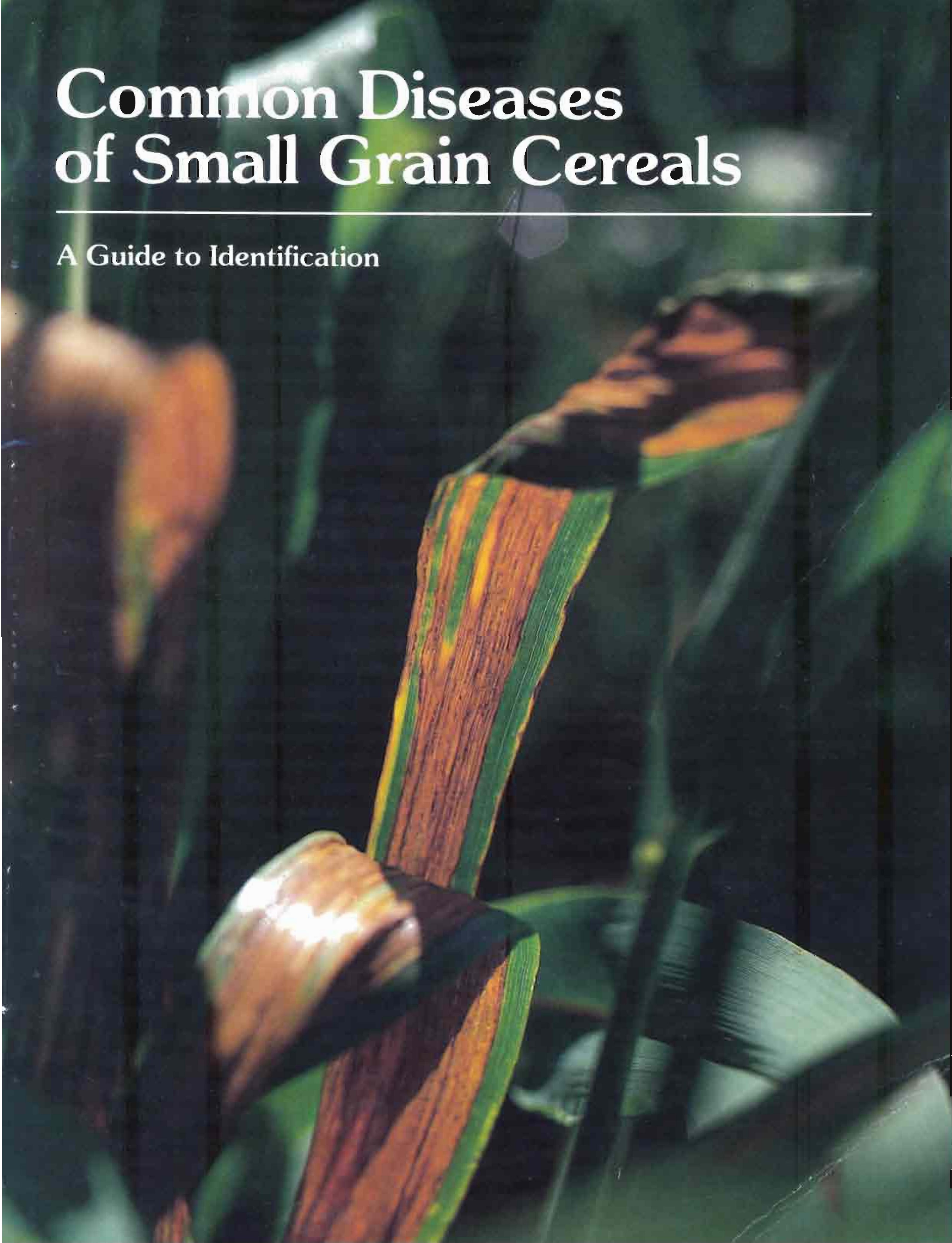
Common Diseases of Small Grain Cereals

A Guide to Identification



Common Diseases of Small Grain Cereals

A Guide to Identification

F.J. Zillinsky

CIMMYT

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Cover: Bacterial stripe, caused by *Xanthomonas translucens*, on barley (photo: S. Fuentes)

Cover design: Anita Albert

Acknowledgements

As with nearly all major publications, this book is the result of the interest and efforts of many people. For their help I am indebted and greatly appreciative; they are mentioned here in no particular order.

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Finally, a special note of appreciation to my wife, Hilda, whose support and faith over the years have enabled me to pursue my career wherever it led me. It is to her that I dedicate this book.

Dr. Frank Zillinsky

Preface

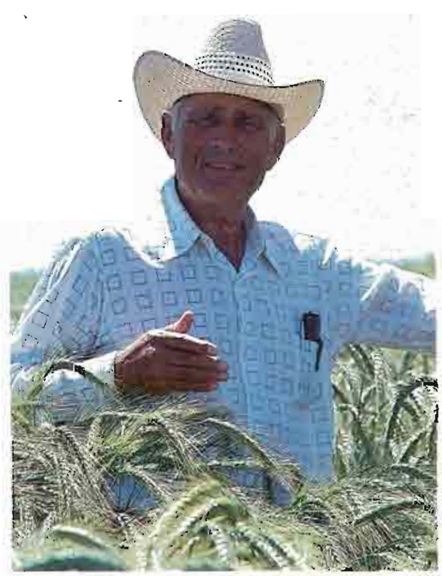
Dr. Frank Zillinsky came to CIMMYT in December, 1967, from the Ottawa Agricultural Research Station, where he was employed for some 17 years by the Canada Department of Agriculture as a Cereal Crop Specialist. He was appointed as the leader of CIMMYT's triticale improvement program, and held that position until his retirement from CIMMYT in July, 1982. Under Frank's guidance, triticale germplasm underwent very rapid improvement, from not much more than a biological curiosity to the threshold of widespread commercial cultivation. That alone is an accomplishment which would satisfy most agricultural researchers, yet Frank offered more.

Though not formally trained as a cereal pathologist, Frank was often confronted with the need to identify diseases afflicting various small grain cereals. Characteristically, Frank studied in his spare time to develop this necessary expertise. To his disappointment, he found no publications designed specifically to help a person in his situation: a plant breeder who needed to identify the diseases affecting his crops.

For many years, Frank worked closely with cereal pathologists from all over the world, studying, learning, and accumulating thousands of photographs and disease observations for his own edification. About nine months before Frank's scheduled retirement from CIMMYT, Norman Borlaug suggested that hundreds of agricultural researchers in developing countries and elsewhere could benefit from the kind of book Frank had sought but not found. He also suggested that Frank was the person best qualified to write such a book. Hence, this guidebook was prepared, drawing heavily on Frank's own collection of photographs and knowledge of small grain diseases, but with significant input from friends and collegues the world over.

Plant diseases are probably the greatest constraint to increasing global small grains production in the 1980s and beyond. Accordingly, CIMMYT is placing a very high priority on breeding wheat, triticale and barley for enhanced resistance to various diseases, and we encourage national programs in developing countries to do the same. In fact, the essence of CIMMYT's wheat improvement strategy lies in multilocation testing and a heavy reliance on national program staff for performance evaluations of germplasm. Moreover, many developing countries now have sophisticated plant breeding programs capable of making valuable contributions to improving disease resistance.

Success in breeding for enhanced disease resistance will be the result of a cooperative effort among many national programs and CIMMYT, and will require the free exchange of germplasm and performance data. To develop useful breeding objectives and effective crossing plans, accurate information as to which diseases are affecting which cultivars is needed, as well as data on the severity of affliction. This guidebook addresses the problem of accurate disease identification. We strongly believe it is a necessary addition to the literature and that it will prove useful to thousands of agricultural researchers all over the world. It is a significant achievement, a fitting capstone to Frank's distinguished career, and perhaps the most important publication to be recently forthcoming from the CIMMYT Wheat Improvement Program.



Dr. Frank Zillinsky

Byrd C. Curtis
Director, CIMMYT Wheat
Improvement Program

Facing page: Triticale seed (wheat x rye)

Contents

Introduction

The main purpose of this guidebook is to assist in the identification of diseases and of pathogens causing diseases in cereal crops. It is not intended as an authoritative text on diseases, nor does it provide any new information on diseases or diagnostic techniques. Its primary audience includes breeders, agronomists, and young scientists-in-training, especially in developing nations, who are responsible for identifying and recording trial data, screening segregating populations for disease resistance, and verifying the disease reactions of various types of plant material in their programs.

At many of the research stations in developing countries, the responsibility for recording disease reactions and making selections belongs to breeders, production agronomists, and their assistants. Most of them are familiar with conspicuous diseases, such as the rusts, smuts and mildews, and feel confident that the data they report are reliable. Their confidence tends to fade, however, when faced with the need to identify foot rots, head blights and leaf blights. This guidebook is intended to help cereal crops researchers improve their knowledge and increase their confidence in dealing with the plant diseases they encounter.

The diseases affecting bread wheat, durum wheat, barley, oats and triticale provide the main focus of this guidebook. Triticale is now emerging as a commercial food and feed crop in some countries and production appears destined to increase. Because it is a relatively new crop, published information on the reaction of triticale to specific diseases is meager at this time. Important observations by CIMMYT personnel and national program cooperators regarding diseases associated with triticale are included here.

The scope of this guidebook is limited to the use of simple techniques helpful in identifying some of the most common causal organisms of cereal crop diseases and associated saprophytes. It is recognized that many pathogens, such as those causing foot rots and viral diseases, require more elaborate techniques for accurate diagnosis. There are many excellent books available to those who wish to delve deeper into the study of plant diseases. As a start, the *Compendium of Wheat Diseases* (1977) and the *Compendium of Barley Diseases* (1982), published by the American Phytopathological Society are especially recommended.

In an effort to minimize the confusion that currently exists in the taxonomy of fungi, bacteria and viruses, the most commonly used generic and species names are used in this publication. The perfect or sexual stage is footnoted and discussed in those instances where it serves as an important stage in the initiation, recognition or spread of the disease.

Considerable variability exists in nature among microorganisms, just as among higher plants. In the past, there has been a tendency among mycologists to give species status to minor morphological variants or forms that are pathogenic on different host species. Greater awareness among scientists of the natural variation that exists among all life forms has led to the establishment of fewer species groups. This trend is appreciated by many biological scientists outside the realm of taxonomy.

Figure 1. This guidebook is particularly useful to nonpathologists charged with the responsibility of identifying less than conspicuous diseases (photo: C. Dowswell).

Field and laboratory techniques and equipment

Many cereal crop researchers whose work brings them in contact with crop diseases are not familiar with routine laboratory techniques used by phytopathologists and mycologists. Thus, only simple field and laboratory techniques and equipment, those well within the means and capabilities of researchers and technicians, are suggested here. Where these methods fail to provide reliable identification, it is recommended that one seek assistance from qualified pathologists.

Collecting and preserving samples

The collection of diseased samples becomes necessary when identification of the causal organism requires observation or verification in the laboratory. These same samples can then be preserved as references for future observation, or for comparative purposes, especially as symptoms change with the progressive development of the disease or maturation of the host.

Disease symptoms are more typical and the causal organism more easily identified if sampled when the disease is actively developing. As disease progresses, necrosis increases, and other organisms (both parasites and saprophytes) invade weakened or damaged plant tissues. These changes increase the complexity of identifying the major (primary) pathogen.

Collected samples should be dried and pressed flat as quickly as possible. Two systems of preserving the collected samples are suggested here, and the appropriate choice will depend upon what form of reference is desired, as well as the amount of information the researcher decides to collect.

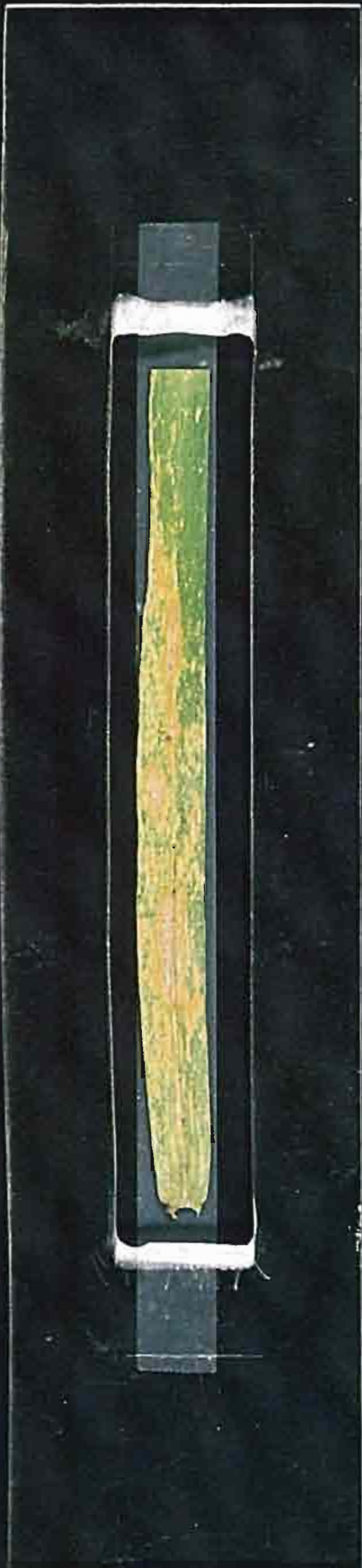
Transparent cellophane tape: Thin plant material, such as leaves, stem sections, seeds, and spikelets can be pressed between two layers of cellophane tape. If maintained in a file away from sunlight, the color and shape of the sample will remain relatively stable for 10 years or more. The collected sample should have dry surfaces and be pressed flat so that the disease symptoms are readily apparent.

A useful and easily made tool that will facilitate sandwiching of collected samples in cellophane tape is shown in Figure 2. This is a clear plastic frame approximately 10 cm wide and 30 cm long. In the center, a rectangular section 20 cm long and 3 cm wide is cut out. The plastic frame holds the sample flat while tape is applied, first to one side, then the other. Important information, such as the identity of the crop, sample, nursery and the date, can be recorded directly on the tape at the time of collection.

Permanent data sheets (see Figure 3) should be used to record field observations. Information recorded on the tape of each sample should also be recorded on the corresponding data sheet. This will help avoid confusion, especially if several samples are taken from the same crop and nursery. Field observations should be as complete as possible in anticipation that other diseases may occur on the same crop in the same field. Laboratory observations may be made later to verify the identity of disease causing organisms.

Glassine envelopes: Diseased plant material from all parts of cereal crops can be preserved in envelopes made of glassine paper. These envelopes allow plant material to dry quickly, and are not damaged by moisture. A very useful type is one originally designed for hand pollination of maize plants (see Figure 4). All relevant field observations can be recorded on the envelope, either in pencil or with a marking pen. This method has a further advantage in that several samples of similar material can be placed in the same envelope. Leaf material stored in envelopes must be pressed and dried thoroughly or it will tend to twist, curl and become brittle.

Figure 2. The plastic frame shown here helps to hold thin plant material in place while cellophane tape is applied, first to one side, then the other (field photos: C. Dowswell).



PLANT DISEASE COLLECTIONS

Sample number: B 81-48

Crop and variety or plot no.: TRITICALE, MERINO S-1481

Date collected: 19 SEPT. 1981

Nursery or location: El BATAN, MEXICO

Sample

Field observations when collected:

- LARGE GRAYISH TO STRAW COLORED BLOTCHES, OVAL TO ELONGATE IN SHAPE, ON LOWER LEAVES
- NO BLOTCHING OR LESIONS ON UPPER LEAVES
- NO APPARENT STRESS TO PLANTS. NORMAL RIPENING, SPIKES CLEAN, GRAIN PLUMP

Laboratory observations:

Under low magnification, dry: CENTERS OF DISEASE LESIONS COVERED WITH DARK SUPERFICIAL FRUITING STRUCTURES

Under low magnification, in petri dish: BLACK SHINY CONIDIA AND DARK BROWN CONIDIOPHORES DEVELOPING ACROSS WHOLE AREA OF LESION

Under high magnification on slides: CONIDIA OLIVE BROWN, 5-7 CONSPICUOUS SEPTA, 60-100 µm x 15-22 µm, THICK WALLS, CYLINDRICAL WITH ROUNDED ENDS

Disease identity:

SPOT BLOTCH
(*H. SATIVUM*)

References: "GRAMINICOLOUS SPECIES OF HELMINTHOSPORIUM" JOUR. OF AGRIC. RESEARCH, 24: pp. 640-740 C. DRESCHSLER, 1923

Remarks:

NO FOOT ROT OR DISEASED NODES

Figure 3. When samples are collected in the field, permanent data sheets such as the one shown here (facing page) should be used to record field observations. Laboratory observations can then be made to verify the disease-causing organism.

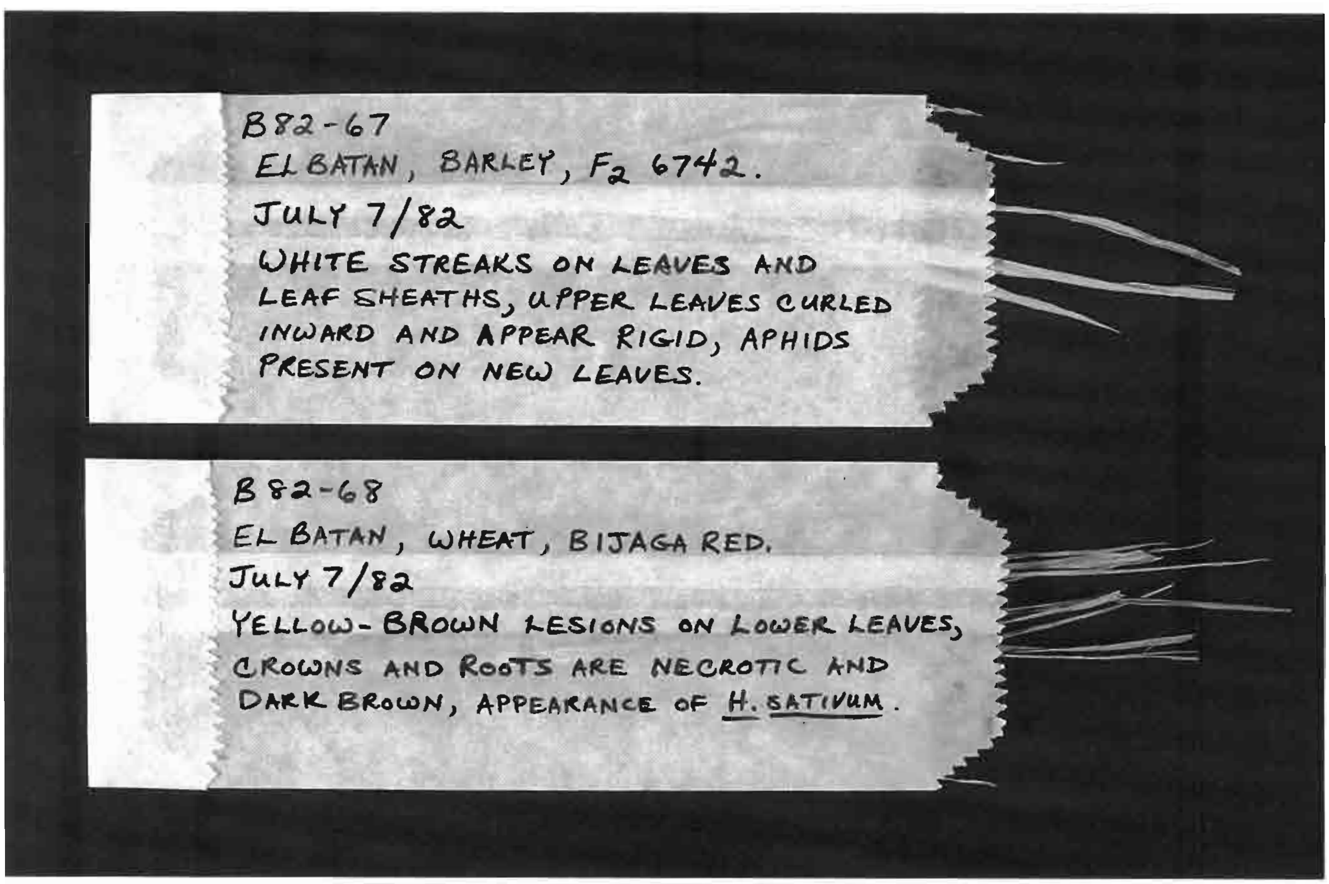


Figure 4. Envelopes made from glassine paper can be used effectively to preserve diseased samples. They allow rapid drying of samples (leaf material should be pressed flat) and are not damaged by moisture.

