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Diseases of Trees in the Great Plains

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Abstract

Hosts, distribution, symptoms and signs, disease cycle, and management strategies are described for 84 hardwood and 32 conifer diseases in 56 chapters. Color illustrations are provided to aid in accurate diagnosis. A glossary of technical terms and indexes to hosts and pathogens also are included.

Keywords: Tree diseases, forest pathology, Great Plains, forest and tree health, windbreaks.

Cover photos by: James A. Walla (top left), Laurie J. Stepanek (top right), David Leatherman (middle left), Aaron D. Bergdahl (middle right), James T. Blodgett (bottom left) and Laurie J. Stepanek (bottom right).

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Background

This technical report provides a guide to assist arborists, landowners, woody plant pest management specialists, foresters, and plant pathologists in the diagnosis and control of tree diseases encountered in the Great Plains. It contains 56 chapters on tree diseases prepared by 27 authors, and emphasizes disease situations as observed in the 10 states of the Great Plains: Colorado, Kansas, Montana, Nebraska, New Mexico, North Dakota, Oklahoma, South Dakota, Texas, and Wyoming.

The need for an updated tree disease guide for the Great Plains has been recognized for some time and an account of the history of this publication is provided here. When the Pest Management Task Force of the Great Plains Agricultural Council, Forestry Committee, met in 1980, the task force requested that the committee support its publication of a tree disease handbook for the Great Plains, and assist it in obtaining the necessary funding. The request was approved.

During the task force's 1981 meeting, a working group was formed to prepare a prospectus for the handbook. The working group members, Edward Sharon, James Walla, Mark Harrell, and Jerry Riffle, chose specific diseases for inclusion in the handbook, selected potential authors for each chapter and determined chapter format to emphasize diagnosis, biology, damage, and management of diseases. In 1982, the Pest Management Task Force agreed that Riffle would serve as coordinator of the handbook, with Glenn Peterson as co-coordinator. Riffle contacted all potential contributors in May 1982, and their response was excellent; 31 persons agreed to be authors or coauthors of chapters on 64 diseases. The source of funding for publication of the handbook was resolved in 1985 when it was proposed that it be published as a general technical report by the U.S. Department of Agriculture (USDA) Forest Service, Rocky Mountain Research Station (RMRS).

This proposal was approved by the Executive Committee of the Great Plains Agricultural Council, Forestry Committee. The original *Diseases of Trees in the Great Plains*[1], published in 1986, has been an extremely popular publication used by a wide spectrum of natural resource professionals in the Great Plains and beyond.

During the Great Plains Tree Pest Council annual meeting in 2012, a discussion was initiated about updating the original publication to include new diseases and to reflect the current available knowledge about tree diseases in the Great Plains. Upon agreement to the update, a group was formed consisting of James Walla, William Jacobi, Mark Harrell, Judith O'Mara, John Ball, James Blodgett, Jared LeBoldus, Simeon Wright, Laurie Stepanek, Les Koch, Rachel Allison, Nicole Ricci, and Aaron Bergdahl to consider which new diseases to include and potential authors and reviewers. Letters of support for the effort were requested from the state foresters of the Great Plains states and subsequently approval of the update was given by RMRS Station Director Sam Foster. Possible funding sources were researched, and Michele Schoeneberger, from the USDA National Agroforestry Center, offered assistance and set aside funds for printing the publication. Later, Roy Mask of USFS Region 2 came forward with additional funding in 2015 and Gregg DeNitto and Marcus Jackson of Northern Region Forest Health Protection facilitated funding in 2016.

The chair of the Great Plains Tree Pest Council at that time, Aaron Bergdahl, and Alison Hill of the RMRS offered to organize the update effort and serve as technical editors. Work on the update began by requesting the contribution of 27 authors with forest pathology experience in the Great Plains. The response from the authors was very positive. The chapter content and technical reviews were completed in late 2013 and 2014. The updated publication, which is available in print and online versions, is the culmination of the cooperative efforts of numerous professionals dedicated to the health of forest resources in the Great Plains from several state and federal agencies and research institutions.

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[1] Riffle, Jerry W.; Peterson, Glenn W., tech. coords. 1986. Diseases of trees in the Great Plains. Gen. Tech. Rep. RM-129. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station. 149 p.

Dedication

This update of *Diseases of Trees in the Great Plains* is dedicated to the coordinators of the original manuscript, Jerry Riffle and Glenn Peterson. These two retired U.S. Forest Service researchers are to be commended for their many years of dedication and contributions to the understanding of tree diseases and health in the Great Plains region. Their passion for their field of study and Great Plains forest resources was embodied in the first edition of this valuable guide. The authors of this update have done their utmost to carry on the traditions of scientific inquiry and excellence personified by Riffle and Peterson in Great Plains forest pathology.

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Introduction

Scope

The purpose of this handbook is to assist users in diagnosing tree diseases and reducing their impact. Included are the most damaging diseases, along with some that are the subject of frequent inquiry although of minor importance. The diseases included were those encountered primarily on trees in conservation plantings such as windbreaks, farmstead plantings, and living snow fences and in landscape plantings in recreational areas and near rights-of-way. Additionally, diseases affecting tree nurseries and Christmas tree farms in the Great Plains are included. By association, there is great overlap with the most common diseases encountered in native woodlands and urban plantings.

Diseases of fruit and nut trees are included when they are encountered on trees that are used in the above types of plantings. Information on nutrient deficiencies is not included, except in the chapter on chlorosis. There is no extensive coverage of damage by airborne pollutants except that caused by herbicides. The chapters are grouped as hardwood diseases and conifer diseases; within each of these categories they are classified as to the part of plant affected: foliage, branch and stem, vascular wilt, and root and soilborne. Most of the diseases are treated separately in the following standard format: "Hosts and Distribution," "Symptoms and Signs," "Disease Cycle," "Damage," "Management," and "Selected References." Select chapters cover several similar diseases affecting a particular host or similar types of pathogens, as in the chapter on stem decays (chapter 30). The emphasis of this tree disease guide is on disease situations in the Great Plains. Accordingly, no special effort has been made to include the total distribution of pathogens, or to list all of their hosts. Similarly, "Symptoms and Signs" and "Disease Cycles" are described primarily as observed in the Great Plains.

Diagnosis

In making a diagnosis, the reader is referred first to the host index to see which diseases are listed for the tree host in question. Then the reader should check the writeup on the diseases for that host and also the photographs to see if they conform to the disease under consideration. A glossary of technical terms is included for a more complete understanding of the chapters. English units of measure are used for the most part; metric units are used primarily in descriptions of pathogens.

Management

Information on management (reducing damage) is provided. Where chemicals are suggested or recommended, the reader must determine if the material is currently registered for the intended use in the area of application. Most of the articles listed in "Selected References" were chosen because they will be useful in understanding the recommendations for reducing damage. Because information on the cycle of some diseases is limited, the recommendations for their control are in some cases quite general.

Damage from pathogens can often be reduced by increasing the vigor of trees. Thus, the "Management" section of several of the chapters in this guide includes recommendations for increasing vigor. Vigor of trees in the Great Plains usually can be increased by supplemental watering. Application of fertilizers sometimes can result in increased tree vigor; however, the wholesale application of fertilizers for increasing tree vigor is not recommended. Use of fertilizers should be based on evidence that the tree in question (considering species and age) will respond to fertilizers on the site involved.

History

The demand for trees in the Great Plains stems from early settlers' needs for orchards, woodlots, windbreaks, and hedges. Long before the Federal Timber Culture Act of 1873, individuals, companies, associations, and local governments had promoted tree planting in the Great Plains. Establishment of dry-land Agricultural Experiment Stations at Mandan, North Dakota; Woodward, Oklahoma; and Cheyenne, Wyoming; resulted in increased planting of trees by farmers and ranchers, who cooperated with these stations in tests of tree species, cultural methods, spacing of trees, and the number of rows needed

(Droze 1977). Their work aided the USDA's tree distribution program authorized by the Clarke-McNary Act, which began in 1924. This program, under the direction of the Extension Service of the USDA, encouraged tree planting in the Great Plains.

The biggest impetus to tree planting came, however, when President Franklin D. Roosevelt, by executive order in July 1934, authorized the Shelterbelt Project. This project, later called the Prairie States Forestry Project, operated between 1935 and 1942. It led to the planting of 223 million trees in 18,600 miles of field windbreaks. Tree planting for protection purposes continued after 1942 under the direction of the USDA Soil Conservation Service. Tree planting was not as extensive as during the Prairie States Forestry Project, but was fairly steady. The Soil Bank program of the 1960s resulted in an increase in plains tree planting as considerable land was taken out of crop production and planted to trees.

Rising energy costs spurred tree planting for many additional years, not only on farms and ranches but in communities as well. Trees were also planted along roads for "living snow fences." In more recent years, tree planting in communities has continued, but rural tree planting has declined. Furthermore, strong crop prices and larger farm machinery have led to the loss of shelterbelts, with farmers opting to remove older shelterbelts rather than renovating or replanting.

Although many pest problems were noted in the early plantings in the Great Plains, very little systematic study of tree diseases and insects was conducted, with the exception of diseases of seedlings. Research on diseases of tree seedlings in the Great Plains was initiated some 100 years ago by Carl Hartley and USDA colleagues. Their research resulted in a good understanding of damping-off disease of pines and other conifers and provided some information on other diseases of conifer seedlings.

Little additional tree disease research occurred in the Great Plains until the period of the Prairie States Forestry Project (1935–1942). With the expansion of existing nurseries and the establishment of many new nurseries to provide trees for that project, a number of disease problems were encountered, particularly on hardwood seedlings. Thus, during this period, Ernest Wright and USDA colleagues concentrated their research on disease problems of hardwood seedlings, with some work being done on plantation diseases such as Phymatotrichum root rot and Cytospora canker of cottonwood (*Populus deltoides*).

When the Prairie States Forestry Project was ended in 1942, tree disease research in the Great Plains by the USDA was discontinued. Research by land grant institutions at this time was minimal as well. Agricultural extension personnel in the Great Plains accumulated some information on tree diseases, but they had little time to devote to tree problems. Likewise, pest management specialists assigned to U.S. Forest Service regional offices devoted little time to tree disease problems in the Great Plains, other than those associated with federal nurseries and national monuments.

A turnaround came in 1958 when a plant pathologist position was established in the Lincoln Unit of the U.S. Forest Service's Rocky Mountain Forest and Range Experiment Station. Nursery diseases received attention first; diseases in pine and juniper plantings were emphasized later. With the hiring of another pathologist in 1972, research was expanded to include additional research on hardwood diseases and on mycorrhizae. Land grant institutions in North Dakota, South Dakota, Nebraska, Kansas, and Oklahoma expanded their research efforts and more extension plant pathologists were hired. U.S. Forest Service pest management specialists increased their activities in the Great Plains, and pest management specialists were assigned to several state forester offices. Additionally, The ARS added a tree disease research project to support a tree improvement project in the Northern Plains.

The increased effort on tree diseases prompted the Great Plains Agricultural Council, Forestry Committee to establish a Pest Management Task Force to increase communication among workers and to coordinate activities in research, pest management, and extension. This task force sponsored the original development of this handbook.

Since the publication of this guide in 1986, land grant institutions in the Great Plains have continued to support their capacity to study tree disease problems of concern, and federal programs have maintained their support of state forest health programs and research groups within the U.S. Forest Service. But because funding for tree disease

research and forest health programs has not increased at a rate sufficient to sustain the number of positions and programs that existed when the original version of this guide was published, the future state of forest pathology knowledge in the Great Plains is at the moment uncertain. Further, the number of personnel in the Great Plains focusing on diseases of trees is decreasing and fewer positions are being replaced after retirement. While this guide was being updated, three senior active members of the Great Plains Tree Pest Council and the greater forest pathology scientific community retired. With several more senior researchers scheduled to retire in the coming years, the future state of forest pathology knowledge in the Great Plains will diminish without a committed effort to maintain and rebuild well-funded programs and increase pathology expertise in the region. With respect to these current challenges, updating this disease guide at this time was critical to fully capitalize on the wealth of expertise held by senior members of the Great Plains Tree Pest Council.

Use and Safe Handling of Pesticides

Some states have restrictions on the use of certain pesticides. Check your state and local regulations. Also, because registrations of pesticides are under constant review by the U.S. Environmental Protection Agency, consult your state department of agriculture to be sure the intended use is still registered. The rules below should be followed when handling pesticides. These rules should be read by all persons involved in pesticide use. Copies of these rules should be posted in several places, particularly in the areas of pesticide storage.

Pesticides are poisonous and always should be used with caution. The dangers associated with mishandling and misapplication of pesticides include possible injury to the operator and handler and damage to target and non-target plants, to the equipment, and to the environment. Read the health and safety codes of your organization pertaining to use of toxic chemicals prior to use of pesticides.

1. **Read the label**. Handlers should read, understand, and follow all instructions on the label. Notice warnings and cautions before opening the container. Repeat the procedure every time, no matter how familiar you think you are with the directions. Apply the material only in the amounts and at the times specified.
2. **Avoid contact**. Avoid inhaling sprays and dusts. Avoid contact with skin and eyes. When directed by the label, wear the proper protective clothing, a mask, and all other recommended personal protective equipment. Do not eat, drink, smoke or chew on anything while spraying or dusting. Wash thoroughly before eating.
3. **Apply safely**. Use only the specified dosages and mix as directed. Do not use your mouth to siphon liquids from containers or blow out clogged lines or nozzles. Use clean, well-functioning equipment to apply the pesticides. Do not spray with leaking hoses or connections. Do not work or allow others to work in the drift of the spray or dust.
4. **Wash immediately**. Stop and wash off any pesticide spilled on the body. Remove contaminated clothing. Wash and change to clean clothing after spraying and dusting. Also, wash clothing each day before re-use.
5. **Dispose of containers properly**. Always dispose of empty containers so that they pose no hazard to humans, animals, or plants (either terrestrial or aquatic). When unsure about proper disposal procedures, contact the nearest waste management authority.
6. **Store safely**. Keep pesticides stored together outside the home or office away from food and usual working areas. Keep them under lock and key. Label and sign the area well and do not store other chemicals among the pesticides. Always keep the pesticides in the original containers, and keep them tightly closed.
7. **Report illness.** If symptoms of illness occur during or shortly after dusting or spraying, call a physician or poison control number or 911 and get the patient to a hospital immediately.

1. Environmental Stresses

John Ball; revised from Donald F. Schoeneweiss (Riffle and Peterson 1986)

Environmental stresses, which are nonliving (abiotic) disorders, are commonly overlooked causal agents when tree problems are diagnosed in the Great Plains. This oversight is unfortunate as many of the problems submitted as samples to diagnostic clinics are due, at least in part, to environmental stresses. Because no signs are associated with these disorders, identification of the specific stresses can be difficult with only a leaf or twig sample. An accurate diagnosis of environmental stresses requires knowledge of the normal appearance of the species and cultivar, its growing requirements, site conditions, and plant history. Obtaining this information may require a site visit and interview with the tree's owner.

Environmental stresses are sometimes defined as only weather-related stresses; extremes in temperature or precipitation, or snow, ice, and wind are considered separate from soil-related stresses and those imposed by human activity. However, most diagnostic guides cover all these as environmental stresses but within three broad categories:

- Human activity-related: air pollution, deicing and dust suppressant road salts, herbicides, improper planting, mechanical operations.
- Soils-related: nutrient availability, soil moisture.
- Weather-related: ice, precipitation, snow, temperature, wind.

This chapter will cover the most common environmental stresses in the Great Plains. Two of these stresses, nutrient deficiencies resulting from alkaline soils (chlorosis) and herbicide, are covered in more detail in other chapters as they are very common tree stresses in the Great Plains. Other environmental stresses such as ice, snow, and wind loading, and other mechanical stresses such as mower or string trimmer damage, are omitted from this chapter as they are fairly simple to identify in the field. Environmental stresses related to air pollution are also omitted. High ozone concentrations in the lower atmosphere are one of the most common air pollution concerns for trees and forests, but ozone injury to trees in the Great Plains is rare.

Hosts and Distribution

Environmental stresses are ubiquitous; they are not limited to specific hosts or distribution in the Great Plains. One key field diagnostic clue for determining the presence of environmental stresses is that they occur across tree species, genera, and families. It is common to find trees of differing genera expressing similar symptoms from exposure to the same environmental stress. This symptom pattern is rare with biotic stresses, which are generally limited to a specific host genus or species.

However, different genera and species do vary in their tolerance to specific environmental stresses. For example, the susceptibility to damage from road deicing salts varies among genera; ashes (*Fraxinus* spp.) are generally tolerant and arborvitaes (*Thuja* spp.) are very sensitive to exposure. Tolerance levels can vary considerably even within a genus. Eastern white pine (*Pinus strobus*), for instance, is very sensitive to exposure to road deicing salts, whereas Austrian pine (*P. nigra*) is tolerant. This same variation is found for many other environmental stresses, so knowledge of a specific species' tolerance to the suspected environmental stress is critical to the diagnostic process.

Environmental stresses are common throughout the Great Plains, but stresses related to soil and human activity are usually limited to specific sites. Flooding injury, for example, occurs in riparian areas; damage from road deicing salt is most apparent in trees lining roads. These symptom patterns can also be important clues for determining the presence of environmental stresses. Biotic stresses, such as infectious diseases, often spread randomly on a site depending on the tolerance or resistance of individual host trees to a specific pathogen. Environmental stresses usually follow regular patterns, such as the damage being most apparent just above the snow line on a marginally hardy shrub

(fig. 1-1). It is much easier to identify environmental stresses in the field, rather than from samples, because a field visit allows symptom patterns to be observed and information to be gathered on the plant's history.

Symptoms

No signs are associated with environmental stresses. Instead, symptoms, which are physical changes in the tree's appearance, are used in the field to determine the presence and role of these stresses. But symptoms are not as diagnostic as signs; very similar symptoms may be associated with different environmental stresses (table 1-1). Marginal necrosis (scorch)—blackened or brown tissue along the edge of the leaf blade—may be the result of a late spring frost or a summer drought. Symptoms associated with environmental stresses may also mimic those expressed by injury from insects or pathogens, thereby complicating diagnosis of their role. For example, a stunted pine shoot may be a result of drought, road deicing salts, pathogens, or borers. Another difficulty in the diagnostic process is that environmental stresses may not be limited to a single stress agent, but may occur as multiple stresses, such as drought and saline soils. The combination of two or more environmental stresses may result in a symptom pattern different from that of a single stress acting alone.

Figure 1-1—A common dieback pattern associated with abiotic stresses. The definite line to the dieback here was the snow depth on the privet hedge that protected the lower buds from winter injury (John Ball, South Dakota State University, used with permission).

Determination of the stress or stresses responsible for tree damage may require further investigation in a laboratory. Although the symptom pattern on roadside trees may indicate use of road deicing salts, tissue samples may be necessary to determine the presence or absence of salts and whether their concentrations were sufficient to cause the damage observed. This step may also be required when associating symptoms with nutrient deficiencies or herbicide drift.

Table 1-1—Common environmental stress agents and their symptoms

	Foliage symptoms						Canopy symptoms			
Environmental stress agent	**Chlorosis**	**Premature autumn color**	**Premature foliage drop**	**Scorch**	**Small foliage**	**Wilting**	**Adventitious shoots**	**Dieback**	**Reduced shoot growth**	**Split trunk**
Alkaline soils—iron/manganese deficiencies	x			x	x			x	x	
Drought			x	x	x	x		x	x	
Flooding		x	x	x	x	x		x	x	
Freeze				x	x	x		x		x
Frost			x	x	x	x		x		x
Planting errors		x	x	x	x	x		x	x	
Poorly drained soils	x	x			x			x	x	
Road deicing/dust suppressant salts			x	x			x	x	x	
Saline soils	x			x		x		x	x	
Stem-girdling roots		x	x	x	x			x	x	

Cause and Damage

Alkalinity and iron or manganese availability

Chlorosis is covered in more detail in chapter 5 of this publication, but the symptoms are so prevalent in Great Plains trees that it is worth mentioning in this chapter as well. Most tree species perform best in soils that are slightly acid (pH 6.1 to 6.5). Soil pH greater or less than this range can result in excessive or reduced availability of specific soil nutrients. Slightly alkaline soils, those with a pH between 7.4 and 7.8, are common in the Great Plains. These soils may lack sufficient available (soluble) iron or manganese to support certain tree species.

Two of the species most sensitive to reduced availability of micronutrients are pin oak (*Quercus palustris*) and red maple (*Acer rubrum*). When planted on slightly alkaline soils or even neutral soils, these trees express symptoms of interveinal chlorosis, which is yellowing of the leaf tissue surrounding the veins (fig. 1-2). There is also a gradient of these symptoms along a branch, with the symptoms more severe on the newest foliage. These micronutrients are immobile, so they cannot be transferred from older to newer foliage tissue.

Most tree species can tolerate slightly alkaline soils and there are a few tree species such as black walnut (*Julgans nigra*) and bur oak (*Q. macrocarpa*) that may not show any symptoms of chlorosis until the soil pH exceeds 8.0. Although chlorosis is most often linked to alkaline soils, it can also appear in trees on saline or poorly drained soils. There are also tree cultivars grown in the Great Plains that have yellow or golden ornamental foliage, which may be confused with chlorosis. These cultivars include 'Princeton Gold' Norway maple (*A. platanoides* 'Princeton Gold'), Golden Shadow® pagoda dogwood (*Cornus alternifolia* 'W. Stackman'), 'Taylor's Sunburst' pine (*P. contorta* 'Taylor's Sunburst'), and golden red oak (*Q. rubra* 'Aurea').

Drought/heat

Excessive heat and water deficit stresses usually occur in combination, so the two are generally discussed together with water deficits considered the more serious stress of the two. Deciduous trees affected by water deficits may exhibit foliage that droops, curls, and become scorched (fig. 1-3). The scorching will usually move deeper into the leaf tissue between the veins. Severely injured foliage will discolor, often turning yellow to brown, and abscise prematurely. Conifer needles may turn brown, droop slightly near the base, and shed prematurely. Pines affected by long-term drought may retain only their one- and two-year-old needles.

Shoot extension may be reduced either during the year of the drought or the next year depending upon the season the drought occurred and whether the affected species has preformed shoot growth (the environmental conditions one year determine the extent of shoot growth the next year) or sustained shoot growth (the conditions during the present year determine the growth that same year). Trees such as pine and spruce (*Picea* spp.) that exhibit preformed growth will produce abnormally shortened needles and shoots the year following a drought (fig. 1-4). This "bottlebrush" growth pattern is often

Figure 1-2—Chlorotic leaf from a swamp white oak (*Q. bicolor*) (John Ball, South Dakota State University, used with permission).

Figure 1-3—Browning and wilting oak leaves affected by drought (John Ball, South Dakota State University, used with permission).

Figure 1-4— "Bottlebrush" appearance of blue spruce (*Picea pungens*) shoots, an indication of past stresses (John Ball, South Dakota State University, used with permission).

accompanied by excessive production of cones, both of which are very common symptoms of water deficits from the previous growing season. Trees such as birch (*Betula* spp.) that have sustained growth will show reduced shoot growth during the summer when the drought occurred. However, symptoms of late-summer and fall drought injury may not appear until the next year. Late-season drought may also increase the potential for winter freeze injury.

Flooding

Flooding is a frequent occurrence along the many streams and rivers in the Great Plains. There are many factors to consider when determining the impact of flooding on a tree. Early spring flooding that occurs while the trees are dormant imposes less stress than flooding that occurs during the growing season. Colder and fast-moving water causes less injury than warm, stagnant water. Flooding depth is also a factor, with water covering the trunk or canopy more injurious than a flood that only saturates the soil. Season and duration of flooding are factors as well. Many trees are able to tolerate spring flooding for several days. Most tree species can survive flooding for several weeks, but few can survive for time periods longer than two months. The list of species that can survive long flooding periods includes boxelder (*A. negundo*), green ash (*F. pennsylvanica*), plains cottonwood (*P. deltoides* ssp. *monilifera*) and willow (*Salix* spp.). Flooding symptoms often appear first as premature fall foliage color followed by wilting (fig. 1-5). Dieback and decline of the canopy may follow, depending upon the species' tolerance to flooding and the depth, duration, and season of the flood.

Frost

Late frost may occur while a tree is breaking bud and the tender shoots and leaves are beginning to expand. This tissue is vulnerable to cold temperatures and if exposed to freezing temperatures, it can be injured or killed. Late spring frosts result in wilted and scorched foliage on deciduous trees (fig. 1-6) and curled and distorted needles on conifers. Stems of young deciduous trees may develop vertical splits.

Figure 1-5—Premature fall foliage color, a common symptom of flooding injury (John Ball, South Dakota State University, used with permission).

Figure 1-6—Wilting of newly expanding foliage, a common symptom associated with late frosts (John Ball, South Dakota State University, used with permission).

Planting errors

Improper planting is another common environmental stress agent. Errors at planting cannot be easily corrected later and may result in long-term growth reduction, decline, and premature death. The most common long-term planting stress is from planting too deep. Placing soil around the trunk and covering the trunk/root interface can result in a slow decline. A tree that was planted too deep may persist for years, even decades, but the shoot growth is usually much less than normal and the leaves may be small. The most common clue that the tree has been planted too deep is that the stem comes straight out of the ground without the wider taper that normally occurs at the flare. Most ornamental trees are budded, so the bulge or crook associated with the budding onto a rootstock should still be above ground on young trees (fig. 1-7).

Poorly drained soils

High precipitation combined with clay soils or a perched water table can result in excessive soil moisture, which contributes to root decline. Trees growing on poorly drained soils may appear stunted and the foliage may become chlorotic (fig. 1-8). The foliage may also turn red or yellow by late summer. Some tree species are less adapted to this environment than others. Spruces are poorly suited to such sites, whereas willows may thrive.

Road deicing and dust suppressant salt applications

Deicing and dust suppressant road treatments involve applications of liquid or solid forms of magnesium chloride or sodium chloride. The chlorine ion is considered to be the most injurious to trees. Chloride can be deposited on the buds and foliage and absorbed into the tissue or enter the tree through the root system from salt runoff. Symptoms of chloride toxicity on conifers typically begin as browning foliage, starting at the needle tips, and on the side of the tree facing the road (fig. 1-9). The injured foliage will shed prematurely so that perhaps only the one- and two-year-old needles persist. Deciduous trees will have buds fail to open, resulting in branch dieback. Witches'-brooms, which are clusters of adventitious shoots, usually occur on the side of the canopy facing the road. Some tree species are more sensitive to these salts: little-leaf lindens (*Tilia cordata*) may be affected, yet nearby honeylocust (*Gleditsia triacanthos*) do not show any symptoms.

Saline soils

Some regions of the Great Plains have soils that are naturally high in salts or become saline from soluble salts in irrigation water. High salt concentrations can disrupt the movement of water from the soil into the roots. High soil salt concentrations can also change soil structure. The salts can bind to clay particles, reducing pore spaces and thereby potentially compromising gas exchange and water drainage. Damage from saline soils most often occurs in seedlings rather than in mature trees. Tree seedlings in saline

Figure 1-7—A tree that was planted too deep. The slight bulge at the base of this budded tree should have been above the soil surface (John Ball South Dakota State University, used with permission).

Figure 1-8—Declining spruce in poorly drained soils (John Ball South Dakota State University, used with permission).

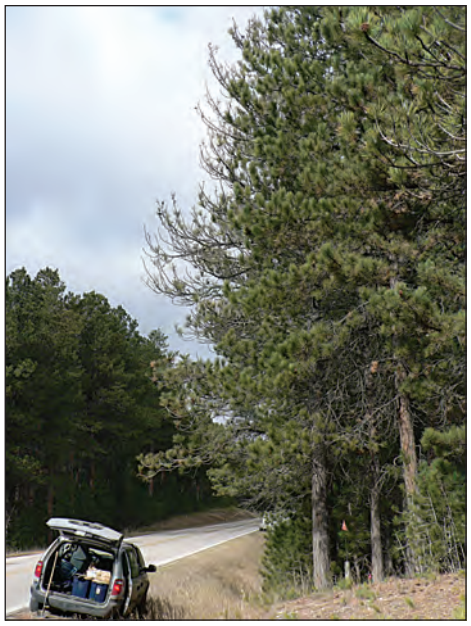

Figure 1-9—A ponderosa pine (*Pinus ponderosa*) exhibiting symptoms of injury from road de-icing salts (John Ball South Dakota State University, used with permission).

soils may exhibit foliage scorch or wilting. The trees may also become stunted or die if soil salt concentrations are too high. The soil salt concentration can be determined only by soil testing, and soils with a conductivity greater than 8 deciSiemens/meter (dS/m) measured as a saturated paste are harmful to all but two tree species, Russian-olive (*Elaeagnus angustifolia*) and mugo pine (*P. mugo*). Many tree seedlings can become established and grow on slightly saline soils, that is, those with a conductivity near 4 dS/m.

Stem-girdling roots

Associated with burying the trunk in the soil at planting is the development of stem-girdling roots. The development of a stem-girdling root requires a portion of the stem be below grade, which allows a root to rise to constrict the stem (fig. 1-10). Stem-girdling roots are more common in species that generate roots that grow tangential to the trunk following harvest or root pruning. Maple, ash, and linden are the three most common genera associated with this disorder. Stem-girdling roots may also occur with root-bound container stock if the circling roots are not shaved off at planting. Stem-girdling roots can reduce tree stability because of the distorted root system. The girdling can also restrict photosynthate movement to the roots, and water and nutrient movement to the foliage. The most common symptoms associated with stem-girdling roots are leaf scorch, abnormally small foliage, premature fall foliage color, premature foliage drop, dieback, and death. The stem may also appear flattened along one side.

Figure 1-10—A stem-girdling root appearing on a tree (John Ball, South Dakota State University, used with permission).

Winter injury

Planting a species farther north than its native range may result in cold winter temperatures killing the tree or, as is often the case with marginally hardy tree species, branch dieback, which provides an entry wound for pathogens. Thornless honeylocust (*G. triacanthos* var. *inermis*) cultivars that originate from trees native to milder climates may be injured by the cold winter temperatures in the Great Plains and become more susceptible to canker diseases.

The different parts of the tree—buds, sapwood, roots—acclimate to different midwinter minimal temperatures, so cold injury might be limited to the loss of specific tissue. The sapwood is less cold tolerant than the vegetative buds. Cold injury to the sapwood may not be noticed until the spring, when the expanding foliage on the affected shoot wilts and dies. A brown streak is often present in the sapwood of these injured trees.

Roots are the more cold-sensitive part of a tree and the woody roots for many tree species cannot tolerate temperatures below 15 °F. Cold injury to roots can occur during winter on sites with bare soils and inadequate snow cover. Trees with extensive cold injury to the woody roots will often partially leaf out in spring before wilting. This injury can occur on tree cultivars that are generally considered hardy to the region if the rootstock is

from a more southern seed source or even from a cold-sensitive species. Winter injury has been observed in the Great Plains when State Street® maple (*A. miyabei* 'Morton'), which is hardy to much of the region, is budded onto hedge maple (*A. campestre*) rootstock, a tree hardy only to the southern Great Plains.

Much of the winter injury in the Great Plains does not occur in midwinter but occurs during late fall and early spring as trees are acclimating to or de-acclimating from freezing temperatures. The timing and rate of acclimating to cold winter temperatures differ among species and may even differ among seed sources within a species. As the photoperiod shortens in late summer and early fall, trees begin the process of acclimation to the subfreezing conditions of winter. This process accelerates with exposure to freezing night temperatures. If the fall is abnormally warm with very few freeze/thaw cycles before cold winter temperatures begin, the trees may not be fully acclimated. Injury can also occur in late winter and early spring when trees are deacclimating and preparing to resume growth. If a tree has fulfilled its chilling requirement—exposure to a minimum period of cold temperatures—and the weather turns unseasonably warm then back to more-normal cold temperatures, freeze injury may result. Because of the continental climate of the Great Plains, this region experiences more unseasonable temperature fluctuations between fall and spring than areas along the east or west coast, where oceans help in moderating temperatures.

A type of winter injury common to evergreens is winter desiccation injury, commonly referred to as winter burn. This injury occurs during sunny and windy winter days where temperatures are high enough that water can be lost to transpiration. This water cannot be replaced as the soil and trunk are still frozen, so the foliage desiccates. The injured foliage turns red or brown (fig. 1-11), though this color change may not occur until spring as the evergreen begins to resume growth.

Figure 1-11—Browning of concolor fir needles from winter burn (John Ball, South Dakota State University, used with permission).

Management

Environmental stresses are common, but often overlooked, causal agents when diagnosing tree problems. They are easy to miss as environmental stresses do not produce signs, only symptoms, and these symptoms can also be associated with multiple abiotic and biotic agents. Identification of environmental stresses requires not only knowledge of the symptoms associated with a particular stress but also site information and history. For example, are the soils alkaline or saline, or was there a late frost the past spring? Determining environmental stresses is difficult to do from a sample or a picture attached to an email and often requires site information and a history of the plant's planting and care.

Another reason that environmental stresses may be overlooked is their underlying role in a tree's decline. Although environmental stresses can be primary factors in tree mortality, they more often serve as predisposing factors in tree decline. The gradual decline of a tree can be described as a mortality spiral as the tree's susceptibility and sensitivity to abiotic and biotic agents increase as the tree declines. The spiral begins with predisposing factors, followed by inciting and finally contributing factors. Predisposing factors are long-term stresses that reduce the tree's health on a particular site and increase susceptibility to pests. The most common predisposing factors in tree decline are environmental stresses such as poorly drained soils and planting errors.

Trees may endure predisposing stresses for many years until an inciting factor occurs. Incitants are short-duration stress agents that further compromise the tree's health and its capability to defend itself from pests. Common incitants are late frosts and drought, or defoliation by leaf-feeding insects or pathogens. These inciting factors add to the stresses

imposed by the predisposing factors and the combined effect will be reduced capability of the tree to defend against secondary pests.

Secondary pests, referred to as contributing factors in the decline spiral, are generally biotic; common examples are bark beetles, root diseases, and canker diseases. Detection of these organisms in a tree often coincides with symptoms of extensive canopy dieback. The observer may associate the appearance of dead branches with the presence of borers or cankers. These organisms often are assumed to be the sole cause of the decline and the predisposing factors and incitants ignored.

Making this assumption often results in managing only the contributing factors. Merely treating the contributing factors may not arrest the tree's decline. Tree managers should instead address or at least acknowledge the underlying stresses, both predisposing and inciting, and try to understand their role in the tree's decline.

Once the environmental stresses are identified, there is often little that can be done to alleviate their effects. A marginally cold-hardy tree subjected to winter injury will be injured every winter. Predisposing factors are not usually correctable or alleviated by any treatment. Environmental inciting factors, though not correctable, may be mitigated by tree care practices such as watering and mulching to reduce their impact.

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2. Anthracnose Diseases of Broadleaf Trees

Scott C. Redlin; revised from Robert W. Stack and Kenneth E. Conway (Riffle and Peterson 1986)

Many trees are affected by diseases caused by fungi with asexual stages that produce asexual spores (conidia) in acervular conidiomata. Diseases caused by these fungi are commonly called anthracnose and are usually connected to Ascomycete fungi (Diaporthales: Gnomoniaceae[1]). The site of infection, symptoms and signs, and severity of the anthracnose disease differ from species to species and often vary from year to year. The only symptom commonly found on maples (*Acer* spp.) and elms (*Ulmus* spp.) may be leaf spot or leaf blight (figs. 2-1 and 2-2). In other species, such as sycamore (*Platanus* spp.) and white oaks (*Quercus* spp. such as *Q. macrocarpa*), the entire range of anthracnose symptoms may occur, including bud, twig, and shoot blights, leaf spots, and stem cankers. Several of the causal fungi are now considered latently infecting or endophytic. Fungi on host trees have asexual stages in several genera including *Asteroma, Discula*, and *Marssoniella.* Some species have two or more anamorphic stages (synanamorphs). Numerous names for the anamorphs of *Apiognomonia errabunda,* which occurs on basswood (*Tilia* spp.), oak, and other hosts have been proposed, but few are nomenclaturally acceptable.

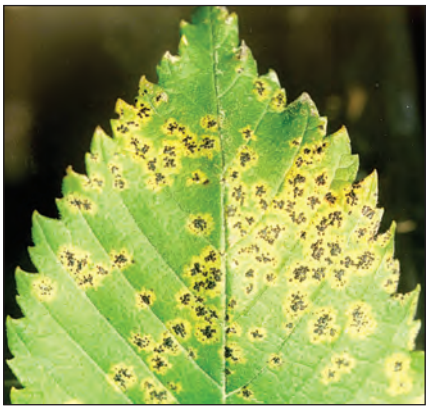

Figure 2-1—Black leaf spot of elm caused by *Stegophora ulmea* (Bruce Watt, University of Maine, used with permission).

Figure 2-2—Symptoms of maple anthracnose caused by *Aureobasidium apocryptum* (Paul Bachi, University of Kentucky, used with permission).

Hosts and Distribution

Anthracnose diseases caused by Gnomoniaceae are widespread throughout the native and planted ranges of their respective hosts. The pathogens and their hosts growing in the Great Plains are summarized in table 2-1. Each fungal species is specific for a particular host genus, with little or no cross pathogenicity as far as is known. The taxonomy of the causal fungi of anthracnose diseases has been revised. For each host, the symptoms may differ based on the part of the tree attacked (table 2-1).

Symptoms and Signs

Leaf infections early in the season result in blotching, distortion, and large necrotic areas. These symptoms are typical of ash (*Fraxinus* spp.), oak, and sycamore anthracnose (fig. 2-3 through fig. 2-6). If symptoms occur early in the season, they can be confused with frost damage. Later leaf infections result in discrete small to large spots, often

[1] The fungi in the order Diaporthales causing anthracnose symptoms discussed here have been proposed to be reclassified to include the families Sydowiellaceae and Valsaceae in addition to Gnomoniaceae.

Table 2-1—Pathogens causing anthracnose diseases of trees in the Great Plains

Host attacked	Name of pathogen[a]	Parts of tree attacked
Ash, especially green ash	*Plagiostoma fraxini* Syn.: *Gnomoniella fraxini* Anamorph: *Discula fraxinea*	Leaves, twigs, samaras
Basswood	*Apiognomonia errabunda* Anamorph: *Discula* sp.	Leaves, twigs
Elm	*Stegophora ulmea* Anamorph: *Asteroma ulmea*	Leaves
Maple	*Aureobasidium apocryptum* *Colletotrichum gloeosporioides* *Discula campestris* *Discula* spp.	Leaves
Oak, especially white oaks	*Apiognomonia quercina*[b] Anamorph: *Discula quercina*	Leaves, twigs, shoots, buds
American sycamore; London plane-tree	*Apiognomonia veneta* Anamorph: *Discula platani*	Leaves, twigs, shoots, buds
Walnut	*Ophiognomonia leptostyla* Anamorph: *Marssoniella juglandis* (syn.: *Marssonina juglandis*)	Leaves, twigs, nuts

[a] The anamorph names of most of the anthracnose fungi require further study for accurate taxonomic placement. They are provided in table 2-1 because the anamorph, commonly observed during spring and summer, is the basis of disease diagnosis.

[b] *Amphiporthe leiphaemia* is proposed as the most recent nomenclature for this pathogen.

surrounded by a dark ring or a chlorotic halo. Such spots are the principal symptoms in basswood, elm, and maple (figs. 2-1 and 2-2). Severe leaf spot infection may cause extensive defoliation, especially in ash, sycamore, and walnut (*Juglans* spp.).

On sycamore, four phases of anthracnose can be distinguished: twig blight, bud blight, shoot blight, and leaf blight. Leaf blight is already described above. In the twig blight phase, small, 1- or 2-year-old twigs are girdled and killed (fig. 2-5). Later, larger twigs and branches may be cankered and killed. In the

Figure 2-3—Leaf distortion and necrosis of green ash caused by *Plagiostoma fraxini* (Michael Kangas, North Dakota Forest Service, used with permission).

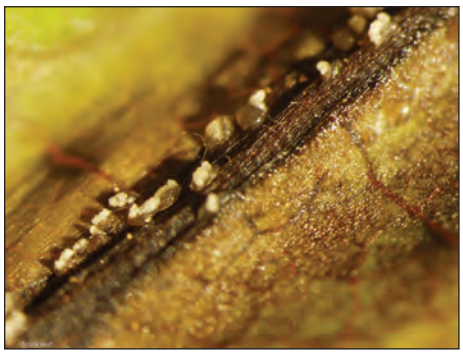

Figure 2-4—Acervuli of *Apiognomonia quercina* on midvein of oak (Bruce Watt, University of Maine, used with permission).

Figure 2-5—Leaf necrosis and twig dieback of sycamore caused by *Apiognomonia veneta* (Joseph O'Brien, U.S. Forest Service).

Figure 2-6—Leaf necrosis of sycamore caused by *Apiognomonia veneta* (American Phytopathological Society, used with permission).

bud blight phase, buds are penetrated by the fungus and killed before they expand. In the shoot blight phase, new young shoots and expanding leaves are suddenly killed. In sycamore (and probably other hosts), severe shoot blight depends on temperature, with infection greatest when temperatures are 50 to 59 °F. In walnut, optimal infection temperature is 70 °F.

Disease Cycle

The life cycles of these anthracnose fungi are similar in that they require water from rain, dew, or fog to infect a tree. However, most anthracnose fungi do not incite all four phases of symptoms, and *Apiognomonia veneta* does not represent the typical life cycle in the Midwest. The fungi survive the winter primarily on fallen infected leaves and sometimes in infected twigs and branches. With ash anthracnose, petioles colonized by the powdery mildew fungus, *Phyllactinia fraxini*, often persist through winter and provide a major source of anthracnose inoculum close to the buds, from which susceptible shoots emerge in spring. In most situations there is abundant inoculum for infection if conditions are favorable. Asexual spores (conidia) are the most important stage for dissemination and infection by anthracnose fungi. In several species, the sexual spores (ascospores) may also be produced and serve as primary inoculum; however, their relative importance compared to the conidial stage is undetermined.

Spores are disseminated by wind and splashing rain to buds, shoots, and expanding leaves, where infection begins if conditions are favorable. In some species, infections develop during favorable periods in summer, giving rise to late-season spots or leaf blight.

Damage

Anthracnose fungi can cause defoliation and contribute to branch dieback, which disfigures the tree. If anthracnose is severe for several seasons, the tree may be weakened and start to decline, or may become susceptible to other diseases or insect pests. Trees subject to other stresses such as root restriction, drought, or heavy scale infestation, are much less tolerant to anthracnose, and may show decreased vigor after only a single season of severe anthracnose defoliation.

Management

Because individual trees may vary in susceptibility to anthracnose, there is potential for selection of resistant clones or seed sources of highly resistant trees. The true London plane-tree (an interspecific hybrid, *Platanus x acerifolia*) is much less susceptible than the native American sycamore (*Platanus occidentalis*). Oaks in the white oak group are much more susceptible to anthracnose than those in the red oak group (for example, *Q. palustris*).

Several cultural practices can help manage anthracnose diseases and include sanitation and pruning to remove infected twigs and branches during the dormant period. In walnut anthracnose, removing some branches by pruning increases air movement through crowns, facilitates faster drying of leaves after rain, and reduces infection of leaves.

Application of nitrogenous fertilizers promotes tree vigor and could result in less-severe disease.

Fungicide sprays have been used in attempts to control anthracnose on boulevard and residential plantings but are rarely warranted because anthracnose diseases do not occur every year and seldom seriously affect tree health. Specific protectant fungicides are registered for certain tree species. Follow label instructions for times of application and dosage rates. Specific recommendations on type of spray and timing depend on locality. Consult your local extension plant pathologist or forester for current recommendations for your area. Several systemic fungicides have been registered for tree injection to control anthracnose fungi.

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3. Bur Oak Blight

Thomas C. Harrington and Douglas L. McNew

Many species of *Tubakia* are capable of causing late-season leaf spots and necrosis of leaf veins on oak (*Quercus* spp.), chestnut (*Castanea* spp.), and other hardwood species in Europe, Asia, and North America. But the recently described fungus *T. iowensis* is also capable of causing petiole necrosis and death of whole leaves on bur oak (*Q. macrocarpa*), sometimes killing nearly every leaf on a susceptible tree. Damage to bur oak was first noted in the mid-1990s in the northern Great Plains and the Upper Midwest, and the incidence appears to be increasing in this region, perhaps due to more abundant spring rainfall.

Hosts and Distribution

Substantial necrosis of leaf veins and petioles by *T. iowensis* occurs only on bur oak. Bur oak blight is most common in Iowa and Minnesota, but the disease has been noted in western and southern Wisconsin, northern Illinois, northeast Kansas, eastern Nebraska, and eastern South Dakota, with isolated groups of affected trees in counties of Illinois and Missouri that border Iowa. This disease distribution roughly coincides with the limited distribution of a small-acorned, fire-adapted variety of bur oak, *Q. macrocarpa* var. *oliviformis*. Bur oak blight is most severe in relic savanna groves on upland sites (fig. 3-1). On bottomland sites, few trees show severe symptoms, and the disease has not been recorded where the large-acorned *Q. macrocarpa* var. *macrocarpa* is common. The disease does not seem to be present in eastern North Dakota, where *Q. macrocarpa* var. *depressa* predominates on sites with sandy soils. Ornamental plantings of bur oak trees with large acorns are not seriously affected, but planted bur oak with small acorns may have severe symptoms (fig. 3-2).

Figure 3-1—Two mature severely blighted bur oak trees (left). Other bur oak trees in the grove appear healthy, presumably because of resistance (Thomas C. Harrington, Iowa State University, used with permission).

Figure 3-2—A planted bur oak tree with small acorns, showing substantial leaf symptoms in the lower crown (Thomas C. Harrington, Iowa State University, used with permission).

Symptoms and Signs

The first symptoms usually appear in June in the form of elongated, purple-brown lesions on veins of the leaf undersurface, sometimes with numerous small, reddish-brown necrotic spots on the leaf blade between veins (fig. 3-3). Sporulation is not evident until there is more substantial necrosis of the leaf veins in late June and throughout summer (fig. 3-4), when leaf symptoms could be mistaken for anthracnose caused by *Amphiporthe leiphaemia (*formerly *Discula quercina)*. Fruiting structures that bear asexual spores (conidia) of *T. iowensis* appear on or near the necrotic veins. On the upper leaf surface, masses of conidia are typically covered with a shield of red-brown, radiating, setal-like structures (fig. 3-5). Unshielded conidial masses form on the veins on the underside of the leaves. The necrosis may expand down to the base of the leaves, and the leaves are shed. Substantial defoliation may be seen during wet summers (fig. 3-6). Veinal necrosis and fruiting structures with radiating shields are evident on the fallen leaves.

Figure 3-3—Initial symptoms of bur oak blight in June: purple-brown lesions on the veins on the underside of leaves (Thomas C. Harrington, Iowa State University, used with permission).

Figure 3-4—Midsummer symptoms of bur oak blight: veinal necrosis, with the fungus producing shielded masses of conidia along the necrotic veins on the upper surface and naked conidial masses on the veins of the lower leaf surface (Thomas C. Harrington, Iowa State University, used with permission).

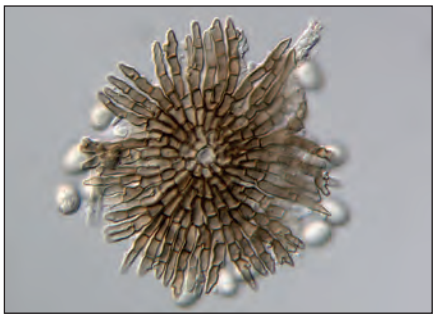

Figure 3-5—Top view of a fruiting structure of *Tubakia iowensis* from the upper leaf surface, showing the shield of radiating, pigmented hyphae and conidia produced under the shield (Douglas L. McNew, Iowa State University, used with permission).

Figure 3-6—Bur oak blight-infected trees with thin crowns. In wet summers, infected trees may shed most of their symptomatic leaves (Thomas C. Harrington, Iowa State University, used with permission).

Coincidental with veinal necrosis and defoliation, necrosis on the base of the petiole and death of whole leaves appear in late July and into August (fig. 3-7). Such leaves may remain hanging on the tree, and black pustules begin to form in late summer on the necrotic petiole tissue. However, these pustules typically do not mature and produce conidia until the next spring, at the time of leaf emergence (fig. 3-8). Healthy bur oak branches typically shed all of their leaves heading into winter.

The fruiting structures and conidia of *T. iowensis* (fig. 3-5) are difficult to distinguish from those of other *Tubakia* species. However, the shielded fruiting structures along the veins on the upper leaf surface and naked conidial masses on the veins of the lower leaf surface are typical for *T. iownesis*. Only *T. iowensis* forms black, crustose fruiting structures on the lower 0.4 inch of overwintering petioles of bur oak (fig. 3-7), but other fungi, such as *Botryosphaeria* species, may form similar black structures on midveins, petioles, and twigs. A twig blight associated with *B. corticola* may also leave dead leaves hanging on branches throughout the winter, but in this case the dead leaves are clustered on dead branch tips instead of scattered among the twigs. The conidia of *Botryosphaeria* species are much larger than those of *T. iowensis*.

Figure 3-7—Leaves endophytically infected by *Tubakia iowensis*. Necrosis of petioles may eventually be shown, leading to dead leaves that remain firmly attached to branches until the next spring, when conidia are produced from black pustules on the petioles (Thomas C. Harrington, Iowa State University, used with permission).

Figure 3-8—Black, crustose pustules of *Tubakia iowensis* on the petioles of overwintering leaves. Pustules produce rain-splashed conidia at the time of shoot emergence in spring (Douglas L. McNew, Iowa State University, used with permission).

Dead leaves and leaves with veinal necrosis are typically most evident in the lower crown (fig. 3-2). Trees severely defoliated in consecutive years may have thin crowns, and twig death and branch dieback are sometimes seen. However, the branch dieback is most likely associated with the two-lined chestnut borer, *Agrilus bilineatus*. Branch dieback associated with the two-lined chestnut borer is commonly found on trees affected by drought, root rots, and other primary stresses, so branch dieback is not diagnostic for bur oak blight. It is likely that some trees with bur oak blight have been misdiagnosed as oak wilt, which may also result in dead foliage and branch dieback.

Disease Cycle

Black, crustose pustules formed on last season's petioles provide the initial inoculum in spring. The fungus may produce sexual spores (ascospores) in spring, but these have not been found and are not likely of major importance. The petiole pustules on hanging leaves expand and break open with wetness in spring, and the conidia are dispersed by rain, leading to endophytic and symptomless infections of expanding shoots and leaves (fig. 3-8). After wet springs, nearly every leaf on a susceptible tree is endophytically infected but remains symptomless for two months or more, when petiole necrosis may become evident. This petiole necrosis apparently prevents normal leaf abscission in the fall.

When mature leaves are infected during the summer, veinal necrosis and leaf death may be conspicuous, and the fungus produces abundant conidia on both the upper and lower leaf surfaces. Many leaves infected during the summer are shed, and it is not clear if summer-infected leaves remain attached and produce petiole pustules the next spring. Wet summers tend to lead to more veinal necrosis and defoliation, but dry summers following wet springs may lead to more petiole necrosis and many dead leaves hanging on the tree. Consecutive years of normal or above-normal spring rainfall seem to lead to buildup of the disease in individual trees. The conidia are dispersed by rainsplash, which might be why the lower crowns of the trees are typically more seriously affected than the upper crowns (figs. 3-2 and 3-6).

Damage

Bur oak blight has been most severe in former savanna groves. However, both the host and pathogen appear to be native, and even in the most severely affected groves, many of the trees show few or no symptoms (fig. 3-1), presumably because of resistance. Within the known range of the disease, fewer trees appear to be affected on bottomland sites as compared to upland sites, perhaps because bottomland ecotypes of bur oak tend to be more resistant. Most ornamental bur oak trees do not show symptoms of bur oak blight, but some planted trees are seriously affected (fig. 3-2). When acorns have been seen on planted bur oak trees with bur oak blight, the acorns have been small, typical for *Q. macrocarpa* var. *oliviformis*.

Severe symptoms in late summer appear to follow periods of high rainfall during the previous spring, and a recent shift in climate towards more spring rainfall is believed to be responsible for the relatively high incidence of the disease in Iowa and Minnesota. Consecutive years of high spring rainfall may lead to buildup of overwintering inoculum (pustules on petioles) in the crown of susceptible trees. High summer moisture may also lead to an abundance of secondary infections with veinal necrosis and defoliation. In contrast, summer drought following normal spring rains may lead to more dead leaves hanging on the trees.

Some trees with repeated defoliation may show twig and branch dieback, generally attributable to the two-lined chestnut borer. Aside from the borer damage, relatively few trees affected by bur oak blight appear to die from the disease. In most areas, root rots and stem decays are more commonly associated with branch dieback and mortality of mature bur oak trees.

Management

Because the initial inoculum is produced in the tree crown, sanitation of leaves on the ground is not believed to be an effective control. Experimental injections of fungicide have been found to be effective in controlling the disease in some high-value trees, but only trees showing moderate to severe levels of disease should be treated. Injection of dilute formulations of propiconazole, following directions for oak wilt control, have been most effective and may reduce symptoms for two or more seasons. Inject trees as soon as the leaves are fully formed in late May or early June, before symptoms appear. However, leaves and small branches of some trees may show phytotoxicity with propiconazole treatments, and not all treated trees show reduced disease levels. In some treated trees, severe symptoms did not return until the second year after treatment, and individual trees need not be treated more than once every three years.

For ornamental plantings in regions with bur oak blight, grow bur oak trees produced from large acorns as these seem to be mostly free of the disease, as are other species of oak. In management of existing groves, the most susceptible trees should probably be removed, especially if they have substantial branch dieback. In mature groves, practice sanitation to reduce populations of the two-lined chestnut borer. In the absence of the borer, the frequency of mortality from bur oak blight alone is quite low.

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4. Cherry Leaf Spots

Gerard C. Adams, Ned Tisserat, and William R. Jacobi

Cherry leaf spot and shot-hole disease are common diseases of foliage of sweet (*Prunus avium*) and sour (*P. cerasus*) cherry, Nanking cherry (*P. tomentosa*), and chokecherry (*P. virginiana*) in the Great Plains (fig. 4-1). Trees that are susceptible to these diseases are planted throughout the Great Plains: fruit-bearing cherry in yards and orchards, ornamental Nanking cherry, and chokecherry in windbreaks and other resource conservation plantings.

Figure 4-1—Cherry leaf spot and shot-hole symptoms on commercial cherry (William R. Jacobi, Colorado State University, used with permission).

Hosts and Distribution

Cherry leaf spot and shot-hole disease are caused by bacterial and fungal plant pathogens of several genera. All of these pathogens and the related diseases are common throughout the range of their hosts and are particularly important in the Great Plains and eastern United States.

- **Bacterial spot** caused by *Xanthomonas arboricola* pv. *pruni* is particularly damaging in commercial stone fruit trees, as well as in ornamental and native species grown in nurseries for home garden, shelterbelt, or soil conservation plantings. This pathogen is becoming the more prevalent and damaging of the shot-hole leaf spot pathogens.
- **Bacterial canker** pathogen *Pseudomonas syringae* pv. *syringae* also may cause leaf spots and shot-hole symptoms and cankers with gummosis; the cankers often lead to susceptibility to winter injury, branch breakage, and problems in replanting.
- **Cherry leaf spot** has been the common disease name for leaf spots caused by the fungus *Blumeriella jaapii* (syn. *Coccomyces hiemalis* and *C. lutescens*) on fruit-bearing cherry, Nanking cherry, sandcherry (*Prunus besseyi*), and chokecherry.
- **Shot-hole blight** or **Coryneum blight** caused by the fungus *Stigmina carpophila* (syn. *Coryneum carpophilum, Wilsonomyces carpophilus,* and *Thyrostroma carpophilum*) has been the more prevalent and damaging disease with shot-hole symptoms on cherry in the past. The typical symptoms may be confused with the other leaf spots and shot-hole pathogens of cherry leading to inappropriate disease management treatments.
- **Cercospora leaf spot** caused by the fungus *Mycosphaerella cerasella* is generally a less damaging leaf spot pathogen even on commercial stone fruit trees.

Symptoms and Signs

The term "shot-hole" refers to leaf spot diseases where the necrotic tissues in small circular lesions on leaves drop out, leaving circular openings or holes.

Bacterial leaf spots caused by *Xanthomonas arboricola* pv. *pruni* and *P. syringae* pv. *syringae* are similar to those caused by *B. jaapii* and *S. carpophila*. Lesions are reddish brown, are angular or circular, and frequently fall out, giving infected leaves a tattered appearance (fig. 4-2). Lesions may coalesce to form large areas of necrotic tissue. Heavily infected leaves are chlorotic and may be shed prematurely. *Pseudomonas syringae* pv. *syringae* also may cause depressed black lesions on fruits, and cankers on twigs and, as seen in figure 4-3, branches and the main stem. Gummosis commonly occurs around cankers. Bacterial leaf spots can be differentiated from fungal leaf spots by crushing a young leaf spot in water and examining under a microscope for the diagnostic bacterial streaming.

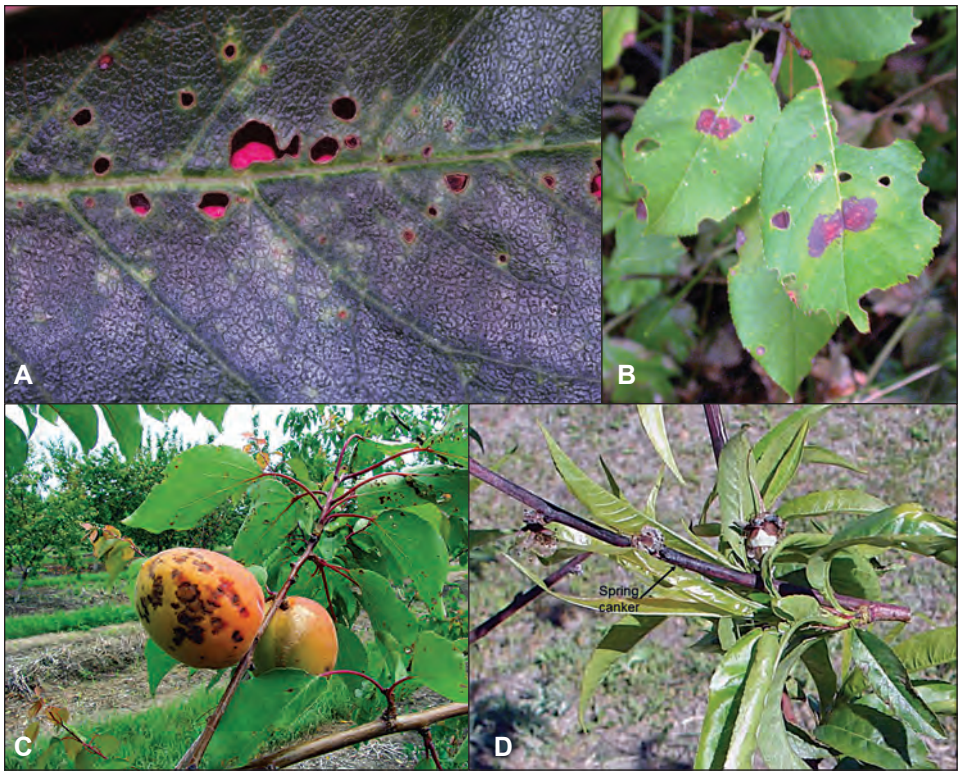

Figure 4-2—Typical bacterial leaf spot and shot-hole symptoms caused by *Xanthomonas arboricola* pv. *pruni*: (A) Shot-hole on leaf of sandcherry (Kevin Korus, University of Nebraska Lincoln, used with permission); (B) Coalesced leaf spots forming necrotic blotches on chokecherry (Ned Tisserat, Colorado State University, used with permission); (C) Infection of apricot (*Prunus* spp.) fruit (Tyre Proffer, Kent State University, used with permission); (D) Spring canker on twig. Spring canker on twigs caused by *P. syringae* pv. *syringae* would appear similar but exhibit gummosis (David Ritchie, North Carolina State University, used with permission).

Figure 4-3—*Pseudomonas syringae* pv. *syringae* bacterial canker causing copious gummosis and death of scaffold branch on residential cherry (Gerard C.Adams, University of Nebraska, used with permission).

Fungal leaf spot and shot-hole blight caused by *B. jaapii*, *S. carpophila*, and *M. cerasella* first appear as small, chlorotic, or off-color (purple to reddish) circular spots, which later enlarge and turn brown and necrotic (fig. 4-4 through 4-6). Spots are usually 4 to 5 mm in diameter; they may be few in number or may become numerous enough to coalesce and form large irregular necrotic areas. If the leaf spots are caused by fungal pathogens, then during humid periods, whitish (for *B. jaapii* and *M. cerasella*) or brownish (for *S. carpophila*) spore masses (conidia) may be visible in the center of lesions. These spore masses are more numerous on the underside of leaves for *B. jaapii* and occur on both sides of the leaves for *M. cerasella*. To determine the fungal pathogen involved in cherry leaf spots, place cherry leaves with intact spots into a moist chamber for 24 hours and then examine the conidia formed on the leaf spot. The conidia of *B. jaapii* and

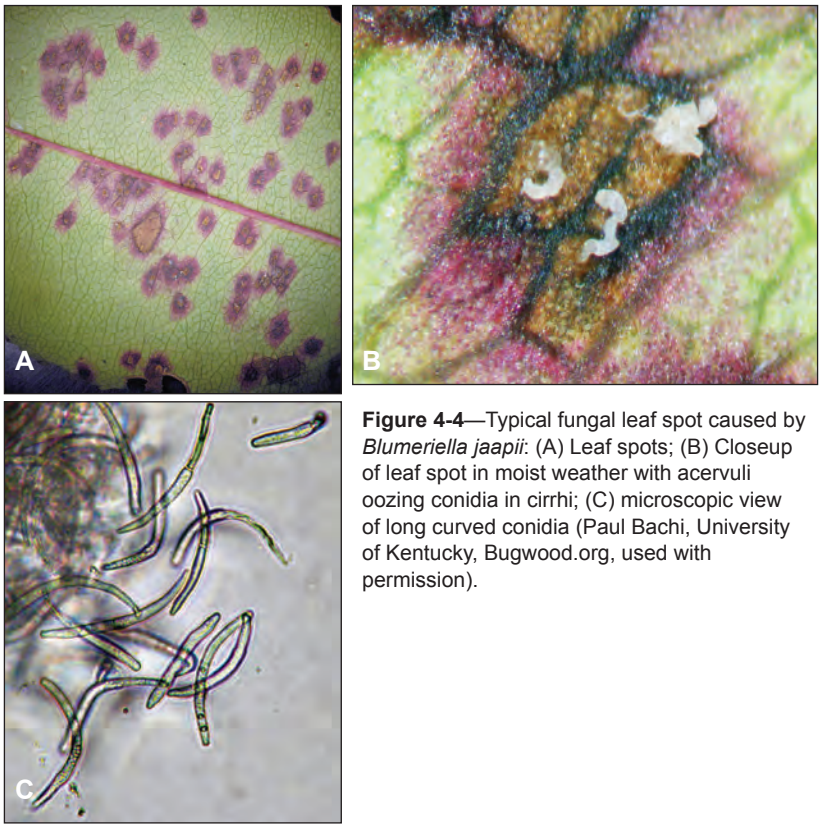

Figure 4-4—Typical fungal leaf spot caused by *Blumeriella jaapii*: (A) Leaf spots; (B) Closeup of leaf spot in moist weather with acervuli oozing conidia in cirrhi; (C) microscopic view of long curved conidia (Paul Bachi, University of Kentucky, Bugwood.org, used with permission).

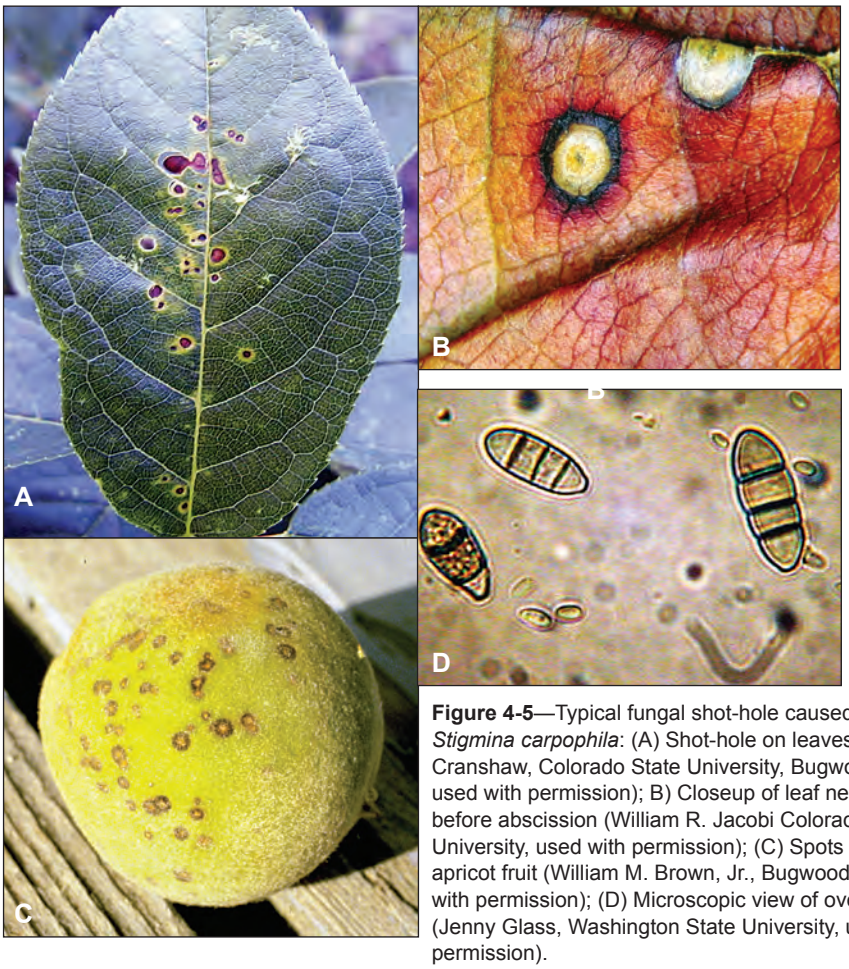

Figure 4-5—Typical fungal shot-hole caused by *Stigmina carpophila*: (A) Shot-hole on leaves (Whitney Cranshaw, Colorado State University, Bugwood.org, used with permission); B) Closeup of leaf necrotic spot before abscission (William R. Jacobi Colorado State University, used with permission); (C) Spots on infected apricot fruit (William M. Brown, Jr., Bugwood.org, used with permission); (D) Microscopic view of ovoid conidia (Jenny Glass, Washington State University, used with permission).

