Diseases of Tree Fruits in Michigan
Michigan State University
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Brown rot of apricot

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By Alan L. Jones, **assistant professor, Botany and Plant Pathology Department**

Contents

Year after year, plant diseases lower both yield and quality of Michigan apples, pears, peaches, cherries, and plums. This bulletin is designed to assist the fruit grower or his consultant achieve better control of tree-fruit diseases.

Proper recognition of the problem is the first step in successful and efficient control of disease pests. Growers who can identify fruit diseases will be in a position to select the proper and most efficient control methods. Wherever possible, photographs of symptoms, many in color, have been included as diagnostic aids.

Each orchard disease has a developmental pattern or cycle it passes through during the year. Control chemicals and methods are generally effective only at certain times or stages in the disease cycle. If the pest is not in a susceptible stage of development when control is attempted, results will be disappointing. Throughout this bulletin, an attempt has been made to outline the life cycles of the various diseases as they are known to occur in Michigan. Those portions of the cycle known to be important in developing successful control procedures are stressed.

Weather plays an important part in determining the incidence and severity of diseases. Temperature, rainfall, and relative humidity can alter the development of the disease, the plant, or both. Pest programs must be adjusted annually according to prevailing environmental conditions to achieve the best results. Interrelationships between the pathogen, the host plant, and the environment are discussed in some detail as a guide in deciding when control procedures can be safely modified.

Most of the diseases of tree fruits in Michigan are described here. Specific chemical control recommendations for most of these diseases may be found in the Fruit Spraying Calendar, Extension Bulletin 154, which is updated annually.

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APPLE SCAB

Scab is caused by the fungus *Venturia inaequalis*. It occurs in most areas of the world where apples are grown. It is less severe in semi-arid regions than in cool, humid areas with frequent rainfall. The climate in Michigan is extremely favorable for scab. Fortunately, successful control procedures have been developed and losses from scab can be prevented.

Symptoms

Apple scab occurs on the leaves, petioles, shoot growth, blossoms, and fruit. The most striking symptoms are on the leaves and fruit.

Leaf infections (Figure 1) usually appear first on the flower bud leaves. Lesions develop primarily on the undersurface of the leaf, the side exposed when the fruit buds first open. Once the entire leaf has unfolded, both sides may be infected.

Diseased leaves are often distorted. Severe infection can also dwarf leaves and cause defoliation. Trees severely defoliated two or three years in a row are weakened and susceptible to low temperature damage.

Lesions develop as velvety brown to olive spots which turn black with age. At first, the margins (edges) of the lesion are indefinite, but later have distinct limits.

Fruit infections (Figure 2) resemble leaf infection when young but with age become brown and corky. Early scab infection results in uneven growth of the fruit and cracking of the skin and flesh. Lesions often develop around the blossom end of the fruit early in the season, later they develop anywhere on the surface.

If infection occurs in late summer or early fall, rough, black, circular lesions may develop on the fruit in storage. These lesions are usually small, varying in size from specks to one-fourth inch in diameter.

Disease Cycle

Primary Cycle: The fungus overwinters in leaves on the orchard floor (follow Figure 3 when reading the text on disease cycle). In late fall and early spring, microscopic, black, pimple-like structures, called perithecia, are produced in these dead leaves. Within each perithecium are asci, each with eight ascospores. The ascospores produce the first or primary infections to the new growth.

Perithecial development is favored by alternating periods of wetness and dryness in late winter and early spring. In years with good snow coverage and no deep-seated frost, the fallen leaves are well protected and perithecia mature early. Ascospore production begins during mild days in February. Spores develop gradually at first, but much faster as the temperature increases to the mid 60's. Perithecial development is reduced and spore production delayed in dry springs.

Normally, some perithecia have mature ascospores when the blossom buds start to open. When the leaves on the orchard floor become wet, spores are forcibly ejected into the air. Air currents carry them to the emerging tissues where infection takes place. Maximum spore discharge occurs within 30 minutes of wetting; complete discharge requires about two hours. Maturation and discharge of ascospores usually lasts five to nine weeks.

Germination begins as soon as a spore lands on new, green leaves or fruit, providing a film of moisture is present. The number of hours of wetting required for infection varies with prevailing temperatures. Growers should record the beginning of rainfall and average temperatures, and determine the length of time it takes for infection to occur (Table 1). For example, at an average temperature of 58°F, primary infection will occur 10 hours after the start of the rain. After 22 hours of wetting, the degree of infection will be severe. This simple calculation is useful in deciding when fungicide sprays are needed.

Lesions are not visible until several days after the fungus first infects the leaf or fruit. The average temperature after penetration is important for determining the time required for lesion development. About nine to seventeen days are required from inoculation to the appearance of the olive-green, velvety scab lesions. Within the lesion are secondary spores (conidia) for perpetuating the disease in summer.

Secondary Cycles: Secondary infections are initiated by conidia produced in primary lesions. Since conidia may develop as soon as seven to nine days after infection, secondary infection, if not controlled, may start to occur during bloom. This is particularly true when ascospores infect the apical portions of sepals and leaves, the first susceptible parts exposed.

Conidial formation is favored by high humidity and moderate temperatures. Although wetting is not

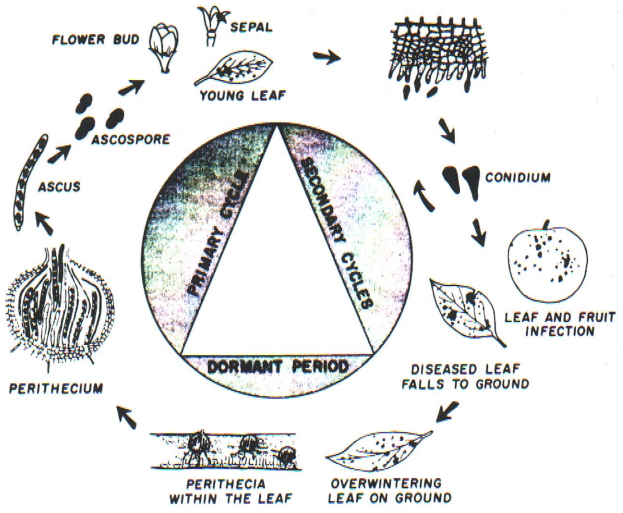


Figure 3—Disease cycle of apple scab.

Figures 1 and 2—Leaves with apple scab (left); primary apple scab on calyx end of McIntosh fruit (right), with small secondary lesions developing on the side.

required for spore formation, heavy dews or rain are required to detach the conidia. Spores are disseminated primarily by splashing rain. Unlike ascospores, they are not scattered long distances by wind. Conidia germinate and infection occurs under the same conditions as for ascospores, except that they require about two-thirds of the time indicated in Table 1 for primary infection at the same temperature.

Secondary infection to fruit can occur in the fall but not show up until the fruit have been stored for several months. The disease can also build up on the leaves. Since the fungus overwinters in these leaves, perithecia may be present in quantity to start the new season even though a good spray program was followed the previous year.

Control

Chemical control: Apple scab is controlled primarily with fungicide sprays or dusts. A large number and variety of chemicals are available. How and when they should be used depends upon their mode of attack.

Protectant fungicides prevent the spores from germinating or penetrating. To be effective, they must be present on the surface of susceptible tissues before infection. These chemicals may be applied periodically at pre-determined intervals, or according to anticipated rainfall (infection periods).

Eradicant fungicides kill the fungus after the spores have landed on the plant. The relative ability of fungicides to eradicate scab is referred to as "back-action" or "kick-back" action—the number of hours that the chemical can be effectively applied after the infection period begins. Ratings of "kick-back" action are made for specific temperatures. The eradicant action for most fungicides is limited to a few hours or days after infection.

Some fungicides or mixtures of fungicides have both eradicative and protective properties. Generally, they can not be applied as frequently as strictly protectant materials, but are the type most used today.

Other types of fungicides also interrupt the life cycle of scab. Systemic chemicals, which are translocated to the new growth as it emerges, are becoming available. These chemicals may not completely

Table 1. Approximate number of hours of wetting required for primary apple scab infection at different air temperatures and the length of time required for development of conidia*

Average Temperature	DEGREE OF INFECTION			Days Incubation[a]
	Light	Moderate	Heavy	
°F	hrs.[b]	hrs.	hrs.	days
78	13	17	26	
77	11	14	21	
76	9½	12	19	
63 to 75	9	12	18	9
62	9	12	19	10
61	9	13	20	10
60	9½	13	20	11
59	10	13	21	12
58	10	14	21	12
57	10	14	22	13
56	11	15	22	13
55	11	16	24	14
54	11½	16	24	14
53	12	17	25	15
52	12	18	26	15
51	13	18	27	16
50	14	19	29	16
49	14½	20	30	17
48	15	20	30	17
47	17	23	35	
46	19	25	38	
45	20	27	41	
44	22	30	45	
43	25	34	51	
42	30	40	60	
33 to 41	2 days			

a Approx. no. days required for conidial development after primary scab infection.
b The infection period is considered to start at the beginning of the rain.
* From W. D. Mills, Cornell University.

kill the fungus after it has entered the plant, but suppress production of spores and secondary spread. They are important in scab spray programs where infections have gotten started.

Prevention of perithecial formation in overwintering leaves would probably eliminate scab. Unfortunately, complete elimination of perithecia has not been achieved under orchard conditions with current techniques. Just one percent of the perithecia is sufficient inoculum for severe scab infection, under ideal weather conditions. Scab control the following year can be simplified by making fall nitrogen applications to hasten leaf decomposition, plus post-harvest fungicide sprays to inhibit perithecial formation.

Resistant varieties: Apple varieties resistant to scab are being developed. Purdue University, in cooperation with several other universities, recently released the scab resistant variety Prema. Although this variety has not been evaluated in Michigan, it may have value for specific markets. More important, it indicates that the many years of research efforts in this area are beginning to pay dividends. Hopefully, other selections will be available for limited trial in the near future.

FIRE BLIGHT OF PEAR AND APPLE

Fire blight, caused by the bacterium *Erwinia amylovora*, is the major production problem faced by Michigan pear growers. Blight can also significantly reduce yields on susceptible apple varieties. Blight now causes greater loss to the apple industry than to the pear industry, because of a recent decline in pear production. The disease causes an annual loss of blossoms and fruit, but its most serious effect is reduced future production due to destruction of branches and scaffold limbs.

Symptoms

Infected blossoms become water-soaked and dark green as the bacteria invade the succulent tissues. Within a few days, the entire fruiting spur may be invaded (Figure 4). Infected tissues wilt and turn dark brown to black on pear, brown to dark brown on apple. The disease usually moves into the leaves through the petiole, resulting in discoloration of the mid-vein first, followed shortly by a darkening of the lateral veins and surrounding tissue.

Infected terminals wilt from the tip and often develop a crook or bend at the growing point. At first, the tissues are water-soaked and dark green, later turn brown to black. The disease sometimes progresses into the shoot from its base, blighting the lower tissues and girdling the parts above. Infected leaves and fruit often persist into winter as a reminder of the previous summer's activity.

Following infection, blight can move long distances within the living tissue and kill an entire tree in one season. Bark of invaded branches and scaffold limbs

Figure 4—Fire blight blossom infection killing fruit spur.

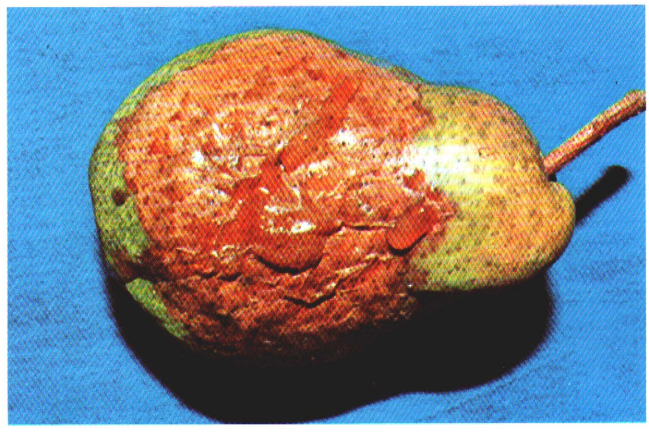


Figure 5—Bartlett pear infected with blight. Note bacterial ooze.

is darker than normal. When the outer bark is peeled away, the inner tissues are water-soaked with reddish streaks when first invaded; later the tissues are brown. When development slows down, the margins become sunken and sometimes cracked, forming a canker. The presence of reddening helps to distinguish fire blight cankers from low temperature damage to bark.

Under certain conditions, apple and pear fruit develop a brown to black decay from blight (Figure 5). The rotted areas remain firm but the fruit eventually shrivels into mummies and persists on the tree.

During wet, humid weather, blighted tissues often extrude a milky, sticky liquid or ooze containing the bacteria. These droplets are produced in areas where the bacteria are actively multiplying. The ooze turns brown upon exposure to air. Appearance of ooze on the surface of diseased blossoms, terminals, fruit or wood is the most obvious characteristic of blight.

Disease Cycle

Bacteria overwinter at the margins of cankers on branches (Figure 6). Survival is most likely to occur in cankers with indefinite margins located on large

branches. The proportion of these "holdover cankers" is highest following mild winters. Ooze containing the bacteria begins to appear on the surface of the cankers when the trees are in the pre-bloom stage of development. Bacteria must move from overwintering cankers to exposed flowers before primary infection can occur. This is accomplished through the action of splashing rain and by flies and other insects which visit both the bacterial ooze and the blossoms.

Only a small portion of the blossoms are infected in this manner, particularly if care has been taken to eliminate overwintering cankers before growth begins. Eventually, however, a honeybee visits a diseased blossom and picks up pollen or nectar contaminated with bacteria. Once this occurs, spread can be rapid and infection severe. Secondary infections also occur from splashing rain.