Preparing samples for the laboratory

Microscopes that can provide several levels of magnification constitute the most important equipment needed for making laboratory observations. The best combination of microscopes is a stereo dissecting scope, with an upper magnification of not more than 50X, and a compound microscope, with magnification ranging from 40X to 400X. The researcher will need petri dishes (the plastic type are satisfactory), filter paper, dissecting needles, scissors, a scalpel, tweezers, a water dropping bottle and glass slides with cover slips (Figure 5).

Techniques: Once again, the emphasis here is on simplicity and ease of execution. Such techniques as staining, sterilizing, isolation on nutrient media, and the reproduction of disease on healthy plants are very useful, but beyond the scope of this guidebook. However, for researchers requiring more sophisticated techniques, the *Herbarium Handbook* prepared by the Commonwealth Mycological Institute, Kew, England (1960), is recommended.

Clean samples possessing disease symptoms are cut to fit within the petri dishes. The identity of the sample is recorded on the dry filter paper previously placed in the bottom of each dish (Figure 6a). Just enough water is added to moisten the paper; there should be no excess or free water in the dish (Figure 6b). Plastic bags that will hold several petri dishes can be used as moisture chambers to prevent drying during incubation (Figure 6c). Samples should be incubated for 24 to 48 hours at room temperature. Samples from the lower portions of the plant (near or in the soil) should be washed thoroughly before being placed in moisture chambers. Surface sterilization with calcium hypochlorite or common chlorine bleach (at 1 percent solution for 1 to 5 minutes), followed by rinsing, may also help eliminate surface contaminants.

Figure 5. Equipment needed for making laboratory observations includes two kinds of microscopes (dissecting and compound types), petri dishes, filter paper, tweezers, dissecting needles and scalpel, a water dropping bottle, and glass slides with cover slips.

Fungi that are pathogenic on cereal crops will generally sporulate within 48 hours on fresh or dried samples (Figure 6d). The main exceptions to this are fungi that develop sexual fruiting structures, and thus require more time to induce maturation of sexual spores. One to two weeks under semi-dry conditions, or alternate periods of drying and moistening, is all that is required for some ascomycetes. Others require extended periods of outdoor weathering.

The incubated material should be examined within 48 hours; otherwise, saprophytic contaminants tend to obscure the main pathogens. The fruiting structures can be observed and, with practice, identified under the dissecting scope at low magnification (5X to 50X). For observation at higher magnifications, spores or fruiting structures can be picked out or scraped from the surface of the plant material and placed in a drop of water on a slide. A cover slip should be applied gently to avoid air bubbles, and the slide examined under higher magnification. Using an eyepiece with a calibrated scale will help to establish the size and identify other features of the spores being examined. However, with experience the relative size of spores can be established without using a scale, and disease causing organisms can be identified at lower magnification.

PLEASE NOTE: Most of the photographs of spore forms included in this guidebook were taken at an original magnification of 10 x 40 (400 times the actual size). These photographs were then further enlarged 250 percent for printing purposes. Thus, the final magnification of a 10 x 40 photomicrograph equals 10 x 40 x 2.5 (1000 times the actual size). In the case of close-up photographs of conidiophores, etc., which were generally taken at 50 x, the final magnification equals 50 x 2.5 (125 times the actual size). The original magnification of each photograph taken with the aid of a microscope is provided in each respective caption. Simply multiply that magnification by 2.5 to arrive at the final magnification.

Figure 6(a). Diseased leaf material is first cut to fit in a previously prepared petri dish...

... **(b)** the filter paper is then moistened and excess moisture is poured off ...

...**(c)** the samples are incubated for 24 to 48 hours at room temperature in a plastic bag that acts as a moisture chamber...

...**(d)** and usually within 48 hours, pathogenic fungi will sporulate. Shown here is sporulating *Helminthosporium sativum*.

Disease groups and their general symptoms

The diseases included in this guidebook are grouped principally on taxonomic or morphological similarities of the pathogens. Diseases caused by fungi not belonging to one of these groups are arbitrarily placed in the category of miscellaneous diseases and separated by their propensity to affect either the lower or upper portions of host plants. Some common saprophytic and weakly pathogenic fungi are also included here, not because of their importance in causing disease, but to help the researcher distinguish them from primary pathogens. Some common noninfectious problems are described briefly in the sections dealing with injuries from nematodes and other pests, and physiological, nutritional and environmental stresses.

Disease Group	Symptoms	Page
The rusts	Conspicuous raised pustules (uredia), yellow-orange to dark red, occur on leaf blades and sheaths, stems or glumes. These pustules release loose, colored spores. Black, covered or exposed teliospores may be produced as plants approach maturity.	10-19
Helminthosporium diseases	Foot rots, leaf spots, leaf blotches, seedling blight, leaf stripe and head blight are the main diseases caused by this group of fungi. The *Helminthosporium* species are identified by the type of fruiting structures produced and the morphological features of the spores. The conidiophores and conidia develop in colonies on the surface of the host tissue from dark embedded and superficial mycelium. The conidia are smooth, septate, subhyaline to dark, mostly cylindrical, often with a dark, colored scar on the basal cell.	20-34
Septoria complex and septoria-like diseases	Leaf blotches develop tiny dark spots (pycnidia) within irregularly shaped dry lesions. When moist, pycnidia exude masses of colorless, threadlike, septate conidia.	35-47
Smuts and bunts	Florets and/or grain are replaced by black spore masses. These spore masses may be loose, enclosed in greyish white membranes, or encased in dark spore balls similar in shape to the grain replaced. Others form black spore masses in the leaf blades and sheaths, and occasionally stems.	48-58
Fusarium diseases	Scab, foot rot, and snow mold are the main diseases caused by *Fusarium* species. The fungi are most readily recognized by the colored spore accumulations and aerial mycelium, which varies from white, pink, peach to red in color on the affected areas. *Fusarium nivale* may also cause a grey-brown leaf blotch.	59-69
Miscellaneous root and crown diseases	Discolored or rotted roots and crowns (foot rots), dark mycelial development and/or eye spots on basal internodes, white heads or sterile spikes, as well as lodging, are often associated with root rots. Symptoms of other diseases in this group include broad stripes on basal leaves, thickened leaves, dwarfed plants and distorted heads.	70-81

Disease Group	Symptoms	Page
Miscellaneous leaf and head diseases	The fungal pathogens included in this group have little in common except their tendency to invade the upper parts of the plant. Each fungus produces rather distinct disease symptoms, and they usually develop their fruiting structures on the surface from submerged mycelia. *Claviceps purpurea*, the cause of ergot, differs in its development of fruiting structures; large dark sclerotia replace the kernels in infected florets.	82-89
Saprophytic and weakly pathogenic fungi	Several common saprophytes and facultative parasites are included in this group. Normally these fungi are aggressive spore producers, producing dark, thick-walled spores on dead or dying tissue. The affected plant parts take on a dull greyish appearance. Under conditions favorable to the fungi they may invade living plant tissues or developing grain, usually during the maturation stage.	90-99
Bacterial diseases	Water-soaked streaks, spots, and blotches appear on leaves, stems or glumes. When leaves are moist with dew, droplets of bacterial exudate may develop on lesions and newly infected areas. When dry, the bacterial exudate may form crystals or a thin shiny film that flakes when scraped. Streaks may appear dark by reflected light, but often are translucent by transmitted light.	100-107
Viral and mycoplasmal diseases	Viruses produce no visible fruiting structures. They develop a broad range of symptoms on cereal crop plants, including stunting, spotting, striping, streaking, yellowing or general chlorosis. Many viral diseases are transmitted by insects or small animals (such as aphids and nematodes); others are transmitted mechanically or by the fungus *Polymyxa graminis*. The identity of the vector is important and often essential to the identification of the disease. There may be a tendency for the onset of necrosis to be delayed longer in viral diseases than in fungal diseases.	108-121
Injury from nematodes and other pests	Injury can occur to below- or above-ground portions of the plant. Damage to roots by cutting, chewing or boring indicates worms, larvae or other insects. Abnormal branching and deformation of the roots in the form of knots, swellings or galls indicates a nematode infestation. Above ground, mechanical damage such as chewing, clipping, boring or stripping of leaves, stems or heads indicates insects or mammals. Nematodes may form seed galls, replacing grain in the heads. Other nematodes may infest the nodes, causing swelling and deformation.	122-129
Physiological, nutritional and environmental stresses	Abnormal plant development caused by mechanical damage, chemical injury, environmental and physiological stresses, or genetic abnormalities; not caused by pathogens.	130-138

The rusts

Puccinia species

The rusts are perhaps the most destructive and also the most widely recognized of cereal crop diseases. They occur in almost all areas of the world where cereal crops are grown. Any of the above-ground portions of the plant may become infected, from seedling stage to maturity. The fungi, *Puccinia* species, are mostly obligate parasites with highly complex life cycles. Three spore forms develop on small grains and grasses, and two additional spore forms are produced on the alternate host (where it exists).

Of these spore forms, urediospores (summer spores) are most important, because they enable repeated cycles of the disease to spread from field to field and survive from year to year. These spores vary in color from yellow-orange to reddish brown. They are produced in pustules (uredia), which erupt through the epidermis exposing the powdery spore masses (Figure 7).

Teliospores (winter spores) develop as the crop approaches maturity, and they usually remain covered by the epidermis of the host plant. Teliospores differ from the urediospores in color, form and function. They are dark brown, two-celled, have thick walls, and function as the first stage in the sexual cycle of the fungus. After weathering, or overwintering, the teliospores germinate and produce basidiospores that are able to infect the alternate host (but not cereals or grasses). The sexual cycle on alternate host plants creates diversity through genetic recombination, and establishes primary inoculum for the new crop cycle. The fungi, however, usually persist from cycle to cycle as urediospores, or as mycelium established in the host tissue.

Figure 7. Uredia of stem rust (*Puccinia graminis* f.sp. *tritici*) on wheat (facing page). Note the loose epidermal tissue along the margins of the sori.

At least five *Puccinia* species attack cereal crops; forms within species vary in their capacity to parasitize crops and grasses. The different forms are given form species (f.sp.) names. For example, stem rust is caused by *Puccinia graminis*. The form species that infects oats but not wheat or barley is identified as *P. graminis* f. sp. *avenae.* Within forms there are physiologic races that are distinguished by their virulence on specific host genotypes. The occurrence of new physiologic races of rust fungi creates difficulties in developing and maintaining resistant plant varieties.

Rust diseases are most effectively controlled by the use of resistant varieties and by the eradication of the alternate host. Fungicides applied as foliar sprays can be used effectively to protect crops threatened by pathogenic races, but are usually uneconomical as control systems.

Distinguishing the species

Urediospores of *P. graminis* f.sp. *tritici* (10 x 40)

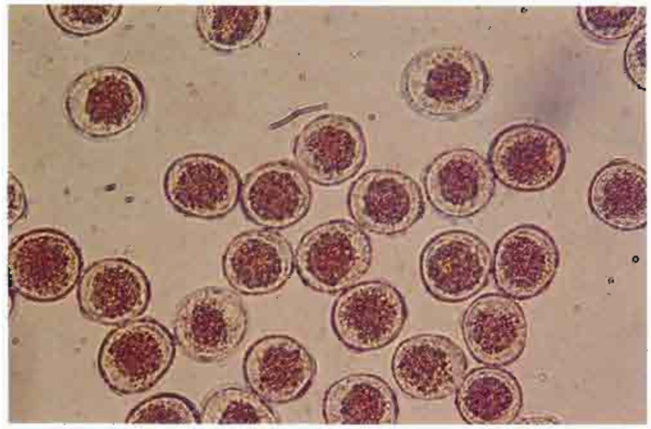
The rust species described here usually can be distinguished by the characteristics of the uredia, color of the urediospores, and the host species they infect.

Puccinia graminis (stem rust)

Large elongate spore masses are reddish brown with loose epidermal tissue conspicuous at the margins of the pustules. Uredia may develop on both the upper and lower surfaces of leaves, and on heads and stems. Urediospores are dark red, elliptical, echinulate, and measure 24-32 μm x 18-22 μm.

Puccinia recondita (leaf rust of wheat)

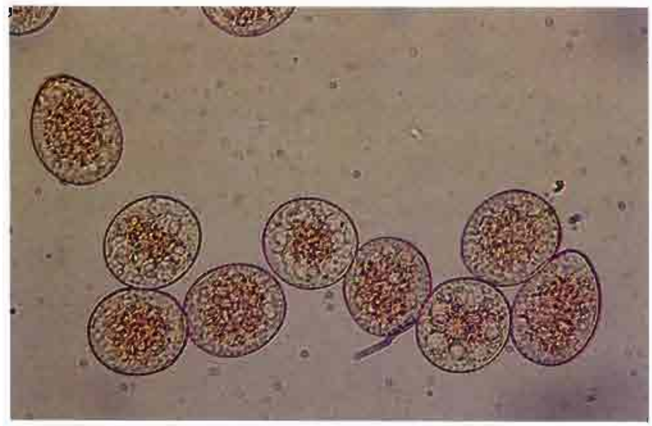
Uredia are red, oval shaped, and scattered, without conspicuous loose epidermal tissue at the margins. They are usually produced on the upper side of leaf blades. Urediospores are orange-red to dark red, spherical, and measure 20-28 μm in diameter.



Urediospores of *P. recondita* (10 x 40)

Puccinia hordei (leaf rust of barley)

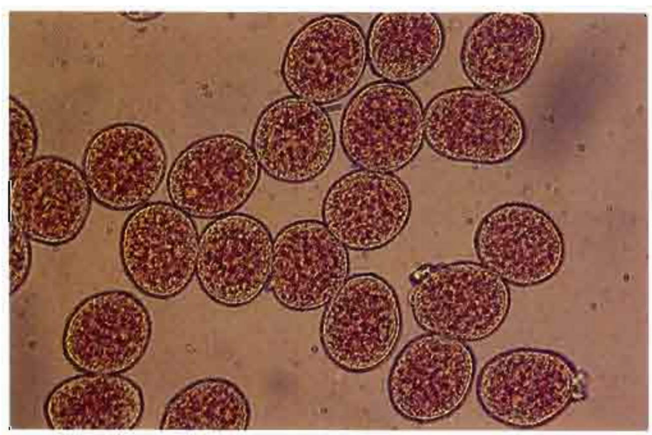
Uredia are yellow-orange, oval shaped, scattered, and occur generally on the upper side of the leaf blades. Urediospores are yellow-orange, relatively large, egg shaped to spherical, and measure 28-36 μm x 24-28 μm. This fungus is pathogenic only on barley and its close relatives.



Urediospores of *P. hordei* (10 x 40)

Puccinia striiformis (stripe rust)

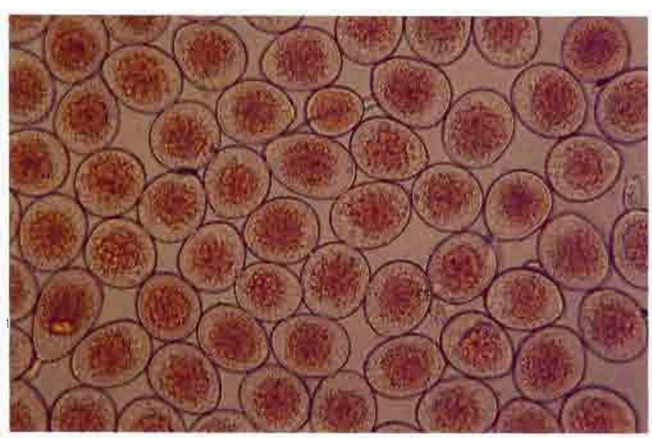
Uredia occur in conspicuous narrow yellow stripes on leaf blades and sheaths. Urediospores are yellow, more or less spherical, and large, measuring 28-34 μm in diameter. Glumes and lemmas often become infected. Narrow black stripes are formed on leaves during telial development.



Urediospores of *P. striiformis* (10 x 40)

Puccinia coronata (crown rust of oats)

This fungus is pathogenic on oats, but not on other cereal crops. Uredia are oval to elongate blisters with bright yellow-orange urediospores. The urediospores are spherical to egg shaped, and 20-32 μm in diameter. The teliospores are broad and have projections on the upper cells.



Urediospores of *P. coronata* (10 x 40)

Stem rust

Puccinia graminis

Stem rust is a well recognized disease of cereal crops, occuring almost everywhere these crops are grown. *Puccinia graminis tritici* infects wheat, barley and triticale. *Puccinia graminis avenae* infects oats, and *P. graminis secalis* infects rye. Disease symptoms most commonly appear on the stems and leaf sheaths, but leaf blades and spikes may also become infected (Figure 8). Three spore forms are produced on the cereal hosts. Urediospores develop in pustules (uredia) that rupture the epidermis and expose masses of reddish brown spores (Figure 9). The pustules are larger than those of leaf and stripe rust, oval shaped or elongated, with loose or torn epidermal tissue along the margins. They may appear on both the upper and lower surfaces of the leaf. The urediospores of *P. graminis* are reddish brown, elliptical to egg shaped, echinulate structures measuring 24-32 µm x 18-22 µm (Figure 10). They continue to be produced until the plants approach maturity.

Figure 8. Heavy infection of stem rust on triticale. Note that leaf blades and sheaths are also affected.

Figure 9. Uredia of *P. graminis*. Note the loose epidermal tissue.

Figure 10. Urediospores of *P. graminis* are oval to egg shaped (10 x 40).

As the plants approach maturity, urediospore production ceases and teliospores develop, either in the same uredia or in different fruiting structures called telia. The teliospores are dark brown, two celled, and somewhat wedge shaped. They retain a portion of the pedicel or stalk, have thick walls, and measure 40-60 μm x 18-26 μm. The apical cell is rounded or slightly pointed (Figure 11). The teliospores frequently erupt through the epidermis, persist in the plant tissue during the winter, and may serve as the first stage in the sexual cycle by producing basidiospores. Common barberry, *Berberis vulgaris,* and *Mahonia* species are alternate hosts for *P. graminis* (Figure 12).

The urediospores of stem rust are carried over long distances by the wind. During the active growth period of host crops, repeated cycles of uredia are produced every 14 to 21 days, which accounts for the rapid spread of the disease over wide areas. This disease, along with leaf rust, has received much attention from pathologists, plant breeders, and other scientists. In the past, the creation of new physiologic races via the sexual cycle on the alternate host, barberry, resulted in serious crop losses and reduced the effectiveness of resistant varieties. In North America, the eradication of barberry, and deliberate breeding of resistant varieties, have been important factors in improving the stability of resistance to stem rust in varieties and reducing the incidence of epiphytotics.

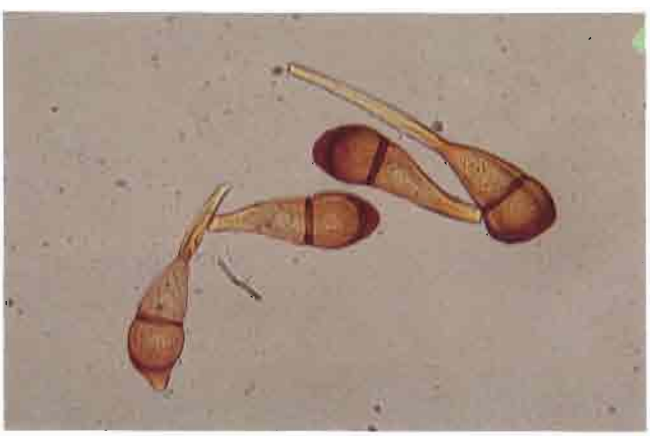


Figure 11. Teliospores of *P. graminis* (10 x 40).

Figure 12. Aecia developing on an alternate host of *P. graminis*, common barberry (*Berberis vulgaris*).

Leaf rust of wheat

Puccinia recondita

Leaf rust is a serious disease of wheat, rye, triticale and many grasses. The pathogen is mildly parasitic on barley, but does not infect oats. Another closely related species, *P. hordei*, is a common pathogen of barley (page 16).

Leaf rust appears as small, oval-shaped, dark red pustules scattered on leaf sheaths and the upper surfaces of leaf blades. The pustules (uredia) break through the epidermis, but do not cause loose epidermal tissue at the margins as is characteristic of stem rust uredia (Figure 13). The urediospores of *P. recondita* are orange red to dark red, echinulate, spherical, and usually measure 20-28 μm in diameter (Figure 14). The teliospores are dark brown, two celled with thick walls, and are rounded or flattened at the apex (Figure 15). The telia develop during the later stages of plant development in sori on leaf sheaths and on both surfaces of the leaf blades. The teliospores remain in the leaf tissues and are covered by the epidermis.

