# A DETAIL LABORATORY AND LIBRARY STUDY

of

# FRUIT DISEASES

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Minor Thesis in Plant Pathology

for

The Degree of Master of Science

-

Submitted

to

Professor H. L. Price

Dean of Agriculture

Virginia Polytechnic Institute

by

R. C. Moore

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APPROVED:

COURSE ADVISOR

APPROVED:

DEAN

A DETAIL LABORATORY AND LIBRARY STUDY OF FRUIT DISEASES.

## Outline

I. Laboratory Studies.

(a) A careful microscopical study of each pathogen and its physical relation to the host.

(b) An accurate description of the disease.

(c) Where feasible, the pathogen isolated into pure culture and infection studies conducted.

II. Library Work consisting of a study of the economic importance of each disease in the United States. Control measures advocated and their relative values in Virginia.

1. Host relationships.
2. Losses and economic importance of each disease.
3. Factors influencing the development of the disease.
4. Control.
5. Important references found.

## Diseases included in the Study

APPLE

1. Scab - Venturia inaequalis (cooke) Aderholt.
2. Bitter Rot - Glomerella cingulata (Stonenson) Spaulding and Schrenk.
3. Fire Blight - Bacillus amylovorus (Burrill) Trevisan.
4. Black Rot - Physalospora malorum (pk.) Shear
5. Cedar Rust - Gymnosporangium juniperi-virginianae (Schweinitz)
6. Blotch - Phyllosticta solitaria (Ellis and Everhart)
7. Mildew - Podosphaera Leucotricha (Ellis and Everhart) Salmon
8. Black Root Rot - Xylaria mali (Fromme)

PEACH

1. Brown Rot - Sclerotinia Fructicola (Winter) Rehm.
2. Scab - Cladosporium carpophilum (Thumen)
3. Bacterial Spot - Bacterium pruni (E. F. Smith)
4. Leaf Curl - Exoascus deformans (Berkley) Fuckel

PLUM

1. Brown Rot - Sclerotinia fructicola (Winter) Rehm.
2. Black Knot - Plowrightia morbosa (Schweinitz) Saccardo.
3. Leaf Spot - Coccomyces Prunophorae (Higgins)

## Diseases Included in the Study

CHERRY

1. Brown Rot - Sclerotinia fructicola (Winter) Rehmi
2. Leaf Spot - Coccomyces hiemalis (Higgins)

GRAPE

Black Rot - Guignardia Bidwellii (Ellis) Viala and Ravaz.

Downy Mildew - Plasmopara Viticola (Berkley & Curtis) Berlese and De Toni

Estimated reduction in yield due to diseases - 1928-29-30.

Percent

| | In Virginia | | | In United States | | |
|---|---|---|---|---|---|---|
| APPLES | 1928 | 1929 | 1930 | 1928 | 1929 | 1930 |
| Scab | 2.5 | 4. | .5 | 9.2 | 8.7 | 6.7 |
| Fire Blight | trace | .5 | .2 | .8 | 1.1 | 1.3 |
| Cedar Rust | 5. | 2.5 | trace | 1.1 | .7 | .4 |
| Bitter Rot | .8 | .5 | " | .6 | .5 | .1 |
| Black Rot | 1. | 1.3 | 1. | .6 | .5 | .2 |
| Blotch | trace | trace | trace | .7 | .7 | .4 |
| PEACH | | | | | | |
| Brown Rot | 13.6 | 5.9 | trace | 10.6 | 4.9 | 1.8 |
| Scab | 2. | 3. | " | 2.6 | 1.2 | .6 |
| Leaf Curl | 2. | 1. | " | 1.7 | 2. | .3 |
| Bacterial Spot | --- | --- | --- | --- | --- | .3 |
| PLUM | | | | | | |
| Brown Rot | 5. | 2.5 | trace | No data | | |
| Other Diseases | 1.5 | 1.5 | " | | | |
| CHERRY | | | | | | |
| Brown Rot | 3.5 | 1.5 | --- | No data | | |
| Leaf Spot | 1. | 1. | --- | | | |
| Other diseases | 1. | 1. | --- | | | |
| GRAPE (east of Great Plain) | | | | | | |
| Black Rot | 10. | 8. | trace | 3. | 6.6 | .8 |
| Other diseases | 1.5 | 1.5 | trace | 7.7 | 1.1 | 1.1 |

ACKNOWLEDGMENT

Appreciation and thanks of the writer are expressed to Dr. S. A. Wingard and Professor A. B. Massey of the Plant Pathology Department for helpful advice in making the studies included in this paper.

April 1932 Robeft C. Moore

## INTRODUCTORY

Studies detailed in this paper are not original research but a summary of the literature with microscopic and cultural studies intended to familiarize the writer with some of the more important fruit diseases. Material where available was brought into the laboratory and microscopic studies made of the host symptoms and causal organisms.

Hesler and Whetzel's "Manual of Fruit Diseases", "The Plant Disease Reporter" and "The Experiment Station Record" were found to be of much value in descriptive and reference work and "The Plant Disease Reporter" was considered authentic for nomenclature of the organisms. The pen and ink drawings accompanying each disease are diagrammatic representations and comparative magnifications are not given.

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## PART I

### Apple Diseases

Over the United States as a whole scab is probably the most serious of all apple diseases. Heavy infections of fire blight take place in some years, while in others losses are small. Bitter Rot is mostly confined to the southern apple belt and to such susceptible varieties as Albermarle Pippin. Black Rot is of relative unimportance in well sprayed and well pruned orchards. Where present the leaf spot form is most serious. Cedar Rust is serious in years of heavy rainfall with winds in May and June in sections where red cedars are numerous in the vicinities of orchards. It is practically confined to the Eastern Half of the U. S. Blotch is one of the most serious diseases in the middle west but is of minor importance in Virginia except on such varieties as Northwestern Greening and Limbertwig, which are not grown extensively here. Mildew is not of economic importance in most sections of the country. In Washington state and other sections and where Jonathan and Spitzenberg are extensively grown it may cause severe damage to one-year twigs, on unsprayed trees. Black root rot is found in the South Atlantic states and a few of the middle west states, but its chief damage has been in the Shenandoah Valley of Virginia, where it is thought to be the most important single cause of death to apple trees. In order of their importance these diseases are probably as follows:

| | |
|---|---|
| Scab | Black Rot |
| Cedar Rust | Bitter Rot |
| Fire Blight | Black Root Rot |
| Blotch | Mildew |

# VIRGINIA APPLE SPRAY PROGRAM

| | NAME | TIME | MATERIAL FOR 100 GALLONS OF SPRAY | PURPOSE |
|---|---|---|---|---|
| 1. | Delayed Dormant | When green can first be seen in blossom buds | Lime-sulphur, 32°Baume, 12 gallons, and 40% nicotine sulphate 1 pint. Oils may be used at 3% strength, and are recommended for scale and red mite control. | Scales<br>Aphids<br>Red Mite |
| 2. | Pink | When the majority of the cluster buds have separated | Lime-sulphur, 32°Baume, 10 quarts. When curculio is present, include lead arsenate 3 pounds | Scab<br>Mildew<br>Leaf Spot<br>Curculio<br>Bud moth |
| 3. | Petal-Fall | When most of the petals have fallen. | Lime-sulphur, 32°Baume, 10 quarts, and lead arsenate, 3 pounds. (If scab is not a serious factor, one of the following fungicides may be substituted for liquid lime-sulphur: Dry Lime-sulphur, calcium sulphide or one of the wettable sulphurs.) | Scab<br>Mildew<br>Leaf spot<br>Codling moth<br>Curculio<br>Leaf roller<br>and other chewing insect |

## VIRGINIA APPLE SPRAY PROGRAM Cont'd

| | NAME | TIME | MATERIAL FOR 100 GALLONS OF SPRAY | PURPOSE |
|---|---|---|---|---|
| 4. | 3-Weeks' Spray | About three or four weeks after the petal-fall stage, or as advised by the Spray Service. | Lime-sulphur, 32°Baume, 10 quarts, and lead arsenate, 3 pounds, or Bordeaux 4-8-100, as advised by Spray Service.[1] (If scab is not a serious factor, one of the following fungicides may be substituted for lime-sulphur: Dry lime-sulphur, calcium sulphide or one of the wettable sulphurs.) | Scab<br>Frog-eye<br>Mildew<br>Blotch<br>Curculio<br>Codling Moth |
| 5. | 5-Weeks' Spray | About five or six weeks after petal-fall stage as advised by Spray Service | Lead arsenate, 3 pounds, in 4-8-100 Bordeaux as advised by Spray Service. | Codling moth and other chewing insects<br>Blotch<br>Bitter Rot |
| 6.[2] | Mid-Summmer Spray | Usually in July as advised by Spray Service. | Lead arsenate, 3 pounds, in 4-8-100 Bordeaux as advised by Spray Service. | Codling moth and other chewing insects<br>Blotch<br>Bitter Rot |

[1] Choice of material in this spray is governed by weather conditions. If it is hot, 85° or over, use Bordeaux as recommended. If it is cool, use a sulphur fungicide. About 6 pounds of hydrated spray lime should be used in each 100 gallon solution whenever lead arsenate is combined with lime-sulphur. Spray lime is preferable.

[2] GROWERS who apply an arsenical spray in July must be prepared to remove any excessive residue that may persist at picking time. SPRAY No. 6 is very important where late worm injury is a factor.

# APPLE SCAB

## HOST

Only the apple is attacked by this organism. There are scabs of pear, peach and other fruits but these are caused by different organisms.

## GEOGRAPHICAL DISTRIBUTION

Apple scab is almost universally found wherever apples are grown, but is likely to be most severe in sections where cool moist weather prevails in the spring. Northern parts of the U. S. seem to be particularly troubled with this disease. It is rare in the irrigated sections of the country, due probably to the fact that moisture is a controlling factor in its development and dissemination.

## ECONOMIC IMPORTANCE

In the more humid sections of the country scab is considered the most serious disease of the apple, due to its omnipresent mature. Proper spraying will hold it in check but it is always a constant menace to fruit growing. Often abundant rainfall, which so greatly favors infection in the spring, will also hinder or prevent timely spraying operations, so necessary to its control. It may readily be seen then that losses may occur even in well-cared-for commercial orchards. Scab attacks the leaves, blossoms and fruit, causing a partial defoliation, a poor set of fruit or a high percentage of low grade fruit at harvest. In years in which conditions are favorable for development of scab losses may run as high as 30% to 35% of the crop in

average commercial plantings and as high as 100% in unsprayed orchards. In general, if a proper spray schedule is adhered to each year, losses should not exceed 3% to 5% of the crop. Perfect control is generally too costly to be warranted.

## Over-wintering Stage

The organism passes the winter as immature perithecia in dried leaves on the ground.

## Method of Dissemination

Rainfall is necessary to ejection of the spores, which drift around in air currents until they alight on the apple tissue and germinate in the presence of water.

## Symptoms

LEAVES. Scab first makes its appearance in the spring as velvety olive brown areas on the upper surface of leaves, generally about the latter part of May. At first these spots are slightly lighter than the normal leaf color, but they soon turn darker and finally almost black. Some of these spots or lesions are circular in shape, while others tend to extend along the leaf veins in an irregular manner diffusing into the surrounding healthy tissue. Lesions may be few and scattered, or so numerous as to converge and cover a large portion of the leaf surface. In some cases the leaf maintains its normal form, while in others the lesions become swollen and puffed on the upper surface with a corresponding concavity on the lower surface. As the season progresses these diseased areas frequently

die, shrink and crack, and where large portions of the leaf are involved they drop prematurely. The velvety appearance is characteristic of scab.

FRUIT. Lesions on the fruit may appear at any time after it is about the size of small marbles as dark spots. These may be almost black from the start, or may be olive brown at first later turning dark. In either case the velvety appearance is characteristic as on the leaves. Spread of the lesion is slower and the line of demarkation between diseased and healthy tissue more pronounced on fruit than on the leaves. With age lesions change considerably. The center becomes bare, brown and corky, while the advancing margin remains black and velvety. On some lesions a white band appears surrounding the black margin. This is the ruptured cuticle ofthe fruit. Several enlarging spots may coalesce to cover a rather large area. This diseased area is dwarfed, and as the surrounding healthy tissue grows, a deformed apple results. Further dwarfing may cause the diseased area to crack open revealing the underlying tissue, and frequently rot organisms may gain entrance through these cracks. Later in the season small lesions may appear on the fruit, popularly termed "pin-head scab". These rarely become bare in the centers, but usually maintain their velvety appearance, and enlarge considerably before there is any rupture of the cuticle. Scab is a superficial disease and effects only the cuticle and upper epidermal cells.

. Blossoms. In years favorable to heavy infection, some of the failures of blossoms to set fruit may be attributed to scab. Lesions may be seen as raised, dark, velvety areas on the pedicel and base of the blossom, which cause the blssom to shrivel and drop. Infection of young twigs occurs on McIntosh but neither blossom nor twig infection is nearly so common as leaf and fruit infection.

## Life History

### Cycle of Infection.

The fungus passes the winter as immature perithecia in dead leaves on the ground. In early spring ascospores mature in these perithecia and during rainy spells just before blooming are forcibly ejected into the air. These ascospores alight on the leaf surface and germinate invading the area just beneath the cuticle. All perithecia do not mature their ascospores at one time, and the spore discharge continues over a period of one to two months. About three weeks after the spore has germinated the spots may be seen on the leaves. Conidiaphores or spore stalks protrude from the lesions and conidia or secondary spores form on these stalks. Conidia are carried by rain and wind to other leaves and fruit and set up secondary infection. Secondary spore infection cycles take place throughout the growing season. Diseased leaves drop to the ground and during the fall and early winter perithecia are formed in the vicinity of the lesions. These perithecia remain immature over winter ready to initiate primary infection the following spring.

## MORPHOLOGY and PATHOGENESIS

### Perfect or Sexual Stage

Perithecium. The organism over-winters on dried leaves which were diseased the year before. Examination of these leaves in the latter part of March will reveal minute, shiny, dome shaped bodies, which project slightly above the surface of the leaf. Some of these may be seen with the naked eye, but they are readily seen with a hand lens. They are found on the uppermost surface of the dried leaves on the ground. A cross section of one of these bodies reveals a jug shaped cavity (Fig 1) which is bounded by an inner wall of thin-walled cells and outer layers of thick-walled cells. Attached to the inner wall are numerous spore bearing sacs (asci). Each ascus contains eight ascospores (Fig 2). These ascospores are 2 celled, one cell being more blunt and narrow than the other. At maturity they are olive brown in color. All the spores within a perithecium ripen at approximately the same time, however all perithecia do not mature at the same time. The first soaking rain after maturity of the ascospores gorges the perithecium with water, and sets up a pressure, which bursts the covering, leaving a neck like opening (the ostiolum). The free end of each ascus opens out and ascospores are forcibly ejected into the air clear of the dried leaf surface. Within five minutes after dry leaves become wet, spore ejection may take place, and it has been estimated that under a large tree, where perithecia are fairly numerous, it is possible for 8 million spores to be ejected during forty-five

minutes of rainfall. Once discharge of spores from a perithecium has started, and if wet weather continues this particular perithecium will exhaust its entire contents within twenty four hours.

Dissemination. Ascospores are light and float in air currents like dust particles. A relatively small number of them alight on moist surfaces of leaves, blossoms or young fruits, and germinate in the water left by the rain. A period of damp weather following the rain, during which the tree remains wet for several hours, is ideal for infection. Showers late in the evening, leaving the trees wet over night are quite common in the spring and present very favorable conditions for germination of the spores.

Germination. The narrower, more blunt cell of the two-celled ascospore sends out a germ tube into the leaf tissue puncturing the outer cuticle and extending between the cuticle and epidermal cells, (Fig. 3).

Incubation. After spore germination a period of approximately three weeks elapses, during which the germ tube branches out into mycelium and the mycelium occupies the space between the cuticle and epidermis. Hyphae (or strands) of the mycelium become closely packed between the host cells, and form division walls. These division walls in the hyphal strands break the mass of mycelium into one or more layers of densely packed rounded cells (the stroma).

## Secondary Stage- Conidiaphores

A pressure exerted by these cells in the stroma finally pries loose the cuticle of the apple tissue. In the open ruptured area thus formed, short spore stalks (conidiaphores) arise from the stromatic cells. Conidiaphores are reddish-brown, somewhat ovate and sac-like at the upper or free end. They have hyaline inner walls, which protrude from the outer wall at the free end of the conidiaphore. As this inner wall extends itself a cross wallis gradually formed at the point of protrusion. Part of the contents of the conidiaphore passes into this newly formed area, which now becomes a conidium or secondary assexual spore. See Fig. 4.

Conidia. Conidia are at first rounded and hyaline (or clear). With maturity they become more lanceolate, reddish brown and sac-like at their free ends. When ripened these spores break loose from their conidiaphores and are carried by rain and wind to other surfaces of leaves and fruit, where under favorable moisture conditions, they germinate, setting up secondary infection. Formation of the reddish brown conidiaphores and conidia impart the characteristic olivaceous velvety appearance to the lesion. This process of secondary summer spore formation may be repeated several times during the growing season, when rainfall is sufficient for dissemination. After dissemination of spores, the conidiaphores turn dark and finally black. Some lesions on the fruit are black from the outset due to the darker color of the stromatic cells.

Formation of Perithecium. Diseased leaves falling to the ground carry the fungus mycelium which carries over winter. Mycelial strands spread out from the diseased areas, and at irregular intervals form tangles. This tangling is brought about by a crossing and recrossing of the hyphal strands to form a knot. Cross walls are formedat the intersections and the knot becomes a mass of cells. Cell walls in the interior break down and produce a cavity, containing a protoplasmic mass. Probably this stage is attained by late fall and winter is passed in this condition. In late winter and early spring the inner cells of the perithecium gradually give rise to asci, within which ascospores are formed. By the time blossom buds have reached the pink stage, ascospores are usually mature and ready to be ejected to initiate primary infection.

## CONTROL

Control measures are based upon the time of spore ejections and since spore ejections correspond somewhat to seasonal development of the tree, earlier sprays are recommended at these seasonal stages of the tree. The following sprays are usually recommended for scab control:

(1) Pink spray- when the individual blossoms of the cluster have separated and show pink.

(2) Calyx spray- when blossom petals are about seven-eighths fallen.

(3) Three-weeks spray- Three weeks after the calyx spray.

Standard 32 degree Baume' test Concentrated lime-sulphur is used in these three sprays at the rate of one part to forty parts of water. Bordeaux mixture 4 pounds of copper sulphate and 8 pounds of lime to 100 gallons of water is used five weeks after petal-fall and again about July 10 for control of secondary infections. Schneiderhan (3) found that usually the pink and calyx sprays together account for approximately 80% of the control secured by the spray schedule. A spray 10 days after petal fall (recently replaced by the 3 weeks spray) accounted for 10% control; the 5 weeks spray for 5%; and all other sprays for 5%.

Variations in season and location cause variation in effectiveness of the individual sprays, but it is generally conceded that the Pink, Calyx and Three Weeks sprays are most important. Such materials as Calcium Mono-sulphide and Dry Lime-Sulphur have proved effective in scab control, but none have supplanted the Liquid Lime-sulphur-Bordeaux schedule. Dusting has given good results in some states under some conditions, but is not recommended as a general practice in Virginia. Some investigations have found control value in the delayed dormant and pre-pink sprays, but Schneiderham (3) could assign no value to these sprays for scab control.

## VARIETAL RESISTANCE

While some varieties show a resistance to scab, this cannot be relied upon too much in planning control measures, because in some years, even the resistant varieties may become severely infected. Under average conditions the following varieties are listed as susceptible:

Winesap, Rome Beauty, Stayman, Delicious, Gano, Ben Davis, Black Twig, Lowry, Virginia Beauty, King David and Early Harvest. Less susceptible varieties are:- York Imperial, Grimes Golden, Jonathan, Oldenburg and Wealthy.

## REFERENCES

The following experimental work carried on in different sections shows some results secured under a variety of conditions:-

In Virginia Schneiderhan (3) found that none of the sprays prior to the Pink spray gave any control of scab in the years 1922 to 1925 inclusive. At least there was not enough control to justify the added expense of application. He found that where there was a very early spore discharge these early spores seldom produced infection.

Sherbakoff and Andes (5) found in Tennessee that in a rainy season, every one of the seven sprays of the schedule was important for control. It was found that the delayed dormant, pre-pink and pink sprays may greatly influence the effectiveness of the schedule, and if the weather is rainy earlier in the season, spraying should start earlier than the pink spray, if effective control is to be secured.

In Ohio, May (8) points out that three factors determining timeliness of spraying are (1) development stage of the tree, (2) development of the scab fungus (3) relation of weather conditions to the fungus infection. He states that the time to apply scab sprays cannot be judged by condition of the tree alone. The other two factors must also be taken into consideration. In the period

1924 to 1928, it has sometimes been the delayed dormant, some times the pre-pink, and again the pink, and in at least one case an application in the bloom stage, which has been most important for control.

Martin found that in New Jersey total rainfall is not so important as rainfall in relation to development of the tree. Any conditions, where moisture is retained on the tree after a rain, will increase severity of infection. He also showed the need of spraying before rather than after rainfall.

Martin and Clark (10) found with Stayman and Winesap varieties, pre-blossom sprays are of extreme importance in scab control in New Jersey. The delayed dormant spray was of greater value than the pre-pink. No difference was observed in the value of delayed dormant pink or calyx sprays for control of scab.

In Ohio, Stover and Johnson found that lime-sulphur and Bordeaux mixture both gave good control; and that the pink spray was the most important single spray. The pre-pink spray had practically no value.

Folsom (13) found in Maine that after primary infection had taken place later spraying checked the spread of scab. The amount of scab on the fruit was determined by the efficiency of the current seasons fungicidal schedule, rather than by the amount of scab present the year before. Liquid lime sulphur cost slightly less per tree than dry lime sulphur. The extra control secured by a pre-pink spray did not justify its cost of application. New

scab spots develop in storage, which were not apparent at packing time. Sprayed fruit developed less scab in storage than did unsprayed fruit.

Folsom and Ayers (12) in Maine found that ascospore development varied with season and location, hence financial returns from a schedule or particular spray will also vary. Variety plays an important part. Location of trees in the orchard was more important to variation in degree of infection than was vigor induced by nitrogen fertilization. Spraying the foliage with copper sulphate in the fall did not appreciably decrease ascospore development the following spring.

In Wisconsin, Keitt and Wilson (7) report that leaves falling early in the fall, develop ascospores sooner the following spring than leaves hanging on the trees later in the fall.

APPLE SCAB - FIG. A.

1. - Two different forms of scab lesions on apple leaves. Those on leaf to right are irregular, fusing with surrounding tissue. Those on left leaf are sharply defined.

2. - Buckling of leaf surface by scab lesions.

3. - Lesions on young fruit, showing dwarfing effect on tissue. White margins of ruptured cuticle may be seen on some.

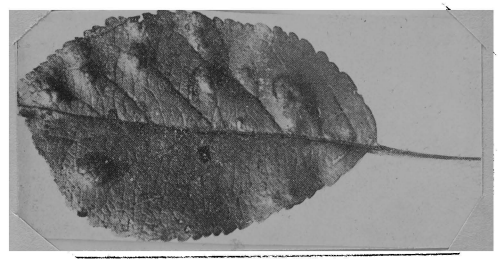
4. - Severely scabbed fruit on which growth was checked, causing cracks.

5. - More mature fruit with scab lesions. Scab is more noticeable on green or yellow varieties.

6. - Almost mature apple upon which little dwarfing took place. Growth of the fruit tissue has cracked and peeled up some of the corky tissue.

APPLE SCAB - FIG. A.

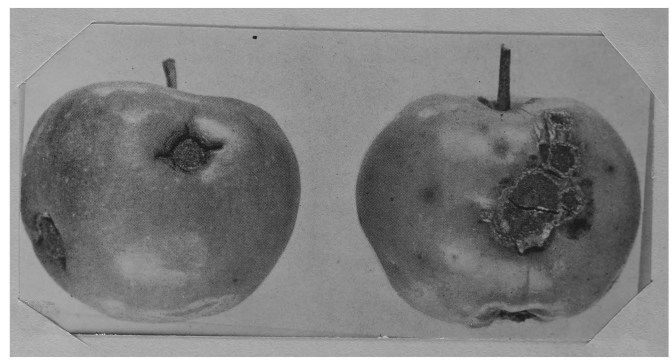
1.



2.

3.

4.



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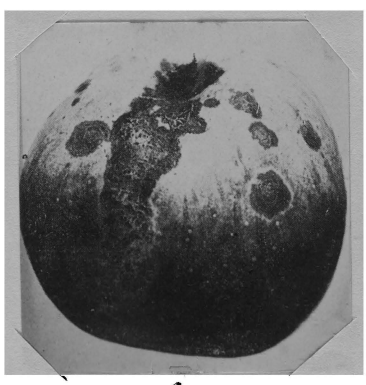

6.

Fig. 1- Sexual winter stage of apple scab, showing a perithecium, with asci or spore sacs, each containing 8 ascospores. Also pseudo cell construction with outer layer of thick walled cells, and inner layer of thin walled cells.

Fig. 2- Individual ascus with mature ascospores.

Fig. 3- Germinating ascospore or surface of a leaf.

Fig. 4- Stroma from which spore stalks (conidiaphores) arise bearing the conidia or summer spore of scab.

# APPLE SCAB

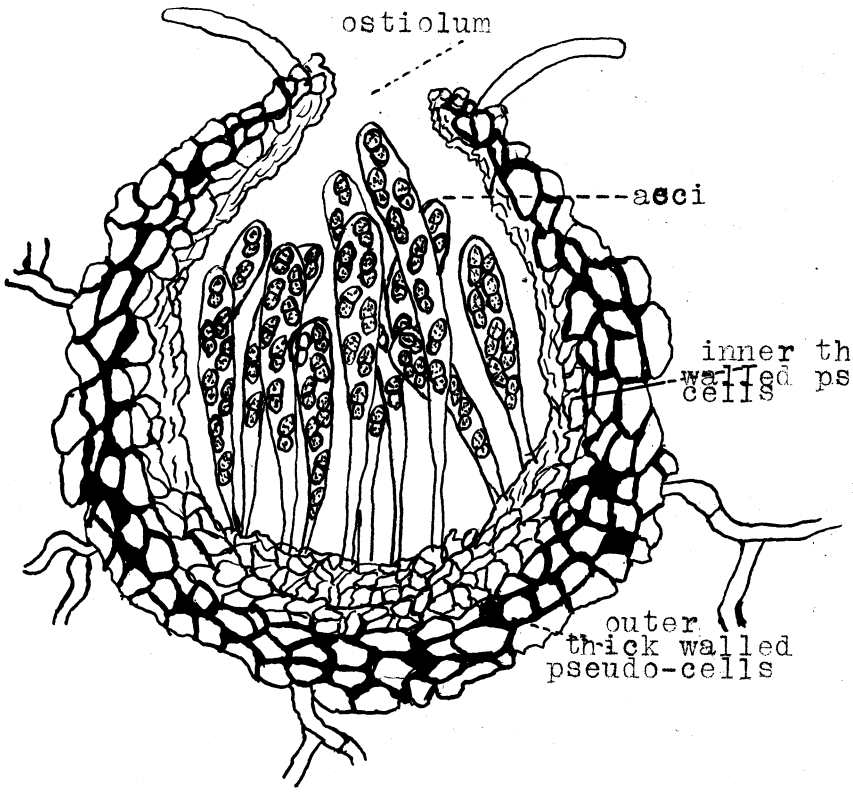


FIG. 1

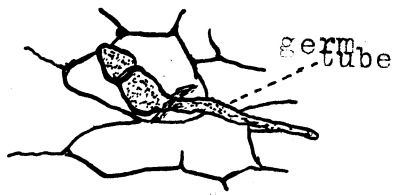


FIG. 3

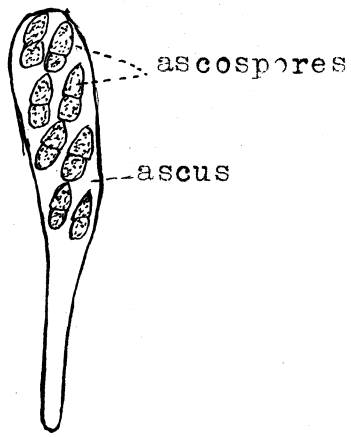


FIG. 2

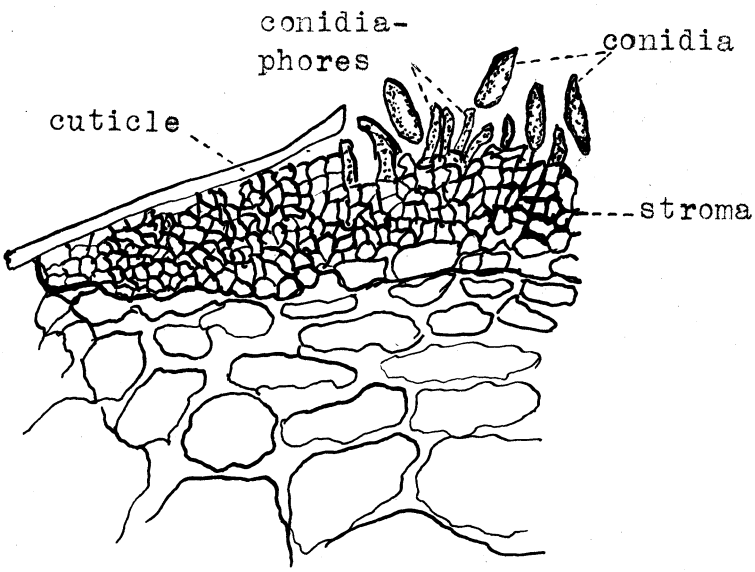


FIG. 4

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## FIRE BLIGHT

### History

This organism, while being known for a long time, was first described by T. J. Burrill in 1883. It was believed to have originated on the wild crab apple and hawthorn, and was first discovered on the cultivated apple about 1780 in the Hudson River Highlands by William Deming, an orchardist. It worked Westward with the plantings of the settlers, being now found throughout the U. S. and Canada.

