INDIAN AGRICULTURAL RESEARCH INSTITUTE, NEW DELHI.
# CONTENTS

## INTRODUCTION
- General Instructions ........................................... 1
- Directions for Use of Microscope .................................... 1
- The Preparation of Temporary Mounts .................................. 3
- Koch's Postulates .................................................................. 3
- Recording Experiments ...................................................... 4
- References .............................................................................. 4

## BACTERIAL DISEASES OF PLANTS

### Soft Rots
- Soft rot of vegetables: *Erwinia caratovora* ........................................ 5

### Leaf Spots
- Angular leaf spot of cotton: *Xanthomonas maliacearum* ......................... 6
- Bacterial blight of bean: *Xanthomonas phaseoli* .................................. 6
- Bacterial spot of peach: *Xanthomonas pruni* ...................................... 6

### Extensive Blights
- Fire blight: *Erwinia amylovora* .................................................................. 10

### Vascular Wilts
- Bacterial wilt of alfalfa: *Corynebacterium insidiosum* ......................... 11

### Galls
- Crown gall: *Agrobacterium tumefaciens* ........................................ 13

### Actinomycetes
- Common Scab of potato: *Streptomyces scabies* .................................. 15

## DISEASES CAUSED BY PLASMODIOPHORALES
- Clubroot of crucifers: *Plasmodiophora brassicae* ................................ 17

## DISEASES CAUSED BY PHYCOMYCETES
- Damping-off of seedlings: *Pythium* spp. ........................................ 21
- Late blight: *Phytophthora infestans* ........................................... 23
- Downy mildew of grapes: *Plasmopora viticola* ................................ 25
- White rust of crucifers: *Albugo candida* ......................................... 28
- Rhizopus soft rot of sweet potato: *Rhizopus nigricans* ................. 30
### DISEASES CAUSED BY ASCOMYCETES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peach leaf curl</td>
<td>Taphrina deformans</td>
<td>34</td>
</tr>
<tr>
<td>Powdery mildew of cereals and grasses</td>
<td>Erysiphe graminis</td>
<td>37</td>
</tr>
<tr>
<td>Apple scab</td>
<td>Venturia inaequalis</td>
<td>39</td>
</tr>
<tr>
<td>Ergot of grains and grasses</td>
<td>Claviceps purpurea</td>
<td>42</td>
</tr>
<tr>
<td>Brown rot of peach</td>
<td>Sclerotinia fructicola</td>
<td>44</td>
</tr>
</tbody>
</table>

### DISEASES CAUSED BY FUNGI IMPERFECTI

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fusarium wilt of cotton</td>
<td>Fusarium oxysporum f. basijectum</td>
<td>49</td>
</tr>
<tr>
<td>Anthracnose of bean</td>
<td>Colletotrichum lindemuthianum</td>
<td>51</td>
</tr>
<tr>
<td>Southern blight</td>
<td>Sclerotium rolfsii</td>
<td>53</td>
</tr>
</tbody>
</table>

### DISEASES CAUSED BY BASIDIOMYCETES

#### Smut Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn smut</td>
<td>Ustilago maydis</td>
<td>56</td>
</tr>
<tr>
<td>Loose smut of barley</td>
<td>Ustilago nuda</td>
<td>59</td>
</tr>
<tr>
<td>Loose smut of wheat</td>
<td>Ustilago tritici</td>
<td>59</td>
</tr>
<tr>
<td>Bunt of wheat</td>
<td>Tilletia caries and T. foetida</td>
<td>60</td>
</tr>
<tr>
<td>Loose smut of oats</td>
<td>Ustilago avenae</td>
<td>60</td>
</tr>
</tbody>
</table>

#### Rust Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stem rust of wheat</td>
<td>Puccinia graminis tritici</td>
<td>63</td>
</tr>
<tr>
<td>Cedar apple rust</td>
<td>Gymnosporangium juniperi-virginiane</td>
<td>67</td>
</tr>
<tr>
<td>Bean rust</td>
<td>Uromyces phaseoli typica</td>
<td>69</td>
</tr>
</tbody>
</table>

#### Pathogenic Gill Fungi

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Armillaria root rot</td>
<td>Armilleria mellea</td>
<td>71</td>
</tr>
</tbody>
</table>

### DISEASES CAUSED BY NEMATODES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Root knot</td>
<td>Meloidogyne spp</td>
<td>74</td>
</tr>
</tbody>
</table>

### VIRUS DISEASES OF PLANTS

#### Mosaic Group

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cucumber mosaic</td>
<td>Cucumber mosaic virus</td>
<td>79</td>
</tr>
</tbody>
</table>

#### Yellows Group

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aster yellows</td>
<td>Aster-yellows virus</td>
<td>81</td>
</tr>
</tbody>
</table>
PREFACE

This manual was prepared for use in an introductory course in plant pathology at the undergraduate level. It is designed as a guide around which students can base their laboratory studies. The authors hope that it will contribute to an understanding by the students of the principles of plant pathology and to an appreciation of the importance of plant diseases to agriculture.

The material presented in this manual has been gleaned from original publications and reference books on plant pathology. In order to conserve space, credit has not been given for original publication on details of established facts in regard to diseases or pathogens.

Many persons have undoubtedly influenced the authors in the ways in which various portions are presented. It is impossible to acknowledge all of these individuals since such a list would include most of the plant pathologists with whom the authors have been associated and under whom they have received their training. The authors would, however, like to thank Dr. Ferne L. Fulton for critical reading of the manuscript and Mrs. Marilyn Crow for careful preparation of the final copies of the manuscript.
INTRODUCTION

General Instructions

Laboratory work discussed in this manual consists of a study of representative parasitic plant diseases. Although specific diseases are studied, they present examples of general types with principles that frequently apply to other similar diseases. To understand the development of any plant disease it is necessary to have a thorough knowledge of the normal as well as the abnormal (diseased) plant and to understand the relationship of the causal agent (inciting cause) to the host plant in causing that disease. All phases of the development of the causal agent, both while associated with the host plant and while not so associated, are studied to understand disease occurrence and to recognize the principles underlying plant disease control.

The laboratory work is divided into units. The various units to be considered are diseases due to:

1. Bacteria
2. Plasmodiophorales
3. Phycomycetes
4. Ascomycetes
5. Fungi Imperfecti
6. Basidiomycetes
7. Nematodes
8. Viruses

This manual provides a study outline which is used in conjunction with your laboratory work. Your laboratory instructor will present the specific requirements in regard to the material to be studied.

The laboratory furnishes you the opportunity of observing diseased plants. Detailed microscopic observations are made, in many cases, of diseased plants and of the pathogens causing the disease. A record of laboratory observations is made either in written form or by means of labelled drawings. To economize laboratory time, many drawings have already been prepared from specimens similar to those available to you. Details of these drawings should be labelled. Drawings may also be modified to conform to the particular specimen you are observing. You will make many drawings other than those already prepared. The permanent laboratory record must be neat and accurate. You will be given instruction regarding drawings and written material.

Directions for Use of Microscope

It will be necessary to use the microscope frequently to study the details of host tissues and structures of the pathogen which are too small to be clearly seen with the unaided eye. It is very easy to ruin both the microscope and mount if certain precautions are not observed. The following procedure will reduce this hazard to a minimum and aid in quickly bringing the object into sharp focus.
1. The position of the microscope should be near the chest so the eye is brought over the ocular without tilting the microscope. If the microscope is tilted, difficulty will be experienced in obtaining sufficient light, and liquid mounts will flow off the slide.

2. Adjustment of light may be attained by directing the substage mirror toward the light source so that a maximum of light is seen through the ocular. The amount of light is controlled by an adjustable diaphragm beneath the microscope stage. Various mounts will require different amounts of light. Modify the amount of light so that the greatest detail is obtained.

3. The following technique should be used to focus the microscope.
   a. Raise the tube with the coarse adjustment so that the mount may be placed on the stage.
   b. Clamp the mount in place directly over the hole in the stage by means of the spring clamps on either side.
   c. Lower the tube until the low power objective is within 1/8 inch of the mount. Never focus downward while looking through the microscope!
   d. Raise the tube slowly with the coarse adjustment while looking through the microscope. When the object is nearly in focus use the fine adjustment.
   e. Move the mount so as to bring the desired part into the center of the field.
   f. For higher magnification, rotate the nosepiece so that the high-power objective swings in place under the tube. (Caution: this should be done gently. Do not force the objective in place. If difficulty is experienced, ask for help from the instructor.)
   g. Change the fine adjustment upward or downward slightly to bring the object into sharp focus. You will find that under high magnifications only certain parts of the field will be in focus at any one time. It will be necessary to make slight adjustments in focus as different parts of the field are studied. When focusing is complete, make readjustments in lighting until the best arrangement is attained.

4. Eyestrain. Closing one eye while using the other to look through the microscope tends to tire certain eye muscles. Learn at the start to keep both eyes open. Another source of eyestrain results from imperfect focusing. The eye is forced to make unaccustomed adjustments in order to bring the object into view. This can be obviated by keeping the hand on the fine adjustment and continually making slight changes in focus as different parts of the field are studied.
5. Cleaning the lenses. When the lenses become dirty or dusty, clean them with lens paper. Never use ordinary tissue paper or cloth which might contain grit. Fold the lens paper several times, hold against the lens lightly and twist slightly. Repeat with a new surface of the paper. Never rub heavily or you may scratch the lens. If further cleaning is necessary, check with your instructor.

6. Do not remove lenses from their mounts or unscrew the objectives from the nosepiece. If the microscope seems to be out of order, bring it to the attention of the instructor.

The Preparation of Temporary Mounts

Temporary mounts of diseased plant parts or pathogens are frequently required for microscopic examination. To make such a mount, first prepare a clean glass slide and a clean cover slip. Place a drop of water on the slide. Add the specimen to be studied to the drop of water. The specimen is then properly aligned on the slide with dissecting needles. In many cases specimens must be torn and teased apart with needles. The cover slip is then placed on top of the preparation. This is done by placing one edge of the cover slip on the glass slide in contact with the drop of water. Using the tip of a dissecting needle gently lower the cover slip into position. If this procedure is done correctly, the mount should be free of air bubbles.

The most common error in making temporary mounts occurs from using too much or too thick material on the slide. Only very thin objects can be studied with the compound microscope. The cover slip must lie flat. If it visibly tilts to one side, the specimen is too thick. The specimen and area under the cover slip must be flooded with water or other mounting medium. Avoid the presence of water on the rest of the slide or on top of the cover slip.

Koch's Postulates

Robert Koch in 1881 introduced the plate method of isolating bacteria and fungi. Using this method he was able to demonstrate that certain bacteria were associated with diseased human and animal tissues. He formulated a series of "rules of proof" or postulates which must be satisfied in order to determine the causal relation of a particular organism in a disease. Although his postulates were formulated with animal and human diseases, they apply equally well to many plant diseases caused by parasitic organisms. These postulates may be stated as follows:

1. The organism in question must always be found associated with a particular diseased condition of a plant.

2. The organism must be isolated and grown in pure culture.

3. The organism grown in pure culture must be inoculated into a healthy plant and induce the characteristic disease.

4. The organism must be reisolated from the second plant and compared with the first culture. Both the diseased condition induced by inoculation, and the organism recovered from the inoculated plant must correspond to the original diseased condition, and to the first organism isolated, respectively.
Recording Experiments

In some of your work, experiments will be performed to illustrate principles regarding a disease or pathogen. Concise and adequate records should be kept of experiments as they are conducted. Facts that may, at the time, appear irrelevant should also be recorded as, in the light of further evidence, they may be important. At the conclusion of the experiment, the data should be interpreted and recorded in a suitable form as soon as possible. Students will use the following form for recording experiments:

1. Subject or title.
2. Object or aim.
3. Methods and materials used. Describe the method of procedure and enumerate the materials used.
4. Experimental results. State the results briefly and concisely.
5. Conclusions. This is an explanation of the results. Conclusions must be supported by adequate evidence from the experimental results. Be careful not to overlook the apparently obvious conclusions.

References

The diseases discussed in this manual are presented in a concise form. Additional information on each disease can be found in one or more of the following books.

BACTERIAL DISEASES OF PLANTS

Since 1878, when bacteria were first definitely established as plant pathogens, many bacterial diseases have been characterized. Cultural and morphological characteristics of these pathogens are variable. Except for species of Streptomyces, however, they are all nonsporeforming rods.

In general, bacteria penetrate host surfaces through wounds or natural openings, such as stomata, hydathodes, lenticels, and nectaries. Intercellular invasion is common. Some become vascular parasites.

Based on symptoms produced, bacterial diseases may be separated into five categories:

1. Soft rots
2. Leaf spots
3. Extensive blights
4. Vascular wilts
5. Galls

Consideration will be given to representative diseases of each type. In addition, common scab of potato is presented as an example of a disease caused by an actinomycete.

Soft Rots

SOFT ROT OF VEGETABLES: Erwinia caratovora (L. R. Jones) Holland

Bacterial soft rot is widespread and destructive on many vegetables in storage and transit. Under favorable conditions the disease may also affect vegetables at various stages of growth in the field.

Symptoms:

A soft, mushy rot is characteristic of most vegetables affected by soft rot. Variations as to color, odor, and consistency are encountered among different vegetables. Diseased specimens are readily available from local markets handling fresh produce. Specimens in varying degrees of deterioration may be produced by artificial inoculation.

Pathological Histology:

The bacteria causing soft rot do not enter the host cells in the early stages of infection, but are active between the cells. By enzymatic secretions, the bacteria cause the dissolution of the middle lamella. During initial stages of cellular disorganization, the cell walls and cell contents remain intact. Loss of water from the cells and plasmolysis occur as the intercellular spaces increase in solute concentration. Temporary mounts for microscopic examination can be prepared by making thin freehand sections from
the margin of the rotting area. Such a preparation allows study of unaffected tissue as well as disrupted tissue in the affected area.

Causal Organism:

*Erwinia caratovora* was originally isolated from carrot and characterized by Jones in 1907. On agar, colonies of the bacteria appear circular, convex, smooth, grayish-white, moist, and glistening, with sharp, entire margins. There is disagreement as to whether a single species of related organisms or different species are involved in the soft rot group of bacteria.

Life Cycle of the Pathogen:

1. Penetration: The bacteria enter primarily through wounds. However, entrance through lenticels of potato tubers and stomata of spinach is reported.

2. Dissemination: Bacterial exudate and bacteria released by the disintegration of infected plant parts make distribution possible. Spattering rain, contaminated tools, seed-corn maggot and several related species of flies (black rot of potato) and direct contact between diseased and healthy plant parts furnish means of spreading the pathogen.

3. Overwintering: The bacteria survive in the soil and crop refuse.

Favorable Environment:

This disease of vegetables is favored by a variety of poor handling and storage practices, i.e., excessive bruising and wounding in harvesting and storing produce, storage at warm temperatures and with inadequate ventilation, harvesting and storage of vegetables under abnormally wet conditions, and storage of certain types of vegetables without "curing" to heal wounds. When this disease affects bulbs, tubers, or roots in the field, conditions are usually excessively wet so that the plant part is not able to "heal-off" wounds.

Control:

1. The crop should be harvested, shipped, and stored in a manner that would prevent excessive bruising.
2. Practices that aid in the healing of wounds and removal of surface moisture should be followed before storing.
3. Cool, aerated storage reduces the decay produced by the soft-rot bacteria.

Leaf Spots

ANGULAR LEAF SPOT OF COTTON: *Xanthomonas malvacearum* (E.F.S.) Dow.


The bacterial leaf-spotting diseases listed above are of major economic importance on the respective crops. Although these diseases have certain characteristics in common, inherent differences in the suscepts (hosts) and pathogens exist.
A. Bacterial Blight of Bean, affected leaf
B. Bacterial Blight of Bean, affected pod
C. Bacterial Blight of Bean, diagram of cross section of affected pod
D. Bacterial Blight of Bean, cross section of affected leaf
Symptoms:

On the respective plants, lesions are formed on leaves, stems, and fruit. The lesions first appear water-soaked and later become necrotic. Leaf lesions are frequently surrounded by chlorotic bands. The infected areas in the leaves of some plants drop out to give a "shot-hole" appearance. Symptoms of each disease are best appreciated by a careful study of diseased specimens and photographs.

Pathological Histology:

Each of the above bacterial pathogens exerts a somewhat similar influence upon its respective host. After entrance through stomata, the bacteria develop in the sub-stomatal chambers. The parasites are intercellular and dissolve away the middle lamella, plasmolyze the cell contents, and cause necrosis. In some circumstances X. phaseoli enters the vascular system of the host.

Causal Organisms:

Xanthomonas malvacearum was originally described by E. F. Smith in 1901 as being pathogenic on cotton. Colonies of this organism on agar are typically convex, smooth, pale yellow, and glistening. Colony margins are irregular. Strains of the pathogen have been reported.