Thalictrum and *Anchusa* species, the alternate hosts of *P. recondita*, occur in some areas of Europe, but are unimportant as a source of new races of this fungus. In temperate climates, the pathogen survives from year to year as mycelium or urediospores on winter cereals and grasses. In the tropics, it oversummers at higher elevations and in cooler areas.

Leaf rust is best controlled by the use of resistant cereal crop varieties or multilines. Fungicides, such as *Triadimefon* (bayleton) and *Butrizol* (indar), are effective in controlling leaf rust and can be used economically in epidemic situations.

Figure 13. Uredia of *P. recondita* on a wheat leaf.

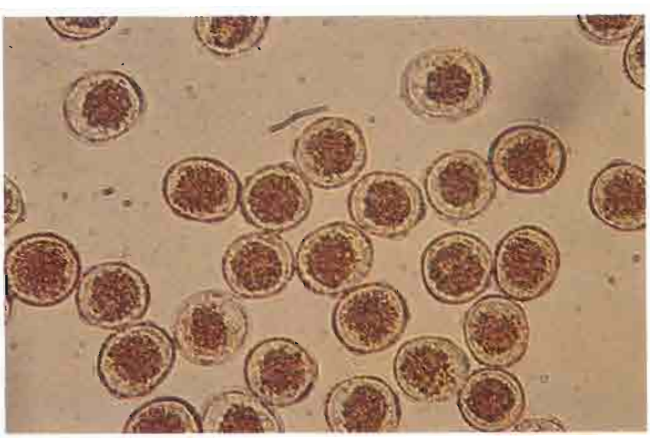


Figure 14. Urediospores of *P. recondita* tend to be more spherical than those of *P. graminis* (10 x 40).

Figure 15. Teliospores of *P. recondita* (10 x 40).

Leaf rust of barley

Puccinia hordei

Leaf rust occurs extensively in many barley growing areas of the world, but the crop losses it causes are usually light. The pathogenicity of this fungus is restricted to cultivated barley and its close relatives. Barley is also affected by some races of *P. recondita,* but to a lesser extent.

The uredia of *P. hordei* are small, oval to round, and yellow-orange in color. They develop almost exclusively on the leaf blades and sheaths (Figure 16). The urediospores are egg shaped to spherical, yellow, and relatively large, measuring 28-36 μm x 24-28 μm (Figure 17). As the crop matures, dark brown telia form in the leaf tissues and initially remain covered by the epidermis. The alternate host, *Ornithogalum umbellatum* (the star of Bethlehem), can be a source of aeciospores in some areas of Europe, but does not appear to become infected in North America; it is unlikely that it plays an important role in the perpetuation or dissemination of new, diverse forms of the pathogen.

The development and spread of leaf rust of barley is favored by warm humid weather. The uredial stage overwinters on fall-sown barley in areas having mild winters. In subtropical areas the fungus survives summer temperatures in the cooler highlands. The use of resistant varieties is the most economical means of control.

Figure 16. Uredia of *P. hordei* on a barley leaf.

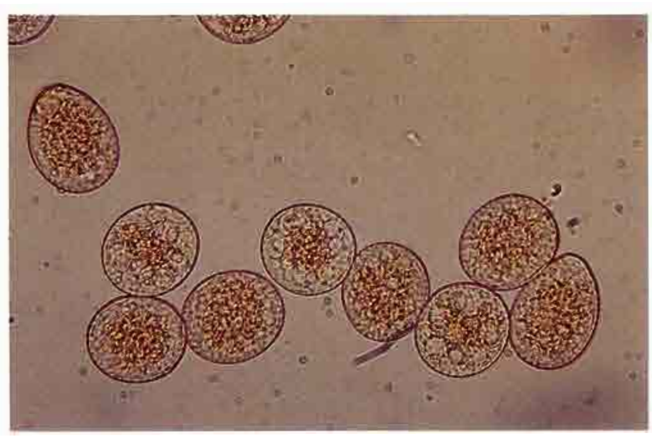


Figure 17. Urediospores of *P. hordei* vary from spherical to egg shaped (10 x 40).

Stripe rust

Puccinia striiformis

Stripe rust is a serious disease of wheat and barley, and moderately affects triticale, rye and some grasses. Oats appear to be immune. No alternate host is known, and the disease is confined by environmental factors to a greater extent than either stem rust or leaf rust. Disease development occurs at lower temperatures than are optimal for leaf and stem rust.

Uredia develop in narrow, yellow, linear stripes mainly on leaves and spikelets (Figure 18). When the heads are infected, the pustules appear on the inner surfaces of glumes and lemmas, occasionally invading the developing kernels (Figure 19). The urediospores are yellow to orange in color, more or less spherical, echinulate, and 28-34 μm in diameter (Figure 20). The teliospores apparently serve no function. They are dark brown, two celled, and similar in size and shape to those of *P. recondita*. The telia develop on leaf blades and sheaths as dark brown to black stripes and remain covered by the epidermis.

The urediospores and mycelium persist through the winter on fall-sown cereals and grasses in areas where temperatures are not extreme. Aggressive secondary spread occurs during the cool temperatures of spring. New, highly virulent races of stripe rust have recently appeared in Europe, Australia, and the Andean region of South America.

Figure 18. Uredia of *P. striiformis* on a wheat leaf. Note that they form narrow linear stripes.

Figure 19. Uredia of *P. striiformis* on wheat florets (glumes and lemmas).

Figure 20. Urediospores of *P. striiformis* (10 x 40).

Crown rust of oats

Puccinia coronata

Crown rust is a significant disease of oats and a few grasses, but does not affect the other cereal crops. The disease is prevalent wherever oats are grown. The diversity in physiologic races is very great, due at least in part to the wide distribution of the alternate host, *Rhamnus cathartica* (buckthorn), and related species.

The uredial pustules develop mainly on the leaf blades, but may also appear on leaf sheaths and spikelets. They occur as small, scattered, oval-shaped blisters with exposed urediospores that are bright orange in color (Figure 21). The epidermis is slightly lifted along the margins of the pustules. The urediospores are spherical to oval in shape, echinulate, orange-yellow in color, and measure 20-32 µm in diameter (Figure 22). Telia develop on affected leaves as the plants approach maturity. The telial sori may develop as conspicuous black borders around the uredia, but they remain covered by the epidermis. The teliospores are distinctive in shape from those of other species. The apical cells of the teliospores are broad and tipped with several projections, from which the name "crown" rust was derived (Figure 23).

The fungus persists from season to season in the warmer climates by continuous production of urediospores. In areas with lower temperatures, the disease is initiated by spores carried by wind currents from warmer areas, or from aeciospores developing on buckthorn (*Rhamnus* species).

In many oat producing areas, such as Africa, South America and Mexico, breeding for resistance has not kept pace with the occurrence of new physiologic races of the pathogen. Oat production in these areas has declined because of losses from crown rust and stem rust, as well as from barley yellow dwarf virus.

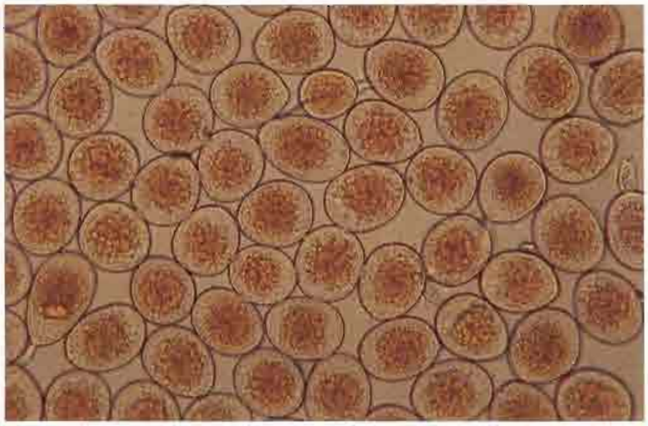


Figure 22. Urediospores of *P. coronata* (10 x 40).

Figure 21. Uredia of *P. coronata* on an oat leaf.

Figure 23. Teliospores of *P. coronata* have short projections on upper cells, which gave rise to the name "crown rust" (10 x 40).

Hyperparasite of rust

Darluca filum

Darluca filum is an obligate parasite of the rust fungi, occurring most commonly in warmer climates. The fungus develops dark fruiting bodies (pycnidia) inside the rust pustules. Its role in controlling disease spread is probably insignificant, but the fungus is included here because of the frequency with which it is observed on cereal crops affected by rust diseases.

The fruiting structures appear in clusters and consist of several black spherical pycnidia within the rust sori (Figure 24). Conidia are released through the ostiole of the pycnidium as a whitish or light grey mucous-like mass (Figure 25). The conidia are colorless, two celled, ellipsoidal to oblong in shape, and measure 14-16 μm x 3-4 μm with a few short bristles projecting from each end (Figure 26). *Darluca filum* progresses too slowly during the rust cycle to serve as an effective biological control of the rusts.

Figure 24. Black pycnidia of *D. filum* forming in the pustules of *P. recondita* (leaf rust of wheat).

Figure 25. Whitish to light grey cirrhi (containing conidia) are exuded from the pycnidia of *D. filum*.

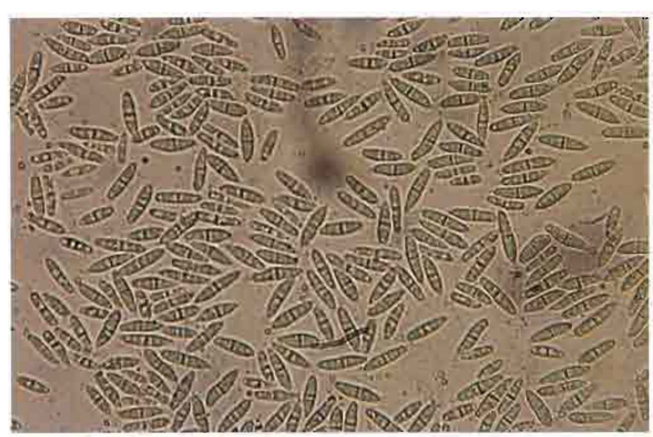


Figure 26. Conidia of *D. filum* (10 x 40).

Helminthosporium diseases

Five *Helminthosporium* species are injurious pathogens of cereal crops and are widely distributed around the world. Two less common species that have unique characteristics are also included here. The *Helminthosporium* species are treated as a group in this guidebook, not because of similarities in the disease symptoms produced, but because of similarities (1) in the types of fruiting structures developed during sporulation, and (2) in the general shape of the conidia. As a group, they are probably second in importance to the rusts as destructive pathogens of cereal crops the world over.

Various types of foliar lesions, such as blotches, stripes, spots and discolorations are caused by most of the species (Figure 27). Some species also cause seedling blights, foot rots, node and stem decay, and head blights.

Figure 27. Early leaf-spotting symptoms (facing page) caused by *Helminthosporium tritici-repentis* (yellow leaf blotch or tan spot).

The asexual fruiting structures develop on the surface of plant tissues from mycelia within the tissue. The spore-bearing stalks (conidiophores) are as long as, or longer than, the conidia, develop singly or in clusters, are erect, septate, light brown to dark brown in color, and are mostly unbranched. Each conidiophore produces one to several conidia, which are cylindrical to slightly barrel shaped (ellipsoid), may be straight or slightly curved, vary in color from tinted (subhyaline) to dark brown or black, and have several cells with very conspicuous septa.

Distinguishing the species

Conidia of *H. sativum* (10 x 40)

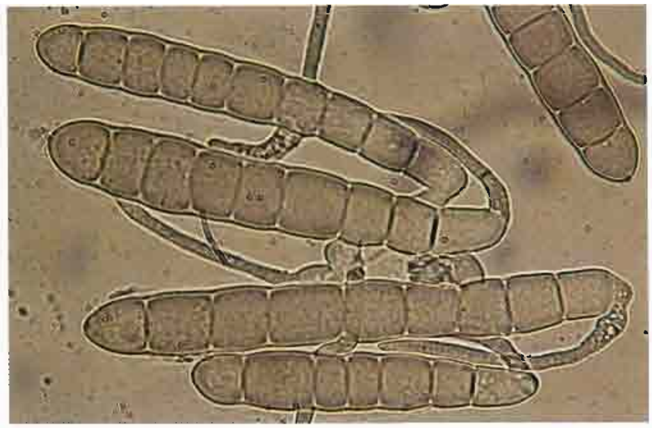
The species within this group can be distinguished mainly by the characteristics of the conidia, the crops they infect and the disease symptoms produced.

Helminthosporium sativum (spot blotch)

Conidia appear black and shiny under low magnification, but under higher magnification they are a dark olive brown. The conidia are thick walled, typically have five to nine cells, are elliptical, may be straight or slightly curved, and measure 60-120 µm x 12-20 µm. This pathogen can affect all small grain cereals, but is less common on oats.

Helminthosporium tritici-repentis (yellow leaf blotch or tan spot)

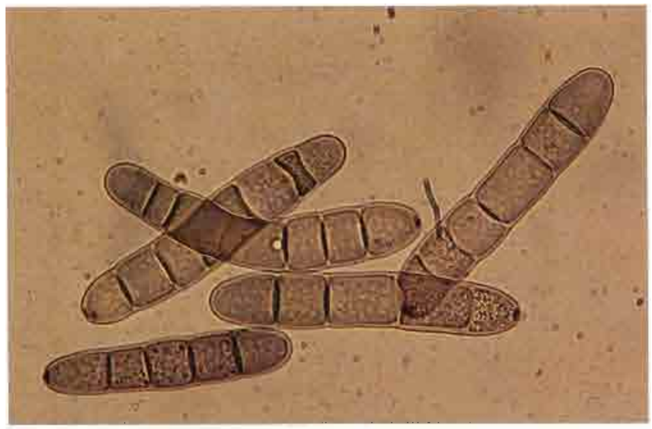
The early leaf spots caused by this disease may have distinct yellow borders around straw to grey-brown, oval-shaped lesions. Young conidia are subhyaline, becoming yellow-grey with age. They are cylindrical, thin walled, taper slightly toward the top, and measure 80-170 μm x 12-24 μm with five to ten septa. The basal end cell is a rounded cone suggestive of a "snake's head." This fungus is pathogenic on wheat and triticale, and less frequently affects rye and barley.



Conidia of *H. tritici-repentis* (10 x 40)

Helminthosporium avenae (leaf blotch of oats)

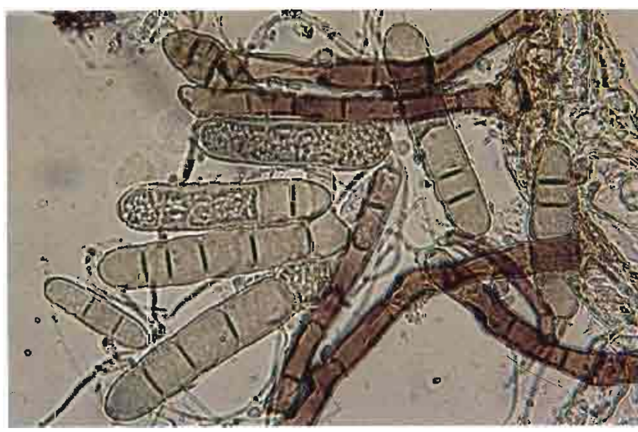
This disease is restricted to oats. The conidia are light greyish brown, cylindrical, straight to slightly curved, four to six septate, and measure 80-120 μm x 12-18 μm. They are very similar in size and shape to the conidia of *H. teres.*



Conidia of *H. avenae* (10 x 40)

Helminthosporium teres (net blotch)

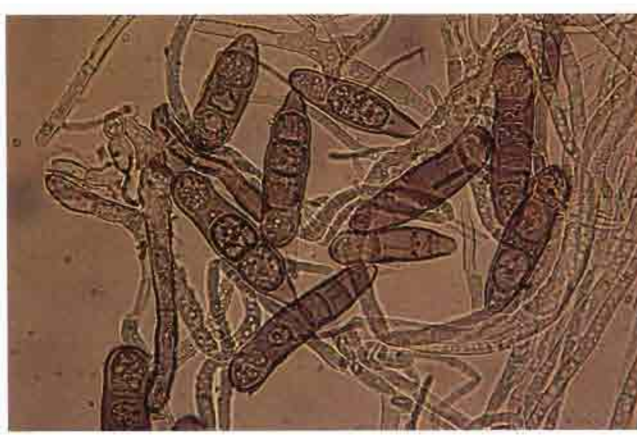
The conidia may be easily confused with those of *H. gramineum*, but the disease symptoms are quite distinct. Net blotch lesions start as small brown spots or short streaks and expand to irregularly shaped blotches with internal brown pigmentation. The conidia are straight, cylindrical, four to six septate and have rounded end cells. They are subhyaline at first, darken to yellow-brown with age, and measure 60-120 μm x 16-24 μm. This disease is most often found on barley, but occasionally affects wheat and triticale.



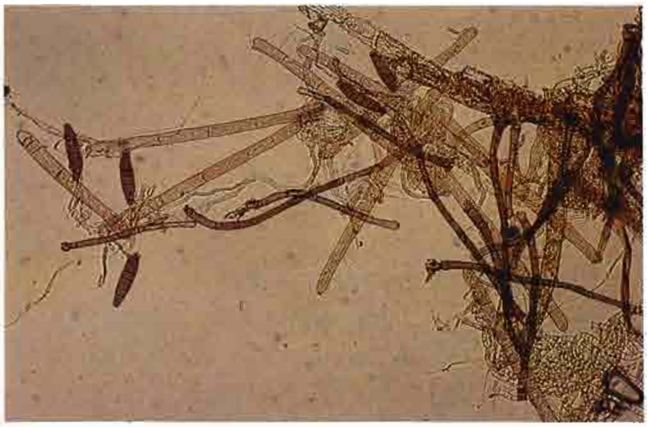
Conidia and dark conidiophores of *H. teres* (10 x 40)

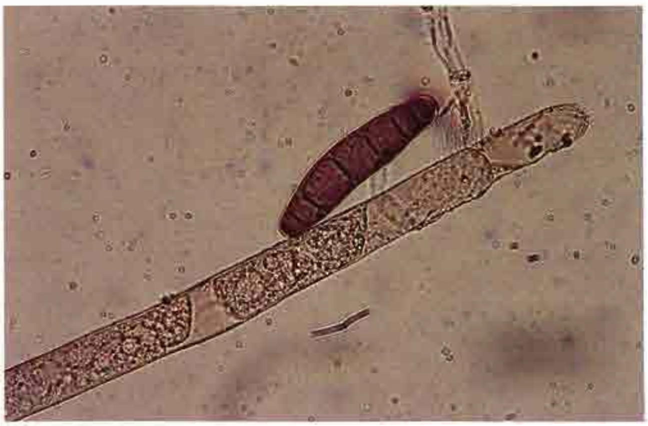
Helminthosporium gramineum (barley stripe)

This disease is systemic and is restricted to barley. As the plant develops, each succeeding leaf shows stripes that are yellow at first and then turn brown or dark grey. The leaves tend to split and fray. The conidia are usually light brown in color and seldom exceed 100 μm in length. Conidiophores develop in clusters of two to six, without the pronounced swollen basal cells that occur in *H. teres.*



Conidia of *H. gramineum* (10 x 40)

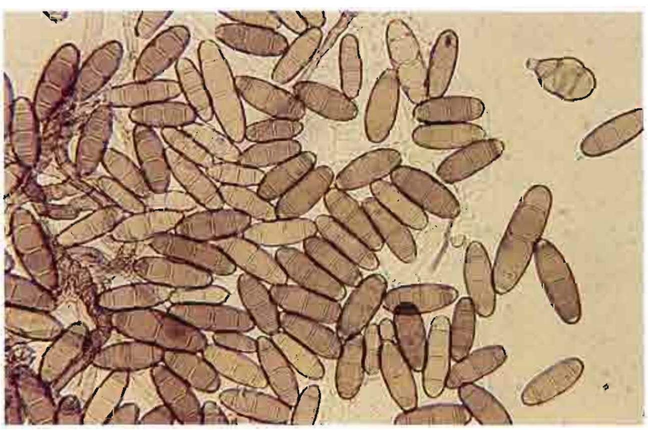




Conidia of *H. giganteum*—the long, cylindrical, light colored structures (10 x 10, top; 10 x 40 bottom)

Helminthosporium giganteum (zonate eye spot)

The lesions of this disease are distinctive, appearing as small yellow spots surrounded by dark brown borders. The conidia are straight, subhyaline, cylindrical, measure 350-400 μm x 15-20 μm and have few septa. The disease can affect all small grain cereals, and is restricted to humid subtropical regions.



