Suscepts: All pines, including Scots, red, eastern white, ponderosa, jack, Virginia, shortleaf, and longleaf, are susceptible.

Range and site description: This disease occurs throughout much of the range of its suscepts. It is particularly important on longleaf pine seedlings in the Southeast. The disease has been of no importance in New England. Brown spot needle blight is found on all types of sites but favors areas with poor air drainage because of the associated long periods of moisture.

Diagnostics: The symptoms of this disease are similar to those incited by *Dothistroma pini* (red band needle blight). Initially, the observed chlorotic mottle and spotting may appear similar to ozone- or SO_2-induced injury. A small droplet of resin is commonly associated with each spot. The base of an infected needle remains green for some time after the rest of the needle dies.

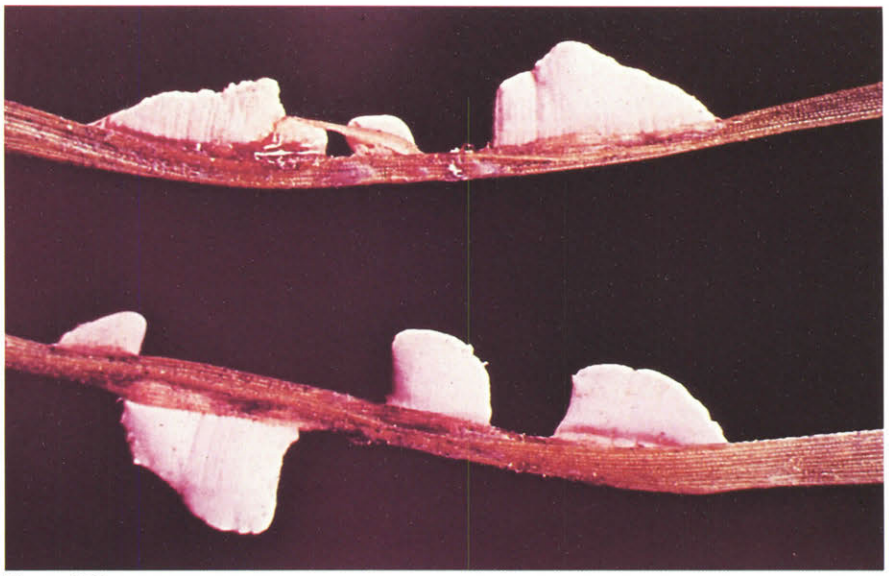

Figure 111

Figure 112

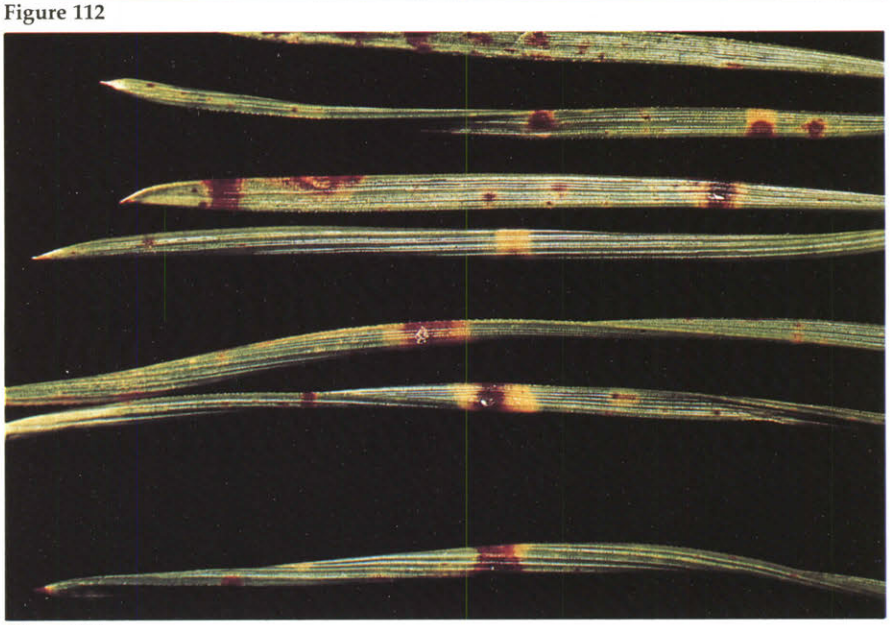

Figure 113

Figure 111. Aecial fruiting bodies of a needle rust fungus erupting through the surface of infected pine needles.

Figure 112. Needle rust on spruce as evidenced by orange-colored telial pustules erupting from yellow infected spots.

Figure 113. Brown spot needle blight symptoms exhibiting the yellow band, brown spotting, and resin droplet usually present with this disease.

RED BAND NEEDLE BLIGHT

Disease description: Symptoms usually do not develop on first-year needles until fall. At first, infected needles appear mottled with dark green bands and yellow to tan spots (Figure 114). The mottled areas soon turn brown to red-brown. Infected needles may have dead tips and healthy green bases; fruiting bodies develop in these dead spots in the fall. Infected needles are cast in the fall season or may remain hanging in the tree until the following spring and summer.

Trees of any age, including seedlings, may be attacked. The entire tree may be affected, but infection is usually more severe on the lower branches.

In the spring, dark brown to black fungus fruiting bodies erupt through the epidermis within dead portions of the needles. The fruiting bodies are barely visible to the naked eye but are readily seen with a hand lens. They vary in size and shape, ranging from small round "pimples" to elliptical ridges several times longer than wide. Fruiting bodies of the causal fungus lack the longitudinal slit characteristic of the fruiting bodies of the fungi causing needlecast.

Cause: The fungus *Dothistroma pini* causes red band needle blight.

Suscepts: While most pines are susceptible, Austrian and ponderosa are very sensitive. Scots and red pines are resistant. Douglas-fir and European larch that are close to infected pines may become infected.

Range and site description: Red band needle blight occurs worldwide in the temperate and tropical highland regions.

Diagnostics: Late-season symptoms of red band needle blight are yellow to tan spots on needles. Spots may girdle the needle, killing terminal portions. The base of an infected needle usually remains green for some time after the rest of the needle dies. Examination of symptomatic needles on the tree or in the duff layer under the tree reveals the fungus fruiting bodies described above.

Twig and Stem Diseases

Little is known about the many twig and stem canker diseases. Most are rather common and are characterized by a slight constriction in the bark that may or may not be accompanied by resin bleeding.

DIPLODIA TWIG BLIGHT

Disease description: The diplodia twig blight fungus rapidly kills infected young, succulent shoots, usually before needles fully elongate. Needles on such shoots are often stunted. The fungus can also attack older shoots through wounds, including those caused by insect injury. Such infection seldom leads to girdling and death but usually results in the formation of perennial bleeding twig and stem cankers and twig death (Figure 115). Sometimes only a portion of the young shoot is killed; in such instances, the junction between diseased and healthy stem tissues is distinctly constricted. The fungus persists for several years in infected dead needles and cone scales. Fruiting bodies may not appear on these dead tissues until fall or the following spring. Readily seen under a hand lens, fruiting bodies are embedded in host tissues; small, black, conical beaks protrude from the surface of these tissues (Figure 116).

Cause: The fungus *Diplodia pinea (= Sphaeropsis ellisii)* causes diplodia twig blight.

Suscepts: Douglas-fir and most hard pines, especially Scots, Austrian, red, and ponderosa, are susceptible. Diplodia twig blight can be very destructive to young Douglas-fir during springs with abnormally high rainfall. On such diseased Douglas-fir, the elongating shoots suddenly wilt, droop, and turn brown.

Range and site description: This disease is found in eastern North America, from South Carolina and Oklahoma northward. The disease is uncommon on pine trees under fifteen years old, except in abandoned plantations, but may become a problem in younger trees after severe spittlebug infestations.

Diagnostics: Death and flagging of young shoots and stems, a slight crook, and the presence of black fruiting bodies on the canker surface aid in diagnosis. A disease that appears similar to diplodia twig blight is atropellis canker, but in the latter disease the fruiting structure of the causal organism is somewhat larger.

Figure 114. Spots and needle necrosis typical of red band needle blight on Austrian pine.

Figure 115. Needle death and branch mortality caused by infection of young twigs by *Diplodia pinea.*

Figure 116. Fruiting bodies (pycnidia) of *Diplodia pinea* erupting through infected pine needles.

Figure 114

Figure 115

Figure 116

Disease description: Branch dieback is the most common symptom of this disease, with branches in the lower crown being among those most affected (Figure 117). Needles on cankered branches turn red and may remain attached for an entire growing season. The base of a dying branch usually has an inch or so of twig bearing yellowish needles. A faint constriction in the bark may appear within this zone or between it and the portion bearing healthy needles. If the constriction is not visible to the eye, it sometimes can be felt by rubbing the fingertips along the bark in this area. Such cankers are not associated with any visible wounding. Resin flow at the point of cankering along the main stem is usually in evidence or is deposited on branches and needles below cankered branches (Figure 118).

The causal fungus can cause either a girdling basal-stem canker or a branch and twig dieback. The fungus is considered to be a weak parasite, attacking only stressed trees. It produces slime spores that ooze out in yellowish, sticky drops, or, during wet weather, in tendrils (Figure 119).

Cause: The fungus *Cytospora* spp. causes Cytospora canker and twig dieback.

Suscepts: Most conifers are susceptible. The girdling basal-stem canker has been observed on Norway, red and blue spruce, Douglas-fir, and on white, Fraser, and balsam firs.

Figure 117

Figure 118

Figure 117. Branch and lower- to mid-crown mortality of red spruce; caused by *Cytospora*.

Figure 118. Pitch oozing from a *Cytospora*-incited canker on the basal portions of spruce limbs.

Range and site description: This disease is found throughout the range of its hosts but is most severe where trees are suffering from other stresses, such as drought or nutrient deficiencies.

Diagnostics: The disease usually strikes limbs at the base of the tree first and then progresses up the stem. Pitch ooze at the base of infected limbs is quite common.

SCLERODERRIS CANKER OF NORTHERN CONIFERS

Disease description: The most characteristic symptom of this disease is an orange-brown coloration of the bases of needles along an infected shoot (Figure 120). Spores of the causal fungus infect through buds or needles during midsummer, and the fungus rapidly colonizes the twig and stem tissues. The following spring, needle bases will discolor and cankers may develop on twigs and the main stem. A green stain is usually present on freshly cut tissues underneath the canker surface (Figure 121). Numerous cankers on a young tree will cause mortality; when conditions are proper, a major epidemic may occur, resulting in the death of large trees over extensive areas

Figure 119

Figure 120

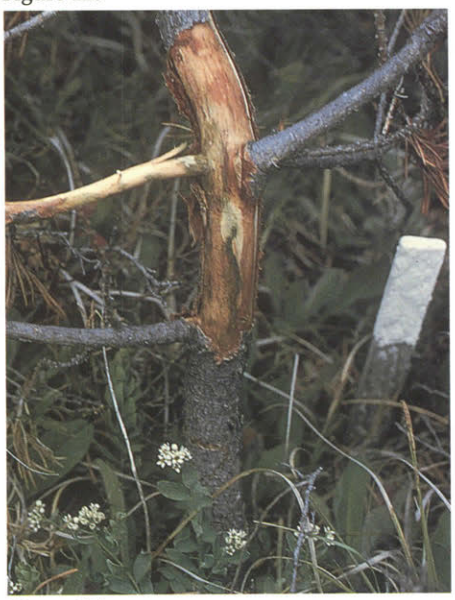

Figure 121

Figure 119. Spore tendrils of *Cytospora* emerging through the bark of an infected spruce branch following a wet period.

Figure 120. Typical dying of the base of needles in red pine following infection of twigs by *Scleroderris abietina*.

Figure 121. Green streaking (stain) in the wood of young stem infected by *Scleroderris*.

Figure 122

(Figure 122). Fruiting structures of the fungus, i.e., black pycnidia and red-brown apothecia, are produced on the needles, twigs, and canker surfaces (Figure 123).

Cause: Scleroderris canker is caused by the introduced fungus *Gremmeniella abietina (= Scleroderris lagerbergii)*. Two strains have been observed in North America: the Lake states and the European strains. Damage from the European strain is much greater in that it kills large trees by causing extensive branch mortality.

Suscepts: Scleroderris canker has been reported on red, jack, Scots, pitch, ponderosa, lodgepole, Virginia, and eastern white pines; on black, white, and Norway spruce; and on Douglas-fir and larch. Major epidemics have been observed in red, jack, and Scots pine plantations.

Figure 122. Stand of red pine severely infected by *Scleroderris*. Note ragged crowns, dieback, and dead trees.

Figure 123. The reddish brown apothecia fruiting structures of *Scleroderris abietina* on the bark of an infected red pine.

Figure 123

Range and site description: This disease occurs throughout the Lake states and into Vermont and New York. The fungus is known as a cool-temperature organism and is favored by conditions in low-lying or frost pocket sites. The potential for spread from its current range is great.

Diagnostics: The most useful diagnostic symptom involves the initial coloration and death of needle bases. Infected needles fall from a slight touch, and bud and branch tips will be brown. Small cankers on twigs and a green coloration of tissues under the bark are also diagnostic. The presence of this type of disease in low-lying areas may also assist in correct identification.

EASTERN DWARF MISTLETOE

Disease description: The most prominent symptom of eastern dwarf mistletoe infection in spruce is the witches' broom, a proliferation of host tissues that produces a slight swelling of the branch, followed by the formation of a compact, bushy mass of branches and twigs that tend to have a positive phototropic growth habit. Witches' brooms can vary in size from a few small twigs to compact brooms measuring up to about 120 inches (3 m) in diameter (Figure 124). Swellings on main stems may be a result of old, but still active, bole infections (Figure 125).

Figure 124

Figure 125

Figure 124. Witches' broom on red spruce, indicative of infection by the eastern dwarf mistletoe plant, *Arceuthobium pusillum*.

Figure 125. Swelling of main stem of red spruce due to an old, but continuing, bole infection.

The rootlike system of the dwarf mistletoe plant grows within host branches and eventually gives rise to shiny, small, separate male and female aerial shoots (0.5 to 1.5 inches or 1 to 4 cm long), with greenish brown, scalelike leaves (Figures 126, 127). These aerial shoots die and fall off soon after pollen production in spring and seed dispersal in fall, leaving basal cups on host branches. The basal cups can be easily seen with a hand lens, and, along with the aerial shoots, serve as a sign of dwarf mistletoe presence.

Within stands, irregular, circular patches of infected trees can be observed. Infected trees have witches' brooms, swollen branches, spike (dead) tops, reduced growth, and poor wood quality. In addition, trees are weakened and predisposed to infestation by other pests, such as fungi and insects. Mistletoe-infested stands usually become poorly stocked. They also tend to be more open, so that infected living trees, especially those with large witches' brooms, are more susceptible to windthrow.

Cause: The parasitic plant *Arceuthobium pusillum* is the eastern dwarf mistletoe organism.

Suscepts: White, red, and black spruce are considered to be the principal hosts.

Range and site description: Eastern dwarf mistletoe is found on black, red, or white spruce growing in pure and mixed stands in eastern North America.

Black spruce usually grows in pure, even-aged stands in cool sphagnum bogs with deep, wet, organic soils. White spruce grows in extensive pure stands in its northern range. It prefers moist loam or alluvial soils and is typically found along stream banks, lake shores, and adjacent areas. White spruce along the coastal areas of Maine is heavily infected with eastern dwarf mistletoe. Red spruce has recently been found infected in mixed stands in several New England states.