X. phaseoli was first isolated by E. F. Smith in 1898 from infected bean leaves. On beef extract agar the colonies appear circular, amber-yellow, and smooth, with entire edges. Strains of the species are common.

X. pruni was reported in 1903 by E. F. Smith. Isolates from Japanese plum were shown to be pathogenic to plum and peach. On beef extract the colonies are yellow, circular, smooth, and convex, with entire edges.

Life Cycle of the Pathogens:

1. Penetration: Bacteria enter the host primarily through stomata, although wounds may also be entry points.
2. Dissemination: Bacterial exudate is spread by meteoric water and wind. Bacteria of bean blight and angular leaf spot of cotton are seed-borne and those of bacterial spot of peach are spread on nursery stock.
3. Overwintering: X. malvacearum remains viable in infested and infected seed, and probably in plant refuse; X. phaseoli persists in infested and infected seed, and plant refuse; X. pruni survives in infected terminal twigs and buds.

Favorable Environment:

In these three diseases an environment which is favorable for rapid growth of the host is favorable for the disease. This is particularly true if the moisture is obtained from frequent rains. In arid areas, where the moisture is furnished by furrow irrigation, bacterial diseases of this type are rare.
Plate II

A. Angular Leaf Spot of Cotton, affected leaf
B. Angular Leaf Spot of Cotton, affected boll
C. Bacterial Spot of Peach, affected fruit
D. Bacterial Spot of Peach, affected leaf
Control:

Angular leaf spot of cotton:

1. Acid delinting and chemical seed treatment reduce the amount of primary infection.

2. Varietal resistance has been recorded.

3. Volunteer seedlings are frequently infected and should be removed.

4. Cotton defoliation reduces late boll infection.

Bean Blight:

1. Use clean seed from the Rocky Mountain states and California.


3. Some varietal resistance has been recorded.

Bacterial Spot of Peach:

1. Late fall applications of copper sulfate followed by applications of lime reduce the overwintering bacteria in the terminal twigs.

2. Foliage applications of a zinc-lime mixture protect the foliage and fruit from infection.

Extensive Blights

FIRE BLIGHT: Erwinia amylovora (Burrill) Winslow et al.

Fire blight is a common and destructive disease on pear and apple. Commercial cultivation of some desirable pear varieties has been abandoned in many areas because of the ravages of this disease. Of historical importance is the fact that with this disease a bacterium was first shown to be capable of causing plant disease.

Symptoms:

Fire blight first attacks the blossoms causing a blossom blight and then a progressive blighting of spurs and twigs. Spread of the pathogen may continue, resulting in cankers on branches and trunks of trees.

Pathological Histology:

After the bacteria gain entrance to the host tissue they develop in the intercellular spaces. The cortical parenchyma is involved first. The bacteria then migrate to the phloem, cambium, and vascular tissue. Bacteria are found in large numbers in the intercellular spaces of the involved tissue. They dissolve the middle lamella causing cell separation, plasmolysis, death, and, finally, break-down of the cell wall.
Causal Organism:

*Erwinia amylovora* was first described by Burrill in 1882. Proof that it caused fire blight had been presented in 1878. On agar the bacteria produce colonies that are white, elevated, wet-shining, circular, and with irregular margins.

Life Cycle of the Pathogen:

1. Penetration: Bacteria enter the host through the nectarics in the receptacle cup of blossoms, through stomata in sepals and leaves, and through wounds.

2. Dissemination: Bacterial exudate from cankers is spread by spattering rain and pruning tools. Bees and other nectar-seeking insects spread the bacteria in nectar from blossom to blossom.

3. Overwintering: Bacteria have been shown to remain alive over winter in cankers on cultivated and wild hosts and in beehives.

Favorable Environment:

This disease is favored by conditions which induce rapid, succulent growth of apple or pear during the time of blossoming. Abundant rainfall during the spring months is usually associated with severe outbreaks. The promotion of succulent growth by the use of nitrogenous fertilizers will increase the severity of damage.

Control:

1. Satisfactory control of this disease on susceptible varieties has not been demonstrated.

2. Spraying during the bloom period with various materials having bactericidal properties has shown possibilities. Some of the antibiotics are especially promising.

3. Planting the more resistant varieties aids in control.

4. Pruning out blighted twigs and small cankers is a questionable remedy. The purpose of pruning is to remove sources of primary infection. This practice is not often recommended because greater succulence of the tree results and, with it, increased susceptibility to fire blight.

5. Soil fertility should be regulated to reduce the degree of succulence of new growth. Nitrogenous fertilizers are to be applied with extreme caution.

Vascular Wilts

**Bacterial Wilt of Alfalfa:** *Corynebacterium insidiosum* (McCull.) Jens.

Bacterial wilt of alfalfa occurs throughout the alfalfa-growing areas of the United States. Since 1926 the importance of this disease to alfalfa pro-
duction has increased until it is now recognized as the most important malady of the crop. Alfalfa plants are killed so rapidly that forage production often becomes unprofitable after 3 or 4 years.

Symptoms:

Wilt affects all portions of the plant. Plants become dwarfed and reduced in vigor, the leaves are small and yellowish, and the plants finally wilt and die. The disease is most serious in its effect on root tissues. The tap root shows a yellowish-brown discoloration in the outermost part of the woody cylinder just beneath the bark. Root discoloration is the most important and constant characteristic of the disease.

Pathological Histology:

Bacteria are present in the xylem bundles. As the bacteria reproduce they progress up and down the stem in the vascular tissue. Tangential spread occurs into the parenchymatous tissue and adjacent xylem bundles, resulting in distribution around the circumference of the root.

Causal Organism:

_Corynebacterium insidiosum_ was originally described by McCullough in 1925. This is one of the few gram-positive bacteria pathogenic to plants. On beef agar, colonies are pale yellow, circular, smooth, and glistening, with entire edges.

Life Cycle of the Pathogen:

1. Penetration: Bacteria enter the roots through natural root wounds or wounds caused by winter injury or insects.

2. Dissemination: Bacteria are released into the soil by the disintegration of infected plant tissue and from fissures in the root resulting from winter injury. Drainage water, soil, and farm machinery spread the bacteria about the field or from field to field.

3. Overwintering: The bacteria persist in infected plants, crop residue, and soil.

Favorable Environment:

The disease is most severe on heavy types of soil. Excessive amounts of soil moisture favor infection.

Control:

Resistant varieties offer the only satisfactory means of control. Two tolerant varieties of alfalfa, Ranger and Buffalo, have been developed. These varieties have lessened the damage incurred by bacterial wilt, but do not have complete resistance to the disease. Precautionary measures to be observed which aid in retarding the disease are to

1. Avoid excess and waste irrigation water
2. Avoid wounding plants
3. Rotate infested fields with crops other than alfalfa.
Galls

CROWN GALL: *Agrobacterium tumefaciens* (E.F.S. and Towns.) Conn.

Crown gall occurs on a wide and diverse number of plants and the disease is of special importance on tree and cane fruits. Infected plants are stunted and unthrifty since food conducting tissues are disrupted. Growers who produce nursery stock may experience heavy losses because any plants with galls must be discarded. The relationship of this plant disease to the cancer problem in humans has received much consideration.

Symptoms:

Galls vary in size, reaching a diameter of several inches, and have a more or less convoluted surface. On trees the galls are frequently produced at the base (crown) of the trunk or belowground. Galls are commonly produced on the aerial branches of herbaceous plants and brambles. Non-parasitic wound overgrowths are easily confused with crown gall.

Pathological Histology:

After infection takes place the bacteria grow and multiply in the intercellular spaces. In general they are found between the parenchyma cells near the outer surface of the gall. Their presence stimulates the cells to hyperplasia. The conductive tissue is disrupted and scattered throughout the overgrowth.

Causal Organism:

*Agrobacterium tumefaciens* was first described by E. F. Smith and Townsend in 1907. On agar, the colonies are small, white, circular, glistening, translucent, and entire. Inoculation tests have shown this bacterium to be pathogenic on representative plants in over 40 families. Other species of *Agrobacterium* incite similar symptoms.

Life Cycle of Pathogen:

1. Penetration: The bacteria enter only through wounds.
2. Dissemination: Diseased plants carry the bacteria from one area to another. Grafting tools spread the pathogen from plant to plant. As diseased plants disintegrate bacteria are released into the soil where they may be disseminated by water or in soil particles.
3. Overwintering: Bacteria overwinter in diseased plants, infested plant refuse in soil, and as free organisms in the soil for a few years.

Control:

1. Produce clean nursery stock.
2. Inspection and certification is required by law and eliminates conspicuously diseased plants.
Plate III

A. Fire Blight of Apple, affected twig
B. Crown Gall, affected peach crown
C. Common Potato Scab, affected tuber
3. Sanitation of nursery storage facilities is recommended.

4. Eradicate and burn diseased stock in the nursery row.

**Actinomycetes**

**COMMON POTATO SCAB:** *Streptomyces scabies* (Thaxt.) Waks. and Henrici

Common scab occurs in America and Europe and probably wherever the potato is grown. The damage from the disease results from the unsightly lesions which increase paring waste, change the taste and odor of the potato, and make it more susceptible to storage rot. Beets, turnips, mangels, carrots, and radishes may also be affected. Losses to the potato crop in the United States amount to several million dollars annually.

**Symptoms and Signs:**

Scab infection initially shows itself as minute, reddish to brownish surface areas, later enlarging to form circular or irregular corky lesions on the tubers. The lesions may appear as shallow "russetting" or may develop into deep pits. Symptoms on stolons and on subterranean stems are much less noticeable than those on the tubers.

**Pathological Histology:**

After infection the pathogen is first situated between the host cells and moves through the tissue by what appears to be dissolution of the middle lamella. Substances are formed which greatly stimulate cambial activity, producing great numbers of cells with walls that become suberized (corky). As the organism continues its activity a layer of dead tissue forms over the cork and a scabby area results.

**Causal Organism:**

The taxonomic position of this group is in dispute. Some authorities consider it fungal, others designate it bacterial. For the sake of convenience it is here placed with the bacteria.

Aerial growth of the organism consists of prostrate filaments with many lateral branches. The hyphae which bear the spores are spiral in form. Septa cut off spores which are rod-shaped and germinate by means of one or more germ tubes.

**Disease Cycle:**

1. **Penetration:** Young tubers are most vulnerable. Lenticels constitute the most common means of invasion. Wounds, stomata and the cuticle (when it is thin) are all avenues of entrance.

2. **Dissemination:** Spores are produced on the surface of all affected parts. Infected seed tubers and wind- and water-borne soil are all important means of distribution. Passage through the digestive tracts of animals does not kill the spores and manure can be badly infested.
3. Overwintering: The pathogen may subsist indefinitely in soil favorable for its development.

Favorable Environment:

Temperatures ranging from 20° to 22° C. are optimum. Greatest disease development occurs in well aerated soil under moisture conditions which favor rapid growth of the host. A pH range of 5.2-8.0 is most favorable for the disease. Liming the soil may increase the incidence and severity of the disease.

Control:

Cultural and Sanitation Practices: Potato seed tubers can be treated with various materials. Formaldehyde and mercuric chloride soaks have been widely used. Organic mercury dips have more recently become common. However, where the scab organism is prevalent in the soil, tuber treatments are almost valueless.

Green manures have been shown to reduce scab. A 4 to 6 year alfalfa rotation is said to be effective. In naturally acidic soils, rotation with acid-tolerant crops reduces the disease. The use of chemicals to lower pH of potato land has been used very little commercially.

Resistant Varieties: The use of resistant varieties holds the most promise of control. Russet-skinned varieties are more resistant than smooth varieties. Russetting has been found to be linked genetically with resistance. Varieties such as Russet Rural and Russet Burbank are resistant only so long as the scab incidence is moderate.

Certain European varieties are very resistant and this germ plasm has been incorporated into several American varieties such as Menominee, Ontario, Seneca and Cayuga.
DISEASES CAUSED BY PLASMODIOPHORALES

The Order Plasmodiophorales is composed of a small group of organisms which includes parasites on seed plants, algae, and fungi. In most cases the effects on higher plants are mild. Plasmodiophora brassicae and Spongospora subterranea are the only two species causing diseases of economic importance. The classification of this group of organisms is in doubt. It is usually placed in a group of organisms, the Myxothallophyta, which is neither fungal nor bacterial. Some authors retain it in the Phycomycetes, the most primitive Class of true fungi.

CLUBROOT OF CRUCIFERS: Plasmodiophora brassicae Wor.

Clubroot was first recorded in England in 1735. The disease occurs in most countries of the world, but is probably most destructive in Europe. In the United States, it is found principally in the North Central and Northeastern sections of the country. Woronin, a Russian scientist, conducted a masterful investigation of this disease in 1873-78. His paper has been available in English since 1934 as Phytopathological Classic 4. The members of the mustard family (Cruciferae) are the only hosts for the pathogen. Losses from the disease are difficult to estimate, but in some areas they have been extensive.

Symptoms:

The clubroot organism may attack crucifers at any stage of growth. Infected roots develop "clubs" which to some extent are characteristic for the host involved. Cabbage roots form various types of spindle-shaped enlargements. Other hosts exhibit clubs of different shapes. enlargements are first light in color, smooth, and brittle, but later turn dark-colored, rough, and flabby, because rot producing organisms soon penetrate them and cause a rapid decay.

Aboveground parts of the host may show a temporary wilting or "flagging" during the hottest part of the day. This may continue day after day until the host finally dies. In other cases only a gradual decline of the plant is noticed. The lower leaves are dropped, foliage becomes chlorotic, and, at the end of the growing season, plants are stunted and yield little or nothing.

Pathological Histology:

The pathogen appears within the host cells as multinucleate plasmodia which develop from uninucleate myxamoebae that apparently can migrate both vertically and horizontally in the cortex, the cambium, or the medullary rays of the host root. Infected cells are stimulated to enlarge (hypertrophy) and also to divide repeatedly (hyperplasia). Accelerated cell division aids the parasite both in multiplication and in distribution in the host tissue, since portions of plasmodia accompany daughter cells. Cambial tissue appears most sensitive to the parasite, and great numbers of unspecialized cells develop at the expense of xylem and phloem production. Thus, the host vascular system is drastically reduced.

- 17 -
A. Club root of Cabbage, affected roots

B. Clubroot of Cabbage, cross section of affected root

C. Clubroot of Cabbage, affected root cell containing plasmodia of *Plasmodiophora brassicae*

D. Clubroot of Cabbage, affected root cell containing spores of *Plasmodiophora brassicae*
Causal Organism:

*Plasmodiophora brassicae* exists in the soil as resting spores. These spores germinate to produce biflagellate zoospores which enter the host through root hairs. Plasmodia in different stages of development can be found in the host cells. Eventually each nucleus in the multinucleate plasmodium is walled off, and the thick-walled, resistant, resting-speres are formed. These spores are released into the soil by the disintegration of diseased roots.

Disease Cycle:

1. Penetration: Root hairs and young root tissue are apparently easily penetrated. Older roots and aboveground stems are not invaded except through wounds or leaf scars. Once within the root, the parasite moves from cell to cell as myxamoebae (possibly also as very small plasmodia).

2. Dissemination: The resting spores are released into the soil by the rotting of the host roots. Spores may be distributed by drainage water, implements, wind-borne soil, animals, and transplants.

3. Overwintering: Resting spores carry the organism from one growing season to the next. Moreover, they are known to remain dormant in the soil for as many as ten years.

Favorable Environment:

Soil temperature appears to have relatively little effect on infection since disease occurs in a temperature range of 9°C to 30°C. Soils must have a water content of at least 45% of their water-holding capacity before infection takes place. Soil pH is an important factor in disease incidence. Spores do not germinate, or do so only weakly, in an alkaline medium.

Control:

Extreme care should be taken to assure that transplants are grown in non-infested soil. All contaminated manure, crop residues, and drainage water should be kept from clean land.

Rotations appear impractical in most cases because of the longevity of the resting spores.

Liming the soil has been effective in many instances, but it is not feasible in certain muck soils because of their buffer capacity.

Mercuric chloride (1-1500) used at the rate of 60-125 ml. per transplant in planting cabbage and cauliflower has aided in control on mildly-infested soil.

Resistant Varieties: In Europe progress has been made in developing resistant varieties of turnip, rutabaga, and marrow kale. The existence of physiologic races of the parasite complicates the search for resistance. A number of turnip and rutabaga varieties in the United States are resistant to strains of the organism found here, but are susceptible when grown in England. No resistance in cauliflower is known, and work on resistant cabbage varieties has only begun. Selections from a kale-cabbage hybrid, utilizing resistance found in kale, may alleviate the problem to growers of cabbage in the future.
DISEASES CAUSED BY PHYCOMYCETES

The Class Phycomycetes is composed of a heterogeneous group of organisms, both with reference to morphological characteristics and to types of diseases produced by pathogens in the group. It is considered the most primitive of the true fungi, and the group is commonly called the algal-like fungi.

