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Small Fruit Diseases in Michigan

COOPERATIVE EXTENSION SERVICE

Michigan State University East Lansing

CONTENTS

SMALL FRUIT DISEASES
In Michigan

*By Robert H. Fulton**

Small fruit growers in Michigan suffer varying degrees of financial loss from certain diseases. In any given year the severity of the loss depends on climatic conditions. Such losses are incurred by reduced yield and quality of fruit and increased susceptibility to winter injury.

The purpose of this bulletin is to aid fruit growers to recognize the different bacterial and fungal small fruit diseases in all stages, to outline the disease cycles, and to point out critical periods of these disease cycles when control practices should be carried out.

The diseases common to Michigan are treated under the following headings: importance, symptoms, disease cycle including conditions favoring the causal organism and control. Specific chemical controls are not included because of yearly revisions. The grower should consult the Extension Fruit Spraying Calendar, Bulletin 154, for current chemicals controls.

Wherever possible, photographs illustrate the symptoms. Diagrams of the disease cycles of the major small fruit diseases show the different stages the fungus passes through in causing a disease.

Visible virus diseases and virus-like disorders are described for blueberry and strawberry. To date, there is no research data to indicate virus in grape. For raspberry, the grower should refer to Extension Folder F-275, "How to Recognize and Control Raspberry Viruses".

I. BLUEBERRY DISEASES

CANE GALL *(Agrobacterium tumefaciens)*

Galls on blueberry branches are observed frequently in fields and propagation frames. These aerial galls may kill the affected branches, reduce yield and increase susceptibility of affected parts to winter

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injury. Furthermore, infected rooted cuttings in propagating frames cannot be certified for sale and should not be used for planting.

Symptoms

During the summer, small dark slits appear on the stems or twigs from which light brown nodules emerge. They may form as single pea-size galls, elongated ridges of gall tissue, or may completely encircle the stem as several large spreading galls. With age the galls become dark brown or black and are rough and hard in texture (Fig. 1).

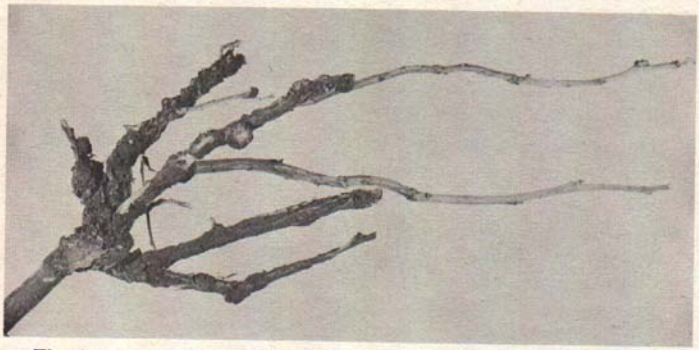


Fig. 1. Severe cane gall infection on a young blueberry plant. Note the elongated ridges of gall tissue.

Disease Cycle

The bacterial organism remains active within the plant from season to season. This is evidenced by the formation of new galls above or below old gall tissues that were formed the previous season.

However, gall develops and spreads slowly on an affected stem. Several years may elapse before these infected plant parts succumb to the bacteria.

Control

Studies on this disease indicate that infection occurs only through plant wounds. This suggests that cane gall in commercial blueberry fields and propagating frames spreads when the bushes are pruned or soft wood cuttings are made.

Prevention of this bacterial disorder is the best control. The nurserymen should obtain his cuttings from bushes completely free of the

disease. The planting medium in the propagating frame should be steam-sterilized previous to rooting.

Severely infected bushes in the field should be removed (Fig. 1). Where only slight infection is noted, pruning out well below this infection site should curb the disease. In this case, disinfect the pruning tools *before each cut* by dipping in a solution of mercuric chloride. (Disslove ½ gm. tablet mercuric chloride in a pint of water, using a glass container.)

MUMMY BERRY *(Monilinia Vaccinii-corymbosi)*

This disease is common throughout the cultivated highbush blueberry fields in Michigan. During wet, muggy springs it can be most destructive, for blighting many new shoots and blossom clusters. The greatest loss is incurred with fruit infection, for diseased berries shell off the bush before harvest.

Symptoms

Shoots, Leaves and Blossoms—In the spring, after growth begins, certain tips of succulent new shoots become water-soaked, then wilt, and turn brown. On leaves the infection is similar to that on the stem; this condition progresses up along the mid vein of the leaf, soon

Fig. 2. Blossom cluster infection, note tufts of spores on blighted stems.

encompassing the entire leaf. Diseased blossoms display brown water-soaked areas at the base of their clusters (Fig. 2). These infected plant parts soon turn gray due to the formation of spore masses by the fungus. This occurs rapidly during cool, wet weather.

Fruit—Berry infection takes place on the stigmas of the open flowers. The berry will continue to develop until nearly full size, then growth stops. At this point the berry turns from a normal immature green color to a tan or cream color, begins to shrivel and mummify and shatters easily off the bush. However, infected berries will *not* spread the disease to other healthy developing blueberries in the fruit cluster (Fig. 3).

Life Cycle

The complete life cycle of the mummy berry fungus consists of two phases: (1) Primary, (2) Secondary. Follow Fig. 4 as you read the text.

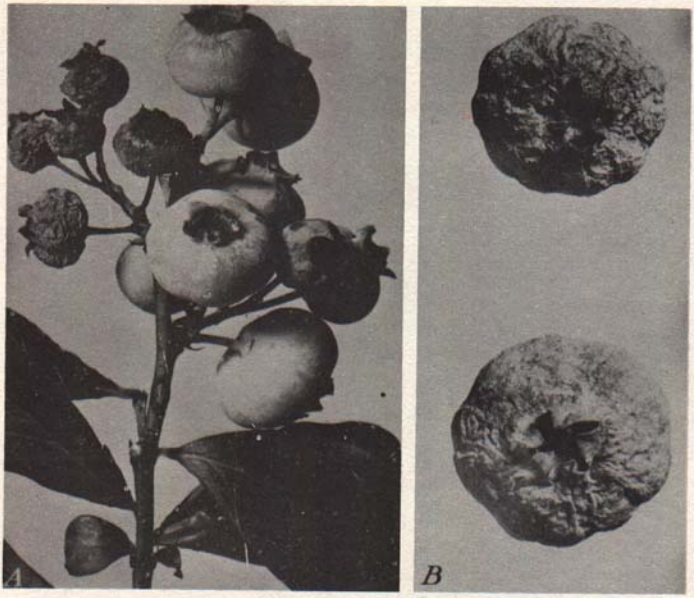


Fig. 3. A. Cluster of blueberries, of which five are affected by mummy berry; B. Infected berries, note shriveled, mummified appearance.

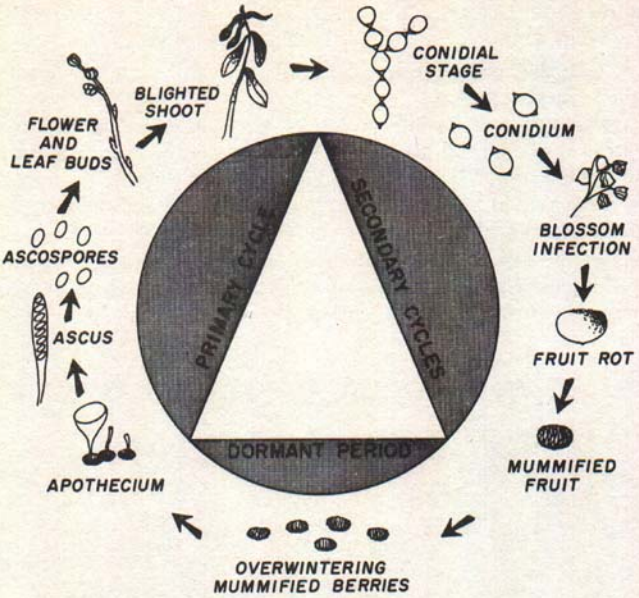


Fig. 4. Life cycle, mummy berry disease.

The primary infection begins during the first weeks in April, as the plants are breaking dormancy, by the formation of apothecia (cup-like mushrooms) on the overwintered, fallen, mummified berries (Fig. 5). These miniature, inconspicuous apothecia are lined with sacs (asci) containing ascospores which are forcibly discharged into the air and wafted up into the bushes by air currents. Some may land on the succulent new growth, and if humid conditions prevail, will germinate, causing the primary infection.

The fungus can be re-established on the bushes only by means of the ascospores, since it does not overwinter as cankers in the previously infected plant parts on the bush. In turn, these blighted plant parts become covered with conidia, summer spores. These conidia cause the secondary infections. They may be dispersed by wind or insects to the pollinating surfaces of blossoms, ultimately causing fruit infection (Fig. 3).

Control

A control program to eradicate the primary inoculum—the apothecia—in the spring prevents the disease from re-establishing itself in the bushes.

Cultivation and hoeing have been tried as means of destroying apothecia. The power-driven hoes that cultivate the areas around and between the bushes have improved and reduced the cost of this practice compared to hand hoeing. However, cultivation is incomplete, because equipment cannot clean out the crowns of the bushes where many of the infected mummified berries become lodged. Therefore, for additional control measures and the use of chemicals to eliminate sources of primary infection, consult the current Fruit Spraying Calendar.

Fig. 5. Apothecia arising from mummified berries infected in previous season.

POWDERY MILDEW *(Microsphaera alni)*

This leaf disease is becoming more prevalent every year in Michigan. It is difficult to evaluate the over-all effect that powdery mildew has on the productivity of affected bushes. However, early defoliation as a result of severe infection reduces the size of fruit and leaf buds.

Symptoms

Mildew is first noted on the lower curfaces of young leaves as an etched, water-soaked spot (Fig. 6). Later, red lines appear outward

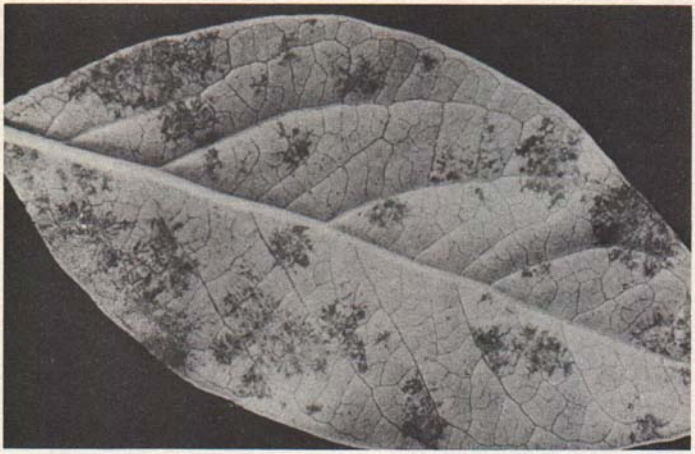


Fig. 6. Blueberry powdery mildew on lower leaf surface. Note the water-soaked (dark) etched circular lesions.

from the center of the lesion, following the hyphal strands of the fungus. The spot enlarges as the season progresses culminating in a yellow leaf.

On those varieties which are attacked only on the lower leaf surfaces, the hyphae may be scarcely visible, but red-yellow lesions occur and may be seen on either leaf surface. On susceptible varieties, the fungus is noted on upper leaf surface by masses of white mycelial growth presenting the powdery appearance. Also infected leaves become bumpy and develop wavy margins due to uneven growth of leaf caused by the infection (Fig. 7).

In late August, small, brown to black bodies can be noted scattered over the fungal growth. These bodies are termed cleistothecia, the overwintering stage of the fungus.

Disease Cycle

In late spring, the overwintered perithecia on fallen leaves rupture, releasing ascospores, which are carried by wind currents to the young, succulent growing leaves. This causes the primary infection of leaf tissues and the re-establishment of the disease on the bush.

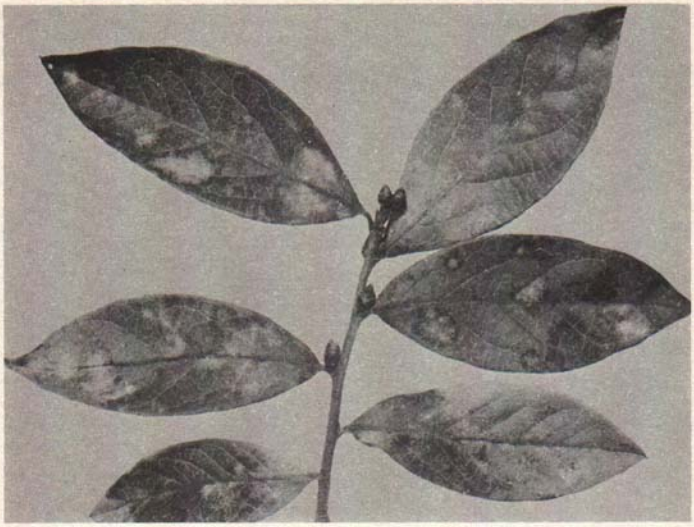


Fig. 7. Blueberry powdery mildew on upper leaf surface. Note the typical white patches of fungal growth. Small, black, round perithecia can be seen, especially on lower right leaf.

After the leaf infections are established, the affected areas become covered with a powdery layer, consisting of the fungal growth and summer spores, the conidia. These spores serve to spread secondary infections throughout the summer to susceptible leaf tissues.

Control

Blueberry varieties vary considerably in their susceptibility to powdery mildew. The varieties, Adams, Concord, Jersey, and Rubel, are susceptible, Rancocas, Stanley and Weymouth are quite resistant. No variety is completely immune. At present, newer varieties are being developed that show a high degree of resistance to powdery mildew; they will replace several of the old susceptible varieties now being grown.

To date, control of powdery mildew by fungicide applications has not been successful under Michigan conditions.

II. BLUEBERRY VIRUS DISEASES

Virus diseases affecting blueberry are transmitted only by insects and grafting. Losses due to the several viruses attacking blueberry are high, since an infected bush never recovers and development of bearing wood and blossom buds decline rapidly. There is further loss in the time in years and cost of culture involved in replanting until a healthy bush is mature and in full production.

STUNT

The first symptoms are noted in the spring as pale yellow tips and margins on the terminal 2 to 5 leaves of young shoots. Soon, the yellowing extends into the leaf areas between the lateral veins; the veins remain green. These leaves become round and small and their margins cup—either up or down (Fig. 8).

Fig. 8. Stunt infection showing round, small, marginal cupped leaves as noted in center of bush.

The leaves on the old canes develop a premature fall reddening. This red coloration lies in two bands inside the leaf margin, parallel to the mid-rib, the other portion of the leaf being yellowish-green.

The new cane growth developed by stunt-affected bushes rarely grows higher than 2 feet. These short canes have a tendency for excessive terminal branching that produces short, stubby side twigs at sharp angles with the stem. Older canes produce many short, thin, fruiting laterals that fail to produce fruit buds.

9. Blueberry mosaic. Note the stippling of yellow on the leaves at the left, the terminal leaves appearing to be normal.

Blueberries borne on affected bushes color later than normal, are very small and bitter, and cling tightly to the stems even though apparently ripe.