Diagnostics: The presence of witches' brooms is the best diagnostic tool; however, not all brooming in spruce results from infection by dwarf mistletoe. Some brooms are thought to be caused by genetic mutation, others by the spruce broom rust fungus, and still others may be caused by repeated snow, wind, or ice breakage of leaders. No other diseases mimic the symptoms associated with eastern dwarf mistletoe infection. However, since the causal organism is so small and difficult to detect until brooms are established, early symptoms may be confused with other decline situations. A tufting of small branches and foliage has been reported on spruce showing progressive symptoms of dieback and decline. The cause of these symptoms is unknown.

Figure 126

Figure 126. *Arceuthobium pusillum* plants, showing growth habit on branches of red spruce.

Disease description: The blister rust fungus infects pine needles in late summer or early fall. The fungus grows through the needle and twig into larger branches or the main stem of the tree, where it grows throughout the host cambium and bark. During the second growing season, a slightly sunken, reddish brown area may appear. At the end of the third growing season, tissues above the cankered area may swell. Two to three years after initial infection, the blister rust fungus produces spores that can be identified on the bark surface by ooze which remains shiny after drying. The fungus continues to colonize adjacent tissues each year, and the cankered area enlarges. One year after the first spore stage, a second spore stage produces bright yellow-orange pustules directly on the canker surface (Figure 128). These spores are windblown and often appear as a dust cloud emitted from a tree that has been blown or shaken by the wind. Second-stage spores infect currant or gooseberry, the alternate hosts.

Cankers on infected pines eventually girdle branches or main

stems, resulting in dead branches or dead treetops. While both small and large trees may die from infection, the ability of the fungus to kill a tree depends upon the diameter of infected stems and the growth rate of the fungus. In many instances, hundreds of cankers have been found on a single tree. Tops of trees with trunk cankers usually turn pale green or yellow several months before dying (Figure 129). Old cankers are characterized by abundant pitch flow and the remnants of the earlier spore stages.

Cause: The fungus *Cronartium ribicola* causes blister rust of white pine.

Suscepts: All five-needle (white) pines are susceptible to some extent to the attack of the causal fungus. No commercially important white pine species are resistant to infection. In the past, the disease has been particularly damaging to eastern white pine. The alternate host is *Ribes* spp. (wild currant) or *Grossularia* spp. (gooseberry).

Range and site description: Blister rust of white pine affects stands in the East from Maine to North Carolina and westward to Minnesota in a band across the northern states.

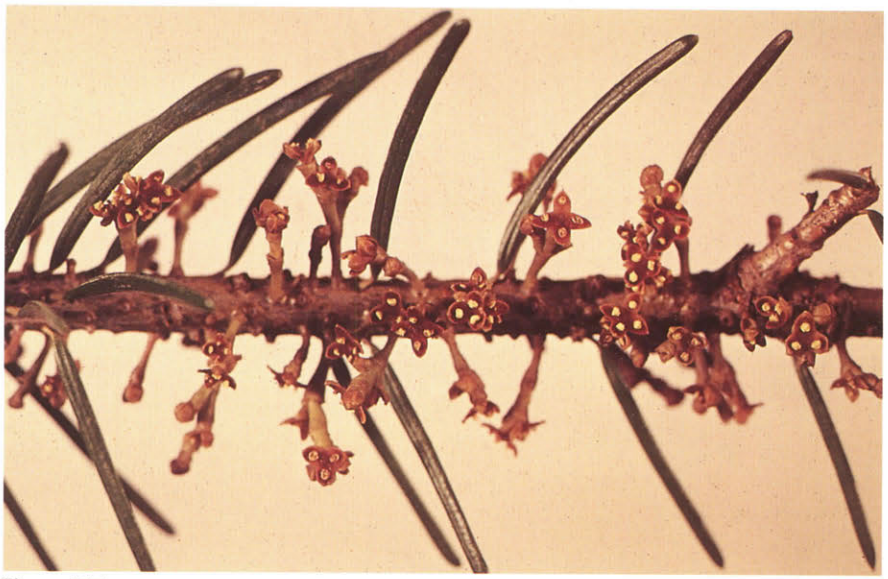

Figure 127

Figure 128

Figure 129

Figure 127. Close-up of mistletoe plant, showing its size relative to the needles of its host plant, red spruce.

Figure 128. Yellow-orange pustules (aecia) erupting through the bark of eastern white pine infected by *Cronatium ribicola*, the blister rust fungus.

Figure 129. Eastern white pine tree declining from blister rust infection, showing off-color of crown that will be followed by crown death above the cankered area.

Diagnostics: Stem and branch cankers are evident below foliage areas that have died or become chlorotic. Fruiting bodies of the causal fungus may be present on the cankered surface in late spring or early summer. Trees with dead tops should be closely examined for main stem cankers.

COMANDRA BLISTER RUST

Disease description: Comandra blister rust is very similar in symptomatology and disease cycle to white pine blister rust. Following infection of young shoots, the causal fungus becomes established in the inner bark and young wood tissues. Fruiting of the pycnial stage may occur one to three years later, with typical shiny ooze in evidence on swollen, spindle-shaped areas of stems and twigs. Later in the disease cycle, the bright orange, rust-colored aecial spore stage is produced on infected bark surfaces (Figure 130). Developing cankers are rough-surfaced and usually bleed resin profusely. Yellowing and necrosis of foliage occurs distal to the cankers, with associated twig, branch, and main stem death following girdling of tissues. Spiketops will occur on larger trees.

Cause: The fungus *Cronartium comandrae* causes comandra blister rust.

Suscepts: The major eastern forest trees susceptible to this fungus include loblolly, jack, pitch, Scots, shortleaf, Table-Mountain, and Virginia pines. The disease is economically important on loblolly pine. Young pine plantations less than ten years old are most severely affected. The alternate host is the false-toad-flax.

Range and site description: This disease is primarily found in the central portions of the eastern United States, i.e., in Tennessee, Kentucky, and North and South Carolina. However, the disease may occur in most eastern states wherever the alternate host is found.

Diagnostics: Small swellings on twigs and the presence of fruiting stages of the rust fungus are most helpful in diagnosis. Cankers and resin flow may also indicate the presence of the disease.

FUSIFORM RUST

Disease description: Fusiform rust is characterized by spindle-shaped galls on branches or the main stem of pine. Galls are elongate, often with deep fissures, and seldom show a pronounced ridge between healthy and infected tissues. In early spring, pustules containing bright orange spores form on the galls, making them very conspicuous (Figure 131). Many galls may be present on one tree. Galls on the main stem of young trees may cause breakage or may girdle the stem.

Oak serves as the alternate host for this rust fungus, which produces fine, brown, hairlike projections on the lower surface of infected leaves. Severely infected leaves may exhibit chlorotic spots and some distortion, but oak leaves usually remain symptomless.

Suscepts: Loblolly and slash pine are highly susceptible to infection. Longleaf pine is somewhat resistant, and shortleaf and Virginia pine are considered immune. Alternate hosts include water, willow, and laurel oaks as being most susceptible, with other red and black oaks being less favorable hosts. White oaks are rarely infected.

Range and site description: Fusiform rust has been found from Maryland to Florida and Texas. Sites that tend to have high relative humidity and warm temperatures for extended time periods favor development of fusiform rust epidemics.

Cause: The fungus *Cronartium quercuum* f. sp. *fusiforme* causes fusiform rust.

Diagnostics: Spindle-shaped galls on stems and/or branches are characteristic symptoms. Main stem breakage often occurs at the point of gall development. Fusiform rust has been known to decimate entire stands.

PINE-OAK RUST

Disease description: Pine-oak (also known as eastern gall) rust is characterized by brain-shaped galls on the main stem of the pine hosts (Figure 132). Hundreds of galls have been observed on a single tree. Usually galls are quite distinct from the main branch, and a collar of bark separates diseased from healthy tissue. Severe infection may lead to tree deformity, reduced growth, breakage at the point of gall formation, and death. On larger stems, galls may serve as the entry point for decay fungi. During spring, the bright orange spores of the fungus make the galls very conspicuous.

Oak serves as the alternate host. Oak leaves seldom exhibit symptoms of infection unless severely infected, and then only a mild chlorosis or chlorotic spotting may occur. Brown, hairlike projections of the fungus on the lower leaf surface are normally the only sign of infection.

Cause: The fungus *Cronartium quercuum* f. sp. *banksianae* causes pine-oak rust.

Figure 130. The aecial stage of *Cronartium comandrae* on a limb of loblolly pine.

Figure 131. Spindle-shaped fusiform rust gall on loblolly pine stem, showing bright yellow aecial spore stage of the causal fungus.

Figure 132. Brain-shaped pine-oak rust gall on Virginia pine branch with the aecial stage of the causal fungus in evidence.

Figure 130

Figure 131

Figure 132

Suscepts: Many species of pine and oak are susceptible to infection by the various stages of this rust fungus. Pine hosts include Virginia and Scots pine. Red, black, scarlet, and pin oaks are considered the most susceptible. Infection of white oak, American chestnut, and chinkapin has been noted in the southeastern United States.

Range and site description: This disease is found throughout the eastern United States and has been considered a serious problem in nurseries in the Great Lakes states. Many jack pine plantations have been severely attacked and rendered useless for timber production.

Diagnostics: Round galls on stems and branches are characteristic symptoms. Severely infected trees may be deformed and may suffer breakage at the gall area.

ABIOTIC STRESSES OF HARDWOODS AND CONIFERS

The foliage of an entire tree often turns brown or off-color with no obvious signs of causal agents. Closer examination of roots, stems, or foliage for other symptoms may yield little additional information. Problems of this nature may be due to local weather conditions, including drought, winter drying, or ice. In addition, nutrient imbalances can induce locally mild to severe symptoms.

Temperature and Moisture Injury

WINTER INJURY (DRYING)

Disease description: Winter injury is most prominent on conifers, and, because of its many possible symptoms, is usually hard to recognize. Symptoms of winter drying do not appear until the following spring and may include marginal scorch, tipburn of needles, mottling and death of leaves, and various amounts of

leaf fall (Figures 133, 134). Dead leaves are a uniform tan, and the afflicted leaves and twigs that remain on the tree are very dry and brittle. Tip dieback may affect some hardwood species and is most likely due to early spring frosts that kill terminal buds and twigs; when new growth begins, affected plants appear to be tufted.

Cause: Damage is most likely to occur on warm winter days that have been preceded by a prolonged period of very low temperatures. Under such conditions, soil water is frozen and is no longer available to the plant, and/or the water present in the plant stem is frozen and blocks the upward movement of water to the leaves. During periods of warm weather when a slight wind is accompanied by low relative humidity, the tissues of leaves, stems, and buds begin to lose water to the air. This water cannot be replaced due to the frozen soil and stem water. The extent of leaf damage, i.e., marginal scorch or death of the entire leaf, and survival of the entire plant depend upon the length of exposure to such conditions. Winter drying is not the same as blackening of foliage due to late spring frosts.

Suscepts: Any species of plant is sensitive to winter drying when exposed for a sufficient length of time. Plants that retain their leaves over winter, however, are more likely to be injured than are deciduous species.

Conifers of all types are particularly susceptible to winter drying. Portions of the plants that lie be-

Figure 133

Figure 134

Figure 133. Winter injury (drying) on red spruce trees, showing injury to previous years' foliage and defoliation.

Figure 134. Close-up of winter injury on red spruce branch with orange coloration and shoot death evident.

neath the snow cover are usually not affected. Large numbers of trees, especially loblolly pine, have been damaged during recent winters characterized by little snow cover and subfreezing temperatures.

Range and site description: Winter drying is most common where species are at the northern edge of their range, but it may also damage species of plants native to an area well within their range. Trees exposed to high winds during severe cold, such as those found on the edges of stands adjacent to large expanses of open terrain, are sensitive to winter injury.

Diagnostics: Winter drying is most severe on conifers. A chlorotic mottle or uniform yellowing of tissue may exist in early spring; a uniform browning or tan coloration follows (Figure 135). Injury is often found on the southwest facing side of exposed trees, i.e., the side facing the prevailing winds. Many root diseases caused by biotic pathogens produce symptoms similar to those that result from winter drying.

FROST

Late spring or early fall frost injures sensitive plant tissues. Late spring frosts are the most damaging, since the young foliage is succulent at that time of year. Diagnosis is easy, as the frozen tissues turn blackish brown (Figures 136, 137). Mild freezes or

Figure 135

Figure 136

Figure 137

Figure 135. Close-up of winter injury on red spruce needles.

Figure 136. Frost injury on current-season needles of Scots pine, following a sudden temperature drop in the late summer.

Figure 137. Frost injury on new growth of a dogwood, resulting in blackened leaf tissues.

even cool temperatures may cause slight tissue collapse and/or chlorosis that may resemble air pollution-induced symptoms, such as severe SO_2 injury. Weather records and personal observations can assist in identifying injury caused by late spring frosts. Initiated by early fall frosts, premature autumn coloration and leaf drop are common in several species of plants. Green foliage may drop from the tree, and remaining foliage may have light brown margins.

HIGH TEMPERATURE

Periods of extremely high temperature combined with windy conditions can injure many tree species, even when adequate moisture is available in the soil. Injury symptoms appear at the periphery of crowns and include a marginal scorch of leaf tissues, with a fairly sharp line between affected and healthy areas. Symptoms may resemble those induced by fluorides.

During spring, periods of cool, wet weather that are followed by sudden extremely high temperatures may increase the extent of heat injury. High temperatures may also injure plants growing in shaded areas that are suddenly exposed to direct sunlight.

DROUGHT

The symptoms of prolonged drought are easily recognizable; awareness of recent drought at a particular site will assist in diagnosis. Typical wilt symptoms, including drooping or cupped leaves, pendant needles, and browned terminal portions of foliage, may be evident. On dry sites during particularly long, dry periods, wilting symptoms may appear in late afternoon. Crowns may look thinned due to the cupping and rolling of leaves. In severe instances, defoliation and other dieback and decline symptoms may follow. (See section on diebacks and declines.)

EXCESSIVE WATER

Excessive amounts of water may injure trees. Symptoms are usually associated with man-made changes in grade levels around logging sites or adjacent to new highways where streams have been blocked. Symptoms include dwarfing of leaves, general chlorosis of all foliage, and dieback. Affected plants may die gradually, and the cause of death can be readily identified by examining drainage conditions in the area.

Chemical Injuries

SALT SPRAY INJURY (HIGHWAYS AND INLAND OF OCEANS)

Disease description: The accumulation of salt on or within plants often causes severe injury. This injury may occur on plants growing where deicing salts are used or up to tens of miles inland from ocean sources. In long-term impact areas, tree dieback, decline, and death have been noted to occur. Uptake occurs through direct foliar applications of wind-driven sprays or through root absorption of salts accumulated in soils. Due to their year-round foliage, conifers may have symptomatic foliage during winter months. Terminal portions of needles turn red-brown then become darker brown and very brittle (Figure 138); wind breakage of dead needle portions is common. Hardwood foliage shows a marginal chlorosis, followed quickly by marginal necrosis and defoliation (Figure 139). Premature fall coloration can also be a symptom of salt injury. Bud, twig, branch, and tree death occur in both conifers and hardwoods.

Cause: Salts like NaCl and $CaCl_2$ in aerosol form, roadway application of deicing salts, natural saltwaters, and cooling tower spray drift from use of saline waters at large power plants have all been shown as important sources.