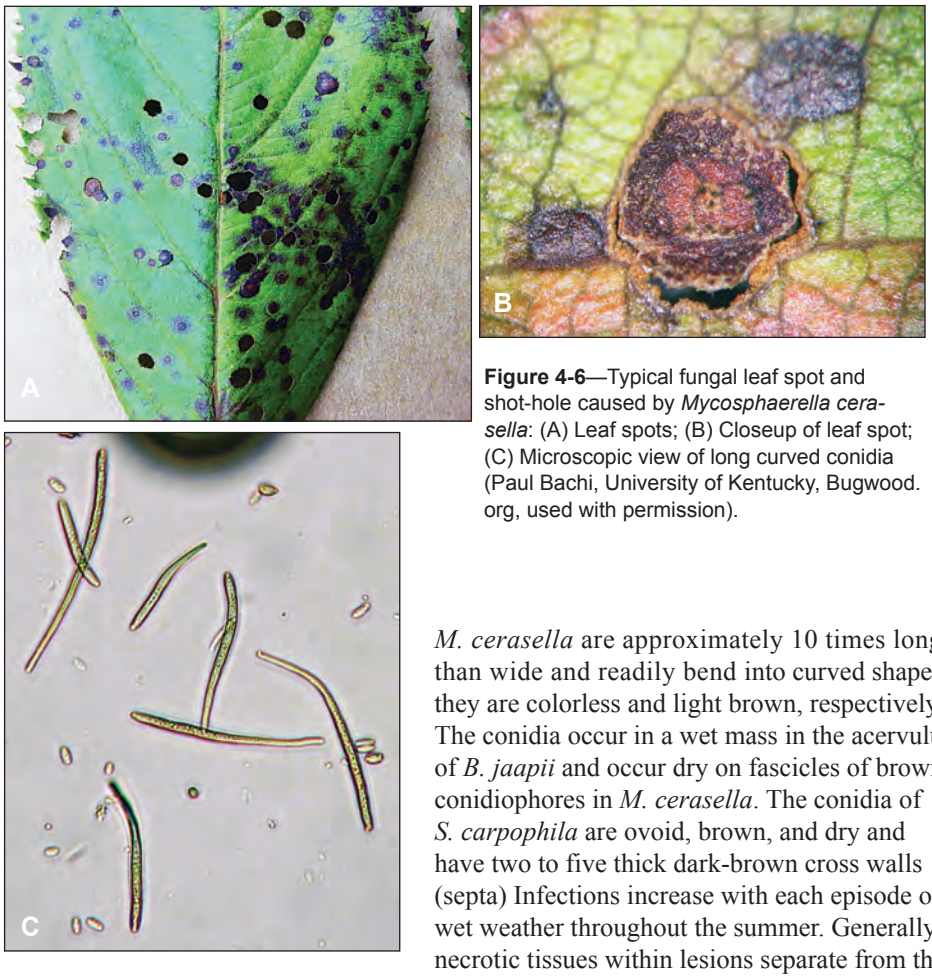

Figure 4-6—Typical fungal leaf spot and shot-hole caused by *Mycosphaerella cerasella*: (A) Leaf spots; (B) Closeup of leaf spot; (C) Microscopic view of long curved conidia (Paul Bachi, University of Kentucky, Bugwood.org, used with permission).

M. cerasella are approximately 10 times longer than wide and readily bend into curved shapes; they are colorless and light brown, respectively. The conidia occur in a wet mass in the acervulus of *B. jaapii* and occur dry on fascicles of brown conidiophores in *M. cerasella*. The conidia of *S. carpophila* are ovoid, brown, and dry and have two to five thick dark-brown cross walls (septa) Infections increase with each episode of wet weather throughout the summer. Generally, necrotic tissues within lesions separate from the surrounding leaf tissues, forming characteristic shot-holes. During severe stages of disease, leaves infected by *B. jaapii* become highly chlorotic, and trees may appear yellow.

Circular black lesions can also form on twigs infected by *S. carpophila* and later these enlarge and become sunken in appearance. This pathogen can infect buds, blossoms, and fruit (fig 4-5c). It is the infection of buds that leads to infection of twigs and canker formation. *M. cerasella* generally remains as a 1- to 2-mm-diameter reddish-brown leaf spot with a lighter border (fig 4-6a, b). However, spots occasionally may coalesce into necrotic patches. Leaf spots caused by *M. cerasella* can be distinguished by the presence of dark fascicles of conidiophores arising from a stroma (fig. 4-6).

Disease Cycle

Bacterial leaf spots of cherry

Xanthomonas arboricola pv. *pruni* overwinters in buds, leaf scars, and twig cankers. The twig cankers in spring can be an important source of inoculum for new infections.

P. syringae pv. *syringae* overwinters within cankers and also is a common epiphyte that occurs on the fruit, limbs, and leaves of both susceptible and nonsusceptible plants. Infection begins when the epiphytic bacteria increase in population owing to physiological changes in the host and favorable weather. However, the inoculum for new infection usually arises from the margins of cankers as bacterial ooze.

Both bacterial pathogens are spread to leaves and cause infections in spring during cool, wet weather. Infection is correlated more closely with favorable weather conditions than with the availability of inoculum. Free-standing water (leaf wetness) is necessary for leaf infection. The bacteria enter stomata primarily, but also can enter lenticels, nectaries, hydathodes, leaf scars, and wounds. Wounds, such as those made during pruning, are common infection courts. Repeated infections develop throughout the growing season during wet periods. Heavily infected leaves fall prematurely.

Fungal leaf spots of cherry

Cherry leaf spot—Infection by *B. jaapii* begins in spring, and is caused by ascospores from fruiting bodies (apothecia) that form on previously infected, fallen leaves. Ascospores are wind dispersed and cause infection during April and May in the southern Great Plains. Leaves are especially susceptible shortly after budbreak. The summer form of the pathogen (*Phlyctema padi* or formerly *Cylindrosporium padi*) has an asexual fruiting body called an acervulus that forms a gelatinous white tendril (cirrhus) of conidia within the center of the leaf lesion during moist weather (fig. 4-4b). Conidia are splash dispersed by rain and wind and cause secondary infections throughout the summer. Fresh pruning wounds can also become infected. Warm temperatures are especially conducive to disease development. Heavy infection may cause premature leaf fall; some trees may be completely defoliated by July.

Shot-hole blight—Infection by *S. carpophila* begins in spring and is favored by cool, wet springs. The pathogen overwinters in blasted buds and cankers, and the primary inoculum generally originates from such tissues, as sporulation occurs during wet spring weather. Fall applied protectant fungicides protect the buds from fungal infection during the late and dormant seasons. Peach (*Prunus persica*) and apricot (*Prunus armeniaca*) are more susceptible to *S. carpophila* than cherry, but the pathogen can infect most species of *Prunus*.

Cercospora leaf spot—Leaf spots and shot-hole symptoms caused by *M. cerasella* are favored by wet springs. The pathogen overwinters on fallen leaves and produces multiple sexual fruiting bodies in each former leaf spot. The primary inoculum is the ascospores, which are discharged in air currents during spring and infect leaves with wet surfaces during moist weather. The summer form of the pathogen (*Passalora circumscissa,* formerly *Cercospora circumscissa*) is a stroma with fascicles of conidiophores and conidia. Conidia that form in the necrotic leaf lesions become windblown and cause secondary infections throughout the summer during conducive weather conditions. The pathogen is common on other species of *Prunus.*

Damage

Cherry leaf spotting diseases cause defoliation; severe disease reduces production and storage of carbohydrates and causes reduced growth. Repeated infections and early defoliation alter the condition of the trees going into winter dormancy and reduce the number of leaves formed and the fruit yield the next year. The reduced vigor the following spring sometimes can result in mortality. Additionally, diseased trees become more sensitive to bud mortality after frost injury. Trees with repeated infections decline over several years.

Cherry leaf spot caused by *B. jaapii* greatly damages commercial sweet and sour cherries in parts of the United States. Although effects on Nanking cherry, chokecherry, and sandcherry are less dramatic, the pathogen commonly causes severe defoliation in crowded nursery beds of chokecherry and sandcherry under sprinkler irrigation in Great Plains nurseries. Planting of chokecherry has been reduced in parts of the Great Plains because of this disease.

Fungal shot-hole blight from repeated infections by *S. carpophila* causes considerable branch dieback owing to the accumulation of bud mortality (blasting) and annual twig cankers. As a result, fruit-bearing and flowering branches are lost, and aesthetic value and vigor are diminished.

Bacterial leaf spot and shot-hole blight caused by *X. arboricola* pv. *pruni* is less severe on cherry than other stone fruits, and annual cankers and shoot and twig dieback are not usually present on cherry. The perennial deep-seated cankers that occur on plum (*Prunus* spp.) do not occur on cherry. This disease is the most economically damaging because of the severe infection of fruit. When fruit production is not the objective of planting, the disease becomes of little significance on cherry except in the nursery, where management of shot-hole is problematic.

P. syringae pv. *syringae* is more important as a canker-causing pathogen of residential and commercial stone fruit trees than as a leaf spot pathogen. Large perennial cankers on the trunk and major scaffold branches produce copious gummosis and are highly destructive. The effect of this pathogen on Nanking cherry, sandcherry, and chokecherry is limited, although some windbreak and ornamental plantings may be severely infected.

Management

Usually, managing these leaf spot diseases is necessary only in commercial fruit growing areas and in nurseries. *B. jaapii* can be controlled with spring curative fungicide and summer protectant fungicide treatments applied directly to foliage. An epidemiological model of the disease cycle is available, which aids in predicting periods of high disease probability in commercial cherry orchards (Eisensmith and Jones 1981). The number of fungicide applications necessary to control the disease has been reduced by use of curative fungicides for spring application to decrease ascospore infection, and use of this predictive model (Holb and others 2010).

Leaf spot caused by *S. carpophila, X. arboicola* pv. *pruni,* and *P. syringae* pv. *syringae* can be reduced by incorporating sanitation practices, such as dormant pruning of bud-blasted and cankered wood and removal of leaves and other debris in the fall. Dormant pruning of cankered branches is especially important for *P. syringae* pv. *syringae*. Fungicides can be used to suppress disease development of *S. carpophila* in the season and also during dormancy. Bactericides and fixed copper applications can decrease the damage of the bacterial diseases, but use copper compounds only in the early season as they can blemish fruit destined for market. Best disease control is obtained with several foliar applications of fungicides or bactericides coordinated with periods of high infection hazard. Fungicides or bactericides alone do not provide good control. A combination of sanitation and fungicide application is recommended.

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5. Chlorosis

Laurie J. Stepanek, Mark O. Harrell, and Rachel A. Allison; revised from Mark O. Harrell and Mark W. Andrews (Riffle and Peterson 1986)

Chlorosis describes any condition in which leaves or needles develop an abnormally light green or yellow color. The most common cause of chlorosis in trees is a deficiency of usable iron, either in the soil or within the tree. But the problem is complex, often involving multiple factors, and can be difficult to correct.

Hosts and Distribution

Chlorosis may develop wherever trees are grown in alkaline soils (soils with a pH greater than 7). Alkalinity generally increases from east to west across the Great Plains, and with it, incidence of chlorosis. Chlorosis is also common in urban areas, where excavation during construction of buildings brings alkaline subsoil to the surface and where poor cultural practices lead to unhealthy soils and root damage in trees.

Pin oak (*Quercus palustris*) and silver maple (*Acer saccharinum*) are commonly affected by chlorosis, but many other tree species are susceptible as well (table 5-1). Trees susceptible to chlorosis differ somewhat among the various areas of the Great Plains.

Table 5-1—Partial list of trees susceptible to chlorosis.

Common name	Scientific name	Common name	Scientific name
Amur maple	*Acer ginnala*	Crabapple	*Malus* spp.
Red maple	*A. rubrum*	Pine	*Pinus* spp.
Silver maple	*A. saccharinum*	Peach	*Prunus persica*
Freeman maple	*A.* x *freemanii*	Pear	*Pyrus* spp.
Serviceberry	*Amelanchier* spp.	Aspen	*Populus* spp.
River birch	*Betula nigra*	Cottonwood	*Populus* spp.
Catalpa	*Catalpa speciosa*	Poplar	*Populus* spp.
Flowering dogwood	*Cornus florida*	Swamp white oak	*Quercus bicolor*
Hawthorn	*Crataegus* spp.	Pin oak	*Q. palustris*
Holly	*Ilex* spp.	Northern red oak	*Q. rubra*
Juniper	*Juniperus* spp.	Baldcypress	*Taxodium* spp.
Sweetgum	*Liquidambar styraciflua*	Yew	*Taxus* spp.
Magnolia	*Magnolia* spp.		

Symptoms and Signs

The degree of yellowing varies from a yellowish-green in only slightly chlorotic leaves to lemon yellow and almost white in leaves that are moderately to severely chlorotic (figs. 5-1 and 5-2). The yellowing is most intense in the interveinal areas of leaves while the veins remain green. Brown necrotic spots may develop between the veins and along the leaf edges, giving the appearance of leaf scorch (fig. 5-3), and leaves may be abnormally small.

Symptoms of chlorosis may be uniform throughout the entire tree or be confined to one or a few branches. If chlorosis continues for several years, shoot growth is reduced, branches begin to die back, and the tree may eventually die (fig. 5-4). Symptoms among trees of the same species growing on the same site can vary; even normal green trees can be located next to severely chlorotic ones owing to soil variations within the site and genetic differences among trees (fig. 5-5).

Figure 5-1—Chlorotic Freeman maple leaves showing typical yellow interveinal areas and green veins (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 5-2—Chlorotic baldcypress foliage (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 5-3—Necrotic spots, which may develop along leaf edges and between the veins (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 5-4—Branch dieback in a chlorotic crabapple. Symptoms developed shortly after an irrigation system was installed and the landscape was overwatered (Rachel A. Alison, Nebraska Forest Service, used with permission).

Figure 5-5—Variation in symptoms among oak trees (Mark O. Harrell, Nebraska Forest Service, used with permission).

Cause

Deficiency of iron is the most common cause of chlorosis of trees in the Great Plains. In most cases, iron in the soil is present in sufficient quantity, but under alkaline conditions, it becomes less soluble and thus less available for plant uptake. Even if absorbed, iron may remain in a form that the tree is unable to use. Iron is used in the production of chlorophyll, so a deficiency of iron prevents the leaves from producing the normal amount of chlorophyll.

The availability of other micronutrients such as manganese and zinc also is limited in alkaline soils and therefore may lead to chlorosis as well. An excess of some nutrients, especially nitrate-nitrogen and phosphorus, can cause chlorosis by interfering with the uptake of iron in the soil or its movement and utilization in the tree. High levels of bicarbonates, such as those found in calcareous soils and many sources of irrigation water and groundwater, interfere with these processes as well. A lack of nitrogen or other macronutrients may cause chlorosis, but the focus of this discussion is on micronutrient deficiencies associated with high pH soils.

Chlorosis frequently develops in wet, compacted soils, such as those found in overwatered landscapes (fig. 5-4). Saturated soils limit gas exchange, allowing the buildup of carbon dioxide, which leads to increased bicarbonate levels. Extensive rains in spring can

cause temporary chlorosis. Planting trees too deeply, piling soil over roots, and digging or trenching near trees can damage roots, resulting in limited nutrient uptake and the development of chlorotic tissues.

Damage

Trees with severe chlorosis typically decline and die over several years. In many areas of the Great Plains some species, especially pin oak, are no longer being planted because this disorder severely reduces their chances of survival.

Management

Chlorosis is difficult to correct. Soils resist changes in pH, and several factors that contribute to the chlorotic condition may be involved. Treatments that work in one area may not work in other areas, and some trees respond better to certain treatments than others. A general discussion of control options is provided here. Consult your local university extension office, state forestry department, or the U.S. Forest Service for specific information.

Cultural practices should be included in any treatment plan. Water trees thoroughly but infrequently if it does not rain, which will give time for the soil to drain and allow gas exchange to occur. Automatic sprinklers that run daily or every other day can severely stress or kill trees. Woodchip mulches improve the soil environment for tree roots, mycorrhizal fungi, and other beneficial soil microbes that aid in nutrient uptake, as well as provide a natural supply of micronutrients. Use fertilizers with prudence. Trees generally do not need extra nitrogen fertilizer if the lawn is already being fertilized.

When planting trees, choose those that are more adapted to alkaline soils. Specific trees will vary widely across the Great Plains so consult local sources for recommendations. Avoid pot-bound trees with circling roots and prepare a wide, shallow planting site (not a deep hole) to keep the main roots level with the soil surface. Remove all twine, wire, and burlap and spread the roots out as much as possible, which will help the tree establish a healthy root system.

Several chemical treatments are available to help correct chlorosis, including soil applications, trunk injections or implants, and foliar sprays. Soil and leaf tissue tests can provide information on pH and nutrient levels, which can help with treatment selection.

Soil treatments

Micronutrient deficiency in soils is uncommon. The goal of most soil treatments, therefore, is to lower the soil pH to improve nutrient availability. Elemental sulfur is commonly used to achieve this. Soil bacteria convert the sulfur to acid, which lowers the pH. Results are variable and slow to work (symptoms may not improve for one to two years after treatments), but the effects can last several years.

A broadcast application of sulfur can be applied below the canopy of the tree. High rates are needed: one to five pounds or more per 100 ft^2, depending on initial pH and soil texture. Raking the sulfur into mulched areas and aerating turf areas will increase sulfur/soil contact and speed up the acidification process. Watering thoroughly after application and during hot, dry weather will help prevent turfgrass burn. Annual followup applications of sulfur can help maintain desired pH levels. Broadcast applications may be less successful on clay soils, which are highly buffered and more resistant to pH changes than sandy soils.

Alternatively, sulfur may be combined with iron sulfate or a micronutrient mix and placed into holes dug in the ground around the tree. Pockets of soil are created that are rich in readily available iron and other micronutrients. This method may be a better option for clay soils.

Calcareous soils contain high levels of free lime (calcium carbonate) and are extremely difficult to correct. Excessive amounts of sulfur are needed to neutralize the free lime before the pH can be lowered. To test for free lime, moisten a small amount of dry soil with vinegar. If the mixture fizzes or bubbles, it has free lime.

Another soil-applied chemical specifically for trees deficient in iron is iron chelate, which provides iron in a form easily absorbed by plants. Look for products containing

EDDHA (FeEDDHA), which works better than other chelates when soil pH is above 7.2. This chelate has been shown to be effective even in calcareous soils. Iron chelates generally provide control for one to two years.

Trunk injections and implants

In areas where soil treatments may be ineffective or impractical, trunk injections and implants are commonly used to treat chlorosis. For injections, holes are drilled into the trunk flare, and a liquid micronutrient product is injected into the holes; or the product is delivered to the tree through a needlelike injection tool. Implants are plastic cartridges containing a powdered material and are placed into holes drilled into the trunk. Most injections and implants are effective for one to three years.

One major disadvantage of injections and implants is the physical damage caused by the drilling and the internal damage caused by the chemical itself. Treatments should not be re-applied until the drill wounds seal over with new trunk tissue, which may take more than a year. Injection methods that use small, shallow holes are less damaging. Although some products are available to consumers, the potential for failure and significant tree injury is high. Injection treatments performed by a certified arborist who is well trained in the procedure can reduce these risks.

Spray treatments

Spray treatments for chlorosis consist of a solution of iron or manganese sulfate or a chelated compound. This treatment improves the condition of only those leaves present when the foliage is sprayed. It has little or no effect on leaves formed after spraying, and the effectiveness does not carry over into the next growing season. Used in combination with a soil treatment, a spray can provide iron to the tree until the slower acting soil treatment becomes effective.

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6. Herbicide Damage to Trees and Shrubs

Aaron D. Bergdahl and William R. Jacobi

Intensified use of herbicides in recent decades has increased the hazard to nontarget vegetation. Disorders or mortality caused by herbicides is particularly prevalent among sensitive tree species in residential and agricultural settings where herbicides are commonly used. Areas at higher risk for exposure include windbreaks, sites near rights-of-way, railroads, roadsides, or areas treated for noxious weed control.

Herbicide Classification and Modes of Action

Herbicides that commonly cause injury to nontarget plants fall into three general groups: 1) those that are used to kill a certain type of established vegetation (post-emergence selective herbicides), 2) herbicides intended to prevent germination of seeds or emergence of seedlings (preemergence selective herbicides) or 3) those that kill all vegetation (nonselective herbicides). Herbicides are classified as either contact herbicides, which affect only the points of contact with the active ingredient, or systemic herbicides, which are absorbed, are translocated, and typically show a broader pattern of symptoms. Herbicides kill plants by interfering with essential plant functions; this interference causes disruption of cell components and inhibits proteins essential to photosynthesis, growth, and cell formation. The modes of action of herbicides most commonly found to damage trees in the Great Plains are described here.

Plant growth regulator herbicides are typically used on turf to selectively kill broadleaf weeds. Herbicides in this group include the phenoxyacetic acids (2,4-D, 2,4-DP, MCPA, MCPP, and related compounds), benzoic acids (for example, dicamba), and pyridine carboxylic acids (for example, clopyralid, fluroxypyr, picloram, and triclopyr). Herbicides of this type can move systemically to growing points, where they disrupt plant growth

Figure 6-1—(A) Phenoxy-type herbicide injury to green ash (Aaron D. Bergdahl, North Dakota Forest Service, used with permission); (B) Simazine damage on poplar (John Sharpe, Prairie Farm Rehabilitation Administration, Indian Head, Saskatchewan, used with permission); (C) 2,4-D damage on maple (Robert Stack, North Dakota State University, retired, used with permission); (D) Glyphosate plus imazapyr damage on linden (Michael Schomaker, Colorado Forest Service, retired, used with permission).

hormone balance, causing various developmental deformities such as narrow elongated growth (leaf strapping), cupping, and twisting (figs. 6-1a, c, 6-2, 6-3, 6-5, and 6-7a and b).

Exposure to thin barked species by these chemicals may result in deformation, blistering, and cankering of bark (fig. 6-4). Symptoms develop within a few days to a week after exposure. Pyridine carboxylic acid herbicides can remain active in the environment (residual period) for more than one year.

Figure 6-2—Symptoms of 2,4-D damage showing twisted new growth and cupped leaves on (A) linden, and (B) elm (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 6-3—Strapping and discoloration of bur oak (*Quercus macrocarpa*) foliage resulting from herbicide exposure (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 6-4—Blistering of bark on honeylocust from herbicide exposure (William R. Jacobi, Colorado State University, used with permission).

Commonly used nonselective herbicides belonging to the group known as photosynthesis inhibitors include the following (with examples in parentheses): triazines (atrazine, simazine), phenylureas (tebuthiuron), uracils (terbacil), benzothiadiazoles (bentazon), nitriles (bromoxynil), and carbamates (desmedipham). These herbicides cause bleaching of leaves followed by leaf browning and drying out (fig. 6-1b). Effects may be exaggerated in higher-pH soils and the residual period may be several months.

Nonselective herbicides, also called bare ground or total vegetation control herbicides, function via a variety of modes of action, ranging from interfering with amino acid formation to disrupting lipid formation (plant cell membrane disruption). Nonselective herbicides like glyphosate and bromacil are applied directly onto actively growing target plants. The development of crop varieties that are glyphosate resistant has led to greatly expanded use of these compounds in recent years, resulting in increased reports of glyphosate damage to trees, especially in field windbreaks (figs. 6-1d and 6-6). Nonselective herbicides may have long-lasting residual activity, so effects can be persistent. Some products also include multiple chemicals and therefore multiple modes of action, creating variable symptoms that are not as diagnostic.

Preemergence herbicides are applied directly to soil or on turf and then incorporated into the soil, usually with water, to kill germinating weed seedlings. Preemergence herbicides are less commonly associated with tree disorders in the northern Great Plains, mainly because of the mode and timing of application.

Symptoms

Symptoms vary depending on the type and concentration of the chemical, frequency of use, species and condition of plants, and the timing of chemical application. The more common expressions of injury from phenoxy or hormonal-type herbicides include cupped leaves, parallel leaf venation on normally net-veined leaves, chlorosis, nastic growth, and wavy or curled leaf margins (figs. 6-1a and 6-1c, 6-2, and 6-5). Lindens (*Tilia* spp.), honeylocust (*Gleditsia triacanthos*), redbud (*Cercis canadensis*), and boxelder (*Acer negundo*) are among those that readily exhibit these effects.

Figure 6-5—Picloram damage to catalpa (*Catalpa speciosa*) (Ned Tisserat, Colorado State University, used with permission).

Leaves of some trees, such as pin oak (*Quercus palustris*), become waxy and stiff, while maples may develop pebbled foliage that appears weather-beaten. Loss of apical growth is typical of phenoxy or hormonal herbicide injury. Affected trees may suffer a gradual crown dieback and eventually die. Lateral leaf development may also be hindered. Exposure to 2,4-D may result in the production of fewer than normal leaves, flowers, and fruits. Leaf scorch can indicate herbicide injury, particularly when exposure is heavy. Ash (*Fraxinus* spp.) and cottonwood

Figure 6-6—Glyphosate damage to red oak (*Quercus rubra*) and adjacent white spruce (*Picea glauca*) (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 6-7—Examples of herbicide damage to evergreens. (A) Spiral pattern typical of a point exposure to herbicide in spruce; Aaron D. Bergdahl, North Dakota Forest Service, used with permission), (B) MCPA direct exposure to Scots pine (*Pinus sylvestris*) (James A. Walla, Northern Tree Specialties, used with permission); (C) Spruce injury from soil sterilant exposure (William R. Jacobi, Colorado State University, used with permission).

(*Populus deltoides*) may exhibit scorched leaves without expressing broader effects of herbicide exposure. High rates of exposure of 2,4-D to Siberian elm (*Ulmus pumila*) have been shown to result in bark abnormalities. A single 2,4-D exposure may produce injury for two years or more. Honeylocust bark can become spongy, woody swellings (galls) can form at branch crotches, and the cambium may be killed by high concentrations of phenoxy herbicides (fig. 6-4).

Evergreens are generally resistant to phenoxy herbicides, but they may be injured under certain circumstances. On conifers symptoms can include abnormal swelling, twisting, and curling of branchlets, as well as bleaching, browning, and casting of needles. Broadleaf trees and especially conifers have shown sensitivity to dicamba often used in "weed and feed" lawn applications. Dicamba can be absorbed via the roots and injury to trees ranges from leaf distortion, discoloration, and defoliation to branch dieback, often expressed in a spiral pattern in conifers (fig. 6-7a), and sometimes tree mortality. When farm-grade broadleaf weed control products that frequently combine more than one herbicide mode of action are applied to lawns, they may cause tree mortality in landscape settings, especially on lighter soil types.

Nonselective herbicides, which are used for bare ground or total vegetation control, tend to halt growth and cause chlorosis of new and old foliage. At high doses, foliar browning, foliar drop, and dieback of twigs and branches may occur (fig. 6-7c). Trees and shrubs injured by these herbicides are less likely to recover than trees injured by plant growth regulator herbicides. While light exposure to some herbicides may not cause noticeable symptoms or damage, partial or total mortality can result from exposure to nonselective herbicides.

Diagnosis of herbicide damage can be aided by careful collection and analysis of residue samples. The cost of analysis is usually prohibitive, but if analysis is required, the testing laboratory will need to know what herbicides to test for in the plant samples. It is important that the samples be collected as soon as possible after exposure. When possible, residue samples should be kept frozen until they reach the laboratory. It may be advisable to contact an analytic laboratory for specific instructions on collection and care of samples.

Damage

All possible sources of chemical exposure should be considered when dealing with suspected herbicide damage. Harmful exposure to herbicides can result from drift of the spray particles, movement of volatiles, application to the soil or movement in soil (or water) resulting in root exposure, and direct application. Spray drift volatiles can move considerable distances, resulting in damage several miles from where the herbicide was applied. Direct application may cause damage if sensitive trees are not carefully avoided.

In urban areas, desirable plants are most likely to be damaged by repeated lawn sprays or fertilizers that contain herbicides. Damage is also commonly associated with soil applied herbicides used around driveways, sidewalks, and structures to remove vegetation. In situations where damage has resulted from an apparent misuse of herbicides, it may be appropriate to contact your state's pesticide regulatory agency or department of agriculture for guidance.

Management

Desirable trees located where herbicide exposure is likely will require protection. Persons who apply herbicides should be informed of locations of desirable trees, and products that pose a minimal risk to the trees should be used. Exercise extra caution to ensure proper calibration of equipment and application. Herbicides that are mishandled or are applied under improper conditions often end up injuring nontarget plants. Avoid misapplication to nontarget plants by applying sprays only when it is not windy so that drift of herbicide will be unlikely. Additionally, vapor drift can occur with many plant growth regulator herbicides if temperatures are too high during application or soon afterwards.

When applying broadleaf herbicides to turf, remember that tree roots can extend up to five times beyond the diameter of the tree crown and herbicides can be absorbed by tree roots present in the area of application.

Once exposure has occurred to plants, little can be done to minimize the initial effects. Washing herbicide off foliage can be effective if it is done immediately after exposure. Soil-active herbicides can be deactivated on a limited scale with activated charcoal or similar products. Generally, any effort that promotes tree vigor should help reduce the effects of the herbicide exposure. Trees weakened by herbicides may be predisposed to insects, pathogens, and other types of environmental damage. Many trees can survive moderate herbicide damage if given care after exposure. Trees should be given at least one year to recover before declaring the tree not salvageable. Inappropriate and high, acute dosages of herbicides can kill trees, especially if the trees are stressed from growing under droughty conditions along roadsides or in windbreaks. Always check with your state's pesticide regulatory agency for current registered use of pesticides and strictly follow all aspects of product labels.

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7. Taphrina Diseases of Shade and Fruit Trees

Mark L. Gleason, Hayley M. Nelson, and Megan M. Kennelly

Species of fungi in the genus *Taphrina* infect a wide range of woody plants, but primarily oak (*Quercus* spp.), maple (*Acer* spp.), elm (*Ulmus* spp.), plum (*Prunus* spp.), and peach (*Prunus persica*) in the Great Plains. These fungi cause conspicuous blistering and browning of leaves as well as distortion of fruit and defoliation.

Hosts and Distribution

Species of fungi in the genus *Taphrina* are host specific, meaning that each one infects only certain tree species. *Taphrina caerulescens* infects many oaks, primarily those in the red oak group: red (*Q. rubra*), scarlet (*Q. coccinea*), black (*Q. velutina*), and pin oak (*Q. palustris*). Sometimes it also infects those in the white oak group: bur (*Q. macrocarpa*) and white (*Q. alba*) oak. Within *T. caerulescens* there are subgroups that are specific to certain types of oaks.

Taphrina carveri (named after the famous plant scientist George Washington Carver, who originally described its sexual spores) attacks maple species, primarily red (*A. rubrum*) and silver (*A. saccharinum*) maple, and *T. ulmi* attacks elm leaves. Both *T. communis* and *T. pruni* cause plum pockets on plum fruit, but *T. communis* is much more prevalent in the Great Plains. A number of cultivated and wild plums are susceptible, but cultivated plums are less so. Peach leaf curl, caused by *T. deformans*, can be a damaging disease in commercial and backyard peach and nectarine (*Prunus* spp.) orchards.

Figure 7-1—Leaf blistering and discoloration caused by oak leaf blister (Cornell University, used with permission).

Figure 7-2—Yellow and brown discoloration caused by oak leaf blister (Cornell University, used with permission).

Symptoms and Signs

Most Taphrina diseases attack leaves, and symptoms typically appear in late spring to early summer. **Oak leaf blister** appears on the upper leaf surfaces as circular, raised, wrinkled, yellowish-white spots, and on the leaf undersides as yellowish-brown to gray depressions of the same size directly below the raised areas (figs. 7-1 and 7-2). The blistered tissue later turns reddish brown with yellow margins, and finally becomes dull brown. When several blisters merge, leaves may become curled and fall off prematurely. **Maple leaf blister** produces grayish-brown to black, irregularly shaped spots that are sometimes slightly raised. As the leaf spots age, their centers often turn a lighter color (fig. 7-3). Multiple infections cause the leaf shape to become distorted (fig. 7-4). The leaf spots' somewhat rounded shape helps to distinguish them from spots caused by maple anthracnose, which tends to produce irregularly shaped, angular leaf spots. **Elm leaf blister** causes yellow spots that enlarge into dull brown, somewhat angular spots with light green halos (fig. 7-5) and some areas of puckered, light green tissue (fig. 7-6).

Figure 7-3—Coalescing leaf spots caused by maple leaf blister (Sabrina Tirpak, Plant Diagnostic Lab, Rutgers University, used with permission).

Figure 7-4—Figure 4. Distortion caused by maple leaf blister and anthracnose. (Gail Ruhl, Purdue University, used with permission).

Figure 7-5—Leaf spots caused by elm leaf blister (Megan Kennelly, Kansas State University, used with permission).

Figure 7-6—Blistering and browning of leaf caused by elm leaf blister (Megan Kennelly, Kansas State University, used with permission).

Symptoms of **plum pockets** are most noticeable on fruit. Small, whitish spots appear on young fruit, then expand in size to cover the fruit. Seed fails to form in the infected fruit, and the fruit become hollow and enlarge to many times their normal size (fig. 7-7). They sometimes take on a red to grayish cast and a thickened, leathery texture (fig. 7-8). **Peach leaf curl** results in puckered to severely distorted, downward-curling leaves that are crisp in texture and show red and purple discoloration (fig. 7-9). Later, the leaf surface turns grayish as the fungus begins to produce spores. Diseased leaves eventually die, fall off the tree, and are replaced by a new crop of leaves in early to midsummer. Extensive defoliation can lead to a reduced fruit crop the following year. Though less apparent, fruits and flowers can also be directly infected, often falling off the tree. Young shoots can also be stunted and deformed.

Disease Cycle

Taphrina fungi overwinter as dormant spores in bud scales and bark crevices. These spores germinate in spring during cool, rainy periods and infect young leaves and fruit. The spores are produced on infected tissues and later spread by wind and rain, but do not cause additional infections during the same growing season. Instead, surviving spores remain dormant until favorable weather occurs the next spring. As a result, Taphrina diseases have only one cycle of infection per year.

Damage

Damage to trees is primarily cosmetic, and most Taphrina diseases do not endanger plant health. On peach and nectarine, however, defoliation by peach leaf curl can weaken trees and reduce yield if not controlled.

Figure 7-7—Plum fruit infected by plum pockets is yellow and abnormally enlarged (Thomas Whitlow, Cornell University, used with permission).

Figure 7-8—Gray discoloration of plum fruit caused by spores of the plum pockets fungus (Aaron Bergdahl, North Dakota Forest Service, used with permission).

Figure 7-9—Discoloration and puckering caused by peach leaf curl (Sabrina Tirpak, Plant Diagnostic Lab, Rutgers University).

Management

Most Taphrina diseases do not need to be controlled because their threat to tree health is negligible. The exception is in peach and nectarine orchards, where peach leaf curl can cause economic losses; in those situations, a single fungicide spray, applied between the time that 90 percent of leaves have been shed in fall and the time that leaf buds begin to swell in spring, is highly effective at controlling the disease. Appropriate water and fertilizer may reduce stress and promote tree vigor in affected trees. Although full resistance is not available, some peach cultivars have reduced susceptibility.

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8. Leaf Tatter and Leaf Tatters

Laurie J. Stepanek and Mark O. Harrell

Leaves of broadleaf trees sometimes sustain various types of abiotic injury that cause the leaves to appear lacy or tattered. This tattering condition has been referred to by several similar terms, which are sometimes used interchangeably. "Tatter" (also "leaf tatter" and "tatter-leaf") appears in early scientific literature and service publications, where it is broadly applied to tattering symptoms that are associated with unknown abiotic causes and that affect a variety of tree species. The similar term "tatters" (or "leaf tatters") is found in more recent research and describes damage caused by specific herbicides on a limited number of species.

In this discussion, "leaf tatter" will refer to a general abiotic tattering disorder without regard to cause or host, and "leaf tatters" will be used exclusively for the specific herbicide-induced disorder affecting oak (*Quercus* spp.) and common hackberry (*Celtis occidentalis*).

Hosts and Distribution

Leaf tatter occurs on many broadleaf trees throughout the Great Plains, including species of maple (*Acer*), oak, linden (*Tilia*), birch (*Betula*), ash (*Fraxinus*), crabapple (*Malus*), hawthorn (*Crataegus*), dogwood (*Cornus*), and hackberry. Maple species, especially sugar maple (*A. saccharum*), are particularly prone to leaf tatter. Susceptible cultivars include 'Green Mountain®,' 'Wright Brothers,' 'Bonfire,' 'Endowment,' and 'Fairview.'

Leaf tatters, the herbicide-induced condition of tattering, occurs on hackberry and oak, particularly bur (*Q. macrocarpa*), white (*Q. alba*), and northern red (*Q. rubra*) oak. Leaf tatters occurs principally in midwestern states but is known to occur in the eastern Great Plains. Many cases occur in rural areas or near crop fields where agricultural herbicides are used. Native stands of trees as well as planted ornamental trees can be affected.

Symptoms

Leaf tatter may show a range of symptoms. One symptom found in many species is the presence of numerous holes between the veins (fig. 8-1). The holes may extend to the margins, affecting the form of the leaf and creating a ragged appearance.

Other symptoms, often seen in maples, are shredding, tearing, or absence of tissue between the lobes of the leaves (fig. 8-2). The tips of the lobes may develop a water-soaked appearance, followed by browning, drying, and disintegration of the tissues. The palmate shape typical of maple leaves may be lost when tatter is extensive (fig. 8-3).

Figure 8-1—Leaf tatter on birch associated with a late spring freeze (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 8-2—Leaf tatter of sugar maple showing missing tissue between the lobes (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Symptoms of **leaf tatters** on oak and hackberry appear on young leaves as they unfold and begin to expand in spring. Leaf edges and interveinal areas turn brown, and leaves may curl or pucker. As the damaged parts of the leaves die and drop off, the leaves develop a lacy appearance (fig. 8-4). Leaf damage can vary from only a few scattered holes to leaves that are little more than veins (figs. 8-5 and 8-6). Trees severely affected by tatters have thin crowns in spring (fig. 8-7). A new flush of undamaged leaves usually develops, although some twig and branch dieback may be present.

Cause

Leaf tatter has been associated with several environmental factors, although causation in many cases has not been shown. Frost or freeze that occurs in spring as buds are beginning to open may be a potential cause for some cases. Injury is thought to be initiated when bud scales loosen and tiny areas of tender leaf tissue become exposed to cold temperatures. As leaves unfold and expand, the injured or killed areas develop into holes (fig. 8-1).

Wind tunnel experiments have produced tatterlike symptoms in maple, and tattering is frequently observed on young, tender foliage of many trees following strong winds in spring. One study of sugar maple leaves suggests that the anatomical makeup of the leaves of some cultivars may play a role in their susceptibility to tatter.

Figure 8-3—Sugar maple with missing leaf tips resulting in loss of palmate shape (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 8-4—Bur oak leaf with tatters. Dead tissue falls away, leaving mostly veins (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 8-5—Hackberry leaves with varying degrees of damage from tatters (Laurie J. Stepanek, Nebraska Forest Service, used with permission)

Figure 8-6—Bur oak leaves reduced to veins (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 8-7—Thin crowns of hackberry caused by leaf tatters (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Leaf tatters of oak and hackberry has been shown in studies to be caused by herbicides containing acetochlor, metolachlor, and dimethenamid. These herbicides are frequently used in corn production, which could explain the high number of tatters reports occurring in the Midwest, where corn production is common.

Leaf tatters symptoms develop when herbicide exposure occurs as the leaves are unfolding and expanding. Leaves still in the bud and leaves fully expanded are not affected. Exposure may potentially occur through spray drift, volatilization, or contaminated rain or dust, thereby affecting trees both near and far from the application.

Damage

Leaf tatter and leaf tatters typically affect only the appearance of the trees. Severely affected trees may produce a new flush of leaves if they have sufficient energy reserves, but the stress of leaf loss can make trees more susceptible to other damaging agents. Branch dieback or tree death may occur in trees that have been severely affected over several years, especially if they have suffered prior stress, such as from drought or other poor growing conditions.

Management

Sugar maple selections less susceptible to maple tatter include Legacy® Maple (*Acer saccharum* 'Legacy', PP4,979) Commemoration® Maple (*Acer saccharum* 'Commemoration', PP5,079) Oregon Trail® Maple (*Acer saccharum* 'Hiawatha1') John Pair Maple, (*Acer saccharum* 'John Pair') Autumn Splendor Maple (*Acer saccharum* 'Autumn Splendor') Flashfire® Maple (*Acer saccharum* 'JFS-Caddo2', PP23,361) 'Oregon Trail' is a northeastern Kansas selection; 'Fall Fiesta' originated in Minnesota; and the last three are Caddo maples, which are a population of sugar maple in western Oklahoma.

Oak and hackberry tatters may be reduced by application of crop herbicides before or well after budbreak to avoid sensitive stages of leaf growth. Large droplet size, low boom heights, and the addition of drift inhibitors can help minimize drift. Soil incorporation can minimize volatilization.

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9. Marssonina Leaf Spot and Blight of *Populus* Species

Michael E. Ostry and Jared M. LeBoldus; revised from John E. Watkins and David S. Wysong (Riffle and Peterson 1986)

Marssonina leaf spot, a widespread and serious disease of native and hybrid poplars (*Populus* spp.), can severely defoliate susceptible genotypes well before normal leaf drop. This disease is caused by at least four different species of fungi in the genus *Marssonina*.

Hosts and Distribution

There are four relatively well studied species of *Marssonina* affecting poplar in Europe, North America, Asia, Australia, and New Zealand: *M. brunnea* (teleomorph = *Drepanopeziza punctiformis*), *M. castagnei* (*D. populi-albae*), *M. populi* (*D. populorum*), and *M. balsamiferae* (teleomorph unknown). These fungi affect balsam poplar (*P. balsamifera*), plains cottonwood (*P. deltoides* subsp. *monilifera)*, quaking aspen (*P. tremuloides*), white poplar (*P. alba*), and various hybrid poplars. *Marssonina* species are known to be host specific. On willows (*Salix* spp.), *M. brunnea* and *M. salicicola* (teleomorph = *Drepanopeziza sphaerioides*) are the major species of importance. A fifth taxon, *M. populicola*, is known to affect poplars in China. Species in section *Populus* (aspens, white poplars) are susceptible to *M. brunnea* f. sp. *trepidae,* and species in section Aigeiros (cottonwoods, black poplars) are susceptible to *M. brunnea* f. sp. *brunnea. M. balsamiferae* was reported occurring on *P. balsamifera* in Ontario and Manitoba. *M. castagnei* infects *P. alba*, and *M. populi* is common on *P. tremuloides*. *Marssonina populicola* has been reported in China on *P. davidiana*, *P. euphratica*, *P. euramericana*, *P. laurifolia*, *P. pseudosimonii*, *P. simonii*, and *P. tomentosa*. Severe defoliation of *Populus* species and their hybrids by *Marssonina* has been reported across the Great Plains.

Symptoms and Signs

Dark brown flecks (fig. 9-1), often with yellow margins (fig. 9-2), appear on leaves within a few weeks of leaf emergence. These spots gradually enlarge to 1 to 2 mm in diameter. Individual spots on severely affected leaves may coalesce to form angular, necrotic blotches. Diseased leaves on affected trees appear smaller than normal, turn yellow to bronze, and fall prematurely (fig. 9-3). Symptoms develop progressively upward into the crown. If viewed from a distance, the diseased leaves appear bronzed. On highly susceptible clones lens-shaped lesions develop on petioles (fig. 9-4) and current-year shoots (fig. 9-5). Subcuticular fruiting bodies (acervuli) containing asexual spores (conidia) measuring 11 to 16 µm × 3.5 to 7.0 µm (fig. 9-6) in a white gelatinous mass form within leaf, petiole, and stem lesions of infected hosts.

Figure 9-1—Angular leaf spots caused by *Marssonina brunnea* on a poplar leaf (Michael E. Ostry, U.S. Forest Service).

Figure 9-2—Marssonina leaf spot on aspen with necrotic leaf spots with yellow margins (Joseph G. O'Brien, U.S. Forest Service).

Figure 9-3—Healthy green poplar leaf and yellow poplar leaf infected by *Marssonina brunnea* (Michael E. Ostry, U.S. Forest Service).

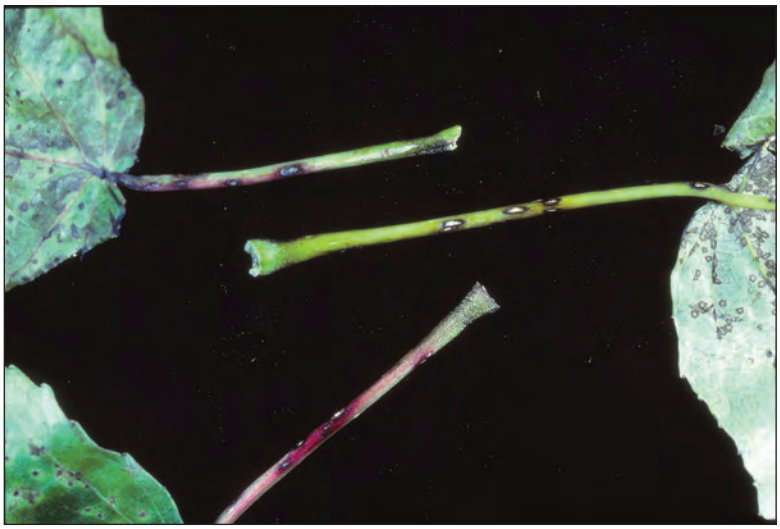

Figure 9-4—Elliptical lesions on petioles of poplar leaves caused by *Marssonina brunnea* (Michael E. Ostry, U.S. Forest Service).

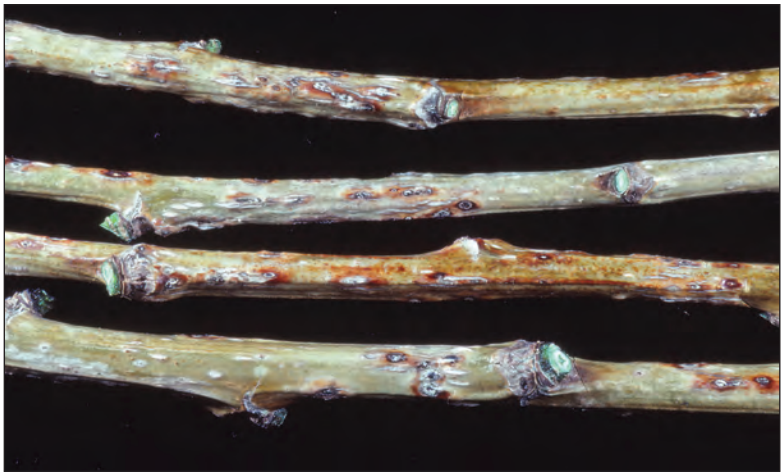

Figure 9-5—Lesions on poplar stems caused by *Marssonina brunnea* (Michael E. Ostry, U.S. Forest Service).

Figure 9-6—Conidia of *Marssonina brunnea.* Scale bar is 10 µm (Michael E. Ostry, U.S. Forest Service).

Disease Cycle

The fungus overwinters in fallen leaves and in infected shoots. In spring, ascospores produced in the leaves and conidia from lesions on the shoots are released in wet weather. Leaves and new shoots are infected throughout the growing season by rain-splashed conidia, intensifying disease severity. Epidemics coincide with extended or frequent wet weather.

Damage

In nurseries and plantations, Marssonina leaf spot causes only slight damage to cuttings and young seedlings until sufficient inoculum develops on infected shoots and on fallen leaf debris. In more established plantings and in native stands, repeated outbreaks result in branch and twig dieback and predispose trees to other pathogens or pests and to injury from low temperatures.

Management

The preferred method of control is to plant clones resistant to the disease. Pruning diseased twigs and branches and removal of fallen leaves may also limit disease incidence and severity. Fungicide has also proven to be an effective means of control; however, there is currently no fungicide labeled for use in plantations. To avoid spreading this pathogen to new areas, cuttings should only be taken from disease-free shoots. However, it is important to remember that infected leaves and shoots may remain asymptomatic and that this pathogen is seed borne. As a result, care must be given to avoid inadvertent shipment of infested seed or asymptomatic plant material.

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10. Melampsora Leaf Rust of Cottonwood, Aspen, and Willow

Phillip A. Mason and Jared M. LeBoldus; revised from Glenn W. Peterson and Robert W. Stack (Riffle and Peterson 1986)

Cottonwood (*Populus deltoides*), aspen (*Populus* spp.), and willow (*Salix* spp.), including both native species and their hybrids, are susceptible to infection by the fungal leaf rust pathogens in the genus *Melampsora*. On highly susceptible trees the disease results in premature leaf drop, which can be deleterious to tree health and vigor.

Hosts and Distribution

Melampsora leaf rust is widely distributed, affecting numerous *Populus* species, hybrids, and clones from North Dakota to Texas. In the Great Plains the *Melampsora* pathogen is often found on various *Populus* species and their hybrids, quaking aspen (*P. tremuloides*), and willow (*Salix* spp.). The alternate host of these rust species is larch (*Larix* spp.). The most common rust species in the Great Plains are *Melampsora medusae* on cottonwood and aspen and *Melampsora epitea* on willow. However, other *Melampsora* species have been reported to be expanding their known host ranges, and natural rust hybrids are occurring.

Symptoms and Signs

The most commonly recognized sign of this disease is orange pustules (uredinia) on the lower (abaxial) leaf surface (figs. 10-1 and 10-2). The orange powdery pustules are composed of a mass of urediniospores that usually form on the underside of leaves in the lower crown of trees. As the leaves become severely diseased, they begin to senesce. At this time brown to black crusts (telia) begin to form on leaves among the orange uredinia and become prevalent on fallen leaves. Premature leaf drop occurs in midsummer.

Disease Cycle

Melampsora medusae and *M. epitea* require two hosts to complete their life cycle. The fungi cycle most often between the genera *Populus* and *Larix*. In spring the disease cycle begins on fallen, infected *Populus* leaves, when the overwintering teliospores on telia germinate, giving rise to basidia, another spore-bearing structure where basidiospores form. The basidiospores can infect only the conifer host (larch), which later gives rise to aecia, a spore-bearing structure on larch needles where aeciospores develop. Aeciospores are windblown and infect the *Populus* or *Salix* host (cottonwood, aspen, or willow). Uredinia then develop and production of the urediniospores occurs. These spores are released and infect other poplar or willow hosts (figs. 10-1 and 10-2) and often re-infect the same host plant continuously until leaf drop. Telia form on the leaves in late summer, and the infected, fallen leaves serve as the overwintering site for the pathogen, completing the complex life cycle of the fungus. In many areas of the Great Plains where the larch is not present, it is believed that the urediniospores are spread via wind in a north to south direction.

Figure 10-1—Orange powdery urediniospores of *Melampsora* on the abaxial surface of willow leaves (Phillip A. Mason, Animal and Plant Health Inspection Service).

Figure 10-2—Orange powdery urediniospores of *Melampsora* on the abaxial surface of a hybrid poplar leaf (Kelsey L. Dunnell, North Dakota State University, used with permission).

Damage

Depending on leaf rust severity, premature leaf drop can occur, resulting in loss of vigor and reducing the tree's ability to defend against other stresses. Premature defoliation in successive years can reduce carbohydrate reserves and contribute to the decline of *Populus* species in the Great Plains. Although *M. epitea* has been reported to cause stem cankers on young willow stems, cankers have not been reported to occur with *M. medusae* on cottonwood or aspen.

Management

Fungicides are effective in protecting trees from infection by *Melampsora*, but this is not an economically practical solution when control is desired in larger plantings like that of a windbreak. The disease impact in the Great Plains can be reduced by planting a variety of *Populus* hybrids and clones, specifically those that exhibit various levels of resistance to Melampsora leaf rust. The recognition of Eurasian poplar leaf rust (*Melampsora* × *columbiana*), a new *Melampsora* hybrid in the Pacific Northwest, and *M. occidentalis* in the central United States suggests that a *Populus* hybrid or clone resistant to one species of *Melampsora* can be susceptible to another *Melampsora* species. Use of trees with various levels of resistance to *Melampsora* species should help limit the impact of the disease on larger plantings of *Populus*.

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11. Mycosphaerella Leaf Spot of Ash

Judith O'Mara and Megan M. Kennelly

Mycosphaerella leaf spot of ash is caused by two species of *Mycosphaerella* (*M. effigurata* and *M. fraxinicola*). The disease occurs on all ash (*Fraxinus*) species grown throughout the Great Plains. In most years the disease occurs late in the growing season and causes minor leaf spotting and defoliation.

Hosts and Distribution

White ash (*F. americana*) and green ash (*F. pennsylvanica*) are the dominant ash species in the Great Plains. The composition of ash in urban and woodland plantings within the Great Plains ranges from 10 to 15 percent in Kansas, Oklahoma, and parts of Missouri to greater than 50 percent in North and South Dakota. The Mycosphaerella leaf spot disease complex occurs primarily on ash in the eastern states of the Great Plains. No resistant ash species are known, although they can differ in their level of susceptibility.

Symptoms and Signs

The symptoms for both *Mycosphaerella* species are distinctive, but can be difficult to distinguish visually as both pathogens may be present at the same time. *M. fraxinicola* produces large, irregular leaf spots that start out light green and then turn brown with yellow halos (5 to 15 mm) (fig. 11-1). Individual lesions may coalesce to form larger necrotic blotches. *M. effigurata* produces numerous small, yellow to brown spots (1 to 3 mm) on the upper leaf surface. At mid-season, masses of white asexual spores (conidia) may be found in acervuli on the upper leaf surface (fig. 11-2). Late in the fall, black spots (stromata) develop in lesions on the lower leaf surface. The disease starts on the lowest portion of the tree and moves up (fig. 11-3). Severely infected trees may prematurely defoliate four to six weeks early.

Figure 11-1—Mycosphaerella leaf spot on ash (Ward Upham, Kansas State University, used with permission).

Disease Cycle

Mycosphaerella leaf spot of ash is caused by two pleomorphic fungi, *M. effigurata* and *M. fraxinicola*. *M. effigurata* initiates symptoms earlier in summer; *M. fraxinicola* tends to occur August through September. During dry years, the impact of the disease is minor. During wet growing seasons in Kansas, *M. fraxinicola* triggers visible symptoms by early-to-mid July and entire trees may be completely defoliated.

M. fraxinicola has a complex life cycle with three stages. Pseudothecia overwinter and mature in ash leaf litter. The ascospores are 8 to 10 µm × 4 to 5 µm, oval, two-celled, and colorless. Initial infection by ascospores takes place late spring to early summer. The anamorph (*Pseudocercosporella fraxini*; syn. *Cercospora fraxini* and *Cylindrosporium fraxini*) develops in summer and produces masses of colorless, cylindrical, multicelled conidia primarily on the upper leaf surface. The spermatial state of the *Asteromella* type produces tiny black structures that appear late summer in lesions on the lower leaf surface.

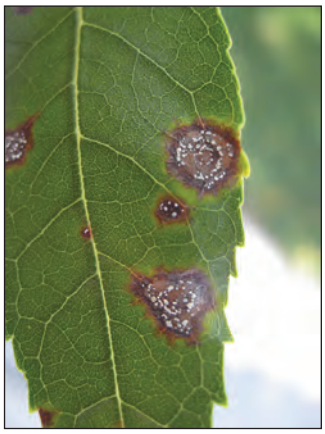

Figure 11-2—Mycosphaerella leaf spot with white masses of conidia produced by *Pseudocercosporella fraxini* (Megan M. Kennelly, Kansas State University, used with permission).

Figure 11-3—Extensive defoliation on ash resulting from *Mycosphaerella fraxinicola* (Ward Upham, Kansas State University, used with permission).

M. effigurata can also be found on ash leaves infected by *M. fraxinicola*. Lesions of *M. effigurata* tend to be smaller and more numerous. Ascospores are 4 to 14 µm long, overwinter in leaf litter, and initiate infection in late spring to early summer. During midsummer, acervuli produce clear, two-celled conidia on the underside of the leaf (*Septoria fraxinicola*; syn. *Marssonina fraxini*). Late in the season a black *Asteromella* spermagonial state follows on the underside of the leaf.

Damage

Mycosphaerella leaf spot can be found on ash trees during any given year. In most years, it is a late-season disease that causes minor shedding. During wet summers, high disease pressure can result in rapid defoliation and complete leaf loss one to two months earlier than normal autumn leaf fall. Leaf spot diseases that occur late in the growing season are generally considered to have minimal impact on tree health. However, successive years of wet weather and high disease pressure can result in ash trees with poor emergence, low vigor, and branch dieback.

Ash trees of all ages are susceptible to Mycosphaerella leaf spot, with younger trees in nursery settings more severely affected. Owing to the continuing spread of the emerald ash borer (*Agrilus planipennis*), ash trees are no longer widely grown in nurseries. Thus, until a time when effective controls for EAB or resistant species or genotypes are developed, the impact of Mycosphaerella leaf spot of ash on young trees will be primarily in landscaped and wooded areas.

Management

Mycosphaerella leaf spot of ash generally occurs late in the growing season with minimal defoliation preceding normal fall maturity. When wet years result in extensive defoliation, raking leaf litter in the fall may help reduce overwintering disease inoculum. Trees undergoing multiple years of heavy disease pressure will benefit from proper pruning to promote good air flow and regular irrigation to help maintain tree vigor. Chemical control measures are not available for this disease complex.

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12. Powdery Mildew of Lilac

John Ball; revised from Richard Dorset and Michael W. Ferguson (Riffle and Peterson 1986)

Lilacs (*Syringa* spp.) are some of the most common shrubs planted in the Great Plains, and the many species within this genus can be found in ornamental plantings as well as in windbreaks. Common lilac (*S. vulgaris*) is the most common lilac species in the region and is prized as much for its winter hardiness and adaptation to the harsh growing conditions of the Great Plains as for its fragrant late-spring flowers.

Lilacs are not without their stressors, however, and lilac/ash borer (*Podosesia syringae*), bacterial blight, and powdery mildew are all frequent problems. Powdery mildew is a disease often noticed by people in late summer or early fall as they pass by infected plants.

Hosts and Distribution

Powdery mildew is the common name for a disease caused by a number of closely related fungi that produce similar symptoms in their hosts. There are almost 100 different powdery mildew species affecting more than 10 different genera of woody plants in North America alone. Woody plant genera most commonly infected with powdery mildew are *Aesculus* (buckeyes), *Quercus* (oaks), *Rosa* (roses), *Vitis* (grapes), and lilacs. A powdery mildew species will not spread among these different genera, as mildew fungi are specific to a host genus. Although powdery mildew is common on many tree species, the disease is primarily a concern on lilacs. The remainder of this chapter will focus on powdery mildew of lilacs (*Erysiphe syringae*). This is a different disease from downy mildew, a disease that rarely affects lilacs.

Symptoms and Signs

Symptoms appear first on the lower leaves and spread upward along the branch as the growing season progresses. The upper surface of infected leaves develops small white to gray, almost dusty-appearing, patches of the mycelium (fig. 12-1). These colonies continue to enlarge through the summer and by early fall the entire leaf, and most of the leaves on the plant, may be covered with a white powdery material. Although the leaf surface appears powdery, the foliage itself may turn yellow. Severely infected leaves also become distorted and twisted and even occasionally wilt. The wilting is sometimes mistaken for injury from herbicide drift.

Figure 12-1—Late-summer appearance of common lilac infected with powdery mildew (John Ball, South Dakota State University, used with permission).

Figure 12-2—Closeup of overwintering fruiting body (chasmothecium) and appendages of powdery mildew species (Harold Larsen, Colorado State University, used with permission).

Disease Cycle

Small, almost pinpoint-size brown to black structures (ascocarps) begin development in late summer within the colonies and are easily seen with a 10× hand lens. The pathogen overwinters in the fallen, infected leaves as partially developed ascospores in an ascus enclosed in a fruiting body (chasmothecium; fig. 12-2). The ascospores mature during the early spring. As daytime air temperatures exceed 70 °F, they are released through cracks in the fruiting body and are carried by the wind to susceptible hosts. This is also the time when lilac shoots and foliage begin to expand. Once spores land on the leaf, germination can occur within a few hours if environmental conditions are favorable. Unlike many other pathogens, free water inhibits germination of the spores. The disease requires different environmental conditions for spore formation and dispersal. The disease develops best with repeated cycles of cool (60 °F), humid nights (spore formation) and warm (80 °F), dry days (spore dispersal). Infection is generally limited to the succulent new growth; mature leaves are resistant. Ascospores initiate the disease cycle on a plant, with secondary infection by conidia during the growing season. The conidia are invisible to the naked eye, but chains of conidia on the hyphae (conidiophores) contribute to the overall powdery appearance. The conidiophores generally arise from the epiphytic hyphae.

Damage

The disease appears most commonly on the foliage, though occasionally shoots can be affected. Powdery mildew is unusual in that it lives on top of as well as beneath the leaf surface. The hyphae live on the leaf surface but also produce haustoria that penetrate the epidermis and extend into the elongated palisade mesophyll cells that are the primary sites for photosynthesis. The haustoria extract water and nutrients from these cells. The disease is rarely fatal to lilacs. The appearance of the symptoms is usually the major concern with the lilac owner, who may view them as aesthetically unattractive.

Management

The simplest management of powdery mildew is to alter the growing environment, making it less favorable for development of the disease. This approach requires pruning and clearing lilac beds to decrease night humidity and improve air flow. Removing overhanging trees or, if the tree cannot be removed, pruning off the lower limbs (those lower than 12 feet) may provide sufficient air flow and reduced night humidity to minimize

infection. However some lilacs, particularly the common lilac and its many cultivars, may be covered with powdery mildew even in open growing conditions.

Another management option is to plant lilac species that are rarely affected by powdery mildew. Not all of these species have the same form or flowering characteristics of the common lilac but may be acceptable substitutes in many planting locations.

The lilac species and hybrids in which powdery mildew is rarely a problem in the Great Plains include: early flowering lilac (*S.* × *hyacinthiflora*), Meyer lilac (*S. meyeri*), littleleaf lilac (*S. microphylla*), Manchurian lilac (*S. patula*), Pekin tree lilac (*S. pekinensis*), Preston lilac (*S.* × *prestoniae*), Japanese tree lilac (*S. reticulata*), late lilac (*S. villosa*).

Chinese lilac (*S.* × *chinensis*) can occasionally be infected with powdery mildew and Persian lilac (*S.* × *persica*) and its cultivars may also become infected, but the species most severely infected is the one that is planted the most: the common lilac. Almost all cultivars of common lilac are susceptible to this disease, including the French hybrids; however, there are a few cultivars that may not become infected or at least exhibit barely noticeable symptoms of the disease: 'Adelaide Dunbar,' 'Charles Joly,' 'Hulda,' 'Jan Van Tol,' and 'Katherine Havemeyer.'

If the disease does not appear until late summer, there is little concern that the disease will seriously harm the health of the lilac. The shoot growth and flowering will not be reduced the next spring. If the disease begins to develop earlier in the season, the infection may result in reduced growth. Regardless of whether the disease symptoms appear early or late in the growing season, the appearance of "dusty" leaves may be unacceptable to the plant's owner.

Because the disease-causing fungus occurs primarily on the leaf surface, powdery mildew is much easier to manage with fungicides than are many other diseases. The choice of fungicides will vary with availability and registration within a particular state or province, but generally a fungicide labeled for powdery mildew on lilacs can be found in garden stores. A fungicide application can be made when the disease symptoms are first noticed and then continued every two weeks while conditions are favorable for the disease. Once lilac finishes its growth, usually in midsummer, leaves become resistant to the disease and treatments can be discontinued.

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13. Tar Spots of Maple

Aaron D. Bergdahl and Kelsey L. Dunnell

The fungi that cause tar spots of maple are becoming a concern in the Great Plains as the popularity of planting maple (*Acer*) species and their cultivars increases throughout the region. Although tar spots do not often severely affect tree health, they have the potential to significantly reduce the aesthetic appeal of trees.

Hosts and Distribution

Tar spot is a disease of maple leaves caused by one of three species of fungi in the genus *Rhytisma*. All three fungi occur in the central United States. Tar spot pathogens have been known to cause diseases on maple species throughout the eastern and Great Lakes regions, in the Pacific Northwest, and in Manitoba, Canada. In the central United States, tar spots have been observed on *Acer* species in Kansas, Missouri, Nebraska, North Dakota, South Dakota, and Wyoming. Refer to table 13-1 for maple hosts and associated *Rhytisma* species.

Symptoms and Signs

In mid- to late July one or more small greenish-yellow spots appear on the upper leaf surface indicating early infection (fig. 13-1a). In late summer conspicuous black, tarlike structures form within the chlorotic areas (fig. 13-1b,c,d). The lower leaf surface opposite the tarlike structures is cupped and less black.

The tar spot fungus most often associated with Norway maple (*A. platanoides*) is caused by *R. acerinum* (fig. 13-1b) and was apparently introduced from Europe in the early 1900s and first formally reported in North America in 1942. Tar spots caused by *R. acerinum* can exceed 0.5 inch in diameter, which is why it has been referred to as "giant tar spot" in some earlier references. In the Great Plains the fungus *R. americanum* (fig. 13-1c) primarily infects red maple (*A. rubrum*), silver maple (*A. sacharrinum*), and their hybrids. In the past, the causal fungus *R. americanum* has often been misidentified as *R. acerinum* and the confusion between these two fungal pathogens continues owing to their similar appearance and previously used common names. The extent to which speckled tar spot (*R. punctatum*) (fig. 13-1d) occurs in the Great Plains is uncertain. Other *Rhytisma* species commonly cause tar spot on willow (*Salix* spp.), holly (*Ilex* spp.), and tuliptree (*Liriodendron tulipifera*), although these are not discussed here.

Table 13-1—Maple species and their susceptibility to three species of tar spot fungi.

	Tar spot species		
Maple species	***Rhytisma acerinum***	***R. americanum***	***R. punctatum***
Acer campestre (hedge maple)	X		
A. circinatum (vine maple)	X		X
A. ginnala (Amur maple)	X		
A. glabrum (Rocky Mountain maple)		X	X
A. negundo (boxelder)	X	X	X
A. macrophyllum (bigleaf maple)		X	X
A. pensylvanicum (striped maple)			X
A. platanoides (Norway maple)	X		
A. pseudoplatanus (sycamore maple)	X	X	X
A. rubrum (red maple)	X	X	X
A. saccharinum (silver maple)	X	X	X
A. saccharum (sugar maple)	X	X	X
A. spicatum (mountain maple)	X	X	X

Adapted from Hsiang and Tian (2007).