To understand the relationship of the Phycomycetes to disease development, it is essential to know something of their form and reproduction. The vegetative hyphae are non-septate and multinucleate (coenocytic). Septations develop in conjunction with spore development. At times the mycelium develops special structures (haustoria) for food absorption which branch off from the main mycelium.

Reproduction is by means of asexual and/or sexual spores. Asexual spores are produced either within differentiated structures called sporangia or directly upon differentiated hyphae (conidiophores). Spores produced within sporangia are of two types:

1. The non-motile called sporangiospores (e.g. Rhizopus spp.)
2. The motile called zoospores (e.g. Pythium spp.)

In order to distinguish a sporangium producing sporangiospores from one producing zoospores, the latter structure will be called a zoosporangium. Spores produced exogenously on conidiophores are known as conidia (e.g. Peronospora spp.). Another type of spore which is associated with some of the Phycomycetes is the chlamydospore. Chlamydospores are thick-walled resistant spores formed by modification of a hyphal segment (e.g. Pythium spp.).

Two types of sexual reproduction are encountered in the Phycomycetes. In one of these, sexual reproduction is by the union of differentiated sex cells (e.g. Phythium spp., Phytophthora spp.). The larger of the cells, the oogonium, is the female structure, and contains the female gamete or egg. The smaller, the antheridium, produces many male gametes, which are discharged into the oogonium. Fusion of male and female gametes occurs inside the oogonium to produce an oospore or oospores. The second type of sexual reproduction is typified by the union of sex cells which are morphologically similar (e.g. Rhizopus spp.). Upon union of gametangia, the confluent end walls between them dissipate and fusion of gametes occurs. The resultant spore which develops from this union is a zygospore. Both oospores and zygospores are thick-walled and resistant to adverse conditions.

A wide diversity of types and degrees of pathogenicity occur within the Phycomycetes. Some species are relatively low-grade pathogens, generally spending much of their life history as saprophytes. When environmental conditions are favorable for their development as pathogens, they attack plants causing direct necrosis, softening of tissues, and a rapid killing (e.g. Pythium spp., Rhizopus spp.). Others are obligate parasites and establish a host relationship from which they derive food over a long period of time without killing the plant (e.g. Albugo spp., Plasmopora spp.).
Representative diseases are presented which illustrate different types of structures and pathogenicity found within the Phycomycetes. The above discussion introduces a number of terms which may be new to you. Repeated use and comparison of these terms in studying the following diseases should clarify their meanings.

DAMPING-OFF OF SEEDLINGS: Pythium spp. and others.

Damping-off occurs widely on young seedlings under conditions of high soil moisture. Disease development is also influenced by temperature. As a rule, seedlings of warm weather crops are more severely damaged by damping-off when cool temperatures prevail, while seedlings of cool weather crops are more severely damaged when the temperatures remain warm.

The extent of damage attributable to this disease is impossible to estimate. Direct losses of 50 to 100 per cent of plantings are not rare. Losses include the cost of replanting, broken stands, and reduced yields from surviving but damaged plants.

Almost any plant in the seedling stage of growth is susceptible to damping-off. Severe damage is inflicted annually upon legumes, tomatoes, cotton, forest seedlings, and many others. Seedlings grown under glass are often severely attacked.

Symptoms:

Damage from damping-off occurs in two phases:

1. Before the seedling emerges from the soil (pre-emergence damping-off)
2. During the seedling stage (post-emergence damping-off).

In the pre-emergence phase, the prospective seedling is killed before it reaches the surface of the soil, at times even before the hypocotyl emerges from the seed coat. This phase of the disease is not often recognized, and the failure of seedlings to appear is often attributed to low vitality of the seed. The term "damping-off" characterizes the post-emergence phase of the disease. Typically, the stem of the young seedling becomes soft and water-soaked at the soil line, which results in the seedling "dropping dead".

Pathological Histology:

Damping-off pathogens are found inter- and intracellularly in the diseased host tissue as fine, branched, continuous threads of hyphae. It is reported that enzymes and toxins are secreted which aid in the breakdown of the host cellular structure. The relationship of the fungal mycelium to the host tissue may be studied by teasing apart affected tissue and observing it through the microscope. The addition of a drop of dilute solution of cotton blue aids in differentiating the fungal mycelium from the host tissue.

Causal Organism:

Although damping-off and related maladies may be caused by fungi other than Pythium spp., only pythiaceous fungi will be considered at this time. The reproductive stages of these fungi are not readily observed on, or in, the host.
Plate V

A. *Pythium* sp., zoosporangium and intercalary zoosporangium

B. *Pythium* sp., oogonium and antheridium

C. *Pythium* sp., oospore
Under laboratory conditions, reproduction of the pathogen may be profuse. Predominantly sexual or asexual reproduction of *P. debaryanum* may be induced in water culture, depending upon the food source. Sexual organs predominate when the fungus is grown on hemp, oats, or wheat seed, while asexual reproduction predominates when the fungus is grown on squares of carrot.

**Disease Cycle:**

1. **Penetration:** These fungi enter the young, unprotected epidermal cells by direct, mechanical pressure.

2. **Dissemination:** *Pythium* spp. produce zoosporangia on the surface of diseased tissue. Oospores are produced in a similar manner. These fungi also grow readily and reproduce sexually and asexually on dead organic matter in the soil. For this reason pythiaceous fungi are almost universally present in the soil. Distribution is by drainage water, crop residues, and soil.

3. **Overwintering:** Oospores and chlamydospores remain in the soil and crop residues. In southern areas, such as Arkansas, vegetative growth of the fungus may continue through the winter in the soil.

4. **Favorable Environment:** An abundance of moisture accompanied by other conditions which retard germination of seeds and growth of seedlings promotes damage from damping-off.

**Control:**

1. Treatment of seeds with appropriate chemicals effectively protects the germinating seeds from pre-emergence damping off, and reduces post-emergence damping-off.

2. Damping-off is decreased by cultural practices which minimize high soil moisture, reduce humidity, and enhance the growth of seedlings.

3. In seedbeds, coldframes, or greenhouses, the following practices have been used to control damping-off:
   - Soil sterilizing by heat or chemicals
   - Planting seed in a manner to avoid excessive moisture
   - After seedlings emerge, watering only in the morning as the temperature is rising, avoiding over-watering
   - Regulating temperature to promote rapid germination of seed and growth of seedlings.

**LATE BLIGHT:** *Phytophthora infestans* (Mont.) DEB.

Late blight is primarily a disease of potato and tomato, although some other members of the Family Solanaceae are susceptible. This disease is world wide in distribution. Total losses are frequently experienced in the field, with losses continuing in storage on potatoes from the more mildly diseased fields.
In 1844-1846, exceptionally severe epidemics of late blight practically eliminated potatoes as a source of food in Europe and materially contributed to the Irish famine and mass migration of peoples. In the United States, the disease is most disastrous in the northeastern section associated with moist, cool, summer weather.

Symptoms:

On aerial portions of plants small, water-soaked spots of variable size develop, becoming brownish to purplish as the lesions mature. Under favorable conditions, the lesions enlarge rapidly to produce a general blighting of the foliage. On the under surface of the leaves a white mildew appears on the advancing margin of the lesions. When the disease is progressing rapidly, a characteristic offensive odor prevails. In drier weather, the initial lesions tend to remain localized and dry up.

On potato tubers, a dry or wet rot may be associated with infection. In the absence of any secondary complications, a dry rot is characteristic, typically involving only the outer one-half inch of the tuber. Tomato fruits are attacked at any stage of maturity. Large, poorly defined, brownish lesions appear on the fruit. The subsequent rot is firm and remains somewhat superficial unless the fruit is picked and placed in a humid atmosphere to allow the fungus to develop internally.

Pathological Histology:

As the fungus invades the host tissue, the main mycelium is intercellular with haustoria found inside the cells. Parenchyma tissue of leaves, tubers, fruits, and outer portions of stems are attacked. After the cortical cells of the stem have been killed, the mycelium frequently invades the pith. Infected host cells are killed rapidly. Shortly after necrosis of the tissue develops, the fungal mycelium in that area also dies. Only the mycelium in the advancing margin of the infection remains active.

Causal Organism:

Vegetative growth of \textit{P. infestans} is by filamentous coenocytic mycelium. In the presence of high humidity, a characteristic growth (sporangiophores or conidiophores) is observed protruding from stomata on the lower surface of leaves. Attached to this growth are zoosporangia or conidia, depending upon prevailing temperatures. In cool weather (near 12° C.), zoosporangia are formed which, upon germinating, release many biflagellate zoospores. Under warmer conditions (near 25° C.), conidia are produced which germinate directly to produce a germ tube. No sexual stage has been definitely associated with this organism.

Disease Cycle:

1. Penetration: Germ tubes produced by zoospores or conidia penetrate the cuticle directly, or enter through stomata or lenticels.

2. Dissemination: Zoosporangia or conidia are spread by splashing rain, wind, or insects. The fungus may be distributed about the country on potato tubers or young tomato plants.
3. Overwintering: The fungus remains alive as mycelium in infected tubers. In southern areas such as Florida and Texas, where tomatoes and potatoes are grown through the winter months, the pathogen remains active. In the spring as the crops are successively planted toward the northern areas, primary inoculum is available from these tomato and potato winter-growing areas.

Favorable Environment:

Cool (10°-20° C.), moist (90-100% humidity) nights are required for rapid increase of inoculum. Temperatures between 21° and 24° C. favor development of the disease after infection has occurred.

Control:

1. Varieties of potato resistant to some strains of the pathogens are available. Resistance has been recognized in some tomato selections, and commercial varieties may be developed.

2. Protection of the crop by the application of fungicides allows efficient control. Protection of tomato foliage and fruit with fixed coppers or zineb, an organic fungicide, is recommended. Bordeaux mixture, as well as these fungicides, is used on potatoes.

3. Forecasting of late-blight epidemics on the basis of weather trends and development of the pathogen has achieved considerable success in various areas. Timely applications and more efficient use of fungicides result from this service.

4. Killing of potato foliage a few days before harvest is recommended to eliminate possible contamination of tubers during harvest.

5. Disposal of tuber refuse from the field and from storage removes such material as a source of primary inoculum.

DOWNY MILDEW OF GRAPE: *Plasmopora viticola* (Berk. and Curt.) Berl. and DeT.

Downy mildew is a common disease of grape. Although the damage attributed to this disease in the United States is not great, heavy losses are frequently reported in localized areas. Losses are seasonal and range from a trace to 10 per cent.

In 1878, downy mildew was found in France in a nursery of grape seedlings which had been imported from the United States. In the grape-growing areas of France, the pathogen was introduced into a favorable environment for development, and the varieties of grapes under culture were extremely susceptible. Downy mildew rapidly became a devastating disease. Associated with this disease historically is the development of Bordeaux mixture. Had it not been for the discovery of Bordeaux mixture in 1885, the grape-growing industry of Europe would have been destroyed.

This disease presents an example of a group of closely related diseases. Downy mildews occur on a wide variety of hosts, both wild and cultivated. The fungi of this group, although closely related, represent a large number of distinct species.
Plate VI

A. Downy Mildew of Grape, affected leaf

B. Plasmopora viticola, sporangiophore and zoosporangia

C. Plasmopora viticola, hyphae and haustoria in host tissue
Symptoms:

New infections on leaves appear as small, water-soaked areas on the upper surface of leaves, becoming chlorotic and enlarging to about ½ cm. in diameter. On the under surface of the leaf lesions, a downy growth of sporangiophores bearing zoosporangia is apparent. The center of the lesion on the upper surface of the leaf frequently turns orange, then brown.

Other plant parts are also attacked. Infection of young shoots and tendrils may become systemic, causing a stunting of these parts. Depressed, dark-colored lesions are produced on more mature shoots. Young fruits are more severely injured than maturing fruits. Brownish spots which become covered with mildew during wet weather are typical on young fruits.

Pathological Histology:

The mycelium develops intercellularly in the parenchymatous tissue of the leaves and shoots. Prominent, knob-shaped haustoria are formed abundantly within the cells. In the leaf, the mycelium congregates near the stoma, through which the sporangiophores protrude. Sexual structures and the resultant oospores are formed in the intercellular spaces of the affected parts.

Causal Organism:

*Plasmopora viticola* is an obligate parasite. The mycelium is coenocytic. Sporangiophores protrude from the lower surface of leaves. The type of branching of the sporangiophore is used as a characteristic for classification of different downy mildew fungi. In *P. viticola* sporangiophores are branched and retrrenched at right angles, with egg-shaped zoosporangia being borne on the tips of the branches. Oospores are formed in leaf tissue and, in the spring, germinate to produce a single zoosporangium bearing zoospores.

Disease Cycle:

1. Penetration: Zoospores encyst, germinate, and send infection tubes through stomata.

2. Dissemination: Zoosporangia produced on sporangiophores are spread by wind and rain. The disintegration of host tissue releases oospores.

3. Overwintering: In some areas of this country oospores in leaves on the ground are the primary means of overwintering for the fungus. In other areas perennial mycelium in buds and shoots is probably of primary importance.

Favorable Environment:

Temperature between 18° and 25° C. and high humidity (80-100%) accompanied by rains, fogs, or heavy dews favor infection and disease development.

Control:

1. American grape varieties contain a degree of resistance to downy mildew. Complete immunity, however, has not been attained in commercial varieties.
2. In Arkansas and the eastern United States where grapes are grown, the grape-spray schedule is based on the control for black rot. If certain materials are used, downy mildew is controlled also. Bordeaux mixture applied immediately after bloom and again two weeks later effectively controls downy mildew. Ferbam, sometimes used for the control of black rot, is, however, ineffective against downy mildew.

3. Sanitation of the vineyard, involving the removal of infected prunings and diskling in the fall to turn under diseased leaves, aids in reducing potential inoculum.

WHITE RUST OF CRUCIFERS: \textit{Albugo candida} (Lev.) Kuntze

White rust occurs on many members of the mustard (Cruciferae) family. Both wild and economic plants are affected. Various species of the Capparidaceae are also attacked. In North America, radish and horse-radish are usually the most common economic hosts, although cabbage and cauliflower plants grown for seed production sometimes suffer. Shepherd's-purse is a very common weed host. The disease occurs generally throughout the world, but serious crop losses from it are relatively small.

The group of diseases called white rusts is caused by different species of the genus \textit{Albugo}. All of the fungi in this genus are obligate parasites. Other diseases in this group include white rust of spinach, white rust of sweet potato, and white rust of salsify.

Symptoms:

White rust may be found on any aboveground portion of the host. Infection may be local or systemic. Lesions first appear as light yellow spots which soon change to creamy white areas with a narrow yellow margin. The epidermis eventually ruptures and rolls back, exposing a white powdery mass consisting of zoosporangia of the parasite. Invasion of young plant tissue brings about systemic infection. In such cases cells of the stem and floral parts become enlarged and distorted. Seeds are not produced.

Pathological Histology:

The mycelium of the parasite is intercellular with intracellular haustoria. With local infection there is produced, immediately below the epidermis, short, club-shaped sporangiophores bearing chains of sub-spherical zoosporangia. The pressure exerted upon the host epidermis by the growth of these structures results in rupture of the epidermis to form white, shiny pustules. In young stems and floral organs the infection may become systemic. Hypertrophy and hyperplasia of the host cells result from stimulating substances secreted by the pathogen. The host tissues become greatly distorted and flowers may lack their normal shape and color.

Causal Organism:

\textit{Albugo candida} produces zoosporangia in chains. Each zoosporangium usually produces, upon germination, biflagellate zoospores. The mycelium is coenocytic and the haustoria are knob-shaped.
A. Late Blight of Potato, affected leaf

C. White rust, diseased stem of shepherds-purse

E. Albugo candida, antheridium and oogonium

B. Phytophthora infestans, sporangiophore and zoosporangium

D. Albugo candida, sporangiophores and zoosporangia

F. Albugo candida, oospore
Antheridia and oogonia develop between the host cells, especially in those host tissues with systemic infection. A fertilization tube is produced through which an antheridial nucleus passes to fertilize the oogonial egg. A single oospore is formed which, upon germination, is said to produce a vesicle containing zoospores. Presumably, reduction division occurs at this time. The character of the oospore wall is one criterion used in distinguishing among species of Albugo. Races of the parasite have been identified.

Disease Cycle:

1. Penetration: *A. candida* usually produces zoospores whose germ tubes penetrate the stomata of the host. In rare instances sporangia germinate directly, with the resulting germ tubes entering stomata.

2. Dissemination: Zoosporangia are carried by air currents and splashing rain. Oospores in plant refuse can be carried by any mechanical means.

3. Overwintering: Oospores are very resistant to adverse conditions and are a principal means of survival. Perennial mycelium in the systemically-infected hosts also persists from one season to the next.

Favorable Environment:

In general, this disease requires damp, cool conditions for rapid spread. A film of moisture on the host is necessary for infection. Zoosporangia require a chilling and drying period before they will produce zoospores. Once these conditions have been satisfied, the spores germinate over a relatively wide temperature range (10°-20° C.).