Suscepts: Most major forest tree and understory tree species are sensitive to salt accumulations and sprays. Notable among these are sugar maples adjacent to roads throughout New England and eastern white pine at distances in excess of 300 yards (300 meters) downwind of heavily travelled highways. Loblolly and other coastal species have been injured by wind-driven sea spray up to distances of 12 to 18 miles (20 to 30 km) inland.

Range and site description: Salt-spray injury can occur almost anywhere salts or sea spray exist. Low-lying areas adjacent to roads allow accumulation for eventual root uptake. Damage is often most severe on trees located downwind of major highways.

Diagnostics: Observing the location of injured trees is most helpful in diagnosis. Symptoms resemble those of abiotic stresses, including acute air pollutant injury. Weather patterns like recent hurricanes should also be checked when injury is inland of ocean sources.

PESTICIDE SPRAY DRIFT

Herbicide application for control of undesirable vegetation is a fairly common forest management practice during site preparation or tree release operations. Herbicides are also used to control brush within and along power lines and various pipeline rights-of-way. Such chemicals are also used along highways and rail lines and on expanses like golf courses, agricultural fields, and similar areas to control competitive vegetation.

Figure 138

Large acreages have also been sprayed with various insecticides in control efforts aimed primarily at defoliating types of insects like the gypsy moth and spruce budworm. Fungicides or other disease-control chemicals are seldom applied to forest trees, with the noted exception of needle disease-control programs on Christmas tree plantations.

Pesticide application practices occasionally result in injury to non-target vegetation through drift of the chemical, improper conditions for application, or improper dosage application. Herbicidal drift is the major cause of such induced injuries under forest conditions.

Figure 138. Salt spray injury on eastern white pine located next to road surface treated with deicing salts.

Figure 139

Figure 139. Marginal scorch of basswood leaves due to toxic accumulations following uptake of deicing salts.

On broadleaf species, symptoms may include chlorotic or necrotic spotting, marginal scorch, leaf cupping and curling (Figure 140), bud death, tree dieback, decline, and perhaps mortality. Injury to conifers may include similar spotting patterns, but banding, tip necrosis, and curling of new shoots may also occur (Figure 141). A pattern of injury to nontargeted vegetation may be evident from the actual location of pesticide application, through a progression of symptoms, to a lessening and eventual disappearance of symptoms. Knowledge of direct or nearby pesticide applications, amounts, conditions, times of application, and actual chemical(s) used will assist in proper diagnosis. Tissue analysis may be necessary to confirm and properly identify such chemical injuries.

Figure 140

Figure 141

NUTRIENT IMBALANCES

Much is known about the symptoms associated with the shortage or excess of nutrient supply to plants. The effect of either extreme is, however, sometimes difficult to diagnose and may require chemical tests of soil and plant tissue.

Symptoms of a specific nutrient deficiency may appear on all parts of a tree but are most common on foliage. The coloration exhibited by the foliage is often the best means of determining the element involved. Lists of nutrient disorders are available. Some nutrient-imbalance symptoms are similar to those induced by air pollutants.

Unfavorable soil pH may cause mineral imbalances in the soil solution. These imbalances are usually manifested in the foliage as nutrient excesses or deficiencies. Low or high pH, for example, may lead to the release of minerals like aluminum that become toxic to the plant or to the immobilization of minerals like iron or phosphorus that are essential for plant growth. In the eastern United States, low soil pH is the most common cause of foliage yellowing in some species, resulting in stunting, loss of leaves, and sometimes death. Soil analysis should be conducted where symptoms are present, and, if possible, a comparison should be made to optimum nutrient levels for the specific species involved.

Figure 140. Herbicide injury to red maple leaves. Note curled, twisted, and elongated leaf deformation.

Figure 141. Herbicide injury to Colorado blue spruce, showing tip necrosis of newest needles.

Forest Tree Insects

Numerous insect species cause injury to trees in the temperate forests of the eastern United States. Insects attack all parts of trees, including foliage, shoots, cones, seeds, stems, and roots. Injury may be negligible or present as scarcely noticeable feeding on leaves, or it may be catastrophic, as when defoliation of millions of acres leads to decline and death of trees, or when the girdling activities of feeding bark beetles lead to extensive mortality. Only selected examples of insect injury are included in this manual; many other species are found in the field. For diagnostic purposes, the observer should at least be able to identify the feeding groups of these other species, and, in most cases, to separate insect-induced injury from injury caused by other agents. In many instances, the insect being reviewed has not been associated with specific sites; however, its general range has been presented.

This part of the manual is divided into two major sections: Hardwood Insects and Conifer Insects. In some instances, the same species of insect attacks both hardwood and conifer hosts. Each major section is further divided into discussions based on feeding groups.

Leaf-eating insects (defoliators) may be divided into three subgroups: chewers, leafminers, and skeletonizers, depending on the location and kind of feeding activity. *Chewers* are insects that consume entire leaves or large portions of leaves, frequently including vascular tissues. Especially during the early instars, some species eat only holes in individual leaves, making them appear as if they had been blasted with a shotgun. This kind of leaf-eating is known as shothole feeding. As larvae grow, they consume larger portions, often beginning at leaf edges and eventually removing entire leaves. *Leafminers* live between the epidermal layers of leaves, sometimes consuming vascular tissues as well. *Skeletonizers* consume leaf tissue, often from the lower side of the leaf, leaving only the upper epidermis or vascular tissues.

Sucking insects have sucking rather than biting and chewing mouthparts. These species insert their mouthparts into plant tissues and draw nutrients and fluids from their host. Noningested plant tissue is frequently discolored, misshapen, or has galls as a result of excessive tissue growth.

Meristematic insects may attack any of the growing tissues of a tree, including shoots, twigs, cones, seeds, roots, or the phloem-cambium areas. Depending on the tissues involved and the intensity of the attack, damage symptoms may be local, i.e., limited to single cones or seeds, or extensive, e.g., a bark beetle attack in the bole that girdles and kills the whole tree.

Wood and wood-boring insects are common, especially as secondary invaders, but their symptoms are not likely to be confused with air pollution damage, so they are not included as a separate group in this manual.

HARDWOOD INSECTS

Because the majority of important insect pests of hardwoods are leaf-eaters, such insects are emphasized in this manual. A few examples of insects with sucking mouthparts and a meristem feeder are also included for reference.

Leaf-eating Insects (Defoliators)

GYPSY MOTH

Description of injury: Eggs hatch in the spring at about the time of budbreak and early leaf expansion. Young, black, hairy larvae about ⅛ to ¼ inch (3 to 6 mm) long crawl up to the tops of trees, spin down on silken threads, and are blown by the wind from tree to tree or over even greater distances. Once settled on a suitable host, larvae begin to feed. Early feeding when leaves are expanding removes small pieces of leaf tissue (Figure 142); this is called shothole feeding. By late spring, large pieces of leaf are consumed and leaves may be severed at the petiole, giving the appearance of premature leaf fall.

Trees may be partially (Figure 143) or completely defoliated. From a distance, light defoliation may make the tree appear unthrifty; closer examination reveals missing portions of leaves or needles. For many hardwoods, heavy defoliation (foliage loss exceeding 60 percent) triggers refoliation during midsummer; a single complete defoliation frequently kills coniferous hosts.

Hosts: The insect does especially well on white and chestnut oaks. Other common hosts include red and black oaks, aspens, gray, white, and river birches, willows, and especially for the older larvae, beech, hemlock, Douglas-fir, and all species of pines and spruces. During population outbreaks, all forest tree species except yellow-poplar and ash may be partially or entirely consumed.

Range and site description: The gypsy moth occurs over large areas in the northeastern United States. Isolated infestations have been discovered in several midwestern, southern, and western states.

Trees growing under all sorts of conditions may be attacked — trees on ridges, slopes, bottomlands, in urban environments and forests, on good or poor sites, in pure stands, or in mixed forests.

Insect: The gypsy moth, *Lymantria dispar*, overwinters in the egg stage. Single egg masses may range from the size of a dime to larger than a half dollar; egg masses are circular to oval and are buff-colored (Figure 144). Masses

Figure 142

Figure 143

Figure 142. Defoliation caused by gypsy moth larval feeding on chestnut oak. Shotholes are caused by young larvae; larger holes and edge feeding are caused by larger larvae.

Figure 143. Moderate defoliation of oak tree canopy; caused by gypsy moth feeding.

may be laid almost anywhere but are most commonly seen on boles of trees, especially those with rough or fissured bark. Undersides of large branches in the lower crown are frequently used for egg deposition. Fully grown larvae are from 1 to 3 inches (2 to 8 cm) long, dark gray in color, and have five pairs of blue tubercles (dots) and six pairs of brick red tubercles on the dorsum, or upper side (Figures 145, 146). From mid-June to late July, brown pupae may be found; the pupal stage lasts ten to fourteen days. Pupae are loosely attached to the substrate, often under leaves or bark flaps, or in hollows of trees or fissures in the bark. Adult males, which are fairly strong daytime fliers, are brownish with dark wavy bands across each forewing; nonflying females are larger, off-white, and, like males, have wavy bands and marginal dark spots on their forewings (Figure 144).

Diagnostics: Except for partially consumed and then abandoned needles that may discolor, feeding does not normally cause discoloration of foliage left on trees. There is little chance of confusing defoliation with disease or air-pollution damage if individual leaves are examined. During the fall, winter, or early spring, look for the egg masses and other signs of attack by agents associated with dieback and decline diseases.

Figure 144

Figure 145

Figure 146

Figure 144. Adult female gypsy moth laying a mass of eggs in a bark crevice on oak. Egg masses may be numerous, and their evidence may remain for a second season.

Figure 145. Fully grown gypsy moth larva, showing five pairs of blue tubercles followed by six pairs of brick red tubercles on the dorsum.

Figure 146. Large gypsy moth larvae, showing red color phase typical of heavy infestations (left) and gray color phase typical of light to moderate infestations (right).

Description of injury: Larvae hatching before budbreak enter and mine leaf buds, destroying a number of leaves; those hatching later consume emerging leaf tissue, normally from the edges of leaves. Trees under attack may appear to have thin crowns if leafroller populations are not particularly heavy; when populations are heavier, most leaves may be damaged (Figure 147). Close observation reveals consumed portions of leaves, and, especially from early summer onward, many rolled leaves (Figure 148). These rolled leaves will often contain larvae, or, later on, pupae or cast pupal skins.

Early-season defoliation may trigger refoliation of host trees that lose more than 60 percent of their foliage. Heavy defoliation for two or three successive years causes tree mortality. Because the energy reserves of the trees are so depleted, stump sprouts or root sprouts rarely occur.

Hosts: While oak leafroller larvae favor oaks, they also feed on maple, witch-hazel, and apple, especially during population outbreaks. The oak leafroller may occasionally cause extensive defoliation over large areas of forest, especially in forests with large proportions of red and white oaks.

Range and site description: Outbreaks normally occur in mature to overmature forests and have been recorded in some of the northeastern and northern midwestern states. During the last several decades, severe outbreaks have been reported in Michigan, Pennsylvania, and New York. Pennsylvania suffered extensive

Figure 147

Figure 148

Figure 147. Defoliation of oak species caused by the oak leafroller, leading to thin crowns, dieback, and decline.

Figure 148. Leaf showing skeletonizing and rolling by oak leafroller on red oak.

tree mortality during the late 1960s and early 1970s, probably because a prolonged drought intensified the effects of defoliation. Some of the heaviest defoliation has been recorded on trees on some of the better bottomland sites.

Insect: The oak leafroller, *Archips semiferanus,* overwinters in the egg stage. Egg masses are closely appressed to the bark of the tree (Figure 149). Numbers of egg masses are concentrated on the lower bole and in the lower portion of the crown. Egg masses may also be found on some of the larger, lower branches.

At about the time of foliage flush, newly hatched larvae are less than ⅛ inch (3 mm) long. Through a series of molts, they grow to approximately ¾ inch (2 cm) long. They are yellowish green and have a dark head capsule (Figure 150). Larvae continue to feed on the succulent young and expanding leaves until mid- to late June, at which time they pupate in rolled leaves, bark fissures, or other sheltered places. New pupae are light brown and darken with age. Adults are ½ inch (1 cm) long, reddish brown moths.

Diagnostics: Close examination of foliage reveals that pieces of leaf have been removed. Numbers of rolled leaves, cast larval skins, pupal cases, and hatched or unhatched egg masses on the bark are easily seen, particularly in moderate to heavy infestations.

Figure 149

Figure 150

Figure 149. Closely appressed egg masses of the oak leafroller, *Archips semiferanus,* on the bark of oak. Topmost egg mass shows exit holes from hatched larvae.

Figure 150. Larvae of the oak leafroller feeding on young, expanding leaves of oak.

Description of injury: Egg masses of the forest tent caterpillar may be observed on twigs of the hosts from midsummer through to the following spring (Figure 151). Young larvae sometimes skeletonize leaves, whereas older larvae typically consume entire leaves. Defoliated forests appear silvery gray from a distance or from the air. Early feeding by larvae is done mainly in groups; larger larvae feed singly. In spite of its common name, this insect does not form tents. Diebacks and declines may follow defoliation.

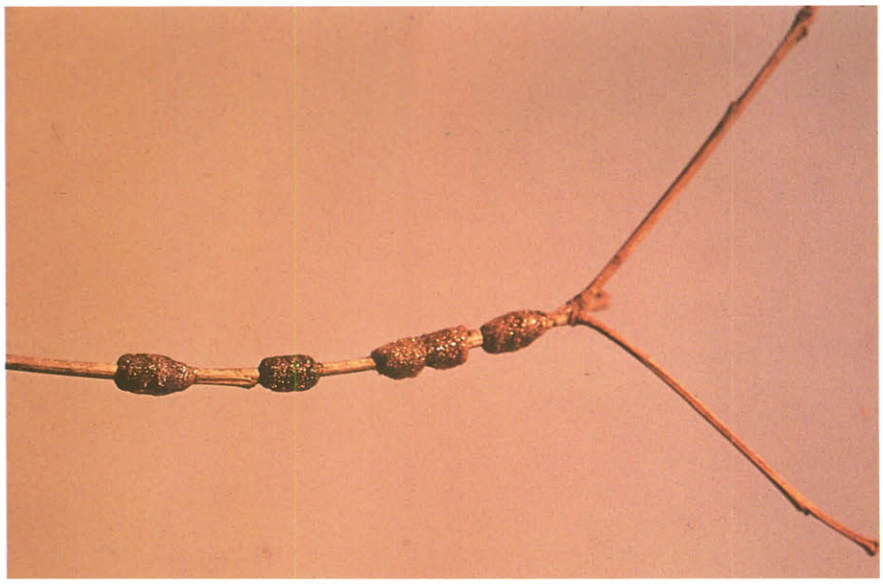

Figure 151

Hosts: Forest tent caterpillars attack many different kinds of hardwoods, but they prefer trembling (quaking) aspen, sugar maple, blackgum, tupelo, and sweetgum; various species of oaks are commonly attacked in the South.

Range and site description: This insect thrives throughout most of the United States and Canada wherever suitable hosts are present. Trees on bottomlands and along waterways are commonly attacked.

Insect: Fully grown larvae of *Malacosoma disstria,* the forest tent caterpillar, may be 2 to 3 inches (5 to 8 cm) long and have light blue heads with black mottling. Each abdominal segment has a brownish yellow, keyhole-shaped spot in the middle of the upper side (Figure 152). Larvae are bluish gray with a gray lateral stripe running the length of the body.

Figure 152

Figure 151. Egg masses of the forest tent caterpillar deposited along a twig of an unidentified host species.

