

LABORATORY OUTLINES IN PLANT PATHOLOGY

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LABORATORY OUTLINES
IN
PLANT PATHOLOGY

BY

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AT

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PREFACE

THESE outlines are designed solely for the purpose of most effectively acquainting the student with the laboratory materials presented in this course. They are the result of several years of experience and test in actual practice work. They have been frequently revised. It is expected that they will now be revised and reprinted at least every two years.

Although the acquisition of a body of facts is an important and necessary part of the work in such a course a more vital feature is the training in logical methods of acquiring them. The student is urged to follow his outline carefully, making sure at each step, that the outline and the materials before him agree. The same sequence of treatment is followed throughout all the outlines. This sequence in procedure should be mastered promptly. The term papers will afford opportunities for determining how well the student has grasped the logic of this procedure.

The grouping of diseases here presented is, we believe, an important step in diverting attention from the domination of systematic mycology in phytopathological teaching and writing, and of directing it toward the more logical classification and study of diseases on the basis of the pathological phenomena exhibited. At the same time the subgrouping of the diseases, according to the chief etiologic factor involved, provides for a point of view still generally presented in the teaching of plant pathology.

It is not expected that all the diseases herein outlined will be covered in a three hours' course. The instructor will make such selections from the different groups as will best serve his purpose in illustrating the fundamentals of the subject in the case of the students in his classes. No laboratory practice in the methods of control of the diseases studied is provided. That phase of the subject is fully treated in the course based upon and following this, namely, *The Principles of Plant Disease Control*.

These outlines are designed specifically for the work as given at Cornell University and without any attempt to adapt them for use in other institutions. It is hoped, however, that teachers elsewhere may at least find them helpful and possibly usable in their classes.

Acknowledgments are due Mr. Chas. Chupp, instructor in the department of Plant Pathology for the preparation of a number of the outlines. We also gratefully acknowledge the friendly advice and assistance of the Comstock Publishing Company in our effort to make the cost of these outlines to the student as reasonable as possible.

THE AUTHORS.

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FIELD TRIP

This first exercise consists of a field trip taken to points close to the laboratory. The purpose is to introduce the student to a variety of plant diseases as they appear in the field. Attention will be directed to such diseases of all sorts of plants as are found.

The student himself should seek to discover diseased plants. He will be expected to collect at least ten specimens of diseased plants for study and report. Healthy specimens for comparison should also be taken. (See REPORT at end of exercise.)

For field identification of diseases one must depend largely upon *symptoms* and *signs*. By symptoms is meant those changes induced in the diseased plant which distinguish it from the healthy. Signs of disease are incidental or experimental evidences of disease and not the direct results or expressions of the diseased conditions.

SYMPTOMS

Symptoms are usually the expression of structural changes of one kind or another (histological or morphological). Diseases in plants are of three general types within each of which are exhibited a considerable variety of symptoms. These three types of disease with some of their more characteristic symptoms are:—

NECROTIC DISEASES— those in which the most striking effect on the host is the death of the affected tissues resulting in such symptoms as:—

Rot—the term applied when the killed tissues become discolored and decayed. If the tissue in such lesions are dry and firm it is called *dry rot*; if soft and mushy, *soft rot*; if firm or tough but very watery or soggy, *wet rot*; if white in color, *white rot*; if black, *black rot*. The term rot, coupled with the name of the part or organ affected serves to designate a variety of symptoms such as, *stem-rot*, *bud-rot*, *crown-rot*, *collar-rot*, *root-rot*, *heart-rot*, *foot-rot*.

Blight—the term applied when there is rapid killing of the affected parts often accompanied by wilting and withering of the foliage. This term coupled with the name of the host organs gives, *twig-blight*, *blossom-blight*, *body-blight*, *leaf-blight*, and the like.

Wilt—the term applied where all or a portion of a plant becomes limp due to loss of turgor. Wilt differs from blight in that the causal factor does not directly affect the wilting or dying organs. The injury results from the activities of the causal factor usually in the vascular system somewhere below the wilting organs.

Spot—the term used to designate necrotic areas, usually those in leaves, fruits and herbaceous stems. This term is used in numerous combinations to designate a variety of spot-symptoms as, *leaf-spot*, *fruit-spot*, *pod-spot*, and the like.

Shot-hole—the term applied to limited necrotic spots on foliage where the dead tissue drops out leaving a hole. Peculiar largely to the foliage of peaches, plums and cherries.

Damping-off—used to designate the symptom resulting from the rotting of the stems of seedlings at the base and the consequent falling-over of the tops. A form of stem-rot.

Canker—the term applied to definitely delimited necrotic lesions in the bark of woody plants or the cortex of herbaceous stems; smooth or roughened, sunken or raised.

HYPOPLASTIC DISEASES—those in which the most striking effect on the plant is a halting in some feature of its normal growth and development resulting in such symptoms as:—

Dwarfing—the term applied in those cases where the plant or organ does not reach normal size. Special terms like, *curly-dwarf*, *leaf-roll*, *spindling-sprouts*, and *little-peach*, are but disease names used to designate peculiar forms of dwarfing.

Chlorosis—used to designate the failure of or insufficient development of chlorophyl. Especially characteristic forms of chlorosis have been designated by such names as, *mosaic*, *calico*, *frenching*, and *yellow*s.

METAPLASTIC DISEASES—those in which the most striking effect of the disease is the overgrowth or overdevelopment of the affected tissues or organs. Stimulation of growth and development beyond the normal results in such symptoms as:—

Hypertrophy—the term used in its broadest sense to designate all abnormal overgrowths in size of diseased parts or organs. Among the specially designated forms of hypertrophy are, *galls*, *knots*, and *tubercles*, which are more or less globose swellings of leaves, stems, fruits or roots resulting from the stimulating activities of such causal factors as insects, fungi, bacteria and mechanical injuries. Combinations with the names of the plant-organs affected give the terms, *crown-gall*, *root-gall*, *root-knot*, and *root-tubercles*.

Witches'-brooms—broom-like growths resulting from the dense fasciculation or clustering of branches due to the forcing of adventitious buds from or about the diseased tissues.

Hairy root—abnormal root development of the same character and nature as witches'-brooms on the limbs.

Scab—definite areas on fruits, tubers and other organs usually due to injury followed by abnormal cork-development resulting in a rough raised or sunken spot.

Curl—a term applied usually to overgrowths of leaves resulting in a thickening and fluting or puffing of the diseased areas, usually accompanied by abnormal coloration.

SIGNS

The more striking and diagnostic signs of disease are the characteristic fruiting structures of the pathogene. Some of these have received pathologic designations, as:—

Smut—the black powdery spore-masses developed by the so-called *smut-fungi*. Found usually in the fruiting structures of the host, although sometimes in leaves and stems.

Rust—the yellow, red, brown or black powdery spore-masses of the *rust-fungi* usually produced in small pustules on leaves and stems.

Mildew—the superficial mycelial or conidial structures developed by certain fungi on the lesions. When such growths are mealy or silky they are designated *powdery mildews*, when fluffy, *downy mildews*. The former are usually white and on the upper surface of the leaves, while the latter are usually grey or purplish and on the under surface.

Mould—grey or black fluffy growths, mycelial or conidial structures, usually accompanying necrotic lesions; sometimes used synonymously with mildew.

Ooze—sticky gummy or fluid exudates from certain lesions.

Punks—sporophores of basidiomycetous shelf-fungi whose presence on the surface of apparently healthy trees is evidence of heart- or sap-rot.

Attempt to identify with one of the above, each symptom or sign of disease observed on the trip. Classify the signs and symptoms represented in the collected materials.

The instructor in charge will explain the cause, stages of development and other points of interest concerning some of the diseases met with.

REPORT

1. Describe and sketch the signs and symptoms of ten diseases, illustrating the different types observed during the trip.

THE LITERATURE OF PLANT DISEASES

The literature on plant diseases is widely scattered. Abstracts of the current literature on the subject (American and some of the foreign) appear regularly in each number of the EXPERIMENT STATION RECORD. Other journals and publications in which such reviews and abstracts of phytopathological papers are to be found, follow:—

Phytopathology. (Official organ of the American Phytopathological Society.)

Zeitschrift für Pflanzenkrankheiten.

Botanisches Centralblatt.

Centralblatt für Bacteriologie und Parasitenkunde.

Just's Botanische Jahresbericht.

Index au Bulletin Bibliographique Herbdomadaire Institute International d'Agriculture.

Hollrung's Jahresbericht über das Gebeit der Pflanzenkrankheiten.

Mycologia, formerly Journal of Mycology.

Texts and reference books in which references to the literature of the subject are more or less brought together, are:—

Duggar, B. M. Fungous Diseases of Plants. 1909.

Stevens, F. L. The Fungi Which Cause Plant Disease. 1913.

Sorauer, P. Handbuch der Pflanzenkrankheiten. 1909.

Smith, E. F. Bacteria in Relation to Plant Diseases, vol. I-III. 1905-1911-1914.

Massee, G. Diseases of Cultivated Plants and Trees. 1910.

PROCEDURE IN PREPARING A BIBLIOGRAPHY

Choose from the list of term-paper subjects, according to directions there given, the disease on which a term paper is to be written.

Write in three different columns on one of the reference-sheets provided; (a) the names of the host-plants; (b) different names applied to the disease; (c) different names applied to the pathogene.

Note:—Begin each column with the names given in the subject-list, adding to each; other hosts, other names of the disease and other names of the pathogene, discovered as the work proceeds. These will constitute *key-words* under which references to the disease in hand will be looked for in the indices.

To find and copy references:—

A. Select the *latest* volume of the Experiment Station Record available and, turning to the index, locate under key-words the references to the disease chosen.

1. Write on a sheet of paper the number of the volume in hand.

2. Record under this volume number, in numerical order, the pages on which references to the disease are abstracted. (See Information p. 12.)

Suppose for example the key-words as found in the subject-list for the term paper are:—

Apple

Bitter Rot

Glomerella cingulata

First consult the index under apple, noting the page of every item that may possibly refer to the disease bitter rot, and record as directed above.

Then consult the index under bitter rot, for you may find there references to the disease not listed under apple.

Finally consult the index under the names of the pathogene.

3. Examine each reference in the text carefully, and copy according to directions under B below, any of the references which refer in any way to the disease.

B. Copy the references on the 5 x 8 sheets of paper provided using *one sheet for one reference only*. *Begin at the very top*. Write lengthwise of the sheet and arrange the data as follows:—

1. Record the source of the reference in the *very upper right-hand corner* thus:—E.S.R. 18 : 748. (See Information on,—Source of reference p. 12)

2. Place the surname of the author, followed by his initials in the *upper left-hand corner* on the line below that of the source of the reference.

3. Directly following the author's name record in order:—
(title) (publication) (series) (vol.) (part) (pages) (plates) (figures) (year)
Bitter rot. Jour. Agr. Sci. 2 : 47 : 3 : 37-47, pl. 1-4, fig. 1-9. 1906.
(See sample sheet below.)

Note:—No matter what the arrangement and punctuation given in the source of the reference or in the original, it should be arranged and punctuated as above.

When in doubt on any point, consult "Information" under the proper heading following the sample-sheet, page 12.

C. Key-words. These should always appear on the reference-sheet. (See sample-sheet below.) First the name of the host or hosts,

SAMPLE-SHEET

		E.S.R. 18:874
HASSELBRING, H. Bitter rot. <i>Jour. Agr. Science.</i> 2: 47: 3: 37-47, pl. 1-4, fig. 1-9. 1906.		
Apple	Bitter rot	[<i>Glomerella cingulata</i>]
Grape	Anthracoise	<i>Glomerella rufomaculans</i>
Pear	Ripe rot	<i>Gloeosporium fructigenum</i>
Abstract from original.		
HOSTS		
Hosts listed are apple, pear, etc. p. 37.		
VARIETAL SUSCEPTIBILITY		
Most susceptible, Greening, Baldwin; least susceptible, Winesap, etc. p. 39.		
DISEASE		
NAMES		
Following names listed; bitter rot, anthracnose, ripe rot. The first most commonly applied. p. 40.		
(Continue on other sheets under the pertinent heads and subheads as used in the outline for the term paper.)		

especially dealt with in the article should be written close to the left margin of the sheet and one or two spaces lower than the last line of the reference, in a column one above the other.

In the same manner place the names of the disease at the middle of the sheet and the names of the pathogene at the right of the sheet.

These key-words should be for each sheet, *only those which appear in that particular reference*. If none appear in the reference (for example, if the name of the pathogene is entirely omitted), write the name generally accepted and enclose in brackets thus:—

Apple Bitter rot [G. cingulata]

The chief purpose of these key-words is to assist in assorting and identifying the references later.

INFORMATION

Source of reference. Having this with each reference will enable one to readily verify it or correct errors made in copying. It should include the abbreviated name of the publication, volume-numeral (or of year or number as case may be), colon (:) followed by the page on which the reference occurs. If the article is first discovered in the original, indicate thus:—"Orig."

Author. Surname, comma, initials, period, is the order of the arrangement. Where there are two or more authors, arrange each name in the same way connecting the names with "and" or its foreign equivalent, or with "commas" and "and" in the case of more than two authors. In the case of several authors one may write for example,—Stewart, F. C. and others, or et al. In the case of anonymous articles, write in place of the author's name the word, Anonymous.

Title. The title of an article follows directly after the author's name and should always be in the language of the original if possible. When only the translation of the title is given precede it with the name of the original language and the word, title, in brackets, thus:—[Italian title] "Concerning the influence, etc." When both the original and the translation of the title are given, the translation follows the original and is to be inclosed in brackets. No abbreviations of the title should be used except such as appear in the original. Only the first word and proper names in the title may be capitalized, except when the title is in German. The title terminates with a period.

Name of society or organization publishing the work follows directly after the title, the second line beginning directly under the fourth letter of the author's surname. It is followed by a period; abbreviation allowed.

Name of publication. Journal, Berichte, bulletin (properly abbreviated) follows directly after the name of the society or organization, or where these are wanting, directly after the title. When the last word is not an abbreviation, no punctuation-mark follows.

Volume-numerals are always to be in arabic and are to be *under-scored* with a wavy line, indicating bold-faced type in printing. The word volume or its abbreviation (vol.) *should not* appear in a reference.

Reports are often issued for a given year as for example the Alabama Agr. Exp. Sta. Report for 1896, no volume-number being given. In such

a case the *year-numerals take the place of volume-numerals* and the above would appear thus:—Alabama Agr. Exp. Sta. Rept. 1896:1–87.

Number-numerals when indicating consecutively paged parts of a volume (as for example, vol. 27, No. 6 pp. 70–83) *should not* appear in the reference. When not paged consecutively the number-numeral appears between volume-numeral and page-numeral.

Bulletins, circulars, memoirs, leaflets and the like are usually numbered and are not considered as constituting consecutively paged numbers of a volume. The numerals appear therefore in place of the volume-numerals, but *are not to be underscored*. The proper abbreviation for the word Bulletin is Bul. with a capital B. Where qualified, as for example, Technical bulletin, it is abbreviated but not capitalized.

Series, Abteilungen. If a periodical is issued in two or more separate series the *series-numeral precedes the volume-numeral*, separated from it by a colon and is *not* to be underscored, as:—3:46:23–87. Sometimes the series is indicated thus:—Science n. s. 42:47–56. The n.s. = New Series.

Parts. If parts of a volume are paged separately, the part-numeral *follows* the volume-numeral and is separated from it by a colon:—thus, 82:2:241–256.

Pages. The page-numerals when preceded by volume- or number-numerals and a colon are to be cited *inclusively*, i.e. first and last pages separated by a dash thus:—47–83, and followed by a period.

Tables, plates and figures follow directly after the pages, properly abbreviated, followed by the proper numerals in arabic.

Year. This must be the *actual* year of publication. It may not always be determined with certainty except by consulting the original. Exercise care on this point and leave the space blank until the original can be consulted, if not absolutely sure of it from the reference.

Books. In cases where *no volume* is given, cite the pages thus:—p. 27 or p. 1–441.

Citation *from* books. Following the date, give the title of the chapter or paragraph, especially referred to, then, *In*, then the title of the book followed by the letter p. to indicate page, then the page number and finally the date thus:—

Ellis, J. B. Dothidea pomigena. Schw. *In* The North American Pyrenomycetes. p. 605. 1892.

Government publications are always to be cited in the following form;—U. S. Agr. Dept. Yearbook; or U. S. Plant Ind. Bur. Bul. 37:

State Experiment Station publications are always to be cited thus:—Alabama Agr. Exp. Sta. Bul.; or New York (Geneva) Agr. Exp. Sta. Rept.; Cornell Univ. Agr. Exp. Sta. Bul. 237:46–91.

Note:—For further details and examples of correct citation see pamphlet “Notes for guidance of author’s.” A copy may be checked from the departmental library.

PREPARATION OF AN ABSTRACT OF AN ARTICLE

The abstract should always be made from the *original*, unless for some good reason this is impossible. Do not copy the abstract of the article as it appears in the Experiment Station Record or elsewhere.

Begin the abstract on the reference-sheet directly beneath the key-words. Use both sides of the paper or use additional sheets or both if desired.

One should abstract as he reads, assorting and entering data under the proper head as indicated in the outline for writing term paper. (See term—paper outline and sample-sheet in this outline p. 11.)

Follow each entry of data with the number of the page on which it was found in the original. This will enable one, when writing the term paper to readily cite the source of any statement made.

The abstract should be full enough that it will not usually be necessary to refer again to the original in preparing the term paper.

REPORT

Hand in the bibliography of the subject chosen, arrange as follows:—

1. The references, each on a separate sheet. Arrange the sheets in the manilla folder provided, in the order of importance of the references,—the most important first.

2. One or more of the most important references carefully abstracted as above described.

3. The folder labeled thus:—

(Name of host)	(Name of disease)	Bibl.
(Name of student).		

NECROTIC DISEASES

FIRE BLIGHT

This is the most common and best known bacterial disease of plants occurring in this country. It affects apples, pears, quinces and occasionally plums, apricots and a few ornamental and wild plants related to the apple family.

SYMPTOMS

The symptoms of this disease will be studied in the order in which they manifest themselves during the season on different parts of the tree, beginning with the appearance of the cankers in the spring.

Hold-over cankers. These are the sources of inoculum for the first infection of the blossoms in the spring. Study the typical cankers on the limbs of apple and pear trees provided and OBSERVE:—

1. The smooth, more or less sunken area in the bark,—the canker; its margin sharply defined by a definite crack. In cankers in which the pathogene is active this margin is not sharply defined. (See illustration specimens if available; photograph 1; Cornell Bul. 272, fig. 16, or 329, fig. 114.)

2. The margin. Note that it is irregular, the crack being formed by the drying away of the diseased tissue from the healthy when the active progress of the pathogene is suddenly checked. Dry or cold weather may thus check the enlargement of the canker. These specimens were collected in the autumn or winter.

3. The surface of the canker. Note that it is smooth, seldom roughened or wrinkled. It is often checked at the margin by drying. Compare with the healthy bark in this respect. Locate the lenticles. What is their structure and function?

Make a V-shaped cut across the margin of the canker and DETERMINE:—

4. How deeply the disease penetrates. What tissues are affected?

Make a DRAWING of the canker studied. Label fully. These cankers are formed during the summer and early autumn and in many of them the bacteria pass the winter dormant, or only slightly active in the partially living tissues along the margin. With the increased temperature and the beginning of growth-activities in the spring, these bacteria become active, work rapidly into the adjoining healthy tissue, increase the area of the canker and ooze out through the lenticles to the surface in sticky, milky drops. Study photograph 1; Cornell Bul. 272, fig. 16, or 329, fig. 114. OBSERVE:—

5. That the advancing margin of the canker is not distinctly evident here. The diseased area covers nearly all the surface shown in the illustration, except on the extreme left.

6. The large viscid milky drops oozing out and running down the limb; above to the left, two small globules just oozing out from the lenticles. Read Cornell Bul. 272:40-41, also Ontario Bul. 176:15-23.

Make a DRAWING from photograph 1; Cornell Bul. 272, fig. 16, or 329, fig. 114. Label fully.

Blossom-blight. Bees and flies visit these active cankers in the spring, to feed on the exuding sap and then visit the opening blossoms where they leave behind them some of the bacteria which adhered to their bodies. Here in the nectar and in the injuries made by the insects' claws in the tender tissue of the flower, the bacteria multiply rapidly killing the blossoms. Study the specimens provided; Ontario Bul. 176, frontispiece; Cornell Bul. 329, fig. 112; or photograph 2. OBSERVE:—

7. The dead and blackened flowers. The leaves of the spur are also dead and brown. The bacteria have spread down the pedicles into the spur. These dead and blackened blossom-spurs are usually the first striking evidence of the disease in the spring. The oozing cankers are usually overlooked. Make an outline DRAWING of a blighted blossom-spur.

Fruit-blight. Frequently only one blossom on a spur is infected and by the time the bacteria have killed it and worked their way down the pedicle to the spur itself, the uninfected blossoms have developed fruit of a considerable size. From the spur the bacteria now work into the base of these fruit-pedicles and by way of them into the growing fruit. Study photograph 3; Ontario Bul. 176, fig. 15-19; or illustration specimen. OBSERVE:—

8. The blackened pedicle and discolored lower half of the fruit. A fruit affected in this way usually shrivels and drops from the tree. It may cling to the twig as a blackened and shriveled mummy. The loss from fruit-blight is sometimes heavy. The curculio and aphids frequently introduce the bacteria into the fruit through their punctures. The pathogene does not always enter the fruit by way of the pedicle. Note that the leaves of the spur are also dead and shriveling. In rainy, muggy weather the bacteria ooze from these blighted fruits and blossoms in sticky drops as they do from the hold-over cankers. Study photographs 4a and 4b. OBSERVE:—

9. The discolored and slightly sunken tissues about the base of the stem and extending toward the blossom end. The sticky, milky drops oozing from the diseased area. A large one on the pedicle. Make DRAWINGS (from specimens or photographs) showing these different phases of fruit-blight.

Twig-blight. The bacteria from the diseased blossoms and fruits are carried by sucking insects to the tips of the growing shoots and water-sprouts and are there introduced through the wounds or punctures made by the insects, into the tender succulent tissues. Here they multiply rapidly killing the shoot, causing the form of the disease known as twig-blight. Blighted and healthy twigs (fresh and preserved) are provided. Examine and OBSERVE:—

10. The contrast between the diseased and healthy portions of both the twigs and leaves. You may be able to find the dried ooze. (See illustration specimen.) SKETCH to show contrast between diseased and healthy twigs.

11. That in some of the specimens the dormant buds in the axils of the leaves just below the blighted portion have been prematurely forced. Explain this. (See illustration specimen or photograph 6.)

Origin of the cankers. Examine the specimens of cankers again very carefully and OBSERVE:—

12. That the cankers almost always surround the base of the spur, twig or watersprout, or that there is at least one or more within the area. Sometimes it is a fruit-spur, the bacteria had entered by way of the blossoms; more frequently it is a water-sprout, the tender succulent growth of which is very favorable for the rapid development of the disease. The bacteria kill the water-sprout down to the trunk or limb and spread out into the bark often for a considerable distance around the base of the sprout. (See illustration specimens and photograph 8.) If the previous DRAWING of the canker does not show the dead spur or water-sprout, DRAW to show it.

Cankers occasionally originate in other ways. Sometimes the pruning-knife carries the inoculum. Cankers may start from insect-wounds in the bark. (See Ontario Bul. 176:33-38.)

ETIOLOGY

The organism that causes this disease is *Bacillus amylovorus* (Burrill) Trevisan.

Life-history. Laboratory studies in the life-history of this pathogene, aside from the facts brought out in the above study of symptoms, are necessarily limited to a short study of the morphology of the bacillus. Before proceeding with the following observations the student should have read carefully that portion of the text* dealing with the life-history of the organism.

If pure cultures are available, examine them carefully and OBSERVE:—

13. The character of the growth on the surface of the solid media. The effect on the bouillon. Why? How long has it taken to produce this effect? What does that indicate as to the rapidity of multiplication? Make a mount from a pure culture and before covering introduce a bee's foot. OBSERVE:—

14. The size and motility of the organism; cell-unions. Compare it in size with the claw of the bee's foot. It may now be understood how a bee or fly might carry thousands of these on its feet. DRAW the bee's claw and some bacteria beside it. Maintain relative proportions.

Study photograph 9 or the stained mount under the demonstration microscope and MAKE OUT:—

15. The cilia; their number, length and distribution over the thallus. They are peritrichic. It is this character that places the organism in the genus *Bacillus*.

If fresh material is available crush a bit of the most recently affected tissue in water on a slide, cover and try to DEMONSTRATE:—

16. The living bacteria. How do they compare in size, form and abundance with those from the pure culture? Are they motile?

Pathological Histology. Cross-sections of fresh growing twigs showing both healthy and diseased tissues are to be studied (supplemented with stained section provided). Examine the healthy portion and OBSERVE:—

17. The reddish brown outer coat, made up of the epidermis and the several layers of cork-cells beneath.

*This refers to any text which is available.

18. The large globose or oval cortex-cells, closely packed next the cork-layer but with looser arrangement toward the wood. Their color and contents.

19. Just beneath and partly surrounded by the cortex-cells, bundles of white densely packed sclerenchyma-fibers (bast).

20. Toward the center in the following order, the phloem, cambium, and xylem.

21. At the center of the cross-section,—the pith; the medullary rays distributed radially in the vascular cylinder.

Study the diseased portion of the twig, comparing the blighted tissues with the healthy tissues already examined. OBSERVE:—

22. The affected area. Which tissues are involved? How recognized? Are all the cells within the diseased region killed?

23. The effect of the pathogene on the epidermis, cork, cortex, sclerenchyma-fibers, medullary rays, phloem, cambium, xylem and pith. Compare with the healthy condition of the cells as to form, size, color and contents.

24. The relatively large cavities scattered throughout the affected portion. How may these be accounted for?

25. The stratified appearance just outside of the phloem. To what due?

26. The apparent absence of the bacillus. Why not evident?

DRAWINGS:—(a) Make a diagrammatic drawing to show the healthy and diseased tissues. (b) A much larger and detailed drawing of a portion of a cross-section of the affected twig. Make the drawing include both healthy and diseased bark.

Study closely the margin of the lesion. In case the section was taken from a twig in which the pathogene was inactive the diseased and healthy tissues will be separated by a cork-layer. If the pathogene was active no cork-layer will have yet been formed. Determine which was the condition in the twig from which the section provided was cut. In the apex of growing twigs, the parasite may invade the xylem-ducts and thus migrate some distance in them down the twig.

REPORT

1. In working out methods of control, of what importance are the following facts about fire-blight:—

- a. It occurs only in North America.
- b. The bacteria causing the disease pass the winter in hold-over cankers in any of its numerous hosts.
- c. The bacteria get into the host through wounds.
- d. The chief agents of inoculation are certain insects.
- e. The bacteria are usually introduced into the young and growing parts of the host where in these succulent tissues they multiply and develop the disease very rapidly.

STREAK OF SWEET PEAS

This bacterial disease affects not only sweet peas but many other legumes including clover.

SYMPTOMS

Examine the specimens provided (fresh, dry, or in liquid). OBSERVE:—

On the stems.

1. The streak-like lesions; color, extent, part of stem affected. Look for young isolated lesions; old lesions. How do they differ as to extent and color. DRAW to show variation in stem-lesions. Label to indicate colors.

On the leaves and tendrils.

2. The similarity of the lesions to those on the stem. Find a stem-lesion that has extended along the petiole into the blade at its base. Note the reddening of the affected veins and the dead blade-tissues between. DRAW.

3. Locate isolated lesions or spots in the leaf. Study carefully. DRAW and label fully as to markings and colors.

On the flower.

4. The character of the lesions on the petals. Especially striking in dark flowers. DRAW.

ETIOLOGY

The cause of this disease, long unknown, has recently been shown to be *Bacillus lathryi* Manns and Taubenhau. This pathogene appears to be widely distributed in England and United States.

Life-history. Little is certainly known of the habits of this parasite. It is supposed to pass the winter in the soil and decaying plant parts which were killed the previous year.

The **Primary Cycle** is initiated about the time the peas begin to blossom.

Pathogenesis. The bacteria in the soil are splashed by the rain upon the plants and gain entrance to the tissue through the stomata. Peel the epidermis from the stem of a healthy sweet pea. Mount outside up and OBSERVE:—

5. The epidermal cells; their shape and arrangement. The stomata; numbers and structure. DRAW. These stomata are the infection-courts. The bacteria in the moisture on the stem pass through the stomatal opening into the substomatal cavity. Here they multiply and by their toxic secretions kill the adjoining cortical cells. The disorganized juices of these cells diffuse into the substomatal cavity affording food for further growth and multiplication of the pathogene.

Make sections through a young lesion on the stem (or use prepared slides). Examine and if possible DETECT:—

6. The bacteria in the tissues. Are they between or in the cells? DRAW to show the relation of the bacteria to the cells.

Saprogenesis. Whether the pathogene is able to live and multiply in the soil is an open question. It is, however, readily cultivated

on nutrient media where it can be better studied than in the tissues of the host. Examine the plate-colonies of *Bacillus lathryi*. OBSERVE:—

7. Their form, size, color and consistency. DRAW.

Mount a bit of one of the colonies in a drop of water. Cover and OBSERVE:—

8. The size, shape, motility and cell-unions of the bacteria. DRAW; and COPY also from Delaware Bul. 108:22, fig. 1.

Secondary Cycles originate from bacteria oozing from the primary lesions and spattered by rain to nearby leaves and stems. They do not differ in any other way from the primary.

Pathological Histology. Study the prepared sections (cross- and longi-sections) through lesions on the stem. OBSERVE:—

9. That at first the bacteria occupy the intercellular spaces, adjacent to the substomatal cavity.

10. That these soon break down the cell-walls and invade the cell-cavities. (See Delaware Bul. 108, pl. I.) To what is the color in the lesion due?

11. That not all the tissues of the stem are invaded. Determine which. Show this in a DRAWING of the section through the stem-lesion.

Pathogenicity Studies. Under the direction of the instructor the student will, if fresh material is available, make isolations from stem-lesions on nutrient agar or potato-agar in a petri-dish. Set the petri-dish away until the next exercise at which time compare with the pure culture studied above.

If growing sweet peas and clover are available, inoculate under the direction of the instructor, with these pure cultures of the pathogene. Examine the plants after two days and again later for evidences of infection. Record in your notes the length of the incubation-period.

REPORT

1. Describe fully how the pathogene was isolated, character of its growth in the media used, how the inoculations were made and the results.

BEAN BLIGHT

This is the common bacterial disease of beans. It apparently affects all varieties of *Phaseolus vulgaris* L., the field- and garden-bean, as well as the Lima bean, *Phaseolus lunatus* L.

SYMPTOMS

This disease affects all parts of the host above the ground, leaves, stems, pods and seeds.

On the leaves. Examine the leaves provided and OBSERVE:—

1. The location, size, color and general appearance of the lesions on both sides of the leaf.

2. The zonate character of the spots. How do you account for each zone, especially the opaque, green water-soaked zone which shows best when the leaf is held to the light; also the pale green outermost zone?

DRAW to show these characters and indicate fully in the labeling, the color and appearance.

On the stems. The affected stems show no distinct lesions since only the vascular system, as a rule, is usually involved. Diseased stems finally shrivel following the wilting of the leaves. This symptom is not common.

On the pods. Examine the pods provided and OBSERVE:—

3. The location, size, color and water-soaked appearance of the lesions. Pod-lesions are sometimes red-bordered. (See illustration specimens.)

4. Depth to which the lesions penetrate. Determine by cutting across the pod through a lesion. DRAW to show form and depth of the lesion. Label fully as to color and character.

On the seed. Examine the lesion with a hand-lens and OBSERVE:—

5. The discolored, roughened surface. To what is this due? What relation do the lesions on the seed bear to the pod-lesions? Make a DRAWING showing the character of the lesions on the seed.

Examine the passe-partouts provided and compare the symptoms of this disease with those of the *anthracnose*, a fungous disease of the bean which is also very prevalent. Make DRAWINGS of a pod, seed and leaf affected with anthracnose, showing how the symptoms of that disease differ from those of the bean blight.

ETIOLOGY

The cause of this disease is *Bacterium Phaseoli* E. F. Smith (= *Pseudomonas Phaseoli* EFS.), a monotrichic bacterium which produces yellow colonies on agar.

Life-history. This organism is a restricted parasite, normally passing its entire life within the bean. It probably has no true saprogenic phase in its life-history.

The **Primary Cycle** is initiated in the spring from the cotyledons of diseased seedlings.

Pathogenesis. Diseased seed have been soaked a short time in water. Peel the seed-coat from the lesion. Make two mounts, one from the diseased seed-coat and one from the diseased cotyledon, chopping and crushing the tissues. OBSERVE:—

6. The minute, short rod-shaped bacteria to be detected only with the high-power. Are they inside or outside the cells? DRAW.

The bacteria in these cotyledons are carried by insects or, oozing out upon the surface, are splashed by rain to the leaves. The insects introduce the pathogene into the wounds which they make or the bacteria may enter through the stomata, thus initiating primary infection. The primary infections which are seldom to be observed, exhibit the same type of leaf-lesion as the secondary infections. The primary cycle is probably never completed, the bacteria in the primary lesions dying with the disintegration of the leaves.

The **Secondary Cycle** infections are far more numerous and therefore are more destructive than the primary. They are the ones most commonly observed. They are repeated frequently throughout the season, initiated at first from the primary and later from earlier secondary infections.

Pathogenesis. The pathogene is carried by insects or rain from the primary lesions to healthy leaves and pods. Examine several of the leaf-lesions carefully to see if you can **LOCATE**:—

7. The point at which the bacteria were introduced. Was the inoculating agent a biting or sucking insect? Was the lesion a result of stomatal invasion?

With a scalpel, chop in a drop of potassium hydroxide (do not crush) a bit of leaf-tissue from the outer opaque green zone of the lesion. Cover carefully and under low-power **OBSERVE**:—

8. The bacteria oozing from the cut ends of the veins. Make a **DRAWING** of a vein with oozing bacteria.

Examine several deep pod-lesions, cutting across the pod through the lesion or shelling out the beans. The bacteria in the pod invade the seed, where, with the ripening of the seed, they become dormant, thus completing the secondary cycle. Can evidences of invasion of the seed beneath the lesion be detected?

Saprogenesis. Although there is no evidence that this pathogene lives and multiplies outside the living tissues of the bean, some investigators hold that it may live over winter in the stems and leaves on the ground (Ontario Bul. 136:12.). It will, however, grow readily on culture media. Examine the plate- and tube-cultures and **OBSERVE**:—

9. The color, form and consistency of both the buried and surface-growing colonies; effect on the bouillon-cultures. **DRAW** and label to indicate color and other characters.

Make a mount from one of the surface-colonies and **COMPARE**:—

10. The bacteria with those studied from the tissues as to size, shape, cell-unions and motility. **DRAW**.

For flagella see Cornell Bul. 239:211. **COPY**.

Pathological Histology. Examine the sections (freehand or prepared) of diseased pods, stems, or leaves. **OBSERVE**:—

11. Which tissues are chiefly invaded in the organ examined.

12. The relation of the bacteria to the cells; within or between?

13. The effect on the cells; on the middle lamella and protoplast.

Make **DRAWINGS** necessary to bring out these points.

REPORT

1. Detail the life-history of *Bacterium Phaseoli* EFS., describing the symptoms resulting from its activities in the different organs of the host. Follow the sequence and indicate the headings, used in this outline.

BLACK ROT OF CRUCIFERS

This is a very destructive bacterial disease of cabbage and cauliflower. It also affects kale, radishes, rape, turnips and mustard.

SYMPTOMS

This disease is detected chiefly by the lesions on the leaves, by the effects on the plant as a whole and on the vascular system of the mid-ribs and the stalk.

On the leaves. Examine the cabbage or cauliflower leaves provided and OBSERVE:—

1. The large irregular dead or dying areas in the blade. The yellowish or light-brown color of the lesions.

2. Their location in the leaf. What does this indicate with respect to the point of initial invasion?

3. That when held to the light the veins of the affected area are black. (See in N. Y. (Geneva) Bul. 232, pl. I and II; or photograph 1.)

4. In fresh material, if available, the difference in texture of the diseased and healthy parts of the leaf.

DRAW to show the character of the leaf-lesions.

On the plant in general. Study the diseased heads provided. OBSERVE:—

5. The long bare stalk and loose dwarfed head often reduced to a mere tuft of leaves (photograph 2).

6. The stubs or leaf-scars on the lower part of the stem where the leaves have died and dropped away.

7. That in some heads the leaves have dropped away from one side only.

8. That diseased seedlings are dwarfed and fail to head. They often wilt and die early. (See N. Y. (Geneva) Bul. 251, pl. I.)

In Smith's *Bacteria in Relation to Plant Diseases* 2, pl. 18, is shown a peculiar effect due to slow killing of the margins of the leaves. Note carefully.

Make DRAWINGS to show these characteristic effects on the plant as a whole.

In the vascular system. Examine the leaf-scars on the diseased stalks. OBSERVE:—

9. The black points,—the ends of diseased vascular bundles. This is a typical vascular disease. Break a leaf from the diseased stem, cut across the petiole and NOTE:—

10. That here some of the bundles are also black. Split the petiole and midrib lengthwise. How far does the blackening extend?

Make transverse and longitudinal cuts through the diseased stalks. OBSERVE:—

11. In the vascular cylinder, the blackened strands which here and there curve out to the margin. Explain.

Make a DRAWING to show the stem with blackened strands both in cross- and longisection.

ETIOLOGY

The causal organism of black rot is *Bacterium campestre* (Pammel) Erwin F. Smith. It is related to the bean blight pathogene, *Bacterium*

Phaseoli EFS., forming like that bacterium, yellow colonies on agar. It is also monotrichic.

Life-history. This pathogene passes the winter either in the debris of the host in the soil or on the seed.

The **Primary Cycle** begins in the seedlings in the seed-bed or in the plants after they are set in the field.

Pathogenesis. If the seed-plants have been diseased the seed may become contaminated with the pathogene when thrashed. On the oily coat of the seed, the bacteria may remain alive for a year or more. Examine the agar-plates on which contaminated cabbage seed have been sown. OBSERVE:—

12. The yellow colonies developing about some of the seed. DRAW. With your needle remove a bit from a colony to a small drop of water on a slide. Cover, examine and MAKE OUT:—

13. The short rod-shaped bacteria swarming in the mount. Just how the pathogene on the seed gets into the seedling is not known, but it probably enters through the young rootlets. DRAW.

The inoculum in the soil in the field is carried to the leaves of the growing plants by snails, insects and probably by the wind.

Examine the cabbage plant under the bell-jar. Here the conditions of humid weather are reproduced. OBSERVE:—

14. The beady drops of water along the leaf-margins. This water is extruded from the water-pores at the ends of the veins. DRAW. In these drops the bacteria multiply and enter through the pore into the cavity at the end of the vascular bundle. Here they multiply rapidly, penetrate into the xylem-vessels and, traveling downward, kill and disorganize the vascular tissues causing them to blacken. The pathogene thus reaches the stalk by way of the leaf-veins.

Saprogenesis. It is not certainly known whether the organism multiplies in the dead and rotting leaf-tissues and in the soil; probably not to any great extent. It grows readily, however, in culture media as has been observed, forming characteristic yellow colonies.

Secondary Cycles are initiated by bacteria carried by gnawing or sucking insects which have fed on diseased tissues. In this way the pathogene is carried from plant to plant and epiphytotics often result.

Pathological Histology. Study the prepared cross-sections and OBSERVE:—

15. The deeply stained diseased vascular bundles. What tissues of the bundle are affected?

16. The relation of the pathogene to the cells; inter- or intracellular?

17. The numberless bacteria filling the spiral and annular vessels. Study the effects of the pathogene on the structure of these vessels as shown both in the cross- and longisections.

Make DRAWINGS to show these effects on the tissues.

REPORT

1. Describe fully the ways in which *Bacterium campestre* (Pam.) EFS. may possibly hibernate and show how these determine the control measures to be employed.

CARATOVORUS SOFT ROT

This is the best known and most common soft rot disease of plants. It affects a great variety of hosts in each of which it produces more or less characteristic and peculiar symptoms.

SYMPTOMS

In carrots or turnips. It was in the fleshy roots of the carrot that this disease was first carefully studied. Examine the diseased roots provided and OBSERVE:—

1. The soft mushy character of the rotted tissues. Crush a bit of it between the thumb and finger.

2. The fetid odor of putrefaction.

3. That the rot is largely confined to the root.

Cut through the lesion and NOTE:—

4. The variations in color and firmness of the diseased tissue from the center of the rotted mass toward the healthy tissue.

DRAW to show these characters.

The rotting of the roots in the field is accompanied in its advanced stages by the sudden wilting of the leaves.

In cabbage and cauliflower. In these hosts the soft rot frequently follows the black rot and causes in cabbage the so-called "stump-rot." Study the diseased heads provided and OBSERVE:—

5. That the rotting is largely in the base of the fleshy leaves (or in the fleshy head of the cauliflower).

6. That the cabbage head is thus loosened from the stalk from which it may be readily lifted leaving a slimy rotten "stump."

DRAW to show these symptoms.

7. The soft mushy character of the rotted tissues; color?

8. The fetid odor of the decomposing tissues.

Split one of the partially rotted mid-ribs of the leaf or a diseased branch of the cauliflower head. Examine and NOTE:—

9. The differences in color and consistency of the diseased tissues as the healthy tissue is approached.

In iris. This disease is very destructive to the iris both in Europe and America. Examine the diseased leaves provided and OBSERVE:—

10. The soft rotted basal portion. Crush a bit of it between the fingers. If fresh material is available, split a healthy and a diseased leaf and compare as to color and odor.

11. That the epidermis of the diseased portion is more or less intact forming a rather firm covering for the rotten mass within.

12. The upper advancing portion of the lesion, firmer than below but dark and water-soaked in appearance. SKETCH external and internal view.

13. That the tips and margins of some of the leaves rotted at the base are dying and drying out. How may this be explained?

The most striking field-symptoms are the falling-over on the ground of the outer affected leaves or the shriveling and browning of the tips of the leaves of the cluster. This latter symptom is, however, easily confused with the dying of the tips due to a fungous spot-disease common on iris. (See demonstration specimen.)

SKETCH to show field symptoms. (See demonstration material.)

14. Diseased rhizomes if available. Make a similar comparative study and DRAW.

ETIOLOGY

The pathogene responsible for this soft rot in such a variety of hosts is *Bacillus caratovor* Jones. It was first described from carrots.

Life-history. This organism doubtless hibernates in host-debris or in the soil itself.

The **Primary Cycle** is initiated when the particular host concerned is in the best stage of its development for the attack. Succulent tissue is a requisite. The iris is usually affected very early in the spring while carrots, turnips, cabbage and cauliflower are first attacked late in summer or autumn.

Pathogenesis. The source of the primary inoculum is doubtless the soil or rotting host-tissue in the soil. The bacteria are carried to the roots or rhizomes by snails, insects and probably flowing water. They can enter apparently only through wounds. Once within susceptible tissue with abundant moisture present they multiply rapidly and cause the disintegration of the parenchyma.

Remove a bit of the rotten tissue to a drop of water on a slide, cover and examine for the bacteria. OBSERVE:—

15. Their abundance, and relation to the cells of the host. Are they within or without the cells; motile or non-motile; single or in threads? Are spores present? DRAW.

16. The photomicrograph showing the flagellate stage of the pathogene. How many flagella has each bacillus and how are they arranged? DRAW.

Saprogenesis. This organism is doubtless able to multiply in host-debris in the soil so that virulent active inoculum is always present where the pathogene has flourished the previous season. It grows readily on culture-media.

Examine the pure culture provided. SKETCH and DESCRIBE:—

17. A typical colony as to form, color, consistency and odor.

Make a mount from one of the colonies and compare the bacteria with those from the tissues as to the points indicated in No. 15.

Secondary Cycles are initiated from, and develop in the same manner as the primary cycles.

Pathological Histology. Make several thin longisections through a small piece of the healthy organ (root, leaf-base or rhizome). IDENTIFY:—

18. The various tissues present. The cell-contents and organs in each. Make a DRAWING detailing a portion of the section, a few cells wide, from epidermis toward the center. Show with especial care the intercellular spaces and contiguous walls of the cells.

Remove the epidermis from over the soft portion of a lesion. With a needle or point of a scalpel remove a bit of the rotten tissue to a drop of water on a slide, cover and gently press on the cover. Study and OBSERVE:—

19. That the individual cells are separated from each other but are still intact. Identify the kinds of cells seen with those in the healthy leaf. Compare their shape with corresponding healthy cells.

20. The disorganized and plasmolized character of their contents. What organs of the cell can still be identified?

DRAW to show these points and the relation of the bacteria to the cells.

Examine the prepared longisections through the advancing margin of the lesion and see if it can be DETERMINED:—

21. Just where the diseased cells leave off and healthy ones begin. On what characters are the conclusions based? What tissues are affected?

22. How the cells are separated from one another. Relation of the bacteria to the separating cells.

DRAW to show the cell-structures, dissolving middle-lamella and bacteria at the advancing margin of the lesion.

Pathogenicity studies. To give the student some idea of the rapidity with which the bacteria destroy tissue, a simple inoculation experiment may be made as follows:—

(a) Cut three short pieces of healthy leaves of iris, cabbage or cauliflower and three thin slices of roots of carrots or turnips and place them in clean petri-dishes on wet filter-paper. (b) Flame a needle and puncture one piece of each; these constitute the checks. (c) Flame the needle and when cool, draw the point through the pure culture and then puncture a second piece of each. (d) Flame the needle and when cool, draw the point through the soft diseased tissue and puncture the remaining pieces. Label carefully and set away until next exercise when they should be examined. Record by sketch or description the results of the experiments.

The instructor will demonstrate the method of isolating the pathogene to get it in pure culture.

REPORT

1. Describe and explain in detail the pathological effects of *Bacillus caratovorius* Jones on susceptible plant-tissues.

DAMPING-OFF OF SEEDLINGS

The name damping-off is applied to a type of disease affecting seedlings rather than to any one specific disease. Many species of quite different groups of fungi are known to cause damping-off, but as here used the name will be restricted to the diseases produced by certain phycomycetous fungi.

SYMPTOMS

The most striking symptom and the one which gives to this type of seedling-disease the name damping-off, is the peculiar effect of a necrotic lesion in the stem at or near the surface of the ground. Often the lesion originates just below the surface of the soil. A wilting and then a disintegration of the tissues affected, results and the plantlet falls over and dies. Root-rot may accompany damping-off and may prove fatal except where adventitious roots are put out above the lesion and remain healthy.

Study the damped-off seedlings provided and photographs 1, 2, 3, 4 and 5. OBSERVE:—

1. The withering of the stem-tissues at or near the surface of the soil.

2. The sharp angle made by the fallen stem. While affected plants usually fall over, they may remain erect and wilt if the vascular tissues are sufficiently rigid.

3. The dying of all the plants in affected areas. These damped-off spots are quite characteristic of infested seed-beds.

DRAW to show these characteristic symptoms.

ETIOLOGY

A rather large number of fungi are now known which produce the above symptoms in seedlings. (See text.) As indicated above, only phycomycetous forms will be here considered. Of these it is one of two species that is usually responsible, either *Pythium de Baryanum* Hesse or *Phytophthora Cactorum* (Cohn and Lebert) Schroeter. These two species are very much alike in structure and habits so that the following outline should serve for either.

Life-history. These pathogenes exhibit in their life-history both primary and secondary cycles, with a short period of pathogenic activity and a well-marked saprogenic phase, especially in the case of *P. de Baryanum*.

The **Primary Cycle** is initiated at or shortly after the germination of the host. Sometimes the seedlings are killed before they get above ground.

Pathogenesis. Both fungi pass their saprogenic existence in the soil, probably always in association with host-debris. Both produce resting sexual spores,—the oospores, which carry them through the winter. These germinate and produce the inoculum for the primary cycle. Mount and examine some of the mature oospores provided (in pure cultures).

OBSERVE:—

4. The form, size and color of the sexual bodies.

5. The old oogonial sac, the outer covering enclosing the single oospore. The thick smooth wall of the oospore and its densely granular, oily contents. DRAW.

Lying in the soil near the seedling, these oospores germinate (in the case of *P. Cactorum* at least) by sending forth a germtube at the tip of which is produced one or more conidia. Study Cornell Bul. 363, fig. 18. COPY one or more of the drawings.

These conidia germinate either by swarmspore-formation or by germ-tubes and infect the tender tissues of the seedlings. (See Cornell Bul. 363, fig. 16-17.) Conidial germination will be studied later.

The mycelium developed from the primary inoculum spreads rapidly in the succulent tissues of the seedlings, secreting enzymes which enable it to penetrate the cellulose walls, and toxins which kill the protoplasm. Study thin longitudinal sections (freehand or prepared) through diseased stems. OBSERVE:—

6. The relation of the mycelium to the host-cells; inter- or intracellular? Are haustoria produced?

7. The structure of the mycelium; densely granular, branched, non-septate, often knobbed and swollen. The form and structure may often be better made out in mounts from pure culture.

Make and study mounts from cultures provided. OBSERVE:—

8. That frequently there is a streaming of the cytoplasm in the long non-septate cells.

DRAW, showing structure of the mycelium and its relation to the host-tissues.

From this mycelium is sent forth from the diseased tissues short fruiting branches,—the conidiophores, on which are developed conidia just like those produced from the oospore. Make mounts from diseased stems or from pure cultures showing conidia. Study and OBSERVE:—

9. Their peculiar form, apical papillae and coarsely granular contents.

10. Their attachment to the conidiophore at their larger end. DRAW showing conidiophores with conidia. These conidia function as the inoculum for the secondary cycles. In *P. de Baryanum* it is held that mycelium spreading through the soil from seedling to seedling usually functions to initiate the secondary infections. This may also be true at times for *P. Cactorum*.

Saprogenesis. The mycelium in the dead tissues continues to grow and produce conidia so long as the food supply is not exhausted. As the nutrients begin to fail there develops on the mycelium within the tissues the sexual bodies, which eventually mature into oospores. Tease apart in a drop of water bits of diseased stems which contain forming oospores or use mounts from pure cultures provided. OBSERVE:—

11. The large globose bodies,—the oogonia, and the smaller closely appressed antheridia.

12. The manner in which they are borne on the mycelium and their positional relation to each other.

13. Their thin, hyaline walls and granular contents.

14. The aggregation in some, of the denser protoplasm in the center,—the oosphere, and the surrounding layer of hyaline protoplasm,—the periplasm.

15. The impregnation-tube penetrating from the antheridium, through the oogonium wall and periplasm to the oosphere. What is the function of this tube?

Make **DRAWINGS** showing the structure of the sexual bodies and fertilization of the oosphere.

The fertilized oospores now go into a resting-period and will not germinate at once. Normally this dormant period extends through the winter. During the spring, changes in the oily protoplasm of the oospore take place which make its germination possible. With germination of these oospores (already studied) the primary cycle is completed.

Secondary Cycles are initiated repeatedly during the growing-season as has already been pointed out, either by conidia or by mycelium spreading through the soil. If viable conidia are available study conidial germination on the slides provided; or Cornell Bul. 363, fig. 16-17; and read p. 97-99.

Make **DRAWINGS** illustrating ways in which conidia (sporangia) germinate; also the structure and germination of the swarmspores. How do the germtubes gain entrance to the host-tissues? Why can they not penetrate mature tissues?

The further developments of the pathogene during the secondary cycles duplicate those of the primary.

Pathological Histology. The pathologic effects of these pathogenes are of the simplest sort—a direct killing of the protoplasts—necrosis. Study sections through the advancing margin of the lesion, using fresh material. **OBSERVE:**—

16. The plasmolyzed and disorganized cell-contents.

17. The water-soaked appearance of the diseased tissues due to the diffusion of the cell-sap into the intercellular spaces, forcing out the air.

DRAW to show these effects.

Pathogenicity Studies. The rapid destructiveness of these pathogenes in a bed or flat of closely crowded seedlings is strikingly brought out by a simple inoculation experiment.

Under the direction of the instructor each student will inoculate the seedlings provided, with pure cultures of one or the other of these fungi. Keep under moist conditions and examine daily for first evidences of infection and successive symptoms. Make notes on conditions under which the inoculated plants were kept, length of incubation-period, effects of removing plants to dry conditions before all go down and other facts observed.

REPORT

1. Describe in detail and explain the functions of the structures developed by either of these pathogenes during its life-cycles.

2. Prepare a complete report on the pathogenicity-studies made.

LATE BLIGHT OF POTATOES

This is the most destructive disease of the potato in the humid temperate regions of the world. It is especially severe in northeastern United States.

SYMPTOMS

During damp, muggy weather in August and September, circular areas in the fields are often to be observed in which all the tops of the potatoes are dead or dying (photograph 1). (See also N. Y. (Geneva) Bul. 241, pl. VII, and 264, pl. II, III, VIII, XIV, XV.)

On the leaves. Examine the material provided and OBSERVE:—

1. The large spots or lesions with indefinite margins. Their position in the leaflets (photograph 2).

2. On holding them to the light, the dark water-soaked or opaque appearance of the diseased areas.

3. On the lower surface of the leaflet, a white frost-like mildew about the margins of the diseased spots. (See illustration specimen; photograph 2; or N. Y. (Geneva) Bul. 241, pl. XI.) In dry weather this mildew may be difficult to see.

4. That the lower leaves are usually the first attacked. They die and shrivel on the stem. (See illustration specimen; or N. Y. (Geneva) Bul. 241, pl. X.)

Compare these leaf-spot symptoms with those of the *Alternaria* blight as shown in the material and photographs provided.

Show in DRAWINGS the difference in leaf-symptoms of these two diseases. Show both sides of the spots.

On the tubers. The disease may also occur on tubers, causing a reddish brown rot. (Read Connecticut Ann. rept. 1904:364, second paragraph and see pl. XXXIII; also photographs 4a and 4 b.)