Climatic conditions are important in determining amount of spread and severity of blossom blight. Temperatures between 65°F and 90°F favor infection. The nectar must be diluted with moisture for fire blight bacteria to grow. At the optimum temperature of 76°F, blossoms begin to show blight in four to five days. At lower temperatures, symptom development takes much longer.

Although bacteria invade the flower primarily through natural openings, wounds are important in the infection of terminal shoots, leaves, and fruit. Water sprouts and shoots may be inoculated directly by piercing insects, primarily aphids and leafhoppers, or indirectly through the feeding wounds left by these pests. Wounds from hail stones are frequently invaded by blight bacteria, leading to severe disease outbreaks. Any fresh wound can serve as an infection point, but wounds become less susceptible with age. Under prolonged periods of wetness and high humidity, infection of leaves, shoots, and fruit through natural openings may occur.

Control

Many practices help reduce the severity of fire blight in the orchard. Not all measures are necessary or feasible in every planting. No single control method is adequate however, and a conscious effort must be made to control the disease each year on susceptible varieties. Otherwise, grow other crops or varieties.

Removing sources of infection: In summer, periodically inspect orchards and remove infected spurs or terminals. This reduces spread of the disease and prevents extensive loss of bearing surface. It is best to break out infected twigs, thereby reducing the possibility of spread on the pruning tools. If infections are cut out, use Chlorox, diluted 1:10 with water, to sterilize the tools. Remove at least 12 inches of normal-appearing bark below the lowest evidence of disease to insure elimination of the infection. Dormant pruning of *all* overwintering cankers on young apple trees and pear trees is important. Remove cankers one-half inch or larger from older apple trees. Make cuts about four inches below any evidence of dead bark killed by the disease.

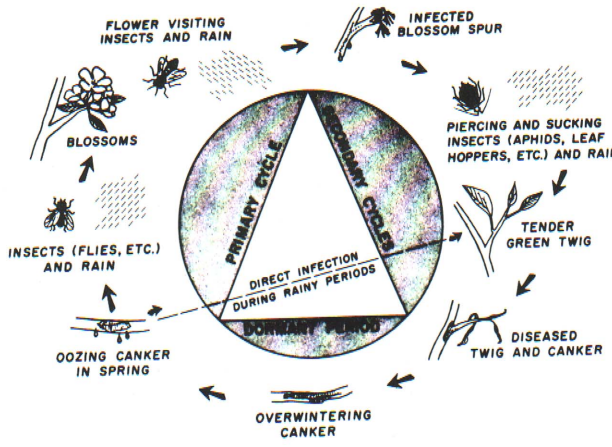


Figure 6—Disease cycle of fire blight.

Insect control: Sucking insects such as aphids, leafhoppers, plant bugs, and pear psylla inoculate growing terminals and watersprouts and create wounds that bacteria can enter. These pests must be controlled before bloom and throughout the summer.

Resistant varieties: Although varieties vary in resistance to fire blight, none are immune. Susceptible apple varieties include: Tompkins King, Twenty Ounce, Rhode Island Greening, Yellow Transparent, Jonathan, Idared, Fenton (Beacon), and many crab apple varieties. Some years, Golden Delicious, Delicious, McIntosh, and Stayman develop spur infections, but the disease seldom enters the main branches. Bartlett, Bosc, and Clapp's Favorite, the main pear varieties in Michigan, are highly susceptible.

When establishing new orchards, consider varietal susceptibility. Blight control is easier if plantings of susceptible crops can be isolated. Susceptible apple varieties should not be interplanted with pears or in fields adjacent to pear plantings. In mixed variety planting, varieties susceptible to blight should be set in solid rows for ease of spraying with blight control chemicals.

Cultural practices: Lesion development and damage from blight is more severe when tree growth continues late into the season. Use management systems that promote early cessation of growth without reducing tree vigor excessively. Plant orchards on well-drained soil, apply nitrogen fertilizer early, and cultivate no later than July 1 to stop growth.

Chemical control: Bactericides have been highly effective against the blossom phase of fire blight. These sprays are important because they often prevent fire blight from getting started. Once the disease is established, control is difficult. Although currently available chemicals have some effect on terminal blight, other chemicals and procedures are being studied to find more efficient methods to control this phase of the disease.

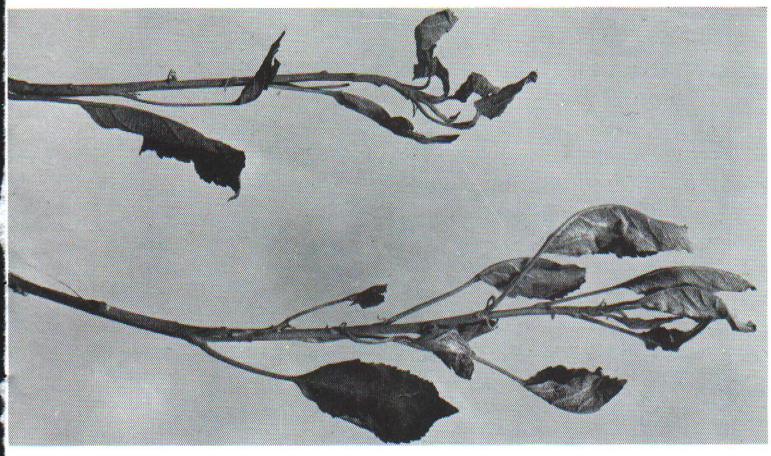


Figure 7—Powdery mildew infected twigs with distorted leaves.

POWDERY MILDEW

Powdery mildew, caused by the fungus *Podosphaera leucotricha*, is often an important problem in Michigan orchards, but is not a major disease, compared to apple scab. It becomes destructive mainly on susceptible varieties, but only after it has built up to high levels. The problem is greatest when a mildewcide is not included regularly in the scab program.

Symptoms

Mildew occurs in the nursery on terminal growth and in the orchard on leaves, flowers, shoots, and fruit. On leaves, lesions first appear as whitish, felt-like patches of fungal mycelium and spores on the undersides and along the margins. The lesions spread rapidly, engulfing the entire leaf. Infected leaves are narrower than normal, folded longitudinally, and become stiff and brittle with age (Figure 7). When severe, early defoliation occurs.

Infected blossom buds open several days later than normal and are sometimes killed outright by low winter temperatures. Flower parts and leaves are usually covered with the white mycelium when they emerge. Infected terminal or shoot buds also produce diseased leaves and shoots. Shoots from infected buds are shorter than those from healthy buds.

The fruit is not usually infected unless the disease has built up to high levels. Infected fruit are severely russeted, sometimes dwarfed (Figure 8).

Disease Cycle

The mildew fungus overwinters as mycelium in buds infected the previous summer. As the infected buds open in spring, the fungus, already established on the emerging leaves, begins to produce spores (conidia). The conidia, circular to barrel-shaped, are produced in chains. They are carried by wind to the emerging tissues to initiate secondary infections.

Unlike apple scab, which requires a film of moisture to germinate, the mildew spores germinate readily at high relative humidities and temperatures between 60° and 80°F. Spore germination and mycelial development at temperatures between 40° and 50°F is slow; slight at temperatures above 90°F. Mycelium from the germinating spores ramifies over the surface of the leaf, putting down small "roots" into the epidermal cells for nutrition. More spores are quickly produced and the cycle repeated. Secondary cycles continue to occur until tree growth stops in late summer. Small, dark brown, globular, fruiting bodies (perithecia) are sometimes formed in the mycelial mat in late summer. Each structure has one ascus containing eight ascospores, but it is doubtful they play an important role in overwintering the fungus.

Control

Mildew susceptible varieties include Johathan, Rome Beauty, Cortland, Baldwin, Monroe, and Idared. Where susceptible varieties are grown, include a mildewcide in the scab program to provide control of both diseases. Start sprays early and continue until terminal growth stops.

RUST DISEASES

Cedar-apple rust is the most common of the three apple rust diseases in Michigan. Unlike southern U.S. fruit growing areas, Michigan is not seriously affected by these diseases. The diseases and causal fungi include: 1) cedar-apple rust, *Gymnosporangium*

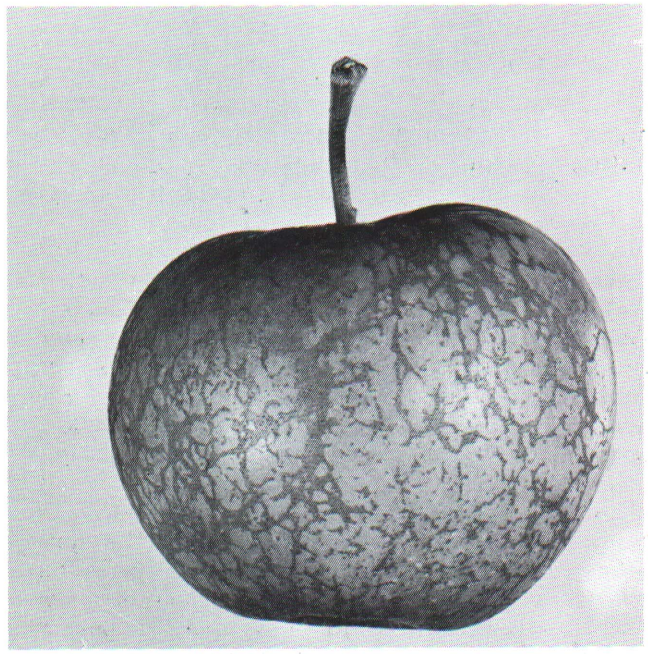


Figure 8—Fruit showing net russetting from powdery mildew infection.

juniperivirginianae; 2) quince rust, *G. clavipes;* and 3) hawthorn rust, *G. globosum.* All three fungi spend part of their life cycle on red cedar. The northern limit for red cedar is roughly a line across the state through Grand Rapids and Lansing. This explains why rust diseases are restricted to the southern portion of Michigan. Because life cycle and control of these diseases are similar, only cedar-apple rust will be considered.

Symptoms

On Apple—The bright color of the lesions produced by this disease make it easy to identify (Figure 9). Small, pale yellow spots develop on the leaves shortly after bloom. These spots gradually enlarge and turn orange. Orange-colored drops of liquid may be observed in the spots when they are about one-eighth inch in diameter. Later, black dots (pycnia) appear in the spot on the upper surface. The lesions become evident on the undersurface and cylindrical tubes or protuberances (aecia) appear in late summer. The ends of the tubes split open and curl back. Severe infection results in heavy defoliation and weakens the tree.

Fruit infection is most common near the calyx end. Lesions are similar in color to those on leaves except the border is a darker green than normal. The black pycnia often develop in the lesion, but formation of the cylindrical tubes (aecia) is less common.

On Cedar—The fungus produces brown to reddish-brown galls from one-quarter to two inches in diameter. During rainy periods in spring, bright orange, gelatinous, spore-horns protrude from depressions on the surface of the galls (Figure 10).

Disease Cycle

The disease cycle of cedar-apple rust is complex (Figure 11). Two plants (apple and cedar) and three fruiting structures (telia, aecia, and pycnia) are involved. The pathogen requires two years to complete its life cycle.

In July and August, wind-borne aeciospores from apple infect cedar leaves. The reddish-brown galls or "cedar apples" require nearly two years to mature. When wet in spring, the galls extrude gelatinous tendrils or "horns" consisting of two celled teliospores. Each spore produces four basidiospores from each of the two cells. Air currents carry the basidiospores to the apple leaf and fruit where they infect within four hours under favorable conditions.

Rust lesions develop in one to three weeks. On leaves, the yellow rust spots develop on the upper surface. Black dots (pycnia) develop in the lesions followed by appearance of the disease on the underside. Cup-like fruiting structures (aecia) appear in these lesions in July and August and produce aeciospores. The aeciospores infect cedar leaves, thus completing the life cycle of the fungus.

Figure 9—Apple leaves and fruit severely infected with cedar apple rust (Courtesy J.D. Moore, U. of Wisconsin).

Control

Removal of cedars located within a two-mile radius of the orchard interrupts the life cycle and makes fungicidal control easier. For complete control, remove all cedars within four to five miles of the orchard.

Several fungicides are highly effective against the rust diseases. They should be applied periodically from the pink stage of bud development to third cover to protect the emerging leaves and developing fruit.

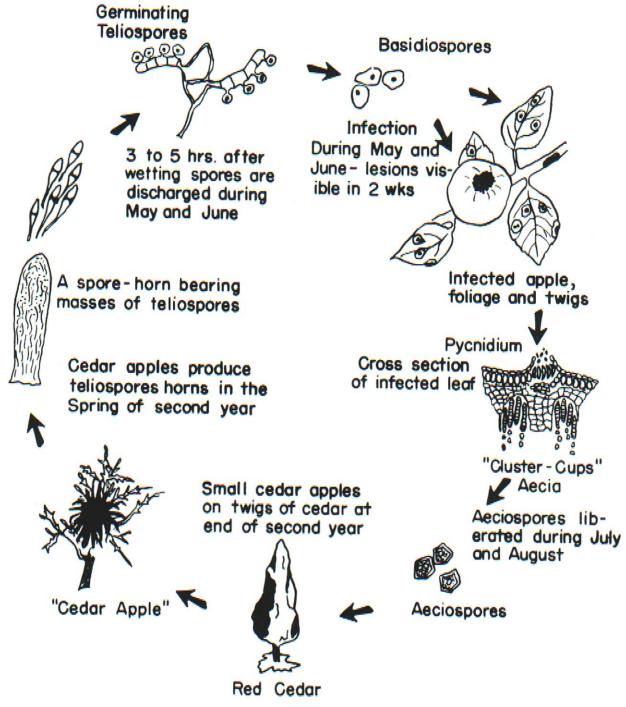


Figure 11—Disease cycle of cedar-apple rust (Courtesy K. D. Hickey, Virginia Polytechnic Institute).

Figure 10—Cedar-apple rust gall on cedar.