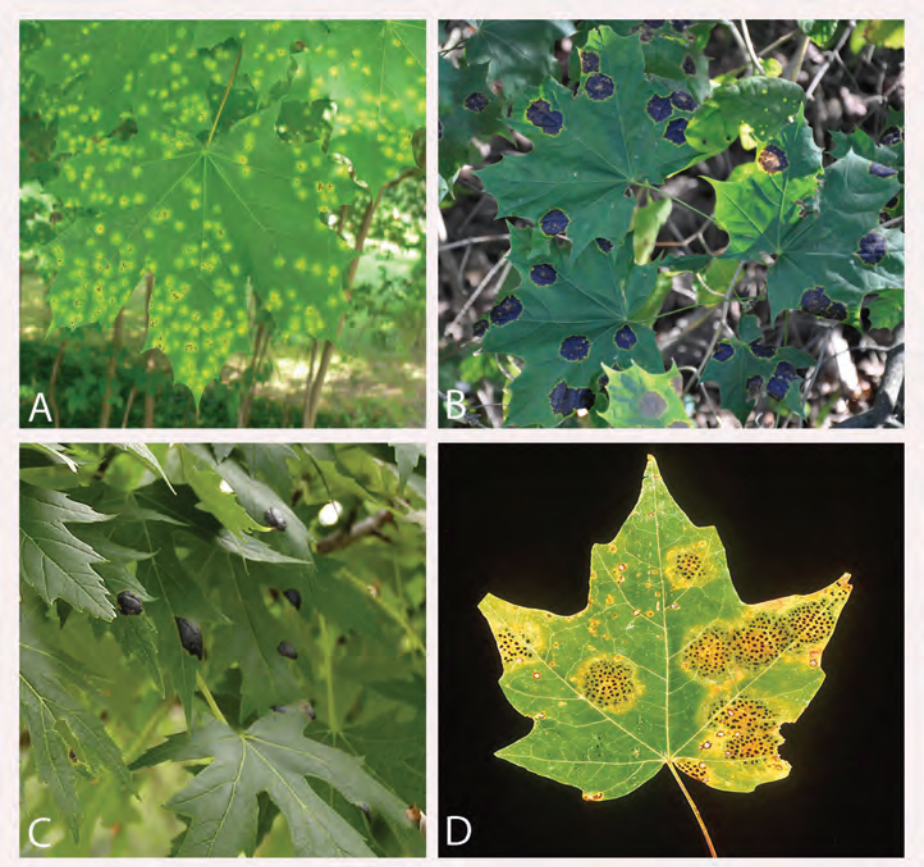

Figure 13-1—Symptoms of infection: (A) Typical symptoms of early infection by *Rhytisma acerinum* (George W. Hudler, Cornell University, used with permission); (B) Late-season tar spot (*R. acerinum*) on Norway maple (Aaron D. Bergdahl, North Dakota Forest Service, used with permission); (C) *R. americanum* on silver maple (Simeon Wright, Missouri Department of Conservation, used with permission); (D) Infection by speckled tar spot *R. punctatum* (George W. Hudler, Cornell University, used with permission).

Disease Cycle

Ascospores of the tar spot fungus generally begin to develop in the black spots (stromata) before leaves are cast in fall and continue development when leaves are on the ground. In spring the stroma begins to split, and mature needle-shaped ascospores are forcibly ejected upward from apothecia on fallen leaves during favorable conditions. The spores are ejected into the air for a limited distance and then carried upward on air currents, where they make contact with expanding foliage of susceptible trees. Germ tubes from the germinating spores enter the leaves through stomata, and a black gummy substance is produced within the tissues.

Symptoms are generally visible within two months of infection. At this time each lesion appears as a defined blackened area fringed with a yellowish-green border. First, numerous spore-producing structures develop in a group at the center of the stroma. They resemble small pimples with a minute hole in the center, and represent the imperfect (asexual) stage. Simple or branched conidiophores are formed within each fruiting body, producing clear (hyaline) or partially clear (subhyaline), rodshaped spores that measure 6 µm × 1 µm. It has been proposed that these single-celled spores are not infectious and play a role in sexual reproduction (therefore, they are often referred to as spermatia formed in spermagonia), although this has not been confirmed. Later, apothecia begin to

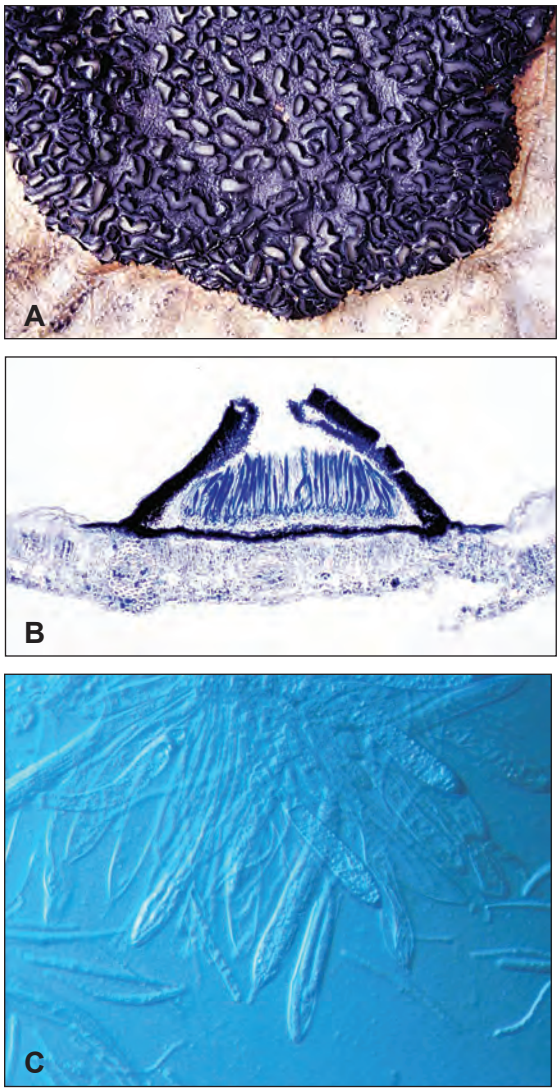

Figure 13-2—Stages of disease cycle of tar spot of maple disease: (A) Splitting *Rhytisma* stroma after an overwintering period prior to spore dispersal; (B) Cross-section of mature *Rhytisma* apothecium; (C) Asci and ascospores of *Rhytisma* species (George W. Hudler, Cornell University, used with permission).

develop within this tissue before the leaves drop. Stromata continue to expand, developing radiating wrinkles in which apothecia form (fig. 13-2a). Asci slowly develop through the winter and by early spring their development is completed. Each ascus within an apothecium (fig. 13-2b) contains eight needlelike ascospores (fig. 13-2c), approximately 130 µm × 10 µm. The stroma splits along the radiating wrinkles and ascospores are ejected, completing the disease cycle (fig. 13-3).

Damage

Tar spot seldom severely affects the health of trees and is not known to kill established trees, although it may lead to seedling mortality after heavy infection. In sheltered situations, such as in the forest, trees can become heavily infected and defoliate prematurely, whereas open-grown trees are typically less infected. However, during years of prime conditions for infection, large open-grown trees can have a severe level of infection by *R. acerinum*. Successive years of defoliation may constitute a significant stress, predisposing trees to other pests. However, two successive years of significant premature defoliation seldom occurs because heavily infected leaves are prematurely shed. Thus, the stroma on shed leaves has not had sufficient time to fully develop asci for reinfection the next spring. Further, in a forest setting early shed leaves are often covered by leaves of other tree species, creating a barrier to spore dispersal. Premature defoliation of infected leaves gives the appearance of crown thinning.

Management

The disease can be controlled in shade trees by raking and burning fallen leaves to destroy overwintering inoculum. Efficient composting of leaves is also an effective practice for reducing inoculum. Typically, use of fungicides to control this disease is discouraged. If spraying is warranted, copper-based fungicides are considered satisfactory. Although management of *R. acerinum* may be achieved with one well-timed fungicide application, just as the leaves on Norway maple reach full expansion, applying fungicide at two- to three-week intervals during leaf emergence may provide best results.

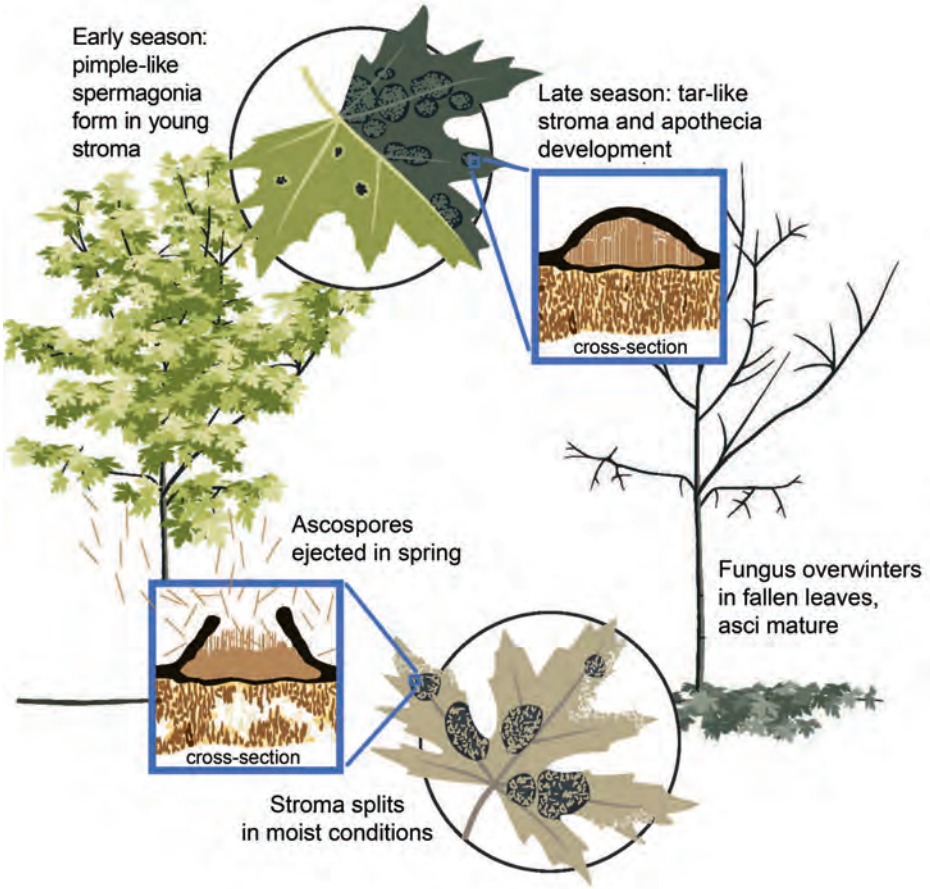

Figure 13-3—Disease cycle of tar spot of maple (David Haasser, North Dakota State University, used with permission, Adapted from Riffle and Peterson, 1986).

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14. X-Disease of Chokecherry

James A. Walla; revised from Glenn W. Peterson and David W. Johnson (Riffle and Peterson 1986)

Chokecherry (*Prunus virginiana*) is a native shrub used in conservation, ornamental, and fruit production plantings in the central and northern Great Plains. Chokecherry in many of these plantings is infected by X-disease phytoplasmas, '*Candidatus* Phytoplasma pruni,' RFLP group 16sRIII-A. Phytoplasmas are closely related to Gram-positive bacteria. Previous names for X-disease include virus-X, eastern X-disease, and western X-disease.

Hosts and Distribution

X-disease seriously damages common chokecherry (*Prunus virginiana* L. var. *virginiana*), sweet (*P. avium*) and sour (*P. cerasus*) cherries, and peach (*P. persica*). Also susceptible are western chokecherry (*P. virginiana* L. var. *demissa* (Nutt.) Torr.), apricot (*P. armeniaca*), domestic plum (*P. americana*), Japanese plum (*P. salicina*), and some other *Prunus* species. The red-leaf strains of common chokecherry, such as 'Schubert' chokecherry and 'Canada Red' cherry, are also damaged. Leafhoppers are thought to be the primary vector, but the specific vectors are not known in the Great Plains. X-disease occurs in the northern half of the United States and southern half of Canada, including the central and northern Great Plains states and the Prairie Provinces.

Symptoms

X-disease usually causes a slow decline, but symptoms vary with phytoplasma genotype, host species and genotype, disease development stage, host age, and site conditions. In chokecherry, one or more years after infection, leaves on an infected stem typically become greenish yellow a few weeks before normal onset of fall color. They may have green tissue remaining along the veins and an orange or red tinge or necrosis on their borders, and there may be leaves with different stages of disease on a single shoot (fig. 14-1). Leaf discoloration, deformation, and stunting begin earlier and become more pronounced each subsequent year, until first discoloration is at budbreak over the entire plant and the leaves may turn deep red or orange by six to eight weeks after budbreak (fig. 14-2). Moderately or severely diseased leaves feel thicker and waxier than healthy leaves. Shoots and roots are stunted, and rosettes may result from shortened internodes at shoot tips. Fruit may be small and somewhat pointed and may ripen to bright red, not the normal dark purple of healthy fruit (fig. 14-3a). Buds growing from current-season leaf axils and small late-season flowers may develop (fig. 14-3b). Twigs do not harden off as early, and twig dieback may result. After two or more years, branch, whole stem, or whole plant dieback may occur. In red-leaf chokecherry strains, X-disease causes similar decline, but leaf discoloration is masked; severely diseased leaves are lighter red than healthy leaves.

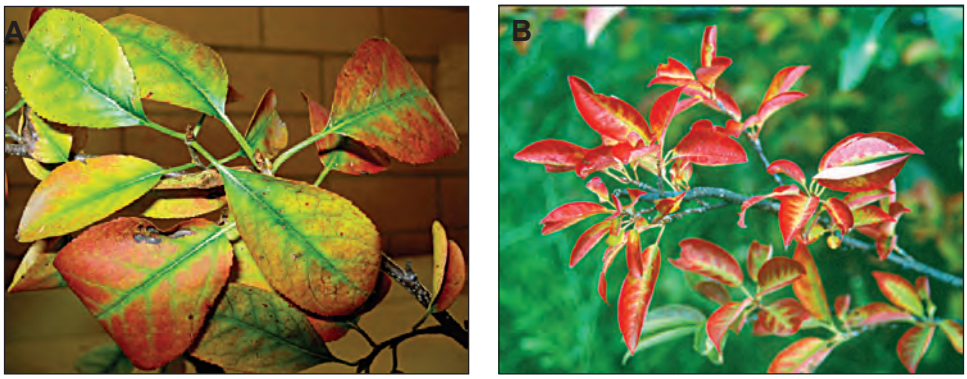

Figure 14-1—Chokecherry leaves with X-disease symptoms. Leaves become various shades of (A) yellow, (B) red, and orange before normal fall color develops on unaffected plants. Leaf veins tend to be darker green than surrounding tissues (A: James A. Walla, Northern Tree Specialties, used with permission; B: Bruce Neill, Agriculture and Agri-food Canada, used with permission).

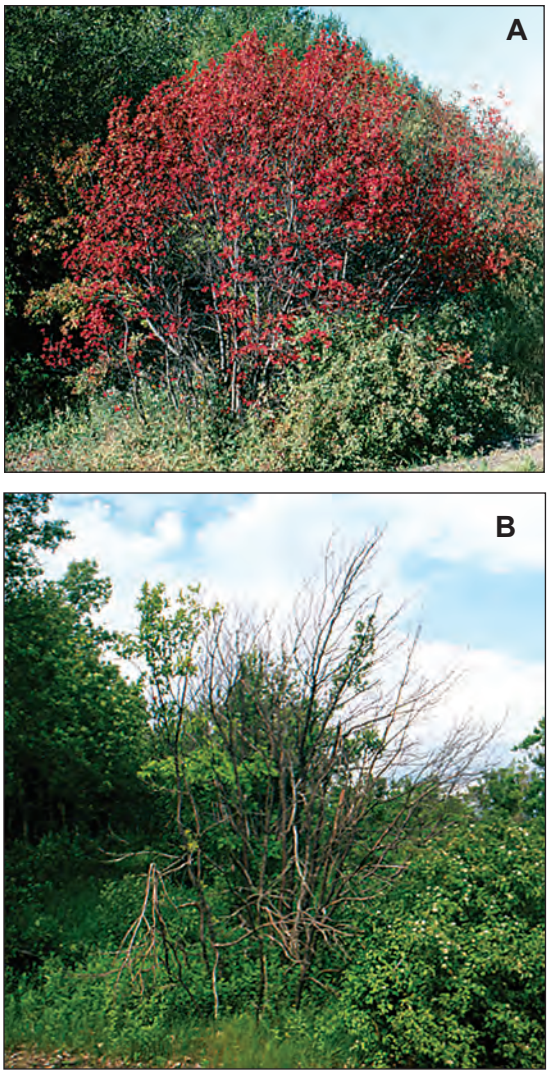

Figure 14-2—Increasing effect of X-disease in chokecherry. (A) In 1993, this severely diseased chokecherry plant had symptomatic red leaves when other plants still had green leaves. (B) The same plant was nearly dead by 1997 (James A. Walla, Northern Tree Specialties, used with permission).

Discolored leaves and stem dieback may also be associated with other declines caused by drought, cankers, bark beetles, and herbicides. At early onset, X-disease cannot be reliably differentiated from healthy plants or plants with other problems. With more advanced disease, the texture of leaves, the pattern of leaf discoloration, the combination of symptoms and signs, and the pattern of disease progression become more reliable, but confirmation requires serological or molecular diagnostics. Confirmation is generally justified only for research or to discern the role of other possible chokecherry problems.

Disease Cycle

Research in eastern and western United States indicates that the pathogen is usually transmitted to *Prunus* hosts by leafhoppers in mid- to late-summer when infected insects feed on the phloem in leaves of healthy plants. In larger plants, phytoplasmas are distributed systemically to all plant parts over a period of years. They survive over winter primarily in roots and are distributed to upper plant parts a few weeks after budbreak. Leafhoppers feed on phloem of infected leaves to acquire X-disease phytoplasmas, and can transmit the phytoplasmas after four to five weeks.

Damage

X-disease is the most limiting problem of chokecherry in plantings and native stands in the central and northern Great Plains. Growth and fruit production are reduced. Most infected plants gradually decline and have a shortened lifespan. In a study in eastern Nebraska, symptoms appeared on more than 80 percent of chokecherry plants within three years after X-disease was introduced to a plot, and mortality was more than 50 percent within eight years. Damage to infected plants is less pronounced in the Prairie Provinces. In North Dakota, the most susceptible large shrubs die within 10 years of infection, and most infected plants die within 20 years of infection, compared to expected longevity of 70+ years. A small percentage of infected common chokecherry remains healthy for many years. Younger or more stressed plants are more quickly and seriously damaged than older or less stressed plants. Infected plants have reduced ability to recover from other stresses. Awareness of damage caused by X-disease limits the use of chokecherry in new plantings. Infected American plums are not damaged. Black cherry (*Prunus serotina*) and Japanese flowering cherry (*Prunus serrulata*) are apparently not susceptible.

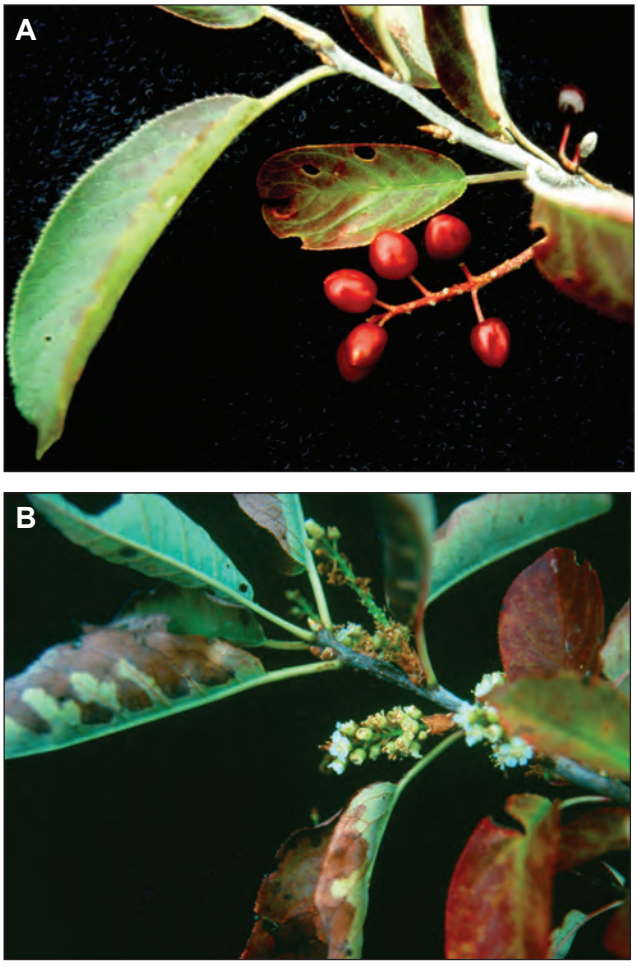

Figure 14-3—Effects of X-disease. The disease can cause (A) fruit to remain red and pointed compared to the normal dark purple and round berries on unaffected plants, and (B) stunted, deformed flowers to be formed late in the season (Bruce Neill, Agriculture and Agri-food Canada, used with permission).

Management

Promoting vigorous growth of infected plants (for example, with proper management of weeds, soil moisture, and soil fertility) will slow the rate of decline. Consider planting resistant or nonhost plants in areas where X-disease is a problem. American plum can be included in plantings containing susceptible hosts with confidence that the X-disease pathogen will not seriously damage it. However, the disease could be transmitted from American plum to susceptible plants. To reduce the spread of the disease by insect vectors, avoid establishing new plantings near any *Prunus* species. Removal of other susceptible plants such as wild chokecherries, from a zone (500 feet suggested for eastern United States) around desired susceptible plants, such as a stone fruit orchard, will reduce the rate of transmission. Clean cultivate or mow the area in and around plantings to reduce resting habitat for vectors. Mix one or more nonhost plants among *Prunus* species in new plantings.

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15. Biscogniauxia Canker of Oak

Judith O'Mara and Nicole Opbroek

Oaks (*Quercus* spp.) are an important forest and landscape tree species found throughout the Great Plains from the Dakotas to Texas. Biscogniauxia canker (pronounced 'Bisk-o-nee-ox-e-a', formerly Hypoxylon canker) can cause substantial damage to oaks in wooded areas and urban landscapes. Disease severity is generally associated with stressful environmental and site conditions.

Hosts and Distribution

Biscogniauxia canker is caused by the fungi *Biscogniauxia atropunctata* (syn. *Hypoxylon atropunctatum*) and *B. mediterranea* (syn. *H. mediterraneum)*. In North America, both species of *Biscogniauxia* can be found as far north as Ontario, and as far south as Texas and Georgia. *Biscogniauxia* is also distributed across central and eastern states as well as in the western states of California, Oregon, and Washington.

Outbreaks of Biscogniauxia canker are common in the Great Plains following multi-year periods of heat and drought, particularly in Kansas, Missouri, Oklahoma, and Texas. Biscogniauxia canker is primarily a problem on oaks in the red oak group, although it can also affect white oaks. Other hardwood species affected include maple (*Acer* spp.), hickory (*Carya* spp.), sycamore (*Platanus occidentalis*), elm (*Ulmus* spp.), hackberry (*Celtis* spp.), hornbeam (*Carpinus caroliniana*), and basswood (*Tilia americana*).

Symptoms and Signs

Symptoms of Biscogniauxia canker resemble drought stress. Affected trees exhibit a general thinning of the canopy, yellowing and wilting of the foliage, and branch dieback. As the disease progresses, a mass of fungal tissue (stroma) is formed underneath the bark in the cambium. The pressure from this mass causes the bark to pop off, exposing a crusty, tan-colored mat of fungal tissue (fig. 15-1). As the outer layer of the stroma wears off, the canker turns silver (fig. 15-2) and then black and crusty, similar in appearance to asphalt (fig. 15-3). The black, crusty stroma is diagnostic for Biscogniauxia canker.

Figure 15-1—Dusty-brown conidial stroma of Biscogniauxia canker on post oak (*Q. stellata*) (Jake Weber, Kansas State University, used with permission).

Figure 15-2—Silver stroma of Biscogniauxia canker on oak (Nicole Opbroek, Kansas Forest Service, used with permission).

Figure 15-3—Mature black *Biscogniauxia* stroma on oak (Nicole Opbroek, Kansas Forest Service, used with permission).

Figure 15-4—Black zone lines and wood decay caused by Biscogniauxia canker (Nicole Opbroek, Kansas Forest Service, used with permission).

Disease Cycle

Biscogniauxia canker can affect trees of any age. Sexual spores (ascospores) are spread by blowing wind, insects, or splashing rain. They enter trees through natural openings or wounds and the fungus can persist for many years within the sapwood and bark of healthy trees without causing disease symptoms. Vigorous trees can keep the fungus in check until a stress event triggers the onset of disease. Factors such as drought stress, fire, root injury, chemical damage, compacted soil or addition of fill soil, improper pruning, storm damage, or damage by insect pests may weaken the tree. These stress events in turn can reduce the water potential in the tree and predispose it to infection. Canker development often occurs in the year after a major stress event, although *B. atropunctata* can produce cankers within two months.

Biscogniauxia canker is favored by low moisture content within the tree and warm, dry growing conditions. Both *Biscogniauxia* species are adapted to dry conditions with optimal growth from 85 and to 95 °F. Growth of *B. atropunctata* can occur at temperatures up to 104 °F. After infection, development of stromatic tissue causes bark to slough off, exposing a tan stroma containing asexual spores (conidia). As the stroma matures, the tan conidial layer wears off and exposes a silver stroma with a black ascospore-producing perithecial layer beneath. Old cankers appear black and crusty. *Biscogniauxia mediterranea* tends to produce small patches, whereas *B. atropunctata* produces cankers that range from a few inches to several yards along the length of the tree. As the fungus grows into the sapwood, it can coalesce into areas of yellowish decay with distinctive black zone lines (fig. 15-4) at the edge. As the disease progresses, infected trees can lose large strips of bark and die within one to two growing seasons.

Damage

Historical reports indicate a link between periods of drought stress, oak decline, and the presence of Biscogniauxia canker. A drought during 1980 to 1981 in South Carolina was associated with lowered water tables and large areas of red oak mortality along the coastal highway. Almost all dead and declining oaks showed the distinctive tan-silver stromata associated with Biscogniauxia canker. Regional records suggest that a 1980

Figure 15-5—Biscogniauxia canker on maple (Jake Weber, Kansas State University, used with permission).

drought in east central Arkansas resulted in heavy oak mortality, affecting 85 percent of the oak trees within a 494-acre area. *Biscogniauxia* canker was found on 95 percent of the dead oaks, as well as in oak trees exhibiting dieback.

More recently, multiyear droughts have significantly affected oak populations in wooded areas and landscapes in eastern Kansas and Oklahoma and western Missouri. After a severe drought from 2010 to 2012, Biscogniauxia canker was observed by the author on 10 percent of the oaks in two watersheds in northeast Kansas. A multi-county survey in central and eastern Oklahoma documented Biscogniauxia canker on a range of hardwood trees, including maple, goldenrain tree (*Koelreuteria paniculata*), hackberry, hickory, pecan (*C. illinoinensis*), American elm (*U. americana*), sycamore, and many species of oak. Maple trees (fig. 15-5) were severely affected by Biscogniauxia canker in Kansas, Oklahoma, Missouri, and Colorado during this same period.

Management

There are no curative measures for trees with extensive Biscogniauxia canker. It might be possible to slow disease progression on trees by pruning affected limbs and improving the vigor of the tree with regular care. Remove severely affected trees down to ground level, as the disease can affect the remaining stump. Destroy firewood taken from infected trees so that the disease does not spread. Preventive strategies in timber stands include sanitation cutting of infected trees, removing infected debris from the area, and pruning trees with storm-damaged limbs.

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16. Botryodiplodia Diseases

Mark O. Harrell and Laurie J. Stepanek;
revised from Jerry W. Riffle and Joseph M. Krupinsky, Botryodiplodia canker of elms; Glenn W. Peterson and Harrison L. Morton, Botryodiplodia disease of Russian-olive; and Robert Lewis, Jr., and Kenneth E. Conway, Botryodiplodia canker of sycamore (Riffle and Peterson 1986)

Trees weakened by drought, heat, or other stressful conditions in the Great Plains are often damaged by the fungi *Botryodiplodia hypodermia* and *Lasiodiplodia theobromae* (formerly *B. theobromae*), which cause cankers, dieback, and tree mortality. Siberian elm (*Ulmus pumila*), Russian-olive (*Elaeagnus angustifolia*), and sycamore (*Platanus occidentalis*) are commonly attacked. Additional information on *L. theobromae* can be found in chapter 20.

Hosts and Distribution

B. hypodermia (also known as *B. ulmicola*, *Sphaeropsis hypodermia*, and *S. ulmicola*) is found on American (*U. americana*), Siberian (*U. pumila*) and English (*U. procera*) elms in the United States. It occurs principally on Siberian elm in North Dakota, South Dakota, Nebraska, Kansas, Oklahoma, Minnesota, and Montana.

L. theobromae (once known as *Botryodiplodia theobromae*, *Diplodia gossypina*, *D. natalensis*, *D. theobromae*, and *B. rhodina*) occurs around the world and attacks woody and herbaceous plants in at least 280 genera. In the Great Plains *L. theobromae* is most damaging to Russian-olive and sycamore, but will also attack ailanthus (*Ailanthus altissima*), American holly (*Ilex opaca*), flowering dogwood (*Cornus florida*), hickory (*Carya* spp.), oak (*Quercus* spp.), persimmon (*Diospyros virginiana*), and walnut (*Juglans* spp.). In some tree species the fungus is only weakly pathogenic and may cause cankers only when the trees are severely stressed.

Symptoms and Signs

B. hypodermia cankers on Siberian elm develop a reddish-brown to black discoloration on the bark surface (fig. 16-1). Infected inner bark tissues turn reddish brown to brownish black (fig. 16-2) and become water soaked and soft. Sapwood beneath the

Figure 16-1—*Botryodiplodia hypodermia* canker on Siberian elm (James A. Walla, Northern Tree Specialties, used with permission).

Figure 16-2—Siberian elm stem with reddish-brown inner bark killed by *Botryodiplodia hypodermia* (James A. Walla, Northern Tree Specialties, used with permission).

Figure 16-3—Loose bark caused by *Botryodiplodia hypodermia* on Siberian elm (James A. Walla, Northern Tree Specialties, used with permission).

infected bark turns reddish brown to brownish black as well. The bark frequently splits longitudinally and may become loose (fig. 16-3). When the fungus girdles an infected branch or trunk, the foliage wilts and branch dieback or top kill occurs. Sprouts often develop below girdling cankers. Yellow leaves on infected American elm branches may somewhat resemble symptoms caused by Dutch elm disease.

L. theobromae in Russian-olive and sycamore typically kills bark and cambium in strips that may extend many feet along the trunk and branches. On Russian-olive the cankers may initially be inconspicuous and indicated only by the presence of small dead branches along the strip. Mortality of these small branches is most readily detected in the upper crown during July and August. Major branches and stems may have more than one strip of dead tissue, and the strips may spiral along the stems. Infected Russian-olives may exude gum from stems and branches, but gum is not a reliable indicator of this disease because gumming also can be caused by other pathogens.

On sycamore, *L. theobromae* cankers may appear as tan to dark brown streaks in the normally green to white smooth bark. Cankers may develop cracks as they enlarge and age. Twig and branch dieback may occur either on an individual branch or throughout most of the crown. In cases where the trunk is affected, sprouts may grow from the base of the trunk (root collar) and on the trunk below the canker. Stressful environmental conditions, such as drought, can cause cankers to develop more rapidly and become more severe.

Both *B. hypodermia* and *L. theobromae* fruit on killed bark. The fruiting bodies (pycnidia) appear as small black pimplelike structures embedded in the outer layer of dead bark. If pycnidia are cut through by shaving the bark, they often appear to have black walls and white contents. Pycnidia of both species produce asexual spores (conidia) that may be one- or two-celled, and clear or brown (fig. 16-4). *B. hypodermia* conidia are 20 to 36 µm × 13 to 18 µm in size. *L. theobromae* conidia are 20 to 30 µm × 10 to 15 µm.

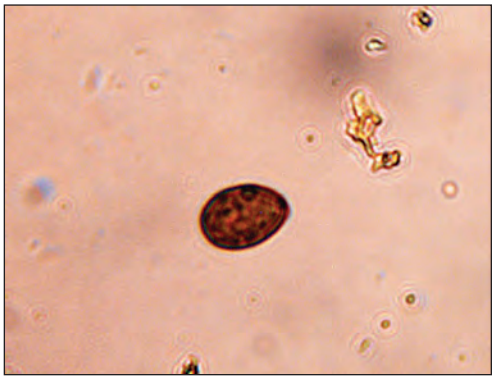

Figure 16-4—Brown single-celled conidium of *Botryodiplodia hypodermia* (William R. Jacobi, Colorado State University, used with permission).

Disease Cycle

Conidia on dead bark are the primary inoculum for new infections. Most conidia are probably dispersed by splashing rain or wind, but may also be carried by insects and pruning tools. When conidia make contact with wounded stems, branch stubs, and other suitable infection courts, they germinate and colonize host tissues.

Colonization does not always result in a girdling canker. Susceptibility of the tree, virulence of the fungal strain, and environmental conditions are determining factors in canker formation. The most virulent strains can cause cankers in nonstressed trees, but the least virulent strains do not. Canker development is favored by high temperature and water stress.

Damage

The effects of infection can range from small, inconspicuous cankers to tree mortality. Small cankers have little effect on trees. Large cankers slow the growth rate and cause wood defects, which weaken stems and makes them more vulnerable to wind breakage. Dieback changes the form of individual trees and severe dieback may kill trees.

Management

Losses from Botryodiplodia cankers can be reduced by cultural practices. Avoid wounding stems and making branch stubs. Prune during late fall or winter when fungal colonization is reduced because of low temperatures. Prune dead, dying, or severely cankered branches from infected trees during winter or before spring rains to prevent fungal spores from splashing to new infection sites.

Because water stress favors infection and disease development, avoid planting trees on dry sites. Use a mulch bed of wood or bark chips around trees on dry sites to improve soil conditions. Trees in landscapes should be watered appropriately during dry periods. When possible, plant seedlings that are adapted to a particular geographic or climatic site. Select genetically improved stock with resistance to *Botryodiplodia* if available.

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17. Brown Rot of Stone Fruits

John Ball and James A. Walla; revised from David S. Wysong and James A. Walla (Riffle and Peterson 1986)

Common chokecherry (*Prunus virginiana*) and American plum (*P. americana*) are two common windbreak and native trees in the Great Plains. These trees are also used as ornamentals for their attractive spring flowers. The fruit from these trees is collected and used for jams and jellies. Other stone fruits commonly planted in the region include apricot (*P. armeniaca*), peach (*P. persica*), hybrid plum, and sour cherry (*P. cerasus*). These trees suffer from numerous pests, one of which is brown rot.

Hosts and Distribution

Brown rot on stone and pome fruits in North America is caused by several fungi such as *Monilinia fructicola, M. amelanchieris, M. demissa*, and *M. laxa*, with *M. fructicola* likely predominating on the northern Great Plains.

The disease affects primarily stone fruits, members of the genus *Prunus.* Chokecherry and American plum are the most commonly affected species on the northern Great Plains, whereas all stone fruits are affected in the warmer, more humid southern Great Plains. Less frequently, it can also be found affecting pome fruits, such as apple (*Malus* spp.), pear (*Pyrus* spp.), and serviceberry (*Amelanchier* spp.).

Symptoms and Signs

Brown rot has two main phases: blossom and shoot blight, affecting the flowers, leaves, and new shoots; and brown rot, affecting the fruit. The blossom and shoot blight phase begins in early spring. The brown rot phase involves the browning and decay of affected fruit, usually as the fruit ripens and after harvest, as a result of both shoot and fruit infection. A twig canker phase may also occur.

Figure 17-1—Shoot of western sandcherry (*Prunus pumila*) infected with brown rot (Simeon Wright, Missouri Department of Conservation, used with permission).

Figure 17-2—Formation of ash-gray mold on surface of fruit infected with brown rot (Simeon Wright, Missouri Department of Conservation, used with permission).

Blossom blight begins with infection of anthers and pistils in the flowers, eventually spreading to kill the entire flower and the peduncle. Petals turn brown and water soaked, and then shrivel and wilt but remain hanging from the twig, often until midsummer. Shoot blight begins with infections of new leaves or shoots, with the infection usually spreading to girdle the shoot, causing the shoot to wilt and hang down (fig. 17-1).

Immature fruit may be infected at any stage of development, but such infections are often latent. Infection of mature fruit is more common. Fruit decay usually begins from recent and latent infections, when sugar content increases as fruits near maturity. Decay first appears as tan-brown, almost circular spots. Within a few days, these spots coalesce and the entire fruit will turn a light brown with a gray powder on the surface (fig. 17-2). The hanging, discolored fruit will also have a semi-watery appearance before beginning to dry and harden. The dry, infected fruit may fall to the ground or shrivel and become mummified while still hanging from the twig. These mummified fruits generally drop later in the season although some may remain hanging for the winter.

Twig cankers sometimes develop through expansion of blossom, shoot, or fruit infections. Infected twigs will have a sunken, oval or elliptical canker. As cankers girdle or nearly girdle twigs, the foliage distal to the canker will often become yellow and wilt, but remain attached. Gum will often ooze from the canker edge, most commonly with apricot. This gum "glues" infected flowers or fruit to the twig.

Signs of the pathogens are apothecia, in which sexual ascospores are produced, and sporodochia, on which asexual conidia are produced. The rarely seen apothecia are wine-glass-shaped tan mushroomlike structures usually 10 to 15 mm across that arise in spring from fruit mummies on the ground. Sporodochia appear as ash-gray to tan tufts that may cover part or all of infected plant tissue under moist conditions at any time of the growing season and in storage. When abundant, sporodochia often form in concentric rings. Identification of many *Monilinia* species has been difficult, but gene sequences have been described and proven useful.

Disease Cycle

Windblown ascospores from apothecia and splashed, windblown, or insect-carried conidia from mummified fruit or twig cankers are the inoculum sources for infection of opening flowers and expanding shoots in spring. Some *Monilinia* species (including *M. fructicola*) produce repeated secondary cycles of conidia throughout the season; others produce only one secondary cycle of conidia. Secondary conidia infect immature and mature fruit, primarily through wounds, but also through stomata and hair sockets. Fruits

infected by both groups will develop brown rot, mostly as they ripen, and the fruit will become mummified and overwinter to continue the disease cycle. Fungus survival is reduced in colder climates for fruit mummies that hang on tree branches, but not for those on the ground.

Damage

The disease is more common in warmer, and wet, climates, such as those of the eastern United States. Brown rot can result in the loss of more than half the fruit in an orchard if not treated, particularly during summers with above-normal precipitation and humid conditions. The disease often is more noticeable as blossom and shoot blight on chokecherry. Both phases, blossom and shoot blight and brown rot, appear on American plum.

Management

Sanitation is a key practice in reducing this disease. Mummified fruit on and under the tree as well as infected twigs should be removed and destroyed before spring. Remove wild chokecherry or plum trees from the vicinity. Also, improve drying conditions in and around susceptible trees, for example by pruning stone fruits for an open center; increasing the distance between susceptible trees; tilling or mowing ground cover; and pruning, thinning, or removing overhanging and surrounding trees. Control vectoring insects that make or visit wounds in the fruit, such as fruit flies (*Drosophila* spp.), oriental fruit moth (*Grapholita molesta*), and plum curculio (*Conotrachelus nenuphar*). Fungicide treatments are often necessary.

Brown rot is usually managed with fungicides during two time periods: the spring to reduce blossom and shoot blight and the summer to reduce brown rot on fruit. One to three fungicide sprays are applied to manage blossom blight: the first as the flowers open, the second at full bloom, and the third at petal fall. A similar schedule is used for shoot blight, beginning as the leaves unfurl. Spring may be the only season that treatment is needed if the ornamental value of the flowers and foliage is the only concern. If the fruit is important, however, then summer fungicide applications are made beginning about three weeks before harvest. Several applications may be needed during fruit ripening, and fungicide labels should be checked for preharvest intervals. If brown rot infestation remains too high, control of latent infections may be needed in future years. Many populations of brown rot fungi have developed resistance to individual fungicides, so use different fungicide groups in alternating applications for disease control.

Infection can easily spread from infected to healthy fruit during storage. To reduce storage losses, harvest before fruit is overripe, avoid wounding the fruit, sort out any distorted and discolored fruit, cool the fruit promptly after harvest, and periodically sort stored fruit to remove those with decay.

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18. Black Knot of Cherry and Plum

Mark O. Harrell and James T. Blodgett; revised from David S. Wysong and Mark O. Harrell (Riffle and Peterson 1986)

Black knot, caused by the fungus *Apiosporina morbosa*, can significantly reduce fruit production and kill small branches in heavily infected trees.

Hosts and Distribution

Black knot occurs throughout the United States. It affects cherries, plums, and most other members of the genus *Prunus*. Common hosts in the Great Plains include bird (*Prunus padus*), bitter (*P. emarginata*), black (*P. serotina*), choke (*P. virginiana*), mahaleb (*P. mahaleb*), Nanking (*P. tomentosa*), pin (*P. pensylvanica*), sand (*P. pumila*), sour (*P. cerasus*), and sweet (*P. avium*) cherries; apricot (*P. armeniaca*) and peach (*P. persica*); and American (*P. americana*), Canada (*P. nigra*), and domestic plums (*Prunus* spp.).

Symptoms and Signs

The fungus produces elongate woody swellings (galls) or knots on twigs, branches, and small stems (figs. 18-1 and 18-2). The knots are usually about one to eight inches long and up to one inch thick, but some may reach one foot or more in length. On large branches and stems, infections can result in cankerous galls or rough bark, or both. Scattered black masses of fungal tissue (stromata) that produce fruiting bodies also may be present. Knots become greenish and soft in late spring about one year after infection, but become black and hard over time (fig. 18-2). Old knots may be covered with a white to pink mycoparasitic fungus (*Trichothecium roseum*) (fig. 18-3), and insects often tunnel into the knots.

Figure 18-1—Black knot on chokecherry (James A. Walla, Northern Tree Specialties, used with permission).

Figure 18-2— Old (black) and new (olive green) black knot swellings (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 18-3—Old black knot swelling partially covered by a white parasitic fungus (James T. Blodgett, U.S. Forest Service).

When mature, knot surfaces are covered with hard black fungal fruiting bodies (pseudothecia) composed entirely of stromata. The inner knot is composed of both host and fungal tissues. Pseudothecia produce two-celled club-shaped, olivaceous spores (ascospores) measuring 13 to 18 µm × 4.5 to 7.5 µm.

Disease Cycle

Ascospores (sexual spores) of the fungus are discharged in spring from fruiting bodies on the surface of the knots. Sporulation may occur from the time that tips of green leaves emerge from buds until shoot growth stops. Sporulation is often greatest from the pink/white blossom stage to two weeks after bloom. Rain is required for spore discharge, and spores are carried by rainsplash and wind. Germinating spores penetrate unwounded surface tissue of current-season growth and may also infect older tissues through wounds. Infection is most severe when prolonged moist conditions are accompanied by temperatures between 55 and 77 °F.

Knots appear several months after infection. Depending on host species, cultivar susceptibility, and length of the growing season, some knots are visible by late summer; others do not appear until the following spring. Knots that appear during fall or spring continue growing through the next summer. Conidiospores (asexual reproductive spores) are produced on developing knots beginning about when ascospore discharge ends from mature knots and continuing into midsummer. These spores are considered to have little role in infection.

At least one year and usually two years are required before new knots produce mature fruiting bodies. Fruiting bodies on the old knot die after releasing ascospores in spring, but the fungus often grows into adjacent wood the year before sporulation and produces ascospores there the next year. Some infections may expand for several years, potentially expanding into large branches and stems.

Damage

Severely infected trees can have little value due to physical and aesthetic effects. Heavy black knot infections can significantly reduce plant vigor and fruit production. Older branches beyond knots frequently die. New branches beyond knots often do not develop or new shoots may leaf out and wilt in early summer.

Management

The disease is kept in check by less than optimal infection conditions; thus, management is usually not needed. Unbalanced host-pathogen phenology, partial host resistance, and the presence of mycoparasites can also reduce the need for extensive disease management.

Damage from black knot can be reduced by pruning. Damage can also be mitigated by removing or pruning infected wild hosts in nearby wooded areas and fence rows within 600 ft of the desired host trees. Make pruning cuts three to four inches or more below the knot, as the fungus may extend beyond the swelling. The best times to prune are late winter and early spring, because knots are most easily seen before the tree has leaves and because the source of spores should be removed before the next infection season begins. Pruned knots on the ground are another source of spores that can cause new infections if left in the area, so gather and dispose of all pruned knots before April 1.

Because the disease usually has a two-year life cycle, stems should be closely inspected and knots should be removed for two consecutive years. Infections from the previous year will often be small or not visible when mature knots are pruned the first year. In subsequent years, less removal is required to prevent disease buildup.

If black knot is severe within 200 yards of a planting site, do not plant *Prunus* species, or select resistant species and cultivars when possible. The pathogen is often host specialized, and a species or cultivar thought to be resistant may be susceptible to a pathogen form present in the area that is different from where the plant was tested, or the plant may become infected if new pathogen forms move into the area.

Fungicide sprays can provide some protection, but the protection is not sufficient if sanitation and pruning are not included. Fungicides registered in some states for use against black knot include chlorothalonil, copper hydroxide, mancozeb, and thiophanate-methyl. Full protection would require the fungicide to be present throughout the sporulation period. However, if infection pressure is not severe, then protection from the pink/white blossom stage until two weeks after bloom may be sufficient. Follow label instructions for application timing, precautions, and other information.

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19. Black-Spot Nectria Canker

Marcus B. Jackson; revised from Tubercularia Canker of Siberian Elm and Russian-olive, by Joseph M. Krupinsky and James A. Walla (Riffle and Peterson 1986)

Black-spot Nectria canker causes dieback and mortality of trees weakened by drought, freeze injury, root damage, poor site adaptability, and other stresses. The disease is caused by the fungus *Nectria nigrescens* and is usually identified by mature fruiting bodies of its anamorph (asexual stage) *Tubercularia ulmea,* which resemble raised black spots on the bark of affected trees and shrubs.

Hosts and Distribution

N. nigrescens is found on more than 30 species of maple (*Acer*); birch (*Betula*); peashrub (*Caragana*); bittersweet (*Celastrus*); Russian-olive (*Elaeagnus*); ash (*Fraxinus*); honeylocust (*Gleditisia*); creeper (*Parthenocissus*); ninebark (*Physocarpus*); poplar, aspen, and cottonwood (*Populus*); plum, peach, nectarine, and cherry (*Prunus*); buckthorn (*Rhamnus*); sumac (*Rhus*); currant (*Ribes*); willow (*Salix*); elderberry (*Sambucus*); buffaloberry (*Shepherdia*); mountain-ash (*Sorbus*); lilac (*Syringa*); linden (*Tilia*); and elm (*Ulmus*). This fungus occurs in Europe and temperate North America with extensive damage occasionally observed in northern Great Plains states and the Prairie Provinces. The most severe damage in the Great Plains has been in nonnative Siberian elm (*U. pumila*) and Russian-olive (*E. angustifolia*), as well as in honeylocust (*G. triacanthos*) trees planted outside their native range.

Symptoms and Signs

Flags (recently killed branches with dead leaves still attached) may be the first indication that black-spot Nectria canker is affecting a tree (fig.19-1). Closer examination near the point of infection may show oval to elongate areas of sunken bark, which are sometimes darkened on small, thin-barked branches (fig. 19-2). Gum exudates may occur on some hosts, such as Russian-olive. Removing bark within the sunken area will show dead, discolored (often dark brown to black) cambium and outer sapwood indicative of a branch or stem canker (fig. 19-3). Many different pathogens can cause cankers on the dozens of *N. nigrescens* hosts, so identification of the pathogen by examination of fruiting structures is required.

Figure 19-1—Flagging of Siberian elm caused by black-spot Nectria canker (Marcus B. Jackson, U.S. Forest Service).

N. nigrescens-caused canker is usually confirmed by identifying the anamorph, which fruits more readily both in nature and under culture than the sexual stage (teleomorph). Cushion-shaped fruiting bodies (sporodochia) of *T. ulmea* are fairly common on *N. nigrescens*-killed branches (fig. 19-4), but are not always present. They are 0.25 to 1.7 mm high and 0.3 to 1.7 mm wide with a stalk that may not be readily evident as it is often covered by the bark surface. The tops can be white, cream, tan, orange, red, brown, or black at first, but will typically turn black within two to three weeks after sporodochia form. Stalks are white to whitish red, or, rarely, dark red. One-celled, clear (hyaline), straight, or slightly curved ellipsoidal to cylindrical conidia measure 4.6 to 8.4 µm × 1.5 to 3.0 µm (fig. 19-5). They form on curved (sometimes straight or coiled) hyaline conidiophores on top of sporodochia (fig 19-6).

Figure 19-2—Sunken bark in a canker on the stem of a two-year-old Siberian elm inoculated with *Nectria nigrescens* (Marcus B. Jackson, U.S. Forest Service).

Figure 19-3—Canker margin exposed by peeling bark on a Siberian elm (Marcus B. Jackson, U.S. Forest Service).

Figure 19-4—Fruiting bodies (sporodochia) of *Tubercularia ulmea* (anamorph of *Nectria nigrescens*); lower right, cross-section of a sporodochium (Marcus B. Jackson, U.S. Forest Service).

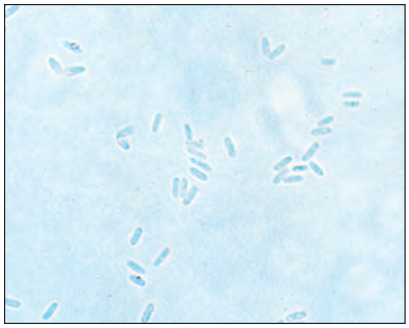

Figure 19-5—Spores of *Tubercularia ulmea* (anamorph of *Nectria nigrescens*) (Marcus B. Jackson, U.S. Forest Service).

T. ulmea fruiting bodies can be confused with those of the coral-spot Nectria canker anamorph, but the former blacken with age, whereas *T. vulgaris* typically retains a lighter color.

Teleomorphic fruiting bodies of *N. nigrescens* are infrequently found in nature. They are red to reddish-brown single or clustered perithecia on raised stromata and are sometimes clustered around the base of a sporodochium. Perithecia are 0.27 to 0.42 mm high and 0.24 to 0.41 mm wide with aseptate to three-septate (mostly one-septate) ascospores that measure 10.5 to 22.0 µm × 2.5 to 8.0 µm (figs. 19-7 and 19-8). They are easily confused with *N. cinnabarina* fruiting bodies but have minor microscopic differences. Rapid field discrimination between the two *Nectria* species is by the color of mature sporodochia. When fruiting bodies are not present, the fungus can be cultured from wood chips taken from the edge of a young canker. *T. ulmea* can produce sporodochia after two to six weeks on potato dextrose agar at room temperature.

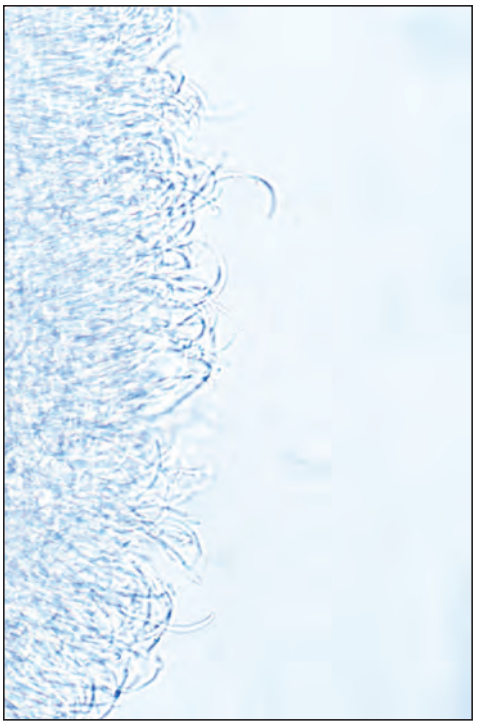

Figure 19-6—Spore-producing structures (sporophores) of *Tubercularia ulmea* (anamorph of *Nectria nigrescens*) (Marcus B. Jackson, U.S. Forest Service).

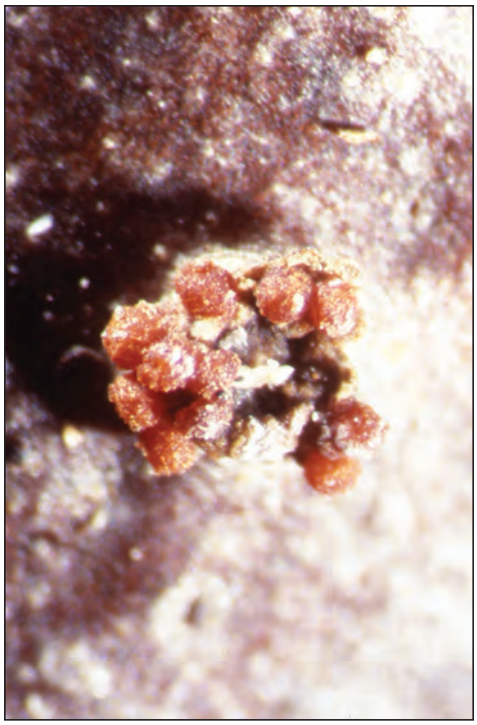

Figure 19-7—*Nectria nigrescens* fruiting bodies (perithecia) (Marcus B. Jackson, U.S. Forest Service).

Figure 19-8—*Nectria nigrescens* ascospores within a spore sac (ascus) (Marcus B. Jackson, U.S. Forest Service).

Disease Cycle

N. nigrescens overwinters as mycelium and fruiting bodies in cankered bark. Although windborne spores of the teleomorph are important for pathogen adaptability and long-distance spread, they are probably insignificant for local disease outbreaks owing to their rare occurrence. Most infections are caused by asexual spores (conidia) that are held in place by a viscous material until liberated by water. They are spread by rainsplash, by adhering to birds or insects, or on pruning tools. Conidia infect bark wounds caused by hail, wind, freeze damage, snow breakage, animals, cultivation, pruning, and possibly herbicides. The fungus invades the sapwood, cambium, and living portions of the bark. It is considered a weak pathogen, causing more damage to stressed trees. In otherwise healthy trees, only small cankers usually develop, which will seal over with woundwood tissue in the next growing season. *N. nigrescens* generally grows more quickly in weakened branches and stems, resulting in more girdling infections that cause flagging and provide a large amount of substrate for more conidial production.

Damage

Although *N. nigrescens* has a large host range, widespread damage is rare and is largely restricted to stressed trees. Root damage from transplanting or excavating near trees, drought stress, freeze injury, and chronic herbicide exposure have all been implicated in significant damage caused by *N. nigrescens*. Extensive damage sometimes occurs on trees planted beyond their native range, such as Russian-olive, Siberian elm, and honeylocust in the Great Plains. Trees that are well adapted to their environment generally have little or no damage from *N. nigrescens*. Therefore, ongoing problems with this pathogen may indicate trees are poorly adapted to a given site or a site requires some form of mitigation to reduce the stress on affected species.

Management

Impacts of black-spot Nectria canker can be reduced with proper woody plant selection, plant stress mitigation, and sanitation. Select well-adapted species suitable to soils, weather conditions, and maintenance capabilities of the site. Some cultivars have been developed with improved tolerance to cold, drought, and common diseases and insects that weaken trees and shrubs. These cultivars may be less prone to *N. nigrescens* damage. Planting adapted species and cultivars may reduce damage from this disease. Because healthy trees and shrubs are less susceptible to damage, maintain proper irrigation and fertilization and avoid injury from soil compaction, root disturbance, and herbicides. Manage other diseases and insects when feasible.

Avoid unnecessary bark wounds. Complete pruning during cool, dry periods when spore dispersal and germination are limited, making infection less likely. Dead branches and trees can be burned or removed from the site to reduce inoculum for future infections. See chapters 20 and 21 for more specific information about managing *N. nigrescens* on host species.

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20. Canker and Dieback of Russian-Olive

Gerard C. Adams, Ned Tisserat, and William R. Jacobi

Russian-olive (*Elaeagnus angustifolia*) is used in windbreaks, shelterbelts, and landscape plantings. It has been planted widely in windbreaks in the northern Great Plains because it survives rigorous environmental conditions. It is particularly tolerant of drought and salinity. In some regions it has become an invasive plant in riparian environments with major negative impacts on native plants. In the Great Plains the tree is susceptible to several fungal canker diseases:

- Phomopsis canker caused by *Phomopsis arnoldiae* (syn. *P. elaeagni*)
- Botryodiplodia canker caused by *Lasiodiplodia theobromae* (formerly *Botryodiplodia theobromae*)
- Black-spot Nectria canker caused by *Nectria nigrescens* (asexual state *Tubercularia ulmea*) and also called Tubercularia canker.

Hosts and Distribution

Phomopsis canker is the most important canker disease of Russian-olive throughout the Great Plains, the North Central states, and the Northeast. The disease was first found in Missouri in 1963 and subsequently in Illinois, Ohio, Delaware, Michigan, and New York. The only other recorded host of *P. arnoldiae* is black walnut (*Juglans nigra*) nursery seedling stock in Indiana. Other species of *Phomopsis* cause cankers and dieback on many plant species. Many other plants also harbor opportunistic *Phomopsis* species that are common endophytes. Inoculation tests can aid in distinguishing aggressive pathogens from opportunists.

Botryodiplodia canker is common on many woody plants. *Lasiodiplodia theobromae* is an opportunist latent pathogen, and is an endophyte with worldwide distribution. The pathogen commonly causes cankers on trees suffering from environmental stress such as drought. More information on Botryodiplodia canker can be found in chapter 16.

Black-spot Nectria canker on Siberian elm (*Ulmus pumila*), Russian-olive, and other common hosts is discussed in chapters 19 and 21. *N. nigrescens* (a member of the *N. cinnabarina* species complex) is a common pathogen of trees suffering from environmental stress such as freezing before full dormancy has been reached.

Symptoms and Signs

Recently killed branches with dead leaves still attached (flags) indicate the presence of a canker that has girdled the stem or branch. Phomopsis and black-spot Nectria cankers are elongate, girdling, reddish-brown to purplish-black, and mostly on small branches, while those of Botryodiplodia cankers are long, narrow strips mostly on stems. There is an obvious canker margin in the bark tissue, especially in younger tissues, visible on the bark surface for Phomopsis and Tubercularia cankers (fig. 20-1), but only by excising the bark for Botryodiplodia cankers. The sapwood immediately beneath the bark canker is brown, and this browning may extend beyond the margin of the canker. Frequently gummosis occurs around the canker margins. Phomopsis and black-spot Nectria cankers often develop on current-year shoots. Site of development of Botryodiplodia cankers on Russian-olive is not known, but most cankers are found on larger stems. Young shoots are girdled quickly, wilting the new silvery foliage. Cankers also have been found on branches up to four inches in diameter, and larger for Botryodiplodia cankers. Black-spot Nectria and Phomopsis cankers often look similar, and all three cankers may be present in the same area or tree, particularly in the central Great Plains.

The asexual fruiting bodies (pycnidia) of *Phomopsis* form on infected bark in the same season the bark is killed. Pycnidia with multiple locules are numerous and clustered in cankers, erupt through the bark, and measure approximately 500 µm × 275 µm. Asexual spores (conidia) in pycnidia, or exuding in moist weather, are of two types known as alpha and beta. Alpha conidia are short (5 to 11µm long), ovoid, blunt, and straight (fig. 20-2); beta conidia are long (15 to 26 µm), filiform, and curved (fig. 20-3). Both spore types are produced on plant tissue and sometimes in culture. The beta conidia act as spermatia and are not infective.

Figure 20-1—Discolored bark of Phomopsis canker on young Russian-olive branch with asexual fruiting bodies (pycnidia) beginning to erupt through bark in cankered area (Ned Tisserat, Colorado State University, used with permission).

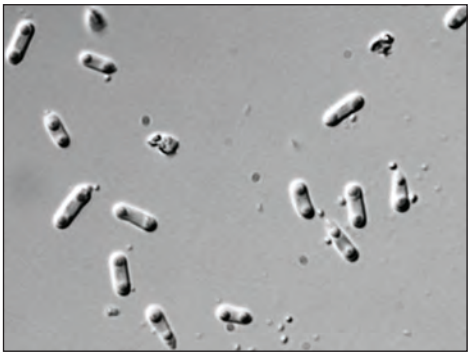

Figure 20-2—Alpha conidia of *Phomopsis* spp. are oval and colorless, 5-11 µm long (Paul Bachi, University of Kentucky, Bugwood.org, used with permission).

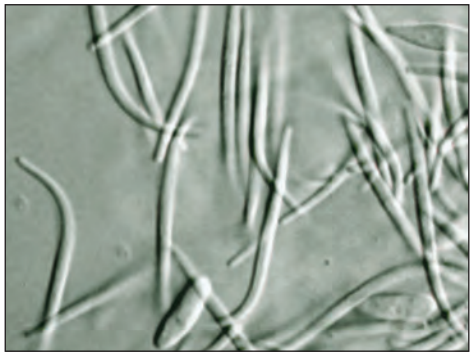

Figure 20-3—Beta conidia of *Phomopsis* spp. are filiform and curved, 15-26 µm long (Bruce Watt, University of Maine, Bugwood.org, used with permission).

The pycnidia of *L. theobromae* also can form on infected bark in the same season the bark is killed. Additionally, they are often observed on twigs and branches that died a few years earlier. The pycnidia erupt through bark separately (fig. 20-4) or as groups in fused clusters. The fused clusters may superficially appear as one fruiting body with multiple locules, but individual pycnidia have one locule. The compound fruiting body is large (1-5 mm), whereas the individual pycnidia are about 250 µm in diameter. The conidia begin as colorless single cells and then become brown two-celled bodies ornamented with striations (figs. 20-5 and 20-6). *Lasiodiplodia* is readily distinguished from *Phomopsis* by the large ellipsoid conidia that measure 20 to 30 µm × 10 to 15 µm (fig. 20-6).

The asexual fruiting bodies (sporodochia) of black-spot Nectria canker are usually observed on twigs and branches that have died the previous year but may appear in the same year as infection, particularly in branches that are rapidly killed. Sporodochia are distinctive cushions erupting through the bark. Initially they may be cream, tan, reddish, brown, or black but turn black about two or more weeks after eruption (fig. 20-7). In North Dakota and South Dakota, *N. nigrescens* is more frequently isolated from the cankers than *Phomopsis* or *Lasiodiplodia*. The conidia are colorless and oval (fig. 20-8).

When fruiting bodies are not present, the pathogens are confirmed by culturing wood chips taken from the edge of the canker, on potato dextrose agar or similar media. *Phomopsis* produces white fluffy aerial hyphae and pycnidia in stromata. *Lasiodiplodia*

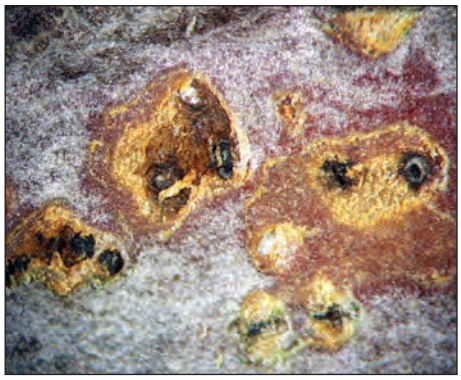

Figure 20-4—The asexual fruiting bodies (pycnidia) of the Botryodiplodia canker pathogens erupt through the bark separately or in clusters (Paul Bachi, University of Kentucky, bugwood.org, used with permission)..

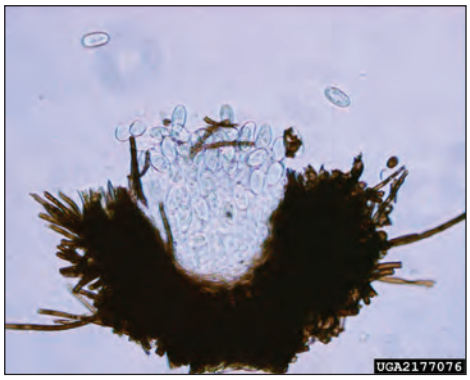

Figure 20-5—A cross-sectional cut through a pycnidium demonstrates that the large conidia (20-30 × 10-15 µm) of *L. theobromae* are colorless and one-celled at first, before becoming dark brown and two-celled (Cesar Calderon, USDA APHIS PPQ, Bugwood.org, used with permission).

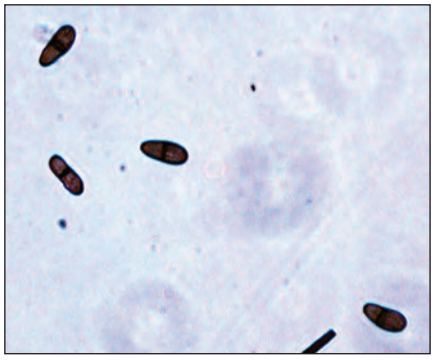

Figure 20-6—The mature conidia of *L. theobromae* are two-celled, dark brown, and large (20-30 × 10-15 µm) (Photo: Cesar Calderon, USDA APHIS PPQ, Bugwood.org, used with permission).

Figure 20-7—The black-spot fruiting bodies (sporodochia) are usually visible on the bark of the infected branch (Bruce Watt, University of Maine, Bugwood.org, used with permission).

Figure 20-8—The conidia of the black-spot Nectria canker pathogen are produced on the sporodochia and are colorless and oval (Bruce Watt, University of Maine, Bugwood.org, used with permission).

produces gray aerial hyphae and pycnidia in stromata. *Nectria* produces sporodochia usually within two months of incubation on potato dextrose agar at room temperature.

Disease Cycle

The life cycle of these parasites has not been demonstrated, but studies of Phomopsis, Botryodiplodia, and black-spot Nectria cankers in other hosts indicate that conidia infect through wounds and become established as endophytes in the sapwood. While the fungi are in the previous-year sapwood and sometimes the leaves, new growth remains free of the fungi for a period and is susceptible to infection. It is assumed that new growth becomes infected by conidia. It is not known whether infections can occur throughout the growing season. Cankers develop within a few days following natural infection or artificial inoculation.

Sporulation on infected tissue also follows quickly on black-spot Nectria and Phomopsis cankers, but is several weeks delayed for Botryodiplodia canker. Black-spot Nectria and Phomopsis cankers are annual and delimited by an active host response. Cankers on older tissues occur during periods of stress when the host defense response is compromised. Infection on older tissue is believed to result from expansion of endophytic colonies of the previously latent pathogen. Botryodiplodia cankers are typically perennial with no apparent host response.