Study the material provided and OBSERVE:—

5. The sunken spots on the surface of the diseased tubers provided. To what due?

6. The color and consistency of the diseased tissues shown on cutting the tuber open. How deep do the lesions extend?

DRAW to show tuber-symptoms as observed in specimens and photographs.

This disease is also destructive to the tomato in some sections of the United States. If material is available study the symptoms on this host and show in DRAWINGS. (See California Bul. 175:9 and fig. 3, 4 and 5; and Virginia Bul. 192:7-10 and fig. 5, 6, 7 and 8.)

ETIOLOGY

One of the phycomycetous fungi, *Phytophthora infestans* (Montagne) de Bary, is the cause of this disease. The species of *Phytophthora* are all very virulent and destructive pathogens.

Life-history. So far as is certainly known this pathogene remains in continuous association with the living host throughout its life-cycles.

The **Primary Cycle** originates from the activity of the mycelium in diseased tubers after they are planted in the spring.

Pathogenesis. The fungus passes the winter as mycelium in diseased tubers. Make thin tangential sections of the diseased tubers, stain with eosin and OBSERVE:—

7. The mycelial threads between the cells. Their densely granular contents. Note the absence of haustoria. (Read Connecticut Ann. rept. 1904:364, last paragraph.)

8. The general absence of septa in the mycelium; this fungus is a cenocyte. Toxines secreted from this mycelium kill the adjoining host-cells and their soluble contents are set free for the fungus.

DRAW, or COPY a part of fig. 6 in California Bul. 175 to show character of the mycelium and its relation to the cells of the tuber.

The mycelium in the diseased tuber grows out into one or more of the shoots resulting in a systemic invasion. The diseased shoot is weak and dwarfed. From such shoots conidia are produced in great abundance. Such plants become centers of dissemination or sources of inoculum for the primary infections. After these conidia are produced and dispersed the fungus in these systemically invaded shoots dies with the host. (Read in Jour. Agr. Research 5:99–102.) COPY pl. VI, fig. 1.

The primary infections occur on the leaves; are relatively few and serve to afford inoculum for the secondary cycles. With the death of the leaves the pathogene in them perishes and the primary cycle is not completed. The lesions and pathogene-structures are like those resulting from the secondary infections in the leaves.

The **Secondary Cycles** arise from the conidia produced on the primary lesions. These conidia are distributed by wind during rainy or foggy weather, to nearby healthy plants where they germinate.

Pathogenesis. Make mounts of these conidia from material provided (freshly blighted leaves or cultures, if available) and OBSERVE:—

9. The shape, contents and color of conidia. These conidia upon germination usually act as sporangia, giving rise to swarmspores.

Study germinating spores, if available; N. Y. (Geneva) Bul. 141, pl. XII, and California Bul. 175, fig. 8; or photograph 5. MAKE OUT:—

10. The manner of germination; number and form of swarmspores and their motility by flagella.

DRAW to show points observed or COPY from plates or photographs studied.

After swimming about for a short time in the drop of rain or dew on the host-leaf, the swarmspores come to rest, round up, invest themselves with a thin cellulose wall and germinate by a germtube. Study slides; figures in the bulletin; or photographs 5 and 6. OBSERVE:—

11. The relative size of the germinating swarmspore and the conidium. The difference in form of the active and germinating swarmspores.

12. The two methods of entrance of the swarmspore-germtube into the leaf. COPY to show this.

This germtube gives rise to mycelium within the leaf-tissues like that observed in the tuber. Haustoria-like branches are often sent from the intercellular mycelium into the leaf-cells. From this mycelium, branches are put forth on the underside of the leaf to form the conidiophores.

Scrape some of these conidiophores from the leaf and mount in water. OBSERVE:—

13. Their form and structure. Distinguish the conidiophores from the large leaf-hairs. Note the peculiar swellings on the branches.

14. The ovate conidia; their manner of formation and their attachment to the conidiophores. (See also photograph 5 or N. Y. (Geneva) Bul. 141, pl. XII.)

If freshly diseased leaves are available, peel epidermis from lower surface of spot. Mount and study, MAKING OUT:—

15. How the conidiophores emerge from the tissues of the leaf. (See also N. Y. (Geneva) Bul. 241, pl. XII, fig. 2; Maine Bul. 169, fig. 15 or California Bul. 175, fig. 7.)

Make a DRAWING to show the leaf-surface, conidiophores emerging and conidia attached both at the apex and side of the branch.

These secondary conidia in turn give rise to other secondary infections on healthy leaves or they fall to the ground during rains and produce swarmspores in the soil. These find their way to the growing tubers which they penetrate by a germtube. This gives rise therein to the mycelium that is to carry the pathogene over to the next season and invade the shoots and develop conidia for the primary infections. Thus are the secondary or at least the late secondary cycles completed.

Saprogenesis. As already indicated, a true saprogenic development of this pathogene in nature is not known. It will, however, grow as a saprophyte on certain culture-media. Here it will sometimes produce not only conidia but also resting-spores, called oospores. These have never certainly been observed in the tissues of the host. One might expect to find them in the dead tops killed by the pathogene or in the rotting tubers since other species of *Phytophthora* regularly form oospores which are long-lived and serve to carry the fungus through the winter or drought-periods. Worthington G. Smith thought he had discovered them. (See *Diseases of Field and Garden Crops* p. 295-310.) No observer since Smith has been able certainly to find them in nature. Jones of Vermont, found in his pure cultures, bodies which are strikingly like the oospores figured by Smith. (See *Science* 29:271.) Finally Clinton of Connecticut, succeeded in growing the oospores of *P. infestans* on oat-agar, and has even produced what he believes are hybrid oospores of *P. infestans* with *P. Phaseoli* and also with *P. Cactorum*. (See *Connecticut Ann. rept.* 1909-1910:753-774, pl. XXXVIII-XL.)

REPORT

1. Discuss tuber-infection; where and how it occurs; two methods of prevention, explaining why each method is efficient.
2. Give detailed directions for the control of the disease.

DOWNY MILDEW OF CUCURBITS

This is a well known and much dreaded disease of cucumbers and muskmelons, ranging from the tropics to the north temperate regions. It sometimes affects squashes and watermelons.

SYMPTOMS

The lesions of this disease are confined to the leaves. The oldest leaves are first affected. The fruit is dwarfed by the loss of food-supply.

The general effect of the disease on the plant as a whole is quite striking. The affected leaves at the center of the hill, yellow and soon die and shrivel like leaves killed by the frost. (Study Connecticut Ann. rept. 1904, pl. XXX and read N. Y. (Geneva) Bul. 119:158.)

On the leaf. Study the specimens provided. OBSERVE:—

1. The location or distribution of the spots in the leaf-blade.
2. The form, size, nature and color of the spots, both above and below. In the fresh condition the lower surface of the spot shows a purplish tinge due to the color of the conidia of the fungus. This color is lost in drying.

3. Relation of the margin of the lesions to the leaf-veins. Hold to the light to see this.

Make a DRAWING to show the character of the spots on upper and lower surfaces.

4. That after the spots have coalesced the entire leaf becomes brown, shrivels and drops.

Make a DRAWING of a leaf showing this advanced stage of the disease.

ETIOLOGY

This disease is caused by the phycomycetous fungus *Peronoplasmopara cubensis* (Berkeley and Curtis) Clinton. It is one of the "downy mildews."

Life-history. Unlike most of the members of the order Peronosporales, *P. cubensis* appears to develop no sexual or resting-spores (oospores). It is believed that it depends entirely on conidia for reproduction, passing the winter on living hosts in the tropical islands of the West Indies or the extreme southern United States, spreading northward with the advancing summer weather. It may in some cases winter on greenhouse cucumbers. There are then strictly speaking no primary cycles.

Secondary Cycles initiated in the south repeat themselves, spreading northward so that they reach the region of northern United States in late July and August. No saprogenic phase is certainly known for this pathogene.

Pathogenesis. The conidia produced in abundance on the lower surface of the lesions constitutes the inoculum. Scrape some from the diseased leaves provided, mount and OBSERVE:—

5. The large lemon-shaped conidia each with a papilla at the tip. The thin wall and the brown granular appearance of the contents. Make an enlarged DRAWING of a single conidium.

6. That the conidia germinate by the formation of swarmspores. COPY the figures showing the different stages of germination from Connecticut Ann. rept. 1904, pl. XXXI.

These swarmspores swim about in the water on the leaves for a short time, then round up, secrete a wall about themselves and send forth a germ-tube which penetrates the leaf and develops a mycelium within the tissues. The germ-tube may enter through a stoma or bore directly through the walls of the epidermal cells. COPY the figures of swarmspore-germination and penetration from Connecticut Ann. rept. 1904, pl. XXXI.

The mycelium in the leaf is difficult to study, and as it is of the same character as that of the pathogene of grape mildew, the grape stem will be used instead. Cut a thin longitudinal section of the stem provided, stain slightly with eosin and OBSERVE:—

7. The granular mycelial threads between the cells. The mycelium is cenocytic.

8. The button-like haustoria which branch from these threads and push through the cell-walls of the host.

Make a DRAWING illustrating the mycelium with some of the host-cells and the haustoria pressing through the walls.

The mycelium rapidly kills the tissues and as it does so it sends forth through the stomata on the under surface of the lesion long branched fruiting stalks,—the conidiophores.

Scrape with a scalpel several spots on the under side of the leaf and place the substance on a slide in a drop of water. With the high-power, OBSERVE:—

9. The long sparingly branched conidiophores with slightly swollen base, and the beak-shaped tips at the ends of the branches, to which are attached the conidia.

Make an enlarged DRAWING of a conidiophore, showing conidia attached.

These conidia are disseminated by the wind and initiate new secondary cycles.

This pathogene sometimes attacks the wild cucumber, *Echinocystis lobata* (Michx.) T & G., but there is on this host another downy mildew pathogene, *Plasmopara australis* (Spegazzini) Swingle. To show the difference between these two pathogenes examine diseased leaves of *E. lobata* provided and OBSERVE:—

10. The downy white spots on the lower side of the cucumber-leaf.

Make a DRAWING of a leaf showing one of these spots.

Place some of the fungus on a slide and with the high-power, OBSERVE:—

11. The short conidiophores branched at right angles; the blunt tips with short sterigmata; and the smaller almost globose conidia.

Make a DRAWING showing the points observed.

REPORT

1. Discuss fully the control of the downy mildew of cucurbits.

SCLEROTINIA ROT

This is a very common disease of a great variety of plants. It is especially destructive in rainy seasons or in very moist situations. Due to the different symptoms exhibited in a great variety of hosts it has come to be known under several names as; stem-rot in seedlings, soft rot in stored roots and bulbs, and "drop" in lettuce.

SYMPTOMS

The most usual point of injury is the base of the stem, but the leaves of such plants as cabbage and lettuce, especially when packed together in heads are commonly affected. Fleshy roots, when stored together often suffer from this disease. Pods, fruits and twigs sometimes show lesions.

On the stems. The name stem-rot is applied to the disease in those cases where the lesions are chiefly at the base of the stem. The symptoms are in general the same on the different hosts. Examine the diseased stems provided. OBSERVE:—

1. A bleaching of the normal color of the tissues accompanied by a softening and sometimes a water-soaked appearance. If fresh material is available, examine for these characters.

2. The appearance, in a short time if the air is moist, of a densely matted pure white felt of mycelium on the surface of the lesion. Examine diseased specimens which have been kept for a time in a moist-chamber.

3. The large black bodies,—sclerotia, on the surface of the lesion or more frequently within the pith of the stem. These sclerotia are at first soft cream-colored bodies embedded in the white mycelial felt or in the pith. They soon turn black and become firm and tuber-like.

Make SKETCHES to show these distinguishing symptoms.

On the heads of lettuce. "Drop" is frequently a serious disease of lettuce. Examine the diseased plants provided and OBSERVE:—

4. The collapsed and wilting state of the plant. It has "dropped." SKETCH a diseased and healthy plant.

5. The point of injury; extent of the lesion.

6. The soft slimy condition of the affected tissues. Note also odor, color, and consistency of the affected parts. How does this rot compare with the bacterial soft rot?

7. The loose white felty masses of mycelium, on and between the decomposed leaves. Larger masses of very compact aggregations of mycelium which are the beginnings of sclerotia.

8. In somewhat older material the mature sclerotia which have become very hard with black, tough outer walls.

On fleshy roots. As a storage-rot of carrots and turnips, this disease is often exceedingly destructive. Examine the diseased roots provided. OBSERVE:—

9. The white felty mat of mycelium covering the surface of the lesion.

10. The hard tuber-like sclerotia; some mature black and hard, others just forming as white cottony masses on the mycelial felt.

11. The soft decay of the tissues beneath the mycelial felt; odor, color, taste.

12. The water-soaked zone in the tissue about the soft mycelium-coated center of the lesion.

DRAW to show the external appearance of rotted roots.

Cut the root lengthwise through the lesion. OBSERVE:—

13. The depth to which the lesion extends. What tissues of the root are involved?

DRAW to show internal characters of the rot in the roots.

ETIOLOGY

The disease is caused by the ascomycete, *Sclerotinia Libertiana* Fuckel (= *Sclerotinia sclerotiorum* (Libert) de Bary).

Life-history.

Primary Cycle.

Pathogenesis. The source of inoculum for the primary cycle is the apothecium developed in the spring from the overwintered sclerotium. Examine the specimen provided. OBSERVE:—

14. The large black sclerotium and the trumpet-shaped apothecium with stalk and concave disk. DRAW. From this apothecium are shot forth the ascospores which constitute the inoculum of the primary cycle.

The character of this inoculum may be determined as follows: Gently break the seal and remove the cover from the glass preparation-dish in which are living apothecia. Watching sharply, OBSERVE:—

15. The tiny cloud of spores that puff from the disc of the apothecium. This cloud is composed of ascospores which caught by the breeze are borne about and lodging in a suitable infection-court initiate the cycle. All of the ascospores of an apothecium are not discharged at one time.

With your forceps carefully remove a bit from the edge of the apothecium and crush in a drop of water on a slide. Cover and study. OBSERVE:—

16. The ascospores; their form, number in each ascus, and color. DRAW to show several ascospores much enlarged.

Once within the infection-court where sufficient moisture is available, these germinate forming a germ-tube which soon branches to form mycelium and finally penetrates into the host-tissues. Examine the germinating ascospores provided. OBSERVE:—

17. From what place on the spore the germ-tube arises; relative size of spore and germ-tube, branching, contents. DRAW. This germ-tube branches forming the mycelium which spreads through the tissues, causing the rot.

Remove a bit of the rotten tissue and tease apart in a drop of water on a slide. Cover and examine for mycelium. OBSERVE:—

18. Size, septation, branching, color, relation to host-cells, (inter- or intracellular?). Do haustoria occur?

As this pathogene grows readily in culture, the mycelium may be advantageously studied in the agar-culture in the petri-dish provided. Carefully cut out a small square from the advancing margin of the colony, transfer to a slide in a drop of water, cover and with little or no crushing, study the mycelium, comparing this with that found in the tissues.

DRAW to show the characters of the mycelium of *S. Libertiana*.

Saprogenesis. No conidia are probably ever produced by this fungus during its normal life-cycles; certainly not during pathogenesis.

Sometimes in very old artificial cultures or where the ascospores germinate in non-nutrient media, so-called microconidia are produced. (See text.) These have never been observed to germinate.

When the mycelium has killed the tissues, it rapidly extracts and appropriates the food-substance therein. As this food-supply begins to dwindle the mycelium forms gnarls of short intertwining hyphae at different points in or on the lesion. These rapidly enlarge, forming the mature black sclerotia.

Make thin sections of one of the sclerotia and OBSERVE:—

19. Its structure; (a) the black outer layer or rind made up of about two or three layers of cells whose walls are thick and brown to black; (b) the inner white medulla made up of densely interwoven thick-walled hyphae. These sclerotia are storage-organs and the food-substance stored is largely in the thickened walls of these medullary hyphae.

DRAW to show structure of rind and medulla much enlarged. (See de Bary, *Morph. and Biol. Fungi* p. 30–32, fig. 14.)

During a drought or cold weather all the mycelium, except that in these sclerotia, dies. The sclerotia pass the winter in the soil or old host-debris, in a dormant condition. In the spring they germinate; that is, from them develop the apothecia in which are produced the ascospores. (See de Bary, *Morph. and Biol. Fungi* p. 52–53, 218–219.)

Make thin sections through the apothecium provided or use prepared slide. MAKE OUT:—

20. Asci with discharging-pore at apex; ascospores, paraphyses, and hypothecium; also structure of stipe and excipulum. DRAW a portion of the section to show these structures in the proper relation to one another.

Secondary Cycles. These may originate from mycelium which spreads through the soil from diseased to neighboring healthy plants. In this case the mycelium serves as inoculum. Aside from this the secondary cycles duplicate the primary.

Pathological Histology. Mount a bit of the diseased tissue from just back of the advancing margin. DETERMINE:—

21. The effects of the pathogene on the host-cells. DRAW to show this.

This mycelium secretes a toxine which kills the host-cells. De Bary believed he had shown this to be oxalic acid. When the sclerotia are forming, large drops of liquid are extruded upon their surfaces. This also contains the toxine.

If cultures showing these drops on the sclerotia are available, remove with the end of a needle-handle one of the drops to a clean slide; quickly place a small drop of calcium nitrate solution beside it, place under the low-power and OBSERVE:—

22. The formation of calcium oxylate crystals as one causes the two drops to come in contact, using a needle. Cover and study these crystals with the high-power.

The mycelium also secretes a cytolytic enzyme which dissolves the middle-lamella from between the host-cells.

Make thin sections of the young succulent portion of the host and mount in a large drop of the liquid from a bouillon-culture of *S. Libertiana*. Mount similar sections in water. Cover in a petri-dish to prevent evaporation. After an hour or so, EXAMINE:—

23. Compare the two mounts as to changes in the tissues. RECORD the results in the form of notes or sketches.

Pathogenicity studies. The pathogenicity of *S. Libertiana* and the development of the symptoms of the disease may be easily studied as follows:—

24. Inoculate at the base of stems of beans, sunflower or lettuce with an agar-block containing mycelium (from petri-dish cultures). Cover inoculum with moist cotton and wrap with strip of tinfoil.

25. Inoculate roots of carrot or turnip by placing inoculum on the surface (injured or uninjured), cover with a bit of wet cotton and place the roots in a moist-chamber.

26. If living ascospores are available, inoculate some host-plants with a suspension of these in water.

KEEP NOTES on method of inoculation, character of infection-courts, length of incubation-period and development of symptoms.

Reisolate *S. Libertiana* from the lesions appearing on inoculated plants.

REPORT

1. Drawings and report on inoculation-experiments.
2. Detail the best method of control of this disease in one of the crops it affects, explaining how the life-history of *S. Libertiana* Fckl. warrants the procedure proposed.

BOTRYTIS BLIGHT OF PEONIES

This is a common and very important disease of the peony. There appear to be in fact two Botrytis blights so similar in symptoms as to be distinguished only by a careful examination of the pathogenes involved.

SYMPTOMS

On young shoots. Study photographs and specimens provided.
OBSERVE:—

1. That when the plants first come up in the spring shoots from six to ten inches high, suddenly begin to wilt and fall to the ground. The entire shoot quickly withers and dries.

2. At the base of these wilting shoots, the brown rotten lesion usually involving the entire circumference of the stem and extending some distance above the surface of the soil. The wilting noted above is due to this basal stem-rot.

3. Covering the surface of some of these lesions the greyish brown felty covering of conidiophores of the pathogene, appressed or broken away from the dry specimens. Very evident in fresh specimens.

4. On some of the lesions in their more advanced stages, small black bodies the size of a pin-head imbedded in the cortical tissues; most usually found at the base of the stem where it was covered by the soil. These are the sclerotia of the pathogene.

SKETCH (full page size) to show the symptoms in blighted young shoots.

As the season advances shoots continue to wilt from lesions at their base. However, the proportion of such cases decreases rapidly as the stem-tissues mature and harden. Toward the middle of the season the stems attacked at the base wilt but do not fall over. The development of the lesion is much slower and the wilting is often preceded by a yellowing or a purpling of the foliage.

Study such specimens and photographs as are available showing the characters of the stem-rot in the older shoots.

On the buds. The bud-rot is the next symptom of the disease to develop after the stem-rot of the young shoots. The symptoms exhibited by affected buds are of two general types; the "bud-blast" of the small immature buds when they are from the size of a pea to half an inch in diameter and the "bud-rot" which is the name applied to the disease as it manifests itself in affected buds after they are nearly or quite mature, just before blooming.

Bud-blast. Examine the material available. OBSERVE:—

5. Black or brown undeveloped small buds in the cluster. It is usually the first or leader-bud of the shoot that is affected. Note that some of the secondary buds are larger and healthy. They grow rapidly after the death of the main-bud.

6. The brown felt of conidiophores with grey conidia. This is commonly wanting or difficult to detect with the naked eye especially in dry specimens.

SKETCH to show bud-blast.

Bud-rot. Examine one of the large buds provided. OBSERVE:—

7. The general discoloration of the floral parts. The bud is killed. It fails to open.

8. The felt of conidiophores, usually most noticeable about the base of the bud.

9. That the lesion often extends down the stem for some distance below the bud. (Study illustration specimens and autochromes.)

SKETCH to show the external characters of bud-rot.

Some of the dry rotted buds have been moistened to put them in a more characteristic condition. Carefully dissect one of these and OBSERVE:—

10. That the innermost parts of the bud are involved, the petals being very soft and decayed.

11. That the petals do not separate readily from one another being held together by the mycelium of the pathogene. By carefully tearing apart two adjoining petals the stretched and breaking hyphae may sometimes be detected.

12. Mats or wefts of mycelium which may sometimes be observed in the cavities of the bud.

The bud-rot phase of this disease is probably its most serious feature. Why?

On the blossoms. Blossom-rot. Sometimes the half-open buds are attacked. They turn brown and collapse and become a rotted mass. This occurs only during an epiphytotic of the disease in very wet weather.

Examine the dried specimens provided. They give only a partial idea of this condition as it appears in the field. Compare with healthy blossoms as shown in the autochrome. SKETCH.

On the upper stem. It has already been seen that the stem at the base of the bud may and usually does become involved. Examine specimens that show upper stem-lesions. OBSERVE:—

13. The brown discoloration and shrunken character of the diseased stem.

14. In many cases, very characteristic alternating light and dark bands or zonations especially marked on stems just below rotted buds. (See autochrome.)

15. On some of the lesions, conidiophore-felts. These are usually wanting on stem-lesions.

16. Occasionally stem-lesions showing the small sclerotia of the pathogene as small black bodies bursting through the epidermis. (Examine illustration specimens or photographs showing these.)

17. That sometimes the pathogene spreads from blasted small secondary buds or from blighted leaves into the stem, forming canker-like lesions. (Examine illustration specimens, photographs and autochromes.)

SKETCH to show different types of upper stem-lesions.

On the leaves. Leaf-lesions while they may occasionally appear early in the season become characteristic and abundant usually at the time of, or just after the blossoming-period. Examine specimens and photographs provided. OBSERVE:—

18. The location of the majority of the lesions, i.e. the part of the blade involved.

19. The average size and color characters of the lesions. Note evidences of zonation in some of them similar to those on the stem.

20. Conidiophores. Where most abundant? That they are more scattered, i.e. less felty than on shoots and buds.

SKETCH to show both surfaces of leaf-lesions.

On the root. It has recently been found that the crowns and roots may also become involved, resulting in the long-known "brown rot" of the roots. If specimens are available study, NOTING:—

21. The firm more or less dry brown decay of the tissue. The entire diameter of the root is involved. Compare with specimens of the so-called "black rot" of the roots.

ETIOLOGY

There are two pathogenes which cause Botrytis blights of peonies. The small-sclerotial species is *Botrytis paeoniae* Oudemans. The large-sclerotial species is *Botrytis (species?)*. The *Botrytis paeoniae* Oudemans appears to be the more common and destructive and is the one here considered.

Life-history. This fungus exhibits in its life-history both primary and secondary cycles with characteristic pathogenic and saprogenic phases.

The **Primary Cycles** are initiated and completed, chiefly at least, on the young shoots which become diseased shortly after they come up in the spring.

Pathogenesis. So far as is known conidia alone constitute the primary inoculum. These are produced in early spring from overwintered mycelium in the old diseased stubble or the stems on the ground. Possibly also from germinating sclerotia in the old stems.

Diseased stems have been brought in from the field and placed in moist-chambers. Examine the material and OBSERVE:—

22. The numerous tufts of brown conidiophores covered with grey conidia,—the primary inoculum.

SKETCH to show these conidial clusters on the stems.

Mount some of the conidia and OBSERVE:—

23. Their form, size and color. These conidia are carried by the wind and insects, chiefly ants, to the young shoots that are coming up nearby. Here they lodge and if conditions of moisture and temperature are favorable they germinate and penetrate the tender young tissues. Study the germinating conidia on the slides provided. DRAW.

After penetration by the germtube, infection occurs, the first evidence of injury showing in two or three days. The mycelium ramifies the tender tissues in all directions killing them presumably by toxins which it secretes.

Make thin sections of diseased stems provided and STUDY:—

24. The morphology of the mycelium and its relation to the host-cells. Is it inter- or intracellular? DRAW.

From the mycelium, conidiophores are soon thrust forth to the exterior and conidia quickly and abundantly produced. Make mounts of conidiophores from diseased stems. OBSERVE:—

25. Their form, structure and the way in which conidia are borne. DRAW.

Saprogenesis. After the shoot dies the mycelium begins to form sclerotia at the surface of the lesion, especially below the surface of the soil, where moisture conditions are more favorable.

Examine the sclerotia growing in pure cultures or on diseased shoots. COMPARE:—

26. With the sclerotia of *Sclerotinia Libertiana* as to size, color and relation to substratum. Make comparative SKETCHES.

Crush or section one of the minute sclerotia of *B. paeoniae* and OBSERVE:—

27. Its structure; the outer black rind-cells and the central white medulla. DRAW:

The diseased shoots shrivel and fall to the ground, harboring the sclerotia until next season. The mycelium frequently spreads down from the diseased shoots into the crowns and roots causing root-rot.

It appears probable that mycelium as such and not in the form of sclerotia, hibernates in the old tissues and even spreads through them as a saprophyte giving rise in the spring to conidia for the primary infections. Examine the old stems in the moist-chamber. They have just recently been brought in from the field. OBSERVE:—

28. The numerous tufts of brown conidiophores covered with the grey conidia. From what do the conidiophore-clusters arise? Dissect one of them carefully away from the stem. Do they ever appear to arise from a sclerotium?

Secondary Cycles. The conidia produced on the diseased young shoots are carried chiefly by ants to the developing buds where in the sugary exudate they quickly germinate, penetrate and cause the bud-blast.

Other secondary cycles are exhibited later in the bud-rot, blossom-rot, upper stem-rot, and leaf-blight. The fungous structures produced; their relation to, and effects upon the tissues are the same as those of the primary cycles. Sclerotia are rarely produced, however, in any of the host-organs other than the stem. The mycelium of the secondary cycles in old leaves and especially stems gives rise in the moist days of spring to great numbers of conidia which may also initiate primary cycles.

REPORT

1. Write a letter to a peony-grower in reply to the query "How shall I proceed to control the Botrytis blight in my peonies?" It is to be assumed that the correspondent knows the various symptoms of the disease and that it is caused by a fungus.

BROWN ROT OF STONE-FRUITS

This disease is especially destructive on plums, peaches and sweet cherries. Apples, pears and quinces in America are sometimes affected. In the case of certain varieties of apples, affected fruits become jet black instead of brown. (See illustration specimen.) The brown color of the affected tissues of plums and peaches has given to the disease the name of brown rot.

SYMPTOMS

The disease is usually confined to the fruit, although it may appear also on other parts of the host,—blossoms, leaves, twigs and limbs.

On the blossoms. The blossom-blight form of the disease is the first symptom to appear if the spring weather is at all favorable. Examine the specimens provided and OBSERVE:—

1. Effect of the disease on the blossoms; similar to the effect of the fire-blight on apple blossoms.

2. The grayish brown spore-tufts of the fungus on the petioles and young fruits.

DRAW to show blossom-blight symptoms.

On the fruit. Diseased fruits of plum, peach, cherry or apple have been provided. (*Do not remove preserved specimens from bottles.*) OBSERVE:—

3. The brown discoloration of the diseased area. The margin of the spot, distinct or indefinite?

4. That at first the diseased tissues do not collapse, the diseased portions retaining their original plump character. Compare this with a fruit in an advanced stage of decay. To what is the difference due?

DRAW to show fruits in early and advanced stages of the rot.

With the finger TEST:—

5. The relative firmness of the diseased and healthy portions of the fruit; strength and character of the epidermis.

Cut into the fruit and NOTE:—

6. The effect of the disease on the tissues as to discoloration, firmness, taste and odor.

The fruit is often attacked and killed before it matures and for this reason clings to the tree, instead of falling as is the case with ripe or prematurely ripened fruit. Such diseased fruits dry and wrinkle forming the so-called mummies of brown rot. In the dry material provided OBSERVE:—

7. The dry and shriveled condition of the fruit.

8. Often a cluster of the mummies clinging together, bound to each other by threads of mycelium where they touch.

9. The tufts of the fungus crowded or scattered over the surface; color?

SKETCH a mummied plum.

On the twigs. In some seasons the twig-blight form of this disease is severe, especially on plums and peaches. Examine the specimens provided and OBSERVE:—

10. The dead and shriveled brown leaves of the upper part of the twig, in appearance not unlike the effect of fire blight on apple or pear twigs.

11. The old blossom imbedded in a drop of gum at the point where the twig was girdled; or—

12. The old mummied fruit or fruit-pedicle where the twig was girdled.

DRAW to show the twig-blight form of the brown rot.

On the limbs. Brown rot cankers are very common on large and small limbs of peach trees in some peach-growing sections. They originate usually from the blighted twigs, blossom or fruit-spurs. Examine the cankered limbs provided and OBSERVE:—

13. The rough open cankers with evidences of repeated callus-formation. What does this indicate as to the age of the canker?

14. Evidences of the diseased twig or spur about which the canker developed.

15. Cankers breaking out above or below the original lesion,—evidences of metastasis.

DRAW to show the character of brown rot cankers.

ETIOLOGY

The pathogene causing the brown rot of stone-fruits is *Sclerotinia cinerea* (Bonorden) Woronin. A closely related species, *Sclerotinia fructigena* (Persoon) Schröeter, is the common cause of brown rot of apples, pears and similar fruits in Europe. These fungi belong to that group of the ascomycetous fungi known as the Pezizales which are characterized by having an open cup-shaped apothecium in which the asci are borne. In the conidial stage the pathogene has been commonly known as *Monilia cinerea* Bonorden.

Life-history. This fungus has a more complicated life-history than most pathogenes causing diseases of the more common crops. This is due to the fact that its conidia may live over winter and initiate primary cycles.

The **Primary Cycle** may be initiated from more than one source and by two sorts of inocula, as brought out in (a) and (b) below.

Pathogenesis. (a) The mummies which fall to the ground give rise in the spring to apothecia which discharge myriads of ascospores about blossoming-time. The apothecia are very short lived, appearing over a period of not more than two or three weeks. Specimens have been provided. OBSERVE:—

16. The goblet-shaped fruit-body,—the apothecium with a long slender stipe. Upon what does the length of the stipe depend?

17. The size and color of the apothecium. The upper surface (bearing asci) is when fully expanded, usually flat or even convex with recurving margins. They retain almost their natural color in alcohol.

18. The attachment of the stipe to the mummy. To which side, upper or lower, is it attached? Why?

Make a diagrammatic DRAWING showing the anatomy of the apothecium, its relation to the mummy and to the surface of the soil.

Crush a bit of one of the apothecia especially provided for this purpose and OBSERVE:—

19. The minute ovate ascospores,—the inoculum. Some of them are still in the asci in which they are formed. DRAW. They germinate by a simple germtube.

(b) The mummies which cling to the trees through the winter or the cankers on the limbs harbor the pathogene as living mycelium or as thick-walled conidia. The warm spring rains cause the formation of multitudes of thin-walled conidia which it is believed may also function as primary inoculum. Mummies have been brought into the laboratory and placed for several days in a moist-chamber.

Examine one of the mummies and NOTE:—

20. The grayish brown powdery spore-tufts. Make an enlarged DRAWING of a part of a fruit showing these cushions as they appear under the hand-lens.

With the point of a scalpel remove a bit of the spore-mass to a slide, cover and examine. OBSERVE:—

21. The rather large hyaline globose or oval bodies scattered through the mount,—the conidia. Variations in form, size, and thickness of the walls.

22. The granular, sometimes alveolate character of the protoplasm of the spore. (See also under the oil-immersion demonstration microscope.)

Make an enlarged DRAWING of a conidium to show the structure.

Slides showing germinating conidia have been prepared. Examine and OBSERVE:—

23. The long septate germtubes. Simple or branched? How many from each conidium? DRAW.

The ascospores or conidia are carried to the opening blossoms, where if the weather be rainy they quickly germinate and the germtubes penetrate the calyx or exposed corolla. The blossom-blight form of the disease is thus initiated.

Mount one of the diseased petals provided. Stain with eosin and STUDY:—

24. The mycelium in the tissue; its form, size, septation and branching. Does it go through or between the cells? DRAW.

The mycelium ramifies throughout the blossom and on protruding conidiophores forms masses of grayish brown conidia,—the inoculum for secondary cycles. From the blighted blossom the mycelium follows down through the fruit-spur to the twig or branch killing the bark and causing twig-blight or young cankers. In the cankers on the limbs the fungus may remain alive throughout the winter and provide, as indicated above, conidial inoculum for the primary cycles the next year.

The primary cycle does not include a saprogenic phase unless developed on fruits, which is probably rare.

The **Secondary Cycles** are in all probability initiated from conidia developed on the blighted blossoms or from other secondary infections on fruits and twigs. The blighted twigs, blossoms and green fruit continue to harbor and multiply the parasite during the early part of the season until the maturing fruit offers opportunity for its very general spread and destructive work.

Pathogenesis. Diseased specimens of ripe fruits showing the pathogene are provided. With forceps, remove a bit of tissue from a badly rotted fruit. Tease apart in a drop of water on a slide, cover and study under the microscope. OBSERVE:—

25. The form, structure, contents, septation branching, size and color of the mycelium. Note variation in diameter of the mycelial threads. (Staining with eosin or methylene blue will bring out the mycel-

ium.) Are the cells penetrated? If available, study the mycelium growing in agar.

DRAW to show the mycelium in the fruit and its relation to the cells.

This mycelium soon amasses at certain points beneath the skin of the fruit and bursting the skin pushes forth a cushion of short branched conidiophores. Make thin sections through these cushions,—sporodochia, and the skin of the fruit (or use prepared slides), study and OBSERVE:—

26. The cluster of conidiophores protruding through the ruptured epidermis. The cuticle of the fruit pushed back on either side.

27. The densely packed mass of mycelial cells from which the conidiophores arise,—the stroma. Note the pseudoparenchymatous structure.

28. The conidiophores; their form and mode of branching, length and diameter, color, contents and septation. (Use material from the petri-dish culture.)

29. The conidia; their mode of formation,—moniliform. It was from this character that the conidial stage first received the name *Monilia*.

Make a careful DRAWING of the section through the sporodochium to show its structure and relation to the host.

From the diseased fruits, especially if attacked when green, the mycelium may penetrate through the cortex of the fruit-pedicle to the bark of the twig and cause twig-blight. Then passing on down to the larger limbs, it spreads into the bark and initiates the large limb-cankers.

Saprogenesis. Many of the rotted or mummified fruits fall to the ground in the autumn or cling to the tree. Those on the tree may, as already seen, harbor the parasite throughout the winter. Those which fall to the ground become more or less buried in the soil and in them just beneath the cuticle in the epidermal regions is formed a crust-like black stroma. Make sections (or use those provided) through the skin of one of these fallen black mummies. Study and OBSERVE:—

30. The black thick-walled cells of the stroma. Their relation to the old dead host-tissue. DRAW.

From this stroma there arise in the spring the apothecia, the external features of which have already been studied. Examine a thin section through the apothecium and OBSERVE:—

31. The densely packed hyphae making up the greater portion of the section,—the excipulum. This is made up of much septate interwoven threads of the mycelium. It forms the bulk of the apothecium and serves as a protection and foundation for the layer of asci that lines the cup,—the hymenium.

32. The make-up of the hymenium; the long slender tube-like asci containing the ascospores. How many ascospores in each ascus? Their form and structure; the thread-like paraphyses standing up between the asci, septate or not?

The paraphyses at first form the hymenium but as the apothecium expands the asci are formed and push up between the paraphyses and become a part of the hymenium. Make out the pore at the apex of the ascus. Confirm your observations with the prepared slides.

Make DRAWINGS to illustrate fully the structure of the apothecium.

REPORT

1. Prepare a graphic illustration (cartoon) showing the life-history of *Sclerotinia cinerea* (Bon.) Wor.

BEAN ANTHRACNOSE

This is one of the most serious diseases with which the vegetable-gardener has to contend. It occurs wherever beans are grown and, under conditions favorable for the fungus, will ruin an entire crop.

SYMPTOMS

Lesions are found on all parts of the plant above the root.

On the seedlings provided, OBSERVE:—

1. The elongated, blackened lesions on the stems, with the centers shrunken and in some cases the host-tissue cracked open.

2. The lesions on the cotyledons which resemble very much those on the pods. Where do the lesions first appear, on the stem or on the cotyledon? Why?

Make a DRAWING of a diseased seedling.

On the leaves provided, OBSERVE:—

3. The browned or blackened lesion on the under side. The areas between the veins are seldom injured. On the young growing leaves the veins are often killed while the tissues between still continue to grow, so that the leaf becomes distorted. Do the lesions show on the upper side of the leaf? DRAW.

On the pods provided, OBSERVE:—

4. The distinct regularly outlined, much depressed spots on the pod; their shape and size.

5. The margin of each, bordered with a red zone, and that where two spots are near together, the red zones coalesce.

6. The blackened center and, occasionally in the larger lesions, the host-tissue so shrunken that it is cracked open. Are there any spots lacking the black centers?

Make DRAWINGS of several lesions on a pod to show the above points.

On the seeds provided, OBSERVE:—

7. The browned or blackened areas. Do they resemble in any way those on the pods? Are they depressed? Is the seed-coat ruptured at any point in the lesion? DRAW.

Compare the symptoms of this disease with those of bean blight, shown in the illustration materials.

ETIOLOGY

The pathogene causing bean anthracnose is *Colletotrichum Lindemuthianum* (Saccardo and Magnus) Briosi and Cavara. It is one of the Melanconiales, a group of the Fungi Imperfecti. Its perfect form is not known but is doubtless an ascomycete of the genus *Glomerella*.

Life-history.

The **Primary Cycle** is initiated by conidia from the lesions on the cotyledons. When diseased beans are planted they contain mycelium of the pathogene, which at the germination of the host also begins to grow and is ready to produce spores when the cotyledons push above the surface of the soil.

Pathogenesis. Scrape some material from a lesion on the cotyledon of a seedling provided. Mount and examine. OBSERVE:—

8. The numerous small hyaline conidia mixed with the debris of diseased tissues. Their shape, size and color. DRAW. These constitute the primary inoculum. They are washed or splashed by rain to the stalks and first leaves of the seedling which thus inoculated show within a few days the lesions of the primary infection. The cotyledons soon shrivel and fall carrying with them the source of primary inoculum. If dry weather prevails from the time the beans come up until the cotyledons fall, few or no primary infections occur and a clean crop is assured.

The conidia, washed from the cotyledon-lesions, germinate on the stems or leaves by a germtube. This penetrates directly into the tissues or forms a resting-cell at its tip,—an appressorium, from which later a germ-tube penetrates the cell on which it rests. Study germinating conidia provided and OBSERVE:—

9. The number and positional origin of the germtubes. Note that the identity of the conidium is nearly or quite lost in the developing mycelium.

10. The darker thick-walled appressoria at the ends of some of the germtubes. What is their function?

DRAW germinating conidia.

There are soon developed in the primary lesions on the seedling-leaves and stems, minute fruit-bodies. Examine the lesions and OBSERVE:—

11. The number and location of these fruit-bodies,—the acervuli. Color? Some of them may show (if fresh specimens) the pink mass of conidia oozing out on the surface of the lesion. DRAW to show the appearance of these acervuli in the lesion as seen under the hand-lens.

Mount and examine some of the conidia from a primary lesion to see that they are the same as those from the cotyledons. Conidia from the primary lesions are produced for a considerable period and initiate the secondary cycles.

Saprogenesis. The mycelium in the old primary lesions may continue to live and spread saprophytically into the dead tissues of the leaves and stems, on which in the spring it may produce conidia. These conidia probably seldom function in initiating primary infections. The sexual stage may develop from the saprophytic mycelium in old stems and leaves but is not certainly known for this pathogene.

Secondary Cycles develop on the later leaves and on the pods. The same phenomena of inoculation and infection are exhibited as in the primary cycle.

Pathogenesis. The lesions on pods afford the best material for a study of the structure of the fruit-bodies of the pathogene.

Study cross-sections through a lesion. OBSERVE:—

12. The saucer-shaped acervuli, at the surface of the lesion; their relation to the host-tissues, especially the epidermis.

13. The thin mat of mycelium forming the base of the acervulus, from which arise the conidiophores.

14. The size and shape of the spores; the manner in which they are attached to the conidiophores. What are the bubble-like bodies found embedded in the granular contents of each? Do the spores have septa?

15. The dark tapering septate threads which extend above the conidiophores and have somewhat the appearance of spines. These

are the setae, which distinguish the genus *Colletotrichum* from *Gloeosporium*, another anthracnose-producing group of organisms.

Make a diagrammatic DRAWING of a lesion showing the relation of the acervuli to the host-tissue. Make an enlarged DRAWING of a portion of an acervulus, showing its structure.

If fresh pods showing the oozing spores are not available examine the cultures provided and OBSERVE:—

16. The pink masses of spores embedded in a gelatinous matrix. It is in this manner that they ooze from the acervulus and are later washed or splashed by the rain to initiate new infections. They are formed so fast by successive constrictions of the conidiophores that many millions may be formed on one badly diseased pod.

If conditions are favorable, the mycelium in the lesions on the pods grows through the tissue and into the seed below it. It is in this manner that the bean seed becomes infected with the fungus-hyphae which produce spores on the growing cotyledons the following season.

Cut through a bean-pod directly over a lesion and OBSERVE:—

17. The depth of the diseased area and the relation which the pod-lesion bears to the discolored area on the seed. Make a DRAWING showing this relation.

In this manner the pathogene usually completes the cycle without going through a saprogenic period of existence.

Saprogenesis. The mycelium in the pods and stalks develops saprophytically after they are harvested or fall to the ground. Here are produced conidia whenever sufficient moisture and favorable temperature allows. If such debris is scattered with manure on land planted to beans, conidia produced thereon might cause primary infections in the spring. Observation indicates that this rarely occurs and that diseased seed is the usual source of the primary inoculum.

Pathological Histology. Study cross-sections through lesions on cotyledons, stems or pods. (If freehand, stain with methyl blue or eosin. Wash thoroughly.) OBSERVE:—

18. The broken-down mass of cells causing a deep depression in the tissues, leaving a blackened layer of disintegrating cell-walls at the base of the depression.

19. The numerous mycelial threads which are both inter- and intracellular, sometimes almost completely filling the cell; the individual hyphae which are septate and much branched, and the granular contents with numerous bubble-like bodies scattered throughout the threads. Are these bodies vacuoles or nuclei?

20. The effect on the cell-contents, i.e. protoplast, starch or chloroplasts.

Make a DRAWING showing points brought out above.

REPORT

1. Outline a method of eradication by which clean crops of a susceptible variety of beans may be eventually obtained, starting with diseased seed; (a) for a northern grower; (b) for a grower in the Gulf states or the southwest.

ANTHRACNOSE OF SYCAMORES AND OAKS

The anethracnose of sycamores and oaks is a serious disease, especially in the northeastern part of the United States where it is frequently epiphytotic. It is enphytotic on most of the species of the genus *Platanus*. In Europe as well as the United States the sycamores are more commonly and destructively attacked than are the oaks. However, in certain regions some species of oak suffer severely.

SYMPTOMS

The leaves, twigs and small branches are affected. In addition to the characteristic lesions produced on these parts of the tree, there are certain general symptoms by which a diseased individual may be detected at a distance.

General symptoms. In the photographs provided, OBSERVE:—

1. The dead, leafless twigs.
2. The irregularity of the direction taken by smaller branches. These twisted and deformed branches are characteristic of this disease.
3. The excessive twigging on the smaller limbs, producing clumps of branches,—witches'-brooms. Note that many of the branches of the broom are dead. Make a DRAWING of the broom-like growth of the branches.

In the case of trees badly affected, the foliage becomes thin due to the falling of many leaves, and those remaining appear scorched owing to the large dead areas in them. In the early spring when the leaves are developing, all the leaves on a twig may wither and die due to the death of the twigs. If this type of injury is general it may be confused with frost-injury.

On the leaves of sycamore. In the material provided, NOTE:—

4. The large brown areas in the leaves,—the lesions.
5. That usually the lesion is located on one of the larger veins of the leaf.

The lesions, when first noticeable, are narrow dead areas paralleling the veins on both sides. This dead area then rapidly becomes larger as the tissues of the vein are killed and the portion of the lamina dependent on the vein for water dies and turns brown. Why do most of the lesions extend to the margin of the leaf?

6. The lesions on the petioles and the pimple-like fruit-bodies of the pathogene.

DRAW sycamore leaves showing these symptoms.

On the leaves of oak. In the material provided, OBSERVE:—

7. That in some cases the lesions are similar to those on sycamore leaves in that the dead areas are located along the veins; but that more commonly numerous small lesions occur without any relation to the position of the veins. How can the difference in size between the lesions not located on the veins and those located on the main veins be accounted for? Make a DRAWING of an oak leaf showing both types of lesions.

8. On the under surface of the lesions, minute brown spots,—the fruiting structures of the pathogene. DRAW as seen with the hand-lens or low-power of the microscope.

On twigs. Twigs from oak or sycamore, collected in the early spring, are provided. OBSERVE:—

9. That the twigs are (or were) still alive and the buds formed the previous autumn have reached maturity.

10. The fruiting stage of the pathogene formed under the cork-layer, raising it up in pimple-like pustules. Later the cork-layer is ruptured and the fruit-bodies are exposed.

11. The terminal portion of the twig is partially or entirely invaded by the pathogene, as indicated by the distribution of the fruit-bodies noted above.

Twigs thus affected may put out leaves in the spring which, however, usually wither and die before they reach full size. This is due to the rapid advance of the pathogene in the twigs, killing them.

On the branches of sycamores. In the affected branches of sycamore NOTE:—

12. The small diseased areas around the bases of the twigs,—cankers.

13. Larger cankers with scars of old twigs at the center, showing the origin of the canker.

14. The cortex of the bark raised up and broken through by the fruit-bodies of the pathogene. In some cases these fruit-bodies are gone and only a cavity is left in the bark.

15. Older cankers in which the dead bark has fallen away and the wood is left bare.

16. That callusing takes place rapidly in some cases and the open wound is covered.

17. That swellings of irregular shapes are often formed at points where witches'-brooms originate.

ETIOLOGY

The anthracnose of sycamores and oaks is caused by the ascomycetous fungus, *Gnomonia veneta* (Saccardo and Spegazzini) Klebahn. Previous to the discovery of the ascigerous stage of the pathogene, it was known by several names because of the variable types of asexual fruit-bodies which the fungus forms. The commonest of the names for the imperfect forms is *Gloeosporium nervisequum* (Fuckel) Saccardo, this being the name for the acervulus-stage which occurs on the lesions on the leaf-blades and petioles. On dead leaves, a pycnidial stage is formed,—the *Sporonema* stage and on the twigs, a pseudopycnidial form,—the *Myxosporium* stage.

Life-history. The polymorphic character of the asexual fruit-bodies found on the different parts of the hosts which are attacked, makes the life-history of this pathogene complicated. Their identity was established by Klebahn in 1905. Klebahn also connected the polymorphic conidial stage with the ascospore stage which is developed on overwintered leaves on the ground.

The **Primary Cycle** may be initiated by several sorts of inocula. No definite investigations have been made to determine the relative effectiveness of these in nature. The possible primary inocula may be, ascospores from overwintered leaves, conidia from the *Myxosporium* stage on twigs and cankers, and conidia from the *Sporonema* stage on overwintered

leaves. It seems most probable that the primary inocula in the spring are the conidia from the twigs and cankers or the ascospores in the overwintered leaves.

Pathogenesis. The fungus passes the winter in the living twigs. In autumn, fruit-bodies begin their development on the affected twigs and reach maturity early in the spring. In prepared sections of developing fruit-bodies on oak twigs, NOTE:—

18. That, while in the lower half of the fruit-body an opening has formed, the upper half is made up of mycelium and is still covered by the cork-layer of the bark.

Before early spring the mycelium in the upper part of the fruit-body disappears and the cork-layer is ruptured so that the conidia may escape. Examine with the hand-lens the twigs with the mature Myxosporium stage. Make freehand sections at right angles to the twig. Stain with eosin, mount and OBSERVE:—

19. That the top of the fruit-body is open. Has it a definite ostiolum?

20. The abundance of conidia.

21. The size, shape and color of the conidia.

These conidia are produced in great abundance and ooze out of the fruit-bodies in creamy drops or strings. In the presence of abundant moisture they may be washed or splashed to the young unfolding leaves. It seems that the conidia thus disseminated in early spring from the fruit-bodies in the twigs are the most efficient of the various possible primary inocula. The other types of primary inocula undoubtedly play some rôle and must be considered. In the overwintered leaves, specimens of which are provided, NOTE:—

22. The small pimple-like bodies on the surface.

Remove one of these fruit-bodies to a slide, mount and crush. OBSERVE:—

23. The abundance of conidia coming from the pustules.

24. The remains of the walls which enclosed the spores.

25. The size, shape and color of the conidia. Compare with the conidia obtained from fruit-bodies on the twigs.

Make DRAWINGS of this stage.

The pycnidia-like fruit-bodies thus formed on overwintering leaves come about by growth of the vegetative hyphae around the margin of the acervulus, arching-up over the conidia until they are enclosed. This is the Sporonema stage and is formed on leaf-petioles as well as on the blade. Study the Sporonema stage on old petiole-lesions if material is available. DRAW.

The conidia in the Sporonema pustules on dead leaves remain viable through the winter and may cause primary infection in the spring if they reach susceptible parts of the hosts.

In the same overwintered leaves showing the Sporonema stage, search for the perithecia. NOTE:—

26. Small hair-like spines protruding from ruptured places in the leaf-epidermis.

27. On the opposite side of the leaf, that a distinct bulge in the surface is evident.

Cut out small bits of the leaves containing perithecia. Mount in water and tease away the leaf-tissues. NOTE:—

28. The shape of the perithecium with its beak. What is the function of the beak? DRAW.

29. Upon crushing the perithecium, the contents.

30. Shape and size of asci; thickness of their walls.

31. Shape and size of ascospores; number in each ascus; septate or nonseptate?

Make DRAWINGS of the perithecium, asci and ascospores. (If material is not sufficient see Bot. Gaz. 45: 379 and pl. XI.)

The ascospores are probably forcibly ejected from the perithecia, as most ascospores are, and thus they may readily be borne to the unfolding leaves and produce primary infection in the spring. That primary infection is commonly initiated by ascospores is evidenced by the fact that leaves on the lower branches are usually the first to show the disease in the spring.

From whatever source primary infection may result, a new mycelium is started near the veins of the young leaves. As the lesions enlarge the acervulus-type of fruit-body is formed. In the prepared sections of acervuli, OBSERVE:—

32. The aggregation of hyphæ radiating from the base of the fruit-body.

33. The chains of conidia cut-off from the ends of the conidiophores. These form so abundantly that they pile up into balls or strings and are held together while dry by a gelatinous matrix.

34. The individual conidia, as to shape and size. Compare with conidia from *Myxosporium* and *Sporonema* stages.

Make DRAWINGS to show the structure of an acervulus in cross-section, including a portion of the adjacent leaf-tissue and the vegetative hyphæ.

Saprogenesis. When the leaves fall to the ground the mycelium spreads rapidly to all parts of the leaf and lives saprophytically forming more acervuli and, in moist situations, the pycnidia of the *Sporonema* stage. The perithecia begin their development after the mycelium has existed saprophytically for a time. The ascospores are mature by early spring.

Secondary Cycles originate from the conidia developed in acervuli on the leaf- and petiole-lesions. It is suggested by some that the mycelium, extending down from the diseased petioles into the twigs, plays an important part in the infection of the current year's twigs. This has not been proved, but it happens either in this way or by infection during the summer resulting from conidia. The infected twigs are not killed but develop normal buds which put out leaves the following spring. However, the mycelium in the twigs gains enough headway during the autumn and early spring to cause the death of the twigs at a time when the leaves are only partially developed.

REPORT

1. From what is known of the life-history of *Gnomonia veneta* (Sacc. and Speg.) Kleb., work out possible control-measures and give reasons for each step, based on the life-history of the pathogene.

BLACK ROT OF GRAPES

The black rot is an American disease, known as a destructive malady in our vineyards for nearly a century before its appearance in Europe in 1885. It is markedly epiphytotic in its occurrence on cultivated grapes.

SYMPTOMS

All green parts of the vine, including the young canes, may exhibit lesions of the black rot. Old woody parts are never affected.

On the leaves. Spots on the leaves are reddish brown and more or less circular. The spot at first appears as a small blanched area. The spread is concentric but not perfectly circular. Study the material before you and OBSERVE:—

1. The size and distribution of the lesions over the surface of the leaf. Note the color.

2. The shape of single spots and their relation to the finer veinlets. Study the margin closely; even or crenulate?

3. That certain spots show minute black bodies,—structures of the pathogene. Are they superficial or sunken in the leaf-tissue? What is their arrangement on the lesion? On which surface are they found?