### Hosts

Apple, pear, quince, hawthorn, mountain ash, Burbank plum, cultivated rose, spirea, and japanese quince.

### Economic Importance

It may be classed as one of the most important apple diseases, because of its epidemic nature, difficulty of control when it has once obtained a start, and ease with which it spreads.

### Dissemination

Heretofore it has been thought that insects were the most important agents in spreading the bacteria, but recent observations indicate that splashing of raindrops account for most of the spread, and that insects play a minor role in this matter. Healthy tissue is easily invaded through the stomata without artificial means.

## Symptons

Blossoms: Blossoms are usually the first portions of the tree to exhibit blight symptons in spring. Clusters with their surrounding leaf tufts appear brown and wilted and later turn black (Fig. B). From blossoms the disease extends into spur and may even extend to larger twigs and branches causing characteristic browning and wilting.

Twigs: The affected twig first wilts, then turns brown and finally black (Fig. B). The first browning resembles partially burned green brush. There may or may not be a sticky milky substance oozing from the lenticels on the surface of the twig in small drops. If this ooze is present, it later oxidizes into an amber yellow color, becoming a brown and finally black gum. Newly affected shoots frequently show a reddish-brown discoloration at the tips. Finally the shoot or twig turns black and shrivels becoming brittle and hard, with its tip inclined to curl. It retains its shrivelled blackened foliage and gives the appearance of being blasted by fire. Hence the name "fire blight."

With favorable conditions of succulency of the bark, the disease may spread down through twigs into limbs and some times into the trunk of the tree. Here it usually spreads out radially around the base of the shoot to form a cankered area. The canker first shows as a circular darkened area, which later becomes sunken and cracks away from the surrounding healthy tissue (Fig. D). Any checking of moisture tends to arrest the

spread of the organism and form a callus around the edge of the canker, thus forming a sharp line of demarkation between diseased and healthy parts. Later the diseased bark sloughs away exposing bare wood with its margin of callus. Fig. D shows a one year old canker surrounding a blighted twig, through which the organism gained entrance.

Fruit: The organism enters green fruit generally through its pedicel, coming first down the blighted shoot (Fig. B). The characteristic browning shrivelling and later blackening of the tissue is apparent here as in the shoot. Milky drops may ooze from the lenticels of the green fruit also. As the young apple becomes involved, it shrivels and turns black, becoming mummified and hanging to the tree from one season to the next.

## LIFE HISTORY

The organism causing this disease is a minute bacterium (Fig. 5) discernible only under the highest power microscope. These bacteria multiply by simple division. In winter they collect into cysts (Fig. 6) along the margin of cankers in the living bark, and pass the winter in an inactive state. Those are called "hold over" cankers. In spring activity commences, the bacteria multiply rapidly, and there is a pressure exerted which forces the bacteria out through lenticels of the bark.

Multiplication is very rapid in early stages. Each bacterium forms a cross wall at its mid point and divides into two parts. Each of these parts grows to a normal sized bacterium and in turn divides. The milky ooze is a mixture of decomposed tissues formed in the metabolism of the bacteria with cell sap of the tree. Raindrops splash bacteria to open blossoms, to twigs and to shoots. In the blossom bacteria quickly multiply (Fig.7) and extend down the stems into the cluster, causing characteristic browning and wilting. Bees may carry bacteria from flower to flower.

H. R. Rosen (1) has found that heavy blossom infection took place, when there was no noticeable ooze from the cankers at blossoming time and that infection of the young shoots may take place without the aid of a mechanical injury.

In the tender tissue of the shoots the bacteria multiply rapidly and work their way down the shoot. They may spread out in the bark at the base of the shoot or may be checked in the shoot. Spreading out in the surrounding bark tissue they break down the cell walls dissolve the cellelular tissue and cause a collapse which makes the sunken characteristic canker. An abundance of moisture with succulent growth favors spread of the bacteria, while dry weather, which tends to toughen the tissue, checks its spread. As this checking takes place bacteria gather at the margin of the cankers and become inactive. During the fall they gather into cysts (Fig. 6) and remain in this condition over winter ready to commence activity in the spring.

## Control

The control method long recommended has been that of cutting out cankered tissue in the dormant season. Cankered twigs are removed, the cuts being made well below the actual cankered area. Cankers on larger limbs are usually chiselled out including a layer of healthy tissue to insure the removal of all infected tissue. After each cut tools are dipped into a 1 to 1000 bichloride of mercury solution for disinfection. Removal of pears, wild crab apples and hawthorns from the vicinity of the orchard is also recommended. The above method is tedious and costly and not likely to be followed carefully by the average grower.

McClintock and Fackler (3) have found that a canker treatment has been effective in at least greatly reducing the amount of blight. This does not necessitate the cutting out of cankers. A solution is applied to the canker with a brush before growth starts in the spring, which penetrates the canker tissue and kills the bacteria without injuring the wood tissue to any great extent. Directions for this solution follow:- Add three ozs. of concentrated hydrochloric acid to one quart of hot water in an enamel kettle. In this liquid dissolve 9 pounds of dry zinc chloride powder, heating if necessary to insure dissolving. Cool the mixture and pour it into 7 pints of denatured alcohol, thoroughly mixing. Keep tightly stopped until ready for use.

The sooner the canker is detected and painted the easier the killing and quicker the healing. The entire canker including the margin should be thoroughly covered with the solution. Blighted spurs and twigs also may be effectively treated.

About three or four weeks later another inspection should be made to check up results, and any new cankers should be treated at this time.

This method was found to be more successful than the old surgical treatment.

McClintock (5) states that resistant varieties of pears are generally not very desirable commercially. Pears generally harbor bacteria over winter more than apples, although yellow transparent is worse in this respect than most apples. Rain is more often the disseminating agent for primary infection than insects. Sucking insects are absent when most of the infection takes place.

Brooks (6) found that wild host plants are of little importance in the over-wintering of the bacteria. The source of primary infection is the exudate from blighted twigs and cankers in the apparently healthy tissue surrounding or adjacent to the dead area. Bacteria ooze at a temperature of 65 to 85°F and humidity above 80%. At least 90% of twig infection was traced to water-borne bacteria from hold-over cankers and blighted

twigs. Attempts to secure stomatal infection failed. Asphids were found on the exudate from cankers and pollination insects were believed to carry the organism. Tillage and fertility management, which tend to promote excessive vegetative vigor are conducive to severe blight.

Keitt and Miller (7) in Wisconsin found the first bacterial exudate from cankers and twigs on April 19, and the first blighted flowers before the blossom opened. Rain was considered the most important agent for blossom infection, while aphid and leaf hoppers as well as rain carried the innoculum for twig infection.

In Kentucky (8) almost complete control of blight on a block of Transparent apples and a neighboring block of pears was secured by carefully removing cankers from the pear trees.

FIRE BLIGHT OF APPLE - FIGS. B - C - D.

Fig. B - New shoot blighted, browned and curled in characteristic manner. The cluster of terminal fruits was probably infected through contact of their pedicels with the diseased shoots.

Fig. C - Blossoms and surrounding leaf clusters browned and deadened by blossom infection.

Fig. D.- Canker formed at base of a shoot. Dead shoot broken off. The crack formed around the margin of the oval shaped canker marks the point at which bacterial spread was checked. The surface of the canker is sunken below that of the surrounding tissue.

Fig. 5- Bacillus amylovorus, the fire blight organism, showing the hain-like appendage of locomotion-flagella.

Fig. 6- Winter stage of bacteria, showing a cyst in the serve tube adjacent to the canker.

Fig. 7- Bacteria mass in the stomatal cavity after infection of host tissue in the spring.

Fig. 8- Bacteria filling intercellular spaces and destroying adjacent cells.

Fig. 9- Further destruction of adjacent cells.

FIRE BLIGHT

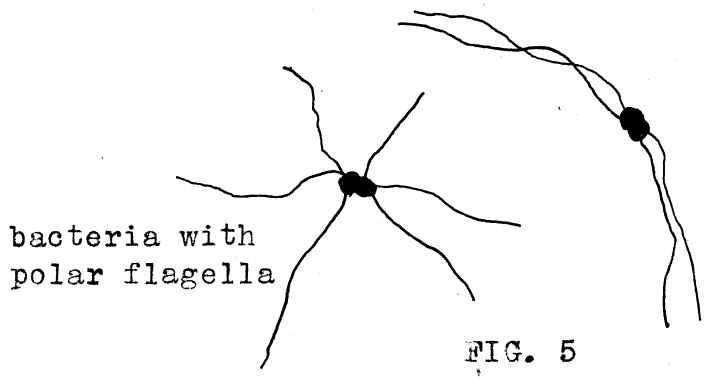


FIG. 5

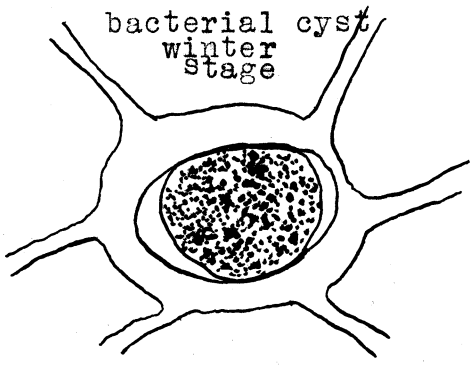


FIG. 6

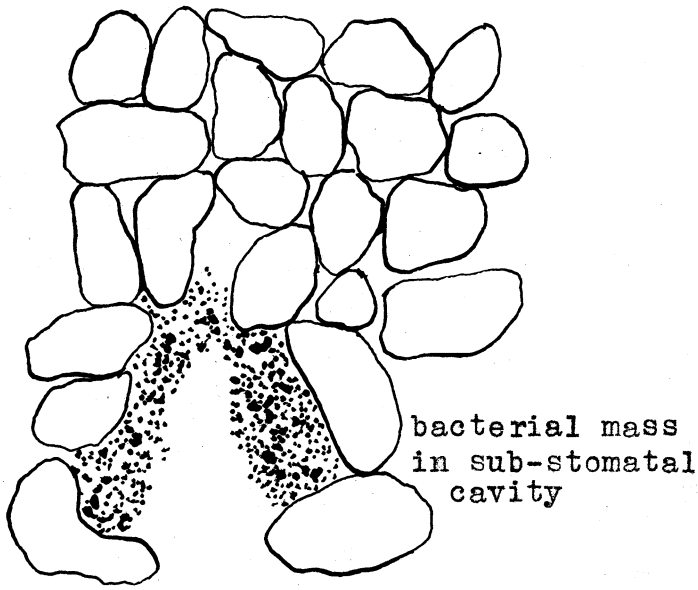


FIG. 7

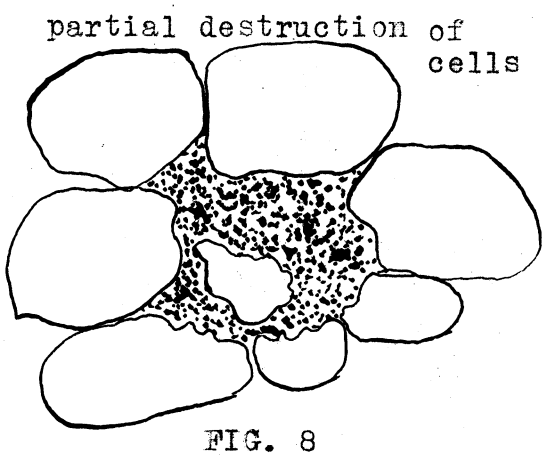


FIG. 8

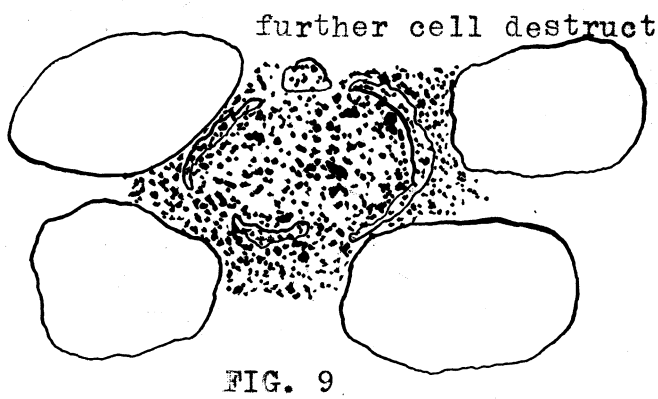


FIG. 9

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## BITTER ROT OF APPLES

### History

This disease first made its appearance in North Carolina in 1867 but was not specifically described until 1874 by Berkeley and Curtis.

### Host

A number of hosts are affected, including apple, pear, quince, and grape, although there is seldom an exchange from one fruit to another.

### Geographical Distribution

While this disease is found in many apple sections its economic range is confined to the eastern half of the U.S. and it occasions the greatest loss in the Southern part of the apple belt, which includes the area from Northern West Virginia to North Carolina and extending westward to southern Kansas and Oklahoma. The fact that it is favored by hot moist weather probably limits its range somewhat. In Virginia it is especially troublesome in the Piedmont section, where the Albermarle Pippin is commercially grown, this variety being notoriously susceptible.

### ECONOMIC IMPORTANCE

In past years the ravages of this disease have been severe, especially in years of heavy summer rainfall with high temperatures, whole crops being destroyed in a short period of time in the summer. With the advent of Bordeaux mixture and the

use of more efficient spraying machinery this disease has been brought under control so that at the present time there is only an occasional orchard in which losses are great.

The grower, whose orchard contains susceptible varieties must be ever watchful, to keep the disease under control. The chief loss is in the rotting of the fruit, although some varieties tend to become infected with limb and twig cankers.

Over-wintering Stage

This fungus organism is carried over winter as mycelium in mummied fruit and limb cankers on the tree, the most common type being mummies hanging on the limbs of the tree.

Symptoms

The first appearance of the disease is generally about the first part of July, although this may vary from season to season with temperature and rainfall, with age of fruit, and with the time of maturity of the spores in the carry over mummies. The first sign of the rot on the fruit is a light brown discoloration under the skin of the apple. These spots are small at first but soon grow larger being circular in outline. When these spots are numerous on the apple they do not become very large, but when only a few spots are present they spread rapidly, After the spot has attained a diameter of about 18 inches, the surface appears distinctly sunken, and the border is sharply defined from the surrounding healthy tissue. When about ½" in diameter, small black raised dots appear at more or less regular

intervals in a ring on the surface of the sunken area. Later these dots break open and allow pink masses of spores to ooze out in coils.

As the rotted spot increases in size more dots are formedin a ring and exude their spores, until 6 or 8 or even more concentric rings are formed. These concentric rings of pink spore masses on the sunken surface of the black rot spot are characteristic symptoms of the disease. Most perfect rings are formed when the spot enlarges rapidly.

The first rain dissolves and washes away the spore masses, leaving the black pustules open and empty. The pink color disappears with the spores and the spot appears black or dark brown. Beneath the surface of the spot the tissue is found to be brown and rotted in the shape of a cone, the apex of which points toward the core of the apple. The larger the diameter of the spot the greater is the depth of the rotted area. This rotted tissue usually has a bitter taste, hence the name "bitter rot". The sinking of the surface of the spot is caused by a shrinking of the underlying tissue, brought about by the collapse of the cells. The size of the diseased areas increases rapidly and may involve practically the whole fruit. There are generally small patches of healthy tissue interspersed among the rotted areas, which hold out a long time Rotted fruit generally falls to the ground, its ripening processes having been hastened. However some fruits become dry and cling to the twigs, forming the mummies, in which the mycelium lives over winter.

## Cankers

Cankers of bitter rot may be found on limbs of different sizes from small spurs to limbs 3 or 4 inches in diameter, but not on the main trunk. These cankers are rounded, oval shaped to almost oblong, sooty black sunken areas from one to several inches long, having more or less ragged edges. The entire bark is killed and appears cracked and fissured and in some instances broken away. Transverse cracks are very marked in some cankers. As the cells collapse there is a sinking of the bark, which adheres to the under lying wood. Around the margin the callus layer usually forms. The wood beneath the diseased bark is discolored to a considerable depth toward the center of the limb. These cankers are not noticeable for about two years after infection. Cankers are not as commonly found as fruit rot.

## Life History

### Cycle of Infection:

The fungus over-winters as mycelium chiefly in mummied fruit hanging to the trees. In June when weather has become hot, pustules (ascervuli) are formed from which sticky masses of pink spores ooze following rainy weather. Subsequent rains dissolve the mass and wash spores to the underlying fruit, producing infection through the natural openings of the fruit. Within a few days ascervuli are formed in the rotted area on the fruit. Within the ascervuli conidiaphores and conidia are formed and ooze out on the surface of the rotted spot. Conidia are washed to other fruits and cause secondary infection. Some apples drop and others shrivel into mummies. Hot moist weather in

July and August is favorable to production of several cycles of this secondary infection, occuring only a few days apart, and spreading the spores over large portions of the tree. Some infected apples drop and others shrivel and cling to the tree. Mummies clinging to the tree carry the mycelium over winter.

## Morphology

This fungus organism passes the winter as mycelium in mummied fruit, in infected twigs and in cankers on smaller limbs. In the early part of June when the weather has become hot, small black dots appear on the surface of the mummies or twigs. In the presence of moisture, coils of pink to yellowish spore masses ooze from these dots (or pustules), and form an irregular mass around the opening of the pustule. This mass is a sticky medium in which are imbedded large numbers of conidia. As the air dries the mass dries also, cementing itself to the surface of the mummy. Further rains, dissolve the mass, separate out the spores and splash them in a cone shaped shower to the apple below. About 5 to 10 hours of moisture following the spore discharge is necessary for germination of these spores. A hot muggy night following a shower is ideal, although heavy dew is sometimes sufficient. Fig. 10 shows the spores as they are discharged. Germination takes place by the formation of a cross wall in the middle of the spore. The larger spores usually send out two germ tubes, one from each half of the spore. Smaller spores may send out only one germ tube from one half of the spore. Some of these germ tubes form appresoria or dark rounded bodies at their ends.(Fig 11)

The germ tube is able to penetrate the sound tissue of the fruit, probably through the stomates. Mycelium quickly spreads between the cells below the epidermis absorbing its food material from the cell contents. The cells turn brown and finally collapse producing the sunken appearance. The hyphae threads grow equally in all directions from the point of entrance, thus forming a circular spot as the cells are killed. Hyphal threads finally form themselves into mass, which constitutes the fruiting body. In these masses the hyphae are arranged parallel to each other and closely packed with the apex of each hypha toward the surface of the apple (see Fig. 12). These hyphae from the conidiaphores or spore stalks upon which the spores are formed. The general arrangement of the hyphae mass is cone shaped with the apex near the surface. This mass has now become the fruiting body called the acervulum, which appears as the black dot or postule on the surface of the rotted spot. Large numbers of these are found on the surface of a single spot. Pressure exerted by growth of the spore stalks ruptures the pustule; the ends of the spore stalks protrude; and spores are formed on these projecting stalks. The spores are formed from the conidiaphores by a constriction of the side walls, to form a cross wall, which becomes the dividing line between the newly formed spore and the conidiaspore. As mentioned before these spores are imbedded in a pink sticky substance and remain on the surface as a globular mass, until washed away by rain (see Fig. E).

Each spore stalk is capable of producing one spore after another, by the constriction process. Rains wash these spores to other fruits, where they set up secondary infections. From these secondary infections spores arise which carry infection still farther. The number of cycles depends on the temperature and moisture. During hot damp weather sometimes encountered in July and August only a few days intervene between infection of an apple and the formation of new spores which spread infection, so that the spread is very rapid. However, cool dry weather may entirely check its spread. Bitter Rot is not a true rot, but rather the organism acts as a preservative of the tissue. If there is no infection from other fungi, the apple tissue may dry and shrivel into a hardened mummy, in which the hyphae threads remain viable for as long as two years or more. These mummies are the chief carry over agents, which produce the primary infection under favorable conditions the following year.

Some varieties, such as Smokehouse are susceptible to canker formation. Spores are washed to an injured portion of bark or to twigs upon which some injury has been made. Here they germinate forming the mycellium, which carries the disease over winter. The following year these cankers may form spore masses which account for a small portion of the primary infection. Cankers are not numberous in Virginia and are not considered very important as a source of infection, mummies being the chief source.

Hurt & Schneiderhan (1) have found that the stem of the mummied fruit as well as the mummy itself may cause primary infection.

There is a perfect or sexual stage of the bitter rot organism, which, however does not play an important part in the life history. Perithecia are not commonly found in nature. There is formed by a close intertwining and matting of the mycelium a black, hard, carbonaceous nodule. In this nodule the perithecia are imbedded. There may be one or many in each nodule. If only one is present, it is globular, while, if several are formed in the same nodule, they are irregular in shape. Within the perithecium numerous asci are developed in each one of which are eight ascospores, usually arranged in pairs, see Fig. 14 and 15. Ascospores resemble the conidia very closely in appearance and size and germinate in the same manner.

## Control

Bitter rot control is a comparatively easy matter under ordinary conditions. Two Bordeaux mixture sprays are applied; the first one five weeks after petal fall and the second the midsummer spray called for in the spray calendar. However, when there has been a serious outbreak on susceptible varieties it may be necessary to supplement this with removal of all mummied fruit in the fall or winter; or all infected fruit in the summer; or both. In mummy removal it is necessary to remove the stem with the mummy. Once the disease has been brought under control, thorough spraying will hold it in check.

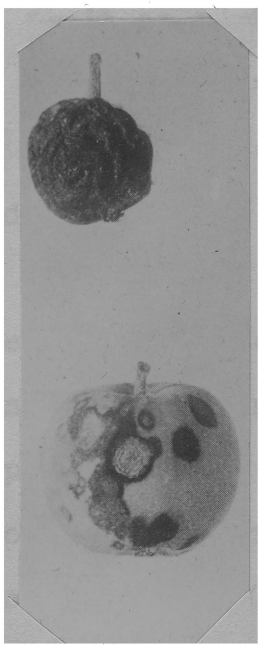
BITTER ROT OF APPLE - FIG. E.

1. - Bitter rot mummy, the source of primary infection. The lower fruit is infected, showing concentric white rings of the fruiting bodies.

2. - Immature fruits badly infected, showing sunken concentric rotted areas.

3. - Close-up of bitter rot lesions showing white tufts of fruiting bodies. Single spot in the center and a cluster of spots on the fruit on the right. The apple on the extreme left shows small spots which have not enlarged much yet.

BITTER ROT OF APPLE - FIG. E.



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Regulatory measures in regard to arsenical residue on fruit at picking time have complicated the control on susceptible varieties. It may be necessary in some cases to apply later sprays and wash the fruit at packing time. Bordeaux mixture 4-8-100 applied 5 to 6 weeks after petal fall protects the fruit against earlier infection, but where bitter rot has been prevalent it is necessary to apply an additional spray from the middle to the last of July. Both of these sprays arecombined with lead arsenate for control of late broods of codling moth, and where the last spray is applied the fruit usually must be washed at picking time.

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Fig. 10- Conidia or summer spores of bitter rot.

Fig. 11- Germination of conidia, showing the formation in some cases of haustoria or feeding bodies.

Fig. 12- A bitter rot pustule-ascervulum-on surface of the fruit. In it may be seen the conidia on their conidiaphores.

Fig. 13- Section of an ascervulum on a canker, bearing conidia.

Fig. 14- Sexual stage- Diagrametic section showing perithecium with asci and ascospores. This stage is not commonly found in nature.

Fig. 15- Single ascus with its eight ascospores.

BITTER ROT OF APPLE

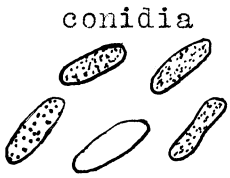



FIG. 10

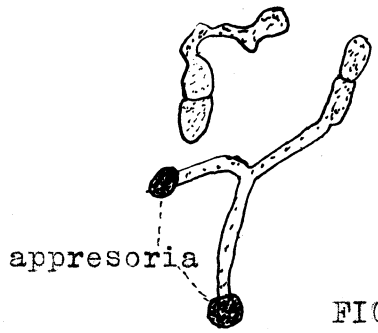
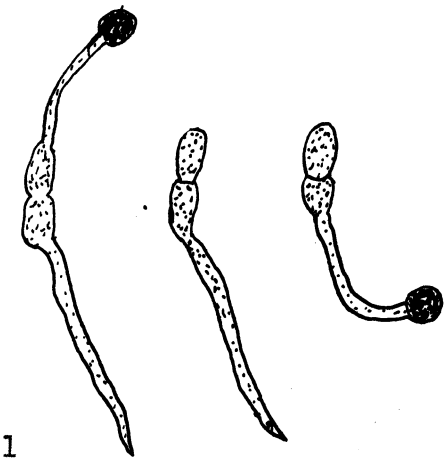



FIG. 11



conidia germinating

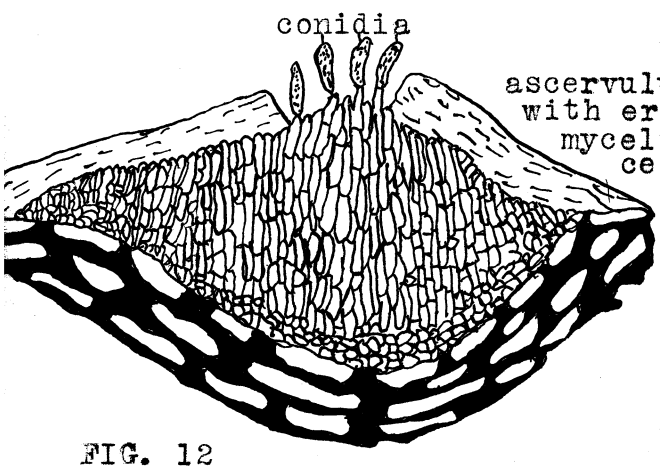



FIG. 12

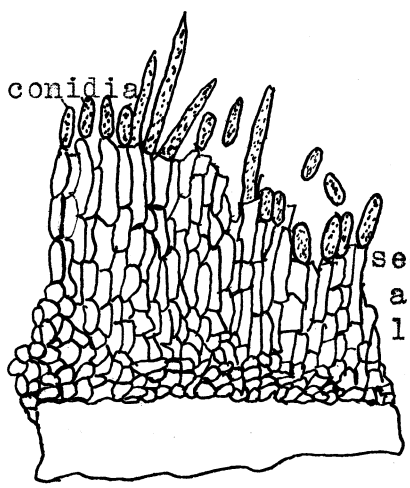



FIG. 13

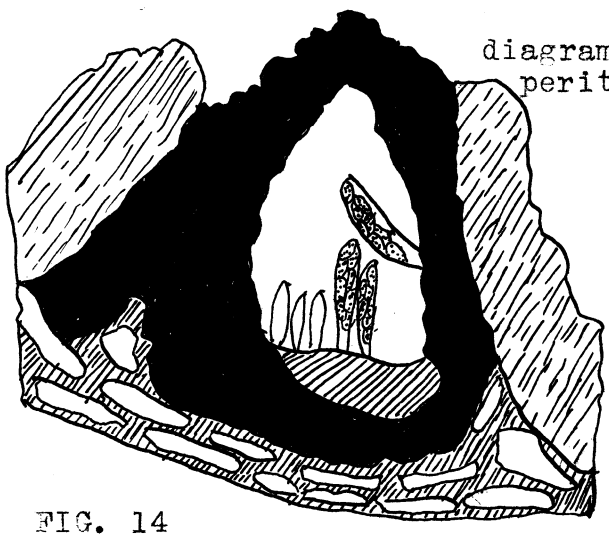



FIG. 14

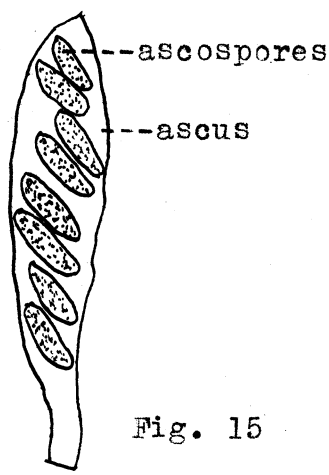



Fig. 15

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## BLACK ROT OF APPLE

### History

This disease has been known for a long time, especially in older orchards of New York. The disease was formerly known as Sphaeropsis Malorum, later as Physalospora Cydoniae and at present as Physalospora malorum.

### Host

Injury by this particular fungus organism is not confined to pomaceous fruits, such as apple, pear and quince, but is also found on a number of other plants such as alder, ash, basswood, dogwood, elder, hawthorn, lilac, maple, oak, pine, rose, sumac, and others.

### Geographical Distribution

This disease, while found in most parts of the world, is most injurious in North America. In western New York and states bordering the Great Lakes the canker form is most prevalent, but in the New England and Middle Atlantic states and the Ozark regions fruit rot and leaf spot forms are most common.