The symptoms of stunt are similar for all major blueberry varieties grown in Michigan. However, Rancocas may not show the typical symptoms. Thus, stunt on this variety would be indicated by a compilation of weak basal shoots with leaves showing green on the main vein and partially out on the lateral veins, with the rest of the leaf red during the post harvest period.

MOSAIC

Mosaic, previously termed "variegation" by growers, may be either of virus or genetic origin. However, the symptoms of both are similar and thus treated as one.

When growth is well started, symptoms of this disorder stand out prominently in a planting. The leaves on one or more canes develop brilliant hues of yellow, yellow-green, and red in a variegated fashion.

Generally, the coloration is more intense on the lower leaves, while the terminal leaves may show no symptoms (Fig. 9).

There appears to be no known immediate effect on young or old canes. However, the number of blossom buds developed begins to decline. The fruit produced on Mosaic-affected bushes appear normal.

Mosaic is observed commonly on the varieties, Concord, Dixi, and Stanley.

SHOESTRING

The first signs of this virus are noted as brown to red discolorations banding the mid vein of a leaf, often extending partially up lateral veins. Such affected leaves develop abnormally—they are wavy, distorted, and often crescent in shape (Fig. 10).

Severely affected leaves are narrow, pointed, and light green to red in color. These strap-like leaves are commonly found on new canes arising from the base of affected bushes. However, the leaf symptoms may be found throughout a bush, even on canes that also bear twigs or normal leaves.

Fig. 10. Leaf symptoms of shoestring virus, Burlington variety. Left – severely affected showing narrow, pointed strap-shaped leaves; center – early symptoms of wavy crescent-shaped leaves and recurving shoot growth; right – healthy shoot.

On the varieties, Burlington, Cabot and Jersey, red streaks or bands varying in length from a small dot to several inches occur along the surfaces of new cane and twig growth, especially on those areas most exposed to direct light (Fig. 11). This red coloration may appear as oval patches around buds, scattered over the cane surface, or it may completely encircle this new growth, especially at the base. Finally, this typical red coloration significant of Shoestring infection, can be observed on this new wood until the following spring (Fig. 12).

The cane growth itself is affected and becomes long, and spindly with a tendency to twist or recurve. It fails to produce blossom buds.

Fruit on affected bushes of the Jersey variety, even with only one or two canes showing disease symptoms, develops a premature red to purple cast on the surface of the berry most exposed to light. This coloring covers part or all of the upper half of the berry, in contrast to the green fruit on healthy bushes. This can be seen in early July.

Control of Blueberry Viruses

It is known that the stunt virus may spread from bush to bush in the field by a leafhopper insect. The mode of field infection by the

Fig. 11. Shoestring infection on new shoot growth is typified by streaking (red) up the cane on the Burlington, Cabot and Jersey varieties.

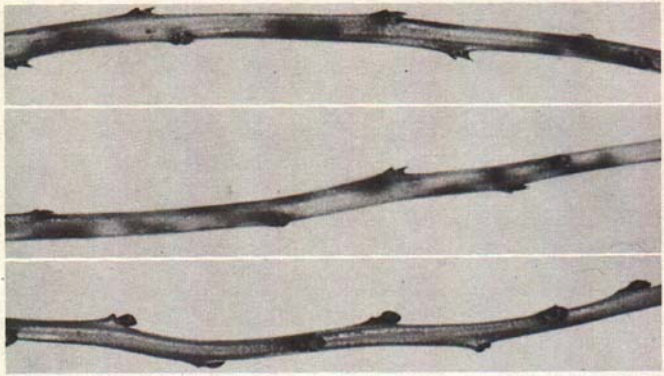


Fig. 12. Dormant symptoms of the shoestring virus. Note patches of discoloration (red) on the 2 one year canes, top and center. Healthy cane, bottom.

mosiac and shoestring viruses is still under investigation. These viruses cannot be transmitted on pruning tools.

In new plantings and resetting established fields, the grower should use only certified, state-inspected nursery stock.

Infected bushes located in the planting should be dug completely and removed from the field. If feasible, the entire root system should be taken with the plant, for any sucker growth that may develop will be virus-infected and serve as a source for further field spread of the disease.

In fields of the Burlington and Jersey varieties, inspections for shoestring can be made also while pruning during late winter or early spring. Due to the absence of leaves the typical red blotches and streaking on the previous season's cane and shoot growth is easily noted. In April, after several warm days, sap flow into this previous years' growth turns the wood an apple green color which makes the red discoloration even more outstanding (Fig. 12). Furthermore, there would be more time in the spring for immediate bush removal, than during the busy growing season.

Finally, early identification of virus-affected blueberry bushes, followed immediately by complete plant removal is essential for effective control.

III. CURRANT AND GOOSEBERRY DISEASES

ANTHRACNOSE *(Pseudopezina ribis)* and LEAF SPOT *(Mycosphaerella ribis)*

Anthracnose is considered the most serious fungus disease attacking these crops. Losses result from reduced fruit size and yields and from premature defoliation. Loss of vigor and defoliation can cause crop losses as great as 80 percent.

Symptoms

The leaves are the most susceptible portion of the plant. In the spring several weeks after bloom, small water soaked areas on the lower leaves will develop into brown patches that are visible on both surfaces of the leaf. The lesions are circular or angular in shape and measure from several millimeters up to ⅛ inch (gooseberry) in diameter (Fig. 13). A close examination of the lesions on the upper surface during periods of high humidity will disclose gray viscous

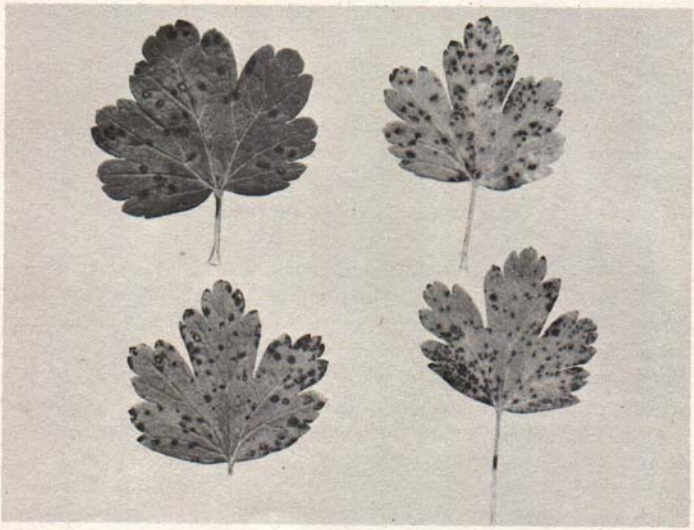


Fig. 13. Anthracnose of gooseberry. Note the numerous small lesions on the leaves. Yellowing apparent on right.

droplets. These are the spore masses (anthracnose). The leaves generally turn yellow, leaving a green margin around the lesion; or a red to purple margin. Defoliation follows and is usually complete by the middle of July. In severe infections, leaves quickly turn brown, wither as if scorched, and then fall.

On young stems, the lesions are elliptical and light brown. Similar symptoms are noted on the leaf and fruit stems. On the fruit the disease is noted only as small black specks, common on currants.

Though the fungus causing anthracnose attacks both crops, another leaf disease sometimes occurs, termed leaf spot, *(Mycosphaerella ribis).* Its symptoms are similar to those of anthracnose except that in the case of leaf spot the lesions are larger and small, black pycnidia are present in the spot (Fig. 14).

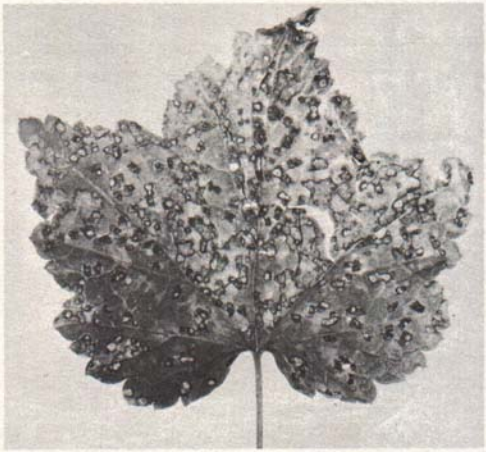


Fig. 14. Leaf spot on currant. Note the small, black pycnidia in the spots.

Disease Cycle

The disease cycle for anthracnose and leaf spot are quite similar. In the spring, ascospores are discharged from cup-like apothecia (anthracnose) or perithecia (leaf spot) from overwintered infected leaves. In the presence of moisture, the spore germinates with subsequent primary infection of the leaf. Primary infections may also be incited

by overwintered conidia. Infections may occur on either leaf surface, but more commonly on the lower surface.

After the leaf lesions are evident, abundant conidia—secondary spores—are ready for dissemination from acervuli (anthracnose) or pycnidia (leaf spot). Thus, spread of conidia by wind and rains results in renewed secondary infections for the remainder of the season.

Control

The fungal incitants of anthracnose and leaf-spot overwinter mainly on fallen leaves. Therefore, cultural practices that remove or destroy these leaves will reduce or eliminate much of the primary inoculum.

Chemical control of these diseases is successful if the first application is made 2 to 3 weeks after bloom, or about the time the first few lower leaves in the center of the bushes show signs of lesions or spots. This is the period of ascospore discharge which initiates the primary infection on the plants. Subsequent sprays, generally in the post-harvest period, will prevent secondary spread of the diseases.

POWDERY MILDEW *(Sphaerotheca mors-uvae)*

Mildew is most damaging to gooseberry. Affected plants are stunted and reduced in vigor. If leaf infection is severe, fruit buds fail to form. The greatest damage is to the fruit in the form of cracking and russeting which makes it unsaleable.

Symptoms

In May or June, superficial white powdery patches may be seen on the lower leaves, tips of young shoots, and berries. These patches may extend to cover large portions of the affected plant parts. The leaves on the tips of young shoots become stunted and distorted, and they soon defoliate. Affected areas on the shoots and berries turn rusty-brown in color as the persistent fungus mycelium ages. About this time, small, black perithecia can be observed on the brown patches on the shoots. The mycelium can be scraped off the berries but the skin is roughened and discolored wherever the fungus growth occurred.

Disease Cycle

The fungus overwinters in the perithecia on the shoots or as mycelial strands on previously infected plant parts. In the spring, just after fruit set, the ascospores are ready to discharge. This occurs during rainy periods and within 10 days primary infection is established.

Conidial production is then underway, spreading the disease further over the developing plant parts. The spread is dependent upon cool, wet periods during spring and early summer.

Control

Cultural practices to provide good air drainage and thus allow quick drying are essential.

Chemical sprays can control powdery mildew if the after-bloom application is made as soon as the fruit has set. Since this is the critical period of primary infection, a one-week delay will decidedly increase the amount of mildew on the plant, especially the fruit.

CURRANT CANE BLIGHT *(Botryosphaeria ribis)*

The fungus kills the canes. Once established on a cane, it may spread to all of the canes of the bush—thus a total crop loss for infected bushes.

Symptoms

During the ripening period of the fruit, canes may suddenly wilt and die. An examination of the wood and pith of affected canes discloses a blackened appearance. On young shoots the terminal portions die-back.

Later in the season, on canes infected the previous year (not removed) and on current season's growth, black, wartlike fruiting bodies, termed stromata, appear on the canes in parallel rows (Fig. 15).

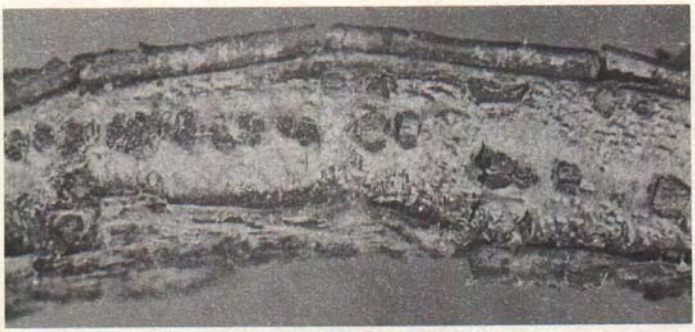


Fig. 15. Currant cane blight. Note the row of wart-like fruiting bodies.

Disease Cycle

The fungus overwinters in diseased canes. In the spring, ascospores are discharged which infect young developing shoots. In late June, pycnidia are evident on these infected sites and spores are spread from them during July and early August to further infect new growth. After infection, the mycelium develops down through the bark wood and pith. Such infection on a lateral shoot often develops to encircle the main cane.

Control

Systematic cutting out and burning of the diseased canes has served to reduce the disease. This pruning would best be done in early May, since the absence of leaves will disclose infected canes better.

IV. GRAPE DISEASES

BLACK ROT *(Guignardia bidwellii)*

Black rot of grape is common throughout Michigan. In at least 1 out of every 3 years, it reaches epidemic proportions. The principal loss is through destruction of the berries. Early infections kill blossom clusters or cause newly developing berries to "shell off" the cluster. Later, when the berries are more than half grown, they can rot in the cluster, reducing yields and distracting from the appearance of the cluster.

Symptoms

The causal fungus can infect all of the new growth of the vine. In late June circular spots appear reddish-brown. They are usually in the interveinal leaf area. Later, when the spots are about $\frac{1}{16}$ inch in diameter, the center becomes brown with the margin appearing as a black line. The identifying characteristic is the presence of small, black pycnidia arranged in a definite ring near the inside margin of the lesion. (Fig. 16).

The lesions on leaf and fruit stems, shoots and cluster stalks are purple to black in color, depressed and elliptical in shape. The small, black pycnidia (pimple-like) are scattered over the surface of these lesions, not definitely arranged as on the leaf. Furthermore, as these plant parts develop, the epidermis tends to split along the axis of the lesion.

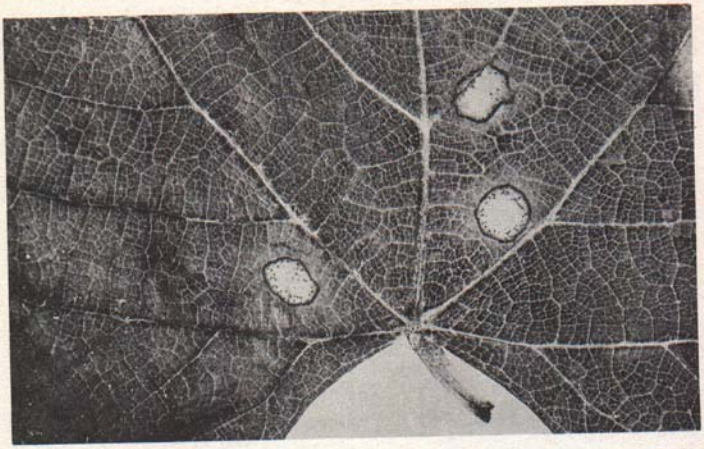


Fig. 16. Black rot infection on a grape leaf. Note the dark margin and the ring of pycnidia.

Lesions on cluster stalks and fruit stems tend to girdle, causing shelling of the lower or individual berries. (Fig. 17).