Make such SKETCHES as are necessary to bring out the character of the spots on the leaves available. (See photograph 1.)

On the berries. The first signs of the disease on the berries are not readily detected except by careful observation. The lesions rapidly become apparent, however. Study the diseased berries provided. OBSERVE:—

4. The different stages in the development of the lesion from a small blanched or brown discoloration to the black and shriveled mummy. (See photographs 2 and 3.)

5. The characteristic black fruit-bodies in the lesion; position and arrangement. At what stage in the development of the lesion do they appear? (See photographs 3 and 4.)

DRAW to show the development of the symptoms on the berry.

On the canes and tendrils. In the material provided, OBSERVE:—

6. The character of the lesions. Compare and contrast with those on the leaves; with those on the fruit.

7. The extent of the lesion on the cane; on the tendril. Are the lesions limited and definite? Is there a tendency to girdle?

8. The number of lesions; their form, size, and the presence of the black fruit-bodies of the pathogene.

Study photograph 5, noting lesions on canes, petioles, midribs and tendrils.

Make DRAWINGS to illustrate these studies.

ETIOLOGY

The pyrenomycetous fungus, *Guignardia Bidwellii* (Ellis) Viala and Ravaz, is the cause of the black rot of grapes. Its conidial form has been given the genus name *Phyllosticta*, when occurring on the leaves but when on the canes, *Phoma*. A variety of specific names have been applied to it. (See Cornell Bul. 293:307.)

Life-history. This pathogene exhibits in its life-history all the typical spore-forms and pathological phenomena of a large number of the leaf-spotting pyrenomycetes.

Primary Cycles are initiated in late spring and throughout the summer (Cornell Bul. 293: 310). The fungus winters largely in the mummies which lie on the ground or cling to the vines. (Note in the latter connection, photograph 6.) Canes and tendrils may also harbor it.

Pathogenesis. Some of the mummies are provided for study. With a hand-lens, OBSERVE:—

9. The presence of very numerous elevations,—the perithecia of *Guignardia Bidwellii*; the ostiolum at the apex.

With the hand-lens and needle remove one or more of these perithecia (from the soaked mummy provided) to a drop of water on a slide; cover and examine with the lower-power, exerting slight pressure on the cover-glass. NOTE:—

10. That if the perithecia are mature, the asci with ascospores will be forced out.

11. The average number of asci in the perithecia; the number of spores in an ascus.

DRAW an ascus with ascospores. These ascospores constitute in most seasons the chief inoculum for primary cycles. Discharged from the perithecia during rainy weather, they are blown by the breeze or splashed by rain-drops to young leaves or berries where they germinate and the germ-tubes penetrate the host-tissues. COPY Cornell Bul. 293, pl. V, fig. 28-29. Note the appressorium formed on the end of the germ-tube.

Pycnospores overwintered in canes and tendrils may also serve to initiate primary cycles. (See Cornell Bul. 293: 326.) With hand-lens and needle, remove a few pycnidia from one of the cane-lesions. Crush on a slide in water or potassium hydroxide and study. MAKE OUT:—

12. The numerous pycnospores set free from the broken pycnidia. Their form, size and color as compared with the ascospores. DRAW. These germinate by a simple germ-tube and infect young leaves and fruits. COPY Cornell Bul. 293, pl. V, fig. 34.

After penetration by the germ-tube of the ascospore or pycnospore, mycelium is slowly developed in the surrounding tissue. The first evidences of infection appear in one to three weeks, depending on conditions of the host and the weather. After the appearance of the lesions, the pycnidia develop (see photographs 1, 3, 4, 7 and 8) and form pycnospores with great rapidity. Study prepared slides provided and OBSERVE:—

13. Position of the pycnidium in relation to the host-tissues.

14. The pseudoparenchymatous wall of the fruit-body.

15. The short conidiophores bearing the pycnospores; their origin and arrangement in the pycnidium.

16. The ostium through which the pycnospores are discharged. They are discharged by the pressure resulting from water-absorption by the gelatinous substance in which they are surrounded. These initiate the secondary cycles.

Make a DRAWING to show the structure of the pycnidium and its relation to the host-tissues.

Saprogenesis. The fungus lives saprophytically after the lesion is fully developed until the following spring. From July until October certain fruit-bodies, known as spermagonia, are developed. They are

abundant in August and may develop in the same stroma with pycnidia. They bear spores called spermatia. (See Cornell Bul. 293, pl. III, fig. 19.) Their function is unknown but it is believed by some that they are vestigial male elements. Whether perithecia are produced in the old primary lesions is not certain. Pycnosporos produced in these primary lesions may, however, winter over and new ones may possibly be formed, thus completing the primary cycle. (See Cornell Bul. 293: 325-326.)

The **Secondary Cycles**, especially those initiated in the late summer differ from the primary chiefly in that they certainly give rise to the ascogenous structures which develop into perithecia the next spring.

Pathogenesis. Until the first of August, pycnosporos are produced in enormous numbers on the mummied berries. They are discharged with rain or dew and initiate secondary cycles. Other cycles follow in succession as long as pycnosporos are developed. After about August 1, pycnidial production ceases and instead there are developed in the lesions pycnidium-like bodies called pycnosclerotia.

Study slides provided or Cornell Bul. 293, pl. IV. OBSERVE:—

17. The absence of a central cavity and of spores.

18. The parenchymatous character of not only the walls but of the entire fruit-body.

19. Differences in the character of the central portion as contrasted with the wall-structure.

READ Cornell Bul. 293: 329. DRAW to show the structure of the pycnosclerotium.

Saprogenesis. With the death and shriveling of the tissues in the berry, the fungus continues its activities as a saprophyte. The development of the incipient perithecia (pycnosclerotia) is halted by winter but resumed in late spring. In the thin-walled parenchymatous-like center of the pycnosclerotium, asci are developed and matured. Study sections through mature perithecia and OBSERVE:—

20. The thick dark walls of the cells making up the wall of the perithecium; the mouth or ostiolum.

21. The rather thick-walled asci; their shape, size and position in the perithecial cavity.

22. The ascospores; number and arrangement in the ascus.

DRAW to show the structure of the perithecium. The maturation and discharge of the ascospores completes the secondary cycle.

Pathological Histology. The effect of *Guignardia Bidwellii* on the tissues of its host is always necrotic. Study sections through leaf-, stem- or berry-lesions. OBSERVE:—

23. The contrast between healthy and affected tissues; changes in color, and appearance of cell-walls and protoplasts in the diseased tissues.

24. Which tissues in each organ appear to be most affected. Is there any evidence of an attempt by the host to stay the progress of the pathogene by the formation of cork-tissue about the lesion?

25. The presence of mycelium; inter- or intracellular? Are haustoria present?

DRAW to show the points brought out in the study of the histological effects.

REPORT

1. Discuss the relation of weather-conditions to the disease, keeping remedial measures in mind.

HARD ROT OF GLADIOLI

This is one of the destructive diseases of gladioli. No varieties are known to be immune to it. The disease appears to occur wherever gladioli are grown. So far as known it affects only this host.

SYMPTOMS

Both foliage and corms may be affected. Although it rarely affects the foliage of flowering-sized plants, the disease occurs very commonly on leaves of plants the first year from seed and cormels.

On the leaves. Examine the material provided. OBSERVE:—

1. That the affected leaves show more or less circular reddish brown spots.
2. The several zones of color in the lesions.
3. That the spots usually extend from the edge to the midrib, although sometimes the lesion involves the entire width of the leaf. When this is the case, that part of the leaf above the lesion soon dies.
4. That frequently two or more spots coalesce forming a large, more or less irregular lesion.
5. That the spots show black bodies,—the fruit-bodies (pycnidia) of the fungus.

DRAW an affected leaf showing the several points above indicated.

On the corms. When gladiolus corms are dug in the autumn the lesions are usually very small. The disease develops during storage until by spring many corms are reduced to hard mummies.

Study the diseased corms provided. OBSERVE:—

6. Size, shape and position of the lesions. Is it possible to determine whether or not a corm is diseased without removing the husk (sheathing leaf-base)?
7. That the center of the lesion is sunken; to what due?
8. The black water-soaked margin separating diseased from healthy tissue. When the disease is not vigorously advancing, this margin is absent.
9. That in old lesions the diseased tissue can be readily chipped out with the finger-nail. Cut through a lesion and OBSERVE:—
10. The hardness of the diseased tissue.
11. The depth of the lesion.

Make such DRAWINGS as are necessary to bring out the above points.

ETIOLOGY

The cause of this disease is a fungous pathogene, *Septoria Gladioli* Passerini.

Life-history. The complete life-history of this organism has not been definitely worked out.

Primary Cycles. Just how these are initiated is not clear as the following statements indicate. The corms are lifted in the autumn, stored and set out the following spring. The fungus continues as an active parasite during the winter and is carried to the soil along with the corm. If the corm is not too badly affected it will sprout, sending up a shoot and produce a corm above with side clusters of cormels. Since in the large majority

of cases the corms and cormels from a diseased corm are diseased, the fungus is thus perpetuated without the intervention of a spore-stage. Just how the fungus passes from the old corm to the new one is unknown. It does not grow directly from the one to the other but either works along the sheathing leaf-bases or grows out into the soil from whence it attacks the new corm or cormels.

The fungus is known to winter in the soil, hence the possibility that soil containing mycelium of the pathogene may be splashed by rain onto the foliage. The foliage is often beaten down onto the soil where infection may occur. The latter is much more likely to occur with the tops of seedlings or cormels than of older plants. Why?

Healthy corms set into infested soil are doubtless commonly infected by the pathogene there.

Pycnospores have been found in the spring in pycnidia on leaves which have remained out of doors all winter, but only in very limited numbers. These pycnospores will germinate and may constitute the primary inoculum for the foliage of seedlings or cormels; by the washing of these spores into the soil they may infect the corms themselves.

Pathogenesis. Of the three probable sources of primary inoculum, diseased corms, infested soil or pycnidia in overwintered foliage, the last is most readily studied. Examine some of the leaves gathered in the autumn. OBSERVE:—

12. The old lesions on the leaves.

13. The presence of numerous, black elevations,—the pycnidia.

Is there an ostiolum? DRAW, showing pycnidia on the leaf.

With the needle remove several pycnidia to a drop of water on a slide, cover and study under the low-power. Pressing lightly on the cover with a needle, OBSERVE:—

14. The many pycnospores; size, shape and number of septa.

The mycelium in the diseased tissue of the corm is not easily detected. Make thin sections from the material provided. Stain with methylene blue in weak acetic acid for one hour. Wash thoroughly and study. OBSERVE:

15. The large cells still partly packed with starch-grains.

16. Between them the small blue-stained mycelial threads, varying in thickness with the space at their disposal.

DRAW a few host-cells with the intercellular mycelium. No fruit-bodies or spores are known to be produced on diseased corms.

On infected leaves, pycnidia appear in the lesions simultaneously with, or soon after the lesions become evident. Study the pycnidia in section, using prepared slides. OBSERVE:—

17. The outer wall of pseudoparenchymatous tissue.

18. The inner layer of thinner-walled cells from which arise the conidiophores.

19. The conidiophores bearing pycnospores.

20. The mycelium in the tissue of the host.

DRAW, showing the structure of a pycnidium seen in longisection.

The pycnospores from the primary lesions on the leaves initiate secondary cycles.

Saprogenesis. The fungus is able to live in the soil without the presence of the living host for at least four years. Whether it feeds on organic matter and develops in the soil or merely remains alive in a dormant condition until corms of gladioli are again available for attack, is not

known. It grows slowly in culture on agar making a very characteristic growth. Examine the plate-cultures provided. OBSERVE:—

21. The form and color of the colonies. The fungus is readily obtained in culture by planting in agar bits of diseased tissue from within the corm.

Mount some of the mycelium from the edge of the culture. Study and DRAW to show its form and structure. This may possibly be the form in which it persists in the soil.

Secondary cycles. Pycnospores from the primary lesions, blown during the summer, fall upon healthy leaves where, under proper conditions of temperature and moisture, they germinate and produce secondary infection. From the large percentage of diseased corms formed by plants growing from seed and cormels, the foliage of which was attacked by *Septoria Gladioli* Passer., it appears that secondary infections also occur from spores washed by rain down into the soil, where they germinate and infect the corm.

So far as known the secondary cycles duplicate in details the primary cycle.

Pathological Histology. The lesions of this disease are strictly necrotic. The pathogene promptly kills the protoplasts of the invaded tissues.

Study prepared sections through a lesion on the leaf. OBSERVE:—

22. The collapsed and shriveled cells in the diseased portion. Compare with the cells in the healthy region. What tissues are affected?

23. The effects on the cell-organs, i. e. nucleus and chloroplasts.

24. The mycelium of the pathogene; its relation to the host-cells.

25. Position and relation of the pycnidia to the host-tissues.

DRAW to show conditions and structures of the fungus in the diseased leaf.

Study sections through a lesion in a corm. OBSERVE:—

26. The absence or the small amount of starch in the diseased as compared with that in the healthy tissue.

27. That many cells in the diseased area are collapsed; due to what?

28. The layer of cork-cambium laid down between healthy and diseased tissue. May this be associated with the fact that the diseased tissue can be chipped out with the finger-nail?

29. The presence of intercellular mycelium.

DRAW to bring out these points, showing both healthy and diseased tissue.

REPORT

1. A grower plants annually over one hundred acres of gladioli. About fifty per cent of his corms are diseased. Outline a scheme, bringing in the principles of eradication and protection, whereby he can gradually work from diseased to healthy stock, probably requiring ten years or longer. Indicate the difficulties to be encountered in carrying out such a scheme.

STRAWBERRY LEAF-SPOT

There are at least two leaf-spot diseases of strawberries, but the one to which this name is applied is by far the most prevalent and injurious.

SYMPTOMS

The lesions on the leaves are most numerous and conspicuous, but the petioles and fruit-pedicles are often affected. In the material provided, OBSERVE:—

1. The spots on the upper side of the leaf, ranging in size from mere pink flecks to areas almost one centimeter in diameter. The large spots always have white centers and pink or brick-red margins.

2. That some of the spots have coalesced to form larger lesions which may cover a large part of the leaf-area.

3. The character of the under surface of the spots as compared with the upper surface. To what extent are the veins affected?

4. The elongated spots on the leaf-petioles and on the fruit-pedicles; the white centers of the lesions, which are very conspicuous. Are they surrounded by red margins as in the leaf?

5. The small and dried berries on the diseased pedicles. One or two berries may develop normally before the food supply is cut off, after which the others shrivel and are worthless.

Make DRAWINGS to show the symptoms on the different parts of the host.

Compare these symptoms with those of the *Marssonia* leaf-spot on strawberry.

ETIOLOGY

The pathogene causing this disease is an ascomycetous fungus, *Mycosphaerella Fragariae* (Tulasne) Lindau, belonging to the family Mycosphaerellaceae of the order Sphaeriales.

Life-history.

The **Primary Cycle** is initiated by the ascospores of the sexual stage found in the dead leaves on the ground. These ascospores, while still in the fruit-body, germinate sending out a long germtube which pushes out through the mouth of the perithecium and produces a conidium at the tip. (See Cornell Bul. 15: 178-180.) Make a diagrammatic DRAWING to show production of primary conidia.

Pathogenesis. The conidium is blown to a young leaf where it produces a germtube that directly penetrates the upper epidermis and invades the tissues beneath.

Cut out a diseased spot with the scalpel, and include a margin of the healthy tissue. Place the small piece in the casserole with a dropperfull of potassium hydroxide solution, and boil over a microburner for half a minute. Rinse two or three times with water, then mount and OBSERVE:—

6. The browned or blackened appearance of the diseased area in contrast to that of the healthy tissue immediately adjoining it.

7. The mycelium ramifying all the diseased tissue; its color, width, septation and branching. Does it extend into the tissues which still contain chlorophyl?

Make a DRAWING of the tissue as seen in this mount, showing the mycelium and the extent of its penetration.

The hyphae invade the cell, using up the contents, and permit the air to enter the tissue, thus giving the white appearance to the center of the lesion. These hyphal threads grow upward and produce a stroma just below the cuticle; the cuticle is then ruptured permitting the fruit-stalks to emerge.

With the aid of pith, cut thin sections through the diseased area of the leaf. Mount these in water (or use prepared slides) and OBSERVE:—

8. The location of the mycelium and its relation to the diseased cells.

9. The mat of mycelium or stroma just below the cuticle.

10. The three or more short stalks or conidiophores which arise from each stroma. Are they septate? A single conidium is borne on the tip of each conidiophore. The conidia will probably not be present in this mount. Why?

Make a DRAWING of a cross-section of a leaf showing the location and structure of the stomata with their conidiophores.

For study of the conidia, scrape several of the spots on the leaf with the scalpel and make mounts in potassium hydroxide. (If spores are not present, consult text books of Duggar, Stevens, or Tubeuf and Smith.) OBSERVE:—

11. Their size, shape and septation. DRAW.

Saprogenesis. The infected leaves which drop to the ground are still farther invaded by the mycelium of the pathogene. This mycelium finally develops a sexual fruit-body or perithecium. This is at first covered by the tissue of the leaf, but soon breaks through the epidermis.

In the leaves provided OBSERVE:—

12. Small black bodies just large enough to be seen with the naked eye, near or in the old lesions. They are never numerous. Make a DRAWING of part of a leaf showing these small black perithecia. Compare their size and appearance with the acervuli of *Marssonina* found on old fallen leaves of strawberry. (The two fungi are not related.) How can the perithecium be distinguished from the acervulus?

With the aid of pith, cut thin cross-sections of a leaf containing perithecia (or use prepared slides). Under the microscope, OBSERVE:—

13. That the perithecia are more or less hemispherical.

14. That the wall of the perithecium seems to be made of two layers, the outer one being made up of thick-walled pseudoparenchymatous cells, and the inner one of thin-walled cells.

15. That there is an opening at the tip of each perithecium,—the ostiolum.

16. That arising from the inner base of the perithecium are club-shaped bodies,—the asci containing eight hyaline, uniseptate spores with acute ends.

Make a DRAWING of a cross-section of the perithecium, with the surrounding host-tissue. Make an enlarged DRAWING of a single ascus with its spores.

It is from these spores that the primary cycles start, the following spring. The mycelium may also live during the winter in the partially green leaves and produce conidia the following spring, thus initiating primary cycles.

This is probably quite as often the means of wintering over as that afforded by the perithecial stage.

The **Secondary Cycles** originate from the conidia on the young diseased leaves. These conidia infect the petioles, pedicels and other leaves. The pathogene completes its development in the secondary cycles in the same manner as in the primary.

REPORT

1. Illustrate in cartoon-like drawings the life-history of *Mycosphaerella Fragariae* (Tul.) Lindau.

PSEUDOPEZIZA LEAF-SPOT OF ALFALFA AND CLOVER

This is an enphytotic disease of alfalfa and clover in most regions where these crops are grown. It annually reduces the yield, especially of alfalfa but rarely, if ever, kills out the plants.

SYMPTOMS

The lesions of this disease are confined to the leaves and stems. They are much less common and of little importance on the latter.

On the leaves. Study the specimens provided and OBSERVE:—

1. The spots on the alfalfa leaf. Do they show on both surfaces? Note their size, shape, color and position on the leaf. Compare with those on the clover leaf.

2. That these are usually surrounded by a yellowish zone. Note that this is more pronounced between several adjacent spots.

3. The darker, raised center of the spot made up of the fruit-body of the pathogene. Is this visible on both sides of the lesion?

Make DRAWINGS to show the character of the lesions on both clover and alfalfa leaves.

With the hand-lens or with the low-power of the microscope examine several spots which show the fruit-bodies of the pathogene. NOTE:—

4. The radially split crater-like openings and the waxy-appearing surface within. These fruit-bodies at the center of the spots serve to definitely distinguish the *Pseudopeziza* spot from other leaf-spots common on alfalfa and clover.

DRAW to show the appearance of the fruit-body under hand-lens or low-power.

5. A badly diseased leaf in comparison with one which has but few spots. Is there any difference in the general color? Does a single spot ever involve the entire leaf; if not, how is the leaf killed?

6. The passepartout provided and determine where on the plant the most seriously spotted leaves occur, and what are the ultimate results. Compare photographs 1 and 2.

Make SKETCHES to show the general effects on the alfalfa and the clover.

ETIOLOGY

The cause of this leaf-spot is a discomycetous fungus, *Pseudopeziza Medicaginis* (Libert) Saccardo. The pathogene on the clover is regarded by some as a distinct species and goes under the name, *Pseudopeziza Trifolii* (Bivona-Bernardi) Fuckel. They are probably identical.

Life-history. But one kind of spores, the ascospores, are certainly known to be produced by this fungus. As repeated crops of these are produced throughout the season, the only distinction between primary and secondary cycles is that the former are those first initiated in the spring.

Primary Cycle.

Pathogenesis. The sources of the primary inoculum are the apothecia produced in early spring on overwintered living leaves. These leaves become infected late in the autumn but complete development, including apothecial formation, is halted until spring when with the arrival

of warm weather, the apothecia are developed on these overwintered lesions. It has been held by some that from the lesions on fallen leaves which have laid on the ground over winter, apothecia are produced which supply the primary inoculum.

Crush a mature apothecium (one that is open) from leaves provided, tease well apart, cover and **STUDY**:—

7. The mature ascospores in the ascus; their shape, structure, color, and number in the ascus. **DRAW**.

These spores constitute the primary inoculum. If germinating ascospores are available, study and **SKETCH**.

The germtube penetrates the upper epidermis of the leaf, and produces a locally spreading mycelium with a resulting spot or lesion. At the center of this is shortly developed an apothecium without any intermediate production of asexual spores as is usually the case among the ascomycetes.

Make freehand sections of the leaves through the spots, stain with methyl blue, wash, and examine with the high-power of your microscope. **OBSERVE**:—

8. The brown host-cells; the character of their contents; the relation of the mycelium to these cells, inter- or intracellular?

9. How far the mycelium extends. What causes the yellowing around the brown cells?

10. The blue-stained structure on one side of the brown-spot,—the apothecium. On which surface does it occur? How deep into the leaf does it extend? How does it become exposed?

11. That the lower part of the apothecium,—the stroma, is composed of small cells.

12. That arising from the stroma is the hymenium. Of what is it composed? (It may be easier to observe the structure of the hymenium by crushing a few of the older spots in a drop of water.)

13. Within the asci, the oval ascospores; how many? Note the oil-drops or guttulae at each end. These spores are shot from the asci during moist conditions, thus disseminating the pathogene.

Make **DRAWINGS**, (a) to show the relation of the mycelium to the host-cells, its extent and the apothecium; (b) an enlarged view of the hymenium to show its structure, the spores and their arrangement in the ascus.

Saprogenesis. There is some evidence that apothecia may be formed on spots which were immature when the leaves fell to the ground. This, together with the fact that apothecia may be produced in artificial culture media, indicates that apothecial development may in some cases result in nature from saprophytic activity. Since, however, mature ascospores in apothecia on living leaves are always common and abundant, saprogenesis, as such, is an unimportant if not uncommon phase of the life-cycle.

Secondary cycles. These are initiated by ascospores from the apothecia produced during the primary cycle, and the pathogene repeats in all details the structures and activities of the primary cycle.

REPORT

1. Explain the effects of this fungus on the leaf and on the yield.
2. Show graphically the life-history of *Pseudopeziza Medicaginis* (Lib.) Sacc.

CERCOSPORA LEAF-SPOT OF BEETS

Although this disease is found more often on sugar-beets, it also affects the common garden-beet. The disease is typical of a great many *Cercospora* leaf-spots on different hosts.

SYMPTOMS

Lesions are usually present only on the leaves, though they have been reported on the petioles, the bracts, peduncles of the flowers and on the seed-pods.

In the material provided, OBSERVE:—

1. The variously sized spots with brown or grayish centers surrounded by a zone of red or purple (brown in dried specimens); their shape and distribution over the leaf; differences in the character of the spot on the upper and the lower surface. Make DRAWINGS to show these points.

As the diseased leaves drop off, new ones are formed, thus producing an elongated crown on the root. (See photographs or Duggar, *Fungous Diseases of Plants*, p. 310.)

ETIOLOGY

The pathogene responsible for this disease is *Cercospora Beticola* Saccardo, one of the Hyphomycetes. Only the asexual form of the fungus is known.

Life-history.

The **Primary Cycle** is initiated by conidia developed on overwintered tops and crowns left in the field. They are blown or spattered by the rain to the first young leaves.

Pathogenesis. Scrape several of the spots with a scalpel and make a mount in water of the material thus procured. Under the microscope, OBSERVE:—

2. The thin, long, needle-like spores, which are slightly thickened at one end and tapering at the other. How many septa in each? DRAW.

These conidia germinate and the germtubes enter the leaf through the stomata. Study the germinating spores. OBSERVE:—

3. The number of germtubes developed from each spore; number from each cell. DRAW. Study leaf-invasion through the stomata, as shown by Pool and McKay, *Jour. Agr. Research* 5, pl. LXXXI. COPY one or more of the figures, A, B, C, or F. Label in detail.

The mycelium developed from these germtubes spreads through and kills the tissue. A leaf-lesion or spot thus results. Very shortly this mycelium develops the fruiting structures on the surface of the lesion.

Cut out a spot from the leaf including a margin of healthy tissue. Macerate in a casserole for half a minute in potassium hydroxide over a microburner. Mount in water and OBSERVE:—

4. At the edges of the lesion, the almost hyaline branching mycelium, which may be uniform in width or have occasional cells thickened, with granular vacuolated contents. DRAW.

5. The tufts of brown conidiophores closely crowded at the base and widely separated at the top.

6. That very few of the conidiophores are straight but appear knotted or angled.

7. That at each angle is a scar where a conidium had been borne. How many scars does each conidiophore have?

8. The sclerotia-like mass of cells from which the conidiophores arise; color, size.

Place a lesion, lower surface up, under the binocular microscope and OBSERVE:—

9. The points emphasized in 5 and 6.

10. The relation of the tufts of conidiophores to the host-tissue, especially to the stomata.

11. The attachment of the conidium to the conidiophore. Is the thickened or the thin end attached to the stalk?

Make a DRAWING showing the points brought out under paragraphs 4 to 8.

Saprogenesis. No perfect stage of this fungus has yet been found. The fungus continues to fruit for a time after the leaf-tissues die. The sclerotia-like bodies from which the conidiophores arise remain alive under favorable conditions, in the host-debris. They produce a new crop of conidia the next spring which gives rise to the primary infections.

Secondary Cycles are started repeatedly during the summer by conidia from primary and secondary lesions. They repeat in detail the phenomena of the primary cycle.

REPORT

Review and abstract one of the following papers by V. W. Pool and M. B. McKay.

Climatic conditions as related to *Cercospora beticola*. Jour. Agr. Research 6:21-60. 1916.

Relation of stomatal movement to infection by *Cercospora beticola*. Jour. Agr. Research 5:1011-1038. 1916.

SEPTORIA LEAF-SPOT OF CELERY

This disease is often called late blight in contradistinction to the early blight or *Cercospora* leaf-spot. Both of them will affect the plant while it is still in the seedling-stage, but the *Septoria* leaf-spot is usually most severe later in the season. Both diseases are very destructive to the celery-crop.

SYMPTOMS

This disease is found on all parts of the plant above the surface of the soil.

On the leaves. Examine specimens of both dried leaves and those preserved in liquid. OBSERVE:—

1. The numerous spots scattered over the leaf, both on the upper and lower surface. Do the lesions extend through the leaf?

2. That the spots are irregular in outline and often coalesce with neighboring spots. Do the spots have any definite zones surrounding them as found in lesions on strawberry or beet leaves?

3. That each spot is marked with a large number of black papilla-like bodies,—the fruit-bodies of the pathogene.

Make a DRAWING of a diseased leaf.

On the stems provided, OBSERVE:—

4. The small brown sunken areas, which also show the black fruit-bodies. DRAW.

On the seed. If specimens of diseased seed-capsules are not provided, see *Zeitschrift für Pflanzenkrankheiten* 20:7. 1910; or Michigan Special bul. 77:4. COPY.

ETIOLOGY

The pathogene is *Septoria Petroselina* Desmazieres var. *Apii* Briosi and Cavara.

Life-history. Only the conidial form of the pathogene is known. In this form it is able to survive the winter, hence a sexual stage, if it does exist, is probably of little importance in the perpetuation of the fungus.

The **Primary Cycle** has its beginning with infection by the conidia which are present in the debris of last-years crop, or with those which are found on the seed.

Pathogenesis. Crush in water several of the small black pycnidia from old leaves, cover and OBSERVE:—

5. The thin long cylindrical spores, slightly curved and tri-septate. (Staining with iodine may be necessary to make the septation evident.)

These spores are blown about or spattered to the outer, lowest leaves of the celery, where they germinate, sending germ-tubes through either the upper or lower epidermis. The mycelium which is usually intercellular, soon forms pycnidia.

Cut thin cross-sections of a diseased spot or use prepared slides and under the high-power, OBSERVE:—

6. The large thick-walled pycnidium, which ordinarily is nearly globose but in the sections may be flattened or dented. Does it set deeply into the tissues?

7. The pseudoparenchymatous nature of the pycnidial wall, and the round opening,—the ostiolum.

8. Lining the pycnidial cavity, the short, sharply pointed conidiophores on the ends of which the slender conidia are borne.

9. The mycelial threads penetrating the tissue all about the pycnidium; the septation, contents and the branching of this mycelium.

Make an enlarged DRAWING of a pycnidium with the surrounding host-tissue.

Saprogenesis. There is no vegetative activity during the saprogenic phase. The fungus winters over as spores in the pycnidia in the old leaves and stems which have been left in the field. These spores serve as the inoculum the following spring.

Secondary Cycles. The conidia from the diseased leaves ooze from the pycnidium whenever moisture is present and are transferred by rain, tools or animals to the stems or other leaves. Secondary infections are repeated until the host is killed or removed.

REPORT

1. Make a list of all the *Septoria* leaf-spot diseases found on vegetables, to which you can find references in available literature. Give both the name of the pathogene and of the host.

THE BLACK SPOT OF ROSES

The black spot is a disease peculiar to the rose, and appears wherever this ornamental is grown, in temperate and tropical regions, in greenhouse and garden. It is, next to the powdery mildew, the most serious disease of roses. Few varieties appear to be immune.

SYMPTOMS

The lesions of this disease are confined to the leaves. Examine the material provided. OBSERVE:—

1. That some of the affected leaflets show isolated, more or less circular black spots.

2. That the margins of these spots are radiate-fibrillose. Study with the hand-lens.

3. The distribution of the lesions. Do they occur on both sides of the leaflet? Are they marginal or located more toward the center of the blade?

DRAW a leaf showing the character of the isolated black spots.

4. That some leaflets are nearly or quite covered by the lesions. Explain how this comes about.

5. That the lesions vary somewhat on different varieties of roses. In what respects? Show this in SKETCHES or in NOTES.

The leaves of some varieties become yellow very soon after the spots develop. In others there is little or no yellowing but in either case premature defoliation occurs. The early loss of foliage weakens the plants.

ETIOLOGY

The cause of this disease is an ascomycetous pathogene, *Diplocarpon Rosae* (Libert) Wolf, the conidial form of which has long been known under the name of *Actinonema Rosae* (Libert) Fries.

Life-history. The life-activities of this pathogene are perfectly correlated with the seasonal changes of the temperate zone, although the family, Microthyriaceae, to which this fungus belongs, is largely tropical.

The **Primary Cycles** are initiated in the spring by inoculum from the overwintered infested leaves on the ground.

Pathogenesis. The inoculum consists of ascospores produced in fruit-bodies in the fallen leaves. Remove a bit of tissue containing some of these minute fruit-bodies, crush, and under the microscope, OBSERVE:—

6. The two-celled ascospores, some floating free, others still within the asci. DRAW or if material is not available COPY from Bot. Gaz. 54, pl. XIII, fig. 16-17.

The ascospores do not appear to be shot into the air but simply ooze out on the surface of the old leaf. How may they reach the unfolding leaves of the plant? These ascospores will germinate only on the surface of living rose leaves. COPY Bot. Gaz. 54, pl. XIII, fig. 18.

The germtube penetrates the cuticle and gives rise to hyphae which, spreading just beneath it over the epidermal cells, form radiating strands of subcuticular mycelium.

Examine one of the spots on the leaves provided and MAKE OUT:—

7. These fine branched radiating mycelial strands. What color are they?

To study the structure and arrangement of these subcuticular strands, use prepared stained surface-mounts or make such mounts under direction of the instructor. Some mycelial threads penetrate throughout the leaf-tissue beneath. At certain places on the subcuticular mycelium, the conidial fruit-bodies are developed.

Again examining one of the old dark spots with the hand-lens, MAKE OUT:—

8. The conidial fruit-bodies, minute black specks,—the acervuli.

Make an enlarged diagrammatic DRAWING to show surface view of the subcuticular mycelium and acervuli in their proper relation to each other and to the leaf.

Make thin cross-sections through a spot showing numerous acervuli. Stain with methylene blue, wash, cover and OBSERVE:—

9. The relation of the acervuli to the leaf-tissues; subepidermal, subcuticular, or on the surface? (Compare with prepared stained slides.)

10. Structure of acervulus, conidiophores and conidia.

11. Mycelium, in epidermal cells and palisade-tissues, beneath the acervulus; inter- or intracellular?

Make a detailed DRAWING of a section through the acervulus.

These conidia, either borne by the wind or splashed by rain to healthy leaves, germinate to initiate the secondary cycles. Study germinating conidia and DRAW; or COPY Bot. Gaz. 54, pl. XIII, fig. 3.

Saprogenesis. After the diseased leaves fall to the ground, the subcuticular mycelium forms peculiar shield-like structures, beneath which in the leaf-tissue below the epidermal cells, the mycelium forms a perithecium. These perithecia begin to form in the autumn but do not mature until spring.

If prepared sections are available, study and work out in a DRAWING, the structure of the perithecium, or COPY figures from Wolf's article in Bot. Gaz. 54, pl. XIII.

Secondary Cycles. These are initiated repeatedly throughout the season by conidia from acervuli. Leaves involved in the secondary cycles fall prematurely like those suffering from the primary infections and the next spring also produce a crop of ascospores.

Pathological Histology. The brown or black coloration of the spots is due to the necrosis of the epidermal and palisade-cells and not to the mycelium as one might expect.

12. If fresh leaves are available, make thin cross-sections through a spot and verify the above statement.

SKETCH in detail a few diseased and healthy cells to show effects on the protoplasm and cell-organs.

REPORT

1. Detail in a letter to a grower of outdoor-roses two methods for the control of this disease, one an eradication-method, the other a protection-method and show how one may be made to supplement and strengthen the other. Assume that the grower knows the facts of the life-history of the pathogene.

LEAF-SPOT DISEASES

There are a large number of diseases affecting crops which may be designated under the term leaf-spot diseases. In most cases little more is known than the names of the fungi associated with the lesions. They are, nevertheless, in the aggregate, of considerable economic importance and deserve more careful and exhaustive study.

These diseases take their names from the character of the lesions most commonly exhibited, namely, spots on the leaves. These spots vary in size from mere specks to extensive dead or discolored areas, often involving a large part of the blade. In most cases they are rather definite in outline, more or less circular, and necrotic in character. They may, however, be angular or linear. They usually exhibit fruiting structures of the pathogene either on the upper or lower surface near the center of the lesion. Most of these leaf-spots are due to ascomycetous pathogens which produce during pathogenesis the conidial or imperfect fruiting structures. Hence one may expect to find on these spots either conidiophores, acervuli or pycnidia. In some cases the fruit-bodies of the sexual stage are formed in the lesions. For many of these leaf-spot fungi, the sexual or perfect stage is unknown; in many cases because it has never been sought for. It will usually be found to develop during saprogenesis on the old fallen leaves, either in the areas of the original spots or scattered over the dead foliage.

SYMPTOMS

Specimens of several leaf-spot diseases have been provided. Study the symptoms exhibited by each and make DRAWINGS to show the same. Note that the opposite sides of the lesion may differ. The drawings and labeling should bring out the different color-zones in the lesion, size, distribution in the blade and other characters.

ETIOLOGY

The pathogene in the material provided is in the pathogenic phase of either the primary or secondary cycle. Remembering that it is probably an ascomycetous pathogene, search carefully for the characteristic fruiting structures of ascomycetes. Use your hand-lens or place the leaf on the microscope-stage under low-power and examine with reflected light.

If conidiophores are found, wet the scalpel, scrape some of the conidiophores from the spot, and mount in potassium hydroxide. Study and DRAW conidiophores and conidia. How are the conidia borne on the conidiophores?

If acervuli or pycnidia are found, make an enlarged SKETCH of the lesion as it appears under the hand-lens, showing location and distribution of these fruit-bodies.

To study the internal structures of these fruit-bodies and their relation to the host-tissue, make thin cross-sections from dry material and mount in potassium hydroxide, or from alcoholic material, in water. Show the character of these structures and relations in DRAWINGS.

Determine the name of the pathogene by using Duggar, Fungous Diseases of Plants; Stevens, Fungi which Cause Plant Disease; Cooke,

Fungoid Pests of Cultivated Plants; Hartig, Diseases of Trees; and other books which the instructor may suggest. Study in this way as many as you can of the leaf-spot diseases provided.

REPORT

1. Drawings fully labeled, giving the name of each disease and the pathogene in each case.
2. Consult the available literature on two of the diseases studied and suggest possible methods of control.

BITTER ROT OF APPLES

Although bitter rot is known to occur on many different plants, it is of preëminent importance on the apple in the southern and central states. It is practically unknown in northeastern United States. It has been estimated that American growers of this fruit lose from bitter rot in some years ten million dollars. For the most part the disease has been called bitter rot, but the names anthracnose and ripe rot have been used.

SYMPTOMS

The disease affects the fruit and the bark of limbs and twigs. Leaves never show the lesions.

On the fruit. Spots on the apple ordinarily appear in July and August, although they may be seen as early as June and as late as October. Lesions at first appear as a light-brown discoloration beneath the skin. They are very small but soon enlarge and become firm in texture and circular in outline. When the lesions are three millimeters or more in diameter, the surface is distinctly sunken. Usually each fruit shows only a few spots, but in cases of severe infection more than a thousand separate lesions may occur. In cases like the latter very few if any of the spots increase in size, but instead, after reaching a diameter of about two millimeters, cease enlarging and appear as brown raised blisters. Study the specimen in the jar and OBSERVE:—

1. The stage of development of the affected apple. At what stage, green or ripe, was the fruit affected? Make an outline DRAWING of the apple.

2. The spots on the fruit; their size, color, distribution, appearance (sunken or raised). Estimate the number. DRAW lesions on the apple already outlined. Have any of the many spots enlarged? If so, show relative size in the sketch. See illustration photograph and Jour. Agr. Research 4, pl. VII, fig. 1.

In more typical cases of bitter rot only a few spots occur on each fruit. (See Pl. Ind. Bur. Bul. 93, pl. I and VI.) These spread rapidly, involving the entire fruit in a short time. Ripe apples have been inoculated with pure cultures of the fungus and placed in an incubator at 30° C. One or more specimens are provided for each student. Study the apples with the following POINTS as a guide.

3. The size of the spot; its form, and color. Note any other characters exhibited.

4. Do you find any evidence of the fruiting bodies of the pathogene on the surface of the lesions? If so, note their distribution, color, and relation to the tissues of the apple (sunken or superficial?).

5. Study the following figures so far as available:—Pl. Ind. Bur. Bul. 44, pl. I, II, IV, and VI; Pl. Ind. Bur. Bul. 93, pl. I and VI; Illinois Bul. 118, pl. III and VIII. Read the legend in each case.

Make a DRAWING to bring out the points observed. COPY a figure such as is shown in Pl. Ind. Bur. Bul. 44, pl. II.

Cut the artificially infected apple longitudinally into halves (do not use the razor) NOTE:—

6. Taste of the rotten flesh. Is it bitter? Examine Pl. Ind. Bur. Bul. 44:17, fig. 2-3. Make a DRAWING to show the lesion internally, its depth and shape.

Affected fruits may fall from the tree during all stages of the disease. Explain why. Whether or not they fall, there finally results a much-wrinkled dark-brown mummy. Examine the mummies provided and Pl. Ind. Bur. Bul. 93, pl. I, VI and VII. Read the descriptions. SKETCH to show a fallen and a hanging mummy.

On the limbs. The lesions on the limbs are cankers. Twigs are wholly killed and are then said to be blighted. Examine the canker before you and OBSERVE:—

7. The general appearance of the lesions as to color, size, shape and surface characters. Do you find evidence of a dead twig at the center? (See Pl. Ind. Bur. Bul. 44, pl. VII and Illinois Bul. 118, pl. I and IV.) Make a DRAWING of a typical canker. Young cankers begin as small, dark, sunken, sharply defined areas. (See Pl. Ind. Bur. Bul. 44, pl. IX.)

ETIOLOGY

The pathogene, now known as *Glomerella cingulata* (Stoneman) Spaulding and von Schrenk, is an ascomycetous fungus belonging to the family Gnomoniaceae of the Pyrenomycetes. Its conidial stage has long been known under several names, that usually applied to the fungus on the fruit being *Gloeosporium fructigenum* Berkley.

Life-history. Extensive studies on the life-history of this fungus have been made, especially by American workers, so that the details are now well known.

The **Primary Cycle** is very definite. There are many secondary ones during the season.

Pathogenesis. The mummied fruits hanging on the trees and the cankers are the sources of the primary inoculum. The fungus hibernates in these, producing during the spring conidia in acervuli. Apparently ascospores, which may initiate the primary cycle, ordinarily play but a small rôle in the life-history of this pathogene. Make crushed mounts of acervuli from the mummies or cankers provided and OBSERVE:—

8. The hyaline conidia; their form, granular contents, abundance and variation in size. DRAW several to show these points.

These conidia, washed and splashed by the rains to young fruits, initiate primary infection. Study the germinating spores provided. OBSERVE:—

9. The long septate germ tubes; point of origin.

10. The dark thick-walled appressoria formed at the tips of some of the germ tubes. What is their function? (Compare Pl. Ind. Bur. Bul. 44, pl. V.) DRAW several germinating spores.

The germ tubes, directly from the spores or from the appressoria, penetrate the uninjured skin of the apple or gain an entrance through wounds and a mycelium is rapidly developed resulting, usually within a few days, in a distinct rot-lesion. Fruits generally affected are often in a conical region beneath a mummy or canker. Remove a bit of the diseased apple flesh with the forceps and STUDY:—

11. The mycelium; its character and branching. DRAW.

The lesions soon begin to show developing fruit-bodies,—the acervuli of the conidial stage of the fungus.

Study the diseased fruits provided. NOTE:—

12. The location and concentric arrangement of the acervuli within the lesion.

13. The different degrees of development of these fruit-bodies from the center toward the margin of the spot.

14. The mass of conidia oozing from each mature acervulus; color and consistency of the mass. Remove a spore-mass to a drop of water; note the dissolving effect. The conidia are held together by a gelatinous substance which quickly dissolves in water. (See illustration photograph; Pl. Ind. Bur. Bul. 93, pl. VI, fig. 1A; Illinois Bul. 118, pl. III, fig. 2 and pl. V, fig. 1; Pl. Ind. Bur. Bul. 44, pl. I, II and VI.) These conidia initiate secondary cycles.

Make a careful DRAWING of several acervuli as they appear under the hand-lens.

Make a number of very thin sections through a bit of the affected tissue showing mature acervuli (or use prepared sections). Study and OBSERVE:—

15. The great mass of conidia pushed forth through the ruptured epidermis, the recurved edges of which show at each side of the spore-mass.

16. The mycelial bed, forming the base of the acervulus, from which the short simple conidiophores arise. How are the conidia formed?

17. The mycelium ramifying the adjoining host-tissues; inter- or intracellular?

DRAW to show the structure of the acervulus and its relation to the host-tissues.

Saprogenesis. With the mummification of the fruit and the death of the bark in the cankers, the mycelium does not die but sometimes continues a saprophytic existence. It lies dormant through the winter, resumes its activities in the spring and usually produces acervuli, though sometimes the sexual fruit-bodies, perithecia, are produced. The latter are rare. Acervuli having been studied, a brief examination of the perithecium may be made. Study the prepared sections. OBSERVE:—

18. The long blunt protruding beak of the perithecium.

19. The relation of the perithecium to the host-tissue.

20. The asci; form and arrangement in the perithecium.

21. The structure of the perithecial walls.

22. The ascospores; number and arrangement in an ascus; form.

DRAW a longisection of the perithecium. The ascospores, when produced, doubtless serve to initiate primary cycles.

Secondary Cycles are initiated repeatedly throughout the growing-season on fruits and twigs, causing fruit-rot, twig-blight and limb-cankers. The pathogene passes through the same forms and phenomena as in the primary cycles.

Pathological Histology. Slides are provided showing sections through young spots such as are to be observed on the fruits in the jar. Study the sections and DRAW to show pathological effects observed. Explain the formation of the cork-layer.

REPORT

1. Make a diagram illustrating the cycles in the life-history of *Glomerella cingulata* (Stonem.) S. and von S.

APPLE BLOTCH

This disease is generally known as apple blotch, but it is sometimes called black scab, fruit-blotch, and star-fungus.

It should be clearly borne in mind that apple blotch is entirely distinct from sooty blotch and apple scab, in spite of the common names applied. This disease, like bitter rot, is confined largely to the central and southern states, and in the southern half of the apple-belt it is more destructive, taken year by year, than bitter rot and apple scab combined.

SYMPTOMS

The fruit, bark and foliage are all affected by this disease.

On the fruit. The lesions vary somewhat with the variety. Using available affected fruits, and Pl. Ind. Bur. Bul. 144, pl. I, II, IV, V and VI, study the symptoms and OBSERVE:—

1. That the first evidence of the disease is a very small inconspicuous, light-brown speck, which, under slight magnification, has the appearance of a stellate collection of brown fibers just beneath the epidermis. On account of this stellate character of the spot this disease is at times confused with apple scab. On young apples, the lesions are at first water-soaked, and in wet weather there may be a yellowish gummy exudate therefrom.

2. That spots enlarge radially, attaining a diameter of from 3–10 or more millimeters, becoming darker in color. On light-colored varieties the spots are green, later becoming brown, while on red varieties the spots are at first red, then changing to black. The advancing margin is irregular and jagged and has a fringed appearance.

3. That sometimes the spots are sunken and may or may not show this fringed margin, or they may be raised; gradations between the fringed smooth spot and the umbonate spot (raised and strongly projecting in the center). Where the spots are numerous, they may coalesce, forming large lesions involving a major portion of the fruit. On some varieties at least, like the Ben Davis, the skin ultimately cracks. The crevices, although usually about a half an inch long, may girdle the fruit and extend to the core. Often these cracks intersect, forming a cross.

4. That within a few days after the spots become visible, black pimples (the fruiting bodies of the pathogene) develop thereon.

Make a series of SKETCHES to bring out points observed in 1, 2, 3, and 4.

On the bark. The disease affects the fruit-spurs, twigs and rapidly growing shoots, producing characteristic cankers. Larger limbs and trunks are not usually affected. On the material provided, OBSERVE:—

5. That on the fruit-spurs, the spots are at first purplish or blackish. The center turns brown with age, the margin remaining purplish. The resulting lesion is small but rather conspicuous, with a crack along the margin.

6. That on water-sprouts and on other rapidly growing shoots, the cankers have much the same appearance as that just described, but are larger, sometimes measuring an inch or more in length and often girdling the stem. Longitudinal cracks appear not only along the edge but also through the cankered tissue, eventually giving the surface a rough appearance. This character is noticeable on cankers two or three years

old. The canker may extend at several points along the margin, producing a lobed or somewhat concentric appearance. Frequently the canker is limited permanently by a crack and the wound is calloused.

Make DRAWINGS to show the points brought out in 5 and 6.

On the foliage. Examine the material provided and OBSERVE:—

7. That the spots on the leaves are at first irregular, light-brown, yellowish or whitish and small, measuring 1.5 millimeters or less in diameter. Their distribution over the surface of the leaf is without order. On account of their minuteness, several may appear on a leaf without attracting attention, and perhaps without great injury; in more severe cases several hundred spots frequently appear on a single leaf. Badly diseased leaves may fall prematurely. The leaf-petioles may be girdled and instead of the leaves falling, they may die, turn brown, and hang on the tree.

DRAW to illustrate the symptoms of the disease on leaves, as observed. (See Pl. Ind. Bur. Bul. 144, pl. I.)

ETIOLOGY

The pathogene causing apple blotch is known as *Phyllosticta solitaria* Ellis and Everhart. This name applies to the asexual stage, no sexual stage being known. It may be regarded as an ascomycete, even in the absence of a perithecial stage, but until the missing stage is found, it is temporarily grouped with the Fungi Imperfecti.

Life-history. A number of important links in the life-history of this pathogene remain to be discovered. Enough, however, is known to account for its whereabouts throughout the year.

The **Primary Cycles** are initiated rather late in the spring, usually a month or more after the petals fall.

Pathogenesis. Twig-cankers are the chief sources of the inoculum. The fungus hibernates in these lesions as mycelium which becomes active in the spring and which produces pycnidia along the advancing margin of the canker. Examine the twigs provided. OBSERVE:—

8. The minute black pycnidia in the bark near the margin of the cankers. DRAW to show their appearance under a hand-lens.

Crush a bit of the bark containing pycnidia and examine for pycnospores. OBSERVE:—

9. That they are ovoid-elliptical, hyaline, one-celled. DRAW.

10. That they sometimes exhibit a gelatinous sheath, as shown in Pl. Ind. Bur. Bul. 144, pl. III, fig. 2-5. COPY to show this. These pycnospores are washed or carried to the young fruits, twigs and leaves.

Germination of these pycnospores occurs in twelve to eighteen hours. Examine the germinating spores and OBSERVE:—

11. Number and position of the germ-tubes; branching, septation and contents. DRAW or COPY Pl. Ind. Bur. Bul. 144, pl. III, fig. 6-7.

The mycelium developed from the germ-tubes ramifies the tissues of the fruit in a stellate fashion as may be observed by a careful examination of the lesions. Pycnidia are soon developed by this mycelium beneath the epidermis. Study prepared slides showing the pycnidia in section. OBSERVE:—

12. The position and relation of the mature pycnidium to the host-tissues.

13. The structure of pycnidial walls and conidia-bearing structures.

Make a detailed DRAWING of the pycnidium and adjacent host-cells.

The pathogene produces only sterile pycnidia on the leaves. Apparently on these organs the cycle is never completed. The pycnospores, matured in primary lesions on twigs and fruits, serve to initiate secondary cycles on all susceptible host-organs. In the twig-lesions, the pathogene hibernates either as immature pycnidia(?) or as mycelium from which pycnidia are developed in the spring. Thus is the cycle completed without the appearance of a sexual spore-form.

Saprogenesis. There is no marked tendency exhibited by this pathogene to grow and develop saprophytically. It may feed to some extent on the dead tissues after leaves and fruit have fallen. The formation of spore-producing pycnidia only during the fore part of the season, followed by the development (in the later secondary cycles at least) of pycnosclerotia, suggests that perithecia, similar to those known for *Guignardia Bidwellii* on grapes, may be produced during saprogenesis. They are as yet unrecorded.

Secondary Cycles originate from the pycnospore-crop of the primary infections. They are initiated only during the first half of the summer, and probably do not often initiate other secondary cycles. They duplicate in all respects the primary cycles.

REPORT

1. Compare and contrast in parallel columns the two diseases of the apple:—*blotch and sooty blotch*.

LEAF-CAST OF CONIFERS

Species of the genera *Pinus*, *Abies*, *Picea* and *Larix* frequently suffer from needle-diseases known in common by the general name leaf-cast or needle-cast. These diseases have been much more abundant in Europe than in this country. Frequently they have been epiphytotic on certain hosts in Europe. Although all leaf-cast diseases of conifers are very similar, they have various specific characteristics. The one considered in this outline is common on yellow pine (*Pinus ponderosa* Douglas) in the northwestern part of the United States.

SYMPTOMS

General symptoms. The leaves of certain branches turn brown at any time during the growing-season. In young trees, the leaves of the tips of all of the branches may be attacked while in older trees only the lower branches on the windward side usually show diseased needles. Witches'-brooms of large size are produced on older trees. Often the brooms are heavy enough to cause them to hang pendant from a larger limb. The recent growth of affected young trees is much deformed, due to the forcing of growth back of the tips of the twigs which are killed. (See Jour. Agr. Res. 6 : 277-288, pl. XXXII.)

On the needles. The needles may be infected at any time during the growing-season. They show variable symptoms according to the time of year at which infection takes place and the point on the leaf where infection was initiated. In the material provided, NOTE:—

1. The brown tips of certain needles. This is the first evidence of the disease when the needle becomes infected near the tip.

2. In some cases the entire needle may gradually become straw-yellow in color and then turn brown later in the season.

3. The long rows of black, shiny fruit-bodies of the pathogene on the dead needles. On which surface of the needle do they occur, dorsal or ventral? DRAW.

On the twigs. The mycelium grows from the affected needles into the twig and produces hypertrophy. Later the affected twigs die. The branch bearing affected twigs becomes gnarled and enlarged. Adventitious buds are forced and a witches'-broom is developed. In the specimens provided, OBSERVE:—

4. The hypertrophied twigs and branches.

5. The character of the brooms produced on the older trees. (See Jour. Agr. Res. 6, pl. XXXII.)

DRAW showing the effect on the woody parts of the tree.

ETIOLOGY

The leaf-cast of yellow pine is caused by *Hypoderma deformans* Weir. *Hypoderma* is a genus of the Ascomycetes in which apothecia with valve-like coverings are formed. These valves open, exposing the hymenium, when abundant moisture is present. They close again during dry periods.