Control:

White rust of crucifers is not generally of great economic importance and expensive control measures have not been justified. Rotation, eradication of weed hosts, and destruction of infected plant residues may be suggested where the disease repeatedly causes damage.

**RHIZOPUS SOFT ROT OF SWEET POTATO:** *Rhizopus nigricans* Ehr.

*Rhizopus nigricans* is widely distributed in nature. The fungus is primarily a saprophyte, frequently called black mold of bread. As a pathogen, *R. nigricans* is best known for its ability to cause a soft rot of mature fruits and vegetables in transit and storage. Each year serious losses accrue to growers and consumers of fresh fruits such as strawberries and peaches. Records of damage to these fruits at terminal markets indicate that losses between 10 and 25 per cent of a shipment are not uncommon.

*Rhizopus* soft rot was first reported on sweet potato about 1890. Since this early report, the disease has been found wherever sweet potatoes are grown. It is generally conceded to be the most destructive storage disease of this crop. Annual losses between 1 and 70 per cent, with a mean near 10 per cent, have been reported at terminal markets.
Symptoms:

The disease is typified by a soft, watery rot which rapidly destroys the attacked tissues. The coloration of infected sweet potato roots is not greatly changed in the initial stages of decay. A brownish discoloration, however, develops during the latter stages. A watery exudate is noticeable whenever the skin is ruptured, and a mild yeast-like odor can be detected. From such ruptures on decayed roots, typical aerial mycelium may develop producing sporangiophores bearing sporangia. "Ring rot" develops when rotting occurs in a ring around the root. Desiccation of the attacked tissues leaves a typical sunken collar or ring.

Pathological Histology:

After infection is established, decay is the result of an enzyme, pectinase, secreted by the fungus and acting in advance of mycelial growth. The mycelial strands branch freely and ramify in all directions. Plasmolysis of the disorganized cells occurs to further enhance the soft, watery condition in the decayed portions. Although pectinase production is an important factor in rot development by Rhizopus spp., there is apparently no correlation between parasitism and the ability of a particular species to produce greater or lesser amounts of the enzyme.

A simple experiment aids in demonstrating the production of the enzyme by the fungus and the manner in which decay of the plant tissue occurs. Briefly the experiment involves growing R. nigricans on a liquid medium and demonstrating that an enzyme has been secreted into the liquid by the fungus. A satisfactory medium is made by boiling chopped sweet potato and decanting off the liquid. This liquid is then filtered and put into flasks which are plugged and sterilized. The flasks containing the sterile medium are inoculated with spores of R. nigricans. The fungus grows rapidly on this solution and soon forms a mat of mycelium over the surface. At about the time the first sporangia mature, the liquid is filtered so that none of the fungus remains.

To demonstrate the presence of enzyme in the culture filtrate, thin slices of sweet potato are added to it. Maceration or other changes in the sweet potato tissues are then observed macroscopically and microscopically. A set of suggested observations includes placing sweet potato slices in beakers containing the following:

1. Culture filtrate
2. Culture filtrate which has been boiled
3. Sterile (uninoculated) medium
4. Physiological saline solution

In addition a series of beakers containing the tissue slices in culture filtrate are placed at several different temperatures to observe the speed of enzymatic action as related to temperature. If such an experiment is carried out individually or presented as a demonstration by the instructor, record your observations using the experimental outline. Understand the reasons for each step in the procedure and be able to discuss logically all of the observed results.
Causal Organism:

Although several species of *Rhizopus* are known to cause soft rot of fruits and vegetables, *R. nigricans* probably causes the major damage. *R. nigricans* is a facultative parasite. The coenocytic mycelium anchors itself to a substrate by means of rhizoids. Aerial hyphae, called stolons, connect the rhizoids. At the point of anchorage, sporangiophores develop in an upright manner, bearing terminal, spherical sporangia. A bulbous extension of the sporangiophore, called a columella, extends into the sporangium. Sporangiospores are formed within the sporangium. At maturity, the sporangial wall ruptures releasing the non-motile spores.

Sexual reproduction requires the presence of compatible mating strains referred to as + and -. Lateral segments from hyphae of these strains protrude to form progametangia. Soon terminal cells which are morphologically similar, called gametangia, are delimited. The gametangia remain attached to the hyphae by suspensors. The zygote is formed soon after fusion of gametangia, and develops into a thick-walled resistant spore, the zygospore. This diploid spore remains dormant. Reduction division occurs upon germination to form a sporangiophore and sporangium.

Disease Cycle:

1. Penetration: *R. nigricans* is a wound invader. Actively growing tissue is seldom attacked.

2. Dissemination: Sporangiospores are spread by wind and air currents. These spores appear to be omnipresent. Disintegration of host tissue releases zygospores.

3. Overwintering: *R. nigricans* is such an excellent saprophyte that it is capable of subsisting on crop refuse wherever it might be. Zygospores serve as resting spores, and sporangiospores may also survive for long periods.

Favorable Environment:

Temperature between 15° and 23° C. is optimal for disease development. A relative humidity between 75 and 90% favors disease damage.

Control:

The primary control is the regulation of storage environment. After the crop is stored, raising the temperature to approximately 30° C. for about two weeks allows curing of wounds. The storage temperature should then be reduced to near 5° C. to retard any existing decay.

Measures to insure proper sanitation of the storage room and containers should be practiced.

Care in handling during harvest and storage should be practiced to reduce bruises and wounds as these are areas through which the fungus enters the root.
A. *Rhizopus nigricans*, asexual reproduction

B. *Rhizopus nigricans*, progametangia

C. *Rhizopus nigricans*, gametangia

D. *Rhizopus nigricans*, zygote

E. *Rhizopus nigricans*, zygospore
DISEASES CAUSED BY ASCOMYCETES

The largest Class of the fungi is the Ascomycetes. This Class is characterized by a septate mycelium and by the production of ascospores in an ascus. An ascus is a sac-like body in which spores, called ascospores, are formed as a culmination of the sexual process. Individual asci may arise "naked" without a distinct fruiting body, but more frequently they are grouped in specialized fruiting bodies or ascocarps. Three general types of ascocarps are

1. Cleistothecium, a roughly spherical, entirely enclosed body
2. Perithecium, a globular to flask-shaped structure having an opening (pore) to the exterior
3. Apothecium, a cup- or saucer-shaped, open structure bearing the asci on its upper surface in a palisade-like layer.

A stimulus derived from a sexual process is involved in the initiation of the ascocarp. The sexual processes, however, are not consumated until the ascospores are formed.

Although a few of the Ascomycetes produce only the ascosporic stage, asexual reproduction by means of conidia is common. The manner of conidial production is often used to differentiate species.

In the ascomyceteous plant pathogens, conidia generally occupy a prominent role in the active parasitism and production of disease. The conidia, sometimes called summer spores, serve to distribute the pathogen during the growing season of the host plant. With the advent of unfavorable growing conditions, such as the onset of winter when the host plant matures or becomes dormant, the fungus generally becomes a saprophyte. During the saprophytic phase, the perfect or ascocarpic stage is developed. When more favorable growing conditions for the host occur, such as in the spring of the year, the ascospores mature and are discharged to incite the initial or primary infection on a susceptible host. As the fungus develops in the host tissue, conidia are produced, released from the host, and rapidly spread the pathogen. Of course, exceptions to the general development and association of these pathogens in their saprophytic and parasitic phases occur.

A wide diversity of types and degrees of pathogenicity occur within the Ascomycetes. Representative diseases illustrating the pathogenesis and morphology of these pathogens are presented.

PEACH LEAF CURL: *Taphrina deformans* (Fckl.) Tul.

Peach leaf curl is distributed throughout the world wherever peaches are grown. This disease seriously affects leaves, flowers, tender shoots, and fruit of peach, nectarine, and peach almond. It is most severe in the spring of the year shortly after the trees leaf out. Prior to 1900, losses due to this disease were extremely heavy and constituted a major hazard to peach pro-
duction. The formulation of effective and efficient remedial measures has reduced this disease to a role of relatively minor importance.

Symptoms:

The most characteristic expression of the disease is the puckering of the leaves along the mid-rib and the resultant curling. Chlorosis of the diseased areas is also characteristic. In nature, a whitish bloom, composed of the ruptured cuticle of the host and asci and ascospores of the pathogen, is found on the surface of diseased tissue. This whitish bloom cannot be detected in preserved material.

Pathological Histology:

The septate mycelium of *Taphrina deformans* develops intercellularly among the parenchyma cells of the leaves, cortex of twigs, and superficial tissues of fruits. Hyperplasia and hypertrophy of cells occur in the infected areas. In the leaf, cellular disturbance is most readily observed in the palisade parenchyma, but it extends throughout the parenchymatous tissues. The affected leaf areas are thicker than normal. Observation of affected cells in the leaf reveals the absence of chloroplasts.

Causal Organism:

*Taphrina deformans* is a facultative saprophyte. In the host tissue, three different types of hyphae are recognized:

1. Vegetative hyphae, consisting of cells irregular in size and shape found between the parenchyma cells in the leaf

2. Distributive hyphae, developing beneath the epidermis of diseased peach twigs

3. Fruiting hyphae, similar in appearance to vegetative hyphae except that they bear stalk cells between the cuticle and epidermis of the host.

Sexual organs have not been found for this organism; however, a binucleate condition of the mycelium develops soon after infection. Ovoid ascogenous cells develop beneath the cuticle on the upper surface of the leaf. Fusion of nuclei takes place to form a diploid nucleus. From each ascogenous cell, an ascus is formed. No ascocarp is produced. The asci rupture the host cuticle and arise "naked" on the upper surface of the leaf. After three successive nuclear divisions in the ascus, one of which is reductional, eight ascospores are formed. Ascospores are capable of budding in a yeast-like manner before or after discharge from the ascus. These secondary spores should be referred to as bud-ascospores and not conidia; therefore, asexual reproduction in the true sense is lacking.

Disease Cycle:

1. Penetration: Direct penetration through the cuticle of young leaves occurs.
Plate IX

A. Peach Leaf Curl, affected twig and leaves
B. Taphrina deformans, asci and ascospores
C. Peach Leaf Curl, cross section of diseased leaf
D. Erysiphe graminis, conidiophores and conidia
E. Erysiphe graminis, cleistothecium
2. Dissemination: Ascospores and bud-ascospores are forcibly discharged from the asci to become wind-borne. No secondary spread of the pathogen occurs.

3. Overwintering: Ascospores and bud-ascospores develop thick spore walls and persist on twigs and leaf scales.

Favorable Environment:

Relatively cool temperature, between 10° and 16° C., wet spring weather, and cloudy skies favor infection and disease development.

Control:

Peach leaf curl is readily controlled by a single fungicidal application during the dormant season. Either a 5-50 solution of lime-sulfur or a 3-4-50 solution of Bordeaux mixture is a suitable material. These materials act as eradicants rather than protectants in eliminating the disease.

POWDERY MILDEW OF CEREALS AND GRASSES: Erysiphe graminis D. C.

This disease is presented as an example of a group of diseases known as "powdery mildews". The powdery mildew pathogens are all obligate parasites which attack a wide range of host plants. Some species attack many different plants; others are relatively specific. The most common genera are Uncinula, Phyllactinia, Podosphaera, Microsphaera, Sphaerotheca, and Erysiphe. Most species produce, on the host surface, a grayish growth consisting of mycelium, conidiophores, conidia, and, later, the much darker cleistothecia. The mycelium is, in most cases, superficial, with haustoria entering the epidermal cells. Many of these mildews are most severe under dry climatic conditions.

The cleistothecia, or fruiting bodies, of these fungi have peculiar appendages attached to their outer walls. The shape of these appendages, in conjunction with the number of asci involved, constitute the major factors in the identification of the various genera.

Contrary to the situation with many powdery mildews, powdery mildew of cereals and grasses is more severe under moist weather conditions. Wheat and barley usually suffer the most damage, with other cereals and grasses affected to a lesser extent. In the United States, the Great Lakes, the Pacific coast, and the Atlantic seaboard are regions where this mildew is most prevalent.

Symptoms:

Gray to white areas appear first on aerial parts of the host. These may spread and coalesce. Conidia accumulate as a dusty powder on the host surface. Chlorosis of the host ensues and there is a gradual decrease in host vigor. Cleistothecia may, or may not, appear as black dots scattered throughout the mycelium. A reduction in both size and quality of kernel, as well as yield, results from the attack of the parasite.
Pathological Histology:

The mycelium is entirely superficial on the surface of the host, and specialized feeding organs called haustoria take food for the parasite from the living host cells. The haustorial development is roughly inversely proportional to the resistance of the host variety concerned. Host tissue gradually loses its chlorophyll and in severe cases may become necrotic. Apparently, light exclusion by the heavy mycelial mat is also a factor in reduced photosynthetic activity.

Causal Organism:

Erysiphe graminis forms septate, branched mycelium which produces great numbers of erect conidiophores, each bearing a chain of one-celled, hyaline, oval or rectangular conidia. Haustoria in the epidermal cells may have numerous slender extensions, or may be only a lobed knob, depending on the host variety.

Cleistothecia may appear in late summer as the result of sexual reproduction. An antheridium and an ascogonium are produced. Fusion of sex cells is thought to take place after which ascogenous cells arise resulting eventually in asci and ascospores. Within the simple-appendaged cleistothecium are several asci. Each ascus contains from 4 to 8 elliptical ascospores.

The fungus has been shown to be highly specialized with reference to its hosts. At least 7 forms (subspecies) are now known, i.e., E. graminis tritici, hordei, avenae, secalis, poae, bromi, and agropyri. In addition, physiologic races within these forms have been identified.

Disease Cycle:

1. Penetration: Conidia and ascospores germinate by germ tubes, form sucker-like discs or appressoria, and enter the host through the cuticle by means of a fine infection peg.

2. Dissemination: Conidia, which are produced in abundance on the superficial mycelium on the host, are readily wind-borne. Cleistothecia may be scattered by the wind and other means. This stage, however, seems to be of minor importance in the life cycle of this pathogen.

3. Overwintering: The chief manner of survival appears to be as mycelial mats on overwintering grasses and cereals. To a lesser extent, cleistothecia serve to carry immature ascospores from one season to another.

Favorable Environment:

A cool, moist or foggy climate favors development of this disease (most other powdery mildews are favored by dry conditions). Conidia germinate best at about 10° C. and at humidity levels of 95% or above. Conidia can germinate, however, to some extent at very low moisture levels. Infection, disease development, and spread of inoculum are optimum within the temperature range of 10°-20° C. Proper development of cleistothecia depends upon fluctuating temperatures. Ascospore formation is accelerated by alternate wetting and dry-
ing. Vigorous, succulent host plants are most susceptible to the parasite. Indications are that excessive nitrogen fertilization of the host increases the disease.

Control:

Resistant varieties: Cereal varieties differ in their resistance to the various races of the pathogen. A goal of the plant breeder is to produce a variety having resistance to all races of the fungus. Several genes for resistance to certain races have been found. Present practice, however, is to avoid any highly susceptible varieties for the locality involved.

Fungicides: Powdery mildew may be effectively controlled by sulfur applications. These are usually too expensive for cereals, except for seed plots or show plantings.

APPLE SCAB: *Venturia inaequalis* (Cke.) Wint.

Apple scab is considered the most serious disease of apple in nearly every part of the world where the crop is grown. The disease is most destructive in those areas that have a relatively cool humid climate. In north central and northeastern United States, apple scab is the limiting factor in apple production. During the ten-year period from 1928 to 1938, losses from apple scab in the United States were estimated at 8 per cent of the crop.

Symptoms:

Apple scab affects leaves, flowers, and fruit. On certain apple varieties, scab is found occasionally on twigs. Lesions develop on both the upper and lower surfaces of leaves. In general, the lesions on the upper surface of leaves are conspicuous, olivaceous spots with definite margins. The lesions on the lower surface, however, tend to follow the veins of the leaf and the margins are indefinite. Leaf distortion is observed frequently. On the fruit, scabby, dark-colored spots are clearly demarcated. The uplifted cuticle may be observed as a whitish band at the margin of the spot. As the fruit matures in regions having a warmer climate, the spots commonly appear as russetted scars. As the fruit develops in size, the previous infection of immature fruit results in cracking and distortion of the area associated with scab lesions.

Pathological Histology:

In leaf, fruit, and twig infection, the invasion of the fungus is chiefly sub-cuticular, i.e., between the cuticle and epidermis. In later stages of disease development, the cells of the host beneath the infected area may die. The fungus is capable of developing intercellularly in this region, forming a stroma one to several layers thick. Conidiophores bearing conidia are produced on this stroma. Corky protective cells of the host develop to limit expansion of the fungus. When the leaves drop in the fall, the fungus becomes a saprophyte, and the mycelium permeates the entire leaf.