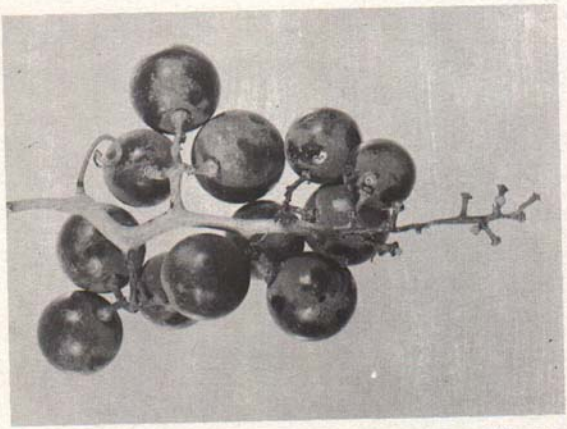


Fig. 17. Effect of Black rot infection on the cluster stalks. Note the right portion where berries have shelled off.

Fig. 18. Stages of development of black rot infection on the grape berry.

The berries become infected when they are about half grown. The first sign is a white spot that soon is encircled by a brown ring. This ring widens rapidly and within 48 hours may encompass half of the berry. Simultaneously the center of the lesion becomes soft and depressed. The color now begins to change to black with the skin becoming more wrinkled. Soon the whole berry is infected. The fruit becomes mummified, its surface studded with small, black pycnidia (Fig. 18). Several or all of the berries in a cluster may become infected (Fig. 19). Generally, after mummification of the fruit occurs, the affected berries "shell off' the cluster.

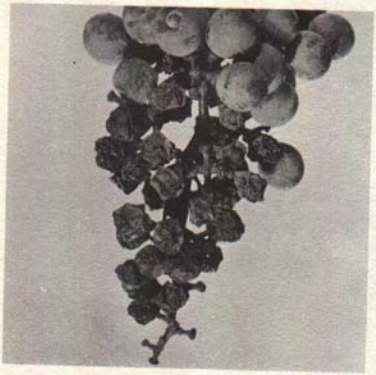


Fig. 19. Black rot infection on the grape cluster.

Disease Cycle

Follow Fig. 20 of the disease cycle as you read the text. The fungus overwinters in the previously infected canes and berries as developing perithecia. Mummified berries partly buried by the soil become soaked during spring rains and easily discharge ascospores during the pre- and post-bloom period of the plant. The ascospores are carried by air currents to the succulent growing plant parts. Within 48 hours they germinate and penetrate the plant tissue directly if 2 or 3 days of humid weather exists. The fungal mycelium then ramifies through the host tissues and is evident about 2 weeks later as visible lesions. Pycnidia develop quickly in these lesions and ooze out spores in great quantities that are carried by rains throughout the vine for subsequent waves of secondary infections.

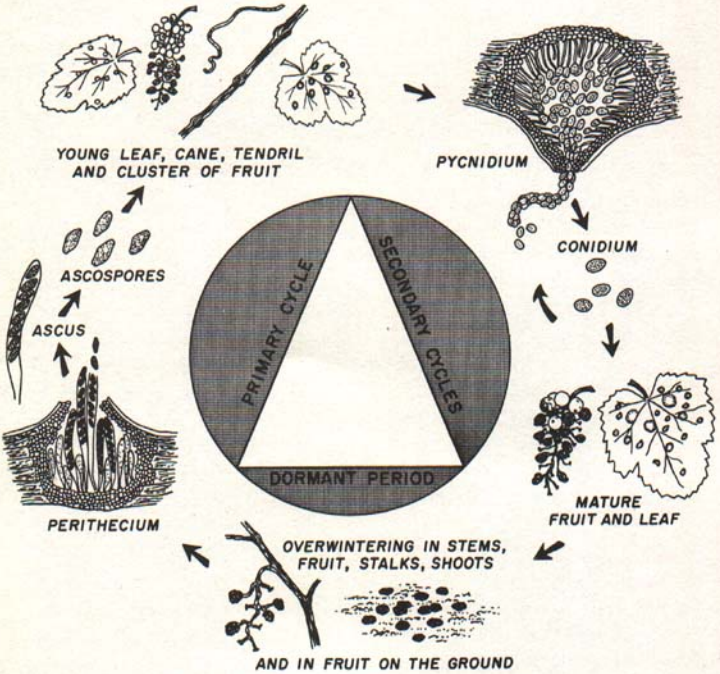


Fig. 20. Life cycle. Black rot of grape.

These infection periods can be traced back to rainy spells about 10 to 14 days after new lesions or rot occurs. In August, the pycnidia cease to produce spores and begin transformation into perithecia, the overwintering and primary infection stage of black rot.

Control

Of the varieties of grapes grown in Michigan, only Champion, Delaware, and Worden are resistant to black rot.

Cultural practices to effect weed control increases air drainage and also permits better spray coverage. Trimming or tying up low hanging vines prevents early disease build up on the vine. Finally, grape hoeing to throw soil up under the vines, early in the season, will cover fallen infected berries. The mummified fruit will not produce ascospores if covered by soil.

Black rot can be controlled successfully by chemical sprays. However, spraying must be timed with plant development and before predicted rains.

POWDERY MILDEW *(Uncinula necator)*

Within the past five years, this fungus disease has attained economic importance in Michigan vineyards because of the rise of organic fungicides. It reduces the food manufacturing of leaves and delays ripening of fruit. In severe cases the berries fail to ripen or "shell off" the vine.

Symptoms

In early summer, indistinct white powdery patches appear on the upper leaf surfaces. The lesions may enlarge so that most of leaf surface presents a gray dusty coating. Later in the season, numerous small, black perithecia appear on the mildew infected lesions. (Fig. 21).

On fruiting stems, the disease also presents a white powdery growth that often encircles and tends to girdle these plant parts (Fig. 22).

Control

Cultural practices to increase air circulation and to reduce shading aid in initially reducing mildew. Weeds common in Michigan vineyards are attacked by a powdery mildew, but it is *not* the same species that attacks the grape.

The powdery mildew fungus is superficial on the plant in that it

Fig. 21. Perithecia of the powdery mildew fungus evident on grape leaves late in the season.

sends root-like structures into the host tissue to gain its nourishment. Therefore, sprays to control mildew should thoroughly wet the mycelial mats.

Since the disease has been noted early in the growing season, two sprays of copper, one immediately after bloom, the other 10 to 14 days later, should reduce the disease. This may have to be done several seasons. Furthermore, the inorganic copper will also control the black rot and downy mildew diseases.

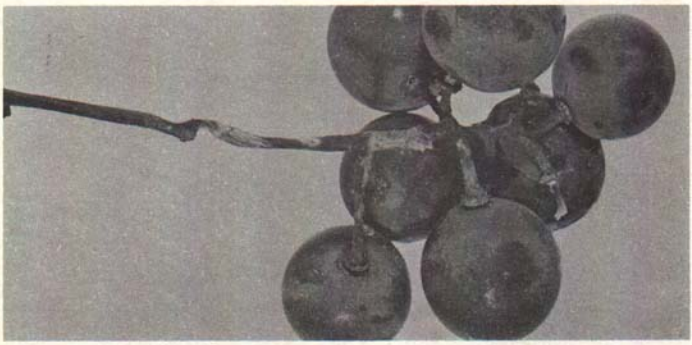


Fig. 22. Powdery mildew evident on a grape stem. Note the girdling effect.

DEAD ARM *(Cryptosporella viticola)*

The principal losses as a result of dead arm disease are loss of bearing vines, reduction in yield of diseased vines, and the loss in yield which occurs until replacement vines come into full bearing.

The effect of the disease on yield does not seem to be appreciable until leaf symptoms have developed. In yield experiments with the dead arm infected Concord vines, a yield reduction of nearly 30% was recorded.

Symptoms

The most easily recognized symptom is the dead arm on the vine, or the vines themselves may be dead practically to the ground, with suckers growing up from the base. This is observed in the spring when the vine either fails to put out shoots or the shoots die back after a few weeks. Badly lesioned vines are weakened and usually die during the cold, dormant period. In June or early July the young branches or shoots from the diseased arm are stunted and have shortened internodes which become progressively shortened and stunted from year to year. The primary leaves are about half the size of healthy ones and are often misshapen, cupped along the margins, and markedly *chlorotic* except along the veins (Fig. 23). Occasionally browning between the veins results in a shredding of the leaf.

In July, secondary branches develop from axillary buds and bear leaves which are normal in color and may be only slightly stunted and

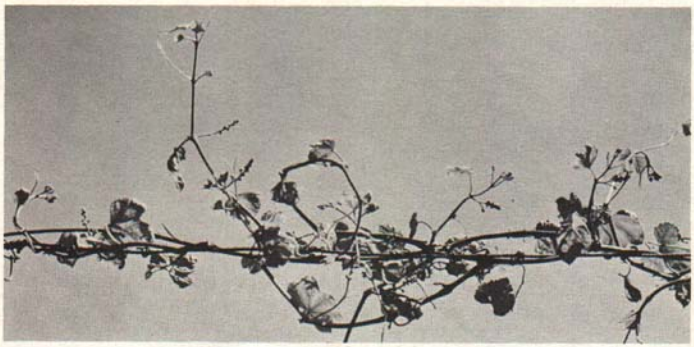


Fig. 23. Symptoms of dead arm on the new growth. Note the small, cupped, distorted leaves.

curled. By late July or August most of the primary leaves have dropped from the vines. Those which still persist retain their chlorotic character and may even show more necrosis and shredding than they did in June. Thus, the early symptoms are masked by the development of secondary growth.

On the new cane growth, small purple to black sunken lesions may be seen on the first 3 or 4 internodes. These lesions may also occur on the leaf stems and veins. On the older canes and trunk of the vine necrotic lesions can be noted under the bark. Sections through these plant parts will disclose V-shaped segments of the discoloration extending into the pith (Fig. 24).

Fig. 24. Section through a grape cane showing the typical, infected, discolored lesions of dead arm.

This lesion stage is a fairly advanced stage of the disease and may have existed for at least 3 years before any leaf symptoms appeared. The lesions may be quite far removed from the arms showing symptoms. They may be quite extensive, extending up and down the stem for a distance of 3 feet or more and may completely encircle the stem. Since the old dead bark must be removed before the lesions can be observed, they are often overlooked. Lesions may show a conspicuous zonation due to successive annual attempts to overgrow the necrotic area.

Final confirmation of the disease on these cane and trunk lesions is the presence of numerous small, black pycnidia on the surface under the bark (Fig. 25).

Dead arm may occasionally be observed on the berries. It is recognized by a black *flattened* lesion with only a few pycnidia present.

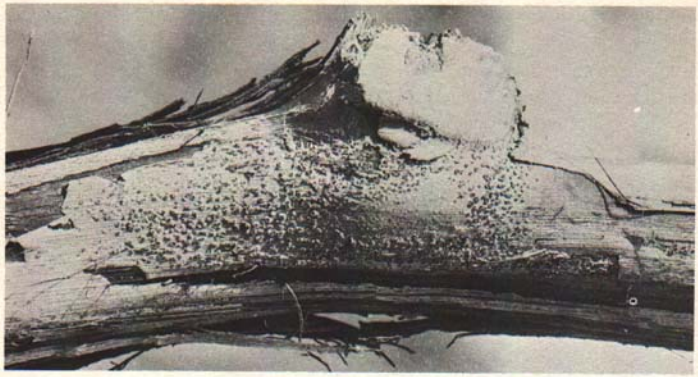


Fig. 25. Pycnidia of the dead arm fungus on an infected grape trunk.

Disease Cycle

Dead arm has been assumed to overwinter in the stem cankers both in the mycelial and the pycnidial stage. Infected berries will overwinter the pycnidial stage of the fungus, but this has not been found to influence the spread of the disease significantly.

The pycnidia mature shortly after the bursting of the buds in spring (May, June). Rains or heavy dew cause the spores to exude in reddish-yellow tendrils containing several thousand spores. Spattering drops of rain spread this inoculum to young shoots below or along the infected arm.

A period of continous mist or fog is necessary for spore germination on the host. Generally, only a single period of infection occurs early in the growing season as evidenced by the fact that the lesions appear almost entirely at the base of the shoots, usually between the first 3 internodes. The susceptible parts of the vine are the tender, more succulent growth, while older tissues are resistant. However, infection may occur through pruning wounds and stubs on the trunk or heavier arms.

After infection, the fungus invades nearly all the stem tissues and causes a wedge shaped necrotic area extending to the center of the wood. Later young pycnidia form immediately beneath the layer of cork of the outer bark. As it enlarges, it lifts through the cork layer and the larger portion of the pycnidium appears on the outside.

Control

Dead arm can be controlled by two practices, sanitation and spraying.

Sanitation: Early in the season the vineyards should be inspected for the characteristic dead arm symptoms. Each infected vine should be marked for special pruning. If feasible, note should be made indicating to what extent the arms are infected, so that in pruning, it may be possible to save healthy bearing wood. However, when dead arm is noted in the lower trunk area and all four arms show symptoms, immediate pruning is suggested. This not only removes inoculum but permits a renewal stem to be started.

Partial pruning during this early growth period may be dangerous as this is the period when the fungus is active and fresh wounds should be avoided.

During the normal pruning period, it may be necessary to strip away many layers of old bark in order to prove the presence of dead arm, for marked vines may show similar growth to neighboring healthy vines. Normal appearance of a marked vine is no criterion for neglect to properly prune out diseased portions.

Prunings should be made as close as possible to supporting wood to prevent prominent pruning stubs. All dead arm prunings should be destroyed, for the fungus can live and reinfect from old prunings up to 14 months after removal.

Finally, dead arm has been spread in vineyards by contaminated saws. Therefore, in the course of pruning out dead arm wood, it is advisable to disinfect pruning tools.

Spraying: Since the fungus also overwinters in the previously infected new shoots, these shoot lesions serve as a great source of inoculum in the spring. Spraying to eliminate this source in order to control new shoot infections is possible. Generally, a single spray at the 2 to 3 leaf stage of plant growth should greatly reduce infection of the basal internodes of the shoots. However, this spray should be applied thoroughly for best effectiveness and *before* a predicted rain period.

DOWNY MILDEW *(Plasmopara viticola)*

This fungus disease will cause more loss by its effect on the vine than from rotting of the fruit. Early infection may kill out entire fruit clusters. Later, infections cause defoliation and killing of leaf tissues, resulting in a failure of the berries to ripen. Attacks stunt

young shoots so badly that there is a decided reduction in bearing wood for the next season.

Symptoms

On the upper surface of the leaves, small, pale yellow irregular lesions appear. The spot may appear translucent in the early stages of development. Directly opposite the yellow lesion on the under surface the white downy masses of the fungus can be noted.

Soon the leaf tissues becomes brown with the undersurface fungal masses a dark gray. The lesions may be numerous on a leaf and can coalesce, resulting in defoliation. On varieties such as Delaware and Niagara, the lesions are irregular while on Concord they are delimited by the leaf vines and are yellow to orange.

Infection on leaf stems, young shoots, and fruit stalks causes severe stunting, distortion, and extreme thickening (hypertrophy) of the tissues (Fig. 26). Soon these plant parts may be covered with the white downy fruiting structures of the fungus. Later, the tissues turn brown and die.