Life-history. The life-history of this pathogene has only recently been investigated by Wier. (Jour. Agr. Res. 6 : 277-288.) Other closely related species causing leaf-cast of other conifers have been studied for years and the life-histories of all these species are similar. Where pycnidial

forms are known, but little attention is paid to them since it seems that the asexual spores play no rôle in the life-history. Some believe that the pycnospores are not even capable of germinating.

The **Primary Cycle** is initiated by the ascospores which are forcibly ejected from the open apothecia on dead needles at any time during the growing-season when sufficient moisture is present.

Pathogenesis. The apothecia are formed on dead needles of healthy twigs and witches'-brooms. Although the twigs are penetrated and killed by the mycelium, the apothecia are formed only on the needles. In the material provided, **OBSERVE:—**

6. The longitudinal slit running the length of each apothecium.

DRAW.

7. That, in material which has been in a moist-chamber, the two halves forming the covering of the apothecium are pulled apart exposing the hymenium. **DRAW.**

In prepared cross-sections of needles with apothecia, **NOTE:—**

8. That the apothecium is formed under the epidermis in the mesophyl-tissue.

9. The black mycelial covering of the fruit-body.

10. The asci and paraphyses arising from the base of the fruit-body.

11. The ascospores; two-celled and much longer than broad. **DRAW** showing the structure of the apothecium and contents.

On the same needles with the apothecia are the smaller asexual fruit-bodies,—the pycnidia. In the material, **NOTE:—**

12. The resemblance between the apothecia and pycnidia. Crush one of the pycnidia in water. **OBSERVE:—**

13. The elongated conidia. The conidia of the leaf-cast pathogenes have never been shown to cause infections.

The ascospores are shot from the asci when sufficient moisture is present to open the valves covering the hymenium. If material is available, **NOTE:—**

14. The milky deposit on the glass slides suspended over the discharging apothecia.

15. By examination with the microscope, that this milky deposit is due to the large number of ascospores ejected upon it.

Saprogenesis. There is no distinct or obligate saprophytic existence required in the life-history of this pathogene. Leaves infected in early spring are killed and while still attached to the tree produce mature apothecia in the late autumn, although usually they do not mature until the following spring or summer. Pycnidia are formed among the apothecia at the same time.

Secondary Cycles are apparently lacking in the case of this pathogene, since it takes the fungus a year to develop new inoculum.

REPORT

1. Formulate control measures for this disease in a nursery which is surrounded by a mixed forest containing yellow pine. Give reasons for all steps in proposed measures based on the facts known about the life-history of the pathogene.

BLACK ROT CANKER OF APPLE

This disease affects chiefly the apple, but other pomaceous fruits are by no means free from the trouble. Marked variation in susceptibility of varieties is shown only where the canker-form of the disease appears. In western New York the Twenty Ounce is noted for its susceptibility to the canker. As a fungous disease of the apple in this state, it is perhaps second only to apple scab, the chief losses occurring as a result of cankers on the large limbs.

SYMPTOMS

Lesions appear on limbs, leaves and fruit, known on each respectively, as cankers, leaf-spots and black rot.

On the limbs. Larger limbs are more often affected than twigs or trunks. The lesions are cankers and are usually on the upper sides of the limbs. At first the bark is reddish brown in color and sunken. (See Cornell Bul. 379 : 62-63, pl. VIII, and IX, fig. 4). Study the specimens available and OBSERVE:—

1. The size of the lesion; its surface, appearance, shape, color and margin. Is the margin definite?

2. The pimple-like fruit-bodies of the pathogene; their numbers and distribution within the lesion-area. Supplement the above with a careful study of the illustrations in Cornell Bul. 379 to show the characters of the cankers.

On the leaves. The first evidence of a spot on the leaf is the appearance of a minute purplish speck which soon enlarges until it has reached a diameter of 2 to 10 millimeters. It then becomes brown. Examine the the specimens provided and OBSERVE:—

3. That the spots are at first circular, but that older ones show a lobing or a concentric appearance due to secondary spreading; the center appears lighter in color and the whole effect is that of a frog's eye. The disease is therefore called "frog-eye" by certain writers.

4. The presence or absence of the fruit-bodies of the pathogene. If present they will be much smaller than those on the cankers.

Study, in addition to the specimens, the illustrations in Cornell Bul. 379, pl. VII, fig. 3; Virginia Bul. 209; Pl. Ind. Bur. Bul. 121, pl. III, fig. 1-2. Defoliation results in cases of severe infection. (See last named bulletin, plate IV.)

On the fruit. Black rot is primarily a rot of ripe fruit, although green fruits may be attacked. Symptoms may show anywhere on the surface.

Study, the material and illustration specimen provided. OBSERVE:—

5. That the lesions appear first as small brown spots, which may turn dark at once or remain brown until the entire fruit is involved.

6. Concentric color-zones in the lesion.

7. The firmness of the rotted tissues; taste and odor.

8. The black fruit-bodies of the pathogene just under the skin; their distribution and arrangement within the lesion.

DRAW to show the characters brought out above. (See Cornell Bul. 379, pl. VII, fig. 1-2.)

The lesion usually surrounds an injury of some sort, as for EXAMPLE:—

9. A codling-moth wound in either green or ripe fruits, as seen in the illustration specimens. DRAW.

10. The blossom-end of the fruit, a type common in Missouri and other regions.

DRAW from illustration specimens or from figures in the bulletins.

After a month or so the entire fruit becomes a waxy and finally a dry mummy which may fall or may hang on the tree for a year or more. Study specimens provided and show in a DRAWING the character of these mummies.

ETIOLOGY

The name of the pathogene is *Physalospora Cydoniae* Arnaud. Until recently, it was known as *Sphaeropsis Malorum* Berkley which name is most commonly found in literature. The pathogene is very closely related to *Guignardia Bidwellii* (Ellis) Viala and Ravaz, the cause of black rot of grapes. Both organisms have similar perithecial and pycnidial stages.

Life-history. This pathogene is evidently a facultative parasite. It develops most vigorously as a saprophyte on dead plant-tissues. It depends almost entirely on its asexual spores for propagation. The perithecial stage appears but rarely, at least in northern United States.

The **Primary Cycle** is initiated by pycnospores, or by the ascospores, when the latter are produced. Only the former will be considered here.

Pathogenesis. The fungus passes the winter as pycnospores in pycnidia and as mycelium in the cankers from which pycnidia with pycnospores are produced. Remove several of the pycnidia from the cankered bark provided and crush in a drop of water on a slide. Examine under the microscope and OBSERVE:—

11. The great number of pycnospores; their color, size, shape and septation. DRAW to show variations in the spores.

These pycnospores constitute the chief primary inoculum. Washed by rain or possibly carried by insects to leaves or to wounds in the fruit or to dead tissue in the bark of the limbs, these spores germinate and initiate the first infections. Study the germinating spores provided and OBSERVE:—

12. The long germ tubes; color, contents, septation and branching. How many from each spore? DRAW.

From the infection-court the mycelium penetrates in various directions into the living tissues, killing them as it spreads.

To study the mycelium in the tissues, mount a bit of the rotted fruit well teased apart, or study prepared slides of leaf-lesions in surface view, or sections through diseased bark. OBSERVE:—

13. The color, size, septation and manner of branching. How does this mycelium compare with the germ tubes from the pycnospores?

14. The relation of the mycelium to the host-cells (best seen in rotted fruit-tissues); inter- or intracellular? Are haustoria developed? (See Cornell Bul. 379, fig. 31.)

DRAW to illustrate the character of the mycelium in the tissues.

The mycelium near the center of the lesion soon begins to form gnarls of hyphae which rapidly develop into pycnidia containing pycnospores. (See Cornell Bul. 379, fig. 23.) Study the sections (freehand or prepared) through pycnidia. OBSERVE:—

15. The dark globose structures, seen in longisection; their relation to the tissues in which embedded; the ostiola.

16. The structure of the pycnidial walls; origin and shape of the conidiophores; the spores and manner in which formed on the conidio-

phores. When ripe these pycnospores are disseminated and initiate secondary cycles.

Make a DRAWING of a section through the pycnidium. (See Cornell Bul. 379, fig. 22.)

Saprogenesis. The fungus readily infests the dead bark of a great many plants. (See list in Cornell Bul. 379: 95 and 98.) Pycnidia may be formed on such plants. Occasionally perithecia are also formed from the saprogenic mycelium. The conditions under which they are produced are not clearly understood.

Study sections through a perithecium under the demonstration microscope. Make a DRAWING to show its structure. (Compare Cornell Bul. 379, fig. 18.)

Secondary Cycles are not different from the primary except as to the time of year at which they occur.

Pathological Histology. The lesions produced on the host are necrotic. Laboratory studies of histological changes will be confined to the more striking features of the canker. (Read Cornell Bul. 379:111-116, and study figures 34-37.)

The mycelium attacks the cortex, phloem, cambium, medullary rays, vascular bundles and pith. The outward changes evident to the naked eye, however, are the results of a necrosis of the cortex and phloem. These tissues are affected most generally and most extensively. Study slides showing sections cut through the center of limb-cankers and MAKE:—

17. Diagrammatic DRAWINGS of both cross- and longisections through the center of the canker, showing healthy tissue above and below the lesion.

The fungus breaks away from the lesion at different points and spreads in a streak up and down the limb. If this is attempted in the cortex, the advance of the fungus is halted by the development of a cork-layer. There results, then, a pocket. If the pathogene leaves the cortex and advances in the sap-wood, no check on its progress is made by a cork-layer as just described above. On the other hand it enters the sap-tubes of the xylem and travels a considerable distance above and below the canker. As it does so the wood-cells, adjacent to the xylem-ducts, are discolored and filled with a brownish deposit. Consequently a streak is visible to the unaided eye where the mycelium is advancing. SHOW this streak in the diagram of a longitudinal section through the canker.

Study prepared sections through the margin of a young canker. NOTE:—

18. The appearance of the tissues involved. Study the margin between healthy and diseased tissues. What sort of tissue is evident? Find the mycelium. Make a DRAWING including a portion of healthy and diseased tissues and the mycelium.

Study prepared sections cut through the stem at some distance above the canker. DRAW to show points observed.

REPORT

1. Discuss the parasitism of *Physalospora Cydoniae* Arnaud.

ENDOTHIA CANKER OF CHESTNUT

This disease has become the most destructive epiphytotic of tree diseases in this country. It is interesting in many respects. In the first place, the causal fungus is an importation, and the destruction caused is typical of what may often occur when new and unsuspected pathogens are transferred to a different flora and climate. Secondly, it is as good an example of efficient correlation of parasite with its host and of extreme susceptibility of the host, as any disease could demonstrate.

SYMPTOMS

The lesions of this disease appear chiefly in the body and larger limbs of the tree. The symptoms are of two general sorts: the lesions or cankers themselves, and the secondary effects produced by these cankers in uninvaded parts of the host. For convenience the latter are to be studied first.

General symptoms. The diseased tree may often be detected at a distance by the following symptoms which are to be studied in the photographs provided. OBSERVE:—

1. That limbs with developing cankers, which have almost girdled them, do not produce normal leaves in the spring. They are usually lighter green in color and never develop to full size. This symptom is quite noticeable during May and June. (See illustration photographs 2 and 3.)

2. That dead leaves and last year's burrs often hang to limbs which have been killed by the fungus during the summer. (See illustration photographs 3 and 4.)

3. The dead limbs, the death of which can be traced to distinct cankers, that are of the type described below. (See illustration photographs 1, 2, 3 and 4.)

4. The abnormal development of suckers on trunk or limbs. Examination may prove that these suckers grow from below a cankered area. (See illustration photographs 2 and 3.)

Cankers. The normal healthy bark of the chestnut remains smooth and green for several years and thus the cankers are very distinct on young growth. In the material and photographs provided, NOTE:—

5. That the cankers may be either sunken (necrotic) or raised and swollen (hypertrophic). (See illustration photographs 5 and 6.)

6. That the surface of the diseased bark is always of a reddish cast, thus contrasting sharply with the green bark. (See frontispiece in Cornell Bul. 347.)

7. That in a canker of the necrotic type, the bark is roughened and often split longitudinally; also the "pimples" or small eruptions all over the surface of the diseased area,—the fruiting stromata of the fungus. (See illustration photograph 5.) DRAW.

8. That in a canker of the hypertrophied type, the outer bark is split longitudinally because of the abnormal development of the tissues beneath. (See illustration photograph 6.) DRAW.

The sunken cankered area, reddish brown in color, is the usual evidence of the disease. When the fungus attacks rough-barked portions of the tree, its effects are not noticeable externally until the stromata of the

fungus break through in the cracks of the bark. This is the condition existing at the base of most trees attacked, and only the most careful scrutiny will reveal the lesion. (See illustration material.)

ETIOLOGY

This canker disease of the chestnut is caused by the pyrenomycetous fungus, *Endothia parasitica* (Murrill) Andersons. It is a member of the order Sphaerales. The conidial stage, considered by itself, falls in the genus *Endothiella* of the Fungi Imperfecti.

Life-history. Primary and secondary life-cycles are not as sharply differentiated in *E. parasitica* as is usual with pyrenomycetous pathogens. This is due to the lack of marked correlation between the periodicity of spore-form production in the fungus and seasonal periodicity. Cycles are initiated by ascospores or conidia at any time when moisture and temperature permit. Those initiated early in the spring are to be regarded as the primary cycles.

The **Primary Cycles** may be initiated, as indicated above, by either of two kinds of inocula.

Pathogenesis.

(a) Ascospores are produced within perithecia developed in superficial stromata on the older portions of a canker or on dead twigs and bark. The production of ascospores is not limited to any specific time of year. It takes a certain time (depending on seasonal conditions) to develop perithecia after a new infection. If this period is interrupted by winter, development is simply arrested until spring. Therefore only those perithecia which were in a certain stage of development are mature in the early spring.

In the material and photographs, NOTE:—

9. The perithecial stromata, dark chocolate-brown in color. The surfaces of the mature stromata are covered with small projections or papillae, each with a small black dot at the apex. This is the ostiolum of a perithecium embedded in the stroma. (Illustration photograph 11.)
DRAW.

Crush a bit of this stroma and OBSERVE:—

10. The ascospores; two-celled, hyaline, eight in each ascus (photographs 34-37). DRAW. These ascospores are forcibly discharged by the bursting of the asci as they accumulate on top of the papillae after being crowded out through the ostiolum.

If material is available study the violent discharge of the spores. With the hand-lens, OBSERVE:—

11. The tiny points of light popping from the ostiola of the perithecia.

Make a SKETCH to show ascospore-discharge.

Hold clean dry slides at different heights above this material and determine the distance to which spores are shot in a vertical direction; also in a horizontal direction. From these slides, NOTE:—

12. That the whole contents of the ascus is ejected at once. Ascospores germinate as soon as discharged, if they fall in water. DRAW a germinating ascospore showing the germ tubes (Cornell Bul. 347:568).

(b) Conidia are produced within subcortical or superficial pycnidia (illustration photograph 10a and photograph 12). Pycnidial

development may be arrested at any stage by winter conditions and be resumed in the spring. Study the material provided and OBSERVE:—

13. The long, twisted, yellow, tendril-like masses of pycnospores as they are pushed out from the subcortical pycnidia which appear as “pimples” on the thin-barked twigs. Study either fresh material in moist-chambers or photographs. These strings of spores are composed of thousands of the pycnospores stuck together by a mucilaginous substance. (See illustration photographs 8, 9 and 10.) DRAW.

Make a mount of a portion of one of these spore-threads in a drop of water on a slide and NOTE:—

14. That the spores in the yellow string separate immediately.

Place under the microscope and OBSERVE:—

15. The small hyaline pycnospores. They are among the smallest pycnospores of the fungi. (See illustration photograph 13, d and e, and photographs 13–14.)

Some conception of the large number of pycnospores produced from one canker may be obtained by noting the number observed in the mount from only a portion of one small thread. These spores, on account of their clinging together, cannot be blown about by the wind. Then too, the fact that they separate when water is present, would indicate that eventually these conidial strings find lodgment very near the place of formation, if not carried away by insects, birds or other animals. The possibility of bark-boring insects and insectivorous birds acting as inoculating agents in transferring these conidia to a healthy tree is plausible.

These pycnospores will not germinate in pure water but require the presence of substances from the bark in solution. The process of germination begins with enormous swelling of the minute spore. Study pycnospore-germination, either from slides or from Cornell Bulletin 347 : 566.

DRAW to show three or more stages in spore-germination.

Ascospores or conidia, when introduced into a wound of any sort in the bark which exposes the tissues beneath the cork, germinate and develop a mycelium. (See illustration photograph 12.)

The further developments of the pathogene are the same regardless of whether ascospores or conidia initiate the cycle. The period of incubation is an uncertain one depending on many factors; weather conditions (especially temperature), depth of inoculation and thickness of bark. In general, however, in the thin bark of twigs, definite lesions may be discerned three weeks after inoculations made with conidia, and four weeks after inoculation with ascospores.

Wounded tissue and a brief saprophytic growth, sufficient to produce mycelial fans, are necessary before living tissues can be invaded and infection accomplished. Examine material and illustration photograph provided. OBSERVE:—

16. The form, size and color of these mycelial fans; relation to the host-tissue.

17. In mounts under the microscope, the structure of the fans, branching of hyphae and relation to each other.

DRAW to show general appearance of the fans and also their structure.

After infection is accomplished and the mycelial fans begin to invade and kill living bark-tissues, the canker develops rapidly. Pycnidia are produced abundantly just back of the advancing edge of the canker. Study material or photographs of young cankers and NOTE:—

18. The appearance of the "pimples",—the subcortical pycnidia. (See illustration photograph 8, and photographs 1-5.)

Make sections or study prepared slides of sections through the pycnidia.

NOTE:—

19. The mass of mycelium of the fungus aggregated in the outer bark just at the base of the fruiting structures. Such a "bed" of mycelium is termed a stroma. (See illustration photograph 2c.)

20. That this stromatic layer of mycelium encases the spore-bearing cavities. The fruit-bodies are said to be encased or enclosed in a stroma.

21. The irregular convoluted cavities in the stroma,—the pycnidia. (See illustration photograph 13c.)

22. The compact layer of pycnospores lining the entire inner wall of the pycnidium (photograph 6).

23. The manner of the cutting-off of the pycnospores (photograph 6).

24. That each stroma contains a single pycnidium and that the spores produced are pushed out of the ostiolum through a rupture in the bark in the long thread noted above. These spores are disseminated and initiate secondary cycles.

Make DRAWINGS to show these points.

After a short period of pycnospore-production, the stroma grows to the extent that the bark is pushed back and a cushion-like structure is raised considerably above the surface. These stromata are at first reddish in color, but later at maturity of the spores they are a chocolate-brown and covered with numerous small projections or papillae. (See illustration photograph 11 and photographs 23, 30-33.) In the material provided, OBSERVE:—

25. The extent of the stroma as compared with that of the pycnidial stage. (See illustration photograph 13c.)

26. The cavities here are simple flask-shaped perithecia. (See photograph 11 and illustration photograph 14.)

27. The thick, lead-colored walls of the perithecia.

28. The long black necks leading from the perithecial cavity to the surface of the stroma where they open through the papillae by the ostiola. (See illustration photograph 14.)

29. The numerous asci contained in the perithecia and floating about (photographs 34-37).

DRAW showing the perithecium in longisection.

The ascospores produced on the canker are washed down the tree and can initiate secondary infections at any slight wound in the bark. Eventually the tree becomes covered with cankers and dies.

Saprogenesis. The fungus does not require saprogenic conditions for the continuance of its cycles. On the other hand it is capable of existing as a saprophyte producing both spore-stages, the perithecia following the pycnidia as in pathogenesis. Whenever a saprogenic habit is assumed, it is purely incidental and not a definite stage providing for something which cannot be produced during pathogenesis. Strictly speaking, however, the perithecial stage may, although produced abundantly on cankers on living trees, be considered as the saprogenic stage since it is developed far back from the advancing margin and probably is largely or entirely fed by saprophytic mycelium.

In the **Secondary Cycles** all the structures and phenomena of the primary cycles are exactly repeated. Secondary cycles differ in no way from the primary except in the time of year at which they are initiated.

Pathological Histology. The mycelium of the fungus kills the cells of the bark, cambium, and sap-wood. The cells are killed in advance of the mycelium, presumably by excreted toxins.

In the sections of cankers provided, OBSERVE:—

30. The discoloration of the tissues affected. Make a diagrammatic DRAWING illustrating the enlargement of the canker.

31. The septate and abundant mycelium. Often mycelial fans are formed in the bark. By stripping off the bark of a fresh canker, this is easily observed. (See demonstration microscopes for cross-sections of healthy and diseased twigs or see Phytopath. 4:191-200.)

REPORT

1. Bearing in mind the pertinent facts of the life-history of *E. parasitica* (Murr.) Andersons, enumerate and discuss the reasons why the Endothia disease is difficult to control.

RHIZOCTONIA STEM-ROT

This disease affects a great variety of plants. It appears to have an extensive range, occurring both in the new and the old world, in the temperate zones and in the tropics.

SYMPTOMS

The symptoms vary considerably depending upon the host affected. Studies here will be confined to seedlings, potatoes and carnations.

On seedlings. The disease on seedlings is often known as damping-off. The base of the stems are affected, sometimes also the roots. Examine the diseased seedlings provided and OBSERVE:—

1. The form, size and color of the lesions.
2. Their location on the stem with respect to the surface of the soil.
3. The depth to which the lesion penetrates (cut across the stem through the lesion). Are any of the stems girdled?
4. A rotting of the roots in some seedlings.
5. In some cases, the reddish brown mycelial strands or mats of the pathogene in the lesions.

The symptoms of this disease in seedlings may be those resulting from a stem-rot or root-rot. They vary slightly with the seedling-host affected. (See Illinois Bul. 189:308-337.)

Make SKETCHES to show the symptoms of the disease as exhibited in the seedlings provided.

On potatoes. A striking field-symptom is a marked unevenness in the stand. Many hills are wanting or the shoots are small and weak. If on digging up the seed-tubers, where plants have failed to come up, diseased sprouts are found, it is an almost certain evidence of this disease. Examine the specimens of diseased sprouts provided and see Maine Bul. 230, fig. 62-67. OBSERVE:—

6. The dark-colored canker-like lesions; their size, location on the sprouts and depth to which they penetrate.
7. That some of the sprouts may get above ground before they are rotted off. They may even develop weak plants which grow slowly and persist for a time.
8. That many of the sprouts which are rotted off send up new sprouts from a node below the lesion. These may in time become diseased and even rot off or they may produce nearly normal tops.

Make a series of SKETCHES to show the various symptoms and effects exhibited by diseased potato sprouts.

Study the older stalks of diseased plants provided. OBSERVE:—

9. The necrotic lesions at the base of the stem just below the soil-level. In some specimens the stem is nearly rotted off.
10. The effect of the disease as exhibited in the tops, especially the production of clusters of small tubers about the base of the stem at the surface of the soil and the production of aerial tubers in the axils of the leaves.
11. The rosette-symptoms exhibited by the tops of some affected plants. (See Ohio Bul. 139, fig. 4; and 145, fig. 1.)

Make DRAWINGS to show symptoms exhibited in the older stems and tops.

A variety of symptoms have been described for the tubers of affected plants. In the specimens or photographs provided, OBSERVE:—

12. The large number of small potatoes from a diseased hill as compared with those from a healthy hill. Lesions on the tuber-stolons cut the young potatoes off before they can mature. The plant is thus stimulated to a production of more tubers which in turn are likewise prevented from reaching mature size. (Maine Bul. 230, fig. 69.) DRAW.

13. That tubers themselves may be affected, showing a russeting or even deep cracking of the surface. Cankers or pits in the tubers are held by some investigators to be frequent symptoms of this disease. (Maine Bul. 230, fig. 72-73.) DRAW.

The most striking and certain diagnostic sign of this disease is the presence on tubers of the sclerotia of the pathogene. Examine the tubers provided. OBSERVE:—

14. The numerous, dark-brown or black sclerotia scattered over the surface of the tuber.

15. Their irregular size and shape.

16. That on attempting to scrape them off, they cling very tightly, but when they come away it is evident that they are entirely superficial. DRAW a tuber to show the character and distribution of the sclerotia. These sclerotia are not usually associated with lesions on the tuber and are exceedingly common on potato-tubers, even from apparently healthy plants.

Sclerotia may appear on the stems or roots as well as on the tubers. (See illustration specimens.)

On the carnation. This is probably the most serious carnation disease with which florists have to contend. Both cuttings and older plants are affected. Cuttings are attacked below the surface of the soil and rot or damp-off like seedlings.

Examine the specimens of diseased plants provided. OBSERVE:—

17. The silvery gray color of the foliage as compared with the grey-green of the healthy plants.

18. The erect and withered habit of the diseased plants.

19. The soft and rotted cortex of the stem just at, or above, the surface of the soil. It comes away very readily from the wood.

20. In the crevices of the bark, the dark-brown sclerotia of the pathogene; not always easily discovered.

DRAW to bring out the contrast, so far as you can, between diseased and healthy plants.

ETIOLOGY

The cause of this disease, so cosmopolitan both as to hosts and range, is the basidiomycetous fungus, *Corticium vagum* Berkley and Curtis, long known in its mycelial form as the sterile fungus, *Rhizoctonia Solani* Kühn.

Life-history. This pathogene presents some unusual features in its life-history. True conidial bodies are wanting. Sexual spores, the basidiospores, are formed, but apparently with no regularity and only under certain very favorable conditions. Sclerotia of a very simple type are produced in great abundance and doubtless are the only structures which serve to carry the pathogene over from one season to the next.

The **Primary Cycle** is initiated in the spring. The source of inoculum is, without much doubt, the sclerotia on overwintered host-debris, in the soil, or on resting organs of the host such as tubers or roots.

Pathogenesis. Exactly how the primary infections are initiated does not appear to have been observed. The following studies suggest the probable phenomena involved. Tease apart, in a drop of water on the slide, one of the sclerotia from the material provided (on potato tubers or in pure culture). Examine and OBSERVE:—

21. The barrel-shaped cells in profusely branched chains which make up the sclerotium.

22. The color of the walls and the granular protoplasmic contents of many of the cells.

23. That these cells break away readily in groups of several each.

Make DRAWINGS of several of these cells together.

Many of these cells germinate like conidia. Study the slides provided showing germination. OBSERVE:—

24. The different types of germination presented, polar and equatorial.

25. The diameter of the germtubes as compared with that of the parent-cell and the number of germtubes from each.

26. The granular and vacuolate character of the contents of the germtubes.

27. The difference in the thickness and color of cell-walls in germinating cells and germtubes.

DRAW several germinating cells to show the different types observed.

These barrel-shaped cells constitute the inoculum. Just how they find their way to the infection-courts is not always clear. The germtubes may be capable of growing for considerable distances through the soil to the susceptible stem or root which they penetrate and infect.

The vegetative mycelium, developed from these germtubes, is quite different in character from that composing the sclerotia. Mount some of the growing mycelium from the advancing margin of the agar culture provided. OBSERVE:—

28. The long parallel hyphae, much branched and frequently anastomosing.

29. The characteristic manner of branching: the acute angle formed by the branch; the construction of the branch at the point of origin; and the septum laid across it a short distance above the point of origin. These characters usually hold for the actively growing mycelium in the tissues of the host and serve as a diagnostic sign of the disease.

30. The colorless character of the young mycelium; the yellowing and browning with age.

Make thin longitudinal sections of diseased stems (or use prepared slides) and OBSERVE:—

31. The mycelium in relation to the cells of the host-tissue; inter- or intracellular? What tissues are invaded? How does the mycelium here compare with that studied from the culture?

DRAW a portion of the section in detail to show the character of the mycelium and its relation to the cells.

Hyphae may grow out from a diseased plant and, spreading through the soil, reach and infect nearby healthy plants. It is probable that external mycelium and sclerotial cells, formed in the soil, may be carried in cultiva-

ting and in other ways to the neighborhood of healthy plants and so initiate secondary cycles.

Saprogenesis. The evidence seems to show that this pathogene may vegetate as mycelium in old host-debris and often in the soil. At least it develops on dead plant-parts and many strains appear to be almost obligate saprophytes.

A sexual form of the pathogene, the *Corticium* stage, is at times developed, and always on the surface of the living plant-parts above the ground, i. e. on stems and leaves. Apparently it does not always occur in the life-cycle. It may appear on the healthy parts above a *Rhizoctonia* lesion or on the stems and leaves of plants which show no such lesions or other evidences of injury. There is no indication that the fungus is ever pathogenic in this stage.

Examine the material provided, OBSERVE:—

32. The thin, white, web-like coating over the surface, very difficult to discern in dried or pressed material.

Scrape and mount some of this coating; examine under the microscope and OBSERVE:—

33. That it is made up of a much-branched and septate mycelium. How does it compare with the vegetative mycelium of the *Rhizoctonia* stage as to size, branching and septation?

34. The swollen tips of many of the branches each bearing two to four pointed sterigmata. These are the basidia.

35. Scattered through the mount, the small, hyaline, ovate basidiospores. Some half-formed or nearly matured spores still attached to the sterigmata may sometimes be found.

Make a DRAWING of the mycelium of the sexual stage, showing the fruiting structures or COPY from Colorado Bul. 91, pl. III.

The mycelium of the sexual stage appears to develop from the sclerotia on the underground parts of the host, or from the mycelium in the dead tissue of lesions, or from host-debris in the soil. It spreads upward over the surface of the living parts, without causing any direct injury, to a position where spores may be formed and readily disseminated.

The basidiospores are scattered by the wind. It is almost certain that they never function in directly infecting a healthy plant but germinate and develop a saprophytic mycelium which may go into the sclerotial stage or at once attack healthy plants.

The development of a considerable amount of saprophytically nourished mycelium in and about the infection-court seems to be a necessary prelude to an attack on living tissue.

Secondary Cycles are frequently initiated during the growing-season by spreading or broken-off, vegetative mycelium. Sclerotia are readily formed and their barrel-like cells germinate at any time when conditions favor, so that they are doubtless a frequent source of secondary inoculum. So far as known, secondary cycles duplicate the primary in all details.

REPORT

1. Enumerate the facts in regard to the morphological structures and life-habits of *C. vagum* B. & C. which make its control difficult and explain why in each case.

2. Outline a plan for reducing the ravages of this disease in one of the crops which it affects.

POLYPORACEOUS WOOD-ROTS OF TREES

The wood-rots of trees, as a class of diseases, cause more economic loss than any other type of enphytotic disease of trees. The difficulty in controlling wood-rot diseases is due largely to their insidious nature and to the relatively small amount of work which has been done on them. The control of wood-rots in the forest is one of the important measures in forest-management, which must soon be recognized. Since the fruit-bodies of the fungi causing wood-rots are the chief signs by which these diseases may be recognized, it is important that the student gain a working-knowledge of their identification.

SYMPTOMS

Sections from different deciduous and coniferous trees, showing the character of a few common wood-rots, are provided. OBSERVE:—

1. That the decayed wood is, in some cases, darker in color than in the normal, usually reddish or brownish. Such wood-rots are called "red-rots." The color is caused by residual substances left by the causal fungus. DRAW.

2. In other cases the rotted wood is lighter in color than the normal wood and often is almost white. Such wood-rots are called "white-rots." The change in color is due to the delignification of the wood-elements, leaving the whitish cellulose cell-walls. No colored residue is left as in the red-rots. DRAW.

3. That certain wood-rots at first appear in the heart-wood and work out toward the sap-wood, while others work from the start chiefly in the sap-wood. The former are known as "heart-rots" and the latter as "sap-rots." DRAW.

4. That, in some wood-rots, the tissue is entirely decayed leaving holes or pockets. Usually these holes are surrounded by a white border that represents the cellulose fibers which were not dissolved. DRAW.

5. That, in other cases, the decay results in a uniform change to a weak cellulose structure but in no place are the tissues entirely destroyed. DRAW.

6. The black lines bordering the decayed portion of the wood in many wood-rots. This line is usually due to both the color of the feeding-hyphae and to the first products of the decay-process in solution in the sap. DRAW.

ETIOLOGY

The majority of wood-rots are caused by basidiomycetous fungi belonging to the two large families, Agaricaceae (toad-stools) and Polyporaceae (polypores or bracket-fungi). The polypores are by far the more important of the two groups.

The main distinction between the Agaricaceae and Polyporaceae is in the morphology of the under side of the fruit-body. The hymenium, each basidium of which bears four spordia on sterigmata, is spread over the plate-like or tube-like surfaces which hang vertically from the cap or pileus.

Examine the prepared sections of the hymenium of a toad-stool or polypore with the microscope. OBSERVE:—

7. The irregular tangled mycelium making up the trama or supporting-structure for the hymenium.

8. The more or less regularly arranged tips of hyphae extending at right angles to the general direction of the trama-hyphae,—the subhymenium. The hyphae of the subhymenium always lie in a horizontal direction when the fruit-body is in place.

9. The layer of basidia borne on the tips of the hyphae of the subhymenium. Each basidium has four sterigmata on the free end. The layer of basidia and the spores borne on them make up the hymenium.

10. The four spores borne on each basidium, one on each sterigma.

Make a DRAWING to show the structure of the trama, subhymenium, basidia and spores in relation to one another.

Study the specimens of toad-stools and polypores on the tables and classify each numbered specimen as to the family to which it belongs, using the following KEY:—

- A. Hymenium usually on the underside in the form of radiating plates or strong folds. AGARICACEAE
- B. Hymenium usually below or on the outer surface when the plant is spread over the substratum; honey-combed, porous, tubulose or reticulate. POLYPORACEAE

The numbered specimens of polypores on the tables are next to be studied and the genus to which they belong determined for each. All the steps in using the key must be written on sheets provided, to show how each determination was arrived at, thus:—1. Polyporus. 2, B, b, x,⁰⁰. The older terminology is used here because of its use in most of the literature and because it is the more simple. The rearrangement of the members of this group and the new genera proposed may be consulted in North American Flora 7, and Murrill, Northern Polypores, p. 1-64.

The following key to the Polyporaceae (i.e. the Polyporeae as given in the text on nature of decay in wood) is rearranged from that of Overholt, The Polyporaceae of Ohio. Missouri Bot. Gard. Ann. 1:81-155.

- 1. Sporophore entirely resupinate; pileus none. PORIA
- 2. Sporophore sessile or stipitate, sometimes effused-reflexed but normally not entirely resupinate.
 - A. Hymenium either daedaloid, labyrinthiform or lamellate, at least in part.
 - a. Context white.
 - x. Pileus minutely velvety to glabrous; context more than 1 mm. thick DAEDALEA
 - y. Pileus hirsute to villous; context 1 mm. or less thick.
 - ⁰Hymenium lamellate, at least in part. LENZITES
 - ⁰⁰Hymenium daedaloid but never lamellate. DAEDALEA
 - b. Context brown.
 - x. Plants woody and perennial, more than one cm. thick; hymenium not at all lamellate. TRAMETES
 - y. Plants coriaceous or corky, less than 1 cm. thick; hymenium often lamellate. LENZITES
 - B. Hymenium poroid or sometimes broken up into teeth.
 - a. Hymenium broken up into teeth.
 - x. Tubes or teeth 5 mm. or more long. IRPEX
 - y. Tubes or teeth less than 5 mm. long.
 - ⁰Hymenium labyrinthiform at first and remaining so at the margin. DAEDALEA

- °°Hymenium never labyrinthiform.
 - μPileus less than 1 cm. broad; fungus mostly resupinate IRPEX
 - μμPileus more than 1 cm. broad; fungus not mostly resupinate..... POLYPORUS
- b. Hymenium poroid, not broken up into teeth.
 - x. Pores large and hexagonal; stipe present.
 - °Stipe lateral, often rudimentary; pores usually radiating and longer in the radial direction.. FAVOLUS
 - °°Stipe usually central or sub-central; pores not radiating..... POLYPORUS
 - y. Pores small, and circular or angular; stipe present or absent.
 - °Tubes in a single layer; plants annual.
 - μTubes not in a distinct stratum but appearing to be sunken to different depths into the context..... TRAMETES
 - μμTubes forming a well marked stratum entirely distinct from the context.... POLYPORUS
 - °°Tubes in two or several layers; plants perennial.
 - μHymenium bright yellowish brown; plants growing only on the wood of coniferous trees..... TRAMETES
 - μμHymenium whitish, flesh-colored, dull brown, etc., but not bright yellowish brown; plants growing on the wood of either coniferous or deciduous trees..... FOMES

The specimens of polypores, labeled as to name, should next be studied. The names of each should be written down, together with an original description of the specimen in the student's own words. These notes should be as full as possible, especially as to all the striking differences.

Life-history. There is great similarity in the life-histories of the polypores causing wood-rots. Wounds in which the wood is exposed are required for infection. The basidiospores, carried by the wind, constitute the only kind of inoculum except by the direct growth of the vegetative mycelium from a diseased individual to a healthy one.

Primary Cycles only, exist, since there is only one crop of basidiophores formed annually. A single cycle may require several years to be completed.

Pathogenesis. The spores formed on the basidia are forcibly thrown from their sterigmata and fall out from between adjacent projections of the hymenial surface (pores or gills).

Examine figures in Buller, *Researches on Fungi*, and make diagrammatic DRAWINGS illustrating the anatomy of a toad-stool and polypore in longitudinal section, showing the path taken by an ejected spore in each.

The type of infection-court required differs with the species under consideration. In general, those fungi causing heart-rots require very different types of wounds for successful infection from those causing sap-rots. In any case the basidiospore, borne by the wind may find lodgment in a suitable infection-court or, if not, it may later be washed by water to a suitable place. The spores are relatively short-lived and proper conditions for germination must be provided in a short time if the spores

are to function. Under proper conditions, the spore germinates and a new mycelium develops in the wood. The steps in the decay-processes from the time of infection to the production of the mature fruiting body differs with the species of polypore concerned and somewhat also with the kind of tree affected. In the case of the fungi which attack the woody parts of trees, delignification of the cell-walls usually takes place, at least the lignin-cellulose composition of the cell-wall is destroyed. It is supposed that an enzyme similar to hadromase, isolated by Czapek, is excreted by all wood-rotting fungi. The products of the activities of cytolytic enzymes then, form the main food for the fungus. Many other compounds, such as tanorins and proteids, are used by some of the wood-rotting fungi.

Saprogenesis. The majority of the polypores may live purely as saprophytes. Some do so more commonly than others. However, this saprophytic habit is only incidental, since it does not form a required phase in the life-history which produces anything that is not produced in the pathogenic phase. The same kind of inoculum, however, is formed in saprogenesis as in pathogenesis and this may initiate new primary cycles.

Pathological Histology. To illustrate the process by which the mycelium of wood-rotting polypores causes the decay of the wood, prepared cross-sections and longitudinal sections of beech wood, rotted by *Fomes pinicola* (Swartz) Fr., are provided. The sections are stained to show; lignified cell-walls, light green (methyl green); delignified cell-walls or cellulose, brownish red (congo red); and middle lamella, light red (ruthenium red). OBSERVE:—

11. The abundant and light-colored hyphae of the fungus in the partially decayed wood. DRAW.

12. The large dark-colored hyphae where new tissue is being delignified. DRAW.

13. The delignification of the wood-elements. Which layer of the cell-wall is delignified first?

14. The characters of sound beech wood, elements present, and thickness of walls of different elements.

Make a DRAWING of healthy beech wood at a point showing both spring and summer wood with all the elements properly arranged and detailed. Label fully.

15. The characters of the decayed wood, noting the changes brought about in the elements.

Make a DRAWING of decayed beech wood showing the same elements as in the drawing of healthy wood. Label fully, and bring out all the changes caused during the decay-process. If necessary, make DRAWINGS of the intermediate steps between the healthy and decayed wood, using the cell-wall of a single element to illustrate the point.

REPORT

1. Hand in report-sheets filled out giving your determination of the family and genus of each of the numbered specimens.

2. Hand in the notes taken on the named specimens and an original dichotomous key (arranged like the one in the outline) by which each species can be determined.

RING-SHAKE OF SPRUCE

This wood-rot of spruce is also common in pine, larch, fir and other conifers. It is not known to affect junipers. It has been estimated that this disease causes more loss than the aggregate of all the other numerous wood-rots.

SYMPTOMS

Trees affected by this disease, as in the case of other wood-rots, do not show any symptoms externally until limbs begin to die. The fruiting bodies of the causal fungus, produced at the broken stubs of limbs or from the fissures in the bark, are usually the first signs of disease.

External signs. Study U. S. Agr. Dept., Veg. Phys. and Path. Div. Bul. 25, pl. XII and the specimens provided. OBSERVE:—

1. The various forms of sporophores which constitute the signs of this wood-rot.

2. The sporophores may be shelving or more or less resupinate.

3. The sporophores may emerge from broken stubs of branches or from the fissures in the bark.

Make outline DRAWINGS from the specimens or plate referred to, showing the above points.

Internal symptoms. In the specimens of transversely and longitudinally-sawed wood of spruce affected by this wood-rot; and in plates VI, VII, and VIII of the above mentioned bulletin, NOTE:—

4. That the first effect on the wood is the red-brown discoloration. Such timber is said to be "red-hearted."

5. That later in the decay-process the color becomes lighter.

6. That white areas appear here and there in the wood in somewhat later stages and that these areas gradually increase in number and size. DRAW.

7. That the centers of the white areas are entirely dissolved, leaving holes surrounded by white borders. DRAW.

8. Around most of the holes, a little distance away and usually midway between adjoining pockets, a definite black line. DRAW.

9. That the size of the pockets and the amount of relatively sound wood left between the pockets depends on the kind of wood attacked.

10. That the rotted area enlarges longitudinally in the wood much faster than transversely and often the decay involves either partially or completely a certain number of annual rings. This is the character which gives the name "ring-shake" to this wood-rot.

ETIOLOGY

The cause of wood-rot known as ring-shake of conifers is the polyporaceous fungus, *Trametes Pini* Fries (= *Porodaedalea Pini* (Thore) Murrill).

Life-history. The species of polypores which cause wood-rots of living trees are similar in all the major facts of their life-histories. (See pages 96-97).

The **primary cycles** are initiated by the basidiospores which are wind-borne.

Pathogenesis. It has been shown that the basidiospores are shot from the sterigmata with just enough force in each case to bring them to

the center of the perpendicular pore from which they fall and are then caught by the wind and blown about in all directions. (Buller, *Researches on Fungi*, p. 133-152). If material of discharging spores is available, NOTE:—

11. The clouds of spores falling from the sporophore. DRAW.

12. The deposit of white spores on the black paper under the sporophores.

Mount some of these spores in water and under the microscope, OBSERVE:—

13. The size and shape of the spores. DRAW.

The sources of the primary inoculum are the shelving or resupinate sporophores produced in great abundance on the outside of diseased trees. In the material provided, NOTE:—

14. That the fruit-bodies are perennial.

15. The black, checked upper surface of the pileus in the case of the shelving form.

16. The dull-brown pore-layer,—the compound hymenium.

17. The small round pores making up the pore-layer.

DRAW a shelving and resupinate fruit-body.

Make freehand sections of a fruit-body provided, stain with eosin, mount and examine with the microscope. OBSERVE:—

18. The character of the mycelium forming the trama of the pileus and the substance between the pores. DRAW.

19. The subhymenium and the two kinds of cells produced in the hymenium, basidia and setae. DRAW.

20. The form of the basidium; the four spores borne on each.

DRAW.

If material does not show points mentioned above, see *Veg. Phys. and Path. Div. Bul. 25*, pl. IX, fig. 3. COPY.

If the spores reach the exposed heart-wood of susceptible conifers, they germinate under favorable conditions and the mycelium extends down into the trunk of the branch. Why are exposed wounds in the sap-wood of conifers not infected?

The mycelium usually confines its activities to the annual rings first invaded, extending longitudinally and circumferentially in these rings much more rapidly than radially in the wood. Thus, completely decayed rings in the wood often result as the first evidence of disease. This is the ring-shake symptom. Soon, however, the mycelium invades the rings of wood adjacent to the shake and spreads inward and outward toward the sap-wood. The progress outward depends upon the amount of resin present (i.e. in pine). The mycelium rarely penetrates the sap-wood. In spruce, however, it not only decays the sap-wood but penetrates and kills the tissues of the bark and produces small sporophores in the fissures of the bark.

Saprogenesis. The ring-shake fungus may live in the wood of susceptible conifers after the tree is dead. Fruiting bodies are formed also in the saprophytic stage and furnish inoculum for new infections. Saprogenesis is not an obligatory stage, however, for the production of anything in the life-history of the fungus which is not produced in the living tree.

Secondary Cycles are not considered as existing in cases of wood-rot pathogenes since only one source of inoculum is developed annually. The period

over which the dissemination of primary inoculum takes place may, however, be long, as the sporophores of polypores often cease spore-production under adverse weather-conditions. With more favorable conditions it is resumed.

Pathological Histology. The decay-process caused by this fungus in spruce is of two kinds. In the cross- and longisections of spruce wood where pockets are not formed, NOTE:—

21. That the medullary ray-cells are destroyed and from these the mycelium spreads to the adjoining tracheids. DRAW.

22. That the elements of the spring wood are destroyed before the thicker-walled summer elements.

23. That the first noticeable change in the cell-walls is the separation of the middle wall-layer from the outer layer. It has not been determined that this is due to the shrinking of the middle layer as a result of the disintegration of the outer layer. DRAW.

24. That when a hypha makes a hole through a cell-wall the hole is at first cylindrical but changes by enlarging in the middle. DRAW.

25. That the outer wall-layer of each cell separates from the outer wall-layer of the neighboring cell. DRAW.

26. That, following this stage, the middle wall-layer is changed into cellulose. Then there are left for a time only the outer and inner lignified layers which later also change to cellulose. DRAW.

27. That the middle layer disappears first, then the outermost layer is dissolved. DRAW.

28. That the only portion of the wall resisting decay is the innermost layer of the cell-wall. DRAW.

In cross- and longisections of spruce wood where pockets are formed, NOTE:—

29. The general appearance of the pockets under low-power; their location, abundance of mycelium, and the black lines surrounding the pockets. DRAW.

30. The black line under the high-power. Of what is it composed? DRAW.

31. The fine mycelium around the margin of the pocket. DRAW.

32. That the progress of the decay around the margin of the pocket proceeds as follows:—The entire wall is changed into cellulose; the elements become separated by the dissolving of the middle-lamella; the outermost wall-layer then dissolves and the wall becomes thinner and thinner until only the innermost layer is left. DRAW showing the progressive stages.

REPORT

1. From the studies made of this decay-process, write a concise account of the steps involved. (See U. S. Agr. Dept., Veg. Phys. and Path. Div. Bul. 25:31-40, pl. VI-X.)

IGNIARIUS HEART-ROT

This is the most common of the numerous heart-rot diseases of deciduous trees and may be taken as a type. The fungi causing heart-rots of deciduous trees are more or less alike in their life-habits and the methods for their control are similar. (In this connection, see von Schrenk and Spaulding, Diseases of Deciduous Forest Trees. Pl. Ind. Bur. Bul. 149: 1-85, pl. 1-10, fig. I-X, and extensive bibliography.)

SYMPTOMS

External signs. In the material provided, OBSERVE:—

1. That the only external evidence of the disease is the bracket-like sporophore of the fungus.
2. The point at which the sporophore emerges. Why? (See Pl. Ind. Bur. Bul. 149, pl. I.)
3. The variation in the size of the sporophores. Upon what two factors does this variation depend?
4. The age of different sporophores.
5. The characteristics, shape and color of the sporophores. Compare with other specimens on the demonstration table.

Make a DRAWING to show characteristics of the sporophore in position on the host.

Internal symptoms. In the transverse and longitudinally sawed sections of diseased wood provided, OBSERVE:—

6. The rotted wood at the center of the lesion; white or yellowish white and more or less soft and crumbly.
7. The distinct black line or lines bounding the white rotted areas.
8. The light-brown or yellowish brown zone of firm hard wood just outside the outermost black line. Cause of the discoloration?
9. The brown felty character of the mycelial growth from the cut ends of the sticks.

Make DRAWINGS to show the symptoms as seen in the transverse and longitudinal sections.

ETIOLOGY

This disease is caused by a basidiomycetous fungus, *Fomes igniarius* (Linneus) Fries, one of the perennial species of the family Polyporaceae.

Life-history. This fungus, although largely confined in its activities to the heart-wood of its host, may be regarded as a true pathogene, because it develops only in living trees.

Primary Cycles only are known, the infections occurring once a year. With our present limited knowledge of this fungus, no sharp line can be drawn, if any exists, between its saprogenic and pathogenic activities.

The source of inoculum is the compound sporophore on the living tree. Study several of the sporophores provided and OBSERVE:—

10. The cracked and checked, hard black upper surface of the sporophores.
11. The uprolled reddish brown margin of the last-formed annual pore-layer.
12. The brown surface of the pore-layer.

13. The minute almost microscopic pores making up the pore-layer. Study with the hand-lens. The basidiospores sift forth from these pores, and are often discharged in such quantities as to be visible to the naked eye, forming a white coating on the bark and leaves beneath the sporophore.

Mount some of the spores (discharged or in crushed mounts of the the pores). Examine and OBSERVE:—

14. Their form, size and color. DRAW.

Some of these spores, carried by the wind, find lodgment in dead branches or the stubs of broken-off limbs of their favorite hosts. Here they germinate sending forth a germtube into the dead host-tissue. This germtube develops into a mycelium which penetrates to the heart-wood where it spreads in all directions but most rapidly up and down, feeding and accumulating a reserve of food.

The character of this mycelium and its relation to the host-tissue can be best studied in prepared sections made through the region of the black line. Study the sections provided and OBSERVE:—

15. The black line. Of what is it composed?

16. The invasion of the medullary rays and wood-fibers surrounding the tracheae in advance of the black line.

17. The character of the mycelium in the region of the black line and when advancing into uninvaded tissue.

18. The character of the mycelium inside of the black line where the tissues have already been delignified.

Make DRAWINGS showing the above points.

After a year or more, a sporophore develops from this mycelium, usually emerging from the old infection-court. The lower surface develops a pore-layer which is annually renewed thereafter for many years.

Examine the sporophore that has been sawed through and NOTE:—

19. That it is composed of many pore-layers or strata, one below the other, each succeeding one being more extensive than the preceding. Normally one layer is formed each year. DRAW.

20. The rusty brown color of the inside of the sporophore with white flecks through it.

21. The length of the pores, that is, the width of the pore-layer. Are the pores continuous from year to year?

22. The shape of the pore-openings or mouths. Compare them, as to size and shape, with the pores of other species on the demonstration table.

Make a DRAWING of the longisection through the sporophore; also an enlarged DRAWING to show shape and arrangement of the pore-openings.

Make thin cross-sections of bits of the pore-layer and NOTE:—

23. The mycelial structure of the pore-layer.

24. The basidia with spores lining the inside of the pores.

Detail in a DRAWING the structures exhibited in the cross-section of a pore.

The spores sift out of these pores and are disseminated by the wind. Make a diagrammatic DRAWING showing the pores in longitudinal section and the path of a spore in getting out of the tube. (See Buller's book, *Researches on Fungi*, p. 189.)

Pathological Histology. Prepared cross- and longisections of wood from a living maple tree are provided. These have been stained to show:

lignified cell-walls, light-green (methyl green); delignified cell-walls or cellulose, brownish red (congo red); and middle lamella, light-red (ruthenium red). The dark color of the large vigorous hyphae when advancing into healthy tissue is natural. In the cross-sections, OBSERVE:—

25. The dense growth of the large dark-colored hyphae making up the black line. At this point the fungus is advancing in a solid line toward the sap-wood. DRAW a few cells included in the black line, detailing the hyphae.

26. The scattered areas, especially around tracheae and in the medullary rays, where the same dark-colored hyphae are found. How do these centers of decay originate ahead of the general advance? Corroborate your answer by two DRAWINGS; one diagrammatic, showing the relation between the black line and the advanced centers of decay; the other detailed, showing the actual cells.

27. The accumulation of green-staining material around the cells newly invaded. What does this indicate?

28. The delignification of the cell-walls near the advanced centers of decay. Do the hyphae of the fungus have to be in contact with a cell-wall to cause its delignification? Proof? How is the delignification brought about? Detail answers in DRAWINGS.

29. The character of sound maple wood; elements present and thickness of the walls of different elements. Make a DRAWING of healthy maple wood showing an annual ring with all elements properly arranged and detailed. Label fully.

30. The characters of the decayed wood some distance inside the black line, noting changes in the elements. Make a DRAWING of the decayed maple wood showing the same elements as in healthy wood. Label fully and bring out all changes brought about by the decay-process.

Study the longitudinal sections and OBSERVE:—

31. The mycelium advancing into healthy tissue.

32. The change brought about by the decay-process inside the black line as compared with the healthy tissue outside. Make DRAWINGS of healthy and decayed maple wood, contrasting the conditions as seen in the longitudinal sections of each.

REPORT

1. Compare and contrast the decay-process caused by *F. igniarius* (L.) Fr. in maple with that caused by *T. Pini* Fr. in spruce.

FENCE POST-ROT

One of the items of expense on the farm is the building and keeping up of fences. The life of the posts is considerably shortened by various saprophytic fungi which often attack and quickly rot them. A knowledge of the character of these rots and of the conditions favorable to them is of service in devising means of treatment which greatly lengthen the life of the timber.

SYMPTOMS

Sections of affected timber are provided. OBSERVE:—

1. The light-colored region about the margin, inside of the bark,—the affected wood. Compare the hardness of this with that of healthy wood. Is the bark similarly affected?

2. That the affected wood in the early stages includes only the outer three or four annual rings, but that ultimately the penetration is deeper, showing that although the sap-wood is first invaded the heart-wood will finally also be destroyed. Compare with the blocks that have been affected longest.

Make DRAWINGS of the transverse and longitudinal aspects.

3. On the larger blocks, the shelving or resupinate sporophores of the fungus causing this decay. Why are some shelving and some resupinate? Make an outline DRAWING showing these sporophores attached to the post.