### Economic Importance

In Virginia leaf spot is the only form of the disease of economic importance, although in years of severe codling moth injury, fruit rot may be associated with injury from this insect. Cankers found on older trees, sometimes take a heavy toll. In past years there has been a gradual annual loss from dying of branches and limbs, and a lack of potentiality for yields. In cases of fruit rotting, the whole apple is involved. "Frog-eye"

spots on the leaves interfere with photosynthetic processes, and if spots are numerous, they may coalesce to involve a large portion of the leaf. Such leaves drop prematurely. This disease, throughout the entire country is classed as one of the most important diseases, although in the well cared for Virginia commercial orchard it is of minor importance.

Over-wintering Stage

This fungus passes the winter as mycelium and pycnidia in cankers on twigs or limbs.

Symptoms

Leaves: Soon after the leaves come out in spring, spots appear on the upper surfaces. At first the spots are as minute purple specks, which soon enlarge to about 1/8" in diameter. As tissue in the center of the spot becomes killed, it turns a yellowish brown circular, and has a distinct margin, (Fig. F) The margin becomes raised and the infected area sunken. Further-growth of the spot causes circular growth rings within the dead area. The center of the spot becomes grayish brown, and the general appearance is that of a frog's eye, hence the name. Secondary growths of the spot may produce a lobed effect. When spots are numerous they may coalesce to form a large deadened area, in which circular outlines of the individual spots remain more or less distinct.

Cankers: Freshly infected areas are found on the limbs from about May on to the end of the growing season, generally on the upper surfaces. They are most often seen at the base of a smaller limb or around a wound. Cankers on the trunks are

not common, but, where found, are generally on the southwest side. In earlier stages of the canker, the bark is slightly sunken and reddish brown in color. The diseased area slowly increases in size and darkens, and although not conspicuous from a distance, is readily noticed upon close observation. Some lesions remain very small, the canker usually dying out at the end of the season. Others enlarge from year to year, covering a largearea of bark.

A callus may develop around the margin of the canker, temporarily inhibiting its spread, but as the canker extends further a secondary margin is formed. Repetition of this process gives the canker a lobed appearance. Black rot canker differs in this respect from bitter rot canker, in which there is no callus formation or lobing of the bark. The bark of black rot canker remains closely oppressed to the underlying wood for at least a year. Later the dead bark cracks and falls away, exposing the wood with callus around the margin of the diseased area. Cankers formed early in the season, show numerous black raised fruiting pustules scattered over the central portion. These may not become apparent until the following year. Cankers may enlarge sufficiently to girdle the limb, in which case leaves turn yellow and drop, and the fruit and bark shrivel and die. The fruit sometimes clings to the dead twigs as mummies for a year or more. Fall blossoming of trees may be brought about by the presence of cankers on a tree.

Fruit: Black Rot is primarily a ripe rot of fruit, although it may appear several weeks before maturity. It may appear anywhere on the surface or at the blossom end, but is most often centered about an insect or mechanical injury. There is generally only one spot on an apple. The first symptom is a small brown area, which later turns darker and finally black. On green fruit the spot soon turns black, but on ripe fruit it continues brown until a large area of the fruit is involved. Usually concentric bands of different color shades and of uniform width appear about the center of the spot. The affected area is distinct from the surrounding tissue. The taste of the rotted tissue is not unpleasant as in the case of some decays. The spot enlarges in all directions to finally involve the whole apple, the surface of which is wrinkled and shrivelled, and typically becomes covered with black fruiting pustules. These characters may be assumed in one month or less time. Finally a dry mummy is formed, which clings to the twig for a year or more. Characteristics of the black rot mummy are the long duration of plumpness of the tissue and the black pustules.

Brown Rot produces a coal black, shiny mummy which is smooth. Bitter Rot forms a much sunken roughened and generally lop-sided mummy.

## Life History

Cycle of Infection: Pycnospores ooze from pycnidia in cankers from April on through the growing season whenever enough moisture is present. These pycnospores alight on the leaf surfaces and set up infection which causes "frog-eye" spots. Later pycnospores

infect ripe or almost ripe fruit. In the rotted areas on the fruit pycnidia are formed from which pycnospores ooze and are washed to other fruit surfaces setting up secondary infection. Pycnospores which lodge on wounded surfaces in the bark of limbs and trunk cause canker formation. Mycelium in these cankers forms pycnidia which live over winter and initiate infection the following spring. Cankers may become perennial, enlarging each season and forming pycnidia in the advancing margin.

Morphology and Pathogenesis:

The fungus passes the winter in cankers as mycelium and as pycnidia. In the spring, pycnospores ooze from the pycinidia in coil like masses, during the latter part of April. Moisture governs formation of coils of black spore masses within the pycnidium. The ostiole opens only in mild weather and in the presence of moisture to allow the mass to ooze out. Drops of rain splah these masses to leaves beneath, where, if they remain in a drop of water, the gelatinous material expands scattering the spores. Other drops of rain scatter them still further. In the presence of moisture these pycnospores germinate within a few hours, sending out germ tubes, which penetrates healthy leaf tissue, but can gain entrance to the bark only through a wound, or insect injury. Fruit infection usually takes place through a wound.

In germinating the pycnospore send out one or two germ tubes at or near the end. Moisture and mild weather are favorable to germination. The mycelium hyphae extend out in all directions

through the tissue growing between the leaf cells. The walls of the mycelium are thick and lacking in color at first, later going through stages of yellowish green to dark brown. As the organism draws its nourishment from the cell contents the cells collapse and die. All the leaf cells are involved. Fruiting bodies or pustules rarely form on the leaf spots.

On the bark pycnospores germinate in the same manner and mycelium ramifies the tissue, but the process is much slower and as previously mentioned, the formation of cork tends to check its spread.

Later in the season pycnospores are carried to the fruit and alighting at a point where the skin is broken send their germ tubes into the tissue, forming mycelium which grows between the cells. Cells are turned brown, but do not collapse, remaining rather plump and retaining moisture.

Mycelial-hyphae grow and extend in all directions, gradually consuming the entire apple. Alternate growing and checking of growth causes the various colored bands seen on the outer surface.

As the fungus becomes well advanced in the canker or decayed fruit the fruiting bodies are formed. These are pycnidia which arise from the mycelium. The mycelial threads become tangled and twisted and thicken and enlarge into a knot. The inner cells of the knot become densely granular and break down forming the cavity of the pycnidia. The ostiole is formed in the same manner by breaking down of cells. The cells forming the inner wall of

the cavity arch outward into the cavity and continued growth in the same direction forms the spore stalks (or pycnosporophores). The tips of the spore stalks swell and the walls are constricted to cut off the spores from the stalk. These spores (pycnospores) are at first colorless later becoming dark brown. A pycnidium may contain only one celled spores; or it may contain only two celled spores; or it may contain a mixture of one and two celled spores. Fig. 19 shows a pycnidium and Fig. 21 the pycnospores. Fig. 20 shows the process by which the pycnospores are formed from the spore stalks.

There is a perithecial stage, which apparently plays no important part in the life history of the organism. Fig. 16 shows a perithecium with asci and ascospores. The asci are thickened at the tip, and are abundant. They are interspersed with narrow filamentous threads called paraphyses. The typical number of ascospores in each ascus is eight, but this may vary. Fig. 17 shows the ascospore and Fig. 18 the type of germination.

The mycelium and pycnidia formed in the margin of old cankers carries the disease over winter.

## Control

The pink, calyx and three weeks sprays as recommended in the spray calendar will control the leaf spot. The rotting of the fruit is only brought about through injuries so that proper control of codling moth will also aid in control of black rot of the fruit. Cankers should be cut out as soon as observed and disinfected. All large pruning cuts should be coated with a white lead and linseed oil dressing.

BLACK ROT OF APPLE - FIG. F.

1. - Twig canker, a common source of infection and over wintering stage of the fungus. Note old fruiting pustules on the surface.

2. - Leaf spot form of black rot . Note the concentric arrangement, which gives these spots the popular name "frog eye". The spots are brownish with gray centers.

3. - An apple consumed by black rot. The apple tissue remains fairly firm for considerable while after it has been permeated by the fungus mycelium . Most of such fruit drops, but that which clings to the tree eventually becomes a black, shiny mummy. Black fruiting bodies may be seen on the fruit surface in this picture.

BLACK ROT OF APPLE - FIG. F.

1.

2.

3.

Fig. 16- Perithecium with thick walled asci containing irregular shaped ascospores. Note the slender filaments- paraphyses- interspersed among the asci. This stage apparently plays no important part in the life history.

Fig. 17- Type of ascospores, with air spaces-vacuoles.

Fig. 18- Germinating ascospores.

Fig. 19- Pycnidium, showing pycnosporophores (spore stalks) upon which pycnospores are formed. Winter stage from which primary infection starts.

Fig. 20- Method of formation of pycnospores from the spore stalks. Stages in formation from left to right.

Fig. 21- Germinating pycnospores.

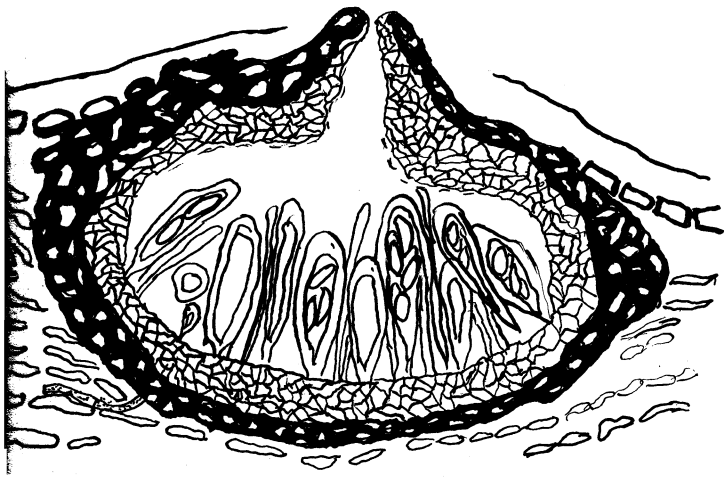
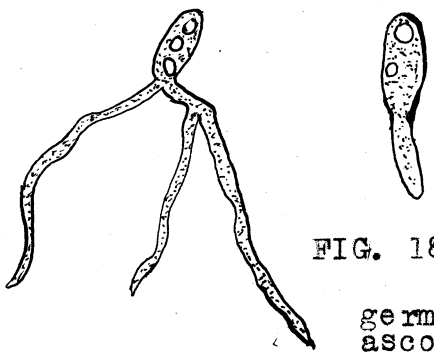


FIG. 16

perithecium(not connected with life history

FIG. 17



germinating ascospores

FIG. 18

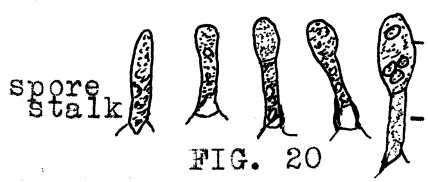



FIG. 20

formation of pycnospore by enlargement and constriction of the spore stalk

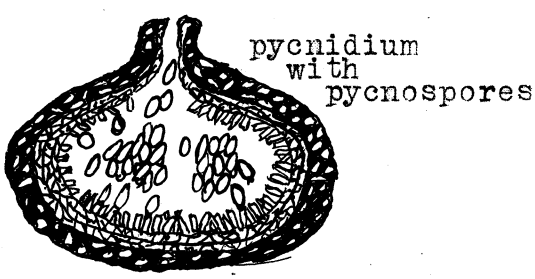
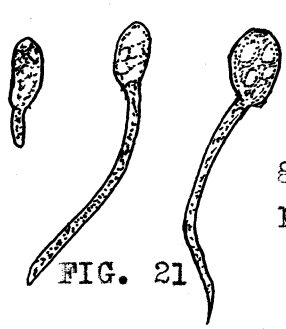



FIG. 19



germinating pycnospores

FIG. 21

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## CEDAR RUST OF APPLES

### History

Cedar rust was first described by Schweinitz (1832)

### Host

Red cedar and apple.

### Stages

The disease passes considerable of its life history on cedars, and the balance on apples. About 21 months elapses from the time aecidiospores infect the cedar until galls are mature and produce spores. Only about 3 months is required for the sporidia from the cedar to produce infection on the apple and produce the aecidiospores, which carry infection back to the cedar. The apple infection is the sporophytic stage and the cedar infection is the gametophytic stage.

### Economic Importance

The loss from this disease varies from year to year because of the effect of weather conditions. It is considered one of the most important diseases in many states, especially where susceptible varieties are grown. The loss is not only in low quality fruit, but also in defoliation and weakening of the trees, with the resulting decrease in their efficiency. In areas in which cedars have been removed the disease is no longer of economic importance, but in some sections, where cedars are abundant, apple trees may lose ¼ of their foliage and losses may run high. In Virginia, parts of which are included under the cedar ban, losses are generally 2% to 2½%, the greatest loss being in sections in which York Imperial is the chief variety grown.

## Mode of Dissemination

Wind blown spores following rainy weather carry the organism from the cedar to apple and from the apple back to cedar.

## Symptoms

The first symptoms of cedar rust on the apple are pale yellow spots on the foliage in spring, usually in May. These are seen on the upper surface of leaves as minute pale yellow areas, becoming darker to an orange color and enlarging in size. On the surface of the spots about one month after infection cup shaped pustules develop, which later turn black. The tissue beneath the spot becomes swollen and distorted, due to the enlargement and multiplication of the spongy parenchyma cells. A cup shaped body appears on the lower surface of the leaf about the first part of July. This is the aecidium in which are produced the aecidiaspores, which carry infection to the cedar. If many aecidia are formed the leaf becomes curled and may turn yellow and drop prematurely. There are sometimes as many as 200 to 300 aecidia on a single leaf. These aecidia appears as orange colored thickened cup-shaped spots. On the fruit aecidia are formed as on the leaf and appear more numerous around the calyx. Early infection of the fruit stops growth in the infected area causing a dwarfing of the fruit. Galls are formed on the cedar by infection from the apple. Growth takes place the following season and a gray or greenish gall continues to grow all season attaining its full size by fall. This gall is caused by an enlargement and distorcion of

the cedar leaf tissue. During late autumn and early spring of the second winter these galls become pitted with small depressions. In the spring orange colored gelatinous horns grow from these depressions/

## Life History

### Cycle of Infection:

The two year cycle of cedar rust is quite complex, being divided into two parts (1) that passed on the apple (2) the time passed on the red cedar. The diagram shown in Fig. 22 gives the cycle of infection of the two hosts. From galls on the cedar orange colored horns protrude during rainy weather in April. These horns contain teleutospores. These teleutospores germinate on the horn by sending up a 5 celled tube (pros mycelium). From four of the cells of the promycelium four basidiaspores are formed, one from each cell. Wind carries these basidiaspores to the leaf and fruit surface of the apple. Here they germinate and produce infection. Mycelium spreads in the tissues and cu-shaped bodies form on the under sides of the leaves and on the fruit. These are the aecidia, which produce within themselves aecidiaspores in July. Aecidiaspores are expelled and blown by wind to cedar trees where they germinate in the fall. The following spring growth of the cedar gall starts and continues during the growing season. In the fall, one year after infection of the cedar, the galls are full grown. They remain dormant during winter and form gelatinous horns the following spring to start primary infection in another cycle.

Morphology and Pathogenesis:

Teleutospore Formation:

From a mass of mycelium in the cedar gall, a layer of erect, oblong cells arises. These cells at first have no nuclei, but later become bi-nucleate. From these oblong cells the teleutospores gradually take shape by formation of two cross walls. One wall separates the base of the spore from the oblong cell and the other wall divides the spore in two parts. The oblong cell which has not become a spore stalk for the teleutospore, rapidly elongates, carrying the spore outward and upward. Large numbers of these pedicels with their teleutospores on the ends constitute the gelatinous horns (sori) which are seen about the first part of April. Mature teleutospores (Fig. 23) are carried up on their stalks out of the way of other spores which are continueing to form. The nucleus of one cell of the teleutospore passes into the other cell and fuses with the mucleus therein. From this latter cell a germ tube is sent out (pro-mycelium) Fig. 24. All the teleutospores do not germinate at one time, but after each rain there is a gelatinization of the horn and germination of some of the teleutospores. A horn will gelatinize only about two or three times before drying up, but germination of teleutospores on gelatinous horn over the cedar tree as a whole lasts about six weeks, depending upon rainfall.

Basidiaspores (Spordia):

The pro-mycelium sent from the teleutospore divides into five cells (Fig. 24). From each of the four outer cells a basidiaspore (sporidium) buds out. The fifth cell next to the

Teleutospore does not produce. The asmosphere must remain highly humid for about 3 hours to allow gelatinization, germination, and sporidial production. Sporidia are very light and with the return of dry air may be blown several miles to alight upon the surface of apple leaves and fruit. Very few spores are blown over two miles. In the presence of moisture sporidia germinate on the apple tissue (Fig. 35) and send germ tubes through the cuticle. Mycelium develops around the point of infection and occupies the space between the cells. Haustoria are sent into the cells of the spongy parenchyma and the fungus on the host through these haustoria. Host tissue becomes swollen and distorted in the diseased areas. Later the fruit tissue becomes dwarfed in the vicinity of the rust spot. Only young leaves are infected by sporidia, the older leaves apparently not being susceptible. About one month after infection, the first fruiting bodies may be seen on the upper leaf surfaces as orange colored pustules, within the diseased area. These pustules (spermagonia) are formed, immediately under the upper epidermis of the leaf, from mycelium, which arranges itself into a dense mass of converging strands. These strands finally rupture the cuticle and protrude. Chains of spores (spermatia) are formed from these strands, continuously until all the mycelium is used up (Fig. 36-a). The function of spermatia is not known. They apparently do not play an important part in the life history.

Aecidia:

The tissue beneath the diseased spot on the leaf becomes swollen and distorted due to enlargement of the spongy parenchyma

cells. A globular mass of mycelium forms in the parenchym tissue, which mass gradually enlarges and elongates toward the lower surface of the leaf. A thickened orange colored cup-shaped spot appears on the under side of the leaf about the first part of July. This is the aecidium, within which chains of aecidiaspores are formed from mycelial strands. The outermost chains of aecidiaspores change to a single layer of cells; which forms the wall or covering for the aecidiaspores within.

On the fruit aecidia (Fig. 27) are formed in much the same manner as on the leaves. Mycelium invades the pseudo-parenchymatous cells of the fruit and a globular mass forming here extends outward toward the surface to form the thickened orange colored cups. These usually appear more numerous around the calyx end of the fruit. Early infection of the fruit checks growth in the infected area, dwarfing the fruit at this point.

Aecidiaspores:

Aecidiaspores are formed within the aecidia by conjugation. At the base of the aecidium two mycelial hyphae lie side by side. The end cells of these hyphae make a contact and the cell walls break down between the two at the point of contact. The nucleus from one cell passes over into the other cell but does not fuse with the nucleus there. This forms a bi-nucleate cell, from which one or more new cells bud off. The two nuclei in the parent cell divide, and two of these new nuclei pass over into each new budded cell. These new cells are aecidiaspores (Fig. 26c) Further division of the numerous hyphae from the gametophytic to

the sporophytic stage. Aecidiaspores are dark brown, minutely pitted almost spherical bodies with thick dark walls, some of which are inclined to be almost hexagonal.

Cedar Galls:

The cedar leaf or needle becomes hypertrophied from invasion of its tissue by mycelium formed after infection from the aecidiaspore. In the spring following fall infection from the apple, mycelium invades the intercellular spaces of the parenchyma and branches and divides into cell-like portions of different lengths, between which cell division is very indistinct. Each of these cells contains two nuclei. There is a tendency of the mycelial strands to grow in a straight line, thus making the mycelial not appear as a mass of sharp angles. Mycelium does not penetrate the twig but is checked at the point of union of the leaf with the twig. Haustoria or feeding bodies are sent into the parenchyma cells only, and do not penetrate many of these cells until about the time of teleutospore formation. About 20 months elapses from the time of infection of the cedar leaf in the fall until teleutospores are formed. During this period mycelium establishes itself in the leaf and the gall grows to maturity, (Fig. F) Young galls are a grayish-brown color and fairly smooth, but as they grow they turn more brownish and become deeply pitted. Gelatinous horn exude from these pits at the proper time. When gelatinization of the gall is completed it turns black and becomes very brittle and hard and may persist on the tree for a considerable time until destroyed by insects or the elements.

## Control

Both the apple and cedar tree are necessary in the immediate locality, forthe completion of the life cycle of the rust disease. Removal of all red cedars within a radius of two miles of the orchard is the only successful practical method of control, although if cedar galls can be thoroughly and consistently removed during winter from specimen trees this will also serve to save some cedars in case they are valuable as ornamentals. Varietal resistance plays an important part. Such varieties as York, Rome, Jonathan and Wealthy are quite susceptible, while Winesap, Stayman, Grimes, Black Twig, Yellow Transparent and some others are resistant.

Legislation has been passed in some states and counties in which apple growing is an important industry, requiring the cutting of all cedars. Spraying is not practical in large orchards because of the limited time, during which it may be successfully done. In some years as high as 21 spore ejections from cedar galls have been recorded from April 2 to June 23rd. If heavy spore ejection takes place before apple foliage comes out infection may not be so severe. Heavy infection takes place within two miles of cedars. High winds may extend this distance considerably. Defoliation takes place in proportion to the number of spots per leaf. Five or more spots per leaf may cause heavy defoliation.

RUST OF APPLE AND CEDAR - FIG. G.

1. - Gall on red cedar ready for extrusion of gelatinous horns.

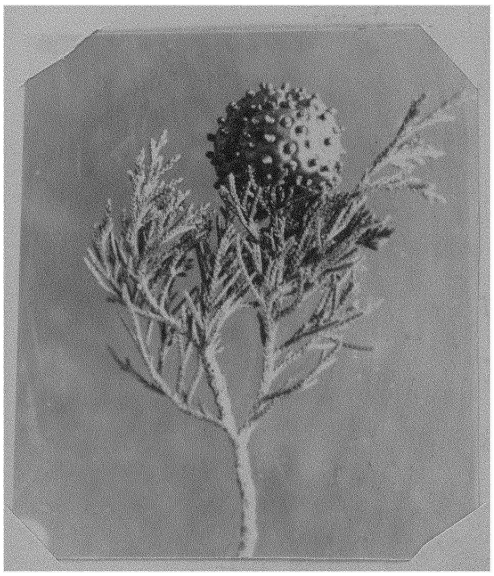
2. - Cedar gall with horns extruded. These horns carry spores, which produce infection on the apple.

3. - Orange spots on upper surface of leaf. Numerous spots cause leaves to drop prematurely.

4. - Later stages of rust on leaves, with formation of pustules on the surface.

5.& 6. - Characteristic formation of pustules around calyx end of fruit . Numerous pustules in a limited area impart the roughened orange colored appearance.

RUST ON CEDAR AND APPLE - FIG. G.



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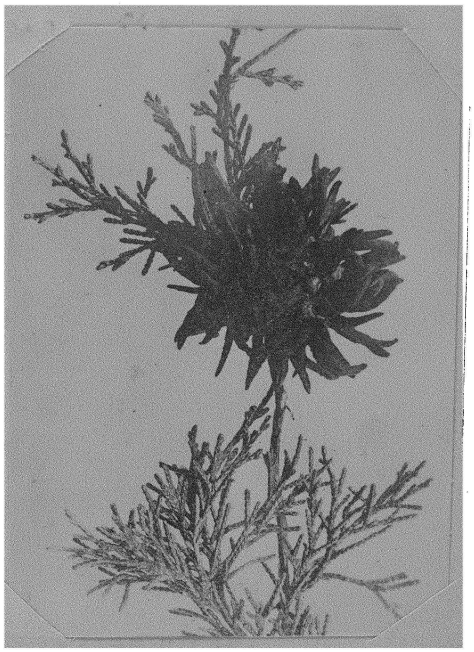

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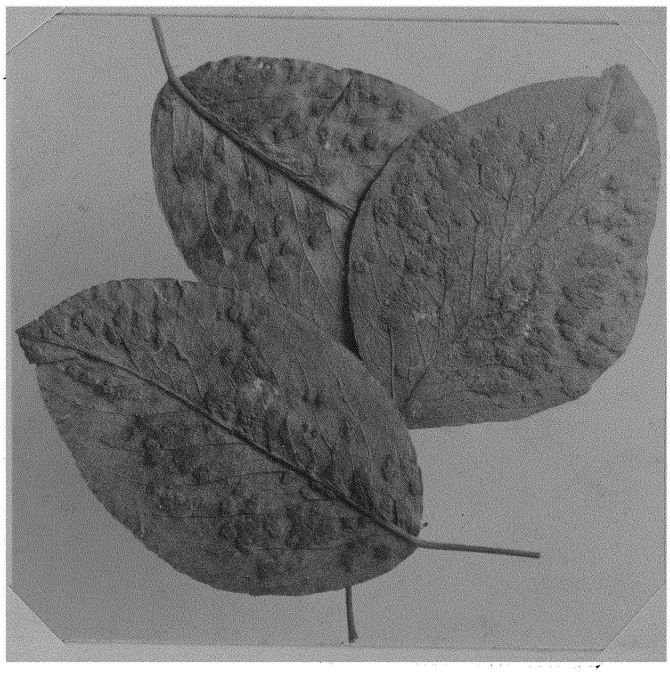

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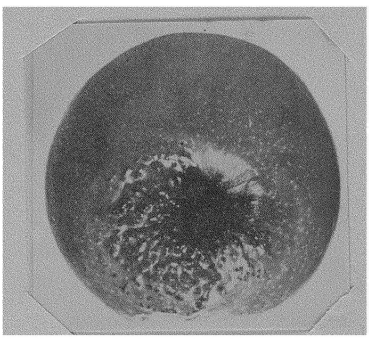

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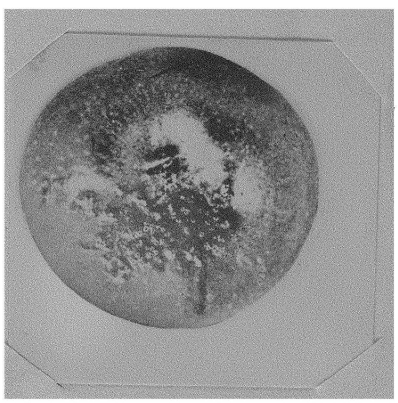

6.

Fig. 22- Life cycle of cedar rust fungus.

Fig. 23- Teleutospore borne on the gelatinous horns of the cedar gall.

Fig. 24- Teleutospore germinated showing the five celled promycelium with one sporidium- (basidiaspore)- on each of the top four cells.

Fig. 25- Types of germination of basidiaspores.

Fig. 26- Section of a leaf infected by cedar rust.

a- pycnium in upper part of leaf with spermatia extruding.

b- cup-shaped aecidium on under side of leaf containing aecidiaspores.

c- Aecidiaspores

Fig. 27- Immature aecidium in tissue of the apple fruit.