Damage

In the northern Great Plains, these canker diseases contribute to the decline of Russian-olive health, and damage to the function of shelterbelts and windbreaks that include the species. The canker diseases are usually restricted to trees under environmental stress and the stress may be present in subsequent years. Such trees exhibit increasing dieback of branches, resulting in decreasing health and aesthetic appearance of the canopy over the years. Both disease incidence and severity increase with age until the quality of plant appearance is no longer acceptable and the plants become increasingly susceptible to other pathogens and pests.

Infected Russian-olive and black walnut nursery stock may suffer severe damage from Phomopsis canker, especially in the North Central states. Terminal dieback of black walnut nursery stock and outplanted saplings leads to multiple-stemmed trees. Careful inspection of healthy-appearing Russian-olive ornamental plants may reveal that as much as 52 percent have the disease, as was found in a study in southeastern Michigan. It is recommended that diseased nursery stock be disposed of by being buried, burned, or composted away from production plants.

Management

Because healthy trees are less susceptible to infection and damage, manage trees for optimum vigor. If possible, water trees during prolonged droughts. Establish new plantings on good sites with vigorous planting stock and control weeds. Reduce chances of infection by preventing wounds to the bark. Because infected or dead branches of trees are a source of fungal inoculum, remove the branches from the site and burn them. Prune infected branches back to the nearest living branch six inches beyond the canker. Prune trees during dry weather, and disinfect pruning tools after each cut.

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21. Cankers and Root Collar Rot of Honeylocusts

William R. Jacobi and James A. Walla

Branch and stem cankers are among the most serious problems of honeylocusts and can be caused by various fungal pathogens. Thyronectria canker is caused by *Pleonectria austroamericana*, formerly *Thyronectria*. Black-spot Nectria canker, formerly called Tubercularia canker, is caused by *Nectria nigrescens*, the recently described sexual stage of *Tubercularia ulmea*. More information on this canker can be found in chapter 19. Coral-spot Nectria canker is caused by *Nectria cinnabarina*, the sexual stage of *Tubercularia vulgaris*. Root collar rot of honeylocust acts like a girdling canker at the soil line; the pathogen is not known.

Hosts and Distribution

Thyronectria canker occurs on native thorny honeylocust (*Gleditsia triacanthos* var. *triacanthos*), all cultivars of thornless and podless honeylocust (*G. triacanthos* var. *inermis*) and oriental honeylocust (*G. japonica*). The fungus *P. austroamericana* also colonizes and produces spores on dead tissue of several other hardwoods including American elm (*Ulmus americana*), white ash (*Fraxinus americana*), mockernut hickory (*Carya tomentosa*), willow (*Salix* spp.), and bur oak (*Quercus macrocarpa*). The disease has been reported from Colorado and Oklahoma eastward to Massachusetts and Georgia. The disease is common in the central United States, where it can cause major damage. It is uncommon in the Northeast and in Great Lake states such as Minnesota and Wisconsin, and it has not been found in the northernmost states and provinces of the Great Plains, so fungus distribution may be restricted by colder climates.

Black-spot Nectria canker is common on many hardwood and coniferous trees and shrubs. The fungus *N. nigrescens* is found on honeylocusts, maples (*Acer* spp.), Russian-olive (*Elaeagnus angustifolia*), winged euonymus (*Euonymus alatus*), green ash (*F. pennsylvanica*), American (*U. americana*) and Siberian elm (*U. pumila*), and many other woody plants. Among honeylocusts, those planted outside of their native range are the most susceptible to damage from this disease. The disease is found commonly from Colorado eastward and from the northern Great Plains states northward to Manitoba and Alberta.

Coral-spot Nectria canker is found on many tree species, and honeylocusts are a common host of *N. cinnabarina* in some areas of the Great Plains. The disease can be found from the northern to the southern Great Plains but is the least common canker disease on honeylocusts of these three described.

Root collar rot is a common disease on honeylocusts in the drier regions of the Great Plains, where irrigation practices can keep the soil saturated at the base of the tree. Occurrence of the disease is also related to weather conditions that apparently promote the disease in the dormant season. Thus, the disease can be seen girdling trees for several years in a row, and then there can be several years without return of the disease.

Symptoms and Signs

Symptoms of the four diseases include dieback of affected branches, reduced foliage, yellow foliage, premature fall coloration, and early leaf drop.

- **Cankers** range from slightly flattened surfaces to distinctly sunken areas with large ridges of woundwood at the canker margin. Loose bark and woundwood at the tree base can indicate root collar rot. Thin-barked areas of stems and branches may have a red-yellow discoloration associated with the canker. Usually there is no discoloration where the bark is thicker. Wood beneath infected bark turns wine-red to yellow for Thyronectria canker.
- **Honeylocust root collar rot** symptoms include discolored and loose bark and discolored wood at the soil line. Early fall coloration of a portion of the tree may indicate a large amount of damage. Small drops of gum on the stem or root sprouting usually indicate girdling. Loose bark and discolored (yellow to brown instead of white) wood just below the bark indicate initial collar rot and are the most indicative symptoms.

Signs of the pathogen often allow for better disease identification than do symptoms.

- **Thyronectria cankers** have irregularly shaped clumps of stroma (fig. 21-1, 21-2, 21-3) containing irregularly shaped chambers lined with conidiophores (fig. 21-4) (stromatic conidiomata) instead of neatly organized round pycnidia with necks. Stromatic conidiomata are light pink or yellow-brown when fresh and become blackened with age. Perithecia are yellowish brown or reddish gray with dark, often black, shining apical regions and also darken with age (fig. 21-5). Both fruiting body types are usually found in natural openings such as lenticels in thick-bark areas and scattered on bark surfaces in thin-bark areas. Asexual fruiting bodies (pycnidia) exude milky masses of conidia (fig. 21-6). Conidia are hyaline, ovoid to ellipsoid,

Figure 21-1—Thyronectria canker on a honeylocust with asexual fruiting bodies (pycnidia) growing through bark lenticels (William R. Jacobi, Colorado State University, used with permission).

Figure 21-2—Asexual fruiting bodies (pycnidia) in Thyronectria canker in cracked bark (William R. Jacobi, Colorado State University, used with permission).

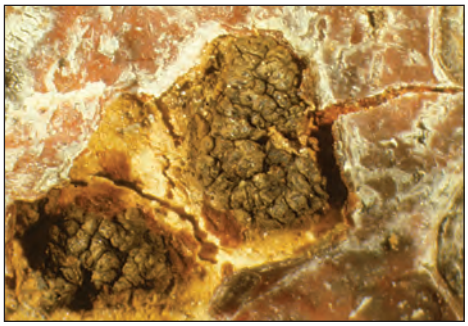

Figure 21-3—Asexual fruiting bodies (pycnidia) in Thyronectria canker (William R. Jacobi, Colorado State University, used with permission).

Figure 21-4—*Pleonectria austroamericana* conidia and conidiogenous cells (Yuuri Hirooka, University of Maryland, used with permission).

Figure 21-5—Perithecia in Thyronectria canker (Yuuri Hirooka, University of Maryland, used with permission).

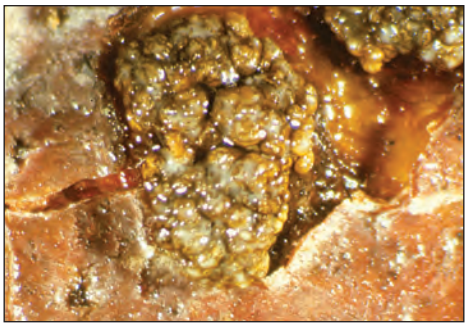

Figure 21-6—Creamy-white asexual spores (conidia) oozing from fruiting bodies of Thyronectria canker (William Jacobi, Colorado State University, used with permission).

one-celled, and 1.6 to 4.2 µm × 0.8 to 2.7 µm in size. Sexual fruiting bodies (perithecia) develop in rounded clusters in a stroma, and are smooth-surfaced and egg-shaped with a short neck. Spore sacs (asci) contain eight ellipsoid to pear-shaped ascospores that are hyaline to pale yellow with both transverse and longitudinal septa and range in size from 8 to 16 µm × 5 to 9 µm (fig. 21-7, 21-8). Cultures of *P. austroamericana* on potato dextrose agar have somewhat waxy margins with orange, slimy centers containing masses of conidia (fig. 21-9).

- **Black-spot Nectria cankers** (fig. 21-10, 21-11) appear very similar to Thyronectria cankers, and are best distinguished by cultural characteristics, morphology of the asexual fruiting structures, and ascospore morphology. The asexual sporodochia are creamy to peach colored when fresh, but normally turn dark brown to black within a few days of drying conditions (fig. 21-12). They usually form exposed on the bark surface or erupt from a stroma under a thin layer of bark. A fresh squash mount of the sporodochia will show curved conidiogenous cells (fig. 21-13, 21-14). Perithecia are reddish brown and form singly or in groups of up to 20 on a raised fungal stroma and can be found clustered at the base of sporodochia (fig. 21-15, 21-16). Ascospores are ellipsoid to fusiform, smooth walled, one- to three-septate, and 13.5 to 18.0 µm × 3.5 to 5.5 µm in size (fig. 21-17). *N. nigrescens* cultures are white and fluffy.

Figure 21-7—*Pleonectria austroamericana* asci with multicelled sexual spores (Yuuri Hirooka, University of Maryland, used with permission).

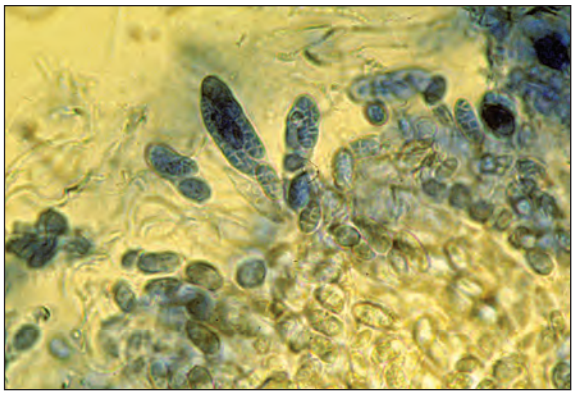

Figure 21-08—*Pleonectria austroamericana* sexual spores (ascospores), mostly in asci (William Jacobi, Colorado State University, used with permission).

Figure 21-9—Culture plate of *Pleonectria austroamericana* with orange, slimy center (Yuuri Hirooka, University of Maryland, used with permission).

Figure 21-10—Black-spot Nectria canker with black sporodochia on honeylocust (William R. Jacobi, Colorado State University, used with permission).

Figure 21-11—Black-spot Nectria canker at branch crotch (William R. Jacobi, Colorado State University, used with permission).

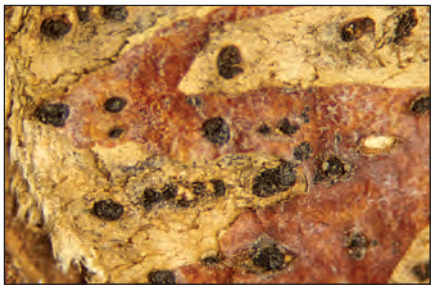

Figure 21-12—Black-spot Nectria canker with old darkened sporodochia on bark (William R. Jacobi, Colorado State University, used with permission).

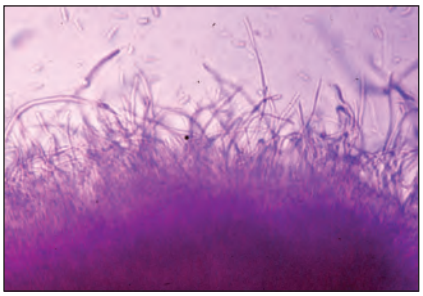

Figure 21-13—Sporodochia with curved conidiogenous cells (spore-bearing structures) and conidia of *Nectria nigrescens* (William Jacobi, Colorado State University, used with permission).

Figure 21-14—Curved conidiogenous cells and spores (conidia) of *Nectria nigrescens* (Yuuri Hirooka, University of Maryland, used with permission).

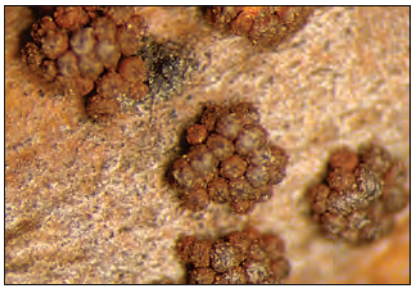

Figure 21-15—Clumps of fruiting bodies (perithecia) in black-spot Nectria canker (William R. Jacobi, Colorado State University, used with permission).

Figure 21-16—Creamy colored sporodochia surrounded by red fruiting bodies (perithecia) in black-spot Nectria canker (Yuuri Hirooka, University of Maryland, used with permission).

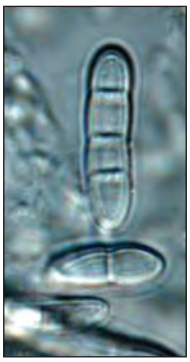

Figure 21-17—Sexual spores (ascospores) of *Nectria nigrescens* (Yuuri Hirooka, University of Maryland, used with permission).

- **Coral-spot Nectria cankers** (fig. 21-18) appear similar to the two other cankers, but *N. cinnabarina* may form sporodochia that are creamy to bright coral colored when young (usually for several weeks) and tan, brown or black after overwintering (fig. 21-19). Perithecia form in late summer singly or in groups of up to 15 and are bright red to reddish brown (fig. 21-20, 21-21). Ascospores are one- to two-septate, fusiform, and 14.0 to 17.5 µm × 4.0 to 5.5 µm (fig. 21-22), similar to *N. nigrescens* but unlike those of *P. austroamericana*. *N. cinnabarina* cultures are white and fluffy.

Disease Cycle

The three canker-causing fungi overwinter as mycelium and fruiting structures on infected trees. Conidia presumably are spread by rain and ascospores by wind. Infections take place through wounds and natural openings. Because these fungi can exist as saprophytes, they can become established on dead wood such as branch stubs or wound edges.

Figure 21-18—Discolored honeylocust bark from coral-spot Nectria canker (William R. Jacobi, Colorado State University, used with permission).

Figure 21-19—Sporodochia of coral-spot Nectria canker in honeylocust (William R. Jacobi, Colorado State University, used with permission).

Figure 21-20—Red fruiting bodies (perithecia) of coral-spot Nectria canker (William Jacobi, Colorado State University, used with permission).

Figure 21-21—Red fruiting bodies (perithecia) surrounding white sporodochia of coral-spot Nectria canker (Yuuri Hirooka, University of Maryland, used with permission).

As pathogens, the fungi grow in the cambium and outer xylem, where they eventually kill the cambium and surrounding cells. Trees, or affected parts, die because of cambial death and possibly vascular dysfunction. Sporodochia can form within one month after tissue is colonized, and are abundant on bark of dying or dead trees. Perithecia form in affected areas but are not as common or abundant as pycnidia or sporodochia.

The disease cycle of honeylocust root collar rot involves interactions with pathogenic soil organisms through unknown processes. Frequent irrigation that keeps the soil wet at the tree base allows soil microorganisms to kill the bark and cambium just below soil line. *P. austroamericana*, *N. cinnabarina*, or *N. nigrescens* then may infect the weakened tree above the area previously killed by root collar rot and cause large cankers that can be mistaken for the primary disease.

Damage

Cankers at the tree base are usually fatal. Cankers at the main stem or branch crotch may cause complete girdling, depending on the tree's health. Stressed trees cannot defend against the fungi, whereas vigorous trees should be able to stop the fungus invasion and recover. The amount of damage by the canker diseases across the United States is not known, but they are found on many hosts and in particular on honeylocust in urban, rural, and windbreak plantings. Root collar rot can kill trees rapidly.

Management

Honeylocust cankers and root collar rot should be prevented rather than controlled. Physical damage and wounds should be avoided or properly cleaned and allowed to dry. Susceptibility to these pathogens is increased by a variety of stresses, including drought, overwatering, herbicide damage, and restricted area or restricted oxygen for root growth. Trees should be watered adequately but not excessively. Frequent irrigation of turf is a

Figure 21-22—Sexual spores (ascospores, left) of *Nectria cinnabarina* (Yuuri Hirooka, University of Maryland, used with permission). Asexual spores (conidia, right) and conidiogenous cells of *Nectria cinnabarina* (Yuuri Hirooka, University of Maryland, used with permission).

factor that creates conditions favoring root collar rot. This disease can be prevented by keeping the soil at the base of trees as dry as possible. Place sprinklers and sprinkler heads far away from trees to minimize the amount of water falling on the tree stem and at the tree base. Remove flowers, turf, or other vegetation that requires frequent irrigation from around the tree's base and replace with small gravel or organic mulch. When planting honeylocust trees, place the top of the root ball six inches above grade to allow for drainage away from the tree trunk. Black-spot Nectria canker rarely occurs on 'Northern Acclaim' honeylocust in the northern Great Plains, likely owing to its superior cold hardiness. 'Sunburst' honeylocust is the most susceptible to Thyronectria canker. 'Imperial,' 'Skyline,' and thornless selections are more resistant.

Pruning infected branches can reduce the chances of other infections. Trees should be pruned in cool, dry weather when the presence of fungal spores is reduced and when exposed bark will be sealed over the soonest. Small cankers may be scribed out if the tree is reasonably vigorous.

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22. Crown Gall

Simeon Wright; revised from Michael W. Ferguson and Richard Dorset (Riffle and Peterson 1986)

Crown gall is a tumor-forming plant disease caused by *Rhizobium radiobacter* (formerly *Agrobacterium tumefaciens*) and related bacterial species occurring around the world. It is a serious issue for the nursery industry and affects both broadleaf and conifer species (fig. 22-1). Crown gall can sometimes be challenging to diagnose because of similarities with other galls and the prevalence of nonpathogenic strains of *Rhizobium*.

Figure 22-1—Crown gall on juniper seedling (William R. Jacobi, Colorado State University, used with permission).

Hosts and Distribution

More than 600 host plants in more than 90 families are susceptible to crown gall, but natural symptom development has not been observed in the field for all species. Species and strains of *Rhizobium* are highly species specific. Apple and crabapple (*Malus* spp.), cottonwood (*Populus* spp.), plum and cherry (*Prunus* spp.), and willow (*Salix* spp.) are frequently reported as hosts in the Great Plains, although other species are reported as well.

Symptoms and Signs

Individual swellings (galls) develop on roots or stems, often at the base (root collar) of the tree (fig. 22-2). Several galls may be present along a stem or root (fig. 22-3). Gall diameter varies from less than 1 inch to more than 12 inches (fig. 22-4). The exterior surface of galls is usually rounded with a rough, irregular surface that darkens with age (fig. 22-5). The interior can be spongy or hard and solid with irregular structure. Galls caused by insects, mites, and fungi may look similar but often contain holes or chambers or have a nodular exterior.

Disease Cycle

R. radiobacter is a soil-inhabiting bacterium infecting fresh wounds that result from mechanical or environmental injury such as propagation, pruning, freezing, insect or nematode damage. After entering the plant, bacterial DNA is transferred to the host, stimulating increased cellular growth and division within that region of the host tissue through the production of phytohormones. The swollen tissue that develops forms the gall. Trees may be stunted when the woody tissue is disrupted, preventing flow of water

Figure 22-2—Crown gall symptoms at the root collar (Aaron D. Bergdahl, North Dakota State University, used with permission).

Figure 22-3—Multiple galls along tree roots (William R. Jacobi, Colorado State University, used with permission).

Figure 22-4—Large aerial gall on cottonwood (William R. Jacobi, Colorado State University, used with permission).

Figure 22-5—Rough gall exterior at cottonwood tree base (William R. Jacobi, Colorado State University, used with permission).

or nutrients. Chewing insects or tools can carry the bacteria from plant to plant. Long-distance movement is also possible by transport of infected nursery stock or by contaminated soil, irrigation water, or tools such as pruning or grafting equipment. Symptoms may not appear for several weeks after infection, depending on weather conditions. Gall development is favored by temperatures near 72 °F. Bacteria return to the soil from galls and can persist for long periods of time, colonizing root surfaces of nonsusceptible plants or as a latent infection on susceptible hosts. Crown gall is favored by soils of neutral or higher pH.

Damage

Infection of large trees is infrequent and may not result in economic loss. Infection of nursery stock can be quite extensive, especially when inoculation takes place during propagation. Small trees may become stunted and more susceptible to environmental stress. In most states there is no tolerance for nursery stock infected with crown gall; all infected plants are destroyed.

Management

Always use disease-free planting stock and propagation materials. Extension guides list resistant species that can be planted in infested areas. Avoid mechanical wounds on young trees or bushes. Sterilize cutting instruments frequently by using a disinfectant such as 70 percent alcohol. Remove and destroy all infected plants and rotate with a nonsusceptible crop (corn, small grains, other grass) for two to four years. As a preventive pre-planting treatment, nursery stock can be dipped in a suspension containing a related antagonistic bacterial species *R. rhizogenes* (syn. *Agrobacterium radiobacter*) (Strain K84 or K1026) to protect against damage from sensitive strains of *R. radiobacter*.

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23. Cryptodiaporthe (Dothichiza) Canker of *Populus* Species

Michael E. Ostry and Jared M. LeBoldus; revised from David W. Johnson and Robert W. Stack (Riffle and Peterson 1986)

Disfiguring and often lethal stem cankers caused by *Cryptodiaporthe populea* affect primarily black poplars (*Populus nigra* varieties) in nurseries, plantations, and landscape plantings in North America.

Hosts and Distribution

Cryptodiaporthe canker (also known as Dothichiza canker) caused by the fungus *C. populea* is a widespread disease affecting poplars in nurseries, plantations, and landscape plantings throughout the Northern Hemisphere and in South America. Susceptibility to the disease varies by poplar species, but hybrids in sections Aigeiros (cottonwoods, black poplars) and Tacamahaca (balsam poplars) are the most frequently and severely affected. Lombardy poplar (*P. nigra* var. *italica*), which was commonly used for ornamental line plantings, is so highly susceptible that its life is greatly shortened and it is no longer recommended for use. The disease is not significant on aspen (*P. tremuloides*) in native stands.

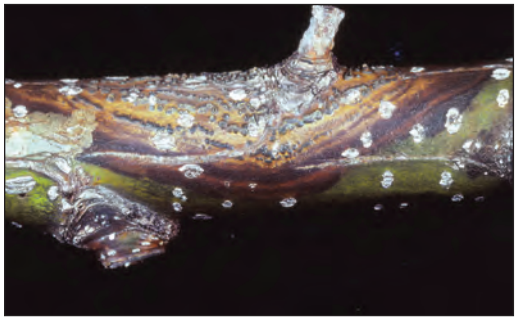

Figure 23-1—Young branch Cryptodiaporthe canker with fruiting bodies (pycnidia) protruding through the discolored bark (Michael E. Ostry, U.S. Forest Service).

Symptoms and Signs

Young, developing cankers, often at branch axils, begin as discolored patches of bark on twigs, branches, and stems of trees of any age. Cankers enlarge and the bark within the canker margins becomes slightly sunken. Raised, dark, pimplelike fruiting structures (pycnidia) containing spores (conidia) of the asexual state of the fungus (*Discosporium populeum*; syn. *Dothichiza populea* and *Chondroplea populea*) develop within the infected bark (fig. 23-1). Later, the bark cracks and extensive woundwood often develops around the perennial cankers, resulting in rough, warty areas on larger branches and stems (fig. 23-2). Adventitious shoots often develop below cankers that have girdled stems.

Figure 23-2—Perennial, rough stem Cryptodiaporthe cankers with extensive woundwood (Michael E. Ostry, U.S. Forest Service).

Disease Cycle

Conidia, lemon shaped and 9 to 11 µm × 7 to 8 µm in size (fig. 23-3), are released in cream-colored tendrils from pycnidia within the canker margins during wet weather throughout the growing season (fig. 23-4). The conidia are dispersed by rainsplash or inadvertently carried by insects or birds to infection sites such as buds, leaf scars, lenticels, and various wounds on adjacent trees. Ascospores, produced by the sexual state of the fungus, develop in small, flask-shaped fruiting bodies (perithecia), and are not as commonly encountered as the conidia. These spores presumably are also infective

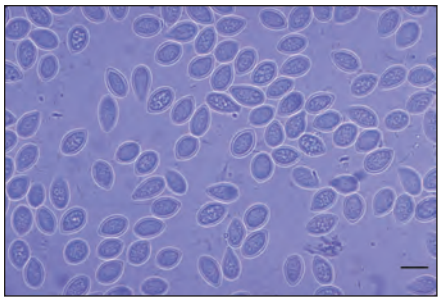

Figure 23-3—Lemon-shaped asexual spores (conidia) of the Cryptodiaporthe canker. Scale bar is 10 µm (Michael E. Ostry, U.S. Forest Service).

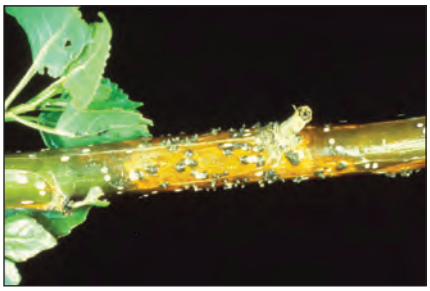

Figure 23-4—Cream-colored tendrils of asexual spores (conidia) exuded from fruiting bodies (pycnidia) in a Cryptodiaporthe canker (Michael E. Ostry, U.S. Forest Service).

Figure 23-5—Stem breakage at Cryptodiaporthe canker (Michael E. Ostry, U.S. Forest Service).

and involved in spread of the fungus. The pathogen overwinters as spores and mycelium within cankers. After infection, cankers can rapidly expand in the bark of trees under moisture stress, but their growth may be arrested by tree defensive mechanisms, such as woundwood production, with the return of normal growing conditions. However, because the fungus can persist in the bark and wood, the cankers may begin to expand when trees are again exposed to stressful conditions.

Damage

The fungus causes bark necrosis, cankers, dieback, and death of affected trees in nurseries and established tree plantings. Affected trees are disfigured and the longevity of highly susceptible trees used in landscape plantings is greatly reduced. Cankers girdle or weaken tree branches and stems, and invasion by decay fungi into the open cankers results in reduction in wood quality and stem breakage (fig. 23-5). The disease is most severe on trees under moisture stress and trees stressed or damaged by other abiotic and biotic agents.

Management

Poplar varieties known to be vulnerable to Cryptodiaporthe canker should not be planted. In addition, avoid poplar cultivars and clones highly susceptible to leaf diseases, because premature defoliation stresses trees. The resulting leaf scars provide infection sites for *C. populea* during the growing season. Only vigorous, disease-free stock should be planted. The fungus can be harbored on hardwood cuttings and nursery stock without visible symptoms. Apply nursery practices that maintain tree vigor such as prudent fertilization and weed control. Remove diseased trees and plant material to reduce fungal inoculum. Store and handle planting stock to avoid moisture stress and conditions that favor the development of the pathogen. Avoid wounding trees and prevent moisture stress to minimize the risk of infection and damage by the fungus in landscape plantings and plantations.

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24. Cryptosphaeria Canker of Cottonwood and Aspen

James J. Worrall, Kelly S. Burns, and Jennifer Juzwik; revised from Thomas E. Hinds and Jerry W. Riffle (Riffle and Peterson 1986)

Cryptosphaeria canker, caused by the fungus *Cryptosphaeria ligniota* (syn. *C. populina*), is a destructive disease of aspen and other poplars (*Populus* spp.). Although the fungus had been collected from Colorado as early as 1897, it was not associated with a canker disease until 1969. The anamorph (asexual stage) is a *Cytosporina* species (often referred to as *Libertella* species).

Hosts and Distribution

Quaking aspen (*P. tremuloides*) is the principal host of *C. ligniota*, but the disease also occurs on other *Populus* species of the Great Plains, including narrowleaf (*P. angustifolia*), eastern (*P. deltoides*), and plains (*P. deltoides* ssp. *monilifera*) cottonwoods; Lombardy (*P. nigra* var. *italica*), balsam (*P. balsamifera*), and black (*P. nigra*) poplars; and some hybrid poplars. The disease is widely distributed and has been found throughout much of North America and in Europe.

Symptoms and Signs

Cryptosphaeria cankers are usually much longer and narrower than other cankers of aspen and may spiral snakelike around the stem, following the grain of the underlying wood (fig. 24-1). For this reason, the disease is sometimes referred to as "snake canker." Cankers may be only two to four inches wide but 10 ft long. They may extend for much of the stem length, but more often occur primarily in the portion of the stem with live branches. Bark recently killed near the margin of the canker becomes discolored light brown to orange. Reddish-brown fluid may bleed from the margin. Within one to two years, dead inner bark becomes stringy, black, and sooty, and contains diagnostic small (0.5 to 2.0 mm), lenticular, light-colored spots (fig. 24-2). Annual woundwood formation may be visible but does not restrict canker growth. Dead bark adheres tightly to cankers.

Figure 24-1—Cryptosphaeria canker killing an aspen. The canker spirals around the stem up to the live crown (James J. Worrall, U.S. Forest Service).

The disease is a canker rot, which means that the fungus advances in and decays wood, causing cankers where the infection reaches the cambium and bark. Wood is stained in hues of gray, brown, yellow, orange, and even pink. The decay is brownish and mottled and is probably a type of soft rot similar to white rot, as is typical of wood-decaying ascomycetes. Wood staining can extend longitudinally three feet or more above and below the canker margin. The pathogen can be isolated readily from cankers and underlying wood.

Sexual fruiting bodies (perithecia) form in narrow patches up to 0.5 inch wide and 1 foot long on the surface of bark that has been dead at least one year

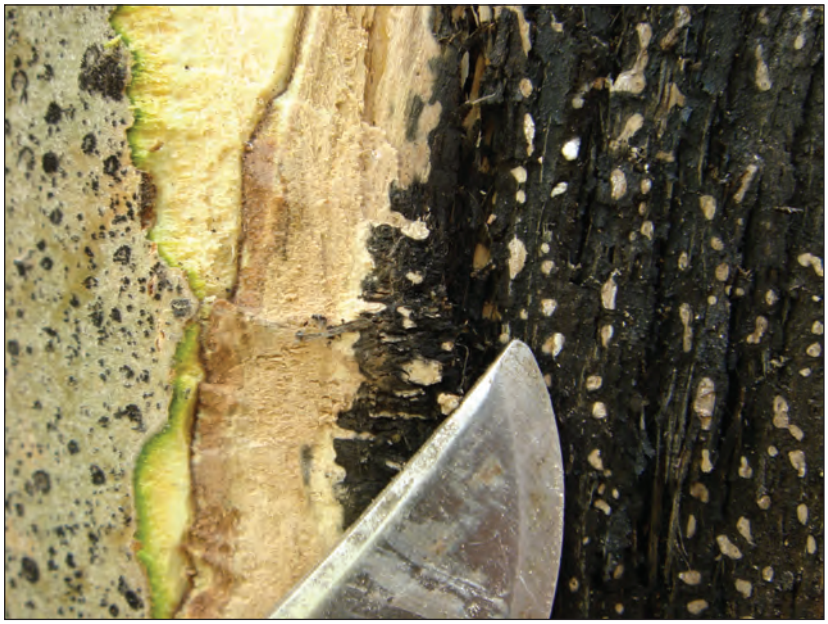

Figure 24-2—Light-colored lenticular spots that frequently appear in older dead bark of Cryptosphaeria cankers. Live bark is visible at the left-most margin of the cut (James J. Worrall, U.S. Forest Service).

(fig. 24-3). The fungus readily fruits on branches and smooth bark, but perithecia are uncommon both on small trees and on thick, corky bark of old trees. Light-orange fruiting bodies (acervuli) of the *Cytosporina* (*Libertella*) stage are occasionally found near the canker margin on aspen.

Cytospora species frequently infect and fruit at the margin of Cryptosphaeria cankers and quickly colonize trees after they die, so the cause of death may be erroneously attributed to Cytospora canker. For this reason, and because the pathogen may not fruit reliably, diagnosis should be made by looking for the lenticular, light-colored areas in bark and the staining in the sapwood.

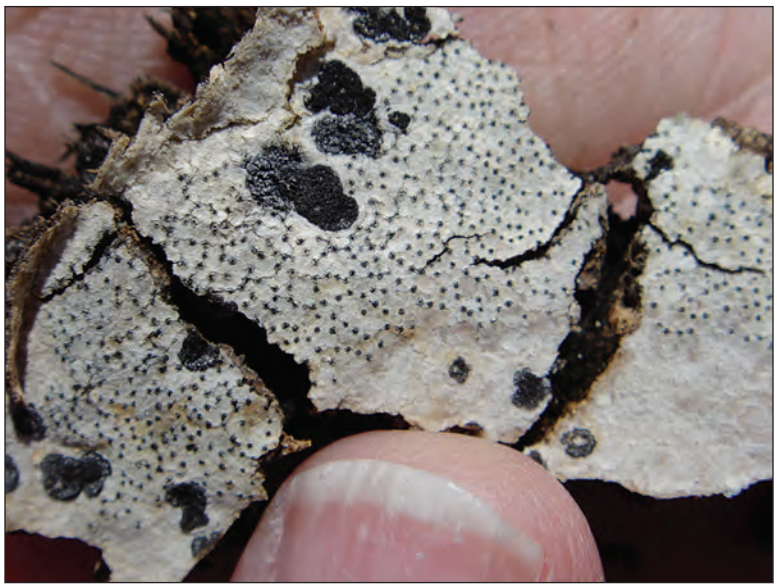

Figure 24-3—Sexual fruiting bodies (perithecia) of *Cryptosphaeria ligniota* (numerous small, dark dots) (James J. Worrall, U.S. Forest Service).

Disease Cycle

Infection occurs primarily through branch and trunk wounds. Initially, the pathogen colonizes the heartwood and sapwood (xylem), causing discoloration and decay, but the fungus eventually grows out from the wood to the cambium and bark (phloem), killing the cambium and causing a canker. Wood staining can extend longitudinally three ft or more above and below the canker margin. Trees usually die before the canker completely girdles the stem.

Black perithecia may appear scattered on bark that has been dead for at least one year. The perithecia develop singly in a grayish pseudostroma made of dense fungal hyphae and dead bark just beneath the surface. Sexual spores (ascospores) are forcibly ejected from perithecia during wet weather and are presumably dispersed by wind. Spores from the *Cytosporina* stage likely function as fertilizing elements rather than causing infection.

Damage

Cryptosphaeria canker is an important killer of aspen. In one Colorado survey, 83 percent of 30 surveyed sites had Cryptosphaeria canker, and 26 percent of aspen mortality was attributed to the disease. It seemed to kill mostly small to midsize trees. Cryptosphaeria was the canker most frequently found on trees in diameter classes ranging from three to six inches and was found only on trees less than 11 inches in diameter. It is estimated that eight percent of aspen in Colorado have decay caused by *C. ligniota*. Cryptosphaeria canker is one of a number of wound-infecting cankers that lead to mortality following partial cutting of aspen stands and also in developed recreation sites established in aspen.

Mortality usually occurs in small-diameter trees after only a few years and before the canker girdles the stem. A large volume of sapwood can be killed, causing more damage than the canker itself. On larger trees, infections may start in branches, and then spread into the stem, where they can grow up and down and girdle other branches, killing the tree one branch at a time. The importance of this disease on *Populus* species of the Great Plains is largely unknown.

Management

Wounding should be prevented. However, many infections result from natural infection courts, and practical means of preventing such infections are not known. Pruning diseased, dying, and dead branches on high-value trees may reduce impacts by preventing the fungus from spreading into the stem. Development of recreation sites in aspen stands is strongly discouraged because aspen is prone to many diseases that can lead to stand deterioration.

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25. Cytospora Canker of Hardwoods

Gerard C. Adams and William R. Jacobi

Cytospora canker, caused by various species of *Cytospora* (sexual forms, *Valsa* and *Leucostoma*), is a common disease of many hardwoods including poplars (*Populus* spp.), willows (*Salix* spp.), maples (*Acer* spp.) elms (*Ulmus* spp.), alders (*Alnus* spp.), ash (*Fraxinus* spp.), peach (*Prunus persica*), mountain-ash (*Sorbus* spp.), quince (*Cydonia* spp.), chokecherry (*Prunus virginiana*), serviceberry (*Amelanchier arborea*), and various woody plants in the Great Plains. *Cytospora* species are generally latent pathogens that form cankers on hosts subjected to environmental stresses.

Hosts and Distribution

Susceptible hosts include those growing on poor sites or injured by drought, frost and winter injury, sunscald, fire, herbicides, mechanical damage, severe pruning, insects, or disease such as fire blight (see chapter 26). In alders, cankering is associated with unusually hot weather. The latent pathogens often colonize senescing branches and twigs without forming evident cankers. For example, *Cytospora* species colonize nearly every senescing branch and twig on maples planted in suitable urban sites. In unsuitable urban sites the same *Cytospora* species will cause damaging, often lethal, perennial cankers on the living trunks of maples. Occasionally, *Cytospora* species can be opportunistic invaders of bark and twigs that have been killed by other causes, and these species are generally not listed in host indexes. Differentiating between a latent pathogenic and a saprophytic *Cytospora* species is generally possible by inoculating hosts during early or late dormancy or when the plant is slightly stressed such as by drought, or by inoculating excised living twigs. Summer inoculations during active host defense response often fail to form cankers.

Cytospora canker is widespread in North America and other regions. Many *Cytospora* species have been inadvertently distributed worldwide with their preferred host as endophytes in propagation cuttings. Resistance to Cytospora canker varies among hosts, but in general there are no commercially available resistant genotypes.

Symptoms and Signs

Cytospora species cause branch dieback and cankers on trees of any age. Cankers on trunks and limbs are often elongate, slightly sunken, discolored areas in the bark (fig. 25-1). Bark often splits along the canker margin because of woundwood formation by the host. The fungus may quickly girdle and kill twigs without forming cankers. Symptoms vary with host species and stage of disease development. Bark above infected cambium may appear sunken and yellow, brown, reddish brown, gray, or black. Diseased inner-bark and cambium turn reddish brown to black, and become watery and odorous as the tissues deteriorate. Gummosis occurs in *Prunus* species. Wood below the cambium is stained brown.

Figure 25-1—Cytospora canker on stem of peach showing shrunken discolored bark (Gerard C. Adams, University of Nebraska, used with permission).

Fruiting bodies develop in dead bark. Asexual fruiting bodies (pycnidia) form first and appear as small pustules on smooth bark (figs. 25-1, 25-2). Pycnidia are less conspicuous on rough bark. Superficial cuts in cankered bark may expose small (generally 0.5 to 1.5 mm diameter) dark pycnidia. Pycnidia generally have white deposits of dried spores encircling

Figure 25-2—Cytospora canker on stem of poplar showing shrunken bark and minute pustules of fruiting bodies (William R. Jacobi, Colorado State University, used with permission).

Figure 25-3—Asexual (pycnidia; small gray structures) and sexual (perithecia; large black structure) fruiting bodies of Cytospora on aspen bark (William R. Jacobi, Colorado State University, used with permission).

Figure 25-4—Orange spore masses and tendrils above erumpent Cytospora pycnidia (William Jacobi, Colorado State University, used with permission).

the openings (ostioles). During moist weather, yellow to reddish-brown spore masses or tendrils may exude from pycnidia (fig. 25-4). Dried tendrils may be the first sign noticed on some cankered tissues. The spores (conidia) are clear (hyaline), sausage-shaped (allantoid), one-celled, and generally 3 to 5 µm × 1.0 to 1.5 µm. Sexual fruiting bodies (perithecia) are sometimes found in bark after pycnidia mature in the year after death of the cambium. On the host, the fungal stroma forms larger pustules (2 to 8 mm in diameter), each containing perithecia (each about 0.3 to 0.5 mm in diameter) in groups of six to 20 with necks converging toward the surface of a disk of whitish to dark brownish tissue. The disk (upper surface of the stroma) breaks through the bark (fig. 25-3). Sometimes the necks extend through the disk as visible beaks. Ascospores from perithecia are hyaline, allantoid, one-celled, and about 7 to 12 µm × 1.5 to 2.5 µm.

Disease Cycle

Sexual and asexual spores of *Cytospora* species are infective, especially when freshly wounded tissue is inoculated. The spores are dispersed after fruiting bodies have absorbed sufficient moisture. Asexual spores (conidia) ooze out of saturated fruiting bodies and are dispersed by rainsplash and blown by wind. Occasionally insects or birds transport conidia. Sexual spores are forcibly discharged from saturated fruiting bodies into air currents, but occasionally they will ooze out as intact asci and then are forcibly discharged. *Cytospora* species infect senescing fruit spurs, shaded twigs, and senescing buds and bud scars. The fungus becomes an established and symptomless endophytic latent pathogen in scattered locations in the living wood, often residing in branch crotches. Trees with the latent pathogen may never exhibit disease or produce fruiting bodies on senescing tissues. The absence of the pathogen fruiting bodies in senescing tissues is due in part to rapid drying of the cambium tissues in climates like that of the Great Plains. Fruiting bodies are seldom evident in shed or broken branches when branch death or fall occurs during dry winter weather. Actively growing nonstressed trees limit expansion of Cytospora canker by their internal defense processes. Therefore, annual cankers occur on some trees, but perennial cankers occur on trees that do not have the genetic capability to limit canker expansion. Commercial peach cultivars have lost the capability of limiting canker expansion and have highly destructive perennial cankers, whereas other *Prunus* species generally retain tolerance and have annual cankers.

Fruiting bodies form in infected bark to complete the life cycle. The fungus overwinters as fruiting bodies and mycelium in bark. Several studies have concluded that despite the production of immense numbers of conidia on cankers, rarely produced ascospores are responsible for most infections. This disease cycle has been suggested for peach perennial canker indirectly through the use of population genetic markers, and was operating in orchards in which the sexual fruiting bodies were not found despite careful searching.

Damage

Cytospora canker can cause dieback or death of planted or native trees. It is a limiting factor in the establishment and growth of some trees. In nurseries, it can attack cuttings used for propagation of almost any hardwood, including poplars and willows. A disease called blackstem of cottonwood is caused primarily by *C. chrysosperma* (sexual stage *Valsa sordida*) and other canker fungi. It can develop during storage or after outplanting (fig. 25-5) and can result in severely reduced nursery production.

Figure 25-5—Development of blackstem symptoms following outplanting of poplar cuttings in a nursery setting (James A. Walla, Northern Tree Specialties, used with pemission).

Management

Healthy trees are less susceptible to infection and damage. Drought stress is one of the most common predisposing factors with this disease. If possible, water and fertilize trees as needed to maintain optimum vigor. Reduce chances of infection by preventing wounds. Do not bring infected plant materials into the area. Plant resistant varieties if they are known or alternative species if they are acceptable. Severely cankered branches or trees should be removed and destroyed. Yearly, prune cankered branches back to a live branch beyond the canker. Small cankers on stems can be removed by excising all affected bark. Shape wounds into an ellipse to promote rapid healing. Prune during dry weather when rain or irrigation will not occur for two weeks (for example, in winter). Disinfect pruning tools with alcohol after each cut.

Fungicides are not normally effective in preventing infection or canker enlargement after infection.

To avoid cankers on nursery cuttings, maintain healthy stock blocks (mother blocks) for selecting your propagation wood. Collect shoots from stock blocks in the fall before very cold weather causes bark moisture to fall. Process and store cuttings quickly: dip cuttings in a fungicide solution before winter storage, store at a constant temperature below 35 °F, and plant cuttings in beds after soil warms to allow rapid plant growth.

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26. Fire Blight of Apple, Pear, and Other Ornamental Rosaceous Shrubs and Trees

Megan M. Kennelly and Mark L. Gleason

Fire blight is caused by the bacterium *Erwinia amylovora*. The pathogen infects flowers, fruits, shoots, branches, and roots, leading to a wide array of symptoms. Flowers, fruits, shoots, branches, and even entire trees can be killed. Fire blight damages susceptible ornamental trees and shrubs and is a major threat to apple and pear production.

Hosts and Distribution

Fire blight occurs on many ornamental trees and shrubs in the family Rosaceae. This disease was first reported in New York in the 1700s and is now found throughout the United States. As of 2012 it had been reported in 47 countries in North America, Europe, the Mediterranean region, and the Pacific Rim. The fire blight pathogen is a quarantine organism in several important apple- and pear-producing countries. In the Great Plains, fire blight is common on apples and crabapples (*Malus* spp.) as well as fruiting and ornamental pears (*Pyrus* spp). Fire blight also occurs on cotoneaster (*Cotoneaster* spp.), hawthorn (*Crataegus* spp.), serviceberry (*Amelanchier* spp.), mountain-ash (*Sorbus* spp.), firethorn (*Pyracantha* spp.), and quince (*Cydonia oblonga*).

Symptoms and Signs

Infected flowers exhibit a water-soaked appearance, droop, then turn black or dark brown (fig. 26-1). Infections commonly begin with the flowers, and the pathogen then spreads into the shoot and leaves. Infected leaves and shoots turn dark brown or black, and shoot tips curl downward into a characteristic "shepherd's crook" shape (fig. 26-2).

Figure 26-1—Flowers infected with fire blight. They droop and turn black or dark brown (Penn State Plant Pathology Collection, used with permission).

Figure 26-2—Fire blight-infected shoot with characteristic "shepherd's crook" symptom (Megan M. Kennelly, Kansas State University, used with permission).

Figure 26-3—Fire blight-infected spur branch with blackened, "scorched" leaves (Megan M. Kennelly, Kansas State University, used with permission).

Shorter side branches (spur branches) can also become infected (fig. 26-3). The blackened dead leaves and flowers remain attached, giving a scorched appearance to the tree. Gummy bacterial ooze may be present on shoots or fruit. Cankers can develop on branches of any size as well as the trunk (figs. 26-4, 26-5, and 26-6). The bark on cankered areas is dark and may be sunken or cracked. Canker margins may be rough or smooth. When disease is active, cankers may have a water-soaked appearance with bacterial ooze present (figs. 26-4 and 26-5). Tissue under the bark exhibits reddish-brown streaking (fig. 26-6). Branches with cankers are girdled and often die.

Figure 26-4—Canker from fire blight exhibiting discoloration, cracking, and gummy bacterial ooze (George Sundin, Michigan State University, used with permission).

Figure 26-5—Canker from fire blight exhibiting bacterial ooze (Patricia McManus, University of Wisconsin – Madison, used with permission).

Figure 26-6—Small branch canker from fire blight with reddish-brown streaking under the bark (Penn State Plant Pathology Collection, used with permission).

Disease Cycle

Fire blight is most severe during moderately warm temperatures with rainfall. The optimum temperature range for infection is 70 to 81 °F, with a minimum of 65 °F and maximum of 90 to 95 °F. *E. amylovora* overwinters at the margins of cankers. During warm, wet weather in spring, the bacteria multiply, often dripping in a sticky ooze at canker margins. Bacteria are spread by rain, wind, and insects (primarily bees and flies) to cause primary infection on open flowers. The pathogen multiplies rapidly on the stigmas, and the bacterial cells can spread down into the nectaries. Once initial flowers are colonized, pollinating insects transfer the pathogen to other blossoms, and the bacteria can spread from flowers to supporting branches. Bacteria also infect the plant through natural openings and wounds in leaves and succulent shoots. These secondary cycles can occur throughout the growing season as long as conducive weather occurs and susceptible tissue is available. Wounding from wind and hail increases the risk of infection. Once inside the plant, bacteria can spread into larger branches, trunks, and roots. Root infection generally causes tree decline and death.

Damage

Fire blight is a serious disease of commercial pome fruit in the United States and many other countries worldwide, reducing yields as well as directly killing entire branches and trees. Fire blight damage in ornamental trees and shrubs reduces ornamental value and functionality, with potential management and replacement costs. Disease severity varies from year to year based on weather conditions.

Management

There are several practices that should be used in combination, if possible, to prevent and manage fire blight. The following practices are focused on ornamental plantings. Detailed information on fire blight prevention in intensively managed commercial orchards is available from other sources and can be obtained from local Cooperative Extension personnel.Resistant varieties are available for some fruiting and flowering species, with varying levels of resistance. Consider using resistant varieties in new plantings. Cultural practices are also important to reduce the risk of fire blight. Succulent growth is more susceptible to fire blight. Therefore, maintain plants with appropriate irrigation, fertilization, and pruning to avoid overstimulation of growth. Several references listed below provide useful guidelines on cultivar selection and maintenance practices.

If fire blight occurs, sanitation is critical to reduce further spread. The optimal time for pruning is during the dormant period, from mid- to late winter, and pruning cuts should be made at least 12 inches below any visible symptoms. If pruning in summer, however, conduct pruning during dry weather and make cuts at least 12 to 18 inches below any visible symptoms. In addition, wait to prune until late summer when new growth has ceased, as new succulent tissue is particularly susceptible. Disinfect pruning tools with a 1:10 bleach dilution between cuts. Pruned-out branches should be burned or discarded, not left at the site. Remove heavily infected trees.

Chemicals (primarily antibiotics and copper compounds), growth regulators, and biological controls are available to prevent fire blight development. If a tree has a history of fire blight, copper products can be applied before budbreak to reduce overwintering inoculum. Avoid applying copper once new growth begins, as phytotoxic reactions can occur, and follow all label instructions. Certain antibiotics (such as streptomycin products) are labeled for application during bloom to prevent blossom blight. Follow all label instructions to reduce the risk of antibiotic resistance, such as limits on number of applications per year and avoiding applications after symptoms have developed. Strains of *E. amylovora* with resistance to antibiotics have been reported in pome fruit orchards in several states. Exact details on the use of chemicals and biological controls are beyond the scope of this publication. Be sure to read, understand, and follow all label instructions for any pesticide including biological controls or growth regulators. Further information on fire blight management for commercial and home production of fruiting apple and pear is available in other references (see list below) and resources. Disease forecasting models to optimize the timing of applications are available for commercial apple and pear producers.

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27. Hypoxylon Canker of Aspen

Michael E. Ostry and Jared M. LeBoldus; revised from Thomas E. Hinds and Mark O. Harrell (Riffle and Peterson 1986)

Quaking aspen (*Populus tremuloides*) is the most widely distributed forest tree in North America, abundant throughout the northern boreal forest of Canada and extending as far south as Mexico. The tree is found in the northern states from Maine to Washington, but its occurrence in the Great Plains is sporadic, with small stands in Nebraska, North Dakota, and South Dakota. Fast-growing poplar hybrids are also commonly planted in the Great Plains. These natural and planted stands of *Populus* spp. are susceptible to infection by Hypoxylon canker.

Hosts and Distribution

Hypoxylon canker, caused by the fungus *Entoleuca mammata* (syn. *Hypoxylon mammatum*), is one of the most damaging diseases of quaking aspen in many areas of North America. Bigtooth aspen (*P. grandidentata*) is occasionally infected, as are hybrids of various *Populus* species. However, the disease is not nearly as damaging to these species as it is to quaking aspen, its most susceptible host.

Hypoxylon canker of aspen is abundant east of the Rocky Mountains and is a major cause of mortality of quaking aspen in some Great Lakes States (Minnesota, Wisconsin, and Michigan) and in the Northeast. Hypoxylon canker is of far less importance on aspen in its western range and has not been found in Alaska. In Canada the disease is widely distributed and in Europe Hypoxylon canker affects *P. tremula*. Although the disease is present in aspen stands on the northern Great Plains, its ecological importance in these scattered stands is unknown.

Symptoms and Signs

Cankers can develop anywhere on branches and stems of aspen of all ages, and symptoms vary depending on the stage of disease development. Dead leaves ("flags") remaining on branches and twigs killed by the disease in the upper crowns are especially visible during the dormant season and often a symptom of the disease. Young cankers are slightly sunken, yellowish-orange areas with irregular margins (fig. 27-1). Later, the bark becomes blistered and ruptures, exposing the powdery gray mat of fungal tissue with bristlelike conidial pillars (hyphal pegs) (fig. 27-2), which bear single-celled, clear (hyaline) spores measuring 1.5 to 4.0 µm × 5.5 to 8.0 µm.

Cankers become rough and black as patches of bark flake off. In the oldest areas of the cankers, fruiting bodies (perithecia) develop, embedded in hard, cushionlike stromata. Stromata are white when young and turn gray to black as they age (fig. 27-3). Within the fruiting bodies dark brown single-celled sexual spores

Figure 27-1—A young Hypoxylon canker with sunken, yellowish-orange bark and irregular margins (Michael E. Ostry, U.S. Forest Service).

Figure 27-2—Hyphal pegs beneath blistered bark of a young Hypoxylon canker (Michael E. Ostry, U.S. Forest Service).

Figure 27-3—A Hypoxylon canker with rough, blackened bark and gray perithecial stromata (Michael E. Ostry, U.S. Forest Service).

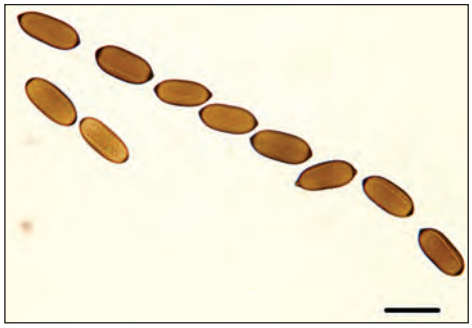

Figure 27-4—Sexual spores (ascospores) of *Entoleuca mammata*. Scale bar is 10 µm (Michael E. Ostry, U.S. Forest Service).

(ascospores) measuring 9.0 to 12.0 µm × 20.0 to 33.0 µm develop (fig. 27-4).

Advancing canker margins are yellowish orange and often slightly sunken. Trees only occasionally are able to develop callus and subsequent woundwood to limit growth of the fungus, and most infections result in the characteristic diffuse-type canker. Cutting into cankers reveals a mottled black and yellowish-cream color characteristic of diseased sapwood, often with white or gray mycelial fans of the fungus extending well beyond the visible canker margin (fig. 27-5). Holes and areas where the bark has been removed at the margins of cankers of all ages are common. This damage is the result of birds searching for and excavating insect larvae of wood-boring insects frequently associated with the diseased wood.

Disease Cycle

Aspen bark contains chemicals toxic to the fungus, so a wound through the bark and into the xylem is needed for infection. Oviposition wounds on branches made by several species of insects are commonly the sites of infection. Birds and insects could also inadvertently introduce bits of diseased tissues or mycelium of the fungus into wounds during their foraging activities. The fungus colonizes and obtains energy from the sapwood to advance under the bark beyond the wound and produces a toxin that inhibits wound callus formation. This toxin kills the bark, resulting in canker development. The fungus can have a latent period of two years or more before symptoms develop.

Approximately one year after initial symptoms appear, the hyphal pegs develop and rupture the bark, exposing hyaline spores that are not infective but rather function as gametes playing a role in the production of perithecia one to two years later. Single-celled, dark brown ascospores are released from perithecia in the cushionlike stromata during wet weather and infect trees through various wounds, completing the life cycle of the fungus.

Damage

Hypoxylon canker girdles and kills twigs, branches, and stems of trees. Disease incidence and impact are the greatest in the first 20 years of a developing aspen stand. Mortality of trees of this age is common because cankers are lower on the stem than on older trees. Older trees can recover from stem cankers higher in their crowns by developing new leaders. Wind breakage of weakened stems at cankers is common owing to the wood decay that develops (fig. 27-6). Aspen in plantations and ornamental landscapes are especially vulnerable to damage by Hypoxylon canker because infection of persistent lower branches on widely spaced trees often results in stem canker development.

Figure 27-5—Mottled black and yellowish-white sapwood and white mycelial fan beneath a Hypoxylon canker (gray perithecial stromata, U.S. Forest Service).

Management

There are no direct control measures proven to minimize the impact of Hypoxylon canker over a large area. Susceptibility to the disease varies by clone. Therefore, favoring any resistant clones identified in native stands or selecting and planting genetically resistant trees have been suggested as ways to reduce damage in natural stands and plantations.

Hypoxylon canker frequently infects trees through wounds on branches made by various insects that are common in aspen along stand edges, in understocked stands, and in plantations with widely spaced trees. As a result, maintaining fully stocked stands and avoiding openings are recommended control strategies. Self-pruning of lower branches that often are infected and result in lethal stem cankers is enhanced by shading in dense stands and has been found to be a genetic trait of canker-resistant aspen in test plantings. Individual trees may be saved by early pruning of diseased branches before the fungus enters the main stem.

Figure 27-6—Stem breakage at a Hypoxylon canker (U.S. Forest Service).

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28. Phomopsis Canker of Poplar

Michael E. Ostry and Jared M. LeBoldus; revised from: Theodore H. Filer Jr. and Edward M. Sharon (Riffle and Peterson 1986)

Cottonwood (*Populus* spp.) occurs along streams and rivers throughout the Great Plains. In most of its range, cottonwood grows on moist, well-drained soils; but in the western semi-arid one-third of its range, it is found only along streams. Most species of *Populus* and their interspecific hybrids are susceptible to Phomopsis canker.

Hosts and Distribution

Phomopsis species cause dieback and cankers on many tree species in North America, as well as in Europe and Japan. *Phomopsis macrospora* commonly affects *Populus* species in nurseries and young plantations. The teleomorph of the fungus has not been reported in North America. Outbreaks of Phomopsis canker have been reported from Minnesota to Mississippi and in Japan.

Figure 28-1—Phomopsis stem canker on hybrid poplar (Michael E. Ostry, U.S. Forest Service).

Symptoms and Signs

Cankers caused by *Phomopsis* species develop on cottonwood twigs, limbs, and boles; they are inconspicuous during early development (fig. 28-1). Small pustules (pycnidia) on cankers appear as small black dots protruding through bark epidermis (fig. 28-2). *Phomopsis* fruits profusely on dead tissues (fig. 28-3). Spores are pushed out of the pore of the pustules under moist conditions and often form sticky white, orange, or reddish spore tendrils (fig. 28-4). Two types of spores are produced: spindle-shaped alpha spores that are 11.0 to 20.5 µm × 2.5 to 5.0 µm and J-shaped beta spores 10 to 20 µm × 1.3 to 2.5 µm (fig. 28-5).

Figure 28-2—Poplar cuttings affected by blackstem disease caused by *Phomopsis macrospora* during improper storage, with small black fruiting bodies (pycnidia) protruding through the bark (Michael E. Ostry, U.S. Forest Service).

As the canker develops and enlarges, the phloem tissue dies, forming a sunken area. When the stem is girdled, the distal portion dies; the resulting dead leaves cling to the branches for several weeks. Similar symptoms are caused by *Fusarium*, *Cytospora*, and other canker-causing fungi.

Disease Cycle

The asexual spores (conidia) are the primary inoculum. Conidia can be dispersed by wind and insects, and rainsplash often spreads the spores to wounds on other parts of the tree and to adjacent trees. The fungus is considered a wound parasite, and requires a wound or natural opening before it can penetrate and colonize tissues. The size of the canker that develops depends on tree vigor. On fast-growing cottonwoods, canker development is usually limited by woundwood development, which restricts fungal growth. On slow-growing

Figure 28-3—Phomopsis canker on planted cutting (Michael E. Ostry, U.S. Forest Service).

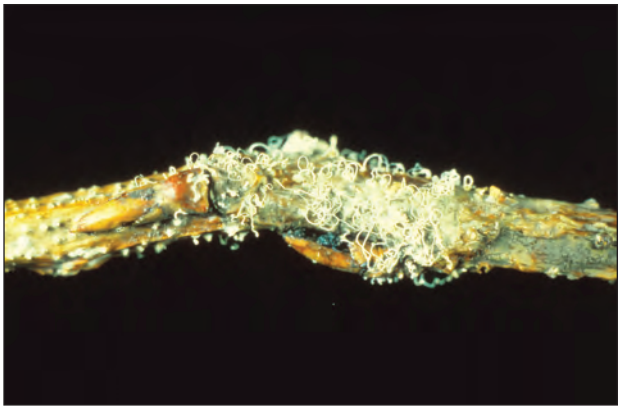

Figure 28-4—Spore tendrils of *Phomopsis macrospora* on diseased cutting (Michael E. Ostry, U.S. Forest Service).

trees (trees growing under moisture stress or in poorly aerated soils), cankers can girdle the stem and cause tree mortality.

Damage

Phomopsis is one of the most important pathogens that cause mortality of water-stressed planted cuttings. In most cases the fungus is on or in the bark of cuttings when they are harvested from the nursery. If cuttings are under water stress the fungus can girdle the cutting within a few weeks of planting.

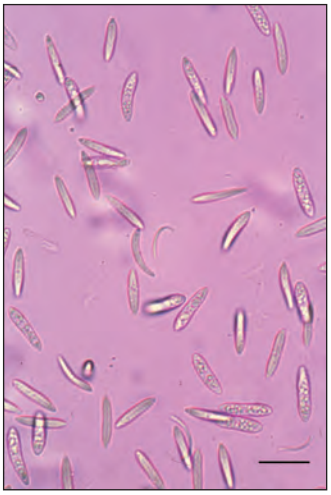

Figure 28-5—Spindle-shaped alpha spores and J-shaped beta spores of *Phomopsis* spp. Scale bar is 20 µm (Michael E. Ostry, U.S. Forest Service).

Management

Good cultural practices can reduce the likelihood of mortality of shade trees. Water at the rate of two inches per week during periods of moisture deficit, and control insect defoliators and leaf diseases to reduce tree stress and promote tree vigor. Fertilize prudently, applying needed nutrients only if a soil test shows a deficit.

To prevent losses when planting poplars, select healthy, canker-free cuttings or seedlings. Prevent cuttings from drying in storage; soak them for 48 hours in water to initiate root primordia before planting. Cultivation is needed during the first year to keep trees growing vigorously. Plant poplar clones that are resistant to other diseases whenever possible.

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29. Septoria Leaf Spot and Canker of Poplar

Jared M. LeBoldus, Michael E. Ostry, and James T. Blodgett; revised from Joseph M. Krupinsky and David W. Johnson; Jerry W. Riffle and David S. Wysong (Riffle and Peterson 1986)

Sphaerulina musiva (syn. *Septoria musiva*) causes a leaf spot disease and branch and stem cankers on native and hybrid poplars (*Populus* spp.). The sexual form of this fungus is *Mycosphaerella populorum*. There are two closely related species, *S. populicola* and *M. populi*, which also cause a leaf spot disease of poplar, as well as *M. jaczewskii* and *S. aceris*, which infect caragana (*Caragana* spp.) and maple (*Acer* spp.), respectively.

Hosts and Distribution

S. musiva is indigenous throughout much of the United States and Canada and occurs across the Great Plains. This fungus causes leaf spots on native cottonwood (*P. deltoides*) and hybrid poplars. It also causes a branch and stem canker disease of hybrid poplars and introduced *Populus* species. In the North Central region of the United States hybrids among cottonwood, balsam poplar (*P. balsamifera*), black poplar (*P. nigra*), Japanese poplar (*P. maximowiczii*), black cottonwood (*P. trichocarpa*), and laurel poplar (*P. laurifolia*) are common hosts.

Symptoms and Signs

Leaf spot symptoms vary according to time of infection, host species, and age of leaves. Four types of leaf spot symptoms have been described: (a) brown, mostly circular leaf spots (1 cm in diameter) that may have a brown or yellow margin, with black, pimplelike fruiting bodies (pycnidia) clustered within; (b) small flecks, commonly with very angular margins; (c) white or silvery spots, mostly 1 to 3 mm in diameter; and (d) irregularly shaped spots that are light tan in the center with brown margins (fig. 29-1). Numerous black pycnidia are commonly clustered in the center of these large spots. Immature pycnidia are light brown and turn black when mature (fig. 29-2).

Cankers are formed on the main stem and branches of the current season's growth. Cankers are often flat-faced or have swollen marginal callus and early woundwood (fig. 29-3). The bark over young cankers is dark brown or black (fig. 29-4) and depressed.

Figure 29-1—Leaf spot symptoms on three balsam poplar leaves (Jared M. LeBoldus, Oregon State University, used with permission).

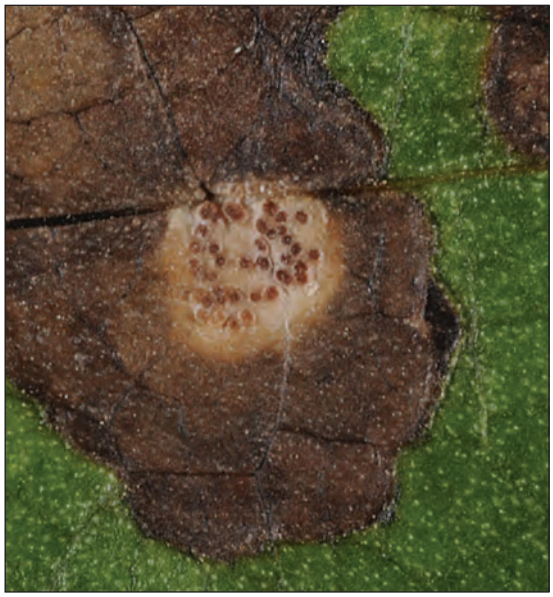

Figure 29-2—Immature asexual fruiting bodies (pycnidia) clustered in the center of a leaf spot (Jared M. LeBoldus, Oregon State University, used with permission).

Figure 29-3—Multiple cankers caused by *Sphaerulina musiva* on a seven-year-old balsam poplar stem with a broken top (Jared M. LeBoldus, Oregon State University, used with permission).

Infected cambium is killed and small black pycnidia may develop in the bark on the ashy-white central area of cankers. Continued development of cankers may result in girdling and death of affected branches and stems during late summer. Affected stems may be infected by other canker fungi, such as *Cytospora chrysosperma*, resulting in additional damage.

Two types of spores develop within fruiting bodies in infected host tissues. Asexual spores (conidia) develop in pycnidia on cankers and leaf spots throughout the growing season, and are exuded in pink or white tendrils during wet weather. Conidia are clear, cylindrical, straight or curved, and one- to four-septate, and measure 20 to 56 µm × 3 to 4 µm

Figure 29-4—Canker (necrotic lesion) caused by *Sphaerulina musiva* on a young poplar shoot (Jared M. LeBoldus, Oregon State University, used with permission).

(fig. 29-5). Sexual spores (ascospores) are produced in perithecia that develop on fallen, infected leaves. The ascospores are clear, one-septate, and 16 to 28 µm × 4.5 to 6.0 µm. Fungi other than *M. populorum* can cause similar leaf spots (listed above). Microscopic examination of spores or a molecular technique (i.e, internal transcribed spacer [ITS] sequences) is necessary to identify these pathogens.

Disease Cycle

The pathogen overwinters on fallen infected leaves and in branch cankers. In spring, ascospores from fallen leaves and conidia from cankers are discharged during wet weather. These spores are dispersed by wind and rainsplash to infect newly emerging leaf and stem tissue. Infections may occur through stipules, petioles, buds, lenticels, or bark

Figure 29-5—Asexual spores (conidia) of *Sphaerulina musiva*. Scale bar measures 20 µm (Jared M. LeBoldus, Oregon State University, used with permission).

wounds. Leaf infection usually precedes stem infection. Leaf spots develop soon after leaf emergence and the fungus spreads to stems and branches, causing cankers. Cankers begin as necrotic lesions on the current season's growth. Pycnidia usually develop shortly after and produce conidia that are dispersed and cause secondary infections. Disease development is enhanced by warm temperatures and long periods of high humidity.