BLACK ROT

Black rot is not as severe in Michigan as in southern fruit production areas. The disease is also known as "frogeye leaf spot" in areas where leaf infections are important. In Michigan, the canker phase, caused by the fungus *Physalospora obtusa*, is the most important.

Symptoms and Disease Cycle

Cankers are found on branches weakened by heavy shading, sun scald, or winter injury. Pruning cuts and old fire blight cankers are the major points of entry. Apparently, the fungus is not able to infect vigorous trees without the presence of bark injury. The disease is rarely found on non-bearing trees, only on trees that have produced crops for a few years.

Infected areas on limbs and branches are reddish-brown and slightly sunken (Figure 12). Cankers expand a little each year, eventually becoming several feet in length. The bark in older areas of the canker can be removed easily—bark recently killed clings firmly. Infected branches are sometimes weakened enough to break with heavy crop loads; sometimes they are killed outright.

In Michigan, the black rot fungus overwinters primarily in cankers. In spring, conidia are formed in pycnidia found in pimple-like protuberances on the surface of cankers. Spores are produced for several months on infected fruit and leaves in addition to branches.

Fruit infections have been found occasionally in southwestern Michigan. Diseased fruit develop a brown to black rot with alternating light and dark bands. Infections start either around the calyx (blossom) end of the fruit or in an injury. Although severely diseased fruit may dry up and persist under some conditions, mummies are relatively rare in Michigan.

Leaves injured by spray chemicals are sometimes infected by black rot. Margins of lesions become irregular; centers have alternating bands of light and dark. Presence of black pycnidia helps distinguish black rot lesions from similar types of leaf injuries caused by other factors.

Control

Although some spray programs may reduce the incidence of this disease, none are completely effective. Control of the canker phase is particularly difficult.

Cultural practices designed to keep trees in a vigorous condition help reduce canker infections. Pruning should be confined to the dormant season as much as possible. Keep pruning tools sharp so that cuts are clean. Remove all broken branches and other weak or injured wood which might become infected. Avoid long pruning stubs. If cankers are found, cut well below any evidence of diseased bark.

Figure 12—Black rot canker on apple.

PHYTOPHTHORA COLLAR ROT

Collar rot caused by the fungus *Phytophthora cactorum* was a minor problem when apple trees were propagated on seedling rootstock. The disease was rare except where Grimes apples were grown. The incidence of *Phytophthora* collar rot has increased as semi-dwarf rootstocks, particularly Malling Merton clones, have replaced seedling rootstocks.

Symptoms

The disease produces cankers below the ground line in areas where the roots become attached to the crown or lower trunk (Figure 13). Infected bark becomes brown and is often slimy when wet. Cankers may extend from the point of origin into the root system and up the trunk to the bud union. If the scion variety is susceptible to *Phytophthora*, the disease may extend above the union. More often, the disease stops at the ground line. Older cankers form a definite outline when the bark dries out and callus tissues develop at the margins.

The cankers girdle the roots and lower trunk, resulting in poor terminal growth and foliage discoloration. Severely infected trees eventually die. The leaves on damaged trees may become yellow in summer or reddish to bright red in early fall. Because other root problems cause similar symptoms, these symptoms only indicate the need for further examination of the root system. Often, isolation of the fungus is required before a positive diagnosis can be made.

Young trees show the disease most when fruit production begins. This could be in the third year on some early bearing rootstocks. Infected trees usually linger two or three years before dying. However, if conditions are optimum for infection, trees can be killed in one growing season. Trees over 10 years old are usually able to tolerate the disease.

Figure 13—Phytophthora collar rot on the crown and root system. Rootstock is MM-104.

Disease Cycle

The pathogen survives in soil for several years as oospores. These thick walled structures can resist periods of unfavorable environment, such as drought, and are relatively resistant to chemical treatment. They occur in greatest numbers in old orchard soils. The fungus is also believed to overwinter as mycelium in diseased tissue.

Fungal growth and infection are favored by damp, cool periods after harvest and in spring about the time leaves and flowers are emerging. Ability to produce many spores (primarily zoospores), allows the fungus to build up to high levels from a few oospores under favorable conditions. Zoospores swim in films of moisture to the crown area where they infect. Others may be splashed on the fruit and cause a rot. Phytophthora fruit rot is most common in areas where contaminated water is used in overhead irrigation systems.

Control

Problems with collar rot can be reduced or eliminated by carefully selecting the orchard site and rootstocks for the new planting. Susceptible rootstocks should not be planted in orchards (or areas of the orchard), with heavy, poorly drained soils. Tiling of wet areas in an otherwise well drained location often improves internal soil drainage sufficiently to avoid the disease in low areas. Where the soil is variable, it becomes necessary to use several different rootstocks in the orchard, depending on their soil preference and susceptibility to collar rot.

Observations in Michigan and in other Midwest states indicate Malling-Merton stocks MM-104 and MM-106 are very susceptible. EM-VII and EM-II are considered susceptible but extensive use of these stocks in Michigan indicate they are adaptable to many locations. EM-IX is considered resistant. Tests and observations on other rootstocks have been limited. Before using them, check with your District Extension Horticultural Agent and Agricultural Agent. Seedling rootstocks of Delicious are moderately resistant, while those of Melba, McIntosh, and Wealthy are apparently resistant.

Once infection occurs, it is difficult to eradicate the disease and save the tree. Probably the most effective method is the use of "nurse trees." This practice involves inarching one year-old whips of a resistant variety into the trunk above the lesion. Results are best when treatments are made early, but are of limited value if more than 50 percent of the circumference of the trunk is infected.

Chemical control procedures are being tested. At best, they are "stop-gap" measures to be used in emergency situations, and are not a substitute for selecting rootstocks adapted to the intended orchard site. Contact your local Cooperative Extension Agent for latest recommendations.

Figure 14—Sooty blotch on surface of Northern Spy apple.

SOOTY BLOTCH AND FLY SPECK

Sooty blotch and fly speck are common names of two diseases usually found on apple fruit at the same time. They are caused by different fungi; sooty blotch by *Gloeodes pomigena*, fly speck by *Microthyriella rubi*. They do little or no actual damage to the fruit, but lower market value by their presence on the surface. Rarely important in Michigan, these diseases are sometimes found in poorly sprayed orchards and in backyard plantings.

Symptoms and Disease Cycle

Sooty blotch appears as superficial sooty or cloudy blotches on the surface of the fruit (Figure 14). Lesions are brown to olive green, indefinite in outline, and can be removed by vigorous rubbing. The fungus apparently overwinters on infected twigs of many woody plants. Infection by conidia produced in pycnidia occurs from June to September. Because disease development is favored by moderate temperatures and high humidity, late summer infection is the most severe.

Fly speck, often associated with sooty blotch, is favored by the same environmental conditions. The disease appears as sharply defined, black shiny dots in groups of 10 to 20 specks (Figure 15). Infection is by wind-borne ascospores and conidia from wild hosts.

Control

Both diseases are easily controlled with fungicides. Where the disease is observed, use scab fungicides in late summer that are effective against both sooty blotch and fly speck. The disease can develop if spray programs are stopped too early.

BLISTER SPOT

Blister spot of apple is caused by the bacterium *Pseudomonas syringae*. The disease is rarely serious.

Symptoms and Disease Cycle

Symptoms appear in June and are associated with natural breaks in the wax layer covering the fruit (lenticels). Infections initially look like water-soaked areas on the surface of the fruit, and turn black to brown. The spots become raised, giving the fruit a pimply appearance (Figure 16). Red to purple discoloration often develops in the skin bordering the lesion and helps distinguish the disease from pesticide injury to fruit.

Disease development is usually erratic and only a few fruit in the orchard may be affected. In some orchards of Mutsu, however, nearly all fruit have become infected under favorable conditions. Severely infected fruit are not suitable for fresh market sale. Other susceptible varieties include Jonathan, Red Delicious, and Golden Delicious. Rome Beauty and Duchess have been reported susceptible outside Michigan fruit growing areas.

The bacteria are believed to overwinter in twig infections although cankers are difficult to observe. The pathogen also causes a blossom blight on apple, but the relation of blossom infection to fruit infection is uncertain. The disease has been most severe in years and locations of heavy rainfall.

Control

Due to the minor nature of this disease, control procedures have not been developed. Severe outbreaks of blister spot in young Mutsu plantings indicate caution should be taken in selecting this variety for fresh-market use until more is known about its susceptibility.

Figure 15—Fly speck on surface of Northern Spy apple.

Figure 16—Blister spot on young Mutsu apple fruit.

NECTRIA TWIG BLIGHT

In 1970, Nectria twig blight, caused by the fungus *Nectria cinnabarina*, was identified on Rome apple in Michigan. Recognition of the disease is important, even though it is only a minor problem. It can be confused with fire blight for which control measures are quite difficult.

Symptoms and Disease Cycle

In June, leaves on infected twigs begin to wilt and die. Small, sunken cankers may be found at the base of the diseased twig. Unlike fire blight, no signs of blighted blossom clusters remain on the twigs and the terminals do not appear to die from the tip. In mid to late summer, a few bright-pink or coral-red structures appear on the surface of the cankers (Figure 17). These smooth, globular structures distinguish the disease from other canker diseases of apple or pear.

The disease has been noted primarily on varieties such as Rome Beauty, Ben Davis, and Northern Spy, with a large cluster-bud base. Worst infections appear to follow late maturing seasons, suggesting infection may occur in the fall after harvest. However, experimental evidence for this is lacking. Optimum fungus growth occurs in culture at 70°F, moderate growth at 80° to 85°F, and light growth at 37°F. Picking wounds, which probably take longer than usual to heal in late maturing seasons, combined with low temperatures, favor the establishment of infection.

Control

The disease is not usually severe enough to require special control measures. As a result, chemical control procedures have not been developed. Removal of infected twigs helps reduce the disease.

BLOSSOM END ROT

Blossom end rot is seldom serious in Michigan and usually not more than five to ten percent of the fruit in an orchard show infection. The disease is caused primarily by *Botrytis cinerea.* A nearly identical disease, caused by *Sclerotinia sclerotiorum*, has been reported on McIntosh from New Hampshire.

Symptoms and Disease Cycle

Affected fruit become visible in the orchard in June, although infection apparently occurs in bloom. As infected blossoms develop, the fungus spreads into the tissues surrounding the calyx (blossom) end of the fruit (Figure 18). A small, quarter- to half-inch area of shallow dry rot develops adjacent to the blossom end. The rot is often surrounded by a red colored border.

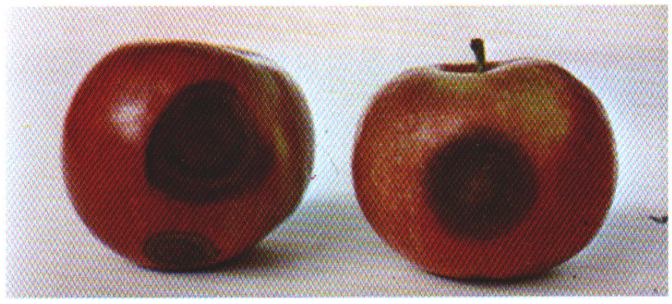
Botrytis prefers cool weather and is observed primarily in years when an extended cool, rainy period exists in bloom, or shortly after. The disease is noted most often on McIntosh, Rome Beauty, and Red Delicious although other varieties are undoubtedly infected. Often, the rotted area corks over before harvest.

Control

Blossom end rot has not yet caused sufficient damage to justify a study of control measures. Many of the fungicides used to control apple scab apparently help suppress the disease.

In apples with higher levels of infection, prolonged storage may be undesirable due to secondary rot problems. This is particularly true of Red Delicious where blossom end rot often leads to moldy core problems.

Figure 17—Bright orange fruiting structures of Nectria twig blight fungus (enlarged at right).



Figures 18 and 19—Blossom end rot of McIntosh apple (left); bull's-eye rot (right). In bull's-eye rot photo, note light color in center of apple on right and alternating areas of tan and brown on fruit at left.

BULL'S-EYE ROT

Bull's-eye rot or "Northwestern anthracnose" is primarily a problem on fruit in storage although infection originates in the orchard. It is a minor disease in Michigan. The disease develops slowly on stored fruit and is most troublesome following wet falls. The fungus causing the disease is *Neofabraea malicorticis*.

Symptoms

Small, elliptical, sunken cankers develop in the bark, on twigs, and branches that are less than two inches in diameter. Old cankers have a pronounced ridge of callus around the margin. New infections appear in the fall as small reddish-brown areas on young twigs. As the cankers enlarge during the fall and spring, the bark remains smooth. Later, cracks develop between the canker and surrounding healthy tissue.

Infected fruit develop brown, sunken, round lesions on the surface (Figure 19). The centers of the infected areas are often light colored and surrounded by alternating areas of tan and brown, giving the characteristic "bull's-eye" appearance. Lesions tend to be relatively small. Whole fruit are rarely rotted by one lesion. The surface of the rot is sometimes covered with cream-colored spore masses (acervuli).

Disease Cycle

Conidia of the bull's-eye rot fungus are washed by rain from the cankers on the twigs and branches to the apples and twigs below. Ascospores formed in two-year-old cankers are forcibly discharged and disseminated by wind. Infection is mainly through the lenticels on the fruit, but wounds may also be infected. Fruit decay does not usually show up until after harvest. The disease is generally worse in falls with frequent rains and moderate temperatures and on fruit harvested beyond optimum maturity. Twig infections develop for two years before completing the life cycle. In the first fall after infection, conidia are produced in acervuli on the surface of the canker. Ascospores are produced in apothecia borne on cankers the second year.

Control

Removal of twigs with cankers aids control by reducing the number of fungal spores. Chemical sprays near harvest help control infection to the fruit and twigs.

SOFT ROT

Soft rot or blue mold is the most common storage rot of apple in Michigan. It is caused primarily by the fungus *Penicillium expansum*, but other species of *Penicillium* produce a similar rot. It occurs on apple and pear and numerous other crops.