ETIOLOGY

The cause of this rot is a very common polyporaceous fungus, *Polystictus versicolor* (Linneus) Fries. The fungus is ordinarily a saprogyne but is known to cause destructive heart-rot of catalpa. It attacks a great variety of timbers of broad-leaved trees but never affects coniferous wood.

Life-history. There are apparently no secondary cycles, the infection occurring in the earlier part of the season with usually no production of fruit-bodies until the end of the season. There is of course no pathogenic phase.

The **Life Cycles** are initiated by basidiospores from the fruit-bodies during the wet weather of spring and early summer, and possibly also to some extent from early-matured sporophores in the autumn.

Saprogenesis. Examine the black paper under the sporophores which have been in a moist-chamber for several hours. OBSERVE:—

4. The tiny piles of white spores, each representing the spores discharged from a single pore.

Mount some of these spores in water and examine under high-power. OBSERVE:—

5. Their form, size, color and contents. DRAW. Study and DRAW to show spore-germination as seen in slides provided.

The germ-tube penetrates to the dead sap-wood, where it develops a vigorous mycelium. This mycelium spreads through the wood, disintegrates it by means of enzymes and feeds upon the products of this disintegration.

. Study a thin longisection of the diseased wood and OBSERVE:—

6. The mycelial threads, especially abundant in the pitted vessels and medullary rays.

7. The form, size, branching and septation of the mycelium.

8. The relation of the hyphae to the cells and vessels. Do they penetrate between or through the tissue-elements?

DRAW showing a few cells and vessels with the ramifying mycelium.

This vegetative activity continues during the season. No conidia are formed in nature so far as known. Toward autumn the mycelium begins to form sporophores which break forth through the lenticels or cracks in the bark, as may be seen in the specimens provided. Examine the sporophores further and OBSERVE:—

9. The velvety upper surface. Note the zonations and coloring.

10. The lower or pore-surface. Note the numerous small pores; their form and size; color of the pore-surface.

Make a DRAWING of a sporophore.

Make thin cross-sections of the pileus and examine with high-power.

NOTE:—

11. That it is made up of interlacing strands of mycelium.

12. The parallel columns which are the sides of the tubes; lining these tubes the basidia bearing the spores.

Make an outline DRAWING of the cross-section of a pileus to indicate the relation of parts.

Carefully examine the hymenium and MAKE OUT:—

13. The club-shaped basidia, bearing at their tips four spores on short sterigmata.

Make a large DRAWING of the hymenium showing its structure.

These sporophores are not killed even by long drying and when wetted again mature and discharge more spores. A single mature sporophore will, if kept moist, continue to discharge spores for more than two weeks.

The mycelium lives over in the rotten tissues and, spreading into adjoining sound wood, continues the destruction and produces a crop of sporophores year after year until the post is destroyed.

Pathological Histology. In order to more clearly understand the changes which have taken place in the rotted wood, a study of the normal wood must first be made.

Cut thin cross- and longisections of the healthy wood from the center of the blocks. Stain two minutes with methyl green and 45 seconds with congo red. Examine carefully. OBSERVE:—

14. The large tracheae in the spring wood. Note the heavy walls of these ducts; within the tracheae, the cell-like structures. These are ingrowths of the walls of surrounding cells, now dead, and are termed tyloses.

15. The heavy-walled wood-cells surrounding the ducts.

16. The medullary rays between the xylem-wedges. The methyl green stains the lignified tissues; and the congo red, the tissues the walls of which are partially or wholly of a cellulose nature.

Locate each of these in the cross- and longisections. Make a detailed DRAWING of a part of the cross-section to show the relation of the tissues, the size of the cells and the thickness of the walls.

Now examine the similarly stained cross- and longisections of the decayed wood. OBSERVE:—

17. The dense masses of mycelium in the tracheae and the absence of the tyloses. Compare the walls of the tracheae here with those in the sound wood.

18. The mycelium in the wood-cells about the ducts and the medullary ray-cells. Examine the cell-walls. They are thinner than in the healthy wood. Note that the spring wood of each annual ring is more affected than the autumn wood.

19. That some of the tissues in the diseased wood take the stains differently than they did in healthy wood; which tissues? In the diseased wood, the mycelium also is stained red.

The destruction or removal of the lignin from the cell-walls explains the lighter color of the decaying wood; the partial destruction of the cellulose walls accounts for its crumbly nature.

Make an enlarged DRAWING of a cross-section of the affected wood to show all changes which have occurred and the location and character of the mycelium.

REPORT

1. Give the method of treating the fence posts and explain the philosophy of the treatment. (See Farmers' Bul. 387.)

STIPPEN

This disease goes under a variety of names; Baldwin-spot, bitter pit, and fruit-pit being the most commonly used. It is one of the non-parasitic diseases. It occurs the world over, wherever apples are grown.

SYMPTOMS

So far as is known, lesions are evident only in the fruit. Examine the specimens provided, studying carefully the external characters of the lesions. OBSERVE:—

1. The slightly depressed areas or “pocks”; their size, color and shape.

2. Their distribution over the fruit, usually most numerous in what region?

3. The differences between these lesions and those of the New England fruit-spot, specimens of which are provided.

Make a SKETCH of an affected apple to show the general character and distribution of the lesions.

Make an enlarged detailed DRAWING of a lesion of the stippen and beside it one of the New England fruit-spot for comparison. Label fully.

Divide the apple (with the scalpel) lengthwise through the core. OBSERVE:—

4. The internal appearance of the lesion; color, size and shape.

5. The distribution of the lesions; relation to the surface-pits and to the vascular bundles.

SKETCH the cut surface to show the form, size and distribution of the lesions as seen in this aspect.

Placing the halves together, cut the fruit across the core. STUDY:—

6. The character and distribution of the internal lesions as exhibited in this aspect, especially their relation to the vascular strands. SKETCH to show the lesions as seen in the cross-section of the fruit.

Study the individual internal lesions more critically AS TO:—

7. Usual form, color, texture and taste. Compare with the adjacent healthy tissues. Are there any indications of a bitter taste?

ETIOLOGY

Many theories have been advanced with respect to the cause of stippen. Pathologists appear to be generally agreed that it is due to extreme variations in the water-supply to the tree during the growing-season. (For a full discussion of etiology see the text or McAlpine, Bitter Pit Investigations. 1:110; 2:93-95; 4:73-75.)

Pathological Histology. The effect on the host-tissue is necrotic. Select one of the lesions next to the skin of the fruit which shows no cavity as yet. Make numerous thin sections with a sharp razor through the lesion at right angles to the surface of the fruit. Float these at once in clean water in a watch-glass. Mount one of the thin but entire sections. Cover and STUDY:—

8. The diseased cells; their collapsed walls, contents, and color as compared with the adjacent healthy cells. Treat the section with iodine-solution and examine for starch. Where found and why present?

9. The relation of the diseased cells to the vascular cells and vessels.

DRAW to show the points brought out in 8 and 9.

REPORT

1. Write a full but concise description of the symptoms of stippen and characterize briefly other fruit-spots with which it may be confused.

2. Detail a plan of procedure by which a fruit-grower may hope to reduce the stippen to a minimum in his orchard.

HYPOPLASTIC DISEASES

DOWNY MILDEW OF GRAPES

This is a common disease of both wild and cultivated grapes. It is in some years very destructive to certain varieties in American vineyards but is far more destructive to cultivated grapes in Europe. It also affects the five-leaved ivy, *Psedera quinquefolia* (L.) Green.

SYMPTOMS

This disease affects the leaves, young canes and fruit of the grape.

On the leaves. Examine the leaves of cultivated grapes provided and OBSERVE:—

1. The location, size, color and general appearance of the spots on both surfaces of the leaf.

2. Difference in the appearance of the old and young lesions, especially on the upper surface. The tissues are not directly killed, but show a brownish yellow color contrasting sharply with the green of the healthy parts. (See autochrome 1.)

3. The downy white growth covering the under surface of the lesion,—the fruit-structures of the pathogene.

4. The above characters as exhibited by the lesions on the smooth leaves of the wild grape.

Make SKETCHES of both sides of affected leaves to show the characters of the lesions as observed.

On the canes. The cane-lesions may be either local or systemic. Study the specimens showing local invasion. OBSERVE:—

5. That the lesions are usually on one side of the stem, causing it to bend or curl, the diseased area being on the outer side of the curve.

6. A slight increase in the size of the affected portion of the cane.

7. The white downy growth of the pathogene, covering the lesion in many cases.

SKETCH to show a localized stem-lesion.

Examine the specimens (illustration specimens) and photographs of systemically invaded shoots. OBSERVE:—

8. The distinct dwarfing or hypoplasia of the shoot and its organs,—the leaves and tendrils.

9. The continuous white coating of the fruiting structures of the fungus.

SKETCH a portion of a shoot to show these characters. Only very young shoots show systemic lesions.

On the fruits. Examine the specimens and photographs provided. OBSERVE:—

10. That certain of the berries in the bunch are covered with the downy white growth observed on leaf and stem-lesions.

11. That the affected berries are brown in color, in striking contrast to the green healthy fruits on the bunch. The disease is, on this

account, sometimes known as "brown rot." Compare with grape affected with anthracnose or black rot. (See illustration specimens.)

Sometimes, particularly in the older berries, the fungus does not seem to be able to send forth the white downy fruiting structures. Such berries fail to ripen and have a whitish opaque color. This character is not well seen in preserved specimens.

SKETCH a bunch of grapes to show the contrast between diseased and healthy berries.

ETIOLOGY

The fungus which causes the downy mildew is a Phycomycete known as *Plasmopara Viticola* (Berkley and Curtis) Berlese and De Toni. It is a native of America and was introduced into Europe about 1878 where it has since wrought great destruction.

Life-history. Thanks to the extensive investigations devoted to this pathogene, our knowledge of its life-history is now relatively complete.

The **Primary Cycles** are initiated shortly after growth of the host starts in the spring. The sources of the primary inoculum are in the previously diseased overwintered leaves on the ground.

Pathogenesis. Examine bits of overwintered leaves or prepared mounts of the same. SEARCH carefully for:—

12. Rather large, globose bodies,—the oospores, embedded in the tissues of the leaf. They are readily distinguished by the circular band-like appearance of the thick hyaline inner wall. These are resting-spores which serve to carry the fungus through the winter.

These germinate during wet weather in the spring in the old leaves as they lie on the ground. The study of actual germination of these spores is attended with difficulties; therefore, study separates of Gregory's article from *Phytopath.* 2:237, fig. 2. OBSERVE:—

13. The large rough oospores embedded in the disorganized leaf-tissues.

14. The slender stalk,—conidiophore, sent forth from a crack in the wall of the oospore and bearing at its tip a large egg-shaped conidium (sporangium).

COPY, fig. 2 a and b. Label fully.

When mature, this conidium is readily broken off by the breeze or splashing rain-drops and carried to young leaves or growing canes. Here in a drop of water, the conidium, a potential sporangium, germinates by the division of its protoplasm into (usually) 5-8 swarmspores, which are emitted through the papillate apex as shown in Gregory's article, fig. 3.

If viable conidia are available, attempt to germinate them, proceeding as directed by the instructor. Watch the process carefully.

Make DRAWINGS, either from observed germination or from Gregory's drawings, to show swarmspore-formation.

These swarmspores swim about by means of two flagella (Gregory's figures 4c and 7). COPY drawings to show swarmspore-germination and invasion through a stoma.

From the swarmspore-germtube, a mycelium is developed which rami-fies the tissue, passing between the cells.

Make thin tangential sections through the cortex of a diseased cane or berry. Mount in chloral hydrate to clear. OBSERVE:—

15. The large granular mycelium, fitting so closely into the intercellular spaces that its walls are not readily distinguished from those of its host-cells; septate or non-septate?

16. The numerous small globose haustoria extending through the walls of the host-cells. They push the plasma-membrane inward but do not penetrate it.

Make a DRAWING of the mycelium showing its structure and relation to the host-cells.

This mycelium soon develops special branches which are sent forth through the stomata and branching, form the conidiophores. These conidiophores constitute the downy white growth on the under surface of the lesion. Scrape some of the conidiophores from the specimen provided for this purpose. Mount in potassium hydroxide. Examine and OBSERVE:—

17. Their color, form of branching and shape of the ultimate tips or branchlets on which the conidia (sporangia) are borne. Estimate carefully the number of conidia that may be borne on each conidiophore.

18. The egg-shaped conidia lying all about in the mount. They separate from the conidiophore very readily when mature. By which end were they attached? A mount from near the margin of a young lesion may show the immature conidia still attached to the conidiophores.

Study sections through a leaf-lesion. OBSERVE:—

19. The relation of the conidiophores to the host-structures; emergence through the stomata; number from each stoma; and constriction at the stomatal opening.

Make a large DRAWING of a conidiophore with mature conidia attached to a few of the branches.

These conidia are disseminated by the wind. They fall upon leaves, fruits and stems and initiate secondary cycles.

Saprogenesis. The mycelium within the dying leaf-tissue begins the sexual formation of long-lived resting-spores to carry the pathogene through the winter.

The early stages in the development of these oospores are not easily studied in the case of *Plasmopara Viticola*, but may be readily observed in pure cultures of related phycomycetous fungi. Make mounts from the culture provided and OBSERVE:—

20. The large densely granular swollen tips of many of the mycelial branches,—oogonia or female organs. The cross-wall cutting off some of the older ones from the mycelium.

21. The much smaller granular body, also a modified mycelial tip, applied closely to the side of the oogonium,—the antheridium or male organ. From this a fertilization-tube is sent into the oogonium and a nucleus passes through to fuse with a female nucleus in the oogonium. The contents of the fertilized oogonium now round up to form a single oospore.

DRAW to show the early stages in oospore-formation.

Oospore-formation may probably begin while the mycelium is still drawing nutriment from the living host-tissues. The oospores are, however, not matured until after the tissues in which they are formed are dead. The oospores mature in the fallen leaves on the ground. Study the mature oospores provided. OBSERVE:—

22. The thin outer hyaline sack,—the old oogonial wall. Is there any trace of the old antheridium?

23. The uniformly thick hyaline inner wall surrounding the oily protoplasm,—the living part of the oospore; why oily?

24. The dark colored irregularly thickened outer wall enclosing the inner wall. This consists of worthless unused protoplasm contracted about the inner wall as a protection.

The thicker inner wall serves as a stored food-reserve for the germinating oospores. It dissolves from within as the germinating spore develops the long conidiophore with the primary conidium (already studied).

DRAW to show the structure of a mature oospore.

Secondary Cycles are initiated repeatedly during the season on leaves, stems and fruits by the conidia from primary and other secondary lesions. These conidia germinate, as do the primary conidia, by swarmspores. The secondary cycles repeat in all details the phenomena of the primary cycles. Only in the leaves, so far as known, are the cycles completed by the formation of oospores.

REPORT

1. Describe in detail two methods, one an eradicatory, the other a protective method for the control of the downy mildew of grapes, and state concisely why each should be effective.

DOWNY MILDEWS OF THE RANUNCULACEAE

There are two downy mildew diseases of the Ranunculaceae in America, the *Plasmopara* mildew of anemones and hepaticas and the *Peronospora* mildew of buttercups. While at present it is chiefly the wild species that suffer, these diseases constitute a serious menace to the development of garden-varieties from the wild species.

SYMPTOMS

The symptoms of the two diseases are very similar. The lesions are of two general sorts, localized and generalized or systemic, that is, involving the entire plant or shoot. It is chiefly the leaves that exhibit the effects of the disease.

Local lesions. Examine the affected leaves of the different hosts provided. OBSERVE:—

1. The pale-gray or brownish color of the upper surface of the affected parts, in striking contrast to the green unaffected areas, especially noticeable in the anemones; the angular shape of the lesions; to what due? Note that nearly the entire blade may be involved. SKETCH.

2. The white felty coating covering the lesions on the under surface of the anemone leaves; violet-gray on the buttercup leaves. This felt is composed of the conidiophores and conidia of the pathogene, and constitutes the most distinctive sign of these downy mildew diseases. (Compare with grape mildew, illustration specimens.) SKETCH to show the appearance of the lower surface of the lesion.

Systemic lesions. The hypoplastic or dwarfing effect of these diseases is especially well observed in the individuals which harbor the living pathogene in the rootstocks during the winter. The entire leaves from such rootstocks, at least the earlier leaves, are affected. In the specimens provided, OBSERVE:—

3. A marked dwarfing of diseased as compared with healthy leaves, especially striking in the buttercups.

4. The more greyish color of affected leaves. This is very striking in affected plants of *Ranunculus acris* L. in early spring.

5. The continuous coating of conidiophores on the lower surface. The affected leaves or portions of leaves soon die and turn black, shrivel and become brittle.

Make SKETCHES of diseased and healthy leaves to show contrast in size.

ETIOLOGY

The disease in anemones and hepaticas is caused by *Plasmopara pygmaea* (Unger) Schroeter, while *Peronospora Ficariae* Tulasne attacks various species of *Ranunculus*. They are closely related phycomycetous fungi.

Life-history. The activities of these two pathogenes throughout their life-cycles are essentially alike. They develop the same kind of organs and structures which are, however, distinctive for each parasite.

Primary Cycle. These pathogenes are obligate parasites, the period of saprogenesis being probably one of rest and of development at the expense of stored food.

Pathogenesis. There are two quite different sources of primary inoculum; one the dead overwintered leaves, diseased the previous season; and the first leaves put forth from systemically invaded plants.

(a) Inoculum from overwintered leaves. Overwintered hepatica leaves, showing last year lesions, have been cleared by special treatment and placed in alcohol in vials. Holding one of these to the light, examine with the hand-lens and OBSERVE:—

6. The great number of minute globose dark-colored oospores imbedded in the tissues. SKETCH a portion of the leaf to show these.

These germinate *in situ*, sending forth from each oospore a slender conidiophore bearing one or more conidia. Oospore-germination is not readily obtained. It probably does not differ materially from that described by Gregory for the oospores of *Plasmopara Viticola* in grape leaves. (Study Gregory's article, separate from *Phytopath.* 2:237, fig. 2). COPY and label to explain the use of these figures.

Borne by wind or splashed by rain-drops, these conidia reach the developing leaves of their respective hosts and initiate the primary infections.

(b) Inoculum from systemically invaded plants. The pathogene winters as mycelium in the rootstocks of such plants. As the first leaves develop the mycelium develops with them and shortly after the leaves unfold, sends forth numberless conidiophores through the stomata on the under surface. These conidiophores develop conidia (primary inoculum) in every respect like those produced from the oospores. Mount some of the down from the under surface of a systemically invaded leaf. OBSERVE:—

7. The numerous globose or egg-shaped conidia; their thin walls and densely granular contents. DRAW. Carried by the slightest breeze, these conidia fall upon young leaves of their host-plants and, if moisture is present, germinate and infect the plants.

Conidia, whether produced from oospores or from mycelium from within the living leaf, germinate in the same way. If they are conidia of *Plasmopara* they form swarmspores. If they are conidia of *Peronospora* they germinate by a germtube. If viable conidia of either or both are available, study germination and DRAW to show the structures produced or COPY drawings provided by the instructor.

Mycelium is rapidly developed from the germ tubes of conidia or swarmspores and spreads through the tissues.

Make thin sections of diseased leaves of anemone or hepatica (if dry leaves, mount sections in potassium hydroxide) and study carefully to LOCATE:—

8. The mycelium. Is it intercellular or intracellular? Are haustoria present; what form?

9. Mycelial branches projecting forth through the stomata; how many through each stoma? The form of these conidiophores is best studied in material scraped from the under surface of a lesion. Scrape a bit of the white felt from the under surface of one of these lesions on anemone or hepatica leaves, mount in potassium hydroxide and examine with the low-power. OBSERVE:—

10. The short, rather stout, scarcely branched conidiophores. Do not confuse them with the long pointed thick-walled leaf-hairs. It is from the unusual shortness of the conidiophores that this fungus gets

its specific name, *pygmaea*. It has the pygmy conidiophores among the species of *Plasmopara*. Make a mount of *Peronospora Ficariae* from *Ranunculus* leaves provided, along with some of *P. pygmaea* in the same drop of water so that they can be compared as to size, form and branching. LOCATE:—

11. A single entire conidiophore of each species well isolated in the mount. Study it carefully as to structure, thickness of walls, branching and arrangement of ultimate branchlets on which the conidia are borne. Make a large SKETCH of an entire conidiophore of each. Try to find in the mount a conidiophore with conidia still attached. Mounts from young leaves or the margins of lesions will often give conidiophores with conidia attached. If one can be found, study a branch to determine the manner of attachment of the conidia.

Study the mature conidia scattered through the mount. OBSERVE:—

12. Their form; the slightly raised apical papilla to be discerned on some of them; the thin wall and granular contents. DRAW to show the form of the conidia of the two pathogenes.

These conidia, produced in great abundance, are carried by the wind to healthy plants during rainy weather, initiating secondary cycles.

Make an enlarged DRAWING of a section through a leaf of one of the hosts to show the pathogene-structures within and without the leaf.

Saprogenesis. As the diseased tissue begins to die, the mycelium of the pathogene produces branches, the tips of which swell and round up between the cells to form oogonia and antheridia. Fertilization takes place and before the dead and fallen leaves begin to disintegrate, oospores are matured.

(To study the sexual structures and development of the oospore, follow the procedure outlined under Downy Mildew of Grapes on p. 111, No. 20 and 21.)

Oospores of *Plasmopara pygmaea* are produced in great abundance in leaf-lesions in *Hepatica triloba* as already observed under No. 6. To understand the structure of the mature oospores and their relation to the tissues, examine prepared slides and make mounts from leaf-tissue macerated in potassium hydroxide. OBSERVE:—

13. The numerous dark-brown bodies imbedded in the tissues.

14. The oospore proper, with its uniformly thick smooth hyaline inner wall, and its outer brown wall, irregular in thickness; enclosing the oospore, the transparent thin old oogonial wall usually collapsed tightly against the outside of the oospore; remnants of empty hyphae. As these oospores are nearly mature, no trace of antheridia will probably be found.

DRAW carefully to show the structure of a mature oospore, much enlarged.

These oospores germinate in the spring as they lie in the old leaves on the ground, each giving rise to a conidiophore with conidia and so provide the primary inoculum as already seen.

Secondary Cycles are initiated by the conidia produced during the primary cycle. Ordinarily, as in the case of the primary lesions, local lesions result. In the case of the secondary cycles initiated late in the season, the mycelium arising from the germtube may, instead of causing a local lesion, spread throughout the stem and root of the plant without killing it. It becomes perennially associated with the tissues of the living host. It grows up into the new leaves put forth in the spring and sends

forth from their entire under surfaces, conidiophores bearing conidia. Such invaded plants are said to be suffering from "systemic infection". (See text.)

Systemic infection is very common in the case of *Peronospora Ficariae* in the buttercup, *Ranunculus acris*. Examine one of the diseased leaves of *R. acris*. NOTE:—

15. That the conidiophores cover the entire under surface of the leaves. Where this occurs, one may be quite sure it is a case of "systemic infection" and not a local lesion.

If fresh material is available, make sections through some part of systemically infected plants (crown or rootstock). Stain with methyl blue, wash thoroughly, cover and locate the mycelium in the tissues; inter- or intracellular? Haustoria? DRAW.

The mycelium continues to live in the gradually weakening host, producing one crop of conidia each season from which primary infections, local or systemic, in character may arise. It eventually perishes with the host.

REPORT

1. If a gardener discovered some of his perennials to be suffering from systemic infection, what methods of control should he employ? Why? If all the infections are local, what should be the treatment? Why?

2. Show in a graphic diagram the life-cycles of either of the pathogenes studied in this exercise.

POWDERY MILDEWS OF FLORISTS' CROPS

Powdery mildew diseases frequently affect various ornamental plants of greenhouse and garden. They are sometimes very destructive and commonly troublesome. Among such plants which most often suffer from the powdery mildews are roses, phloxes, sweet peas, willows, hawthorns, lilacs, honeysuckles, bittersweet and dogwoods.

SYMPTOMS

The leaves are usually the organs affected, although stems, blossoms and even fruits may be diseased. Powdery mildews are usually most common and conspicuous in gardens and borders toward the latter part of the season. The white mealy coating which is formed on the affected organs is usually very striking. The minute black perithecia of the pathogene frequently appear in great numbers late in the summer or early autumn, in some cases standing out sharply against the white mycelial mats on which they rest. Where the mycelium is sparse or webby, the black perithecia may not be easily detected. In many cases they are but rarely formed. A tendency to stunt or dwarf the host is commonly to be observed. This is much more striking in some cases than in others.

On the rose. Examine the diseased shoots provided. OBSERVE:—

1. The white felt, covering large areas on the canes and running out over the thorns; in some cases localized about the base of large thorns.
2. The powdery and less felty character of the white coating of the leaves. Which surface is affected?
3. The curling and dwarfing effect on the leaves, especially marked in hothouse-roses and in ramblers.
4. The abnormal coloration sometimes exhibited by the leaf under and about the mildewed spot.
5. The dwarfing of the entire tips or branches of some shoots, most frequently observed in ramblers. This results from bud-infection explained later.
6. The white mycelial felt, coating young buds and hips. The buds are often so stunted that they fail to open or the affected hips are dwarfed and do not ripen.

Make DRAWINGS to show the symptoms exhibited in the material studied.

On phlox. All above-ground parts of this host are likely to be affected. The mildew-spots are most prominent on the leaves. Examine the specimens provided. OBSERVE:—

7. The felty white mycelial patches on the leaves. How do the patches on the two surfaces of the leaf differ?
8. The purple coloration often developed beneath and about the spots.
9. The yellowish color of the mycelial mat in the older patches and their tendency to coalesce and cover the larger part of the leaf-surface.
10. The brown centers of many of the spots due, as may be seen with the hand-lens, to the perithecial fruit-bodies of the pathogene.
11. The mycelial patches on stems and inflorescence; less felty, often hardly discernible but usually covered with the brown perithecia.

12. The dwarfing effect on the inflorescence. Flowering is often partially or entirely prevented.

Make a series of DRAWINGS to show the symptoms exhibited by mildewed phlox.

On peas. Both sweet peas and garden- or field-peas are affected. Examine the material provided and NOTE:—

13. That the mycelial coating spreads almost uniformly over the entire leaf-surface, stems and pods.

14. That it is much thinner than that on rose or phlox, and is web-like instead of felty.

15. That there is little difference in the character of the mycelial coating on the upper and lower surfaces of the leaf.

16. The general effect on the growth of the plant. Is dwarfing marked? Peas are usually affected late after growth is largely completed.

17. The minute black perithecia in groups here and there in the mycelial web; not prominent; usually not found on sweet peas.

Make DRAWINGS to show the powdery mildew on peas.

On lilacs. The powdery mildew on the lilac is so common as to be almost always found on lilacs wherever grown and every year. The leaves are the organs affected. Examine the material provided and OBSERVE:—

18. The character of the mycelial coating. On which surface of the leaf does it develop?

19. The minute black perithecia; their arrangement and distribution on the leaf.

20. Any evidences of pathological effects on the leaves.

Make DRAWINGS to show the symptoms of the mildew on lilac leaves.

On bittersweet. This mildew is not only common on *Celastrus scandens* L. but affects the foliage of many shrubs and trees. Examine the leaves provided and OBSERVE:—

21. The size and location of the spots; the habit which this particular mildew-pathogene has, of sending mycelial branches into the leaf-tissues through the stomata, is responsible for the location of the spot.

22. The chlorotic effect on the leaf-tissue beneath the mildewed area as evidenced through the upper surface.

23. The character of the superficial mycelial growth.

24. The comparatively large and numerous perithecia in all stages of development, the younger ones smaller and brown or yellow in color.

DRAW to show a leaf with a mildewed spot.

ETIOLOGY

Powdery mildew pathogenes all belong to the Erysiphaceae, a family of ascomycetous fungi. They are characterized among other things by their habit of growing externally over the surface of their hosts. They attach themselves by means of short haustoria sent into the epidermal cells. One or two species are known to send intercellular hyphae through the stomata into the tissues. The diseases above studied and their respective pathogenes are:—the powdery mildew,

of rose, caused by *Sphaerotheca pannosa* (Wallroth) Léveillé;

of phlox, caused by *Erysiphe Cichoracearum* DeCandolle;

of peas, caused by *Erysiphe Polygoni* DeCandolle;
of lilac, caused by *Microsphaera Alni* (Wallroth) Winter;
of bittersweet, caused by *Phyllactinia Corylea* Karsten. Besides the genera above represented, two more, *Uncinula* and *Podosphaera* are known, species of which occur on trees or shrubs of the yard and garden. Examples:—*Podosphaera Oxyacanthae* (DeCandolle) de Bary, on species of *Crataegus*, *Prunus*, *Spirea* and others; *Uncinula Salicis* (DeCandolle) Winter, on species of *Salix* and *Populus*.

Life-history. The powdery mildew fungi exhibit such similarity in structure and life-habits that the same outline will serve for the study of the life-history of any of them. From this point the student will follow the outline as given for the Powdery Mildews of Trees and Fruits, p. 125, including the subject designated for the report.

POWDERY MILDEW OF CEREALS AND GRASSES

This is a very common and sometimes serious disease of wheat, rye and other cereals. It is also to be found commonly on various wild grasses, especially species of the genus *Poa*.

SYMPTOMS

The powdery mildews are detected chiefly by the pathogene-structures developed upon the exterior of the host. There are also some accompanying affects or symptoms exhibited by the host. The lesions are confined largely to the leaves and leaf-sheaths. In the material provided, OBSERVE:—

1. The densely matted white, sometimes brownish, mycelial patches on the surface of the leaf (upper and lower). In the fresh condition these patches are powdery due to the abundance of conidia, hence the name, powdery mildew.

2. On the wheat leaves especially, the minute black bodies buried in the mycelial mass, usually most abundant at the center of the spot. These are the perithecia of the pathogene.

3. The effect on the tissues of the leaf beneath and about the mycelial mat. Note that in some cases the entire leaf has turned brown and died.

Make SKETCHES showing these symptoms.

Where the attack is severe, there is a dwarfing of the heads or a shriveling of the maturing grains, or both. If material is available, study and compare with healthy heads and grains. Make SKETCHES to show the comparison.

Examine the illustration specimens of powdery mildews on various hosts provided and NOTE:—

4. The marked similarity in the symptoms exhibited by all of them. Select one of the specimens and SKETCH. Label fully.

ETIOLOGY

The powdery mildew of cereals and grasses is caused by *Erysiphe graminis* DeCandolle, the conidial form of which is known as *Oidium monilioides* Link. Like all the other powdery mildew pathogens it is an ascomycetous fungus belonging to the family, Erysiphaceae. They all develop externally upon the surface of their host except for short haustoria sent into the epidermal cells or, in the case of one or two species, an occasional mycelial thread sent through stomata into the tissues.

Life-history. This pathogene exhibits during its life-cycles all of the characteristic structures of the powdery mildew fungi.

The **Primary Cycles** are initiated in the spring. The sources of inoculum are the overwintered perithecia on the leaves and stubble of the host.

Pathogenesis. Remove some of the minute black perithecia from the mycelial mats on the old overwintered host-leaves. Crush in a drop of water by pressing on the cover-glass. OBSERVE:—

5. The large ellipsoidal ascospores forced from the perithecia; some still in the asci. Determine the number of asci in each peri-

thecium. These constitute the primary inoculum. DRAW to show the form and structure of these ascospores.

When the ascospores are mature and the perithecium is thoroughly wetted, it cracks open and the ascospores are forcibly discharged. Borne by the wind, they fall upon the growing leaves of the host and germinate. If viable ascospores are available, study spore-germination as seen in the slides provided. DRAW, or COPY illustrations provided by the instructor.

As soon as this germtube has developed a food-relation with the host by means of haustoria in the epidermal cells, a mycelium begins to develop, branching and spreading in all directions over the leaf-surface. Examine, under the binocular microscope, one of the white mycelial mats on the diseased green leaves (fresh or preserved) provided and OBSERVE:—

6. The tangle of silver-white hyphae with long spreading branches about the margin of the lesion.

7. The numerous erect chains of conidia borne on short conidiophores. Many of these conidia have fallen off and give the mealy appearance to the mildew-spots.

Scrape the mycelial mass from the surface of the leaf. Mount in water, cover and EXAMINE:—

8. Conidia; large ellipsoidal, flattened slightly at the ends; their thin hyaline walls and densely granular protoplasm.

9. Conidiophores; short, with a swollen base just above the point of attachment to the mycelium. Try to find a conidiophore with several conidia still attached.

10. The mycelium; very crooked and much broken in scraping from the leaf; septate or nonseptate?

As the mycelium spreads over the surface of the leaf it sends minute branches through the outer cell-wall of the epidermal cells. This branch enlarges and branches within the cell to form the finger-like haustoria. Study these in the slides provided or from the drawings by Smith, Bot. Gaz. 29, pl. XI and XII.

Make a composite DRAWING to show a cross-section of the epidermal cells of the host with haustoria, mycelium, conidiophores and conidia in normal relation to each other.

These conidia break off at the top of the chain as fast as matured and, scattered by the wind, initiate secondary cycles.

Conidia continue to be produced for a time by the mycelium on the primary lesions. As the primary lesions are largely on the young or seedling-leaves of the host, the mycelium probably perishes along with the young leaf before the sexual fruit-bodies can be developed. These appear later on the mycelium of the secondary cycles. There is, therefore, no saprogenic phase in the primary cycles.

The **Secondary Cycles** are initiated by conidia from the primary cycles.

Pathogenesis. The fungus exhibits the same conidial structures in the secondary cycles as those just studied. As the host-tissues begin to mature, conidial production ceases, and the mycelium begins the development of sexual structures. The detailed study of the development of these structures cannot well be followed out in this laboratory exercise. (See de Bary, Morphology and Biology of the Fungi, p. 226, fig. 107.)

The structure of the perithecium may, however, be readily studied. Examine the specimens provided under the binocular microscope. OBSERVE:—

11. The much coarser and more densely matted mycelium; not pure white but yellowish or brownish.

12. The globose perithecia of varying sizes and colors enmeshed in the mycelial mat.

SKETCH to show the appearance under the binocular microscope.

Saprogenesis. The perithecia usually begin to appear while the leaf is still green, but do not mature their ascospores until the leaves die and are overwintered. The perithecia on the dead leaves on the ground pass the winter in an inactive condition. The rains and warm weather of spring cause the asci to mature their ascospores.

To simulate the spring conditions, some of the leaves bearing immature perithecia have been placed in warm water for several days. They are now mature.

Remove some of the perithecia to a slide in water and cover with a cover-glass. Crush by pressing firmly on the cover with the handle of a scalpel, while watching the perithecia under low-power. What comes out of the perithecium? How many? What is the character of their contents? Does the perithecium have an ostiolum?

DRAW to show a perithecium with its contents. Show the structure of the perithecial wall and mycelial attachment.

With the maturity and discharge of the ascospores, the secondary cycles are completed.

REPORT

1. Explain the significance of biologic strains in *E. graminis* DC., with respect to control.

2. Discuss eradication measures in the control of powdery mildews.

POWDERY MILDEWS OF TREES AND FRUITS

Powdery mildew diseases have been reported on about 1500 species of wild and cultivated plants. Several of them are frequently very injurious to fruit-trees and sometimes to shade- and forest-trees.

SYMPTOMS

Leaves and young shoots are usually the parts of the host that are affected. Throughout the latter part of the summer the powdery mildews are conspicuous and give to the infected parts of the host-plant a whitish, mealy or dusty appearance, due partly to the superficial white web-like mycelium of the pathogene, and partly to the presence of myriads of rapidly formed white conidia. Later in the summer, and in the autumn, there usually appears, on the affected parts, the small black spherical perithecia of the sexual stage. In the autumn these are more in evidence than the whitish growth; the latter often disappears. These signs, the fruit-bodies of the pathogene, are usually the most striking evidences of the disease. Definite and characteristic symptoms resulting from the effects of the pathogene on the host are, however, not wanting in many cases.

On the cherry. On this host, the leaves and twigs show the effects of the disease. Examine the specimens and photographs provided and OBSERVE:—

1. The upward rolling or curling of the leaf-blades parallel with the mid-rib.
2. The shorter and thicker internodes of diseased twigs as compared with healthy ones.
3. The weft-like coating of fine white hyphae on the leaves, especially on the under surface. The mycelium of most of the powdery mildew pathogenes is entirely superficial.
4. Patches of the mycelial weft dotted with the minute black perithecia of the pathogene.

Make a DRAWING of a diseased twig to show the signs and symptoms exhibited.

On the apple. The young leaves, flower clusters and shoots are affected. Examine the diseased shoots and U. S. Agr. Dept. Bul. 120, pl. I and VI, provided. OBSERVE:—

5. The marked hypoplastic effect exhibited in the dwarfed foliage.
6. The mealy white coating of the diseased leaves,—conidia and mycelium of the fungus.
7. The thick felty mycelial coating on the watersprouts collected in the autumn. Note the dirty white or brownish tinge as compared with the pure white of the growth on the leaves.
8. The minute perithecia, more or less embedded in the mycelial mat on the shoots.

Make DRAWINGS to show the appearance of affected leaves and watersprouts.

On the peach. Not only the leaves and twigs, but also the fruits of the peach, are subject to the disease. Study the specimens and photographs provided. OBSERVE:—

9. That the leaves are narrow, have failed to expand and are curled and deformed. Chlorophyll is not developed properly and the leaves show red and yellow tints. Defoliation often results from a severe infection.

10. The effect on the more succulent upper parts of the twigs.

11. The felty character of the superficial mycelium which forms in white patches over affected leaves and twigs. This thick mycelial felt persists on the twigs after the leaves fall, becoming a dirty gray-brown in color.

12. The more or less circular white patches of mycelium on the fruit. When very young fruits are affected they soon fall.

Perithecial fruit-bodies rarely appear.

Make DRAWINGS to show the symptoms exhibited by the powdery mildew of the peach.

On the grape. This disease is more destructive and develops more typically in the Pacific Coast regions than in eastern United States. All herbaceous parts of the host are affected. Examine the specimens provided. OBSERVE:—

13. The whitish patches on the upper and lower surfaces of the leaf. How do they compare with the spots of the downy mildew? These spots may spread to form a whitish, mealy coating over the greater part of the leaf-surface. Badly diseased leaves may curl upwards about the edges.

14. The small black perithecia scattered over affected areas on the leaves.

15. The diseased canes. They also show the superficial greyish white patches of mycelium beneath which the tissues of the cane soon darken, making it spotted. (See California Bul. 186, fig. 3.)

16. The diseased berries. (See California Bul. 186, fig. 4.) Blossoms and young fruits when affected, quickly fall. The disease may often cause shelling of the large green berries when the fruit-pedicles are affected.

Make DRAWINGS from specimens and illustrations to show the symptoms of the powdery mildew of grapes.

On the gooseberry. In case of the gooseberry mildew, it is chiefly the young shoots and the fruits that are affected. In the material provided, OBSERVE:—

17. The mycelial mats coating the shoots; color, thickness and distribution.

18. That the superficial mycelium spreads out over the leaves. How is the growth of leaf and stem affected?

19. The character of the mycelial patches on the fruits. Does the growth and development of the fruit appear to be affected?

20. The black perithecia of the pathogene embedded in the mycelial felt.

This same gooseberry mildew may sometimes seriously affect some varieties of currants, as may be seen in the specimens provided.

Make DRAWINGS of mildewed shoots and fruits of gooseberry or currant.

On the chestnut. The leaves are the organs affected. This powdery mildew affects not only chestnut but a great variety of trees, shrubs and woody vines. Examine the diseased leaves provided. OBSERVE:—

21. That the mildew-patches are confined to the under surface of the leaf.

22. The rather thick felty character of the mycelial growth; color and extent.

23. The numerous perithecia sitting on the mycelial mat, not embedded in it; larger than the perithecia of the other mildew-pathogenes observed; some of them immature as indicated by their small size and light color.

24. Any evidence of injury showing on the upper surface opposite the mildew-spot.

DRAW a leaf showing the characters of the mildewed areas.

On the willow. Many species of willow and also poplars are affected. The leaves are usually the only organs involved. In the specimens provided, OBSERVE:—

25. The location and distribution of the spots; hypophyllous or epiphyllous?

26. The characteristic dense white mycelial border of the spot with darker center, especially in older lesions, due to the numerous black perithecia. Willows are often defoliated by this disease.

27. Any evidence of injury to the tissues.

DRAW a willow leaf showing the mildew-spots.

ETIOLOGY

The powdery mildew diseases are all caused by species of ascomycetous fungi belonging to a single family, the Erysiphaceae. Each disease above studied is caused by a different species of pathogene. Even within some of these species there are doubtless biologic species. The diseases studied with their respective pathogenes are as follows:—the powdery mildew,

of cherry, caused by *Podosphaera Oxycanthae* (DeCandolle) de Bary;

of apple, caused by *Podosphaera leucotricha* (Ellis and Everhart)

Salmon;

of peach, caused by *Sphaerotheca pannosa* (Wallroth) Lév., var. *Persicae* Woronichin;

of grape, caused by *Uncinula necator* (Schweinitz) Burrill;

of gooseberry and currant, caused by *Sphaerotheca Mors-uvae* (Schweinitz) Berkley and Curtis;

of chestnut, caused by *Phyllactinia Corylea* Karsten;

of willow, caused by *Uncinula Salicis* (DeCandolle) Winter.

Besides the four genera represented, *Podosphaera*, *Sphaerotheca*, *Uncinula* and *Phyllactinia*, two more are known, species of which are very common in this country. Examples:—*Erysiphe graminis* DeCandolle, on grasses and cereals and *Microsphaera Alni* (Wallroth) Winter, on lilac. (See demonstration specimens.)

Life-history. These powdery mildew pathogenes all exhibit such similarity in their structures and life-habits that the following outline should serve for the study of any of them. As they are strictly obligate parasites, saprogenesis is a period of rest or a maturation-process carried on at the expense of stored food-reserves.

The **Primary Cycles** are initiated in the spring or early summer. The primary inoculum is usually the ascospores from overwintered perithecia. In some cases, as in that of *S. pannosa* (Wallr.) Lév., or of *Podo-*

sphaera leucotricha (E. and E.) Salm., the mycelium may winter in a semi-dormant condition within the host-buds on the embryonic leaves. As these buds open and the leaves and shoots develop in the spring, the mycelium grows out over them and produces conidia, which, scattered to healthy shoots, may initiate primary infections. (See U. S. Agr. Dept. Bul. 120:9-10.)

Ascospores are, however, responsible for the primary infections in the case of most powdery mildews and often even in those in which hibernating mycelium is known.

Pathogenesis. Remove with the scalpel, several perithecia of one of the pathogenes indicated by the instructor. Mount in a drop of potassium hydroxide. Cover and, while examining it under the low-power, crush the perithecium by gently pressing on the cover-glass with the point of the scalpel. OBSERVE:—

25. The irregular crack in the perithecium from which one or more asci are forced out. How many in this case?

26. The large ellipsoidal ascospores usually remaining within the asci; number in each ascus; color; contents.

27. The thin place in the wall of the ascus at the apex. At maturity this dissolves as the perithecium cracks open and the ascospores are forcibly ejected into the air.

Make a diagrammatic DRAWING of a cracked perithecium with protruding asci discharging spores.

Borne by the breeze, these ascospores fall upon young shoots or leaves of the host and germinate. The germ-tube grows out over the surface and sends a haustorium into the epidermal cells, from which point the branching mycelium develops. Study germinating ascospores or illustrations provided, especially Bot. Gaz. 29, pl. XI-XII. Make a diagrammatic DRAWING of a germinating ascospore with haustorium.

To study the mycelium, scrape some from the surface of young spots; tease apart in water or potassium hydroxide; cover and examine. OBSERVE:—

28. The broken pieces of irregular, branched mycelium; septa, color and contents.

29. The large, ellipsoidal or ovoid conidia; color and contents. Several may be found attached in a chain or even still on the conidiophore.

30. The short conidiophores,—upright branches which bear the conidia in a chain. (See the demonstration specimen under the binocular microscope; or U. S. Agr. Dept. Bul. 120, fig. 2.)

Make a diagrammatic DRAWING to show the vegetative structures in position on the leaf-surface.

The conidia are produced in great quantities. They give the powdery appearance so characteristic of these mildews. They are scattered by the wind and initiate secondary cycles.

After a period of conidial production, the mycelium begins to form the sexual fruit-bodies,—the perithecia. These usually begin to develop toward the end of the growing-season but before the leaves fall. In some cases as in the apple mildew-pathogene, *P. leucotricha*, the perithecia are formed on the twigs. The detailed study of the development of the sexual bodies and the formation of the perithecium cannot well be undertaken in this exercise. (See de Bary, Morphology and Biology of the Fungi. p. 226, fig. 107.)

Saprogenesis. The perithecia do not complete their development until the late autumn, usually after the leaves fall. Some do not mature until the following spring. They develop and mature at the expense of food gathered by the parasitic mycelium. Select from the material provided, representatives of two or more genera. Study the perithecia. OBSERVE:—

31. The shape, color and structure of the perithecium; the nature of the appendages. DRAW one perithecium with its appendages. Study species from the three remaining genera, OUTLINE the perithecia, but DRAW carefully a typical appendage for each. (See Salmon, Monograph of the Erysiphaceae, pl. 1-7; also Burrill, Parasitic Fungi of Illinois, p. 395-397.) Crush the perithecium in each case and examine. In this connection the following key:—

A. Perithecia with one ascus.

1. Appendages simple, undivided at tip SPHAEROTHECA
2. Appendages once or more dichotomously divided at the tip
 PODOSPHERA

B. Perithecia with several asci.

1. Appendages never more than slightly swollen at the base.
 - a. Appendages simple, or irregularly branched; without tip peculiarities ERYSIPIHE
 - b. Appendages once or more dichotomously branched at the tip MICROSPHERA
 - c. Appendages spirally rolled at the tip UNCINULA
2. Appendages swollen at the base so as to form an enlarged plate . . .
 PHYLLACTINIA

With ascospore-discharge in the spring and early summer, saprogenesis of the primary cycle ends.

The **Secondary Cycles** are, as pointed out above, initiated by the conidia from the primary lesions. They normally repeat in all details the phenomena and structures exhibited in primary cycles. Secondary cycles may repeatedly initiate other secondary cycles during the season.

REPORT

1. Discuss control of one of the powdery mildews which may be selected, treating the subject under the headings of eradication and protection and explain how the life-habits of the pathogene make effective the measures described.

APPLE SCAB

Of all the diseases of the apple, this is the most common and best known to the growers. It is the one fungus disease for which they spray. It is world wide, occurring practically wherever the apple is grown. While there is a marked difference in the susceptibility of varieties, all will suffer some under conditions especially favorable to the fungus causing the disease. The scab of the pear is very similar in its symptoms to the apple scab but is caused by a distinct but closely related species of fungus.

SYMPTOMS

The disease affects the leaves, flowers, fruit and rarely the twigs. Material is provided showing the different symptoms.

On the leaves. The first evidence of the disease in the spring is on the unfolding leaves. The scab-spots usually appear first on the lower surface, but later new spots appear on the upper surface. Examine the leaves provided and OBSERVE:—

1. The size, form and character of the spot. The radiating character of the lesion. To what due?

2. The character of the injury to the leaf. Does the injury show on the surface opposite the spot?

3. The difference in the character of the upper and under surface of the leaf itself; and of the scab-spots on the two surfaces of the leaf.

4. The variations in the character of the scab-spots on different leaves. (Compare Cornell Bul. 335, pl. I.)

Make DRAWINGS to show the characters of the scab-spots on the upper and under surfaces of the leaves.

On the flowers. The disease may appear on the pedicle and calyx of the flower before the petals fall and may be severe enough to prevent the setting of the fruits. (See Cornell Bul. 335, pl. VII.) In the material provided, OBSERVE:—

5. The location, form and character of the scab-spots and the effect on the flower. Make DRAWINGS to show these symptoms.

On the fruit. Where the infection of the calyx is not severe enough to prevent the fruit from setting, the apple as it grows shows the enlarging scab-spots. These become very evident as the season advances. In the young apples provided, OBSERVE:—

6. The black scab-spots. Their form, size, and effect on the fruit. To what region on the apple are they largely confined?

7. The felty black center of the spot. In some cases, this felt has disappeared and the center of the spot is hard, reddish brown and often cracked. Note the scab-spots on the mature apple provided.

8. The papery rim bordering the spot; best seen in the younger spots. This consists of the cuticle of the apple which has been loosened by the fungus as it spreads out from the center of the spot. (See Cornell Bul. 335, pl. VI, fig. 2.)

Make DRAWINGS to show the points brought out in 6, 7 and 8.

Sometimes these spots cause a dwarfing of the apple on the affected side. (See demonstration specimens.)

On the twigs. This form of the disease appears to be rare except on certain varieties like the Lady apple. In Maine and other very northerly apple sections it is not uncommon on other varieties. In the material provided, OBSERVE:—

9. The rough blistered character of the lesions, confined to the growth of the current year. DRAW.

ETIOLOGY

The apple scab is caused by the conidial stage, *Fusicladium dendriticum* (Wallroth) Fuckel, of an ascomycetous fungus known as *Venturia inæqualis* (Cooke) Winter (= *V. Pomi* (Fries) Winter). It belongs to that group of the ascomycetes known as Pyrenomycetes which have their asci enclosed in a more or less globose fruit-body, called a perithecium.

Life-history. It is in the conidial stage that this fungus exhibits its parasitic nature. It lives superficially on the host or nearly so, simply prying off the cuticle or upper part of the epidermal cells, and applying its mycelium closely to the host-tissues.

The **Primary Cycle** is initiated by ascospores from perithecia in old leaves on the ground.

Pathogenesis. Crush in potassium hydroxide a bit of the old leaf provided, examine and OBSERVE:—

10. The 2-celled olivaceous ascospores. DRAW. These ascospores are shot from the ascus which protrudes through the ostiolum of the perithecium. Ascospores are discharged only during rains or very moist weather in spring. They are carried to the young leaves just emerging from the buds. Here they germinate.

Study Cornell Bul. 335, pl. IX and X, and OBSERVE:—

11. That but one cell of the ascospore gives rise to a germ-tube. Which cell? DRAW to show three stages in the development of the germ-tube. This germ-tube pierces the cuticle of the leaf or young fruit and initiates the scab-spot. COPY Cornell Bul. 335, fig. 185. Again examine the scab-spots on the leaf and with the hand-lens or low-power, MAKE OUT:—

12. The radiating branched mycelial threads. Why do they radiate from a center? Make an enlarged DRAWING of a scab-spot to show this habit of the mycelium.

Study the mycelium, as shown in the prepared slides of apple leaves which have been cooked in potassium hydroxide and the epidermis, bearing the fungus, peeled off. OBSERVE:—

13. The form, size and septation of the mycelium. Its color and method of branching. No haustoria are sent into the host-cells and the mycelium does not at this stage penetrate beyond the epidermal cells.

14. That the conidiophores arise in clusters or singly from this spreading mycelium. Note their form, length and color.

15. The conidia lying about, which have been broken off from the conidiophores; their form, size and color and point where they were attached to the conidiophore. Several conidia may be produced from near the same point on a conidiophore. How? See if you can find a conidiophore which shows this.

Make DRAWINGS to show the mycelium, conidiophores and conidia in their proper relations to each other and to the host.

Scrape some of the conidia from the scab-spot on an apple. Mount and study. Compare them with those found in the prepared slide. They are sometimes 1-septate.

Make DRAWINGS to show the variations in size, form and septation of the conidia.

Saprogenesis. The mycelium in the primary lesions on the leaves continues to produce conidia until the leaves fall. The fungus, which has up to that time lived practically on the surface, now sends new branches of the mycelium throughout the dead tissues of the leaf. From this mycelium are formed the globose perithecia. They are formed just beneath the epidermis of the lower or upper surface of the leaf, being most abundant near the surface facing upward as the leaf lies on the ground. Examine the old apple leaves provided and OBSERVE:—

16. The evidence of the old scab-spots of the summer.

17. With the hand-lens the pimple-like perithecia scattered over both surfaces of the leaf just beneath the epidermis. Note the color, size, distribution, relation to the old scab-spots, and relative abundance on two sides of the leaf.

Make a DRAWING of a portion of the leaf to show the perithecia as seen with the hand-lens.

With the scalpel, cut from the leaf a small square of tissue showing an abundance of the perithecia. Place between pieces of pith and with a razor make freehand sections through the perithecia. Mount and study. OBSERVE:—

18. The shrunken dead condition of the host-tissue.

19. The imbedded perithecia.

20. The mycelium of the parasite throughout the tissues of the leaf; its form and character.

21. The structure of the perithecium. (Best made out in the stained sections.) The walls, their relation to the host-tissues, form, size, mouth or ostiolium and asci, with ascospores. How many ascospores in an ascus?

Make a DRAWING to show the structure and contents of the perithecium and its relation to the host-tissues.

The perithecia begin to develop in the autumn but do not mature their ascospores until spring at the time when the apple leaves and blossoms are unfolding.

Secondary Cycles are initiated by the conidia from the lesions on leaves and fruits on the tree. Aside from their conidial origin, the secondary cycles which are continuously initiated throughout the season, do not differ from the primary.

22. Study the germinating conidia. DRAW.

The fungus forms on the fruit a thick stroma-like growth from the upper surface of which arise the short conidiophores bearing the conidia. In order to study this structure and the relation of the parasite to the tissue of the fruit, make thin cross-sections through the scab-spots on the half-grown fruits. OBSERVE:—

23. The thick stroma-like mass of the mycelium. Note its pseudoparenchymatous structure; thickest toward the center of the spot.

24. The short conidiophores with conidia. Find conidiophores that show different stages in their formation and development.

Make a DRAWING to show the structures observed.

Pathological Histology. Study the prepared sections made through a scab-spot on the fruit. OBSERVE:—

25. That the fungus works in the cuticle above the epidermal cells, splitting off and forming the papery rim of cuticle that covers the advancing margin of the fungous growth. This is best made out at the very edge of the lesion.