Conidia of *H. spiciferum* (10 x 40)

Helminthosporium spiciferum (cereal leaf blotch and foot rot)

The leaf blotches vary in color from light brown to grey-brown, are irregularly shaped, and usually spread across the leaves. The conidia are the smallest among the helminthosporium cereal crop pathogens. They measure 25-40 μm x 8-12 μm, and have three and sometimes four septa. This pathogen attacks wheat, barley and related grasses.

Spot blotch

Helminthosporium sativum *

A voracious pathogen that attacks wheat, triticale, barley and many grasses, *H. sativum* causes spot blotch, root and crown rots, node cankers (black joint) and head and seedling blight (Figure 28). Rye is somewhat more tolerant, while oats are seldom infected. The fungus also is fully capable of surviving as a saprophyte on dead plant tissue.

Infection by *H. sativum* may occur at any stage of plant development, but symptoms are usually more pronounced after heading. Infected seedlings develop dark brown necrotic lesions on roots, crowns and lower leaf sheaths (Figure 29). Lesions on leaf sheaths may extend to the blades. Root and crown infections occurring before or during the flowering period usually kill plants before seeds are formed. This often occurs in association with other root-rotting pathogens, such as *Ophiobolus graminis* and *Fusarium* species. Node cankers appear as brownish black discolorations at the lower nodes. The diseased nodes may be partially or completely invaded, thus restricting the flow of plant nutrients. Diseased nodes take on a greyish black, velvety appearance due to dense sporulation (Figure 30).

Leaf blade and sheath infections (the spot blotch stage) develop as distinct oval to elongated blotches, light brown to dark brown in color, that contrast sharply with the normal green of the leaf (Figures 31, 32). The dead areas of leaf blades and sheaths eventually take on a dull greyish appearance. Under conditions favorable for the development of the disease, spikelets may be affected, causing shriveling of the grain. Black point, which develops as a dark staining of the

* Perfect stage: *Cochliobolus sativus*

Figure 28. Typical leaf-spotting symptoms on barley, caused by *H. sativum* (photo: S. Fuentes).

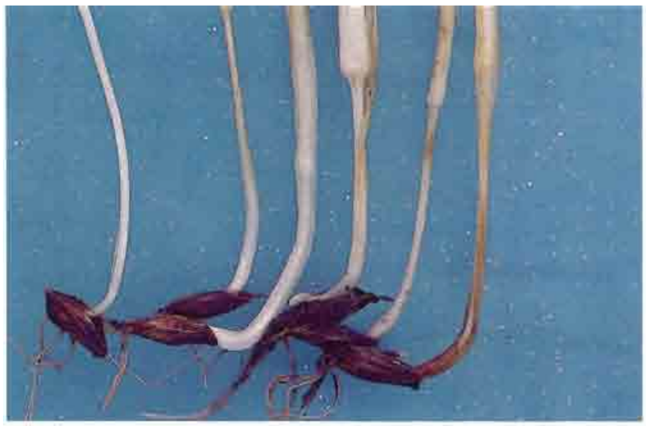


Figure 29. Barley seedlings infected by *H. sativum*. Note the dark brown necrotic lesions.

Figure 30. Sporulation of *H. sativum* on a diseased wheat stem node. Note the black, velvety appearance.

embryo end of the seed, occurs under conditions favorable to the disease (warm temperatures and high humidity). The disease develops faster at temperatures above 20°C (68°F).

The fruiting structures, which develop readily on moistened diseased plant tissue, serve as the most reliable diagnostic feature of this pathogen. The conidiophores develop singly or in small clusters, are erect, unbranched, 100-150 μm x 6-8 μm, and have several septa (Figure 33). Conidia develop laterally from pores just below the conidiophore septa. The conidia are olive brown, oblong, tapered toward the end, slightly curved with smooth walls, and have a prominent basal scar. They measure 60-120 μm x 12-20 μm in size and have three to nine septa (Figure 34). Isolates vary considerably in virulence. The perfect stage rarely occurs in nature.

Helminthosporium sativum is among the most widely distributed of the cereal crop pathogens. The diseases caused by this fungus probably reduce the yield of wheat and barley in subtropical areas more than any other pathogen. Its ability to survive in soil debris and on many grass species reduces the effectiveness of crop rotation as a control measure. Seed treatment can be beneficial in areas where head blight results in the production of infected seed. Breeding for resistance is a slow and elusive process, but appears to offer the best probability of success in controlling this pathogen.

Figure 31. Spot blotch lesions on a durum wheat leaf, caused by *H. sativum*.

Figure 33. Conidiophores of *H. sativum* on wheat (50 x). See also Figure 6d, page 7.

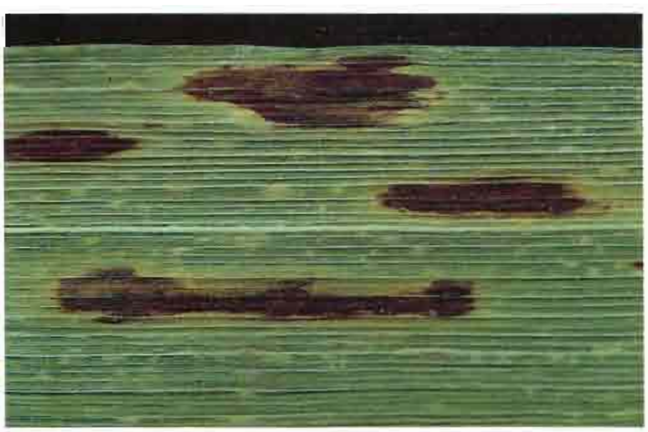


Figure 32. Typical spot blotch lesions caused by *H. sativum* on barley (photo: S. Fuentes). Compare these lesions with those produced on wheat (Figure 31).

Figure 34. Conidia of *H. sativum* are dark, thick walled, and have conspicuous septa. Note that the conidia shown here are slightly larger than most encountered (10 x 40).

Yellow leaf blotch (tan spot)

*Helminthosporium tritici-repentis**

Yellow leaf blotch is chiefly a foliar disease of wheat, triticale and several grasses. Rye and barley are less frequently affected and oats appear to be immune. The first symptoms appear as small yellow-brown spots that develop into oval-shaped, light brown blotches, usually with yellow borders (Figure 35). As the lesions merge, large areas of leaves turn yellow and die (Figure 36). Necrosis often begins near the tip and progresses toward the base of the leaf. Heavy sporulation causes the centers of lesions to darken.

* Perfect stage: *Pyrenophora tritici-repentis*

Sporulation occurs readily in moisture. The conidiophores are dark brown, septate, and measure 100-400 μm x 6-8 μm. The conidia are pale yellow-grey, cylindrical, 80-170 μm x 12-24 μm in size and have four to ten septa. The basal cell tapers to a rounded cone resembling a "snake's head" (Figure 37).

Figure 36. More advanced leaf spotting on wheat, caused by *H. tritici-repentis.*

Figure 35. Typical yellow leaf blotch lesions on wheat, caused by *H. tritici-repentis.*

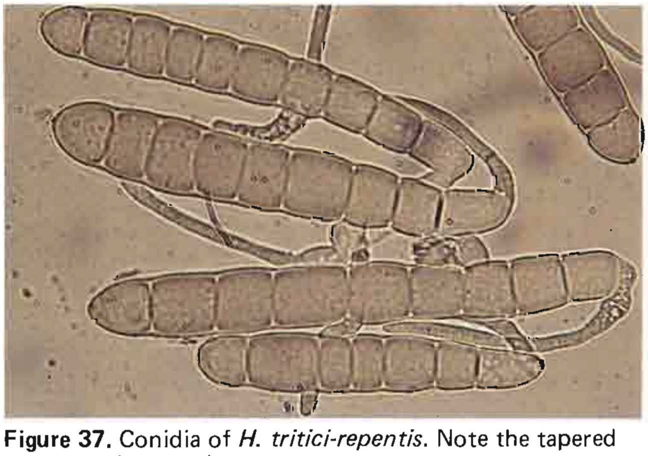


Figure 37. Conidia of *H. tritici-repentis.* Note the tapered basal cells (10 x 40).

The perfect stage, *Pyrenophora tritici-repentis,* develops on weathered wheat stubble and straw. The perithecia are black, erumpent, with dark spines surrounding the short beaks (Figures 38, 39). The mature ascospores are yellow-brown, oval shaped, have three transverse septa and one or two longitudinal septa. They measure 40-60 μm long by 18-25 μm wide (Figure 40). Conidiophores and conidia frequently develop on the perithecia. Ascospores and conidia may both serve as primary inoculum.

The disease caused by *H. tritici-repentis* occurs in most of the wheat-growing regions of the world. The pathogen appears to tolerate cool temperatures more readily than *H. sativum*. Losses from yellow leaf blotch in wheat crops occur regularly in the Andean region of South America, the highlands of East and North Africa, the High Plateau of Mexico, and the Middle East. Clean cultivation and fungicides are suggested as possible control measures; however, resistant varieties offer a more realistic means of keeping losses to a minimum.

Figure 38. Perithecia of *Pyrenophora tritici-repentis* (the perfect stage of *H. tritici-repentis*) developing on weathered wheat straw.

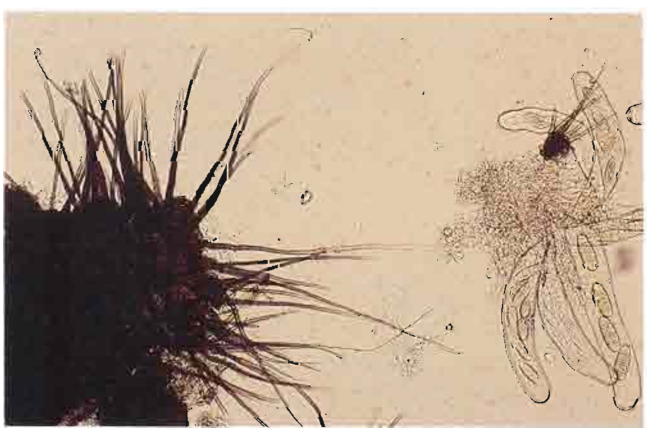


Figure 39. A closer view of a perithecium of *P. tritici-repentis*. Note the dark spines surrounding the short beak (10 x 40).

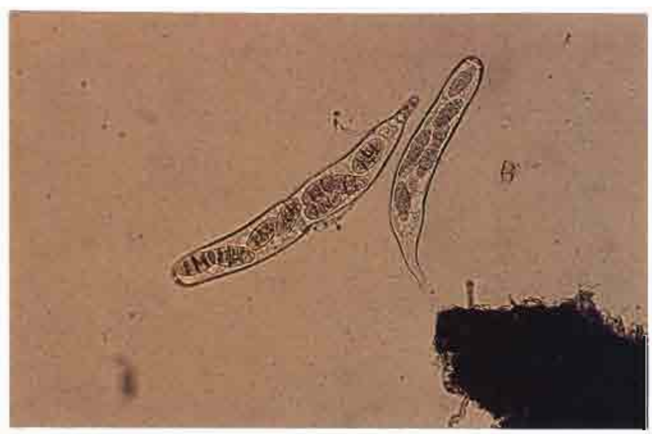


Figure 40. Asci containing ascospores of *P. tritici-repentis*. Note that the ascospores have transverse and longitudinal septa (10 x 40).

Leaf blotch of oats

*Helminthosporium avenae**

Leaf blotch, caused by *H. avenae,* is a common disease of oats. Apparently restricted to this crop, the fungus may be carried over from one oat crop to the next on the grain. The disease starts with the coleoptiles becoming infected. Subsequent infections develop from conidia produced on lesions. Diseased leaves develop oblong to elongated reddish brown spots (Figure 41). The leaf symptoms at the early stages of disease development are good diagnostic features. As the disease progresses, the brown merges into reddish shades with indefinite margins. The lesions gradually spread across most of the leaf blade, and eventually the affected leaves die and dry out as the disease advances. Under severe conditions, panicles can be infected, and the fungus becomes established in the hulls and kernels where it remains until the next crop cycle.

The fruiting structures of this fungus are similar in shape and color to those of *Helminthosporium teres* (net blotch) on barley (page 29). However, since *H. avenae* is restricted to oat species and *H. teres* does not infect oats, there should be no confusion as to the identity of the fungus. The conidiophores are scattered, develop singly or in clusters of two or three, and are thickened, measuring 8-12 μm thick and up to 200 μm long (Figure 42). The conidia are light yellow-grey when young and turn to a darker brown with age. They are straight or slightly curved, cylindrical with rounded ends, have four to six septa and measure 80-110 μm x 12-18 μm. The basal cell has a prominent scar (Figure 43). The perfect stage, *Pyrenophora avenae*, occurs infrequently in nature, and its role in disease development has not been established with certainty.

Helminthosporium avenae is widespread in most oat-growing regions of the world, but crop losses are generally light. Crop rotation and seed treatment with chemical fungicides reduce the severity and incidence of the disease.

* Perfect stage: *Pyrenophora avenae*

Figure 41. Leaf blotch of oats, caused by *H. avenae*. Note the reddish brown color.

Figure 42. Sporulation of *H. avenae* on a diseased oat leaf (50 x).

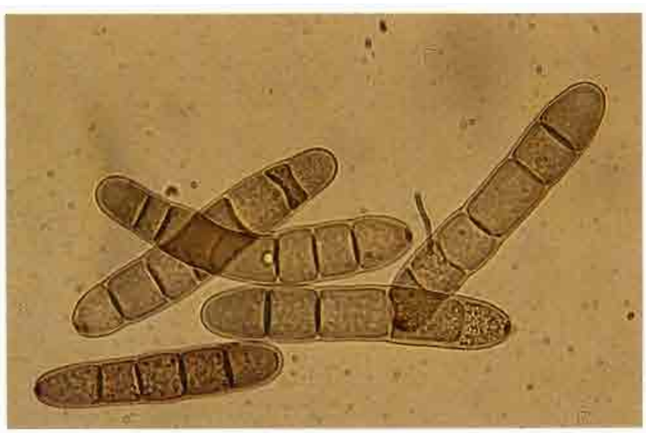


Figure 43. Conidia of *H. avenae*. Note the prominent scar on the basal cell (10 x 40).

Net blotch

*Helminthosporium teres**

Net blotch is primarily a leaf disease of barley. It occasionally affects wheat, triticale and other grasses. Disease symptoms appear first as small brown spots, usually near the tip of the blade (Figure 44). These spots soon elongate, becoming short stripes or blotches with internal brown pigmentation that develop in a transverse and longitudinal netlike pattern (Figure 45). Spikes may become infected as plants approach maturity.

Conidial development is somewhat unpredictable. Rapid sporulation is usually observed on fresh leaves after flowering, but often fails to develop on dry leaves after a few months in storage. Conidiophores develop singly or in groups of two or three, are light to medium brown, and are up to 200 μm long and 7-11 μm thick. The basal cell is usually enlarged. The conidia are straight, cylindrical, and light grey when young, turning greenish brown with age. Most of the conidia measure 60-120 μm x 16-23 μm and have four to six septa (Figure 46). They are similar in size and shape to *H. avenae* conidia (page 28).

The perfect stage, *Pyrenophora teres*, develops on overwintered straw and stubble. The perithecia are black, with setae (bristles) around the short beak. Mature ascospores are yellow-brown with three transverse and one longitudinal septa. The spores are constricted at the septa. The ascospores are important as inoculum in some areas where barley is produced.

Net blotch may develop from infected seed, or from ascospores released from perithecia on weathered straw and stubble. The disease is common in all cool and humid barley-growing regions of the world. A spot blotch phase of *H. teres* occurs on barley in some highland areas, particularly the Andes of South America. The disease can be distinguished from spot blotch, caused by *H. sativum* (page 24), only by observing the spores. Seed treatment with an appropriate fungicide controls the seed-borne pathogen. Resistance to *H. teres* has been derived from old varieties of barley originating in China, Turkey and Ethiopia.

* Perfect stage: *Pyrenophora teres*

Figure 44. Early symptoms of net blotch on barley, caused by *H. teres*.

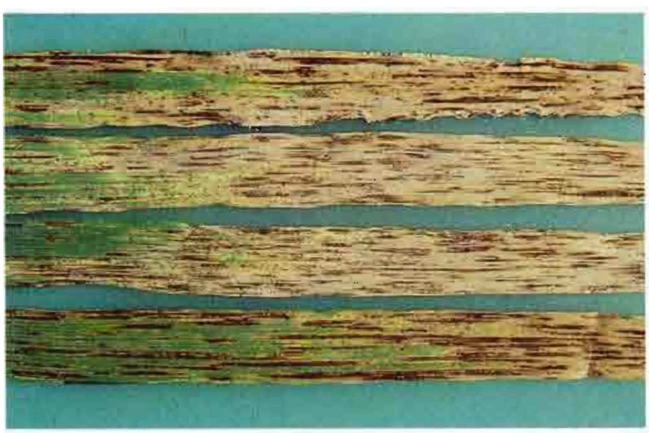


Figure 45. Advanced symptoms of net blotch on barley.

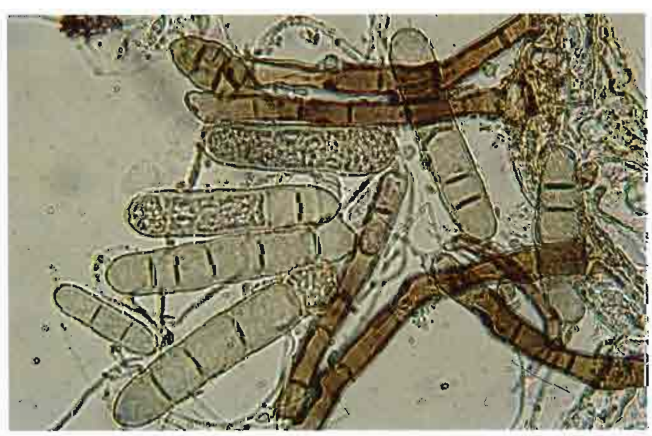


Figure 46. Conidia and dark conidiophores of *H. teres* (10 x 40).

Barley Stripe

Helminthosporium gramineum *

Barley stripe is a systemic disease that affects barley, but no other cereal crops. The fungus is seed-borne, and becomes active during seed germination, invading the coleoptile and infecting each of the leaves as they develop. Barley stripe symptoms provide excellent diagnostic features, and characteristic symptoms appear at the tillering stage. Narrow yellow stripes develop the full length of the leaf (Figure 47). These soon turn reddish brown and then dark brown. The leaf tissues die and dry out, causing the leaves to split along the stripes (Figure 48). Plants are dwarfed; the leaves turn dark, twist and fray; the spikes usually fail to emerge. Although disease symptoms appear on leaf blades and sheaths during the seedling stage, sporulation is delayed until the flowering period of the barley crop. Floral infection of healthy plants occurs from air-borne conidia. The fungus becomes dormant after invading the hulls of developing seed.

* Perfect stage: *Pyrenophora graminea*

Figure 47. Early symptoms of barley stripe, caused by *H. gramineum*. Note that the narrow yellow stripes develop the full length of the leaf blade (photo: S. Fuentes).

Figure 48. Advanced symptoms of barley stripe. Note the dark stripes and splitting of the leaf blades along the stripes.

The conidiophores appear in clusters of two to six and are grey to dark brown in color (Figure 49). The basal cell of the conidiophore is enlarged. The young conidia are light grey, turning dark brown with age. They are straight, cylindrical, slightly tapered, have rounded ends, and typically measure 60-90 µm x 15-18 µm with three to six septa (Figure 50). An unusual feature of this species during sporulation is the frequent development of secondary conidia from the apices of the primary ones. The perfect stage, *Pyrenophora graminea*, seldom occurs in nature and is of no importance in the disease cycle, but is included here because the name is commonly used by mycologists to identify the fungus.

The pathogen can be kept under control with fungicide seed treatment and resistant varieties. Its importance in Europe and North America has diminished considerably in recent years, though the disease is still common in some areas of South America, Africa and Asia.

Figure 49. Sporulation of *H. gramineum* on a barley leaf (50 x).