Symptoms and Disease Cycle

Soft rot can usually be distinguished by its light color and the soft, watery texture of the decayed areas. The rot develops rapidly under favorable temperatures, often rotting the whole fruit in about two weeks. When humidity is high, the fungus develops gray-blue, cushion-like structures on the surface of the rot (Figure 20). These structures produce spores (conidia) important in spread of the disease.

Spores of the soft rot organism are extremely resistant to drying. The fungus survives on many types of surfaces including those of packing and picking equipment. Thus, spores are nearly always present to cause trouble when conditions favor infection and disease development. Spores often build up in water used in dumping bulk boxes of fruit or in solutions used to treat apples for scald.

Soft rot is a disease of ripe fruit which develops mostly on apples picked at an advanced state of maturity. It rarely occurs on immature fruit or in the orchard, except on fallen fruits. The pathogen infects the fruit through skin breaks or lenticels. Although stem punctures are usually considered the most important point of entrance, infection through lenticels in bruised areas may be serious at times.

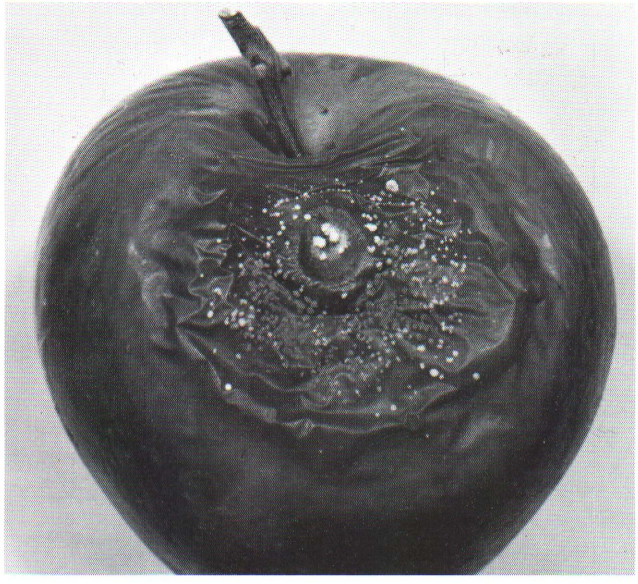


Figure 20—Delicious apple with soft rot. Note cushions of fungus growth on surface of lesion.

Control

To reduce soft rot to fruit, harvest at optimum maturity, handle carefully to prevent bruises and other injuries, and store as soon as possible after harvest under good, cold storage conditions. If fruit are to be stored late into the season, use disinfectants or fungicides in the water during grading to reduce spread to healthy fruit. Contact your local extension agent for suggested chemicals.

PEAR SCAB

Although pear scab resembles apple scab, it rarely develops in Michigan pear orchards even in years when apple scab is severe. The disease occurs sporadically in isolated Bartlett pear orchards near Lake Michigan, more frequently in backyard plantings of little known or exotic pear varieties.

Pear scab symptoms are similar to those of apple scab (Figure 21). Unlike apple scab, however, twig infections are common and can be damaging. Twig infections and perithecia produced in the fallen leaves are the source of primary inoculum in spring. There are several strains of the pear scab fungus, *Venturia pirina*. Because strains of the fungus differ in varieties they infect, varieties resistant to scab in one region may not be resistant in another.

Control

Many of the fungicides that control apple scab also control pear scab. Check label clearance, however, before using a particular compound.

STONY PIT OF PEAR

Stony pit is a virus disease which severely misshapes and gnarls Bosc pear fruit, making them unsalable (Figure 22). Less severely infected fruit may be dimpled, a symptom often confused with plant bug injury. Grit cells concentrate beneath the pits and make it difficult to cut the fruit with a knife. A rather pronounced roughened bark or "measleslike" symptom has been associated with some stony pit strains. Leaf symptoms are known but are difficult to distinguish in the orchard. Bartlett has not shown symptoms in Michigan, but has occasionally in other regions of the country.

Control

Although Bosc is a minor variety, it is important as a pollenizer for Bartlett. Incidence of stony pit virus in Bosc trees in Michigan is high even though evidence of field spread is lacking. Nursery propagation is the most important means of stony pit spread. To eliminate further incidence of this virus in new plantings, select trees known to be virus-free. While it is best to remove infected trees, removal of all infected Bosc trees in some orchards could result in a drop in Bartlett pear production due to lack of sufficient pollenizers.

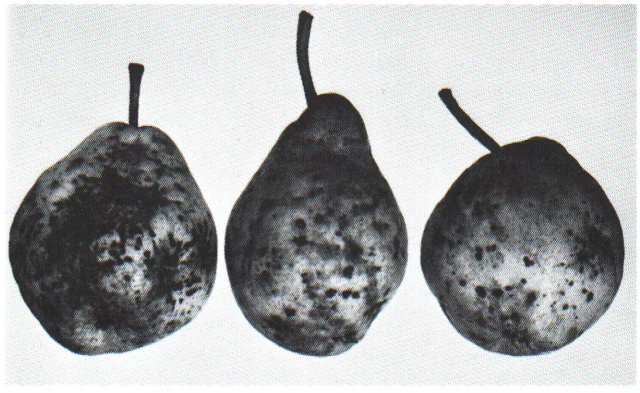


Figure 21—Pear scab on fruit.

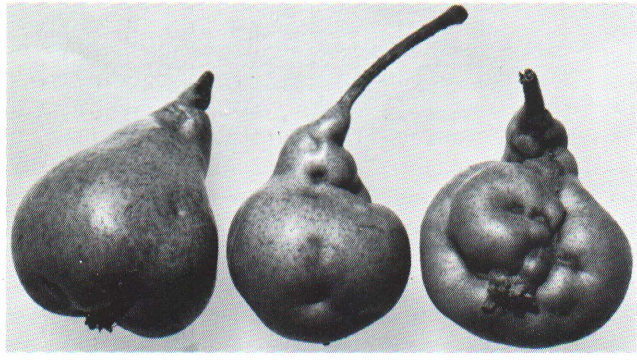


Figure 22—Pear fruit severely gnarled from stony pit virus.

Stone Fruit Diseases

CHERRY LEAF SPOT

Cherry leaf spot, caused by the fungus *Coccomyces hiemalis*, when uncontrolled, has a marked influence on the yield and quality of sour cherry fruit. For this reason, it is considered the most important fungus disease of sour cherry. The fungus attacks sweet cherries and a closely related species or "form" of the pathogen *(C. prunophorae)* is occasionally a problem on plum. In some cherry growing regions, the disease is referred to as "shot-hole", although this symptom is more common on plum. Cherry leaf spot is the preferred common name for the disease on cherry.

Symptoms

The disease appears first on the upper side of the leaf as small, circular, purple spots (Figure 23). Later, they may turn brown and form a definite boundary. On sweet cherry, the spots are often larger and more nearly circular than those on sour cherry. During rainy weather, light-pink to white masses appear on the underside of the leaf in the center of the spots.

After the cherry leaves become infected, they turn yellow and fall. Only a few lesions are required to produce leaf yellowing and dropping. When control measures are inadequate, complete defoliation often occurs before harvest (Figure 24). The fruit on such trees fail to mature properly, are light colored, low in soluble solids, soft and watery. They may be smaller or larger than normal, depending on time and severity of defoliation. Early loss of leaves reduces tree vigor. The buds and wood are more susceptible to injury from low temperatures in winter. This becomes a severe problem when regrowth occurs late in the season, preventing the trees from hardening off properly in the fall.

Although the disease occurs primarily on the leaves—fruit, stems, and leaf petioles may become infected when the disease is severe. Fruit infection is sometimes so severe that the crop is unmarketable.

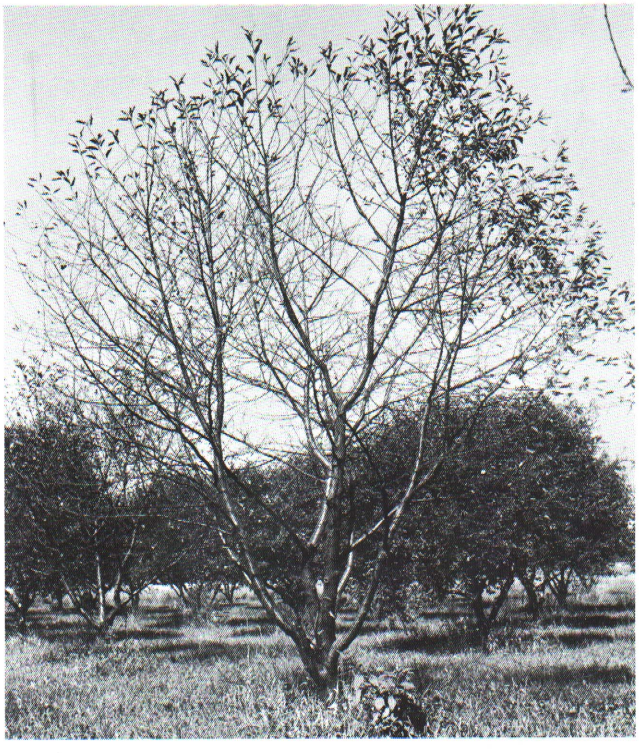


Figure 24—Premature defoliation of unsprayed tree from leaf spot: trees in background were on a complete spray program.

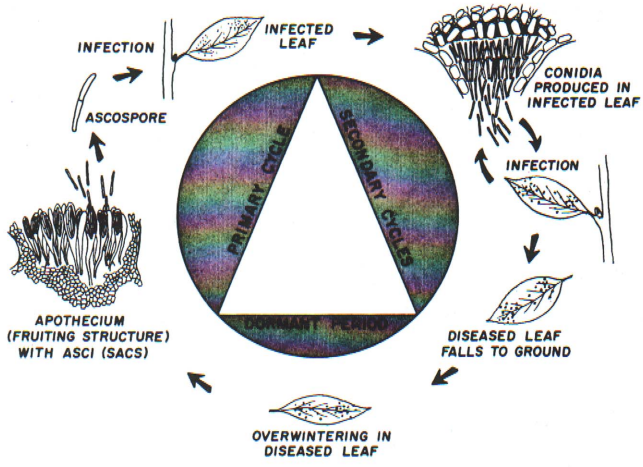


Figure 25—Disease cycle of cherry leaf spot.

Disease Cycle

The fungus overwinters in infected leaves on the orchard floor (Figure 25). In late winter and early spring it produces fruiting bodies (apothecia) which normally mature during the blossoming period. Upon wetting, the ascospores are released. Greatest ascospore discharge occurs at temperatures of 61°F and higher, somewhat less at 54°F, and very little from 39° to 46°F.

Infection will not take place until the leaves have developed mature stomates, (breathing pores), in the underside of the leaf. The leaves are usually not unfolded sufficiently for infection until petal fall. However, in years when blossom bud development is delayed by low temperatures, leaf development may be sufficient for infection prior to petal fall. A wetting period of only a few hours is required for infection when favorable temperatures exist. Ascospores ma-

ture gradually over an extended period and complete spore discharge from the apothecium may not occur until mid-summer.

Development of visible lesions occurs in 5 to 15 days depending on temperature and moisture conditions. Optimum conditions for lesion development are temperatures of 60° to 68°F with rainfall or high humidity. Soon after the lesions appear, the acervuli, containing flesh-colored masses of spores (conidia), are visible on the under surface of the leaf. The conidia are readily carried by splashing rain and air currents to the lower leaves of the tree. Secondary spread and infection by conidia continues repeatedly during favorable weather until the leaves drop to the ground in the fall.

Control

The disease cycle could be broken and the disease eliminated by completely preventing the fungus from developing fruiting bodies on fallen leaves. It is not currently possible to completely inhibit apothecial formation in the orchard. However, several experimental chemicals have been highly effective for this purpose in small scale tests. Fall fertilization helps hasten decay of leaves and reduce the number of spores discharged by the fungus in the spring rains. Cultivating the orchard in spring or mowing late in the fall also helps reduce inoculum.

The primary approach to the control of leaf spot is use of fungicide sprays.

Figure 23—Spotting of cherry leaves due to cherry leaf spot fungus.

Figure 26—Severe black knot infection to scaffold branches of prune.

BLACK KNOT OF PLUM

Increased interest in prune and plum production in Michigan has made black knot, caused by the fungus *Dibotryon morbosum*, more important to commercial fruit growers. While it is severe on wild cherry seedlings, the disease is not known to occur on commercial cherry varieties. However, infected wild cherry are dangerous because the fungus can spread from wooded areas containing diseased cherry to the commercial or backyard plum planting. Bearing capacity is markedly reduced when the disease is allowed to get out of hand, since heavy pruning is required to remove the resulting knots.

Symptoms

The disease is characterized by the production of elongated swellings or knots on the limbs of susceptible prune and cherry (Figure 26). These corky outgrowths predominate on small twigs and branches but may also be located on larger scaffold branches and in the trunk area. Knots, which tend to be longer than wide, may be a foot or more in length.

Newly formed knots are greenish and soft, but become hard and black with age (Figure 27). Old knots may be covered with a white or pink parasitic fungus during summer and be riddled with insect bores.

Disease Cycle

In spring, when one-half inch of new growth is present, ascospore discharge is initiated from perithecia located on the surface of the knots. Spore discharge occurs during periods of rainfall. Spores are carried by wind and rain to the site of infection. Infection takes place through the unwounded surface and is most severe when wet conditions are accompanied by temperatures between 55° and 77°F. Spore discharge and infection are generally most severe one to three weeks after bloom. Infection continues to occur until terminal growth stops.