26. The suberization of the cells of the host just under the fungous stroma, indicated by the browning of the cell-walls. Why does this occur?

REPORT

1. Give a concise account of the life history of *Venturia in-æqualis* (Cke.) Wint. Arrange your data under and show the heads and subheads used in this outline.

ERGOT OF RYE

This disease is one of the earliest known and most frequently studied of ascomycetous diseases of cereals; due doubtless to the poisonous effects of the sclerotia of the pathogene on man and beast, rather than to the reduction of crop-yields which it causes. The host of greatest economic importance is rye.

SYMPTOMS

The heads alone show the disease which is exhibited chiefly by the presence of the pathogene structures. In the early stages, the pathogene produces a honeydew which exudes and runs down over the young spikelets. Examine the dried specimens labeled "honeydew-stage," and photograph 1, fig. 2, 4 and 22, and photograph 9. OBSERVE:—

1. Smudgy traces of the now dried honeydew. This honeydew is often difficult to see in dried specimens but in fresh specimens it is often very copious and sticky.

Carefully dissect away the glumes in the smudgy region. OBSERVE:—

2. The whitish kernels with corrugated surfaces. Compare with normal kernels from the same or healthy heads of the same age, as to size and color.

3. The glumes about the diseased kernels. Are they diseased? Compare with healthy.

It is from these diseased kernels that the honeydew oozes, but in the dried specimens, there is evident only the whitish covering of the dark diseased kernels.

Make a DRAWING of the organs of a single diseased and healthy spikelet to bring out these characters.

Later there is developed the ergot stage. Examine the specimens and photographs provided and OBSERVE:—

4. The black spur-like bodies projecting here and there from between the glumes,—the ergots; average number from each head. Examine illustration specimens of other ergot-infested grasses. DRAW a head of rye and a head of one other grass showing the ergots.

Examine the loose ergots provided. NOTE:—

5. The variation in size and shape; consistency; the surface, often checked and cracked. These ergots are sclerotia of the pathogene developed, in place of the host-kernel, from the structures of the Sphacelia stage, the shrunken remnant of which may often be observed still clinging to the tip of the sclerotium.

6. On breaking one open, the light colored interior. These ergots are poisonous.

Make an enlarged detailed DRAWING of two or more of the ergots.

ETIOLOGY

The ergot is produced by the ascomycetous fungus, *Claviceps purpurea* (Fries) Tulasne, the conidial or honeydew-stage of which was given the name, *Sphacelia segetum* Léveillé.

Life-history. Several careful researches have resulted in a rather complete knowledge of the life-history of this pathogene. Primary and secondary cycles with sharply marked pathogenesis and saprogenesis occur.

The **Primary Cycles** are initiated in early summer at the time the rye or other grass-hosts begin to head-out.

Pathogenesis. Ascospores which are produced in the spring constitute the inoculum for the primary cycles. From the ergots (sclerotia), which overwinter on the ground, there develop in the spring, stalked fruit-bodies. In the heads of these are developed numerous perithecia containing the asci with long slender ascospores. Examine the specimens provided or photograph 4, fig. 22. OBSERVE:—

7. The little pimple-like dots scattered thickly over the globose head of the fruit-body,—the ostiola or mouths of the perithecia.

From these ostiola the long thread-like ascospores are shot into the air and, carried by air-currents, lodge in the infection-courts,—the open blossoms of the rye or other grasses.

A bit of material from a mature stroma will be provided (take clean slide to materials room). Crush, cover and study. OBSERVE:—

8. The long slender ascospores; some floating free, others still in the asci; continuous or septate? DRAW several ascospores. (Keep this slide for No. 20.)

These germinate within the blossom. Examine photograph or de Bary, *Morphology and Biology of the Fungi*, p. 227, for germinating ascospores. NOTE:—

9. That upon germination the spore at once becomes, to all intents, a part of the mycelium. COPY.

The branching germtubes quickly penetrate the young ovary and, growing throughout its tissues, develop the Sphacelia or conidial form of the fungus.

Study cross-sections (freehand or prepared) of the upper end of the ovary (made during the Sphacelia stage). Examine with the high-power. OBSERVE:—

10. The numerous irregular cavities lined with erect conidiphores. Note the irregular dark masses of host-cells.

DRAW a small portion of the hymenium with underlying host-tissue, showing the conidia and the manner in which they are borne.

The honeydew consists of a sweet fluid, exuded by the pathogene, in which great numbers of these conidia find their way to the surface of the spikelet. Insects attracted by the honeydew serve as inoculating agents in starting the secondary cycles.

Very soon after, the conidia mature and the mycelium in the basal portion of the ovary begins to develop the sclerotium, which, as it grows and develops, replaces the host-cells and pushes forth as a long black spur with the shriveled Sphacelia structures at its tip. It is at first light colored, inclined to purplish but soon turns black. (See illustration specimen.)

Examine photographs 3, fig. 25 and 4, fig. 13-14. NOTE:—

11. That while the sclerotium usually replaces the entire ovary, this is not always the case. COPY from the above figures to show this.

Make very thin cross-sections of one of the mature sclerotia and study under the high-power. OBSERVE:—

12. The pseudoparenchymatous structures.

13. The thick walls and small lumina of the hyphae composing the sclerotium. Are there any evidences of host-tissue?

14. The dark color of the outer coat or rind; number of cells thick; the white color of the medulla.

Detail in a DRAWING the structure of the sclerotium.

Saprogenesis. These sclerotia, when mature, fall to the ground or find their way at threshing into bins along with the rye grains. With these they may be sown and find their way to the soil. Here they remain dormant until spring, when the fungus again becomes active. At any point beneath the rind, growth-centers may be set up from which are developed slender stalks with globose tops. These are developed at the expense of the food-materials stored in the cell-walls of the sclerotial medulla.

Examine the so-called germinating sclerotia provided, OBSERVE:—

15. The small, capitate bodies arising from the sclerotium,—the stromata. Note the comparative size of the stem and the head. Examine the stromata carefully with a hand-lens, noting the small dots or punctations. Make a DRAWING to bring out all the external characters of the stromata and how they arise from the sclerotium.

Prepared slides of cross-sections of the stromata are furnished. Examine with the low- and high-powers and OBSERVE:—

16. The ovate acuminate cavities,—perithecia. Note the dense pseudoparenchymatous wall.

17. The location of the perithecia about the periphery.

18. The opening at the top,—ostium. Note that it protrudes. Make an outline DRAWING of the cross-section of the entire stroma.

19. The asci arising from the bottom of the perithecium.

20. Within the asci, the bundle of eight thread-like spores. Supplement at this point with the crushed mount prepared under number 8. Stain by running a little methyl blue under the cover-glass.

Make an enlarged DRAWING of a perithecium containing the asci; and of a single enlarged ascus with the ascospores.

Secondary Cycles initiated by the conidia of the Sphacelia stage, multiply and spread the pathogene during the blossoming-period of the host. The Sphacelia stage, followed by the development of the ergot, is produced as in the primary cycles.

REPORT

1. Make one or more cartoon-like sketches to illustrate the life-history of *Claviceps purpurea* (Fr.) Tul.

ONION SMUT

This disease is most prevalent in the northeastern part of the United States where in certain localities it causes a considerable loss. Because of its striking symptoms it has been known to onion-growers for many years and has been the subject of study by scientists for at least the past forty years.

SYMPTOMS

Examine a diseased seedling. OBSERVE:—

1. The narrow, yellow lesions extending parallel with the leaves. These are the first symptoms to appear.

2. The narrow, black areas which are most numerous near the base of the onion and sometimes extend almost to the tip of the leaf. The yellow areas will later become darkened like these.

3. That these black masses are all enclosed within the tissue of the leaf, or that occasionally the epidermis is ruptured.

4. That in cases where the leaf is badly affected, its tip has withered and droops.

Make a DRAWING of a diseased seedling.

Examine one of the larger diseased onions. OBSERVE:—

5. That in this case each badly diseased leaf has turned brown and has fallen over.

6. That the black lesions have now broken open, forming long black open sori from which a sooty-like mass is sifting. DRAW.

ETIOLOGY

The pathogene responsible for this disease is *Urocystis Cepulae* Frost, a basidiomycetous fungus of the order Ustilaginales. There are two families in this order, the Ustilaginaceae and Tilletiaceae. To the former belong those organisms which produce the loose smuts of wheat, oats and corn, while to the latter belong the pathogenes like those producing the stinking smut of wheat, and the onion smut.

Life-history.

The **Primary Cycle** has its origin in the chlamydospores found in the infested soil.

Pathogenesis. These spores, when near germinating onion seeds, also germinate sending out a long branched mycelial thread. This mycelium probably enters the host directly, or it may possibly produce conidia or sporidia borne on the tips of the mycelial branches. In the latter case it is the germtube which grows from the sporidium, that enters the tissue of the host. Infection always takes place under ground and only when the seedling is just emerging from the soil. No new infections occur after the onion becomes older. This mycelium develops within the leaf-tissues, giving rise to the long black sori. Scrape a few spore-balls from a sorus and, under the microscope, OBSERVE:—

7. The size, color and surface markings of the spore-ball. It is made up of a central thick-walled resting-spore (one or more celled) surrounded completely by thin-walled pseudospores. Only the central cells are viable. If the inner structure can not be determined from the mount, consult Duggar, *Fungous Diseases of Plants*, p. 382.

Make a diagrammatic DRAWING showing the structure of the spore-ball. If no germinating spores are available, sketch the illustration shown in the above reference.

Cut a cross-section of an onion seedling through a closed lesion or use prepared slides. OBSERVE:—

8. The depth to which the sorus penetrates.
9. The black mass of spores lining the sorus. Are they attached to stalks? How are the spores of a smut-pathogene usually borne? (See McAlpine, Smuts of Australia, p. 19.)
10. The cuticle still intact and retaining the spores which later escape.
11. The mycelium penetrating the tissues adjoining the sorus. Do any of these cells appear dead?

Make a DRAWING of a cross-section of a sorus with the host-tissue surrounding it.

Saprogenesis. There is no vegetative activity during saprogenesis. The spores lie dormant in the soil or host-debris. They have been known to live over in the soil as long as twelve years.

Secondary cycle. Unlike most fungi this pathogene has no secondary cycle. Infection takes place only during a limited period while the host is in the seedling stage. They are initiated only by overwintered spores. Consequently, even if onion sets are transplanted to badly infested soil, the onions will remain free from the disease.

REPORT

1. Discuss the use of sulphur and of formaldehyde in the control of this disease, including the advantages or disadvantages in methods of application, the amounts used, cost, and the preference of the onion-grower of the present day.

LOOSE SMUT OF WHEAT

This disease is exceedingly common and often destructive in the wheat-fields of eastern United States and Canada. It is readily distinguished from the stinking smut. The latter, while not uncommon in the east, is much more general and destructive than loose smut in the great wheatlands of the west. While the loose smuts of wheat and oats are much alike in general appearance, they differ strikingly in certain features of the life-history of the respective pathogenes.

SYMPTOMS

The evidences of the disease are to be observed chiefly in the heads at blossoming-time. Compare the diseased and healthy heads provided.

NOTE:—

1. The general effect of the disease on the form and appearance of the head. Does it affect the rachis as to length and size; length of internodes? DRAW both diseased and healthy heads.

2. The effect on the individual spikelets. Determine this by carefully dissecting out the parts of the (moistened) healthy spikelets and flowers. DRAW. Dissect and DRAW the parts of the diseased spikelet.

3. That the culms of a diseased plant at first grow more rapidly and outstrip those of the healthy plant. Eventually, after spore dispersal, the healthy culms push up above the diseased ones. (See whether later studies will explain this.)

4. The naked rachides from which the spore-masses have disappeared. DRAW.

ETIOLOGY

The loose smut of wheat is caused by the basidiomycetous parasite, *Ustilago Tritici* (Persoon) Jensen. It is a member of the order of primitive basidiomycetes, the Ustilaginales commonly known as the smut-fungi. There are two families in this order, the Ustilaginaceae to which *U. Tritici* belongs and the Tilletiaceae to which belongs the stinking smut-pathogenes, *Tilletia Tritici* (Bjerk.) Wint. and *Tilletia foetens* (B. and C.) Trel.

Life-history. While the parasite causing this disease has long been known, it is but recently that its life-history has been completely understood. (See Pl. Ind. Bur. Bul. 152:10-12.) It differs, along with *U. nuda* (Jens.) Kell. and Sw. in barley, from most of the other smut-fungi whose life-history is known, in that infection occurs through the stigmatic surfaces of the pistil at flowering-time. There are no secondary cycles.

The **Primary Cycle** requires a full year for its completion. There is no saprogenesis, the pathogene remaining in continuous association with the living host except for the few minutes during transfer from the source of inoculum to the infection-court.

Pathogenesis. The black smutted heads emerging from the upper leaf-sheaths at flowering-time constitute the sources of inoculum.

Mount some of the sooty mass in a drop of potassium hydroxide, cover and examine with the high-power. OBSERVE:—

5. The numerous brown globose bodies scattered through the mount,—the chlamydospores. These, in the case of this smut-fungus, constitute the inoculum.

6. Their color (lighter on one side); markings; variations in form. (See demonstration microscope.) DRAW five different spores.

While the chlamydospores of most smut-fungi are resting-spores, those of *U. Tritici*, on account of the blossom-infecting habit of the pathogene, have largely lost their ability to remain viable for a long period.

The dusty dry chlamydospores are scattered by the wind over the blossoming wheat. Some spores fall upon the exposed stigmas protruding from the open glumes of healthy heads. Here they germinate, sending a long germtube along and into the stigmatic filaments and by way of them into the young ovary. If germinating spores are available, study and OBSERVE:—

7. The long germtubes, septate and with knee-joint fusions.

8. The uniform absence of sporidia.

DRAW several germinating chlamydospores to show variation.

This germtube is morphologically a basidium which in most other species of *Ustilago* produces basidiospores or sporidia as they are called. In this species, sporidial production is unnecessary. After penetrating to the ovary, the germtube branches and, establishing itself near the growing-point, goes into a dormant condition along with the ripening kernel. The invaded kernel shows no evidence of injury.

As the embryonic plant develops upon planting the kernel, the mycelium, aroused by the same condition of heat and moisture (and probably by the activities of the embryo as well), begins active growth, branches and spreads into the stooling culms, and forces its way upward through the tissue as the stem lengthens. When the wheat heads begin to form, the mycelium of the fungus takes possession, completely replacing the more tender or succulent tissues of the spikelet with its dense mats of profusely branching mycelium, thus forming the black, easily ruptured sori of the smut. The diseased heads emerge from the sheath about the time the flowers on the healthy heads are in full bloom. Why?

REPORT

1. A farmer writes for information on the nature and control of the loose smut of wheat. Write him a letter giving clearly and concisely the information he needs.

LOOSE SMUT OF OATS

There are two smuts of oats, the loose and the covered. Loose smut is much the more common and the one usually referred to as oat smut. This disease occurs wherever oats are grown and may often destroy from 25 to 50 percent of the crop. Its control is exceedingly simple and effective.

SYMPTOMS

The evidences of loose smut are usually confined to the inflorescence, though the leaves may rarely show lesions. Compare the specimens of this disease with healthy specimens provided. OBSERVE:—

1. The striking difference in the panicles of the two; form, size and color. DRAW.

2. In the entire plants (illustration specimens) the relative length of straw, number of stalks in the stool, amount and character of leafage.

3. The differences in the spikelets of each; empty glumes, hull and flower parts (most easily determined by dissecting specimens in water). Make enlarged DRAWINGS to show comparatively the effects of the disease on the parts of the spikelet.

Compare specimens of loose smut with those of covered smut. SKETCH to show differences.

ETIOLOGY

The pathogene causing the loose smut of oats is *Ustilago Avenae* (Persoon) Jensen. It is closely related to *Ustilago Tritici* (Pers.) Jens. but differs strikingly in certain features of its life-history. (See p. 137) The covered smut of oats, caused by *Ustilago levis* (Kell. and Sw.) Mag., occurs along with *U. Avenae* from which it is to be distinguished certainly only by its smooth, granular spores.

Life-history. There are in the life-history of this pathogene only primary cycles. While not so intimately associated with the living host during the resting-period as is *Ustilago Tritici* (Pers.) Jens., it normally exhibits little saprogenic activity under natural conditions.

The **Primary Cycles** are initiated at blossoming-time, at which time inoculation occurs.

Pathogenesis. The smutted plants scattered through the field are the sources of inoculum.

Remove a bit of the black mass from a smutted head to a drop of potassium hydroxide on a slide; cover and examine. OBSERVE:—

4. The numerous brown, globose bodies scattered through the mount,—the chlamydospores.

5. Their color (lighter on one side); markings and variations in size. DRAW three different spores. (See demonstration microscope, oil-immersion.)

These chlamydospores are resting-spores and may retain their vitality for several years.

The chlamydospores, disseminated when the oats are in blossom, lodge within the oat hull next to the kernel. They do not germinate at once as do those of *U. Tritici* but lie dormant as the oat hull closes about the maturing kernel. The chlamydospores are thus enclosed

along with the ripe kernel. When the oat grain is planted in the soil and germinates, the same conditions that cause the seed to grow, start the spores into activity.

Spores have been germinated on the slides provided. Carefully cover the drop of water containing the germinated spores, and examine. OBSERVE:—

6. The rather long hyaline germtube or promycelium,—the basidium of this basidiomycete.

7. That it is septate; number of cells into which it is divided; the crack in the chlamydospore-wall where the promycelium emerges.

8. That from each cell at the septum (above or below?) arises one or more oblong sporidia,—the basidiospores.

9. The variations from the normal germination.

Make DRAWINGS to show germination of chlamydospores and formation of sporidia, with such variations as are observed.

10. Examine the mount of germinated spores to see if you can find any of the sporidia germinating. DRAW.

As the seedling bursts through the seed-coat and pushes up through the hull, it is penetrated by the germtubes of the sporidia. The mycelium of the parasite grows and branches, pushing its way upward between the cells of the host as the culms of the oat come out and shoot upward. Usually every culm in the stool is infested. The mycelium at first stimulates the growth of the plant. The activities of the mycelium continue throughout the growth and development of the host until the formation of the flowers begins. In these rapidly developing embryonic tissues, the mycelium takes possession, appropriates the abundant food-substances and begins the formation of chlamydospores. The formation of these chlamydospores takes place as follows:—The mycelium in the flower at the time that spore-formation begins shows a nodulate appearance and the branches are closely fasciated like clusters of grapes. Within each swollen part of the mycelium a chlamydospore is formed. As the chlamydospores mature, the encompassing walls of the parent-hyphae and much of the general mycelium which is not differentiated into spores, gelatinizes or otherwise breaks away and the spores are set free in large masses.

Practically all the floral parts of every spikelet are destroyed, transforming them into the black sori of the pathogene. The smutted panicles emerge, the spore-masses ripen, and the spores are scattered when the healthy panicles are in full bloom.

REPORT

1. Write a concise explanation of why the formaldehyde-treatment succeeds with loose smut of oats and fails with loose smut of wheat.

STINKING SMUT OF WHEAT

Although the loose smut is the more common on wheat in eastern United States, the stinking smut is frequently serious. In the great wheat-fields of the west it is the common and destructive wheat smut. The losses from the stinking smut may be very large, not only from the reduction in yield but also from the lowering of the market-value of grain with which the smutted kernels become mixed. The life-history of the causal fungus is very different from that inducing the loose smut of wheat.

SYMPTOMS

Heads of wheat affected with the stinking smut are readily distinguished from the healthy heads as soon as they emerge from the leaf-sheath. Read Barrus, *Phytopath.* 6:21-28; study fig. 2; and the diseased and healthy specimens collected in different stages of development.

NOTE:—

1. The difference in size and color of the heads.
2. On dissecting flowers, the differences exhibited by ovaries and stamens; size, color, form and odor.

Confirm, so far as available material will permit, the observations of Barrus. Make a series of DRAWINGS to show the comparative morphology of diseased and healthy heads and flowers, from flowering-time to maturity.

ETIOLOGY

This disease is caused by *Tilletia Tritici* (Bjerkander) Winter, or by *Tilletia foetens* (Berkley and Curtis) Trelease, (= *T. laevis* Kühn). This is a case in which two distinctly different species of fungi cause the same type of disease, in fact the two species may occur together in the same ovary. It is the latter form, *T. foetens*, which is most commonly found in eastern United States and will be the one here considered. These are species of the Ustilaginales, family Tilletiaceae.

Life-history. This pathogene exhibits only the primary cycles in its life-history. Since the chlamydo-spores usually become attached to the healthy grains during harvesting or threshing and so are almost continuously associated with the living host, this smut-fungus like most others may be said to have normally no saprogenic phase. Infection from spores in the soil appears to be rare for this species. The sown wheat in the Pacific Coast regions is said to become inoculated by wind-borne spores of *T. Tritici*.

Pathogenesis. Inoculation, as indicated above, occurs at threshing-time, when spores from the crushed smutted kernels become attached to the healthy grains. Crush one of the diseased grains between thumb and finger. NOTE:—

3. The greasy character of the spore-mass. This evidently helps to attach the spores to the seed-coat.

Mount a bit of the spore-mass in a drop of water. Cover and examine. OBSERVE:—

4. The numerous, more or less globose, smooth brown spores, —the chlamydo-spores. DRAW.

5. The chlamydospores of *Tilletia Tritici* under the demonstration microscope. Note the reticulated surface of the spores, the globose shape and smaller size. DRAW.

Make a mount containing chlamydospores of both *Ustilago Tritici* and *Tilletia foetens*. Compare as to size, shape, color and markings. DRAW to show the contrast.

When the inoculated grains are planted, the spores of the pathogene are planted with them. The conditions which favor seed-germination also favor spore-germination. A promycelium is formed bearing sporidia which in turn produce germ-tubes capable of penetrating the seedling, but only at some point below the first node or tillering-point. Study the germinated spores provided, or figures in Washington Bul. 126:7, and OBSERVE:—

6. The germ-tube arising from the chlamydospores,—the promycelium. From which side of the spore does it arise?

7. The cluster of sickle-shaped sporidia at the end of the promycelium.

8. That frequently two of the sporidia have joined by a short tube forming an H-like figure. This is very common and was once supposed to be a sexual process but it is not now so considered.

9. Some of the sporidia germinated, forming secondary sporidia.

Make DRAWINGS to show chlamydospore-germination and formation of secondary sporidia, or COPY from Minnesota Bul. 133, pl. XXIV and XXV.

The germ-tubes from sporidia or secondary sporidia penetrate the seedling as it emerges from the grain and, branching, forms a mycelium. This mycelium quickly reaches the growing-point and grows upward with the culm causing no apparent injury to the host. At the time of flower-formation, however, the hyphae develop rapidly and, growing into the ovary, destroy the contents and replace them with the black spore-mass.

REPORT

1. A farmer who knows how to control stinking smut desires to understand the cause of the disease and just how it differs in this respect from the loose smut of wheat. Write him a letter giving this information in a form which he will understand.

ASPARAGUS RUST

This disease is of European origin, and first appeared in America in several localities along the Atlantic seaboard about 1896. It rapidly appeared farther and farther west until by 1902 it had become an important factor in asparagus-growing in California. It is much more destructive in this country than in Europe. It is the most important disease of asparagus in America.

SYMPTOMS

Most rust diseases exhibit themselves in some form of hypoplastic or metaplastic effect. While the very first effect of the asparagus rust is probably hypoplastic in character, this rapidly passes over into a necrotic condition, the host-tissues being rather quickly killed. The signs and symptoms of rust diseases group themselves about certain distinct spore-producing stages of the pathogene. In the case of the asparagus rust there are three: the cluster-cup stage, the red rust stage and the black rust stage. All of these occur on asparagus.

The cluster-cup stage. The disease in this stage affects only the stalks in the young condition shortly after they come up in the early spring. Examine the specimens provided and OBSERVE:—

1. The oval-shaped, light-green patches on the canes near the base, covered with minute honey-colored pimples. These are the spermagonial fruit-bodies of the pathogene,—the pycnia.

2. On the lesions as they grow older, larger pustules arranged in concentric order within the lesions, especially definite in the older spots; averaging how many to a lesion?

3. That finally these pustules break through the epidermis and appear as round cup-shaped bodies,—the aecia, exposing the yellow spore-mass within.

This phase of the disease is usually to be found only on volunteer plants along roadsides and hedgerows, on plants in abandoned plantations and in young uncut plantations. Make a DRAWING to show the lesion of the cluster-cup stage.

The red rust stage. This usually appears early in the summer on the plants which are allowed to grow up after the cutting-season. In the specimens provided, OBSERVE:—

4. The rusty brown linear pustules scattered very abundantly over the stem, branches and even on the needles,—the uredinial sori or uredinia.

5. Their size, number and arrangement on the surface; relation to the host-tissues.

6. The dusty character of their contents.

7. Any pathogenic effects exhibited by the tissues about the sori.

Make a DRAWING of a diseased stem; also a DRAWING of a uredinium as seen under the hand-lens.

The black rust stage. This phase of the disease begins to develop in late summer on the same branches and needles along with the red rust stage. During the transition, affected plants take on a brown color which, however, soon gives place to a distinct black, as uredinial develop-

ment ceases and the black resting-spores of the pathogene predominate. Examine the old diseased branches and needles provided and OBSERVE:—

8. The crowded black sori or telia. How do they compare with the uredinia as to prominence, shape, size, arrangement on the stems and branches, and dustiness of their spore-masses?

9. That the host-tissues covered with the telia are dead and shriveled. This is the last phase of the disease, the autumn or winter condition of rusted plants.

Make a DRAWING of a diseased stem, and also of the pustules as seen under the hand-lens.

ETIOLOGY

The asparagus rust is caused by *Puccinia Asparagi* DeCandolle, a species of the Uredinales, an order of fungi, the members of which are the most highly specialized of the Basidiomycetes. It is an autoecious pathogene producing its different spore-forms all upon the same host.

Life-history. It produces during its life-cycles all the known fruiting structures of the rusts: pycnia (O), aecia (I), uredinia (II), telia (III), and basidia (IV), each with their characteristic spores. It is therefore said to be a Eupuccinia, that is, a true Puccinia. (See McAlpine, Rusts of Australia, p. 10-11.)

The **Primary Cycles** have their development early in the spring on the young shoots before July first; often as early as March but usually during April and May, depending upon the locality and season. Pycnia and aecia only are developed during the primary cycles.

Pathogenesis. The inoculum for the primary infections consists of the basidiospores or sporidia produced on the promycelium from the overwintered teliospores on diseased stalks and needles. Examine the germinated teliospores on slides provided; or study California Bul. 165, fig. 27. OBSERVE:—

10. The long slender promycelium (basidium) put forth from each cell of the teliospore; 4-celled near the apex.

11. The long pointed sterigma from each basidial cell, bearing at its apex a thin-walled ovoidal basidiospore. Each spore contains one nucleus and finely granular protoplasm.

DRAW or COPY to show sporidia ready for dissemination.

These sporidia are carried by the wind or splashing rain to nearby shoots just coming up. There in the moisture on the surface they send forth a germtube which penetrates the tender epidermis and gives rise to a locally spreading mycelium. From the mycelium, within less than a month, are produced first pycnia and promptly after them, aecia.

Study cross-sections of the stalk (freehand or prepared) through an aecial lesion; or California Bul. 165, fig. 18. OBSERVE:—

12. The large cup-like aecia and scattered among them the minute flask-shaped pycnia (spermogonia).

Locate a pycnium and study its structure under the high-power. MAKE OUT:—

13. The very slender interwoven mycelium forming the pycnial wall.

14. The long slender sporophores arising from the wall-mycelium and converging at the center of the pycnium about a small cavity which is filled with the very minute bacterium-like pycnospores (spermatia).

15. The protruding neck of the ostiolum through which the spermatia are discharged. These pycnosporos have now no known function. They will not germinate and are supposed to be vestigial male cells (sperms).

Study the aecia and OBSERVE:—

16. That they are sunken in the tissues; at first covered by the epidermis of the host. Locate one that has not burst through the epidermis. The pycnia are more superficial; subepidermal or subcuticular?

17. The fine interwoven mycelial threads between the host-cells about the aecia. Do they ever penetrate into the host-cells?

18. That it is from a densely interwoven mat of this mycelium that the structures of the aecium are developed; to be seen at its base.

19. The closely packed club-shaped sporophores in the base of the cup, arising from the mycelial mat.

20. The chains of spores cut off from each sporophore, forming the parenchyma-like mass that fills the cup. Why are the immature aeciospores angular in outline? Note that the maturing spores at the mouth of the aecium become globose.

21. The large cells forming a lining to the cup,—peridial cells. They also arise in chains from short mycelial branches similar to the sporophores. Note the difference in the thickness of the wall of the inner and outer faces of the cells. This lining of the cup is pseudoparenchymatous and is called the peridium.

The mycelium of the aecial stage arises from the one-celled basidiospore, and each cell is uninucleate. When the formation of the aecium begins, there is a sexual fusion of many pairs of mycelial branches in the region of what is to be the base of the cup. From each pair of branches arises a single binucleated stalk,—a sporophore of the aecium. A chain of spores are developed from each of these binucleate sporophores by successive conjugate nuclear divisions and a laying-down of a septum between the pairs of nuclei thus formed. Examine stained sections carefully under high-power and LOCATE:—

22. The paired nuclei in the sporophores; in the aeciospores.

Make a DRAWING to show the structure of the aecium, the pycnium and their relation to the host-tissues as seen in longisection.

Remove with a needle or scalpel some mature aeciospores from the diseased stalks provided. If dry, mount in potassium hydroxide. Study and OBSERVE:—

23. The form, size, color, contents and markings on the epispore. DRAW several aeciospores.

But one crop of aeciospores is produced. With the maturity and discharge of the last of them the activities of the primary cycle cease. The aeciospores initiate secondary cycles through which alone the pathogene perpetuates itself until the following year. There is no saprogenesis in the primary cycle.

Secondary Cycles develop on the stems, branches and needles of stalks affected by the aecial stage and on those of neighboring asparagus plants.

Pathogenesis. The first secondary cycles are initiated by aeciospores which fall upon the host. These germinate in the dew which forms at night and send out each a germtube which rapidly grows along

over the surface until its tip reaches a stoma through which it penetrates to the tissue within. The protoplasm of the spore passes out into the germ-tube keeping constantly in its tip as it grows. Within the substomatal cavity, the tip of the germtube applies itself to one of the parenchymal cells and sends into it a short blunt branch,—a haustorium. Having thus established a food relation with the host, it grows and develops a ramifying binucleate mycelium. This mycelium spreads only locally and produces within two or three weeks mature uredinia. Scrape some uredospores from a diseased stalk, mount in potassium hydroxide and OBSERVE:—

24. The form, size, color and contents of the uredospores as compared with the aeciospores; markings on the epispore if any. DRAW several aeciospores.

Study cross-sections (freehand or prepared) of a stem through a uredinium and MAKE OUT:—

25. The structure of the sorus and its relation to the host-tissue.

26. The intercellular mycelium. Are haustoria formed? (See California Bul. 165, fig. 16.)

27. The stout sporophores arising from the mycelium, each bearing at its apex a uredospore in some stage of development or mature. How can one tell whether the spore is young or mature?

Make a DRAWING of a part of the cross-section through the uredinium.

These uredospores are usually wind-disseminated and initiate other secondary cycles. They germinate at once in moisture, even in a moist atmosphere. They infect the host in a manner and under conditions similar to the aeciospores. The germtubes emerge through special pores or thin places in the spore-wall. Mount and examine uredospores that have been treated with acetic acid. OBSERVE:—

28. The size, number and location of the pores. DRAW. For germination and penetration into the host, study California Bul. 165, fig. 21–22. Supplement with study of germinating spores on slides, if available. Make DRAWINGS to show the points brought out.

From the mycelium produced from the uredospore, other uredinia with mature uredospores may be produced in twelve days under favorable conditions. This is repeated over and over throughout the growing-season. The mycelium remains binucleate throughout all the uredospore-generations, the spores being binucleate also.

As the host begins to mature, there is developed from the mycelium in the secondary lesions, not uredospores but another spore-form,—chlamydospores, black resting-spores known as teliospores (teleutospores). They often arise at first in the same sorus with the uredospores, later they appear in sori in which no uredospores are formed. Mount some teliospores in potassium hydroxide and STUDY:—

29. Their form, size and structure as compared with the uredospores; color, where located, in walls or contents?

30. The thick walls and densely granular oily contents.

31. The light spot in the center of each cell; not always evident. The nuclei are located here. These spores are at first binucleate but at, or just before the germination the nuclei fuse and sexual fertilization, begun by the association of nuclei in the aecium, is now completed.

The structure of the telium and its relation to the host-tissues does not differ materially from that of the uredinium. DRAW several teliospores to show structures and variations in form and size observed.

Saprogenesis. With the formation of the teliospores, the host has succumbed. The rusted twigs and needles fall to the ground while the old stalks stand erect during the winter or fall under the weight of snow. On these stalks, twigs and needles, the pathogene in the form of dormant teliospores passes the winter. Under the conditions of moisture and temperature that start the host into growth in the spring, the teliospores on the old host-debris become active. They germinate *in situ* forming a promycelium from each cell of the spore. Four basidiospores are produced on each basidium. Each is uninucleate, its nucleus having been derived through the reduction division of the fusion nucleus of the teliospore. These sporidia, as we have seen, constitute the primary inoculum which infects the young asparagus shoots and produces the aecia.

REPORT

1. Prepare a DIAGRAM showing the spore-forms, and the activities of *P. Asparagi* DC. in proper sequence through the primary and secondary cycles.
2. Give in some detail three fundamentally different methods of controlling the rust of asparagus, arranging them in order of practicability and efficiency. Justify by explanation and argument, the position to which each method is assigned in the sequence.

BLACK STEM-RUST OF CEREALS AND GRASSES

There are many rust diseases of cereals and grasses. The black stem-rust is perhaps of greater economic importance than any of the others, especially on wheat. It is one of the earliest-recorded rust diseases and the subject of many researches by mycologists and plant pathologists. Early in the spring it may affect the barberry (*Berberis vulgaris* L.). At the same time or later in the season it appears on wheat, oats, barley, rye and many other grasses. On cereals and grasses it is characteristically a stem-rust.

SYMPTOMS

This disease is far more injurious to cereals and grasses than to the barberry. The signs and symptoms exhibited by rust diseases are associated with certain distinct spore-producing stages of the causal fungus. In the case of the black rust of cereals there are three: the cluster-cup stage, the red rust stage and the black rust stage.

Cluster-cup stage. The leaves and flower-racemes of the barberry are the organs affected. Examine the leaves provided and OBSERVE:—

1. The small spot-lesions; size, location, color both above and below, and the thickening of the leaf-tissues in the lesion. The disease exhibits distinct metaplastic effects on this host; there is a slight over-growth of affected tissues.

2. With the hand-lens, the tiny dark amber or nearly black pimples embedded in the tissues of the upper surface,—the pycnia (spermatogonia), functionless structures of the pathogene. They normally accompany or precede, in the same lesion, the aecial fruit-bodies next to be observed.

3. On the lower surface, the sunken cups, exposing their yellow spore-masses. These are the aecia or cluster-cups.

Make DRAWINGS to show the cluster-cup symptoms on both surfaces of the leaf.

Examine the diseased flower-clusters. OBSERVE:—

4. The swollen and malformed flowers and pedicels; color of the affected organs.

5. The abundantly developed aecia. Are pycnia present?

SKETCH a diseased flower cluster.

Red rust stage. Examine the specimens of diseased wheat or oats provided and OBSERVE:—

6. The large reddish yellow pustules scattered over the leaf-sheaths,—the uredinia; not usually so common nor so numerous on the blades.

7. The size, arrangement and relation of these pustules to the host-tissues. Note the torn edges of the epidermal covering and the dusty spore-mass of the ruptured uredinia.

8. The effect on the host-tissues; color effects.

SKETCH a rusted leaf-sheath to show the symptoms of the red rust stage. Make a DRAWING of one of the uredinia as it appears under the hand-lens.

Black rust stage. This develops during the latter part of the growing-season along with the red rust stage so that the two are often observed together on the leaf-sheaths and stems. In the material provided, OBSERVE:—

9. The large black linear sori extending up and down the stem,—the telia. Most of them have ruptured the epidermis, exposing the black spore-mass within.

10. The thin epidermal covering, ruptured in most of the telia but partially or wholly covering others.

11. The telia developed on the inflorescence. What organs are affected?

12. Kernels from badly diseased plants in comparison with those from healthy plants. How do they appear to be affected? Telial sori are occasionally developed under the seed-coat of the kernel. (See *Phytopath.* 1:150–154, fig. 1–2.)

SKETCH an internode and also parts of the inflorescence to show the black rust symptoms. Make an enlarged DRAWING of a telium as seen under the hand-lens.

ETIOLOGY

The black rust of cereals and grasses is caused by the fungus, *Puccinia graminis* Persoon. It is one of the Uredinales which are perhaps the most highly specialized of basidiomycetous pathogenes. It is a heteroecious parasite producing its aecial structures on the barberry, a host widely remote in relationship from its grass-hosts on which the other spore-forms appear. This species, *Puccinia graminis*, includes a variety of biologic forms, morphologically alike, and all having their aecia on the barberry but restricted rather sharply in their uredinial and telial stages to definite cereal- or grass-hosts. (See McAlpine, *Rusts of Australia*, p. 122.)

Life-history. This pathogene produces, during its life-history, all the known fruiting structures of the rust-fungi: pycnia (O), aecia (I), uredinia (II), telia (III), and basidia (IV), each with their characteristic spores. It is therefore said to be a Eupuccinia, that is, a true Puccinia. (See McAlpine, *Rusts of Australia*, p. 10–11.)

The **Primary Cycles** are normally those which develop only upon the barberry and exhibit the pycnia and aecia. It seems certain, however, that the fungus may winter over in the uredinial form (probably as mycelium) and by uredospores initiate primary infections on grass- and cereal-hosts. This second type of primary cycle is essentially secondary in all respects except as to the time it originates. It will not be considered separately in this exercise.

Pathogenesis. The inoculum for the primary infections consists of the basidiospores (sporidia), produced on the basidium (promycelium) from the overwintered teliospores on diseased straw. Examine the germinated teliospores on slides or study the drawings provided, and OBSERVE:—

13. The long slender basidium put forth from each cell of the teliospore; 4-celled near the apex.

14. The long pointed sterigma from each basidial cell bearing at its apex a thin-walled ovoidal basidiospore (sporidium). Each sporidium contains one nucleus and finely granular protoplasm.

DRAW or COPY to show sporidia ready for dissemination.

These sporidia are carried by the wind or splashing rain to nearby barberry bushes where, in the moisture on the surface, they send forth a germ-tube which penetrates the tender epidermis and gives rise to the locally

spreading mycelium. From the mycelium, within less than a month, are produced first pycnia and promptly after them, aecia.

Study cross-sections of the leaf (freehand or prepared) through an aecial lesion. OBSERVE:—

15. The large cup-like aecia and opposite them on the upper surface of the leaf, the minute flask-shaped pycnia (spermagonia).

Locate a pycnium and study its structure under high-power. MAKE OUT:—

16. The very slender interwoven mycelium forming the pycnial wall.

17. The long slender sporophores arising from the wall-mycelium and converging at the center of the pycnium about a small cavity which is filled with very minute bacterium-like pycnosporos (spermatia).

18. The protruding neck of the ostiolum through which the spermatia are discharged. These spermatia have now no known function. They will not germinate and are supposed to be vestigial male cells (sperms).

Study an aecium and OBSERVE:—

19. That it is sunken in the tissues; at first covered by the epidermis of the host (see one that has not burst through). The pycnia are more superficial; subepidermal or subcuticular?

20. The fine interwoven mycelial threads between the host-cells about the aecia. Do they ever penetrate into the host-cells?

21. That it is from a densely interwoven mat of this mycelium (to be seen at the base) that the structures of the aecium are developed.

22. The closely packed club-shaped sporophores arising from the mycelial mat in the base of the cup.

23. The chains of spores cut off from each sporophore, forming the parenchyma-like mass that fills the cup. Why are the aeciospores here angular in outline? Note that the maturing spores at the mouth of the aecium become globose.

24. The large cells lining the cup,—peridial cells. They also arise in chains from short mycelial branches similar to sporophores. Note the difference in thickness of the wall of the inner and outer faces of the cells. This lining of the cup is pseudoparenchymatous and is called the peridium.

The mycelium of the aecial stage arises from the uninucleate sporidium and each mycelial cell is uninucleate. When the formation of the aecium begins, there is a sexual fusion of many pairs of mycelial tips in the region where the base of the cup is to be. From each pair of branches arises a single binucleate stalk,—a sporophore of the aecium. A chain of spores is developed from each of these binucleate sporophores by successive conjugate divisions of the paired nuclei and the laying-down of a septum between the resulting pairs thus formed. Examine stained sections carefully under high-power and LOCATE:—

25. The paired nuclei in the sporophores; in the aeciospores.

Make a DRAWING to show the structure of the aecium, pycnium and their relation to the host-tissue as seen in section.

Remove with the needle or scalpel some mature aeciospores from the cups on diseased leaves provided. If dry, mount in potassium hydroxide. Study and OBSERVE:—

26. Their form, size, color and contents; markings on the episporium. DRAW several aeciospores.

But one crop of aeciospores is produced. With the maturity and discharge of the last aeciospores, the activities of the primary cycles cease. The aeciospores initiate secondary cycles on the cereal-hosts through which the pathogene perpetuates itself until the following year. There is no saprogenesis in the primary cycles.

Secondary Cycles, especially the late ones, develop chiefly on the leaf-sheaths and stems of the wheat, oats or grass-hosts. Leaves may also become infected.

Pathogenesis. The first secondary cycles are initiated by aeciospores which, falling upon the host, germinate in the dew or raindrops sending out a germtube which rapidly grows along over the surface until its tip reaches a stoma through which it penetrates to the tissues within. The protoplasm of the spore passes out into the germtube keeping constantly in its tip as it grows. Within the substomatal cavity, the tip of the germtube applies itself to one of the parenchymal cells and sends into it a short blunt branch,—a haustorium.

Having thus established a food-relation with the host, a binucleate ramifying mycelium is developed. This mycelium produces mature uredinia within two or three weeks.

Study cross-sections (freehand or prepared) of a stem through a uredinium and MAKE OUT:—

27. The structure of the sorus and its relation to the host-tissue.

28. The intercellular mycelium. Can the globose haustoria be found?

29. The stout sporophores arising from the mycelium, each bearing at its apex a uredospore in some stage of development, or mature. How can one tell whether the spore is young or mature?

Make a DRAWING of a cross-section through the uredinium.

Scrape some uredospores from a diseased stalk; mount in potassium hydroxide and OBSERVE:—

30. The form, size, color and contents of the uredospore as compared with the aeciospores; markings on the epispore, if any.

These uredospores are usually wind-disseminated, initiating other secondary cycles. They germinate at once in moisture, even in a moist atmosphere. They infect the host in a manner and under conditions similar to the aeciospores. The germtubes emerge through special pores or thin places in the spore-wall. Mount and examine uredospores that have been treated with acetic acid. OBSERVE:—

31. The size, number and location of the pores. DRAW.

For germination and penetration into the host, study illustrations provided. Supplement this with a study of germinating spores on slides, if available. Make DRAWINGS to show the points brought out.

From the mycelium produced from the uredospore, other uredinia with mature uredospores are produced. This may be repeated seven or eight times during the growing-season. The mycelium remains binucleate throughout all the uredospore-generations, the spores being binucleate also.

As the host begins to mature, there is developed from the mycelium in the secondary lesions, not uredospores but another spore-form,—chlamydospores, black resting-spores known as teliospores (telcutospores). They often arise at first in the same sorus with the uredospores, later they

appear in sori in which no uredospores are formed. Mount some teliospores in potassium hydroxide and STUDY:--

32. Their form, size and structure as compared with uredospores; color, where located, in walls or contents?

33. The thick walls and densely granular oily contents.

34. The light spot in the center of each cell; not always evident. The nuclei are located here. These spores are at first binucleate but at, or just before germination the nuclei fuse and sexual fertilization, begun by the association of nuclei in the aecium, is now completed.

The structure of the telium and its relation to the host-tissue does not differ materially from that of the uredinium. DRAW several teliospores to show structure and variations in form and size observed.

Saprogenesis. With the ripening or death of the host, teliospores have been formed usually in great abundance on the straw. When the grain is harvested, many teliospores are left on the stubble. Those on the straw go with it into the stack, then into the manure pile and finally with the manure back on the land. Under the conditions of moisture and temperature that starts the barberry into growth in the spring, the teliospores on the old straw become active. They germinate *in situ* forming a promycelium from each cell of the spore. Four basidiospores are produced from each basidium. Each is uninucleate, its nucleus having been derived through the reduction division of the fusion nucleus of the teliospore. These sporidia, as we have seen, constitute the primary inoculum which affects the young leaves of the barberry. They cannot, so far as is known, affect any of the cereals or grasses.

REPORT

1. Prepare a diagram showing the spore-forms and activities of *Puccinia graminis* Pers. in their proper sequence through the primary and secondary cycles.

2. Explain the effect of the black stem-rust on the tissues of its host, and point out how it causes reduction in yield.

CARNATION RUST

This is a disease of European origin, which was first observed in this country in 1890. It rapidly developed in epiphytotic form, shortly appearing in every carnation house throughout eastern United States. It was for a decade the most destructive and most feared of carnation diseases, but is now enphytotic and rarely serious, due to the development of resistant varieties. It also affects some other species of the genus *Dianthus* and species in related genera. In Europe the cluster-cup stage of this disease is known, affecting *Euphorbia Gerardiana* Jacq.

SYMPTOMS

Since the cluster-cup stage of the carnation rust is unknown in America, the disease will be studied only as it appears on the common greenhouse carnation. The red and black rust stages appear together and are so much alike that the symptoms they induce will be considered together. Both leaves and stems are affected. Study the materials provided and OBSERVE:—

1. The pale-green or yellowish areas on the leaves; which surface?
2. In some of these spots, dark-brown or black pustules,—sori of the pathogene. The lighter-colored ones are uredinia, the darker ones, telia.
3. The arrangement of these sori within the lesion; scattered, confluent, linear?
4. Some of the sori burst open exposing the dark-colored rusty spore-mass within; the ruptured epidermis forming a papery fringe.
5. The sori on the stems; usually darker than those on the leaf. Stem-sori are largely telia.
6. The general effect of the disease on badly rusted plants.

Make DRAWINGS to show the characters of the lesions on leaves and stems.

ETIOLOGY

The causal organism is a urediniaceous fungus, *Uromyces Caryophyllinus* (Schrank) Winter. It is heteroecious, having all the spore-forms characteristic of the rust-fungi.

Life-history. Being in this country a parasite of a greenhouse-crop, the fungus is able to propagate itself continuously through secondary cycles. The primary cycle therefore will not be here considered.

The **Secondary Cycles** are all initiated by uredospores from sori on leaves or stems of living plants (except of course when aeciospores occur). They are constantly being initiated as uredospores are matured and disseminated.

Scrape some of the uredospores from a sorus; mount and study. OBSERVE:—

7. The form, size, color, thickness of wall and markings on the epispore. Treat some with acetic acid and see if the number and arrangement of the germ-pores can be determined. DRAW several uredospores.

These uredospores are scattered by wind or splashed, in watering, to nearby healthy leaves and plants. They germinate very readily in water. Examine the germinating spores on the slides provided. OBSERVE:—

8. The number of germ-tubes produced from each spore. Are they septate?

9. The densely granular protoplasm crowded into the tips of the long germ-tubes. The empty germinated spores. Compare with those not germinated.

DRAW two or more germinated uredospores.

The germ-tube enters through a stoma and forces its tip between the palisade-cells, sending haustoria into them, thus establishing a food-relation with the host. Make thin cross-sections of a leaf through a sorus; clear in chloral hydrate; study and OBSERVE:—

10. The intercellular mycelium; often in gnarled and interwoven meshes, especially in the intercellular spaces, prying apart and isolating the host-cells; densely granular, septate.

11. The peculiar lobed haustoria in the host-cells; their very thin walls and densely granular protoplasm.

12. The uredinium; its structure and relation to the host-tissue.

13. The matted mycelial bed from which arise the short closely crowded uredosporophores.

14. The globose uredospores; thin walled, covered with short spines and much lighter colored than the one-celled teliospores, some of which are usually to be found in a uredinium.

Make a DRAWING of a portion of a cross-section to show the pathogene-structures and their relation to the host-tissue.

The mycelium is supposed to spread very generally throughout the leaf- and stem-tissues beyond the point where distinct lesions appear, so that cuttings from diseased plants are usually invaded. It is believed that this habit of the pathogene is responsible for its wide and rapid distribution in this country. Make thin sections through the leaf at some distance from the lesion; clear in chloral hydrate; examine and DETERMINE:—

15. Whether or not mycelial invasion is general.

Select a dark-colored sorus,—a telium (usually more abundant on stems); mount some of the teliospores. Study and OBSERVE:—

16. That they are much darker in color than the uredospores, although of about the same size and shape; walls slightly thicker, and episporium nearly or quite smooth; stalk often still attached. DRAW several.

The teliospores arise from the same mycelium on which uredospores are produced, usually more abundantly in the sori formed as the affected organ grows older.

Saprogenesis. The teliospores do not germinate at once as do the uredospores, but require a period of rest. They are chlamydospores, and, under natural conditions, germinate in the spring producing a promycelium with four sterigmata, each bearing a basidiospore (sporidium). Study the drawings provided. COPY.

These sporidia produced by the germination of the teliospores can, so far as known, infect only the Euphorbia host. As *Euphorbia gerardiana* does not grow in this country, the sporidia cannot function in the propagation of the pathogene, and primary cycles cannot develop.

REPORT

1. Prepare a diagram showing the complete life-history of *Uromyces Caryophyllinus* (Schr.) Wint. as it may occur in Europe.

DODDER

The term dodder is applied as a common name to the pathogenes as well as to the diseases which they cause. It is used for all the diseases caused by species of phanerogamous parasites of the genus *Cuscuta*. The different dodder diseases are often distinguished by adding the host-name as, alfalfa dodder, clover dodder and the like. The pathologic effects are in all cases similar.

SYMPTOMS

All parts of the host above ground, especially the stems, are directly attacked by dodder pathogenes. The most striking sign is the dodder parasite itself. The tangled yellow stems matted about its host form conspicuous yellow patches in the field. These doddered areas, often several feet in diameter, are at first more or less circular. Examine the photographs showing spots in the field. OBSERVE:—

1. The dwarfed and dying host-plants at the center of the doddered area.
2. The densely matted dodder stems.
3. The long spreading runner-like dodder stems about the margins of the area, extending out among the healthy plants.

Examine the specimens provided and OBSERVE:—

4. The yellow dodder vine entwined about the host-stems; size, color, shape in cross-section, branching.

5. Evidences of injury to the host, to be detected by comparison of diseased and healthy plants; dwarfing and chlorosis.

Make a DRAWING to show the general appearance of a dodder-affected plant.

ETIOLOGY

Dodder diseases are caused by species of *Cuscuta*, a genus of the family Convolvulaceae. Some near relatives in the same family are the morning-glories, bindweeds, buckwheat and sweet potatoes. There are about ninety species of *Cuscuta*, of which about half occur in America. The most common one on cultivated crops like clover and alfalfa is *Cuscuta epithimum* Murray, a species introduced from Europe.

Life-history. This pathogene has an annual life-history. It is known, however, that it commonly overwinters in a vegetative condition on perennial hosts like clover and alfalfa. (See N. Y. (Geneva) Bul. 305:369-374.)

The **Primary Cycles** are initiated by the seeds which have lain on the ground over winter.

Pathogenesis. Examine seed of *C. epithimum*. NOTE:—

6. Their surface markings; color; and that the one side is convex while the other is flattened in two or more places. Measure several. DRAW:—(a) several to show variations in size and form; (b) two seeds greatly enlarged showing all their characteristics. Describe the color in the notes on the margin of the paper.

7. Examine the seed of the other species provided. These may all occur on clover or alfalfa. Make enlarged DRAWINGS of each so that they will be comparable in size with those of the drawings of *C. epithimum*.

8. The embryo is coiled in the seed and imbedded in the food substance. Examine the photographs of plates taken from Koch's work. DRAW.

9. If available, examine the dodder seedlings and note how they first attack the host-plant. Examine N. Y. (Geneva) Bul. 305, pl. 3, and Koch's pl. 1, fig. 1-3. Make DRAWINGS to show the characters of the seedling and its method of attacking the host.

As soon as the seedling has attached itself to the host, the vestigial root by which it was anchored in the soil, shrivels and dies. The twining stem, however, feeds on the host, grows rapidly and by branching spreads to neighboring hosts. Study the dodder plant on the host provided. OBSERVE:—

10. The type of branching; opposite or alternate?

11. The leaves; their location, size, shape, color and abundance. How are these foliage conditions to be accounted for?

Untwine and separate a portion of the dodder thread from the host-stem. OBSERVE:—

12. That it is attached at some points. At such places the spirals are shorter and are tight about the stem.

13. With the hand-lens, the rows of minute punctures in the host-stem where the dodder was attached; the oval depression about each.

14. With the hand-lens, the corresponding row of concave sucker-like disks on the dodder stem,—the attachment-disk; the minute elevation at the center of each disk,—the broken-off haustorium.

15. The aborted haustoria in places along the dodder stem where it failed to come in contact with the host.

Make a DRAWING of a portion of a host-stem with entwined dodder to show all the points brought out in 10-15.

Cut thin cross-sections through the clover and dodder stems at the point where haustoria are attached. Cover and examine under the low-power. OBSERVE:—

16. In the clover stem, the large pith, the vascular cylinder, the cortex, and the epidermal layers. In the vascular cylinder, note the xylem containing the large ducts, and the phloem which consists of soft sclerenchyma within and the lighter area of sclerenchyma-fibers without.