Fruit infection symptoms vary depending upon the stage of maturity. Berries infected up to the half-grown stage fail to develop and are soon covered with the downy, white fungal growth (Fig. 26). If infection is late in the season, the berry becomes leathery and slightly wrinkled, and develops a reddish-marbling coloration. The berry stem dies.

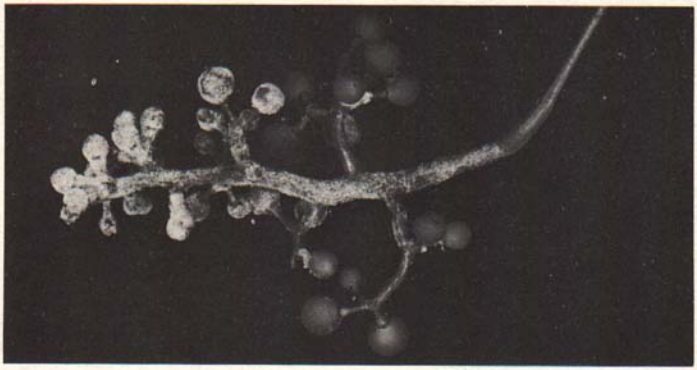


Fig. 26. Downy mildew infection on a fruit cluster of grape. Note the thickening of the stem and white downy fungal growth.

Disease Cycle

The causal fungus overwinters in dead fallen grape leaves as *oöspores.* During the winter and early spring these leaves disintegrate, thus freeing the oöspores. If these fruiting bodies of the fungus have been subjected to long periods of low temperatures, they will germinate and give rise to conidium which release many swarm spores (motile) during wet periods. They are splashed onto leaves near the ground and primary infection takes place through stomata (leaf air pores) generally on the lower leaf surface. Mycelia then develop through the host tissue, soon giving rise to conidiophores (spore stalks) which push through the leaf stomata. This is the downy, white growth noted visually on infected plant parts. The conidia borne on conidiophores are released and carried to other plant parts where they germinate to produce many swarm spores. The swarm spores after moving about for a short period, come to rest, germinate and infect through stomata on the plant tissues.

At the end of the season, oöspores are developed in the old infected leaves.

Control

Since the primary infection source is in the overwintered leaves, removal or early covering with soil is an excellent sanitation practice.

Chemical sprays to control downy mildew are successful. Since the Concord variety is quite resistant, only those growing Champion, Delaware, Niagara or Worden should be faced with this disease. The suggested times of application and chemicals are given in the Fruit Spraying Calendar, Extension Bulletin No. 154.

V-a. BRAMBLE DISEASES
Bacterial

CROWN AND ROOT GALL *(Agrobacterium tumefaciens)*

Crown gall is considered one of the limiting factors in bramble production, especially on light sandy soils. Severe infections may stunt all or part of a plant, reduce yields, or prevent new cane development so that such affected plants die after harvest. Its presence on planting stock results in condemnation, a great economic loss to nurserymen.

Symptoms

The first sign is a slight swelling of the affected crown or root tissue followed by rapid development of white, soft, irregular round-shaped galls that may become several inches in diameter. As the galls approach full size they turn dark brown, are woody in texture, later decaying (Fig. 27).

Black raspberry roots are seldom injured by this disease. However, galls may form abundantly in the crown region where buds for new canes are produced. This limits the number of new canes produced or it may kill all of the buds.

Gall on red raspberry is commonly found on the roots. Severe infections produce weak, stunted new cane growth.

Purple varieties may display root galls, but the crown is the region of greatest occurrence of gall, resulting in damage similar to that of black raspberry (Fig. 28).

Fig. 27. Crown and Root gall affecting red raspberry.

Fig. 28. Crown gall on purple raspberry, illustrating sparsity of new cane development.

Disease Cycle

Under suitable soil mosture conditions, crown gall bacteria are given off continuously from the surfaces of the living galls into the surrounding soil area where they remain active for several years. Infection on the underground plant parts of brambles occurs throughout the season but only through wounds. Within 10 days to 3 weeks after initial infection, gall formation is noted.

Control

Planting sites for new bramble fields known to have a history of crown gall should be rotated to grain crops several years prior to planting. The grower should only use certified stock. Prior to planting, this nursery stock should be checked for any gall formation that could have been in an undeveloped stage of infection at the time of state inspection.

CANE GALL *(Agrobacterium rubi)*

This disease is sporadic in its appearance in commercial black and purple raspberry plantings. It causes the fruiting canes to split open, dry out—resulting in death to the fruiting cane or production of small, seedy berries.

Symptoms

In late May and early June, small, round, white projections may be seen on the fruiting canes. These form on the main stem, laterals, and around the base of the fruiting shoots. These warty excrescences increase in size, forming elongated ridges extending for short distances along affected canes, often encircling the cane. Canes generally crack open along the line of the elongated ridges. Later, the gall tissue turns brown to black in color and decomposes rapidly (Fig. 29).

Disease Cycle

The mode of overwintering of this disease is still to be understood. However, the facts that (1) galls are not found on new cane growth but do appear early in the season on fruiting canes, (2) the bacterium form pockets within these canes, (3) the survival period in the soil is only a few months all point to overwintering of the bacterium in the aerial portions of affected plants.

Gall formation in the spring is responsible for disease spread during the season. Bacteria cells are readily formed on the surface of these

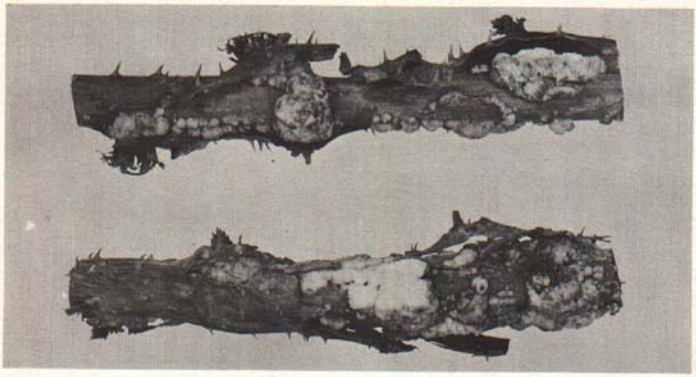


Fig. 29. Black raspberry cane gall; note ridges of young warty gall tissue.

galls and washed or splashed, during rain periods, to other plant parts. However, infection on these new sites will occur *only* through wounds as produced by insects, spines or during cultivation.

Control

There is no practical way, at present, for controlling cane gall formation after the plants are started in the field. Therefore, plants showing this disorder should *not* be tipped for propagation. Only vigorous black and purple raspberries should be used so as to assure the grower of cane gall-free planting stock.

V-b. BRAMBLE DISEASES
Fungal

ANTHRACNOSE *(Elsinoe veneta)*

Most growers consider anthracnose the most serious fungus disease of raspberry, dewberry, and blackberry. Anthracnose reduces yields, makes affected plants more susceptible to winter injury, and shortens the life of the planting.

Symptoms

Canes—In the spring, when the new canes (shoots) are about 6 inches high, small reddish-brown spots appear. As the canes grow, these lesions enlarge, become round, turn white to gray in color, and develop a slightly sunken center. These spots sometimes are so numerous that individual ones grow together and cover large portions of the cane (Fig. 30).

As the canes develop in size, the severely infected portions split open. This has a girdling effect on the cane and reduces the flow of sap and food materials to the maturing fruit. Severely infected canes also may dry out and die during the winter or break off during the next fruiting year.

Leaves—The first symptoms generally appear on the leaves in late June as small, irregular, yellow spots. These spots soon turn red, enlarge somewhat, and become light colored with a red margin. Quite often, the leaf tissue in these diseased spots drop out, giving the leaf a "shothole" effect (Fig. 31). Infections may also occur on the leaf veins and stems. These are similar in appearance to those noted on canes but are more elongated.

Severe leaf infections may cause the plant to lose part of its leaves. This can delay the normal ripening period of the fruit.

Fruit parts—The most damage caused recently by anthracnose in Michigan has been on the fruiting stems and spurs. The symptoms are similar to those on the canes and cause the fruiting stems to be lopsided in their growth, and quite brittle. Fruit borne on these stems is small and may dry up before it ripens. Furthermore, at picking time, the entire cluster with immature berries may come off when the ripe berries are picked. Direct fungus attack on the ripening berries causes cessation of individual drupelet growth presenting a flat-sided berry. Also, these areas do not develop normal maturing coloration, but are a greenish-grey.

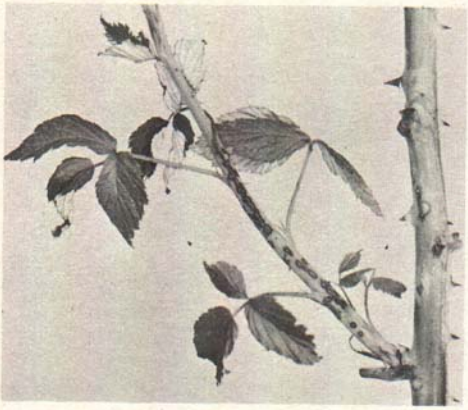


Fig. 30. Raspberry fruiting lateral showing different stages of anthracnose infection.

Disease Cycle

(Follow Figure 32 as you read the text.)

Primary—The fungus overwinters in the lesions formed on the new canes the previous season. In the spring during wet periods, ascospores and conidia formed in the lesion are discharged or washed onto surfaces of newly growing plant parts and become firmly attached. In the presence of moisture these spores germinate and directly infect the exposed, susceptible plant part.

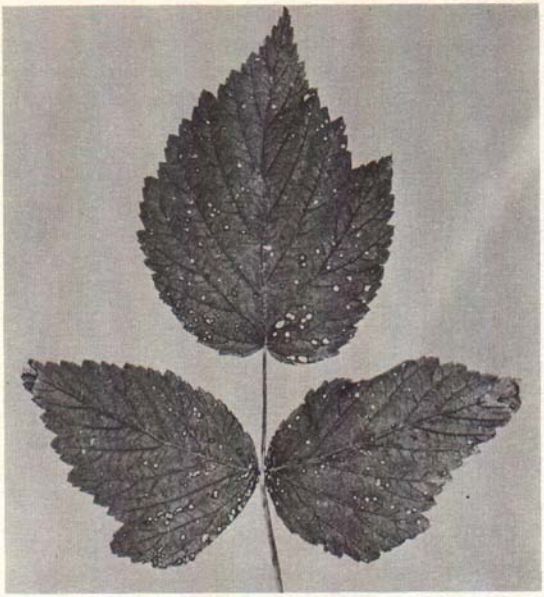


Fig. 31. Raspberry leaf showing anthracnose lesions with dark margins and the "shothole" symptom.

Secondary—The fungus penetrates and develops between the cuticle and epidermal cells. Later this infected area becomes sunken, its surface covered with fungal growth that produces great numbers of infective conidia throughout the growing season.

Infection of bramble plant parts is confined only to young, succulent growth during wet weather. Therefore, anthracnose lesions may be noted in groups along the length of a cane or a lateral, thus marking the rainy periods of the growing season. Growth produced during dry periods is relatively free of the disease (note Fig. 30).

Control

The Anthracnose fungus can infect most of the commercial bramble varieties. However, some are more susceptible than others. Black Hawk, a new black raspberry variety, is a highly resistant; Dundee and Evans moderately resistant, whereas Bristol, Cumberland, and

Logan are highly susceptible. To date, the red raspberry and blackberry varieties common to Michigan are susceptible. Purple raspberry varieties are considered as a rule to be resistant to anthracnose.

Anthracnose *can be eliminated* in a new planting if the practice of early "handle" removal is used.

Propagation of raspberry is either by tip-layering or by suckers. In either case, a portion of the parent cane, termed "handle", is left on these newly rooted plants by nurserymen to make planting easier. However, these "handles" are infected generally with anthracnose; these should be cut off and destroyed before the new cane growth appears (Fig. 33).

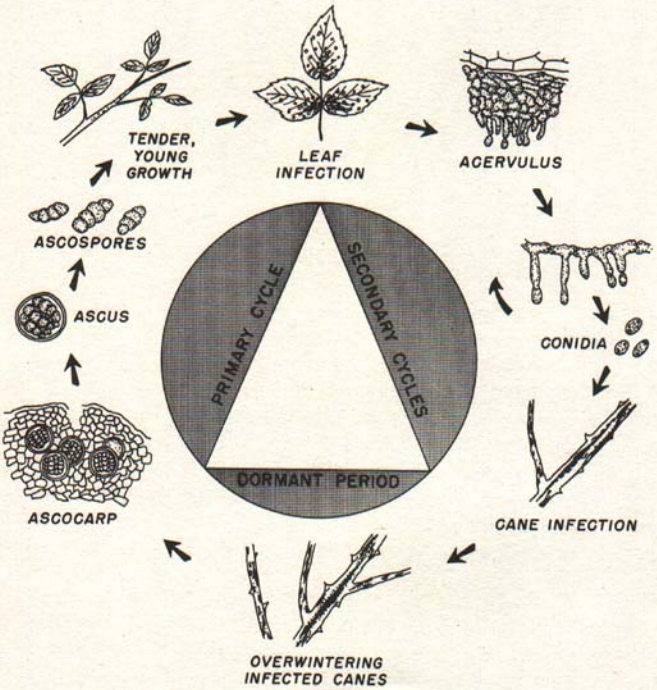


Fig. 32. Life cycle, raspberry anthracnose.

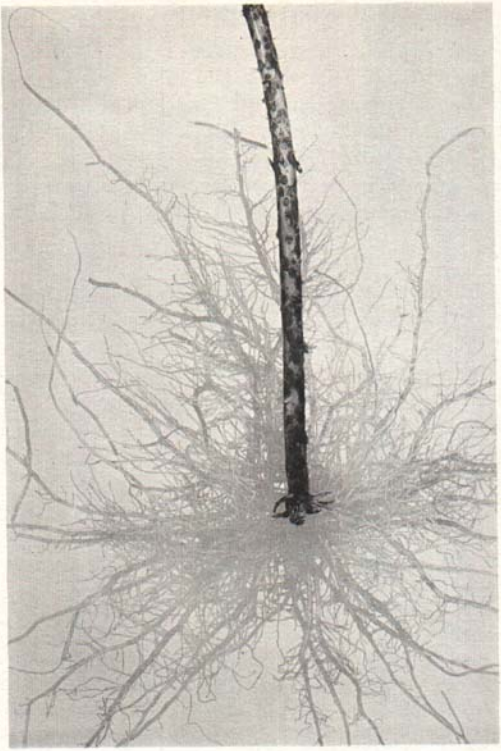


Fig. 33. Young raspberry plant with anthracnose infected "handle."

In both new and old plantings, clean cultivation in and between the rows during May and June aids in removing weeds that not only hold moisture but also interfere with proper spray coverage of the raspberry plants.

In established plantings where anthracnose is present, the only control is by spraying. This can be successful if the sprays are applied thoroughly, timed with plant development and *before* predicted rains.