Several months are required between initiation of infection and appearance of knots. Some knots are visible by late summer, others not until spring the next year. At least one year (usually two), are required before new knots produce mature perithecia. The fungus in old knots progresses during fall and early spring months, extending the knots several

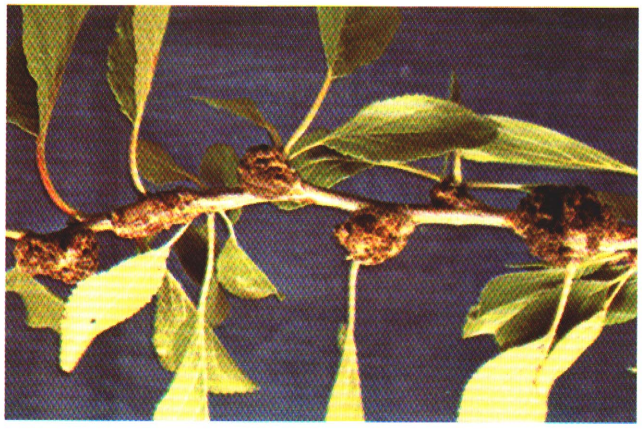


Figure 27—Black knot infection to the current season's growth.

inches in length. This advancement is usually not visible until late spring.

Control

The two major plum varieties grown in Michigan, Stanley and Damson, are susceptible to black knot. Bluefre is also susceptible. Recent varietal susceptibility tests in Pennsylvania show Shropshire and Stanley to be highly susceptible; Methley, Milton, Early Italian, Brodshaw, and Fellenburg moderately; Shiro, Santa Rosa, and Formosa slightly susceptible and President apparently resistant to the black knot fungus.

Plans for preventing the disease from building up in new plantings should be developed before the orchard is established. Wild plums and wild seedling cherries, other than black or pin cherry, should be removed from fence rows and nearby wooded areas. Establish and maintain at least a 600-foot border free of wild hosts. Do not plant new plum orchards next to old plantings with black knot. These simple precautions will greatly reduce problems with the disease and increase orchard longevity.

Once established, the orchard and surrounding wooded areas should be patrolled annually for signs of infection. Although infections are more difficult to find in mid-August, removal of knots at this time is desirable because they are generally fully extended. Cuts should be made two to three inches below the swelling. Remove knots on the trunk or main branches by cutting away the diseased tissue down to the wood and at least one-half inch outward beyond the margin. When infection is severe, leave knot removal until late winter or spring so as not to increase vigor excessively. Since the fungus may have extended beyond the swelling, cuts should be made well below the visible infection. Remove the knots from the orchard and burn before April 1 since they can serve as a source of inoculum if left on the orchard floor.

Spraying, if done in conjunction with a sanitation program, will help prevent the disease. Spraying alone will not control the disease.

Figure 28—Decay of Stanley prune and sporulation of the brown rot fungus.

BROWN ROT OF STONE FRUITS

Brown rot is a destructive fungus disease of apricot, peach, nectarine, plum, and cherry in Michigan. The disease reduces yields in the orchard by infecting blossoms, twigs, and fruit. After harvest, fruit decay poses a constant threat. In seasons with climatic conditions favorable for infection, entire crops may be lost, almost overnight.

There are two brown rots of stone fruit crops in Michigan. American brown rot is caused by the fungus *Monilinia fructicola* and occurs throughout the state. European brown rot, caused by *M. laxa*, occurs primarily on tart cherry in areas north of Oceana County along Lake Michigan. The European fungus is generally a blossom and spur blighter, not a fruit rotter.

Symptoms

Brown rot attacks blossoms, spurs, shoots, and fruit. Symptoms may develop on a few or all of these plant parts. Infected blossoms wilt, turn brown, and persist into the summer. The fungus of American brown rot may spread from the blossom into the flower cluster base and into the spur, particularly on apricot, peach, and nectarine; infrequently on sour cherry. European brown rot infection commonly spreads into the fruit spur on sour cherry.

When the fungus moves down the spur of blighted twigs to the branch below, small cankers are formed.

As they expand, cankers may girdle the branch or twig and cause the terminal growth to wither and die. Gummosis may accompany the blighting of spurs and formation of cankers. Succulent shoots are sometimes blighted by direct infection near their tip.

Fruit decay is worst on mature fruit although immature fruit may develop the disease under certain conditions (Figure 28). At first, a small, circular, light brown spot develops on the surface of the fruit. The disease expands rapidly under favorable conditions, destroying the entire fruit in a few hours. Rotted fruit may fall to the ground or persist as mummies on the tree.

Under wet, humid conditions, ash-gray tufts of fungus growth develop over the surface of the lesions. These structures, called sporadochia, produce conidia or spores important in spread of the disease. Appearance of the fungus on a lesion is the most obvious characteristic of brown rot.

Disease Cycle

Brown rot fungi overwinter in mummies on the tree or ground and in twig cankers (Figure 29). While fruit buds are opening, small, cup-shaped mushrooms called apothecia arise from mummies on the ground. Formation of the apothecial stage is most important for American brown rot since the sexual stage of the European form is rare. Moisture is required for apothecial development. Development is also favored by temperatures of 63° to 68°F.

Spore containing structures called asci, each with eight ascospores, are produced within the apothecia. Upon wetting, the ascospores are forcibly ejected into the air and carried by wind to the blossoms where they infect. Apothecia disintegrate by early summer and do not contribute directly to infection at harvest.

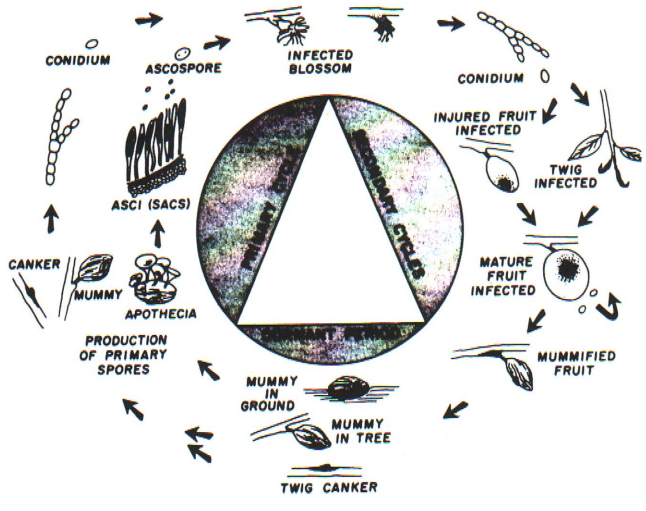


Figure 29—Disease cycle of brown rot.

Infection may also arise from conidia produced on the surface of mummies and cankers in the tree. Spores are carried by wind or splashing rain to susceptible tissues. Relative humidity of 85 percent or higher is necessary for conidial production.

In summer, brown rot activity is reduced but activity increases as the fruit start to mature. Infected blossoms and green fruit are the main source of conidia for infecting fruit at harvest. Infection may occur directly through the cuticle or through natural openings in the fruit. Wounded fruit are infected much more readily than unwounded fruit. Since rotting and spore production can occur in a few days, the disease is able to build up rapidly, even from low levels.

Environment plays an important role in development of the disease. Warm, wet, humid weather is particularly favorable for brown rot. The hours of wetting necessary for blossom infection decrease from 18 hours at 50°F to 5 hours at 77°F. Infection rate decreases above 80° and below 55°, but may continue at temperatures as low as 40°F. Mature fruit decays in 36 to 48 hours under optimum decay conditions.

Control

The entire brown rot control program, although initiated well before bloom, is designed to reduce decay of mature fruit. Use of several control measures will prevent disease buildup at harvest.

Control starts with removal of all fruit, mummies, and blighted twigs from trees after the last picking. This reduces the amount of brown rot overwintering in mummies and twig cankers. Trashy cultivation, just before bloom and no later than mid-bloom, will destroy the apothecia by disturbing the mummies.

Spray applications: Blossom sprays are an important consideration in the overall control program. Severe crop loss can occur from blossom infection on highly susceptible crops like apricot, nectarine, peach, and sweet cherry when weather conditions are favorable. Even light to moderate levels of blossom blight must be avoided to insure against fruit infection later.

Timing of sprays is based on development of the blossom buds and climatic conditions. The pistil is the most susceptible flower part to American brown rot. During wet weather, the first spray should be applied when many of the pistil tips (stigmas) extend above the flower, even unopened flowers. A protective spray is needed earlier when periods of warm, wet weather occur, since infection of the petals is possible. If the orchard has a history of brown rot or blossoms are frost-injured, adjustment in timing of sprays will be necessary. The number of sprays required during bloom varies from year to year. Spray applications every four to five days are necessary if favorable conditions for brown rot persist.

European brown rot infects tart cherry blossoms primarily through the anthers and stigma. Use protective sprays if the anthers and stigmas are exposed and favorable weather for infection anticipated.

Pre-harvest sprays: To control brown rot on ripening fruit, apply pre-harvest fungicides. Three sprays beginning two weeks before harvest may be needed in rainy seasons. Where brown rot has built up, the schedule should be initiated earlier and additional sprays or dusts made at closer intervals. Control of plum curculio, tarnished plant bug, and oriental fruit moth are essential to prevent early infection to immature fruit.

Post-harvest control: To prevent decay during storage and in transit, fruit should be picked and handled with care. Use only clean containers and remove ripe and rotting fruit from packing areas periodically. Hydrocool fruit soon after harvest to remove field heat and reduce rot development. Hot-water treatments, fungicide dips, wax-fungicide treatments, spraying of fruit while grading and controlled atmosphere storage all help reduce decay.

BACTERIAL SPOT

Bacterial spot is a problem on susceptible apricot, peach, nectarine, and plum varieties. It causes severe defoliation and fruit spotting, which weakens the trees and makes the fruit unmarketable.

The disease was originally described by Erwin F. Smith in 1902 on Japanese plums from Michigan. He attributed it to a bacterium known today as *Xanthomonas pruni.* Widespread planting of susceptible fruit crops has increased the significance of this problem in Michigan.

Symptoms

The bacteria attack the leaves, fruit, and tender growing shoots. Leaf lesions are small and generally angular in outline. Initially, they appear as water-soaked areas on the underside of the leaf, but eventually develop into brown to black spots (Figure 30). The disease is generally worst at the tip of the leaf, where an inch or more may be killed. In Michigan, typical leaf lesions are found primarily on peach, nectarine, plum, and prunes.

Severely infected leaves soon turn yellow and fall to the ground. On sensitive varieties, a few lesions result in severe defoliation; tolerant varieties require many more. Heavy defoliation early in summer reduces fruit size and weakens the tree.

Fruit infected early in the growing season develop cracks or checks in the skin (Figures 31 and 32). Lesions extend into the flesh, resulting in deep pits which must be removed in processing. Under certain weather conditions, fruit lesions show gumming. Late season infections are superficial and usually only give the fruit a mottled appearance.

Infection in the current season's growth can result in the production of two types of cankers. Lesions apparent during the year of infection are called summer cankers. These are usually located between the nodes. Infections occuring in the fall but not visible until the following spring are referred to as spring cankers. These tend to be found at buds or nodes. Twig infection is nearly always less common than leaf or fruit infection.

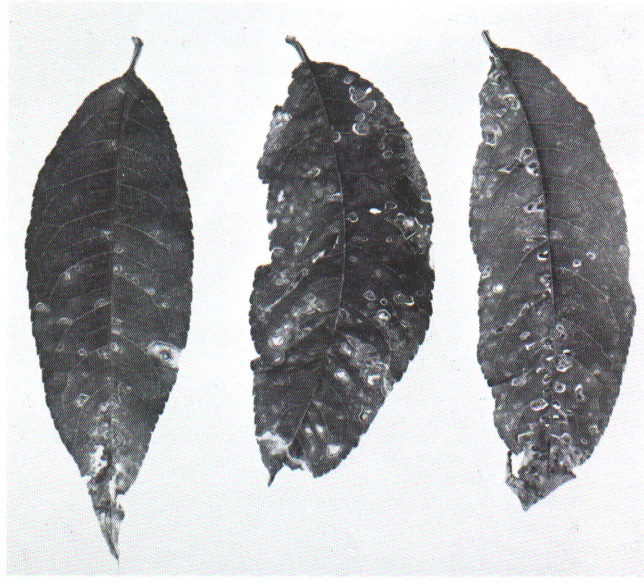


Figure 30—Angular lesions of bacterial spot on peach leaves.

Figure 31—Bacterial spot infection to Suncling peach fruit.

Disease Cycle

Overwintering occurs in spring cankers (Figure 33). Recent evidence indicates these are initiated primarily through leaf scars. The scars left by late maturing leaves are most susceptible to infection. Survival of bacteria in summer cankers on peach is rare in northern states, more common in the South. On plum and

Figure 32—Bacterial spot on apricot.

apricot, bacteria may survive in summer cankers for more than one season.

When canker development is resumed in spring, the bacteria ooze out of the lesions and are carried in water droplets to young leaves, fruits, or shoots. Moisture from fog and dew is important in the dissemination process. Hard, driving rains are more important than gentle rains in initiating new infections. This explains why bacterial spot can be proportionally more severe on one side of the tree than on the other.

Secondary spread from summer cankers, leaf lesions, and fruit infections continue whenever environmental conditions permit. Periods of frequent rainfall with moderate temperatures and fairly high winds favor infection. The disease may become quite severe if these conditions persist through June and July. Extended periods of hot, dry weather reduce disease spread and development.

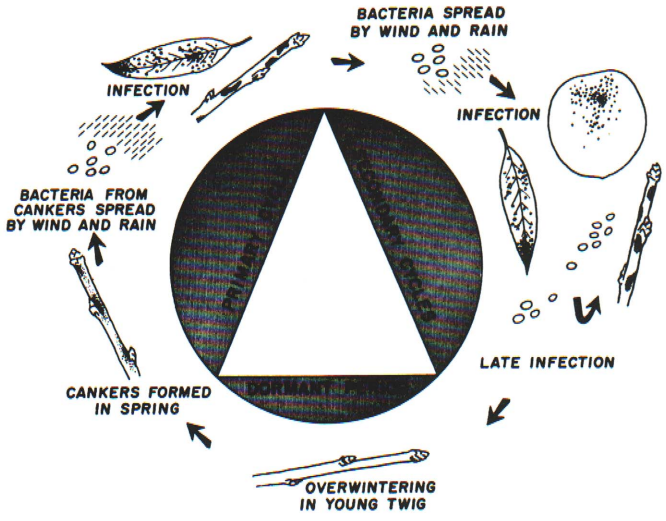


Figure 33—Disease cycle of bacterial spot.