Damage

S. musiva damages poplars of all ages, but damage is most severe in nursery environments, young plantations, and windbreaks. Numerous leaf spot infections can result in premature defoliation of susceptible clones. Multiple cankers can girdle stems, and affected trees are susceptible to wind breakage. Cankers are infection courts for other pathogens such as *Cytospora*, *Phomopsis*, and *Fusarium* and decay fungi, all of which can contribute to the damage.

Management

Damage caused by *S. musiva* is reduced primarily by the use of resistant cultivars. Clones resistant or moderately resistant to leaf spot may also be more resistant to stem canker and should be selected for planting. Use vigorous, disease-free planting stock for maximum early growth and to avoid introducing pathogens into new plantings.

Cultural treatments, such as cultivation or raking in the fall to remove leaf litter harboring fungal inoculum, will minimize primary infection in spring if inoculum from adjacent trees is not a factor. Planting moderately susceptible trees at a wide spacing to provide good air circulation within the canopy will reduce the duration of free moisture on leaves and minimize infection by *S. musiva*.

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30. Stem Decays

James J. Worrall

Stem decay is the decay of wood in aboveground stems of live trees. Fungi that cause stem decays in the Great Plains occur in several orders of phylum Basidiomycota, class Agaricomycetes. Almost all are polypores, with fruiting bodies that are often called conks.

Hosts and Distribution

Stem decays occur in all tree species throughout the Great Plains, but tree species differ greatly in the frequency and severity of decays. Decays also may vary in frequency over the distribution of the host.

Symptoms and Signs

Conks are often the first outward evidence of infection (fig. 30-1), but they only appear years after decay begins and may never appear, even when the tree is extensively decayed. In advanced cases, decay may be detected by sounding: hitting the bark with a mallet or back of a hatchet and listening for a somewhat hollow sound. Often, decay is exposed in broken stems, branch stubs, split forks, or other openings. Otherwise, the only recourse for diagnosis is cutting the tree open or using an increment borer to sample the wood.

Figure 30-1—Conk of *Fomes fomentarius* on common horsechestnut (*Aesculus hippocastanum*) (Andrej Kunca, National Forest Centre—Slovakia, Bugwood.org).

Crown symptoms are not common with stem decays, but can occur. Some stem decay fungi attack the sapwood and can even cause cankers. Crown thinning and dieback of branches often result.

Fungi that attack live trees may continue to decay in and fruit on the tree after it dies. Other fungi grow mostly on dead trees; some common representatives are included here. It is important to recognize that fungi fruiting on dead trees or dead portions of trees may not have decayed wood or killed tissues in the live tree.

Two major types of wood decay are caused by the fungi considered here. In uniform white rot, all structural components (cellulose, hemicelluloses, lignin) are decomposed, and partly decayed wood has about the same proportion of components as undecayed wood. Wood loses strength and becomes soft, but retains its fibrous nature, often becoming stringy. Decayed wood leaves a fibrous, splintery fracture surface when it breaks. Usually white-rotted wood does not darken appreciably and may become bleached white.

In brown rot, cellulose and hemicelluloses are consumed, but lignin remains more or less intact. Cellulose is depolymerized early in the decay process, so that the fibrous nature of the wood is lost and wood breaks readily across the grain as well as with it. In advanced decay, the residual wood shrinks, giving a cubical appearance. Decayed wood leaves relatively nonsplintery, cross-grain, flat surfaces when it breaks (often called a brash failure). Wood with advanced brown rot crumbles readily to a powder when dry. The lignin becomes somewhat darker during the decay process, turning the residual wood brown.

Disease Cycle

Polypores are so named because the conk has on its lower surface thousands of small, vertical tubes, each of which ends at the bottom with a pore (fig. 30-2d, e). Spores (microscopic propagules) are produced in the tubes, fall through the pores, and are then wind dispersed. If a spore chances to land on a suitable point of infection under moist

Figure 30-2—*Fomitiporia texana* on oneseed juniper (*Juniperus monosperma*) in Whitby Canyon, Comanche National Grassland, Colorado. All photos are from the same tree, which had eight conks. (a) Crown with estimated 90 percent dieback. (b) Four of the conks on the stem. (c) Closeup of the lowermost conk in B). (d) Pore surface. (e) Vertical section through a conk showing several layers of tubes and context at top left. (f) Vertical section of the stem at 3 to 6 ft above ground. The sapwood is extensively decayed on both sides, and an inner column of decay is connected to a branch. (g) Cross-section with conk about 6 ft above ground. Decay is much more extensive in the sapwood than in the heartwood, and only a slight amount of sapwood is left alive. (h) Uniform white rot with zone lines. (i) White stringy rot where fine whisps of brown mycelium may be observable (James J. Worrall, U.S. Forest Service. Location and dissection provided by Jeremy Schoonover, U.S. Forest Service).

conditions, it will germinate and the fungus will grow into the host. The infection site differs among fungi and is not well known in many cases. For many fungi, especially saprobic species, the infection sites generally are substantial wounds, such as large branch stubs or mechanical scars. More highly adapted parasites may infect smaller sites such as dead twigs or twig scars. In either case, microscopic, branching filaments (hyphae) grow into the wood and eventually cause decay. Over many years, the wood provides sufficient resources for fruiting. For most fungi, fruiting also requires that two individuals of the same species and compatible mating type encounter each other in the wood and fuse together. Fruiting completes the cycle.

Damage

Decay in stems and branches increases the likelihood of mechanical failure, resulting in loss of portions of the crown or sometimes the entire live crown. Assuming a concentric decay column, likelihood of failure increases substantially when less than one-third of the stem radius remains sound.

Such mechanical failure can be hazardous if people or their property are nearby. In developed sites (around buildings, roads, points of interest, and recreation areas, where people are concentrated), trees should be managed to assess such hazard and mitigate it as needed. Recognizing indicators of decay, understanding how decay develops, and identifying fungi as parasitic versus saprobic can all be important in assessing trees for hazard.

Incidence of decay is generally correlated with tree age. Young trees usually are free of decay. As trees age, branch stubs, wounds, and time increase the opportunity for infection. Once decay is established and begins expanding, time favors the fungus. Older trees are less vigorous, have a greater proportion of nonliving heartwood, and grow more slowly. They are thus less resistant to decay pathogens, have more resources available for fungi that become established in the heartwood, and are less likely to keep up with decay by adding wood faster than it is lost.

Many windbreaks in the Great Plains were established during the Prairie States Forestry Project, 1935-1942. For most species, these have reached an age of declining vigor and increased damage from stem decays.

Buffaloberry (*Shepherdia* spp.) can be severely damaged by *Perenniporia ellisiana* in the northern Great Plains. This fungus may destroy windbreak plantings that are only 20 to 25 years old. Buffaloberry is so susceptible that its extensive use in the northern Great Plains is discouraged.

The most common stem decay of black locust (*Robinia pseudoacacia*) is caused by *Fulvifomes robiniae*, which is host-specific. The disease is common in the central and southern Great Plains, particularly in Oklahoma, where about 25 percent of surveyed black locusts in 40-year-old windbreaks were infected. As with stem decays in general, incidence increases with tree age.

Fomitiporia punctata attacks many hardwoods and is common in the northern Great Plains, particularly in North Dakota. There, the disease occurred in about one-third of sites with trees more than 20 years old. Based on incidence of conks, *F. punctata* was the most important stem decayer of green ash (*Fraxinus pennsylvanica*) in Prairie States Forestry Project windbreaks in North Dakota. It tends to cause a canker-rot, both decaying the inner wood and growing to the surface and killing patches of sapwood and bark.

The most common stem decay of ashes in the Great Plains is caused by *Perenniporia fraxinophila* (fig. 30-3). This fungus occurs on other species as well, including bur oak (*Quercus macrocarpa*) and boxelder (*Acer negundo*). Green ash in woodlands and windbreaks of the central and northern Great Plains is often heavily infected. In native woodlands of Nebraska, more than 10 percent of trees were estimated to be infected. A consistent straight-line relationship was found between diameter and percent infection (fig. 30-4). Conks were less frequent in a large sample of windbreaks 35 to 42 years old in Nebraska, occurring in 90 percent of windbreaks and 5.5 percent of trees. A contemporary study in North Dakota found only 47 percent of windbreaks and 0.3 percent of trees with conks. Of course, these estimates are conservative because additional trees were infected but without conks.

Figure 30-3—Conks of *Perenniporia fraxinophila* (James J. Worrall, U.S. Forest Service).

More recently, in woodlands of east-central Montana, *P. fraxinophila* was associated with widespread canopy dieback of green ash. Stands had on average 38 percent incidence of conks; in some stands as many as 67 percent of trees had conks. Trees with conks had greater crown dieback than trees without conks. Evidence suggested that *P. fraxinophila* is a cause of the widespread crown dieback, but is unlikely to be the sole cause.

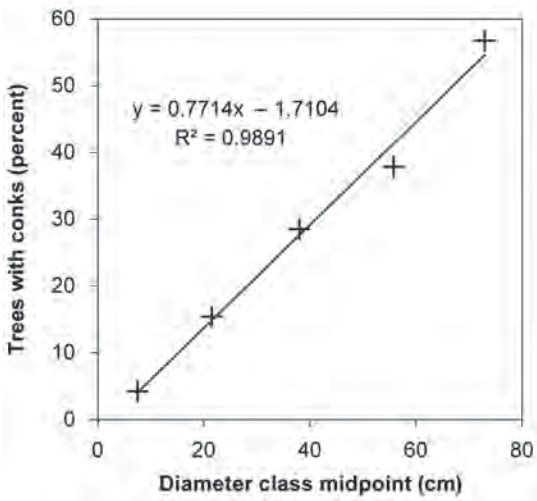

Figure 30-4—Percentage of green ash trees with conks of *Perenniporia fraxinophila* in relation to diameter at breast height (based on data from Riffle and others. [1984]).

Both *F. punctata* in North Dakota and *P. fraxinophila* in Nebraska windbreaks were found fruiting more commonly in the east than in the west. In Nebraska windbreaks, the difference was suggested to have been in part because the higher precipitation in the east promoted more fruiting and infection. In Nebraska woodlands, Riffle and others (1984) did not suggest any pattern in *P. fraxinophila* incidence across the state. However, Lesica and others (2003), reanalyzing Riffle and others' (1984) data with their single datum from eastern Montana, suggested that the pattern was the opposite, and that drier areas had more fruiting.

Eastern redcedar (*Juniperus virginiana*), a major component of field windbreaks in the Great Plains, is frequently infected by *Antrodia juniperina* in the southern and central Great Plains. This pathogen may also attack other juniper species. The disease does not occur everywhere the host does, and its distribution is erratic. The fungus apparently enters through dead branches and wounds, then causes extensive decay in the stem, killing sapwood in the process. Stems become hollow and often break during storms. A windbreak may deteriorate if infected junipers are a major component.

As noted above, decay fungi can sometimes contribute to low vigor, thinning crowns, and branch dieback, to the extent that they kill sapwood from the inside. If trees are not able to produce new sapwood as fast as it is lost, they will suffer from the loss of water conduction and food storage. An example is decay of junipers by *Fomitiporia texana* (fig. 30-2). This aggressive pathogen attacks the sapwood at an early stage, leading to crown thinning and dieback of branches. It eventually kills cambium and can kill the tree. Trees with a single conk may already have thin crowns, several conks are usually associated with dieback, and trees are often near death when the number of conks approaches 10.

Taxonomy and Identification

Some basic terminology is helpful in identifying conks:

Context—the internal tissue of the conk above the tube layers (or for resupinate fungi, between the tube layers and substrate).

Effused—spread out over the substrate indeterminately.

Pileate—having at least a portion of the conk with a sterile upper surface over a fertile lower surface. This portion is called a pileus, although the term may be used more specifically in describing the upper surface.

Reflexed—mostly resupinate, but with a small portion at the upper end that is curved out, creating a small pileus.

Resupinate—lying back on the wood or bark, thus not having a pileus. Resupinate polypores generally show only a pore surface surrounded by a narrow margin.

Sessile—Pileate, without a stem, and not effused; typical conk form

Trama—the tissue between the tubes.

Key to Major Wood Decay Fungi of the Great Plains

The key primarily uses field characters. However, a microscope and knowledge of polypore structure and anatomy will be needed in some groups and are helpful for confirmation of all species. Species characters are based primarily on Gilbertson and Ryvarden (1986), Lowe (1942, 1957), and Overholts (1953). Most species in the key are those included in Riffle and Peterson (1986); table 30-1 cross-references modern and 1986 names.

1. Causing a brown rot; pileus (at least when young), pore surface and context white, cream, yellow, orange, pale brown or pale pink to rosy brown when fresh; hyphal system dimitic (if trimitic, then also with fusiform cystidioles); generative hyphae with clamp connections (if simple-septate, then accompanied by binding hyphae rather than skeletal hyphae); spores cylindrical or allantoid (if ovoid-ellipsoid, then generative hyphae simple-septate), hyaline, thin-walled . . . . . **Fomitopsidaceae**
1. Causing a white rot; not with the above combination of characters **2**
2. Pore surface when fresh white, cream, or yellowish buff, bruising to dark brown or purplish brown; conk perennial; context and tubes dark brown to purplish brown; spores with a truncate apex, ovoid, some broadly ellipsoid, brown, with a two-layered wall . . . . . **Ganodermataceae**
2. Fresh pore surface not substantially changing color when bruised or bruising dull red and spores not as above **3**
3. Conk perennial, usually hard and woody; pileus dark yellowish to reddish brown or black; context dark yellowish or reddish brown, turning permanently black in potassium hydroxide (KOH); pore surface brown; pores circular and usually quite small with thick dissepiments; generative hyphae simple-septate; setae often present in the hymenium . . . . . **Hymenochaetaceae**
3. Conk annual or perennial, tough and leathery to corky rather than woody; pileus, context, and pore surface usually white to pale brown; setae absent. If colors are similar to Hymenochaetaceae (such as in *Fomes fomentarius*), then with a distinct granular core in the context at the point of attachment **4**
4. Conk annual (if perennial then tube layers not separated by layer of context); hyphal system dimitic to trimitic; hyphae with clamp connections; cystidia absent . . . . . **Polyporaceae**
4. Conk perennial, with distinctly stratified tube layers separated by layer of context; pileus (at least when young), pore surface, and context cream to buff; pores circular and small, 5–7 per mm; hyphal system monomitic; hyphae simple-septate; hymenium with encrusted cystidia . . . . . ***Oxyporus populinus*** **(**fig. 30-7)

Fomitopsidaceae

1. When fresh, pileus yellow to orange; pore surface yellow to white; context white; generative hyphae simple-septate and accompanied by binding hyphae; spores ovoid to ellipsoid . . . . . ***Laetiporus sulphureus*** sensu lato (fig. 30-8)
1. Colors different from above; generative hyphae with clamp connections and accompanied by skeletal hyphae; spores cylindrical or allantoid. **2**
2. Conk resupinate to effused-reflexed (rarely sessile); white, cream or pale woody brown throughout (pileus may blacken with age); pores large (≤ 4 per mm), round and regular but elongated and sinuous on sloping surfaces . . . . . ***Antrodia***
2. Conk sessile to effused-reflexed (rarely resupinate); colors pinkish to rosy brown or ivory/tan; pores smaller (≥ 4 per mm), round to angular . . . . . ***Fomitopsis***

Antrodia

a. Typically on live junipers, especially eastern redcedar; conk effused-reflexed to nodulose; pileus pale buff to cork-colored, weathering gray to nearly black; context cream-colored; pore surface white; pores up to 1–3 mm wide and angular to daedaleoid; spores 6.5–9.0 µm x 2.5–3.5 µm . . . . . ***Antrodia juniperina*** (fig. 30-9)

a. Typically on dead hardwoods, especially willows; not with the above combination of characters . . . . . **b**

b. Pileus and pore surface white to cream; pores 2–3 per mm on horizontal surfaces, but tending to become lamellate; spores 10–14 µm x 3.5–5.0 µm . . . . . ***Antrodia albida*** (fig. 30-10)

b. Pileus and pore surface pale woody brown; pores 3–4 per mm on horizontal surfaces; spores 7–10 µm x 2.5–4.0 µm . . . . . ***Antrodia malicola***

Fomitopsis

a. Conk perennial; pink to rosy brown throughout; pores 4–5 per mm; hyphal system dimitic; spores allantoid; uncommon . . . . . ***Fomitopsis cajanderi*** (fig. 30-11)

a. Conk annual or biennial; ivory to tan throughout; pores 5–7 per mm; hyphal system trimitic; spores cylindrical and slightly fusiform . . . . . ***Fomitopsis meliae***

Ganodermataceae

1. On various parts of live and dead hardwoods; conk perennial; crust on pileus usually thick and hard; pore surface white when fresh, quickly bruising dark brown, becoming dull buff when dry . . . . . ***Ganoderma applanatum*** (fig. 30-12***)***

1. On dead hardwoods; conk usually biennial; crust on pileus thin and easily indented; pore surface cream to yellowish when fresh, bruising or drying dull purplish brown . . . . . ***Ganoderma lobatum***

Hymenochaetaceae

1. On live shrubs, mostly currant (*Ribes* spp.); conk soft when fresh but hard when dry; pores almost invisible to naked eye, 6–7 per mm; hyphal system monomitic; spores pale yellow, subcylindrical; setae absent; widespread but uncommon in the Great Plains . . . . . ***Phylloporia ribis***

1. On live or dead trees; conk hard and woody; pores mostly larger than above **2**

2. Typically on dead trees or dead portions of live trees; pileus not becoming rimose (deeply cracked, figs. 30-2c and 30-5); setae abundant; spores hyaline, ellipsoid to subglobose, nondextrinoid **3**

2. On live trees; pileus becoming rimose; microscopic features variable. **4**

3. Conk annual or short-lived, sessile or occasionally effused-reflexed; causing decay of dead or live trees, especially green ash, black locust, maple, and willow; setae arising from subhymenium; encrusted hyphae in hymenium or dissepiments; spores thin-walled, ellipsoid to ovoid . . . . . ***Fuscoporia gilva***

3. Conk perennial; sessile to resupinate; causing decay of primarily dead hardwoods; hyphae not encrusted; setae arising from tramal hyphae; spores somewhat thick-walled and ovoid to subglobose . . . . . ***Phellinopsis conchata*** (fig. 30-13)

4. Spores hyaline **5**

4. Spores yellowish to reddish brown **6**

5. Context usually dark reddish brown; hyphal system distinctly dimitic; spores not dextrinoid . . . . . ***Phellinus***

5. Context brass-colored (yellowish brown); hyphal system weakly dimitic; spores strongly dextrinoid . . . . . ***Fomitiporia***

6. Practically restricted to black locust; context light reddish brown; hyphal system dimitic; spores thin-walled, ellipsoid to ovoid, and flattened on one side . . . . . ***Fulvifomes robiniae*** (fig. 30-14)

6. On oak, walnut, or occasionally other hosts; context dark reddish brown; spores thick-walled, ovoid to subglobose; hyphal system monomitic in context and dimitic in trama **7**

7. On live walnut in the central and southern Great Plains; setae rare to frequent, 20–52 µm x 6–14 µm; spores pale yellowish brown . . . . . ***Inonotus weirianus*** (fig. 30-6)

7. Typically on live oaks; setae frequent to abundant, 16–36 µm x 5–10 µm; spores distinctly reddish brown . . . . . ***Phellinus everhartii*** (fig. 30-5)

Fomitiporia

a. Conk sessile; on live junipers or hardwords; known only in the southern Great Plains . . . . . ***Fomitiporia texana*** (fig. 30-2)

a. Conk resupinate; on live or dead hardwoods in most of the Great Plains . . . . . ***Fomitiporia punctata*** (fig 30-15)

Phellinus

a. On live aspens; conk triangular in profile, upper and lower surfaces at approximately 45° from horizontal . . . . . ***Phellinus tremulae*** (fig. 30-16)

a. On other hosts; pore surface orientation variable . . . . . **b**

b. On live *Prunus*; pores 7–9 per mm . . . . . ***Phellinus pomaceus*** (fig 30-17)

b. On many live hardwoods; pores 5–6 per mm . . . . ***Phellinus igniarius*** sensu lato (fig. 30-19)

Polyporaceae

1. Conk perennial; pores round; spores either large (12–18 µm long) or thick-walled, truncate, and dextrinoid **2**

1. Conk annual; pores round, angular, elongated, daedaleoid, or even lamellate; spores ≤12 µm long (or if longer, pileus densely hirsute to hispid), thin-walled, neither truncate nor dextrinoid **3**

2. Conk sessile, ungulate, woody; pileus gray to brown in sulcate zones, with a hard glabrous crust; pore surface concave, brown; context thin, dark yellowish brown, with a granular, mottled core at point of attachment; older tubes often stuffed with white mycelium; spores thin-walled, neither truncate nor dextrinoid, 12–18 µm x 4–7 µm . . . . . ***Fomes fomentarius*** (fig. 30-1)

2. Conk resupinate to sessile; pileus smooth, cream to black with age; pore surface flat or convex; pore surface and context white to light tan; spores thick-walled, truncate, dextrinoid . . . . . ***Perenniporia***

3. On dead hardwoods; conk resupinate to effused-reflexed; pileus dark brown to black; pore surface white to pale brown . . . . . ***Datronia***

3. On dead or live hardwoods; conk pileate and sessile; pileus and pore surface usually white to medium brown **4**

4. Pileus finely tomentose to glabrous; pileus, context, and pore surface white to creamy buff when fresh . . . . . ***Trametes***

4. Pileus usually hirsute to hispid but may become glabrous; pileus, context and pore surface usually distinctly colored, usually brown to gray **5**

5. Conk sessile or effused-reflexed; pileus up to 6 cm x 8 cm x 0.5 cm, villose to hirsute, brownish to gray in concentric zones; pore surface white becoming gray; pores usually daedaleoid, breaking up into teeth with age; context with a soft, dark upper layer merging into the tomentum and separated by a thin black zone from a corky, pale lower layer; spores 5–7 µm long . . . . . ***Cerrena unicolor*** (fig. 30-20)

5. Conk pileate, may have an effused lower portion; pileus hirsute to hispid or matted-strigose to glabrous, zonate or not; pores round or elongate to daedaleoid; context without black zone; spores ≥8 mm long. **6**

6. Conk usually sessile, may be effused-reflexed, up to 3 cm thick; pileus matted-strigose to glabrous, buff to light brown; pores variable but usually daedaleoid; spores allantoid . . . . . ***Daedaleopsis confragosa*** (fig. 30-18)

6. Conk usually effused-reflexed, usually ~1 cm thick or less; pileus hirsute to hispid, creamy-buff or brown to gray; pores round to angular, less often daedaleoid; spores cylindrical . . . . . ***Coriolopsis***

Coriolopsis

a. Conk sessile, effused-reflexed, or rarely resupinate, up to 6 cm x 12 cm x 2 cm; pileus hirsute to coarsely hispid, cream to clay-buff; pore surface white to clay-buff; pores angular to daedaleoid, averaging 3 per mm but some 1 mm in diameter; context white to cream buff, duplex, 2–5 mm thick; spores cylindric, 9–12 µm x 2.5–4.0 µm . . . . . ***Coriolopsis trogii*** (fig. 30-21)

a. Pileus, pore surface, and especially context darker in color; pores round to angular . . . . . **b**

b. Conk sessile to resupinate; pileus up to 3 cm x 6 cm x 0.3 cm but larger if fused, brown to gray, hirsute to tomentose; pore surface cinnamon-brown to gray, becoming darker; pores 2–4 per mm; context duplex, dense lower part up to 4 mm thick, light brown or golden-cinnamon and shiny where cut, temporarily black in KOH; spores cylindric, 8–11 µm x 2.5–4.0 µm . . . . . ***Coriolopsis floccosa***

b. Conk sessile, but sometimes several pilei connected with common effused part; pileus cream to clay-buff, up to 6 cm x 12 cm x 2 cm, densely hirsute to hispid, yellow-brown weathering to gray, zonate or azonate; pore surface brown to gray; pores angular, 1–3 per mm, becoming elongated and split; context 2–10 mm thick, rusty to umber brown, temporarily blackish in KOH; spores cylindric, 10–16 µm x 4–5 µm . . . . . ***Coriolopsis gallica***

Datronia

a. Conk corky to hard, pileate to effused-reflexed; pileus glabrous; pores round to slightly angular, 4–5 per mm . . . . . ***Datronia scutellata***

a. Conk tough but flexible, effused-reflexed or resupinate; pileus velutinate to tomentose; pores angular to daedaleoid, 1–2 per mm . . . . . ***Datronia mollis***

Perenniporia

a. On live buffaloberry; pores 2–3 per mm; spores 6.5–8.5 µm long . . . . . ***Perenniporia ellisiana*** (fig. 30-22***)***

a. On other hosts, pores ≥3 per mm; spores ≥9 µm long . . . . . **b**

b. Typically on live ashes; conk large, up to 25 cm x 40 cm x 10 cm; pores 3–5 per mm; spores 9–11 µm long . . . . . ***Perenniporia fraxinophila*** (fig. 30-3)

b. On various dead hardwoods; conks small, up to 3 cm x 3 cm x 2 cm; pores 5–7 per mm; spores 13–16 µm long . . . . . ***Perenniporia ohiensis***

Trametes

a. On live or recently dead willows and sometimes other hardwoods; pileus with a pleasant anise odor when fresh, up to 10 cm x 16 cm x 4 cm, white, gray, or pale yellow-brown; context white; pore surface white to pale buff; pores round to angular, 2–3 per mm; spores cylindrical, 7–12 µm x 3.0–4.5 µm . . . . . ***Trametes suaveolens (***fig 30-23)

a. On dead (occasionally live) willows and other hardwoods; pileus with no distinctive odor, up to 20 cm x 35 cm x 3 cm, mostly white but often drying gray or yellow; context cream to pale brown; pore surface white but often drying yellow or pale brown; pores highly variable: round, angular (1–2 per mm), daedaleoid, to more or less lamellate; spores cylindrical, 5–7 µm x 2–3 µm . . ***Trametes elegans***

Table 30-1—Major wood-decay fungi of the Great Plains[a]

Fungus	Name in first edition	Hosts	Tree status[b]	Abundance[c]
Antrodia albida	*Trametes sepium*	Willows (*Salix* spp.)	D	C
Antrodia juniperina	Same	Junipers (*Juniperus* spp.), esp. eastern redcedar (*J. virginiana*)	L	C
Antrodia malicola	*Trametes malicola*	Willows	D	C
Cerrena unicolor	*Daedalea unicolor*	Willows	D	C
Coriolopsis floccosa	*Trametes rigida*	Willows	D	C
Coriolopsis gallica	*Trametes hispida*	Willows	D	C
Coriolopsis trogii	*Trametes trogii*	Willows	D	C
Daedaleopsis confragosa	*Daedalea confragosa*	Willows	D	C
Datronia mollis	*Trametes mollis*	Willows	D	C
Datronia scutellata	Same	Hardwoods	D	U
Fomes fomentarius	Same	Birch (*Betula* spp.) most common	D	C
Fomitiporia punctata	*Phellinus punctatus*	Hardwoods, esp. green ash (*Fraxinus pennsylvanica*), Siberian peashrub (*Caragana arborescens*), American plum (*Prunus americana*), willows, black locust (*Robinia pseudoacacia*), common buckthorn (*Rhamnus cathartica*)	L+D	C
Fomitiporia texana	Not in first edition	Junipers, esp. eastern redcedar	L	C
Fomitopsis cajanderi	Same	Conifers and hardwoods	D (L)	U
Fomitopsis meliae	*Fomitopsis melliae* [*sic*.]	Many hardwoods, including poplar (*Populus* spp.), maple (*Acer* spp.), honeylocust (*Gleditsia* spp.), ash (*Fraxinus* spp.), occasionally conifers. Impt. in live peach (*Prunus persica*).	L+D	C
Fulvifomes robiniae	*Phellinus robineae* [*sic*.]	Black locust in central and southern Great Plains	L	C
Fuscoporia gilva	*Phellinus gilvus*	Green ash, black locust, maple, willow	D (L)	C
Ganoderma applanatum	Same	Poplar, maple, honeylocust, ash	L+D	C
Ganoderma lobatum	Same	Hardwoods	D	U
Inonotus weirianus	*Phellinus weirianus*	Black walnut (*Juglans nigra*)	L	C
Laetiporus sulphureus sensu lato[d]	Not in first edition	Hardwoods	L+D	C
Oxyporus populinus	Same	Hardwoods, esp. maples	L	U
Perenniporia ellisiana	Same	Buffaloberry (*Shepherdia* spp.)	L	C
Perenniporia fraxinophila	Same	Ash almost exclusively	L	C
Perenniporia ohiensis	*Perenniporia ohiense* [*sic*.]	Oaks (*Quercus* spp.) and other hardwoods	D (L)	U
Phellinopsis conchata	*Phellinus conchatus*	Hardwoods	D	C
Phellinus everhartii[e]	Same	Oaks	L	C
Phellinus igniarius sensu lato[f]	Same	Birch, also ash, black walnut, poplars, buckthorn, willow	L	C
Phellinus pomaceus[g]	*Phellinus tuberculosus*	Plum, peach, nectarine, cherry (*Prunus* spp.)	L	C
Phellinus tremulae	Same	Aspen (*Populus* spp.)	L	C
Phylloporia ribis	*Phellinus ribis*	Currant and elderberry (*Ribes* spp.)	L	U
Trametes elegans	*Daedalea ambigua*	On many, common on willows	D mostly	C
Trametes suaveolens	Same	Willows, sometimes other hardwoods	L	C

[a] From (Riffle and Conway 1986; Riffle and Walla 1986; Sharon and Riffle 1986; Walla and Stack 1986).
[b] D = on dead trees; L = on live trees; (L) = less commonly on live trees. Note that conks on live trees may still be present after the tree dies.
[c] C = common; U = uncommon.
[d] *Laetiporus sulphureus* in the broad sense. Several species have been delineated within the *L. sulphureus* group, and further collections are needed to determine which occur in the Great Plains. *Laetiporus cincinnatus* has been reported in Kansas.
[e] The generic placement of *Phellinus everhartii* (fig. 30-5) is uncertain. It is very similar to other *Phellinus* species now placed in the *Inonotus linteus* group, such as *I. weirianus* (fig. 30-6) and *I. dependens*. More molecular data are needed, but available data suggest that it does not belong in *Inonotus*. Although it does not share the characters of *Phellinus sensu stricto*, it is retained in that genus here.
[f] *Phellinus igniarius* in the broad sense. Many similar species have been segregated from *P. igniarius* over the years, but their distribution has not been adequately characterized. *Phellinus alni* is a likely species in the Great Plains because it has a wide host range, but it has not been specifically identified there.
[g] Unpublished multi-locus phylogenetic analysis shows that our *Phellinus* sp. on *Prunus*, originally named after the European *Phellinus pomaceus*, which was later synonymized with *P. tuberculosus*, is in fact distinct from the common *P. tuberculosus* in Europe. Furthermore, the *basionym* of *P. tuberculosus*, *Boletus tuberculosus* Baumgarten 1790, is illegitimate (as a later homonym of *B. tuberculosus* Bull 1783). Here the name *P. pomaceus* is used, although it may eventually prove to be a distinct species.

Figure 30-5— *Phellinus everhartii* on bur oak (Derek Lowstuter, North Dakota Forest Service, used with permission); (inset) close-up of a *P. everhartii* conk (James J. Worrall, U.S. Forest Service).

Figure 30-6—Conks of *Inonotus weirianus* on black walnut (*Juglans nigra*) (Whitney Cranshaw, Colorado State University, Bugwood.org., used with permission).

Figure 30-7—*Oxyporus populinus* (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-8—The sulfur shelf, *Laetiporus sulphureus* (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 30-9—*Antrodia juniperinum* on juniper in Nebraska (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

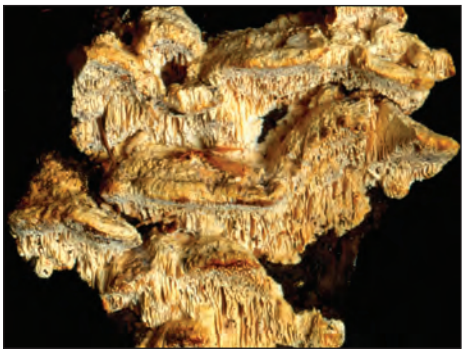

Figure 30-10—*Antrodia albida* (James J. Worrall, U.S. Forest Service).

Figure 30-11—*Fomitopsis cajanderi* (James J. Worrall, U.S. Forest Service).

Figure 30-12—The artist's conk, *Ganoderma applanatum* (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 30-13—*Phellinus conchata* on green ash; (inset) cross section of decay with older conk above (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-14—*Fulvifomes robinae;* (inset) pore surface (James J. Worrall, U.S. Forest Service).

Figure 30-15—*Fomitiporia punctata* on green ash (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-16—*Phellinus tremulae*; (inset) pore surface (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Figure 30-17—*Phellinus pomaceus* (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-18—*Daedaleopsis confragosa* (James J. Worrall, U.S. Forest Service)

Figure 30-19—*Phellinus igniarius* (James J. Worrall, U.S. Forest Service).

Figure 30-20—*Cerrena unicolor* (Joseph O'Brien, U.S. Forest Service, Bugwood.org); (inset) pore surface (James J. Worrall, U.S. Forest Service).

Figure 30-21—*Coriolopsis trogii* on willow (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-22—*Perreniporia ellisiana* on buffaloberry (James A. Walla, Northern Tree Specialties, used with permission).

Figure 30-23—*Trametes* spp. on plum (Aaron D. Bergdahl, North Dakota Forest Service, used with permission); (upper right) pore surface of *Trametes suaveolens* (James J. Worrall, U.S. Forest Service); (lower right) decay of heartwood by *Trametes* spp.(Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

Management

Once decay is established in a tree, little or nothing can be done to stop it. Although there is no cure, prevention can be effective. Avoid unnecessary wounding, such as from livestock, machinery, tree removal, and fire. Where pruning is needed, do it while trees are young and vigorous and branches are small, so that pruning wounds will close quickly. Use proper pruning techniques. Maintaining tree vigor with water during dry periods and fertilizer when soil testing shows a nutrient deficiency may speed wound closure and increase tree defenses. In general, do not apply wound dressing chemicals. There is no evidence that they provide any benefit, and several have been shown to be deleterious.

Where thinning is appropriate, remove trees with large wounds, evidence of decay, and low vigor. If branches break, prune back to the nearest live lateral. When large wounds are fresh, remove any peeled bark fragments. Pruning distal to the wound may help promote wound closure.

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31. Thousand Cankers Disease (TCD) of Walnut

Ned Tisserat and Whitney Cranshaw

Black walnut (*Juglans nigra*) is native to the eastern Great Plains and has been widely planted throughout the region as a nut and ornamental tree. Widespread mortality of black walnut caused by thousand cankers disease was first noted in Colorado in 2001. The disease now threatens black walnuts throughout the Great Plains.

Hosts and Distribution

The walnut twig beetle (WTB; *Pityophthorus juglandis*) and its fungal associate *Geosmithia morbida* cause minor to modest damage to their native host, the Arizona walnut (*J. major*). In contrast, black walnut is highly susceptible to this insect and fungus (known collectively as TCD), and thousands of planted black walnut in cities and rural locations throughout the western United States have been killed. Other native walnuts including butternut (*J. cinerea*), southern and northern California black walnuts (*J. californica* and *J. hindsii*, respectively), little walnut (*J. microcarpa*), and the introduced Japanese and Persian walnuts (*J. ailantifolia* and *J. regia*, respectively) are intermediate in susceptibility to the pathogen.

Symptoms and Signs

Walnut trees affected by TCD initially exhibit yellowing of foliage and thinning of the upper crown, followed by twig and branch dieback. Progressively larger branches die until eventually the main stem of the tree is colonized by the beetle, and the tree is killed (fig. 31-1). The bark remains tightly attached to the tree, and unlike diseases caused by many other canker pathogens, no open-faced cankers are formed. However, external symptoms such as bark cracking or black sap exudation and staining of the bark surface may belie an underlying infection of the phloem. Sprouts may form on the trunks of dying trees, but these also succumb.

Figure 31-1—Branch dieback on black walnut resulting from thousand cankers disease (Ned Tisserat, Colorado State University, used with permission).

The WTB is a minute (1.5 to 2.2 mm in length) reddish-brown bark beetle, about three times as long as it is wide (fig. 31-2). The small entrance/emergence holes of the WTB may be visible on the bark of younger, smooth-barked branches but are not easily found on larger branches or the trunk, where they typically occur in bark fissures. The WTB rarely attacks branches smaller than 1 inch in diameter, so examining small diameter twigs is not helpful in diagnosis. The WTB galleries are only visible in the phloem after the outer bark is removed. Galleries may occur at various depths in the bark tissue, including the bark–wood interface during the final stages of the disease.

The entrance holes and galleries of WTB are surrounded by small, diffuse dark brown to black cankers caused by *G. morbida*. These cankers eventually coalesce, resulting in necrosis of large areas of the bark (fig. 31-3). The light-colored (tan to yellow) fungus often sporulates in beetle galleries, giving them a dusty appearance. The barrel-shaped asexual spores (conidia) measure 2 µm × 5 µm and are formed on verticillate conidiophores (fig. 31-4).

Figure 31-2—The walnut twig beetle, *Pityophthorus juglandis*, can be identified by concentric rows of cuticular bumps or ridges (asperities) on the prothorax (Steven Valley, Oregon Department of Agriculture, Bugwood.org, used with permission).

Figure 31-3—Cankers caused by *Geosmithia morbida*. The outer bark has been removed to expose the cankers, which form around walnut twig beetle galleries (Ned Tisserat, Colorado State University, used with permission).

Figure 31-4—Barrel-shaped asexual spores (conidia) and verticillate conidiophores of *Geosmithia morbida* (Ned Tisserat, Colorado State University, used with permission).

Disease Cycle

The life cycle of the WTB is not fully understood. Generations appear to overlap, and adults may emerge from galleries throughout the year during warm weather. However, in the Rocky Mountain region, peak adult emergence appears to be in late July and early August. The WTB carries *G. morbida* on its body. The relationship of the fungus to the WTB is still unclear, but the fungus appears to begin to colonize the phloem coincident with the construction of the gallery beneath the bark. The infection subsequently causes cankers around the gallery. The total length of time for TCD to kill a mature walnut tree (from initial beetle infestation to tree death) is not known, but trees generally die within four years after symptoms develop.

Management

Currently there are no effective management strategies for TCD. Trees exhibiting more than 50-percent crown dieback should be removed. Wood with bark attached can harbor the WTB for up to 20 months and should not be moved to uninfested areas without first disinfesting by heat or another method. The effectiveness of insecticide drenches or injections in preventing beetle colonization is being studied. Surviving black walnuts in TCD-affected areas have been observed, but it is not yet known whether these trees are resistant.

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32. Wetwood (Slime Flux) and Alcohol Flux in Hardwoods

William R. Jacobi and John Ball

Bacterial wetwood is a common disease affecting the internal xylem of many softwood and hardwood trees. In some species wetwood can affect the cambium and thus is lethal. In other species little damage occurs. Alcohol flux is a yeast-type infection of surface wounds on trees that produces a foaming and sweet alcoholic smell.

Hosts and Distribution

- **Wetwood** affects trees throughout the United States. It is common in elm (*Ulmus* spp.), mulberry (*Morus* spp.), willow (*Salix* spp.), and aspen and cottonwood (*Populus* spp.) in the Great Plains.
- **Alcohol flux** affects primarily willow, although oaks (*Quercus* spp.) and elms can also be hosts in the Great Plains

Symptoms and Signs

Wetwood can be found in the trunk, branches, and roots (figs. 32-1 through 32-4) and has a dark color, foul odor, high moisture content, elevated pH, decreased electrical resistance, abnormally high gas pressure, and increased levels of mobile cations. Elms, aspen, and cottonwoods are often infected early in their life. Dark streaks or bands may appear in the annual rings. Discoloration is most extensive in heartwood or older sapwood, but can occur in current sapwood. Foliage of affected limbs may become prematurely yellow, scorched, and wilted, although many infected trees do not exhibit these symptoms. Wilting may cause dieback of scattered branches, and the entire crown may decline over several years. Premature defoliation may occur, but causes little growth loss. "Bleeding" or slime-fluxing from trunk wounds, cracks, or other injuries is the most conspicuous symptom. Airborne bacteria, yeasts, and other fungi contaminate the sap on the outside of the tree, resulting in a frothy, slimy, foul-smelling liquid. Upon drying, it leaves a light gray to white crust (fig. 32-1). Insects may visit the slime but are not involved with the disease (fig. 32-5).

Figure 32-1—Discoloration of bark of American elm (*U. americana*) affected with wetwood (William R.Jacobi, Colorado State University, used with permission).

Abundant gas is produced in wetwood-affected tissues by the fermenting action of bacteria on carbohydrates and other materials in the xylem. The gas is composed primarily of methane, nitrogen, carbon dioxide, and oxygen. When the gas is confined in the trunk, abnormally high pressures of up to 60 psi develop; 5 to 10 psi is normal for healthy tissues. The accumulation of liquid under pressure results in a water-soaked condition that gives rise to the name "wetwood." The pressure forces the accumulated liquid and gas out of the trunk through cracks in crotches, through pruning and other wounds, and through other natural openings. This liquid can cause localized cambial mortality and prevent callus and subsequent woundwood formation. Foliage wilts when sufficient quantities of the toxic liquid accumulated

Figure 32-2—Wetwood discoloration at branch crotch on American elm (William R. Jacobi, Colorado State University, used with permission).

Figure 32-3—Old pruning cut with wetwood slime inhibiting woundwood growth (William R. Jacobi, Colorado State University, used with permission).

Figure 32-4—Base of tree where wetwood slime has killed the grass (William R. Jacobi, Colorado State University, used with permission).

in the trunk wood are carried into the branches. Leaves first curl upward along their margins, then the petioles become flaccid, and finally the leaves droop and wilt.

Alcohol flux, also known as frothy or white flux, is not related to wetwood, but the symptoms are often confused. Alcohol flux is almost clear liquid although it is more often noticed when it has the appearance of foamy skim milk. The liquid occurs in cracks and fissures, usually near the base of the trunk flare, but can also be found seeping out higher on the trunk (figs. 32-6 and 32-7). The foam usually appears during the summer and may remain for only a few days or so. Alcohol flux has a sweet beerlike odor, whereas wetwood is more rancid. The sweet smell of alcohol flux is also attractive to insects, which may be found swarming around the foamy liquid. Alcohol flux develops just beneath the bark in the outermost sapwood, whereas wetwood is usually in the center of the tree.

Disease Cycle

The bacteria associated with wetwood disease are common soil and water inhabitants and probably enter the tree through root wounds early in a tree's life. Most of the bacteria lie dormant and do not cause wetwood until the tree is much older. Wetwood is primarily associated with various facultative or obligate anaerobic bacteria and archaea; both types of organisms probably play a role in the production of the complex symptoms associated with wetwood. A related form of wetwood called cambial wetwood is more damaging to trees. In this situation the bacterial infestation is found in the outer xylem-cambial area and the fermentation causes the death of the cambium, which can cause small to large cankers that can girdle branches or stems (fig. 32-8).

The yeast microbes responsible for alcohol flux have not been extensively studied, perhaps because the disease causes minimal harm to the infected tree. Many yeast species are associated with the disease, and these organisms produce gas and ethanol during fermentation.

Figure 32-5—Wetwood or alcohol flux slime with visiting wasps (Hymenoptera: Apocrita) and bumble flower beetles (*Euphoria inda*; Whitney Cranshaw, Colorado State University, Bugwood.org, used with permission).

Figure 32-6—White ash (*Fraxinus americana*) with milky alcohol flux (James A. Walla, Northern Tree Specialties, used with permission).

Damage

Wetwood is a chronic disease that may contribute to general decline, especially of old trees and trees of low vigor. The disease affects drought-stressed trees most severely. It causes an unsightly and often foul-smelling bleeding from tree wounds. The disease retards or prevents woundwood formation over wounds, and therefore lengthens susceptibility to decay fungi. Dripping wetwood flux may kill turf beneath infected trees. During drought conditions the bacteria can move to the cambium and cause cankers and girdling of the host. These effects are common in recently transplanted poplars (*Populus* spp.) and related trees.

Wetwood is responsible for substantial losses of wood in the forest products industry. Loss occurs through shake and frost cracks in living trees, checking and collapse in lumber and veneer during drying, and increased drying time in the kiln. The stained wood is also a serious defect in lumber and veneer graded on appearance. Strength properties of wetwood-infected tissues do not differ significantly from those of healthy tissues. In fact, wetwood tissue in living trees is rarely decayed and appears to be resistant to wood-inhabiting fungi.

Alcohol flux can be associated with the death of cambial tissue, which often turns brown before dying. Infected trees may also exhibit dieback, but this effect may be a result of the predisposing stress rather than alcohol flux.

Management

Wetwood cannot be treated by chemicals. The best action is to keep trees healthy and not drought stressed. Drought stress intensifies the severity of disease on many hosts. Installation of drain tubes to lower stem pressures and remove excess liquid is not recommended because the drilling will allow the wetwood pathogens to move outward into healthy sapwood. The wounds also may allow entrance of decay fungi. Remove severely affected trees and limbs in spring. Take care to sterilize tools after each cut to prevent spread from diseased to healthy trees.

Figure 32-7—Milky froth of alcohol flux (James A. Walla, Northern Tree Specialties, used with permission).

Figure 32-8—Wetwood-induced cambial canker on a nursery grown cottonwood (William R. Jacobi, Colorado State University, used with permission).

Alcohol flux infections appear to be stress related and maintaining or improving tree health is the only treatment. Drought and heat stress are the two most common agents associated with alcohol flux, so watering a tree during a summer dry period may be the best treatment. The symptoms often disappear following rain. The disease is also more common on trees that have suffered severe mechanical injury from being struck by grass whips and lawnmowers.

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33. Witches'-Broom of Hackberry

Mark O. Harrell and Laurie J. Stepanek; revised from Mark O. Harrell and Frederick J. Crowe (Riffle and Peterson 1986)

Hackberry (*Celtis occidentalis*) is commonly planted in cities and parks in much of the central Great Plains. Although principally a bottomland tree, it is able to withstand a wide range of moisture and temperature conditions and does well in an urban environment. A common and often disfiguring branch disease of hackberry is witches'-broom.

Hosts and Distribution

Witches'-broom is widespread throughout the range of hackberry in the Great Plains. It is very common in eastern Kansas and Nebraska. The incidence and severity of witches'-brooms on individual hackberry trees can vary greatly. Sugarberry (*C. laevigata*) is rarely affected.

Symptoms and Signs

The most apparent symptom of witches'-broom is a dense clustering or "brooming" of twigs (figs. 33-1 and 33-2). Because brooms do not occur on branches less than one year old, they are not found at branch tips. They occasionally appear to be terminal, however, when a branch leader fails to develop fully or when it dies and breaks off.

Figure 33-1—Witches'-broom of hackberry (Whitney Cranshaw, Colorado State University, Bugwood.org, used with permission).

The first symptom in the development of witches'-broom appears in the buds on branch portions at least one year old. Affected buds are usually smaller than normal, and often many small buds are produced instead of a single larger one. The shoots produced from the affected buds grow vigorously and produce buds that are usually more open and more numerous than normal.

During the next year the development of the broom varies depending upon the vigor of the affected branch. If the branch is vigorous and grows rapidly, its buds develop into branchlets similar to the original, and over a number of years a loose, open cluster of diseased twigs is formed. If the diseased branch is weak, only the buds at the base grow, resulting in a large number of abortive twigs (fig. 33-3). Over several years this type of development produces a compact broom with a firm woody center often 1 inch or more in diameter (fig. 33-1).

Figure 33-2—Hackberry with many witches'-brooms (Laurie J. Stepanek, Nebraska Forest Service, used with permission).

Figure 33-3—Witches'-broom with new and abortive twigs (William M. Ciesla, Forest Health Management International, Bugwood.org).

Disease Cycle

The cause of witches'-broom of hackberry is not well understood. An eriophyid mite *Aceria celtis* (syn. *Eriophyes celtis)* rarely found on symptomless trees is almost always associated with the brooming. The powdery mildew fungus *Podosphaera phytoptophila* is also sometimes found. The original report of the mite-mildew association with brooms has been commonly misinterpreted as a cause-and-effect requirement for witches'-brooms. However, no experimental evidence is available to confirm or disprove the role of these organisms. Whether the growth abnormality results directly from mite activity, whether the powdery mildew is involved but is too obscure to be routinely identified, or whether a virus-like entity is responsible remains uncertain.

Damage

Witches'-brooms on hackberry are more unsightly than harmful to the tree. Brooms can cause branches to break more easily, and extensive brooming can reduce the vigor of the tree, but trees are seldom seriously damaged.

Management

No effective control measures are known for this disease. Pruning back affected twigs to sound wood can improve the tree's appearance.

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34. Dutch Elm Disease

Kelsey L. Dunnell and Aaron D. Bergdahl

Dutch elm disease (DED) is a systemic vascular wilt disease of elm (*Ulmus* spp.) caused by two closely related fungi, *Ophiostoma ulmi* (Buisman) Nannf. and *O. novo-ulmi* Brasier, which are spread by bark beetles and through root grafts. The fungi have been spreading across North America since the 1920s and have killed most of the native elm trees in the United States over the past century.

Hosts and Distribution

Ophiostoma ulmi and *O. novo-ulmi* are exclusively pathogens of trees in the elm family (Ulmaceae). Elm species native to the Great Plains include: American elm (*U. americana*), rock elm (*U. thomasii*), slippery (red) elm (*U. rubra*), winged elm or wahoo (*U. alata*), and cedar elm (*U. crassifolia*). The first four species are very susceptible to the DED fungi and are quickly killed; however, the cedar elm, a native to the southern Great Plains, has some resistant individuals. Several exotic elms have been planted in the Great Plains. The most common is Siberian elm (*U. pumila*), which has been widely used in shelterbelts, on farmsteads, and in urban settings, and is considered moderately tolerant to DED. Among other Asiatic elms, *U. davidiana* var. *japonica*, *U. laciniata*, and *U. villosa* contain a high proportion of resistant individuals; the Chinese or evergreen elm (*U. parvifolia*) is also considered highly resistant to DED. A range of disease resistance occurs within many elm species. Tree breeding programs in the United States, Canada, and Europe continue to work on developing selections and elm hybrids that are resistant to DED.

Symptoms and Signs

New infections established by bark beetles (fig. 34-1) on DED-susceptible elm species, such as American elm, usually involve symptom development on a single branch. The first symptom of DED is yellowing of foliage, followed by wilting, browning, and early defoliation. These symptoms progress downward from the point of infection to all lower branches and eventually the main trunk and root system; the entire tree finally wilts and dies (fig. 34-2). This progression of symptoms may develop in a single season in highly susceptible species, but often takes longer (two or more years). However, observations indicate that when trees are infected via root grafts, wilting often begins in spring in lower branches and progresses upward, leading to mortality during that year; some trees may persist to the next year.

The fungi are strictly vascular parasites, invading the xylem vessels of the host tree and causing disruption of water movement in the tree, which leads to brown streaking in the wood just under the bark (fig. 34-3). Other wilt-causing pathogens may also cause streaking of the wood, so this symptom is not entirely diagnostic for DED.

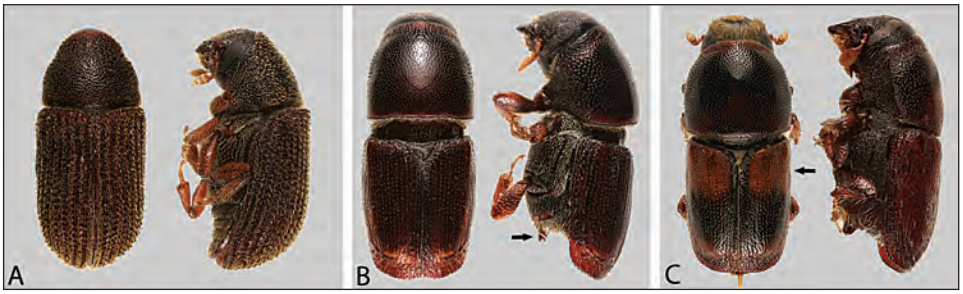

Figure 34-1—Three species of bark beetle found in the Great Plains that spread DED: (A) Native elm bark beetle, *Hylurgopinus rufipes*; (B) Smaller European elm bark beetle, *Scolytus multistriatus* (black arrow indicates prominent abdominal spine); (C) Banded elm bark beetle, *Scolytus schevyrewi*, (black arrow indicates light banding pattern on elytra) (Guy Hanley, Minot State University, used with permission).

Figure 34-2—Infected elm trees showing dying back of branches with yellowing, wilting, and browning leaves (Aaron D. Bergdahl, North Dakota State University, used with permission).

Figure 34-3—A healthy and infected branch of American elm. Brown streaking in the wood (xylem) indicates presence of the fungus in host vascular tissue just under the bark (Jared M. LeBoldus, Oregon State University, used with permission).

Positive diagnosis requires a laboratory test in which wood chips from fresh symptomatic branches are placed on solid culture media for growth. Within seven to 10 days dark fungal stalks (synnemata; 2 to 5 mm high) grow on the wood chips and support a pale, globose ball of sticky liquid containing asexual spores of *O. ulmi* or *O. novo-ulmi* (fig. 34-4).

Disease Cycle

In North America, there are three known vectors for the DED fungus: *Hylurgopinus rufipes* (native elm bark beetle), *Scolytus multistriatus* (smaller European elm bark beetle), and, most recently confirmed, *Scolytus schevyrewi* (banded elm bark beetle) (fig. 34-1). The cycle of infection by the causal fungus is closely linked to the life cycles of the insect vectors. These DED fungi are uniquely adapted for, and dependent on, overland transmission by elm bark beetles. The beetles breed under the bark of weakened or recently killed elm branches, whole trees, or cut logs. If the DED fungus is present at breeding sites, newly emerged beetles will carry spores of the pathogen to healthy elms and introduce the disease when feeding on the xylem of twigs and small branches in the crown. The beetles can fly up to 0.25 miles in search of feeding or breeding sites, but they may be blown many miles by winds.

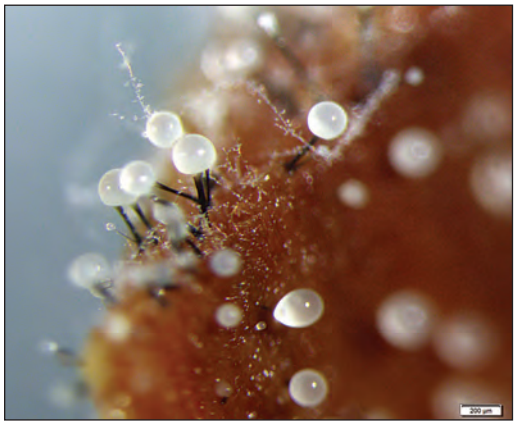

Figure 34-4—Dark-stemmed synnemata supporting a pale globose ball of sticky liquid containing the spores of *Ophiostoma ulmi* or *O. novo-ulmi* (Kelsey L. Dunnell, North Dakota State University, used with permission).

Damage

In the Great Plains, the American elm grows naturally in riparian areas. Because of its many desirable qualities, it was also frequently planted as ornamental and boulevard trees on farmsteads and in communities as well as in windbreaks. Today, most of the American elms in the Great Plains have been lost to DED. However, substantial numbers still remain in the very northern and southern parts of the region, as well as in communities that practice strict sanitation methods to manage the disease. This strict sanitation effort does not come without cost, but it allows communities to maintain thriving elm populations and also minimizes tree removal and replanting costs. The intangible costs of losing a well-adapted tree species for farmstead and windbreak plantings and the loss of a dominant species in riparian forest ecosystems are difficult to quantify, but certainly highly significant.

Management

In urban areas, a strict sanitation program can be extremely effective in slowing the spread of DED. Sanitation consists of removing sources of beetle habitat, which includes pruning and removing any dead or damaged branches and disposal of infected elm wood by burning, burying, chipping, or debarking. To limit the spread through root grafts to adjacent elm trees, the area around infected elm trees should be trenched (midway between trees) to a depth of 36 to 40 inches, and the trench should completely encircle infected trees. For these sanitation efforts to be effective, it is important that newly infected trees be identified quickly, removed, and destroyed promptly. Some cities have strict ordinances requiring prompt removal of infected trees and enforce bans on elm firewood with intact bark. The reward for these DED management efforts is a well-maintained elm population and a rate of tree loss reduced to a level the community can afford, allowing for gradual replacement with a diversity of species adapted to the region.

Unfortunately, in rural areas, windbreaks, and native woodlands, little can be done to forestall the loss of elms susceptible to DED. However, individual high-value trees may be protected by injection of fungicides at one- to three-year intervals into the tree root flares for the entire lifespan of the tree. The level of protection provided by fungicide treatments depends on tree uptake. Although treatment can be relatively expensive and repeated wounding from injections may seriously damage the tree, the value of a mature boulevard, landscape, or specimen tree may justify the cost. Some fungicide injections are labeled for therapeutic treatment, but the success of treating after infection is poor; treatment may nonetheless be worth trying on very high-value trees.

Although no elm species is totally resistant to the DED fungi, some of the Asiatic elms, as well as selections of American and European elms, have high levels of tolerance or resistance to infection by *O. ulmi* and *O. novo-ulmi*. Today, elm selections and hybrids from disease-resistant parents have been propagated and recently released for the commercial trade. Most of the new available cultivars are DED resistant. It is important to use cultivars best suited to the local environmental conditions in which they will be planted. Contact your local extension agent or arborist for current information and availability of DED-resistant cultivars.

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35. Oak Wilt

Jennifer Juzwik and David N. Appel

Oak wilt, caused by the fungus *Ceratocystis fagacearum*, is known to exist only in the central and eastern United States, although susceptible hosts and apparently suitable climate conditions exist in other parts of the country. Oak wilt was likely killing trees in Wisconsin and Minnesota as early as the 1890s based on historical accounts, but the causal fungus was not discovered and described until 1944 in Wisconsin. The origin of the fungus is unknown, although more recent evidence suggests that it may be an introduced pathogen from Central America, South America, or Mexico.

Hosts and Distribution

The disease is currently found in an area bounded by Minnesota to Pennsylvania in the north and Texas to South Carolina in the south. A recent new find (2008) in upstate New York was quickly treated and is now considered eradicated. Within the Great Plains states, the disease is widespread and common in Texas but is reported in eastern Oklahoma, Kansas, Nebraska, and South Dakota.

More than 33 species of oaks (*Quercus* spp.) as well as six other close relatives are known to be susceptible to *C. fagacearum* based on artificial inoculation studies or documented natural infections. Species of the red oak group, such as black (*Q. velutina*), blackjack (*Q. marilandica*), northern red (*Q. rubra*), pin (*Q. palustris*), and Shumard (*Q. shumardii*), are considered to be highly susceptible to the pathogen compared to members of the white oak group, such as bur (*Q. macrocarpa*), post (*Q. stellata*), dwarf chinkapin (*Q. prinoides*), and white oak (*Q. alba*), which display moderate to high levels of resistance. A range of susceptibility occurs in semi-deciduous live oaks (*Quercus fusiformis* and *Q. virginiana*) in Texas, depending on timing of infection, weather conditions, and host genetics.

Symptoms and Signs

Foliar symptoms of oak wilt occur any time from late spring to late summer or early fall. Symptom expression and progression are different in red oaks, white oaks, and live oaks.

Early foliage symptoms in red oaks are characterized by a subtle, off-green to bronze color. Necrosis on wilting leaves starts at the leaf margins and tip, progressing inward to the main vein and the base of the leaf. Foliage symptoms commonly occur first in the upper portion of the tree crown (fig. 35-1). Progression of the disease throughout the crown can be rapid, resulting in complete tree wilt within several weeks to several months. Red oaks infected later in the growing season may produce new shoots in the lower crown the next spring, but these will quickly die. Infected red oaks do not recover from the disease. Light bluish-gray to darker streaking develops in the outermost layer of wood of branches with recently wilted leaves or in the main stem of a completely wilted tree, although such streaking is often hard to detect (fig. 35-4A). Groups of red oaks may die over several years if grafted roots occur between closely spaced trees.

Foliar symptoms in moderately resistant white oak trees occur on scattered branches in the tree crown (fig. 35-2). Discoloration on wilting white oak leaves is more variable than that on red oaks. Reddish-brown to brown discoloration on bur and white oak starts at the leaf margin and toward the tip, but may often be limited to one side of the main vein. Symptomatic leaves of bur oak may be confused with those of bur oak blight, which is caused by the fungus *Tubakia iowensis* (see chapter 3). The veinal chlorosis and necrosis of live oak leaves are unique and diagnostic for the disease (fig. 35-3). Symptoms in white oak species develop more slowly than in red oaks, but time to tree death differs by species. Diseased live oaks in Texas usually die within three to eight months of infection. Moderately resistant oaks, such as bur oak, may die within two to four years of infection. In the highly resistant white oak, *C. fagacearum* infections cause dieback in one or more branches, but affected trees may not die for decades if at all. Such trees may be misdiagnosed as having symptoms of oak decline. Dark brown to blackish streaks in the outer

Figure 35-1—Foliar and crown symptoms of oak wilt in northern red oak (Jennifer Juzwik, U.S. Forest Service).

Figure 35-2—Foliar and crown symptoms of oak wilt in bur oak (Jennifer Juzwik, U.S. Forest Service).

Figure 35-3—Foliar and crown symptoms of oak wilt in Texas live oak (David N. Appel, Texas A&M University, used with permission).

xylem are visible when the bark is removed from an oak wilt-infected branch of some white oak species. This symptom appears as a ring of discolored tissue in the outer xylem in a cross section of the same branch (fig. 35-4B).

Foliar symptoms in live oak are unique and diagnostic for the disease. The veins turn chlorotic and eventually necrotic while the remainder of the leaf remains green. Diseased live oaks in Texas usually die within three to eight months of infection (fig. 35-3).

Mycelial masses, or fungal mats, of the fungus may appear on the inner bark and outer wood within several months or the spring after a tree dies from oak wilt (fig. 35-5). These occur most commonly in red oaks and occasionally, seldom, or never on species in the white oak group. Oak wilt mats are ephemeral in nature and can be difficult to detect on trees with deeply furrowed bark. A vertical crack occurs when special pad-like structures on the light to dark gray-colored mats rupture the bark. The ripe-fruit odor produced by the fungus is attractive to insects, particularly to sap beetles (Coleoptera: Nitidulidae).

Disease Cycle

The oak wilt fungus is spread from diseased oaks to healthy ones through aboveground transmission by insect vectors or through connected root systems. Aboveground spread to a healthy tree occurs when sap beetle vectors carrying spores of the oak wilt fungus visit xylem-penetrating wounds, generally within three days of the wounds' creation. Such spread results in new oak wilt centers (or "pockets"). Susceptible wounds can be created

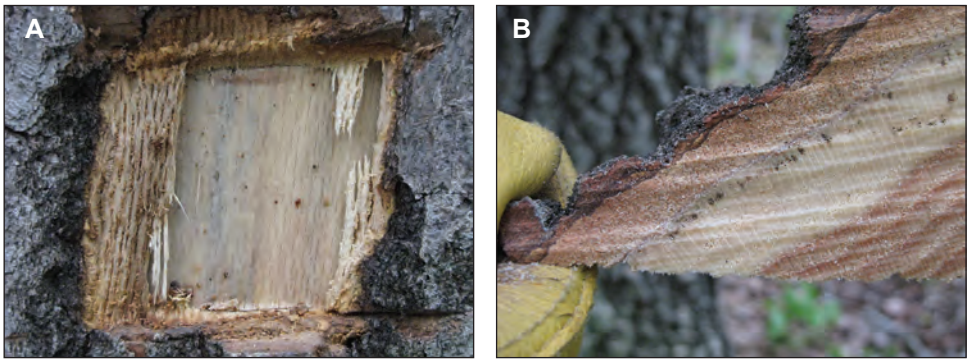

Figure 35-4—Vascular streaking in outer sapwood of northern red oak: (A) Tangential view of blue-gray streaking; (B) Brown discoloration in outer growth ring of stem cross section (Jennifer Juzwik, U.S. Forest Service).

Figure 35-5—Oak wilt fungal mat (dark gray) on the bark–wood interface of northern red oak with pressure pad in the center (Jennifer Juzwik, U.S. Forest Service).

by construction equipment, wind damage during storms, pruning or tree felling activities, vandalism, or any agent that penetrates through the bark and into the sapwood.

Fresh wound volatiles attract the principal sap beetle vectors (*Carpophilus sayi* and other *Coleopterous* spp.). Once the fungus has entered the vascular system of the tree, its asexual spores (conidia) are spread internally by sap flow to distant portions of the tree. Oaks respond to infection by producing balloon-like structures (tyloses) that plug the xylem vessels. This response, along with additional plugging of the vessels by metabolic fungal products, disrupts the sap flow, causing symptoms in the diseased tree. After the tree wilts, the fungus grows outward in its hyphal form from the xylem vessels to the bark-wood interface (the cambium), where the fungal mass accumulates to form the oak wilt mats.

Sexual spores (ascospores) are produced on mats when sap beetles introduce spores of the opposite mating type of the fungus. Sap beetles pick up spores of the fungus while feeding on or crawling across the sporulating mats. As the mats age, the beetles emerge, carrying the fungus inoculum to infect wounds on healthy trees.

Shared root systems of clonally propagated live oak species allow for belowground spread of *C. fagacearum* in those species. Naturally occurring root grafts that connect the vascular system of neighboring oak tree roots also provide a pathway for fungus spread

among oak species. Inter-tree grafts most commonly form between oaks of the same species; however, grafts can also form between different oak species. Root grafts occur most frequently in light textured and deep soils in relatively flat terrain. Root sprouting occurs in live oaks and results in new clonal trees (ramets) that share a common root system.

In the roots of an infected tree, conidia may be translocated through root grafts to an adjacent healthy oak or through the shared root system of clonally propagated live oak species, resulting in belowground spread. Upward movement of the fungus from the roots to the aboveground portion of the tree through the vascular system leads to the wilting process previously described. The fungus can continue to spread to nearby oaks through connected roots, leading to expansion of the disease center.

Damage

Once infected, red oak species and many white oak species die from oak wilt. Although an infected, highly resistant white oak may not die from oak wilt, the death of major limbs may render the tree undesirable as a landscape or specimen tree. Oak wilt is a devastating disease in portions of the oak wilt range where epidemics are ongoing. Tens of thousands of oaks die annually from oak wilt in the Upper Midwest and millions of oaks have died in Texas since discovery of the disease there. In contrast, oak wilt occurs sporadically in the Appalachian Mountains and the Ozark Mountains. The small, isolated disease centers that result from overland spread often die out because of the diversity of species, topography, or other site limitations. Regardless, the disease can cause significant losses in urban and community forests in these regions as well as other parts of the disease range. When consistent, integrated, and sustained suppression programs are used, oak wilt has proven to be a manageable disease in states experiencing ongoing epidemics.