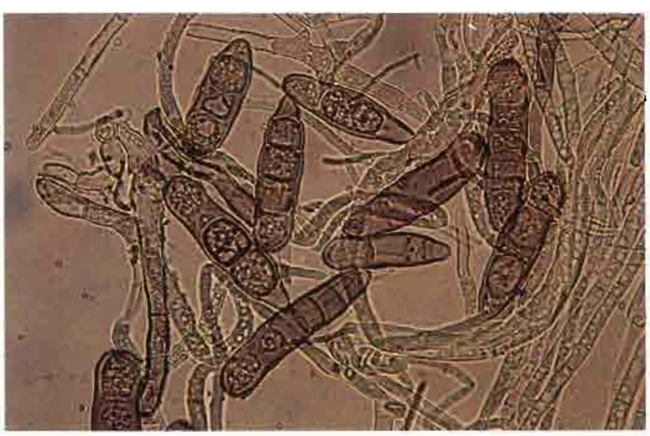


Figure 50. Conidia of *H. gramineum* (10 x 40).

Zonate Eyespot

Helminthosporium giganteum *

Zonate eyespot frequently occurs on wheat, triticale, rye and barley in the coastal areas adjacent to the Gulf of Mexico in the USA and Mexico, extending south into the higher elevations of Central America. It is a common disease on tropical grasses. Early symptoms appear as numerous small, grey-brown, oval to circular spots on green leaves. The centers of the spots soon fade, becoming light grey to straw colored with distinct dark brown margins (Figure 51). The spots tend to remain small, but the tissues between the spots develop an unusual bleached appearance (Figure 52). Eventually the whole leaf turns yellow and dies. The fungus attacks plants that are growing rapidly, from the seedling stage through heading. Sporulation diminishes quickly after severe necrosis develops, and other pathogenic and saprophytic organisms invade the affected plant tissues.

* Also known as: *Drechslera gigantea*

Figure 51. Distinctive zonate eyespot lesion on a wheat leaf, caused by *H. giganteum*. Note the definite dark brown margin.

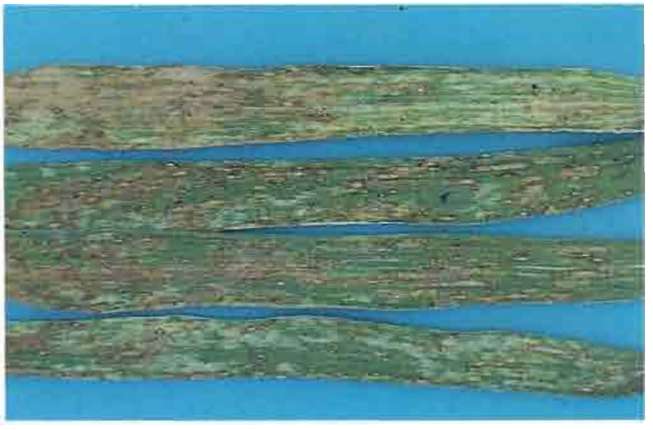


Figure 52. Advanced symptoms of zonate eyespot. Note the unusual bleached appearance of the tissue between the spots.

The disease is easily recognized by the "zonate eyespots" and the fungus by its large fruiting structures. The conidiophores are dark brown, rather thin, but may reach 400 μm in length (Figure 53). The conidia are almost colorless cylindrical tubes with rounded ends, have very thin walls, two to five septa, and measure 350-400 μm long by 15-20 μm wide (Figure 54). The conidia are sensitive to desiccation and lose their ability to germinate after a week or two of drying. The fungus often fails to sporulate after a secondary infection by bacteria.

Although the disease does not usually occur in the temperate zones, it may restrict commercial production of cereal crops in the more humid subtropics. Native tropical grasses apparently provide the major source of primary inoculum. As yet, very little research has been done to identify resistance or tolerance to this disease. Some strains of triticale appeared to be more tolerant than wheat or barley under a heavy natural infection of *H. giganteum* in the Alajuela Nursery, Costa Rica, in 1978.

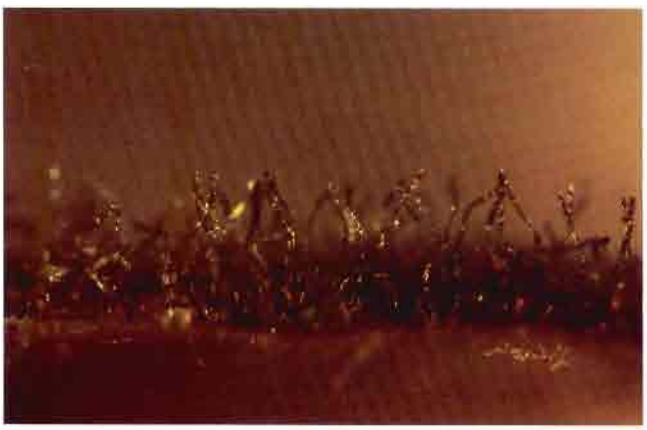


Figure 53. Sporulation of *H. giganteum* on wheat. Note how large these conidiophores are relative to those produced by other *Helminthosporium* species (50 x).

Figure 54. The nearly colorless conidia and dark conidiophores of *H. giganteum* (left photo—magnification 10 x 10); a comparison of relative sizes of *H. sativum* conidium (small, dark brown) and larger conidium of *H. giganteum*, (only part of which is visible at 10 x 40 magnification in the right photo).

Cereal leaf blotch and foot rot

Helminthosporium spiciferum *

This fungus occurs ocassionally on wheat, rice, and other cereals, and has been reported as a serious pathogen on some grasses. It is usually considered as a weak foot rot pathogen of wheat, but has been observed to cause leaf blotch symptoms on bread wheat and durums (in northern India and Pakistan) and on barley (in Mexico). Foliar symptoms appear as dull greyish brown, irregularly shaped blotches extending the full length and width of the leaves (Figure 55).

The fungus is easily identified by the branched superficial mycelia and distinctive conidia. Very heavy sporulation develops on diseased leaves during maturation of the host plant. Conidiophores arise from mycelia developing on the surface of plant tissues. Mycelia and conidiophores are yellow-brown to dark brown (Figure 56). The conidiophores have a knobby appearance from the scars of previous conidia. Conidia are short, oval shaped with rounded ends, straw colored to brown, normally measure 25-40 μm x 8-12 μm, and typically are three septate (Figure 57).

* Perfect stage: *Cochliobolus spicifer*

The perfect stage, *Cochliobolus spicifer*, has been reported, but its role in disease development has not been established. Although the disease has been reported to occur in North America, Europe, Asia and Australia, its influence in cereal crop production is negligible.

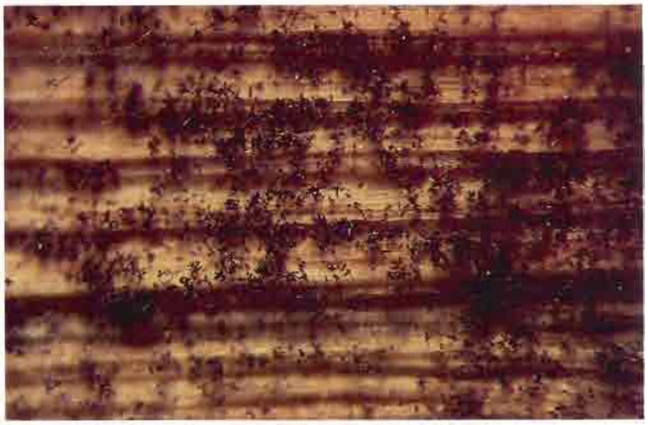


Figure 56. Sporulation of *H. spiciferum* on a diseased barley leaf.

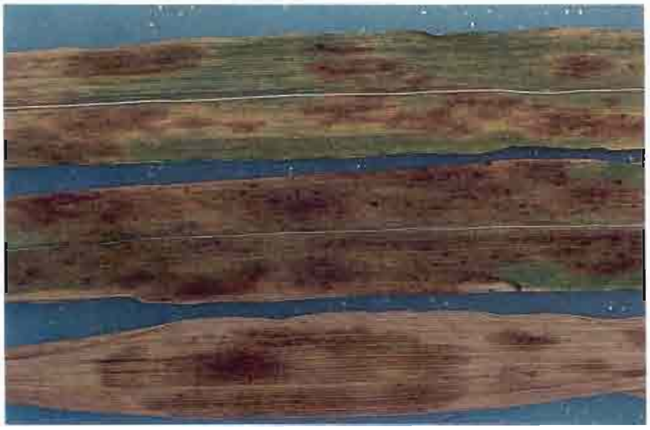


Figure 55. Typical symptoms of cereal leaf blotch on barley leaves, caused by *H. spiciferum.*

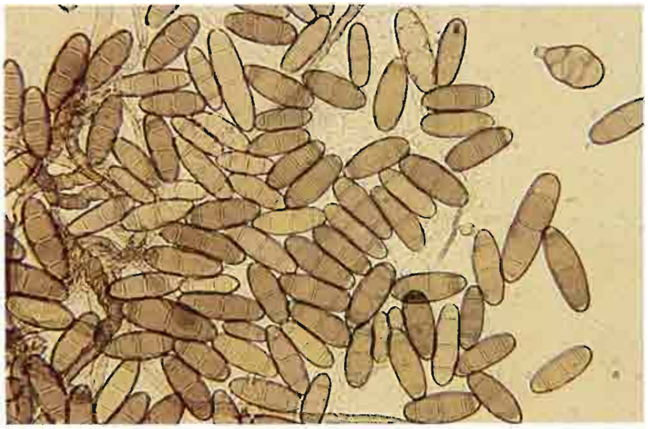


Figure 57. Conidia of *H. spiciferum* (10 x 40).

Septoria complex and septoria-like diseases

Four *Septoria* species are important pathogens of small grain cereals. They are the primary causes of various types of leaf spotting, blotching and necrosis. Depending upon the growth stage of the host and environmental conditions, any of the above-ground portions of the plant may be affected (Figure 58—next page).

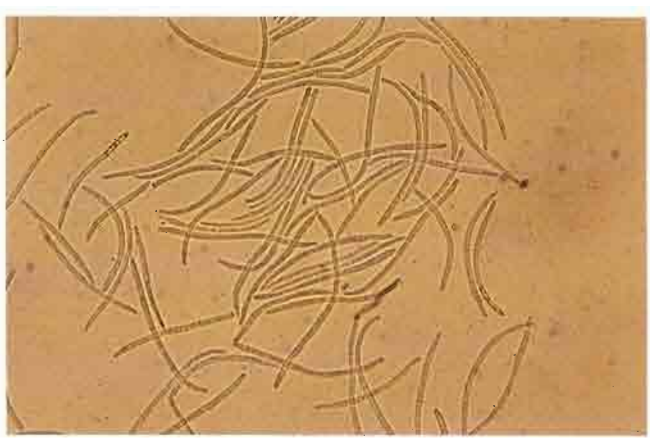
Septoria species possess several characteristics that distinguish them from other genera of pathogenic fungi parasitizing the same crops:

- The pycnidiospores (conidia) develop inside an enclosed, more or less spherical, fruiting structure called a pycnidium.
- The pycnidia develop within the plant tissue (embedded) and, as they mature, break through the epidermis. The conidia are extruded in a spore mass through a pore or ostiole. The spore mass, or cirrhus, is usually slightly pink to yellowish white in color.
- The conidia are threadlike or cylindrical with various lengths and widths depending upon the species. They have rounded ends, may be curved or straight, are hyaline, and usually have two to four cross walls or septa.

The size and length-width ratios of the conidia, the texture and color of the pycnidia or fruiting bodies, and the crop species they infect are important diagnostic features used to identify *Septoria* species.

Septoria diseases occur in all cereal growing regions of the world. The fungi overwinter on stubble and plant debris. Thus, clean cultivation and crop rotation are recommended as control measures in locations where severe septoria infections are frequent. Varieties that are resistant to one species of the pathogen are not necessarily resistant to others.

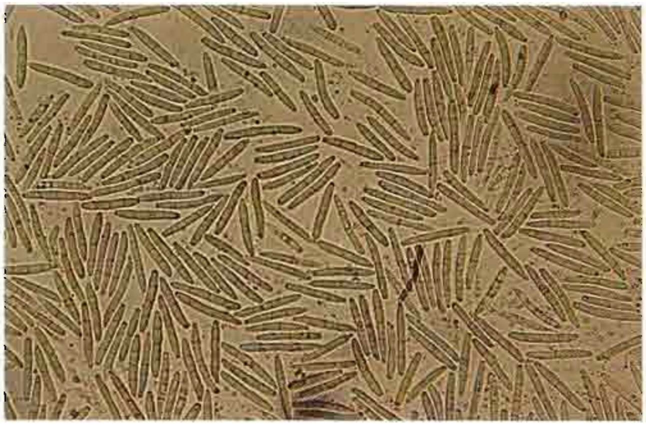
Distinguishing the species



Conidia of *S. tritici* (10 x 40)

Septoria tritici (speckled leaf blotch)

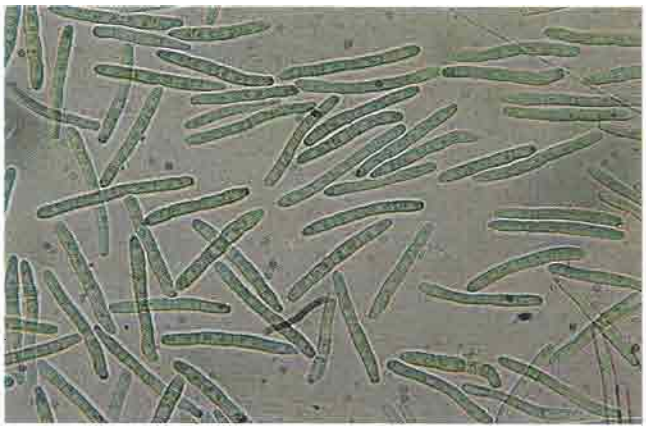
Pycnidia are very dark, and appear as tiny black specks in the lesions on plant tissues. Conidia are long, narrow, curved, threadlike rods, which usually range from 40-80 µm x 1.7-3.0 µm when mature. This species is parasitic mainly on wheat, but occurs on triticale, rye, and infrequently on some oat species.



Conidia of *S. nodorum* (10 x 40)

Septoria nodorum (glume blotch)

Pycnidia are jellylike and straw colored in actively developing lesions, becoming darker and tougher with age. Conidia are shorter, thicker, and straighter than any of the other *Septoria* species, and have one to three distinct septa when mature. Mature conidia are 15-24 μm long and 2.5-4 μm wide. This species is parasitic on wheat, triticale, rye and barley.



Conidia of *S. avenae* (10 x 40)

Septoria avenae (septoria leaf blotch of oats, wheat and triticale)

Pycnidia are soft and slightly darker than those of *S. nodorum*. The cirrhi (exuded spore masses) are distinctly pinkish in color. The conidia are intermediate in length when compared to *S. nodorum* and *S. tritici,* usually 20-45 μm x 2.5-4.0 μm. Forms parasitic on wheat and triticale are classified as *S. avenae triticea*, on oats as *S. avenae avenae*, and on rye as *S. secalis.*

Conidia of *S. passerinii* (10 x 40)

Septoria passerinii (septoria leaf blotch of barley)

This species is apparently restricted to barley. The conidia resemble *S. avenae* in shape but are narrower, measuring 26-42 μm x 1.5-2.0 μm. The mature conidia have two or three septa, and are mostly curved.

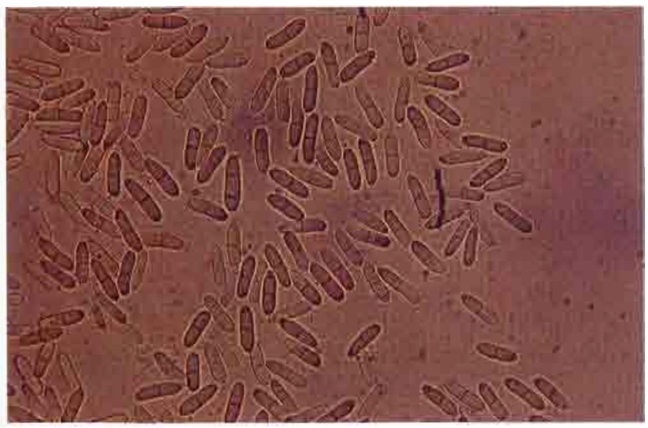
Figure 58. Speckled leaf blotch on wheat (facing page), caused by *Septoria tritici.* Note the tiny black specks, which are the pycnidia (photo: S. Fuentes).

Other similar fungi

Several species of fungi that resemble *Septoria* species in fruiting structures and in the disease symptoms they induce occur on cereals. Some are facultative parasites occurring regularly as leaf-spotting fungi in widely separated cereal crop areas. Two species that parasitize cereal crops and produce dark colored pycnidia are included here. They can be distinguished from the *Septoria* species by the shape and size of the conidia and the number of septa.

Ascochyta graminicola (ascochyta leaf spot)

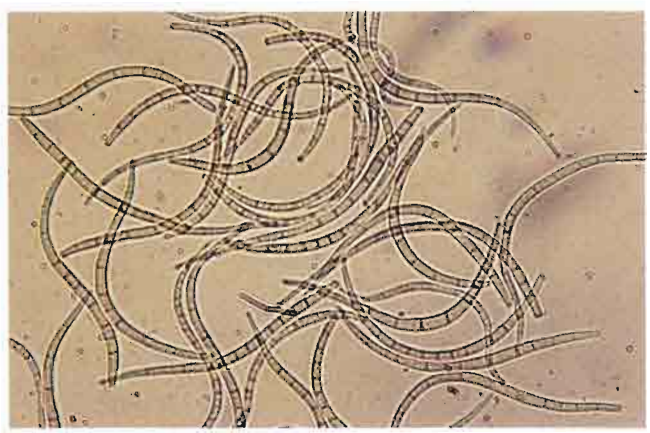
Grey-brown, spherical pycnidia are embedded in the leaf blades and sheaths. The conidia are hyaline, short, cylindrical to oval with rounded ends, have a single septum, and measure 14-22 µm x 4-5 µm.



Conidia of *Ascochyta graminicola* (10 x 40)

Phaeoseptoria vermiformis (phaeoseptoria leaf blotch)

Pycnidia are very dark, large, quite tough, and have short beaks. The conidia are light grey, long, narrow and taper toward the ends. They measure 70-120 µm x 3-4.5 µm, have numerous septa, and resemble nematodes in shape.



Conidia of *Phaeoseptoria vermiformis* (10 x 40)

Speckled leaf blotch

*Septoria tritici**

Speckled leaf blotch results in serious crop losses in many wheat-growing regions of the world. The disease is caused by the fungus *S. tritici,* which primarily infects wheat, though under favorable conditions it also attacks triticale and rye. It is occasionally found on barley and certain oat species.

The symptoms appear first as small, irregular, reddish brown leaf spots that, being restricted by the veins of the leaf, tend to develop longitudinally (Figure 59). As the disease develops, the centers of the lesions become ash colored. These lesions extend and merge, eventually expanding across the leaf, often resulting in complete necrosis. As the lesions enlarge, they lose their dark borders and tend to turn a light greyish color. It is then that the tiny dark specks (pycnidia) appear, hence the name "speckled" leaf blotch (Figure 60).

* Perfect stage: *Mycosphaerella graminicola*

Figure 59. Typical symptoms of speckled leaf blotch on durum wheat, caused by *S. tritici*.

Figure 60. Advanced symptoms of speckled leaf blotch on bread wheat. Note the different coloration of the lesions compared to durum wheat (Figure 59).

This disease usually appears on the lower leaves first. The rate of spread to upper leaves depends upon environmental conditions and the reaction of the variety to the disease. The disease becomes less aggressive as the crop begins to mature. While developing, the fruiting structures (pycnidia) are embedded in the leaf tissue (Figure 61). As they mature, they break through the epidermis and exude white to buff-colored spore masses (cirrhi) (Figure 62). The conidia are hyaline, threadlike (filiform) rods, usually curved, and have rounded ends with three to seven inconspicuous septa. The conidia range between 40-80 μm x 1.7-3.0 μm in size (Figure 63). Sometimes, small spores (microspores) are also produced.

Speckled leaf blotch occurs in the wheat-producing areas of all continents. The crop losses in some areas, such as North Africa and southern Brazil, can be devastating. The breeding of varieties resistant to *S. tritici* is under way in Mexico, Brazil, the USA, East and North Africa, in some countries of the Middle East, and in the Mediterranean region of Europe.