Causal Organism:

*Venturia inaequalis* is a facultative saprophyte. The mycelium of the fungus is uninucleate. It is initially hyaline and branching, but it soon be-
A. Apple Scab, affected leaf  
B. Apple Scab, affected fruit  
C. Venturia inaequalis, conidial stage  
D. Venturia inaequalis, perithecium  
E. Venturia inaequalis, ascus and ascospores
comes dark and stromatoid in the host. Pear-shaped, one or two-celled conidia are borne singly at the tip of conidiophores emerging from the stroma. After each conidium is released, the conidiophore grows slightly and produces another conidium. Cells of the conidiophore and conidium are uninucleate.

Perithecial development is initiated during the fall or early winter as a result of fertilization of the ascogonium by the antheridium, produced on different, sexually compatible mycelia. The perithecia mature in the spring of the year on leaves that have wintered on the ground. The culmination of sexuality is in the production of numerous ascii, each containing eight two-celled ascospores, within each perithecium.

Disease Cycle:

1. Penetration: Appressoria are formed by germinating conidia and ascospores to allow direct penetration through the cuticle.

2. Dissemination: Conidia are released only when wet; therefore, they are spread primarily by rain acting under the influence of wind and gravitation. Ascospores are forcibly ejected from the perithecia as the trees are leafing out. They may be carried some distance by the wind.

3. Overwintering: The fungus persists from one season to another as a saprophyte in the dead leaves on the ground.

4. Sources of Infection: Primary--Ascospores ejected from mature perithecia initiate infection. Secondary--Conidia produced on the surface of lesions from earlier infections continue to spread the pathogen during the current growing season.

Favorable Environment:

Cool, wet, and cloudy weather favors disease development. *V. inaequalis* is capable of growth and reproduction at temperatures above freezing to about 30°C; the optimum being between 16°C to 24°C. Rainy periods are necessary for spore distribution, and moisture favors spore germination and infection of the host. These same conditions favor tree growth, which provides tissue more susceptible to infection.

Control:

Efficient control of apple scab may be achieved by fungicide applications. Fungicide applications may be separated into two categories:

1. Ground sprays
2. Foliage sprays

Application of a ground spray of sodium dinitro-ortho-cresolate, ½ per cent, at the rate of 600 gallons per acre, before bud-break eradicates the fungus from the dead leaves on the ground, thus reducing the potential primary inoculum.
Foliation spray programs must vary to meet conditions peculiar to any area. Protection of foliage must begin before the first infection occurs in the spring. Repeated applications are necessary to protect the developing foliage and fruit during the period the pathogen remains active. The active period of the pathogen is relatively short in the south as compared to the northern apple growing areas.

In Arkansas, sulfur sprays are recommended for scab control as follows:

1. Cluster bud (when buds separate, but before blossoms open; lime sulfur
2. Calyx spray (when 7/8 of the petals have fallen); lime sulfur, 1-50, or a fine mesh wettable sulfur, 4 lbs. per 100 gallons of water
3. First cover spray (2 weeks after petal fall); same as No. 2 above.

Lime sulfur may burn the foliage during hot weather; therefore, a fine mesh wettable sulfur should replace lime sulfur when the temperature approaches 26°C. Various other fungicides, such as ferbam, captan, zineb, and the organic mercurials, are efficient in controlling scab and these may be recommended. The above does not represent a complete apple disease control schedule. In northern areas additional sprays are needed to control scab. In southern areas additional sprays are required to control other diseases.

Apple varieties exhibit differential resistance to infection by V. inaequalis. None of the commercial varieties is sufficiently resistant to permit omission of a fungicidal control program.

ERGOT OF GRAINS AND GRASSES: Claviceps purpurea (Fr.) Tul.

Ergot is a disease of cereals and grasses which is of particular concern because the ergot sclerotia contain alkaloids that are very poisonous to humans and animals. Stock may be poisoned as a result of grazing on diseased grasses. In some areas of the south, Dallis grass (Paspalum dilatatum) is often diseased with ergot and a number of cases of stock poisoning have been recorded. Ergot poisoning in humans has occurred after grain contaminated with ergot sclerotia was used in making flour.

Symptoms:

The most conspicuous evidence of this disease is the dark brown ergot or sclerotium which takes the place of a grain in the inflorescence of affected cereals. This structure is hard, and generally longer and larger than the normal grain. Before ergot formation a sweetish secretion (honey-dew) is present on the affected grain. On Dallis grass ergot has a much different appearance. Affected grains enlarge to produce white to cream-colored sclerotia. Their shape is irregular, resembling exploded popcorn grains.

Pathological Histology:

The mycelium of the fungus ramifies through the young ovaries and produces the conidial stage on the surface. The affected grain enlarges and is ultimately entirely filled with mycelium which hardens into a sclerotium.
A. Ergot, affected rye

B. *Claviceps purpurea*, sclerotium with several perithecial stromata

C. *Claviceps purpurea*, conidia

D. *Claviceps purpurea*, section of perithecial stroma
Causal Organism:

Claviceps purpurea persists during the winter as sclerotia in the ground. In the spring on these sclerotia tan, fleshy structures called stromata are produced. The stroma consists of a swollen head borne on an elongated stipe. Many perithecia are embedded in the swollen head of the stroma. Ascospores are discharged from asci in the perithecia and initiate primary infection. Shortly after young ovaries are infected, tiny conidia develop in profusion on short conidiophores on the surface of the ovary. A sticky fluid is exuded in which the conidia are embedded. This is the "honeydew" stage which is attractive to insects. Infected grains later develop into sclerotia, which drop to the ground as the grain matures or are harvested with the normal grains.

Ergot on Dallis grass is caused by a different species (C. paspali) which develops in a similar manner.

Disease Cycle:

1. Penetration: Ascospores and conidia germinate on the stigmas. Germ tubes grow around the ovary walls and enter at the base of the ovary.

2. Dissemination: Ascospores are forcibly discharged and carried by wind to other inflorescences. Conidia are carried from diseased to healthy flowers by insects. Sclerotia may be transported about the country in harvested grain.

3. Overwintering: The fungus persists from season to season as sclerotia in the soil or in stored grain.

Favorable Environment:

Sclerotia must be subjected to freezing temperatures before stromata are produced. Temperatures around 20° C. favor stromal production. During the blossoming period, cool, cloudy weather favors infection by producing conditions favorable for stromal development and by prolonging flowering.

Control:

Ergot is avoided in cereals by using ergot-free seed.

In pastures the disease is controlled by frequent mowing to prevent the development of flowering heads.

BROWN ROT OF PEACH: Sclerotinia fructicola (Wint.) Rehm.

Brown rot is known as the most generally destructive disease of peach and other stone fruits in the United States, Europe, Australia, and New Zealand. Pome fruits are also damaged as a result of this disease. Losses from brown rot include

1. Destruction of fruit in the orchard and in transit
2. Blighting of blossoms
3. Cankering of twigs
4. General weakening of the tree
In the United States, annual losses due to the destruction of fruit in the orchard and in transit exceed $5,000,000.

Symptoms:

During the blooming period for peaches, blossoms are blighted. Many of these blighted blossoms remain attached to the pedicel over a long period. The pathogen frequently grows through the infected blossoms into the twigs, resulting in the development of cankers. Under favorable conditions, canker development may extend from infected twigs into larger limbs. A gummy substance is exuded from damaged twigs. During moist weather, the affected tissues are covered with masses of conidia.

Fruits are not commonly attacked before the ripening period, although green fruit may be rotted if the surface becomes defective. Initial lesions appear as small, round, brownish discolorations. The entire fruit rapidly becomes rotted. Masses or tufts of conidia form on the fruit surface, frequently in concentric rings. In the later stages of decay, the fruit shrivels to become a dry, hard, wrinkled mummy, hanging in the tree or falling to the ground.

Pathological Histology:

The mycelium of the fungus develops intercellularly through the tissues of the blossoms. In canker development the fungus continues its intercellular development through the cambium and phloem parenchyma tissues. During the earlier phases of fruit infection, the mycelium is intercellular. Secretion of an enzyme, propectinase, in advance of mycelial growth causes the middle lamella between the host cells to be dissolved. Plasmolysis of the cells rapidly follows.

Causal Organism:

_Sclerotinia fructicola_ is a facultative saprophyte. In addition to this pathogen, two other species of _Sclerotinia_, _S. laxa_ and _S. fructigena_, cause brown rot of stone and pome fruits; however, _S. fructicola_ is the common pathogen in most sections of the United States.

Conidia are borne in branching chains on short conidiophores, which arise in tufts from infected tissues.

Sexual organs have not been clearly demonstrated. Microconidia are observed in old cultures and on mummies. Since microconidia function in fertilization in other fungi in the Ascomycetes, it is theorized that these bodies fertilize ascoconidia. The sexual phase is initiated just beneath the surface of the mummies on the ground. In the spring, from these mummies develop one to many, small, cup-shaped, sexual fruiting bodies called apothecia. The stalk bearing the apothecium is called a stipe. The inner surface of an apothecium is composed of a layer of paraphyses or sterile hairs and asci bearing ascospores. This layer is referred to as the hymenium.
Plate XII

A. Brown Rot of Peach, affected fruit

B. Sclerotinia fructicola, conidiophore and conidia

C. Sclerotinia fructicola, mummy with apothecia

D. Sclerotinia fructicola, diagram of cross section of apothecium

E. Sclerotinia fructicola, asci and ascospores
Disease Cycle:

1. Penetration: The stigmas of blossoms are penetrated in a manner similar to pollen tube invasion. The manner of entrance into fruit varies. Immature peaches are attacked through wounds, especially punctures made by insects, such as plum curculio and oriental fruit moth, and lesions from diseases such as bacterial spot and scab. Mature fruit is invaded down hair sockets and through wounds.

2. Dissemination: Ascospores and conidia are chiefly spread by wind and rain.

3. Overwintering: Mummies on the tree and ground serve to carry the fungus from one crop season to another.

4. Sources of infection:
   
   Primary
   a. Ascospores from apothecia develop from mummies on the ground
   b. Conidia are produced on mummies hanging in the tree

   Secondary:
   a. Conidia produced on the surface of diseased tissue
   b. Mycelium passing from infected to non-infected fruits in contact with each other provide secondary infections

Favorable Environment:

A relatively high soil moisture and temperatures between 17° and 20° C. favor the development of apothecia in the spring. Temperature is not critical, however, as apothecia develop to some extent at temperatures ranging from near freezing to about 30° C.

A relative humidity of 85 per cent or higher is required for the production and germination of conidia. The optimum temperature for disease development is near 25° C. As the temperature varies from this optimum, a correspondingly longer period is required for spore germination and infection.

Control:

Cultural and sanitation practices: Removal and destruction of affected fruit before and after harvest eliminate sources of current infection and possible overwintering phases of the pathogen, respectively. Wild Prunus species growing near the orchard should be eradicated to eliminate sources of possible infection.

Pruning trees and thinning fruit simplify fungicide applications to protect the fruit left on the tree.

Disking the orchard to bury mummies is a questionable practice. Mummies may persist for several years and repeated disking will return buried mummies to the surface, allowing apothecial development.
Stericooling of harvested fruit before shipment has become an established practice for reducing the severity of brown rot in transit. Stericooling involves the immersion of packed peaches in chlorinated water held at 0° C. for 15 to 20 minutes. The peaches are then loaded and shipped in refrigerated trucks and railway cars.

Fungicide applications: To control blossom blight, sprays are applied at 3- to 4-day intervals during the blossom period. One to four applications may be necessary, depending upon the duration of bloom. In Arkansas, Phygon, an organic fungicide, 3/4 pound per 100 gallons of water, or a Phygon, 1/2 pound, wettable sulfur, 4 pounds, mixture per 100 gallons of water has proven efficient for protecting open blossoms.

To control fruit rot, complete coverage of fruit one month before harvest with wettable sulfur, 6 pounds per 100 gallons of water, is essential. If the weather is favorable for the development of fruit rot, additional sulfur sprays or dusts should be applied at about 7 day intervals through harvest.

Since wounds are ideal portals of entry for the fungus, control of insects and other diseases of peach which attack the fruit is essential for brown rot control.
DISEASES CAUSED BY FUNGI IMPERFECTI

Fungi Imperfecti (Deuteromycetes) is an artificial assemblage containing those fungi in which a sexual stage is lacking, or has not been discovered. Whenever a sexual stage is found and associated with a particular "imperfect fungus", the organism is then classified in the proper genus in the Ascomycetes, Basidiomycetes, or Phycomycetes.

In the Fungi Imperfecti, classification is based primarily on the absence or presence of asexual spores (conidia) and the morphology of their production. Conidia are produced on:

1. Conidiophores appearing singly or in masses
2. Conidiophores aggregated into an acervulus
3. Conidiophores borne within a pycnidium

Many plant diseases are caused by "imperfect fungi". It is of importance to note, however, that asexual production of spores is associated with active parasitism in the other classes of the fungi, particularly in the Ascomycetes.

FUSARIUM WILT OF COTTON: Fusarium oxysporum f. vasinfectum (Atk.) Snyder and Hansen

Fusarium wilt is considered one of the most destructive diseases of cotton. In 1954 losses were estimated at nearly 1.2 per cent of the crop.

The vascular fusarial wilts comprise a group of economically important diseases on a variety of crop plants. These diseases are caused by strains of Fusarium oxysporum. Each strain of the organism attacks only a limited number of species of plants. An organism with such a limited host range is referred to as being "specific", as contrasted to a "non-specific" pathogen which attacks an unlimited number of different plants. The strain of F. oxysporum attacking cotton has been reported to cause damage to alfalfa, tobacco, and okra.

Symptoms:

Diseased plants are stunted, and produce fewer and smaller bolls and leaves. The leaves turn yellow to brown, wilt, and death of plants ensues. The most characteristic expression of plants attacked by vascular fusaria is the discoloration of the vascular elements. When leaves are detached, this is observed as discolored spots representing the vascular strands. When the stems or roots are cut open, the woody cylinder is observed to be darkened or have dark streaks within it. Sometimes the tip of the tap root or a side root will be darkened and dead. This symptom has been called "black root rot".

Pathological Histology:

The pathogen invades the plant through the fibrous root system. The mycelium is found chiefly in the large xylem elements. Affected vessels become...
browned and partially plugged by the growth of tyloses and the accumulation of
gum-like substances. Pectic enzymes produced by the fungus have been reported
to be involved in this effect. The fungus is also reported to produce a toxin
which has an injurious effect on the host.

Causal Organism:

_Fusarium oxysporum_ f. _vasinfectum_ is an "imperfect fungus" lacking a
sexual stage of development. In the absence of a susceptible host the fungus
can persist in the soil on organic matter for several years as mycelium,
chlamydospores, or macroconidia. Two types of conidia, macroconidia and micro-
conidia, are formed by this fungus. Macroconidia are larger, sickle-shaped,
each composed of several cells, as compared to the smaller, one- or two-celled,
oval microconidia.

_Fusarium oxysporum_ is a species composed of many strains or forms which
differ only in their ability to cause disease in specific plants. This dif­
ference in pathogenicity is not considered sufficient reason for differentiat­
ing into separate species.

Disease Cycle:

1. Penetration: The fungus enters the young roots of susceptible va­
rieties of cotton by direct penetration. Entrance into normally re­
sistant plants is through wounds on the roots.

2. Dissemination: Spores and mycelia of the pathogen are spread by any
means that moves contaminated soil. Macroconidia become air-borne to
a slight extent. The fungus is seed-borne in a low percentage of
cotton seed from infected plants.

3. Overwintering: The pathogen persists in the soil as mycelia, chlamydo­
spores, and macroconidia over a period of several years.

Favorable Environment:

The optimum temperature for growth of the pathogen and infection of the
host is between 25° and 32° C. Growth of the fungus and infection of the host
are reduced as the temperature varies from the optimum, although mycelial de­
development occurs between 10° and 40° C.

_Fusarium_ wilt is more severe on light, sandy soils that have an acid pH.
Although the fungus grows well in culture over a pH range from 2.5 to 9.0, the
disease is of little or no importance in neutral or alkaline soils.

Soil moisture does not appear to be a critical factor in disease develop­
ment. Sufficient moisture for plant growth is apparently sufficient for growth
of the fungus. Damaged plants are more apparent, however, when soil moisture
is limited.

Cotton plants suffering from potash deficiency are more susceptible to
_Fusarium_ wilt. High applications of nitrogen and phosphorus tend to increase
_Fusarium_ wilt. A proper balance of N-P-K in a fertilizer program will enhance
yields and aid in controlling this disease. The rate of application of the
various elements will depend upon their availability in a particular soil.
Plant parasitic nematodes are important factors in predisposing plants to *Fusarium* wilt. Root-knot, root-lesion (meadow), and sting nematodes attack roots of cotton to provide wounds for entrance of the fungus. In addition to the direct damage from the nematodes, normally wilt-resistant varieties of cotton become susceptible to *Fusarium* wilt when roots are damaged.

Control:

The formulation of measures that are uniformly acceptable for the control of *Fusarium* wilt of cotton is impossible.