Management

Management of oak wilt is based on general approaches that either prevent aboveground spread or stop belowground spread of the fungus, whether in a residential lot or a rural forest. The range of oak wilt in the United States cuts across many forest types and dominant *Quercus* species, so that practices or tools to achieve consistent management are not appropriate for every circumstance owing to varying site conditions, costs, and predicted environmental disruption. Availability of equipment may also be a factor in tool selection. In general, effective oak wilt management is a result of using a variety of tools or strategies that limit the spread of the causal fungus.

Early diagnosis of oak wilt is critical to managing the disease effectively and to justify the costs of treatment. Thus, annual monitoring for the disease is important in localities known to have oak wilt. When infected oaks are found, approaches can be developed based on resources available, landowner concern, and established priorities for management. When possible, remove recently wilted red oaks to eliminate sources of inoculum for insect vectors. Removal and proper disposal should occur before mats are produced in the late winter or spring of the year following tree wilt. Logs from diseased oaks should not be removed from the site without being properly treated. Long-distance transmission of the fungus by insects emerging from transported logs has resulted in major extensions of the oak wilt range.

Prevent infection of healthy oaks by avoiding wounding of trees, particularly in spring and early summer, when risk of aboveground spread is highest. Community ordinances may require use of protective measures during construction activities. Utility and arboriculture companies may restrict line-clearing and tree trimming during critical time periods and apply wound treatments when cutting is unavoidable. Educational outreach, such as billboards and public service announcements, raise public awareness of the need to prevent wounding and the hazards of moving oak firewood and logs. Municipal ordinances that mandate removal and treatment of dead oaks in oak wilt areas are also helpful.

Belowground spread of the oak wilt fungus accounts for the vast majority of oaks that die annually from the disease. Common root systems or grafted roots can be disrupted by using mechanical or chemical means to stop this spread. Cutting roots with a trencher to a depth of at least 48 inches or a vibratory plow with a 60-inch-long blade is an effective method in areas where soils and sites are suitable for the equipment. Shallow rocky soils and even-layered rock in Texas have necessitated the use of rock saws able to obtain the same depths for cutting oak roots. Chemical fumigants injected into 18-inch-deep holes evenly spaced along a line have been used in the past to create a narrow strip of dead roots between oak wilt-affected and healthy trees. Chemical toxicity and practical application concerns have greatly reduced or eliminated use of fumigants by practitioners.

Most failures of root cutting or disruption treatments to control oak wilt are attributed to insufficient depth of treatment or poor placement of the treatment line. Several models have been developed to guide management foresters in placing the treatment line. The general approach is to place the line far enough away from the diseased trees that there is low probability of the fungus being in the roots of the symptom-free trees outside the treatment line. However, the line should be close enough that the fungus will die before reaching the treatment line. It is assumed that the fungus will die in cut or otherwise killed roots within several years; then re-grafting can occur. There is some evidence that inserts in the trench may improve their performance by acting as a barrier to re-grafting of roots, but such materials have had only limited use. Felling of all oaks within a treatment line and even extraction of the stumps has been used to increase success of treatments in some state programs.

Systemic fungicides injected into the vascular system of symptom-free, high-value oaks in close proximity to diseased trees have been used to suppress disease development in trees at high risk of infection, but will not limit belowground spread of the pathogen. There are several methods available to inject trees, but only approved, labeled fungicides should be used to control oak wilt. Also, use an injection method only if it has been proven effective through documented scientific study, is relatively easy to use, and is safe for the applicator. Injection and subsequent pruning of symptomatic (<30 percent crown wilt) live, bur, and white oak can also suppress disease development, maintain aesthetic appeal, and extend longevity of high-value trees.

The management of oak wilt requires a sustained, long-term commitment in order to ultimately contain the pathogen and save trees. In this regard, a management plan may also include replanting. If so, any non-oak tree species can be used as long as they are well-suited for the site. If oaks are preferred, then consider only members of the white oak group.

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36. Verticillium Wilt

Mark O. Harrell and Laurie J. Stepanek; revised from David S. Wysong and Mark O. Harrell (Riffle and Peterson 1986)

Two closely related species of fungi, *Verticillium albo-atrum* and *V. dahliae*, are the cause of Verticillium wilt in hundreds of woody and herbaceous plant species around the world. Both species can infect and kill trees and shrubs, but wilt caused by *V. dahliae* is more common.

Hosts and Distribution

Verticillium wilt affects more than 300 species of plants, including food and fiber crops, annual and perennial ornamentals, and landscape trees. The disease occurs in forest stands but is far more destructive in landscape plantings. Valuable ornamental trees can be killed or severely damaged. Verticillium wilt occurs in every country in the world and in every state in the United States. Susceptible trees include apple and crabapple (*Malus* spp.), ash (*Fraxinus* spp.), catalpa (*Catalpa* spp.), cherry (*Prunus* spp.), elm (*Ulmus* spp.), linden (*Tilia* spp.), black locust (*Robinia pseudoacacia*), Kentucky coffeetree (*Gymnocladus dioicus*), magnolia (*Magnolia* spp.), maple (Acer spp.), pin oak (*Quercus palustris*), red oak (*Q. rubra*), poplar (*Populus* spp.), redbud (*Cercis* spp.), Russian-olive (*Elaeagnus angustifolia*), serviceberry (*Amelanchier* spp.), and smoke tree (*Cotinus* spp.). Some species and cultivars of apple, crabapple, linden, poplar, and serviceberry may be resistant depending on the strain of *Verticillium* in the soil.

Figure 36-1—Wilting branch on catalpa caused by Verticillium wilt (John Ball, South Dakota State University, used with permission).

Figure 36-2—Norway maple leaf (*Acer platanoides*) with Verticillium wilt symptoms (Mark O. Harrell, Nebraska Forest Service, used with permission).

Symptoms and Signs

Verticillium wilt in trees has acute and chronic symptoms. Acute symptoms include leaf wilting, curling, and drying on individual branches (figs. 36-1, 36-2 and 36-3). In maple, catalpa, and elm these symptoms are sometimes preceded by a general yellowing or reddening of the foliage. Trees may begin to show leaf symptoms as early as March or as late as November. If early symptoms on a few branches go unnoticed, sudden wilting of the entire crown may be the first symptom seen. Chronic symptoms include a slowing of twig growth, sparse foliage, stunted leaves, abnormally heavy seed crops, and branch dieback. Some trees such as maple may have long areas of dead bark on branches and the trunk. Other trees, such as elms, are more tolerant to infection and may decline slowly over several years.

Vascular streaking is another symptom of *Verticillium*-infected trees. In branches with advanced stages of wilt, the sapwood discolors in the form of bands or streaks that follow the grain of the wood (fig. 36-4). Discoloration occurs most frequently in the earlywood of the current season's growth. In trees that wilt in early summer, the discoloration may not be noticeable when the branch is examined in cross section. However,

Figure 36-3—Norway maple with branches killed by Verticillium wilt (Mark O. Harrell, Nebraska Forest Service, used with permission).

it is usually seen as fine streaks on the surface of the sapwood when the bark is peeled carefully from a wilted branch. In branch cross sections, the discoloration appears as a series of dots in a wood ring. In some cases, the dots are so abundant that the entire ring appears discolored. Trees that have been infected for more than a year may have discoloration in more than one growth ring (fig. 36-5).

In severely wilted trees the discoloration in the sapwood may be abundant and extend to the tips of wilted branches. In others the discoloration may be limited to the trunk sapwood, or it may extend only a few inches into the basal portions of wilted branches. In maple, the discolored wood is light to dark green. In catalpa it is purplish pink, changing to bluish brown upon drying. In elms and many other tree species, it is light to dark brown. Ash wood often shows no streaking, or the streaking may be very light, even when severely infected.

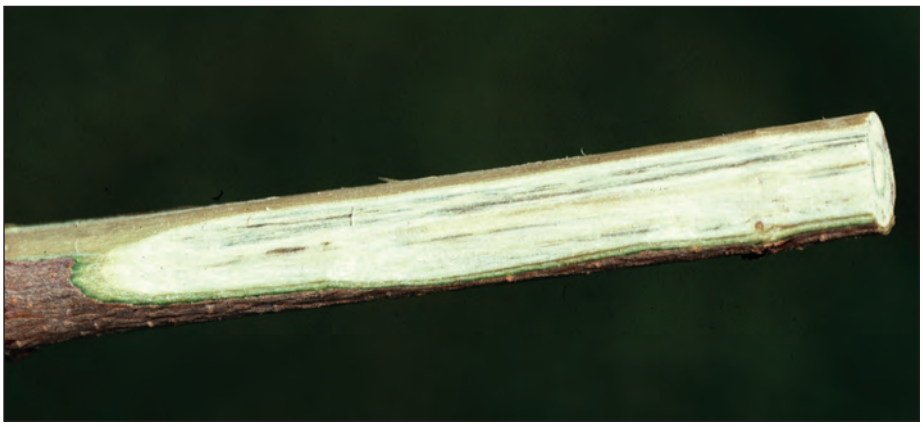

Figure 36-4—Verticillium wilt streaking in Norway maple (Mark O. Harrell, Nebraska Forest Service, used with permission).

Figure 36-5—Norway maple with discolored sapwood and loose bark from Verticillium wilt (Mark O. Harrell, Nebraska Forest Service, used with permission).

Disease Cycle

Verticillium is a soil-borne fungus that invades trees through roots. When roots in infested soil grow close to *Verticillium* hyphae or resting structures (microsclerotia), the hyphae in the soil or from germinating microsclerotia penetrate intact nonwoody roots or enter both woody and nonwoody roots through wounds. After entering roots, the fungus spreads through the tree either by growth of hyphae or by spores (conidia) transported by upward movement of xylem sap. *Verticillium* can exist for several years in roots and trunks of trees killed by the disease.

Microsclerotia allow the fungus to persist in soil for many years away from a parasitized host. These resting structures are most abundant in the top 12 inches of soil. Conidia, which are important in fungal movement within the tree, do not survive in soil for more than a few weeks.

The movement of *Verticillium* to new areas occurs mostly by the movement of microsclerotia. They can be carried in root balls, on bare roots of infected trees, and in wood chips from trees killed by the disease. The microsclerotia contaminate the soil where the trees are planted or wood chips are placed. Because microsclerotia can exist for many years in infected wood, trees killed by *Verticillium* should not be used as mulch. Within nursery fields, microsclerotia can be spread by normal tillage operations and in soil that adheres to equipment that has been used in an infested field.

Damage

Damage caused by Verticillium wilt is variable and depends upon age and species of the host affected. Trees with trunk diameters of 1 to 2 inches can be killed within one year of infection. Older trees may live many years after infection, but they normally decline over time. The disease typically has a greater impact on nursery seedlings and trees in landscape plantings compared to forest stands.

Management

Prevention is the best method for controlling Verticillium wilt. Avoid planting susceptible trees in areas where other plants are known to have died from the disease. Trees resistant or immune to *Verticillium* include arborvitae (*Thuja occidentalis*), beech (*Fagus* spp.), birch (*Betula* spp.), butternut (*Juglans cinerea*), fir (*Abies* spp.), ginkgo (*Ginko biloba*), hackberry (*Celtis* spp.), hawthorn (*Crataegus* spp.), hickory (*Carya* spp.), honeylocust (*Gleditsia triacanthos*), hophornbeam (*Ostrya virginiana*), juniper (*Juniperus* spp.), larch (*Larix* spp.), linden, mountain-ash (*Sorbus* spp.), mulberry (*Morus* spp.), bur oak (*Q. macrocarpa*), white oak (*Q. alba*), pear (*Pyrus* spp.), pine (*Pinus* spp.), spruce (*Picea* spp.), sycamore (*Platanus occidentalis*), walnut (*Juglans* spp.), and willow (*Salix* spp.). Some species and cultivars of apple, crabapple, linden, poplar, and serviceberry are resistant.

Trees with recent or mild symptoms of Verticillium wilt should not be removed immediately because they may recover to some degree after cultural treatments that improve environmental conditions and strengthen tree vigor. Water stress increases a tree's susceptibility to Verticillium wilt. Watering appropriately during periods of drought and using a mulch bed of wood or bark chips to improve soil conditions can reduce water stress and increase resistance to the disease. Do not use mulch from trees that were killed by Verticillium, because microsclerotia can exist for many years in the infected wood. Branches with severe symptoms should be pruned out, but those with mild symptoms could be left to see if they recover. Fungicide treatments are not effective.

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37. Yellows Disease of Ash, Lilac, and Elm

James A. Walla and Les Koch; partially revised from Wayne A. Sinclair and David S. Wysong (Riffle and Peterson 1986)

Yellows diseases are caused by phytoplasmas (bacteria lacking cell walls). Phytoplasmas occur systemically in phloem tissue of their host plants and in their insect vectors (primarily leafhoppers and planthoppers). Ash yellows (AshY) and lilac witches'-broom (LWB) are caused by '*Candidatus* Phytoplasma fraxini'. Elm yellows (EY, previously known as elm phloem necrosis) is caused primarily by '*Ca.* P. ulmi', but it was found to be caused by a member of the clover proliferation phytoplasma group in one area in northern Illinois. There is a wide range of aggressiveness among isolates of many phytoplasmas. Such has been demonstrated for AshY phytoplasmas and would be expected for LWB and EY.

Hosts and Distribution

AshY phytoplasma hosts include at least 13 ash (*Fraxinus*) species, including black (*F. nigra*), green (*F. pennsylvanica*), white (*F. americana*), and velvet ash (*F. velutina*), and 20 lilac (*Syringa*) species, including common (*S. vulgaris*), dwarf Korean *(S. meyeri)*, Japanese tree (*S. reticulata*), late (*S. villosa*), and Manchurian lilac (*S. pubescens* subsp. *patula*). Naturally-infected EY phytoplasma hosts include most native North American elm (*Ulmus*) species—American (*U. americana*), cedar (*U. crassifolia*), September (*U. serotina*), slippery (red) (*U. rubra*), and winged elm (*U. alata*)—Chinese elm (*U. parvifolia*), and the American-Asian hybrid slippery × Siberian elm (*U. pumila*). In New York, the white-banded leafhopper (*Scaphoideus luteolus*) is a known vector of the EY phytoplasma. In Pennsylvania, the spittlebug *Lepyronia quadrangularis* and the leafhopper *Latalus* spp. have been shown to be vectors. Leafhoppers in the genus *Scaphoideus* are probably the principal vector of the AshY phytoplasma.

AshY occurs primarily in North America. In the Great Plains, it is present in most sites with mature ash from Alberta to Colorado and Manitoba to Oklahoma. LWB is known in eastern and central United States. In the Great Plains, it is known only from North Dakota and Oklahoma. EY occurs in eastern and central United States and was present in southern Ontario; it also occurs in Europe. It is known from the eastern Great Plains states and Wyoming, but is rarely recognized. States and provinces in the Great Plains where these diseases are not known have not been adequately sampled.

Symptoms

Symptoms of AshY and LWB may include any combination of reduced growth, deliquescent branching, progressive branch death, abnormal roots, chlorotic or deformed leaves, and witches'-brooms (fig. 37-1). Witches'-brooms are the only diagnostic symptom of AshY and LWB. Witches'-brooms have fine, short, and abnormally upright branches; branches growing from leaf axils; and dwarfed, deformed leaflets. Witches'-brooms are rare on infected green ash in the Great Plains, but are common on trunks of trees that have top dieback or have been cut. In lilac, witches'-brooms are common on the most susceptible hosts (fig. 37-2). Witches'-brooms are rare and small on common lilac. In established plants, symptoms typically develop two or more years after infection.

EY can be mistaken for Dutch elm disease, especially by those not checking for EY. Symptoms of EY may include drooping and yellowing (fig. 37-3) usually in mid-to-late summer, leading to tree death within the next two years. The yellow leaves may drop or suddenly wilt, shrivel, turn brown, and remain attached to the twig. Roots are also affected, with fine roots dying first. The phloem and cambium change color from light cream to tan and then dark brown, typically as uniform discoloration, not streaking, although elongate dark brown flecks may be present. A faint wintergreen aroma emanates from freshly exposed, moist inner bark of infected branches, but this aroma is not always present and, when present, is difficult for many people to detect. In established trees, symptoms typically develop the season after infection.

Figure 37-1—Ash yellows witches'-brooms. (A) Several witches'-brooms on a single green ash trunk, with characteristic lime-green foliage; (B) Witches' broom with dwarfed, deformed, and single leaflet leaves; (C) Witches' broom in dormant season showing upright, narrow-angle branching and branching from leaf axils (James A. Walla, Northern Tree Specialties, used with permission).

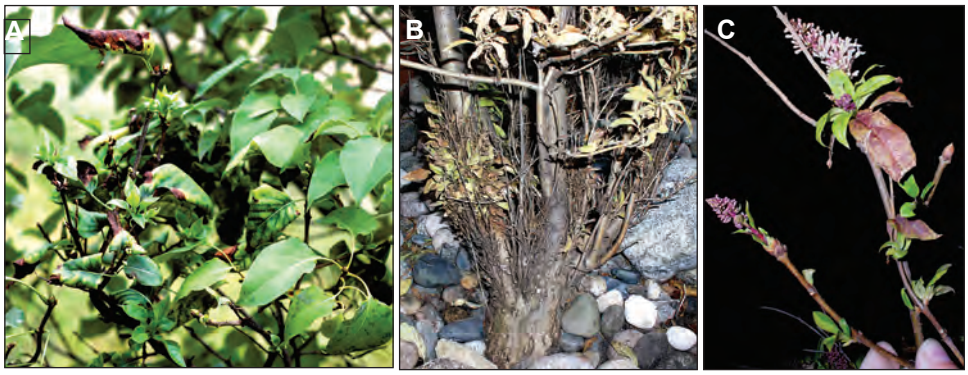

Figure 37-2—Lilac witches'-broom. (A) Late-season growth and deformed, scorched leaves of common lilac; (B) Multiple witches'-brooms at base of late lilac; (C) Late-season growth and late flowering lilac (James A. Walla, Northern Tree Specialties, used with permission).

Figure 37-3—Elm yellows symptoms. (A) Single American elm branch showing symptoms of wilted, yellowing leaves; (B) Witches'-broom on hybrid elm (James A. Walla, Northern Tree Specialties, used with permission).

Symptoms commonly occur on the whole tree over a period of only a few weeks in mid- to late summer, although monitoring from early infection will often show symptoms spreading from one branch or portion of a tree. On slippery elm, similar symptoms develop, but small witches'-brooms also form just before a tree dies, the discolored bark is inconsistent, and the aroma when present is like maple syrup.

Disease Cycle

Vector insects typically transmit the pathogens while feeding on tree leaves in summer and early fall. The pathogens spread in the phloem tissues of infected plants where they overwinter primarily in roots. Pathogen survival may be reduced in aboveground parts in colder winters. In spring, phytoplasmas spread to greater portions of infected plants, and may be acquired by host insects feeding on the leaves. There is a latent period of three to four weeks before the phytoplasmas are transmitted to new hosts to complete the disease cycle. Phytoplasmas can also be transmitted by vegetative propagation. These pathogens are not known to be transmitted through seed.

Damage

AshY causes progressive growth suppression, reduced competitiveness, and decline in susceptible white and green ash, but there is wide variation in these effects. AshY and LWB reduce life expectancy and aesthetic value of susceptible hosts. Reduced pathogen aggressiveness, increased host tolerance, and good growing conditions typically result in reduced damage by AshY and LWB. A moderately susceptible tree may exhibit only a reduced growth rate, which may be desirable in some landscape settings. Decline resulting from these diseases may be triggered by occurrence of stress, whereby infected trees do not recover but noninfected trees do. Surveys of trees in six states and three provinces in the Great Plains did not find an association between tree condition and AshY phytoplasma presence in green ash, likely because there are multiple factors that damage ash. Individual ash and lilac have been severely affected by these diseases in North Dakota (fig. 37-4). Evaluation in North Dakota found very wide variation in both pathogen aggressiveness (fig. 37-5) and green ash tolerance.

In the primary range of EY, there are periodic disease outbreaks, and most North American elm species are killed in those areas. EY kills most North American elm species one or two years after infection. It has been observed that infected slippery elms may survive for several years. In the more tolerant Eurasian elm species, trees that have been inoculated grow slowly and may develop chlorosis and witches'-brooms. As EY affects

Figure 37-4—Damage caused by ash yellows and lilac witches'-broom. (A) Reduced and sparse growth on green ash; the center tree was infected by an aggressive ash yellows phytoplasma isolate. (B) Reduced growth, sparse foliage, and deformed shape of late-blooming lilac (James A. Walla, Northern Tree Specialties, used with permission).

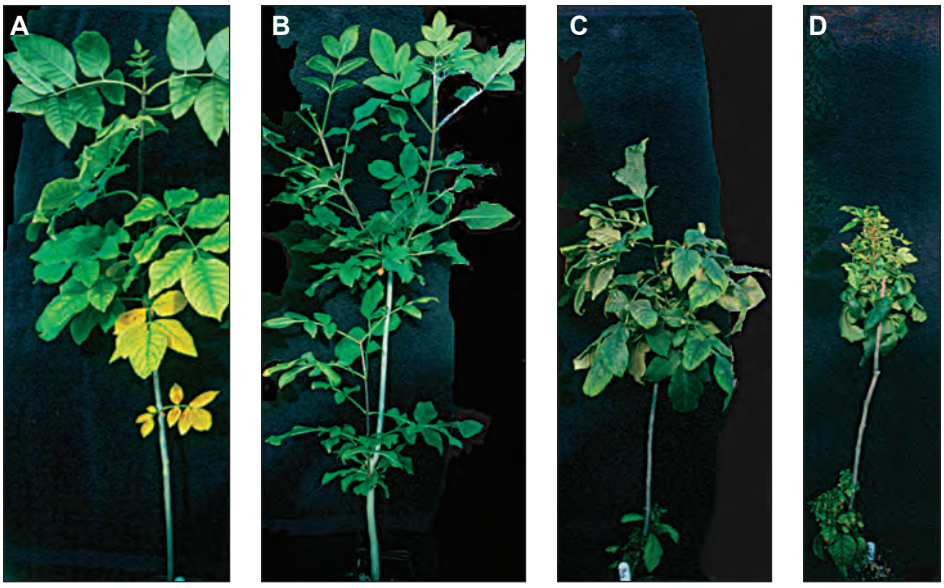

Figure 37-5—Effects of ash yellows phytoplasma aggressiveness on growth of green ash seedlings. (A) Non-inoculated control; (B) Low aggressiveness; (C) Moderate aggressiveness; (D) High aggressiveness (James A. Walla, Northern Tree Specialties, used with permission).

elms, the trees attract and support large populations of the bark beetles that vector the Dutch elm disease fungus. It is not unusual for an elm to have both EY and Dutch elm disease and for Dutch elm disease to increase greatly in a location where EY is active. EY apparently occurs at a very low level of incidence in the Great Plains and is not known to have caused substantial damage there.

Management

The best disease management for AshY, LWB, and EY is use of tolerant species or selections. Green ash and velvet ash are more tolerant than is white ash to AshY. Three of six green ash cultivars ('Bergeson,' "Dakota Centennial®," 'Patmore') and one of five white ash cultivars ('Autumn Applause') are tolerant of AshY. The late-blooming lilac species and hybrids are less tolerant to LWB damage, whereas common lilac is relatively tolerant. Eurasian elms are more tolerant than the native elm species, and they have not been damaged when exposed to natural infection in North America. 'Frontier,' 'Pathfinder,' and 'Patriot' hybrid elms appear to be tolerant to EY, and "Homestead" hybrid elm appears to be resistant to EY.

Calcium oxytetracycline is labeled as a systemic antibacterial for seasonal suppression of AshY, EY, and some other phytoplasma diseases. Injected trees remain infected, but temporary relief from damage may be provided for young infected trees. Disease prevention may be achieved if the antibacterial is used before or at the time of infection. Concern for development of microbial resistance to antibiotics should be considered before they are used for management of phytoplasma diseases. Damage to trees susceptible to some phytoplasmas is substantially reduced by grafting onto rootstocks tolerant to those phytoplasmas; such has not been reported for AshY, LWB, or EY, but would be expected to hold true. Management of AshY should be tempered by consideration of the long-term prospect of loss from emerald ash borer.

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38. Armillaria Root Disease

James T. Blodgett; revised from Lloyd R. Fuller and Robert L. James (Riffle and Peterson 1986)

Armillaria root diseases are caused by several species of fungi in the genus *Armillaria*. These pathogens are common facultative parasites on more than 600 plant species throughout the world. Root diseases caused by these fungi are responsible for considerable economic damage in natural forests, plantations, orchards, and vineyards.

Hosts and Distribution

Root diseases caused by *Armillaria* species occur sporadically throughout the Great Plains, but their distribution in most of the Great Plains states is not well documented. Most trees, shrubs, vines, and some herbaceous plants are susceptible to these pathogens. Armillaria root diseases are common in windbreaks, forests, and landscapes. Some susceptible plants native to or planted in the Great Plains include: apples (*Malus* spp.), aspen (*Populus* spp.), boxelder (*Acer negundo*), cottonwoods (*Populus* spp.), oaks (*Quercus* spp.), paper birch (*Betula papyrifera*), Peking cotoneaster (*Cotoneaster acutifolius*), ponderosa pine (*Pinus ponderosa*), poplars (*Populus* spp.), Scots pine (*Pinus sylvestris*), stone fruits (cherry, peach, and plum; *Prunus* spp.), Tatarian honeysuckle (*Lonerica tatarica*), willows (*Salix* spp.), and members of the rose family (Rosaceae).

Symptoms and Signs

Symptoms are not always diagnostic, and trees with Armillaria root disease do not always show well-defined symptoms. When present, crown symptoms resemble those of other root disorders: chlorotic foliage, reduced terminal growth, premature foliage drop, branch dieback, and stress crop of seeds or cones (fig. 38-1). One or more of these symptoms may be present on a single tree over many years or sudden crown mortality may occur. Sudden crown mortality can occur even if a tree was infected for several years. Hosts infected at the base of the trunk (root collar) may have external basal resinosis or gummosis (fig. 38-2), but these symptoms may not always be present. *Armillaria* species cause white rot, often with zone lines (fig. 38-3), in roots and lower stems. Decay can be difficult to detect unless trees are uprooted or break at the root collar.

Diagnostic signs include mycelial fans under or in the bark, rhizomorphs (a cordlike structure containing mycelia, which resembles roots or shoe strings), and mushrooms. *Armillaria* species produce characteristic mycelial fans (fig. 38-4) that appear as white to light cream-colored, fanlike mycelial mats beneath or inside the bark of roots and lower stems (butts).

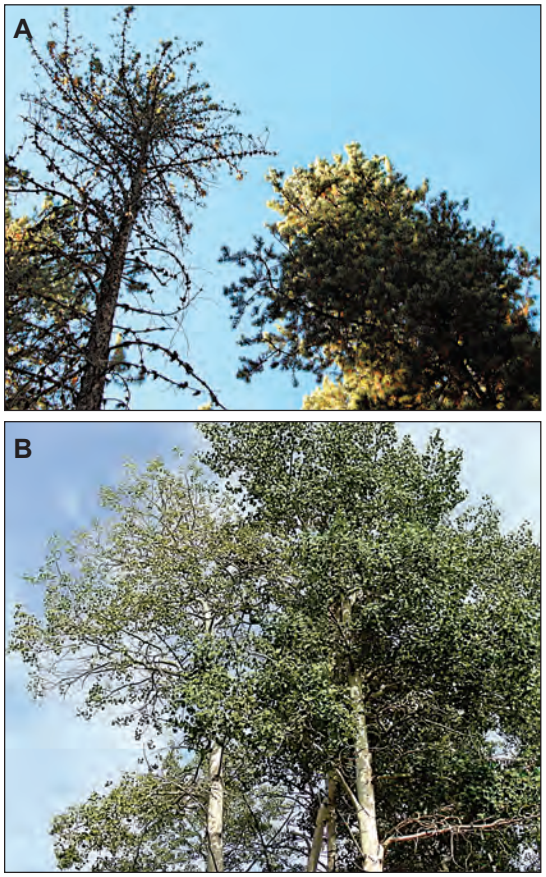

Figure 38-1—Chlorotic foliage with extensive dieback, a typical symptom of (A) lodgepole pine (*Pinus contorta*) and (B) aspen infected with Armillaria root disease (James T. Blodgett, U.S. Forest Service).

Figure 38-2—Lodgepole pine infected with Armillaria root disease showing resinosis at the root collar (James T. Blodgett, U.S. Forest Service).

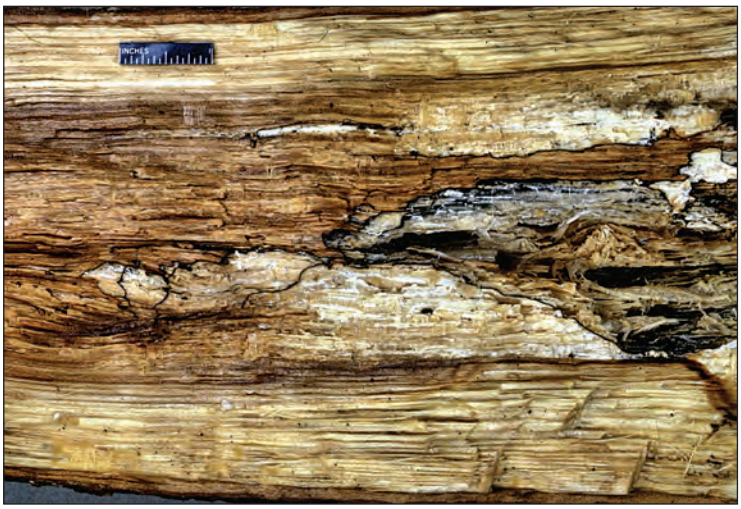

Figure 38-3—Armillaria white rot with zone lines (Daniel H. Brown, U.S. Forest Service, Bugwood.org).

Figure 38-4—Mycelial fan, a diagnostic sign of Armillaria root disease, seen here on aspen (James T. Blodgett, U.S. Forest Service).

Figure 38-5—Rhizomorphs (gray to black, see arrows) in soil growing into a root and root collar, diagnostic signs of Armillaria root disease (James T. Blodgett, U.S. Forest Service).

Figure 38-6—Fruiting bodies of an *Armillaria* species at the base of a paper birch (James T. Blodgett, U.S. Forest Service).

Mycelial fans develop in the cambial zone of roots and root collars, and can spread into stems. The rootlike rhizomorphs may be found on root surfaces, in adjacent soil (fig. 38-5), in decayed wood, or under the bark of living or dead trees. These structures are gray, reddish brown, or black on the exterior with a white inner core and tip. Rhizomorphs branch like roots, but typically have less variation in diameter (0.5 to 2 mm) compared to roots. Rhizomorphs that occur on root surfaces, in soil, and in decayed wood tend to be round; ones beneath bark tend to be flat.

Armillaria species might produce mushrooms in sporadic years (fig. 38-6). Mushrooms are short-lived, but may be present from late summer through fall if moisture is adequate. The mushrooms grow in small to large clusters on living and dead hosts, or on soil near underground wood. *Armillaria* mushrooms can be identified by their honey-yellow caps 1 to 5 inches across with a persistent annulus (ringlike structure) on the upper stem. Caps have fine erect hairs or scales on the upper surface. The white gills on the underside of the cap are attached to the stem. Spores are white to light cream colored.

Differences among the *Armillaria* species and host species result in variation among symptoms and signs, and *Armillaria* species are often somewhat host specialized. *Armillaria* can be identified to species by traditional morphology, mating types, cultural characteristics, and DNA sequences. Additional studies are needed to identify *Armillaria* species present in many areas of the Great Plains.

Disease Cycle

These pathogens spread primarily as vegetative mycelium by root contact, root grafts, or rhizomorphs. Rhizomorphs can grow considerable distances (up to 6.6 ft per year) through soil and initiate parasitic infections or saprotrophic colonization of roots or root collars. *Armillaria* species can occupy sites for many years. In one relatively dry region, a single vegetative clone of *Armillaria* was shown to occupy an area of approximately 2,384 acres and was estimated to be up to 8,650 years old. During favorable late summer or fall weather, mushrooms of *Armillaria* species may produce prolific wind-dispersed spores. However, unlike many other decay pathogens, the spores rarely initiate disease.

Mycelial strands can colonize susceptible host cambium and initiate canker formation. Cankers can remain dormant (quiescent) or progress into lethal infections. Healthy, vigorous hosts or resistant host species may survive for many years. Stressed hosts, hosts with numerous or extensive infections, and susceptible host species can be killed quickly. Successfully infected hosts usually die when the fungus kills the cambium around root

collars. Live trees can be uprooted or break at the lower stem because of mechanical failure resulting from decay. Although such mechanical failures are more frequent during high-wind conditions, they can occur during conditions of low or no wind.

Saprotrophic colonization of wood tissues occurs after trees or tree tissues die. *Armillaria* species can also colonize trees killed by other damage agents. The fungus can survive for decades in dead roots or stumps, and can infect new trees over the course of decades.

Damage

Armillaria root diseases affect trees used for many forest services and products including recreation, aesthetics, timber, windbreaks, watershed protection, soil stabilization, and carbon sequestration. Root diseases in developed sites can produce hazard trees that threaten life and property because of an increased probability of hosts falling. Armillaria root diseases cause direct tree mortality and indirect mortality through uprooting or breakage of lower stems, growth reduction, and wood loss from decay. They also predispose trees to other lethal agents including insects and other diseases. Armillaria root diseases are common contributing factors in many declines and can increase the likelihood of fire by increasing the accumulation of dead fuels.

In some situations, *Armillaria* species are not damaging unless hosts are under stress. Stresses can include extended droughts or dry sites, competition for light or nutrients, insect attacks, infections by other pathogens, or poor planting technique including offsite plantings. Young trees are often quickly killed by *Armillaria* species, whereas 15- to 20-year-old trees tend to be more tolerant.

Management

These pathogens are difficult or impossible to eradicate. Management measures can include removing infected stumps and roots, limiting underground spread of the fungus, managing for resistant species, using proper planting techniques, and maintaining tree health. Management of root diseases over extensive acreage is seldom possible or practical. Valuable ornamental, shade, and orchard trees that are adjacent to infected trees may be protected somewhat by removal of infected stump and root material from the soil. Chemicals have been used to sanitize infested soil and wood, but only professional chemical applicators should apply these chemicals near healthy trees. Favoring healthy existing species and favoring species that are resistant to Armillaria root disease during thinning or planting offer the most promising and long-lasting approaches to disease control. Lists of resistant plants that are well adapted to a locale can be obtained from local extension educators or from the reference by Raabe and McCain (1967). Use proper planting methods, such as adequate site preparation, selection of species suitable for the site, use of foliage that is in balance with root systems, selection of disease-free planting stock, and prevention of J-roots (tap roots pointing up; usually the result of improper planting). Reduce host stress by thinning, weed control, watering during droughts, and managing other diseases and insect pests so that the impact of Armillaria root diseases can be minimized.

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39. Ganoderma Root Rot or White Mottled Rot

James T. Blodgett

Ganoderma root rot, also called white mottled rot, is caused by the fungus *Ganoderma applanatum*. This fungus is found in all 50 states and occurs throughout North America and Europe. It is a pathogen and a common wood-decaying fungus of roots and lower stems (butts) of many deciduous and some coniferous trees species. Ganoderma-caused root rot has been reported in live trees, such as apple (*Malus* spp.), aspen (*Populus* spp.), basswood (*Tilia* spp.), beech (*Fagus* spp.), birch (*Betula* spp.), cherry (*Prunus* spp.), citrus (*Citrus* spp.), cottonwood (*Populus* spp.), elm (*Ulmus* spp.), hemlock (*Tsuga* spp.), hornbeam (*Carpinus caroliniana*), horsechestnut (*Aesculus hippocastanum*), black locust (*Robinia pseudoacacia*) and honeylocust (*Gleditsia triacanthos*), maple (*Acer* spp.), mulberry (*Morus* spp.), oak (*Quercus* spp.), spruce (*Picea* spp.), sycamore (*Platanus occidentalis*), tulip tree (*Liriodendron tulipifera*), sweetgum (*Liquidambar styraciflua*), and willow (*Salix* spp.).

Figure 39-1—Drawing on the lower surface of *Ganoderma applanatum* conk (James J. Worrall, U.S. Forest Service).

G. applanatum is commonly known as the artist's conk. The name comes from the use of its fruiting bodies as a drawing medium by artists (fig. 39-1). When the fresh lower surface is rubbed or scratched, it immediately changes from white to dark brown, producing shading or visible lines. When the conk is dried, drawings become permanent.

Hosts and Distribution

In the Great Plains, *G. applanatum* occurs predominantly in aspen, cottonwoods, and other *Populus* species. This fungal pathogen is irregularly distributed across the Great Plains, but its distribution is not well documented in many areas.

Symptoms and Signs

Crown symptoms in trees affected by *G. applanatum* are not always apparent. When present, symptoms resemble those of other root disorders, including reduced terminal growth, chlorotic foliage, premature foliage drop, and branch dieback. Although *G. applanatum* is often found in dead trees in an affected stand, it can decay roots and butts of otherwise healthy trees in the stand.

Extensive wood decay in roots and butts of infected trees is often overlooked until trees fall. Trees with this root disease often break at the soil line or slightly below with few or no attached roots (fig. 39-2). Often no other symptoms are apparent. Small trees or trees growing on poor sites with dry, shallow soils may be killed before decay leads to

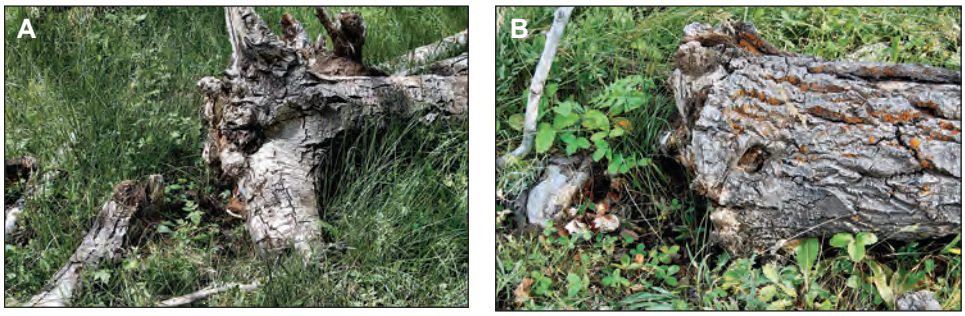

Figure 39-2—Roots infected with *Ganoderma applanatum.* Roots often break (A) near the stem or (B) at root collars. Conks might be present (A) above or (B) below the break (James T. Blodgett, U.S. Forest Service).

windthrow. The fungus causes a white rot, and decayed wood is mottled white and light tan, eventually becomes spongy, and may develop black lines (fig. 39-3).

Fruiting bodies (conks) are the best indicator of host infection, but conks typically are not formed until several years after initial infection. Although conks are not produced on some trees with extensive decay, conks often develop before trees fall or die. Conks also can develop after trees fall or die.

G. applanatum typically produces firm, flat, perennial, shelflike conks at the base of trees, often near old wounds, and usually close to the soil (fig. 39-4). Conks can vary in appearance, starting as a small white projection from bark. The upper surface is initially white, changing to gray, beige, or brown with age. The lower surface and conk margin remain white when growing, and the lower surface is covered with fine pores. When the conk is cut, distinct mottled brown annual tube layers (hymenial tissues) can be observed (fig. 39-5). Context tissues (non-hymenial tissues that compose fungal fruiting bodies) are brown. When conks or parts of conks die, the smooth upper and lower surfaces become dark gray to black and develop cracks. Conks can grow to a diameter of more than 16 inches, and both living and dead conks can persist for years.

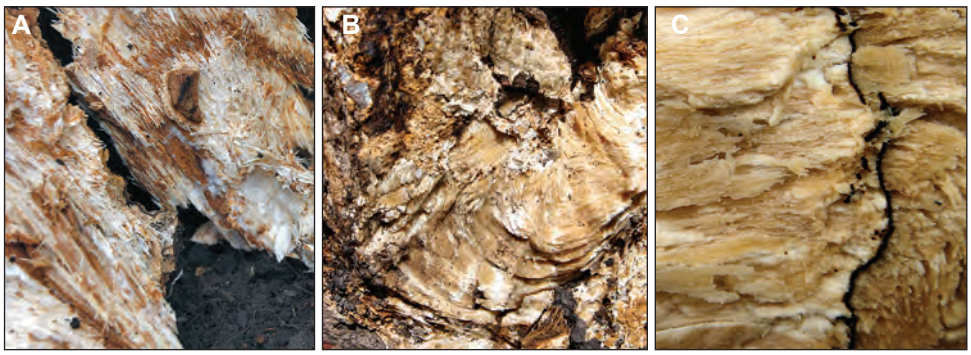

Figure 39-3—White mottled rot caused by *Ganoderma applanatum* (A-C), with (B, C) clear mottling, and (C) a zone line (right) (A: James T. Blodgett, U.S. Forest Service; B, C: James J. Worrall, U.S. Forest Service).

Figure 39-4—Conks of *Ganoderma applanatum*. They can vary in appearance, but have a white margin and underside when growing (James T. Blodgett, U.S. Forest Service).

Figure 39-5—*Ganoderma applanatum* produces perennial conks with mottled brown annual tube layers (hymenial tissues) and brown context tissues (James T. Blodgett, USDA Forest Service).

Disease Cycle

Infections occur from airborne spores or from contact with infected roots of neighboring trees. Conks can produce billions of airborne spores that are released from pores on the lower surface. Although not well understood, infections likely occur at wounds on roots or at the base of trees. Root-to-root spread can result in groups of infected trees called disease centers. In addition, the fungus can persist as a saprobe for several years after the host dies.

Damage

Ganoderma root rot affects trees used for many forest products and services including timber, recreation, and aesthetic value. It is more common in older trees and may be more prevalent on moist sites that are well suited for *Populus* species. Ganoderma root rot causes direct tree mortality, indirect tree mortality by breakage at the base or uprooting, growth reduction, wood loss from decay, and predisposition of trees to other disease and insect agents. Special consideration should be given when this disease occurs in developed sites because live, healthy-looking hosts with this disease frequently fall, which can endanger lives and property. Because the pathogen kills and decays roots, it likely reduces root suckering (vegetative formation of new stems from roots) and regeneration success.

Management

Once Ganoderma root rot is established in trees, practical methods for reducing this disease are unavailable. Because a relationship exists between wounds and decay, avoid wounds to reduce future infections. This disease is a major problem of aspen and other *Populus* species, and only a few other deciduous tree species in the Great Plains, so recognizing the problem and discriminating against susceptible host species is a practical management option. Species conversion could include complete removal of susceptible host tree species from an affected stand, or selective removal of aspen and other host species during thinning to favor nonhost species.

At developed sites with hosts, hazard tree inspectors should be trained to recognize the conks of *G. applanatum*. Conks are the only clear indicator of infection and extensive decay in live, standing trees. Fortunately, conks often develop before trees fall or die, providing an opportunity for removal of hazard trees. Species conversion away from hosts susceptible to Ganoderma root rot should be strongly encouraged on developed sites, especially if this disease is present.

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40. Nematodes of Broadleaf Trees

Timothy C. Todd and Jon A. Appel

Plant-parasitic nematodes have long been implicated as contributors to poor vigor and stunting of seedlings in forest-tree nurseries and decline of trees in windbreaks. These microscopic roundworms feed on root tissues by using a hollow needlelike mouthpart called a stylet (fig. 40-1). Although ubiquitous in agricultural and native soils, most plant-parasitic nematodes become economically important only at large population densities or when the vulnerability of their host is amplified by developmental stage (especially seedling), environmental stress, or opportunistic pathogens.

Figure 40-1— Anterior of an adult female dagger nematode, *Xiphinema americanum*, showing the elongated odontostyle (Tim Todd, Kansas State University, used with permission).

Hosts and Distribution

Information on nematode associations with specific tree species in the Great Plains is sparse. Windbreak surveys have been conducted in several states, but the presence of grasses and weed cover limit conclusions about host status in most cases. Nevertheless, surveys have shown that plant-parasitic nematodes are widespread in windbreaks in the Great Plains. Many of these species have broad host ranges, so it is likely that understory grasses and forbs contribute to conditions favorable for the buildup and maintenance of large population densities.

The dagger nematode *Xiphinema americanum* (fig. 40-1) is the most frequently encountered plant-parasitic nematode in Great Plains windbreak surveys (table 40-1). A survey of 76 South Dakota windbreaks and natural stands conducted during 1965–1967 recovered the dagger nematode from nearly 90 percent of samples; large populations (>100 individuals/100 cm^3 [6 cubic inches] soil) most often were associated with stands of eastern cottonwood (*Populus deltoides*), elm (*Ulmus* spp.), hackberry (*Celtis occidentalis*), and Russian-olive (*Elaeagnus angustifolia*). Dagger nematodes were less prevalent in a 2004-2006 survey[1] of 81 Kansas windbreaks (43 percent of total survey samples; table 40-1), but relatively high frequencies of occurrence (60-64 percent) were observed for green ash (*Fraxinus pennsylvanica*) and hackberry stands (table 40-2). Dagger nematode population densities exceeding 100 individuals/100 cm^3 soil most often were associated with these tree species.

[1] Data on file with the Kansas Department of Agriculture, Topeka, KS.

Table 40-1—Prevalence of nematode genera recovered from Great Plains windbreak surveys

Nematode common name (genus)	Survey (number of samples) *South Dakota (113)*	*Kansas (157)*
	- - - - - - - - - *Percent positive*- - - - - - -	
Dagger (*Xiphinema*)	88	43
Lance (*Hoplolaimus*)	33	<1
Pin (*Paratylenchus*)	75	54
Ring (*Mesocriconema*)	4	8
Root-lesion (*Pratylenchus*)	9	27
Spiral (*Helicotylenchus*)	45	58
Stunt (*Tylenchorhynchus*)	67	38
Stubby-root (*Paratrichodorus*)	7	4

Pin (*Paratylenchus* spp.), stunt (*Tylenchorhynchus* spp.), and spiral (*Helicotylenchus* spp.) nematodes also occur at high frequencies in Great Plains windbreaks (table 40-1). The largest population densities of spiral and pin nematodes in Kansas windbreaks are associated with hackberry and Russian-olive, respectively (table 40-2).

Plant-parasitic nematodes are widely distributed in forest nurseries as well and occasionally are associated with hardwood seedlings, particularly in the southern United States. Notable examples include the root-knot nematode *Meloidogyne incognita* on flowering dogwood (*Cornus florida*) and the needle nematode *Longidorus americanus* on white oak (*Quercus alba*). Although *L. americanus* damage has been observed only for pine (*Pinus* spp.) seedlings, several species of oak may serve as reservoir hosts, leading to severe stunting of loblolly pine (*P. taeda*) seedlings when grown in rotation.

Table 40-2—Prevalence and abundance of nematode genera recovered from four dominant tree species during a three-year survey of 81 Kansas windbreaks.

	Prevalence (percent)				Mean (maximum) number of nematodes/100 cm^3 soil			
Nematode common name (genus)	**Ash (n=15)**	**Elm (n=22)**	**Hackberry (n=11)**	**Russian olive (n=15)**	**Ash (n=15)**	**Elm (n=22)**	**Hackberry (n=11)**	**Russian olive (n=15)**
Dagger (*Xiphinema*)	60	27	64	47	17 (160)	9 (110)	25 (160)	13 (70)
Lance (*Hoplolaimus*)	0	5	0	0	0 (0)	1 (12)	0 (0)	0 (0)
Pin (*Paratylenchus*)	47	64	55	60	133 (900)	116 (930)	43 (320)	252 (2,210)
Ring (*Mesocriconema*)	7	5	27	0	3 (40)	0 (10)	87 (590)	0 (0)
Root-lesion (*Pratylenchus*)	20	14	18	47	17 (200)	3 (40)	4 (30)	18 (80)
Spiral (*Helicotylenchus*)	87	68	91	20	129 (860)	122 (750)	272 (1,300)	8 (60)
Stunt (*Tylenchorhynchus*)	40	50	55	33	7 (40)	14 (60)	38 (120)	45 (270)
Stubby-root (*Paratrichodorus*)	7	14	0	0	0 (4)	2 (30)	0 (0)	0 (0)

Symptoms and Signs

Visual symptoms of nematode damage rarely are diagnostic. Aboveground symptoms of nematode damage to tree roots mimic drought stress and nutrient deficiencies, and include stunting, chlorosis, and a general lack of vigor. These symptoms can be extreme in seedlings. Damage to mature trees, in contrast, often is manifested as a slow decline. Belowground symptoms frequently are manifested as reductions in numbers of lateral and feeder roots, and may include damage that is unique to the nematode species present, such as galling, necrotic cortical lesions, and root tip injury (for example, swelling and necrosis).

A nematode assay of soil and roots collected from the drip line provides the best indication of a nematode problem; however, diagnosis can remain problematic, even with information on types and numbers of plant-parasitic nematodes present in the soil and roots. For perennial hosts, the expression of symptoms related to nematode feeding often is dependent on their interaction with other factors such as environmental stresses and other disease-causing organisms.

Disease Cycle

The life cycle of plant-parasitic nematodes consists of four juvenile stages and an adult stage, each separated by a molt of the cuticle. Species reproduce by laying eggs, and are either sexual, with males and females common, or parthenogenetic, with males rare or absent. The length of life cycle differs with species, but typically requires approximately 1 month for completion.

Juveniles and adults generally are vermiform, but root-knot nematode adult females are swollen and pear shaped. Ectoparasitic species such as dagger, spiral, and stunt nematodes remain in the soil throughout their life cycle and feed by penetrating root cells with their stylet. In the case of the dagger nematode, an elongate stylet permits feeding on internal root tissues. Endoparasites such as root-knot and root-lesion nematodes burrow into roots and feed on cortical or vascular tissues. Root-lesion nematodes remain migratory, moving between roots and soil, whereas root-knot nematodes establish permanent feeding sites and become sedentary.

Damage

Nematode feeding damage varies according to feeding habit and the nature of the digestive enzymes produced but ultimately is associated with disruption of root function (water and nutrient absorption and translocation). Ectoparasitic species that feed on root surface tissues cause little visual damage except at very high population densities. Ectoparasites like dagger nematodes, which feed on internal root tissues, are capable of causing damage at lower population densities, particularly when feeding at or near root tips. Endoparasitic species cause extensive mechanical damage during their migration through root tissues, and often are implicated as partners in disease complexes.

The potential for damage by *X. americanum* has been demonstrated for inoculated greenhouse-grown cottonwood and green ash seedlings, and for nursery-grown golden willow (*Salix alba* 'Vitellina') and green ash transplants in fumigated versus nonfumigated field soil. Growth rates were reduced in the presence of the nematode, and root systems were devoid of feeder roots. Additional evidence for pathogenicity comes from windbreak surveys. Dagger nematode populations exceeding 100 individuals/100 cm^3 soil in South Dakota and Kansas windbreaks are frequently associated with poor tree health.

Other nematodes that may be associated with tree decline in Great Plains windbreaks include the root-lesion and spiral. Root-lesion nematode population densities were inversely related to tree health in the Kansas windbreak survey. Poor tree health was predicted as population densities approached 200 individuals/100 cm^3 soil, a level of nematode pressure that was observed only for green ash. Little additional information is available about the impact of root-lesion nematodes on green ash, and an assessment

of the relationship between tree health and nematode population densities within roots may be necessary for reliable estimates of potential damage. Evidence for a relationship between spiral nematodes and poor tree health in the Great Plains is likewise limited and restricted to hackberry. Spiral nematode population densities exceeding 1,000 individuals/100 cm^3 soil were associated with declining stands of this tree species in Kansas.

Despite numerous reports of the occurrence of plant-parasitic nematodes in forest nurseries, few examples of damage to broadleaf species have been documented. The most serious risks for broadleaf seedling damage in Great Plains nurseries appear to be associated with dagger and root-knot nematodes. In addition to damage due directly to feeding, dagger nematodes are known to transmit several virus diseases of broadleaf trees (for example, tobacco and tomato ringspot viruses (family Secoviridae).

Management

Chemical control of plant-parasitic nematodes in windbreaks is economically unviable. Weed control in windbreaks may limit nematode population increases while reducing competition for soil moisture, resulting in healthier trees that can better tolerate the damage caused by nematodes. Nematode management in orchards, for instance, has shifted in recent years toward stress avoidance by using cultural practices such as irrigation and fertility maintenance that promote better root development.

Soil fumigation is a viable option for nematode management in nurseries, and most alternatives to methyl bromide provide adequate control. An integrated approach often is necessary, however, owing to the high cost of fumigation and the ability of nematode populations to rebound after treatment. Combining soil fumigation with practices such as crop rotation with nonhost cover crops and field fallowing can be an effective strategy for suppressing nematode populations in forest nurseries. The choice of cover crop depends on the target nematode species, and generalizations are difficult. Pearl millet (*Pennisetum glaucum*), for which hybrids with resistance to several nematode species have been developed, is considered to be one of the best options for nematode control.

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41. Brown Spot of Pine

Judith O'Mara and Aaron A. Bergdahl

In the Great Plains, brown spot can be a serious problem in Scots pine (*Pinus sylvestris*) plantings. It is easily confused with another important pine disease, Dothistroma needle blight. Brown spot is also damaging on young longleaf pine (*P. palustris*) trees in the southern United States. The disease is also referred to as brown spot needle blight.

Hosts and Distribution

Brown spot is primarily an issue on Scots pines in windbreaks and Christmas tree plantations in the Great Plains. It is occasionally seen on ponderosa (*P. ponderosa*), mugo (*P. mugo*), and white pine (*P. strobus*) as well. Brown spot also occurs northward into Manitoba and Ontario. In the southeastern United States, brown spot can be a serious problem on longleaf pine.

Symptoms and Signs

Initial symptoms for brown spot are yellow spots that turn reddish brown and eventually girdle the pine needle. These dark bands are surrounded by a yellow halo and frequently develop droplets of dried resin. Needle tissue beyond the band turns brown, producing a half-needle scorch (fig. 41-1). Infected needles eventually turn brown and drop off the tree. Heavy disease pressure can result in extensive needle scorch and branch dieback in the lower portion of the tree (fig. 41-2).

Figure 41-1—Brown spot needle banding and half-needle scorch (Jill O'Donnell, Michigan State University, used with permission).

Figure 41-2—Brown spot damage on Scots pine Christmas trees (Jill O'Donnell, Michigan State University, used with permission).

Figure 41-3—Fruiting bodies (acervuli) on pine needles (Isabel Munck, U.S. Forest Service).

Brown spot is caused by the fungus *Mycosphaerella dearnessii* (syn. *Eruptio acicola* and *Scirrhia acicola*). In the Great Plains, the conidial or asexual state of the fungus (*Lecanosticta acicola*) is found to be associated with the disease. In the southeastern United States, both stages of the fungus can be found on longleaf pine throughout the year. *L. acicola* spores (conidia) form in acervuli that erupt through dead needle tissue (fig. 41-3). The conidia are cylindrical (15 to 35 µm × 3 to 4 µm), multicelled, curved, and olive colored. In culture, the fungus grows slowly and produces a dark olive-green, gelatinous colony (fig. 41-4).

Brown spot symptoms can look similar to another pine disease, Dothistroma needle blight, caused by *D. septosporum* (also referred to in earlier literature as *Mycosphaerella pini* and *Schirria pini*) and *D. pini*, for which no teleomorph has been found (see chapter 43).

The conidia for both brown spot and *Dothistroma* needle blight are similar, the primary difference being that the conidia for *Dothistroma* are colorless. Scots pine is not susceptible to Dothistroma needle blight; ponderosa and mugo pines are susceptible to both diseases.

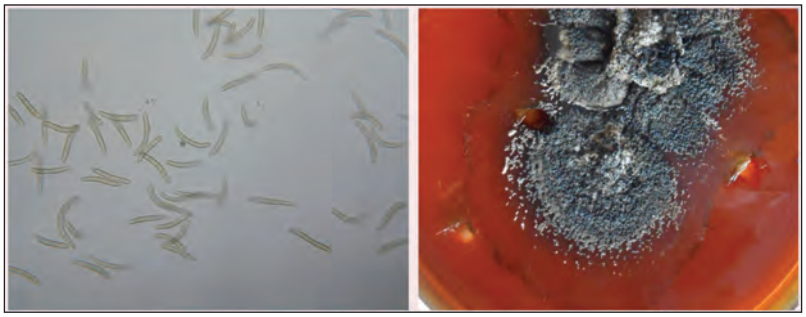

Figure 41-4—Left) Spores (conidia) of *Lecanosticta acicola* (Isabel Munck, U.S. Forest Service); (Right) *Lecanosticta acicola* in culture on malt agar (Kirk Broders, University of New Hampshire, used with permission).

Disease Cycle

In the Great Plains, brown spot overwinters as acervuli on dead needles attached to the tree or on the ground. Spore dispersal and infection takes place in late spring to early summer of the following year (fig. 41-5). Spread of the disease is through rainsplash, insects, and shearing tools. New needle growth is more susceptible to infection than older needles. In the central Great Plains symptoms start developing in late summer and continue through the fall.

Brown spot is favored by warm, wet conditions. In southern states, longleaf pine infection occurs over a range of temperatures, but daytime temperatures of 86 °F and nighttime temperatures of 70 °F are optimal. Similar conditions for infection could be expected for pine species growing in the Great Plains. Infection is also favored by prolonged periods of needle wetness. Crowded plantings, tall grass, or heavy weed pressure can lead to poor air circulation, favoring conditions for disease development.

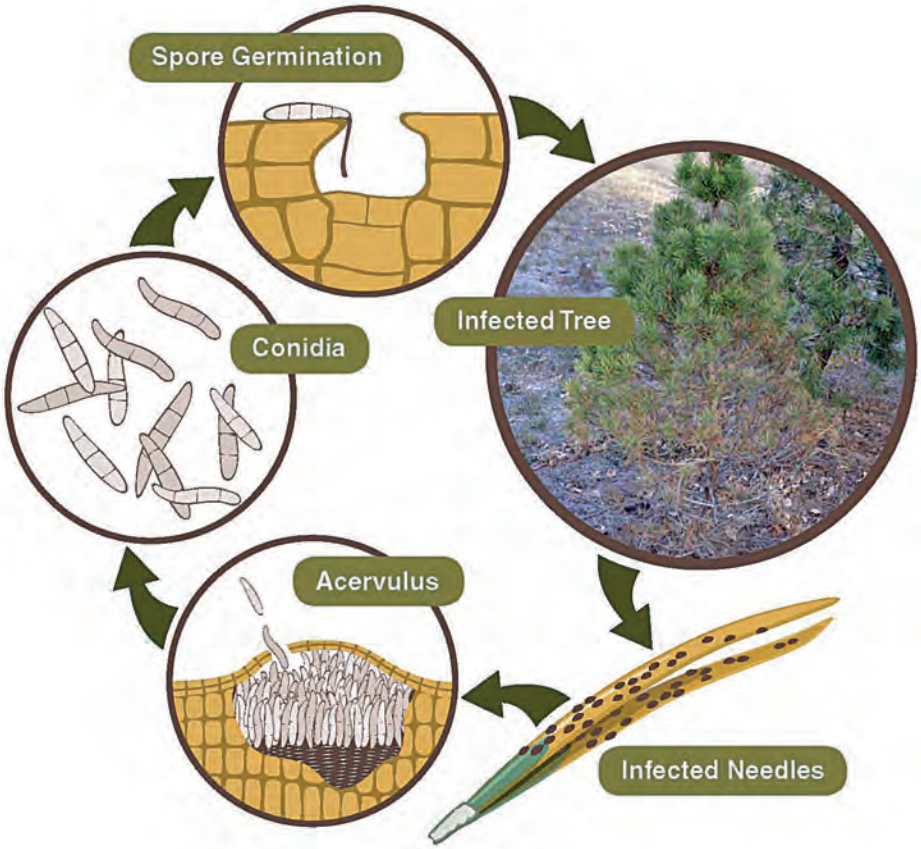

Figure 41-5—Disease cycle on Scots pine (David Haasser, North Dakota State University, used with permission, Adapted from Riffle and Peterson, 1986).

Damage

Occurrence of brown spot on Scots pine has been reduced in the southern Great Plains because of heavy disease pressure from pine wilt, which has destroyed many Scots pine plantings. Although it is less of a problem in landscape plantings, brown spot is still a serious problem in Christmas tree plantations where Scots pine is a staple species. The biggest challenge for Christmas tree growers is that the needle symptoms start to become apparent early in the fall before the harvest period for the Christmas season. In some cases, growers apply green paint to avoid a loss in tree sales during the holiday season.

Management

Management for brown spot needle blight starts with selection. Pine plantings should not be established with only a single species or variety of pine. If planting Scots pine, keep in mind that short-needled Scots pines are more susceptible to brown spot than long-needled varieties. Prolonged leaf wetness can lead to greater disease severity, so improving airflow can help dry out the foliage and reduce disease pressure. For better airflow, use proper plant spacing, control weeds, and keep grass alleyways mowed. Removal of pine needles from around the tree may also help to reduce the amount of disease inoculum that overwinters. In Christmas tree plantations avoid shearing during wet conditions, as the disease can be spread on the shears.

During wet years, crowded plantings may require the use of one to two fungicide applications. Make the first application when needles are one-half their full length. A second application may be necessary three to four weeks later depending on the residual period of the fungicide selected.

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42. Cercospora Blight of Junipers

Glenn W. Peterson and David S. Wysong (Riffle and Peterson 1986)
(updated by Ned Tisserat)

Junipers (*Juniperus* spp.) and other members of the Cupressaceae family are infected by two closely related needle-blighting fungi, *Pseudocercospora juniperi* and *Passalora sequoiae.*

Hosts and Distribution

Cercospora blight, caused by the fungus *P. juniperi* (formerly *Cercospora sequoiae* var. *juniperi*), is a widespread needle disease of junipers in the eastern Great Plains. It is particularly common in the eastern third of Kansas and Nebraska, where relatively high summer humidity and frequent rains favor disease development. The disease is also common in the eastern and central United States. Rocky Mountain juniper (*J. scopulorum*) and its varieties are particularly susceptible to the disease. Eastern redcedar (*J. virginiana*) is less susceptible and Chinese juniper (*J. chinensis*) varieties are very resistant.

A second fungus, *P. sequoiae* (formerly *C. sequoiae*), also causes a disease referred to as Cercospora blight in Arizona cypress (*Cupressus arizonica*), oriental arborvitae (*Platycladus orientalis*), and Chinese juniper and is found throughout the southeastern United States (including Texas and perhaps southern Oklahoma in the Great Plains) and from Pennsylvania to eastern Nebraska.

Figure 42-1—Damage to a Rocky Mountain juniper windbreak. Note thin foliage in the lower half of the tree crown (Mark O. Harrell, Nebraska Forest Service, used with permission).

Symptoms and Signs

Symptoms of Cercospora needle blight first appear in late summer and fall on older, inner spur or scale needles located nearest the main tree trunk (fig. 42-1). Infected needles initially are bronze but later turn more dull red and drop from trees in October and November (earlier in the northern Great Plains). Commonly, all leaves of a branchlet are affected. Defoliation in succeeding years continues from the inner portion of the branch toward the tip, and from the bottom of the tree toward the top. Severely infected trees are open and spindly and may appear as if they had been scorched by a fire. Symptoms caused by *P. sequoiae* on Arizona and Leyland (x *Hesperotropsis leylandii*) cypress are similar.

P. juniperi forms small (140 µm diameter) fuzzy or hairy cushion-shaped fruiting bodies (sporodochia), easily visible with a hand lens, on dead needles (fig. 42-2). These sporodochia produce elongate,

Figure 42-2—Fruiting bodies of *Pseudocercospora juniperi* showing fuzzy gray appearance of spores (conidia and conidiophores) (Glenn Peterson, USDA Forest Service, retired).

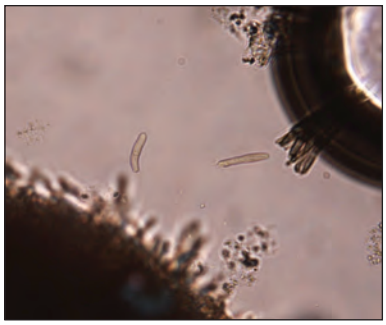

Figure 42-3—Spores (conidia) of *Pseudocercospora juniperi* (James A. Walla, Northern Tree Specialties, used with permission).

multiseptate, light brown spores (conidia) measuring 42 to 57 µm × 2.5 to 3.0 µm (fig. 42-3). The sporodochia of *P. sequoiae* are 50 µm in diameter and also produce multiseptate, light brown conidia (30 to 60 µm × 5.0 to 5.5 µm) that are more tapered than *P. juniperi*.

Cercospora blight is readily distinguished from Phomopsis and Kabatina blights of junipers. The branches of Cercospora-infected trees usually will be devoid of foliage near their bases but will have healthy foliage on their tips, whereas branches of trees infected with Phomopsis and Kabatina blight will have dead needles at branch tips and healthy foliage near the branch base.

Disease Cycle

Spores of *P. juniperi* and *P. sequoiae* are dispersed by rainsplash from late April through October. There is little or no long-distance wind dispersal of spores; almost all spores are deposited within a few feet of the initial site of infection. Infection is more severe when precipitation during the growing season is above average. The period between initial infection and first appearance of symptoms is from two to three weeks.

Damage

Cercospora blight is more destructive than Kabatina or Phomopsis blights in established Rocky Mountain juniper plantings. Repeated defoliation weakens trees, reduces the functional and aesthetic value of windbreaks, and can result in tree mortality.

Management

Rocky Mountain juniper should not be planted for windbreaks in much of the eastern Great Plains (east of longitude 98 degrees west) and where Cercospora blight is a problem because the disease is simply too difficult to manage in tightly spaced plantings. Instead, select eastern redcedar or Chinese junipers that have good resistance to this disease. Cercospora blight is less of a problem on Rocky Mountain juniper in landscape plantings. Nevertheless, the disease may occasionally develop. Proper tree spacing, which promotes good air movement and rapid drying of foliage, will inhibit blight development of both pathogens.

Chemical control may be necessary during wet summers. Although symptoms appear in late summer to fall, fungal infection of needles actually occurs in summer. At least two fungicide applications (copper-based products and other fungicide types) in early June (earlier for Passalora-caused blight) and again in early July are needed. A third application in mid- to late July may be necessary during wet summers. Fungicide applications at other times are ineffective. The foliage must be thoroughly covered, especially the lower two-thirds of the tree crown. Yearly fungicide applications may not be needed once the disease is controlled. However, carefully inspect trees yearly to monitor for recurrence.