17. In the dodder stem, the haustorium. Note the shape of the attachment-disk in cross-section; its relation to the haustorium; where the haustorium arises and how it penetrates the host. Note what happens where it comes in contact with the sclerenchyma-fibers. Toward which tissue of the host-stem does it grow? Why? Note the spiral ducts in the center of the haustorium.

Make diagrammatic DRAWINGS of the host-stem and the dodder stem to show the relative sizes and relation of parts.

With the low-power, examine the stained longitudinal sections of the haustorium of dodder on *Impatiens* (touch-me-not). NOTE:—

18. The long thread-like cells extending out from the tip and sides of the haustorium into the cortex, pith, and sometimes into a bundle, making a connection with a conductive vessel of the host.

19. The layer of crushed cells at the point where the haustorium emerges from the dodder stem. Does the epidermis of the dodder continue about the haustorium? What becomes of it? Are the cells of the host crushed? What is the function of the thread-like cells?

DRAW a single haustorium showing the type of cells in the center and at the ends; the connection of the host and the dodder. Toward autumn flowers may be developed on the dodder vine.

Study a fruiting branch of the dodder and OBSERVE:—

20. The compact flower-clusters. Where do they seem to be most commonly formed? How many flowers in a cluster?

21. Under the hand-lens, the scale-like leaf at the base of each flower-cluster and at the base of each flower.

DRAW a single flower-cluster enlarged three times.

Dissect and study a single mature flower. NOTE:—

22. The structure and arrangement of the calyx, corolla, stamens with fringed scale at the base of each, and the pistil. How many parts of each? DRAW a single flower enlarged, with calyx and corolla removed at one side.

Cut across the ovary, examine with hand-lens and DETERMINE:—

23. How many carpels and ovules are produced. DRAW.

Examine the matured capsule. How does it compare in size with the flower? How many seeds does it contain? Note the remnants of the calyx or the corolla at the base. From what does the capsule develop? DRAW.

Saprogenesis. The seeds fall to the ground where they lie dormant until the following spring. They germinate when conditions favorable to the growth of the host prevail. The vestigial root serves as an anchorage for the simple slender seedling, probably taking up only water from the soil. The seedling seems to be dependent largely on food stored in the seed, although chlorophyll is slightly developed. Seed germination and contact of the seedling with its host completes the primary life-cycle.

Secondary Cycles may be initiated during the growing-season by pieces of dodder stems broken off in cutting and harvesting the host, and falling upon healthy plants, infecting them. Pieces of dodder stems are very tenacious of life and thus afford a dangerous inoculum for spreading the pathogene in cultivated crops. The spreading of the pathogene from one individual host-plant to a neighboring one may also be regarded as a type of secondary infection.

REPORT

1. A farmer had a few spot-infections in an alfalfa field. After cutting the hay, the ground where the dodder occurred was spaded. Soon, however, the dodder appeared about the margins of the same areas. Write a letter outlining the measures to be taken to prevent further local spread of the pathogene and its dissemination over the field.

2. Give directions for determining whether or not there is dodder in the alfalfa seed, and if present, how to get rid of it.

TOBACCO MOSAIC

This is a disease not only of tobacco but also of many other solanaceous plants.

Plants affected with the mosaic disease, or as it is often called, calico, mottled-top, grey-top, or frenching, do not usually die. There is pronounced hypoplasia, especially of young leaves. There is a reduction in size, and chlorophyl-development is halted, so that a true chlorosis results.

SYMPTOMS

The leaves, and sometimes the branches, are the only organs of the host which exhibit symptoms. The plants are often much dwarfed and, in the case of *Nicotiana rustica* L., are sometimes killed. In some of the so-called immune varieties, it has been found that a progressive rotting of the tissues follows inoculations with the virus, but in such cases there are none of the usual symptoms of the disease. (U. S. Agr. Dept. Bul. 40:2.)

The symptoms vary from small blisters on the leaves, or the distinct dark-green islands in large areas of yellow, to an excessive suppression of the development of the lamina of the leaves, in which only the midrib remains normal.

On the entire plant. Study the diseased and healthy tobacco plants provided. OBSERVE:—

1. The comparative size and appearance of the healthy and diseased plants. SKETCH or COPY from U. S. Agr. Dept. Bul. 40, pl. VII. Maintain relative proportions between the healthy and diseased plants.

On the leaves. Examine the plants provided and locate, but do not remove, leaves showing various symptoms. OBSERVE:—

2. The characters of a healthy leaf, its regularity of surface and margin; its size. DRAW.

3. The peculiar green and yellow blotched appearance of diseased leaves. Note the blister-like elevations on the upper surface. Are they swellings or are they archings of the leaf? In which areas do they occur? Is there any relation between the veins and the green areas?

4. That the yellow areas of the leaf are apparently thinner than the green islands.

5. The comparative size of healthy and diseased leaves of the same age.

DRAW a diseased leaf on the same scale as the healthy leaf. Bring out in the drawing and labeling as many of the characters as possible.

6. The distorted leaves. These may be long and ribbon-like or the lamina of the leaf may be entirely undeveloped, although the midrib is not affected. DRAW, or COPY U. S. Agr. Dept. Bul. 40, pl. II, fig. 1.

7. That certain upper leaves may show a mottling (green and yellow) but without the accompanying distortions of the surface. Note how the green areas follow the veins. This is the phase that gives the names, grey-top and mottled-top, and occurs when the plant becomes infected just as the immature flower-heads appear. DRAW, or COPY U. S. Agr. Dept. Bul. 40, pl. I, fig. 2.

On the blossoms. Examine the flowers of the plant provided, or U. S. Agr. Dept. Bul. 40, pl. III and IV. Various species of *Nicotiana*

are shown. Note particularly the pink-flowered form of *N. Tabacum* L. and *N. paniculata*, if available. OBSERVE:—

8. The mottled or blotched appearance of the corollas of *N. Tabacum*. Note the variations and compare with the flowers of healthy plants.

9. That some of the flowers show a depauperate growth of the corolla; the stamens and stigma extrude.

10. That the buds may be depauperate and variously misshapen.

11. That the corolla-tubes of *N. paniculata* are bent and crumpled.

Make DRAWINGS of flowers and buds, both healthy and diseased, to show the various symptoms. Indicate all points, which cannot be shown in drawings, by notes in the margin.

On tomato leaves. Examine the tomato plant and OBSERVE:—

12. The mottling of the leaves; their texture and flexibility; their size and shape. DRAW and note characters in the labeling.

On petunia leaves. OBSERVE:—

13. Their color, size and flexibility. DRAW and label fully. The symptoms on other hosts are similar to these.

ETIOLOGY

The cause of this disease is unknown but there are various theories formulated to explain it. Some investigators (Woods, Chapman and others) believe that enzymes play an important, if not the primary, part in causing it; oxidase and peroxidase are specifically named. It is said that rough handling of the plants, such as breaking the roots in transplanting or severe pruning of the tops, will cause the disease. Disturbance of the equilibrium between root-absorption and leaf-transpiration is also held to be responsible for mosaic.

A much more plausible theory is that recently offered (by Allard and others). The disease is attributed to a filterable virus. It is held that this virus consists of an ultra-microscopic organism, bacterial or protozoan in nature. The extreme minuteness or plasticity of the organism probably allows it to pass through the pores of the Chamberland filter. Such phenomena are known in other cases.

Crush small pieces of healthy and diseased tobacco leaves in small drops of water, cover, and examine each with the high-power. OBSERVE:—

14. The absence of any evidence of an organism in the diseased tissues.

Pathological Histology. The effects on the structure of the leaf are very pronounced. Make thin cross-sections through diseased and healthy tobacco leaves, or if available study prepared slides. OBSERVE:—

15. The arrangement and contents of the cells of the palisade-layers, spongy parenchyma and epidermal tissues of the healthy leaves. DRAW in detail a section across the leaf.

16. The corresponding cells in the green and yellow area of the diseased leaf. Note the suppression of the palisade-tissue in the yellow areas. The relative thickness of the green and the yellow areas. DRAW a section across a diseased leaf at the same enlargement as for the section across the healthy leaf.

Pathogenicity Studies. Although the cause of the disease is not known, it is very easy to inoculate and obtain infections with the virus. A series of infection-experiments will be undertaken by the class as follows:

General instructions. Students will work in pairs, A, B and C, as assigned by the instructor. There will be four experiments, each involving the cooperative action of three pairs of students. Two pairs in each experiment will inoculate the healthy plants provided as directed, while the third pair will prepare check-plants.

Healthy tobacco, tomato and petunia plants will be provided. Do not touch these plants except when directed and then exactly as directed.

Experiment I. Inoculation by touching. PROCEED as follows:—

17. Thoroughly wash the hands and disinfect in alcohol provided.

18. Pairs A and B, squeeze and rub but do not crush, between thumb and finger, a diseased tobacco leaf; then immediately squeeze and rub with the same thumb and finger, leaves on the healthy plants. LABEL.

19. Pair C, do not touch diseased plants but squeeze and rub with thumb and finger (do not crush), the leaves on the healthy plants provided for checks. LABEL.

Experiment II. Inoculation with sap from diseased plants. PROCEED as follows:—

20. Pairs A and B. Each pair will thoroughly clean and rinse a mortar in sterilized distilled water. Grind three diseased tobacco leaves in a little sterilized distilled water in the mortar. The virus is thus freed from the leaf-tissue. Then very thoroughly wash the hands and disinfect in alcohol provided. Flame and cool a needle. Dip the point into the virus and prick the midrib of several young leaves of the healthy plants provided. Dip the needle each time before pricking. LABEL.

21. Pair C, thoroughly wash the hands and disinfect with alcohol. Do not touch diseased plants. Flame and cool a needle. Dip it in sterilized distilled water and prick the midrib of several young leaves of the healthy plants provided. LABEL.

Experiment III. Root inoculation. PROCEED as follows:—

22. Pairs A and B, thoroughly wash the hands and disinfect with alcohol provided. Do not touch the tops of the plants. Sterilize a scalpel by dipping in alcohol and flaming. Thrust the scalpel into the soil about the healthy plants provided, so as to cut some of the roots. Pour into the soil, where the roots were cut, some of the diseased-leaf extract prepared by pairs A and B in experiment II. LABEL.

23. Pair C, thoroughly wash the hands and disinfect with alcohol. Proceed as in number 22 but use sterilized distilled water to pour on the roots, instead of the diseased-leaf extract. LABEL.

Experiment IV. Inoculation through wounds. PROCEED as follows:—

24. Pairs A and B, thoroughly wash the hands and disinfect in alcohol provided. Break a young leaf from each of the healthy plants provided. Rub a diseased leaf between thumb and finger and touch the broken leaf-stub on the healthy plants but do not touch them elsewhere. LABEL.

25. Pair C, thoroughly wash the hands and disinfect with alcohol. Do not touch diseased plants. Break a young leaf from each

healthy plant provided. Rub the healthy leaf between thumb and fingers and touch the broken leaf-stub with the finger. LABEL.

These plants will be kept in the greenhouse and are to be examined every two days for two weeks. Make notes on the first appearance of the disease, i. e. (a) the number of days since inoculation; (b) the maturity of the infected leaves; (c) whether or not inoculated plants show infection.

After two weeks, the plants will be returned to the laboratory for final notes. At this time a tabulation-sheet of the results recorded will be made to obtain the class-result on the different types of inoculation.

REPORT

1. Give the class-record of the inoculation-experiments.
2. State the various ways that the virus can be disseminated.

What precautions should be taken to prevent dissemination. Explain.

METAPLASTIC DISEASES

SPONGOSPORA SCAB OF POTATOES

This slime-mold disease has come into considerable prominence in recent years because of its discovery in the seed-growing potato-sections of northern United States and Canada. It appears to be confined to the potato. Soil and climatic conditions seem to greatly limit the geographical range of the disease. It is evidently a disease of cool, temperate climates or high table-lands and valleys.

SYMPTOMS

The lesions of this disease are confined to the underground parts of the host; tubers, roots and stolons (rhizomes).

On the tubers. Three types of lesions are to be found on the tubers; the scab (the most common form), the wart-form and the canker-form.

Scab-form. Examine the specimens labeled "young sori." OBSERVE:—

1. The small brownish spots (ringed with India ink). These are the first evidences of the disease.

2. The smooth, brown mounds (also ringed with ink),—the covered sori of the pathogene.

3. The crater-like openings of some of the sori; they are mature and are opening for spore-dispersal; form, size and color of the sori.

Examine the specimens labeled "mature sori." OBSERVE:—

4. The large open brown sori. Note the torn and upraised cuticle about the margin.

5. The brown powder within the sorus. Pick it out with the needle.

6. The empty depressions left after this powder is disseminated.

Make a DRAWING of the entire tuber to show the sori and their distribution.

Make a series of enlarged DRAWINGS (at least four), showing the appearance of the sori in different stages of development.

Wart-form. This form is not common but may develop in severe cases in moist soils. Read Maine Bul. 227:94, and study figure 45; or study specimens if available. OBSERVE:—

7. That the lesions are more or less confined to the seed- or eye-end of the tuber.

8. That they are much more enlarged and gall-like than the ordinary scab-form. How does the location of the lesions help to explain their warty character?

SKETCH a tuber showing the wart-form of the disease.

Canker-form. Where the lesions are numerous and coalesce, the tubers may show, in an advanced stage of the disease, large deep cankers. This is held by some to be the result of secondary activities of the pathogene. (See Kunkel, Jour. Agr. Research 4:237.) Study the

illustration specimens provided; Maine Bul. 227, fig. 44; and U. S. Agr. Dept. Bul. 82, pl. III. **OBSERVE:**—

9. The large open cankers, extending deeply into the tuber and lined with the dead and decaying tissues of the host. **DRAW** a tuber showing this form of lesion.

On the roots and stolons. Study the specimens provided. **OBSERVE:**—

10. That here the lesions are gall-like in appearance; form and size; relation to the root or stolon, i.e. on the side only or involving the entire diameter? **DRAW**.

Comparative studies. There are a number of scab-like diseases of potato tubers, the lesions of which are more or less alike. A brief examination of the more common tuber diseases, in comparison with the *Spongospora* scab-lesions, is important. Examine, so far as available, the specimens and illustrations of the **FOLLOWING:**—

11. The black-wart disease. In addition to specimens and illustrations provided, study Pl. Ind. Bur. Circ. 52, pl. I-II. **SKETCH** a tuber to show the character of the lesions of this disease.

12. The common scab. Note the slightly raised irregular corky scab-spot. If this disease is serious, the tuber may be much checked and cracked. **DRAW**.

13. Silver-scurf. This disease does not cause a distinct scab; at most there is but a wrinkling or sloughing-off of the epidermis. (See Schultz, Jour. Agr. Research 6:345-346.)

14. Black scurf or *Rhizoctonia* scab and canker. This disease is very common but not often directly injurious to the tubers. Note the small dark-brown or black sclerotial bodies of the fungus scattered over the tuber. It is held by some that checking of the surface and even the formation of deep cankers are sometimes symptoms of this disease. (See specimens or Maine Bul. 230, fig. 71-73.) **Make DRAWINGS** to show the tuber-symptoms of this disease.

ETIOLOGY

The *Spongospora* scab is caused by the myxomycete, *Spongospora subterranea* (Wallroth) Johnson. It is generally distributed throughout northern Europe, Canada and northeastern United States. It is a native of South America where it occurs as a pathogene of the potato in its native home.

Life-history. This slime-mold does not differ materially from the saprophytic myxomycetes in its structure and life-history, except in so far as it has been modified by its parasitic habit.

Primary Cycle. Spore-balls in the soil constitute the source of inoculum for the primary infections. The spores may apparently remain dormant in the soil for several years. Remove some of the spore-mass from a sorus on the tuber provided; mount in potassium hydroxide and **OBSERVE:**—

15. The rather large dark spore-balls; color and structure. Each cell of the spore-ball is a spore. **DRAW**.

Pathogenesis. The spores in a single spore-ball all germinate at about the same time. There emerges from each cell of the spore-ball, a single, usually uninucleate ameba. (See Kunkel, Jour. Agr. Research 4, pl. XXXIX, fig. 1-2. **copy**).

These amebae soon fuse to form a small plasmodium which penetrates through and between the epidermal cells of the tuber. If conditions are not favorable to the formation of a plasmodium, the individual amebae may encyst. Later, when conditions become favorable, each protoplast escapes from its cyst-wall through a hole and may fuse with others to form the plasmodium.

Once within the tissues, the plasmodium spreads out under the epidermal tissues and down between the cells of the cortex. Study the prepared slides provided; or Jour. Agr. Research 4, pl. XXXIX, fig. 3-5, and pl. XL. OBSERVE:—

16. The densely granular protoplasm of the plasmodium in the intercellular spaces just beneath the epidermal layers.

17. The pseudopodia-like extensions of the plasmodium, forcing their way down between the cortical cells. The middle-lamella is dissolved and the cells are forced apart. The walls become soft and swollen.

18. That the pseudopodia penetrate the cell-walls and portions of the plasmodium enter; these become separated from the main body of the plasmodium.

Make a DRAWING to show the invading plasmodium, outlining the adjacent host-cells.

After a certain period of feeding, all the plasmodia in the host-cells of a particular lesion simultaneously form spore-balls. Examine prepared sections through sori showing mature spore-balls; or study U. S. Agr. Dept. Bul. 82, pl. I. OBSERVE:—

19. That the entire plasmodium is converted into spores. The host-cell serves as a sporangium.

20. The average number of spore-balls in a host-cell.

21. Remnants of the host-protoplast; nucleus, starch-grains, or cytoplasm.

22. Host-cells in which spore-formation is incomplete. Compare nuclei and cytoplasm of the pathogene and host.

Make enlarged and detailed DRAWINGS of the host-cells, (a) containing plasmodia; and (b) containing mature spore-balls.

Saprogenesis. What takes place after the spore-balls are set free in the soil is not certainly known. They probably germinate very shortly, the amebae passing out into the soil where they either form a plasmodium or encyst separately. Whether the organism can live a true saprophytic life in the soil is not known. It has, however, been cultured on artificial media.

Secondary Cycles probably occur, as the spores can apparently germinate as soon as they are mature. The phenomena of the secondary cycles are probably the same as those of the primary cycles. A peculiar type of secondary infection of the deeper tissues of the tuber, surrounding a mature sorus, has recently been described as the cause of the canker-form of the disease. (See Kunkel, Jour. Agr. Research 4:272-273.)

Pathological Histology. There is a marked stimulation of the host-tissues by *Spongospora subterranea*, as may be seen by the comparative study of the cells in the diseased and healthy regions of the tuber.

Cut thin cross-sections (or use prepared slides) through the healthy potato skin. Examine and OBSERVE:—

23. The single layer of thin cells on the outside,—the epidermis, overlaid by the brown cuticle.

24. Beneath the epidermis the layers of the cells which are regularly rectangular in shape. This is the corky hypodermis. The epidermis and hypodermis constitute the skin of the potato.

25. That from the hypodermis there is a rather abrupt transition to the larger, more globose cells of the cortex. Note that the cells of the cortex are filled with starch.

Make a DRAWING of the skin and cortex of the healthy potato.

Cut thin cross-sections (or use prepared slides) through sori of different ages; young and medium old. Put the sections from each into different watch-glasses.

Mount the thinnest sections from the very young sori and NOTE:—

26. Whether the hypodermis and epidermis are affected; the cells where the plasmodium enters are killed.

27. The granular mass between and within the cells,—the plasmodium of the parasite.

28. Any discoloration of the cells. Is there any enlargement of the cells? Note the starch-granules. Iodine solution will stain the starch-grains and make them more evident.

Examine the sections through the medium-aged sori. OBSERVE:—

29. The hypertrophy of certain of the cells; enlarged in which direction? These are called giant-cells. Is the plasmodium of the pathogene to be detected in any of them? Has spore-formation begun?

30. That some of these giant-cells are divided by cross-walls into several smaller cells; hypoplasia occurs as well as hypertrophy.

31. That this overgrowth of the cortical cells pushes up and eventually bursts through the skin.

32. Any effect of the pathogene upon the starch.

33. Effect on the nuclei of the host-cell.

34. Evidences of cambial activity in the uninvaded tissues beneath the sorus.

Make a DRAWING of a section through a sorus showing the histological conditions just prior to spore-formation.

REPORT

1. Give the methods of control to be employed against this disease and explain the philosophy of their use.

2. Consult the literature on club-root of crucifers and write a brief description of the life-history of *Plasmodiophora Brassicae* Wor. comparing and contrasting it with that of *Spongospora subterranea* (Wallr.) Johnson.

CLUB-ROOT OF CRUCIFERS

This is the best known of the slime-mold diseases of plants. It is a distinctly metaplastic disease, causing a marked overgrowth of the affected organs. It is widely prevalent both in the Old and in the New World. It affects most of the common cruciferous crops, especially cabbage, cauliflower and turnips.

SYMPTOMS

The roots are the only organs of the host that show lesions. The specimens provided exhibit the symptoms at different stages in the development of the disease and on different hosts. (See illustration specimens of the disease on various hosts.) Examine carefully the specimens provided and OBSERVE:—

1. The relatively small size and the much more fibrous and branched character of the roots of the healthy plants as compared with the diseased roots.

2. The much swollen and enlarged roots of the diseased plants; occasional healthy roots arising from the stems above the lesion. Why? Determine by diameter measurements how many times the diseased roots have been enlarged. Roots enlarged in this manner are said to be hypertrophied.

3. That the hypertrophy occurs either in the region of the root next to the stem or out in the feeding-roots.

4. That the hypertrophy involves the root in its entire diameter. It is not simply an outgrowth on the side of the root.

5. The specimens of club-root on the different hosts in the illustration jars. (See also Vermont Bul. 185, pl. II-V.) Determine whether there is any uniformity in the shape of the swellings which may be regarded as characteristic of this disease.

6. That the surface of the hypertrophied roots, especially in the old specimens, is much cracked and checked; to what due? These cracks allow the entrance of decay-producing fungi and bacteria. The succulent tissues quickly become soft and rotten and have a foul odor. The club-root pathogene itself does not cause the tissues to soften.

7. That the root beyond the hypertrophy, the branches and fine feeders, seem to be normal and healthy.

8. The abruptness with which the hypertrophy ceases at the base of the stem; this is a disease of the roots.

Nodules, tubercles and galls due to other causes are often produced on roots of plants, as for example: the nematode-gall caused by minute worms; legume-tubercles and crown-gall of trees caused by pathogenic bacteria. (See illustration specimens in jars.)

Make a DRAWING of a "clubbed" root along side of a healthy one. Maintain relative proportions.

Where the roots are badly clubbed, the plants fail to head, or form loose worthless heads. Affected plants often wilt or "flag" during the heat of the day but recover at night. These symptoms exhibited by the tops can be well observed only in the field.

ETIOLOGY

This disease is caused by the myxomycete, *Plasmodiophora Brassicae* Woronin. It is apparently an obligate parasite, saprogenesis being merely a period of rest. It has never been cultured saprophytically.

Life-history. This pathogene has, normally, an annual life-history but its spores may evidently lie dormant in the soil for several years in the absence of a cruciferous crop.

The **Primary Cycle** is initiated by spores that have remained dormant in the soil from a former season.

Pathogenesis. When the affected roots rot, the spores of the pathogene, mixed with the decayed host-tissue, remain in the soil. Selecting a root in an advanced stage of the disease, remove a bit and crush it in a drop of water on a slide; cover and examine with the high-power. **OBSERVE:—**

9. The minute globose hyaline spores, abundant among the crushed tissue-fragments. **DRAW.**

These spores germinate in the spring after the manner of those of the saprophytic slime-molds. The spore-wall cracks and the naked protoplast creeps out. Study the process as illustrated in Vermont Bul. 175, fig. 6. **COPY** to show several stages in spore-germination and the development of the swarmspore or myxameba.

These myxamebae, thus set free in the soil water, come in contact with nearby roots of susceptible plants and infection results. The single uninucleated swarmspore enters through a root-hair. Here by rapid nuclear division and growth, a multinucleate ameba or plasmodium is formed which penetrates into the cortical tissues. By direct migration through the cell-walls and by the division of the meristematic host-cells into which the ameba have penetrated, invasion of the various tissues is effected.

The plasmodium increases in size, destroys the protoplast of the cells in which it is lodged, and gradually fills the lumina of the cells. Make thin sections (or use prepared slides) through roots in the early stages of the disease. **OBSERVE:—**

10. The finely and densely granular multinucleate protoplasm of the pathogene in certain of the enlarged cortical or medullary ray-cells. Compare with the protoplasm of uninvaded cells nearby. (See Vermont Bul. 175, fig. 3-4.) **DRAW** an uninvaded and a normal host-cell, with contents, to show contrast.

These multinucleate plasmodia, when mature, divide into spores, each one-celled. Cut sections (or use prepared slides) of diseased roots containing mature spores. **OBSERVE:—**

11. That the infested cells are packed with spores; uniform size of spores; and absence of capillitium, characteristic of most saprophytic slime-molds. The host-cell serves as a sporangium. **DRAW** an invaded cell with spores.

12. In which tissues of the root the pathogene is most abundant.

Saprogenesis. The affected tissues gradually decay and the mature spores go into a resting- or dormant-period. No saprophytic activities, between spore-germination and infection by myxamebae in the spring, are known to take place.

Secondary Cycles. It is known that the spores will germinate at once or very shortly after they are formed. Therefore, if set free by the

early decay of affected tissues, they may cause secondary infections on roots of the same or later-planted hosts throughout the season. That this commonly happens appears certain. The phenomena are the same as those of the primary cycle.

Pathological Histology. Make freehand sections of a small healthy root (or use prepared slides). With low-power, **OBSERVE:**—

13. The core of the root, made up of alternating wedges of xylem (vessels and wood-cells) and medullary rays. Note that the xylem-wedges are frequently fused toward the center of the stele so that the medullary rays are seldom continuous from the cortex to the center.

Stain the section with a solution of iodine and **NOTE:**—

14. The yellow color taken on by the walls of the wood-cells which, with their thicker walls, are now more sharply contrasted with the medullary ray-cells.

15. The bark of the root surrounding the xylem-cylinder consisting in large part of the thin-walled cortex-tissues.

16. The groups of thick-walled sclerenchyma-cells in the outer cortex, stained deep yellow by the iodine. What is their function?

17. The ends of the phloem-strands, made up of small thin-walled cells in the inner cortex, each opposite a xylem-wedge.

18. The outermost tissues of the root, rough and torn,—the cork-layer.

Make a diagrammatic **DRAWING** three inches in diameter showing the anatomy of a healthy cabbage root as seen in cross-section. Make an enlarged and detailed **DRAWING** of a V-shaped portion of the cross-section showing form and arrangement of cells from center to circumference. Include (in outline) at least one vascular wedge.

Cut, mount and study a thin cross-section of a diseased root (or use prepared slide). **OBSERVE:**—

19. The abnormally increased diameter of the root. To what does this seem to be due; increase in size (hypertrophy) or numbers (hyperplasia) of the cells or both? (Compare with the section of the healthy root.)

20. The xylem-vessels in a few small groups. Note their isolation. To what part of the section are they chiefly confined? Why? (Compare Vermont Bul. 175, fig. 3.)

21. The ends of the phloem-strands composed of small regular cells arranged in rows around the outer end of the xylem-wedge. The relatively large amount of phloem, as compared with the amount of xylem; as compared with the amount of phloem in a bundle in the healthy tissue.

22. The abnormally broad medullary rays, resulting from the increased size of the cells. The dark masses in some of the cells,—the plasmodia or spore-masses of the parasite.

23. The cortex; the most abnormally increased of all the tissues. The cells are enlarged and perhaps increased in number. Some of them are filled with the brown masses of the parasite. Most of them show nucleus and cytoplasm. Note the entire absence of sclerenchyma or stone-cells; why wanting?

24. The layer of cork-cells on the outside. Are they affected?

Make a diagrammatic **DRAWING** showing the anatomy of the diseased root as seen in cross-section.

Make an enlarged and detailed DRAWING of a V-shaped portion of the section from the center to the outside, including one vascular wedge and medullary ray. Indicate the presence of the parasite in the cells by fine stipple-work.

REPORT

1. Describe fully the effect of the disease on the morphology of the root. What is meant by pathological morphology?
2. Explain the difficulties involved in the control of this disease by eradication measures.

LEGUME TUBERCLES

The bacterial tubercles which commonly occur on the roots of legumes are not generally regarded as evidence of a diseased condition. They are nevertheless pathologic in character. That the plant usually profits from their presence on its roots is no argument against their pathologic nature. The roots of all the common leguminous crops usually show these galls.

SYMPTOMS

Roots of various leguminous plants have been provided. (See also the illustration specimens in jars.) Study and compare the tubercles on the different hosts. OBSERVE:—

1. The shape, size and color of the galls; more or less characteristic for different hosts.

2. Location on the root-system; deep or shallow; on small or large roots; lateral or terminal?

3. Types of galls; simple and compound (branched). Make DRAWINGS of the tubercles on pea, bean, clover and alfalfa.

ETIOLOGY

The cause of these tubercles is a bacterial pathogene, *Bacterium leguminosarum* (Frank) E. F. Smith. It is probable that this species includes a number of more or less distinct biologic forms.

Life-history. This pathogene is a highly specialized parasite, developing normally in living plant-tissues which it stimulates to overgrowth. The galls which it induces are usually annual growths. It is probable that in most cases only primary cycles occur.

The **Primary Cycles** are initiated in the spring and summer on the developing rootlets of the host.

Pathogenesis. Active individuals in the soil-water constitute the inoculum. Study a mount from an actively growing pure culture provided. OBSERVE:—

4. The form and size of the bacteria. They are actively motile, having one polar flagellum (which can be seen only in specially stained preparations).

These bacteria in the soil accumulate in small groups on the sides of the root-hairs. By means of some secretion, they gradually soften the wall of the root-hair and penetrate it. The root-hair is stimulated to bend or curl about the mass of penetrating bacteria in a characteristic fashion. Study the prepared slides showing root-invasion. OBSERVE:—

5. The curled tip and the infection-thread extending through the length of the root-hair. DRAW.

6. Under the demonstration microscope an infection-thread within the tissue of the root. Note the funnel-like swellings produced just before the host-walls are penetrated. DRAW.

These bacteria, having penetrated to meristematic cells, multiply rapidly and stimulate abnormal cell-division, resulting in the formation of the gall.

Cut thin sections of tubercles. Examine the central bacterioidal region with the high-power. OBSERVE:—

7. The numerous infection-threads extending from cell to cell. This shows how new cells, formed by the primordial meristem, are infected from the adjoining bacteria-filled cells.

8. The immense number of bacterioids in each invaded cell.

9. That they are for the most part not rod-shaped like the individuals in the soil, but are club-shaped to Y-shaped; most evident in the central cells. As the organisms in the host-cells or in old cultures cease to divide, they gradually take on these so-called involution-forms. (See Virginia Ann. rept. 1909-1910:136-137.)

Some of the individuals in the outer cells of the bacterioidal tissue remain active and rod-shaped. The degenerate bacterioids are attacked by the enzymes of the host, and the nitrogen stored within them is made available for the growth and development of the host.

Saprogenesis. With the death and disintegration of the tubercle-tissues at flowering-time of the host, the active rod-shaped bacteria in the gall are set free in the soil. Whether during their sojourn in the soil, they lead an active saprophytic existence, though highly probable, is not certain. They can be readily grown on artificial media. They are not known to produce spores.

Pathological Histology. Longitudinal sections of tubercles and normal root-branches are provided. Examine and OBSERVE:—

10. The general shape and appearance of the longitudinal section of the tubercle. Compare with that of the young lateral root.

11. The kind of tissues from which the tubercles and the roots respectively arise.

12. The vascular strands. Note their condition at the base of the bacterioidal region and the arrangement of the xylem and phloem. Compare with the condition in the young lateral root.

13. The primordial meristem and root-cap. Are they present both in the tubercle and in the lateral root?

14. The cortex. Is it continuous with that in the root?

15. The epidermis. Is it present in the tubercle as well as in the lateral root?

16. The bacterioidal region. Where is it located in the tubercle? Note the size and contents of the cells. Note the outer cell-layer limiting the bacterioidal region,—the starch-sheath.

What metaplastic conditions predominate in the tubercle, hypertrophy or hyperplasia?

Make a diagrammatic DRAWING of a longitudinal section of a root-tubercle; similar comparative DRAWING of the lateral root. In these drawings only an outline need be made to indicate the comparative shape of the lateral root and the tubercle, and to locate the various tissues mentioned above.

Examine the prepared cross-sections of the tubercles. OBSERVE:—

17. The bacterioidal region, ring of bundles, cortex and starch-sheath. DRAW.

18. The effect of the bacterial invasion on the organs of the infected cells; nucleus, cytoplasm and walls, as compared with those of a homologous healthy cell. (Read Fred, Virginia Ann. rept. **1909-1910**:123, first paragraph.) Detail in enlarged DRAWINGS a healthy and an infected cell showing the cell-organs and contents.

REPORT

1. Present the evidence to show the pathological nature of the legume tubercles. Read in this connection:

Smith, E. F. Bacteria in relation to plant diseases **2**:97-146. 1911.

Fred, E. B. The infection of root-hairs by means of *Bacillus radicola*.

Virginia Agr. Exp. Sta. Ann. rept. **1909-1910**:123-137; and other references given in the text.

PEACH LEAF-CURL

This is a common and frequently destructive disease of peaches in nearly every peach-growing section. It is especially severe in regions subject to cold, wet weather in the spring. One of the most profitable commercial varieties, the Elberta, is particularly susceptible.

SYMPTOMS

It is chiefly a disease of the leaves, although the current season's shoots may also suffer. Shoot-infection is especially serious when nursery-stock is affected. Flowers and fruit rarely show lesions of the disease.

On the leaves. Examine the material provided. OBSERVE:—

1. The flexible character of the normal portions of the leaf. Tear it and note the texture.

2. The wrinkled and distorted portion of the leaf affected by the curl. Tear and compare with the healthy portion as to texture and flexibility. Explain the difference. Compare with the healthy portion as to color and thickness.

3. The character of the distortion. In which direction as regards the long axis is the diseased leaf wrinkled? Why? DRAW.

Examine photographs and Cornell Bul. 276, fig. 83-86. OBSERVE:—

4. That the diseased leaves soon drop, partially or wholly defoliating the tree. Later new leaves are usually put forth.

On the twigs. Study the specimens provided. OBSERVE:—

5. The dwarfing of the shoot due to a shortening of the internodes, giving it a rosette-like appearance.

6. The much thickened fleshy stem,—hypertrophied; more or less definite linear ridges often extending down on the previous years growth.

7. The badly infected leaves. The leaves on diseased twigs are more severely affected than on the healthy twigs, for invasion becomes general or systemic and the leaf-fundamentals become diseased as they are formed. Most of the affected shoots gradually die. (Compare U. S. Agr. Dept., Veg. Phys. and Path. Div. Bul. 20, pl. V.)

On flowers and fruit. If specimens or photographs are available, study the character of the lesions on these organs. DRAW.

The curl on the leaves affects the fruit indirectly by preventing setting or by causing it to fall. (Study U. S. Agr. Dept., Veg. Phys. and Path. Div. Bul. 20, pl. XVII, and table 33, p. 124.)

ETIOLOGY

The cause of this curl is *Exoascus deformans* (Berkley) Fuckel, one of the primitive ascomycetes of the order Protodiscales. No definite fruit-body is formed, the asci developing directly from the mycelium in an exposed hymenium over the surface of the lesion.

Life-history. Relatively little is positively known about the life-history of *E. deformans*. Opinions with respect to the life-habits of this pathogene are based very largely upon circumstantial evidence. No true asexual stage of the fungus is known.

The Primary Cycles are initiated in the early spring when the buds swell and begin to open, by inoculum which has overwintered on the hairy bud-scales. Presumably this inoculum is some kind of spores; just what kind and how they reach the bud-scales is not known.

Pathogenesis. Examine the buds on the peach twigs provided.
OBSERVE:—

8. The hairy character of the outer bud-scales. Remove one and study it under low-power. Minute globose spores may sometimes be detected, lodged among the long hairs on the scale. These may belong to the leaf-curl pathogene, at least present evidence warrants the belief that such spores lodged in this way serve as the primary inoculum. Make an enlarged diagrammatic SKETCH of an infested bud.

The spring rains which cause the buds to swell and to open, also cause the spores to germinate and send forth a slender germtube which enters the bud between the scales and penetrates the tender leaves. As the leaves develop, the mycelium within spreads between the cells and stimulates the growing tissue, resulting in a curling and fluting of the blade. The germtube may also penetrate into the growing tissues of the developing twig.

Make thin sections through diseased leaves or twigs; clear in chloral hydrate; wash and stain for some time in eosin or methyl blue; wash and mount in water. LOCATE:—

9. The intercellular mycelium, fitting tightly in, and conforming with the intercellular spaces. No haustoria are formed. DRAW a bit of the mycelium with adjoining host-cells.

Branches of this mycelium pass toward the surface between the epidermal cells. Make thin tangential sections from the surface of the diseased leaf. Stain as above and study under the high-power. MAKE OUT:—

10. The very abundant subcuticular mycelium; its irregular septate form; its branching and anastomosing habit. From this mycelium arise the asci. DRAW.

Study cross-sections (freehand or prepared) of a leaf through a lesion bearing asci. OBSERVE:—

11. The row of enlarged and angular epidermal cells of the host; their organs and contents.

12. The layer of long, more or less angular asci, containing spores, standing at right angles on the epidermal cells.

In the prepared slides, OBSERVE:—

13. Some of the asci, small and young with deeply stained protoplasmic contents and nuclei.

14. Mature asci containing deeply stained ascospores or empty; shape and size of the spores; number in an ascus.

DRAW a portion of the epidermis showing asci, old and young, in different stages of development.

15. That these ascospores often increase in number within the ascus by budding like yeast (known by the large number of small spores which the asci contain). DRAW.

Saprogenesis. The ascospores when mature are shot through a rupture in the top of the ascus. Their further history is unknown. The mycelium in a lesion produces but one crop of ascospores and then apparently dies with the leaf which soon falls to the ground. There is probably some saprogenic activity on the part of this pathogene between the

dissemination of the ascospore in early summer and primary infection the following spring, but of this nothing is known.

Secondary Cycles may or may not occur. This point is not determined. The ascospores germinate in water by budding or by the protrusion of a short germ tube when placed on leaves of the host. Artificial inoculation of the host has never resulted in infection. (Read U. S. Agr. Dep., Veg. Phys. and Path. Div. Bul. 20, bottom of 38 to 40.) COPY figures from pl. IV, showing two types of spore-germination.

Pathological Histology. Study the prepared cross-sections of a diseased leaf. Select a place in the section where the diseased tissues may be compared with the normal. OBSERVE:—

16. The increased thickness of the leaf in the diseased portion. To what is this due, increase in size, or number of cells (hypertrophy or hyperplasia)?

17. Which tissues are most affected, palisade or spongy parenchyma?

18. The effect on the number, shape and arrangement of the palisade-cells; chloroplasts and cell-walls (hypoplasia and metaplasia). These cells were attacked before they began to differentiate into palisade-cells.

19. The effect on the epidermal cells on the upper surface of the leaf; on the lower surface.

Make a detailed DRAWING showing comparatively the histological structure of the diseased and the normal portions of the leaf.

REPORT

1. Write a letter to a peach-grower, giving brief and current directions for controlling peach leaf-curl.

LEAF-BLISTER OF OAKS

This is a very common leaf-disease of oaks. Frequent epiphytotics, with more or less serious effects on the host, occur in the southern portion of the United States, while in the north very little damage is done.

SYMPTOMS

The leaves only are affected. In the dry material, NOTE:—

1. The blisters; more or less circular; varying greatly in size.
2. That in some cases the spots have become confluent and the entire leaf is much curled.
3. That the blister is usually convex on the upper surface of the leaf and concave on the lower; however, this is not a constant character.
4. That the upper or convex surface of the blister is, in young spots, lighter green than the normal leaf-tissue but, that in the older spots, it becomes more or less flecked with brown areas.
5. That the lower or concave surface of the blister is a bluish gray in the fresh condition but changes on drying to a dirty or brownish gray. The coloration and velvety appearance of the under surface is due to the fruiting stage of the causal fungus.

Make a SKETCH to show the symptoms of leaf-blister.

ETIOLOGY

The pathogene causing the leaf-blister of oaks is *Taphrina coerulescens* (Desmazieres and Montagne) Tulasne, an ascomycetous fungus belonging to the order Protodiscales. No fruit-body is formed by these primitive ascomycetes. The asci are developed directly from the mycelium in a hymenium on the surface of the host. The peach leaf-curl pathogene, *Exoascus deformans* (Berkley) Fuckel, is closely related to *T. coerulescens* (Desm. and Mont.) Tul.

Life-history. As is the case with the peach leaf-curl fungus, the life-history of *T. coerulescens* is imperfectly known. Ascospores, borne on the blisters in the spring and summer, bud in the asci and form numerous secondary spores. These spores are supposed to hibernate in some way and initiate primary infections the following spring.

The **Primary Cycles** are initiated in the early spring by the overwintered inoculum (kind unknown).

Pathogenesis. The pathogenic activities of this fungus are exhibited entirely by the sexual or ascigerous stage. In fact no true conidial form of this pathogene or, for that matter, of any of the entire order, the Protodiscales, to which it belongs, is known.

Examine the stained cross-sections of a blister and NOTE:—

6. The mycelium. To what portion of the leaf is it confined?
7. The layer of more or less angular asci standing at right angles to the lower epidermal cells.
8. The wedge-shaped root-like projections pushed down between the lower epidermal cells and which serve as hold-fasts for the asci.
9. The spores within the asci. They are small and either eight in number or numerous (probably in some cases as many as a hundred). Some authors have suggested that the budding of the eight ascospores,

which results in these numerous asexual spores without a mycelial stage intervening, constitutes in reality asexual reproduction, and that these spores are conidia. This is largely theoretical, however, to account somehow for a conidial stage.

DRAW a portion of the lower epidermis as seen in prepared cross-sections showing mycelium, holdfasts, and young and old asci with spores.

Saprogenesis. The whereabouts and activities of this pathogene during saprogenesis, if such a period ensues, is unknown.

Pathological Histology. Although the mycelium of *T. coerulea* is confined to the space it makes for itself between the lower epidermal cells and the cuticle, the effect of the parasite is seen throughout the thickness of the leaf. In the stained cross-sections, find and compare the healthy with the diseased part of the leaf. NOTE:—

10. The hypertrophy in the lower epidermis, spongy mesophyl, palisade-mesophyl and upper epidermis.

11. The hyperplasia in the lower epidermis and spongy mesophyl-tissues.

12. That in the normal tissue the cells of the lower epidermis are longer than broad. The effect of the fungus is to cause these cells to increase greatly in size and, being so tightly packed together, they elongate in a direction perpendicular to the surface of the leaf. Finally septa may be laid down at right angles to their long axes.

13. That the cells of the spongy mesophyl are increased both in number and size and are tightly packed together, largely eliminating the intercellular spaces so common in the healthy tissue.

14. That the cells of the palisade-mesophyl are longer and wider than the normal.

15. That the cells of the upper epidermis are somewhat larger and more globose than the normal.

16. That the chloroplastids in the diseased cells are much smaller and fewer in number.

Make DRAWINGS showing (comparatively) the histological structure of the diseased and normal portions of the oak leaf. Preserve correct proportions between the two drawings.

REPORT

1. From the literature on leaf-blister of oaks and the methods used for control of peach leaf-curl, suggest possible measures of control for leaf-blister.

BLACK KNOT OF PLUMS AND CHERRIES

This is one of the most striking diseases of stone-fruits. It is an indigenous disease peculiar to our native wild species of plums and cherries and is frequently destructive to our cultivated forms, especially certain varieties of plums and sour cherries. The disease is not known to occur outside of North America.

SYMPTOMS

The disease affects only the woody parts of the host, usually only the twigs, though it may extend from spurs to the larger limbs or body of the tree. (See illustration specimen.) Two seasons are required for the full development of the knot.

Study the material provided showing the character of the knots in the spring of the first season. OBSERVE:—

1. The relation of the knot to the twig. Is the entire circumference involved?
2. The shape and color of the gall.
3. The relation of the swollen tissues to the epidermis and cork-layer of the healthy part.
4. The character of the surface of the knot. The olivaceous lumps or patches scattered over the brown woody surface. These are the conidial stromata. These stromata bear mature conidia in the spring.

Compare several specimens on the above points. SKETCH a typical knot.

The galls are probably initiated at buds or short spurs. Study the very young galls provided and OBSERVE:—

5. The unruptured swellings on the side of the spur or in the twig nearby. What does this indicate as to the infection-court? DRAW.

Study some of the specimens showing the character of the galls the second season. OBSERVE:—

6. The darker color and more hard and woody character of the galls.
7. The continuous black perithecial stroma covering the exposed surface of the gall.
8. The minute pimple-like perithecia arising from, and crowded over the surface of the stroma.
9. Secondary galls arising just above or below some of the primary ones; known by their lighter color and conidial stromata.

DRAW to show the character of secondary galls and secondary gall-formation.

10. That affected twigs may be bent at right angles to the knotted side. DRAW.

ETIOLOGY

The black knot is caused by the ascomycetous fungus, *Plowrightia morbosa* (Schweinitz) Saccardo.

Life-history. This pathogene presents in its life-history one striking variation from the usual type; it requires two years to complete a life-cycle.

The **Primary Cycles** are initiated in the early spring by inoculum produced on two-year-old knots.

Pathogenesis. The ascospores constitute the inoculum for the primary cycles. With the scalpel remove a few of the mature perithecia and crush in a drop of water or potassium hydroxide on a slide. Cover and OBSERVE:—

11. The ascospores; their form, number in ascus, number of cells and color. DRAW.

If living material is available, study the germinating ascospores.

These ascospores mature and are discharged from the perithecia, usually during March. They are carried to the buds on the previous year's growth and infect the twig. The development of the pathogene and the reaction of the host is slow. Slight swelling of the infected twig begin to show by autumn. It develops rapidly during the early days of the following spring and burst through the epidermis. On the exposed surface, the scattered conidial stromata develop on which conidiophores bearing conidia are matured in late spring or early summer. These conidia are disseminated and initiate secondary cycles.

Study cross-sections through the conidial stromata and OBSERVE:—

12. The structure of the stroma; relation to mycelium in host; size of cells; color.

13. The erect conidiophores; septate or non-septate?

14. The conidia; shape, size, color and number of cells; where and how attached to conidiophores?

Make a DRAWING to show these structures.

After the conidia are matured and disseminated, the velvety covering of conidiophores gradually disappears, giving place to the continuous black crust-like stroma of the sexual stage which has been developing, so that by autumn the knot shows the black carbonaceous character, distinctive of its second season, and the minute pimple-like perithecia (fruit-bodies of the sexual stage) developing on the surface.

During the first warm days of the spring (two years after primary infection) ascospores are formed within the asci in these perithecia.

Examine with the hand-lens, the surface of mature galls provided. OBSERVE:—

15. The minute black bodies crowded together over the surface; shape and size; adherent to each other or separate on the stroma?

16. The apex of the perithecia often depressed; a minute opening at the center of each,—the ostiolum.

Study prepared sections through mature galls showing longitudinal sections of perithecia. MAKE OUT:—

17. The relation of perithecia to each other and to the stroma; character of the stroma as compared with that of the conidial stage.

18. The structure of the perithecial wall; ostiolum; relation of asci and paraphyses to the perithecial cavity.

Crush in water one of the mature perithecia. OBSERVE:—

19. The structure of the asci, ascospores and paraphyses.

Make an enlarged DRAWING of a longitudinal section of the perithecium and supporting stroma to show these structures in detail.

The ascospores mature in early spring and are shot, during rainy periods, from the asci out of the ostiolum. Caught by the breeze, they are carried to the trees and infection occurs in the swelling buds or developing shoots. Thus in two full years is the primary life-cycle, starting with ascospores, completed in the production of another crop of ascospores.

Saprogenesis. As the pathogene is continuously associated with its living host, it may be regarded as having no saprogenic phase in any of its life-cycles. However, as a matter of fact, there is often little living host-tissue in the galls during the period of the maturing of the ascospores.

Secondary Cycles are initiated by the conidia, produced on the galls, during the next growing-season after that in which the primary infections were initiated. These secondary cycles require slightly less than two full years for their completion and, like the primary, end with the production of ascospores. The pathogene structures, developed during the secondary cycles, and the host reactions are like those of the primary cycles.

Mycelium spreading from the primary (or secondary) galls into adjacent healthy tissues often give rise to secondary galls. This is regarded by some as secondary infection (Stewart, Amer. Jour. Bot. 1:114).

Pathological Histology. The effect of this pathogene on the host is one of stimulated overgrowth of the invaded tissues. Cut with a knife or scalpel (not a razor) across one of the galls provided for this purpose. Trim the surface smooth and with hand-lens, MAKE OUT:—

20. The anatomical distortions that have occurred in the elements of the vascular cylinder and bark, comparing constantly with the structures in the normal portion of the twig. Make a diagrammatic DRAWING of the entire cross-section showing normal and pathological anatomy. Label corresponding tissues in each.

Study under the microscope the prepared sections through young one-year old galls. OBSERVE:—

21. The character of the host-tissue on which lies the stroma of the fungus; size, shape, contents of cells and tissue-relations.

22. The mycelium in the tissues; inter- or intracellular?

23. The mycelial strands and fans in some places; their pseudo-parenchymatous character. What is their relation to the stroma?

24. The depth toward the pith to which the pathological effects are evident.

25. The broadening of the rays in the diseased region; due to hyperplasia or hypertrophy?

26. The isolation of the cambium at the outer ends of the xylem-wedges; wanting in the abnormally broad rays.

27. The inhibition of xylem-elements and the abnormal development of parenchyma in the xylem-wedges. The enlargement of the gall the second year is largely due to a marked hypertrophy of these parenchymal cells.

28. The isolated groups of summer-wood vessels in the xylem-parenchyma just inside the cambium.

29. The isolated xylem-elements in the diseased bark. These result from the misplaced and broken segments of the cambium opposite the broad rays.

30. The proportional increase in thickness of bark and wood in the diseased region. The outer bark sluffs off before conidial formation.

Make a full-page DRAWING, detailing cells in the different regions, to bring out the histological changes above emphasized.

REPORT

1. Prepare a diagram showing the two types of life-cycles in *Plowrightia morbosa* (Schw.) Sacc. and their relation to each other.

CORN SMUT

This is the most common and most noticeable disease of corn. It is sometimes known as boil-smut to distinguish it from the head-smut. It affects all varieties of field-corn, pop-corn and sweet corn. Some varieties of sweet corn suffer severely from this smut.

SYMPTOMS

All parts of the plant above ground are subject to the disease. Unlike most of the cereal smuts, invasion is local. The lesions are characterized by marked hypertrophy.

On the stalk. Study the specimen provided and OBSERVE:—

1. At what points on the stem the galls are usually located.
2. The large size of the boils and the comparatively small area from which they arise. Is there any evidence of injury to the adjacent host-tissues?
3. The texture of the boils. They are more firm in fresh specimens, especially when yet immature; the tough fleshy covering of such galls.
4. The color; compare old and young boils.
5. The dark dusty spore-mass filling the mature boils.

A relatively large portion of the hypertrophy is composed of pathogene structures (mycelium or spores).

Make a SKETCH to show stem-boils.

On the leaves. Lesions are comparatively rare on the leaves. In the specimens provided, OBSERVE:—

6. The small boils arranged in rows parallel with the veins. Do they seem to arise from or between the veins; which side of the leaf?
7. Any effect on the tissues adjacent to the boils; opposite the boils.

SKETCH leaf-lesions.

On the ear. Affected ears are very common, especially those with exposed tips. In the specimens provided, OBSERVE:—

8. The form, size and location of the boils.
9. That they are each an enlarged kernel. Find kernels in different stages of hypertrophy.

SKETCH a diseased ear to show healthy kernels and diseased ones, hypertrophied in various degrees.

On the tassel. The boils of this smut are very common on tassels but may be confused with those of the head-smut. (See Kansas Bul. 62:199 and pl. VI, VIII–X.) In the specimens provided, OBSERVE:—

10. That the boils on the tassels are but hypertrophied parts of the flowers.
11. That only flowers here and there in the tassel are affected.
12. That the character and degree of malformation varies in different flowers.

Carefully dissect a healthy and a diseased flower. Make a series of comparative DRAWINGS to show the effect of the disease on different organs of the male flower.

ETIOLOGY

The pathogene causing corn smut is *Ustilago Zeae* (Beckmann) Unger, one of the Ustilaginaceae. It was first described as one of the puff-balls by Beckmann in 1768 and has been, since that time, a frequent subject for investigation by mycologists and phytopathologists.

Life-history. It differs markedly in its life-habits from most of the common cereal smut pathogenes. There are normally, in temperate climates at least, only the primary cycles.

The **Primary Cycles** are initiated at any favorable time during the period of active growth of the host. Overwintered chlamydospores, usually in the manure on the field, are the sources of inoculum.

Pathogenesis. Some of the chlamydospores from an old smut-boil have been germinated in manure-extract on the slides provided. Examine under the microscope and OBSERVE:—

13. The large globose spiny dark-brown chlamydospores.

14. From some of these, the protruding hyaline promycelium (basidium).

15. The crack in the epispore through which the promycelium is protruded.

16. The septa in the promycelium, from below each of which arise one to several slender pointed sporidia. They are often produced in great numbers; sometimes in chains.

DRAW a germinating chlamydospore showing abundant sporidial production. These sporidia are carried by the wind to the growing corn. Some of them lodge on the growing tissues of the host near the joints within the leaf-sheath, the emerging silks, or the blossoms on the tassels. Here they germinate, sending out germ-tubes that penetrate these embryonic tissues, and give rise to the mycelium. This mycelium does not spread through the tissues to any great distance from the point of infection; every boil probably arises from a separate infection. The hyphae grow in great masses into the hypertrophied tissues of the boil and form the greater bulk of the excrescence. Chlamydospores are soon developed in chains within short branches of this mycelium. The rest of the mycelium gelatinizes and disintegrates more or less.