A properly balanced fertilizer program with the role of potash emphasized will reduce the amount of *Fusarium* wilt of cotton and increase yields, especially in soils deficient in potash.

Wilt-resistant varieties of cotton are available. Since 1940, various experiment stations have released varieties which are wilt-resistant and agronomically acceptable, such as Coker 100 wilt, Empire WR, Auburn 56, and Plains. Deltapine and Stoneville cottons have little or no resistance to *Fusarium* wilt.

If a soil is infested with both nematodes and *Fusarium*, all varieties of cotton are more susceptible to wilt. Control of nematodes to reduce the severity of the complex is necessary. A rotation of 2 to 3 years with root-knot nematode-resistant crops reduces losses from wilt and increases yields. Economical control of this complex is also obtained by the application of nematicides in the row several weeks before planting. Formulations of ethylene dibromide or dichloropropene-dichloropropane have proven satisfactory for this treatment.

**ANTHRACNOSE OF BEAN:** *Colletotrichum lindemuthianum* (Sacc. and Magn.) Brioso and Cav.

Bean Anthracnose, a disease caused by a seed-borne fungus, was first discovered in Germany in 1875 and first observed in the United States in 1885. It is a major disease in many parts of the world and occasionally causes losses of snap and dry beans in the eastern, midwestern, and southern states. While the disease is most severe on bean, it also affects lima bean, scarlet runner bean, tepary bean, mung bean, cowpea, kudzu bean, and broad bean.

Symptoms:

The disease may occur on any part of the plant above the ground and at almost any stage of growth. Anthracnose is evident on the pods as sunken, irregular, brown lesions exuding pinkish spore masses when moist. Narrow, dark red to black lesions occur along the veins on the underside of leaves. Dark, sunken cankers occur on stems and petioles. Dry beans show dark discolored spots that are most obvious on white-seeded beans. These lesions may involve as much as half the seed surface, and may reach downward into the cotyledons.

Anthracnose may develop on snap beans in transit. In such cases infection takes place in the field about the time the beans are picked, and developing lesions are not visible as they are packed.
A. *Fusarium* Wilt of Cotton, cut stem
B. *Fusarium oxysporum* f. *vasinfectum*, macroconidium, microconidia
C. Bean Anthracnose, affected pod
D. *Colletotrichum lindemuthianum*, conidial stage
E. Southern Blight, affected tomato stem
F. *Sclerotium rolfsii*, sclerotia and hypae
Pathological Histology:

The mycelium of the pathogen is localized in the tissue of a lesion and does not spread internally to other parts. The pathogen may penetrate the seed and may be present as mycelium in the cells of the seed coat or cotyledons, and as spores between the cotyledons, seed coat, and embryo.

Causal Organism:

*Colletotrichum lindemuthianum* produces a mycelium that is branched, septate, hyaline at first, becoming dark-colored with age. Conidia are produced in acervuli developed on stromatic layers. Setae occur sparingly. Conidia are continuous, hyaline, oblong, cylindrical, with ends rounded or somewhat pointed at one end. Presence of the ascigerous stage has not been confirmed. Numerous physiologic races of the organism exist and certain strains produce sclerotia in culture.

Disease Cycle:

1. Penetration: An infection peg forms on the side of the appressorium in contact with the host and penetrates the cuticle by mechanical pressure.
2. Dissemination: The conidia, which are borne on acervuli, are scattered by spattering and wind-blown rain. Cultivation of bean fields while rain or dew is present on the foliage provides another important means of dissemination. Contact of spores with man, animals, and implements spreads the pathogen.
3. Overwintering: The pathogen survives the winter in the seed and on infested plant debris in the soil.

Favorable Environment:

Relative humidity of 92 per cent or above is necessary for infection, the optimum being close to 100 per cent. The optimum temperature for the growth of the fungus is between 21° and 23° C. Rainy weather at frequent intervals is essential for development of an epidemic.

Control:

A rotation period of 2 to 3 years with non-susceptible crops and field sanitation eliminate the fungus on overwintering debris. Western-grown seed, which is free of the organism, is widely used in this country. Crop cultivation when foliage is dry prevents excessive spread. Protective spraying may be employed, but if the organism is eliminated by rotation, sanitation, and clean seed, there should be no need to put other measures into practice.

SOUTHERN BLIGHT: *Sclerotium rolfsii* Sacc.

Southern blight, sometimes called southern wilt, often causes severe losses to a wide variety of crops throughout southern United States. Nearly all vegetables, weeds, and legumes are affected by the disease. Many ornamentals may be seriously injured and killed. Although its complete host range
has never been determined, little or no infection has been observed on such hosts as grasses, small grains, and corn. Cotton may be attacked in the seedling stage but becomes resistant as the plant matures.

Symptoms:

The earliest symptoms, as observed on tomato, are a water soaking and slight darkening of the cortex of the stem at, or just below, the soil line. A collar rot is soon produced, accompanied by wilting, yellowing or leaf shedding by the aboveground parts of the plant. Diseased stems often show a vascular discoloration extending as much as six inches above the necrotic area. A moldy growth of white mycelium intermixed with a large number of sclerotial bodies in all stages of maturity is produced at the base of the stem and on the ground around the plant. The sclerotia are spherical, have the appearance of mustard seeds, and range in color from white to tan depending on the stage of maturity. Fruit in contact with infested soil may be affected with a brown rot which is sometimes soft and watery. The rotted area on fruit is covered with white mycelium and numerous sclerotia. Diseased plants usually have no definite pattern of occurrence but are scattered in a random fashion about the field.

Pathological Histology:

Infection of the underground part of the stem and taproot destroys the cortex. The fungus produces large amounts of oxalic acid in its metabolism. When in contact with living plants this oxalic acid is apparently responsible for death of the host tissue near the mycelium. The host tissue is killed some distance in advance of the mycelium. The fungus thus lives essentially as a saprophyte on tissue killed by its metabolic products.

Causal Organism:

Southern blight is caused by a soil-borne fungus which ordinarily produces no spores and is known as Sclerotium rolfsii in the Fungi Imperfecti. Occasionally isolates or strains are observed which produce basidiospores and it is then identified as Pellicularia rolfsii. The mycelium apparently does not live long in the soil. Under ideal conditions (warm temperature, abundant moisture, and an abundance of organic matter), the mycelium often grows rapidly through a limited area of soil. Growth may suddenly cease, then the mycelium dies and nothing remains but dormant sclerotia. Factors regarding breaking of this dormancy of the sclerotia are incompletely understood. Under some conditions the sclerotia may remain dormant several years.

Disease Cycle:

1. Penetration: As mentioned above host tissue is killed by oxalic acid prior to invasion by the fungus.

2. Dissemination: Since this fungus ordinarily produces no spores it is disseminated by means of sclerotia and mycelia. Sclerotia may be moved from place to place in cultivation or drainage water. Sclerotia and mycelium may also be carried on plants which are transported about the country, such as tomatoes or sweet potatoes.
3. Overwintering: The fungus overwinters as dormant sclerotia.

Favorable Environment:

The disease is favored by temperatures around 30°C, and an abundance of soil and air moisture. It may be severe immediately following the plowing under of large amounts of green manure. The disease is present on all types of soils but it is most prevalent on light, poorly drained soils. The presence of a damaging amount of the disease in a particular field seems to have little bearing on the amount of disease at future dates.

Control:

No adequate control for this disease is known. The destruction of diseased plants and susceptible weeds may help reduce the loss. Rotation with cereals and other resistant crops is a precaution that may assist to some extent. Prevent movement of plants from fields known to be infested.
DISEASES CAUSED BY BASIDIOMYCETES

The fungi in the Class Basidiomycetes are more familiar to the layman than other fungi. Much attention has been directed toward rust and smut diseases of cereals and other plants, and the fleshy fungi (such as mushrooms) are familiar objects because of their prevalence and size.

The characteristic structure of the Basidiomycetes is the basidium. The basidium is a club-shaped, unicellular or multicellular organ which bears 4 basidiospores exogenously. The development of the basidiospores is homologous to the production of ascospores in an ascus in the Ascomycetes. The vegetative mycelium of the Basidiomycetes is septate and branched. The nuclear condition of the mycelium is either binucleate or uninucleate, depending upon whether fertilization has occurred or not.

A wide range of parasitism is found in the Basidiomycetes. Many of the Homobasidiomycetes, a subclass of the Basidiomycetes containing the mushrooms, cause wood decay and rots of trunks and roots of living trees. The rust and smut pathogens comprise the Heterobasidiomycetes, the other subclass of the Basidiomycetes.

Smut Diseases

The smut fungi are all facultative saprophytes. Small grains and grasses are the most common plants attacked, although a wide and diversified number of plants are hosts to specific smuts.

Five species of smuts causing diseases of corn and small grains are presented. These species illustrate the two smut families, the Ustilaginaceae and Tilletiaceae, which are separated by the type of basidium produced. The five presented also illustrate local, blossom, and seedling infection types of smuts. Control of the smut diseases is based upon a knowledge of these fundamentally different infection types.

Although the basidium is characteristic of the Basidiomycetes, the presence of chlamydospores (teliospores) is the conspicuous and constantly associated feature of the smuts.


Common smut of corn is the most conspicuous and easily recognized of all diseases of corn. Teosinte, a large annual fodder grass, is the only other host susceptible to infection by this pathogen. The prevalence of corn smut varies from year to year and from one area to another. In the United States, annual yield reduction of corn attributable to corn smut is estimated to be between 2 and 5 per cent. Percentage losses in sweet corn production are considered to be much greater than for field corn.
Symptoms:

Galls or tumors of variable size and shape are produced on the aerial parts of the plant, including stems, leaves, tassels, and ears. At first, the galls are protected by a glistening whitish membrane of host tissue. As the galls mature, the membrane ruptures to expose a dark, powdery mass of chlamydo-speres.

Pathological Histology:

The fungus develops intercellularly after penetration. As a rule, the mycelium is restricted in its development in the host. Hyperplasia and hypertrophy of the infected tissues occur. Chlamydospores are formed by segmentation of the cells of the septate hyphae as the galls mature.

Causal Organism:

No sexual organ is formed by any of the smut fungi. Fusion of hyphae of two compatible lines occurs, and nuclear migration takes place to change the hyphae from an uninucleate to binucleate condition. A binucleate condition of the hyphae is necessary to allow typical invasion of the host tissue and production of chlamydo-speres. These asexual spores represent the conspicuous phase of the pathogen. Upon germination of the chlamydospore, a four-celled basidium is formed which bears a basidiospore upon a sterigma protruding from each cell. Nuclear fusion and reduction division in the basidium precedes development of basidiospores. The basidiospores can bud in a yeast-like manner to produce secondary basidiospores. In contact with susceptible tissue, these spores germinate and penetrate the plant. Many biotypes of the pathogen have been reported.

Disease Cycle:

1. Penetration: The pathogen invades embryonic tissue directly at any time during the growing season, and through wounds of matured tissue.

2. Dissemination: Rupture of the host membrane covering the smut galls releases the chlamydo-speres. Spread of chlamydo-speres is primarily by wind. Chlamydo-speres may be spread to a slight extent on the surface of seed, and in contaminated soil or manure.

3. Overwintering: The pathogen persists from one crop season to another as chlamydo-speres in the soil and crop refuse.

Favorable Environment:

A relatively high temperature favors germination of chlamydo-speres and basidiospores and infection of the host. Germination of the spores can occur between 50 and 30°C, with the optimum temperature between 260 and 320°C. Moisture conditions sufficient for normal corn growth are apparently ample for spore germination and infection.
A. Corn Smut, affected ear

B. Ustilago maydis, germinating chlamydosporites

C. Tilletia foetida, germinating chlamydosporites

D. Tilletia foetida, secondary basidiosporites
Control:

In commercial plantings of corn, the use of relatively resistant varieties is the only feasible means of control. Sanitation measures and rotation may aid in reducing losses from corn smut in isolated instances.

LOOSE SMUT OF BARLEY: *Ustilago nuda* (Jens.) Rostr.


In the loose smuts of barley and wheat, the young embryo becomes infected in its early formative stages in the flower. The development of the pathogens and their subsequent control are similar. The smut infecting barley, however, will not attack wheat, and vice versa.

Between 1917 and 1939, annual losses in the United States due to loose smut of barley varied between 0.75 and 4.5 million bushels. Annual losses in wheat during this same period varied between 3 and 18 million bushels.

Symptoms:

Infected plants tend to flower slightly in advance of healthy plants. All the floral organs are destroyed by the respective pathogens. Initially the smut masses in the spike are covered with a delicate membrane, which later ruptures to expose the powdery masses of chlamydospores. The chlamydospores are removed by wind at the time normal plants are flowering to leave a bare rachis. Seeds developing from newly infected blossoms appear normal.

Pathological Histology:

The young ovary becomes infected and the mycelium establishes itself inside the seed, where it remains dormant. When the seed germinates, the mycelium begins to grow intercellularly, without materially damaging the plant. The fungus becomes systemic, and finally forms chlamydospores as the plant develops a flowering head.

Causal Organism:

The loose smut of barley and wheat pathogens differ from the corn smut pathogen in the following ways:

1. Few if any basidiospores are produced on the hyphae from the germinating chlamydospores

2. The mycelium becomes binucleate soon after germination of the chlamydospore

3. The chlamydospores are not long-lived and do not function as over-wintering spores.

Physiological specialization of the pathogens exists.
Disease Cycle:

1. Penetration: The binucleate infection tube enters through the flower stigma, and grows through the style into the ovary.

2. Dissemination: Flower parts are replaced by masses of chlamydoospores. They are released at the time of normal flowering by rupture of the thin covering membrane. Spread of the chlamydoospores is primarily by wind. The mycelium establishes itself inside the seed and is seed-born.

3. Overwintering: The fungus persists as dormant mycelium inside the seed during the grain-storage periods.

Favorable Environment:

Humid weather during the flowering period of wheat or barley is essential for infection. Warm soil temperature favors the development of the mycelium in the young seedlings. Very rapid growth of the seedlings may allow the plant to outgrow the systemic invasion of the mycelium.

Control:

A drastic seed treatment to eliminate the internal mycelium is at present the only control. A hot water seed treatment is effective. Seed are presoaked in cold water for 12 hours, immersed in water held at 54° C. (129° F.) for exactly 13 minutes, cooled and dried, and planted immediately. More recently, control has been reported by soaking the seed for 6 hours at room temperature in water, followed by soaking in a 0.2 per cent Spergon solution for about 40 hours. Viability of seeds is greatly reduced by the seed treatments.

BUNT OF WHEAT: Tilletia caries (DC.) Tul. and T. foetida (Wallr.) Lirg.

LOOSE SMUT OF OATS: Ustilago avenae (Pers.) Rostr.

Bunt or stinking smut is one of the most destructive diseases of wheat. Annual losses to wheat production in the United States are estimated to be about 25 million dollars. In addition to losses in yield and quality, damage from bunt may be manifested in increased production cost, losses from fire in harvesting equipment caused by smut explosions, and through allergic effects on humans.

Loose smut is the most important smut disease of oats in several areas. In 1945, aggregate losses due to loose and covered (another seedling infection type) smuts of oats in Wisconsin, Iowa, Illinois, and Minnesota amounted to more than 27 million dollars.

Symptoms:

Bunt of wheat: Heads of wheat infected with bunt can be recognized in the field prior to harvest by their darker bluish-green color, spreading glumes and slim heads. Plants are frequently stunted. The internal contents of the
seed are replaced by mycelium which segments into chlamydospores; however, the pericarp of the seed remains intact. An odor resembling that of decaying fish is associated with the chlamydospore masses and is the basis for the name stinking smut.

Loose smut of oats: The apparent damage to oats from loose smut is confined to the panicle. The entire panicle is transformed into a smutty powder at flowering time.

Pathological Histology:

The development of the mycelium within the host is similar to that found in loose smut of barley and wheat. Infection, however, occurs in the seedling stage rather than through the flower.

Causal Organism:

Ustilago avenae is similar to the corn smut pathogen in its development. The Tilletia spp., however, differ in their characteristic development of the basidium and the basidiospores. The basidium is non-septate. Filiform basidiospores, 8 to 16 in number, are borne at the apex of the basidium. Fusion between the basidiospores to form H-shaped structures upon the basidium is frequently observed. Races of the pathogen have been reported.

Disease Cycle:

1. Penetration: Invasion takes place by direct penetration of young seedlings by means of appressoria in contact with the cuticle.

2. Dissemination: Loose smut of oats--The fragile membrane surrounding the spore masses is ruptured at flowering time and the chlamydospores are spread by wind. Bunt of wheat--The pericarp of the seed is broken during harvest to release the enclosed chlamydospores. Spread takes place by chlamydospores on the seed, and by wind-borne spores released during threshing.

3. Overwintering: Loose smut of oats--The chlamydospores persist on the surface of the seed. Bunt of wheat--The chlamydospores overwinter on the surface of the seed and to some extent in some soils.