Figure 152. Larva of the forest tent caterpillar, *Malacosoma disstria,* showing the characteristic keyhole pattern on the dorsal side.

Diagnostics: Larvae do not form tents but rather lay down silken trails or mats on foliage and tree bark. Pupae are contained in pale yellowish cocoons, often in folded leaves, bark fissures, or other protected places. Close examination reveals portions of leaves removed or entire leaves missing.

FALL CANKERWORM

Description of injury: In early spring, shortly after budbreak, young larvae of fall cankerworm feed as skeletonizers or shothole feeders (Figure 153), primarily in the upper edges of the crown. As larvae grow, they consume entire leaves, sometimes leaving main ribs, and then move down into the crown after they have consumed the tops of the trees. Feeding is normally completed by mid-June.

Outbreaks of fall cankerworm may cause extensive defoliation over large areas of forested land. With repeated successive years of defoliation, trees weaken, decline, and may die. Large numbers of larvae frequently can be observed moving across the ground, seeking new sources of food after they have defoliated a tree.

Hosts: This insect is found on almost all hardwood species in eastern North America but prefers elm, basswood, red, silver, and sugar maple, red oak, apple, hickory, ash, beech, and boxelder.

Range and site description: Fall cankerworm occurs throughout the eastern United States as far south as Georgia and west to Missouri and Montana. In southern Canada, it occurs in the Maritime Provinces and west to Alberta.

Figure 153

Figure 154

Figure 153. Feeding injury caused by fall cankerworm on sugar maple.

Figure 154. Larva of the fall cankerworm, *Alsophila pometaria*.

Insect: The fall cankerworm, *Alsophila pometaria,* has full-grown larvae about 1 inch (2 to 3 cm) long. The head and anal shield vary from light green to almost black; longitudinal stripes run the length of the body (Figure 154). Larvae are loopers, which move by drawing the posterior portion of their abdomen up behind the thorax while looping their middle portion above the surface. Larvae have a single pair of rudimentary prolegs and two pairs of normal prolegs.

Diagnostics: Look for early feeding in the upper periphery of crowns of suitable hosts, followed by heavier feeding that progresses down the crown. Larvae are easily seen until pupation, which occurs in cocoons in the ground. As noted earlier, these are typical looper larvae, which move inchworm fashion.

SADDLED PROMINENT

Description of injury: During the first three stadia, larvae of the saddled prominent feed as skeletonizers, usually on the undersides of leaves. During the remaining larval stadia, the insects consume foliage from the edges of leaves, devouring all of the tissues. During outbreaks, the saddled prominent may heavily defoliate entire trees over extensive areas of forested land (Figure 155). Following a single defoliation, some dieback may be seen in the smaller twigs in the upper portion of the crown; successive years of defoliation reduce annual growth and may kill some trees.

Figure 155. Varying degrees of defoliation caused by the saddled prominent in a mixed hardwood stand.

Hosts: Beech, yellow birch, and sugar maple are preferred, but more than twenty other hardwoods have been recorded as hosts, especially in outbreak conditions.

Range and site description: The saddled prominent is found throughout the eastern United States and southeastern Canada.

Insect: Young larvae of the saddled prominent, *Heterocampa guttivitta,* possess a pair of fleshy, antler-like horns on the first thoracic segment and are dark red to black. Older larvae have a brownish purple "saddle" marking dorsally on the first several abdominal segments (Figure 156). Full-grown larvae are about 1¼ inches (3 cm) long and have a flattened, backward-angled head capsule.

Figure 155

Diagnostics: Larvae can be found feeding on leaves from mid-June through mid-August. Obvious injuries include removal of the epidermal layer during skeletonizing or removal of entire portions of leaves by large larvae.

BIRCH LEAFMINER

Description of injury: Birch leafminer larvae mine within the leaves of the host, excavating blotchy mines (Figure 157). Feeding by several larvae within the same leaf may consume the entire leaf between the epidermal layers. Leaves turn from silvery to grayish to reddish brown, giving heavily infested trees a scorched appearance by mid- to late summer (Figure 158). The several generations per year can cause heavy, unsightly damage by the end of summer.

Hosts: Birch leafminer attacks all species of birches but prefers gray birch, European white birch, and paper birch; sweet birch is seldom attacked.

Range and site description: This insect is found throughout northeastern North America, from Maryland northward and westward to Minnesota and Iowa; it also occurs in eastern Canada. It is common on ornamentals as well as on forest trees.

Insect: Larvae of the birch leafminer, *Fenusa pusilla*, are yellowish white, flattened, and legless and may be found in an active mine.

Figure 156

Figure 157

Figure 158

Figure 156. Larva of the saddled prominent, showing its characteristic saddle marking on the dorsum.

Figure 157. Birch leafminer injury, showing characteristic mining between the upper and lower epidermis of the leaf. Opened mine reveals larva surrounded by fecal pellets.

Figure 158. Overall scorched appearance of a birch tree heavily infested with the birch leafminer.

Diagnostics: Since birch leafminers attack new foliage, the periphery of the crown continues to fall victim to later generations as trees produce new leaves throughout the season. Close examination of the leaves reveals paper-thin tissue containing dark fecal pellets between the epidermal layers (Figure 157). New leaves may bear grayish spots where female adults have injured tissues, probing with their ovipositors during egg-laying (Figure 159).

LOCUST LEAFMINER

Description of injury: Adult beetles of the locust leafminer feed on the lower surfaces of leaves in the spring, skeletonizing them and occasionally eating holes through the leaves (Figure 160). Larvae, which hatch from eggs laid on the leaves, enter and feed as miners between the epidermal layers of the leaves (Figure 161). A single larva frequently uses more than one leaf to complete development. By midsummer, leaves are discolored and have turned red-brown, giving the tree a scorched appearance (Figure 162).

Hosts: This insect prefers black locust but also attacks birch, beech, apple, cherry, elm, hawthorn, and oaks.

Range and site description: The locust leafminer is found throughout most of the eastern United States and Canada wherever its hosts occur.

Figure 159

Figure 160

Figure 161

Figure 159. Leaf injury from ovipositor probing by the birch leafminer.

Figure 160. Locust leafminer injury on locust; adult beetles are also present on the leaf.

Figure 161. Mine caused by larval stage of the locust leafminer.

Figure 162

Insect: Adult *Odontota dorsalis* beetles are about ¼ inch (6 mm) long, elongate, flattened, and orange-brown, with a black stripe on the inner edge of each wing. Larvae are flattened, yellowish white, and have a darkened head and legs (Figure 163).

Diagnostics: Close inspection reveals leaf tissue removed from the lower epidermis by adult feeding, as well as shrivelled or papery leaves hollowed out by larval mining. Mined leaves are dry, brittle, and contain evidence of insect feeding in the form of frass.

Figure 163

Figure 162. Discolored, reddish brown appearance of black locust trees heavily damaged by the locust leafminer.

Figure 163. Left to right, the larva, pupa, and adult locust leafminer.

PERIODICAL CICADA

Description of injury: In midsummer, the female adult periodical cicada cuts jagged slits through the bark and the wood of succulent twigs, into which she deposits her eggs (Figure 164). With multiple oviposition slits in a single twig, leaves wilt, die, and turn brown distal to these injured sites. Flagged tips may break and hang or fall from the tree (Figure 165). In heavy infestations, the periphery of the tree's crown may appear scorched. In the second year, considerable twig dieback afflicts heavily attacked trees.

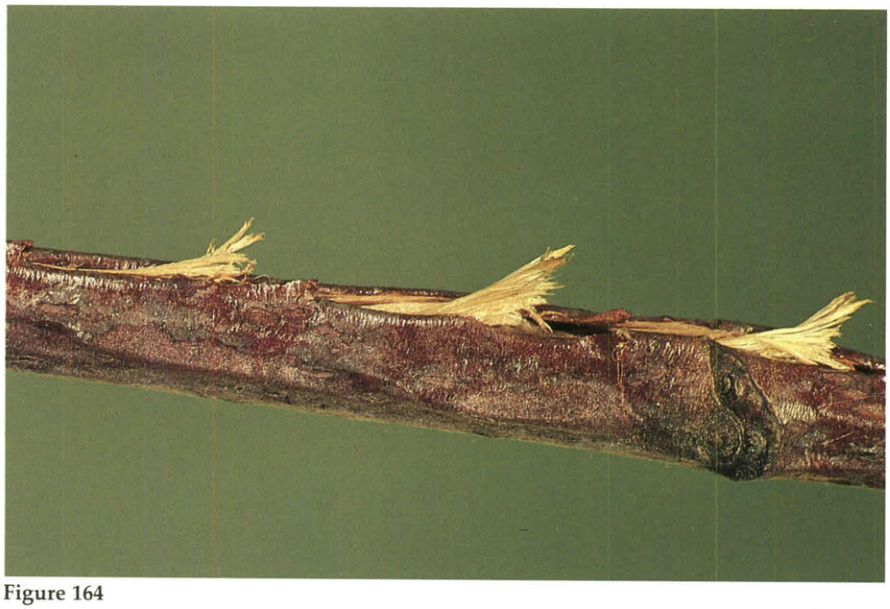

Figure 164

Figure 165

Figure 164. Jagged slits cut into a twig by the ovipositor of the female periodical cicada.

Figure 165. Flagging, or brown branch tips, following oviposition by the female periodical cicada.

Nymphs feed underground on roots for thirteen to seventeen years, causing no readily visible damage.

Hosts: More than seventy species of trees and shrubs are known hosts of the periodical cicada. Oaks, hickory, honeylocust, dogwood, apple, and peach are among the most commonly attacked.

Range and site description: This insect is widely distributed throughout the eastern United States.

Insect: As an adult, the periodical cicada, *Magicicada septendecim*, is about 1½ inches (4 cm) long, stout-bodied, dark-colored, and has clear, stiff, membranous wings held rooflike over the body at rest (Figure 166). Nymphs settle on roots about 18 to 24 inches (0.5 m) below the ground surface.

Diagnostics: Leaves are not damaged directly but merely dry up and turn brown from midsummer onward in years of brood emergence and oviposition. Examination of shoots reveals numbers of ragged slits about ½ inch (12 mm) long through the bark. Slits are often placed end to end, appearing much longer than they actually are.

Figure 166

Figure 167

PEAR THRIPS

Description of injury: In the spring of the year, feeding injury of pear thrips is often confused with late frost damage. Leaves look flaccid and darkened. By August, affected trees turn color prematurely and drop their leaves. Dieback symptoms have been reported in areas known to have thrips infestations. This insect has been recognized only recently as being the agent of the reported damage.

Hosts: Red maple, sugar maple, and beech serve as hosts in forested environments.

Range and site description: Although the range of the insect is known to be much larger, in recent years this insect has been recorded as a pest only in northern Pennsylvania, southern New York, Vermont, and New Hampshire. One report claims that trees on the highest ground are the most severely attacked, but this claim has yet to be verified.

Insect: The pear thrips, *Taeniothrips inconsequens*, is very small, being only about ⅛ inch (3 mm) long and very slender. Immature stages are wingless. Adults are brown and have two pairs of very long, narrow wings that are veinless but have a hairy fringe. This insect is not readily seen except through close observation (Figure 167).

Figure 166. An adult periodical cicada, showing clear, stiff, membranous wings while insect is at rest.

Figure 167. Adult pear thrips on maple bud scales, showing the comparative small size of this insect.

Diagnostics: Pear thrips have rasping and sucking mouthparts and feed on the surface of leaves. Infested leaves look tattered (Figure 168).

Meristematic Insects

BRONZE BIRCH BORER

Description of injury: Bronze birch borers normally attack the crown first, causing individual shoot or branch mortality, chlorosis of foliage, and an overall appearance of decline (Figure 169). Eggs are laid in crevices and cracks, often near branch axils. Larvae mine directly into the cambium and often girdle the branch or bole of the tree with meandering tunnels between the bark and the wood (Figure 170). Tunnels of older larvae may occasionally penetrate the wood and are normally packed with powdery frass. Portions of branches or stems distal to the area of girdling usually decline and die; stems frequently break off at areas of attack (Figure 171). Trees or branches more than about 1 inch (2 to 3 cm) in diameter are subject to attack.

Hosts: European white birch, paper birch, and yellow birch are the preferred hosts.

Range and site description: Bronze birch borer, found throughout the range of birches in the United States and Canada, tends to attack weakened trees or trees on poor sites.

Figure 168

Figure 169

Figure 168. Tattered sugar maple leaf caused by pear thrips feeding.

Figure 169. Declining birch trees following infestation of branches and main stems by the bronze birch borer.

Figure 170. Mines and swollen bark areas caused by larvae of the bronze birch borer on white birch.

Figure 171. Stem breakage in a white birch stand. Most breaks occur at the point of girdling by bronze birch borer larvae.

Insect: Adults of the bronze birch borer, *Agrilus anxius*, are bronze-green and about ¼ to ½ inch (6 to 12 mm) long. Full-grown larvae are about 1 inch (2 to 3 cm) long and are typical of flat-headed wood borers. They are creamy white, compressed dorsoventrally, have an enlarged, wide thoracic area, and have two spines at the posterior end of the abdomen.

Diagnostics: Any aboveground portion of the tree may be attacked. Raised welts over the larval tunnels may be observed on the bark of trees under attack (Figure 170). Breakage of trees, es-

Figure 170

Figure 171

pecially in the winter, commonly occurs at weakened places in the bole. D-shaped exit holes may be seen after beetle emergence, between late May and August. The head and thoracic segments of overwintering larvae are characteristically doubled back over the anterior part of the abdomen (Figure 172).

CONIFER INSECTS

In the Southeast, bark beetles are the most important insect pests of conifers. Along with some of the other meristematic insects, they are most likely to cause symptoms that might be confused with air pollution damage. Defoliators and sucking insects such as the gall adelgids are more important in the northeastern and New England states.

Leaf-eating Insects (Defoliators)

EASTERN SPRUCE BUDWORM

Description of injury: After overwintering in a silken hibernaculum as a second instar larva (Figure 173), the eastern spruce budworm moves to the exterior of the crown early in spring, seeking staminate flowers. Lacking these, larvae mine one to several old needles while awaiting budbreak, at which time they attack new foliage. As the season progresses, larvae consume older foliage as well, foraging out from loosely constructed silken shelters, which they make by tying together needles from adjacent twigs. By early to midsummer, the injured and dying, but not fully consumed,

Figure 172

Figure 173

Figure 174

Figure 172. Overwintering bronze birch borer larva, showing characteristic shape of doubling back of head and thorax over the anterior portion of the abdomen.

Figure 173. Silken hibernaculum of a second instar larva of the eastern spruce budworm on balsam fir.

Figure 174. Larva of the eastern spruce budworm, showing dark head capsule, white spots, and brownish green color.

needles give tree crowns a reddish brown, burned appearance. After repeated defoliation, crowns are fully open, thin, and sparsely foliated. Thousands of hectares have been killed annually in eastern North America.

Hosts: Hosts include balsam fir, white spruce, and red spruce. In the various parts of its range, this insect feeds on at least twenty-five species of conifers, including eight of spruce, six of pine, five of fir, three of hemlock, two of larch, and juniper.

Range and site description: Eastern spruce budworm is the major defoliator of balsam fir and spruce in the northeastern United States, in the Great Lakes region, and across eastern Canada.

Insect: The larva of eastern spruce budworm, *Choristoneura fumiferana,* is fully grown by June or July. It is about 1 inch (2 to 3 cm) long, has a dark head capsule, and is brownish green dorsally and spotted with white laterally (Figure 174).