To study the mycelium in the host-tissue about the base of the boil, make thin longisections of the stalk, just beneath the boil. Stain with methyl blue or eosin. Examine and in the pith-tissues, OBSERVE:—

17. The slender hyphae in the intercellular spaces or penetrating the cells; often in strands of several hyphae or gnarled in the intercellular spaces.

18. Short knotted or irregularly swollen short branches sent into the cells,—the haustoria.

DRAW to show the mycelium in the host-tissue.

To study the distribution of the mycelium in the boil and the formation of the chlamydospores, examine prepared sections through a partly matured boil. Make DRAWINGS to show the structures studied.

Saprogenesis. Mount some of the black spore-mass in potassium hydroxide. Study with the high-power and OBSERVE:—

19. The shape, size and color of the mature chlamydospores; the short spines which thickly beset the spore-wall.

20. Any variation in the spores as to size and shape. Look for bits of mycelium.

Make an enlarged DRAWING of several of the spores.

These chlamydo-spores go with the fodder into the manure and finally out on the land. They winter over in the manure and germinate during the following season. Their dependence upon saprophytic nourishment in germination is evidenced by the fact that they develop sporidia much better in a nutrient solution, e.g. manure-extract, than they do in water.

REPORT

1. Explain why seed treatments, so generally effective with smut diseases, will not control the corn smut.

HOLLYHOCK RUST

This is a rust disease peculiar to a number of malvaceous hosts. It is indigenous to Chili where it was discovered in 1852. It is especially destructive to the hollyhock and is now known as a common disease of this perennial in nearly every temperate country of the globe.

SYMPTOMS

The lesions of this disease occur on all the above-ground parts of the host, but are usually most numerous on the leaves. In severe cases, the calyx and seed-capsules are affected.

On the leaves. Examine the specimens provided and OBSERVE:—

1. The cushion-like pustules scattered over the leaf-surface,—sori of the pathogene; most abundant on which side?

2. The variations in size and shape of the sori.

3. Color. This varies with the age of the lesion. The youngest lesions usually show a distinct bright-yellow color, especially on the upper surface or where they occur on the veins and petioles. A little later the fully developed sorus (telium) takes on a rusty brown color and is surrounded by a yellowish zone. When the spores of the telium begin to germinate, the color becomes a grayish brown. As the pustule dies it turns black.

4. The thickened character of the leaf-tissue in well developed lesions; there is slight hypertrophy.

5. In old lesions, the dead and shriveled sorus surrounded by a narrow zone of necrotic leaf-tissue. This dead tissue sometimes falls out, leaving small round holes in the leaf-blade.

DRAW a diseased leaf to show the characters of the lesions at various stages.

Where the lesions are very numerous, the entire leaf-blade withers, turns brown and finally falls from the stem. The general effect of a severe infection is best observed in the field.

On the stems and the petioles. Examine the specimens provided. OBSERVE:—

6. The shape and size of the pustules; usually larger than those on the leaves. Why?

7. The elongated dead zone about the older lesions. How deep does the lesion extend? The lesions are very limited. They frequently dry out and disappear except for a large oval scar left by the healed wound.

DRAW several stem-lesions.

On the fruit. Study the lesions on the flowers provided (best studied on the fruits of *Malva rotundifolia* L). OBSERVE:—

8. The lesions on the calyx; compare with those on leaves and stems.

9. The sori on the seed-carpels; color, size and location. DRAW. The pathogene is often distributed on seed infested in this manner.

ETIOLOGY

The hollyhock rust is caused by a uredinaceous fungus, *Puccinia Malvacearum* Montagne.

Life-history. It is a so-called lepto-form or Leptopuccinia, i.e. it has neither aecia nor uredinia, producing only telia. The teliospores usually germinate *in situ*, as soon as mature, to form promycelia with sporidia.

The **Primary Cycles** occur on the first leaves developed from the perennial root in the spring. The sources of inoculum are the old dead leaves, diseased the previous season, or overwintered living leaves in which the pathogene has hibernated.

Pathogenesis. The inoculum consists of the sporidia. These are formed on the promycelium which develops from the teliospore. Teliospores which are produced late in the autumn often do not germinate at once but remain dormant on the old dead leaves. More commonly, however, late infections on immature green leaves of the hollyhock or on those of *Malva rotundifolia* do not develop telia until early spring. The teliospores in these telia germinate and sporidia for the primary infections are formed.

Study germinating teliospores; or illustrations in Phytopath. 1, pl. XIII. OBSERVE:—

10. The long slender basidium (promycelium), most commonly developed first from the apical cell.

11. The densely granular protoplasm of the apical portion of the basidium; its final division into four cells, and the formation of a sporidium from each.

12. That sometimes the four apical cells separate before sporidia are developed.

Make a series of DRAWINGS to show sporidial formation.

The sporidia are carried by the wind or splashing rain to the leaves nearby. Here they quickly germinate sending forth a short germtube which penetrates the host.

DRAW several germinating sporidia. These sporidia are uninucleate and develop within the host-tissues a septate uninucleate mycelium. Study thin sections (freehand or prepared) through a lesion. OBSERVE:—

13. The slender, often matted, septate mycelium in the intercellular spaces or forcing the host-cells apart. Are haustoria formed?

14. The matted mycelial stroma from which arise the slender teliospore-stalks, forming the telial sorus.

15. The teliospores; form, size, color, septation and thickness of walls.

16. Young spores in various stages of development.

Make a detailed DRAWING of a telial sorus in section with adjacent host-tissue and intercellular mycelium.

The teliospores germinate *in situ*, giving rise to the sporidia which, scattered by wind or rain, initiate secondary cycles.

Secondary Cycles are initiated repeatedly throughout the season and conform in their development to that of the primary cycles except that in the case of late secondary cycles, teliospores, which have developed, may fail to germinate at once and overwinter on dead leaves and stems. Very late secondary cycles on overwintered living leaves may fail to develop telia until the following spring.

Pathological Histology. Make a comparative study of diseased and healthy areas in prepared cross-sections of the leaf. DETERMINE:—

17. What pathologic changes, if any, in number, form and size of cells has resulted from the presence of the pathogene.

18. What changes have taken place in the protoplasts of affected cells; nucleus, cytoplasm and chloroplasts.

Make DRAWINGS of diseased and healthy tissues to show the pathologic effects.

REPORT

1. Detail a plan for controlling the hollyhock rust in border-plantings. Give satisfactory reasons for each step in the procedure.

RUST OF CEDAR AND APPLE

This is a very common disease of the apple in many sections of the country where the red cedar grows. It is especially prevalent in the Mississippi Valley, northeastern New York, New England, Virginia, West Virginia and other southern states. The effect of the pathogene on both hosts is very marked.

Most of the rust diseases of rosaceous plants are harbored by some species of cedar during the winter. In the case of the rust here considered, the pathogene overwinters on the red cedar, *Juniperus virginiana* L. The summer form of the pathogene occurs on wild crab, *Pyrus coronaria* L., as well as on the cultivated apple. The rusts appearing on pear, quince and hawthorne are usually caused by species distinct from that causing the apple rust; all, however, are closely related.

SYMPTOMS

The symptoms differ strikingly on the two hosts.

On the apple. The material provided was collected in midsummer. Examine the affected leaves and OBSERVE:—

1. That the pathogene produces a leaf-spot. How does it differ from other leaf-spots?

2. The differences in the character of the spot on the upper and lower surfaces as to color and definiteness of outline.

3. The presence of black pimple-like structures on the upper surface,—the pycnia (spermagonia) of the pathogene, probably functionless male structures.

4. The groups of brownish, somewhat cylindrical or fimbriate structures on the lower surface,—fruit-bodies called aecia (aecidia).

SKETCH to show the above characters.

The general effect of the pathogene is to give the trees a striking yellowish color in contrast to the dark green of the healthy trees nearby. Early defoliation follows severe infections. There is a marked variation in the susceptibility of varieties. Wealthy and Jonathan are especially susceptible.

Lesions appear on the fruit when it is about one-fifth grown. They resemble those on the foliage. Examine diseased fruits provided and OBSERVE:—

5. The color and location of the diseased area.

6. The dwarfing effect on the fruit. Recall the effect on the leaf.

7. The kinds of pathogene-structures developed in these lesions.

DRAW a diseased fruit.

On the red cedar. The material provided was collected in early-spring (April). OBSERVE:—

8. The large brown galls. Form, size, consistency, and surface characters.

9. The spore-cushions scattered over the surface,—the telia (teleuto-sori).

10. The attachment and the relation of the gall to the twig. Each gall results from an enlargement of a single leaf, according to certain authors or of the stem in the axil of the leaf, according to others.

Remove one of the healthy leaves and NOTE:—

11. The size, form and relation to the twig.

SKETCH a normal leaf and a diseased or galled leaf; maintain proper proportions.

The most striking sign of this disease on the red cedar is exhibited during warm spring rains in April and May. From the telia scattered over the gall, long yellow gelatinous teliospore-masses protrude, giving the effect, from a distance, of large yellow fruits; whence the popular names cedar-apples and cedar-flowers.

ETIOLOGY

This disease is caused by one of the Uredinales, *Gymnosporangium Juniperi-virginianae* Schweinitz. As already noted, the pycnial stage (O) and the aecial stage (I) occur on the leaves and fruits of the cultivated apple and wild crab. The telial stage (III) is found on the red cedar. A uredinial stage (II) is wanting.

Life-history. The apple rust-fungus continues in close association with the living tissues of its hosts throughout its entire life-history.

Primary Cycle

Pathogenesis. The inoculum for the primary cycle consists of sporidia which are produced on the red cedar. They are developed on a promycelium which in turn arises from a teliospore. Moisture of six or more hours duration is necessary for sporidial formation. As soon as the humidity decreases sufficiently to cause appreciable evaporation, the sporidia are forcibly ejected. This occurs from April to June. Sporidia are carried to the apple by the wind. Germination follows within a short time in the presence of moisture. Two types of germination occur; one in which germ-tubes develop directly from the sporidia, and the other in which secondary sporidia are formed on a short germ-tube. The latter type is more common. (See *Phytopath.* 3:282, fig. 1, and *Nebraska Rept.* 22, pl. III, fig. 5; or *Virginia Tech. bul.* 9, fig. 9.) COPY to show types of sporidial germination.

The germ-tube arising from the secondary sporidium penetrates the cuticle. In the case of the leaf, this occurs on the upper surface. The mycelium developed from the sporidial germ-tube ramifies through a limited area of the leaf, with the result that tissue-changes are effected and certain fruit-bodies (O and I) are developed. Examine the apple leaves again, using hand-lens. OBSERVE:—

12. The pycnia on the upper surface of the leaf; their form and distribution.

13. The aecia on the lower surface of the leaf; their form, distribution and size. The long exerted peridium (aecial-wall),—a prominent character of aecia of this fungus. Rust-fungi showing aecia with such peridia belong to the form-genus, *Roestelia*. Accordingly the apple rust-fungus was formerly called *Roestelia Pyrata* (Schw.) Thaxter.

Make enlarged DRAWINGS to show the pycnia and aecia as they appear under the hand-lens.

Mount in potassium hydroxide some of the peridial cells and aeciospores from the aecium. Study and DRAW to distinguish them.

Examine prepared slides showing sections through lesions on an apple leaf. OBSERVE:—

14. The abnormal tissue-developments. Which tissues are involved? How affected?

15. The pycnia; their form and contents.

16. The aecia; their position with reference to the pycnia, the relative size of the two structures; their form, structure and contents.

Make a DRAWING to show the points observed in 14, 15 and 16. (See Virginia Tech. bul. 9, fig. 13.)

The pycnial stage always precedes the aecial stage in the development of those rust-fungi possessing these spore-forms. Aeciospores never re-infect the apple.

Secondary Cycles

Pathogenesis. The inoculum for the secondary cycles consists of aeciospores which are discharged from July first to the end of the growing-season. They are blown to the red cedar and there initiate the cedar-apples. The aeciospores apparently must undergo a period of rest before germination. The question of whether infection occurs in the fall or spring is yet unsettled. The way in which the germtube gets into the cedar is not known. The manner of germination is not essentially different from that of other fungous spores. A simple germtube is formed; this on entrance into the host-tissue develops into a mycelium which stimulates the growing cells, and a gall results. This becomes evident the following June. Study the young green galls provided. These increase in size during the summer and become brown and full sized by winter. The fungus passes the second winter of its life-history as mycelium in the full-sized gall. From the gall provided, remove a small bit of tissue to a drop of water on a slide and crush. OBSERVE:—

17. The hyphal threads throughout the mount. The character of the host-cells.

Examine prepared sections through mature galls. NOTE:—

18. That the gall shows cortex and vascular elements.

19. The mycelium of the fungus and its relation to the host-cells. Are haustoria present?

In early spring the mycelium forms, at certain places beneath the cortex, stromata from which are developed the telial horns. Examine galls showing these horns. NOTE:—

20. That the horns arise from galls as described.

21. The depression at the base of each horn.

22. The color, size and shape of the horns. What is their consistency when dry; when wet?

On the end of each long stalk-cell (sporophore) a teliospore is developed. These are matured by March and April. Germination of the teliospore results in the formation of a promycelium which bears sporidia. Make a mount of teliospores from the horns provided. OBSERVE:—

23. The size, shape and color of the teliospores.

24. Their manner of germination. (See Virginia Tech. bul. 9, fig. 8, and Nebraska Rept. 22, pl. 3, fig. 5.)

Make a DRAWING to show the points observed in paragraphs 17 to 24 inclusive. (See Virginia Tech. bul. 9, fig. 5-8.)

There is no saprogenesis in either the primary or secondary cycles.

REPORT

1. Discuss the relation of apple rust to environment.
2. Summarize the practical value of such information.

BLISTER-RUST OF WHITE PINE

The blister-rust of five-needled pines and its alternate phase, the felt-rust of currants and gooseberries, has recently become an important disease in eastern United States. Previous to the importation of diseased white pine nursery-stock, this disease was confined to Europe. Recently it has assumed an epiphytotic character in several localities in northeastern United States.

SYMPTOMS

The symptoms produced by this disease will be studied in the order of their seasonal sequence on pine, currant and gooseberry.

Blister-stage on pines. In the material and illustrations referred to, NOTE:—

1. That the first evidence of the disease on the white pine is an indefinite, discolored canker-like area on the trunk or smaller branches.

2. That in some cases a slight or marked hypertrophy of the bark occurs. (See Pl. Ind. Bur. Bul. 206, fig. 3, 4 and pl. I; or Farmers' Bul. 742, fig. 3, 5 and pl. 1.) DRAW OR COPY.

3. That often a diseased tree may be detected by its stunted growth and bushy appearance. (See Farmers' Bul. 742, fig. 1.)

4. That the first external evidences of the pathogene are the minute light-yellow, bladder-like swellings which exude drops of liquid. These small pustules are pycnia (spermagonia) and within them are found minute spore-like bodies called pycnospores (spermatia). These spermatia are most generally believed to be non-functioning male gametes. They do not serve in any way to propagate the fungus. Usually the pycnial stage is formed several months after infection and during the autumn just previous to the production of aecia.

5. The large hemispherical or pustular bladdery aecia pushed out from cracks in the invaded bark. DRAW.

6. The aecia, at first yellow and enclosed by a papery peridium; later irregularly ruptured, allowing the yellow powdery contents to escape,—the aeciospores. The aecia are produced in early spring two or more years after infection.

7. The pock-like depressions in the bark where aecia have been borne. These characteristic markings on the cankered area constitute a reliable means of identification of diseased trees at any time of the year after the first crop of aecia has been produced. DRAW.

Summer stage on currants and gooseberries. Examine the diseased leaves of the different species of *Ribes* provided. OBSERVE:—

8. That in the case of certain species definite leaf-spot lesions are produced; in others the fruit-bodies of the pathogene alone constitute the first signs of the disease. DRAW.

9. The very small hemispherical pustules,—the uredinia, in groups on the under sides of the leaves. They are at first covered by the epidermis and are different in color from the remainder of the leaf, being slightly lighter green. DRAW as seen with the hand-lens.

10. That later the uredinia become more or less orange-yellow in color; the covering (composed of leaf-epidermis and peridium) is ruptured allowing the rusty yellow contents,—the uredospores, to escape.

Felt-rust stage on currants and gooseberries. In the material of gooseberry and currant leaves provided, NOTE:—

11. The conspicuous hair-like growths,—the telia, often arising from the uredinia and more or less scattered over the entire under surface of the leaves. DRAW. In some cases the felt-rust stage accompanies the first uredinia early in the summer but it is typically an autumnal stage, occurring in August and September.

ETIOLOGY

The blister-rust of five-needled pines is caused by the basidiomycetous rust-fungus, *Cronartium ribicola* Fischer von Waldheim. This name is based on the telial stage. The aecial stages of rusts, forming blister-like aecia on the bark or needles of conifers, have all been placed in the form-genus *Peridermium*. The blister-rust of five-needled pines is commonly known in its aecial stage as *Peridermium strobi* Klebahn.

Life-history. This pathogene is a heteroecious fungus requiring two very different hosts for the development of its complete life-history. All the spore-stages produced by rusts are formed by this fungus: aecia (I) on bark of pines; uredinia (II), telia (III) and promycelia (IV) on the leaves of currants and gooseberries. The pycnia (O) are produced on the pine previous to the appearance of the aecia.

Primary Cycles are initiated by the aeciospores produced in the aecia on the pine in early spring. When the peridium is ruptured, the aeciospores dust out and are blown about by the wind.

Pathogenesis. The primary inoculum, aeciospores from the pine, must reach young leaves of currants and gooseberries in order to function in continuing the development of the fungus. The aeciospores cannot infect the pine.

Mount some of the yellow powder from the aecia in potassium hydroxide, cover and examine with the microscope. OBSERVE:—

12. The size, shape and color of the aeciospores; thickness and markings of the spore-wall. DRAW.

The aeciospores, in the presence of moisture, germinate on the under sides of gooseberry and currant leaves and the germtubes penetrate into the stomatal cavities. After the establishment of a food-relation with the host-cells surrounding the sub-stomatal cavity, a mycelium is produced which spreads in a limited area. Within about two weeks after infection the uredinia are formed. In the material studied under symptoms of this stage, NOTE:—

13. The general character of the uredinia in different stages of development.

14. The size, shape and color of the uredospores under the microscope.

15. The thickness and markings of the spore-wall.

DRAW a typical uredospore showing above points.

In the prepared slides showing stained sections through uredinia, NOTE:—

16. The epidermis of the leaf which was raised up and finally ruptured by the developing uredinium.

17. The peridium enclosing the contents of the uredinium.

18. The sporophores each bearing a single uredospore. How many nuclei in each spore?

Make a **DRAWING** of the uredinium in section as seen under the microscope.

The first production of uredospores furnishes inoculum for the secondary cycles, which are repeated often during the summer on currants and gooseberries.

In some cases the telial stage is produced from the same mycelium which produced the first crop of uredospores, and thus the primary cycle may be carried forward to the production of sporidia from the germinating teliospores.

In the prepared slides showing uredinia in section, **NOTE:—**

19. The stroma developed just beneath the uredinium and the radiating mycelium extending into the host-tissue. Find a haustorium. **DRAW.**

In prepared slides showing the telial stage, **OBSERVE:—**

20. That the telial horns arise directly from the same stroma that bore the uredospores. **DRAW.**

21. The telial horn, composed entirely of teliospores cemented together into a compact mass. **DRAW.**

22. The germination of the teliospores, forming a short germtube which is septate,—the promycelium. **DRAW.**

23. The sporidia produced on sterigmata from each cell of the promycelium.

The sporidia, produced as the last step in the primary cycle, initiate secondary cycles on the five-needled pines. The sporidia are formed during periods of wet weather. The teliospores germinate as soon as formed, there being no rest-period required. There is no saprogenesis in the life-history of this pathogene.

Secondary Cycles. There are two sorts of secondary cycles in the case of this pathogene; those initiated by sporidia on pine, and those initiated by uredospores in currant and gooseberry leaves. The repeated succession of secondary cycles, initiated by uredospores, causes the rapid spread of the fungus from the currants and gooseberries harboring the primary cycles, until by the end of summer the fungus may be disseminated over several square miles.

These secondary cycles on gooseberry and currant leaves duplicate the primary cycle, in that after the uredospores are dispersed, telial horns are produced from the same stromata and upon germination of the teliospores, promycelia bearing sporidia are formed. The sporidia of the primary and secondary cycles are the inoculum which initiate the secondary cycles on the five-needled pines.

Very probably the sporidia are forcibly discharged from the sterigmata and they are blown to the pine. They are short-lived and must find suitable conditions for germination soon or they will not function. The sporidia germinate by a germtube and this penetrates uninjured pine bark initiating a new mycelium. The steps in the development of the symptoms on pine have already been observed. Spermagonia may be formed in the autumn of the next year following infection. Aecia may be formed the second spring after infection or the production of the first crop of aecia may be delayed for several years.

From the material of affected pines, take a bit of the peridium; stain with eosin and mount in water. OBSERVE:—

24. The character of the cells of the peridium. DRAW.

Study the sections through white pine twigs bearing aecia. NOTE:—

25. The position of the aecia in relation to the tissues of the twig.

26. The stroma at the base of the aecium and the radiating mycelium in the host-tissue.

27. The origin of the peridium.

28. The sporophores each bearing a chain of aeciospores.

DRAW in detail an aecium with the surrounding host-tissues.

REPORT

1. Illustrate graphically the steps in the primary and secondary cycles of this pathogene.

2. Illustrate graphically the nuclear phenomena exhibited during the life-history of this rust.

MISTLETOE OF JUNIPERS

The mistletoe of junipers is common in western and southwestern United States. A great deal of damage is annually caused by this and other mistletoe diseases of trees.

SYMPTOMS

In the juniper material provided, NOTE:—

1. The irregular and gnarled hypertrophies.
2. The mistletoe plants firmly rooted in the host-tissue.
3. That in many cases, the growth of the branches beyond the parasite is stopped.

Make DRAWINGS showing the above symptoms.

ETIOLOGY

The parasite causing this disease of junipers is *Phoradendron juniperinum* Englemann, one of the many species of the family Loranthaceae, a group of the flowering plants. This mistletoe has only very aborted leaves. They are mere scales closely appressed to the stem.

Life-history. The inoculum in the case of the mistletoe is the small pulpy berry with its single inclosed seed. The berries are mature in Texas about December. Birds seek the berries for food and serve to a large degree as the disseminating agents. The stickiness of the pulp of the berry causes it to adhere closely to the branch of the host.

In the material provided, OBSERVE:—

4. The character of the berry, its pulpy flesh and the enclosed seed. DRAW. (See also bulletin mentioned below, fig. 1.)

COPY Pl. Ind. Bur. Bul. 166, fig. 2 and 3, showing the method of germination of the mistletoe seed and the penetration of the host-tissue.

The seedling establishes its root-like sinker in the conducting tissue of the host and obtains, not only water, but a certain amount of raw and modified plant-food from the host. The mature parasite blooms in December. The seeds require one year to develop and mature, so that they are ready for dissemination the next December.

Pathological Histology. In the small branches of juniper cut longitudinally and transversely, study the roots of the mistletoe. OBSERVE:—

5. The size, shape and number of roots sent from a single plant into the host-tissue. What tissues are invaded? DRAW.

In the prepared cross-sections of juniper twigs, OBSERVE:—

6. The penetration of the host-tissues by the mistletoe root.
7. The elements present in the normal wood and bark.
8. The elements present in the root of the mistletoe.
9. The ultimate connection between the conductive elements of the host and of the parasite.

Make DRAWINGS; (a) to show diagrammatically the relation of the tissues of the host and parasite, outlining the tissues; (b) to show in detail the ultimate connection of the conductive elements.

REPORT

1. Outline a practical method for the control of the mistletoes in forest areas. Explain why it should be effective. Indicate the weak points in the program.

OEDEMA

This disease, although seldom of much importance under field conditions, sometimes becomes destructive in the greenhouse. It is of interest to the plant pathologist in that it is not caused by an organism, but may readily be induced by changes in temperature and humidity or by the application of dilute solutions of certain toxic substances. It affects many plants but especially tomatoes and cabbage.

SYMPTOMS

The cabbage leaves provided were gathered from plants in a vegetable garden. The seedlings had been grown in the greenhouse, and the plants set out in the spring. The season was cold and rainy. OBSERVE:—

1. That all the lesions are on the under side of the leaf. Is there any special reason why they should not be on the upper surface?

2. That these lesions are small raised spots, and are roughened like scab-spots on potatoes. Do they appear on the veins or on the areas between the veins? Is there any definite shape to the intumescences?

Make a DRAWING of a portion of a diseased leaf.

Study and SKETCH the symptoms as exhibited by potato, tomato or other plants provided.

ETIOLOGY

No organism is connected with this disease. It is caused by conditions which produce abnormal turgidity in the spongy parenchyma-cells of any part of the plant above the ground. It has been induced artificially by attaching the cut end of the stem to a hydrant where the water-pressure was very strong. The same result has also been accomplished by spraying the leaves with a dilute solution of ammoniacal copper carbonate. The larger drops always kill the tissue outright, but the smaller particles of spray cause an abnormal increase of the parenchymal cells below the epidermis. In the greenhouse, the disease results when the soil is warm, so that the roots take in a large amount of water, and the atmosphere is cold and the place poorly lighted, so that transpiration does not take place as fast as absorption. This causes such a pressure in the thin-walled tissues that hypertrophy and rupture result.

Under direction of the instructor, the students may undertake some experiments in the greenhouse to demonstrate the causal relation of some of the factors just mentioned.

Pathological Histology. Cut thin cross-sections through a lesion or use prepared slides. OBSERVE:—

3. That the lesion is made up of a number of abnormally large cells, the ends of which are entirely exposed.

4. That this exposure is due to the rupturing and the breaking-away of the epidermal cells. Are fragments of these still remaining?

5. That all the hypertrophied cells are those of the parenchyma.

6. Changes in the organs of affected cells.

Make DRAWINGS to show comparatively the conditions in diseased and healthy tissues.

REPORT

1. Describe in detail the procedure and results in the etiologic experiments, conducted under the instructor's directions.

TERM-PAPER SUBJECTS

Each term-paper exercise will consist in preparing a short paper on one of the diseases listed. The paper shall be essentially a text on the disease chosen.

INFORMATION

1. The instructor will designate those subjects in the appended list from which selections for each term-paper are to be made. Any disease in the designated list may be chosen. The same subject may be chosen by more than one student, if approved by the instructor, but it is generally to the student's advantage to have as few working on the subject as possible because of the limited number of available copies of some of the articles to be consulted.

2. Students choosing the same subject will be expected to work independently in consulting the literature and preparing the paper. Any evidence of disregard of this expectation will rule a term-paper out of consideration.

3. If so desired, the paper may be illustrated. This is not required, but will add to its value. Illustrations may be in the form of text-figures or plates.

4. The student may write on some disease not listed herein, if permission is granted by the instructor.

5. The selection of a subject for the first term-paper will be made at the beginning of the laboratory exercise on Literature of Plant Diseases. Selection of subjects for each of the other term-papers will be made later, at a time designated by the instructor.

6. One laboratory period will be devoted to each term-paper. The work in the laboratory will consist of obtaining references and getting such information and assistance from the instructor as may be needed to make clear the method of procedure.

PROCEDURE

After having located and listed the references bearing on the subject chosen, as outlined in the exercise on Literature of Plant Diseases:—

7. Select three or more of the most important articles (usually the most recent).

8. Read and abstract these carefully according to directions given in the outline on Literature of Plant Diseases, p. 13-14.

9. If specimens and materials of the disease chosen are available, they should be studied supplementary to the literature consulted.

10. Spread out the abstracts and laboratory notes and proceed to correlate and arrange the data in each, according to the following outline:—

OUTLINE FOR ARRANGING DATA IN TERM-PAPER

THE BLACK ROT OF POMACEOUS FRUITS

(References are to be arranged, in order of importance, thus:—)

Brooks, Charles, and DeMeritt, M. Apple leaf-spot. *Phytopath.* 2:181-190, pl. 17, fig. 1-6. 1912.

Paddock, W. The New York apple tree canker. New York (Geneva) Agr. Exp. Sta. Bul. 163:180-206, pl. I-VI.

HOSTS

PLANTS AFFECTED

VARIETAL SUSCEPTIBILITY

Of fruit to rot.

Of foliage to leaf-spot.

DISEASE

NAMES

HISTORY AND RANGE

IMPORTANCE

Nature of losses.

Injury to fruit.

Injury to foliage.

Reduction of yield.

Amount of losses.

SYMPTOMS

On the fruit.

On the leaves.

On the limbs.

ETIOLOGY

Name, history and classification of the pathogene.

Pathogenicity.

Life-history.

The Primary Cycle.....*Pathogenesis.*

Inoculation.

Incubation.

Infection.

Saprogenesis.**The Secondary Cycles**

(If secondary cycles occur and require special consideration, repeat headings as for primary cycle.)

ECOLOGY

Pathological Histology.**Influence of climatic factors.****Influence of soil factors.**

CONTROL

EXCLUSION

Quarantine measures.

ERADICATION

Elimination.**Cultivation.****Rotation.****Disinfection.**

PROTECTION

Manipulation of normal environment.*Interfering with disseminating-agents.**Modifying soil-reactions.**Modifying temperature-relations.**Modifying moisture-relations.***Application of inhibiting substances.***Spraying.**Dusting.**Dressing and filling wounds.*

IMMUNIZATION

Selection.**Crossing.****Feeding.**

NOTES

11. The heads and subheads appearing down the middle of the page are quite definitely fixed and will be the same for any subject chosen. They should appear, properly placed, in the manuscript submitted.

12. The subheadings indicated along the left of the page will vary in character more or less, depending on the disease in hand, *except* in the case of those under etiology which will be uniformly the same for all diseases.

13. Additional subheads of the type at the left of the page may be inserted where the nature of the data requires it.

14. Omission of heads and subheads is to be made when there are no data to record thereunder.

15. References in the body of the text should be inserted at the end of the sentence.

Where one of the references in the list at the beginning of the paper is to be referred to, enclose in parenthesis the author's name, date of publication, colon and page, thus:—(Jones, 1914:27). The author's name may be omitted when it is clear, from the context, to which article reference is made.

When an article not listed in the references given at the beginning of the paper, is to be cited, the parenthetical insertion must include the author's name, name of publication (abbreviated), volume or its equivalent, colon, and pages on which the data are to be found, thus:—(Duggar, Fungous Dis. p. 237. 1909) or (Peck, Journ. Myc. 7:10-14).

16. Confine the discussion to the disease on the host or hosts specified and to closely related hosts.

17. The reference-sheets, including the abstracts, must be handed in with the term paper.

LIST OF SUBJECTS

FIELD CROPS

1. Alfalfa root-gall caused by *Urophlyctis alfalfae* Magnus.
2. European root-rot of alfalfa caused by *Rhizoctonia Crocorum* Fries.
3. Alfalfa stem-blight caused by *Bacterium Medicaginis* (Sackett) E. F. Smith.
4. Smuts of barley caused by *Ustilago Hordei* (Persoon) Kellerman and Swingle, and *Ustilago nuda* (Jensen) Kellerman and Swingle.
5. Clover anthracnose caused by *Colletotrichum Trifolii* Bain (= *C. caulivorum* Kirchner).
6. Clover rusts caused by *Uromyces Trifolii* (Hedwig) Léviellé on white clover, and *Uromyces fallens* (Desmazieres) Kern on red clover.
7. Stem-rot of clover caused by *Sclerotinia Trifoliorum* Eriksson.
8. Ear-rot of corn caused by *Diplodia Zeae* (Schweinitz) Léviellé.
9. Cotton anthracnose caused by *Glomerella Gossypii* (Southworth) Edgerton (= *Colletotrichum Gossypii* Southworth).
10. Root-rot of cotton and alfalfa caused by *Ozonium omnivorum* Shear.
11. Helminthosporium diseases of cereals caused by *Helminthosporium gramineum* (Rabenhorst) Eriksson, *H. teres* Saccardo, *H. sativum* Pammel King and Bakke and *H. turcinum* Passerini.

12. Downy mildew of grasses caused by *Sclerospora graminicola* (Saccardo) Schroeter.
13. Hop mildew caused by *Sphaerotheca Humuli* (Fries) Burrill.
14. Crown-rust of oats caused by *Puccinia Lolii* Nielson (= *Puccinia coronata* Corda.)
15. Early blight of potatoes caused by *Alterania Solani* (Ellis and Martin) Jones and Grout (= *Macrosporium Solani* Ellis and Martin).
16. Leaf-roll and curly-dwarf of potatoes, cause unknown.
17. Black leg of potatoes caused by *Bacillus phytophthorus* Appel (= *Bacillus Solanisaprus* Harrison, = *Bacillus atrosepticus* van Hall, = *Bacillus melanogenes* Pethybridge and Murphy).
18. Potato scab caused by *Actinomyces chromogenus* Gasperini (= *Streptothrix scabies* (Thaxter) Cunningham, = *Oospora scabies* Thaxter).
19. Black wart of potatoes caused by *Chrysophlyctis endobiotica* Schilbersky.
20. Brown rot or wilt of Solanaceae (potatoes, tomatoes and tobacco) caused by *Bacterium Solanacearum* E. F. Smith (= *Bacillus Solanacearum* E. F. Smith, = *Bacillus Nicotianae* Ueda).
21. Rice blight or blast caused by *Piricularia grisea* (Cooke) Saccardo.
22. Rice smut caused by *Tilletia horrida* Takahashi.
23. Cobb's sugar-cane disease caused by *Bacterium vascularum* (Cobb) G. Smith.
24. Root disease of sugar-cane caused by *Marasmius plicatus* Wakker.
25. Smuts of sorghum caused by *Sphacelotheca Sorghi* (Link) Clinton, *Ustilago cruenta* Kühn and *Sphacelotheca Reiliana* (Kühn) Clinton.
26. Timothy smut caused by *Ustilago striaeformis* (West.) Niessel.
27. Timothy rust caused by *Puccinia Phlei-pratensis* Eriksson and Henning.
28. Root-rot of tobacco caused by *Thielavia basicola* (Berkley and Broome) Zopf.
29. Wheat scab caused by *Fusarium cumorum* (W. G. Smith) Saccardo.
30. Flag-smut of wheat caused by *Urocystis Tritici* Körnicke and the stem-smut of rye caused by *Urocystis occulata* (Wallroth) Rabenhorst.
31. Take-all of wheat caused by *Ophiobolus graminis* Saccardo or *Ophiobolus herpotrichus* (Fries) Saccardo.
32. The rôle of insects in the dissemination of pathogenic fungi.

FRUIT CROPS

33. Fruit-spot of apple caused by *Phoma Pomi* Brooks (= *Cylindrosporium pomi* Brooks).
34. European apple tree canker caused by *Nectria galligena* Bresadola.
35. Blister-canker of apple caused by *Nummularia discreta* (Schweinitz) Tulasne.
36. Northwestern anthracnose of apple caused by *Neofabrea malicorticis* (Cordley) Jackson (= *Gloeosporium malicorticis* Cordley).
37. Apple rots caused by the following ascomycetous pathogenes; *Alternaria* sp., *Cephalothecium roseum* Corda., *Volutella fructi* Stevens and Hall, *Endomyces Mali* Lewis, *Sclerotinia cinera* (Bonorden) Schroeter.
38. Water-core of apple, cause not definitely known.
39. Crown-gall of apple and other fruit-trees caused by *Bacterium tumefaciens* E. F. Smith and Townsend.

40. Gummosis caused by *Bacterium Cerasus* (Griffin) (= *Bacillus spongiosus* Aderhold and Ruhland).
41. Leaf-blight (shot-hole or yellow-leaf) of cherries caused by *Coccomyces hiemalis* Higgins (= in part, *Cylindrosporium Padi* Karsten).
42. Powdery mildew of cherry caused by *Podosphaera Oxyacanthae* (Fries) de Bary.
43. Citrus canker caused by *Bacterium Citri* (Hasse) E. F. Smith (= *Pseudomonas Citri* Hasse).
44. Sooty mold of citrus caused by *Meliola Penzigi* Saccardo (= *M. Cammeliae* (Cattaneo) Saccardo).
45. Citrus scab caused by *Cladosporium Citri* Masee.
46. Wither-tip of citrus caused by *Glomerella cingulata* (Stoneman) Spaulding and von Schrenk (= *Colletotrichum gloeosporoides* Penzig).
47. Blue mold or rot of citrus caused by *Penicillium digitatum* (Fries) Saccardo, *P. italicum* Wehmer and *P. expansum* Link.
48. Brown rot and canker or gummosis of citrus caused by *Pythiacystis Citrophthora* E. H. Smith and R. E. Smith.
49. Pod-rot and canker of cocoa caused by *Phytophthora Faberi* Maublanc.
50. Leaf-spots of coffee caused by *Pellicularia Koleroga* Cooke, *Stilbella flavida* (Cooke) Kohl. and other fungi.
51. An anthracnose of currants caused by *Pseudopeziza Ribis* (Libert) Klebahn (= *Gloeosporium Ribis* (Libert) Montagne and Desmazieres).
52. The European currant rust caused by *Cronartium Ribicola* Fischer von Waldheim.
53. Powdery mildew of gooseberries and currants caused by *Sphaerotheca Mors-uvae* (Schweinitz) Berkley and Curtis.
54. Silver-leaf of fruit-trees caused by *Stereum purpureum* Fries.
55. Root-rot of fruit trees caused by *Armillaria mellea* (Fries) Quélet and *Clitocybe parasitica* Wilcox.
56. Injury to fruit-trees caused by frost.
57. Grape anthracnose caused by *Gloeosporium ampelophagum* (Passerini) Saccardo (= *Sphaceloma ampelinum* de Bary).
58. Dead arm or necrosis and ripe rot of grapes caused by *Cryptosporella Viticola* (Reddick) Shear (= *Fusicoccum Viticulum* Reddick).
59. Powdery mildew of grapes caused by *Uncinula necator* (Schweinitz) Burrill.
60. Olive knot caused by *Bacterium Savastanoi* E. F. Smith (= *Bacillus olea-tuberculosis* Savastano).
61. Black spot or bacterial shot-hole of peaches and plums caused by *Bacterium Pruni* E. F. Smith.
62. California peach blight caused by *Coryneum Beijerinckii* Oudemans.
63. Leaf-blight (shot-hole, yellow-leaf) of plums caused by *Coccomyces Prunophorae* Higgins (= in part, *Cylindrosporium Padi* Karsten).
64. Die-back and canker of peaches and plums caused by *Valsa leucostoma* Fries.
65. Peach scab caused by *Cladosporium carpophilum* Thümen.
66. Plum pocket caused by *Exoascus Pruni* Fuckel.
67. Powdery mildew of the peach caused by *Sphaerotheca pannosa* (Fries) Léviellé.
68. Rust of stone-fruits caused by *Puccinia Pruni-spinosae* Persoon.
69. Peach yellows, cause unknown.

70. Little peach, cause unknown.
 71. Leaf-spot of pear caused by *Mycosphaerella sentina* (Fries) Schroeter (= *Septoria Pyricola* Desmazieres).
 72. Pear scab caused by *Venturia Pyrina* Aderhold.
 73. Rust of the pear caused by *Gymnosporangium globosum* Farlow.
 74. Leaf-blight and fruit-spot of quince and pear caused by *Fabrea maculata* (Léveillé) Atkinson (= *Entomosporium maculatum* Léveillé).
 75. Rust of quince caused by *Gymnosporangium clavipes* Cooke and Peck.
 80. Anthracnose of raspberries and blackberries caused by *Gloesporium venetum* Spegazzini.
 81. Orange-rust of raspberries and blackberries caused by *Gymnoconia interstitialis* (Schlectendall) Lagerheim (= *Caecoma nitens* (Schweinitz) Burrill).
 82. Cane-blight of raspberries caused by *Leptosphaeria Coniothyrium* (Fuckel) Saccardo.
 83. Double blossom of dewberry caused by *Fusarium Rubi* Winter.
 84. Powdery mildew of strawberries caused by *Sphaerotheca Humuli* (Fries) Burrill.

GARDEN CROPS

85. Bean blight caused by *Bacterium Phaseoli* E. F. Smith.
 86. Downy mildew of lima-beans caused by *Phytophthora Phaseoli* Thaxter.
 87. Bean rust caused by *Uromyces appendiculatus* (Persoon) Léveillé.
 88. Beet rust caused by *Uromyces Betae* (Persoon) Tulasne.
 89. Heart-rot and leaf-spot of beets caused by *Phoma Betae* Frank.
 90. Black leg and yellows or wilt of cabbage caused respectively by *Phoma oleracea* Saccardo and *Fusarium conglutinans* Wallenweber.
 91. The white rust of crucifers caused by *Albugo candida* Kuntze (= *Cystopus candidus* Léveillé).
 92. Early blight of celery caused by *Cercospora Apii* Fries.
 93. Leaf-spot and stem-rot of clematis caused by *Ascochyta Clematidina* Thümen.
 94. Sweet corn wilt or Stewart's sweet corn disease caused by *Bacterium Stewartii* E. F. Smith.
 95. Scab of cucumbers caused by *Cladosporium cucumerinum* Ellis and Arthur.
 96. Wilt of cucurbits caused by *Bacillus trachiephilus* E. F. Smith.
 97. Angular leaf-spot of cucumbers caused by *Bacterium lachrymans* E. F. Smith and Bryan.
 98. Powdery mildew of cucurbits caused by *Erysiphe cichoracearum* Fries.
 99. Downy mildew of ginseng caused by *Phytophthora cactorum* (Cohn and Lebert) Schroeter.
 100. Alternaria blight of ginseng caused by *Alternaria Panax* Whetzel.
 101. Yellow disease of hyacinth caused by *Bacterium hyacinthi* (Wakker) E. F. Smith.
 102. Lilac blight caused by *Phytophthora Syringae* Klebahn.
 103. Blight of peas caused by *Mycosphaerella pinodes* (Berk. and Blox.) Stone (= *Ascochyta pisi* Libert)
 104. Root-rot of peas, beans, tobacco and ginseng caused by *Thielavia basicola* (Berkley and Broome) Zopf.

105. Dry-rot of sweet potatoes caused by *Diaporthe Batatis* Harter and Field (= *Phoma Batatae* Ellis and Halsted).
106. Soft rot of sweet potatoes and leek of Irish potatoes caused by *Rhizopus nigricans* Ehrenberg.
107. Stem-rot of sweet potatoes caused by *Fusarium Batatatis* Wollenweber or *F. hyperoxysporium* Wollenweber.
108. Scurf of sweet potatoes caused by *Monilochaetes infuscans* Halsted.
109. Fruit-rot of tomato caused by *Phoma destructiva* Plowright.
110. End-rot of tomatoes; cause variously assigned.
111. Leaf-spot or blight of tomatoes caused by *Septoria Lycopersici* Spegazzini.
112. Leaf-mold of tomatoes caused by *Cladosporium fulvum* Cooke.

FOREST TREES

113. Twig-blight of conifers caused by *Pestalozzia funerea* Desmazieres.
114. Twig-blight of conifers caused by *Herpotrichia nigra* Hartig. (= *Neopeckia Coulteri* (Peck) Saccardo).
115. Walnut blight caused by *Bacterium Juglandis* (Pierce) E. F. Smith.
116. Fir blight caused by *Botrytis Douglasii* Tubeuf.
117. Leaf-blight of fir and spruce caused by *Trichosphaeria parasitica* Hartig (= *Acanthostigma parasitica* (Hartig) Saccardo).
118. Tar-spot of maple leaves caused by *Rhytisma acerinum* Fries and *R. punctatum* Fries.
119. Twig-blight of pine caused by *Cenangium Abietis* Rehm.
120. Leaf-spots of walnut and poplar caused respectively by *Gnomonia leptostyla* (Fries) Cesati and de Notaris (= *Marssonina Juglandis* (Lebert) Saccardo) and *Trochilia Populorum* Desmazieres.
121. Powdery mildew of chestnut caused by *Phyllactinia Corylea* Karsten.
122. Powdery mildew of oaks in Europe caused by *Microsphaera Alni* (Fries) var. *Quercina* Neger.
123. Canker of maple and other trees caused by *Nectria cinnabarina* Fries (= *Tubercularia vulgaris* Fries).
124. Canker of spruce caused by *Nectria cucurbitula* Fries.
125. Canker of spruce and fir caused by *Pestalozzia Hartigii* Tubeuf.
126. Root-rot of conifers caused by *Rhizina undulata* Fries (= *Rhizina inflata* Quélet).
127. Root-rots of trees caused by *Rosellinia* sps.
128. Root-rot of forest-trees caused by *Armillaria mellea* (Fries) Quélet.
129. Red-rot of conifers caused by *Fomes Pinicola* (Fries) Cooke.
130. Decay of oak, beach and other trees caused by *Bulgaria polymorpha* Wettstein (= *B. inquinans* Fries).
131. Heart-rot of forest-trees caused by *Polyporus sulphureus* Fries.
132. Heart- and sap-rot of trees caused by *Polystictus versicolor* Fries.
133. Compare and contrast symptoms produced by *Polystictus versicolor* Fries, *Polystictus pergamenus* Fries, *Merulius lacrymans* Fries and *Fomes Pinicola* (Fries) Cooke.
134. Compare and contrast symptoms produced by *Polyporus squamosus* Fries, *Polyporus sulphureus* Fries, *Polyporus Betulinus* Fries, *Hydnum septentrionale* Fries.

135. Compare and contrast symptoms produced by *Trametes Pini* Fries, *Lenzites sepiaria* Fries, *Polyporus subacidus* Peck, *Polyporus carneus* Fries and *Polyporus rimosus* Berkley.

136. Compare and contrast symptoms produced by *Armillaria mellea* (Fries) Quêlet, *Fomes annosus* (Fries) Cooke, *Polyporus Schweinitzii* Fries and *Thelephora galactina* Fries.

137. Compare and contrast symptoms produced by *Fomes ignarius* (Fries) Gillet, *Fomes appalanatus* Wallroth, *Fomes Fraxanophilus* (Peck) Saccardo *Fomes fomentarius* (Fries) Gillet.

138. Host-index of wood-rotting fungi arranged under the names of the fungi.

139. Host-index of wood-rotting fungi arranged under the names of the hosts.

140. Leaf-burn of trees.

141. Smoke and gas injury to trees.

142. Winter injury to trees.

143. Mycorrhiza of tree-roots.

GLOSSARY*

- Agent of inoculation.**—The thing which acts as the carrier in the transfer of the inoculum from its source to the infection-court, as for example:—wind, insects or running water.
- Control.**—The prevention of losses from a disease. Every control measure is based on one of four fundamental principles; exclusion, eradication, protection or immunization.
- Dissemination.**—The act or manner of scattering or spreading the inoculum of the pathogene within an immediate and more or less limited area about the source of inoculum.
- Distribution.**—(a) The act of transporting and establishing a pathogene beyond barriers in other regions. (b) The geographical occurrence of the disease; synonymous with range.
- Ecology.**—That phase in the study or discussion of a disease which deals with the relation of environmental factors to its occurrence, severity and character. The ecologic factors are chiefly, climatic, soil and cultural. They influence the disease indirectly through their influence on the pathogene or the host or on both.
- Enphytotic.**—The opposite of epiphytotic. An enphytotic disease is one regularly occurring in a locality or region and not liable to marked variations in destructiveness.
- Epiphytotic.**—The sudden and destructive appearance of a plant disease in a locality or region. An epiphytotic disease is one the past history of which shows it to have a tendency to appear suddenly and destructively, usually over large areas at rather long intervals. The term is analogous in meaning to epidemic but not synonymous with it.
- Eradication.**—The principle of controlling a plant disease by removing or destroying the pathogene already established within a given area or region. Disinfection, seed-selection, crop-rotation and the like are eradicatory measures.
- Etiology.**—That phase in the study or discussion of a disease, which deals with the chief causal factor, the pathogene, its nature, character and relations with the host.
- Exclusion.**—The principle of controlling a disease in plants by excluding the pathogene from a given area or region. Inspection and quarantine are the exclusionary measures usually employed.
- History of a disease.**—The logically arranged historical facts, relating to the disease itself, as distinguished from those relating more especially to the pathogene.
- Host.**—The plant affected with, or subject to a given disease.
- Hyperplasia.**—That type of pathological condition expressed by abnormal increase in the number of cells, that is, excessive cell-division.
- Hypertrophy.**—That type of pathological condition expressed by abnormal increase in size of cells (dimensions or volume). The term is also commonly used in a less restricted sense to designate swellings or overgrowths of various kinds, due either to abnormal increase in the size of the cells or abnormal cell-division or both. (See p. 8.)

*The definitions apply to the respective terms only as these terms are used in a phytopathological sense, and particularly as used in these outlines. The definitions do not always agree with those to be found in dictionaries, nor always with the variety of senses in which the terms are used even in phytopathological literature.

Hypoplasia.—That type of pathological condition expressed by the failure of plant-cells or organs to complete, in one or more respects, their normal development, that is, arrested development. Dwarfing, failure of chlorophyl-development and the like, are examples.

Hypoplastic diseases.—See p. 8.

Immunization.—The principle of preventing losses from a plant disease by the development of resistant or immune strains of the crop. This may be accomplished by selection and propagation of naturally resistant or immune individuals, by segregation and propagation of resistant or immune individuals obtained by crossing immune and susceptible forms or, artificially by feeding or injecting into susceptible hosts, substances which will make them resistant or immune. The last has, as yet, little or no practical value.

Incubation.—The activities and developments of the pathogene from the moment of its arrival in the infection-court until it has established a pathologic relation with the host.

Incubation period.—The period beginning with the arrival of the inoculum in the infection-court and ending with the first evidence of disease.

Infect.—To initiate or produce disease.

Infection.—The act of producing or initiating a diseased condition in the tissues of the host. Infection is progressive, developing cell by cell and continues as long as the host continues to react to the stimulus of the pathogene.

Infection-court.—The place on or in the host where the incubation-activities of the pathogene take place; the immediate neighborhood of a possible point of infection.

Infection period.—Commonly used to designate the period during which conditions (of host, pathogene, and environment) are especially favorable to inoculation, incubation, and initial infection, or to incubation and initial infection only.

Inoculate.—To transfer the inoculum from its source to the infection-court.

Inoculation.—The act of inoculating. It includes all the phenomena involved in the transfer of the inoculum from its source to the infection-court.

Inoculum.—That structure of the pathogene which may be transferred from its source to the infection-court. It usually consists of spores, seeds, eggs, thalli (of bacteria), or even mycelial fragments or pieces of stems (*Cuscuta*).

Lesion.—A definite region in a plant or in one of its organs, characterized by a pathologic change in structure.

Life-cycle.—The succession of phenomena exhibited during a period of continuous growth and development of the pathogene (in a fungus, from spore-germination to the normal death of the mycelium thus initiated). Most pathogenes exhibit in their life-cycles two rather distinct phases, pathogenesis and saprogenesis.

Life-history.—The complete succession of phenomena characterizing a pathogene throughout the various cycles of its existence.

Metaplasia.—That type of pathological condition expressed by the overgrowth in cells, other than increase in size (hypertrophy) or numbers (hyperplasia). Abnormal starch accumulation, abnormal development of chlorophyl, unusual thickening of cell-walls and abnormal nuclear division or growth are evidences of metaplasia.

Sometimes used in a less restricted sense to include all types of over-growth.

Metaplastic diseases.—See p. 8.

Necrosis.—That type of pathological condition expressed by the rapid destruction of cell-structures and a consequent prompt death of the protoplasts; rots, blights and cankers are examples.

Necrotic diseases.—See p. 7.

Pathogene.—Any factor capable of initiating disease (usually a living organism).

Pathogenesis.—That portion or phase of a life-cycle during which the pathogene becomes and continues directly associated with the living host. Pathogenesis includes inoculation, incubation and infection.

Pathogenicity.—The ability of an organism to produce disease.

Pathogenicity studies.—Experimental studies demonstrating the pathogenicity of a given organism.

Pathological anatomy.—That phase of phytopathology which deals with pathologic changes in form, appearance, arrangement and relation of tissues in plant-organs (gross internal symptoms).

Pathological histology.—That phase of phytopathology which deals with pathologic changes in the individual cells of plant-tissues.

Pathological morphology.—That phase of phytopathology which deals with pathologic changes in form, size, color and the like, of plants or plant-organs (gross or external symptoms).

Primary cycle.—A life-cycle initiated by a primary infection.

Primary infections.—Those infections first initiated by the pathogene after a period of rest or relative inactivity. In temperate regions, pathogenes usually initiate their primary infections in spring or early summer.

Protection.—The principle of controlling a plant disease by placing some protective barrier between the host and the generally-present pathogene. Spraying, dusting and coating with substances inimical to the inoculum of the pathogene but harmless to the host are the usual protective measures employed.

Range.—The geographical regions, areas or countries in which the disease is known to occur.

Saprogenesis.—That phase of a life-cycle during which the pathogene is not in direct association with the living host. Saprogenesis includes the saprophytic activities and dormant period of the pathogene. Some pathogenes exhibit no true saprophytic activities during saprogenesis; some exhibit no saprogenesis, being continuously associated with the living host.

Secondary-cycle.—A life-cycle initiated by a secondary infection.

Secondary infections.—Those initiated by inoculum from the primary or other secondary infections without an interposed resting or dormant period.

Symptoms.—Those pathologic changes by which a diseased plant is distinguished from a healthy one. For names and definitions of different kinds of symptoms, see pages 7–8.

Signs.—Incidental or experimental evidences of disease as distinguished from pathological evidences. For names and definitions of some of the more usual signs of disease in plants, see pages 8–9.

Source of inoculum.—The place or object on or in which the inoculum is produced.

The End.