SPUR BLIGHT *(Didymella applanata)*

This disease has long been a problem in red raspberry plantings. The fungus attacks the leaves, buds, and canes. On infected buds the fungus kills the outer layers and portions of the inner layers, so weakening the bud that dwarfed fruit spurs develop, producing little or no fruit. In addition, the diseased outer layers of the buds shrivel, causing the layers of the bud tip to separate leaving the inner tissue exposed to winter injury and probably death.

Symptoms

Canes—In late June or early July, dark brown to purple discolored areas may be noted on the lower portions of young canes. These lesions develop at the base of the leaves and around the buds in the axils of these leaves. The darkened areas may extend from one node to another and completely encircle the cane (Fig. 34).

Leaf stems arising from infected nodes are so injured that the leaves drop off, leaving the stem attached to the cane.

In July and August, after a rainy period, small brown pycnidia are observed on the discolored surfaces of the cane. By fall, these discolored areas become grey in color, the epidermis splitting along the length of the cane. These cane areas including the buds, are thoroughly studded with small, black perithecia.

Leaves—Small, brown spots are first noted on the leaf veins. These lesions enlarge, spreading rapidly along the vein and becoming angular in shape, the widest portion toward the leaf margin (Fig. 35). Furthermore, the disease may develop down the leaf stem and infect the cane at the point of leaf attachment.

Disease Cycle

The life cycle of spur blight consists of (1) primary and (2) secondary phases.

Ascospores mature in May and June on the overwintered one-year-old canes and discharge during rainy periods. In the presence of moisture they germinate, penetrate the tissues and establish a primary infection. In addition to ascospores as a source of primary infection, pycnidial bodies containing conidia are disseminated also during the spring rains. Pycnidia and perithecia are often intermingled on the overwintered infected canes, but cannot be distinguished separately by visual observation.

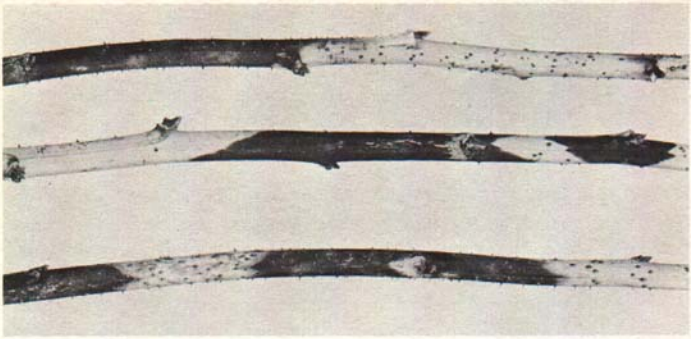


Fig. 34. Primo canes of red raspberry showing spur blight infections (darkened areas).

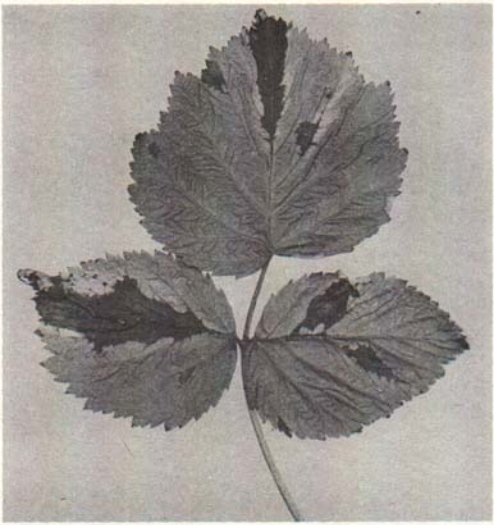


Fig. 35. Spur blight infected red raspberry leaf. Note the dark lesions extending out to the leaflet margins.

The production of pycnidial bodies in the primary infected plant parts is dependent upon moisture. In dry seasons they may not be evident until late July. Nevertheless, they serve to produce and discharge conidia which cause waves of secondary infection during the remainder of the growing season following wet periods.

Control

The spur blight fungus mainly attacks the red raspberry. It may be noted rarely on black raspberry, while blackberry varieties are resistant. At present, there are no known red raspberry selections immune to the ravages of this fungus.

Cultural practices conducive to reducing spur blight are similar to those for raspberry anthracnose. However, "handle" removal cannot be stressed enough as a means to remove the main source of the disease when setting out new plantings.

Since the fungus inciting spur blight is within the plant tissues, control by fungicides is difficult. Eradicant chemicals applied at the green tip stage kill many of the overwintering pycnidia and perithecia on the canes. However, additional protective sprays are required to cover susceptible new growth during the critical spore discharge period in late spring.

CANE BLIGHT *(Leptosphaeria coniothyrium)*

Cane blight can attack all of the varieties of raspberries grown in Michigan. It is considered a relatively minor disease, but under ideal climatic conditions it can kill back primo canes encompassing one to several laterals which reduce yields for that bush as much as 50 percent the next fruiting season. Infections on lower portions of the canes girdle that area causing death to all portions of the cane above the infection site.

Death of canes due to winter injury is sometimes erroneously attributed to cane blight, for the fungus grows readily on any dead or injured bramble cane parts. Thus, in some cases, this disease has received more than its rightful share of credit for the damage produced.

Symptoms

On new cane growth, the disease is apparent late in the growing season as a brown to black discolored area, on those portions of the cane that have been topped back. This may extend down one side of the cane

or completely encircle the terminal portion of the cane from one to several inches. By fall, this affected area is gray in color with small, black fungal bodies concentrated mainly at the margins of the infection (Fig. 36).

Fig. 36. Raspberry cane blight infection at the terminal portion of a primo cane. Note the small, black fruiting bodies of the fungus.

On fruiting canes, the most characteristic symptoms are noted between blossoming and the period of fruit ripening. Affected canes suddenly begin to wilt, leaves turn brown, developing berries shrivel and dry up, and generally all of the affected cane dies. The site of infection and corroboration of cause can be made visually. An examination of the cane may disclose one or more areas varying from one to several inches in length, grayish-brown in color and dotted with small, black fungal bodies (Fig. 37). Sections through the cane in these areas will also show the wood to be deep brown and quite brittle.

Disease Cycle

The fungus produces spores during late spring and summer from pycnidia imbedded in the bark of infected fruiting canes. The spores are pushed out of the pycnidium in long tendrils and spattered onto growing primo canes during rains. The spores germinate during this wet period and enter the canes through wounds caused by insects, wind rub, or topping of new cane growth.

If the infection occurs on a topped cane, the extent of invasion may

Fig. 37. Raspberry cane blight infection on a fruiting cane. Note discolored area and fungal bodies.

be several inches or completely down the cane before the end of the growing season. Infections down on the cane cause no noticeable injury that season. However, by the second year, the fungus has destroyed most or all of the vascular tissue at the site of infection and the cane, now in its fruiting year, is killed.

Rainy weather is conducive to infection. But the extent of fungal invasion is dependent upon high temperatures. This accounts for the slow progress of the disease in fall and early spring followed by rapid development the latter part of June through July. Generally, cane blight is most destructive when a warm wet summer prevails, followed by a wet spring and a dry ripening period the next year.

Control

The causal organism of cane blight is a wound parasite. It is obvious that attempts to eliminate wounding on bramble canes is impossible. However, if the topping of new growth is completed at least three days *before* predicted rain, cane blight injury to those portions of the cane is negligible. Such wounds are hardened off enough to resist infection during the following wet period.

Old fruiting canes that are pruned out of the bushes as a cultural practice should be removed from the planting and destroyed, since the causal fungus may survive as long as four years after the cane is dead.

Chemical sprays to control cane blight have yet to be developed.

ORANGE RUST *(Gymnoconia interstitialis)*

This fungus disease has attained economic importance in brambles since infected plants are a total loss. Furthermore, all new plants developed or propagated from an infected cane will also be infected with the orange rust fungus.

Symptoms

Rusted plants are located easily in the planting after growth is initiated in the spring. New shoots are spindly, *lacking spines* and bear pale green leaves that are stunted or misshapen. Often the leaves are bunched together. In the latter part of April or in early May, small yellow dots appear on both surfaces of the leaves. These spots appear before the leaves have unfolded entirely and they turn black in color. After the leaves have opened, blister-like reddish-orange pustules appear on the lower surface of the leaves. These pustules-aecia-waxy at first and later becoming powdery, are exposed by the rupturing of the leaf epidermis. They may be isolated or cover the entire surface of the leaf (Fig. 38). Masses of bright orange colored spores are produced from these aecia for several weeks, making the affected bush and the disorder recognizable.

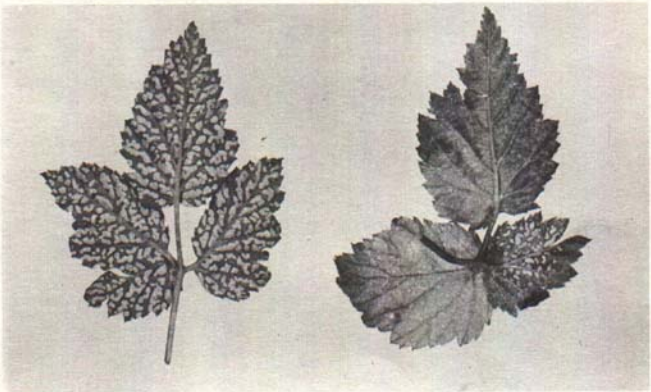


Fig. 38. Under leaf surface of an orange rust infected black raspberry. Note the typical spore cups or aecia.

Buds on the fruiting canes send out a number of lateral shoots in the spring. They not only lack blossoms, but present a "witches broom" effect. (Fig. 39).

Disease Cycle

The orange rust fungus completes its entire life cycle on the *Rubus* host. There are two forms of the fungus—a long cycled form in which true teliospores are produced, common on black raspberry, and a short-cycled form in which teliospores are not produced, common on blackberries and dewberries. Follow Fig. 40 as you read the text.

The fungus mycelium is perennial within the infected plant and overwinters in this manner. New growth in the form of shoots, leaves, and roots is quickly invaded by the fungus. The first external signs of the fungus itself are in the form of small fruiting bodies, pycnia, generally on the upper leaf surface. Orange colored structures, aecia, are noted several weeks later on the lower leaf surface and are filled with great numbers of spores, termed aeciospores.

These spores are carried to other leaves by air currents, where they germinate and penetrate the leaf surfaces directly. After leaf infection has occurred with the long-cycle aeciospore, the formation of telia takes place in early fall. These telia produce two-celled structures, teliospores, each cell germinating to form a four celled tube (basidium), each of which bears another spore, termed a basidiospore.

Fig. 39. Healthy black raspberry plant (left); orange rust plant (right). Note the spindly growth and small leaves.

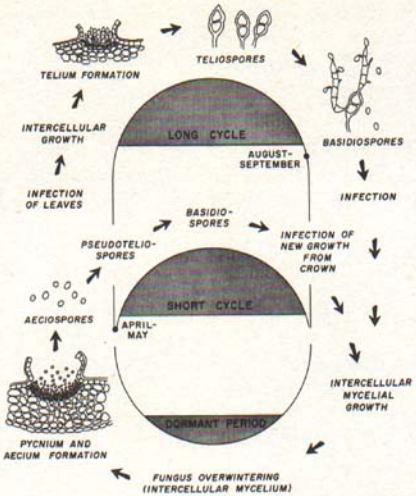


Fig. 40 Life cycle, orange rust.

These basidiospores subsequently infect the buds at the tips of newly rooted canes and young shoots arising from the crown. The plant develops normally during the remainder of the season and no symptoms are visible on this newly infected plant until the following year.

In the case of the short-cycle rust common on blackberry and dewberry, the orange colored aeciospores do not germinate to form telia, but instead they assume the role of a two-celled teliospore and germinate to form basidiospores directly. These in turn infect the new shoots and buds. In either case, the end result is the same: infection of new healthy growth and spread of the fungus through the host tissue.

Control

Since the fungus is systemic and completes its entire cycle within the host, a control program based on infected plant removal is the best control in established plantings. However, it is important that orange rust infected plants be dug completely (including the roots) *before* the orange spores begin to shed from the leaves. It is not necessary to

remove these plants from the planting immediately, as the fungus dies almost as soon as the plant does.

Since orange rust may be carried undetected in planting stock, even first year fields should be watched and infected plants removed if they appear.

Finally, if at all feasible, orange rust infected wild brambles growing in the vicinity of the commercial fields should be destroyed to prevent initial or further infections from these wild sources.

VERTICILLIUM WILT *(Verticillium albo-atrum)*

Infection of brambles by this fungus occurs sporadically in Michigan, but with great losses. The plant dies gradually within several seasons. During this period of decline, infected plants bear less fruit and are subject to winter injury.

Symptoms

In the middle of the season during warm weather, leaves of affected black raspberry plants become pale yellow and cane growth is slow and stunted. Later in the season, with the advent of cooler weather, the plants appear to recover. However, in the second season the symptoms are more marked. The *lower* leaves curl up at the margins, turn yellow, and wilt. Soon a similar condition exists up the cane. A blue stripe extending from the base of the cane upwards is often associated with the wilting of the leaves. The bluish color is the result of the brownish-blue discoloration of the diseased bark as seen through the waxy bloom covering the cane. When the bloom is rubbed off, the bluish color disappears, but the stripe remains brown. In advanced stages of the disease, the entire plant presents a wilted condition (Fig. 41).

Red raspberries show similar symptoms as blacks but are less severe. Leaves on affected canes wilt and die, often leaving a tuft of leaves at the tip of the cane. Some canes are more affected on one side than the other. The cane discoloration, quite prominent in black raspberry, is often absent on red raspberry, or if present, it is hard to distinguish from the normal bronze to reddish coloration.

Disease Cycle

The causal fungus lives in the soil and penetrates the root system of the raspberry plant. Verticillium may be introduced into soils by resistant transplants of potato, tomato, and eggplants. Once in the

Fig. 41. Blask raspberry showing effects of Verticillium wilt.

plant, it disrupts the water conducting tissues, which interferes with the movement of water and food materials to the growing plant parts. The plant begins to wilt and eventually dies.

Control

If feasible, raspberry plantings should not be made in fields that have had a history of potato, tomato, and eggplant within five years. If the soil is known to contain the causal fungus, a 3 or 4 year crop rotation with two or three non-susceptible intervening crops, mostly grains, is effective in reducing the fungus from the soil.

The control of verticillium consists mainly in preventing infection in the future planting rather than eradicating it in an established plantation.

LEAF SPOT *(Mycosphaerella rubi)*

All of the brambles are attacked in varying degrees by leaf spot. Occasionally, infections are severe. The chief damage consists of a general weakening of the plant through defoliation. This is further displayed by reduced yields and susceptibility to winter injury.

Symptoms

Early in the growing season, dark reddish spots may appear on the leaves. These spots are round and the color extends to both leaf surfaces. They average about an eighth of an inch in diameter and are rather uniform in shape and size. Soon the centers of the lesions become brown to gray, surrounded by a red to purple margin (Fig. 42). A careful examination of the affected leaves will disclose the presence of small, black pycnidia embedded in the center of the lesion.