CYCLE OF INFECTION OF CEDAR RUST OF APPLE

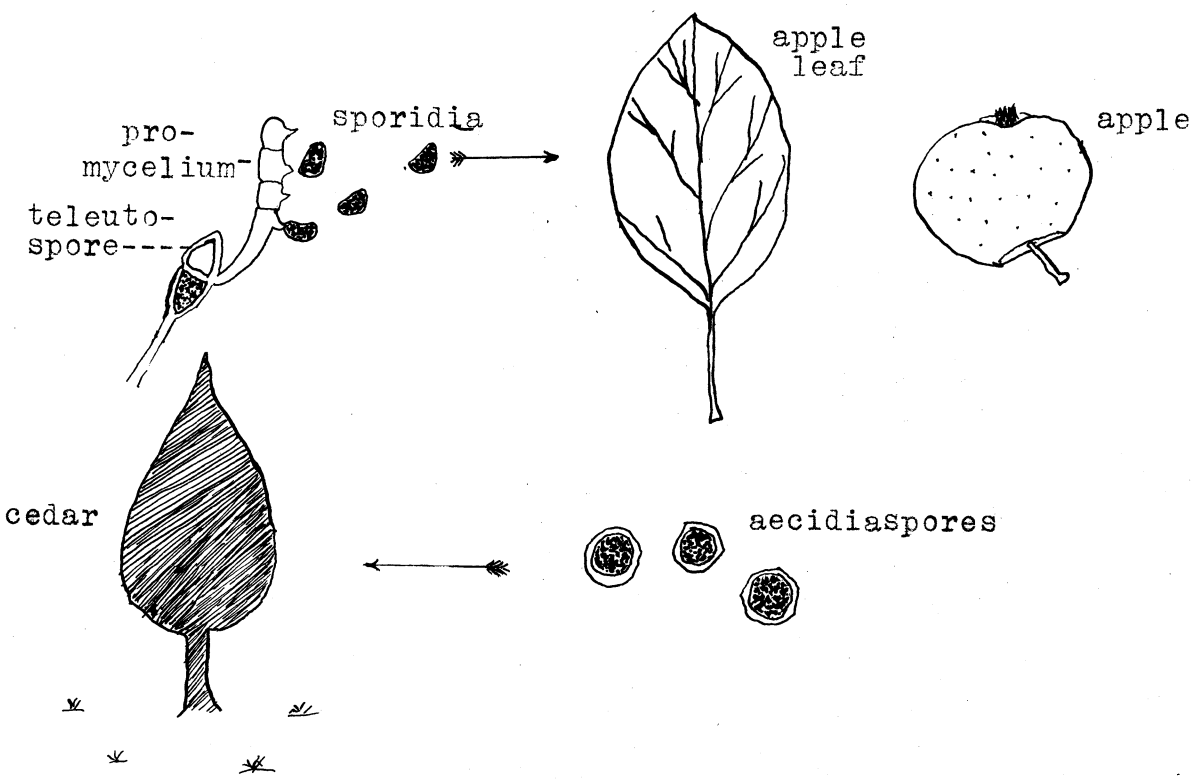


FIG. 22

# CEDAR RUST OF APPLES

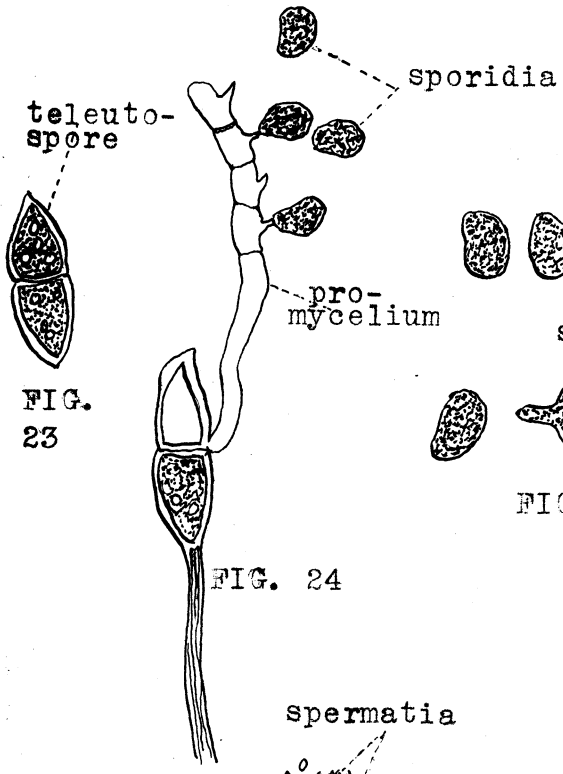


FIG. 23

FIG. 24

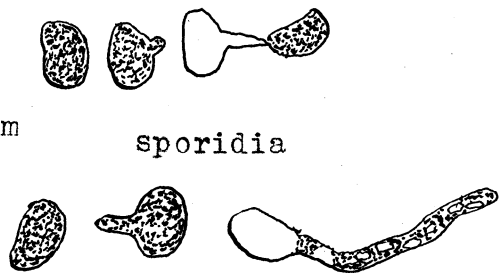


FIG. 25

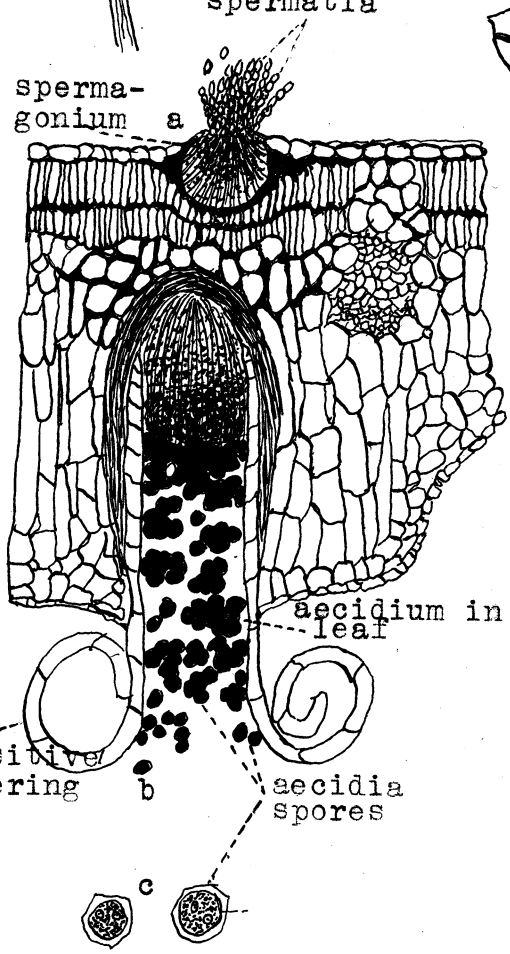


FIG. 26

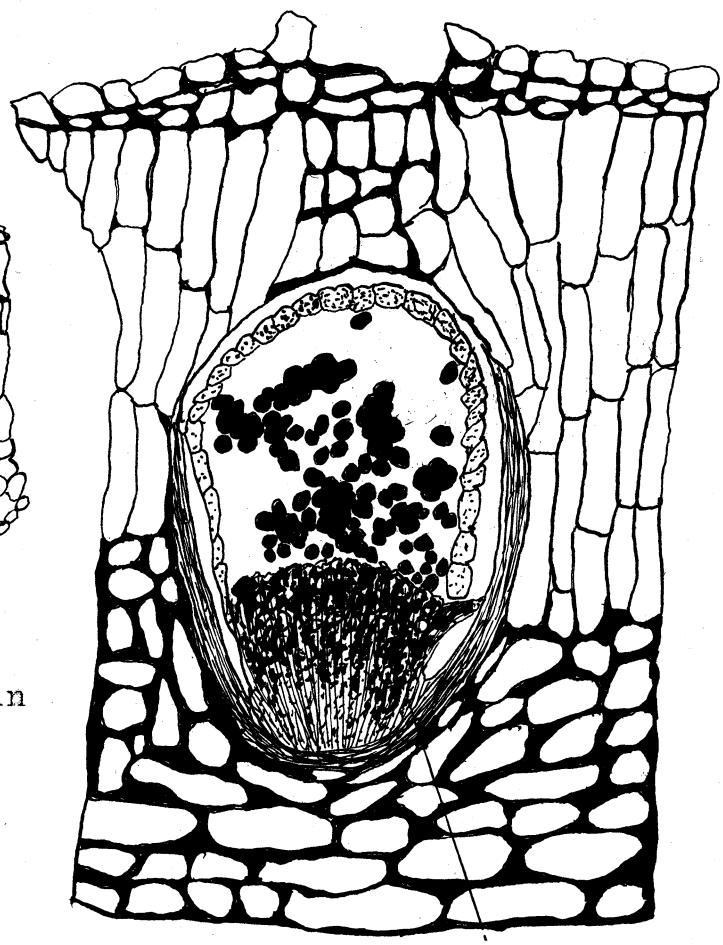


FIG. 27

## LITERATURE

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## APPLE BLOTCH

### Host

Confined to the apple, but found on Wild Crab and cultivated apples.

### History

This disease probably originated on the wild crab apple. It was described on the wild crab in 1895 but it was not known until 1907 that the same fungus affected the apple. Ellis and Everhart first described the causal organism. J. L. Sheldon in 1907 found that the organism described on the wild crab was the same as the blotch organism of the cultivated apple. Numerous workers have contributed tothe life history studies and since 1913 work on control has been in progress.

### Geographical Distribution

The greatest losses from this disease have been in the middle west, particularily in Ark., Ill., Ohio and Indiana also Mo. However it is found on susceptible varieties throughout the eastern half of the U. S.

### Economic Importance

In Virginia the disease is not considered of economic importance as the commercial varieties generally grown in this State are not so susceptible. However, where Northwestern Grening is grown the disease is troublesome. Other moderately susceptible varieties are Ben Davis, York, Limer twig and Pippin. In southern Illinois, Arkansas and Missouri it is prevalent today and while it is not epiphitotic in nature its steady growth takes a large annual toll and places it among the most important diseases in that section.

## THE SPRAY CALENDAR

Specialists in fruit disease and insect work have studied the habits of various orchard pests and from this study have developed a spray program, which gives good control with a minimum number of sprays. Some individual spray applications are timed to control several diseases and in some cases fungicides and insecticides are combined in a spray to control both disease and insect.

Time of application of earlier sprays is based upon development stages of the tree. It has been found that there is generally a correlation between development of fungus organisms and development of the tree in the spring although seasons vary in this respect. At any rate tree development is the most reliable and simple guide for the ordinary grower in applying his earlier sprays.

There is considerable seasonal variation in the time of appearance of different diseases, as well as in climatic conditions favoring infection. This probably accounts for much of the variation in control of the different diseases. Most fungus diseases are dependent upon moisture and temperature for their development, and conditions, which favor early development of disease spores, also may favor early tree development.

The following ten year record of spraying dates and time of appearance of diseases and insects is interesting in studying the efficiency of spray programs.

## Symptoms

The first sign of the disease is a small inconspicuous light brown irregular spot, with the appearance of a star-like collection of brown fibers just beneath the epidermis. The spots spread radially from the point of infection to a size of 1/8 to 1/2 inch, becoming darker. The advancing margin is irregular and jagged and has a fringed appearance. On very young apples the points of infection show as small water soaked areas, from which there may be a yellow exudation in wet weather. Where spots are numerous they may coalesce to form large blotched areas, which may cover half or more of the apple. The fungus kills only the superficial cells, so that continued growth of the underlying tissue causes the diseased spot to crack. The cracks thus formed may be large or small. Some cracks intersect each other to form a cross. The spots may vary in size and character with different varieties. Graduations may be found from large, fringed and ragged spots to small, compact and regular areas. Three general types of spots are recognized as fringed, blistered and pitted spots.

Within a few days after the spots become visible, black pycnidia begin to develop on the diseased areas. From 3 or 4 to many develop on each spot, and may be grouped or scattered. The general effect on the fruit is that of marring its appearance, but cracked fruit is an infection court for black rot. A large percentage of badly infected fruit drops prematurely. The fungus also attacks twigs, spurs and rapidly growing shoots, producing characteristic cankers. The first appearance of infection of

the wood is seen in August as small purplish or blackish blotches, which later become brown in the center, retaining the purple margin, but finally may become gray. The bark soon cracks around the margin, especially on the longer sides. The longitudinal cracks also appear in the center of the canker, giving them a rough scurfy appearance. This is especially noticeable the second or third year. The marginal crack frequently limits the growth of the canker, and later healing over of the canker may take place. Generally cankers are confined to small limbs, but with very susceptible varieties whole limbs or scaffold branches may be killed.

The leaf spot form of blotch is not serious. Small yellowish to light brown spots 1/16 inch or less in diameter, appear on the leaves and leaf petioles, being either scattered or grouped around the veins. Badly infected leaves may drop prematurely or may turn brown and hang on the tree, the latter being caused by the girdling of the petiole.

## Life History

### Cycle of Infection:

Parts of the cycle of the blotch fungus are not definitely known. It is carried over winter in cankers on twigs and small branches. In spring pycnidia form along the margin of cankers. During rains of late spring and early summer, pycnospores ooze from the pycnidia, usually during the last part of May, and are splashed to surfaces of leaves, fruits, and other twigs. Infection of the host tissue takes place, probably through the natural openings. About 20 to 30 days after infection blotch spots appear

on the fruit, leaves and twigs. Pycnidia soon develop on these diseased areas and exude spores which carry secondary infection to other surfaces. Badly diseased fruit drops prematurely. Secondary infection stops in August and just what takes place from this time on is not definitely known. The fruit and leaves probably do not carry the organism over winter. Cankers however do harbor the disease. Scott and Rorer (1) believe that pycnosclerotia (Fig. 32) are formed in the cankers after August, and remain over winter, producing conidia the following spring in some cases. They also believe that mycelium remains in the margin of cankers and forms pycnidia the following spring in other cases. At any rate cankers are known to carry the organism over winter. Cankers may form pycnidia in their margins each year until they are four years old, after which they die out.

## Morphology and Pathogenesis

### Pycnidium:

Pycnidia are rather small black glistening, sub-globose or somewhat depressed bodies (Fig. 28), which appear on the surface as raised black dots. There is a neck-like opening in the apex of the pycnidium through which pycnospores ooze when mature.

Pycnospores are one celled, granular, oval to sub-globose bodies which are usually enclosed in an adhesive, gelatinous covering which may be drawn out into an appendage (Fig. 29) about twice as long as the spore. Spores germinate by sending out one or more germ tubes (Fig. 30) which usually are thickened at the point of attachment to the spore.

Infection:

Germ tubes enter the host tissue and the mycelium occupies the inter-cellular spaces of the epidermis and hypoderma. A protective layer of large dark, rounded cells is formed below the hypoderma, but the spongy parenchyma cells are also affected. My celium is much branched (Fig. 31) and becomes inter twined at intervals to form the pycnidia.

Pycnosclerotia:

According to Scott and Rorer (1), older cankers which have been producing pycnidia on their advancing margins during summer, cease to produce spores in late summer and form pycnosclerotia (Fig. 32). These pycnosclerotia lie dormant over winter and the following March undergo a change in which their inner cavities form cells from which conidiaphores and conidia arise late in the spring. These may bring about primary infection.

## Control

In Virginia three sprays are recommended for control of blotch (1) three weeks after petal fall- lime sulphur 1 to 40. (2) five weeks after petal fall- Bordeaux mixture 4-8-100. (3) mid-summer spray about July 1 to 10. Where blotch has been severe spraying should be supplemented by cutting out of cankered twigs. Trees should be kept well pruned so that spray can reach all parts. Clean nursery stock should be planted.

Gardner, Greene and Baker (2) find that sprays applied at petal fall, 2, 4 and 6 weeks later gave satisfactory control of blotch. Lime sulphur in the petal fall and 2-4-50 Bordeaux in later sprays proved effective in Indiana.

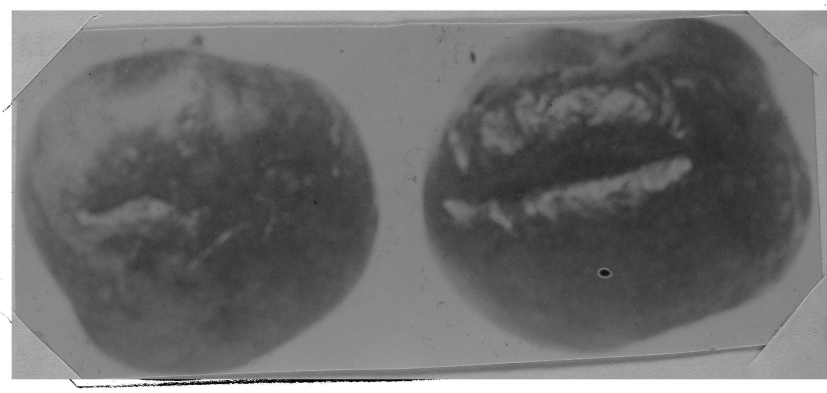
Martin (3) found that blotch did not become severe in New Jersey until sometime between June 3 and June 10 or just after the 17 day spray. Bordeaux mixture proved to be superior to concentrated lime sulphur.

Gardner (5) claims that shaving off or pruning out old cankers and spraying to prevent formation of new ones will effectively eradicate blotch.

Kohl (6) found that the incubation period of apple blotch fungus on the host tissue consumes about 20-24 days. Mycelium is intercellular and occupies only the collenchyma layer.

# APPLE BLOTCH

1.



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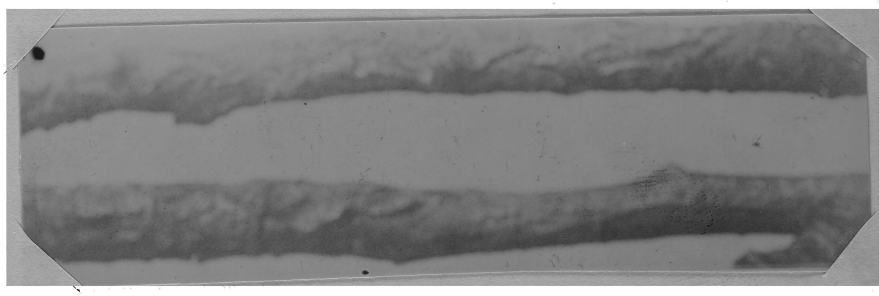

4.

1. - Apples infected with blotch. Note stellate character of the lesions.

2. - Apple so badly blotched as to crack open. A three pointed crack is commonly formed.

3. - Sooty blotch or sooty fungus, sometimes confused with the Phillosticta blotch. This is caused by a different fungus organism.

4. - Cankers on twigs, which hold the fungus over winter and produce primary infection.

Fig. 28- Pycnidium of Apple Blotch with pycnospores.

Fig. 29- Pycnospores with gelatinous appendages.

Fig. 30- Types of pycnospore germination of pycnospores.

Fig. 31- Type of branching of the hyphal threads of the mycelium.

Fig. 32- Pycnosclerotium with conidiaphores and conidia. Note the gelatinous threads connecting the conidia. After Scott and Rorer (1)

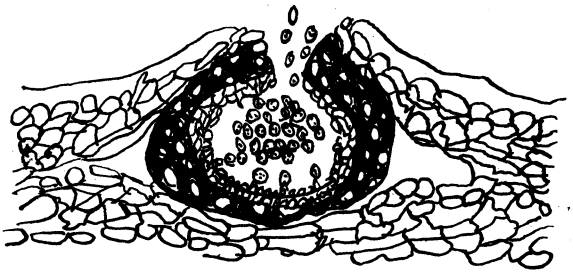

FIG. 28

pycnidium with pycnospores

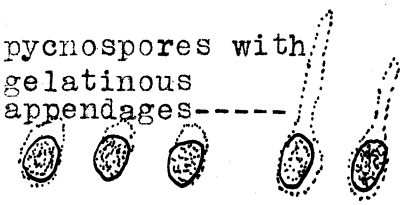


FIG. 29

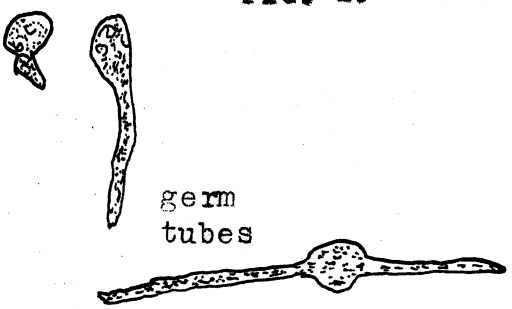


FIG. 30

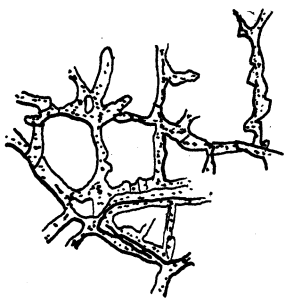


FIG. 31

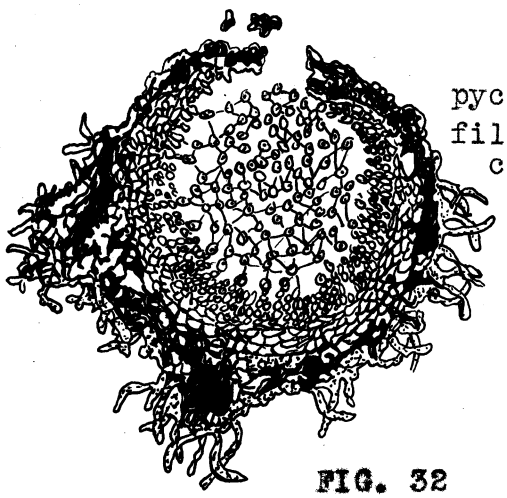


FIG. 32

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## APPLE POWDERY MILDEW

### Hosts

Apple, pear, quince, cherry, plum.

### History

This disease, known for many years for its severe attack of foliage and young shoots of apple nursery stock, later became serious on bearing apple trees.

### Geographical Distribution

It is widely distributed over the world wherever climatic conditions are suitable for its development. In the U. S. parts of California seem to suffer worst although the disease is of potential importance inthe eastern and northern states and somewhat in the middle west.

### Economic Importance

In the Pajaro Valley of California immense losses have occured. Jonathan, one of the most susceptible varieties is largely grown in the Coastal states. In Virginia the disease is classed as important only because of its potentiality for becoming serious. The ordinary spray schedule as applied for control of other more important diseases will also control mildew. Varietal susceptibility is of importance since most of the varieties grown commercially in this state are not very susceptible. Losses therefore are not great.

### Symptoms

Early in the summer, leaves and young shoots show a whitish or grayish powder on their surfaces. Leaves are most

commonly affected on their under surfaces. The whitish areas are small at first, widening to coalesce with other areas. Thus large surfaces are involved. Later mildew is seen on the upper surfaces of the leaves. One year shoots may be entirely covered with the white mycelium of the fungus. There is a marked dwarfing of leaves and shoots. Growth of the shoot is checked, and the leaves are dwarfed, curled and crinkled. Sides of the leaves seem to suffer more dwarfing than the extremities, thus causing them to appear narrow and curled. The general appearance of a severly infected tree is that of sparse foliage. Reduction of leaf surfaces by dwarfing causes diseased shoots to appear more barren of leaves than healthy shoots. In winter, when trees are bare, the heavy coating of mildew on young shoots gives the tree a silvery appearance on bright sunny days. Fruit is not attacked by this disease, it being confined to leaves and young shoots. Summer spores formed in large quantities on the surface of the tissue form a powdery coating, from which the disease takes its name. Badly infected shoots may die during the winter.

## Life History

### Cycle of Infection:

This fungus grows entirely on the surface of the tissue, with the exception of haustoria, which are sent into the epidermal cells and feed the surface mycelium. The organism over winters in the perfect stage as perithecia on infected shoots. Each perithecium contains one ascus with 8 ascospores, which ripen early in the spring and in the presence of moisture fall on the

young leaves and twigs, setting up primary infection. Mycelium also may hybernate in bud scales, producing conidia in the spring and accounting for some of the primary infection. Spores germinate and form mycelium, which sends out haustoria at irregular intervals. Haustoria enter the epidermal cells and derive nourishment. Conidia arise from the mycelium and are carried to other leaves and twigs throughout the season, setting up secondary infection. In July, conidia formation ceases and perithecia are formed in the surface mycelium. Mycelium dies out during late summer and fall except that which penetrates bud scales. Perithecia remain on the shoots over winter ready to initiate infection the following spring.

## Morphology and Pathogenesis

### Perithecia:

An examination of diseased twigs after July reveals smoky patches in the mycelium. Under the lens these patches are seen to contain large numbers of closely crowded, globose dark brown bodies, each of which has a cluster of 4 to 11 long bristly appendages on the upper part and numerous short root-like growths on the lower part. These are perithecia, a cross section of which may be seen in Fig. 35. The outer wall of the perithecium is thick and roughened, its surface being filled with ridges and crevices. Within the center is a single almost round ascus. Eight ovoid ascospores are contained within each ascus. Perithecia are borne on the surface of the twig.

### Conidia:

Mycelial strands, on the surface of the tissue, enlarge at a number of points. These enlar gements extend upward from

the surface to form conidiaphores. From these stalks conidia are formed in chains by constriction of the side walls and formation at intervals of cross walls. Fig. 33 shows this type of spore. Spores germinate by a simple germ tube which branches and re-branches profusely to form mycelium.

Haustoria:

At irregular intervals club-shaped bodies are formed from hyphae of the mycelium. These bodies puncture the surface and find their way into epidermal cells, probably dissolving the cell wall. Withdrawal of nutriment from these cells causes collapse and death. Woodward (3) states that haustoria secrete toxins which destroy the leaf tissue.

## Control

This disease is well controlled in Virginia by application of the pink, calyx and three weeks sprays as recommended in the regular spray program. Vigorous foliage is less damaged by mildew than less vigorous. Any scheme of orchard management, that will invigorate the tree will lessen the severity of infection. Once started, the disease recurs year after year, gradually obtaining stronger hold. Cutting out severely infected twigs during the dormant season will aid in control.

Woodward (3) finds that secondary infections have been over emphasized and that primary infection is most important.

Fig. 33- Conidia arising from the surface of the leaf. Mycelium has sent up conidiaphores Summer stage.

Fig. 34- Conidia or summer spores.

Fig. 35- Perithecium of apple mildew. Cross section showing the thick walled burr like formation with mycelial strands at the base and hair like appendages on the upper part. There is a single globose ascus containing 8 ascospores. This is the sexual or winter stage, which initiates infection in spring.

Fig. 36- Oval-shaped ascospore

APPLE MILDEW

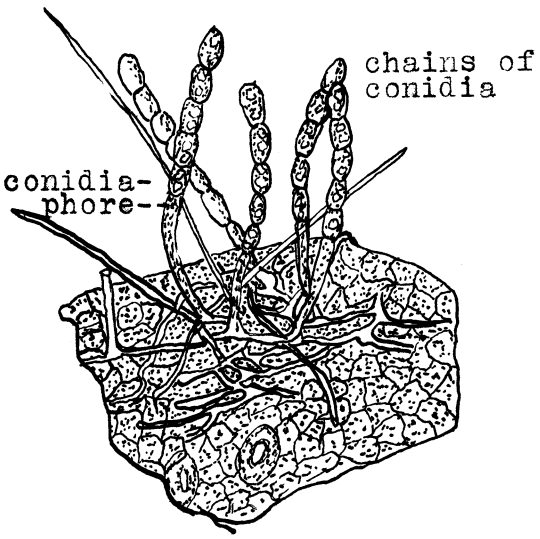



FIG. 33

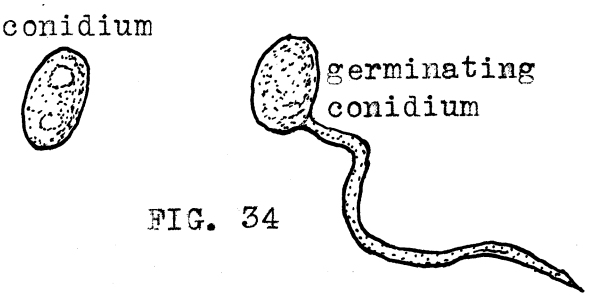



FIG. 34

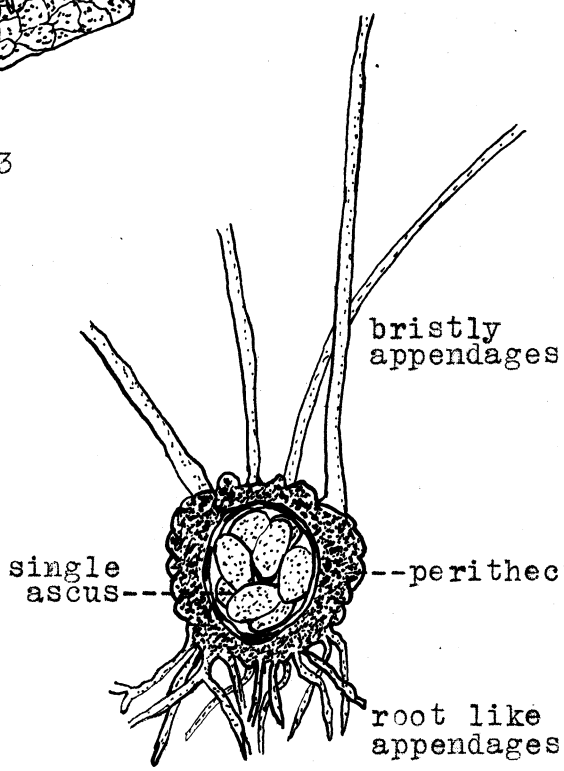



FIG. 35

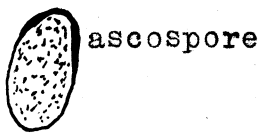
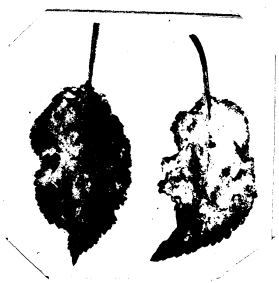



FIG. 36



**Mildew on apple leaves. White mycelium on surface and deadened margins.**

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## BLACK ROOT ROT OF THE APPLE

### Cause

The agent causing the commonly occurring Black Root Rot described by Fromme (1) as being a species of Xylaria and is named Xylaria mali nom. nov.

### History

This disease has become of economic importance only in recent years, being described by Fromme and Thomas (2) in 1917.

### Geographical Distribution

This fungus disease has been most prevalent in Virginia extending up and down the valley and over into the Piedmont section and down the mountainous section into North Carolina. It is now found in scattered locations in West Virginia and in some of the middle-west states.

### Economic Importance

There is a gradual annual toll taken by Black Root Rot and it never becomes epiphetotic. The organism spreads in the soil radially from an infected tree and gradually includes tree after tree. Replants in the infected area soon succumb, even though several years may have elapsed since the death of the original tree. The gradual spread from tree to tree may bring death to a large portion of the orchard. In the Piedmont and Valley sections of Virginia large losses have already occurred with the prospect of still further losses in the future. Where present this disease takes a gradual annual toll, and since there is no known method of control, death of infected trees is almost certain. This diseased is described by Fromme (1) as being the

most important single cause of apple tree death in the Shenandoah Valley of Virginia.

## Symptoms

Checking of growth of the tree, with thin yellowish-green foliage, along with excessive blooming and fruiting and tendency of individual limbs to die, followed by death of the entire limb are the first symptoms. Generally one entire limb or two or three limbs in the same general portion of the tree die first, as the root rot destroys the root which feed these particular parts. As the organism spreads from root to root adjoining limbs die gradually involving additional parts of the tree until the whole tree is dead. Infected roots become punky and brittle allowing the tree to be easily broken down. Death of the entire tree may require from one to several years depending upon the size and age of the tree with its corresponding root area. If roots of the infected tree are examined, black incrustations may be seen on the surface of the diseased roots. These roots will be brittle and easily broken.