Figure 62. Under moist conditions, white to buff-colored cirrhi (containing conidia) are exuded from the pycnidia of *S. tritici.*

Figure 61. It is the dark pycnidia of *S. tritici* that gives lesions a speckled appearance.

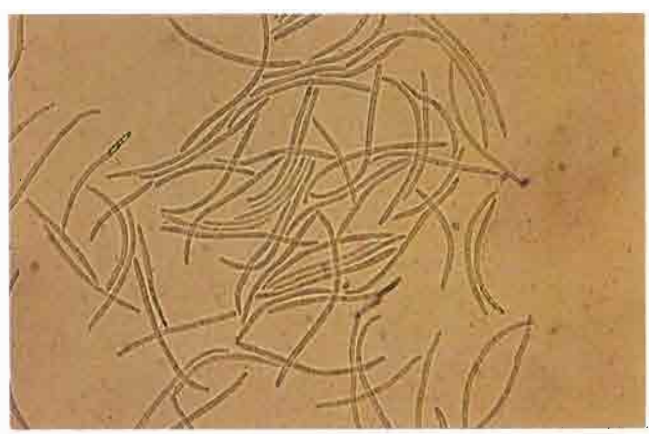


Figure 63. Conidia of *S. tritici* (10 x 40).

Glume blotch

Septoria nodorum *

Glume blotch can be a serious disease on wheat, triticale, rye and barley. The blotches caused by *S. nodorum* occur not only on the glumes, but just as frequently on leaf blades, leaf sheaths and nodes. Early leaf symptoms are yellowish to tan-brown, oval or lens-shaped spots with somewhat darker borders (Figure 64). The young lesions are less restricted by the leaf veins than those produced by *S. tritici.* The spots enlarge and merge, and as the tissues die, the necrotic areas become light grey in color. Light brown pycnidia develop randomly in scattered colonies and are less conspicuous than those of *S. tritici.*

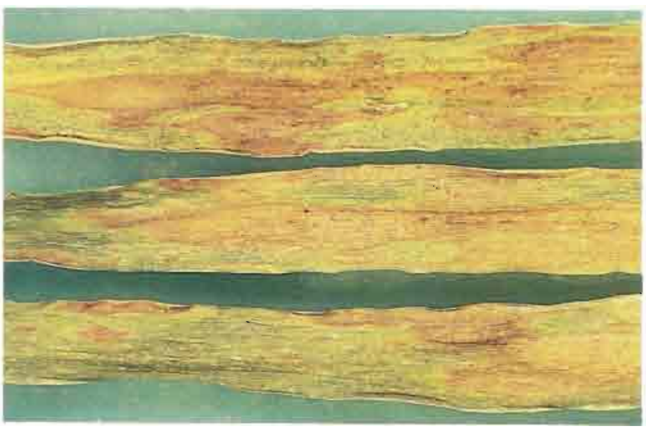


Figure 64. Typical leaf symptoms of glume blotch on wheat, caused by *S. nodorum.*

The pathogen becomes more aggressive as the crop approaches maturity, at which stage the nodes and glumes are primarily affected. The glume blotch stage usually starts at the tip of the glumes and lemmas as greyish discolorations across the glume with a brownish lower border. The disease development is downward and the brown border is eventually replaced by greyish discoloration (Figure 65). Pycnidia usually develop as small, dark grey or brown pimples before the blotch covers the top 1/3 of the glume.

At the glume blotch stage, the disease can be readily distinguished from black chaff, a bacterial disease caused by *Xanthomonas transluscens* (page 102). Black chaff starts developing in narrow, dark brown to black, water-soaked streaks at the base of the awn and tip of the glume (Figure 66). Usually, two or three short streaks develop at first, and globules of yellowish brown bacterial exudate are produced on both sides of the infected lemmas and glumes after incubation on moist filter paper. The lesions caused by *S. nodorum* are dry, turn the affected areas grey, and release white to pinkish-colored spore masses from the protruding pycnidia after 24-48 hours on moist filter paper.

Pycnidia developing on leaf blades and sheaths are honey brown at first, later turning dark grey and thus becoming more difficult to distinguish from *S. tritici.* Microscopic examinations should be

* Perfect stage: *Phaeosphaeria nodorum*

Figure 65. Symptoms of glume blotch on a bread wheat glume. Note the grey-brown discoloration developing from the tip downward, and the brown pycnidia embedded in the tissue.

Figure 66. Compare the typical symptoms of black chaff (shown here), caused by the bacteria *Xanthomonas translucens*, with those of glume blotch. Note the narrow, dark brown to black, water-soaked streaks beginning at the base of the awn.

made within 48 hours after diseased plant material is placed on moist filter paper, as the pink spore masses (cirrhi) are being exuded most actively at this stage and the development of saprophytes is limited (Figure 67). Conidia of *S. nodorum* are hyaline, straight or slightly curved with rounded ends, and usually have three conspicuous septa. The mature conidia are 15-24 µm long and 2.5-4.0 µm wide (Figure 68).

The perfect stage, *Phaeosphaeria nodorum,* develops in older lesions, particularly on diseased glumes. These fruiting structures appear black, and the beaks of the perithecia protrude through the epidermis (Figure 69). Ascospores are slightly curved, light grey in color and have four cells. The two end cells taper to rounded tips (Figure 70). They are indistinguishable from the ascospores of *P. avenaria*, the perfect stage of *S. avenae*. The frequency with which these occur in nature, and their ability to cause infection, suggest that they have some importance in initiating the disease.

The distribution of this fungus is worldwide and at least as broad as that of *S. tritici*. Its prevalence is less erratic and crop losses are probably greater than those caused by *S. tritici*. Varieties resistant to one pathogen are not necessarily resistant to the other. Triticales generally restrict the development of *S. tritici* fruiting structures, but are more susceptible to *S. nodorum*, which occurs quite frequently on the nodes.

Figure 67. Pinkish cirrhi of *S. nodorum* being exuded from pycnidia that are less conspicuous than those of *S. tritici*.

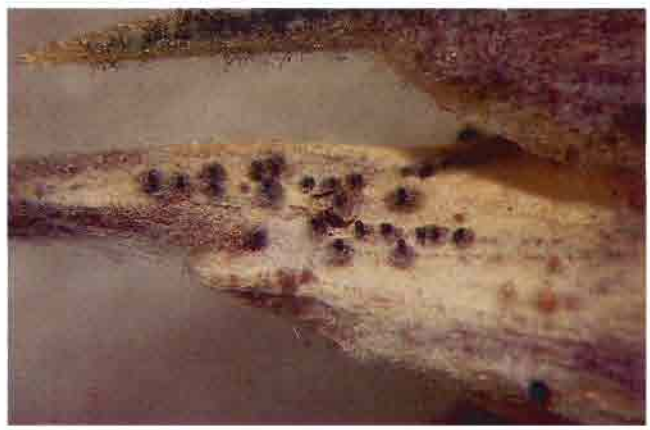


Figure 69. Perithecia of *Phaeosphaeria nodorum* (the perfect stage of *S. nodorum*) developing in an old glume blotch lesion.

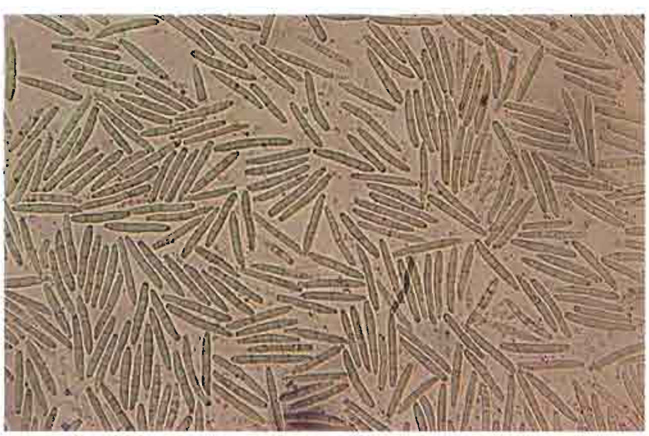


Figure 68. Conidia of *S. nodorum* (10 x 40).

Figure 70. Ascospores of *P. nodorum* (10 x 40).

Septoria leaf blotch of oats, wheat and triticale

*Septoria avenae**

At least three forms of this pathogen parasitize cereal crops. The variants that are pathogenic on wheat, triticale and barley are classified as *S. avenae triticea,* on rye as *S. secalis,* and on oats as *S. avenae avenae.* The pathogen produces similar pycnidial and perithecial forms on all of these crops. The disease caused by this pathogen on oats is generally referred to as septoria black stem (Figure 71). It is one of the most destructive diseases of oats in North America and elsewhere.

Lesions appear first on the leaves as small chocolate brown spots, becoming lens shaped as they expand and merge (Figure 72). The lesions soon turn to a light greyish brown, becoming difficult to distinguish from those caused by other *Septoria* species (Figure 73). This species continues to develop fruiting bodies during the ripening stage well after active plant growth has ceased. It is found also on tissues that have been infected by other pathogens.

* Perfect stage: *Phaeosphaeria avenaria*; until recently, *Leptosphaeria avenaria*

Figure 71. Septoria black stem on oats, caused by *S. avenae avenae*.

Figure 72. Early symptoms of septoria leaf blotch on oat leaves, caused by *S. avenae avenae*.

Figure 73. Advanced symptoms of septoria leaf blotch on oats. At this stage, the disease symptoms become more difficult to distinguish from those caused by other *Septoria* species.

The pycnidia release pinkish-colored spore masses in profusion (more so than *S. tritici* – see Figure 74). The conidia are hyaline, straight or slightly curved, cylindrical with rounded ends, have three to four septa, and typically measure 20-45 μm x 2.5-4.0 μm in size (Figure 75). They are intermediate in length between *S. tritici* and *S. nodorum.*

The perfect stage, *Phaeosphaeria avenaria,* develops readily on diseased straw and stubble that has weathered for some time. Ascospores developing on overwintered stubble and debris provide the initial inoculum for this disease on oats in northern areas of North America (Figure 76). The ascospores are similar in size, shape and color to those produced by *P. nodorum*, the perfect stage of *S. nodorum*.

The disease has been observed on wheat in Europe, Asia, North Africa, South America, the Central Plains of North America and Mexico. Although occurring in the same general areas as *S. nodorum,* losses caused by *S. avenae* on crops other than oats are relatively minor.

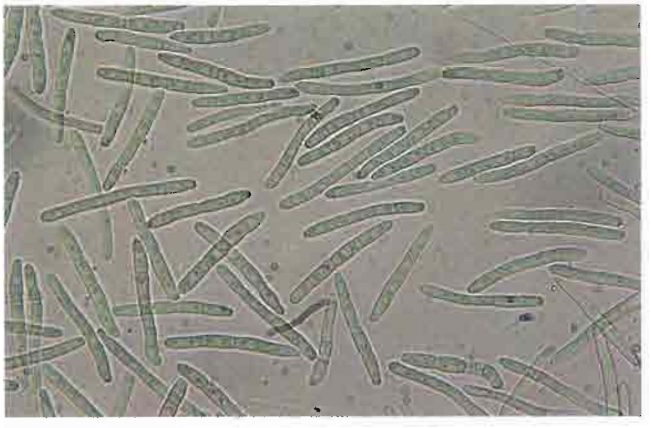


Figure 75. Conidia of *S. avenae* (10 x 40).

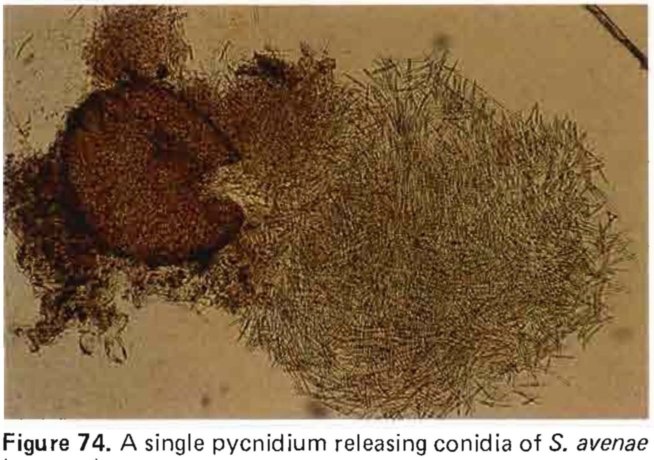


Figure 74. A single pycnidium releasing conidia of *S. avenae* (10 x 10).

Figure 76. Asci and ascospores of *Phaeosphaeria avenaria*, the perfect stage of *S. avenae* (10 x 40).

Septoria leaf blotch of barley

Septoria passerinii

Leaf blotch of barley, caused by *S. passerinii*, appears to be restricted to cultivated barley and some of its wild relatives. The disease is common in the northern area of North America, Europe and Asia, but it is the least damaging of the septoria complex.

The leaf blotch symptoms are similar to those produced by *S. avenae,* including the dark specks (embedded pycnidia — see Figure 77). *Septoria* species that are pathogenic on barley produce dark pycnidia in the lesions, thus microscopic examination is necessary for accurate identification.

Colonies of dark brown pycnidia develop in the elongated lesions on leaf blades, leaf sheaths, and stems (Figure 78). The conidia are hyaline, and may be straight or curved, with one end tapering slightly more than the other. They measure 26-42 µm x 1.5-2.0 µm and usually have three septa (Figure 79). The fungus has been observed in the leaf and stem lesions of mature barley grown on the High Plateau of Mexico. These conidia are sometimes curved, and may exceed 2.0 µm in width (Figure 80).

The fungus persists in plant debris, particularly on the dead leaves of *Hordeum jubatum* or other wild barleys that are common in areas where this disease occurs. Little attention has been given to the development of resistant varieties and other disease control measures.

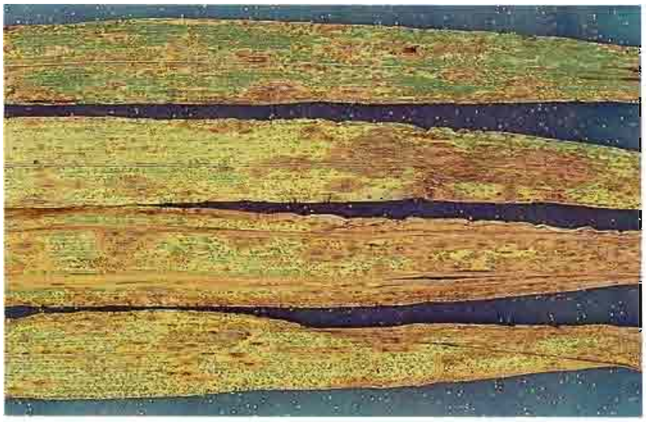


Figure 77. Typical leaf blotch symptoms on barley, caused by *S. passerinii*.

Figure 79. Typical conidia of *S. passerinii* (10 x 40).

Figure 78. A close look at *S. passerinii* pycnidia on diseased barley.

Figure 80. Somewhat different conidia of *S. passerinii* taken from diseased barley on the High Plateau of Mexico (10 x 40).

Ascochyta leaf spot

Ascochyta graminicola

Ascochyta leaf spot is a minor, but widely distributed disease, usually producing leaf spots on the lower leaves of wheat, oats, triticale, barley and numerous grasses. Lesions occur on leaf blades and sheaths, often in association with other leaf-spotting fungi. They develop as circular or oval-shaped chlorotic spots, gradually merging to form light brown to grey-brown diffuse blotches (Figure 81). The disease is often confused with leaf spots caused by *Septoria* species.

The fruiting structures occur as colonies of dark pycnidia in the tissue within the lesion. The conidia are two celled, hyaline, short, more or less straight, cylindrical to slightly oval shaped with rounded ends; they typically measure 14-22 μm x 4-5 μm. A wide range in conidial sizes has been observed. For example, variants with small conidia (measuring 10 μm x 2.5 μm) develop on oats in Kenya (Figure 82), while large-spored variants (measuring 25 μm x 7 μm) have been observed on triticale on the High Plateau of Mexico (Figure 83).

Ascochyta leaf spot is common in areas having mild, temperate climates. The fungus persists on the dead leaves of native grasses. High humidity in the early spring favors sporulation and disease development. Cereal crops are probably infected by spores released from plant debris, or by secondary inoculum from diseased grasses.

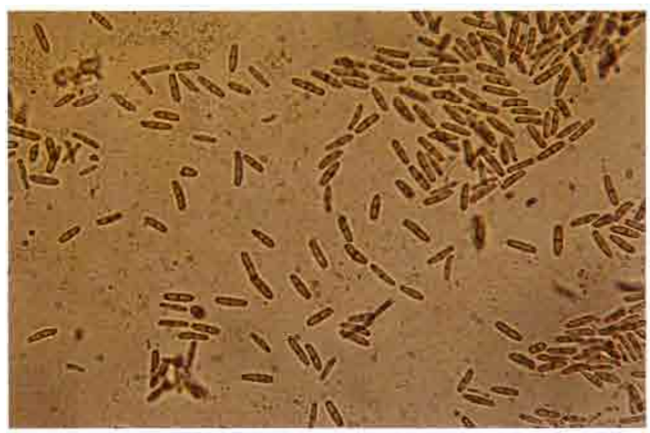


Figure 82. Variants of *Ascochyta graminicola* with small conidia (shown here) are found on oats in Kenya (10 x 40).

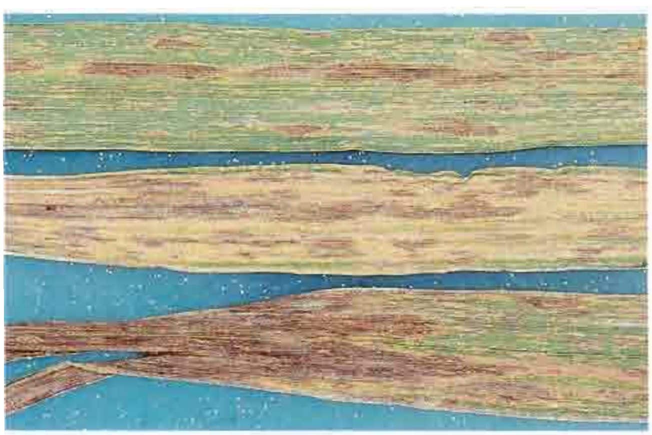


Figure 81. Symptoms of ascochyta leaf spot on diseased wheat leaves, caused by *Ascochyta graminicola*.

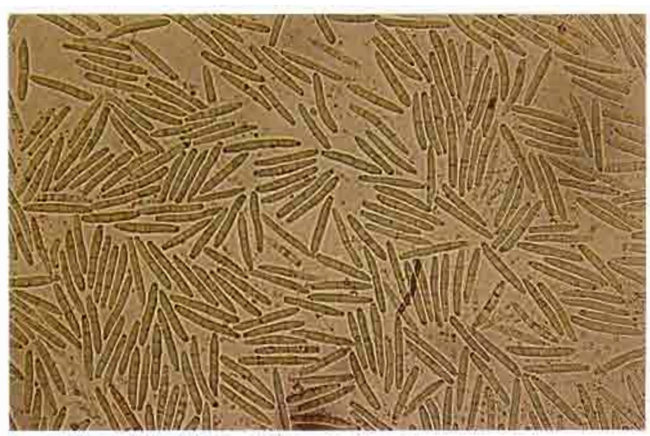


Figure 83. Large-spored variants of *Ascochyta graminicola* have been observed on triticale grown on the High Plateau of Mexico (10 x 40).

Phaeoseptoria leaf blotch

Phaeoseptoria vermiformis

Leaf blotch caused by this fungus occurs in subtropical highlands on wheat, triticale and oats. Although the disease caused by *P. vermiformis* has been observed frequently on the High Plateau of Mexico and in East Africa, its importance as a cereal crop disease is negligible. The fungus was described by Punithalingam and Waller at the Commonwealth Mycological Institute, Kew, England, in 1980.

Leaf lesions caused by *P. vermiformis* are similar to those produced by *Septoria nodorum*, and they often occur together. The blotches are light brown to medium brown with diffuse pale yellow to straw-colored areas extending lengthwise. The lesions frequently occur at the edges of the leaves (Figure 84). The pycnidia are dark brown to black, are embedded in leaf tissue, and have short beaks that rupture the epidermis (Figure 85). Light grey conidial masses are exuded after 24-48 hours in a moisture chamber. The conidia are pale grey, long, narrow, conspicuously septate, curved, and tapered toward the ends. They typically measure 70-120 μm x 3.0-4.5 μm with 8 to 14 septa (Figure 86). They resemble long narrow nematodes with dark septa. The life cycle of this fungus has not been studied. It is probable that the fungus persists on native grasses in its natural habitat.