Occasionally the central portion of the lesions drop out, presenting a "shot-hole" appearance.

On the canes the lesions are grey in color with numerous black pycnidia widely scattered over the lesion.

Disease Cycle

The fungus overwinters in the pycnidia on the affected canes and fallen leaves. In late spring or early winter, during wet periods, spores are washed or splashed onto the young succulent leaves where infection occurs on the lower leaf surface. Only a few lesions may be

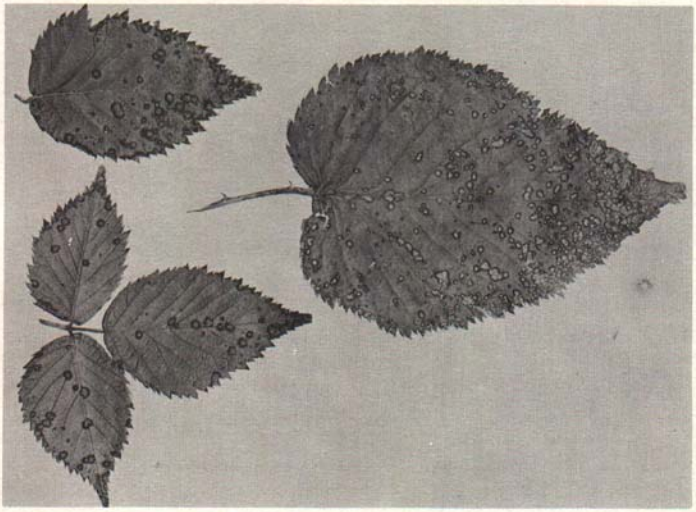


Fig. 42. Leaf spot on blackberry. Note the dark margin and spots growing together in the case of severe infections.

noted during this primary infection period. However, it serves to reestablish the causal fungus on *Rubus*. Then numerous pycnidia bearing great numbers of spores develop in these initial points of infection. Subsequent rains serve to spread the spores to other developing leaves causing waves of secondary infection throughout the growing season.

Control

All of the brambles cultivated in Michigan are susceptible to leaf spot in varying degrees—none are completely immune. However, cultural practices to reduce weed growth and increase air circulation for quick drying of leaves reduces infection. This, coupled with spray recommendations as suggested for raspberry anthracnose will materially reduce leaf spot.

LATE LEAF RUST *(Pucciniastrum americanum)*

This non-systemic rust occurs only on red raspberry and is limited generally to northern Michigan and the Upper Peninsula. It causes premature defoliation, thus weakening the next season's fruiting canes.

Symptoms

In late summer, the undersurface of leaves will display a yellow to orange color, powdery in appearance. The upper surface of leaves below the affected leaves present a speckled yellow dust due to the discharge of the spores from the higher leaves. In severe cases, the spore masses may be observed on leaf stems and shoots.

However, these spore masses of the fungus do *not* have the waxy characteristic of orange rust, nor do they occur early in the season as does orange rust.

Disease Cycle

The causal organism of late leaf rust is heteroecious; i.e., part of the disease cycle occurs on an alternate host, white spruce *(Picea canadensis)*. The spores borne on the raspberry leaf are capable of reinfecting this host. Late in the season another spore form, teliospore, is produced on the raspberry which develop an airborne spore that infects the needles of the white spruce. Within this alternate host the fungus overwinters to produce infective spores the next season attacking the red raspberry.

Control

Since the disease occurs late in the season and is not systemic within raspberry, little has been done to curb the problem. However, reduction of late leaf rust can be achieved by removal of white spruce within the proximity of red raspberry fields.

POWDERY MILDEW *(Sphaerotheca humuli)*

This fungus disease has occurred frequently on red raspberry. With the introduction of new susceptible black raspberry varieties, it is becoming more important. The damage is to the terminal portions of primo canes and laterals. Powdery mildew stunts these areas, reduces yields and may cause death to terminals, ultimately inhibiting tip propagation of black raspberry.

Symptoms

Primarily, mildew attacks the leaves of *Rubus* but is evident on the young succulent terminal portions of the primo canes and laterals. On the leaves, irregular light green to yellow blotches appear on the upper surfaces. Just opposite, on the lower leaf side, the surface is covered with the white to gray mealy growth of the fungus. In certain cases, these lesions may have a water soaked appearance. In severe outbreaks, especially on red raspberry, the fungus may cover both surfaces entirely. As a result of leaf infection, the leaflets are often distorted with margins curled up (Fig. 43).

Cane and stem infections are noted by the white powdery fungus growth. Such infected portions of the plant are dwarfed. If the cane does continue to develop, the leaves are extremely small, presenting a short spike-like or "rat-tail" appearance.

Control

Chemical sprays to reduce powdery mildew have not been developed. Sulphur, a common mildewcide, is too injurious for use on *Rubus.*

Varieties of raspberry vary in resistance to the disease. Blackberry appears to be resistant while most commercial varieties of dewberry and red raspberry are highly susceptible. The Cumberland and Logan varieties of black raspberry are attacked by mildew but to date the damage is negligible. However, the new selection Black Hawk is highly susceptible, resulting in reduced yields and poor propagation.

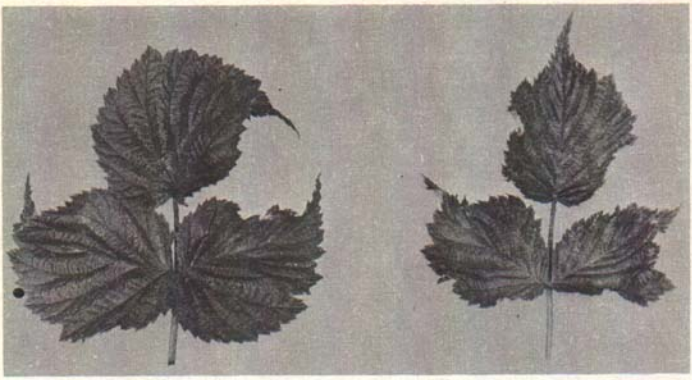


Fig. 43. Powdery mildew on red raspberry leaves. Note light discolored areas and curled margins.

VI. STRAWBERRY DISEASES

RED STELE ROOT ROT *(Phytophthora fragariae)*

In terms of known losses and difficulty of control, this fungus disease is considered the most serious on strawberry in Michigan. Once the fungus is introduced into the field, only resistant varieties can be grown. Losses are not noted until the bearing year and can wipe out an entire crop at this time before harvest. Presence of only one diseased plant in a nursery field makes it necessary to condemn the entire planting.

Finally, the development of new "races or strains" of the fungus, capable of attacking and infecting lines of resistant varieties is making breeding for control complicated and difficult.

Symptoms

Effects of the disease are seen best in the spring of the first bearing year. The above ground symptoms vary with the season. If the spring period is dry, infected plants generally die before or during the blossom period. During wet springs, young leaves on infected plants are small and are a dull bluish-green. The plants develop normally to all outward appearances. During warm days they begin to wilt

and by harvest collapse completely. Only portions of a field may show these symptoms, generally in the low, poorly-drained areas.

Since black root rot or winter injury often presents stunted and wilting plants, corroboration of red stele is made by root examination (Fig. 44.) Affected plants should be removed from the soil carefully. In the first stage of red stele, many small, fibrous rootlets are lost. The remaining succulent, white main roots are bare of rootlets and present a "rat tail" appearance (Fig. 45). Some or all of these main roots may show an external brown rot beginning at the tips. When these roots are split open lengthwise, the central core of the root, (botanically termed stele) may display a brown discoloration near the tip. This color will mask a reddened stele; however, there usually is red color extending up the stelar region from the dead terminal portion (Fig. 46). This symptom is known for no other disease.

Plants on the margin of a red stele pocket in the field appear to recover after harvest. Infected roots rot off and new ones are produced from the crown. This recovery condition is only temporary; the next spring the plants will again become infected. Death of plants due to red stele is more severe during the second fruiting season than the first.

Disease Cycle

During cool, (32° to 55° F), wet soil conditions above 80% water holding capacity in the late fall or early spring, the resting spores, termed oöspores, germinate. The oöspores are thick walled and ca-

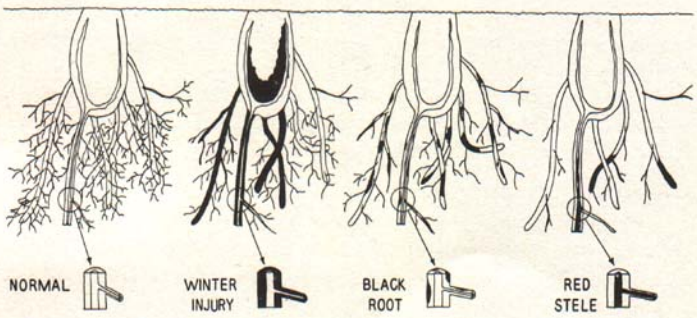


Fig. 44. Common symptoms of strawberry root disorders. Note close up views for internal symptoms.

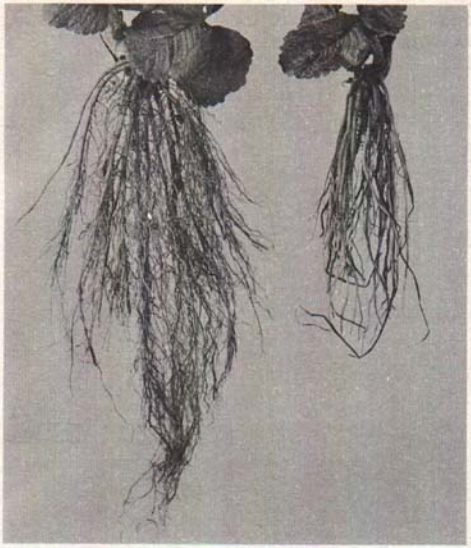


Fig. 45. Roots at right infected with red stele root rot. Note absence of small feeding roots as compared with normal roots, left.

pable of withstanding high soil temperatures, thus serving to carry the causal organism through the summer months and for survival in the soil for many years. Upon germination, a zoösporangium is produced (sac-like) which contains many zoöspores (Fig. 47). These spores are motile for several hours, and can be moved or carried fairly long distances in drainage and soil water. Upon contact with strawberry root tips, the infective zoöspores penetrate through the root cap of the young roots. Fungal strands grow into the root and up the stelar region. Other zoöspore-bearing sacs may arise from the root surfaces for further field spread of the disease.

The resting spore, oöspore, is soon developed in the stelar region of the root, thus completing the disease cycle.

Control

The prevention of red stele is its best control measure. Select a planting site that has good soil drainage, preferably located where

water from adjoining land will *not* drain through it. Start new plantings with certified, state-inspected stock only. Avoid using illegal, free neighborhood, uninspected plants. Use your own machinery for setting out the field and for general cultural practices. Borrowed equipment should be cleaned of all soil and plant debris before use. If the planting site is known to be free of red stele and you wish to grow resistant varieties, then obtain Michigan state-certified stock only. At present, Michigan is the *only* state that requires nursery stock of resistant varieties to be grown on soil free of the red stele fungus.

To date, rotation, spraying, soil disinfection, and dipping plants in chemical solutions have been of no practical value in controlling red stele disease. If you have red stele in your soil, the only control method known, at present, is to use resistant varieties.

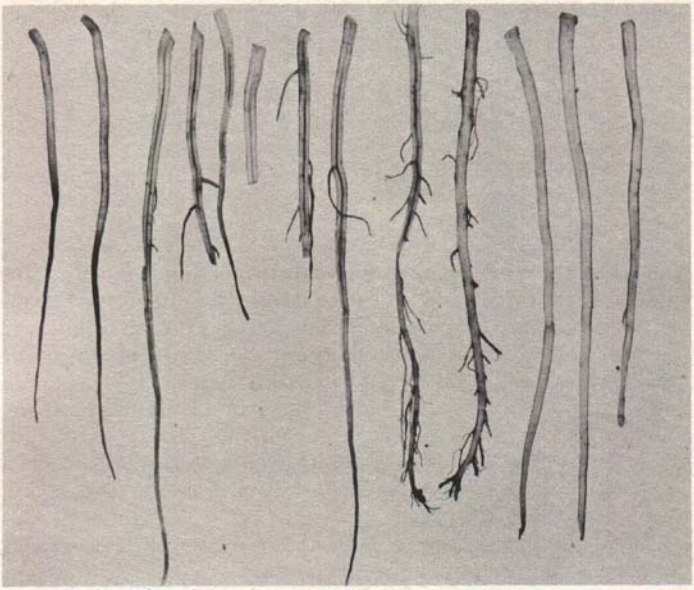


Fig. 46. Strawberry roots showing symptoms of red stele disease. Left – diseased roots showing dead, discolored root tips. Diseased roots split lengthwise to show the reddened core; symptoms. Right – normal roots split lengthwise to show the absence of the red center.

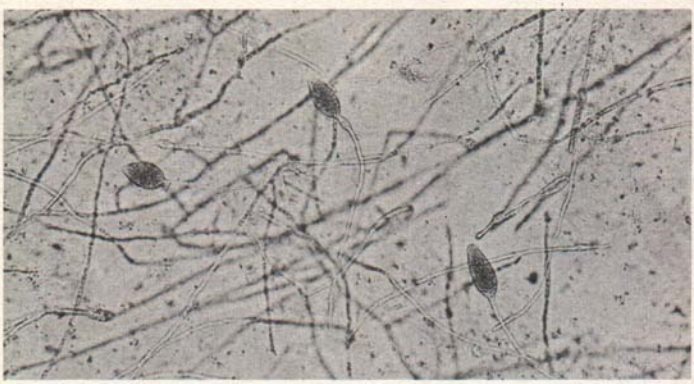


Fig. 47. Fungus growth of the red stele organism. Note the three lemon-shaped spore sacs filled with infective zoospores.

A new problem in fields where red stele is present is arising now when known resistant varieties succumb to the disease. In such an event, consult Table I on reactions of varieties to the several red stele races to determine possible strawberry varieties for trial on that soil complex.

BLACK ROOT *(Fungi complex)*

Strawberry plants affected by black root rot are weakened, low in vigor, and subject to drought. This disease complex serves to deteriorate plantings and resulting yields.

Symptoms

Affected plants are stunted with a tendency to wilt and then recover during the night; but, if dry weather persists, the plants die with the berries hanging green and shriveled.

The leaves may become purplish or bronzed, with red petioles. Such a plant, after being dug up, will show a root system which is black, corky in texture, and apparently dead. However, younger plants display typical root rot symptoms. Several of the main roots may be rotted their entire length, or many brown or black lesions will be noted scattered over the root surface (Fig. 48). These lesions vary in size from ½ inch to several inches in length and may encircle the root. At first, the lesion may be reddish brown, later turning black. Lesions

on the finer roots resemble those on the main roots. It is the attack on the fine rootlets that reduces most of the flow of water and plant-food to the plant.

Finally, close examination of the affected root will disclose that the fungus invasion is limited generally to the cortex or outer cylinders of the root and not the central core or stele. The stele will be a normal white, while the cortex is brown-black and spongy in texture. Later, the entire root structure including the stele decays.