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43. Dothistroma Needle Blight of Pines

Aaron D. Bergdahl and Judith O'Mara; revised from Glenn W. Peterson and David S. Wysong (Riffle and Peterson 1986)

Dothistroma needle blight (DNB) is a common and serious fungal disease of native and exotic pines in windbreaks, Christmas tree plantations, and ornamental plantings throughout the Great Plains. This chapter uses fungal nomenclature for the pathogens causing DNB based on recent molecular work. Earlier work with these pathogens has not been based on these methods and should be interpreted with this in mind. There has been limited confirmation of DNB species by using molecular methods in North America.

Hosts and Distribution

Of the commonly planted pine species in the Great Plains, primary hosts of DNB are Austrian (*Pinus nigra*), ponderosa (*P. ponderosa*), and mugo (*P. mugo*) pines. Scots pine (*P. sylvestris*) is highly resistant to DNB. *Dothistroma* species have been recorded on more than 35 pine species and hybrids in the United States. *D. pini*, which is thought to be the most prevalent species causing DNB in the Great Plains, has been confirmed on whitebark (*P. albicaulis*), Swiss stone (*P. cembra*), limber (*P. flexilis*), Austrian, and ponderosa pines. *D. septosporum* (also referred to in earlier literature as *Mycosphaerella pini* and *Schirria pini*) is found in states neighboring the Great Plains, and thus it is not unreasonable to suspect that it also occurs in the Great Plains.

Symptoms and Signs

Early symptoms of infection, seen in early fall, are resin-soaked, dark green bands and dark green, yellow, and tan spots on needles (fig. 43-1, top). Within one to two weeks, the bands and spots turn brown to reddish brown (fig. 43-1, middle). In the Great Plains, *D. pini* does not develop bands that are as numerous and as red as those on pines west of the Rocky Mountains, where the disease is referred to as red band disease and is likely caused by *D. septosporum*. On the most susceptible trees, the ends of the needles distal to the bands may die (fig. 43-2) during the fall after infection. On others, the needle ends

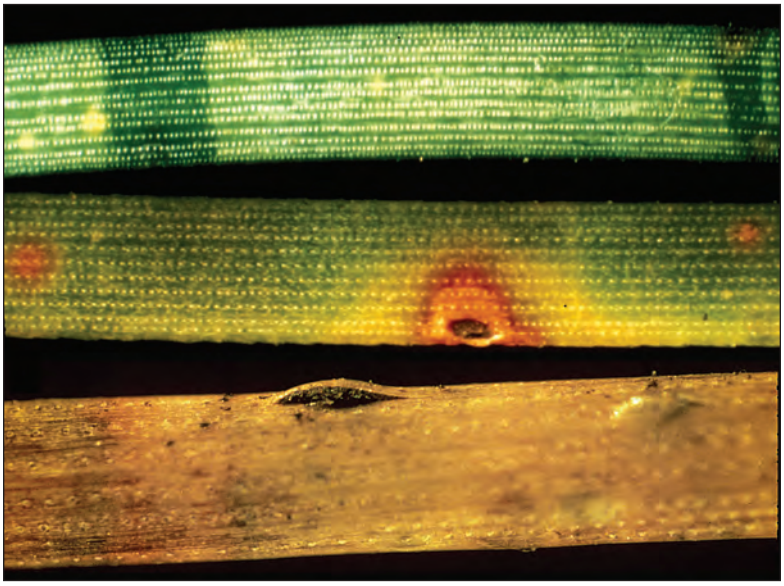

Figure 43-1—Symptoms of infection by *D. pini:* (Top) Dark green banding, an early symptom of infection; (Middle) Reddish spotting at the point of infection as symptoms develop in the late season; (Bottom) A fruiting structure (acervulus) erupting through the epidermis (Glenn Peterson, USDA Forest Service, retired).

Figure 43-2—Second-year ponderosa pine needles with Dothistroma needle blight symptoms (banding, partial scorch) on a tree from a site where only *D. pini* was confirmed by molecular methods (James A. Walla, Northern Tree Specialties, used with permission).

Figure 43-3—Ponderosa pine with severe Dothistroma needle blight symptoms starting in lower and interior portions of the crown leading to tip dieback, needle loss, and a sparse lower crown. Molecular analysis of isolates from multiple trees in the planting found only *D. pini* (James A. Walla, Northern Tree Specialties, used with permission).

may die the next season or in some cases may not die. Within one year after showing dieback, the whole needle typically dies and is shed. Symptoms on most trees occur in the lower portion of the tree on the interior needles (fig. 43-3). Heavily infected trees are often highly susceptible genotypes or trees grown in tight spacing and with poor air circulation.

Disease Cycle

Stromatic acervuli, in which asexual spores (conidia) are formed, develop first near the original infection site, but may later form in any portion of discolored needles. In Nebraska, some acervuli develop and mature sufficiently in the fall after infection to raise and split the needle epidermis (fig. 43-1, bottom), and more become visible the next spring. Acervuli develop and mature earlier in the southern Great Plains, allowing for same-season infection if adequate moisture occurs in the fall. Acervuli develop more slowly and are not visible in the fall of the infection year in the northern Great Plains. A flap of tissue usually covers acervuli as they rupture the epidermis of the needle. Conidia of *D. pini* are three- to five-septate and on average 30.0 µm × 3.5 µm *in vivo*. Conidia of *D. septosporum* are three- to five-septate and on average 28 µm × 2 µm *in vivo*. Owing to a large overlap in spore sizes, this characteristic is not sufficient for differentiation between *D. pini* and *D. septosporum*.

The conidia (fig. 43-4), which are exposed as the epidermis is raised, are dispersed by rainsplash impacting acervuli any time during the growing season. New infections can occur on any age-class of susceptible needles during periods of wet weather or high relative humidity from May to October.

Two growing seasons are required for completion of the life cycle during most years in most areas of the United States (in California and Oregon, what is likely *D. septosporum,* may complete a cycle in one year). In North America, the sexual stage of what is likely *D. septosporum*, has been found only in Alaska, British Columbia, California, and Oregon. The stromata of the sexual stage produce ascospores, whose role in the development of epidemics is not known. In contrast, much is known about the role of conidia in disease development.

growth in late June or July has been used effectively by Christmas tree growers in the central United States. There is some risk in the single fungicide application procedure in southerly regions because infection could occur in previous years' needles prior to the single early-season fungicide application.

If substantial infection occurs during a year in which fungicide has not been applied, a well-timed fungicide application the next year will provide good control. If little or no infection occurs in the year fungicide was not applied, spraying can be skipped for another year. Those managing high-value trees are advised to spray annually if DNB is present in the area. However, annual spraying may not be necessary in park, residential, and similar types of plantings where disease pressure may be lower and exist at a tolerable level.

Removing and disposing of needles from underneath trees will have little effect on DNB because infected needles persist on the tree, many shed needles get caught in the tree, and most infections occur in spring before needles dispersing inoculum are shed in the Great Plains.

Work by Peterson and others in Nebraska in the 1970s and 1980s identified foreign and domestic resistant seed sources of Austrian and ponderosa pines. Needles of all ages were highly resistant in some of these seed sources, as compared to standard seed sources in which current-year needles become susceptible in July and older needles are susceptible. These tree improvement efforts were completed, although the extent to which trees from these sources exist in the Great Plains is unknown. Nurseries in the Great Plains that previously propagated the resistant seed sources were not doing so in 2010.

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44. Kabatina Blight of Juniper

Andrea Ostrofsky and Glenn W. Peterson (Riffle and Peterson 1986)
(updated by Ned Tisserat)

Eastern redcedar (*Juniperus virginiana*) and Rocky Mountain juniper (*J. scopulorum*), native to the Great Plains, are important trees in windbreak, wildlife, and landscape plantings. Branch tips of both species have been damaged by the fungus *Kabatina juniperi*.

Hosts and Distribution

Blight caused by the fungus *K. juniperi* is widespread in North America and especially in windbreak and ornamental plantings of eastern redcedar and Rocky Mountain juniper in the Great Plains. The fungus may also be found in ornamental plantings of Chinese (*J. chinensis*), savin (*J. sabina*), and creeping (*J. horizontalis*) junipers.

Symptoms and Signs

Kabatina blight symptoms typically appear in late winter or early spring a few weeks before new growth resumes and when foliage changes from its dull green winter coloration to a darker green color of summer. The terminal two to six inches of diseased branches scattered throughout the tree or shrub first turn dull green, then red or yellow (fig. 44-1). Small ash-gray to silver lesions dotted with small black fruiting bodies (acervuli) of the fungus are visible at the base of the discolored tissue (fig. 44-2). The brown, desiccated twigs eventually break off at the lesion, usually shortly after new growth resumes but sometimes up to a year after discoloration. Blighting is restricted to the branch tips and usually does not cause extensive branch dieback or tree death except in highly susceptible plants in settings that favor severe disease development. Nevertheless, yearly tip dieback may result in abnormal bunching of the foliage (witches'-broom) and stunting of young trees or shrubs.

Figure 44-1—Branches of Rocky Mountain juniper infected with *Kabatina juniperi*. Note the discoloration and dieback of the branch tips (Ned Tisserat, Colorado State University, used with permission).

Figure 44-2—Lesion with black fruiting bodies (acervuli) on branchlet of Rocky Mountain juniper (Ned Tisserat, Colorado State University, used with permission).

The black, rounded to oval fungal acervuli, visible with a hand lens, develop in the epidermis beginning a few weeks after symptoms appear in spring and become more numerous the next month. Their numbers then decrease throughout the summer partly because some of the dead foliage is shed. The colorless (hyaline), unicellular, ellipsoid spores (conidia) measuring 4.5 to 8.0 µm × 2.3 to 3.0 µm are produced successively at the tips of tapered spore-bearing cells that cover the surface of the acervulus, giving it a granular appearance (figs. 44-3 and 44-4).

Figure 44-3—Fruiting bodies (acervuli) of *Kabatina juniperi* that have broken through the host epidermis. Note the granular appearance of the surface (James A. Walla, Northern Tree Specialties, used with permission).

A second fungus, *Sydowia polyspora* (anamorph *Sclerophoma pithyophila*), causes similar symptoms at about the same time of year on junipers in other regions of North America and also occurs on several other conifers. A third fungus,

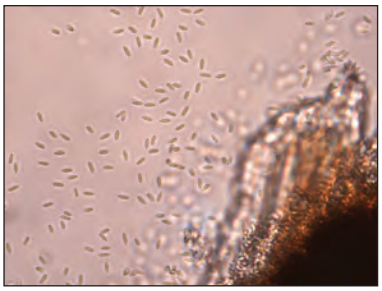

Figure 44-4—One-celled spores of *Kabatina juniperi*, which form on the surface of the fruiting body (acervulus) (James A. Walla, Northern Tree Specialties, used with permission).

Phomopsis juniperovora, also causes twig blighting although symptoms of this disease are most apparent in summer and may continue through the fall. Unlike Phomopsis blight, foliar blighting caused by *K. juniperi* occurs only before new growth appears and does not continue to progress through the summer. Microscopic identification is necessary to differentiate *K. juniperi*, *S. pithyophila*, and *P. juniperovora*. Conidia of *K. juniper*i and *S. pithyophila* are similar to alpha conidia of *P. juniperovora*, but they do not contain refractive oil droplets. Conidia of *S. pithyophila* and *K. juniperi* are similar in size; however, those of *S. pithyophila* develop in pycnidia, whereas those of *K. juniperi* develop in acervuli. The prevalence of *S. pithyophila* in the Great Plains has not been determined, although if present it likely is not as widespread or common as *K. juniperi*.

Disease Cycle

The disease cycle of Kabatina blight is not completely understood. The fungus apparently enters plants through wounds. Many insects, including larvae of species of juniper webworm (*Dichomeris* sp.) and juniper midge (*Contarinia* sp.), are known to feed on juniper foliage and twiglets. Lesions of Kabatina blight frequently develop at these feeding sites. Because disease symptoms develop in late winter or spring as the foliage color changes, it is likely infection of wounded tissue occurs sometime relatively late in the previous growing season or perhaps after freeze damage in late fall. However, the precise period of infection is not known. The fungus, which can survive cold temperatures, probably overwinters within the branchlets.

Damage and Management

Kabatina blight rarely causes serious damage to mature trees in windbreak or farmstead plantings and management is not required. Control in landscape plantings may be desirable for aesthetic reasons, but few management practices have been developed. Control of wounding agents such as insects may reduce infection levels. Pruning and destruction of infected branchlets in late summer may reduce levels of inoculum, but could result in more wounded tissue that is susceptible to infection. The unsightly appearance of infected trees is often improved when lower branches elongate and the dead branch tips fall from the tree. Rocky Mountain juniper varieties are more susceptible to Kabatina blight damage than eastern redcedar. Chinese juniper is very resistant to the disease. Many juniper cultivars are reported to have good resistance; plant them in high-risk landscape areas.

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45. Lirula Needle Blight of Spruce

James A. Walla

The fungus *Lirula macrospora* causes needle blight of spruce (*Picea* spp.) across the Northern Hemisphere. As currently described, it likely consists of a complex of several species, with significant variation in morphology, regional occurrence, hosts, and life cycles. Damage caused by and management of the individual components of the *L. macrospora* species complex are similar if differences in length of the life cycle are taken into account.

Hosts and Distribution

In North America, *L. macrospora* has been reported on the native black (*P. mariana*), blue (*P. pungens*), Engelmann (*P. engelmannii*), red (*P. rubens*), Sitka (*P. sitchensis*), and white (*P. glauca*) spruce and the exotic Norway (*P. abies*) spruce. These represent the majority of diversity among spruce taxa. In the Great Plains, it has been found on white spruce (primarily Black Hills seed sources) and blue spruce.

L. macrospora has been found in all Canadian Provinces and Territories and at least 14 States, including the Great Plains states of North Dakota, South Dakota, Wyoming, and Colorado. It occurs sporadically, with incidence typically at only a small portion of sites, but with wide variation in incidence and severity within sites and over time. The disease is most common in areas with relatively cool temperatures and greater precipitation.

Symptoms and Signs

Only one component of the *L. macrospora* complex (Taxon A in Walla [1998]) has been found in the Great Plains. The remainder of this chapter relates specifically to that component.

The first symptom (yellow bands) appears on needles about 14 months after infection (August to October in North Dakota). The bands gradually become light purplish brown and expand over the entire needle by about 22 months after infection (April to May the next spring in North Dakota). The needles become reddish brown the same spring (fig. 45-1), and, by then, a characteristic black basal band has developed in the abscission layer at the base of the needle (fig. 45-2). Inconspicuous asexual spore-producing structures (pycnidia) develop on third-year needles about 22 months after infection (April in North Dakota). Large, black, smooth sexual spore-producing structures (hysterothecia) develop mostly on the lower surfaces (fig. 45-3) along the length of the needles about 23 months after infection, within one month after pycnidia develop. Hysterothecia are easily visible except when they are just appearing. They are nearly as wide as the needle surface and can be various lengths, ranging up to the entire length of the needle (figs. 45-2 and 45-3). Additional black bands develop around some needles (figs. 45-2 and 45-3) when hysterothecia appear. Infected needles slowly fade to tan (fig. 45-1) by about 34 months after infection. When mature, hysterothecia have a lengthwise slit, which

Figure 45-1—Discolored needles caused by *Lirula macrospora*. (A) Symptoms in spring: light infection of second-year needles, mixed infection of third-year needles, gray fourth-year needles with some broken; (B) Symptoms in fall: new symptoms on second-year needles, light brown discoloration of third-year needles (James A. Walla, Northern Tree Specialties, used with permission).

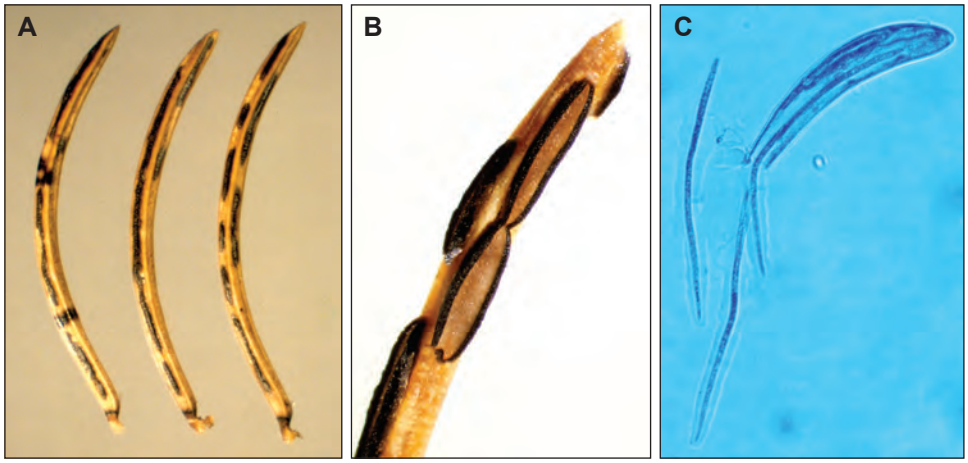

Figure 45-2—Mature *Lirula macrospora* fruiting bodies (hysterothecia), bands, asci, and spores: (A) Closed hysterothecia along length of needles, bands around needles, and basal bands; (B) Open hysterothecia, exposing ascus-bearing surface; (C) Ascus and ascospores stained in aniline-blue (James A. Walla, Northern Tree Specialties, used with permission).

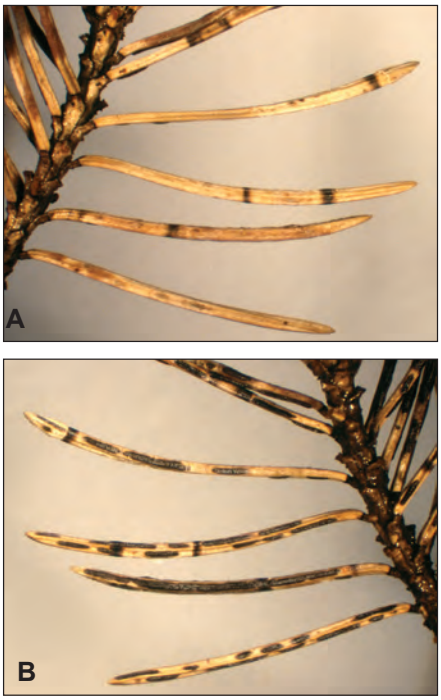

Figure 45-3—Third-year symptoms and signs of *Lirula macrospora* on (A) upper, and (B) lower surfaces of the same needles. Black bands are visible on both surfaces. On these needles, fruiting bodies (hysterothecia) are visible only on the lower surfaces (James A. Walla, Northern Tree Specialties, used with permission).

opens (fig. 45-2B) to release ascospores (115 to 160 µm × 2.5 to 3.5 µm) when the needles are wet. About 40 months after infection (September to October in North Dakota), the needles with mature hysterothecia shrivel and turn gray, but remain attached to branches until they are weathered off, typically another one or more years (fig. 45-1). The same three-year pattern of symptom and sign development occurs if older needles are infected, potentially resulting in a mixture of symptoms and signs on a single age-class of needles (fig. 45-1).

Symptoms, signs, and damage caused by *Lirula brevispora* and *Isthmiella crepidiformis* are similar to those caused by *L. macrospora* types that have a two-year life cycle. *L. macrospora* has often been misdiagnosed as *Lophodermium piceae* despite the distinctive symptoms, signs, and damage.

Disease Cycle

Lirula needle blight in North Dakota consistently has a three-year life cycle, as did samples observed from Manitoba, Minnesota, and South Dakota. Most infection occurs in the two months after budbreak. First symptoms develop about 14 months after infection. Pycnidia develop about 22 months after infection, and likely have an outcrossing role. Hysterothecia develop about 23 months after infection. Hysterothecia mature about one year after they first appear (three years after infection). Ascospore release from mature hysterothecia usually occurs during weeks with rain from just before bud-break through summer (late May through early September in North Dakota), with the greatest releases from June into August. The ascospores cause new infections to complete the disease cycle.

Figure 45-4—Heavy *Lirula macrospora* infection causing dirty-green appearance in (A) center tree, and (B) tree on right (James A. Walla, Northern Tree Specialties, used with permission).

Damage

Damage by Lirula needle blight results from needle discoloration (aesthetic damage) and the loss of functioning needles (growth loss, death of branches). Trees with severe infection for a few years will exhibit a gradual reduction in length of new shoots throughout the crown, but especially on the most seriously affected branches, and if severe enough, those branches will stop growing and die. Because infected needles are not cast, severely infected trees appear to have a dirty green appearance from a distance (fig. 45-4), rather than the sparse appearance caused by needle casts. Long-term damage occurs only if lower branches were killed or if reduced growth allows competing trees to gain an advantage. Even when left unmanaged, Lirula needle blight is often not severe at sites in the Great Plains for more than a few years; reasons remain unclear.

Management

Lirula needle blight is poorly dispersed among sites, so great emphasis should be placed on avoiding introduction on transplants. Spruce should not be transplanted to a site where Lirula needle blight already occurs. There are varying levels of disease resistance among trees within species. Where some trees are severely infected, consider removing those trees to reduce disease pressure on neighboring spruce trees. In mixed plantings, favor retention of blue spruce over white spruce. Cultural actions that reduce the length of time that moisture remains on needles and that increase the distance between spruce trees will reduce potential disease severity. Until severe infection has occurred at least two years in a row, wait to see if it will subside on its own. If the disease has been severe (greater than 50 percent of two needle age-classes infected) for more than two years, fungicide applications would be appropriate. Protection of needles for two months after budbreak in each of three consecutive years readily controls damage from *Lirula*. No fungicides are labeled specifically for Lirula needle blight. Some products containing azoxystrobin, chlorothalonil, copper hydroxide, copper salts of fatty and resin acids, copper sulfate, mancozeb, mono- and di-potassium salts of phosphoric acid, pyraclostrobin, sulfur, and thiophanate-methyl are labeled for use on spruce and have been found to control some foliar diseases. For unknown reasons, application of chlorothalonil in late July, well after initial spore release, provided the same excellent protection as multiple applications per season.

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46. Cyclaneusma Needle Cast of Pines

James A. Walla; revised from Glenn W. Peterson and James A. Walla (Riffle and Peterson 1986)

Needle cast caused by *Cyclaneusma minus* (syn. *Naemacyclus minor*) is commonly present on pines (*Pinus* spp.) and occasionally damages Scots pines (*P. sylvestris*) in young plantings in the Great Plains. A similar species, *C. niveum*, is present in North America, but has not caused damage.

Hosts and Distribution

Cyclaneusma minus infects many pine species, including Scots, Austrian (*P. nigra*), mugo (*P. mugo*), and ponderosa (*P. ponderosa*) pines. It occurs throughout the Northern Hemisphere. Most Scots pine plantings in North Dakota are infected, as well as young plantings in South Dakota, Nebraska, Kansas, and likely Colorado. It also occurs on mugo and ponderosa pine in the Great Plains.

Symptoms and Signs

In the eastern and central United States, where much research on *C. minus* has been done, infected needles typically become necrotic from September to November of their second growing season, with some infected needles becoming necrotic the next spring, summer, or fall. In the northern Great Plains, symptoms typically develop in September to October on third-year or older needles (fig. 46-1). The first symptoms are small light-green spots which gradually lighten and coalesce, turning the entire needle a characteristic dusty yellow or tan (fig. 46-2). Distinct transverse brown bars often occur on necrotic Scots pine needles. Needles are often cast two to three months after becoming fully discolored, but may be retained for several more months. Fruiting bodies (apothecia) 0.2 to 0.6 mm in length develop on necrotic needles (fig. 46-3A). Portions of needle tissue with apothecia will become lighter than surrounding tissue with age. Apothecia have slightly raised needle tissue over them, and are initially inconspicuous when closed, but the covers become lighter tan than the rest of the needle with age and are often conspicuous on recently cast needles and on damp needles. Apothecia are distinctive when they swell during wet weather and the covers swing open to reveal a dull cream to yellow hymenium (fig. 46-3B). Ascospores are clear (hyaline), two-septate, filiform, 65 to 100 µm long, and usually slightly bent like a boomerang.

Figure 46-1—Scots pine with third-year needles discolored by Cyclaneusma needle cast (James A. Walla, Northern Tree Specialties, used with permission).

Symptoms of this disease may be confused with natural and stress-related needle senescence or damage caused by aphids. The timing of *C. minus* symptom development and natural needle senescence overlaps, so needle symptoms must be used to

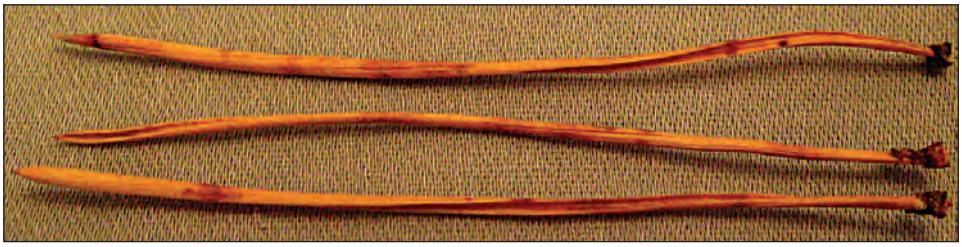

Figure 46-2—Cyclaneusma needle cast symptoms on Scots pine needles, showing tan needles with brown bars (James A. Walla, Northern Tree Specialties, used with permission).

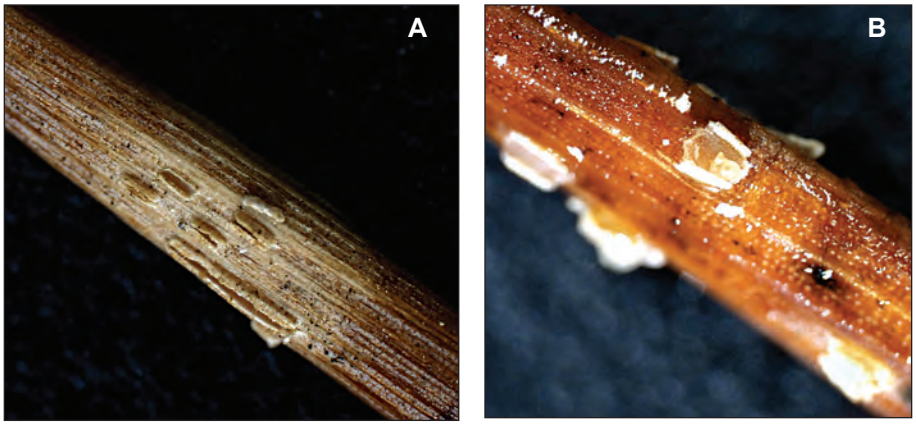

Figure 46-3—*Cyclaneusma minus* fruiting bodies (apothecia) on Scots pine needles: (A) Closed apothecia on light tan portion of a needle; and (B) Open apothecia on moist needle (James A. Walla, Northern Tree Specialties, used with permission).

differentiate the cause of necrosis. Naturally senescing Scots pine needles are bright yellow, and aphid damage may cause brown bars to occur on these needles. The presence of brown bars on dusty yellow or tan needles and presence of apothecia are indicative of Cyclaneusma needle cast.

Disease Cycle

Spores are released and infection can occur throughout the year when temperatures are above freezing. Heavy infection can occur whenever spores are released, but tends to be greatest in spring before budbreak. All ages of needles are susceptible, beginning as soon as needles emerge from the fascicle sheaths. Older needles on susceptible trees develop symptoms as soon as two months after infection, and symptoms develop as soon as 15 months after infection of current-year needles. Infected needles of less susceptible trees remain symptomless for 12 to 24 months longer. Apothecia develop on attached or cast needles one to five months after first symptoms appear. They open when the needles are wet, and spores are dispersed from them by wind. The disease should be managed as if it has a two-year life cycle, although time from infection to spore production on older needles can be less than one year.

Damage

This fungus has caused extensive early yellowing or defoliation of Scots pine Christmas tree plantings in some eastern and central States and up to 60-percent volume reduction in radiata pine (*P. radiata*) plantings in New Zealand. Serious damage occurs only occasionally in some settings in the Great Plains, such as those with highly susceptible seed sources, monocultures, or favorable weather. Retention of two age-classes of green needles is considered sufficient for Christmas tree plantations in the eastern and

central United States, and that is usually achieved without needle protection in the Great Plains. Severe infection results in trees appearing yellow and sparse, thereby reducing aesthetic value, growth, and windbreaking ability. Heavily infected young trees often have uniform yellowing of second-year needles throughout the tree, not decreasing from the base to the top of the trees. The most extensive damage observed in the Great Plains was on Scots pine in neglected Christmas tree plantations in South Dakota, and in a crowded 11-year-old planting in North Dakota. No damage has been apparent on other infected pine species in the Great Plains. *C. minus* does not typically cause needle cast in Europe, but the fungus can still be recovered as an endophyte from some pines.

Management

Control by fungicides is possible, but several applications are needed. In Pennsylvania chlorothalonil applied five times (late March, early May, mid-June, early August, early October) gave good control. Other fungicides also prevent infection, but their reduced longevity on the needles reduces their cost effectiveness. Time of the first application should be when buds of common lilac have swollen so about 1/16 inch of green tissue is showing next to the bud scales. Two years of protection are needed to restore good needle density to sparse trees. If there is a nearby source of infection, annual protection is needed in nurseries to prevent distribution of infected seedlings. Such nurseries should work to eliminate sources of infection.

Silvicultural practices that increase air movement through plantings, or increase distances between susceptible trees, should reduce amount of infection. Improving soil fertility may delay symptom development long enough to provide another age-class of green needles. There is considerable variation in susceptibility to this fungus among Scots pine provenances.

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47. Phomopsis Blight of Junipers

Glenn W. Peterson (Riffle and Peterson 1986) (updated by Ned Tisserat)

Phomopsis blight is a serious problem in nurseries producing juniper (*Juniperus* spp.) seedlings and grafts. It also causes damage to several juniper species in landscape plantings.

Hosts and Distribution

Phomopsis blight, caused by the fungus *Phomopsis juniperovora*, is widespread on eastern redcedar (*J. virginiana*) and Rocky Mountain juniper (*J. scopulorum*) in the eastern United States and the eastern half of the Great Plains, but rare in the more arid, western Great Plains. The fungus may also cause damage to creeping (*J. horizontalis*) and savin (*J. sabina*) junipers, as well as some species of cedar (*Cedrus*), false-cypress (*Chamaecyparis*), and arborvitae (*Thuja*).

Symptoms and Signs

P. juniperovora initially infects foliage, then spreads to and sometimes kills stem tissues. Newly developing needles are especially susceptible while they are still in the yellowish-green stage. After needles become a normal, deep green, they are no longer susceptible. Small yellow spots appear on young needles of eastern redcedar and Rocky Mountain juniper within three to five days of infection. The fungus ramifies within infected needles and rapidly invades and girdles young stems (fig. 47-1). When a side shoot is infected, the fungus progresses to the main stem, which it may girdle if the stem is less than 0.4 inches in diameter. The portion of the plant above the girdled area then dies.

Figure 47-1—Phomopsis blight damage (U.S. Forest Service, Bugwood.org, used with permission).

At first, infected tissues turn light green but then rapidly change to the characteristic red-brown color of dead shoots, which finally turn ash gray (fig. 47-2). Lesions on larger stems frequently develop into cankers, but the stems are not girdled.

Symptoms of Phomopsis closely resemble those of Kabatina blight, and the two diseases may be difficult to distinguish in the field. However, symptoms of Phomopsis blight normally appear in spring and continue throughout the summer months, whereas Kabatina blight symptoms are usually visible by late winter.

Figure 47-2—Foliar symptoms of *Phomopsis juniperovora* on eastern redcedar (Bruce Watt, University of Maine, Bugwood, used with permission).

Disease Cycle

Spores produced in fruiting bodies (pycnidia) formed on leaves and stems infected the previous year are the most important source of inoculum early in the growing season. Pycnidia with viable spores may develop within three to four weeks after seedlings become infected, but usually are not well developed until infected tissues have dried considerably. Pycnidia are found most commonly on tissues that have turned ash gray. The pycnidia are embedded at first in needles and stems, but partially

Figure 47-3—Black fruiting structures (pycnidia) of *Phomopsis juniperovora* (Robert Anderson, U.S. Forest Service).

erupt through the epidermis (fig. 47-3). Two types of spores (alpha and beta) develop in the same or different pycnidia (fig. 47-4) and are extruded in whitish tendrils. The alpha spores are clear (hyaline), one-celled, and ellipsoid; contain two oil globules; and commonly are 7.5 to 10.0 µm × 2.2 to 2.8 µm. The beta spores are hyaline, one-celled, filamentous, slightly curved, and commonly 20.2 to 26.9 µm × 1.0 µm. The fungus can produce spores for as long as two years in dead parts of infected plants. Spores are dispersed primarily by rainsplash.

Damage

Damage is most severe in seedling and transplant beds of eastern redcedar and Rocky Mountain juniper in nurseries. Total loss of first-year seedlings can occur if control measures are not used. Losses are particularly high in areas where water tends to stand, and in beds of new seedlings adjacent to infected older seedlings. Survival of even lightly blighted nursery stock is very poor, because new shoots continue to be infected by spores produced on old, infected tissues.

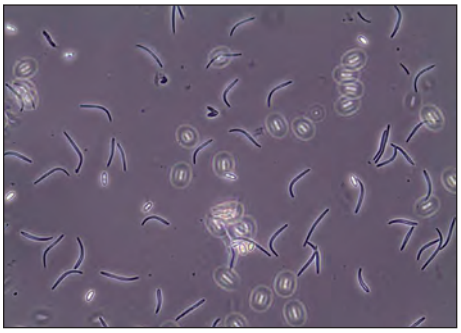

Figure 47-4—Alpha (ellipsoidal) and beta (long) spores of *Phomopsis* species (Lorraine Graney, Bartlett Tree Experts, used with permission).

Damage to junipers in landscape plantings is more severe on shrub forms or in areas with poor air movement. Trees may become unsightly because of numerous dead branch tips, but older trees seldom are killed because only small-diameter stems are girdled. For this reason, Phomopsis blight does not cause significant damage in natural stands of junipers.

Management

Because susceptible new foliage and viable fungal spores are present throughout the growing season in juniper seedling beds, protective fungicides need to be applied at seven- to 14-day intervals. Avoid poorly drained areas because losses are often greater where water tends to stand. If using overhead sprinklers, irrigate seedlings so that water on seedlings dries before nightfall. Infected seedlings should be rogued from the nursery bed. Junipers or other hosts of this fungus should not be used in nursery windbreaks or in landscape plantings on nursery grounds because they may be a source of inoculum (spores) for nursery stock. Such trees are more likely to be extensively infected if pruning results in development of juvenile foliage.

Phomopsis blight is usually not a serious problem on mature juniper trees in the landscape, but it may cause unsightly damage to shrub or prostrate forms in planting beds and require fungicide applications as outlined. In most cases, however, removal of dead tips in early spring and during dry weather is sufficient to control this disease. Susceptibility to *P. juniperovora* varies considerably among junipers. Consider choice of plant material when planting in disease-prone locations.

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48. Rhizosphaera Needle Cast of Spruce

James A. Walla; revised from Darroll D. Skilling and James A. Walla (Riffle and Peterson 1986)

Rhizosphaera needle cast is caused by the fungus *Rhizosphaera kalkhoffii*. It had been the most important needle disease of spruce (*Picea* spp.) in North America until the similar-appearing Stigmina needle cast developed. Needle discoloration caused by insects, mites, environmental stress, chemicals, cankers, and normal old-age senescence may appear similar to that caused by Rhizosphaera needle cast.

Hosts and Distribution

Hosts include blue (*P. pungens*), Engelmann (*P. engelmannii*), Norway (*P. abies*), white (*P. glauca*, including Black Hills seed sources), and several other spruce species. Other conifers, including Douglas-fir (*Pseudotsuga menziesii*), fir (*Abies* spp.), and pine (*Pinus* spp.), may also serve as hosts. In North America, Rhizosphaera needle cast has been most important in the Great Lakes region and northeastern United States. It occurs in the northern Great Plains and causes concern after consecutive years of above-average rainfall. It occurs in natural blue and Englemann spruce stands in Arizona and Colorado.

Symptoms and Signs

Faint yellow bands typically develop on recently infected needles in fall or the following spring, four to 11 months after infection. Small dark brown or black spherical fruiting bodies (pycnidia; fig. 48-1A) emerge through the stomata the spring after infection. They can easily be seen with a hand lens. On severely infected needles, a high proportion of stomatal pits contain pycnidia, giving the appearance of continuous fine black lines on each side of the needle (fig. 48-2B). White wax plugs from the stomatal pits often sit atop the pycnidia. The pycnidia contain asexual spores (conidia; fig. 48-3), which are aseptate, oval, colorless (hyaline), and small (4.5 to 8.6 µm × 2.5 to 4.6 µm). The needles may become fully discolored by early summer of the year after

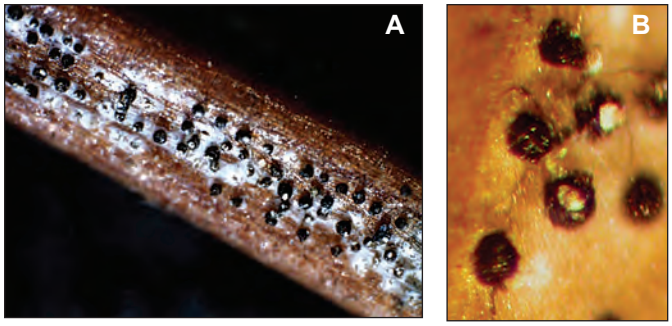

Figure 48-1—*Rhizosphaera kalkhoffii* fruiting bodies (pycnidia) (A) in stomatal pits (James A. Walla, Northern Tree Specialties, used with permission); and (B) showing smooth surface, dark hyphal strands extending from the base, and two fruiting bodies with white wax plugs from the underlying stomatal pits (Kasia Kinzer, North Dakota State University, used with permission).

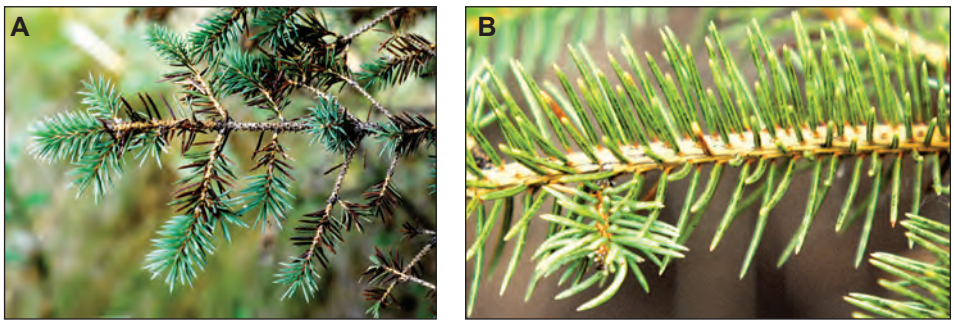

Figure 48-2—Rhizosphaera needle cast in spruce trees showing (A) a reduced number of healthy needle age-classes, and (B) rows of black fruiting bodies (pycnidia) visible without magnification in heavily infected trees (James Walla, Northern Tree Specialties, used with permission)

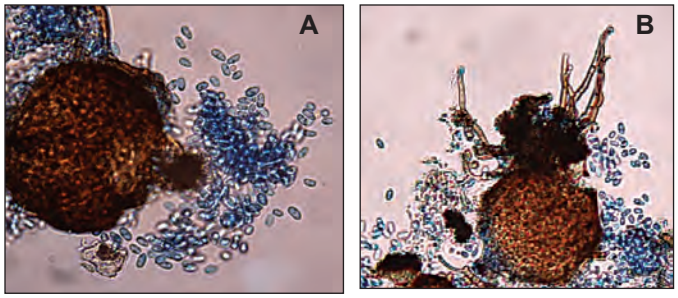

Figure 48-3—*Rhizosphaera kalkhoffii* (A) pycnidia (dark brown structures) and neck where spores emerge from the pycnidium, and (B) small oval spores (stained with aniline blue) and hyphal strands at the base of a pycnidium (James Walla, Northern Tree Specialties, used with permission).

infection and be cast later that year. The disease typically begins close to the ground in the interior crown and progresses upward and outward if conditions are favorable for development. Needles of all ages may be infected, sometimes resulting in a mixture of symptoms on a given age-class of needles (fig. 48-2A). Trees that have had severe infection for three or more years have a sparse, hollow appearance and may have reduced shoot growth (fig. 48-2A) and dead branches in the lower crown (fig. 48-4A).

Blue spruce exhibits a wide range of apparent resistance among trees. Resistance levels are expressed as stepwise increases in the number of needle age-classes that are green. Under severe disease pressure, the most affected branches on susceptible trees will have one age-class of green needles. The next most susceptible trees will have two age-classes of green needles, and so on. With severe disease pressure, trees that have four or more age-classes of green needles have good resistance to Rhizosphaera needle cast (fig. 48-4B).

Stigmina lautii causes a similar needle cast on spruce that can be confused with Rhizosphaera needle cast because its sporodochia very closely resemble of *R. kalkhoffii*. Using a 20× hand lens allows differentiation of the two fungi (compare fig. 48-1A with fig. 49-2).

Disease Cycle

Conidia are released from mature pycnidia for about two months after budbreak during periods of wet weather and are dispersed primarily by rainsplash to cause new infections. Symptoms become readily visible the spring of the year after infection. Pycnidia develop that spring and produce conidia to complete the disease cycle in one year. The disease cycle is longer on more-resistant trees.

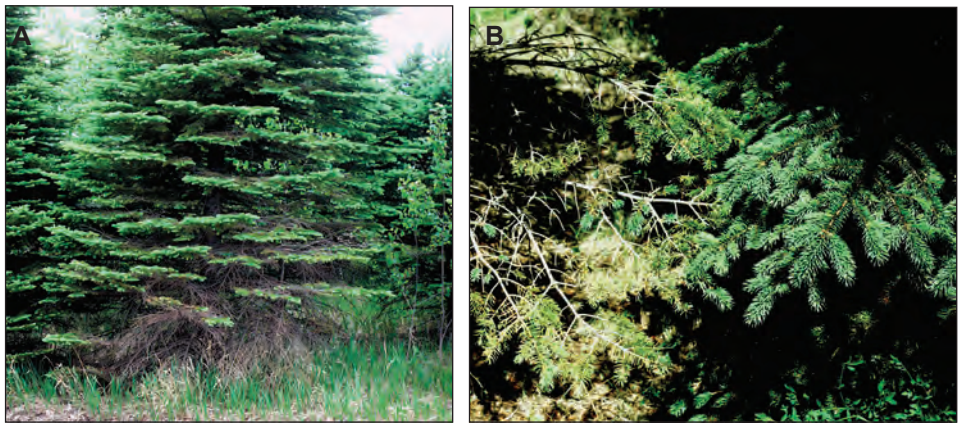

Figure 48-4—Spruce trees with Rhizosphaera needle cast showing (A) hollow, sparse appearance; and (B) wide variation in host resistance, as reflected in amount of defoliation under high disease pressure (James A. Walla, Northern Tree Specialties, used with permission).

Damage

Rhizosphaera needle cast can infect and damage trees of all ages, from nursery seedlings to mature trees. Blue and Engelmann spruce are the most susceptible species, white spruce is intermediate in susceptibility, and Norway spruce is relatively resistant. Although some tree mortality has been observed under epidemic conditions, the primary damage involves premature needle cast. Branches that have less than three age-classes of healthy needles for three to four years will die. Trees with sparse foliage or dead branches are unsightly for ornamental purposes, are unmerchantable for high-quality Christmas trees, and provide reduced benefits in conservation plantings. In the northern Great Plains, consecutive years with greater-than-normal spring rainfall have resulted in serious defoliation and branch dieback owing to Rhizosphaera needle cast on blue and sometimes white spruce. However, because spruce may be damaged in many ways, it is important to realize that presence of a particular pathogen does not mean it is responsible for significant needle discoloration and loss.

Management

As the fungus can be transported on nursery stock, avoid introducing infected stock into plantings. Young spruce planted near older spruce are prone to severe damage even when disease is not severe on the older trees. During outbreaks, the disease becomes widespread. In areas where this disease occurs sporadically, disease severity fluctuates widely with amount of late spring precipitation. Two dry years can reduce disease to a minimum, whereas a few wet years can result in severe needle cast. Awareness of such fluctuations can help in planning management strategies. If the disease has been a problem in the area, consider planting less-susceptible spruce species or other types of trees. Promote good air circulation (wide spacing, pruning lower spruce branches and overhanging trees, mowing grass and brush). Consider removing trees that are repeatedly damaged. Blue spruce plantings should be examined frequently for signs of disease. If the trees are dropping their needles prematurely, determine the cause. Early identification helps avoid damage because light infections can be controlled with only one or two fungicide sprays.

Two years of fungicide protection of needles for the first two months after budbreak will usually restore moderately infected trees to good foliage quality. Heavily infected trees will require more years of treatment. Several fungicides are registered specifically for Rhizosphaera needle cast management in the United States and Canada (a, b, respectively, in the following list). Some products containing azoxystrobin, chlorothalonil[a], [b], copper hydroxide[a], copper oxychloride, copper salts of fatty and resin acids, copper sulfate, mancozeb[a], mono- and di-potassium salts of phosphoric acid, propiconazole[b], pyraclostrobin, sulfur, thiophanate-methyl, and trifloxystrobin[b] are labeled for use on spruce and have been found to control some foliar diseases. Chlorothalonil is most commonly recommended, but recent research on efficacy is lacking. In the United States, fungicides may be used for disease control if they are registered for the intended target site in the state where the application occurs. The products for use in Canada are for commercial or agricultural use.

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49. Stigmina Needle Cast of Spruce

James A. Walla and Aaron D. Bergdahl

Stigmina needle cast is caused by the fungus *Stigmina lautii*. The fungus was first described from native black (*Picea mariana*) and white (*P. glauca*) spruce in Manitoba and Saskatchewan in 1973. It was first reported causing disease in North Carolina in 2002. This disease is often misdiagnosed as Rhizosphaera needle cast.

Hosts and Distribution

Blue (*P. pungens*) and white (including Black Hills seed sources) spruce are generally highly susceptible to Stigmina needle cast, and the disease has been reported on black and Norway (*P. abies*) spruce. Stigmina needle cast was rarely recognized before 2006, but it is now known to be widespread east of the Rocky Mountains in the United States and Canada.

Symptoms and Signs

One or more faint yellow bands and small patches of tiny round black fruiting bodies (sporodochia), which replace the white wax in the stomatal pits, develop in the fall of the same year as infection or early the next spring. Either bands or sporodochia may appear first. By late summer of the year after infection, individual bands or whole needles may be yellow, purple, tan, reddish brown, or brown, and sporodochia may occur in more than half of the stomatal pits. It appears that multiple infections are necessary before whole needles become discolored. Wax from the stomatal pits may rest atop sporodochia. Most needles are discolored and sporodochia mature to produce spores either late in the second season, or early in the third season after infection (fig. 49-1). With dark spores produced mostly horizontal to the needle surface, sporodochia appear en masse as discontinuous fuzzy black lines along the length of each side of a needle, especially on green needles (fig. 49-2). All needle age-classes are usually susceptible, so needles in older age-classes may exhibit multiple stages of disease development. Microscopic observation of the spores is required to confirm Stigmina needle cast. Spores of *S. lautii* are light brown, elongate, five- to eight-septate, and 30 to 45 µm × 5.0 to 5.5 µm when observed with a compound microscope (fig. 49-3).

Figure 49-1—Yellow and brown necrosis and black stippling (fruiting bodies of *Stigmina lautii*) on spruce needles (James A. Walla, Northern Tree Specialties, used with permission).

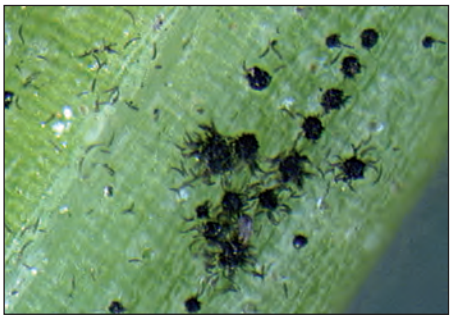

Figure 49-2—Fruiting bodies (sporodochia) in and over some stomatal pits of a spruce needle. Asexual spores (conidia) extend out from the sporodochia, resulting in the appearance of fuzzy fruiting bodies, but some sporodochia may not have attached conidia. Released conidia are often visible lying on the needle surface. (Justin Knott, formerly North Dakota Department of Agriculture, used with permission).

On most heavily infected trees, the two youngest age-classes of needles (first- and second-year needles) are green or mostly green until summer's end (fig. 49-4). Most third-year and any older needles are necrotic. Near summer's end, discoloration expands on second-year needles so that only first-year needles are bright green. Infected needles may drop off shoots as soon as the end of the second season after infection, but they typically begin dropping near the end of the third summer after

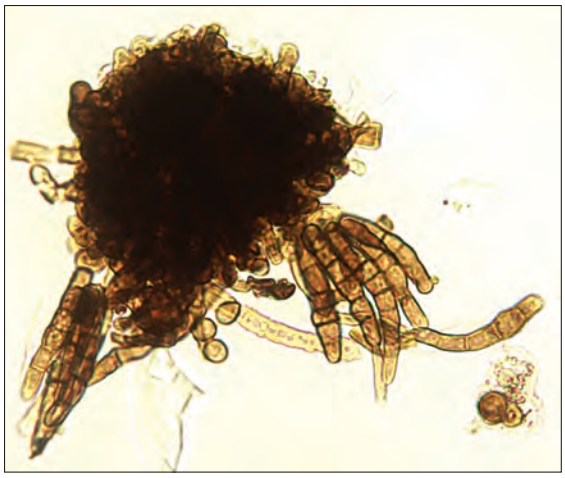

Figure 49-3—Squash mount of *Stigmina lautii* fruiting body (sporodochium), with brown septate spores (conidia; Kasia Kinzer, North Dakota State University, used with permission).

Figure 49-4—Spruce shoots with needles showing symptoms of Stigmina needle cast. Second-year needles have advanced symptoms, and third-year needles have been cast (late August; James A. Walla, Northern Tree Specialties, used with permission).

Figure 49-5—Discolored needles and sparse foliage caused by Stigmina needle cast (James A. Walla, Northern Tree Specialties, used with permission).

infection. Some or all needles may remain attached for another year or two, and spores continue to be produced on them seasonally. A few blue spruce appear to have some resistance to Stigmina needle cast. That apparent resistance is expressed as a delay in infection or development of signs and symptoms by one year, so there is one additional age-class of asymptomatic needles on heavily infected trees with this trait.

Disease Cycle

In the northern Great Plains, *S. lautii* has a two-year life cycle. Some spores are produced in very late fall of the second season after infection, but too late to result in new infections. The next spring, new spores first develop one to two weeks before new shoots begin growing. Spore production and infection can occur for the rest of the growing season whenever temperatures are above about 50 °F, but the major infection period is the first two months after budbreak. On most trees, immature sporodochia develop on needles by late spring about one year after infection and produce spores six to 12 months after formation, continuing the disease cycle.

Damage

Damage from Stigmina needle cast results from the presence of discolored needles and the loss of functioning needles. Stigmina needle cast is proving to be more aggressive and to have longer-lasting epidemics than other spruce needle diseases. Thinning of foliage commonly occurs over the lower two-thirds of the crown of open-grown trees (fig. 49-5), and a greater portion of tree crowns may be affected

in dense stands. In North Dakota, the impact of Stigmina needle cast increased each year from 2006 to 2012, through periods with above-average rainfall and periods with below-average rainfall, to a level well beyond that associated with other spruce needle diseases.

Management

Two fungicide management approaches are available. In the first approach, needle protection for two months after budbreak, repeated every year, will reduce disease so branches retain three or more age-classes of green needles. This approach would be useful for landscape, Christmas, and other trees where loss of the oldest classes of needles is acceptable. In the second approach, needle protection for the entire infection period (usually mid-May to mid-October in North Dakota) every year for four or five years, is meant to prevent infections until previously infected needles drop from the tree. This approach would be necessary for nursery stock where certification requires that plants be disease-free and for those that prefer to maximize the aesthetic value of a tree.

No fungicides are labeled specifically for Stigmina needle cast in the United States. However, fungicides may be used for disease control in the United States if they are registered for the intended target site in the state where the application occurs. For needle diseases on spruce, these fungicides include some products containing azoxystrobin, chlorothalonil, copper hydroxide, copper salts of fatty and resin acids, copper sulfate, mancozeb, mono- and di-potassium salts of phosphoric acid, pyraclostrobin, sulfur, and thiophanate-methyl. In Canada, chlorothalonil, propiconazole, and trifloxystrobin are labeled for use by commercial applicators specifically for management of Stigmina needle cast in Christmas tree nurseries, and copper-oxychloride is similarly labeled except it is registered for management of needle cast diseases in general. In North Dakota, experimental application of chlorothalonil under the first approach (above) was highly effective during the protection period, and experimental application of copper sulfate under the second approach (above) was moderately effective.

Because potential fungicide management options will be long-term or intensive, determine the value of a spruce tree to decide whether disease management is justified. In areas where Stigmina needle cast is epidemic, refraining from planting susceptible spruce and removal of severely damaged spruce are justified. Stigmina needle cast has not been found on a Norway spruce cultivar (‘Royal Splendor’) in North Dakota during multiple inspections since 2006, whereas adjacent blue spruce trees were severely diseased. This apparent resistance has not been evaluated in replicated trials, however. Cultural methods may reduce Stigmina needle cast, but their efficacy has not been established.

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50. Canker Diseases of Juniper and Oriental Arborvitae

Ned Tisserat

Rocky Mountain juniper (*Juniperus scopulorum*) and oriental arborvitae (*Platycladus orientalis*) may be seriously damaged by canker diseases, especially in the southern Great Plains.

Hosts and Distribution

Two canker diseases cause dieback and mortality of juniper (*Juniperus* spp.) and oriental arborvitae in the Great Plains. Botryosphaeria canker, caused by the fungus *Botryosphaeria stevensii*, is a serious disease of Rocky Mountain juniper in the south-central and southeastern Great Plains. The disease also occurs on eastern redcedar (*J. virginiana*) and savin juniper (*J. sabina*). The disease has also been reported on savin juniper in Pennsylvania and is likely widespread on junipers in the eastern United States. A second fungus, *Seiridium unicorne* (teleomorph *Lepteutypa cupressi*), primarily causes cankers on winter-damaged or drought-stressed oriental arborvitae, and occasionally juniper species, primarily from Kansas south into Texas. The fungus may occasionally cause cankers on other tree species in the cypress (Cupressaceae) family.

Symptoms and Signs

Junipers affected with Botryosphaeria canker (fig 50-1) develop elongated, flattened, often resinous cankers (figs. 50-2). These cankers may occur anywhere on woody stems, but are commonly located near branch crotches in the interior portion of the tree crown. Cankers are sometimes difficult to see, and it is often necessary to cut the dead branch off and carefully scrape away the outer bark to expose the chocolate-brown dead tissue in the canker in the living bark (phloem). The surrounding healthy phloem or inner bark is pearl white. Small black fruiting bodies (pycnidia) of the fungus also develop in the canker, but may be partially hidden by thin pieces of dead bark. Pycnidia produce spores (conidia; 25 to 31 µm × 10 to13 µm) that are at first single-celled and clear (hyaline; fig. 50-3), but when mature become dark brown and often two-celled.

Symptoms of Seiridium canker are similar to Botryosphaeria canker, and microscopic examination of the fungal spores may be required to differentiate them.

Figure 50-1—Branch dieback on (A) Rocky Mountain juniper caused by *Botryosphaeria stevensii*, and (B) oriental arborvitae caused by *Seiridium unicorne* (Ned Tisserat, Colorado State University, used with permission).

Figure 50-2—Flattened branch canker on (A) Rocky Mountain juniper caused by *Botryosphaeria stevensii*, and (B) oriental arborvitae caused by *Seiridium unicorne* (Ned Tisserat, Colorado State University, used with permission).

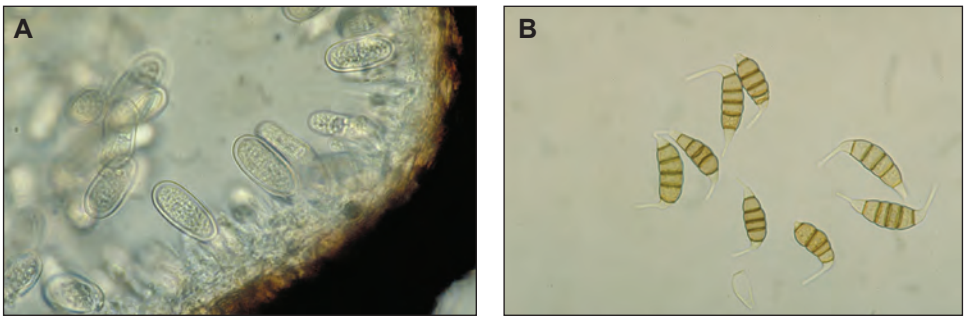

Figure 50-3—Asexual spores (conidia) of (A) *Botryosphaeria stevensii*, and (B) *Seiridium unicorne* (Ned Tisserat, Colorado State University, used with permission).

Seiridium unicorne causes elongated, flattened cankers to form on small branches and main stems. On junipers, these cankers may be larger and more diffuse than those caused by *B. stevensii*. Bleeding or resin formation in cankers, especially on oriental arborvitae, is common (fig 50-2B). Multiple coalescing branch and stem cankers may cause branch dieback or, in some cases, tree death. In the cankers the fungus forms black pycnidia, which produce brown-pigmented, multiseptate conidia with appendages (fig. 50-3B).

Disease Cycle

Spores of *B. stevensii* are dispersed relatively short distances by rainsplash. Peak spore release occurs in late May and June, suggesting this is a major period of infection. Spores of *S. unicorne* are similarly released during rain. Both fungi likely infect through bark wounds. Cankers develop over several months and typically girdle small branches within a year, causing death of the branch distal to the canker. Dead branches scattered throughout the crown may result. Infection by *S. unicorne* is greatly exacerbated in trees suffering from winter damage or drought stress, whereas *B. stevensii* will cause cankers even on vigorously growing trees.

Damage

Occasionally, *B. stevensii* stem cankers will cause girdling and rapid death of the top one-third to one-half of the tree crown. More commonly, the disease causes death of individual branches throughout the crown and a gradual tree decline. This fungus can kill large vigorous Rocky Mountain junipers in both windbreak and ornamental settings. Mortality in windbreak plantings is especially damaging because it may create large gaps. Botryosphaeria canker should not be confused with Kabatina or Phomopsis blight, both of which affect only foliage and succulent branch tips. Seiridium canker may also kill oriental arborvitaes, but more commonly results in scattered branch dieback.

Management

Rocky Mountain juniper is very susceptible to Botryosphaeria canker and should not be planted in locations where the disease is common. Savin juniper also is susceptible, but eastern redcedar and Chinese juniper are more resistant. Fungicides are not currently labeled for control of Botryosphaeria canker. Remove cankers on diseased trees in winter or late spring. Do not prune or shear cankered junipers in May or June, when spores are released. Pruning and sanitation may not completely suppress canker development. Seiridium canker tends to be associated with trees that are suffering from winter damage, drought, or other environmental stresses and thus less able to defend against the fungus. Canker development can be suppressed by planting winter-hardy selections of oriental arborvitae and protecting trees from winter desiccation. Cankered branches should be pruned from trees and destroyed.

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51. Diplodia Shoot Blight and Canker Disease

James T. Blodgett and Glen R. Stanosz; revised from Glenn W. Peterson and David W. Johnson (Riffle and Peterson 1986)

The fungal pathogen, *Diplodia sapinea* (*D. pinea*, *Sphaeropsis sapinea*), causes shoot blights and cankers in pines (*Pinus* spp.) and some other conifers in much of the United States. Damage can be severe in landscapes, windbreaks, parks, and forests. A closely related pathogen, *D. scrobiculata*, occurs in the United States and may be present in the Great Plains although it has not been reported.

Hosts and Distribution

Diplodia shoot blight and canker disease is a common and sometimes serious problem in the Great Plains. Pines are affected from seedling stage to maturity. The disease affects trees in nurseries, Christmas tree and ornamental plantings, plantations, and natural stands. Exotic and native two- and three-needle pines are commonly damaged in the United States. *D. sapinea* is widely distributed in the Great Plains and Diplodia shoot blight and canker disease can be locally severe, affecting Austrian (*P. nigra*), ponderosa (*P. ponderosa*), Scots (*P. sylvestris*), red (*P. resinosa*), and mugo (*P. mugo*) pines. Austrian pine, which has been widely used in landscape, windbreak, and park plantings since the early 1900s, is especially susceptible.

Symptoms and Signs

Symptoms of Diplodia shoot blight on new shoots first occur during late May to late June. The first indications of infection are often resin droplets or one to a few short dead needles on emerging shoots, or a combination thereof. The most conspicuous symptom of Diplodia shoot blight is death of new shoots containing short, light brown, often wilting needles (fig. 51-1) that fade to gray and remain attached to stems. Both needles and stems can become stunted or crooked. *D. sapinea* commonly kills entire new shoots by mid- to late summer. Cankers, often resinous, develop in branches and stems with discolored brown phloem and cambium. Underlying xylem is often stained blue.

Figure 51-1—Typical symptom of Diplodia shoot blight: a dead, crooked new shoot with light brown needles that remain attached (James T. Blodgett, U.S. Forest Service).

Symptomatic, new shoots may be scattered throughout crowns, although damage is often first evident in lower crowns. Symptom severity can vary considerably among major branches. In some cases, damage may be confined to new shoots (fig. 51-2A), particularly on trees affected for the first time. After two or three successive years of damage, extensive branch and top mortality can occur (fig. 51-2B). Repeated outbreaks of Diplodia shoot blight and canker disease result in reduced growth, deformed trees, and tree mortality.

Small black asexual fruiting bodies (pycnidia) are easily observed with a 10× hand lens. However, microscopic examination of the asexual spores (conidia; fig. 51-3) or DNA sequence-based diagnostics are necessary for identification. Pycnidia are often numerous

Figure 51-2—Mortality caused by Diplodia shoot blight in (A) shoots, and (B) top/branches of ponderosa pine (James T. Blodgett, U.S. Forest Service).

Figure 51-3—Spores (conidia) of *Diplodia sapinea* with light young conidia and dark brown older conidia; approximate size 34 to 39 µm × 12 to 13 µm (James T. Blodgett, U.S. Forest Service).

at the base of short, ash-gray needles killed the previous year (fig. 51-4). Pycnidia can develop in stems and may be numerous in scales of mature seed cones (fig. 51-5). To assist in diagnosis, needle and stem samples may be incubated in plastic bags with a piece of damp paper towel. Pycnidia with conidia may develop within two to three weeks.

Disease Cycle

D. sapinea survives as mycelium or pycnidia in killed needles, bark of young shoots, and scales of mature seed cones. Numerous conidia are dispersed during rainfall, especially in spring, but conidial dissemination can also occur throughout the year. Conidia are spread by rainsplash, wind-driven rain, or insects.

Needles and young elongating stems are most susceptible to *D. sapinea* infection in spring from budbreak until around mid-June. Conidia germinate and germ tubes can directly penetrate nonwounded, immature needle and stem tissues. The pathogen also can infect both current-year shoots and older tissues through wounds. Rain or high humidity coupled with cool to moderate temperatures favor spore germination, germ tube growth, and infection. If rain is sparse when new shoots are highly susceptible, disease incidence is usually low. After infection, new shoots are often killed quickly. Pycnidia may be produced within two weeks of shoot infection in spring, or throughout the summer in wet years.

Seed cones bearing pycnidia are often present on pines that otherwise appear healthy. These cones can be a source of large numbers of conidia. The conidia can germinate to initiate new infections on lower branches of the tree, nearby trees, and seedlings. Debris left onsite after harvesting also may serve as a source of conidia for several years.

This pathogen can persist on or in asymptomatic stems and branches of pines. Water stress has been demonstrated to act as a trigger, allowing *D. sapinea* to proliferate and

Figure 51-4—Fruiting bodies (pycnidia) of *Diplodia sapinea* at the base of pine needle with fascicle sheath removed (James T. Blodgett, U.S. Forest Service).

Figure 51-5—Second-year seed cone scales with (A) fruiting bodies (pycnidia) of *Diplodia sapinea*, and (B) uninfected scales (James T. Blodgett, U.S. Forest Service).

cause disease in these trees. This ability to act as a latent pathogen could explain the rapid mortality observed after outplanting or following hail or other wound events.

Damage

Diplodia sapinea can cause shoot blight, canker, crown wilt, collar rot, and root disease. Although shoot blight is the most common type of damage, branch death, top dieback, and tree mortality can occur. Diplodia shoot blight and canker disease can predispose trees to other diseases and attack by insects, including the pine engraver (*Ips* spp.).

Increased damage from Diplodia shoot blight and canker disease has been associated with water deficits, competing vegetation, areas with high soil nitrogen, and offsite plantings. This disease often severely affects trees wounded by hail, snow, pruning, shearing, insects, or other damage. In the Great Plains, hail events are frequently associated with outbreaks of the disease and symptoms develop in one to two weeks. Wounded tissues remain vulnerable to *D. sapinea* infection for several days.

Although pines of all ages are susceptible to *D. sapinea*, damage is more severe in older plantings. In the Great Plains, windbreaks that are 20 to 22 years old have few

diseased pines. However, incidence and damage from Diplodia shoot blight and canker disease increase as trees approach 30 years, and damage is often severe in older pines with abundant mature seed cones.

Management

Reducing water stress, avoiding wounds, and maintaining tree vigor are often the best options to manage Diplodia shoot blight and canker disease. Methods to reduce water stress include selecting species suited to the site, proper planting, managing competing vegetation, and stand thinning. In developed sites, watering during droughts is recommended. Avoid pruning or shearing in Christmas tree or other pine plantings during rain events. Conduct pruning during the dormant season, and disinfect pruning tools. Seedlings, including those in nursery beds, and small trees become infected when they are located close to diseased or cone-bearing pines. Remove cone-bearing trees or plant nonhost trees in the area. Nitrogen fertilization of pines at levels recommended for ornamental and shade trees can result in increased disease incidence and severity, and should be avoided.

Selecting nonhost species or more-resistant pine species is an option for long-term management. Studies have shown that pines differ in susceptibility, but most two- and three-needle pines are susceptible to this disease. Austrian pine is extensively damaged by Diplodia shoot blight and canker disease. Although ponderosa pine is susceptible to this disease, it is much more tolerant than Austrian, lodgepole, or red pine in the Great Plains. Ponderosa, Scots, and jack (*P. banksiana*) pine do not differ markedly in susceptibility to Diplodia shoot blight and canker disease. Scots pine is often recommended for landscape plantings, because it frequently experiences less damage from this disease than Austrian pine, and ponderosa pine planted offsite.