Figure 84. Phaeoseptoria leaf blotch symptoms on triticale leaves, caused by *Phaeoseptoria vermiformis*.

Figure 85. Relatively large, black pycnidia of *P. vermiformis*. Note the beaks of mature pycnidia breaking through the epidermis.

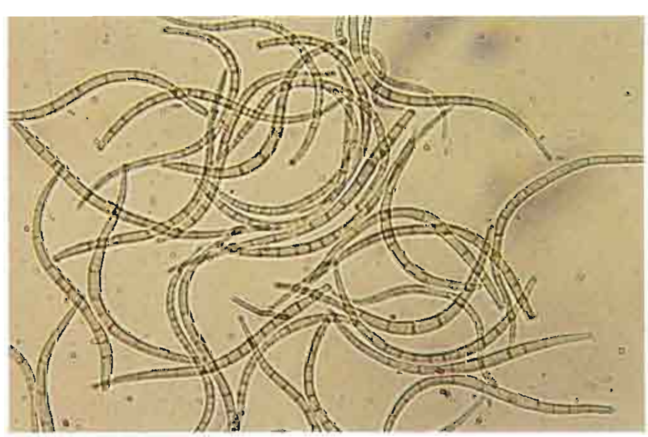
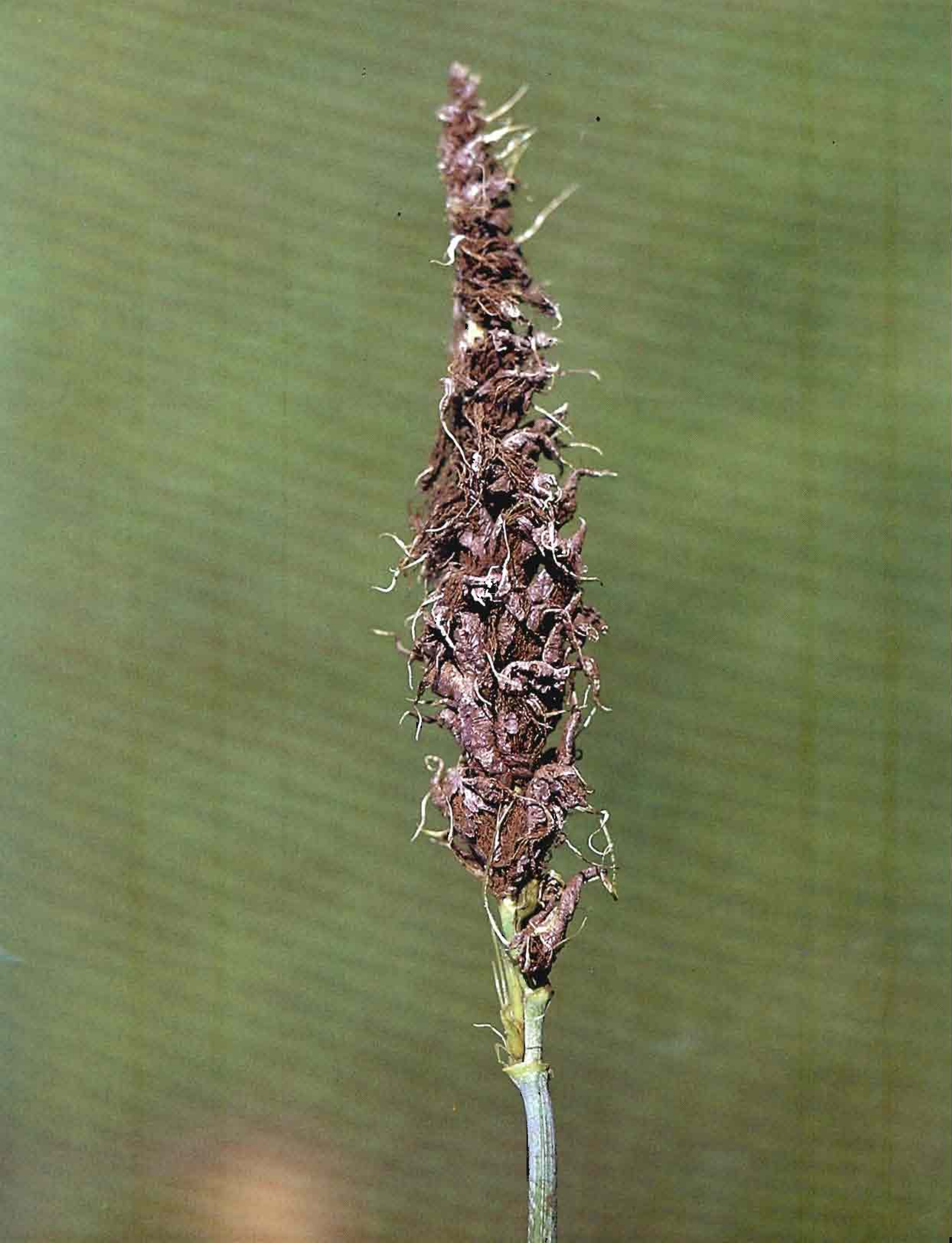


Figure 86. Conidia *P. vermiformis* (10 x 40).



Smuts and bunts

Four genera of Basidiomycetes are included under the general heading of smuts and bunts. They are pathogenic on small grain cereals and many grasses, and produce black spore masses (teliospores*) that partially or completely replace heads, spikelets or kernels (Figure 87). They all tend to be seed- and/or soil-borne pathogens. Smut diseases caused by *Ustilago* species are usually initiated via floral infection. They develop systemically in the host plant during the generation following infection, and smut spore masses are produced instead of grain. In contrast, bunt diseases caused by *Tilletia* species initiate infection at the seedling growth stage of the host plant, develop systemically, and produce bunt balls instead of grain in the same generation that infection occurred. Karnal bunt, caused by *Neovossia indica*, is initiated exclusively via floral infection, does not develop systemically, and produces spores in the infected kernels. Flag smut, caused by *Urocystis* species, produces black spore masses in the leaves and stems of diseased plants, and infection is initiated at the seedling growth stage of the host plant.

Figure 87. A barley spike (facing page) displaying the black spore masses typical of loose smut, caused by *Ustilago nuda* (photo: S. Fuentes).

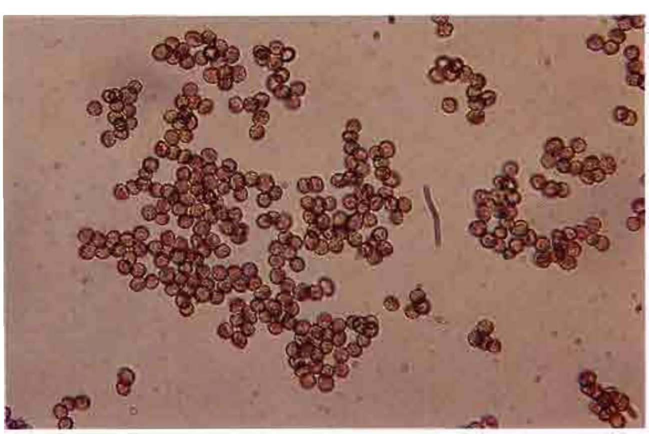
Features common to the smuts and bunts include:

- They are caused by obligate parasites.
- They produce dark spore masses that usually appear at the heading growth stage of the host plant.
- Most of the genera have developed distinctive physiological races.

The smuts and bunts are injurious diseases on cereal crops in the Eastern Hemisphere. They are particularly abundant in the Near and Middle East and the Indian Subcontinent. Numerous wild relatives of cereal crop species are indigenous to these areas, and serve as alternative hosts for the smut- and bunt-causing pathogens. The use of disease-free seed, fungicide seed dressing, crop rotation and resistant varieties (when available) are effective as control measures .

* Teliospores are thick-walled resting spores produced by rust and smut fungi. In this section, the words "spore" and "teliospore" are used interchangeably.

Distinguishing the genera



Teliospores typical of *Ustilago* species (10 x 40)

Ustilago species (loose and covered smuts)

Dark, loose spore masses, enclosed in a fine membrane, completely replace the florets. Teliospores are very small, measuring 5-10 μm in diameter.

Tilletia species (common and dwarf bunts)

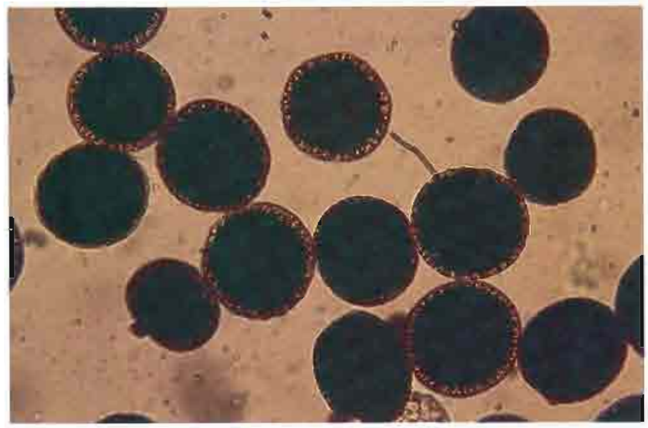
Greyish brown, tough-coated bunt balls containing black spore masses replace the kernels. The bunt balls emit a strong fishy odor when crushed. The teliospores are intermediate in size, measuring 15-23 μm in diameter.

Teliospores typical of *Tilletia* species (10 x 40)

*Tilletia indica** (karnal bunt)

Kernels are partially or completely replaced by black spore masses. Floral infection occurs from sporidia produced by teliospores germinating near the soil surface. Teliospores are large, measuring 25-30 μm in diameter, and also emit a fishy odor.

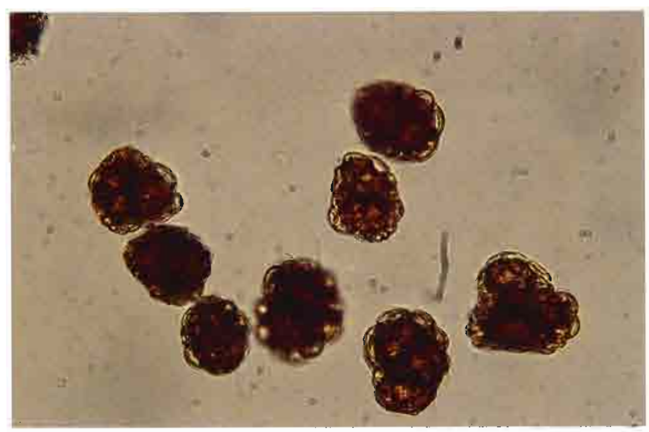
* Formerly *Neovossia indica*



Teliospores of *Tilletia indica* (10 x 40)

Urocystis species (flag smut)

Black spore masses develop in the leaf blades and sheaths. Teliospores are released in clusters of two to four viable teliospores surrounded by a layer of sterile cells. The viable teliospores measure 10-20 μm in diameter.



Teliospores typical of *Urocystis* species (10 x 40)

Loose and covered smuts

Ustilago species

Loose smuts are among the most easily recognized diseases affecting cereal crops. The spikelets of diseased plants are transformed into dark olive brown to black spore masses. The disease becomes obvious only after heading. As the heads emerge, the spore masses are enclosed in delicate greyish white membranes that rupture prior to or during harvest, releasing powdery spore masses. Disease spreads either by floral infection (*U. nuda*), or by embryo infection from spores that may be carried on the seed or that are in the soil (*U. nigra, U. hordei*). The fungus mycelium invades the growing point of the embryo and grows internally with the plant until the floral structures are replaced by spore masses at the heading stage.

Three closely related forms or species are recognized on the basis of morphological characteristics of the fungi, disease symptoms, and life cycle:

- Loose smut of barley and wheat (*U. nuda*).
- Black loose smut of barley and oats (*U. nigra*).
- Covered smut of barley and oats (*U. hordei*).

Physiological specializations based on the reaction of crops and varieties have been established within each of these species. Hybridization has been reported between some forms within the three groups, which suggests that the forms may be more correctly regarded as variants rather than distinct species.

Barley, oats, wheat and many grasses are attacked by smuts of the *Ustilago* species, while triticale and rye are rarely infected. The incidence of loose and covered smuts has decreased substantially in North America and Europe as a result of the use of more effective seed-treatment fungicides, and more resistant varieties.

Loose smut of barley and wheat

Ustilago nuda (U. tritici)

The disease caused by this species occurs via floral infection, and only barley and wheat are susceptible. One form of *U. nuda* affects barley but not wheat (Figure 88); another form, previously identified as *U. tritici*, affects wheat but not barley. Otherwise, these two forms are identical. The heads of diseased plants tend to emerge slightly earlier

Figure 88. A barley spike with loose smut, caused by *U. nuda*. The spike has recently emerged and still retains the delicate, silvery grey membrane that initially encases the spores (photo: S. Fuentes).

than those of healthy plants. The membranes enclosing the spore masses are very delicate, rupturing soon after the head emerges (Figure 89). The olive brown teliospores are dispersed by wind currents. Floral infection is initiated by teliospores landing on the flowers. Teliospores then germinate and produce infectious mycelia, which invade the ovaries and eventually the embryos of developing seed. The fungus remains dormant until the seed germinates. The mycelium grows systemically with the seedling and, as the plant approaches heading, the mycelium penetrates the head tissues and converts them to masses of teliospores.

Teliospores are spherical, finely echinulate (possessing short spines on their surfaces), are of lighter color on one side, and measure 5-10 μm in diameter (Figure 90). Germinating teliospores produce a four-celled promycelium, but no sporidia (Figure 91).

In barley, loose smut caused by *U. nuda* is difficult to distinguish from black loose smut caused by *U. nigra*. Usually, there is more awn development on plants infected by *U. nigra*. However, the variation in awn development among varieties of barley and wheat limits the usefulness of this characteristic as a diagnostic feature. Differences in spore size and degree of echinulation are insufficient to distinguish the species with certainty. Differences in spore color, olive brown in *U. nuda* and dark chocolate brown in *U. nigra*, can be useful if comparative samples are available. A more reliable means of distinguishing the two species is to observe the germination of the teliospores on water agar. *Ustilago nigra* produces sporidia on its germinating promycelia, while *U. nuda* does not.

Ustilago nuda survives between crop cycles as dormant mycelium in the embryo and endosperm of diseased seed. Fungicide seed treatments used to control seed-borne fungi are ineffective. Hot water treatments that were used to disinfect seed have now been superseded by systemic fungicides, such as *Carboxin*. Resistant varieties and good cultural practices have reduced the incidence of this disease.

Figure 89. What remains of a barley spike infected by *U. nuda*. The membrane has broken and most of the spores are dispersed (see Figures 87 and 88).

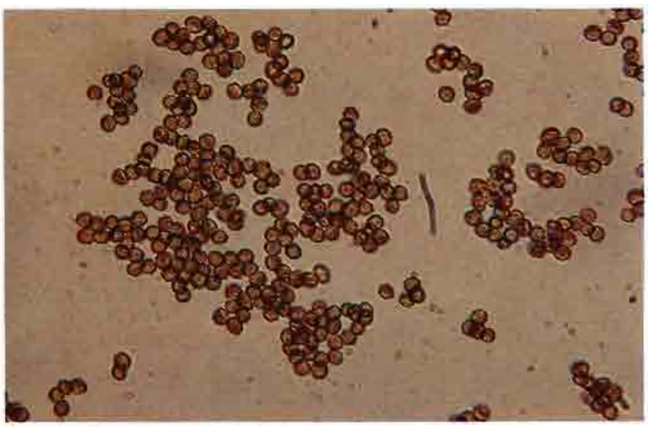


Figure 90. Teliospores of *U. nuda* (10 x 40).

Figure 91. Teliospores of *U. nuda* germinating on water agar. Note the development of branched promycelia, but that no sporidia are produced (10 x 40).

Black loose smut of barley and oats

Ustilago nigra (U. avenae)

Barley and oats are susceptible to forms of this species. One form attacks only barley; another attacks only oats (Figure 92). The disease symptoms are quite similar to those of loose smut. The awns and glumes tend to develop to some extent, particularly in barley (Figure 93), but this characteristic varies with variety and environment. The spore masses are dark chocolate brown to black. The membranes enclosing the spore masses are somewhat tougher than those produced by *U. nuda*, and the dispersal of spores is delayed depending upon the durability of the membranes. Teliospores lodge in the floral bracts, or on the hulls and seed of neighboring plants where they remain dormant until the seed is planted. Seedling infection occurs via direct penetration of the coleoptile by the sporidia of germinating teliospores that are either on the seed or in the soil close to the germinating embryo.

The teliospores are dark brown to black, more or less spherical, 6-7.5 µm in diameter, and vary in the degree of echinulation. They are practically indistinguishable from the teliospores of *U. nuda* (see Figure 90). The germinating teliospores produce a promycelium and oblong sporidia. Secondary sporidia are produced by yeastlike budding. The formation of these sporidia is a useful diagnostic feature.

Seedling infection can be prevented by appropriate fungicide seed treatments. The use of varieties resistant to the local races, as well as seed treatments, usually keeps the disease under control.

Figure 92. Oat spikes with two kinds of smut: black loose smut on the left (caused by *U. nigra*), and covered smut on the right (caused by *U. hordei*).

Figure 93. A barley spike with black loose smut, caused by *U. nigra*. Note the awn development.

Covered smut of barley and oats

Ustilago hordei (U. kolleri)

Serious crop losses from covered smut occur only in barley and oats. The forms causing covered smut have identical morphological characteristics, but differ in their ability to parasitize oats and barley. One form infects barley but not oats (Figure 94), and another form infects oats but not barley (Figures 92, 95). Covered smuts differ from the loose smuts in the degree to which the floral bracts and awns develop, and in the degree of persistance of the membranes enclosing the spore masses. The spore masses tend to remain relatively intact until the crop approaches maturity. The spores are dispersed during threshing, thus contaminating the healthy grain.

The spore masses are dark brown to black in color, but under the microscope the teliospores appear lighter in color on one side. They are subspherical to angular in shape, with smooth walls, and measure 5-9 μm in diameter. Germinating teliospores develop a promycelium and four primary sporidia, from which abundant secondary sporidia are produced (Figure 96).

Figure 94. A barley spike with covered smut, caused by *U. hordei*.

The fungus is carried over from cycle to cycle as a seed-borne pathogen, either as dormant spores or a mycelium in the hulls and seed coat of the grain. Appropriate fungicides used as seed treatments are effective as control measures. Many varieties that are resistant to black loose smut are also resistant to covered smut.

Figure 95. Oat panicles with covered smut, caused by *U. hordei*.

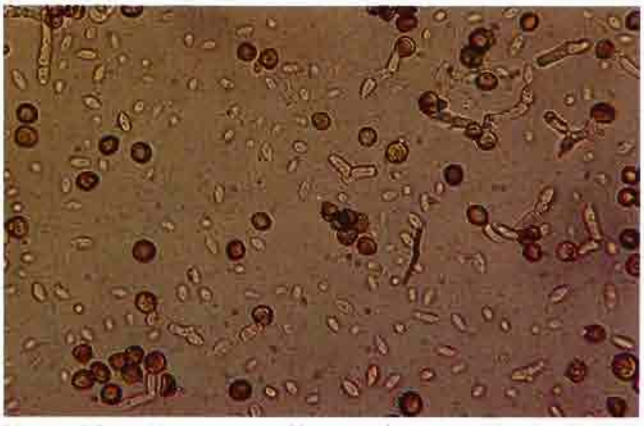


Figure 96. Teliospores of *U. hordei* germinating on water agar. Note the short promycelia and abundant sporidia (10 x 40).

Common and dwarf bunts (stinking smut)

Tilletia species

Common and dwarf bunts are injurious pathogens of wheat, and can also affect barley and certain grass species. Bunts seldom occur on the other small grain cereals. The life cycle of the bunts is similar to that of covered smuts. Diseased plants are usually reduced in height, and often display increased tillering (Figure 97). The degree of dwarfing varies with the race of the bunt fungus and the plant variety. The disease becomes more evident after heading. Diseased spikes are bluish green, and the glumes tend to spread apart slightly to accommodate the bunt balls that have replaced the normal kernels (Figure 98). As the crop matures, the diseased spikes can be distinguished by their darker color, the expanded glumes with the tips of the bunt balls protruding, the variation in height, and the abnormalities in spike shape and size. Diseased club wheat spikes are distinctly more lax and longer than healthy ones.