Disease Cycle

A number of fungi are capable of attacking strawberry roots and causing the cortical rot. However, these organisms are weak parasites. Any condition either climatic or cultural that will reduce the vigor of their root systems will increase root rot.

Control

It is obvious that some cases of root rot are confused with winter injury or fertilizer burn (Fig. 44). Therefore, cultural practices to reduce either problem should be used.

TABLE 1—Reactions of strawberry varieties to races of the red stele fungus

Varieties	RACES				
	A-1 ("common")	A-2 (A)	A-3 (S)	A-4	A-5
Aberdeen	R	R	S	S	S
Fairland	R	R	S	(S)	(S?)
Maine-55	R	(R?)	(S?)	(S?)	(S?)
Marshall	R	—	S	R	—
Mastodon	—	—	S	—	—
Monmouth	R	(R?)	(S?)	(S?)	(S?)
Orland	R	(R?)	(S?)	(S?)	(S?)
Plentiful	R	(R?)	(S?)	(S?)	(S?)
Puget Beauty	R	(R?)	—	—	—
Redcrop	R	(R?)	(S?)	(S?)	(S?)
Redglow	R	(R?)	(S?)	(S?)	(S?)
Siletz	R	—	R	R	S
Sparkle	R	R	S	(S?)	(S?)
Stelemaster	R	R	R	R	S
Surecrop	R	R	R	(R?)	S
Temple	R	R	S	S	S
Vermilion	R	(R?)	(S?)	(S?)	S

R = Resistant; S = Susceptible.

Reactions indicated in parentheses (?) are probable, based on parentage of the varieties, and are not based on experimental results. All other commonly grown United States varieties are susceptible to all races of the red stele fungus as far as is known. (Courtesy U.S.D.A.)

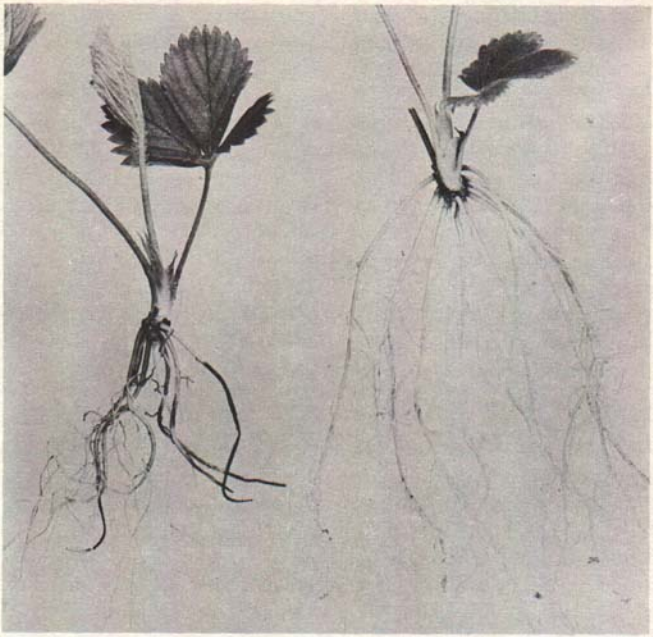


Fig. 48. Black root rot of strawberry. Note the typical die back and lesions on left plant. Right, healthy root system.

In northern Michigan, black root rot has been alleviated greatly through the use of soil fumigation for nematode control. In these areas it was disclosed that several severely affected fields had a high nematode population. Thus, the soil-inhabiting fungi were able to penetrate roots easily through the wounds produced by the nematodes.

In other areas where nematodes were not a problem, but root rot was evident, crop rotation gave economical control. Several rotations of soy beans plowed under as they begin to pod is suggested.

VERTICILLIUM WILT *(Verticillium albo-atrum)*

This soil-borne fungus disease has been recognized on strawberries for many years but is now gaining in importance in Michigan due to the introduction of new susceptible varieties. Infected plants generally

die, or if they live, they produce few berries. Also spot dying of plants may be so extensive that the fields are abandoned.

Symptoms

Verticillium is noted during the summer months in spring-set fields. Individual plants begin to wilt, commencing with the older leaves which show red petioles or stems. These older leaves prematurely turn reddish-yellow and die. Young leaves wilt and have a tendency to curl up along the mid vein.

An examination of the roots may show extensive decay. Sections through the roots and crown discloses a brown discoloration of the tissues. However, for positive diagnosis of the disease, small pieces of the stem tissue must be *surface-sterilized* and placed on *sterile agar media* in order to culture and identify the Verticillium fungus if present.

Control

In Michigan, strawberry plantings proven to be infected with Verticillium have been in rotation with either tomato, potato, or eggplant. Even though the disease was not noticed on those crops, it is often introduced into the planting on resistant transplants. If the fungus was present from former years it will build up on the plant debris from these crops. A crop rotation of several years, if feasible, with non-susceptible grain crops will aid greatly in reducing the inoculum potential of the fungus.

Soil treatments with chloropicrin have been successful and are used in other areas of the country. The cost of the fumigating material and application prevent its wide use in Michigan at the present time.

However, varieties display degrees of resistant and susceptibility to wilt. A recent report indicates that Aberdeen, Blakemore, Catskill, Gem, Premier, Tennessee Beauty, Robinson, and Surecrop are quite resistant. Whereas, Armore, Dixieland, Earlidawn, Jerseybelle, Pocahontas and Sparkle are highly susceptible to this disease.

RHIZOCTONIA BUD ROT *(Rhizoctonia solani)*

This soil borne disease appears sporadically in Michigan plantings. It is most evident in low portions of the field after periods of high rainfall during bloom. The loss is noted in the absence of blossoms or fruit on affected plants.

Symptoms

In bearing fields, the disease is noted between the bloom and harvest period. Several plants in one row or many plants in a circular to oval area of the field may be affected. The terminal bud is water-soaked or dead and can be removed easily from the plant. The previous year's outer leaves assume a flat or horizontal position and are deep green. Several new leaf buds develop from the crown under the infected area. Their growth is weak and spindly.

Some leaves of surviving crown buds are injured before they unfold. After growth, the tips and margins of the leaflets are often brown and an entire leaflet of the compound may be absent completely.

Stolons or runners arising from the affected plant may be girdled at the base and brown in color.

Sometimes plants are killed that season. If not, they produce no fruit that year.

In spring set fields leaf and runner symptoms of bud rot may be noted.

Control

Since Rhizoctonia bud rot is soil borne or introduced into the plantings on soil in nursery stock, the following control is suggested:

Avoid the use of planting stock dug from wet soil. It is better to wait and dig stock after the soil moisture has decreased. Do not set plants deep or cover the crowns during cultivation. These practices will reduce the introduction of contaminated soil into the terminal growing points of the crown.

Finally, attempt to select planting sites that have a well drained soil to alleviate pockets of water on the matted rows after heavy rains.

LEAF BLIGHT *(Dendrophoma obscurans)*

Within the past few years, leaf blight has assumed considerable importance. The increase of the disease has resulted from extensive plantings of susceptible varieties. Large portions of the leaves may be killed resulting in weak runner plants and decline in yields. Furthermore, the causal organism is associated with a fruit rot of strawberry.

Symptoms

The disease may be noted on the leaves in early summer. Young lesions are reddish-purple and circular. If near a main vein, they are elliptical. Later, the lesion presents a purple border with a brown center. Infections located near the midrib of a leaflet form the typical V-shaped lesion of leaf blight, fanning out to the margin (Fig. 49). Later, these infected leaf areas become light brown to silvery due to the eruption of the leaf's waxy layer (cuticle). A careful examination of these lesions will disclose small, black pycnidia embedded in the infected leaf tissues (Fig. 50).

Similar type lesions may be noted also on the sepals or cap of the fruit.

Disease Cycle

Complete information on the disease cycle is lacking. However, mature pycnidia are present in early summer in lesions on overwintered leaves. During rainy periods, after growth begins, infections are noted on the partially mature leaves and sepals of this growth. Further infections occur only during rain periods throughout the remainder of the growing season.

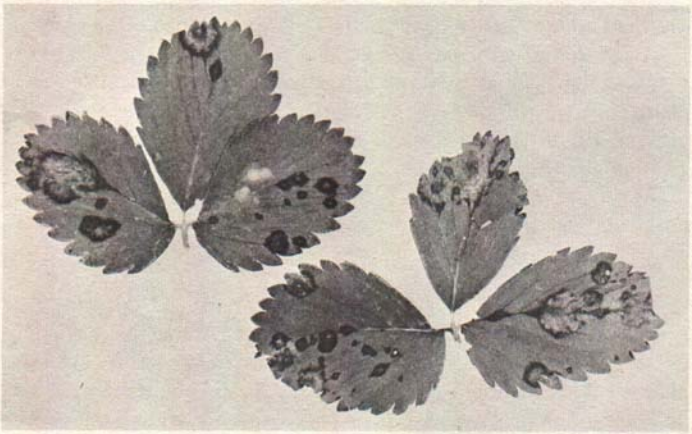


Fig. 49. Strawberry leaf blight on the upper surface of leaves. Note the characteristic V-shaped lesions.

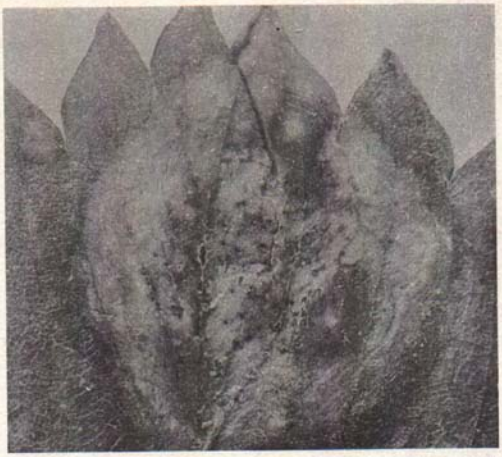


Fig. 50. Leaf blight of strawberry. Note the black pycnidia in the central portion of the lesion.

Control

Strawberry varieties display varying resistance to leaf blight. However, the most promising or commonly used commercial varieties are susceptible.

Economic control of this disease has been achieved by the use of one or two sprays of an eradicant fungicide.

LEAF SPOT *(Mycosphaerella fragariae)*

The presence of this disease in Michigan is sporadic. However, under proper environmental conditions, (see below) leaf spot can attain epidemic proportions. Severe infections can kill entire leaves resulting in reduced yields, lower grade of harvested berries, and a loss in vigor of affected plants.

Symptoms

This fungus disease is first noticeable in the spring as small, purplish, circular spots on the upper surface of young leaflets. As the fungal infection continues, the center of the spot becomes gray to white and is surrounded by a broad reddish-purple margin. The spot

is generally ⅛ to ¼ inch in diameter (Fig. 51). Leaf spot infections on the under surface of the leaflets are similar in appearance to those on the upper surfaces, though less brilliant in color.

In addition, if the main or any lateral leaflets' veins come in contact with a spot, they develop a premature reddish-purple color that generally extends beyond the infected areas. The leaf spot fungus may also attack leaf stems, fruit stalks, and fruit calyxes exhibiting similar spots to those described for the leaves. On the stems and stalks, however, the lesions tend to be more elongated than circular.

Infection of strawberry fruit by this fungus is not too common. When it does occur, it is quite conspicuous on white unripe fruits or on ripe fruits of light-colored varieties. On the fruit, the disease has been called "black seed". Usually one to several seeds on the berry, including the area surrounding them, are brownish-black (Fig. 52). The pulp of the berry under these infected areas is discolored. Although there is no rotting of the fruit, these lesions have such a distracting appearance that they lower the grade or render the fruit unmarketable.

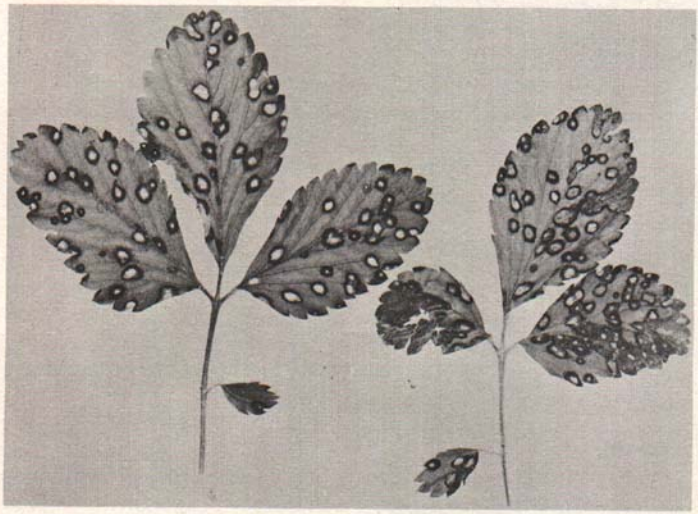


Fig. 51. Strawberry leaf spot on the upper side of leaves. Note the white centers and the typical dark margins surrounding the lesion.

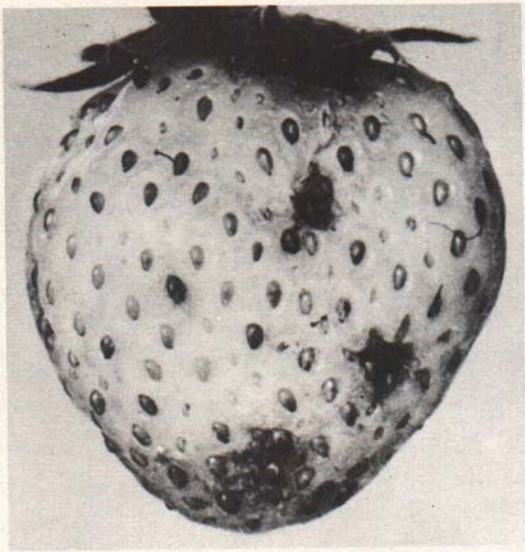


Fig. 52. Black seed disease of strawberry fruit. Note darkened seeds and their surrounding area.

Disease Cycle

The leaf spot fungus overwinters in previously infected leaflets and in other plant parts. In the spring, clusters of spores emerge from these infected spots during periods of high relative humidity (Fig. 53). These are scattered by rains to developing young leaves which are extremely susceptible. Therefore, leaf spot in a given strawberry planting is favored by low spring temperatures and prolonged wet periods. Protracted cold weather delays the maturity of the young leaves, and the prolonged wet periods are conductive to spore production, dissemination, and infection.

Disseminated spores that come in contact with strawberry leaves germinate and infect through the stomata (leaf air pores). Generally, this occurs on the lower leaf surface since in most varieties, the stomata are predominant on the lower side. Within 10 to 14 days, leaf spot symptoms will be evident on the upper surface. Soon, great numbers

Fig. 53. White tufts of spores in the leaf spot lesions following a rain period.

of spores are present in these lesions which cause waves of secondary infections throughout the growing season during prolonged wet periods.

Control

The commercial varieties, Catskill, Premier, and Robinson, are considered to be resistant to leaf spot, whereas Armore, Blakemore, Fairland, Jerseybelle, Pocahontas, Redglow, and Sparkle are quite susceptible.