Control

Use of resistant varieties is the primary method for controlling bacterial spot. Some of the more susceptible varieties: Blake, Suncrest, Suncling, Sunhigh, and Jersey Queen. J. H. Hale, Babygold 5, Elberta, Kalhaven, and Rio-Oso-Gem are moderately susceptible. Relatively resistant varieties: Redskin, Redhaven, Loring, Dixired, Sunhaven, Jefferson, and Madison. Apricot varieties Goldcot (SH-6), SH-50, and SH-7 are susceptible, as well as certain nectarine varieties. Know a variety's relative susceptibility to bacterial spot before buying. Bacterial spot problems can also be reduced by locating new plantings away from older ones containing susceptible varieties. Planting susceptible peach, apricot, prune, and nectarine orchards in close proximity to one another has contributed to build-up of this disease in Michigan.

Balanced fertilization programs should be followed. Excess nitrogen may aggravate the problem, while trees weakened from poor nutrition or other causes are more sensitive to the disease.

Some spray programs help control bacterial spot. However, these programs only suppress development of the disease—they do not eliminate it. Because of the cost and uncertainty of chemical control, the best way to control bacterial spot is through resistant varieties.

POWDERY MILDEW OF PEACH

Powdery mildew is rarely a major problem in Michigan peach orchards. It causes some culling of fruit in years when weather conditions are favorable for infection. The problem is greatest on susceptible varieties and in orchards where sulfur has been dropped from the spray program.

The causal organism is generally considered to be the fungus *Sphaerotheca pannosa*. However, *Podosphaera oxyacanthae*, the common mildew species on cherries, also attacks peach. Due to difficulty in separating the species with certainty, the relative importance of each is not known.

Symptoms

In the orchard, the disease is observed primarily on the fruit. Lesions on young fruit appear as white powdery spots (Figure 34). As the fruit mature and the fungus growth responsible for the early coloration disappears, the spots look hard and leathery. The dark brown spot on the mature fruit is often observed at harvest. The term "rusty spot" is used to describe this symptom in some regions of the U.S.

Leaf and shoot symptoms may occur on nursery stock or on new shoots of older trees. Infected leaves are coated with a thick layer of fungus mycelium and become distorted and narrow. Shoot growth is stunted. Foliar symptoms are generally less prevalent in peach mildew than in apple and cherry mildew.

Figure 34—Mildew on surface of peach.

Disease Cycle

The mildew fungus overwinters as mycelium in dormant peach buds. When infected shoots emerge in spring, the pathogen initiates its growth and produces spores (conidia). The spores are carried to the leaves, twigs, and fruit by wind and rain. Infection may also be initiated in the orchard by spores produced outside the planting. Other stone fruit crops and roses are common external sources of disease.

Fruit have been observed to be most susceptible when young, but they become resistant with pit hardening. Lesion extension continues for a short time after resistance to infection develops. New leaves can be infected throughout most of the season but become resistant with age. The disease is most severe following periods of moderate temperatures and high humidity. Spore germination will occur at relative humidities of 43 to 100%. Free moisture is not required. Optimum temperature for germination is 66° to 72°F. Under favorable environmental conditions, new lesions develop in ten days.

Control

Problems with this disease can be eliminated by avoiding mildew susceptible varieties. Reo-Oso-Gem and Redskin are susceptible under Michigan growing conditions. If these varieties are planted, supplemental sprays with mildewcides are necessary.

Fungicide sprays should be initiated at petal fall and re-applied periodically until the pit hardening stage of fruit development. If a severe problem exists, chemicals can also be used to reduce the severity of leaf and shoot infection. For this purpose, start spray applications early and continue until growth stops.

PEACH LEAF CURL

Leaf curl is a springtime leaf disease of peach, and, to some extent, ornamental species closely related to peach. Loss of foliage in early summer due to this disease does not occur throughout Michigan every year. To insure against it, control measures should be carried out annually.

Symptoms

Leaves infected by the leaf curl fungus *Taphrina deformans* appear in mid-May and are easily distinguished from healthy ones as they become puckered and thick. Puckering of the leaves is primarily along the midrib with part or all of the leaf infected. The leaves are usually flushed with red or purple when they first appear (Figure 35), but later become yellow to brown and drop from the tree.

Twigs infected with peach leaf curl are stunted, swollen, and usually produce curled leaves at their tip. Symptoms are confined to the new growth. Infection of the flowers and young fruit have been reported but affected parts are not often noticed since they fall early.

As the season progresses, areas on the diseased leaves develop a powdery-gray appearance. This results from the production of fungus spores. Spore production is most pronounced following periods of wet, humid weather and occurs primarily on the upper surface of the leaf. The grayish appearance distinguishes leaf curl from curling of leaves by other factors.

Disease Cycle

Leaf curl can cause severe infection even though the disease was light or absent the previous spring. This is because the spores of the leaf curl fungus are relatively resistant to adverse weather conditions. In fact, these tiny spores can remain lodged on the surface of

Figure 35—Peach leaf curl.

the twigs for two or more years. Furthermore, ascospores produced on the surface of diseased leaves can produce large numbers of secondary spores or budconidia. In suitable weather, the ascospores bud profusely, assuring high levels of inoculum for infection.

Infection occurs during a relatively short spring period. Hibernated spores are washed to the surface of leaf buds by spring rains. The spores continue to multiply during periods of moist weather until the leaf buds swell and open. Once the bud scales loosen, the small conidia can be carried in films of water to the leaf tissue where infection takes place.

Environment can limit leaf curl infection and explains why the disease does not occur throughout Michigan every year. Leaf curl is worst when the weather is cool and wet. Low temperatures are thought to retard maturation of leaf tissue, thus prolonging the period of time infection may occur. The fungus can penetrate young peach leaves readily at temperatures between 50° and 70°F, but only weakly below 45°F. Rain is necessary for infection. The tree is susceptible only during the relatively short period of bud swelling and opening.

Figure 36—Typical peach cankers.

Control

A single spray, if applied at the correct time and with the correct material, will provide nearly perfect control of leaf curl. To be effective, the application *must* be made before the buds begin to swell. Fall sprays after the majority of the leaves have fallen, or spring sprays within three to four weeks of bud swell are effective, if applied thoroughly. Once the fungus enters the leaf, the disease cannot be controlled.

PERENNIAL CANKER

Perennial *(Cytospora/Valsa)* canker is caused by two related fungi, *Cytospora cincta* and *C. leucostoma*. Both species attack peach, apricot, prune, plum, and sweet cherry. *C. leucostoma* is the predominant species on peach; *C. cincta* is more common on apricot. The disease is prevalent in many Michigan peach orchards and is a frequent cause of limb dying and death of peach trees. It is less important on other stone fruit crops.

Symptoms and Disease Cycle

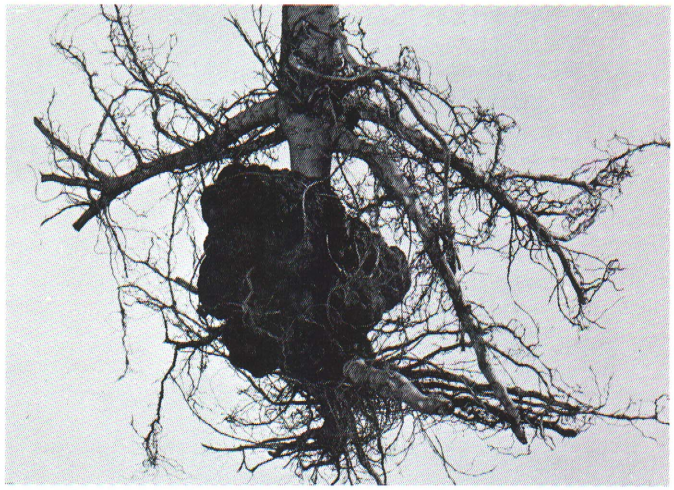
Perennial cankers are oval to linear in outline and are usually surrounded by a roll of callus at the margins (Figure 36). These enlarge gradually on a year to year basis until the limb or trunk is completely girdled. Active cankers often have gum associated with them, but "gummosis" is not unique to canker since it may be caused by several unrelated factors.

The fungi overwinter in cankers or on dead wood. Pycnidia containing small conidia are produced in diseased tissue under the bark. During wet periods or periods of high humidity, the spores are exuded through the bark in tendrils from pycnidia. The conidia are carried short distances by splashing and wind-driven rain—much farther by wind. Infection is through damaged or injured bark. Cold injury is the most important factor predisposing trees to canker (Figure 37), but pruning wounds, mechanical damage, insect punctures, and leaf scars are other entry points.

Moisture is required for spore germination. The rate of canker development following infection depends on temperature and the species of fungus involved. *C. cincta* is favored by lower temperature than *C. leucostoma*. Optimum temperatures for growth of these two fungi are approximately 68° and 86°F, respectively. When temperatures are not favorable for fungal activity, callus formation occurs. Canker activity resumes when temperatures again favor the fungus.

Control

Because of the manner of infection and development of this disease, no single control measure is adequate. Most of the known control methods act indirectly by reducing points of entry or by reducing the level of inoculum.



Figures 37 and 38—Peach canker in crotch angle of tree following low temperature damage (left); crown gall on Mazzard F12/1 rootstock (right).

- Avoid soils with poor internal drainage and remove wet spots by tiling before establishing new plantings.
- Do not plant young peach orchards or replant trees next to older orchards or trees with canker.
- Delay orchard pruning until growth starts in spring. Moderate to severe pruning in November, or earlier, can severely weaken or kill trees. Late pruning promotes quick healing.
- Eradicate cankers and remove badly cankered limbs, branches, or trees. Burn cankered limbs soon after pruning. Sanitation is a must during the early life of the orchard.
- In cultivated orchards, plant a cover crop by July 1 and mow thereafter.
- Fertilize early and according to recommendations.
- Try to avoid mechanical and insect injury and do not leave pruning stubs.
- Apply fungicide sprays after pruning, but before rain.

CROWN GALL

Crown gall does not normally affect the fruit grower directly because infected nursery material is culled before trees are sold. He is affected indirectly, since supplies of trees on susceptible rootstocks may be limited in years when crown gall is severe. This means healthy trees will probably cost more.

Symptoms

Crown gall is characterized by the formation of tumors or galls from one-fourth inch to more than three or four inches in diameter (Figure 38). Galls are usually located on the roots or crown, but, under certain conditions, the disease may develop on the aerial portions of plants. Several galls may occur on the same root or stem. Galls are typically globular in shape but may be elongated or otherwise irregular. Their surface is gnarled and becomes dark with age, often almost black.

Disease Cycle

The bacteria which cause crown gall, *Agrobacterium tumefaciens*, are widely distributed in soil where susceptible crops have been grown. New soils may become contaminated by planting nursery stock dug from problem soils. Once in the soil, the bacteria can survive for several years. When susceptible crops are planted in infested areas, the bacteria enter the roots and crown through wounds produced in caring for, and handling, the nursery material. They may also enter through wounds made by root feeding insects.

In addition to primary galls, secondary galls sometimes develop at some distance from the initial infection. These galls may develop on unwounded tissue and bacteria cannot be found associated with them. Although the mechanism of secondary tumor formation is not entirely known, it is believed a tumor-inducing substance is produced in the primary gall and moves in the plant to induce galls.

Control

Crown gall is severe on Mazzard cherry and peach. It attacks other tree-fruit crops, but is usually less severe. Susceptible nursery stock should not be planted in soils with a history of crown gall. Crop rotation utilizing grain crops helps reduce the disease.

No chemical or soil fumigant has been found to be consistently effective against crown gall. Certain compounds have been reported to selectively kill tumor tissue and thereby rid the tree of infection. These chemicals have not proven effective for nursery stock.

RHIZOPUS ROT

This disease is important primarily when fresh peach, sweet cherry, or plum are stored or otherwise delayed in reaching the consumer. The disease may develop in storage, transit, or market place, depending on temperature and moisture conditions.

Symptoms and Disease Cycle

A rot very similar to brown rot develops on the surface of fruit. The skin of fruit affected with Rhizopus rot readily slips from the flesh underneath, while skin on a brown rot lesion does not. If humidity is low, infected fruit turn black and shrivel into mummies. Under more humid conditions, the infected fruit release liquid and become covered with a "whisker-like" growth (Figure 39).

Infection may occur in the orchard at harvest time or in post-harvest handling of the fruit. Fruit with injuries are most susceptible. However, once the disease becomes established, it can move onto sound fruit nearby. The fungus invades the tissue quickly, producing tufts of whisker-like fungal growth over the lesion. Black sperical structures (sporangia), each containing thousands of small sporangiospores, are produced at the tip of the bushy growth. The outer wall of the sporangia ruptures easily, freeing the sporangiospores, which then float about in the air or drop to the surface. When environmental conditions are not favorable, dark colored spores (zygospores) are formed. These spores have a thick cell wall and can survive periods of dryness or unfavorable temperatures.

Control

Many of the practices used to control brown rot aid also in the control of Rhizopus rot. Careful handling of the fruit to avoid wounds, clean storage containers and warehouse facilities, and proper hydrocooling reduce the incidence of the disease. Fungicide sprays applied near harvest, or post-harvest dips, sprays or impregnated wraps, are effective control procedures.

Figure 39—Rhizopus rot.