Diagnostics: Observe closely to detect early feeding and mining on buds and in needles. Larvae spin down from the foliage on silken threads when limbs are jarred. This insect prefers new foliage, so the tops of trees and the crown periphery are most noticeably defoliated (Figures 175, 176).

Figure 175

Figure 176

Figure 175. Injury to crown periphery of red spruce; caused by the feeding of the eastern spruce budworm.

Figure 176. Defoliation of red spruce branch terminals; caused by the eastern spruce budworm.

Description of injury: Nantucket pine tip moth larvae mine the growing shoots of pine trees. As the shoots die, the tips of the trees turn brown (Figure 177, 178). All new growth may be killed where population levels are high; however, where populations are low, only the buds and the most distal portions of the shoots may be killed. In areas where tip moth populations are high, these insects attack virtually every tree in the stand.

Hosts: Preferred hosts include all southern pines, especially shortleaf pine; longleaf pine is less often attacked, and slash pine is attacked infrequently.

Range and site description: Nantucket pine tip moth is widely distributed in the eastern United States and is of particular importance to pines in the southeastern United States from Maryland to Texas. Populations are especially heavy on stressed sites in the Coastal Plain and on poor sites in the Piedmont areas. In all susceptible areas, heavy moth populations can be expected on intensively managed, mechanically prepared sites and where herbaceous weed control is practiced.

Insect: Adults of the Nantucket pine tip moth, *Rhyacionia frustrana*, are inconspicuous, brownish gray moths with wing spans of less than ¾ inch (18 mm). Larvae are light brown to orange and grow to a maximum length of about ⅓ inch (8 mm).

Figure 177

Figure 178

Figure 177. Terminal bud and shoot death of pine; caused by larval mining by the Nantucket pine tip moth.

Figure 178. Damaged shortened new shoots on Scots pine as a result of attack by Nantucket pine tip moth.

Diagnostics: Early attacks are characterized by silvery webs between buds which are coated with fresh resin. Old attacks can be identified by the dead, brownish shoots that easily crumble when pressed between the thumb and fingers. These dead shoots may have yellow, crystallized resin beads on the buds.

HEMLOCK LOOPERS

Description of injury: Hemlock looper larvae damage trees by feeding on foliage. Heavy defoliation kills individual trees or groups of trees; more moderate defoliation slows tree growth. The larva's habit of consuming only a part of the foliage increases damage. Young larvae eat the edges of needles but leave a central filament which eventually dries out, curls, and turns reddish brown. Older larvae commonly move from needle to needle, taking only a few bites from near the base. These needles then die, showing the same scorched symptoms described earlier. Other needles are severed at or near their base and simply drop to the ground. In a heavy defoliation, enough needles are killed and removed that twigs are exposed directly to sunlight and may die.

Partial defoliation reduces annual growth, and successive years of partial defoliation often weaken the tree enough to allow other pests to become established and ultimately contribute to tree mortality.

Figure 179

Hosts: Hemlock loopers are common in eastern hemlock, balsam fir, and spruce forests. Several species and subspecies of hemlock loopers attack hemlock and other conifers in eastern North America. *Lambdina athasaria* occasionally appears on eastern hemlock. During most years, however, the insect seems to go unnoticed and frequently unreported. *Lambdina fiscellaria,* a more widespread species, commonly feeds on balsam fir as well as hemlock. When populations are very heavy, it may move to other native conifers as well as hardwoods, including birch, maple, and aspen. Outbreaks tend to occur in overmature stands of host trees.

Range and site description: Hemlock loopers are found throughout the northeastern United States and across Canada from Newfoundland, at least as far west as the Rocky Mountains.

Insect: Hemlock loopers, *Lambdina* spp., overwinter in the egg stage. Eggs are frequently laid on twigs near the bases of needles and occasionally on the trunks of trees. They are also laid singly or in small groups under lichens, in bark crevices, in moss growing on old stumps, and on trunks and branches of host trees. Eggs are not covered by any frothy or setaceous protective material. Young larvae initially feed on the succulent new needles. Larvae are greenish gray to brown, with lateral, paler stripes. They move in the typical inchworm fashion, looping along on the needles or branches (Figure 179).

Figure 179. Side view of looping or inchworm nature of hemlock looper larva.

Diagnostics: From a distance, the crown of a tree attacked by hemlock loopers appears thin and unthrifty (Figure 180). Closer examination may reveal a number of severed needles that have fallen to the ground under the canopy and portions of needles, usually discolored and shrivelled, still on the tree. Where pieces of needle have been removed, the physical damage is quite obvious.

INTRODUCED PINE SAWFLY

Description of injury: Larvae of the introduced pine sawfly damage host trees by attacking mature foliage. First-generation larvae consume entire needles of the previous years' growth, thinning the crown, especially in the upper half. The second generation feeds on the remainder of the old foliage and on the hardened-off foliage of the current year. Larvae also may feed on the bark of completely defoliated trees. Sawfly defoliation (Figure 181) commonly retards radial and terminal growth and occasionally kills branches and trees (Figure 182). At high population levels, the sawfly can cause complete defoliation, and, if the buds are also destroyed, can kill the trees as well.

Figure 180

Figure 180. Thin appearance and mortality of crowns of eastern hemlock defoliated by hemlock looper.

Figure 181. Crown defoliation of eastern white pine; caused by feeding activity of the introduced pine sawfly.

Figure 182. Browning of individual needles and shoot dieback; caused by feeding of the introduced pine sawfly.

Figure 181

Figure 182

The early instars feed gregariously, moving from the needle tip to the base and leaving only the central vascular tissue (Figure 183). By the end of the third instar, larvae feed solitarily and consume the entire needle.

Cocoons are constructed on any solid object above ground, commonly in clusters, and in protected areas (Figure 184). Larvae of the overwintering population may spin cocoons in the duff. The cocoon is a tough, cylindrical, silken capsule that is white when newly spun but turns brown with age.

Hosts: The introduced pine sawfly feeds on a wide range of pines, including white, red, Scots, jack, and Austrian. Although other species have been shown to be suitable for oviposition and survival of eggs and larvae, white pine is the preferred and most suitable host. The sawfly rarely causes significant defoliation on other pine species. Defoliation is normally not a problem on seedlings, but all other age classes are susceptible.

Range and site description: This insect, introduced from Europe, occurs in natural stands, plantations, and Christmas tree plantations. It is found from Maine to Virginia and west to the Great Lakes states, as well as in southeastern Canada.

Figure 183

Figure 184

Figure 183. Young larvae of introduced pine sawfly feeding gregariously on a single needle.

Figure 184. Cocoons of the introduced pine sawfly on a totally defoliated eastern white pine.

Figure 185. A large stand of Fraser fir killed by the balsam woolly adelgid.

Insect: The adult female of the introduced pine sawfly, *Diprion similis,* lays small, oval eggs serially in slits that she cuts in the needle. These cuts may create some confusion with symptoms of the needlecast fungi. The eggs are pale blue when first deposited and turn turquoise as they mature.

When first-instar larvae hatch following oviposition in the spring, they are about ¼ inch (6 mm) long and have white head capsules and dull gray bodies. Shortly after emergence, the head capsule turns shiny black, the legs black, and the body a yellowish green. By the third instar, the larvae are slightly darker. By the fourth instar, coloration changes markedly; two black middorsal lines and numerous lateral yellow and white spots appear. The fifth and sixth instars have a distinct mottled pattern and reach a length of about 1 inch (2.5 cm). These instars have a light green to gray head capsule (2 to 3 mm) and very light and dull body markings. When disturbed, late instar larvae characteristically react by arching their heads over their backs and regurgitating a dark, oily fluid.

Diagnostics: Partial defoliation, starting from the top down, appears as a symptom of crown decline and may be confused with crown decline caused by other organisms. For a proper diagnosis, presence of the easily observed larval or cocoon stages should be confirmed.

Sucking Insects

BALSAM WOOLLY ADELGID

Description of injury: The balsam woolly adelgid, formerly known as the balsam woolly aphid, is native to the silver fir forests of central Europe. The insect was introduced into North America about 1900. A continuous, heavy infestation frequently causes crown dieback, and, within two to six years, tree mortality occurs (Figure 185). Infestations can occur on the tree trunk, in the crown, or

Figure 185

both (Figures 186, 187). Stem attack appears to be lethal to the host.

Once it finds a suitable feeding site, the insect inserts its stylet into the bark; it is then restricted to that location for the remainder of its life. As the insect feeds, it secretes saliva through the stylet, thereby stimulating an increase in the size and number of parenchyma cells in the feeding zone. The infestations cause gout, a syndrome characterized by enlarged nodes and buds (Figure 187). Gout reduces growth of new foliage by inhibiting the conduction of water and nutrients. Top kill from gout is common; affected trees assume a flattopped appearance.

Hosts: Fraser fir and balsam fir are particularly susceptible to attack. All size classes are infested. In natural stands, however, mature trees usually support the largest populations, probably because they intercept more wind-dispersed crawlers. Seedlings under a heavily infested overstory occasionally become infested. Fraser fir infestations consist mainly of stem attacks; in bracted balsam fir, crown infestations are more common.

Range and site description: Infestation patterns vary with region and host. Crown infestations predominate in the coastal areas of the balsam fir range, while stem attacks are more common inland.

The adelgid, discovered in the southern Appalachians in 1956, infests Fraser fir in North Carolina and bracted balsam fir in Virginia. It is found on balsam fir from New England to as far south as New York and in eastern Canadian forests.

Figure 186

Figure 187

Figure 186. Balsam woolly adelgid infestation on the trunk of Fraser fir.

Figure 187. Twig gout caused by the balsam woolly adelgid on Fraser fir.

Insect: The balsam woolly adelgid, *Adelges piceae*, overwinters as a dormant first-instar nymph. In spring, the nymphs begin to secrete waxy strands of wool, which eventually cover the entire body. At maturity — by late May — eggs are oviposited within the wool mass; most first-generation eggs hatch by the end of June. At low elevations, additional generations are possible.

Diagnostics: The best diagnostic tools are obvious signs of the insect on the bark of infested stems and the presence of the gout symptom. The flattop and red crown of individual trees in advanced stages of decline are easily seen on sites where the insect has existed over a period of years.

BALSAM TWIG APHID

Description of injury: The unique injury caused by the balsam twig aphid cannot be easily confused with injury caused by anything else. New needles appear curled and twisted, and large numbers of aphids feed within the curled foliage (Figure 188). Most of the twisted needles of mature trees will remain distorted. When populations are extremely high, the aphids are frequently observed migrating up and down the tree (Figure 189). Cast skins and black eggs covered with tiny rods of white wax occur on the needles, twigs, and main stem (Figure 190). These signs can be confused with the damage symptoms of the balsam woolly adelgid.

Figure 188

Figure 189

Figure 190

Figure 188. Curled and twisted foliage of balsam fir; caused by feeding of the balsam twig aphid.

Figure 189. Balsam twig aphid on new foliage; cast skins are also evident.

Figure 190. Black eggs of balsam twig aphid covered with rods of white wax.

Hosts: All fir species of all ages are attacked. Species and varieties that break bud earliest seem most susceptible to injury. The balsam twig aphid has also been reported on white spruce.

Range and site description: Found throughout eastern North America, the balsam twig aphid may injure any sections of the crown, regardless of level or aspect.

Insect: The balsam twig aphid, *Mindarus abietinus*, excretes large quantities of waxy, white wool as well as sticky honeydew. A winged generation allows the aphids to fly to adjacent trees. In the summer months, winged insects are naked, pale green, and are located in clusters on the needles and shoots.

Diagnostics: Twisted and curled new needles and the presence of the aphid are the best diagnostic tools. In addition, the presence of honeydew and the associated sooty mold fungi on the foliage are indicative of attack.

APHIDS ASSOCIATED WITH SOOTY MOLDS

Description of injury: Aphids usually live in colonies up to 3 to 4 inches (7 to 10 cm) long, clustered around a branch or leader. Young trees or individual branches of older trees may be killed. Heavy infestations of aphids can significantly reduce tree growth. Aphids also produce prodigious amounts of honeydew, an excretion that contains sugars; this excretion is frequently colonized by black sooty mold fungi (Figure 191). Sooty mold does not directly injure the tree, but it can interfere with photosynthesis. Infestations in forest stands can consist of individual trees, or, if aphid populations are high, many trees throughout the stand may be affected.

Hosts: All southern and eastern pines and most hardwoods are susceptible.

Range and site description: Infestations are found anywhere hosts grow. Low-vigor trees on poor sites are especially susceptible to aphid attack.

Insect: Aphids associated with sooty mold, commonly *Cinara* spp., are soft-bodied insects ranging in size from 1/25 to 1/4 inch (1 to 6 mm) long. Most aphids are pear-shaped and have a pair of cornicles (projections) on the posterior dorsum of the abdomen. Most pine aphids are brown to black.

Figure 191

Figure 191. Black colonies of the sooty mold fungus growing on aphid or scale exudate on the surface of Scots pine needles.

Diagnostics: Dieback or flagging of branches and terminals are symptoms on heavily infested trees. Sooty mold and ants frequenting a tree are also good indicators of aphid attack. In addition to its association with aphids, sooty mold is also associated with scale insect infestations.

PINE LEAF ADELGID

Description of injury: The pine leaf adelgid attacks spruce and pine and has a complex life cycle that comprises five distinct forms or stages. Injury associated with these stages includes gall formation at the base of spruce buds and feeding at the base of spruce needles. Damage on spruce is not serious, as there are usually sufficient numbers of uninfested buds. On white pine, however, the insect causes shoots to droop and eventually die; needles on affected shoot tips turn red (Figure 192). On white pine, the adelgid produces a waxy covering on the axils of new shoots and then lives in these axils. The insect occasionally kills whole trees.

Hosts: Red and black spruce, white pine, and occasionally red, Scots, and Austrian pine are hosts of the pine leaf adelgid.

Range and site description: This insect is found in Maine, New Hampshire, Vermont, New York, and throughout Canada. Site specificity is not known.

Insect: The pine leaf adelgid, *Pineus pinifoliae,* has five different morphological forms and attacks both spruces and pines. Some adelgids are cryptic, living within galls; others bear a waxy, flocculent covering that is more easily seen than the small nymphs themselves.

Diagnostics: Presence of the dormant first-stage larvae at the base of pine needles is the most distinctive signal for diagnosing an outbreak. Insects are present on new shoots of pine from July until the following May. On spruce, a compact gall resembling a cone may be found on the tip of the new growth.

Dead adults, often lined up in rows, may be found for several months on the needles. On pine, they are invariably oriented with the head toward the base of the needle; on spruce, with the head toward the tip of the needle.

Figure 192

Figure 192. Mortality of eastern white pine shoots; caused by feeding activity of the pine leaf adelgid.

Description of injury: Needles distal to infested portions of branches may turn yellow (Figure 193), die, and fall (Figure 194). Small trees may become stunted, or, rarely, die if heavily infested. Larger trees may appear off-color if under heavy attack, but they seldom suffer any permanent damage if otherwise healthy.

Hosts: White, Scots, Austrian pine, and occasionally spruce serve as hosts.

Range and site description: The species is widely distributed throughout North America, occurring wherever white pines grow.