Sprays for leaf spot should be applied in spring-set fields if the disease is clearly evident. This will aid in reducing the inoculum potential for the next bearing season. On bearing fields that have leaf spot, chemical sprays should be applied early in the season to reduce primary infection not only on the leaves but on the green caps of the developing fruit.

POWDERY MILDEW *(Sphaerotheca macularis)*

This leaf disease is attaining economic importance due to the introduction of new susceptible varieties and the replacement of inorganic fungicides by new organic chemicals. Affected plants are reduced in vigor, the fruit may fail to color properly or present a white powdery appearance.

Symptoms

In early summer, young leaves may show upward curling of their margins, exposing the undersurfaces. Severe infections may cause the individual leaflets to curl to such an extent that the margins overlap—funnel or cigar shaped in appearance.

Accompanying the curled effect of the leaflets, small reddish-purple lesions may be obseved on both surfaces along the main and lateral veins. Commonly noted first on the basal portion of the leaflet. Later the discoloration extends out between the lateral veins resulting in death to the leaflet. A final confirmation of mildew is the presence of the white to gray mycelial mats on the under surface of the leaflets.

Mildew infection on the fruit is evidenced by the frosty white fungal growth on the berry surface. Those portions of the berry attacked fail to color properly.

Control

Here again we find a great degree of resistance to a leaf disease by strawberry varieties. Such commercial selections as Armore, Redglow, Robinson and Tennessee Beauty are highly susceptible to powdery mildew.

Control of mildew on these varieties by various fungicides is feasible. However, it is important to note that thorough coverage is essential for control. Therefore, a wetting agent should be added to the spray before application.

FRUIT ROTS

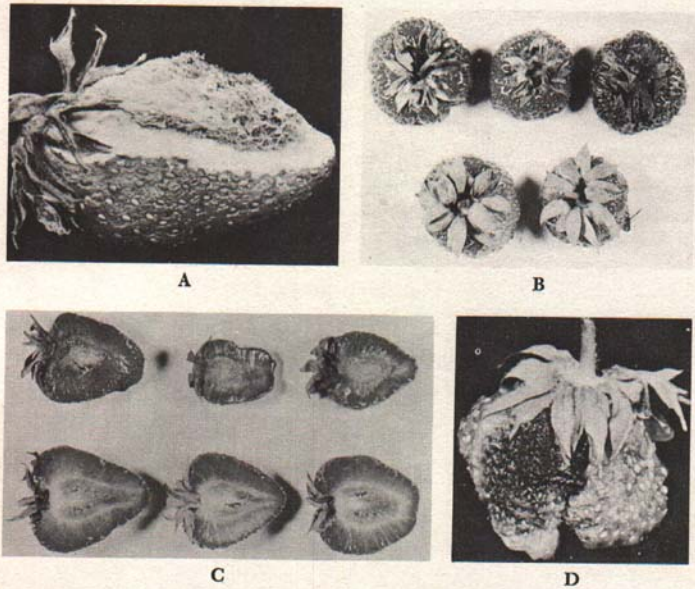
Strawberry fruit rots in Michigan occur sporadically, depending upon local weather conditions. Generally, prolonged rains and moderate temperatures are required for extensive rot outbreaks. Losses of fruit due to the various rots vary from relatively few to a total crop loss.

GRAY MOLD *(Botrytis cinerea)*

Early in the growing season, this disease may be noted as a blossom blight. Portions or all of the blossom and stalk may be affected. The condition is noted as a water-soaking of tissues, followed by a gray coloration—the spore masses. Blossom blight is quite common during frost periods. For any injured plant tissue is invaded readily by the fungus. Within 24 hours after frost, the gray spore masses are evident on frosted blossoms.

Fruit infection occurs mainly from post bloom to pre-harvest, for infected ripe berries are seldom noted. Botrytis infection of the petals or sepals during bloom can result in early fruit infection under or next to the berry cap. Furthermore, infection under the cap is enhanced by a slight depression in the fruit at this point which retains moisture. Thus aiding in rapid spore germination.

The first expression of gray mold on a berry is a small depressed water-soaked area, that appears to be lighter in color than the adjoining tissues. This area rapidly turns brown and begins to wrinkle. The browning extends into and through the fruit. Approximately 24 hours later, the original site of infection is covered with the gray spore masses (Fig. 54A). The fungus soon invades the complete berry, turning it into a wrinkled, mummified gray mass. Later, small black bodies



A B C D

Fig. 54. A. – Berry affected by gray mold fungus. B. – Stem-end fruit rot. Note dead infected caps compared to healthy. C. – Sectioned fruit showing internal symptom. Bottom healthy; top infected with stem-end fungal rot. D. – Rhizoctonia brown rot. Note dark spongy area.

may be noted scattered over its surface. These are weather resistant fungal structures that serve to overwinter the causal organism.

Rainfall followed by temperatures below 70°F are considered the ideal climatic conditions for gray mold outbreaks in the field.

STEM-END OR HULL ROT *(Fungi complex)*

The causal fungus or fungi responsible for this rot are still undetermined. However, outbreaks of the rot are associated with severe infections of the leaf blight disease.

Follow the disease cycle of stem-end rot, Fig. 55, as you read the text. The fungus overwinters in the previously infected leaves, stems and fruits. In the spring as the blossom buds emerge through the crown the fungus is mature and ready to discharge. If a rain period occurs, spores are disseminated to the young sepals and primary infection soon follows. This is evident as a small water-soaked spot, later becoming reddish-brown (Fig. 54B). If wet weather is frequent during this early pre-bloom period, all of the emerging blossom caps can become infected.

After primary infection of a sepal the fungus develops through this tissue, ultimately coming in contact with the fleshy berry under the cap. At this point, the infection progresses down into the berry. The pulp turns brown, soft and watery (Fig. 54C). There is a noticeable line of demarcation between healthy and diseased fruit tissues. The entire berry is enveloped by the infection, becoming wrinkled and mummified. Small black pycnidia are visible on its surface.

RHIZOCTONIA BROWN ROT *(Rhizoctonia solani)*

A soil-borne fruit rot that is initiated when ripening berries come in contact with contaminated soil. There a dry, spongy, dark brown to black rot slowly develops, causing a one-sided berry, to which small quantities of soil are noted clinging to the affected area (Fig. 54D).

Rhizoctonia brown rot may be noted in the field from the time a berry is one-third grown to harvest. The upper side of the berry develops normally, and ripens without any indication of rot on the lower side. Sections through affected fruit disclose a definite line between healthy and decayed tissues, the healthy portion being edible.

This fruit rot will not spread to other berries during storage and transportation.

LEATHER ROT *(Phytophthora cactorum)*

Occasionally in strawberry fields several days after a period of excessive rainfall, leather rot breaks out. Affected berries differ in their symptom expression depending upon the stage of maturity. On young green fruit, brown, water-soaked areas are noted, while on mature berries there is a shading of color from brown to a purplish-red. The areas are soft, becoming leathery. Sections through affected fruit disclose the vascular strands or arteries to be a darker brown than the infected pulp (Fig. 56).

Later, a white, fluffy fungal growth develops over the berry's surface. There is no clear line between diseased and healthy fruit tissues. Final diagnosis can be made by taste, for even slightly rotten berries are bitter to the taste.

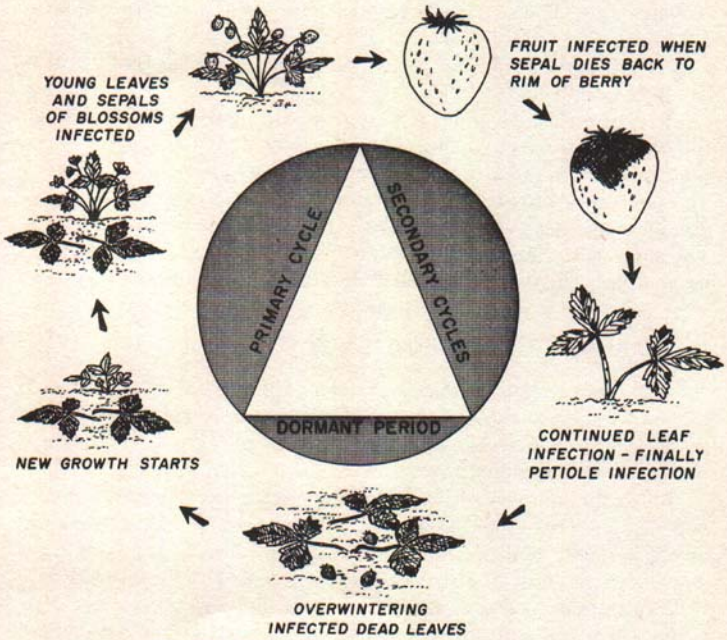


Fig. 55. Life cycle, stem-end rot of strawberry.

Fig. 56. Leather rot, note the darkened vascular strands of the sectioned berry.

LEAK *(Rhizopus nigricans)*

This rot develops in berries during shipment. Infected fruit become covered with the black mold growth of the fungus and collapse with much loss of juice. Leak develops rapidly at temperatures above 70°F and can spread throughout a crate of berries within a day.

Picking of the fruit in the morning while they are still cool and holding or shipping at temperatures below 50°F reduces leak considerably.

VIRUS DISEASES

Virus diseases affecting strawberries in Michigan have received much attention. The great difficulty has been in field diagnosis since several virus complexes are latent or symptomless within an infected plant. The presence of virus for these cases is determined by grafting the runners or excised leaves to a virus-sensitive indicator plant.

The effect of viruses on strawberry may vary between variety. However, the overall effect is reduction in runner plants and resulting yields.

However, there are several viruses and virus-like disorders that display good field symptoms.

ASTER YELLOWS

During the period of bloom to harvest, stunted plants with cupped yellow leaves may be noted. An examination of the flower discloses large green petals while apparent developing berries have elongated to

leaf-like seeds. The fruit never matures, the pulp remains green while the distorted seeds color to a greenish-red (Fig. 57). Quite often, the affected plant dies before the harvest period.

Runner plants from an infected mother plant suddenly wilt and die during July and August. Examination of their root system often discloses a symptom of Aster yellows, flattened, curved root tips.

The symptoms of this virus disease are quite similar for all varieties and in Michigan it has been noted on Catskill, Premier, Red-Glow, Robinson, Sparkle and Surecrop.

LEAF ROLL

Plants affected with this virus display small pale-green leaves supported by long petioles. The margins of the individual leaflets have a definite downward curl from the mid-vein out, especially at the base. A latter symptom is evident when opposite leaflet margins overlap, forming a funnel-shaped tube.

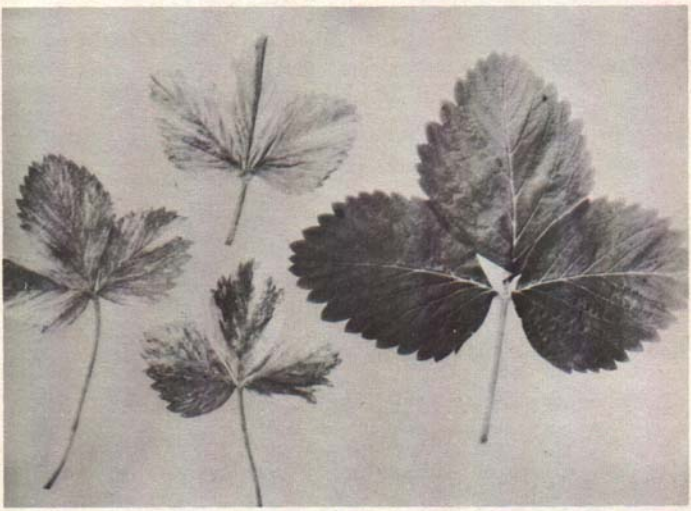
WITCHES BROOM

This virus disease is noted commonly in the Upper Peninsula and the northern half of the Lower Peninsula.

Affected plants have multi-branched crowns with many short, spindly stems and small pale green leaves. The plants have a "bushy"

Fig. 57. Aster yellows, note large green petals, leaf-like seeds, and cupped leaves.

Fig. 58. Witches broom (left) note crowded small leaves on short stems. Right – healthy.



Sec. 59. Left – strawberry leaves showing symptoms of non-infectious variegation or June Yellows. Note the characteristic mottling with yellow and green. Right – healthy leaf.

appearance (Fig. 58). This is enhanced by the fact that runners are very short so that the runner plants root close to the infected mother plant.

VARIEGATION

This disorder, also known as June Yellows or Blakemore Yellows, is not a virus disease. It is a hereditary character found in certain varieties as Blakemore, Climax, Empire and Premier. The leaves are mottled and streaked with yellow or cream and green in the spring (Fig. 59). Symptoms of June Yellows are quite strong at temperatures below 50 degrees F., but disappear within 2 to 3 weeks as temperatures rise above 50 degrees F. Affected plants never recover and are less fruitful than healthy plants. All runner plants which grow from affected plants also have June Yellows.

GLOSSARY

The first 8 terms are not listed alphabetically to facilitate the understanding of their definitions. Those that follow are in alphabetical order.

Fungus—a plant without chlorophyll. The body is made of delicate threads, called hyphae, and reproduces by spores.

Spores—a reproductive body composed of one or more cells, and capable of reproducing the fungus or other lower plant from which it originated.

Fruiting body—A fungous structure that contains or bears spores.

Sporophore—a stalk-like structure on which spores are borne.

Hypha (Plural, **hyphae**)—One of the thread-like parts of the vegetative body, or mycelium, of a fungus.

Mycelium—a mass of hyphae (thread like structures) that forms the body of a fungus.

Sorus—A compact cluster of spores produced in, or on, the host plant by fungi such as the rusts and smuts.

Chlorosis (adjective, **chlorotic**)—yellowing of normally green tissues because of partial failure of chlorophyll to develop.

Necrosis (adjective, **necrotic**)—death or disintegration of cells or tissues.

Acervulus (plural, **ascervuli**)—a shallow saucer-shaped fruiting body that bears spores (conidia).

Aecium (plural, **aecia**)—a cup-shaped spore-producing body developed by certain rust fungi.

Apothecium (plural, **apothecia**)—An open, saucer or disc-shaped fruiting body whose inner surface is lined with asci.

Ascocarp—a fruiting body that contains asci.

Ascus (plural, **asci**)—a sac-like structure in which sexual spores develop.

Ascospores—spores which develop in the asci.

Basidiospore—Small spores borne on a stalk, or basidium, as in the rusts, smuts, and mushrooms.

Basidium (plural, **basidia**)—A club-shaped sporophore, or stalk, on which basidiospores are borne.

Conidium (plural, **conidia**)—spore formed from the end of a special spore-bearing hypha (see above).

Conidiophore—the hypha (thread-like) branch that bears conidia.

Perithecium (plural, **perithecia**)—a flask-like fruiting body containing asci, usually the overwintering stage of certain fungi.

Pycnidiospore—spore produced in a pycnidium.

Pycnidium (plural, **pycnidia**)—flask-like fruiting body containing conidia.

Teliospore—Spore produced by a rust fungi, which upon germination gives rise to a basidium.

Telium (plural, **telia**)—A sorus (cluster) in which teliospores are produced.

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