Favorable Environment:

Loose smut of oats is favored by a temperature between 18°C and 22°C and a relatively low soil moisture during the seedling stage of oats. Bunt of wheat is favored by a lower temperature, between 5°C and 15°C, and a relatively low soil moisture.

Control:

Treatment of seed with appropriate fungicides is the first essential in the control of seedling infecting smuts. New Improved Ceresan and Ceresan M (organic mercury compounds) are widely used for this purpose. In addition, varieties of oats resistant to loose smut are available.
Varieties of wheat resistant to bunt have been developed for areas where this disease is a limiting factor in wheat production. The pathogen is so variable, however, that a continuous breeding program to provide resistant varieties is necessary. Seeding during periods of warm temperatures tends to allow the wheat to escape much infection.

<table>
<thead>
<tr>
<th>Place of primary infection</th>
<th>Seedling Infection Type</th>
<th>Blossom Infection Type</th>
<th>Local Infection Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of infection</td>
<td>Seedling</td>
<td>Blossom</td>
<td>Leaves, etc.</td>
</tr>
<tr>
<td>Transmitted from crop to crop</td>
<td>On the seed</td>
<td>In the seed</td>
<td>In the soil</td>
</tr>
<tr>
<td>Principal control measures</td>
<td>Seed dusting with fungicides</td>
<td>Hot water seed treatment</td>
<td>Sanitation, rotation</td>
</tr>
<tr>
<td>Examples</td>
<td>Bunt of wheat</td>
<td>Loose smut of wheat</td>
<td>Corn smut</td>
</tr>
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<td>Covered smut of oats</td>
<td>Brown loose smut of barley</td>
<td>Head smut of corn and sorghum</td>
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<td></td>
<td>Loose smut of oats</td>
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<td></td>
<td>Covered smut of barley</td>
<td></td>
<td>White smuts of spinach and ornamentals</td>
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<td></td>
<td>Black loose smut of barley</td>
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<td></td>
<td>Covered smut of sorghums</td>
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<td>Onion smut</td>
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<td>Millet smut</td>
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<td>Stem smut of rye</td>
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<td>Leaf smut of rice</td>
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<td>Flag smut of wheat</td>
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<td>Kernel smut of rice</td>
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Rust Diseases

The rusts are all obligate parasites. They are among the most commonly encountered parasitic fungi. A very large number of wild and cultivated plants is attacked by one or more of the pathogens in this assemblage of closely related organisms. Extensive damage to certain crops results from rust epidemics.

Many rusts have complicated life histories. The phenomenon of "heteroecism" is almost unique. The term refers to a condition in which the fungus develops on one host through part of its life cycle, then must complete the cycle on an entirely different host. The majority of rusts is, however, autoecious, completing the entire life cycle on one host.

Rust fungi may produce as many as five different types of spores: spermatia, aeciospores, uredospores, teliospores, and basidiospores. Each type of spore may differ in morphology and function. One type, the spermatium, is sometimes not considered a true spore since it acts only as a sex cell.

Two families of rusts are commonly recognized: Melampsoraceae and Pucciniaceae. Many members of the Melampsoraceae have conifers as their aecial host (White Pine Blister Rust). The Pucciniaceae is the larger of the two families and includes the majority of the more familiar rusts (rusts attacking small grains). Representative diseases caused by rusts in the latter family are presented.

STEM RUST OF WHEAT: *Puccinia graminis tritici* Eriks. and Henn.

Stem rust of wheat is a classical disease. It has received attention in writings from Biblical times to the present. It is certain that for 2500 years or more grain crops of the civilized world have suffered serious damage from this disease. *Puccinia graminis* probably ranks as the most destructive plant pest. Several serious epidemics of stem rust have occurred in the United States and Canada, with damage during a single season amounting to between two and three hundred million dollars.

Stem rust is best known as a disease of wheat. Various varieties of the pathogen, however, attack oats, rye, barley, and a number of grasses. The principal alternate host is the common barberry, *Berberis vulgaris*.

Symptoms:

On wheat, stem rust is characterized by long, narrow streaks (pustules) that erupt on the surfaces of stems, leaf sheaths, and leaves. The pustules first appear dark red (uredia). As the grain approaches maturity, the pustules become black in appearance (telia).

On the barberry, small, yellowish to pinkish spots develop on the leaves. Small, dark bodies (spermagonia) appear in a spot on the upper surface of the leaf, and soon, white, cup-shaped structures (aecia) develop on the lower surface.
Pathological Histology:

In both barberry and grain hosts, the mycelium is intercellular, and haustoria invade the cells. Only parenchyma cells are attacked. Infected cells are not killed. In the grain host, the mycelium is confined to the area between the vascular bundles, and develops in a linear manner. Between one and three weeks after infection, the epidermis is ruptured by developing spores. In the barberry, the infected cells are incited to hypertrophy and hyperplasia. They lack chloroplasts.

Causal Organism:

*Puccinia graminis* is an obligate parasite. It is heteroecious and heterothallic. In the spring, the development of the fungus begins with the germination of the two-celled teliospore, each cell containing a single diploid nucleus. Reduction division occurs in the production of a four-celled basidium upon which are produced the uninucleate, haploid basidiospores. This development occurs on the overwintered grain straw.

The basidiospores are wind-borne to the barberry where they germinate and infect young leaves. The mycelium resulting from this infection is uninucleate. Tiny flask-shaped spermatonia, containing spermatia, are produced by the uninucleate mycelium. No further development of the fungus is possible until the first step in fertilization occurs. This is the initiation of a binucleate condition of the mycelium. It is brought about by the fusion of compatible mycelia or, more commonly, by the transfer of spermatia from one spermatogenic lesion to another by insects. As soon as the binucleate (dicaryotic) condition occurs, the fungus is stimulated to renewed activity. A larger lesion, involving both the upper and lower surfaces of the leaf, is produced. On the lower surface of the leaf cup-shaped aecia, containing binucleate aeciospores, develop.

The aeciospores are wind blown to the grain host. Soon after infection, binucleate, oval-shaped, one-celled uredospores rupture the host epidermis. The uredospores cause secondary infection on the susceptible grain but, as the grain matures, dark, two-celled, thick-walled teliospores are produced upon the same mycelium which bore the uredospores, and gradually replace them in the pustule. The young teliospores have paired nuclei which soon fuse to consummate the sexual process.

The species *Puccinia graminis* is subdivided into seven physiological strains. Each strain has a definite host specialization. For instance, *P. graminis tritici* primarily attacks wheat. Each of these strains is composed of physiological races defined by their ability to incite specific reactions on certain varieties of the grain crop involved. Stakman and co-workers have identified about 240 races of *P. graminis tritici*.

Disease Cycle:

1. Penetration: Aeciospores and uredospores germinate and penetrate the grain host through stomata. The basidiospores germinate and penetrate directly through the cuticle of young barberry leaves.
A. *Puccinia graminis*, spermagonium and aecium

B. *Puccinia graminis*, portion of uredium

C. *Puccinia graminis*, section of diseased leaf with portion of telium
2. Dissemination: Basidiospores, aeciospores, and uredospores are spread primarily by wind. The basidiospores and aeciospores are spread over a radius of one to two miles. The uredospores, however, are carried many miles in the upper air currents. Viable uredospores have been trapped at altitudes exceeding 10,000 feet. Prevailing winds determine the direction of spread over long distances. The teliospores remain attached to the wheat straw by stalk cells. Spermatia are carried from one spermagonium to another by insects.

3. Overwintering: Teliospores on old straw persist through the winter in the northern United States and Canada. They cannot survive the hot summers in the southern United States and Mexico. The fungus remains active through the winter in the southern part of the United States and Mexico as mycelia producing uredospores in fall-sown grain.

Favorable Environment:

Uredospores can germinate at temperatures between 50 and 25°C. The optimum temperature is between 18° and 20°C. A film of water must be present on the surface of the leaf to allow germination and penetration.

Epidemics are favored by a group of interrelated factors:

1. Large acreages of susceptible varieties of wheat
2. Prevalence of physiological races capable of attacking the wheat varieties
3. Mild winters in southern areas which allow abundant uredospore production
4. Prevailing winds from the south to carry the uredospores
5. In northern states, presence of abundant inoculum from barberry

Control:

Although much time and money have been expended for research and control, the disease continues to cause serious losses. The following practices tend to alleviate the damage:

1. Resistant varieties--The development of varieties resistant to the prevalent races of the fungus is a history of successes turned to failure. New varieties resistant to the common races are introduced only to be damaged by a new or different race of P. graminis tritici.

2. Eradication of the barberry--In the northern states, a barberry-eradication program has been in progress since 1918. Although complete eradication of the barberry will not eliminate the disease, some benefits accrue from this program. First, stabilization of the pathogen at the present number of races is accomplished by removing its place of hybridization. Second, later infection of wheat in the north occurs when aeciospores are absent. The later the infection of wheat, the less severe is the damage.

3. Possibility of fungicide applications--Various fungicides successfully protect the grain plant from infection by rust. Although ap-
plications of fungicides for control of diseases of field crops have been considered impractical, this costly method of control may be forced into practice by necessity.

CEDAR APPLE RUST: Gymnosporangium juniperi-virginianae Schw.

Cedar apple rust is native to this country and generally is distributed over the eastern and midwestern parts of the United States. This rust is important on both hosts, apple and red cedar (Juniperus virginiana L.), but is most important on apples when orchards are located in areas where red cedar is native. The rust has only three of the possible four sori, i.e., telia, aecia, and spermagonia. Since there is no repeating stage (uredospores) on either host, the disease can persist only where both hosts are present. Gymnosporangium juniperi-virginianae is the most important of several species of the genus which infect apple in North America.

Symptoms:

On apple the disease appears on leaves as small greenish-yellow spots which gradually enlarge, change to orange-yellow, and become surrounded at the border by concentric red bands. Spermagonia appear on the upper surface of leaf lesions and the yellow exudate is conspicuous. Hypertrophy follows on the lower side of the lesions and later yellow, cup-like aecia develop.

Numerous points of infection may kill the leaf tissue, causing premature defoliation which interferes with normal development of the tree. Lesions appear on immature fruit causing dwarfing and malformation. Twigs of very susceptible varieties may be affected.

On cedar, symptoms are not obvious until the second spring after infection of young twigs by aeciospores. Lesions are then seen as round galls on the twigs, half inch or more in diameter. In moist weather these galls develop long, orange, jellylike horns which consist of masses of teliospores with long gelatinous stalks. This is the most conspicuous stage of the disease. The galls die later but remain attached to the tree a year or more.

Pathological Histology:

The fungus invades the epidermal cells of the apple host to form a primary hypha and proceeds inter- and intracellularly, forming small haustoria. The cedar galls consist chiefly of parenchyma with intercellular mycelium and haustoria of the fungus.

Causal Organism:

Gymnosporangium juniperi-virginianae is an obligate parasite. Since there is no uredial stage, no reinfection of cedar occurs. In the spring the basidiospores are carried to the apple where they germinate and infect the young leaves. Spermagonia are formed on the upper surface of the leaves and spermatia are exuded. Presumably the fungus is heterothallic, and a mixture of exudates is provided by insects and rain. The binucleate stage is initiated in the aecial primordia. Yellow aeciospores develop in the cup-shaped aecia.
A. Cedar Apple Rust, affected apple leaf

B. Cedar Apple Rust, cedar gall

C. Gymnosporangium juniperi-virginianae, germinating teliospore

D. Cedar Apple Rust, cross section of diseased apple leaf
The aeciospores are carried to red cedar and initial gall development starts on the cedar leaf. The cedar may become infected in August or September but no visible signs of infection appear until July. The following spring when moist, moderately warm weather prevails, the telial horns are produced, and teliospores germinate in the gelatinous matrix to produce basidia and basidiospores. Two years are required for this fungus to complete its life cycle.

Several physiological races of the fungus have been reported.

Disease Cycle:

1. Penetration: Basidiospores germinate and penetrate the apple host directly. The apple leaf is susceptible to penetration only when young. Aeciospores germinate and enter the cedar leaf.

2. Dissemination: The aeciospores are borne in chains within the aecial cup and are gradually set free as they mature. The basidiospores are forcibly discharged from the gelatinous matrix. Basidiospores and aeciospores are carried to their respective hosts by wind.

3. Overwintering: The fungus lives as mycelium in the cedar gall.

Favorable Environment:

This rust depends upon moist, cool weather for its development. If the weather is hot and dry in the spring, the spore horns from the cedar galls are inhibited. Basidiospores are easily destroyed by dry air. Since only very young apple leaves are susceptible, the amount of rust that develops is dependent upon the age of the leaves when basidiospores are discharged.

Control:

Resistant varieties of both hosts are available and should be used when it is desirable to have apples and ornamental cedars in the same area. In intensive commercial areas removal of cedars within a mile of orchards has been recommended, but it is difficult to accomplish complete eradication where red cedar is native. Ferbam is an efficient fungicide for the control of cedar apple rust on apple and is used in early season sprays where the disease is severe.

BEAN RUST: Uromyces phaseoli typica Arthur

Bean rust is an example of an autoecious rust, i.e., its life cycle is confined to a single host. It is a very widespread disease which commonly assumes economic importance. Bean, lima bean, and several other species of Phaseolus are affected.

Symptoms:

Bean rust attacks the leaves, pods, and rarely the stems and petioles. It is most conspicuous on the leaves. Symptoms appear first on the lower surface of leaves and are evident as minute, almost white, slightly raised pustules (uredia) in which the rust-colored uredospores develop. Under favorable
conditions uredia are produced in one or two concentric circles around the first uredium. At times uredia are surrounded by a chlorotic halo. Toward the end of the growing season, as the plants become older, the uredium gradually becomes dark as dark-brown to black teliospores replace the uredospores.

Pathological Histology:

The mycelium produced in the early stage by the basidiospores is intracellular. The dicaryotic mycelium produced by the aeciospores is intercellular and the haustoria invade the parenchyma cells. In 8 to 10 days after infection, the epidermis ruptures exposing the uredospores.

Causal Organism:

_Uromyces phaseoli typica_ is an autoecious long-cycle rust. The haploid mycelium from basidiospores penetrates the cuticle directly. The aecial stage is rare in some areas. The dicaryotic mycelium from aeciospores penetrates through stomata and grows intercellularly. Under favorable conditions, the uredospore cycle is completed in 10 to 15 days. The teliospores are dark brown, one-celled, and thick-walled.

More than 20 physiological races of the fungus are known.

Disease Cycle:

1. Penetration: Basidiospores germinate and penetrate the cuticle directly. Aeciospores and uredospores germinate and penetrate the host through stomata.

2. Dissemination: Uredospores are released by eruption of the epidermis, and are carried by wind and by contact of man, animals, and implements.

3. Overwintering: The fungus subsists between crop seasons as uredospores in regions with mild winters, and as teliospores in colder climates.

Favorable Environment:

The optimum temperature for production and germination of the uredospores is between 16° and 25° C. A thin film of water is essential for germination and penetration of aeciospores and uredospores. Epidemics commonly build up from uredospores remaining from previous crops or from wind-blown spores from other areas.

Control:

The disease is effectively controlled by the application of sulfur dust soon after the first signs appear. Some control is obtained by the use of varieties resistant to prevalent strains of the fungus. Crop rotation, elimination of old bean straw stacks, and removal of volunteer beans aid in reducing rust.
Pathogenic Gill Fungi

**ARMILLARIA ROOT ROT: Armillaria mellea (Vahl) Quel.**

Most of the gill fungi are saprophytes, but one species, *Armillaria mellea*, causes root rot of forest and orchard trees. It is pathogenic on many other plants. A dry rot of potato tubers is caused by the fungus. In the United States the disease is probably most destructive to stone fruit and citrus trees in the Pacific Coast states.

**Symptoms:**

Varied manifestations of unthriftiness follow invasion of the root system of trees. Nothing characteristic is noted until the fruiting bodies of the fungus appear, usually in large numbers around the base of the trunk. The fruiting body is a mushroom, and the honey-colored top (pileus) is speckled and has a viscid appearance. On the lower side of the pileus are white gills. Below the pileus on the stalk (stipe) are the remains of the inner veil of the immature fruiting body (annulus). Another distinguishing feature of the fungus is the presence of hard black strands which ramify through the soil and along the roots and trunks. These strands are rhizomorphs and are often called "shoestrings".

**Pathological Histology:**

The rhizomorphs penetrate the cork, and branch within the cortex to form a thick, white, flaky mass of mycelium. If penetration goes deeper, the flakes are laid down in the phloem or in the cambium. Fungal hyphae from a fan established in the cambium travel in a radial direction through the living parenchyma of the medullary rays and penetrate the woody tissue of the tree. The fungus then proceeds from tracheid to tracheid by penetrating the lignified walls.