Insect: The pine bark adelgid, *Pineus strobi,* is small, dark, and covered with white flocculent wax. When numbers of insects occur together, they give the appearance of white, cottony spots or patches on the trunks, or, most commonly, on the undersides of limbs of host trees (Figure 195). They may also occur on twigs at the bases of needles or buds (Figure 196). Extremely heavy infestations may give the trunk of a tree the appearance of having been whitewashed. Eggs are laid in the spring by the mature females that have overwintered; although some winged individuals are produced, most are of the wingless form. This form remains on the pine host and produces up to five generations during the year; winged individuals may fly to other hosts, including spruce, to deposit eggs.

Figure 193

Figure 194

Figure 193. Yellowing of eastern white pine as a result of infestation by the pine bark adelgid.

Figure 194. Pine bark adelgid infestation on Scots pine shoot. Some needles are turning brown as a result of twig attack.

Diagnostics: Small to large white spots or patches, or, in extreme cases, a whitewashed appearance of the trunk, suggest the presence of this insect (Figure 195). On close examination, small, dark insects with sucking mouthparts inserted into the substrate are revealed under the cottonlike waxy covering. Occasionally, yellowish or thinned foliage, especially on younger trees, may indicate the presence of this insect.

Figure 195

WHITE PINE APHID

Description of injury: Tree mortality, stunting, or branch killing may occur as a result of colonies of large numbers of white pine aphids feeding on young trees or on branches of older trees. Large quantities of honeydew, excreted by the aphids as they feed, provide a substrate for the growth of a black, sooty mold that is unsightly, interferes with normal tree growth, and reduces tree quality.

Hosts: Eastern white pine.

Range and site description: The insect occurs from New England to the Lake states and the Carolinas.

Insect: The white pine aphid, *Cinara strobi,* is a shiny, dark brown insect about 1 inch (4 mm) long in its winged form, with a white stripe down the middle of the dorsum, powdery white spots on its sides, and long, stiff hairs on the body. Eggs are laid in a row on needles of the host in the fall and overwinter. In the spring, these eggs hatch to produce

Figure 196

Figure 195. Pine bark adelgid infestation on branches of Scots pine.

Figure 196. Pine bark adelgid nymphs feeding on candles of Scots pine early in the season.

wingless females, which reproduce parthenogenetically through several generations in spring and summer. Feeding occurs colonially in clusters up to about 20 to 25 inches (75 to 100 mm) long around branches or leaders of the trees (Figure 197). In the fall, both male and female winged forms are produced, mating takes place, eggs are deposited, and the annual cycle is completed.

Diagnostics: Colonies of aphids encircling branches or the leader, the presence of unsightly black, sooty mold, and needle discoloration or branch flagging distal to the point of feeding are all evidence of attack by this insect. Ants frequently attend the aphids, feeding on the honeydew produced.

COOLEY SPRUCE GALL ADELGID

Description of injury: On spruces, the Cooley spruce gall adelgid causes swelling of the stem at the bases of needles on new shoots, giving rise to green or purplish galls (Figure 198). By late fall, the foliage and gall tissue turn reddish brown (Figure 199), and cavities appear where the tissues separate to allow the insects to emerge. Galls persist on trees for a year or more.

On Douglas-fir, this insect causes twisted needles with chlorotic spots but no galls.

Douglas-fir trees infested with large populations take on a yellowish green cast and appear unthrifty and chlorotic. A fairly distinct bend, caused by insertion of the sucking mouthparts of the nymphs, exists at some point along each twisted needle.

Figure 197

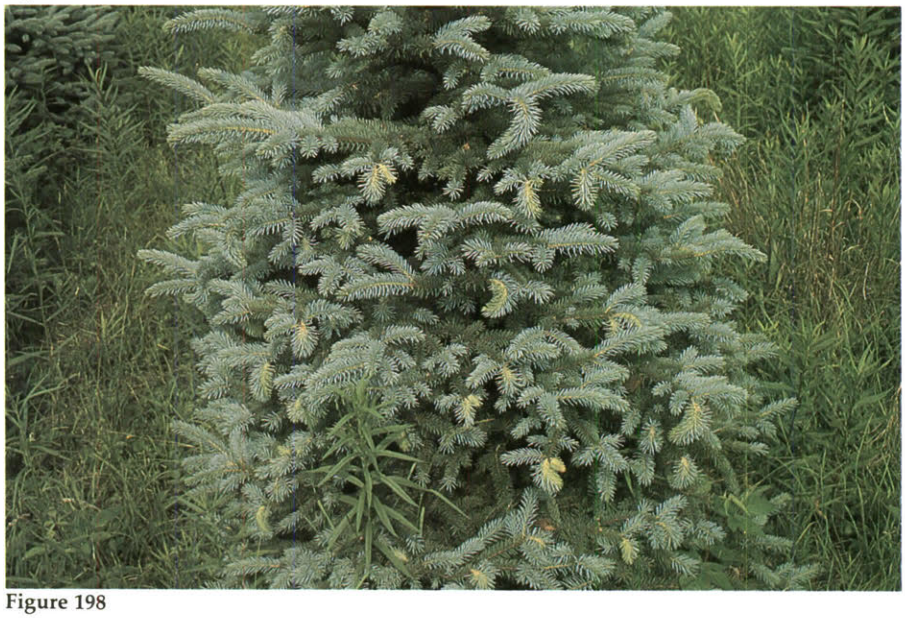

Figure 198

Figure 197. White pine aphid feeding colonially on shoot of eastern white pine.

Figure 198. Green (spring) galls on Colorado blue spruce; caused by the Cooley spruce gall adelgid.

Hosts: In the East, white, Norway, and several ornamental spruces are important hosts; Douglas-fir is less important.

Range and site description: This insect occurs coast to coast in the northern United States and throughout the range of white spruce in Canada.

Insect: The Cooley spruce gall adelgid, *Adelges cooleyi*, has six different morphological forms, depending on time of year and host. The young crawlers, which are very tiny, may be seen in early spring, clustering around the buds on spruce at about the time the bud cap cracks. During most of the rest of their development on spruce, the insects are cryptic.

Diagnostics: This insect is easily identified on spruces by unsightly reddish brown galls and killed shoots while needles are still present. On Douglas-fir, individual twisted needles are not especially noticeable, except in heavily infested trees.

SPRUCE SPIDER MITE

Description of injury: Mites, although not insects, are closely related arthropods; some species cause injuries similar to those caused by insects. The spruce spider mite is probably the most destructive conifer-feeding mite; it feeds by inserting its stylet-like mouthparts into a needle and extracting the contents of the cells, causing needle chlorosis. Under less severe feeding, foliage appears mottled or bleached (Figure 200). Resultant damage may be

Figure 199

Figure 200

Figure 199. Brown (fall) galls on Colorado blue spruce; caused by the Cooley spruce gall adelgid.

Figure 200. Bleached appearance of spruce foliage heavily infested with spruce spider mites.

restricted to a slight reduction in radial and longitudinal growth for the year. In severe infestations, needles may become enwebbed in the fine silk spun by the mites as they move about (Figure 201). Affected needles may become entirely bronzed and drop off prematurely, tree growth may be greatly retarded, and, occasionally, trees may die. Spruce spider mite populations build up most rapidly during hot, dry seasons.

Hosts: The spruce spider mite has a wide range of coniferous hosts in the United States, including many species of spruce, fir, and pine. Other genera that have been attacked by the mite include juniper, cypress, cedar, Port-Orford-cedar, incense-cedar, and sequoia.

Range and site description: The spruce spider mite is found throughout North America. Major infestations may occur on ornamentals and in nurseries, plantations, or natural stands.

Insect: The spruce spider mite, *Oligonychus ununguis,* deposits pale green eggs that turn amber shortly after deposition. They are small, globular, somewhat flattened, and have a distinct central seta. Nymphs and adults have four pairs of legs. Adults vary from dark green to black and have a pale streak extending along the middorsal line (Figure 202).

Diagnostics: Individual spider mites are difficult to observe with the naked eye. A reliable means of detection is to sharply jar a branch over a piece of white paper and watch for tiny crawling objects. A hand lens is useful.

Figure 201. Silken web of the spruce spider mite containing frass and needle debris and surrounding new buds.

Figure 202. Spruce spider mite adult and egg at base of needle. Note small size in comparison to needle width.

The presence of mites can usually be detected by looking for the fine, dust-coated webbing of the insect and for shed needles and similar debris.

Since injury symptoms range from flecking, stippling, or bleaching to total foliage bronzing, needle drop, and tree death, the spider mite injury or damage could be mistaken for something else, especially pollution injury. Suspected foliage should always be examined for one of the spruce spider mite's life stages and for silken webbing. Clear to slightly opaque egg cases are detectable with the aid of a hand lens.

THRIPS: SHORT-NEEDLE OF SCOTS PINE

Description of injury: Short-needle injury as caused by thrips is characterized by a stunting of one or both needles in some fascicles; needles in other fascicles on the same shoot elongate normally (Figure 203). This stunting appears to be caused by the probing of insects into the meristematic area at the base of the needle just before or during needle elongation (Figure 204). Injured needles fail to elongate fully. Heavy infestations render the trees ragged-looking.

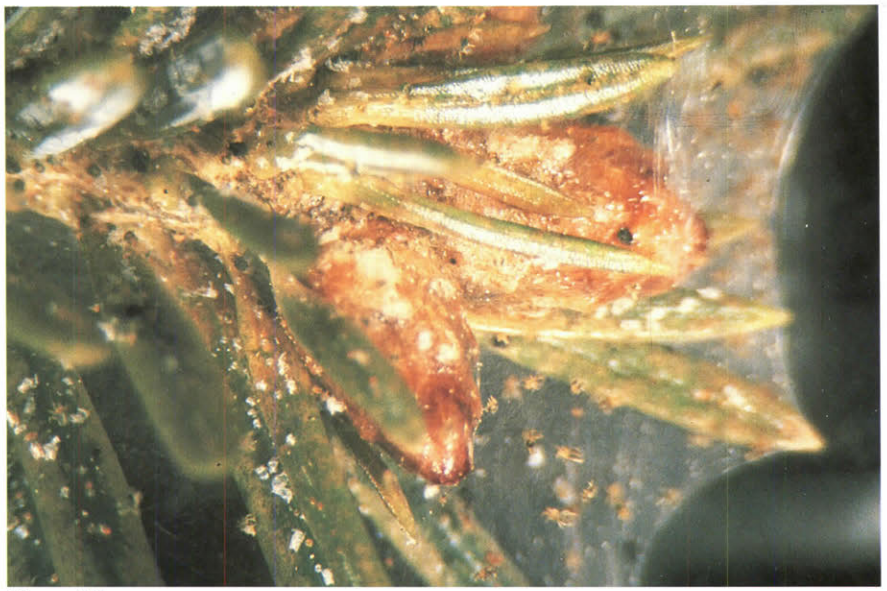

Figure 201

Figure 202

Hosts: Hosts include Scots and Austrian pine and, occasionally, eastern white pine.

Range and site description: Thrips-induced short-needle has been found throughout the mid-Atlantic states and perhaps elsewhere.

Insect: Thrips are in the order *Thysanoptera.* Adult thrips are black, have two pairs of long, narrow wings fringed with hairs, and are usually less than ¼ inch (6 mm) long. The tiny nymphs are active, slender insects that usually hide themselves in protected places, such as between needles in the fascicle. Nymphs resemble adults in shape but lack wings and are orange-yellow.

Diagnostics: Look for needles of different length in the same needle fascicle. With a hand lens, look for injury at the base of needles.

PINE SPITTLEBUG

Description of injury: This insect is a sapfeeder on young shoots and twigs. Heavy infestations may lead to main stem feeding on young trees. Initial injury is difficult to detect and consists only of slight discoloration of fed-upon bark tissues and necrotic spots in the underlying wood. The presence of spittle, evident as frothy masses at bases of needles (Figure 205) or along stems (Figure 206), is the best evidence of insect attack. Prolonged feeding activity will cause shoots and needles to turn red and die. Young trees, especially those in plantations, or

Figure 203

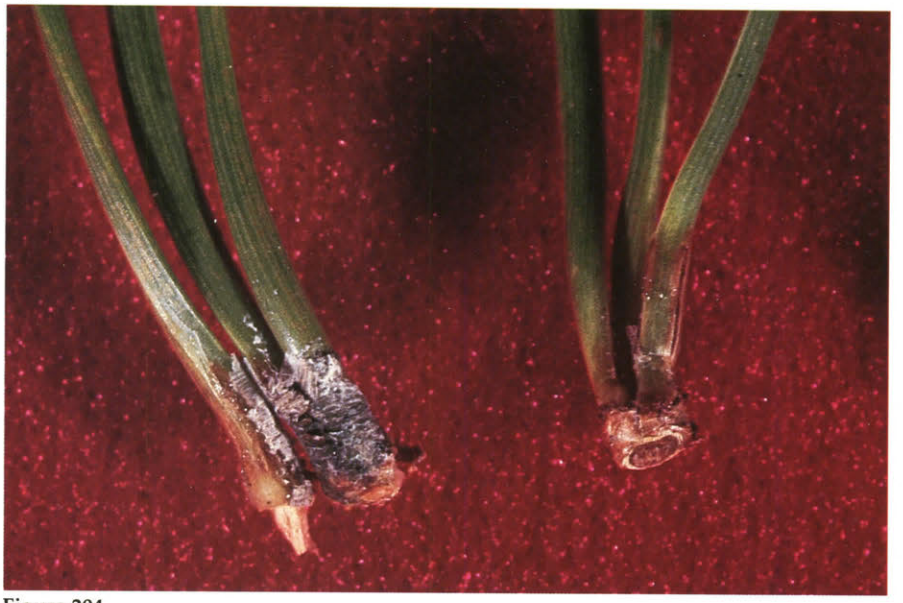

Figure 204

Figure 205

Figure 203. Short-needle of Scots pine; caused by thrips feeding at needle bases.

Figure 204. Feeding injury at needle base of Scots pine; caused by thrips.

Figure 205. Spittle mass caused by pine spittlebug feeding at the base of needles on Scots pine.

trees in stagnating stands may be weakened; they may die in one to three years if attack continues.

Hosts: Scots pine is most susceptible, but eastern white pine and jack pine also serve as hosts. The insect may be found on numerous other pines, balsam fir, hemlock, and spruce species.

Range and site description: The spittlebug may be found throughout the range of its hosts in the eastern United States. The insect does not appear to be site specific but has been found to be damaging in pole-sized Scots pine plantings and Christmas tree plantations.

Insect: The pine spittlebug is *Aphrophora parallela*. Eggs are laid in dead woody tissue or under the bark of shoots in summer and serve as the overwintering stage. Nymphs emerge in the spring and are black in front and orange to the rear. They are enveloped in spittle as they feed. Adults are gray to brownish black, boat-shaped, and about ⅓ to ½ inch (8 to 11 mm) in length. They respond to touch by jumping.

Diagnostics: The presence of spittle as a frothy mass, the insect itself, and reddened twigs and branches are all evidence of insect attack. Most nymphal feeding takes place on growth of the previous year.

Figure 206

Figure 206. Spittle mass surrounding twig of Scots pine as a result of feeding by the pine spittlebug.

Figure 207. Resin exudation and feeding punctures caused by an adult white pine weevil feeding on an eastern white pine terminal.

Figure 208. Mortality of terminal shoot of eastern white pine; caused by the white pine weevil.

Figure 209. Mortality of leader of spruce; caused by white pine weevil.