In later stages of the disease fruiting bodies of the organism can be found on the trunk just at or slightly below the ground line. These consist of a stromata usually in the shape of a fan with finger-like appendages. Most often the base, which is attached to the tree surface, is narrow, broadening out wedge fashion to the point at which the fingers join. The stromata as a whole is fan shaped, and the fingers are con-

siderably flattened. Young stromata may be seen to have mealy white fingers, due to the large numbers of spores borne thereon. Presence of the stromata is the only definite assurance that dying of the tree is caused by this root rot. Other root troubles may cause death of the tree, but no other has these characteristic stromata attached to the trunk.

## Life History

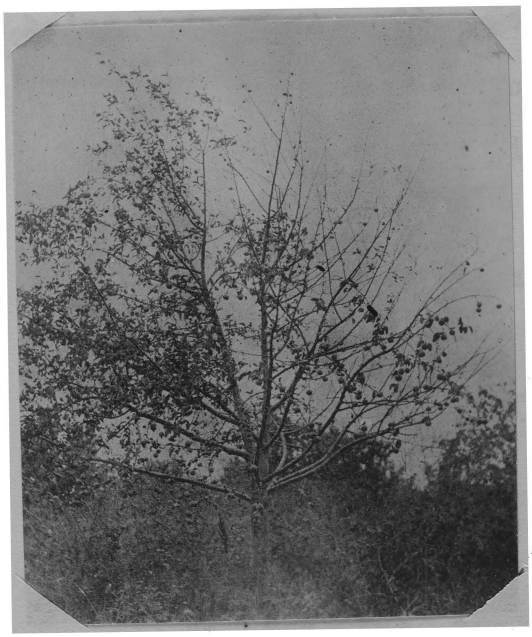
Little is known of the cycle of Xylaria mali except that both the sexual and assexual bodies are present within the flattened stromatic arm there is a dark gray band around the border in which globose to sub-globose perithecia are crowded. These perithecia are deeply imbedded in this layer and covered by a thick hard layer of black stromatic material. There are eight ascospores which are dark brown, elliptical and usually slightly curved.

Stromata start growth in the spring usually about May and reach maturity about the middle of June. The young tips of the stromata become covered with a mealy mass of conidia about the last of June or the first of July. Following conidial production the stroma may cease growth and shrivel and die or perithecia may be formed as described above.

## Control

No satisfactory control has been developed for Black Root Rot disease. Fromme (1) found that any apparent varietal resistance was rather a resistance of the individual than of the variety as a whole, and that no variety withstood infection when replanted in the location of a diseased tree. Studies are

BLACK ROOT ROT OF APPLE.



1.

2.

1. - Characteristic dying back of one side of a tree. Death was caused by dying of the roots, which feed this portion of the tree. Black root rot produced these symptoms.

2. - Stromatic arm of black root rot, showing the fan shaped, black stroma with finger like arms, upon which the fruiting bodies are borne. This stroma is found at the base of the tree near the ground line and is characteristic only of black root rot.

being made of the effect of soil media but no control has as yet been secured. Since the replants are infected as soon as planted it is decidedly unwise to replant where trees have died from root rot at least for several years after death of the original tree.

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## PART II

## PEACH DISEASES

Brown rot is almost universally the most destructive disease of peaches. Curculio and peach moth promote infection. Leaf curl causes considerable damage where the dormant spray is not applied. Some growers do not apply this spray every year, but consistent spraying will effectively control the disease. Scab is potentially serious but is easily controlled. Bacterial spot is becoming increasingly more serious but sprays recently developed have been found effective. These are the most important peach diseases.

## SPRAY CALENDAR FOR PEACHES

| No. | Time of Application | Materials to use | Parasites |
|---|---|---|---|
| 1 | Dormant season (before buds have commenced to swell, February or March. | Lime-sulphur, 32° Baume, 12 gals: add water to make 100 gallons of spray. If oil emulsion is used for scale, it should be used with 3-3-50 Bordeaux mixture for the control of leaf curl. Lime-sulphur, however, may be used with the ligin pitch home-made oil emulsions. If lime-sulphur is used with lignin pitch oil emulsion, it should be used at the rate of 1 to 15 for the control of leaf curl. | Scale<br>Leaf curl |
| 2. | Immediately after the petals drop. | Zinc-lime spray 8-8-100 formula; add two pounds of powdered lead arsenate for each 100 gallons of water. The lime contained in the zinc-lime spray is sufficient. | Curculio |
| 3. | One week after No. 2 | No. 3 same as No. 2 | Curculio |
| 4. | Three weeks after No. 2 | Zinc-lime spray 8-8-100 formula: add one of the following fungicides: calcium sulphide 12½ lbs; or 16 lbs. of dry mix, either formula; or self-boiled lime-sulphur. Add two pounds of powdered lead arsenate for each 100 gallons of water. | Curculio<br>Scab |
| 5. | One month before fruit opens ripens | Calcium sulphide 12½ pounds; or 16 pounds of dry mix; or self-boiled lime-sulphur (16-16-100) to each 100 gallons of water. The Spray Service will notify growers if the zinc-lime spray is necessary in this application. | Scab<br>Brown rot |
| 6. | For late varieties only; three weeks three weeks after No. 5 | No. 6 same as No. 5 | Brown rot |

One of the most important points to be observed in spraying peaches is to use rods with angle nozzles instead of guns with high pressure. In order to properly spray a peach tree, especially when it is in heavy foliage, it is necessary to use from 300 to 325 pounds pressure with rods carrying angle nozzles. The spray should be directed upward and outward from the center of the tree. A peach tree cannot be properly sprayed by simply spraying from the outside of the tree; it must be sprayed from the center outward to the tips of the limbs.

The following materials may be used as fungicides on peaches: Dry mix, either formula; self-boiled lime and sulphur; and calcium sulphide. The zinc-lime spray is not intended as a fungicide but as a control for bacteriosis, and the prevention of arsenical injury to the twigs and foliage.

Arsenical injury and bacteriosis have become serious problems in the last few years and zinc-lime spray is recommended as the most promising material now known for their prevention. It is very important that the zinc-lime material be used in all lead arsenate applications on peaches. When this material is used for the control of bacteriosis, 5 to 7 applications are necessary. The first application should be made as soon as the petals have fallen; the others at two-week intervals thereafter. When bacteriosis is not a problem, the zinc-lime spray may be used only in the lead-arsenate applications. The zinc-lime spray may be used with the common fungicides such as calcium sulphide; dry mix, either formula; and self-boiled lime and sulphur. In

case these fungicides are used with zinc-lime spray, they should be added after the zinc-lime material has been prepared in the spray tank. When lead arsenate is used with the zinc-lime spray alone, or in combination with fungicides and the zinc-lime spray, it should be added last. The directions for preparing the zinc-lime spray are given on page 19.

In orchards where early infection by the brown rot fungus has occurred, resulting in the blighting of the blossoms and dropping of the small fruit, apply one of the summer fungicides when pink begins to show in the buds. Early blossom blight, however, is not common in Virginia, and unless it has been prevalent in the orchard in previous years, follow the regular schedule. The practice of removing the mummies from the trees and ground is a very important measure in the control of the early blight form of brown rot, as well as for the control of brown rot of the mature fruit. Orchard sanitation for the control of both insects and diseases cannot be over-emphasized.

## BROWN ROT OF PEACH AND OTHER STONE FRUITS

### Cause

The causal organism for Brown Rot fungus of stone fruits is Sclerotinia fructicola one of group called "Pezizales", which have an open-cup apothecium in which ascospores are produced.

### History

Brown Rot probably was brought from some foreign country. It was not seriously considered in the U. S. until about 1881, but has now become well established wherever the peach is grown in the U. S.

### Economic Importance

Epiphytotics of the disease occur from time to time in different sections of the country, taking a heavy toll. These occur mostly in warm moist seasons. Sultry hot weather just prior to harvest is particularly disasterous. Weather conditions of this kind are more likely to occur in the southern peach belt than in the cooler northern states, and when this occurs, large losses are encountered by the grower. Destruction is swift and whole crops may be lost within a period of two weeks. The spray program will control this disease in ordinary seasons, but where earlier sprays are not properly applied blossoms may be destroyed and prevent a good set of fruit. This is universally the most serious disease of stone fruits.

## Symptoms

Brown rot is primarily a disease of the blossoms and fruit but may also form cankers on the twigs and limbs.

First symptoms may be seen at blooming time when blossoms turn a grayish brown and wither, but do not fall. The fungus may then spread down the pedicel into the twig causing a browning and subsequent death of the twig, with the withered leaves clinging to it. It may then spread to the branch and form a canker at the point of attachment of the diseased twig to the branch. Cankers are not as commonly seen as blossom blight and fruit rot.

Fruit may be attacked in the half grown stage or at any time from then on until maturity. A smallcircular light brown discoloration first appears on the fruit. This enlarges quite rapidly to cover a considerable area. The margin of the diseased area is rather indefinite and the rotted area at first remains firm. Grayish tufts of fruiting bodies of the fungus soon appear at irregular intervals over the rotted surface; and in a few days the entire area is densely covered with these tufts, which give the surface a moldy appearance. As the rotted area enlarges to cover the entire surface of the fruit, cells collapse and the fruit shrivels into a dark brown mummy. Generally fruits that are attacked in a more mature stage of development fall to the ground, while greener fruits cling to the tree as mummies.

When a rotted fruit is in contact with sound fruits, the fungus may readily transfer itself at the point of contact and involve the entire cluster. Mummies of these clusters may cling together on the tree for two or three years. In winter when the foliage has dropped, they may be readily seen on the twigs and branches and on the ground.

## Life History

### Cycle of Infection:

Brown rot hibernates as mycelium chiefly in mummies on the ground. Early in the spring cup-shaped fruiting bodies arise on stalks from mummies. When mature these cup-shaped apothecia contain asci and ascospores, which are discharged into the air in rainy weather and alight on the blossoms, producing blossom blight. On the pedicels and other parts of blossoms, conidia arise which carry secondary infection to the fruit. As the fruit becomes rotted and falls to the ground it shrivels into mummies, carrying the fungus organism. Usually two years pass before mummies produce apothecia and primary infection.

Morphology and Pathogenesis:

This organism has a rather complicated life history in that there are two sources of primary infection. The most important of these is the sexual stage. Mummies which fall to the ground become partially covered with leaves and dirt and very early the following spring under favorable conditions of rainfall and warmth mycelium in the crust on the uppermost surface of the mummy becomes active and sends out what appears to be a sprout. This is the stalk of the spore case.

This stalk continues to grow until it reaches the surface of the soil and comes in contact with light. Its length depends upon the distance from the mummy to the surface of the soil. Upon reaching the surface this stalk becomes hollowed in the center finally forming a cup-shaped container (Fig. 37), called the apothecium. A single mummy may give rise to only one apothecium and rarely more than 35. Within the apothecium asci are developed as shown in Fig. 38, interspersed with the narrow filamentous paraphyses. Fig. 39 show an ascus with its paraphysis. Each ascus contains eight ovoid ascospores which mature about blooming time.

Infection:

When mature most of these ascospores are discharged into the air during rainy weather and alight upon the open blossoms. Here the spore sends out a simple germ tube into the blossom (Fig. 40). The germ tube branches out to form mycelial threads which invade the entire blossom causing it to turn brown and wither. My celium then grows

into the twig and ties the bloom tightly in place sothat it cannot drop. About two weeks after discharging its spores the apothecium turns brown and dies.

When the mycelium has permeated the blossom it gathers in dense intertwined masses which rupture the surface of the petiole and other parts of the blighted blossom. In this ruptured area short thick conidiaphores are sent up (Fig. 41) which branch out. On each branch a hyaline spore is formed. These conidia spores are variable in form, size and thickness of their walls (See Fig. 41 -42). Most of them are globose or almost round.

Secondary Infection:

These conidia mature when the fruit is about half grown, and are washed and blown to the fruit surface where they set up infection. A large part of this infection takes place through curculio stings, but as the fruit approaches maturity it becomes increasingly easy for the spore to enter the sound surface of the fruit. Conidia send their germ tubes into the flesh of the fruit, and these branch profusely (Fig. 42) to form mycelium which invades the surrounding tissue. As the mycelium grows between cells they are turned brown causing the brownish discoloration. At first the mycelium does not enter cells of the fruit tissue but later these cells are invaded and collapse producing the characteristic mummy (Fig. J) The mycelium then clusters and ruptures the epidermis of the rotted fruit, sending up the tufts of conidiaphores with their conidia (Fig. J). These conidia mature and carry infection to other fruits.

Hot, moist weather is quite favorable for spore formation and germination.

Mummies, which fall to the ground contain the mycelium for formation of the apothecia. Twig and limb cankers and mummies which cling to the trees carry the fungus over winter and early next spring produce conidia which may be another source of primary infection. It is not thought however that this is an important source.

Under very favorable conditions of temperature, moisture and location, mummies produce apothecia the following spring, but generally apothecia are not formed until the second spring. These mummies remain viable for several years provided they do not disintegrate. After being buried for two or three years and not producing apothecia, they may then be uncovered and form apothecia.

## Control

It is noted in the spray calendar for peaches, that generally only one spray is recommended one month before the fruit ripens. The material used should be calcium sulphide, dry mix or self-boiled lime sulphur. If later maturing varieties are to be sprayed another similar spray should be applied three weeks later. Where early blossom blight has occured, an application of spray similar to the above should be made when pink shows in the buds.

Orchard sanitation is very important in controlling this disease as well as in insect control. The spraying program should be supplemented with the removal of mummies from

the trees and ground. These mummies should be buried at least two inches under the ground. If the ground can be plowed this will destroy a large number of them.

McClintock (5) found mummies to be the most important source of primary infection.

Martin (7) states that wind is the greatest factor in dissemination, although the Japanese beetle also carries spores. Innoculation may take place without the necessity of a mechanical injury, but infection through a wound takes place more rapidly.

BROWN ROT OF STONE FRUITS - FIG. J.

1. - Rotted plums resulting from brown rot infection . White fruiting bodies may be seen on the surface.

2. - Peach blossoms browned and shrivelled from blight by brown rot infection.

3. - Partially rotted fruit on which the numerous fruiting bodies may be easily seen.

4. - Cluster of sweet cherries infected by brown rot. Some have shrivelled .

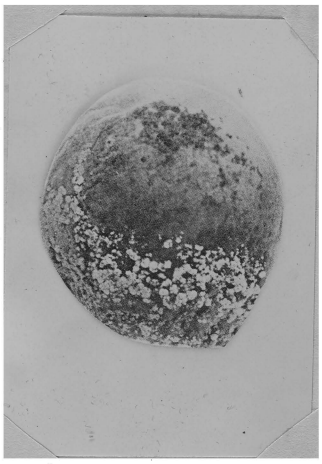
5. - Rotted peach going into the mummy state.

6. - Cluster of peaches severely infected by brown rot. Outbreaks of this kind often bring about almost complete loss of a crop.

BROWN ROT OF STONE FRUIT. FIG. J.

1.

2.



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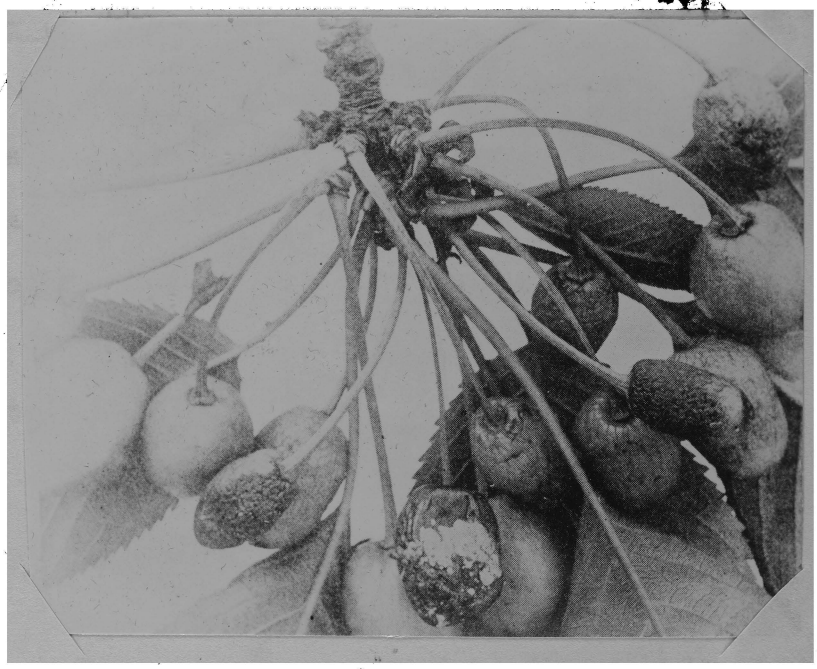


4.

5.

6.

Fig. 37- Brown rot mummy showing apothecia attached to crust by long stipes.

Fig. 38- Section of an apothecial cup showing asci or spore sacks attached to the bottom of the cup, and interspersed with paraphyses.

Fig. 39. Single ascus containing eight ascospores. Adjacent paraphysis.

Fig. 40. Germinating ascospore.

Fig. 41. Ruptured surface tissue in which spore stalks have arisen, bearing conidia or secondary summer spores.

Fig. 42- Conidium and type of germination.

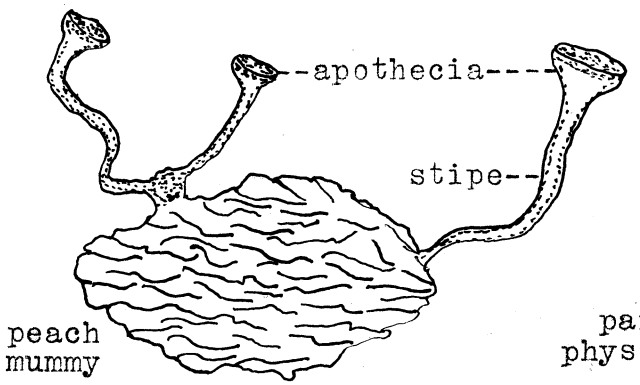



FIG. 37

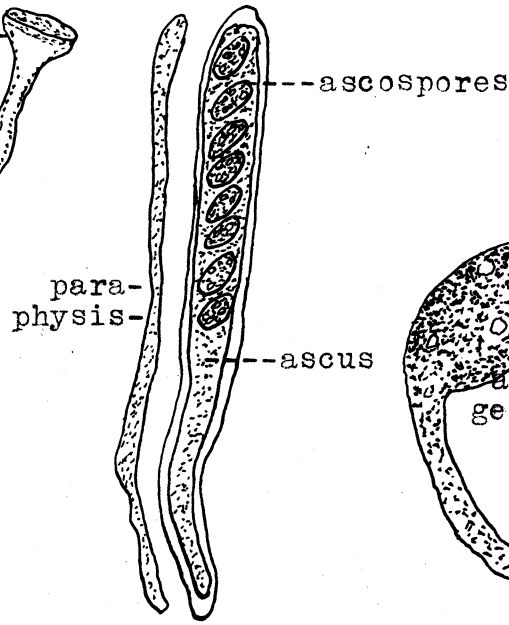



FIG. 39

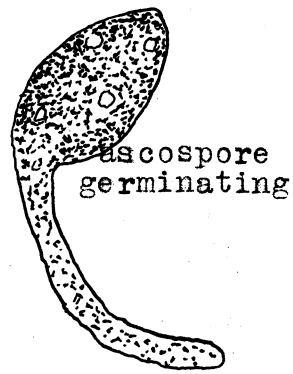



FIG. 40

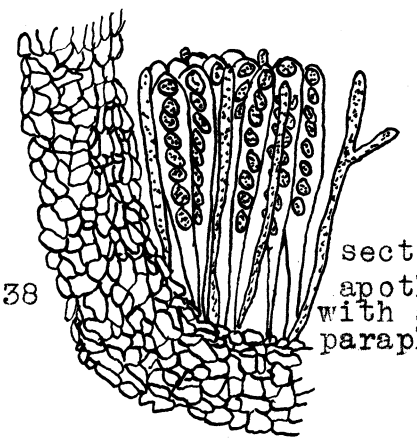


FIG. 38 section of apothecium with asci and paraphyses.

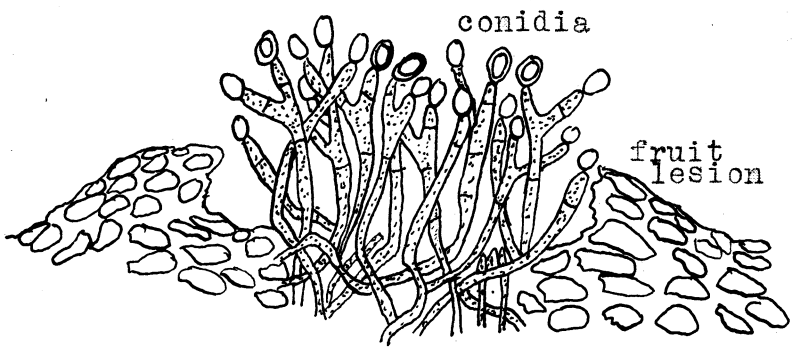
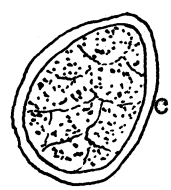



FIG. 41



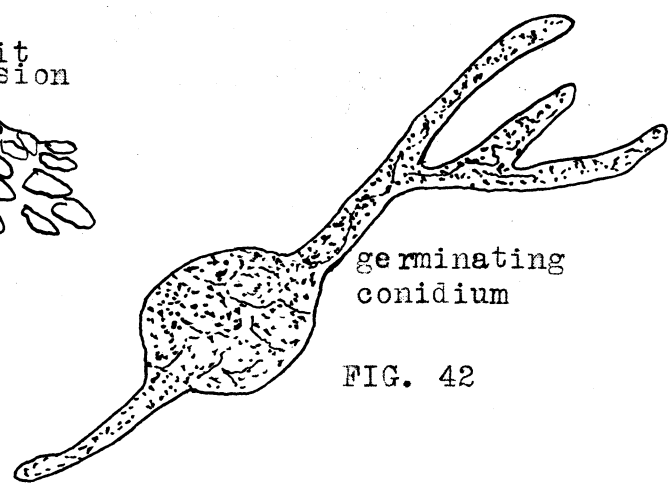




FIG. 42

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## PEACH SCAB

### Hosts

This is a disease primarily of the Peach, Apricot and nectarine.

### History

Peach scab was first described in Austria in 1877, but did not attract much attention in America until 1889, when it was prevalent in Indiana. Investigations in its life history were numerous until in 1909 Scott found that self boiled lime-sulphur satisfactorily controlled this disease. Subsequent work has added to knowledge of its life history.

### Geographical Occurrence

This disease occurs in the U. S. practically everywhere the peach is grown east of the Rocky mountains.

### Economic Importance

If not properly controlled this disease may cause tremendous losses, especially to mid season and late varieties. Fruit suffers the greatest injury, market value of scabby fruit is greatly reduced and in cases of severe infection where cracking takes place, brown rot may follow. Spraying schedules on peaches are so thorough, that scab is readily controlled if spraying is properly done. However it constitutes a serious menace and omission of sprays may be followed by severe infections and losses.

## Symptoms

Fruit:

First appearances of the disease on fruit are small, circular, greenish to brownish areas on the green fruit. These may be numerous, and as they enlarge may converge and make almost a solid covering. As the spots enlarge the color becomes brown to almost black. Spots appearing on the blushed cheek may prevent the formation of the normal red color. These usually appear as brown spots with a greenish margin, which contrasts with the surrounding pink tissue. The skin beneath the spot becomes corky and as the fruit continues to grow in size, cracks may develop where the corky tissue checks growth. As the spots become older and darker, the fuzz dies and disappears and the margins become more definite. The lesions resemble "freckles", except that they are dark. Most of the spots are found on the fruit surface which is most subject to wetting by rainfall and heavy dew, especially portions next to the stem and on down the side of the peach.

Twigs:

Slightly raised, indefinite, circular or oval greenish areas is the first sign of the disease on the young growing twigs. These later turn light brown and finally dark brown. As the spot enlarges, a raised margin of normal green color marks its advance. The interior is brown and level. By the end of the growing season

the spot is seen as a smooth, more or less oval, brown lesion, level with the twig surface and bordered by a slightly raised purplish area, which shades and blends with the surrounding healthy tissue. These are the cankers which carry the fungus mycelium over winter. Early the following spring brownish spore tufts appear on the surface of the cankers. Cankers enlarge slightly during the spring and early summer, but later are obliterated by growth of the bark and rarely survive the second winter. These cankers are found distributed at irregular intervals over the surface of the twigs.

Leaves:

Lesions on the leaves are not so pronounced as on the fruit and twigs, but are first seen as small angular to circular spots almost the same color as the surrounding healthy tissue, on the under surface of the leaves. Formation of olivaceous conidiaphores and conidia soon give the diseased area a greenish-brown color. Later the upper surface of the leaf opposite the lesion shows a pale yellow or purplish color. Lesions on the mid rib and petiole are much the same as those on the shoots. Lesions are scattered at irregular intervals over the leaf surface and may be so numerous as to form almost solid splotches.

## Life History

Cycle of Infection:

Peach scab passes the winter as dark brown, round, thick walled cells of mycelium on infected twigs. In the spring conidiaphores and conidia arise from these cells, and about blooming

time are washed and splashed to the under surfaces of leaves and to surfaces of twigs. Here they germinate setting up infection. Later primary infection takes place on the fruit. About 35 to 45 days elapse between infection and appearance of the disease spots. Due to this long incubation period, secondary infection is of minor importance, except on late varieties. Lesions on twigs carry the mycelium over winter. In the fall dark brown cells are formed which remain in the margin of lesion cankers over winter, ready to produce primary infection the following spring.

Morphology and Pathogenesis -

Primary Inoculum:

The peach scab organism passes the winter as thick-walled mycelium in cankers on twigs (Fig. 43). In the spring this mycelium sends up conidiaphores upon which are borne elliptical, one or two celled conidia (Fig. 44). They are produced singly or in chains on the surface of the canker and in the presence of moisture separate readily from the conidiaphores and are washed downward to the pedicels of leaves and fruits about blooming time. Moisture may splash the spores directly to the surface of the leaves, twigs and fruit or they may be washed down the pedicels and spread out over the wet surface of the fruit and leaves.

Infection:

On the surface of the leaves and twigs these spores germinate by sending out one to three germ tubes, usually from near the ends of the spore (Fig. 44) . These germ tubes become closely oppressed to the surface of the host tissue and send out infection threads or hyphae, which may directly penetrate the tissue or may enter the natural openings. Leaves are almost exclusively infected on their lower surfaces.

Conidia are lodged in the fuzz of the fruit and the germ tubes cling to the fruit surface at the base of the hairs, Fig. 45, entering the cuticle through the basin in which the hair grows. Penetration of the host tissue is not deep. Mycelium spreads out between the cuticle and the epidermal cells, and sets up an irritation which causes formation of the characteristic corky tissue (Fig. 46) Mycelium is at first delicate, thin walled, hyaline and branched (Fig. 47). Later it broadens and the cells walls thicken, becoming brownish and imparting the characteristic brown spotting of the tissue. Finally it turns quite dark.

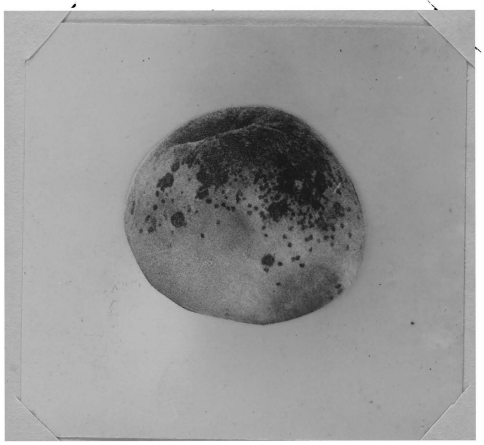
Twig cankers may continue to produce conidia until in early summer, but most of the infection occurs earlier in the season when moisture conditions are more favorable for spore formation and germination. Secondary infection is of little importance, since the period is so long between infection and formation of secondary spores, that the season is well advanced before secondary spores are mature. Fruit twigs and leaves may

all be infected with primary inoculum of conidia from cankers. The first infection usually takes place about blooming time or shortly thereafter, and from 35 to 45 or more days elapse until spots may be seen with the naked eye. Usually the first symptoms appear on the fruit just before ripening date for Carmen, one of the earlier varieties. At this time, it appears on the almost mature Carmen and slightly later on the green fruits of the later varieties. The fruit is harvested and the leaves drop and become decomposed during winter, but the mycelium remains in the twigs along the margins of canker lesions. In the spring this mycelium gives rise to conidia which initiate primary infection.

## Control

As mentioned before scab is readily controlled by application of any of the fungicides commonly used on peaches such as self-boiled lime-sulphur, dry mix and calcium sulphide. In the spray calendar for Virginia the first scab spray is applied about one month after the petal fall spray and the second about one month before fruit ripens. For late varieties such as Smock, Salway and Krummel's an extra spray is recommended three weeks after the second spray. Late varieties are exposed to infection over a longer period of time and hence are liable to heavier infection, if not properly sprayed.