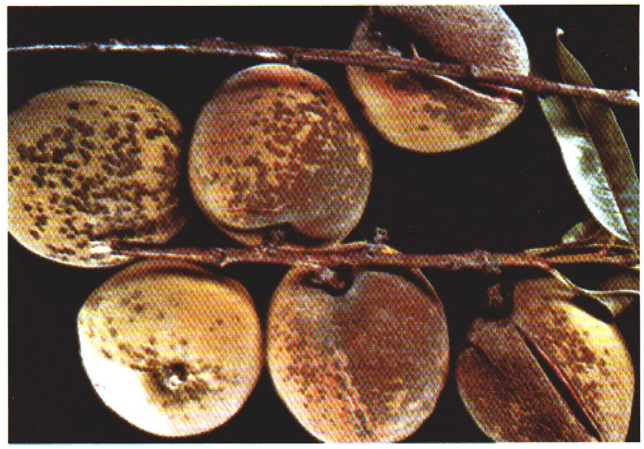


Figure 40—Scab on peach.

PEACH SCAB

Peach scab is a relatively minor disease which occurs primarily in southwestern Michigan. It is worst where a good spray schedule is not followed early in the season. The causal fungus, *Cladosporium carpophilum*, also attacks apricot and nectarine.

Symptoms

Although the disease occurs on the twigs and leaves, it is most often observed on the fruit. Early infections appear as small, greenish, circular spots on the surface of the fruit. These spots do not generally appear until the fruit are half-grown and tend to be concentrated at the stem end. Older lesions become black and velvety in appearance as spores are produced (Figure 40). On apricot, the lesions are more brown than black. Lesions may run together when numerous, resulting in abnormal growth of the fruit, and, in severe cases, cracking of the skin and flesh.

Disease Cycle

The fungus overwinters in lesions on the twigs and conidia are produced in the spring. The conidia begin to infect the peach a few weeks after petal fall. On nectarines and apricot, infection occurs at about shuck fall. The fruit remain susceptible until harvested. Fourty to 70 days elapse from the time the spore lands on the fruit till the disease is visible. Thus, the disease is usually not observed until the fruit are well grown. Spores from the fruit re-infect the twigs and leaves.

Control

The disease is controlled primarily with fungicide sprays, although pruning helps increase air circulation and reduces infection. The first spray should be applied 10 to 12 days after the shuck split stage of bud development on peach, somewhat earlier on

Figure 41—Angular dead spots on Schmidt sweet cherry leaves due to bacterial canker.

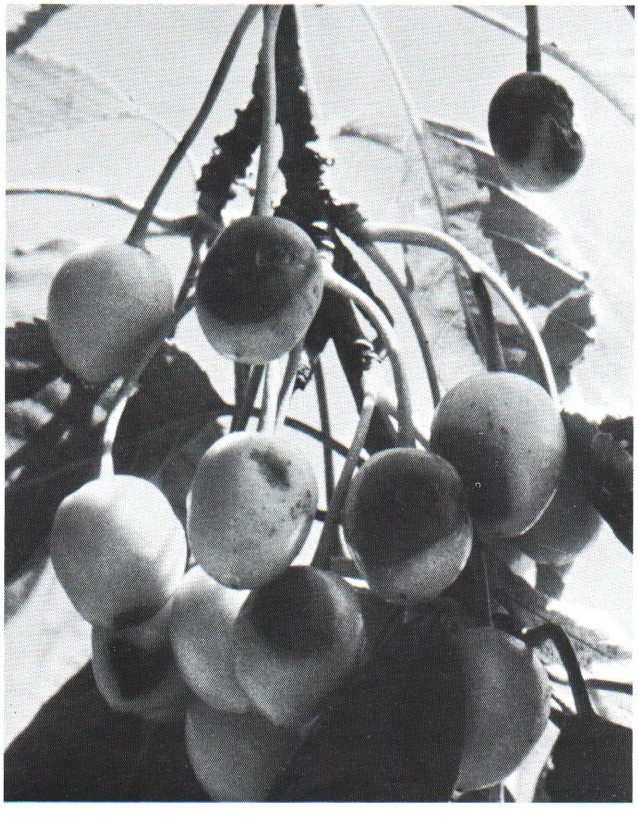


Figure 42—Rot of immature sweet cherry fruit caused by bacterial canker.

apricot and nectarine. Sprays should be applied every 10 to 14 days until 40 days before harvest. Because of the long infection period, late infections will not appear before the fruit are normally consumed. Sprays for brown rot help reduce late infection to the fruit, twigs, and leaves.

BACTERIAL CANKER

Although bacterial canker has probably been present for some time in Michigan, its identification on stone fruit crops in our state is relatively recent. Two closely related bacteria, *Pseudomonas syringae* and *Ps. mors-prunorum*, cause the disease. Bacterial canker has been found primarily on sweet cherry in Michigan although it occurs on peach and plum in other fruit growing regions.

Symptoms

The disease attacks most parts of sweet cherry trees. Branches and twigs are cankered and a heavy exudation of gum is usually associated with these cankers in the fall and again in the spring. Leaves on the terminal portions of severely cankered branches may wilt and die in summer or early fall from girdling. Under severe conditions, large scaffold limbs may be killed.

Leaf and fruit infection are relatively common under Michigan conditions. They appear to be most severe when prolonged periods of wet, cool weather occur after bloom. Infected leaves develop dead areas or spots which drop out, giving them a tattered appearance (Figure 41). Severely infected leaves fall to the ground prematurely. Deep, black depressions develop in the flesh of young cherry fruits and on the fruit stem (Figure 42). Infected fruit are more susceptible to brown rot than healthy fruit and may contribute to outbreaks of this disease.

Leaf and flower buds may be killed while dormant, thus failing to open in the spring. Small cankers sometimes develop at the base of diseased buds. This symptom has also been observed on peach. In some cases, infected spurs open normally, but collapse in early summer, leaving wilted leaves and dried up fruit. Blighting of spurs year after year eliminates bearing surface and makes trees worthless (Figure 43).

Disease Cycle

The bacteria overwinter in cankers, in infected buds, and possibly systemically in apparently healthy tissues of the tree. Infection occurs in the fall through leaf scars and in spring through scars left by bud scales. The relative importance of each area for infection under Michigan conditions is not known.

In spring, bacteria from cankers and infected buds are disseminated by the washing action of rain to emerging tissues. Infection to developing blossoms, leaves, and fruit occurs through natural openings and wounds. Leaves and blossoms are probably more susceptible when injured by frost.

Secondary spread of the disease from primary infections occurs throughout the growing season and is important in build-up of the disease. Periods of frequent rainfall, cool temperatures, and high winds are most favorable for early season infection and spread. Later in the season, leaves are less suscep-

Figure 43—Schmidt sweet cherry tree with severe symptoms of bacterial canker.

tible and temperatures too high for bacterial activity. Disease development remains low until fall when environmental conditions again become favorable for the disease.

Control

Because of the recent recognition of this problem in Michigan, control procedures are not yet established. However, tests of protective spray programs are currently underway.

Extensive field observations indicate that the disease is troublesome on some varieties but not others. Hardy Giant, Schmidt, and Windsor varieties are susceptible and often severely damaged. Hardy Giant appears to be most susceptible and should not be planted. On other varieties, periodic removal of cankers helps to eliminate the disease where present.

ARMILLARIA ROOT ROT

This root rotting disease is caused by the mushroom-producing fungus *Armillaria mellea*. Although Armillaria root rot is not a major problem on stone fruit crops in Michigan, it often eliminates otherwise good orchard sites from production. Old orchard sites known to be infested with the disease should be avoided.

Figure 44—Mushrooms at the soil line and white fungal mat beneath the bark are symptoms of Armillaria root rot.

Symptoms and Disease Cycle

The above-ground symptoms of Armillaria root rot resemble those of other root problems and indicate which trees to select for further examination. Affected trees show early reddening of their foliage in the fall, reduced terminal growth, and in more advanced stages—dieback. One or two scaffold limbs may die initially, followed by death of the entire tree. Trees often die-out in a circular pattern from a central point or localized area in the orchard.

Suspect trees should be examined for root or crown injury. Trees infected with Armillaria have dead roots but some live bark on the crown. Dense, white, fungus growth is evident any time of year throughout the dead tissue (Figure 44). A fan-shaped, fungal mat is often present in the cambium between the bark and the wood. Armillaria is distinguished from other fungi by the presence of dark brown to black threads or rhizomorphs over the surface of the diseased area (Figure 45). In late fall, honey-colored mushrooms are often observed at the base of affected trees.

The rhizomorphs and mycelial strands of fungus on infected roots enable the pathogen to spread from tree to tree. Moreover, the fungus can survive for several years on root fragments in the soil, awaiting planting of a susceptible crop. Since many woody and herbaceous plants are susceptible, care must be taken in selecting crops to plant. In the Midwest, the greatest problem occurs when fruit trees are planted on newly cleared forest land.

Control

Fumigation has been only moderately effective in controlling the disease because of the ability of the fungus to survive deep in the soil. This is particularly true in the Midwest where heavy soils reduce the movement of fumigants.

Newly-cleared forest land or land with an Armillaria history should not be planted to susceptible crops. Cherry on Mahleb rootstock is the most susceptible tree fruit. Pear is considered to be tolerant. Tolerant clones of Myrobolan, St. Julien, and Methly plum are also known.

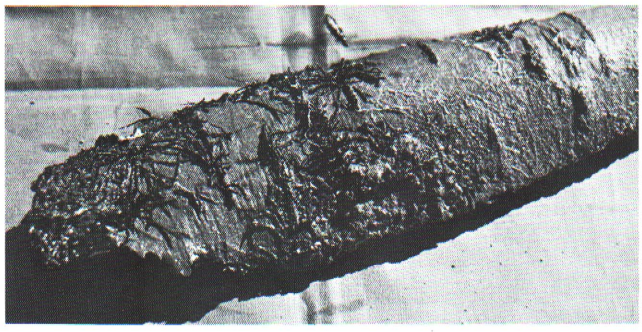


Figure 45—Dark strands (rhizomorphs) of Armillaria on trunk of affected tree.

X-DISEASE

This disease was first identified in Michigan in 1941 when diseased chokecherry bushes were found in Lapeer County. Spread of the disease into commercial stone fruit orchards has occurred sporadically since that time. Peach, nectarine, sweet cherry, and tart cherry are attacked by X-disease.

Distribution of X-disease in Michigan corresponds to the range of the susceptible wild chokecherry. The disease occurs in the fruit growing areas of southern Michigan, but not the fruit areas of the northern part of the lower peninsula. Chokecherry bushes are also more abundant in lower Michigan than in any other part of the state.

Symptoms

The disease is easiest identified on peach. The first indication of its presence is a change in appearance of the foliage. Leaves on isolated branches tend to curl inward in mid-June and July, and develop water-soaked spots which become yellow to reddish-purple (Figure 46). In July, the discolored areas in the foliage drop out, giving leaves a tettered appearance. Leaves on affected branches fall prematurely, starting at the base. Only a tuft of leaves at the tip remains until the end of the season. Two to three years after initial infection, all branches show symptoms.

Presence of leaf symptoms should not be interpreted as proof of X-disease infection, but should suggest that something is wrong with the tree. Similar symptoms may develop as a result of nitrogen deficiency, bacterial spot, winter injury, peach tree borer, peach canker, or mechanical injury. If further examination shows these other factors are not present, and if chokecherries are found within 500 feet of affected trees, it can be assumed the trees are infected with X-disease.

Cherries on Mahaleb root are killed suddenly in mid-summer by the disease. Trees on Mazzard rootstock decline slowly. Their fruit ripens unevently and many small, immature cherries are produced which have an insipid flavor. The fruit is worthless on all infected sweet and tart varieties. Diseased trees are usually winterkilled after a few years. Again, these symptoms only suggest X-disease, since similar symptoms develop from root killing caused by improper soil drainage, girdling by mice, or crown-kill due to low winter temperatures.

Spread of X-Disease

In Michigan, X-diseased chokecherry bushes have always been associated with diseased cherry or peach trees. Disease spread drops sharply when chokecherries are removed from areas near infected orchards, even though infected trees are allowed to remain in the commercial planting.

Extensive research has shown that the disease is transmitted by leafhoppers. The species of leafhoppers involved normally do not live on peach or cultivated cherry foliage, but prefer wild species. Occasionally, they stray or are forced by strong winds into commercial orchards from chokecherry bushes and transmit the disease organism through chance feedings.

Cause

X-disease was believed to be caused by a virus until structures resembling mycoplasma were discovered in association with diseased plants. Mycoplasma are living organisms and were first discovered to cause pleuro-pneumonia-like diseases in animals. Discovery of mycoplasma associated with "yellows" diseases of plants is more recent. The organisms produce spherical to ellipsoid-shaped bodies that are smaller than bacteria, but larger than most virus particles. Although this new information does not alter control procedures for X-disease, hope now exists that chemicals will be found for suppressing or controlling the disease.

Control

Eradication of chokecherry bushes within a 500-foot radius of stone fruit orchards has provided commercial control of X-disease. This chokecherry-free zone is best established before the orchard is planted. If the disease should develop in the orchard, or if new chokecherry bushes are discovered during periodic search of the orchard area, this wild species should be destroyed promptly. An orchard can be ruined in three to four years if the source of mycoplasma remains.

Chokecherry bushes are commonly found in hedgerows, along property lines, in woods, and on other vacant land. Brush killers offer the cheapest and most effective way to kill the bushes. Both summer and fall spray treatments are available. Removal can also be accomplished by bulldozing, deep plowing, burning, or pulling out individual bushes. Once land has been cleared, it should be cultivated regularly to kill sprouts developing from roots left in the soil.

During the growing season following treatment, check the treated area carefully for chokecherry

Figure 46—X-disease on peach. Note one branch with symptoms, another which is apparently healthy.

sprouts. Sprouts, or new chokecherry seedlings should be treated with herbicide sprays or otherwise removed during summer.

Identification of Chokecherries

Chokecherry is often confused with wild black cherry and wild pin or fire cherry which also grow in Michigan. It is important to be able to distinguish this species from others, because only chokecherry is important in the spread of X-disease.