Meristematic Insects

WHITE PINE WEEVIL

Description of injury: Adults of the white pine weevil feed on succulent terminal portions of branches in the fall and on elongating terminals and leaders in the spring, often causing small flows of resin (Figure 207). Eggs are laid in the spring of the year in feeding cavities excavated in leaders. Larvae hatch, form a feeding ring encircling the leader under the bark, and mine their way downward. This feeding activity destroys the cambium and commonly kills the last two to four years of leader growth (Figures 208, 209). Dead leaders break out from the trees. In late summer, larvae excavate chambers in the woody tissue or in the pith, where they pupate and from which they emerge as adults in the fall of the year. The insect not only destroys the leader but also causes forked tops because the damage extends back into stem tissue that is several years old.

Hosts: In the eastern United States, white pine weevil may attack eastern white pine, jack pine, Scots pine, red pine, red spruce, black spruce, and Norway spruce.

Range and site description: This insect is found over much of eastern North America, throughout the range of eastern white pine. Trees ranging from 4 to 25 feet (1 to 8 m) are particularly susceptible to attack. Attacks may be much higher in older trees.

Insect: Grublike larvae of the white pine weevil, *Pissodes strobi*, mine in the cambial area in early summer; larvae or pupae may be found in fibrous cocoons within the leader in late summer. Adult weevils may be seen occasionally in the fall feeding on small twigs or more easily on elongating candles in the spring (Figure 207).

Diagnostics: Look for small, glistening beads of resin on leaders in early spring or on laterals in fall; these beads are caused by adult feeding. In early to midsummer, the needles on currently infested leaders wilt and then turn red or brown; leaders may exhibit drooping crooks (Figures 208, 209). Dead leaders and forked tops, or boles with crooks, signify prior attacks. After adults emerge in early fall, round exit holes are visible in the killed portion of the leader.

Figure 207

Figure 208

Figure 209

Description of injury: Weevils feed on the inner bark of twigs, branches, and main stems of small trees. Especially in the South, infested branches start fading in late winter, and, by spring, foliage on damaged branches is red. The needles have a scorched appearance, particularly in the topmost branches on the tree. Small trees may be killed.

Hosts: Most pines, including Scots, white, Austrian, and southern, and true cedars are hosts.

Range and site description: These weevils are found throughout the eastern and southern United States.

Insect: Adults of *Pissodes* spp. are red to grayish brown and about 1/6 to 1/3 inch (4 to 8 mm) long (Figure 210). The larvae are legless and appear as white grubs that feed in the phloem and xylem.

Diagnostics: On recently attacked trees, feeding scars are evident on the bark of twigs and small branches. These feeding wounds, in combination with fading foliage, are characteristic of weeviled trees (Figure 211). Females lay eggs in the holes made while feeding. The larvae feed in the inner bark — which often swells over the feeding sites — and pupate in chip cocoons, which are shallow depressions or chambers in the sapwood with a layer of long, yellowish white wood fibers on top. Some *Pissodes* species attack stumps or logs. Chip cocoons in dead twigs are good indicators of the presence of these weevils (Figure 212).

Figure 210

Figure 211

Figure 210. Northern pine weevil adult on stem of Scots pine.

Figure 211. Feeding wounds and scars on pine; caused by *Pissodes* weevils.

Description of injury: The most visible symptom of injury is yellow-green to reddish brown foliage (Figure 213). Over a period of weeks, foliage on pine trees fades from green to red, and the dead needles eventually fall from the trees. Secondary signs of injury include the presence of creamy white to reddish brown pitch tubes on the bark and reddish brown boring dust in bark crevices. Occasionally, only the upper portion of the crown of the tree is killed, leaving branches with green foliage at the base of the crown. Within stands, trees are usually killed in groups, although single tree kills are not uncommon.

Hosts: Most pines, including all southern pines, may be attacked.

Range and site description: These insects may be found wherever southern pines grow. Outbreaks of engraver beetles are often associated with prolonged periods of drought. Stand disturbance, such as lightning, fire, or logging damage, can trigger outbreaks of this pest.

Insect: Adult engraver beetles, *Ips* spp., are dark red to brown and about 1/5 inch (5 mm) long. The posterior of the adult beetle has a scooped-out appearance; up to six spines are on each side of the declivity. The creamy white, legless larvae are found in the inner bark of the tree. Pupation also takes place in the bark.

Figure 212

Figure 213

Figure 212. Chip cocoon formed by mature *Pissodes approximatus* weevil larva.

Figure 213. Brown foliage of pine attacked by *Ips* engraver beetles.

Diagnostics: The presence of these engraver beetles is indicated by fading foliage in combination with pitch tubes or boring dust. *Ips* engraver beetles can usually be distinguished from other bark beetles by the presence of Y- or H-shaped egg galleries that parallel the wood grain (Figure 214), in contrast to the meandering egg galleries excavated by other tree-killing bark beetles. Fading of the tree crown is a common symptom caused by all species of bark and engraver beetles.

Figure 214

SOUTHERN PINE BARK BEETLE

Description of injury: Because the southern pine bark beetle develops so quickly, new attacks and infestations occur continuously over periods of months. Attacks often occur in mature and overmature stands on poor sites; trees under stress are also at risk. Large numbers of beetles attack and overcome individual trees, causing large numbers of pitch tubes on the boles (Figure 215). Once trees have been girdled by the construction of meandering, S-shaped oviposition galleries or through the larval feeding activities (Figure 216), their foliage fades from green through yellow to reddish brown, and they die. Trees are typically attacked and killed in patches (Figure 217). Beetles move to adjacent trees after adults emerge.

Figure 215

Hosts: Shortleaf, slash, and loblolly pine are most commonly attacked, but all species of pine within the range of the insect are susceptible, especially under outbreak conditions. Longleaf pine is seldom attacked.

Figure 216

Figure 214. Galleries of *Ips avulsus* beetle larvae and pupal cells in phloem.

Figure 215. Crystallized resin "popcorn" on trunk of pine recently attacked by the southern pine beetle.

Figure 216. Typical sinuous galleries of the southern pine beetle in phloem/cambial area of loblolly pine.

Figure 217

Range and site description: Southern pine bark beetle occurs in the eastern United States, from Delaware south to Florida and west to Texas. Stands with high basal areas, overstocked or slowly growing stands, and stands on poorly drained lowland sites are most susceptible to attack.

Insect: Adults of the southern pine bark beetle, *Dendroctonus frontalis*, are brownish black, less than 1/6 inch (4 mm) long, and, unlike the *Ips* species, have no elytral depression (Figure 218). Larvae are small, off-white, legless grubs with distinct, darkened head capsules. The adults enter the bole of the tree to construct sinuous, S-shaped egg galleries in the phloem-cambium area and to lay eggs in niches along the walls of these galleries. As they feed, larvae mine away from the egg gallery, entering the bark. Adults emerge through small, round holes in the bark.

Figure 218

Figure 217. Stand of loblolly pine killed by the southern pine beetle.

Figure 218. Adult southern pine beetle showing convex posterior.

Diagnostics: Pitch tubes, fine, reddish, powdery frass around the base of a tree and in bark crevices, and small, round emergence holes in the bark are all signs of the southern pine bark beetle. Egg galleries are characteristically serpentine; staining fungi may be evident in the associated wood. Foliage fades from green through yellow to reddish brown as the tree dies. Dead trees are normally found in patches, which may coalesce during major outbreaks.

SPRUCE BEETLE

Description of injury: Spruce beetle first attacks the lower third of the bole above the stump. It may eventually attack the entire bole, except for the upper part under 8 inches (20 cm) in diameter. Limbs are not infested. Beetles attack slowly, continuously, and without major swarming. The attack lasts from a few days to many months, depending on beetle population density, host resistance, and environmental conditions. Feeding eventually causes the foliage to wilt and turn brown (Figure 219). Resin flow and pitch tubes can be seen at the adult entrance holes. Portions of attacked trees may die, while other portions remain green for several months; death may occur one year or more after attack.

Egg galleries are almost straight, parallel to the grain, and often packed with frass (Figure 220). Near entrance holes, the galleries are often hooked diagonally to the left or right for a short distance. After eggs hatch, the larvae feed communally for a while, then burrow lateral, independent galleries that may cross each other.

Figure 219

Figure 220

Figure 219. Thin, dying crowns of red spruce; caused by the spruce beetle, *Dendroctonus rufipennis*.

Figure 220. Typical straight- and parallel-to-the-grain gallery of spruce beetle under the bark of red spruce.

Hosts: All species of spruce within range of the insect may serve as hosts.

Range and site description: Spruce beetle is found from Alaska to Newfoundland and south to Arizona, New Mexico, Michigan, and Pennsylvania. Spruce beetle prefers to attack mature and overmature stands, along with freshly cut logs and shaded slash. It also invades weakened standing or fallen green trees, usually only if they are more than 8 inches (20 cm) DBH. During major outbreaks, any tree in the stand is susceptible to attack.

Insect: Adults of the spruce beetle, *Dendroctonus rufipennis*, are about ⅕ inch (5 mm) long and dark brown; they have reddish brown elytra that darken with age. Larval galleries tend to coalesce early in development. Larvae are legless, white, slightly C-shaped, and have a distinct, dark head capsule.

Diagnostics: Feeding beetles loosen the bark, and feeding galleries are readily discernable under the bark. Reddened top foliage and resin masses at the base of the tree are other diagnostic features.

Common and Scientific Names

FOREST SPECIES

ALDER, HAZEL *Alnus serrulata* (Ait.) Willd.

APPLE, COMMON *Malus pumila* Mill.

ASH, GREEN *Fraxinus pennsylvanica* Marsh.

ASH, WHITE *Fraxinus americana* L.

ASPEN, BIGTOOTH *Populus grandidentata* Michx.

ASPEN, QUAKING (TREMBLING) *Populus tremuloides* Michx.

ASTER *Aster* spp.

BASSWOOD *Tilia americana* L.

BEARBERRY *Uva-ursi uva-ursi* (L.) Britton

BEECH, AMERICAN *Fagus grandifolia* Ehr.

BIRCH, EUROPEAN WHITE *Betula pendula* Roth

BIRCH, GRAY *Betula populifolia* Marsh.

BIRCH, PAPER (WHITE) *Betula papyrifera* Marsh.

BIRCH, RIVER *Betula nigra* L.

BIRCH, SWEET (BLACK, CHERRY) *Betula lenta* L.

BIRCH, YELLOW *Betula alleghaniensis* Britt.

BLACKBERRY *Rubus alleghaniensis* Porter

BLACKGUM *Nyssa sylvatica* Marsh.

BLUEBERRY *Vaccinium* spp.

BOXELDER *Acer negundo* L.

CATBRIER *Smilax glauca* Walt.

CHERRY, BLACK *Prunus serotina* Ehrh.

CHESTNUT, AMERICAN *Castanea dentata* (Marsh.) Borkh.

CHESTNUT, JAPANESE *Castanea crenata* Sieb. & Zucc.

CHICKWEED, MOUSE-EAR *Cerastium viscosum* L.

CHINKAPIN, ALLEGHENY *Castanea pumila* Mill.

COTTONWOOD, BLACK *Populus trichocarpa* T. & G.

CURRANT *Ribes* spp.

DOGWOOD, FLOWERING *Cornus florida* L.

DOUGLAS-FIR *Pseudotsuga menziesii* (Mirb.) Franco

ELM *Ulmus* spp.

FALSE-TOAD-FLAX *Comandra umbellata* (L.) Nutt.

FERN, BRACKEN *Pteridium aquilinum* (L.) Kuhn.

FIR, BALSAM *Abies balsamea* (L.) Mill.

FIR, BRACTED BALSAM *Abies balsamea* var. *phanerolepis* Fern.

FIR, FRASER *Abies fraseri* (Pursh) Poir.

FIR, GRAND *Abies grandis* Lindl.

FIR, NOBLE *Abies procera* Rehd.

FIR, SILVER *Albies alba* Miller

FIR, SUBALPINE *Abies lasiocarpa* (Hook.) Nutt.

FIR, WHITE (CONCOLOR) *Abies concolor* Lindl. & Gord.

GOLDENROD *Solidago* spp.

GOOSEBERRY *Grossularia* spp.

GRAPE, MUSCADINE *Vitis rotundifolia* Michx.

GRAPE, NORTHERN FOX *Vitis labrusca* L.

GREENBRIER *Smilax rotundifolia* L.

HAWTHORN *Crataegus* spp.

HEMLOCK, EASTERN *Tsuga canadensis* Carr.

HICKORY *Carya* spp.

HONEYLOCUST *Gleditsia triacanthos* L.

HORSECHESTNUT *Aesculus hippocastanum* L.

INCENSE-CEDAR *Libocedrus decurrens* Torr.

LABRADOR TEA *Ledum groenlandicum* Oeder.

LARCH, EASTERN (TAMARACK) *Larix laricina* (DuRoi) K. Koch

LARCH, EUROPEAN *Larix decidua* Mill.

LOCUST, BLACK *Robinia pseudoacacia* L.

MAPLE, RED *Acer rubrum* L.

MAPLE, SILVER *Acer saccharinum* L.

MAPLE, SUGAR *Acer saccharum* Marsh.

MILKWEED, COMMON *Asclepias syriaca* L.

OAK, BLACK *Quercus velutina* Lam.

OAK, CHESTNUT *Quercus prinus* L.

OAK, PIN *Quercus palustris* Muench.

OAK, POST *Quercus stellata* Wangenh.

OAK, NORTHERN RED *Quercus rubra* L. Wangenh.

OAK, SCARLET *Quercus coccinea* Muench.

OAK, SOUTHERN RED *Quercus falcata* Michx.

OAK, SWAMP WHITE *Quercus bicolor* Willd.

OAK, WATER *Quercus nigra* L.

OAK, WHITE *Quercus alba* L.

OAK, WILLOW *Quercus phellos* L.

PEACH *Prunus persica* Batsch

PINE, AUSTRIAN *Pinus nigra* Arnold

PINE, EASTERN WHITE *Pinus strobus* L.

PINE, JACK *Pinus banksiana* Lamb.

PINE, LOBLOLLY *Pinus taeda* L.

PINE, LONGLEAF *Pinus palustris* Mill.

PINE, MUGO *Pinus mugo* Turra.

PINE, PITCH *Pinus rigida* Mill.

PINE, POND *Pinus serotina* Michx.

PINE, PONDEROSA *Pinus ponderosa* Laws.

PINE, RED *Pinus resinosa* Ait.

PINE, SAND *Pinus clausa* (Chapm.) Vasey

PINE, SCOTS *Pinus sylvestris* L.

PINE, SHORTLEAF *Pinus echinata* Mill.

PINE, SLASH *Pinus elliottii* Engelm.

PINE, TABLE-MOUNTAIN *Pinus pungens* Lamb.

PINE, VIRGINIA *Pinus virginiana* Mill.

POISON-IVY *Toxicodendron radicans* (L.) Kuntze.

POPLAR *Populus* spp.

PORT-ORFORD-CEDAR *Chamaecyparis lawsoniana* (A. Murr.) Parl.

RAGWEED, GIANT *Ambrosia trifida* L.

RASPBERRY *Rubus occidentalis* L.

REDBUD, EASTERN *Cercis canadensis* L.

SARSAPARILLA *Aralia nudicaulis* L.

SASSAFRAS *Sassafras albidum* (Nutt.) Nees

SEQUOIA *Sequoia gigantea* (Lindl.) Decne.

SPRUCE, BLACK *Picea mariana* (Mill.) B.S.P.

SPRUCE, BLUE *Picea pungens* Engelm.

SPRUCE, NORWAY *Picea abies* (L.) Karst.

SPRUCE, RED *Picea rubens* Sarg.

SPRUCE, WHITE *Picea glauca* (Moench) Voss

SUMAC *Rhus* spp.