# PEACH SCAB



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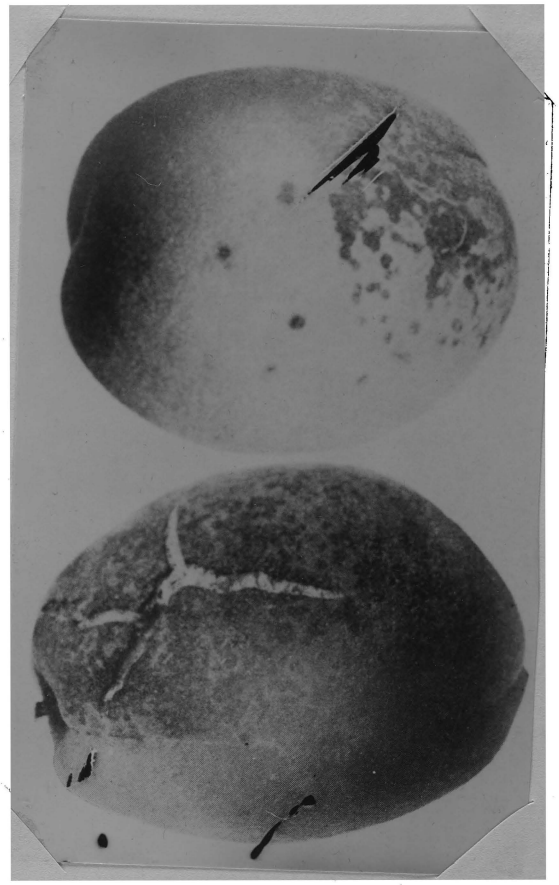


2.

1. - Scab lesions on green peach as commonly seen around the stem end. Some are so numerous as to make almost a solid dark color.

2. - Lesions are so numerous that this peach has cracked open.

Fig. 43- Section of canker lesion showing dark brown mycelial cells from which spore stalks arise. These brown cells constitute the winter resting stage.

Fig. 44- Conidia from 43, showing type of germination.

Fig. 45- Section of fruit showing a single hair of the fuzz with the germ tube of the spore closely oppressed at its base.

Fig. 46- Characteristic cork layer formed on fruit lesions.

Fig. 47- Infection of a leaf by the germ tube of a conidium.

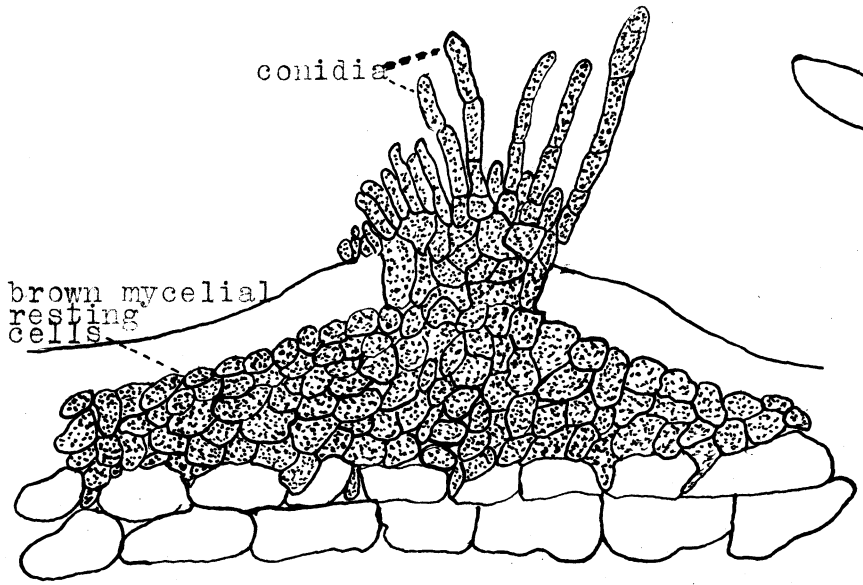



FIG. 43

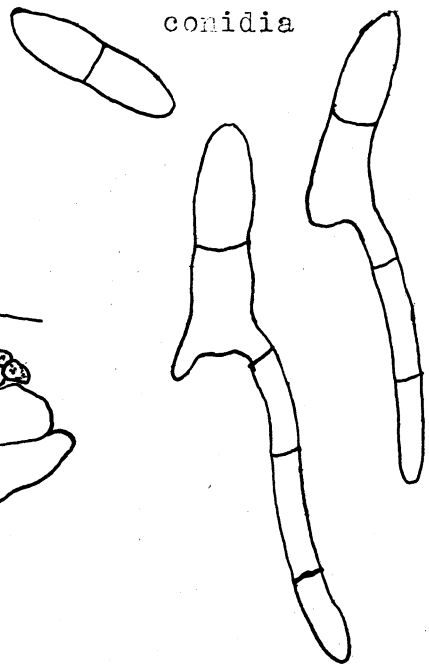



FIG. 44

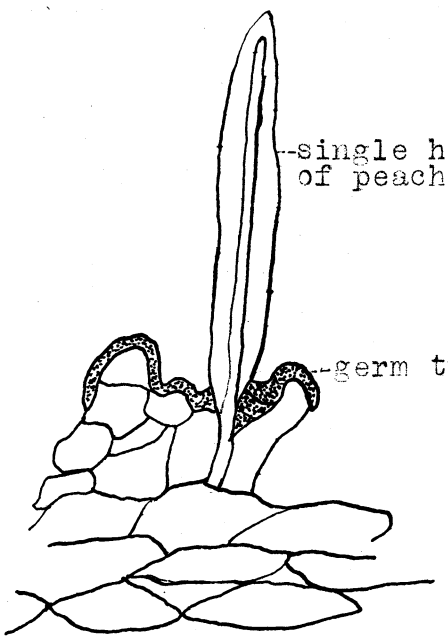



FIG. 45

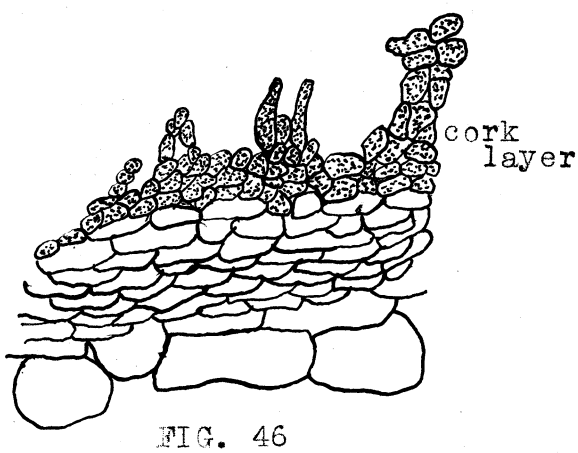
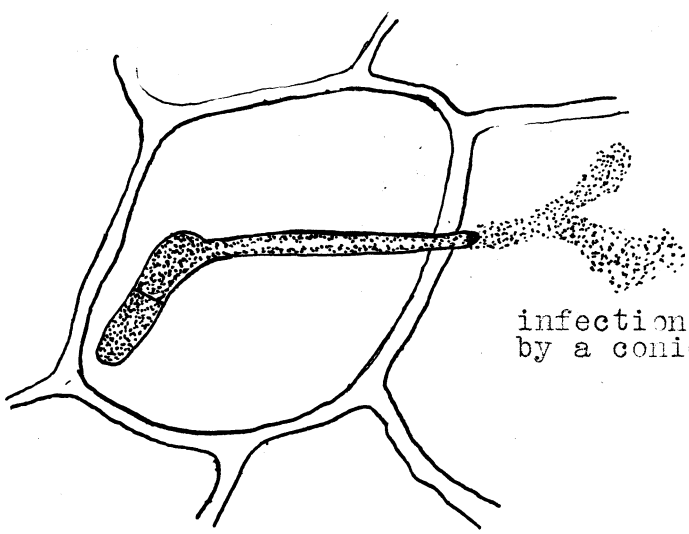



FIG. 46

FIG. 47



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## PEACH LEAF CURL

### Hosts

Peach, Nectarine and peach almond.

### History

This disease was reported in England as early as 1821 however the country in which it originated is not known.

### Geographical Distribution

The disease is known in practically every region in which peaches are grown, being common to Europe, China, Japan, Australia and Africa. In the U. S. it occurs in all portions, but is most serious on the Pacific Coast and in the Great Lakes region.

### Economic Importance

Leaf curl is rated as one of the most important peach diseases due to its damage to the tree, when not checked. It can be kept well under control by spraying, but in seasons especially favorable to its development, spraying may not effectively control it. Also some varieties are very susceptible, Elberta and J. H. Hale being notoriously so. In Virginia the diseased does little damage in well sprayed orchards in the average year, being easily controlled by a dormant spray of Lime Sulphur.

### Symptoms

Symptoms of this disease are characteristic and well known. The most common infection is on leaves, although young shoots and fruit may also be involved. Fig. K shows the abnormal

swollen, thickened and distorted leaves with their sickly yellowish white color, commonly seen on unsprayed Elberta trees. Flowers and young fruit may also be affected, but as these soon drop, the type of infection is not generally observed. Diseased areas may be small or may involve the whole leaf. Where a large portion of the leaf is included, the leaf falls in a few weeks time. Leaves with smaller diseased areas may persist on the tree. Usually in May the spore sack containing the fruiting spores, make their appearance on the surface of the leaves. Giving the leaves a white frosted appearance. As the diseased leaves fall in large numbers, the trees appear quite bare. Vigorous trees will send out new foliage to replace that which has fallen, but the drain on the tree is likely to cause dropping of the green fruit or at best a crop of mediocre sized fruits. Also the chances of fruit bud formation for next years crop are materially reduced. Young shoots infected with leaf curl, show much the same general type of swelling and distortion and usually die.

## Life History

The scientific name of the disease is descriptive in that Exoascus refers to the fact that the asci (or spore sacs) are borne uncovered on the surface of the leaf rather than within the tissue. Deformans describe the characteristic deformity of the leaves.

It is generally believed that the organism over winters as spores in the scales of buds. When buds swell in the spring, spores germinate sending a germ tube into the leaf tissue of the tender leaves within, thus establishing itself before leaves open. The root-like mycelium penetrates between cell walls filling the intercellular spaces. Some sort of an irritation causes an increase in the number and size of the palisade cells in the infected area, bringing about the puffing and thickening of the leaf tissue. Fig. 48 shows a section of healthy and diseased tissue. The numerous small green bodies (Chloroplastids) which fill the cells of healthy leaves, and, which play an important part in food manufacture, are destroyed by the disease organism. The result of this is the yellowish color of the diseased parts. The mycelium finally pushes up between the epidermal cells and grows out under the cuticle, or thin upper surface of the leaf. Here it branches profusely in all directions, and the hyphae, which grow toward the upper surface of leaf form the asci or spore sacs which project from the surface of the leaf (Fig. 49). These asci are so numerous as to give the diseased surface a white frosty appearance. In each ascus is borne eight globose spores. These frequently increase in number by budding so that some asci contain a larger number of small spores. When the ascospores ripen, the upper end bursts open and discharges spores into the air. This spore discharge usually takes place in May. It is assumed that these spores alight at the base of the leaf petioles, become involved in the bud as it is formed and remain dormant until the following

year. However, this is supposition, and, what actually takes place from spore formation until infection the following spring, is not definitely known.

Factors which favor abundance of infection are superabundance of moisture and cool damp conditions. These same conditions render tissue of the peach leaf more succulent, and hence more susceptible to attack of the fungus.

## Control

Effective control of this disease is secured from the dormant spray of Lime Sulphur generally used for scale. On such susceptible varieties as Elberta and J. H. Hale, application of spray should be thoroughly made before the buds have swelled to any extent in the spring. Commercial peach growers, who regularly apply the dormant lime sulphur spray for scale insects, are rarely troubled with Leaf Curl. If it is desired to use oil in the dormant spray for severe scale attacks, lime sulphur must also be used to control leaf curl. A spray of one part of Lime Sulphur to 15 parts of water is sufficient, to control leaf curl but the oil must be used in regular amounts to control scale. A combination spray of Bordeaux and oil emulsion also proves satisfactory. One and one-half gallons of cold mix oil emulsion to 50 gallons of 3-4-100 Bordeaux mixture was found to give control.

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PEACH LEAF CURL - FIG. K

CURLED, puffed and swollen leaves on the right are infected with leaf curl. These diseased areas become a sickly yellowish white or pinkish in later stages. Enlargement of leaf cells and destruction of the green chloroplastids brings about these symptoms.

Fig. 48- Sections of a peach leaf showing diseased and healthy tissue. Note the enlargement of the diseased cells and destruction of the chloroplastids.

Fig. 49- Asci with their ascospores borne or the leaf and twig surfaces.

PEACH LEAF CURL

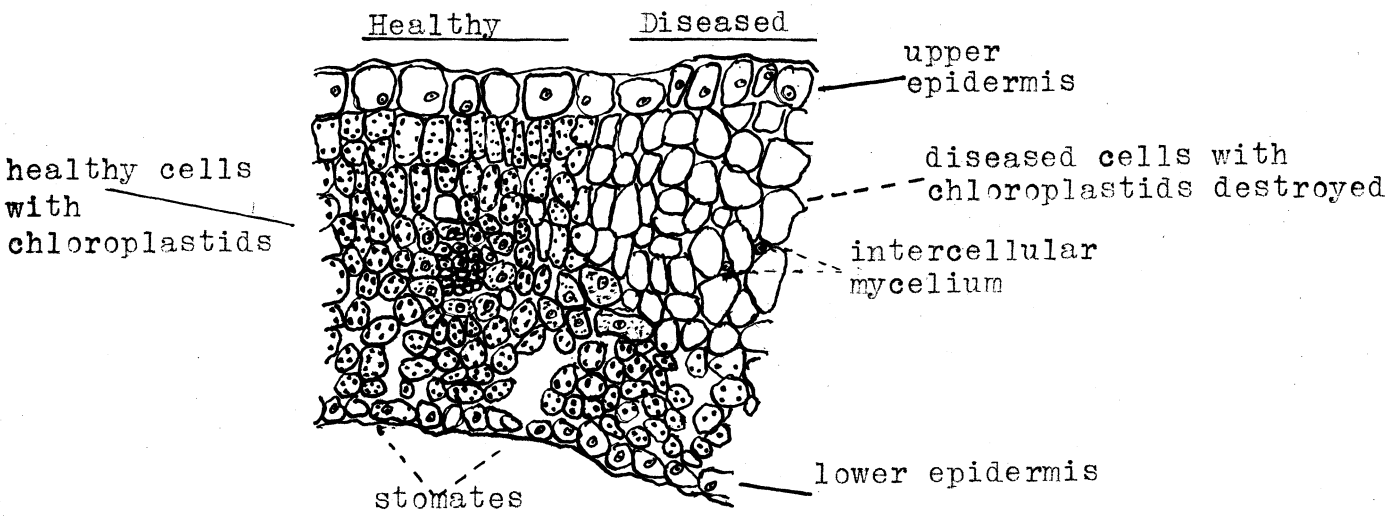



Fig. 48

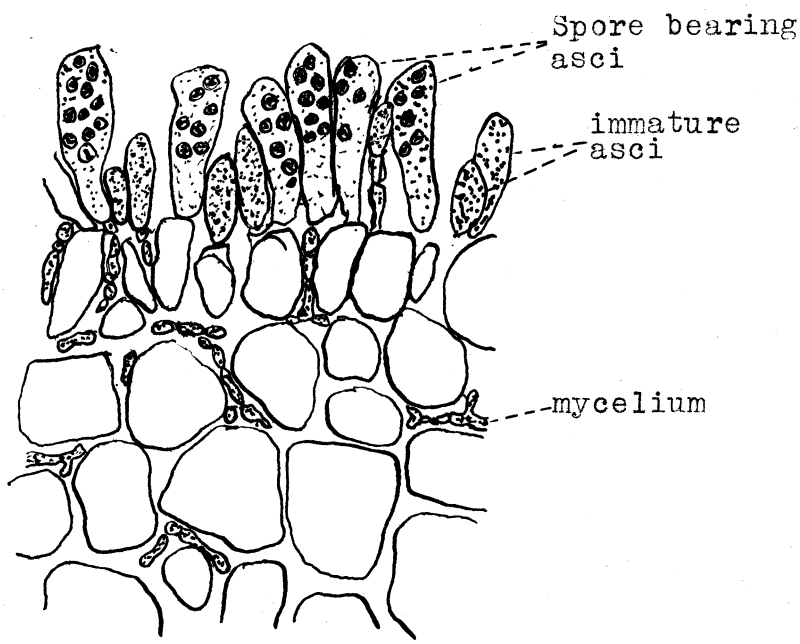



FIG. 49

## BACTERIAL SPOT OF STONE FRUITS

### Cause

This disease is caused by a bacterial organism called Bacterium pruni.

### Hosts

Forms commonly attacked are peach, plum, nectarine and apricot. In general infection is more severe on plums and nectarines than on peach, although varietal differences is quite marked.

### History

History of the disease is rather confusing due to the fact that it was confined with the fungus shot hole disease. Apparently it is of American origin, not having been found in foreign countries. It was reported by Smith, E. F. in 1903.

### Geographical Distribution

It is found in practically all the peach growing sections of the country where the climate is sufficiently humid.

### Economic Importance

Bacterial spot has been very serious in past years in moist climates and on susceptible forms and varieties. Moist seasons especially with warm spring weather are ideal for serious attacks, while cool dry seasons usually retard its seriousness. Japanese plume, Hale and Elberta peach and the nectarines are particularly susceptible and since foliage, twigs, limbs and fruit are attacked, the seriousness of the attack can readily be appreciated. Until quite recently no successful control has been

obtained. However recent control measures have considerably reduced the damage from this disease. Before control measures were applied it was estimated that from 25 to 75% of the fruit was infected in years favorable to infection. Defoliation with consequent weakening of the buds and general vigor of the tree also was an item of considerable importance. In 1929 estimated losses were from 1 to 17% in well cared for orchards. In 1930, a dry year, losses ranged from 1 to 2%. In 1931 losses were considerably reduced by spraying.

Hale and Elberta, two of the most important commercial varieties are quite subject to this disease, but good commercial control may be had if a rigid spray program is adhered to. See control.

Symptoms

The first symptom may be seen generally in May as a gray spotting of leaves. These spots quickly turn purple and enlarge becoming a very deep purple to almost black. The purple coloring is on the under side of the leaf and may be distinguished from the fungus shot hole in that the fungus disease forms white fruiting bodies on the lesions. The bacterial lesion is somewhat angular in outline and as the tissue dies it shrinks and falls out leaving a shot hole effect. When these lesions are numerous, they coalesce and may involve large areas of the leaf. Badly diseased leaves soon fall to the ground and in severe cases 25 to 75% of the foliage may drop from the tree or even more. Usually the leaves, which are infected earliest

in the season are the first to fall. As the leaves fall, a tufted appearance is given tothe ends of the branches caused by the younger leaves hanging later at these points.

Canker formation generally does not take place until in late May or June. On the new shoots raised water-soaked areas may be seen surrounding the lenticels. These are small at first but soon enlarge and lengthen considerably in proportion to their width. Some of these spots may converge and cover such large areas of the twig that it is girdled and deadened. Generally however, these spots remain somewhat small. With age they become sunken,turn brown or purplish brown, then dark brown and finally purplish black or black. Generally these spots grow over and disappear the second year. On plums, however, many of these cankers become perennial, and remain from year to year. Young trees usually suffer more during the first few years than do older trees. They become defoliated and twigs turn black and die and the tree is stunted, bearing small crops of inferior fruit.

About the middle of May numerous gray specks may be seen just beneath the outer skin of the fruit. These soon take on the characteristic water soaked appearance followed by a purple color. Angular cracks appear in the skins, giving the surface a chapped appearance. If these cracks are numerous, they may join together toform a network and extend almost the entire length of the fruit. Cracks are usually not very wide or deep, being more of a surface injury. Certain varieties of nectarine

may have deep wide cracks which are sunken and black. Rots generally enter these cracks and cause decay of the green fruit.

Most of the infected fruits are second grade or culls.

## Life History

### Morphology and Histology:

Bacterium pruni is a rod shaped bacterium rounded at both ends, which forms a yellow colony on culture medium. Under favorable conditions about seven days from time of infection the gray specks appear on the leaves. In ten days the water soaked spots appear. About seven days are required before the water-soaked spots form on the twigs and about 25 days before the spots turn brown. The short rod-like bacteria have from one to several hair-like appendages (flagella) which have a polar attachment. Power of locomotion is attained in this manner.

So far as is known the organism passes its entire life history within the host except for the time necessary to transfer from one point to another. It is generally thought that twig and limb cankers are the source of primary infection in the spring, and that infected buds may harbor the bacteria during winters. There is a possibility that curculio and aphis, high winds and dashing rains may serve to spread the organism from one tree to another. In the presence of water bacteria move to the stomates and lenticels enter the opening and begin to multiply by simple division. At first they remain in the sub stomatal cavity, but soon secrete an enzyme which dissolves the cell walls allowing the bacteria access to the cell contents.

As they multiply and dissolve the cell walls, a cavity is produced which is full of bacteria; and gives the water-soaked appearance. In about ten days or two weeks bacteria may ooze from the stomata or cracks in the fruit, or they may go deeper into the tissue forming wider cracks as the fruit continues growth.

Bacteria invade the leaf somewhat similarly through the stomates on the under side. Water-soaked areas appear a day or two sooner than on the fruit and the ooze comes out sooner.

In the tissue of the shoots apparently travel is more difficult and development of the external symptoms is slower. As the tissue toughens activity of the bacteria slows up and the lesion often heal over during the second year. There are living bacteria in the cankers however, and here they remain dormant during winter until spring weather starts activity and they multiply and produce primory infection. Ooze from infected leaves, fruits and cankers is a source of infection during the spring and early summer.

Warm slow rains in which the tree surface remains moist for a considerable time are ideal conditions for infection. Cool or dry weather holds the organism in check. Drying is fatal to bacteria. Heavy dews favor innoculation, and shade is favorable while sunlight is fatal.

Vigorous well pruned trees are more capable of withstanding attack than less vigorous trees.

## Varietal Susceptibility

Abundance and Burbank plums, most of the nectarines, Royal apricot and Hale and Elberta peach are particularly susceptible. Variations in seasonal conditions cause a difference inthe degree of infection of fruits and leaves.

## Control

A zinc-lime spray has been recently discovered as the most satisfactory material yet found for control of this disease. The formula is as follows:-

| | |
|---|---|
| Zinc Sulphate | 8 pounds |
| Hydrated Lime | 8 pounds |
| Water | 100 gallons |

The zinc sulphate is slowly added to two-thirds of a tank of water with the agitator running. When this is thoroughly dissolved, mix the lime with water to make a thin paste. Wash this paste through the strainer into the tank. Continue agitation until it is thoroughly mixed. If a fungicide is used with this, add calcium sulphide, dry mix or self-boiled lime sulphur last. Combination sprays should be used as soon asmade. The first application should be made as soon as the flower petals have fallen, followed at two week intervals by four to six additional sprays.

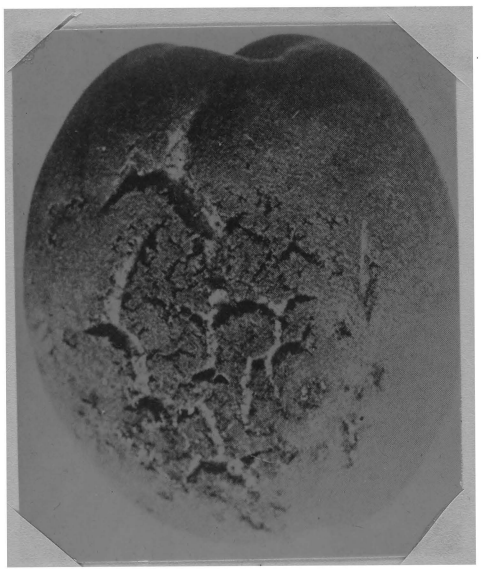
Cultivation and the application of nitrogen fertilizers may be helpful as a supplement to the above sprays. It is necessary that the under sides of the leaves be covered.

Pool (4) found that leaf infection occurs one month later than fruit infection. Peaches borne on branches below, above or at the same level as the cankers were equally infected.

# BACTERIAL SPOT OF PEACH

1.

2.



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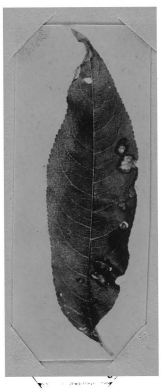

4.

1. - Angular holes in leaf where deadened tissue has fallen out.

2. - Spots on fruit and leaf .

3. - Severely infected peach upon which numerous cracks have resulted from large numbers of spots.

4. - Spray injury sometimes confused with bacterial spot . There is generally some margin injury along with burn spots.

Fig. 50. Rod-like bacteria with their hair-like flagella

Fig. 51. Bacteria concentrated in large numbers in the leaf tissue. Note the destruction of cells. Diagrammatic.

Fig. 52. Bacteria concentrated in the sub-stomatal cavity of fruit. Note some cells are in the process of being broken up.

Fig. 53. Section of a cankered twig showing bacterial pockets and cavities formed in the bark tissue.

BACTERIAL SPOT OF PEACH

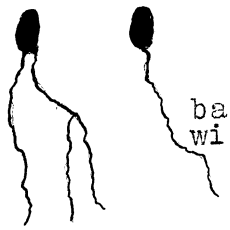


FIG. 50

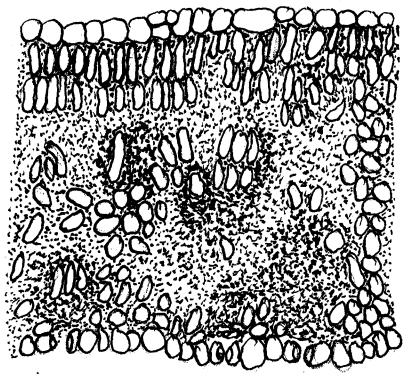


FIG. 51

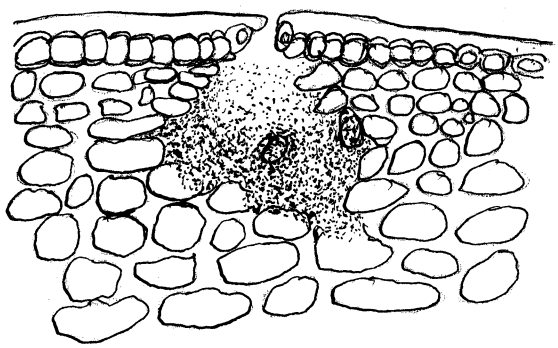


FIG. 52

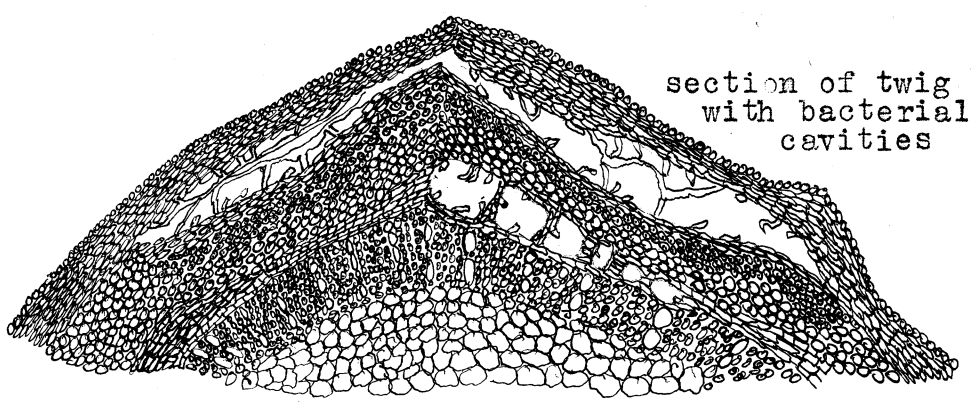


FIG. 53

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## PART III

## PLUM DISEASES

As with peaches, brown rot is the most important plum diseases with black knot probably second and leaf spot least important of the three studied.

## SPRAY CALENDAR FOR PLUMS

| No. | Time of application | Materials to use | Parasites |
|---|---|---|---|
| 1 | Dormant season | Lime-sulphur, 32°Baume, 12 gallons; add water to make 100 gallons of spray | Scale and general clean up |
| 2 | As soon as petals fall | Lime-sulphur, 32°Baume, 12 quarts, and water to make 100 gallons of spray; add two pounds powdered lead arsenate to each 100 gallons of solution. | Curculio Leaf spot |
| 3 | One week after No. 2 | Same as in No. 2 | Curculio Leaf spot |
| 4 | Three weeks after No. 2 | Same as in No. 2 | Curculio Leaf spot |
| 5 | One month before fruit ripens | Self-boiled lime and sulphur | Brown rot and other fungous diseases |

## BROWN ROT OF PLUM

### Hosts

Plum, peach, cherry, nectarine, apricot are all affected by this disease, which is described for peaches, page 73.

### Economic Importance

This is the most serious disease of the plum, and while plums are usually not so generally affected as peaches, it is a serious menace. In years favorable to infection as much as 95% of the fruit on unsprayed trees may be rotted. In 1928 losses from brown rot were reported from .5% in Delaware to 5% in Virginia and as high as 30% in New Hampshire. Varieties differ somewhat in susceptibility especially in the green fruits. Williaman, Pervier and Triebold (1) found that a decrease in skin toughness as ripeness advances is more marked in susceptible varieties. Firmness of flesh and toughness of skin go together, and are important factors in varietal resistance. When ripe all varieties are susceptible. Japanese plums, such as Abundance and Burbank are thought to be more susceptible to rot of the green fruits. This disease is ordinarily well controlled by a spray applied about one month before the fruit ripens. However as with peaches hot damp weather just before ripening may take a heavy toll.