Unlike black cherry and pin cherry which grow like trees to 50 feet or more, chokecherries are shrubs up to 15 feet tall. They are usually found in clumps. Their fruit, which is produced along a central stem, is black when mature, and ripens before those of black cherry. The calyx cup does not persist as it does on black cherry (Figure 47). Pin cherry fruits are borne in clusters like tart cherries and in no way resemble those of chokecherry. Chokecherry leaves are wider and broader than black cherry or pin cherry leaves. Serrations along the margin of the leaves are more prominent and spreading than those of the other two species.

STEM PITTING OF STONE FRUITS

In the summer of 1967, growers and research workers in mid-Atlantic states noted that peach trees were mysteriously dying. A new disorder, "stem pitting", was found to be responsible for the girdling, decline and death of these trees. Surveys in the Midwest show the disease is present in scattered locations.

Although the major problem has been with peach, apricot, and nectarine, stem pitting affects other *Prunus* spp. as well. These include: European plum, Japanese plum, myrobalan plum, Chinese wild peach, Duke cherry, *P. mahaleb*, and sour cherry.

Symptoms

Foliar symptoms develop in late summer after harvest, but before the time of normal defoliation. Leaves on affected trees cup upward, become yellowish, later turn reddish-to-purplish and fall to the ground prematurely. Trees affected for a year or so develop leaf symptoms on one or two branches only—later the entire tree shows symptoms.

Severely affected trees are stunted and weak. Their root system is poorly developed and when trees are pulled out of the ground, socket-like depressions remain where roots have broken away. These trees may break off slightly below ground level from high winds, exposing the disorganized wood in cross section (Figure 48).

When the bark is removed from the wood below ground level, elongated pits or indentation and enations are evident (Figure 49). Severity of pitting depends somewhat on the variety or clone of the rootstock and stage of disease development. Closer examination of the crown area shows that the bark is two to four inches thicker than normal.

Cause

It is believed that stem pitting is caused by a virus but direct evidence is lacking. Graft incompatibility is unlikely, since unbudded seedlings also become infected. Furthermore, trunk enlargement and breakage are not associated specifically with the graft union. Bacteria and fungi have not been isolated or found associated with affected trees.

The apparent spread of the disease within individual orchards suggests involvement of a pathogen. Soil-borne transmission is suspected but direct evidence for this is lacking. It is known, however, that spread occurs through nursery stock.

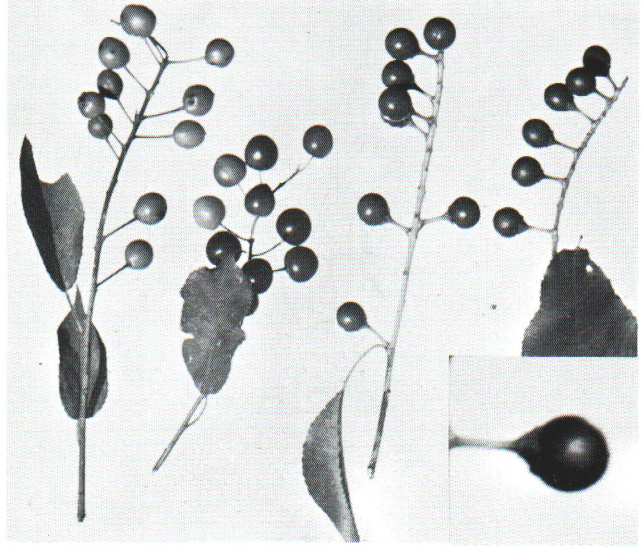


Figure 47—Chokecherry fruit (right) lacks calyx cup while calyx cup persists on black cherry fruit (left). Calyx cup is shown in insert.

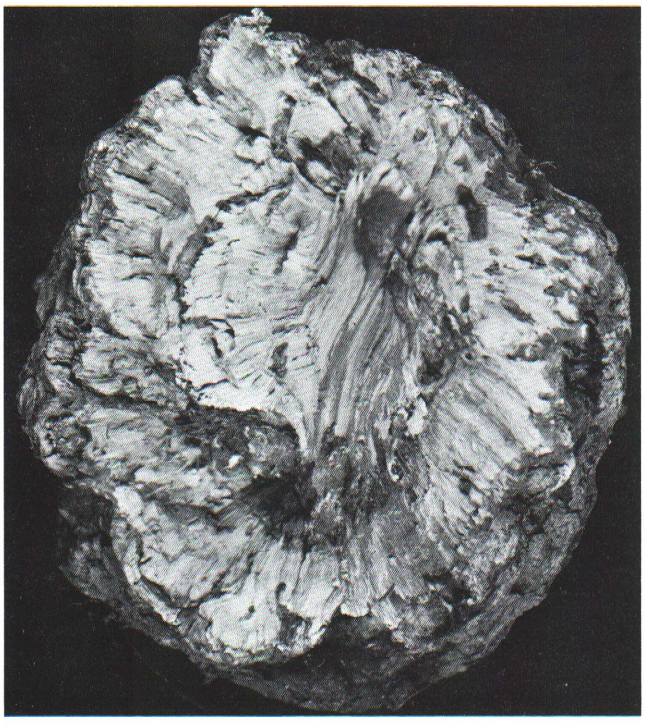


Figure 48—Seven-year-old Redskin tree with highly disorganized woody tissue throughout the trunk from stem pitting disease. Tree broken off at enlarged area of affected trunk (Courtesy S. M. Mircetich, USDA, Beltsville).

Figure 49—Trunk symptoms of stem pitting on peach. Note thickened bark and wood pitting extending to ground level (Courtesy S. M. Mercetich, USDA, Beltsville).

Control

Providing it has an efficient vector for spread, the lethal nature of this disease, its wide host range and extensive distribution gives it the potential for causing great losses. Every effort should be made to exclude this problem by avoiding importation of infected material. If the disease is identified, further spread may be reduced by removing affected trees.

NECROTIC RING SPOT

Before the development of virus indexing programs, necrotic ring spot virus could be found in nearly all orchard and nursery cherry trees. It was also present in peach and plum to a lesser extent. The disease is observed in the orchard primarily on sweet and sour cherry trees. On sweet cherry, the disease is known as "tatter" leaf. In years when symptoms of necrotic ring spot are acute, yields are reduced markedly. Losses for individual trees range from 25 to 50 percent. Severe strains of the virus may reduce yields over 50 percent.

Symptoms

Symptoms of necrotic ring spot are usually present on only a few branches of a given tree the first year infection occurs. The next year, other branches show symptoms but the areas infected initially may not. In the years symptoms occur, the disease is in the acute or "shock" stage. This is when greatest yield reductions occur. Later, the trees appear normal unless they develop sour cherry yellows.

Individual infected branches may be detected in early spring by delayed foliation and blooming. Leaves on affected branches are small and often have depressed fine lines (etching) and partial-to-complete rings on the upper surface. Leaf symptoms are most

Figure 51—Severe yellowing and defoliation of tart cherry due to sour cherry yellows.

Figure 50—Shock symptoms of nectrotic ring spot on tart cherry. Note dieback of terminal shoots.

pronounced during the two-week period following petal fall. Areas of the leaves may become necrotic and fall out, giving the leaves a tattered appearance. Leaves unfolding later often do not show symptoms. Green fruit sometimes show similar arcs and rings.

Terminal and lateral shoots of affected branches are very irregular in length, often stunted or with dieback of the growing points (Figure 50). Occasionally, large areas of bark are killed and show gumming. Severely affected limbs are also more susceptible to low temperature damage in winter.

Disease Cycle

As a result of nursery certification programs, new trees are virtually free of the disease. Virus movement into a young planting occurs primarily through infected pollen. Thus, trees remain free of ring spot until they begin to bloom heavily. The rate of spread and build-up in a new planting is related to proximity to older, infected trees.

Once transmission occurs, a few weeks to two years are required before symptoms develop. Severity of the symptoms depends upon temperature. Leaf symptoms develop readily between 68° and 75°F, but may occur between 50° and 82°F. Die-back of shoots and spurs is more severe at higher temperatures.

Control

Necrotic ring spot may be reduced through the same procedures described for sour cherry yellows.

SOUR CHERRY YELLOWS

Cherry yellows is currently the most serious disease of Montmorency sour cherry in Michigan. It affects trees in such a subtle way that damage is often overlooked until the orchard has become worthless. Initial losses from yellows are not spectacular as with ring spot virus, but increase with time. Yields of sour cherry trees infected while young are reduced by 40 to 50 percent. The longer a new planting can be kept virus free, the closer it will come to reaching its maximum yield potential.

Symptoms

Cherry yellows derives its name from the characteristic yellowing of the leaves (Figure 51) followed by heavy leaf drop. Some leaves drop without first turning yellow but most show different stages of yellow-green mottling. Defoliation generally occurs in waves beginning three to four weeks after petal fall. Severity of each wave depends upon the temperature 30 days prior to the period of defoliation. Low temperatures increase later symptom development.

Yellows-infected trees develop an excess of flower buds on terminals and lateral shoots. In a few years, the bearing surface is reduced since no lateral vegetative buds remain to produce new fruit spurs. The tree exhibits a willowy-type growth with long, bare spaces lacking fruit or spurs.

Flower buds on yellows-infected trees are weaker and abnormally susceptible to low temperature damage. Bloom is often staggered, leading to uneven fruit ripening. Fruit produced on yellows trees are larger than normal, and because of their size, may be objectionable for processing. Sweet cherry trees are also known to contain yellows virus, but no symptoms are produced.

Disease Cycle

Transmission of this disease in the orchard is similar to necrotic ring spot, with one major exception. Before a tree exhibits yellows symptoms, it must be infected with necrotic ring spot virus. The disease is caused by a complex of two viruses—possibly more.

Spread of the cherry yellows virus complex follows two to three years after the spread of necrotic ring spot. Spread is more rapid where high numbers of infected trees are present.

Control

Sour cherry yellows eventually moves into a disease-free orchard unless removed prematurely for other reasons. Care in planning a new orchard, to reduce the movement of ring spot and yellows into the planting, will pay dividends through increased yields and extended orchard life.

The following program has been shown to retard the movement of ring spot and yellows into cherry plantings. Failure to follow any one practice will defeat the objectives of the program.

- Purchase virus-free trees only. Modern nurseries obtain seeds for rootstocks from indexed trees and use virus-free buds to produce trees as free as possible from known viruses. Most states have inspection programs and certify those trees produced from virus-free buds and rootstocks. Trees not certified for freedom from viruses should be refused.
- Isolate plantings. New orchards should be planted at least 100 feet (preferably 500 feet) from existing cherry blocks. Spread of ring spot and yellows is rapid when older, infected trees are nearby. One way to isolate blocks is to plant apple or pear between cherry blocks.
- Plant solid blocks. Replanting missing trees is only profitable for the first five years. Thereafter, avoid replanting. When the orchard becomes unprofitable, remove the block completely.

GREEN RING MOTTLE

Green ring mottle is a virus disease commonly found in older sour cherry orchards which were established before virus-indexed trees were available. Incidence of the disease is high in many of these blocks. It has been detected in latent form in peach, apricot, and sweet cherry in Michigan.

Symptoms and Disease Cycle

Infected sour cherry trees develop yellow leaves similar to sour cherry yellows except for the presence of prominent dark green blotches or rings (Figure 52). Leaf symptoms often occur in waves extending from late June to mid-July, sometimes later. Defoliation is generally not as severe from green ring mottle as from cherry yellows. Both diseases may be present in the same tree and show symptoms either concurrently or separately. A smaller number of leaves may show yellowing along a lateral vein and exhibit slight leaf distortion at the tip of the vein. This is called "constricting chlorosis" and is often present before the yellow-leaf symptom appears.

Fruit on Montmorency trees with green ring mottle are indented with streaks of dead tissue extending to the pit. The fruit from these trees are often worthless.

Very little is known about how the virus spreads other than through infected propagating materials. In orchard plantings, the disease spreads slowly and is not related to the spread of ring spot and yellows. Weed hosts are known but their role in spread is not known. The virus is not transmitted by seed.

Control

Control of green ring mottle virus of stone fruits is based exclusively on starting with virus-free nursery stock and elimination of any tree with disease symptoms.

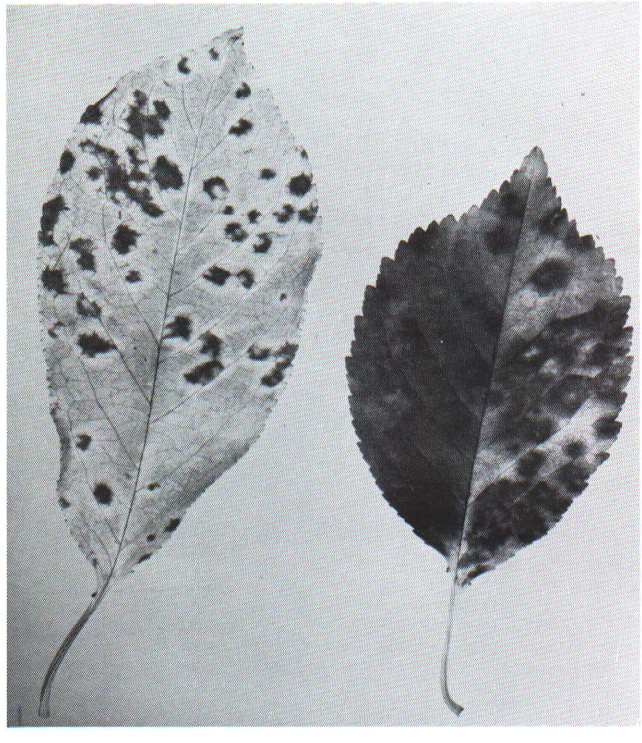


Figure 52—Typical symptoms of green ring mottle virus are dark colored arcs and rings on yellow background.

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