**Causal Organism:**

The mycelium develops in white, fan-shaped felts between bark and wood, and the felts are later replaced by subcortical rhizomorphs. Mushroom fruiting bodies are produced in clusters around the trunk of the host near the ground level. They usually appear in autumn and may not occur until the tree is dead or nearly so. Basidia are borne in the hymenium and are interspersed with paraphyses.

**Disease Cycle:**

1. **Penetration:** Rhizomorphs enter the host chiefly by penetrating cracks in the roots and trunks of trees.

2. **Dissemination:** Basidiospores are discharged in great numbers and are air-borne.

3. **Overwintering:** Rhizomorphs persist from season to season in trees and soil.
Favorable Environment:

The fungus is most pathogenic at temperatures at which host growth is slow, and least so at temperatures which promote rapid root development. Citrus plants are most severely affected at 10° to 18° C. The greatest root growth, in contrast, occurs at 17° to 31° C. In peach and apricot the disease is most damaging at 15° to 25° C. Roots, however, grow most rapidly at 10° to 17° C. Optimum temperature for growth of the fungus on malt agar is 21° to 25° C.

Control:

In orchards where occasional trees become infected and die, local sterilization of the soil is necessary before trees can be replaced. A common precaution is to trench the infested area occupied by the tree to prevent growth of rhizomorphs through the soil to infect roots of healthy trees.

Orchards, vineyards, or tuber crops should not be planted in recently reclaimed forest land since Armillaria is often present in woodlots.
DISEASES CAUSED BY NEMATODES

Nematodes are known commonly as eelworms. They are widespread in nature, occurring in habitats ranging from soil and water to that of parasitism of higher plants and animals. Plant parasitic nematodes seldom exceed one-eighth inch in length and in most cases are seen only with the aid of a microscope. In relation to their size, they have complex internal systems. Each has specialized outer coverings, muscular systems, a digestive system, excretory systems, a reproductive system, and specialized parts for feeding. In their developmental stages,

1. Nematodes usually lay eggs
2. Larvae are differentiated and hatch from eggs
3. Four larval stages precede adult differentiation, each stage being separated by a molt or shedding of external portions of the body
4. Adult females and males are differentiated.

The majority of plant parasitic nematodes is classified in the order Tylenchida. Members of this order possess an organ called a stylet or spear, which is specialized to allow puncture and feeding on cells of higher plants.

Major damage to plants occurs from nematodes feeding on roots. Certain nematodes, however, exhibit a preference for stems, buds, or leaves. Nematodes may burrow and feed inside the plant, or they may remain outside and feed on more superficial tissues.

Damage to plants as a result of feeding by nematodes frequently simulates damage attributed to drought or mineral deficiencies in the soil. Direct damage to plants is often manifest as a rotting, galling, or malformation of affected plant parts. In addition to this damage, other plant parasites may form complex disease associations with nematodes; the resultant crop damage exceeding that caused by either separate pathogen.

As a rule, the significance of nematodes as crop pests is not understood and the damage is greatly underestimated. Several hundred different species of nematodes are known to attack plants. Of this number, many are destructive to crops and, as research advances, others are being implicated as major pests. Serious crop losses are annually caused by

2. Meadow or root lesion nematodes, *Pratylenchus* spp.

and many others.

All crop and ornamental plants are susceptible to attack by one or more species of nematodes. The nematodes, however, vary in their preference for plant food. One species may feed on relatively few kinds of plants; a different species may cause damage to many different kinds of plants.
ROOT KNOT: Meloidogyne spp.

The several species of the root-knot nematode attack some 1865 different species of higher plants. Root knot is a common disease and frequently causes severe losses to a wide variety of crops, especially in the southern United States. Estimates of damage to crops in these states indicate that losses vary from a trace to one hundred per cent, with an average of about 10 per cent.

Symptoms:

Aerial portions of plants affected by root knot show varying degrees of yellowing and dwarfing, with a general unthrifty appearance. Since only the root system is affected, many plants appear normal until they suddenly wilt when soil moisture is limited. Although diseased plants generally remain alive in an unthrifty condition, death of plants may occur. The chief diagnostic symptom is observed as galls on the roots when the plants are removed from the soil. These root swellings vary from small, hardly discernible root enlargements to large spheroid galls. Proliferation of roots is frequently associated with the smaller root-galls.

Pathological Histology:

After entrance, the root-knot nematode larvae feed up and down the cortical region of young roots. Development of the nematode continues inside the plant root. Four molts occur before maturation of adults. The larvae which develop into relatively large, pear-shaped females come to rest in the root cortex with their heads oriented in the endodermis or pericycle and their bodies lying parallel to the longitudinal axis of the root. Males are detected with difficulty. As the nematodes feed on the plant cells by injecting their stylets, hypertrophy and hyperplasia of the cortical cells occur. Giant multinucleate cells are observed in the vicinity of the females' heads. Vascular elements become involved with the lateral and oblique displacement of cells.

The phenomena of hypertrophy and hyperplasia are attributable to both mechanical and chemical stimuli from the parasites. In feeding, the head of the nematode moves from one cell to another, inserting its stylet into the cell, and pulsating. Chemical substances are secreted into the cell through the stylet. The cells are not killed, but are stimulated to increased activity.

As the galls enlarge, the tail of the female is shifted toward the outside of the plant. The swollen female deposits her eggs in a gelatinous mass toward the surface, or on the surface, of the infected area.

Causal Organism:

Root-knot nematodes are obligate parasites of higher plants. Several different species of Meloidogyne cause root knot. In Arkansas, preliminary surveys indicate that M. incognita, M. incognita acrita, and M. hapla are present in destructive numbers. Each species of root-knot nematode tends to attack different plants in varying degrees; cotton is susceptible only to M. incognita acrita, and strawberries are susceptible only to M. hapla. M. incognita is more polyphagous than the other species.
A. Root Knot, affected tomato roots

B. *Meloidogyne incognita*, eggs and larval stages

C. *Meloidogyne incognita*, mature female and eggs in diseased root
All species of root-knot nematodes have essentially the same life cycle. Eggs are released into the soil. The larvae are differentiated in the egg where the first molt occurs. The larvae hatch from the eggs as second-stage larvae. The second-stage larvae must enter and feed on living plant cells. Four additional molts occur in the roots as the larvae grow and mature to adults. Fertilization of the females by males may occur; however, parthenogenesis is common. Eggs are deposited posterior to the female in a gelatinous mass. The number of eggs laid by a single female varies; 300 to 600 eggs are considered average. Depending upon soil temperature, the plant being parasitized, and other factors, the time required to complete a single generation is quite variable; 18 to 21 days is the absolute minimum.

Disease Cycle:

1. Penetration: Second-stage larvae penetrate the epidermal cells of meristematic root tissue.

2. Dissemination: The egg mass is pushed to the surface of the gall or released from the internal areas as the gall disintegrates. Dissemination occurs in the movement of diseased plants and contaminated soil. Movement of larvae through the soil is relatively slow and of little importance in distributing nematodes.

3. Overwintering: Eggs and second-stage larvae in the soil or plant debris, and adults in perennial hosts are all capable of persisting from one crop season to another. In the soil, the egg stage is more capable of withstanding adverse conditions than the semi-active second-stage larvae.

Favorable Environment:

1. Soil: Root knot tends to be most severe on plants grown in light, sandy soil. Extensive damage, however, is frequently observed on plants grown in heavier soils. Under field conditions, the parasite is most prevalent in the top 12 to 24 inches of soil. In association with diseased roots, the nematode has been found in the soil to a depth of 8 feet. Rainfall, height of water table, soil type, and other factors cause variations in depth distribution of root-knot nematodes.

2. Temperature: There is considerable variation among the species of Meloidogyne in their ability to withstand exposure to freezing temperature. The majority of the species is not capable of surviving in soil subjected to prolonged freezing. (M. hapla, however, is apparently adapted to such extremes of temperature, since this species is found commonly attacking potatoes from Minnesota to Maine.) A soil temperature below 9°C practically eliminates larval activity in the soil. Optimum damage to plants occurs at a temperature near 27°C. In general, mild winter temperatures and warm summers favor development of root knot.

3. Moisture: Moisture seems to be of little importance to root knot development. Soil moisture favorable to plant growth allows nematode activity and invasion of plant roots.
Control:

1. Prevention: Root-knot nematodes are frequently introduced into non-infested soil on roots of transplants, nursery stock, and other diseased plant parts. Although state laws prohibit the sale of such diseased plants, growers should inspect and discard plants suspected of harboring root-knot nematodes.

2. Limitation of spread: Machinery used in fields known to be infested with root knot should be carefully decontaminated before proceeding to new areas. Since eggs and larvae are carried in drainage water, run-off water from infested fields should be controlled.

3. Reduction of nematode populations in infested soil:
   
a. Rotation: Development of a suitable rotation program depends upon a knowledge of the species of *Meloidogyne* infesting a field. Crops resistant to the species present can be grown and result in striking reductions of nematode populations.

   b. Summer fallow: Since root-knot nematodes are obligate parasites, elimination of all plant growth during the summer will greatly reduce populations. This method of control is costly since no return is forthcoming on the investment in time, labor, and land. Summer fallow should be followed by fall planting of a green manure crop.

   c. Soil fumigation: Treatment of soil with nematicides is a promising method for combating nematodes. Certain volatile chemicals injected into the soil fumigate and kill the nematodes in the immediate area of injection. Methyl bromide, ethylene dibromide, chloropicrin, and a mixture of dichloropropane and dichloropropene are in commercial use as nematicides in some areas. The preparation for using these materials involves first the development of a level seedbed. Soil moisture should be adequate to germinate seed, and the soil temperatures should be between 90 F. and 280 C. Since methyl bromide is a gas at temperatures recommended for treatment, the material is released under a cover which prevents escape of the gas. The liquid fumigant is introduced into the soil in a number of ways. Holes 6" deep and spaced 12" may be punched into the soil and the proper amount of liquid poured in each hole. Holes are covered immediately. A hand injector may be used to inject the liquid at the same depth and spacing. Furrows 12" apart and 6" deep may be made and the fumigant dribbled into the furrow and covered immediately. On large acreages, tractor mounted equipment is available which introduces the proper amount of fumigant to the right depth. Most of the materials listed above cannot be used less than 15 days before planting date. Since the cost of materials and the cost of labor for treating a continuous acreage is high, it is often profitable to treat only small areas in the immediate proximity of the future crop plants. Spot treat-
ment has been used with peaches and cucurbits. By this method a circular area immediately surrounding the future location of each peach tree or watermelon hill is given thorough treatment. With row crops such as tomatoes, beans, or cotton, the row only is given a treatment prior to planting. All of the above materials are toxic to plants, animals, and humans. Recommendations of the various manufacturers as to handling and use should be rigidly followed.
VIRUS DISEASES OF PLANTS

Virus diseases are caused by pathogenic entities which are not visible by any optical microscope. Laboratory studies are, therefore, limited to observations on the effects of these pathogens on the hosts they attack. Being unable to see the actual causal agent, students sometimes conclude that little is known about this group of pathogens. Such is not the case. A discussion of virus diseases in a recent text should be studied in preparation for the laboratory observations and discussions on this group of diseases.

In general, plant viruses and virus diseases may be separated into two groups on the bases of means of transmission and type of symptom produced in plants:

1. Mosaic group
2. Yellows group

In the mosaic group of virus diseases, interspersion of various degrees of chlorosis with the normal green color of the leaf gives characteristic mosaic patterns of yellow and green. Transmission of most of the viruses within this grouping can be accomplished mechanically. In the yellows group of virus diseases, a nearly uniform reduction of chlorophyll occurs with relatively no mosaic pattern. Transmission of the viruses within this group usually cannot be accomplished mechanically.

Mosaic Group

Some of the most important virus diseases in this group are

1. Tobacco and tomato mosaic
2. Potato mosaics
3. Bean mosaics

CUCUMBER MOSAIC: Cucumber mosaic virus

Cucumber mosaic is one of the major diseases of cucumber. In addition to cucumber, however, an extensive number of plants, including both dicotyledons and monocotyledons, are known to be hosts of one or another of the many strains of this virus. Some of the other important diseases caused by the cucumber mosaic virus are southern celery blight, spinach mosaic or blight, some forms of tomato mosaic, and a mosaic of tobacco. Many common weeds are hosts and serve as reservoirs of the virus.

Symptoms:

Since the common cucumber mosaic virus is actually composed of variants or strains, many of which differ in the type of symptoms induced in certain plants, it is impossible to characterize typical symptoms. In general, the
mosaic type symptom is observed on young leaves of such plants as cucumber and tobacco. This is usually accompanied by a stunting and unthrifty development of the host. Necrotic streaks along leaf veins and stems are observed with some strains of the virus affecting some hosts. Necrotic local lesions are produced when some plants (cowpea, for example) are inoculated by mechanical means.

Pathological Histology:

In those plants showing the mosaic type symptoms, the virus becomes systemic soon after infection. However, symptoms are expressed only in the young leaves formed after inoculation. The virus is apparently both inter- and intracellular. In the mosaic pattern on the leaves, thicker palisade parenchyma is developed in the dark patches, while the palisade parenchyma is underdeveloped in the light patches.

Causal Agent:

The causal agent of cucumber mosaic is a virus. Physical properties are:

1. Thermal inactivation point between 65° and 75° C.
2. Aging in vitro in 6 to 19 days
3. Inactivation by dilution between 1-1000 and 1-20,000.

Systems of classification have been proposed in an attempt to name particular viruses. Each has serious disadvantages. It is acceptable to add the term virus to the name of the disease when it is necessary to refer to a particular virus.

Disease Cycle:

1. Transmission: The virus is readily transmitted mechanically. Since pickling cucumbers are picked small and frequently during a single season, this is an important factor in spread of the disease. Current season spread in many plants is chiefly by means of such aphids as the melon aphid and the green peach aphid. The spotted and striped cucumber beetles are also vectors of transmission. Transmission through the seed of wild cucumber has been reported.

2. Overwintering: Perennial host plants are the most common between-crop reservoir hosts. Such weed hosts as milkweed, ground cherry, pokeweed, and wandering jew are reported to be important reservoirs of the virus in various localities. In many areas of the south the virus is active on economic hosts throughout the entire year. The virus has been reported to overwinter in wild cucumber seed.

Control:

Eradication of wild host plants is feasible in special instances, e.g., control of southern celery mosaic in Florida by eliminating wandering jew near celery fields. Tolerant varieties of cucumber are now available for commercial use. Several varieties of spinach are resistant to this virus.
Yellows Group

Since the viruses in the yellows group are more difficult to manipulate and study, the diseases and viruses in this group are not as well characterized as those in the mosaic group. Some of the most important diseases in this group are

1. Peach yellows and little peach
2. Phony peach
3. Peach rosette
4. Curly top of sugar beets and tomatoes
5. Aster yellows

Aster yellows is the classical example in this group.

ASTER YELLOWS: Aster-yellows virus

Aster yellows is one of the most widespread of the virus diseases in the yellows group. The name was derived from the fact that the disease is conspicuous and destructive on China aster in many parts of the United States. In addition to aster and other ornamental flowering plants, common economic hosts are carrot, celery, lettuce, onion, and potato.

Symptoms:

First indication of disease on aster is a veinclearing in the young leaves. Leaves that are mature when symptoms appear are not affected. Normally dormant, axillary buds are stimulated, and chlorotic, spindly branches with relatively long internodes appear to give a "witches broom" effect. The main stem of the plant, however, is stunted. Leaves are deformed, and flowers are misshapen. This same type of symptom pattern is produced on other affected plants.

Pathological Histology:

The virus becomes systemic in the plant. After infection, an incubation period in the plant of about 38 days is necessary before symptom expression.

The relationship of the virus to the insect vector, Macrosteles spp., is incompletely understood. After feeding on diseased plants, an incubation period of about 10 days is required for the virus inside the insect before it becomes viruliferous. Evidence indicates that the virus increases greatly in the body of the insect, probably associated with the gut cells and the bloodstream.

Causal Agent:

The aster-yellows virus is a member of the yellows group. Since the virus cannot be transmitted mechanically, its physical properties are not known. Many viruliferous leaf hoppers subjected to heat treatments of 12 days at 31°C to 32°C permanently lost their ability to transmit the virus. Heat treatments of periwinkle and Nicotiana rustica plants have permanently inactivated the virus in their tissues.
Disease Cycle:

1. Transmission: The six-spotted leaf hopper, *Macrosteles divisus*, is the most common vector. However, more than a dozen other species of leaf hopper have been proven to be vectors. The virus can be transmitted artificially by grafting.

2. Overwintering: In mild climates, the virus may overwinter in viruliferous adult leaf hoppers. Infected perennial hosts are the common means of carrying the virus from one season to another.

Control:

Partial control of the disease has been accomplished in some instances by controlling or eliminating the insect vector by various methods. Eradication of weed hosts around cold frames may ultimately reduce the incidence of disease in the field. If the crop is of sufficient importance, it may be economically feasible to eliminate weed hosts from around the borders of fields, e.g., lettuce and endive on Staten Island. A general recommendation for control cannot be made.