SWEETGUM *Liquidambar styraciflua* L.

SYCAMORE, AMERICAN *Platanus occidentalis* L.

TANOAK *Lithocarpus densiflorus* (Hook & Arn.) Rehd.

TREE-OF-HEAVEN *Ailanthus altissima* (Mill.) Swingle

TUPELO, WATER *Nyssa aquatica* L.

WALNUT, BLACK *Juglans nigra* L.

WITCH-HAZEL *Hamamelis virginiana* L.

YELLOW-POPLAR *Liriodendron tulipifera* L.

BIOINDICATOR SPECIES

Ozone

ASH, WHITE *Fraxinus americana* L.

BLACKBERRY *Rubus alleghaniensis* Porter

CHERRY, BLACK *Prunus serotina* Ehrh.

DOGWOOD, FLOWERING *Cornus florida* L.

GRAPE, NORTHERN FOX *Vitis labrusca* L.

MILKWEED, COMMON *Asclepias syriaca* L.

PINE, EASTERN WHITE *Pinus strobus* L.

SASSAFRAS *Sassafras albidum* (Nutt.) Nees

YELLOW-POPLAR *Liriodendron tulipifera* L.

Sulfur Dioxide

ASH, GREEN *Fraxinus pennsylvanica* Marsh.

ASH, WHITE *Fraxinus americana* L.

ASPEN, BIGTOOTH *Populus grandidentata* Michx.

ASPEN, QUAKING (TREMBLING) *Populus tremuloides* Michx.

BIRCH, GRAY *Betula populifolia* Marsh.

BIRCH, PAPER (WHITE) *Betula papyrifera* Marsh.

BIRCH, RIVER *Betula nigra* L.

BIRCH, SWEET (BLACK, CHERRY) *Betula lenta* L.

BIRCH, YELLOW *Betula alleghaniensis* Britt.

BLACKBERRY *Rubus alleghaniensis* Porter

PINE, EASTERN WHITE *Pinus strobus* L.

PINE, JACK *Pinus banksiana* Lamb.

RAGWEED, GIANT *Ambrosia trifida* L.

RASPBERRY *Rubus occidentalis* L.

SARSAPARILLA *Aralia nudicaulis* L.

SUMAC, STAGHORN *Rhus typhina* L.

Fluorides

BLUEBERRY *Vaccinum* spp.

BOXELDER *Acer negundo* L.

CATBRIER *Smilax glauca* Walt.

GLADIOLUS *Gladiolus* spp.

GRAPE *Vitis* spp.

GREENBRIER *Smilax rotundifolia* L.

MAPLE *Acer* spp.

PEACH *Prunus persica* Batsch

PINE, EASTERN WHITE *Pinus strobus* L.

SPRUCE, BLUE *Picea pungens* Engelm.

Glossary

ABDOMEN - The third or posterior major division of the insect body; consists normally of nine or ten apparent segments; bears no functional legs in the adult stage, but may bear prolegs or false legs in the larval stage.

ABIOTIC PATHOGEN - A nonliving, disease-causing entity, e.g., drought, salt, air pollutants.

ACUTE INJURY - Injury, usually involving necrosis, which develops within several hours to a few days after a high dose exposure to a pollutant; expressed as fleck, scorch, bifacial necrosis, etc.

AECIAL STAGE (aecium) - A spore stage of the rust fungi; a cuplike structure bearing aeciospores.

ALTERNATE HOST - One of two taxonomically different hosts required by a heteroecious rust fungus to complete its cycle; also applies to some insects.

ANTERIOR - In front; before; opposite of posterior.

ANTHOCYANOSIS - Presence of abnormal red-purple coloration in foliage.

ASEXUAL STAGE - Vegetative; without sexual organs or spores.

AUTOECIOUS - Completing entire life cycle on one host; especially applied to the rust fungi.

BANDING - A foliar symptom characterized by a limited zone of necrotic or discolored tissue traversing the leaf, e.g., the band of tissue on a pine needle injured by SO_2 or O_3.

BASIDIAL STAGE (basidium) - A spore stage of the rust fungi; a specialized structure in the Basidiomycetes bearing basidiospores.

BASIDIOCARPS - Sexual fruiting structure in the Basidiomycetes; conks, sporophore, mushrooms, etc.

BIFACIAL NECROSIS - Death of plant tissues, extending from the upper to the lower leaf surface.

BIOINDICATOR SPECIES - Species, varieties, or cultivars sufficiently sensitive to a specific pollutant to be useful as indicators for the presence of that pollutant.

BIOTIC PATHOGEN - A living organism capable of inciting disease, e.g., fungi, bacteria, viruses, etc.

BLEACHING - Loss of normal color, tending toward white, cream, or tan coloration.

BLIGHT - A common term for several different diseases; usually applied to those where leaf damage is sudden and severe.

BRONZING - A golden brown discoloration that usually appears on the lower surface of leaves and is often an advanced stage of the silvering or glazing typical of injury by PAN and other oxidants; brown coloration on needles due to spider mite infestation.

CALLUS TISSUE - A protective tissue of thin-walled cells developed on wound surfaces, often beginning at the edges of a wound.

CANKER - A plant disease symptom characterized by a sharply defined necrosis of cortical tissue, often sunken below bark surface.

CHEWING INSECTS - Insects that consume all tissues of leaves or portions of leaves, using robust mandibles for chewing.

CHLOROSIS - Yellowing of plant tissue due to failure of chlorophyll synthesis or to chlorophyll destruction.

CHLOROTIC DWARF - An abiotic disease of *Pinus strobus* characterized by reduced growth, chlorosis and mottling of the needles, and premature abscission of all but current needles.

CHRONIC INJURY - Injury which develops after long-term or repeated low dose exposure to an air pollutant; expressed as chlorosis, bronzing, premature senescence, reduced growth, etc.

CLONAL LINES - A group of plants originating from buds or cuttings from the same individual.

CONK - The fruiting structure of a wood-rotting fungus, especially of one of the Polyporaceae.

COCOON - A covering, composed partly or wholly of silk or other sticky fiber, spun or constructed by many larvae as a protection for the pupal stage.

CORNICLES - The posterior dorsal erect or semi-erect tubules of aphids which secrete a waxy defensive liquid to protect the insect against enemies; short, blunt horns or rounded projections occurring on the abdomen.

CRAWLER - The first instar motile nymphal stage of scale insects and mealybugs, which moves to a new feeding site before settling down to a sessile existence for the rest of its developmental life.

CRINKLING - Bending or twisting of foliage without breaking; wrinkling.

CRYPTIC - Hidden or concealed.

DEFOLIATOR - Any chewing insect that consumes the leaves or needles of plants.

DELIQUESCENT - To ramify into fine divisions, such as abnormal numbers of buds, twigs, branches, or leaves, e.g., witches' broom development.

DORSAL - Of or belonging to the upper surface; top.

DORSOVENTRAL - From top to bottom; from the upper surface to the lower surface.

DORSUM - The upper surface; top.

DOSE - A measured concentration of a toxicant for a known duration of time (concentration per unit time).

EGG GALLERY - A long, narrow tunnel along the sides of which eggs are deposited in small niches; the pattern of construction is often diagnostic of a particular species of insect.

EGG MASS - Cluster of eggs, usually in a matrix of body hairs or wing scales from the female adult and/or a mucilaginous cementing secretion.

ELYTRA - The anterior leathery or chitinous wings of beetles and leafhoppers; serve as coverings to the hind wings and commonly meet at rest in a straight line down the middle of the dorsum.

ENGRAVER - Beetle which feeds in the phloem-cambium region of woody plants, often scoring or engraving adjacent sapwood tissues.

EPICORMIC BRANCHING - Branches arising from buds in bark along mainstem, most commonly occurring in trees under crown stress; also called watersprouts.

EPIDEMIC - A change, usually a sudden increase, in a disease within a population.

FOREWINGS - Front pair of wings.

FLECK - White to tan necrotic lesions up to a few millimeters in length or diameter, usually confined to the upper surface of leaves.

FRASS - Solid larval insect excrement; mixed with wood fragments in wood-boring or bark-boring insects.

FRUITING BODY - A specialized structure, often macroscopic, on or in which spores are produced.

FUMIGATION - The natural or controlled exposure of plants to toxic gases or volatile substances.

GALL - A swelling or outgrowth of tissue induced by a pathogen or insect on a plant.

GENERATION - The successive developmental stages from reproduction to reproduction, e.g., egg, larva, pupa, adult.

GIRDLING - Destruction of tissue in a ring around a twig, branch, or stem.

GOUT - Formation of swellings at nodes or at the base of buds.

GREGARIOUS - Living in groups or communities.

GRUB - An insect larva; a term loosely applied, usually to larvae of Coleoptera; larva is thick-bodied with well-developed thoracic legs but no abdominal prolegs.

HETEROECIOUS - Requiring two taxonomically different hosts to be able to complete the entire life cycle, as in the rust fungi.

HONEYDEW - A sweetish excretion produced through the anus by certain insects, notably aphids and scale insects.

HOST - A living organism serving as a food source for a parasite.

HYSTEROTHECIUM - A sexual fruiting structure of the Ascomycete fungi, usually football-shaped or elongate in appearance and occurring on infected needles.

INSTAR - The insect itself during the time between molts in the larva or nymph, numbered to designate the various periods; i.e., the first instar is the insect between the egg and first molt.

INTERVEINAL - Between veins.

INTRAVEINAL - Associated along or within veins.

LARVA (plural = larvae) - The immature instars, between the egg and pupal stages, in an insect having a complete metamorphosis (egg, larva, pupa, adult). Larvae feed and grow but cannot fly, nor can they reproduce.

LESION - A wound; a well-marked, but limited, diseased area; a break or rupture through a tissue, especially a surface tissue.

LOOPER - A caterpillar in which some or all of the middle abdominal prolegs are wanting and which moves by placing the posterior part of the abdomen next to the thorax, forming a loop of the intervening segments, then extending the anterior part of the body forward.

MIDDORSAL - In the middle of the upper side or dorsum.

MIMICKING SYMPTOMS - Symptoms similar to those caused by pollutants but induced by other abiotic or biotic causal agents.

MINER - The larval stage of an insect which makes galleries or burrows between the upper and lower surfaces of leaf tissue.

MITE(S) - Small, often minute, arthropods in the order Acarina of the class Arachnida, which includes spiders, scorpions, and related forms. Mites have four pairs of legs vs. three pairs in insects.

MOLT - To cast off the outgrown skin or cuticle in the process of insect development; changing from one instar to the next.

MOTTLE - Irregular, diffuse patterns of chlorotic areas interspersed with normal green leaf tissue.

MOSAIC - A diseased condition where different portions of a leaf vary in amounts of chlorophyll, thus giving the leaf a mottled appearance; usually caused by viruses.

MYCELIUM - A mass or aggregate of hyphae; vegetative stage of fungi.

NAKED - Larva devoid of body hairs or setae; pupa not enclosed in a cocoon or other covering.

NECROSIS - Death.

NYMPH - The immature stage of insects, following hatching, which does not have a pupal stage, i.e., incomplete metamorphosis (egg, nymph, adult). Late instar nymphs may have nonfunctional rudimentary wings and/or genitalia.

OVIPOSITION - The act of depositing the eggs.

OVIPOSITOR - The egg-laying apparatus; the extended genitalia of a female insect.

PARASITE - An organism which lives on or in another living organism and obtains part or all of its nutrients from that other living organism.

PATHOGEN - Any agent of the environment capable of inciting disease.

PERITHECIUM - A sexual fruiting structure of the Ascomycetes with an opening called the ostiole at or near its top.

POSTERIOR - Hind or hindmost; opposite of anterior.

PREDISPOSITION - The weakening of an organism by some factor(s) of either the physical or biotic environment so as to render the organism more susceptible to a pathogen.

PROLEG - Any process or appendage that serves the purpose of a leg; specifically, the fleshy, unjointed, ventral abdominal projections of caterpillars and certain sawfly larvae.

PUPA - The resting, inactive, nonfeeding instar in all holometabolous insects; the stage intermediate between the larva and the adult.

PYCNIAL STAGE (pycnium) - A flask-shaped spore stage of the rust fungi; oozes out spores in a sticky matrix.

PYCNIDIUM - An open-pored, flask-shaped fruiting structure in which asexual spores called conidia are produced.

RASPING - A type of feeding by insects which rub or grate the leaf surfaces with their mouthparts to obtain particles for consumption.

RESINOSIS - Resin flow through bark or from wounds or cankers on conifers.

RHIZOMORPH - A thread- or cordlike fungal structure made up of hyphae.

RINGSHAKE - Peripheral cracks in woody tissues of stems. The pattern of damage is concentric with the annual rings.

RINGSPOT - A circular area of chlorosis with a green center.

SAPROPHYTE - An organism living on dead organic matter.

SCORCH - Appearing as if tissues were burned by heat; usually affecting marginal portions of leaves.

SENESCENCE - Aging of tissues; growing old.

SESSILE - Attached or fastened; incapable of moving from place to place.

SETA (plural = setae) - Slender, hairlike or bristly projections arising from the epidermal layer on any part of the body of an insect.

SEXUAL STAGE - Reproductive stage of the life cycle of an organism.

SHOTHOLES - Small holes in a leaf caused by feeding activity and giving the appearance of injury via a shotgun.

SIGN - The actual presence of the causal organism in association with the disease symptoms.

SKELETONIZER(S) - Insects which consume leaf tissue, often from the lower side of the leaf, leaving the upper epidermis and vascular tissues intact.

SPORE - A specialized structure consisting of one or few cells and serving any or all of the following three functions: (i) reproduction, (ii) dissemination, (iii) survival.

SPOROPHORE - A spore-producing or supporting structure.

STADIUM (plural = stadia) - The period of time between two successive molts.

STAGE - One of the successive principal divisions in the life cycle of an insect, e.g., egg, nymph, larva, prepupa, pupa, adult.

STAGHEAD - Death of limbs and main branches of a tree in the upper crown, giving the appearance of antlers.

STIPPLE - Pigmented spots up to a few millimeters in diameter, often on the upper surface of leaves.

STYLET - A small, stiff, needlelike tube inserted into a food source to obtain liquid food.

SUCKING INSECTS - Insects that insert their mouthparts into plant tissues and withdraw nutrients and fluids through stylets.

SUSCEPT - Any organism that can be attacked by a biotic pathogen.

SYMPTOM - Visible or measurable manifestation that an organism is diseased; a change in the organism itself.

TELIAL STAGE (telium) - A stage of the rust fungi; a fruiting structure usually appearing as fine, hairlike projections from lower surfaces of infected leaves.

THORACIC - Belonging or attached to the thorax.

THORAX - The body region behind the head, which bears wings and true (jointed) legs if present.

TUFT - Bunching of twigs or needles.

TYLOSIS - Outgrowth of a cell membrane from a ray or axial parenchyma cell through a pit in a xylem vessel wall into the vessel, partially or completely blocking the lumen of the vessel.

UREDIAL STAGE (uredium) - A stage of the rust fungi; a fruiting structure usually appearing as a pustule bearing the repeating spore stage that leads to disease increase.

WATER-SOAKED - A dull green coloration of diseased tissues due to membrane leaking of cellular contents into intercellular spaces.

WILT - A common symptom of disease due to a loss of turgor and resulting in subsequent drooping and collapse of the foliage or succulent tissues.

WITCHES' BROOM - A massed proliferation of the branches of a woody plant.

ZONE LINE - Narrow brown or black lines in decayed or decaying wood.

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