Infection of new shoots can be reduced significantly by applying an appropriate registered fungicide at the time when shoots are highly susceptible, which is usually between late April and mid-May, from budbreak to the end of shoot expansion. Fungicide applications will protect new shoots, but will not prevent infection of cones or eliminate the pathogen in colonized tissues. Fungicide treatments are usually applied to nursery seedlings, Christmas trees, or individual high-value landscape trees. Consult your state or local extension agent regarding appropriate registered fungicides.

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52. Gymnosporangium Rusts

Simeon Wright, James A. Walla, and William R. Jacobi; revised from James A. Walla and Jerry W. Riffle and from Kenneth E. Conway and Mark W. Andrews (Riffle and Peterson 1986)

Many *Gymnosporangium* species occur in the Great Plains. Because it is difficult to differentiate them based on symptoms, diagnosis of species is often imprecise. Species identification is primarily based on size and shape of the various spores, the morphology of fruiting bodies on rosaceous hosts (aecia) and fruiting bodies on *Juniperus* hosts (telia), and on host symptoms. There is much overlap in these traits among species, but each species has a unique combination of the traits. Molecular diagnostics using telial horns or masses of aeciospores offer increasingly useful diagnostic capability. It may not be necessary to identify the rust species unless management involves removing an alternate host or planting resistant varieties.

The use of common names for diseases caused by these fungi does not always exactly fit the hosts being diagnosed. For example, the name cedar apple rust refers specifically to the disease caused by *G. juniperi-virginianae* on junipers and apples (*Malus* spp.). In this case, "cedar" is derived from the partial common name of one of the plants that is infected by the fungus, eastern redcedar (*J. virginiana*), which is a juniper. However, this fungus also infects other hosts.

Hosts and Distribution

Several host-specific *Gymnosporangium* rust species occur in the Great Plains (table 52-1). These species alternately infect two unrelated hosts to complete their life cycle. The aecial stage almost always occurs on rosaceous species and the telial stage occurs on *Juniperus* species. Rust species of particular significance to the Great Plains are individually discussed.

Symptoms and Signs

Symptoms vary with the rust species (table 52-1). Symptoms on rosaceous hosts include yellow, orange, or red lesions on leaves (fig. 52-1), petioles, fruit (fig. 52-2), or new twigs (fig. 52-3) and rough, elongate cankers or gall-like swellings on leaf veins, petioles, twigs, and branches. Fruit can be distorted, and premature leaf drop and dieback can occur. The yellow to black pimplelike sexual fruiting bodies (spermagonia) (fig. 52-4) and later tubelike aecia develop in rosaceous host lesions. Symptoms on *Juniperus* hosts include small lesions on needles, globular branch galls (fig. 52-5), spindle-shaped stem swellings (fig. 52-6), witches'-brooms (fig. 52-7), and branch dieback. Gelatinous, orange, fingerlike telial horns (fig. 52-8) or cushionlike telial masses grow and swell from infections on the *Juniperus* hosts in spring.

Disease Cycle

Spring rains are absorbed by each lesion, gall, swelling, or witches'-broom on junipers, causing telial horns or cushionlike masses to swell. Swelling may begin as early as prebloom or as late as full leafout of the rosaceous hosts, depending on the rust species, its hosts, and the environmental setting. Teliospores in the swollen telial structures germinate to produce basidia that bear basidiospores, which are carried by wind or insects to nearby rosaceous hosts. Telial horns or cushions may may swell and dry down several several times with repeated wetting, allowing production of basidiospores for eight to 10 weeks. After infection of susceptible host tissue by basidiospores, spermagonia develop in yellow to orange lesions, followed by production of aecia. Aeciospores are blown by wind to junipers from late spring to early fall, depending on the species of rust fungus. After infection of the juniper, galls or other structures form during the next one to two years. New telial horns or masses appear the next spring and the cycle repeats. Some galls, swellings, or witches'-brooms produce telial horns or masses annually thereafter.

Table 52-1—Some *Gymnosporangium* species in the Great Plains

Rust species	Distribution	Symptoms on junipers	Major aecial hosts[a]
G. bethelii	Widespread in west	Rough, irregular, spindle-shaped galls	Hawthorn, crabapple, apple
G. clavariiforme	Widespread	Stem swellings, witches'-brooms	*Amelanchier* spp.
G. clavipes	Widespread	Spindle-shaped stem swellings	Many Rosaceae
G. connersii	North	Branch galls	Hawthorn
G. corniculans	North	Branch galls	*Amelanchier* spp.
G. exiguum	South	Foliar lesions	Hawthorn
G. globosum	Widespread	Branch galls	Hawthorn
G. gracile	Texas	Stem swellings, witches'-brooms	*Amelanchier* spp.
G. juniperi-virginianae	Widespread	Branch galls	Apple, crabapple
G. nelsonii	Western and north	Branch gall or no symptoms	*Amelanchier* spp.
G. nidus-avis	Widespread	Stem swellings, witches'-brooms	*Amelanchier* spp.
G. trachysorum	South	Branch galls	Hawthorn

[a] Other Rosaceae may be aecial hosts for some of these rusts.

Figure 52-1—Lesions and fruiting bodies (spermogonia, aecia) on hawthorn leaves (David Leatherman, Colorado State Forest Service, used with permission).

Figure 52-2—Fruiting bodies (aecia) on hawthorn leaves and fruit (Harold Larsen, Colorado State University, used with permission).

Damage

Damage is not usually life threatening on rosaceous hosts, although severe infection reduces yield, fruit quality, vigor, and aesthetic value. Apples and *Amelanchier* species (serviceberry, juneberry, Saskatoon berry) are particularly susceptible to these effects.

Damage to junipers is usually slight with reduced vigor and aesthetic value. Rust fungi that cause stem swellings or galls may kill branches above the infected points when the swellings or galls die. If numerous large galls occur on a juniper, branches can be permanently bent downward. Rusts that cause witches'-brooms can deform trees and serve as nutrient sinks to reduce vigor in the rest of the plant. If broadleaf hosts are nearby and environmental conditions favor infection, spectacular numbers of galls or witches'-brooms can form on junipers. Susceptible groundcover junipers have been killed by branch-girdling galls.

Figure 52-3—Fruiting bodies (aecia) in leaf pustules and stem swellings on serviceberry (James A. Walla, Northern Tree Specialties, used with permission).

The following rusts are common in one or more areas of the Great Plains. Information included in the descriptions provides an indication of the range of symptoms that may be encountered among *Gymnosporangium* rusts and the types of field observations that may be used to provide rough diagnosis.

Cedar Apple Rust

G. juniperi-virginianae is widespread east of the Rocky Mountains and has been reported in some western Great Plains States. The primary aecial hosts are apple and crabapple, where yellow lesions appear on foliage beginning in late spring or early summer. Mature lesion color varies from yellow to orange and red. Lesions may also appear on fruit and, rarely, on twigs. Short aecia (1 to 2 mm long) develop in midsummer, open by aecial walls flaring back from the center, and release aeciospores until fall. Aeciospores infect leaves of eastern redcedar, Rocky Mountain juniper (*J. scopulorum*), and various ornamental junipers. Over the next year, brown galls measuring 0.25 to 2.0 inches in diameter develop on twigs of the juniper host, followed by long, pointed telial horns on galls the following spring. The galls die after one season of sporulation. Resistance levels of many apple, crabapple, and juniper cultivars to this rust are commonly available in extension bulletins and fruit production manuals. The rust has multiple races, however, and cultivar resistance can change if different races become prevalent in a given area.

Figure 52-4—Fruiting bodies (spermagonia) on hawthorn leaves, stems, and fruit (Harold Larsen, Colorado State University, used with permission).

American Hawthorn Rust

G. globosum is widespread in the eastern Great Plains, and has been reported in some prairie provinces and western Great Plains states. The primary aecial hosts are apple, crabapple, and hawthorn (*Crataegus* spp.), although several other rosaceous species are susceptible. Infections of aecial hosts may occur on leaves, fruit, petioles, and twigs, but fruit infection is rare. Mature lesions appear in late spring and vary in color from yellow to orange and red. In contrast with cedar apple rust, long (3 to 5 mm) tubelike aecia extend from lesions in midsummer, open by aecial walls tearing irregularly, and release aeciospores until fall. Aeciospores infect leaves of eastern redcedar, Rocky Mountain juniper, and

Figure 52-5—Globular branch galls on juniper (left: William R. Jacobi, Colorado State University, used with permission; right: Simeon Wright, Missouri Department of Conservation, used with permission).

various ornamental junipers. Over the next year, brown galls (0.1 to 0.6 inch diameter) develop on twigs of the juniper host, followed by short telial horns the following spring. In contrast with cedar apple rust, the galls are smaller, can produce spores for multiple years, and frequently do not kill twigs.

Quince Rust

G. clavipes is widespread in the eastern Great Plains and has been reported in the prairie provinces and some western Great Plains States. Eleven genera of rosaceous plants are aecial hosts: *Amelanchier*, *Aronia* (chokeberry), *Chaenomeles* (flowering quince), *Cotoneaster* (cotoneaster), *Crataegus*, *Cydonia* (common quince), *Malus*, *Mespilus* (medlar), *Photinia* (photinia), *Pyrus* (pear), and *Sorbus* (mountain-ash). Symptoms vary but frequently include fruit distortion and swelling of leaf midveins, petioles, and new shoots. Thorns and young branches are also susceptible and may develop elongate, spindle-shaped perennial cankers. White tubular aecia, 2 to 3 mm long, develop during the summer, becoming torn at the tip or splitting along the side to release aeciospores until fall. Aeciospores infect leaves and soft stems of eastern redcedar, Rocky Mountain juniper, and certain other juniper species. Slight swelling develops around the obscure young infections. It is unknown whether cushionlike telia are produced during the first or second spring and early summer after infection. Most of the infections die after sporulation, but those that survive develop into elongate, swollen (spindle-shaped) perennial cankers on twigs and branches. Perennial cankers often die within four to six years. Those that survive can cause stem swellings 8 to 10 inches in diameter. Severe infection of junipers can reduce winter hardiness.

Figure 52-6—Spindle-shaped stem swelling with cushionlike telia on juniper (Simeon Wright, Missouri Department of Conservation, used with permission).

Juniper Broom Rust

G. nidus-avis is widespread in North America. Several species of serviceberry, including *Amelanchier alnifolia* and *A. stolonifera*, are susceptible, as are common quince and some flowering quince and apple species. Aecia 2 to 5 mm long develop in mid-to-late summer on leaves, petioles, new shoots, and fruit and tear irregularly when mature. Aeciospores infect leaves and green shoots of various junipers, including Rocky Mountain juniper, eastern redcedar, creeping juniper (*J. horizontalis*), and Chinese juniper (*J. chinensis*). On the juniper host, small witches'-brooms may develop throughout the crown. Some of them may become perennial to form one to several witches'-brooms more than 2 ft in diameter. In perennial witches'-brooms, the shoots are dwarfed and highly branched, and there is often a reversion of the needles to the juvenile form. Sometimes portions of the subtending branch are swollen. Infection of stems may lead to spindle-shaped stem swellings without witches'-brooms. Small orange gelatinous telial masses appear on leaves, shoots, and stems throughout the witches'-brooms and stem swellings in spring to early summer.

Figure 52-7—Witches'-brooms in juniper host, showing wet and dry telia (top, middle: James A. Walla, Northern Tree Specialties, used with permission; bottom: Harold Larsen, Colorado State University, used with permission).

Japanese Apple Rust

G. yamadae is a damaging exotic rust from Asia that was first observed in the United States in 2004. As of 2013, it was limited to nine eastern states and one province. The primary aecial hosts are apple species, and the primary telial host is Chinese juniper. On apple leaves, spots are bright red with a pale cream to white center. Infection of fruit is rare. Severe infection occurs on susceptible apple varieties, but susceptibility of apple varieties resistant to cedar apple rust is unknown. *G. yamadae* is morphologically similar to *G. globosum*, but the aecial side walls and telial horn lengths are differentials. Aecia on the lower apple leaf surface are 3 to 7 mm long. On junipers, globoid swellings or small galls are produced on branches. Telial masses 0.2 to 0.3 inches long protrude from swellings like rubbery shelves. The pathogen dies after one season of sporulation.

Juniper Knot Gall Rust

G. bethelii is very common on the plains of eastern Colorado. This rust forms reddish-brown, irregular, perennial galls 0.1 to 0.6 inches in diameter on several juniper species. Telia are chestnut brown, wedge shaped, and 0.1 to 0.2 inches high. The alternate hosts are primarily hawthorn, apple, crabapple, and others. The aecia produce long hair-like tubes on the underside of leaves.

Figure 52-8—Gelatinous telial horns swelling from juniper galls in spring (top: James A. Walla, Northern Tree Specialties, used with permission; bottom: James T. Blodgett, USDA Forest Service).

Management

Rust management is rarely required on junipers with galls, but large witches'-brooms damage junipers and should be removed. A combination of practices may effectively manage these diseases on rosaceous hosts.

Damage can be reduced by not planting susceptible varieties near alternate hosts. Varieties may be resistant to some rust species and not to others. Susceptibility of apple, crabapple, hawthorn, and juniper varieties and species has been determined for some of the most common rusts. When only a few branches are affected, pruning galls or other telia-producing structures from *Juniperus* hosts may reduce infection on alternate hosts. Eradication of the *Juniperus* host from the vicinity may reduce new infections on the rosaceous host because basidiospores are fragile. A two mile separation may be required to minimize rust infections, but a half mile separation will substantially reduce infection.

Fungicides are registered for both rosaceous and *Juniperus* hosts. Application timing and coverage are critical. Make fungicide applications to protect rosaceous hosts according to the fungicide label. The prescribed timing is generally as flower buds expand until telial structures on alternate *Juniperus* hosts become inactive in late spring or early summer (usually four to six weeks). Fungicide applications to protect *Juniperus* hosts are rarely necessary and are challenging to time appropriately. Repeated fungicide applications according to the label from early July through August may help prevent infection.

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53. Valsa (Cytospora) Canker of Spruce

James A. Walla and Aaron D. Bergdahl; revised from James A. Walla and Frederick J. Crowe (Riffle and Peterson 1986)

Valsa canker is caused by the fungus *Valsa kunzei* (syn. *Leucostoma kunzei*). The imperfect stage is *Cytospora kunzei* (syn. *Leucocytospora kunzei*).

Hosts and Distribution

Many species of spruce (*Picea* spp.) are susceptible to Valsa canker, including blue (also called Colorado blue; *P. pungens*), Engelmann (*P. engelmannii*), Norway (*P. abies*), and white (*P. glauca*; including Black Hills seed sources) spruce. Valsa canker may also occur on Douglas-fir (*Pseudotsuga menziesii*), larch (*Larix* spp.), pine (*Pinus* spp.), and other conifers. In North America, it has been reported throughout the Upper Midwest and northeastern United States and adjacent Canada, and in the mountains of Colorado. It occurs in the Prairie Provinces, and the central and northern Great Plains states.

Symptoms and Signs

Branch dieback often occurs first on individual lower branches and then sporadically on other branches laterally and upward in the tree (fig. 53-1). In the northern Great Plains, the first outward symptoms usually occur in spring, when needles of branches girdled since late summer of the previous year begin turning grayish green (fig. 53-1A) and then light purple to brown (fig. 53-1B) as they dry out with warming temperatures. Branch girdling sometimes occurs from late spring to midsummer, resulting in development of the first outward symptoms during summer. One or more months after becoming discolored, needles drop from girdled branches. The youngest blue spruce twigs on dead branches typically remain orange for about one year after needles drop, and then become gray. Resin exudes from cankered branches and may drip onto noncankered branches.

Figure 53-1—Colorado blue spruce trees with Valsa canker. (A) Cankered branch with fading (gray-green) needles (arrow), early August; (B) Same branch in late September when needles have turned brown and started falling (arrow). Twigs remain orange for several months after needles have dropped (James A. Walla, Northern Tree Specialties, used with permission); (C) Serious damage by Valsa canker on blue spruce, showing recently dead branch with brown needles, branch that died the previous year with bleached gray twigs (arrow), and open areas where branches have been removed (Aaron D. Bergdahl, North Dakota Forest Service, used with permission).

The resin dries into white or light blue patches (fig. 53-2A). There is little external evidence of the canker margin, but it can be found by exposing the inner bark. Infected bark tissue and cambium are brown in contrast to the normal cream color of healthy tissue. The wood beneath infected bark is not discolored (fig. 53-2B). Branches that died from other causes may be colonized and contain fruiting bodies of *V. kunzei*, but resin rarely exudes from such branches. Resin often exudes from wounds.

Asexual fruiting bodies (pycnidia) of the fungus develop in infected bark, but are usually not visible on the bark surface. Gentle scraping of cankered bark will expose the small (1 to 3 mm diameter) black pycnidial stromata (fig. 53-2B). Yellow or orange spore masses or tendrils may exude from the pycnidia during wet weather. The spores (conidia) are clear (hyaline), sausage shaped (allantoid), one-celled, and 4 to 6 µm × 1 µm. Sexual fruiting bodies (perithecia) are sometimes associated with pycnidia, but they are usually on branches that have been dead for several years. Perithecia are smaller than pycnidia and are grouped in black stromata. Ascospores from perithecia are hyaline, allantoid, one-celled, and 5 to 9 µm × 1.5 µm.

Disease Cycle

Conidia are released during all seasons except winter, whereas ascospores are released only in spring and early summer. Both the conidial and ascospore stages are infective. Spores from fruiting bodies on cankered branches are spread to branches on the same or other trees by rainsplash, wind, humans, and likely insects and birds. Experimental infection has succeeded only through wounds. The fungus kills bark as it grows, and usually girdles infected branches and stems one or more years after infection. Cankers on girdled branches expand toward the stem until the entire branch is dead. Fruiting bodies form in infected bark. The fungus overwinters as fruiting bodies and mycelium in cankered bark, and spores are exuded from those fruiting bodies to complete the disease cycle.

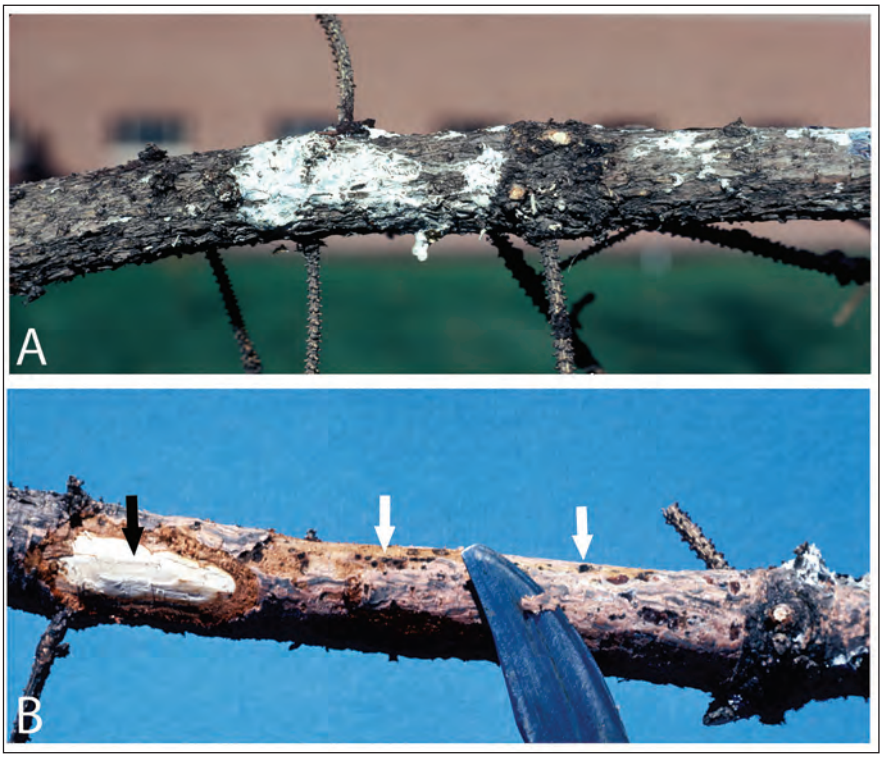

Figure 53-2—Valsa canker on blue spruce. (A) Resin from cankered branches, readily visible on dead bark a few months after infected needles are cast. (B) Black fruiting bodies (pycnidia) of V. kunzei (white arrows) in brown cankered inner bark of infected spruce branches, as revealed by gentle scraping. Wood beneath cankers is not discolored (black arrow) (A, B: James A.Walla, Northern Tree Specialties, used with permission).

Damage

Blue spruce is the most severely damaged host in the Great Plains, and serious damage is more common in the northern United States and in Canada. In most areas, branch cankering is the predominant damage. Cankers may develop on or grow into stems and result in top dieback, particularly on Norway spruce in some other areas. Damage may occur in ornamental, plantation, and windbreak settings. The disease destroys the symmetry of spruce trees; reduces their effectiveness in blocking wind, snow, and noise; and in time, reduces their vigor. Valsa canker rarely kills blue spruce but has the same effect when the trees are removed because of the damage. Damage is increasingly serious as trees get older and larger but may occur on trees less than 10 years old if they are stressed or near infected larger trees.

In some areas, Valsa canker is thought to damage only trees weakened by environmental stress, especially drought. In the northern Great Plains, Valsa canker commonly infects apparently healthy trees, but is more common and spreads more quickly on less vigorous trees.

Management

Healthy trees are less susceptible to damage, so trees should be managed for good vigor. If possible, water and fertilize trees early in the growing season as needed. Reduce chances of infection by preventing wounds and by maintaining good air circulation around the trees. Do not bring potentially infected branches into the area (for example, by discarding or storing spruce boughs or logs near healthy trees). Blue spruce are most susceptible to damage in the Great Plains, so consider planting other species or varieties if Valsa canker is a threat.

If trees become infected, prune out all diseased and dead branches and destroy or dispose of them offsite. Either prune to the trunk, or if only a small portion of a branch is affected, determine the proximal border of the canker and prune to the nearest healthy lateral branch beyond the canker. Major pruning is best done in late winter before spores are released, but may be done during dry periods whenever dead branches are found. It is critical to be aware that there will be asymptomatic cankers during the first round of pruning, so at least two rounds of pruning are required before prior infections are removed (suggested times are late winter and about one month after budbreak). Two consecutive years of thorough sanitation will substantially reduce future infection, but trees should be monitored annually thereafter to find and remove any additional cankered branches. Disinfect pruning tools with rubbing alcohol or 10 percent bleach after each cut. Protective and systemic fungicides have been recommended or labeled for control of Valsa canker on spruce, but efficacy information is not generally available.

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54. Western Gall Rust of Pines

James T. Blodgett and Kelly S. Burns; revised from Glenn W. Peterson and James A. Walla (Riffle and Peterson 1986)

Peridermium (*Endocronartium*) *harknessii* is a rust pathogen that causes woody swellings (galls) and cankers that deform branches and stems of hard pines (*Pinus* spp.). A less common name for this disease is pine-to-pine rust because unlike many rust diseases, it has no alternate host and therefore spreads directly from pine to pine.

Hosts and Distribution

The fungus infects many native hard pines including ponderosa (*P. ponderosa*), lodgepole (*P. contorta*), and jack (*P. banksiana*) pines. It also infects the exotic Scots (*P. sylvestris*) and mugo (*P. mugo*) pines. Western gall rust is found throughout pine forests of western and northern North America, in the northern Great Lakes region, and in scattered locations in eastern North America. It is more common in the western Great Plains, but can occur throughout the region. The western gall rust fungus often causes damage in nurseries, plantations, and landscape plantings, and can cause damage in natural stands.

Symptoms and Signs

Western gall rust is characterized by round or pear-shaped galls on branches (fig. 54-1), and rounded or targetlike cankers on stems (fig. 54-2). Galls are most conspicuous in spring and early summer when the surface ruptures, exposing bright yellow-orange spores (fig. 54-3). Witches'-brooms (bushy masses of branches) sometimes develop in affected trees (fig. 54-4).

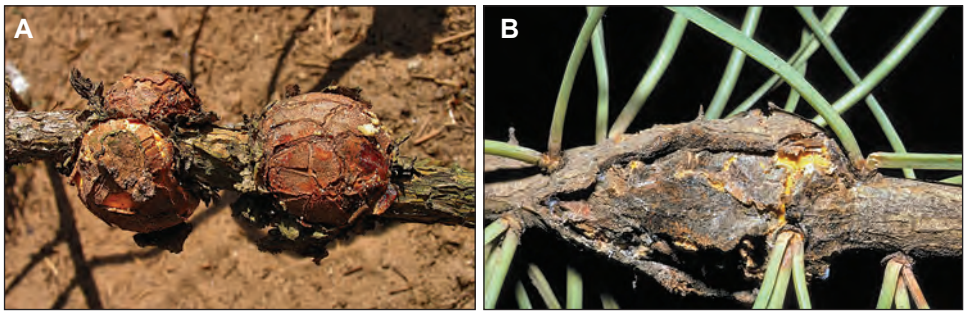

Figure 54-1—Branch galls of western gall rust: (A) Round, and (B) Pear shaped (Joseph O'Brien, U.S. Forest Service, Bugwood.org (left) and U.S. Forest Service, Bugwood.org).

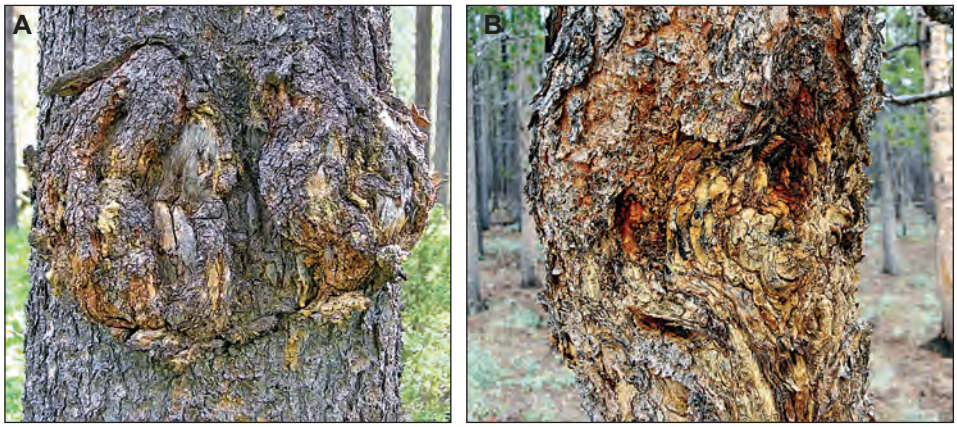

Figure 54-2—Cankers caused by western gall rust: (A) Swollen stem cankers in ponderosa pine, and (B) Sunken targetlike canker in lodgepole pine (James T. Blodgett, U.S. Forest Service).

Figure 54-3—Branch gall of western gall rust sporulating on lodgepole pine (James T. Blodgett, U.S. Forest Service).

Figure 54-4—Witches'-brooms in Scots pine branches caused by western gall rust (Robert L. Anderson, U.S. Forest Service, Bugwood.org).

Disease Cycle

White to orange pustules (aecia-like[1]) full of yellow-orange spores (aeciospore-like[1]) form on galls or, less commonly, at the edges of stem cankers in late spring or early summer. The aecia-like pustules rupture, releasing spores that disperse by wind in May and June. Spores germinate during moist, cool weather and infect expanding needles and young elongating shoots of pines. Infection rarely occurs through wounds on older branches and stems. Stem cankers are usually initiated by infected side branches or branch galls adjacent to the main stem (fig. 54-5). Mass infections tend to occur in wave years when weather is favorable for infection. Galls form mostly in the summer following the year of infection. Sporulation typically occurs in the second or third year after infection, and can continue for a few years. The rust pathogen stimulates rapid division of host cambial cells, which results in gall formation.

Damage

Western gall rust affects pines of all ages, causing growth loss, branch death, deformity, and occasionally mortality. Mortality is most common in seedlings and saplings as galls can quickly girdle small stems. Abundant galls on branches may cause tree stunting. Pines in landscape and Christmas tree plantings may lose value owing to

[1] Aecia-like and aeciospore-like indicate morphology that parallels typical demicyclic heteroecious rusts. The terms aecia and aeciospore do not describe the life stages of typical *Peridermium* species.

Figure 54-5—Stem canker that was initiated by a branch gall of western gall rust (James T. Blodgett, U.S. Forest Service).

stunting, witches'-brooms, and branch death. Stem cankers can severely deform larger trees, resulting in wind breakage or reduced merchantable volume, but they seldom girdle large trees. Uninfected trees of the same species are often common near similar-aged trees with many galls or cankers, or both. This is likely due to genetic variation in resistance to the disease in the population.

Management

Management of western gall rust can be difficult given the lag between infection and symptom development. Management options that can reduce disease impacts include sanitation, removal of the infection source, using disease-free planting stock, planting resistant hosts or nonhost species, and fungicide treatments.

Sanitation by removal of all trees with infections and careful inspection of remaining trees is often the only option in established stands. Eradication of the fungus by gall removal is not feasible in mature trees. In areas where western gall rust incidence is high, it may be necessary to replant with resistant species. Trees with stem cankers can be hazardous in recreation areas and should be given priority for removal.

In nurseries, the most effective and economical management method is the removal of nearby sources of infection. Gall-bearing pines within 0.5 mile of nurseries should be pruned free of all galls or felled. Destroy gall-bearing seedlings before sporulation.

Prevention is the best method of management in field plantings. Examine trees for galls before planting and again the following two springs. Obtain planting stock only from a nursery that is free of western gall rust.

The use of resistant hosts or nonhost species can substantially reduce or eliminate damage from western gall rust. Ponderosa pine from different geographic sources display considerable variation in susceptibility to this rust pathogen. Some highly resistant sources have been identified, but commercially certified, resistant stock may be difficult to find. When planting in areas where western gall rust is known to be a problem, use nonhost species that are adapted to the site.

In some states, fungicide treatments can be used for Christmas trees and high-value landscape trees. Consult your state or local extension agent regarding appropriate registered fungicides.

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55. Pine Wilt

Timothy C. Todd and Mark O. Harrell

Pine wilt is caused by the pinewood nematode (PWN), *Bursaphelenchus xylophilus*, a microscopic roundworm (fig. 55-1) that feeds on the epithelial cells that line the resin canals. The nematode also feeds on fungi that proliferate in the wilting tree and in the pupal chambers of its beetle vector, pine sawyers of the genus *Monochamus* (fig. 55-2). Originally described from specimens collected from longleaf (*Pinus palustris*) and shortleaf (*P. echinata*) pines in Texas, Louisiana, and Virginia in 1934, PWN was first identified (as *B. lignicolus*) as the causal agent of pine wilt in Japanese pine forests in 1971. In 1979, PWN was reported as the causal agent of pine wilt disease in Austrian (*P. nigra*) and Scots (*P. sylvestris*) pines in Missouri. The disease has caused extensive losses to these exotic pine species in the Midwest and Great Plains.

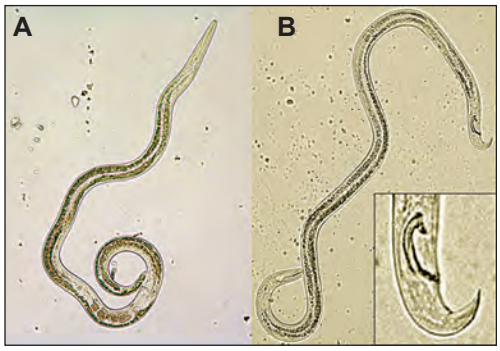

Figure 55-1—Pinewood nematodes: (A) Female, and (B) Male adult. Insert shows the distinctive shape of the male spicule, which is characteristic of the species (Timothy C. Todd, Kansas State University, used with permission).

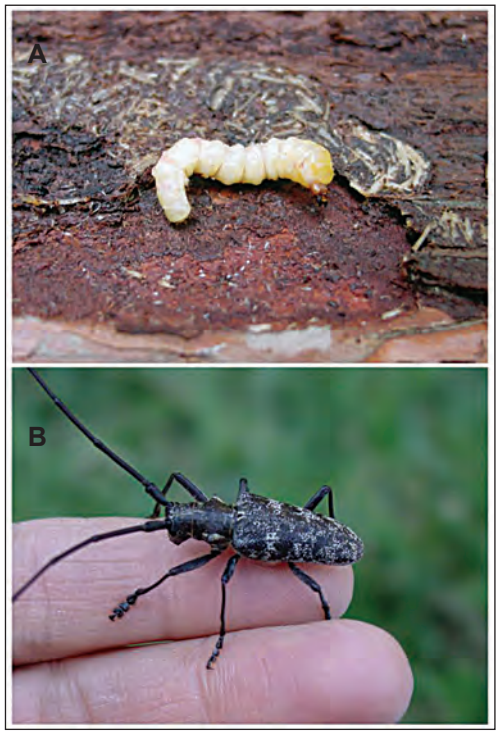

Figure 55-2—Pine sawyer (A) larva, and (B) adult beetle ((A: Mark O. Harrell, Nebraska Forest Service, University of Nebraska, used with permission; B: Laurie Stepanek, Nebraska Forest Service, used with permission).

Hosts and Distribution

B. xylophilus has been isolated from numerous species of pine, and occasionally from other conifer species, including larch (*Larix* spp.), fir (*Abies* spp.), and spruce (*Picea* spp.). Pine wilt, with PWN as the primary agent, has not been documented for conifers other than pines, however, and many of these, including Norway (*P. abies*) and blue (*P. pungens*) spruce, balsam fir (*A. balsamea*), and Douglas-fir (*Pseudotsuga menziesii*), are recommended as replacement trees for Scots and Austrian pines in the Midwest. Some uncertainty also remains about the host status and susceptibility of individual pine species, but species in the *Australes* subsection (for example, loblolly [*P. taeda*], longleaf, pitch [*P. rigida*], and slash [*P. elliottii*]) generally are highly resistant. In contrast, species in the *Pinus* subsection (for example, Austrian, Japanese black [*P. thunbergii*], mountain [*P. uncinata*], and Scots) generally are highly susceptible. In the Midwest, pine wilt most commonly is associated with Scots and Austrian pines, with the primary impact on Scots pine in landscape plantings and windbreaks (fig. 55-3). Categorization of resistance and susceptibility in pine species is further confounded by variability in host preference, pathogenicity, and molecular phenotype among PWN populations in both the United States and Japan. Virulent and avirulent pathotypes appear to be widespread and there is evidence for interbreeding between them.

Figure 55-3—Pine wilt in Scots pine windbreak (Mark O. Harrell, Nebraska Forest Service, University of Nebraska, used with permission).

The PWN is widely distributed throughout the United States, but incidence of pine wilt is concentrated in Iowa, Illinois, Missouri, Kansas, Oklahoma, and Nebraska. Isolated outbreaks of pine wilt associated with the transport of infested firewood have occurred in western Kansas, and the disease recently has been reported on Scots pine in Colorado. Presence of PWN is not always indicative of pine wilt, however, as the nematode can be introduced into dead or dying trees during vector oviposition, and as discussed above, avirulent pathotypes are known to occur.

Studies of the genetic diversity among PWN isolates have concluded that the nematode originated in North America. It was introduced into Japan early in the 20th century, and subsequently spread to China, South Korea, and, more recently, Portugal. *B. xylophilus* and its vectors are listed as A1 quarantine pests by the European and Mediterranean Plant Protection Organization (EPPO).

Symptoms and Signs

Reduction in oleoresin flow and rapid tree death are the characteristic symptoms of pine wilt. Initially needles on affected branches change in color to faded green, then to light brown (fig. 55-4). The discoloration may be restricted to individual branches at first, but it progresses rapidly, often spreading to the entire tree within a few weeks. Trees often die between midsummer and fall, but death can occur at other times of the year. Trees beginning to show symptoms in fall may not become completely brown until after winter, when warmer weather returns.

Rapid death of pines can also be caused by environmental stresses. The presence of PWN can be confirmed by clinical diagnosis of wood samples collected from the base of lower limbs soon after symptoms appear.

Disease Cycle

The life cycles of the PWN and its beetle vectors are closely synchronized. The most widespread and efficient vectors of PWN in the United States are the nearly identical Carolina (*M. carolinensis*) and Southern (*M. titillator*) pine sawyer and the white-spotted sawyer (*M. scutellatus*). The nematode may be transmitted to healthy trees during maturation feeding by newly emerged beetles or to dead or stressed trees during oviposition by mature beetles. Transmission efficiency during *M. carolinensis* feeding has been demonstrated to approach 50 percent of fed-upon branches for mature Scots pines under field conditions. Actual numbers of transmitted nematodes are correlated with numbers of nematodes carried by the beetle but generally appear to be low; infection results from the introduction of a small number of nematodes through one or more feeding wounds.

Figure 55-4—Symptom progression in Scots pine from (A: Mark O. Harrell, Nebraska Forest Service, University of Nebraska, used with permission; B: Nebraska Forest Service, University of Nebraska, used with permission).

The progression of wilt symptoms is accompanied by the emission of volatiles that attract mature beetles to diseased trees, where mating and oviposition occur. Proliferation of fungi in the dying tree provides not only a secondary food source for PWN populations that previously fed upon living plant tissues but also a primary food source for any nematodes that may have been transmitted during oviposition. Pupal chambers, in particular, harbor a diverse assemblage of microorganisms ranging from blue-stain fungi, which are disseminated by *Monochamus* and other beetles and serve as an important food source for the nematode, to nematophagous fungi that capture and kill nematodes and have the potential to reduce the number of nematodes carried by beetles emerging from the wood.

Desiccation and nematode overpopulation in the wood eventually induce the formation of the third juvenile dispersal stage of the nematode, which aggregates around the pupal chambers of vector beetles. Emergence of beetles from the pupal chamber is associated with the release of volatiles that stimulate the nematodes to molt to the fourth juvenile dispersal stage, which then enters the spiracles and trachea of the insect. Adult beetles emerge throughout the spring and early summer. The life cycle of *M. carolinensis* is temperature dependent, resulting in two generations per year in the southern United States but only one generation every two years in the northern United States.

Damage

Unlike other nematode diseases of conifers, the primary impact of pine wilt is not on seedlings but on mature trees in landscape plantings and windbreaks. Susceptible Scots pines can be killed within a few weeks or months. Pathogenicity of the nematode is determined by rates of transmission, dispersal, and reproduction within healthy trees, and by the interaction of secreted and surface coat proteins with host tissue cells. Water deficiency is the ultimate factor in rapid tree mortality, but the proximate cause appears to be the induction of a hypersensitivelike response to nematode feeding in susceptible trees.

Management

Traditional control practices have focused on sanitation involving the timely removal and destruction of trees killed by pine wilt. This practice eliminates the breeding habitat of the beetle vector and prevents nematode transmission to healthy trees. More recently, demonstration of effective PWN control using preventive injections has resulted in the emergence of several new products and injection systems. The active ingredient in these

products is abamectin, a natural fermentation product of the soil bacterium *Streptomyces avermitilis* with nematicidal properties (enhanced gamma-aminobutyric acid [GABA] activity). Abamectin injections in PWN-inoculated, 13- to 20-foot-tall Scots pine trees have been observed to increase the survival rate of treated trees to 75 percent, compared to 25 percent in untreated control trees, up to one year after injection. Similarly, abamectin injections in mature trees exposed to a natural epidemic increased the survival rate of treated trees to 96 percent, compared to 33 percent in untreated control trees, up to two years after injection. Curative injections after symptoms have started appearing, in contrast, are ineffective. Regardless of the efficacy of preventive injections, avoid new plantings of Scots and Austrian pines wherever possible.

Wood chips from PWN-infested trees can be used as mulch, but should be used only around nonsusceptible tree species and should not be placed against fresh wounds on pines. Research has shown that the nematode can survive in infested chips for up to 12 weeks and is capable of infecting wounded roots and stems that are in contact with the mulch.

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56. Root Parasitic Nematodes in Junipers and Pines

Timothy C. Todd and Jon A. Appel; revised from Glenn W. Peterson and Jerry W. Riffle (Riffle and Peterson 1986)

The root-lesion nematode *Pratylenchus penetrans* was the most frequently encountered plant-parasitic nematode in a 1960 survey of Nebraska tree nurseries. Damage from this nematode was reported for Austrian (*Pinus nigra*) and ponderosa (*P. ponderosa*) pine seedlings, but was not observed for windbreak stands of pines and eastern redcedar (*Juniperus virginiana*). In a 2004 to 2006 survey of windbreaks in Kansas[1], root-lesion nematodes were recovered from 25 percent of pine stands and 35 percent of eastern redcedar stands (table 56-1), but, again, damaging population densities were not observed.

Hosts and Distribution

Pratylenchus penetrans (fig. 56-1) is a cosmopolitan pest in North America, with a host range that encompasses grain crops, vegetables, fruit trees, and woody ornamentals. In central Nebraska, this nematode was recovered from roots of eastern redcedar, Rocky Mountain juniper (*J. scopulorum*), white (*Picea glauca*) and blue (*P. pungens*) spruce, Austrian pine, and ponderosa pine.

Nematodes commonly associated with conifers in Kansas windbreaks include pin (*Paratylenchus*) and spiral (*Helicotylenchus*) for eastern redcedar, and dagger (*Xiphinema*) and spiral for Austrian and ponderosa pines (table 56-1). These nematodes also were prevalent in hardwood stands (see chapter 40).

Table 56-1—Prevalence and abundance of nematode genera recovered from eastern redcedar and pine stands during a three-year survey of 81 Kansas windbreaks

Nematode common name (genus)	Prevalence (percent)		Mean (maximum) number of nematodes/100 cm^3 soil	
	Redcedar (n=46)	Pine (n=32)	Redcedar (n=46)	Pine (n=32)
Dagger (*Xiphinema*)	24	50	7 (60)	16 (130)
Lance (*Hoplolaimus*)	0	0	0 (0)	0 (0)
Pin (*Paratylenchus*)	57	38	103 (1,510)	34 (590)
Ring (*Mesocriconema*)	2	16	0 (10)	3 (50)
Root-lesion (*Pratylenchus*)	35	25	12 (90)	6 (50)
Spiral (*Helicotylenchus*)	46	53	31 (420)	45 (520)
Stunt (*Tylenchorhynchus*)	28	34	7 (100)	8 (90)
Stubby-root (*Paratrichodorus*)	0	3	0 (0)	0 (10)

[1]Data on file with the Kansas Department of Agriculture, Topeka, KS.

Figure 56-1—Adult female Pratylenchus penetrans (Timothy C. Todd, Kansas State University, used with permission).

Symptoms and Signs

Aboveground symptoms of nematode damage are most noticeable on nursery seedlings and typically manifest as irregular patches of stunted plants, often accompanied by signs of nutrient deficiency. Symptoms on roots are mostly nonspecific and may consist of formation of woody swellings (galling), restricted root development, and root necrosis. Root-lesion nematodes may produce necrotic lesions in the cortical tissue of feeder roots and can predispose plants to root rots and other disease complexes. Nematode extraction from soil and roots is always necessary to confirm a diagnosis of nematode damage.

Disease Cycle

Root-lesion nematodes are migratory endoparasites that enter and feed in the cortex of the feeder roots of host trees. These nematodes hatch as second-stage juveniles from eggs laid in or near host roots and develop through two additional juvenile stages into the adult stage, typically within four to six weeks, depending on temperature and moisture conditions. Migration between roots and soil can occur continuously, but movement into the soil environment is most pronounced during host senescence.

Dagger nematodes are ectoparasites with elongate stylets that facilitate prolonged feeding on root cortical and stelar tissues. The life cycle of dagger nematodes is longer than that of root-lesion nematodes, requiring one to two years for completion. Reproduction and population increase also are slower; nonetheless, perennial hosts such as trees provide ample time for damaging populations to develop.

Damage

Root-parasitic nematode damage in conifers has been observed primarily for seedlings in nurseries. Root-lesion nematodes were associated with extensive damage to redcedar seedlings in a Nebraska nursery, but were not associated with obvious damage to established pines, junipers, or spruce in windbreaks in Kansas and Nebraska. Nematodes known to damage conifer seedlings outside of the Great Plains include the needle nematode *Longidorus americanus* and the stunt nematode *Tylenchorhynchus ewingi* in pine nurseries in the southern United States, and *P. penetrans* and the dagger nematode *X. bakeri* in spruce and fir nurseries in the Pacific Northwest.

X. americanum population densities above 100 individuals/100 cm^3 (6 cubic inches) soil were associated with poor health of pines in Kansas windbreaks. Similarly high population densities of this nematode also were indicative of poor tree health in hardwood windbreak stands (see chapter 40).

Management

Nematode management options for windbreaks are limited. Weed control may restrict nematode population size, and cultural practices that promote tree health may increase tolerance to nematode feeding pressure. Combine routine fumigation of seedling production areas with sanitation practices and rotation with nonhost cover crops to avoid damage in nurseries. Most soil fumigant alternatives to methyl bromide have been shown to provide adequate control of plant-parasitic nematodes, but all fumigants provide only temporary control of nematodes and must be used routinely.

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Glossary (with plurals in parentheses)

ABAXIAL: referring to the bottom surface of a leaf.

ACERVULUS (ACERVULI): a small subcuticular or subepidermal cushionlike asexual fruiting body, without a covering of fungus tissue, producing conidia in a mass, which escape through a break in the host tissue.

ACIDIC: having a pH below 7: containing or having the properties of an acid.

ADAXIAL: referring to the upper side of the leaf.

AECIOSPORE: one of several kinds of spores produced by a rust fungus. Formed in and released from a fruiting structure called an aecium.

AECIUM (AECIA): a cuplike fruiting structure produced by certain rusts, in which chains of spores (aeciospores) are developed.

AGAR: a substance from certain red algae used to make culture media into gels upon which organisms are grown.

ALKALINE: characterized by high pH. Soils with pH levels above 7.0 are considered alkaline; greater than 8.5 are considered highly alkaline.

ALLANTOID: slightly curved with rounded ends; sausagelike in form.

ALPHA-SPORE: a fertile spore of the asexual stage of the Diaporthaceae (*Phomopsis*) that is fusiform to oblong and biguttulate (having two oil droplets inside the cell); a beta-spore, usually hook shaped, may be produced in addition.

ALTERNATE HOST: one or the other of the two unlike host plants parasitized by a heteroecious (involving two hosts) fungus such as a typical rust fungus, for example, either the juniper or apple host of the cedar-apple rust fungal disease complex.

ANAEROBE: a microorganism that can live and grow where there is no free oxygen.

ANAMORPH: the asexual form of a fungus (the form that is characterized by conidiomata).

ANNULUS: a ringlike partial veil around the stem of a fruiting body after expansion of the pileus (cap).

ANTHRACNOSE: a type of plant disease that typically is a leaf and twig blight. Common on many hardwoods.

APOTHECIUM (APOTHECIA): a cuplike or saucerlike sexual fruiting body that produces ascospores.

ARCHAEA: a kingdom of single-celled prokaryotic microorganisms, once considered bacteria, that inhabit a broad range of habitats.

ASCOMYCETE: fungi belonging to the Ascomycota, the largest group of fungi and for which the ascus is the main diagnostic characteristic.

ASCOSPORE: a spore produced in an ascus (see ASCUS).

ASCUS (ASCI): a saclike cell of the perfect (sexual) stage of the Ascomycetes in which ascospores (usually eight) are produced.

ASEXUAL STAGE: a stage in the life cycle of a fungus in which spores are produced without a previous sexual fusion. Also called IMPERFECT STAGE.

BASIDIOMYCETE: any fungus in the phylum Basidiomycota producing spores on a basidium.

BASIDIOCARP: spore-producing structure of basidiomycetous fungi.

BASIDIOSPORE: the spore produced by the sexual stage of the Basidiomycetes.

BASIDIUM (BASIDIA): a cell, usually terminal, in which nuclear fusion and meiosis occur and each of the four haploid nuclei pass into one of four forming spores.

BETA-SPORE: see ALPHA-SPORE.

BLIGHT: a general term for a plant disease causing rapid death or dieback.

BRASH: the characteristic that causes a piece of wood to break abruptly across the grain, with relatively low resistance to such breaking.

BROOM: see WITCHES'-BROOM.

BROWN ROT: a type of wood decay caused by fungi that degrade cellulose but not lignin, do not produce extracellular phenoloxidases, and generally give negative oxidase tests. Decayed wood appears brown, fractures into cubical pieces, and is crumbly (not to be confused with the disease Brown Rot of Stone Fruits).

CALLUS: undifferentiated cell formation at the site of injured tissue. Note: the term is often used incorrectly to refer to wound closure with woody tissue. See WOUNDWOOD.

CANKER: a definite, relatively localized necrotic lesion primarily of the bark and cambium.

CELLULOSE: long chains of sugar molecules and the main component of the plant cell wall giving wood its mechanical strength.

CHASMOTHECIUM: see CLEISTOTHECIUM.

CHLOROSIS: an abnormal yellowing of the foliage.

CHLOROTIC: abnormally yellow.

CIRRHUS (CIRRUS, CIRRHI): tubelike mass of spores exuded from a fungal fruiting structure. Also called a TENDRIL or sporehorn.

CLAMP CONNECTION: a hyphal outgrowth formed by certain fungal species functioning to allow the transfer of fungal nuclei.

CLEISTOTHECIUM (CLEISTOTHECIA): a closed fruiting body, without an opening, containing asci.

CONIDIOGENOUS CELL: a cell from or within which a conidium is directly produced.

CONIDIOMA (CONIDIOMATA): A specialized multihyphal, conidia-bearing structure.

CONIDIOPHORE: a specialized hypha-bearing conidium.

CONIDIUM (CONIDIA): a spore formed asexually, usually at the tip or side of a hypha.

CONK: a type of fruiting (spore-forming) structure, usually formed by certain wood decaying fungi in the Basidiomycetes. It is often bracketlike and is also referred to as "sporocarp," "sporophore," "FRUITING BODY," "carpophore," "fructification," "basidiocarp," "punk," "bracket," and "shelf."

CONTEXT (of CONK): the inner tissue of the pileus (cap), that is, the tissue lying between the upper surface and the tube or pore layer. Sometimes designated as the "trama" of the pileus.

COVER CROP: a crop, natural or introduced, that is grown alternately with the main crop. Used to prevent erosion, improve soil characteristics, and to favor depletion of plant pathogenic fungus and nematode populations.

CULTIVAR: a group of cultivated plants distinguished by any useful, reproducible character; a cultivated variety.

CUTTING: detached portion of stem or other plant part that, when rooted, produces a whole plant.

DAEDALEOID-PORES: pores with elongated and sinuous mouths.

DECAY: the decomposition of plant tissue by fungi and other microorganisms.

DEFOLIATION: loss of current year's or past years' foliage.

DELIQUESCENT: lack of terminal dominance on branch ends resulting in terminal and lateral twigs that grow about the same amount annually, often restricted to one or a few branches or to sprouts along the trunk.

DEMICYCLIC: Rust life cycle lacking the uredial (repeating) stage.

DEXTRINOID: stained yellowish- or reddish-brown by Melzer's iodine.

DESSICATE: to dry (dehydrate) thoroughly.

DIEBACK: the death of parts of a tree or plant, usually from the top downward.

DIMITIC: referring to fungi that have two types of hyphae.

DISEASE: unfavorable change of the function or form of a plant from normal; caused by a pathogenic agent or unfavorable environment.

DISEASE CYCLE: the chain of events in the development of a disease.

DISSEPIMENTS: the pore walls of polypores.

DUPLEX: (of a context of a sporocarp) in two layers.

ECTOPARASITE: a parasite living outside of its host.

EFFUSED: stretched out flat.

EFFUSED-REFLEXED: spread out over a substratum and turned back at the margin to form a pileus (cap).

ELLIPSOID: In the shape of an ellipse.

ENDOPARASITE: a parasite living inside of its host.

ENDOPHYTIC: describes an organism (endophyte), usually a fungus or bacteria, that is able to live within a plant for at least part of its life cycle without causing disease.

EPIDEMIC: pertaining to a disease that has built up rapidly in a population or area and reached injurious levels.

EPIDERMIS: the outermost layer of cells on the primary plant body.

EPIPHYTE: an organism that lives nonparasitically on the surface of a plant.

ERADICANT: a fungicide, applied to foliage of a plant, that has a direct effect upon organisms which have already invaded the host.

EXUDATE: matter that oozes out or is secreted.

FACULTATIVE PARASITE: an organism that is normally saprophytic but that is capable of living as a parasite only when unfavorable conditions predispose the host so that it is unusually susceptible.

FALLOW: referring to cultivated land allowed to lie idle or unplanted during the growing season.

FASCICLE SHEATH: a sheath around the base of a cluster or bundle of needles.

FEEDER ROOTS: succulent, actively growing rootlets of plants.

FLAGELLUM (FLAGELLA): a whiplike part of some cells, especially of certain bacteria, that is an organ of locomotion.

FOLIAGE: leaves of a plant or tree.

FRUITING BODY: any of a number of kinds of reproductive structures that produces spores.

FUNGICIDE: chemical that is toxic to fungi.

FUNGUS (FUNGI): any of a number of organisms considered by some authorities to be lower plants which lack chlorophyll.

FUSIFORM: spindlelike, narrowing toward the end.

GALL (BURL): a pronounced swelling on a woody plant caused by certain insects and disease organisms.

GIRDLE: to destroy or remove the tissue, particularly living tissue in a rough ring around a stem, branch, or root.

GLABROUS: smooth, not hairy.

GLOBOSE: spherical or almost so.

GUMMOSIS: the exudation of gummy substances.

HAUSTORIUM (HAUSTORIA): absorbing organ originating on a hypha of a parasite and penetrating into a cell of the host.

HEARTWOOD: region at the center of woody plants mainly composed of dead cells, typically darker and harder than the SAPWOOD.

HEMICELLULOSE: polysaccharide component of plant cell walls.

HETEROECIOUS: Describing rust fungi requiring two unrelated hosts to complete their life cycle.

HIRSUTE: having long hairs.

HISPID: having hairs or bristles.

HOST: The organism on or in which a pathogen exists.

HOST RANGE: all hosts that a particular pathogen attacks.

HOST-SPECIFIC: a term used to describe those pathogens that attack hosts of only a certain species or cultivar.

HYALINE: transparent, having no color.

HYMENIUM: the general region of a sporocarp bearing basidia, that is, the layer of tubes. Alternatively, the actual spore-producing layer, made up of basidia and whatever type of non-reproductive tissue that may be present with them and forming a layer lining the inside of the tubes.

HYPHA (HYPHAE): a fungal filament; collectively called mycelium.

HYSTEROTHECIUM: an elongated sexual fruiting structure splitting longitudinally to release spores; of ascomycetes and lichens.

IMPERFECT STAGE: that part of the life cycle of a fungus in which only conidia and no sexual spores are produced. Also called ASEXUAL STAGE.

INFECT: to invade and persist in an organism potentially causing disease.

INFECTION COURT: the area in which the pathogen first established itself on or in the host.

INFEST: to be present within an area in such numbers as to be a disease hazard.

INOCULATE: to place an organism or substance on or in another potential host organism or a substratum.

INOCULUM: the organism or substance, e.g., spores of a pathogen, that can inoculate an organism or substratum.

INTERNODE: the portion of the stem between two nodes.

IN VITRO (or VITRO): taking place in a test tube, laboratory, or otherwise outside of a living organism.

IN VIVO (or VIVO): taking place in a living organism.

JUVENILE LEAVES: foliage characteristic of the immature form of leaves for species that have multiple leaf forms, e.g., awl-shaped needles of junipers.

LAMELLATE: vertical plates on the underside of the pileus.

LATENT INFECTION: an established infection that does not show its presence.

LEAF ABSCISSION: the separation of leaves from plants caused by the weakening of cell walls in a special layer (abscission layer) at the base of the petioles.

LEAF PETIOLE: the slender, usually cylindrical portion of a leaf, that supports the blade and is attached to the stem.

LEAF SPOT: a disease symptom or a type of leaf disease; typically characterized by distinct lesions on leaves.

LESION: a defined necrotic area.

LIFE CYCLE: in the case of diseases, the stage or series of stages between the formation of one form of a pathogen (e.g., spore), development of disease, and reappearance of the same form of the pathogen.

LIGNIN: A complex polymer that with cellulose is a major component of wood and the cell walls of plants, providing rigidity.

LOCULE: cavity in a stroma

MACROCONIDIA: the larger of two types of conidia by certain fungi, such as *Fusarium* species.

MICROCONIDIA: the smaller of two types of conidia produced by certain fungi.

MICROSCLEROTIA: small, melanized (containing dark brown pigments) resting structures produced by some fungi.

MONOMITIC: having one type of generative hypha.

MULTISEPTATE: characterized by having several cross walls that divide hyphae or spores into a number of separate cells.

MUSHROOM: any of various rapid-growing, fleshy fungi that typically have a stalk capped with an umbrella-like top.

MYCELIUM (MYCELIA): a mass of hyphae that forms the vegetative filamentous body of a fungus.

MYCOPARASITIC: describing a fungus-parasitizing organism.

MYCOPLASMA-LIKE ORGANISM (MLO): an organism with apparent features of previously known mammal-infecting bacteria called mycoplasmas, now termed PHYTOPLASMAS, a group of plant-infecting bacteria distinct from mycoplasmas.

MYCORRHIZA (MYCORRHIAZE): type of fungus that engages in a symbiotic (sometimes weakly pathogenic) lifestyle with a vascular plant, growing on or in plant roots, aiding in water and nutrient uptake and receiving carbon in exchange.

NECROSIS: death of plant cells, usually resulting in darkening of the tissue.

NEMATODE: a roundworm with a long, cylindrical, unsegmented body.

NODULOSE: broad-based, blunt, wart-like.

NONSELECTIVE HERBICIDES: herbicides used for control of all vegetation (grass and broadleaf plants).

OSTIOLE: small opening or pore in fungal fruiting structures where fungi release mature spores.

OVIPOSITION: the depositing (laying) of eggs through a specialized tubular organ called an ovipositor (typically in insects).

PARASITE: an organism living on or in, and nourished by, another living organism.

PATHOGEN: an organism that causes a disease.

PATHOGENIC: capable of causing a disease.

PEDUNCLE: the supporting axis of a single flower or a flower cluster.

PERENNIAL: continuing growth from year to year.

PERFECT STAGE: the stage in which the sexual spores are produced. Also called sexual stage.

PERIDERM: the outer protective layer in older stems, consisting of the phellogen and its derivative tissues, phellem and phelloderm.

PERITHECIUM (PERITHECIA): a closed flasklike sexual fruiting body formed by certain Ascomycetes and in which ascospores are produced.

PHLOEM: the tissue of the inner bark responsible for the transport of sugars and growth regulators.

PHYTOPLASMA: a cell-wall-less bacterium that lives in plant phloem tissue and certain insects, causing disease in both hosts, with various taxa causing many plant diseases; formerly known as MYCOPLASMA-LIKE ORGANISM (MLOs).

PILEATE: having at least a portion of a conk with a sterile upper surface over a fertile lower surface (see PILEUS).

PILEUS (PILEI): the upper surface of a conk/sporocarp.

PLEOMORPHIC: the condition in which a fungus has two or more very different morphological forms.

POLYPORE: a group of fungi that form fruiting bodies with pores or tubes on the underside.

PORE SURFACE: the lower surface of a sporocarp in specimens mature enough to have a tube layer. It is the surface at which the pores open.

POST-EMERGENCE HERBICIDES: herbicides applied after germination and establishment of green plants.

PREEMERGENCE HERBICIDES: herbicides applied before seedling emergence, usually designed to inhibit germination.

PRIMARY INFECTION: infection of a host by primary inoculum (first inoculum of a season), for example, from overwintering spores.

PROTECTANT: pesticide applied to foliage of a plant in advance of a pathogen in order to prevent infection.

PRUNE: to remove dead or living parts from a plant to improve its form or condition.

PSEUDOTHECIUM: a sexual fruiting structure (ascocarp) containing randomly organized asci.

PUSTULE: a small, sometimes colored, blisterlike swelling.

PYCNIDIUM (PYCNIDIA): an asexual type of fruiting body, typically flask shaped, in which asexual spores or conidia are produced.

PYRIFORM: pear-shaped.

REFLEXED: describing a decay conk/sporocarp that is mostly resupinate, but with a small portion at the upper end that is curved outward, creating a small pileus.

RELATIVE HUMIDITY: the amount of moisture in the air as compared with the maximum amount that the air could contain at the same temperature, expressed as a percentage.

RESISTANCE: the ability of a host to retard, suppress, or prevent infection or colonization by a pathogen; also the functioning of attributes or processes that do so.

RESUPINATE SPOROCARP: describing a decay conk/sporocarp in which the entire structure lies flat on the substratum, that is, without forming a bracketlike or shelflike body (not having a pileus and having only a pore surface surrounded by a narrow margin).

RHIZOMORPH: a thick strand of vegetative hyphae in which the hyphae have lost their individuality; important in underground expansion of some types of root rot.

RHIZOSPHERE: the soil near a living root.

RIMOSE: cracked, or cracked by radial fissures.

ROT: see DECAY.

SAPROBE: fungus that derives nutrients from dead and decaying organisms.

SAPROPHYTE: an organism using dead organic material as food.

SAPWOOD: the wood beneath the bark that contains living tissue and functions in water and mineral transport and starch storage.

SCLEROTIUM (SCLEROTIA): a firm, frequently rounded, multicellular resting structure produced by fungi.

SECONDARY INFECTION: infection that commonly occurs when the infecting organism produces another crop of spores or infective bodies after a host has already become diseased.

SELECTIVE HERBICIDES: herbicides designed to target specific types of plants, while leaving the desired types of plants unharmed.

SEPTATE: having cross walls that divide hyphae or spores into a number of separate cells.

SEPTUM (SEPTA): a cross wall that divides a hypha or spore into two or more distinct cells.

SESSILE SPOROCARP: describing a decay conk that takes the form of a knob, bracket, or shelf. The conk has no stem or stalk, and the point of attachment to the substratum is typically lateral. Pileate, without a stem, and not effused; typical conk form.

SETA (SETAE): conical or lance-shaped, brown, sterile organs found in the hymenium of some fungal species.

SEXUAL STAGE: the stage in the life cycle of a fungus in which spores are produced after sexual fusion. Also called perfect stage.

SHEARING: cutting foliage and stems from trees with shears.

SHELTERBELT: see WINDBREAK.

SIGNS OF A PATHOGEN: any observable parts of a pathogen.

SILVICULTURE: the art of establishing, growing, and regenerating a forest.

SPERMATIUM (SPERMATIA): a nonmotile, uninucleate, sporelike male structure which empties its contents into a receptive female structure during a type of sexual reproduction known as plasmogamy.

SPERMAGONIUM (SPERMAGONIA): a structure that contains minute, sporelike bodies which in some cases have proved to be functional spermatia.

SPORE: a reproductive or resting structure of the fungi, other lower plants, and some bacteria.

SPORE HORN: a thread-like gelatinous mass of forced-out spores. Also called TENDRIL or CIRRHUS

SPOROCARP: structure on which spore-producing structures, such as basidia or asci, are borne.

SPORODOCHIUM (SPORODOCHIA): a cushion-shaped stroma covered with conidiophores.

SPORULATE: to produce spores.

SPUR LEAVES: foliage characteristic of short (spur) branches.

STOMA (STOMATA): a pore in the leaf epidermis, surrounded by two guard cells, leading into an intercellular space within the plant.

STOMATAL PIT: the space above a stoma that is sunken below the leaf surface.

STRESS: a negative impact on tree health caused by biotic or abiotic factors leading to disruption of normal physiological processes supporting normal growth.

STROMA (STROMATA): a mass of fungal hyphae packed together to form a hard crust in or on which fruiting bodies are formed.

SULCATE ZONES: grooved regions of fungal fruiting bodies.

SUSCEPTIBLE: unable to resist attack by an organism or influence by a nonliving agent.

SYMPTOM: the evidence of disturbance in the normal development and function of a host plant, for example, chlorosis, necrosis, galls, brooms, and stunting.

SYNNEMA (SYNNEMATA): a specialized, spore-bearing structure composed of fused, elongated multihyphal conidiophores.

SYSTEMIC: able to move or be transported internally in a plant.

SYSTEMIC FUNGICIDES: chemicals and natural products that are absorbed into the tissues of plants and are toxic to fungi.

TELEOMORPH: the perfect (sexual) stage of a pleomorphic fungus, characterized by ascomata and basidiomata.

TELIOSPORE: the fourth of five potential spore stages of the rust fungi, from which the perfect stage of the basidium and basidiospore arises. Typically a resting, overwintering stage.

TELIUM (TELIA): a fruiting body or fruiting structure producing teliospores.

TENDRIL: a thread-like gelatinous mass of forced-out spores. Also called sporehorn or cirrhus.

TOMENTOSE: covered with soft matted hairs.

TOMENTUM: a layer of soft matted hairs on plant parts.

TRAMA: the fleshy tissue within a basidiocarp.

TRIMITIC: describing a fungus that has three types of hyphae.

TUBE LAYER OF SPOROCARP: a layer of vertically placed tubes attached to the lower surface of the context of a sporocarp. See HYMENIUM.

TYLOSES: outgrowth of cells in woody tissue that block water-conducting xylem tissue.

UNGULATE: in a form resembling a horse's hoof.

UREDIOSPORE also UREDINIOSPORE: the third of five potential spore stages produced by the rust fungi in their life cycle, capable of infecting the host on which it was formed (so repeating stage). The stage is produced in a fruiting body called a uredium.

UREDIUM (UREDIA, UREDINIA): one of the types of fruiting bodies formed by the rust fungi in their life cycle. Urediospores are formed in this fruiting body.

VECTOR: an organism, usually an insect, that transmits a pathogen from one host to another.

VEGETATIVE MYCELIUM: a mass of hyphae constituting the body of the fungus and without spores.

VELUTINATE: covered with a thick layer of delicate hairs, velvet-like.

VERMIFORM: worm-like.

VILLOSE: covered with long thin, nonmatted hairs (villi).

VIRULENT: vigorously pathogenic.

WETWOOD: a discolored, water-soaked condition of the heartwood of some trees presumably caused by bacterial fermentation. Often associated with distinctive odor, gas production, and an exudation called slime flux.

WHITEROT: a type of wood decay caused by fungi that degrade all chief constituents of wood and leave a whitish or light-colored residue. Affected wood is often fibrous or spongy in texture.

WINDBREAK: a row or rows of trees that serve as a protection from wind.

WITCHES'-BROOM: cluster of branches, twigs, or foliage, often denser than normal, caused by certain pathogens (sometimes insects, or a combination of insects and pathogens), which is not typical of the host's normal pattern of growth.

WOUNDWOOD: differentiated and organized tissue, high in lignin, that is produced by woody plants to cover wounds. Woundwood replaces callus tissue at the site of injury.

ZONATE: having zones characterized by different qualities.

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