For further description of symptoms, life history, etc. see brown rot of peaches, pages 73-81.

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## BLACK KNOT OF PLUM AND CHERRY

### Hosts

Black Knot is caused by the fungus organism Plowrightia morbosa hosts of which are the plum and cherry.

### History and Geographical Distribution

Records show that this disease was found abundantly in the north Atlantic states many years ago on wild plums and cherries. It is probable that the disease may have first affected cultivated fruits about 1800. It was found only in the eastern part of the U. S. until about 1879, when it spread westward. Now practically the whole country as well as part of Canada is included in its range.

### Economic Importance

Plums appear to suffer worst from this disease, although sour cherries are frequently badly damaged. The nature of the loss is that of death of twigs, branches and sometimes the trunk of the tree. Damsons are particularly susceptible and once the fungus organism becomes established on them death of the branches soon takes place. Proper eradication methods along with spraying will hold the disease somewhat in check, but even so, losses are of economic importance.

### Symptoms

As the name implies this disease causes the formation of knots on the twigs, branches and trunks of the tree. These knots may be found on one side of a branch or they may encircle the whole branch. In the former case the function of the branch is impaired. In the latter the branch dies. These knots may be

extended well along the branch, or they may be confined to a thick short knot. During the earlier stages, these knots appear as swellings of the tissue and the bark remains smooth. As the knot enlarges the cambium is bursted and a brownish velvety covering appears on the exposed surface. At this stage the gall is solid but somewhat puffy. Later the knot turns black and becomes brittle and rough and is characterized by many lobes and fissures. Contiguous secondary galls may be formed above or below the main gall. The interior of the older knot becomes honey-combed and gum exudes. Finally there is a pink mold formed on the surface and frequently insects destroy the interior portion leaving only a black hollow shell.

These galls may be found on different portions of the tree. Bulbous growths may be formed at the tip of twigs. Knots may appear in the crotches where two twigs join, on the sides, or tips of fruiting spores, in the leaf axils, on the sides of larger limbs and trunks, and in the crotches of older limbs. The longer knots generally extend spirally, along the branch. Some small twigs are bent at right angles away from the knot. This is brought about by growth pressure of the knot.

## Life History

Two years are required for the completion of the life history of the fungus. Primary spring infection proceeds from two year old knots. Perithecia, which are imbedded in the surface of the black gall, contain ascospores that are disseminated in April. These ascospores lodge in the places mentioned before and germinate sending germ tubes into the cambium layer between the

bark and wood. Mycelial threads from this tube ramify the cambium during the spring and summer setting up an irritation, which causes the tissue to form a large amount of callus. This process is quite slow and the swelling is not so noticeable until toward autumn. The following winter growth is arrested, but very early the second spring becomes pronounced and vigorous rupturing the cambium and bark, and exposing a yellowish surface. Upon this surface conidiaphores or spore stalks are sent up from the mycelium. Each conidiaphore bears an olive brown velvety conidium or summer spore. These are so numerous as to impart a brown velvety appearance to the surface. When the spores are mature, constriction takes place attheir bases and the spores are disengaged from their stalks and carried to the parts mentioned, bringing about a secondary infection.

After the spores are discharged, the brown mycelium turns black and dies. This gives the knot its hard brittle texture. As the season advances the living mycelial threads become entangled and form the perithecium or cavity in which the fungus lives over winter.

During fall and winter these perithecia remain inactive, but with the first warm days of spring ascospores are formed in the asci or sacs within the perithecium. The apex or top of the perithecium is often depressed. In the center at the apex is a small opening called the ostiolum. When the ascospores are mature usually sometime in April, rainy weather softens and swells the perithecia, which open and discharge their ascospores into the

air. Thus it will be seen that two years are required from the time of primary infection until the formation of ascospores for another primary infection. However in the intervening year conidia produce a secondary infection.

## Control

No absolutely satisfactory control method has been developed. The ordinary spray program usually recommended for plums will help somewhat in holding this disease in check. However, when it has once become established it is advisable to cut out the galls and destroy them. A supplement to this is the application of some fungicide starting in March and continuing through May at two to three weeks intervals. Unless all the galls in the vicinity are removed, infections will continue to occur. Spraying is perhaps the most economical practice.

BLACK KNOT OF PLUM - FIG. L.

Black, carbonaceous galls with crevices. These are usually formed on one side of a branch. The large gall in the center has extended practically around the branch. The upper gall started near the crotch of a small twig.

Fig. 54. Conidia of Plum black knot borne on septated spore stalks. Note varying degree of thickness of cell walls of the conidia.

Fig. 55. Conidium germinating by a simple germ tube.

Fig. 56. Perithecium with asci and paraphyses. Note their attachment around inner wall of perithecium.

Fig. 57. Asci with eight ascospores and adjacent paraphyses. Note the pecular septation toward the end of each spore.

Fig. 58. Manner of germination of an ascospore.

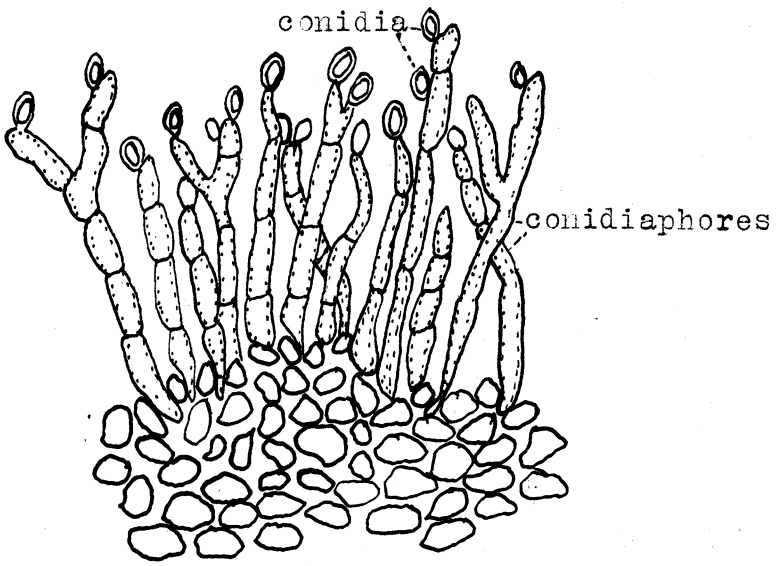



FIG. 54

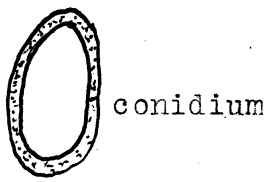
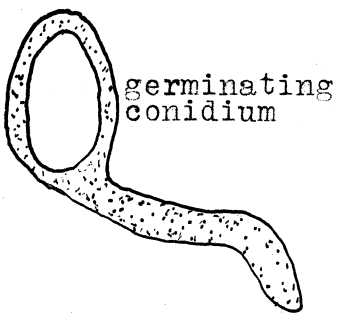



FIG. 55



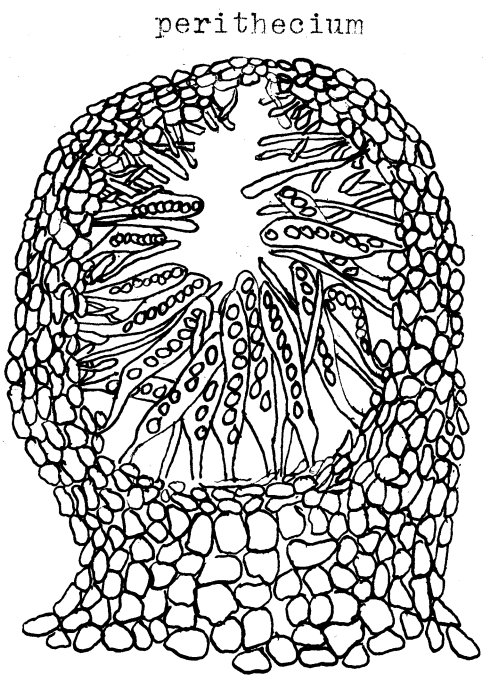




FIG. 56

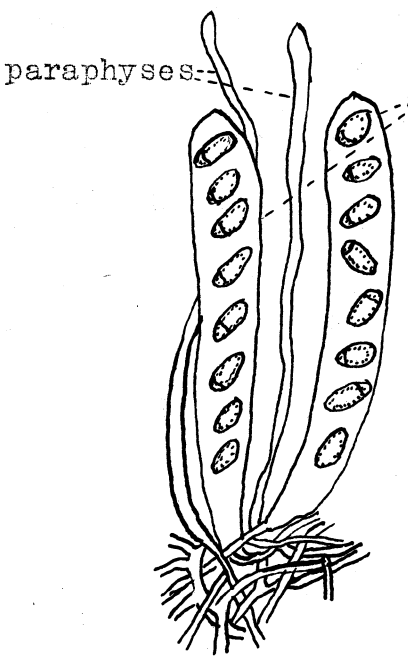



FIG. 57

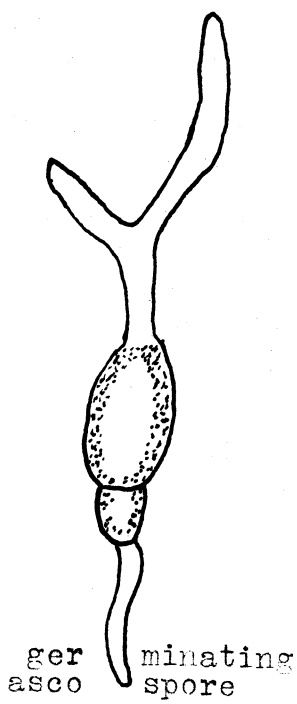



FIG. 58

## LEAF BLIGHT OF PLUM

### Hosts

This organism affects the plum and is very similar to C. hiemalis of the cherry.

### Economic Importance

Leaf-spot, shot-hole and yellow-leaf are synonyms for leaf blight. European varieties of plum such as Bradshaw, Gage, Lombard, etc. are more susceptible than the Japanese group. In some seasons unsprayed trees may have their leaves riddled with holes or sometimes the leaves may turn yellow and drop. Plums as a rule are more subject to shot-holing than the cherry and less subject to yellowing. Losses have been estimated as high as 30% in commercial orchards in New York.

### Control

Three sprays of commercial concentrated lime sulphur, 12 quarts to 100 gals. of water have been recommended to control this disease in Virginia as follows: (1) as soon as petals fall (2) one week later (3) four weeks after petal fall. If properly applied these sprays will satisfactorily control leaf spot.

For symptoms, life history, etc. see Cherry leaf spot, coccomyces hiemalis on page 116.

These diseases are verysimilar in respect to their relation to the host.

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# PART IV

# CHERRY DISEASES

## SPRAY CALENDAR FOR CHERRIES

| No. | Time of Application | Materials to use | Parasites |
|---|---|---|---|
| 1 | Dormant season | Lime-sulphur, 32°Baume 12 gallons; add water to make 100 gallons of spray | Scale |
| 2 | Immediately after petals fall | Lime-sulphur, 32°Baume: Sour Cherry, 12 quarts and water to make 100 gallons of spray. Sweet Cherry, 10 quarts and water to make 100 gallons of spray Add 2 pounds powdered lead arsenate to each 100 gallons of solution | Leaf spot Curculio |
| 3 | One week after #2 | Same as in No. 2 | Leaf spot Curculio |
| 4 | Three weeks after No. 3 | Same as in No. 2 | Leaf spot Curculio Brown rot |
| 5 | Immediately after fruit is harvested | Same as in No.2, but omit the leaf arsenate | Leaf spot |

## BROWN ROT OF CHERRY

### Hosts

This is the same fungus disease which attacks the peach and plum.

### Economic Importance

Brown rot is the most destructive of all cherry diseases just as it is with plum and peach. Fruit is most often rotted just prior to picking but the rot frequently becomes serious in packed cherries intransit or on the market. Blossoms may be killed causing a poor set of fruit. Leaves and twigs are affected to a much lesser extent. Cherries generally are not affected as much as peaches and plums. Wax cherries, such as Napoleon and Governor Wood, as well as Bing and Black Tartarian are generally considered quite susceptible. Such varieties as Lambert and the sour Montmorency are more resistant. When hot damp weather precedes harvest all varieties are more or less susceptible. In Virginia brown rot is considered the most common cause of loss. Estimated losses in 1929 were 3.5% in Virginia and ashigh as 40% in Tennessee for commercial plantings.

### Control

The Virginia Spray Service recommends a spray one month after petal fall on the green fruit for control. If brown rot has been severe in previous years, a spray should be applied just before the blossoms open to prevent blossom blight, the second when the fruit is about the size of a pea and again about three weeks before the fruit is ripe. Concentrated lime-sulphur 12 quarts to 100 gallons of water is used on soul cherries and 10 quarts to 100 gallons of water on sweet cherries. Proper control of curculio

is also an aid in the control of brown rot, since the fungus organism may enter the fruit through curculio stings. For description of the disease see brown rot of peach on page 74.

Keitt and Wilson (3) in Wisconsin find that infection takes place when trees are in bloom. The disease appears later on green and ripe fruit. Spraying for leaf spot holds the disease in check.

In oregon (4) European brown rot S.cinerea pruni was found to be the most important cause of blossom blight, although S.fructicola caused occasional injury.

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## CHERRY LEAF BLIGHT

### Cause

There are two organisms producing this shot hole disease of the foliage, and while the general symptoms and life history are practically similar for the two, there is a difference in the structural characters of the two organisms.

### Hosts

Sweet cherry and the wild choke cherry are more susceptible than the sour cherry or plum, but all of these are hosts for the disease.

### Economic Importance

Since this is a disease primarily of the foliage, damage is not easily measured. Some of the more susceptible forms may be almost entirely defoliated, young trees and nursery stock being particularly damaged. Along with brown rot, leaf spot is considered one of the most important diseases of the cherry. In years of plentiful moisture and warm weather severe defoliation may take place and estimated losses in well cared for plantings may run as high as 30%. Generally the greatest losses occur in the more southern section where climatic conditions are more favorable for early development. There has been a question recently raised as to whether yellowing and dropping of foliage is due entirely to this disease. Gloyer and Glasgow (4) believe there are other causes such as malnutrition which account for part of the loss. Severe winter injury may be associated with premature defoliation brought on by leaf spot.

## Symptoms

Discolored areas appear on the leaves about the last part of May, turning a rather dark blue. These are generally seen on the upper surface of the leaf first, and are quite small. They are rarely over one-eighth inch in diameter and are more or less circular in outline. They may be scattered over the leaf or confined to one portion. In about a week the spots turn reddish brown and have a deadened sunken appearance. Some of the spots may coalesce to involve larger areas when they are numerous and concentrated.

Some of these spots break loose from the healthy tissue at their margins and fall out (Fig. M). Others remain in the leaf as the circular deadened areas. The whole leaf may turn yellow and drop prematurely. Yellowing and dropping of leaves is more common in the cherry.

When spots have developed considerably, whitish masses appear on the under side of the spot during wet weather. Occasionally these masses may be produced in the center of the spot on the upper leaf surface. These are the secondary fruiting spores. Defoliation may take place during early summer about the first of July.

If fruit or leaf pedicels are affected, the spots generally are narrow and extend along the pedicel. In some cases girdling takes place. Pedicel lesions are rarely more than one-fourth inch in length.

## Life History

### Cycle of Infection:

This fungus passes the winter as apothecia in dead leaves on the ground. In spring ascospores are ejected and carried to new leaves where they start infection and produce leaf spotting. On the surface (usually the lower surface) of the spot, a summer fruiting body is formed (ascervulus) from which conidia ooze and are washed to newer surfaces to produce secondary infection. These secondary infections take place throughout the growing season until August. From this time on until leaf fall development of the apothecium takes place. In this stage winter is passed. After all ascospores have been discharged from the apothecia, conidia may be formed in these same apothecia. These conidia may cause infection.

### Morphology and Pathogenesis:

Coccomyces hiemales spends the winter in old diseased leaves on the ground in the sexual stage as apothecia. In the spring, before asci have matured their spores, the covering over the apothecium disintegrates exposing acomparatively shallow cavity containing a number of asci with their maturing ascospores and interspersed with paraphyses (Fig. 59). Ascospores are long cylindrical spores some times having cross walls (Fig. 61). During periods of wet weather, ascospores are ejected forcibly into the air and blown to the leaves usually about the middle of May. They finally reach the under sides of the leaves, germinate and send germ tubes through the stomates. This germ tube rapidly branches out between cells into mycelium (Fig. 62), and some of the mycelial strands send haustoria or feeding bodies into some

of the cells (Fig. 63). After entering the cell haustoria become globose and the leaf cell throws a protective cellulose sheath around the haustorium. In a week or ten days after infection discoloration of the spot may be seen as described above. Soon the cells are killed by a drying process and become brown, imparting the brownish hue to the lesion.

As the season progresses, mycelium becomes massed and entangled just below the leaf surface usually on the under side of the leaf. Cavities are formed, called ascervuli. In the bottom of the ascervilus a disc shaped stroma is formed consisting of one or two layers of cells. From this stroma short conidiaphores or spore stalks arise, upon which are numerous elongated colorless conidia (Fig. 64). As the stroma spreads out between the epidermis and mesophyll cells of the leaf, additional conidiaphores are developed on the new stromatic cells. Accumulation of large quantities of conidia in the ascervulus ruptures the leaf surface. Conidia pile up in large numbers on the surface of the leaf in and around the ruptured area, and white masses become visible to the naked eye. Wind and rain carry these to other leaves, where they germinate (Fig. 66), enter and set up secondary infection. As new leaves appear they become subject to these secondary infections throughout the season until early in August when conidial formation ceases. From the time this occurs until leaves fall, the ascervuli produce small spores about one tenth the length of the conidia. These are called microconidia or spermatia, and are probably borne on the same short spore stalks, which previously produced the conidia. Contemporarily with this spore formation, the stroma grows downward through the leaf cells

involving the mesophyll and palisade cells. Hyphae or threads of the mycelium branch profusely and become densely packed, later differentiating into cells which form a covering on the stroma. Conidiaphores become gelatinous and spread between the scattered epidermal cells of the leaf glueing them together. The cells of the mycelium and of the stroma become thick walled and extend through to the upper epidermis of the leaf. This forms the apothecium, which remains covered above and below by the leaf epidermis. The leaves fall to the ground and the fungus spends the winter in this stage.

During the first warm days of March a row of erect hyphae arise from the stroma toward the lower surface of the dead leaf, causing the apothecium to bulge out on the lower surface of the leaf. These hyphae later become paraphyses which are interspersed with the asci. Asci do not appear until some time in April. They grow rapidly and burst off the covering of epidermal cells. Ascospores mature shortly after the breaking of the covering and are shot out through the pore in the apex of the ascus under favorable moisture conditions and start another cycle in the life history.

The function of the microconidia or spermatia is not known.

When the apothecium has given forth all its ascospores the asci and paraphyses disappear and from the base of the paraphyses short spore stalks arise, which bear long, slender conidia. These are called apothecial conidia and produce infection on the leaves.

## Control

Four sprays are usually applied for control of this disease:-

1. Immediately after petal fall.
2. One week later.
3. Three weeks later.
4. Immediately after fruit is harvested.

Lime-sulphur 32°Baume is generally used at the following rates:

For sour cherry 12 quarts to 97 gallons of water.

For sweet cherry 10 quarts to 98 gallons of water.

A 4-4-50 Bordeaux mixture is sometimes used in case of severe infection in the preceding year.

Orchard sanitation is important in controlling this disease, and where feasible early cultivation should be given before the blooming period to bury diseased leaves. A light covering of soil is sufficient to prevent ascospore discharge.

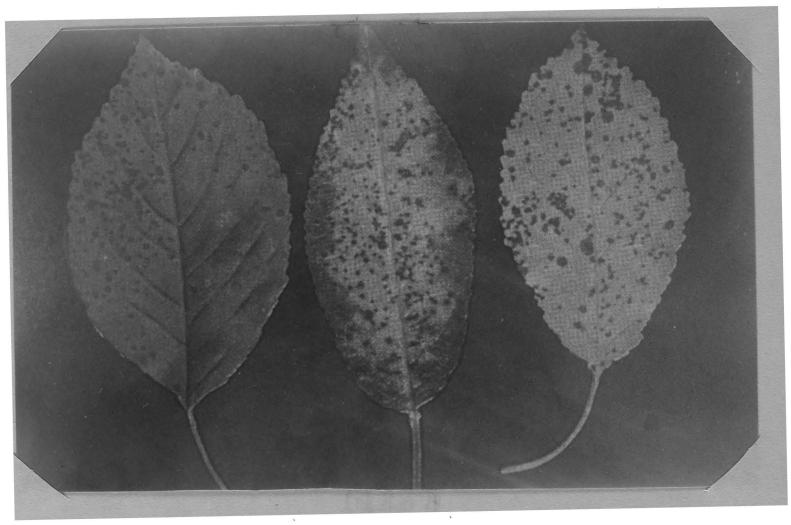
Keitt and Wilson (5) state that Bordeaux 3-3-50 mixture is more effective than lime-sulphur and that 14 days after petal fall is a critical period for control of leaf blight.

In New York, Gloyer and Glasgow (4) find that when early defoliation of trees takes place there is an insufficient supply of stored food and winter injury to the trees takes place. A lime and sulphur spray ten days after petal fall is considered important.

Bradford (6) found that in Michigan sour cherry trees were severely attacked by leaf spot in 1928. Those trees that were defoliated by mid-summer were subject to winter injury. He recommends light pruning and heavy nitrogen fertilization to offset this effect.

LEAF SPOT OF CHERRY AND PLUM. FIG. M.

1.



2.

1. - Plum leaf showing shot-hole effect of the fungus. Shot-hole is more common with plum than with cherry.

2. - Cherry leaves showing spots, and different shades of yellowing. Yellowing of cherry leaves is more common than shot-hole.

Fig. 59. Apothecium of cherry leaf spot containing asci and ascospores.

Fig. 60. A single ascus containing the cylindrical ascospores. Also a paraphysis.

Fig. 61. One, two and three-celled ascospores.

Fig. 62. An ascospore has germinated, entered the stomate and is occupying the intercellular spaces in the leaf.

Fig. 63. Haustoria have punctured the cell wall. Note the cell has thrown out protective cellulose sheaths around the haustoria.

Fig. 64. Ascervulus; found on the under side of the leaf on the leaf spot. Note the numerous conidia, borne on short spore stalks.

Fig. 65. Type of conidia.

Fig. 66. Germination of a conidium.

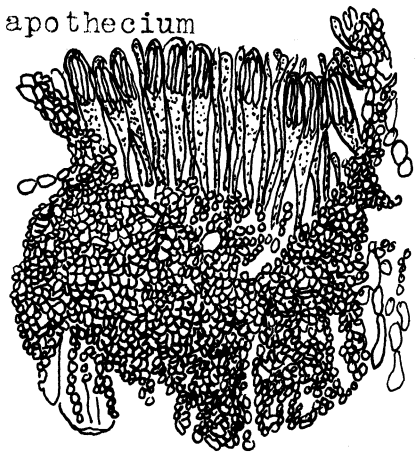



FIG. 59

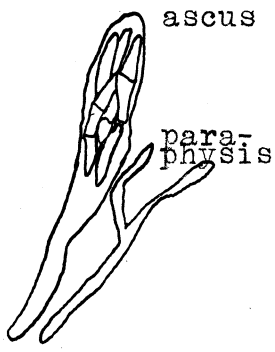



FIG. 60

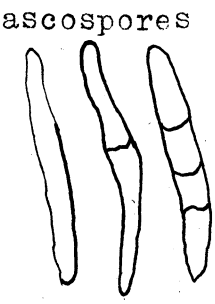



FIG. 61

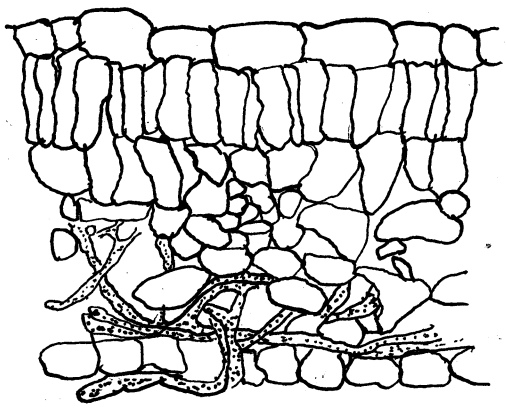


FIG. 62

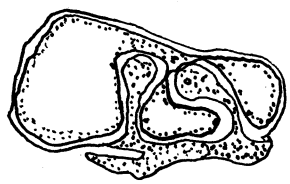



FIG. 63

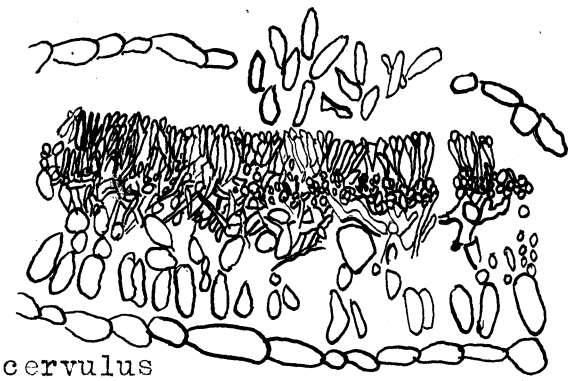



FIG. 64

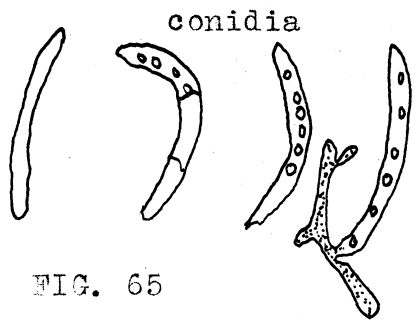



FIG. 65

FIG. 66

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# PART V

# GRAPE DISEASES

## SPRAY CALENDAR FOR GRAPE

| No. | Time of Application | Materials to use | Pest |
|---|---|---|---|
| 1 | Dormant season | Lime-sulphur, 32° strength, diluted 1 to 8 | Scale and general clean-up |
| 2 | When second or third leaf shows. | Bordeaux 4-5-50 | Anthracnose<br>Bitter rot<br>Black rot<br>Mildew. |
| 3. | Before blossoms open | Same | Same |
| 4 | After blossoms fall | Same | Same |
| 5 | Ten to fourteen days later | Same | Same |
| | Then at 2-week intervals until within two weeks of harvest time. | Same | Same |

Leaf arsenate, 2 pounds powder to each 50 gallons of solution, should be combined with Bordeaux if chewing insects make an appearance.

Burgundy mixture may be substituted for Bordeaux in the last spray in order to prevent discoloring of the fruit. The following formula is suggested:

Sodium carbonate (Sal soda) ...1½ lbs.

Copper sulphate. . . . . 1 lb.

Water . . . . . 50 gals.

Prepare and apply same as Bordeaux

## Black Rot of Grapes

### Host

This is a disease confined to the grape family, but is of importance economically only on the grape itself.

### Geographical Distribution

It is found in all vine growing sections of the world.

### Economic Importance

Just as scab of apple and brown rot of stone fruits are the most important diseases of their particular fruits, so is black rot the disease of primary importance in grape growing. This disease may be controlled by spraying but incessant vigilance is necessary during critical periods for infection. On unsprayed vines, severity of infection varies with the season, being more in wet seasons, and less in dry seasons. Also in warm humid regions it is less than in cool dry sections. Thus in North Carolina the entire crop may be lost just after blossoming time, while in New York, greatest losses occur when berries are about one half to two thirds grown. The menace of black rot is ever present and any cessation of spraying along with favorable weather conditions of moisture is likely to be followed by severe rotting of the fruit. Losses in commercial Virginia vineyards may run as high as 25% in years of severe infection. Moisture is the limiting factor in dissemination of the disease.

## Over-wintering Stage

Mummied berries on the ground and clinging to vines are the most important hibernating quarters for the fungus. Although diseased canes, tendrils and petioles may also harbor the disease. It is chiefly carried over as perethecia although pycnidia may also be found.

## Dissemination

By spores during rainy weather.

## Symptoms

Only the young green parts of the vine are attacked. Fruit may be infected at any stage of development.

**Leaves:**

Some time in late June, black rot appears on the leaves as reddish brown more or less circular spots. First indications are a slight blanching of some of the small areas between the veinlets. Spread of a spot is concentric, but the surrounding leaf veins determine its outline. Later the spot appears as grayish brown with a black margin. Still later the margin becomes brown and the browning extends inward until the whole spot is dead and brown. This spot is the result of death of cells and collapse of the parenchyma cells. When spots have reached full size, black fruiting bodies protrude from a ruptured cuticle and dot the entire upper surface of the spot.

**Stems, tendrils, peduncles, petioles and leaf veins:**

Cankers formed are at first small darke red depressions, which soon become brown and finally black. These vary in shape from almost circular to very much elongated. Lesions rarely

extend more than a fourth the way around canes, but may completely encircle leaf petioles. They generally do not extend deeply enough to cut off sap flow except on leaf petioles and berry peduncles.

Fruit:

The most commonly noticed symptom is that of fruit rotting. When the fruit is about half-grown, whitish areas appear on the surface. A brown circular band at the margin with the whitish interior of the spot give a "birds-eye" effect. The spot increases rapidly in size and becomes sunken. Soon numerous minute brown specks begin to appear on the surface of the lesion. These are fruiting bodies which give the spot a dark appearance. Usually the whole berry is rotted and either falls to the ground or shrinks up into a mummy and clings to the cluster. Most of these rotted fruits cling to their peduncles and whole bunches of mummied berries may be commonly seen. Some of the lesions do not spread and cover the whole berry, but are checked and form a much sunken black hardened, spot on the side of the small berry.

## Life History

Cycle of Infection:

Mummies on the ground and clinging to the vines form perithecia and pycnidia with mature ascospores and pycnospores by the last of May or the first of June. With rainfall, spores are discharged and light on the leaf and fruit surfaces and germinate setting up infection and producing rot lesions in from 10 days to three weeks depending upon the weather. In the lesions pycnidia are produced, from which pycnospores ooze during rainy

weather. These spores infect other tissue and carry secondary infection. Ascospores continue to be discharged for a considerable period of time after the first infections and pycnospore formation continues. After the June and early July rains leaf and stem infections are not so common but fruit infections continue until the last of July. After the first of August pycnosclerotia are formed, which change to perithecia during autumn and winter ready to initiate infection the following year. Pycnidia are also carried over winter and set up primary infection the following year.

Morphology and Pathogenesis:

Perithecium:

Black, minute, elevated fruiting bodies are found on the exposed surface of half buried mummied fruit and on the surfaces of mummies clinging to the vine. The latter usually contain a greater abundance. Perithecia and pycnidia are both found. Each perithecium may contain several asci, with 8 ascospores each arranged in single series (Fig. 67). When rain softens the mummy, asci become soaked and elongate out through the ostiolum. When the ascus has protruded almost its original length outside the perithecium, the spores collect toward the apex. Soon the end of the ascus opens and spores are ejected at intervals of a few seconds, one after another until the contents of the ascus are exhausted. Each ascus ejects its spores when it has distended enough to rupture the end. During each succeeding rainy spell the mummy is softened and those perithecia, which have matured in the meantime, discharge their contents into the air. Ascospores are ovoid, hyaline and often have eccentric

swellings at various points. On one end is a gelatinous sheath, which aids in fixing it to the host surface while the slow germination process takes place. Under favorable conditions of moisture, from 2 to 3 days elapse until a germ tube is sent out, This germ tube is usually sent out from the side of the spore (Fig. 69).

Infection:

Germ tubes branch out into mycelium, which is hyaline and very much branched with gnarled laterals. Mycelium invades the cells themselves as well as intercellular spaces, its advance being quite slow. Host cells of the leaves, fruit and young canes are killed in the diseased area, and cells are killed ahead of the advancing mycelium, before hyphae invade them. The period of incubation is usually from 10 days to 3 weeks. Hot moist weather shortens, while cool dry weather lengthens this period. The first leaves to open out are almost never infected. Those in a very succulent tender stage at the time of spore dissemination are generally most heavily infected. Thickening of the leaf cuticle may partially account for this. With berries this thickening does not take place, and they are subject to infection at all times, when moisture conditions are favorable to dissemination and spore germination. Mycelium turns brown with age.

Pycnidia:

Pycnidia are formed by an intertwining and gnarling of the mycelial hyphae. These hyphae turn yellow, then brown and finally black. This transition takes place in the hypodermal

parenchyma region and proceeds rapidly. Intertwining of hyphae forms fungus, cells, which increase in number and size, pushing the adjacent host cells aside. Greatest activity takes place in the center of the gnarl, forcing the flexible outer walls out in all directions, and forming a cavity. At this time the epidermis is uplifted and the cuticle ruptured, exposing the existence of the pycnidium. Spore stalks are formed from the innermost cells of the pycnidium, and pycnospores are constricted from these stalks. Quantities of these spores are formed around the inner wall of the pycnidium (Fig. 70). Most of the pycnidia develop simultaneously in lesions of leaves and stems. On fruit, however, they develop in the center of the lesion first and about 10 days are required to develop them over all portions of the diseased area. Pycnidia are more or less globose. On berries the ostiolum is not always so well developed. Pycnospores are ovoid to globose, with coarsely granular contents, packed full of oily appearing globules (Fig. 71-a). Pycnospores are carried to surfaces of the fruit, germinate (Fig. 71-b) and pierce the cuticle within about 24 hours.

Pycnidia may remain over winter, being found in spring on old canes, tendrils and leaf petioles.

Appresoria:

On the end of a germ tube is sometimes found a brown granular body, which is cut off from the germ tube by a septum (Figs. 69 and 71-c). This is called the appresorium. It is assumed that this is a secondary spore, capable of sending a germ tube into the host tissue, when the original spore fails to gain entrance. Supposedly some unfavorable condition prevents

the germ tube of the ascospore or pycnospore from gaining entrance to host tissue. An appresorium is formed which tides over the germinated spore until conditions become more propitious.

Pycnosclerotia:

Up until about the last of July, pycnidia and pycnospores may be produced in enormous quantities and at a rapid rate to carry secondary infection to grape berries. Each rain followed by damp weather brings a new infection. After about August first pycnidia are produced which have no pycnospores, but are filled with cell like tissue, which is gorged with a whitish substance. These bodies are called pycnosclerotia (Fig. 72) which are thought to chance to perithecia in autumn and winter.

## Control

Methods of control are much the same everywhere, and Bordeaux mixture (4-5-50 formula) is found to be the most effective spray material. The following sprays are recommended:

(1) When the second or third leaf shows

(2) Before blossoms open

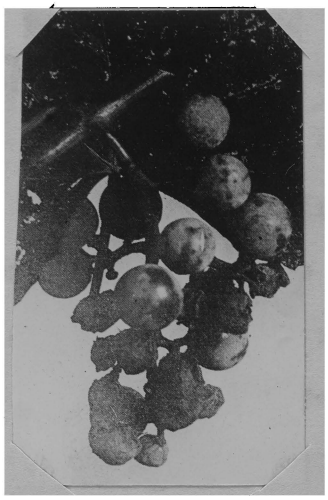
(3) After blossoms fall.

(4) Ten to fourteen days later.

Thereafter at two week intervals until within two weeks of harvest time. These sprays also control mildew, anthracnose and bitter rot.

Sanitation is very important especially where black rot has been severe. Mummied berries should be removed from the vines and destroyed and early cultivation given to plow under mummies and leaves on the ground before infection in the spring.

# BLACK ROT OF GRAPES



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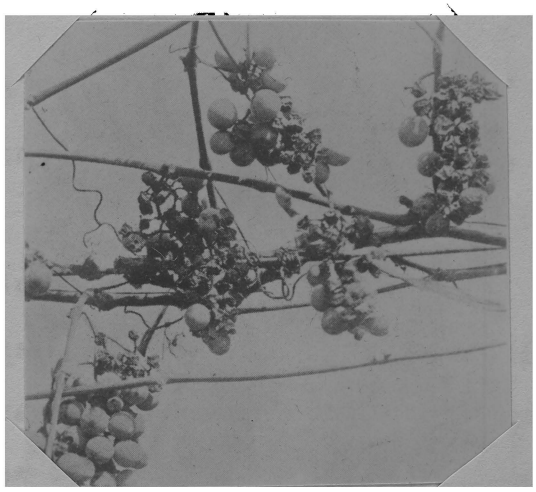

2.

1. - Cluster of grapes showing some sound, some rotting and others shrivelled into mummies.

2. - Heavy infection as seen on this vine causes almost an entire loss of the crop.

Fig. 67. Perithecium of grape black rot as found on a mummied berry in late spring ready to initiate primary infection.

Fig. 68. A single ascus bearing 8 ascospores.

Fig. 69. Irregularly shaped ascospores and type of germination. Note the appresorium formed from one spore. Appresoria are supposedly formed under unfavorable conditions to tide the spore over until germination can take place.

Fig. 70. Pycnidium or summer fruiting body of the fungus found on a diseased berry. Numerous pycnospores are produced from the inner walls.

Fig. 71-a Types of ovoid to globose pycnospores.

b Germination of pycnospores.

c Appresorium developed from a pycnospore.

Fig. 72 - A pycnosclerotium found late in the season filled with pseudo-cell tissue, from which a perithecium and ascospores are formed the following spring.

Fig. 73-a Spermagonium formed from July to October in fully developed lesions. Not connected with the life history of the fungus.

-b Spermatia or spores borne in the spermagonium. These are believed to be vestigial male elements.

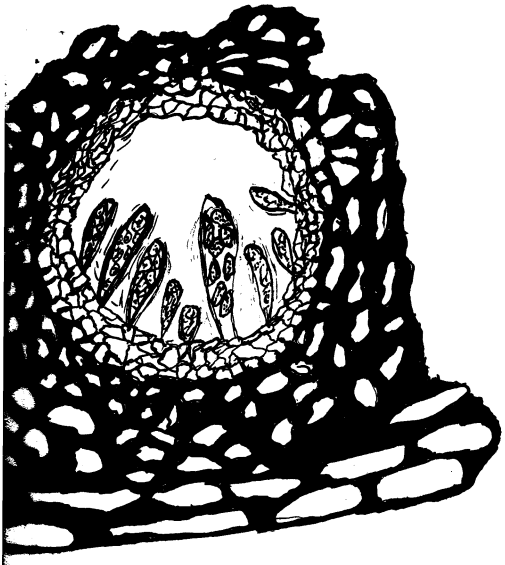

FIG. 67

perithecium from a mummied berry

FIG. 68

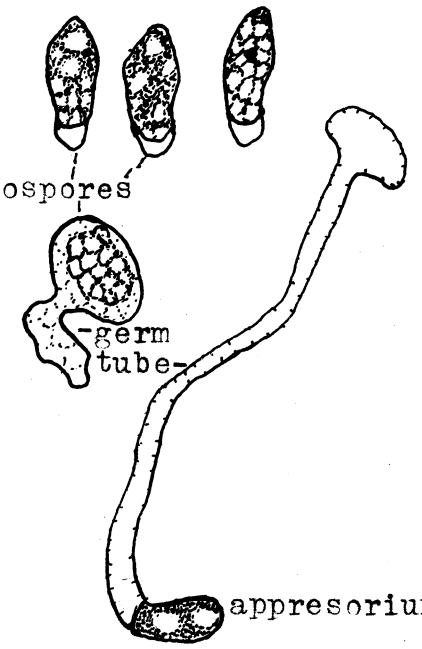


FIG. 69

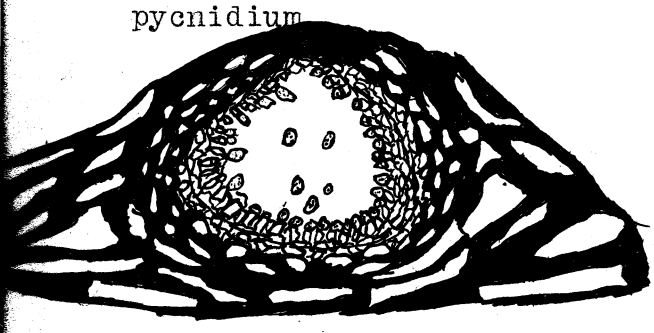


FIG. 70

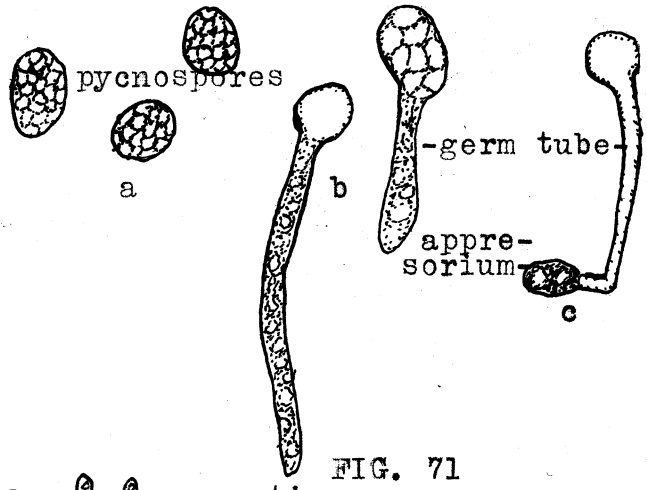


FIG. 71

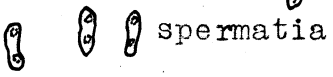


FIG. 73-b

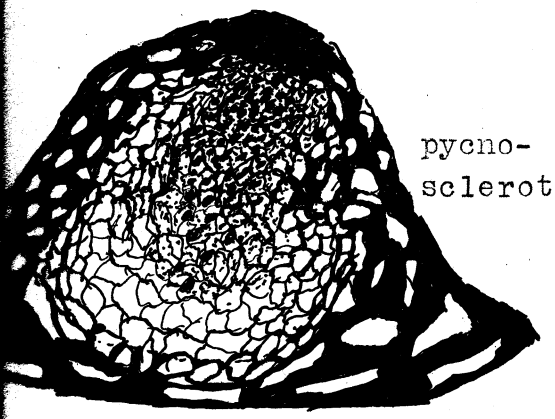


FIG. 72

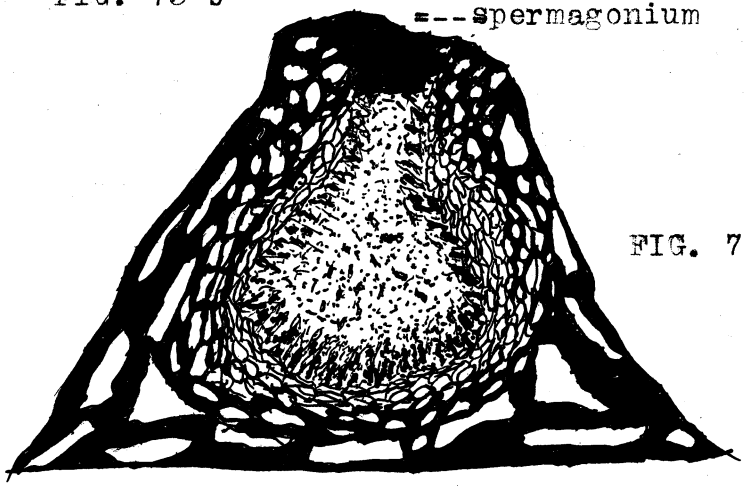


FIG. 73

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## DOWNY MILDEW OF GRAPE

### Cause

Downy mildew is caused by the fungus organism Plasmopara viticola, a Phycomycete.

### History

It is of native origin, being first observed in 1834. By 1878 it had been carried to Europe.

European vineyardists had been warned against the disease but in importing stock for control of phylloxera the disease was brought into France. From here it spread throughout Europe, and has since caused enormous losses.

### Economic Importance

The disease is more destructive to European varieties than to domestic kinds. Delaware is notably susceptible but no variety is very resistant under all conditions. The greatest losses in the U. S. occur in the more northern states, where in cool moist seasons half the crop may be destroyed. Losses in Europe have been enormous. All parts of the vine are attacked. The berries shell off or rot; defoliation often takes place; and dwarfing and death of canes may occur. After thorough investigation, the life history is well understood and control measures usually hold the disease fairly well in check.

### Symptoms

First indications of the disease on the leaf are irregular yellowish spots, the margins of which blend with the

surrounding green. Later the veins become discolored a reddish color, and finally the whole spot becomes reddish brown and dry and brittle. The size of the spot depends upon moisture conditions and age of the leaf. On younger leaves or following wet weather, spots are larger. On less susceptible varieties symptoms are not so pronounced. There is less change in color of the infected area and numerous small brown specks appear on the diseased spot, rather than a browning of the entire area.

On the lower surface of the leaf the first appearance of the disease is similar to that on the upper surface. A white downy covering soon forms on the diseased surface, this consisting of the fruiting bodies of the organism. This downy growth soon disappear from the brown deadened areas.

When the leaf petiole becomes badly spotted or the leaf has a number of large spots, it drops.

Spots on young canes, leaf petioles, and tendrils appear first as shiny water soaked areas slightly swelled above the surrounding tissue. The white covering soon manifests itself and as the diseased areas grow older, they become sunken, brown and dead. In severe attacks young canes are stunted and the leaves remain small.

The fruit is subject to attack at any time from flowering to maturity. Berries become hard and grayish blue in color being coated at this stage with the white downy growth. Later the color changes to reddish brown and finally the berries shrivel into mummies. Diseased berries shell off easily.

## Life History

The organism over winters in the dead leaves on the ground under the vine. Imbedded in the dried leaves are comparatively large globe shaped bodies called oospores, which serve as a resting stage during the winter.

Fig. 74 shows a cross section of an oospore in which the thick wall and circular banded inner wall may be seen. During wet weather in spring this germinates in place by cracking of the wall. Through this crack a slender spore stalk or conidiaphore is projected, which bears a large egg-shaped conidium on its tip (Fig. 75).

When this conidium is matured it breaks loose readily from the stalk and is splashed by wind and rain to the leaf surface especially on the lower leaves. Here in a drop of water the conidium germinates by a division of its contents into 5-8 swarm spores. Fig. 76. At the apex of the conidium there is a portion of the wall which is very thin and protrudes slightly. The spore contents divides into 5-8 parts each one of which has a nucleus. As soon as these become distinct entities, the thin walled protrusion is dissolved and the swarm spores force themselves through the opening. Once outside, the swarm spores form hair-like appendages, one from each side of the nucleus (Fig.77). These are called flagella and are used to propel the swarm spore as it swims around in the rain water for about half an hour. The swarm spore then stops swimming, settles down (Fig. 78) and rounds itself into a globular shape, absorbing the flagella. In about fifteen minutes a protuberance starts on one side (Fig. 79-a) this being the beginning of the germ tube. As the tube lengthens

the contents of the swarm spore passes into this tube (Figs.79-b and c), and the spore gradually becomes empty. This germ tube finds its way into a stomata (Fig. 80) always on the under side of the leaf and branches out into mycelium, which permeates the leaf tissue and sets up the symptoms mentioned before. Shortly the mycelium collects beneath the stomata and sends up from four to ten spore stalks (conidiaphores) through each stomatal opening (Fig. 81). These grow from most of the stomates and give the white downy appearance. The conidiaphores branch out and bear a conidium on each branch. These conidia are blown to other parts of the vine and set up a secondary infection. Diseased leaves carry the mycelium, which forms the resting spore stage for winter dormancy of the organism. This formation probably starts while the leaf is still attached to the vine. The mycelium branches considerably and the tips of some of these branches swell. A cross wall is formed in the mycelium just below the swollen tip. This swollen tip is called the oogonium or female organ (Fig. 82-a) and is very granular with a nucleus. Closely beside the oogonium other mycelial tips swell to form a smaller granular body with a nucleus and a wall separating it from the mycelial thread. The latter body is the antheridium or male organ (Fig. 82-a). A fertilization tube is formed on the antheridium and grows until it enters the oogonium. The nucleus of the antheridium passes through this tube into the oogonium (Fig. 82-b) and fuses with the nucleus there to form the new oospore. The oospore does not mature until the leaf is dead and fallen to the ground. Here it becomes rounded, the walls thicken and it remains within the oogonial cavity until spring. During wet spring weather

the leaves disintegrate freeing the oospore. The surrounding oogonial walls collapses and with proper moisture and temperature the oospore germinates. Thus a very interesting life history is complete.

## Control

Recommended control measures are spraying with a 5-5-50 Bordeaux mixture.

1. When the second or third leaf shows.
2. Before blossoms open
3. After blossoms fall
4. Ten days to two weeks later.
5. Thereafter at two weeks intervals until within about two weeks of picking season.

These are combination sprays for the control of anthracnose, Bitter Rot and Black Rot and should be applied to safely control these diseases.

Since the under sides of the leaves are the points of entrance of the primary infection, obviously it is necessary to thoroughly cover these surfaces as well as other portions of the vines.

Gladwin (4) finds in New York that after trials with numerous spray materials and dusts, Bordeaux mixture 4-5-50 still remains the safest and most efficient fungicide for control of mildew and that the usual sprays for control of black rot fit in nicely for the control of mildew. Since black rot is the most serious disease of grapes and must be controlled, mildew is controlled with the same sprays.

Fig. 74. Cross section of an oospore, the winter hibernating stage.

Fig. 75. An oospore on the ground which has sent up a spore stalk and conidium.

Fig. 76. Various stages of germination of the conidium to form swarm spores.

Fig. 77. Protoplasmic swarm spore with flagella by means of which it swims around.

Fig. 78. The swarm spore after it has come to rest and rounded up.

Fig. 79. Stages in germination of the swarm spore.

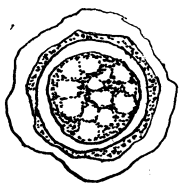
Fig. 80. Infection through stomata (diagrammatic)

Fig. 81. The 4 to 7 conidiaphores projected through the stomata and bearing conidia (diagrammatic)

Fig. 82-a The oogonium (or female cell) and the antheridium (or male cell) lying side by side before conjugation.

b Process of conjugation of oogonium and antheridium. Note fusion of two nuclei in oogonium. The oospore is formed from this body.

DOWNY MILDEW OF GRAPE



oospore

FIG. 74

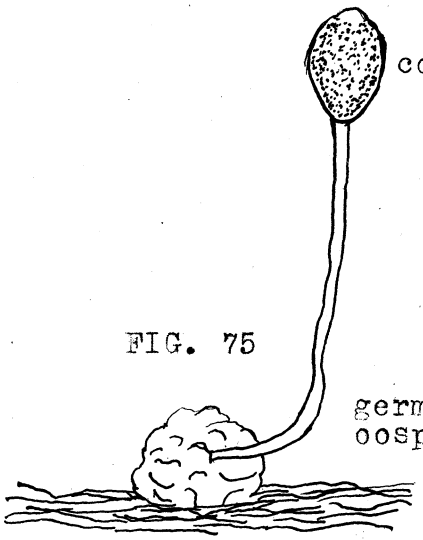



FIG. 75

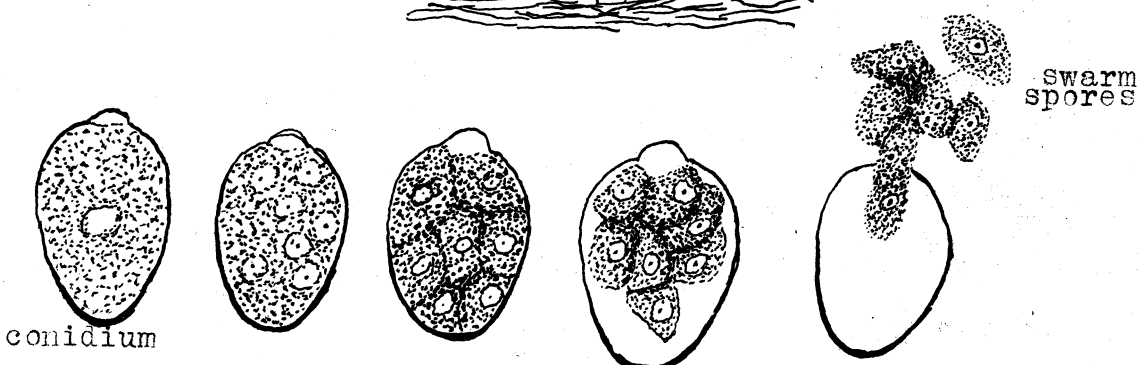



FIG. 76

FIG. 77

FIG. 78

FIG. 79

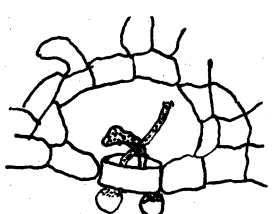


FIG. 80

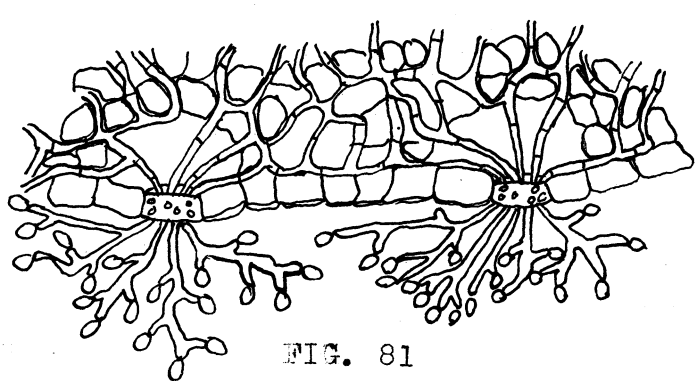


FIG. 81

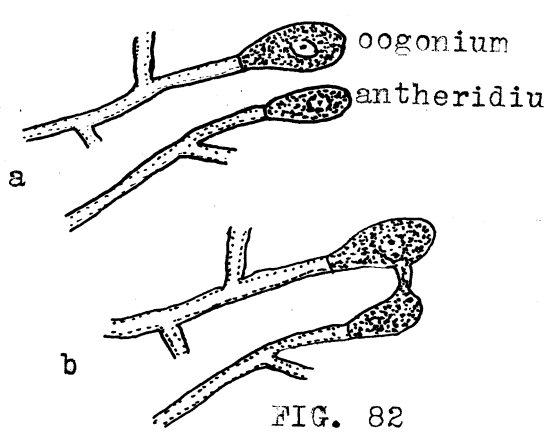



FIG. 82

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| EVENT | 1922 | 1923 | 1924 | 1925 | 1926 | 1927 | 1928 | 1929 | 1930 | 1931 |
|---|---|---|---|---|---|---|---|---|---|---|
| Delayed Dormant Spray | Mar. 22 | Mar. 26 | Apr. 4 | Mar.26-Apr. 8 | Mar. 30-Apr.17 | Mar.16-Apr.5 | Apr.4-14 | Mar.25-Apr.2 | Mar.20 Apr.7 | Apr.11-18 |
| First Rosy asphis | Apr. 1 | Apr. 4 | Apr.15 | Mar. 20 | Apr. 4 | Mar.18 | Apr. 3 | Mar.23 | Mar. 18 | Apr.11 |
| Pink Spray | Apr.9-14 | Apr. 19-25 | Apr.24-30 | Apr. 12-16 | Apr.26-30 | Apr.13-18 | Apr.24-29 | Apr. 5-7 | Apr. 12-16 | Apr.20-30 |
| Mildew on apple | May 7 | May 3 | May 10 | May 1 | Apr. 7 | May 1 | May 6 | ------- | Apr. 18 | Apr. 24 |
| Petal fall spray | May 2-9 | May 5-14 | May 14-20 | Apr. 27 May 7 | May 10-22 | May 4-13 | May 11-21 | Apr.23 May 2 | May 2-10 | May 14-23 |
| Frog eye | May 7 | May 18 | May 12 | Apr. 29 | May 27 | May 1 | May 7 | May 1 | May 11 | May 13 |
| Cedar rust | May 10 | May 17 | May 23 | May 11 | May 30 | May 10 | May 10 | May 1 | May 29 | May 10 |
| Scab | May 15 | May 16 | May 26 | May 10 | June 5 | May 12 | May 20 | May 10 | May 7 | May 21 |
| Three weeks spray | | May 16-26 | June 3-9 | May 13-18 | May 28-June 8 | May 26-June6 | June 1-9 | May 14-23 | May 20-31 | June 3-13 |
| First coddling moth larvae | May 25 | May 27 | June 8 | May 20 | May 20 | May 30 | June 1 | May 18 | May 16 | May 27 |
| Rosy asphis leaving apple | May 25 | May 20 | ------- | May 16 | May 30 | May 19 | May 30 | May 18 | June 8 | May 31 |
| Max. egg laying 1st brood coddling moth | June 1-8 | June 5-12 | June 19-20 | June 4-5 | May 17-25 | June 10-22 | June 1-5 | May 24-June 6 | May 28-June 18 | June 24 |
| Apple blotch | June 5 | June 6 | June 26 | June 12 | June 23 | June 13 | June 10 | ------- | June 25 | June 22 |
| Rose chafer | June 6 | June 2 | June 9 | June 1 | June 10 | ------- | | | May 31 | ------- |
| Five weeks spray | | June 7-14 | June 13-20 | May 29 June 6 | June 18-July 4 | June 13-17 | June 19-23 | June 3-10 | June 10-18 | June 18-25 |
| Black rot | July 1 | July 17 | June 18 | June 10 | June 17 | July 7 | July 8 | ------- | ------- | July 4 |
| Bitter rot | July 1 | Aug 2 | June 24 | July 7 | July 30 | July 18 | July 6 | ------- | Aug. 2 | Aug. 31 |
| Max. egg laying 2nd brood coddling moth | July 22 | July 28 | Aug.9-12 | Sept 14 | Aug. 30 | Aug.6-13 | July 28-Aug 1 | July 26-Aug 8 | July 19-Aug 7 | July 20-Aug 8 |
| Cloud or sooty blotch | Aug. 6 | July 7 | Sept. 16 | Aug 1 | Aug. 4 | Aug. 15 | Aug. 7 | ------- | Aug. 29 | Aug. 20 |
| Mid summer spray | | July 15-22 | July 31-Aug. 9 | Aug 20 | July 23-31 | July 1-7 | July 5-14 | June 24-29 | July 10-19 | July 21-28 |
| Max.egg laying 3rd brood coddling moth | Aug.25-31 | Sept 1-7 | None | Aug. 27 | Sept. 4-8 | | None | Aug.25-28 | Sept. 1-3 | Sept. 1-10 |
| Max. activity of leaf roller | Sept. 5 | Sept. 15 | Sept. 17 | | | | Sept.1-15 | Sept. 1-10 | Sept. 1-20 | Sept. 3-25 |