Bunt balls are greyish brown, similar to normal seed in shape, but usually more spherical (Figure 99). When they are crushed, black powdery spores having a strong fishy odor are released. The spores from diseased plants are dispersed during harvesting operations, contaminating the grain and soil. The teliospores persist on seed or in dry soil until moisture becomes available. Germinating teliospores form a promycelium and 8 to 16 basidiospores, which fuse in pairs forming H-shaped structures, and eventually develop infectious secondary sporidia. The sporidia infect the coleoptiles of young seedlings, or the growing points of young tillers, often before they emerge from the soil. The mycelium of the pathogen grows within the seedling, advancing as the plant develops and eventually invading the meristematic tissues. The pathogen is particularly well suited to developing on winter wheat in temperate climates.

Figure 97. Dwarf bunt, caused by *Tilletia controversa*, results in severe dwarfing and excessive tillering.

Figure 98. Wheat spikes with bunt. Note how the glumes are spread apart by the larger size of the bunt balls that replace the grain.

Figure 99. Two wheat spikes with common bunt (stinking smut). On the left, bunt balls produced by a spike infected with *T. caries*; on the right, bunt balls produced by a spike infected with *T. foetida*.

Three closely related fungi are involved. *Tilletia caries* and *T. foetida* cause common bunt (stinking smut) and *T. controversa* causes dwarf bunt. *Tilletia caries* and *T. foetida* are very similar in morphological features, life cycles, disease development, and in the occurrence of physiologic races. They could be considered variants of one species. They differ slightly in the shape of the teliospores and in the texture of the spore walls. *Tilletia caries* produces dull grey-brown, elongated or kernel-shaped bunt balls, while *T. foetida* produces elongated to round bunt balls (Figure 100). The teliospores of *T. caries* are globose, brownish black in mass, and measure 15 to 23 µm in diameter with reticulate walls (Figure 101). The teliospores of *T. foetida* are globose to elongated with smooth walls, measuring 17-22 µm in diameter (Figure 102).

Tilletia controversa, the cause of dwarf bunt, is very difficult to distinguish from *T. caries* in its morphological features, but differs markedly in the symptoms it causes. A more pronounced stunting occurs in plants with dwarf bunt than with common bunt (see Figure 97). The disease is also restricted to areas where snow cover remains over the soil for extended periods; the spores of dwarf bunt require longer to germinate than those of common bunt. As a consequence of this delayed germination, infection by *T. controversa* usually occurs at the two to three leaf stage of host plant development.

The most effective control of common and dwarf bunts is obtained from a combination of seed treatment and resistant varieties. Among the fungicides that are effective in the control of both seed-borne and soil-borne spores are hexachlorobenzene, pentachloronitrobenzene, *Carboxin* and *Thiabendazole.*

Figure 101. Teliospores of *T. caries* (10 x 40).

Figure 100. A comparison: normal wheat kernels (top, center); bunt balls caused by *T. foetida* (bottom, left); bunt balls caused by *T. caries* (bottom, center); seed galls or cockles caused by the nematode, *Anguina tritici* (bottom, right).

Figure 102. Teliospores of *T. foetida* (10 x 40).

Karnal bunt

*Tilletia indica**

Karnal bunt is primarily a disease of bread wheat, but durum wheats and triticale may be affected (Figure 103). The disease is usually noticed first when broken and partially smutted kernels are seen in the threshed grain. Unless the disease is severe, only a few florets per spike are affected, and these are easily overlooked. While diseased seeds usually retain a partial seed coat, the embryo and part of the endosperm can be converted to masses of dry black spores (Figure 104). They emit a fishy odor similar to that of common bunt. The teliospores of karnal bunt are large (25-30 μm in diameter), more or less spherical, black and have a slightly rough surface (Figure 105). Germinating teliospores form a promycelium from which numerous sporidia are released.

Figure 103. Wheat spike with karnal bunt, caused by *Tilletia indica*.

The main source of inoculum is diseased seed, or soil contaminated with spores from the previous crop. The teliospores germinate, producing numerous sporidia at the soil surface, which are then dispersed by wind and insects. Sporidia infect the ovaries during the flowering stage of the host plant and diseased kernels may be partially or completely displaced by masses of teliospores.

More research on the reaction of varieties and the effectiveness of fungicides is needed before control measures can be recommended. However, the use of disease-free seed is essential. The evidence gathered to date in Mexico on chemical seed treatment for karnal bunt indicates that PCNB (pentachloronitrobenzene), at 2 kg/ton of seed, provides satisfactory control of seed-borne inoculum. Although the disease is indigenous to the Punjab area of the Indian Subcontinent, it is now reported to be in Syria, Lebanon, Iraq and Turkey. Its survival in other parts of the world appears to be restricted by environmental factors.

* Formerly *Neovossia indica*

Figure 104. Wheat kernels with karnal bunt. Note the retention of part of the seed coat (photo: S. Fuentes).

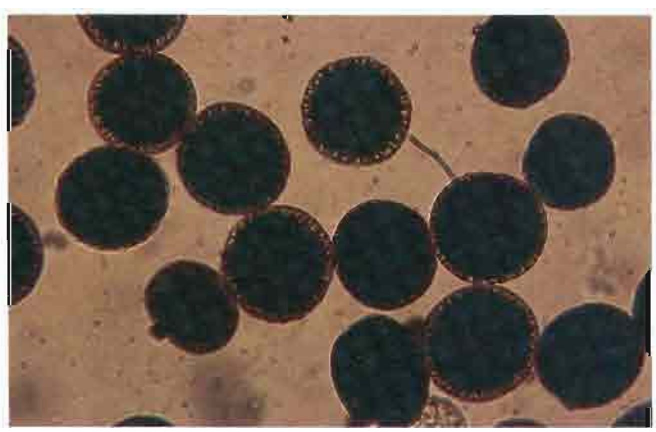


Figure 105. Distinctive black teliospores of *Tilletia indica* (10 x 40).

Flag smut

Urocystis agropyri

Flag smut is primarily a disease of winter wheat in areas with mild winters. It also occurs on spring wheats grown during the winter months in some areas of the Indian Subcontinent. Stalk smut on rye is caused by an organism closely related to *Urocystis agropyri*. Flag smut is easy to identify, but is often overlooked because of the extreme dwarfing of host plants.

Grey to black stripes develop on leaf blades and sheaths. The plants are usually dwarfed, and the leaves twist and split lengthwise after the epidermis breaks open to release the spores (Figure 106). Diseased plants usually fail to head (Figure 107). Spores from diseased plants contaminate the soil and seed. Seedlings of the new crop become infected by sporidia produced by germinating teliospores, either in the soil or on the seed, and the mycelium develops systemically through the diseased plant.

Flag smut sori are filled with masses of spore balls covered with a layer of sterile cells. Each spore ball contains one to four teliospores that are angular to spherical, dark brown, smooth walled, and are 10-20 μm in diameter (Figure 108). Germinating teliospores produce a promycelium with three to four sporidia, which fuse to form the infectious mycelium.

Crop rotation, combined with disease-free seed, can be used effectively to control the disease. Flag smut has been nearly eliminated in Australia through the use of crop rotations and resistant varieties. Systemic fungicides, such as *Carboxin* and *Benomyl*, are effective as seed treatments.

Figure 107. Wheat plants affected by flag smut are distorted, dwarfed, and produce few tillers (photo: R. Metzger).

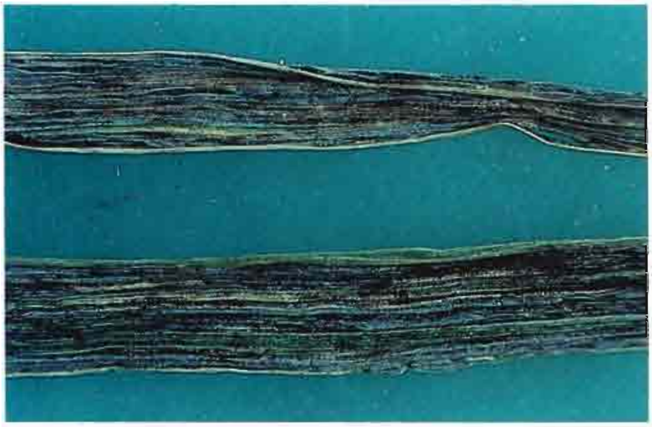


Figure 106. Wheat leaves with flag smut, caused by *Urocystis agropyri*. Note the greyish black stripes.

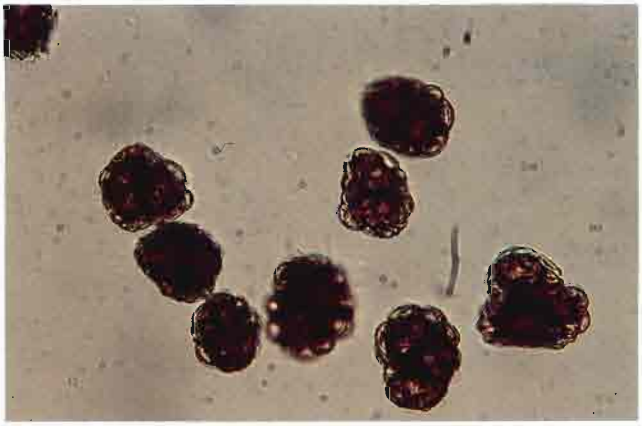


Figure 108. Teliospores of *Urocystis agropyri*. Note the layer of sterile cells surrounding the viable teliospores (10 x 40).

Fusarium diseases

The two most common cereal crop diseases caused by *Fusarium* species are scab and root rot. Most of these *Fusarium* fungi are soil-inhabiting and may be aggressive facultative saprophytes. The root pathogens are difficult to identify without special laboratory techniques and are therefore considered beyond the scope of this guidebook.

Five *Fusarium* species that regularly occur as pathogens on the above-ground parts of small grains are discussed here.* These species develop conidial accumulations on the surface of the affected areas. These appear in various colors, including white, pink, peach, orange, red and tan.

Scab is readily recognized by the appearance of one or more prematurely blighted spikelets after flowering. The infected spikelets develop a bleached straw color, in contrast to the normal green of healthy spikelets (Figure 109—next page). As the crop matures, light pink to orange-colored conidial masses accumulate at the base of the spikelets or along the edges of the glumes and lemmas. The kernels on infected spikelets are usually badly shriveled and nonviable. Diseased grain may contain mycotoxins that are poisonous to humans and animals. The same fungi that cause scab may also cause seedling blight, crown rot, stem and node blight, and grain rot on all small grain cereals.

In early spring, snow mold caused by *F. nivale* invades leaf and crown tissues of fall-sown cereals. The disease can be recognized by the production of white superficial mycelia on the affected areas. *Fusarium nivale* is most destructive at temperatures just a few degrees above freezing. The same species also causes leaf blotch in some subtropical regions.

Fusarium species persist on plant debris in the soil or on untreated seed. Fungicides may protect seedlings against seed-borne infection, but not from inoculum present in the soil or plant refuse. Early infections develop as root rots or seedling blights. Later infections develop as crown rots, often in association with other pathogens, such as *Ophiobolus graminis* and *Helminthosporium sativum.* The occurrence of "white heads" prior to crop maturation may be due to crown rot caused by *Fusarium* species. Spikelet infections result in scab and occur via air-borne conidia during the flowering or grain-filling period.

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* Problems in species identification have occurred as a result of the confused state of nomenclature, and from natural variations in morphological development in response to changes in environment and nutrition. In an effort to bring a measure of order to the classification of *Fusarium*, Snyder and Hansen (1945) proposed that the number of species be reduced to nine. There has been some agreement among mycologists that this was a move in the right direction. However, the concept has created some soul searching among taxonomists regarding the basis for classification of other fungi. For example, differences in morphological features among varieties within the species *Fusarium roseum* are at least as great as the differences that separate species in such genera as *Ustilago* and *Tilletia.*

Dr. C. Booth, at the Commonwealth Mycological Institute, Kew, England, has prepared an excellent review entitled "Genus Fusarium" (1971), which should help researchers who spend considerable time on fusarium diseases. However, few agricultural scientists working with cereal crops have the time or the laboratory equipment to use the techniques suggested. A less complicated system of identification is needed, at least among the *Fusarium* species that infect the above-ground parts of cereal crop plants.

Distinguishing the species

Two types of conidia that differ considerably in size are produced by *Fusarium* species. Small spores, called microconidia, are produced by some species but not others. Microconidia are extremely small and lack features useful in diagnosis *per se*, yet the presence or absence of microconidia can be helpful in species identification. Larger spores, called macroconidia, are produced by all of the species and have morphological features that are useful in species identification.

Five *Fusarium* species regularly occur as pathogens on the above-ground parts of small grain plants. These have distinctive macroconidia and, with practice, it should be possible to identify the species by microscopic examination of fresh macroconidia sporulating on host plant tissue. Infected spikelets, nodes, and leaves usually sporulate adequately on slightly moistened filter paper in petri dishes in 24-48 hours at room temperature.

Distortion of spores occurs with aging, drying, spore germination, or from contamination. Such samples should not be used for identification. When isolates are developing on nutrient media, other diagnostic features, such as growth rate, pigmentation, formation of microconidia and chlamydospores, and the development of perithecia are useful in species identification. The occurrence of these features on host tissue in nature is too erratic for them to serve as reliable guides, but they should not be ignored completely.

Figure 109. Fusarium head scab is easily recognized by the distinctive symptoms that appear after flowering (facing page). Diseased spikelets develop a bleached straw color, in contrast to the normal green of healthy spikelets.

Conidia of *F. graminearum* (10 x 40)

Fusarium graminearum

Macroconidia usually measure between 25-50 µm x 2.5-5.0 µm and have three to seven septa. The main body of the spore is usually straight or slightly curved, and the apical cell curves and narrows sharply to a point. The basal cells are elongated, tapered, and curve slightly. Superficial perithecia often develop on diseased spikes and straw under prolonged periods of warm wet weather during harvest.

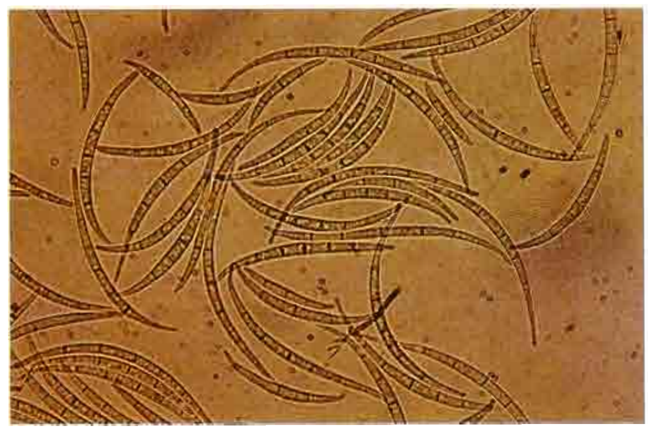
Fusarium culmorum

Macroconidia usually range from 30-60 μm x 4-7 μm in size. They are thick, slightly curved, have three to five distinct septa, pointed apical cells, and well-defined foot cells. Perithecia have not been reported.

Conidia of *F. culmorum* (10 x 40)

Fusarium avenaceum

Macroconidia are long, slender, and curve more or less uniformly throughout their length. They measure 50-80 μm x 2.5-4 μm and have four to seven septa. Perithecia rarely occur in nature.



Conidia of *F. avenaceum* (10 x 40)

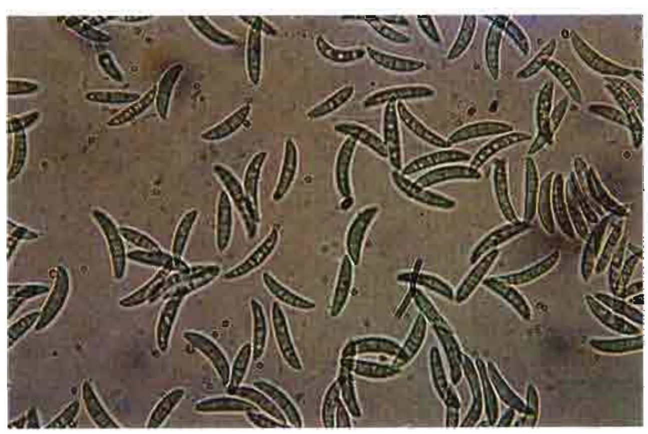
Fusarium equiseti

Macroconidia are more or less intermediate in length and width between *F. culmorum* and *F. avenaceum.* The attenuated apical cell is more strongly curved. The conidia measure 22-60 μm x 3.5-6.0 μm, and have four to seven distinct septa and well-defined foot cells.

Conidia of *F. equiseti* (10 x 40)

Fusarium nivale

This is the most readily identified of the *Fusarium* species that parasitize cereal crops. Conidia are short, curved, taper toward the ends, and foot cells are not well marked. Mature conidia measure 20-28 μm x 2.5-5 μm and usually have three septa. Perithecia develop readily on diseased leaves and leaf sheaths during or after maturity.



Conidia of *F. nivale* (10 x 40)

Scab and root rot

Figure 110. Fruiting structures (conidial masses and aerial mycelium) of head scab on a triticale spikelet, caused by *F. graminearum*.

Figure 111. Conidia of *F. graminearum* (10 x 40).

Figure 112. Perithecia of *Gibberella zeae* (the perfect stage of *Fusarium graminearum*) on a wheat spikelet.

Fusarium graminearum *

Scab, root rot and crown rot diseases are caused by this species world wide. The diseases are especially serious on wheat in China, southern Brazil, Argentina and western Europe. *Fusarium graminearum* apparently favors areas having mild winters and warm humid summers.

Macroconidia are produced in orange to peach colored conidial masses at the base of spikelets, or at the edges of glumes or lemmas (Figure 110). The macroconidia are hyaline, may be straight or slightly curved, and often have a slightly elongated apical cell that curves more abruptly near the tip. They typically measure 25-50 µm x 2.5-5.0 µm, although some collections exceed this range (Figure 111). Microconidia are not produced.

The perfect stage (*Gibberella zeae*) occurs frequently on diseased plants, and probably plays a role in perpetuating the disease from one year to the next. It is the only species of the *F. graminearum* group that produces perithecia regularly in nature. The perithecia develop superficially in clusters on diseased heads of cereal crops (Figure 112). The ascospores are subhyaline to light yellow-brown, fusoid and slightly curved with rounded ends (Figure 113). They resemble the ascospores of *Phaeosphaeria avenaria* (see page 44), but without the enlargement of one of the mid-cells. When mature, the ascospores are three septate and measure 20-25 µm x 3-4 µm.

* Perfect stage: *Gibberella zeae*

Figure 113. Ascospores of *Gibberella zeae* (10 x 40).

Fusarium culmorum

This species is widely distributed, and appears to survive greater extremes of drought and freezing temperatures than *F. graminearum*. Both species cause similar disease symptoms (Figures 114, 115, 116). *Fusarium culmorum* is a serious root rot pathogen in the northwestern USA, western Europe and in some highland areas of the subtropics.

The species produces short, stout macroconidia that are hyaline, uniformly curved, with a pointed apical cell and a distinctive foot cell (shaped like a foot). the conidia are generally three to five septate and measure 30-60 µm x 4-7 µm (Figure 117). Neither microconidia nor perithecia are produced. Chlamydospores develop readily in soil and function as an efficient survival mechanism for this fungus.

Figure 115. Conidial development of head scab on a triticale spikelet, caused by *F. culmorum*. Compare with Figure 110.

Figure 116. Shrivelled, nonviable triticale seed, the result of head scab.

Figure 114. Durum wheat spikes with head scab caused by *F. culmorum*. Note the similarity of disease symptoms with those produced by *F. graminearum* (Figure 109).

Figure 117. Conidia of *F. culmorum* (10 x 40).

Fusarium avenaceum *

This species also commonly causes scab, crown rot, and foot rot diseases. It is widely distributed over a range of temperature environments. *Fusarium avenaceum* is readily identified by its very long and very narrow, bow-shaped macroconidia. It is a frequent scab pathogen on rye (Figure 118) and triticale on the High Plateau of Mexico. Usually, small, light pink spore masses accumulate on the surface of the glumes (Figure 119). The macroconidia are hyaline, uniformly curved, taper gradually at the ends, and typically measure 50-80 μm x 2.5-4.0 μm (Figure 120). The form and size of macroconidia appear to vary considerably. Perithecia are seldom encountered on diseased host plants.

* Perfect stage: *Gibberella avenacea*

Figure 118. Rye spikes infected by *F. avenaceum*.

Figure 119. Conidial development of *F. avenaceum* on a rye glume.

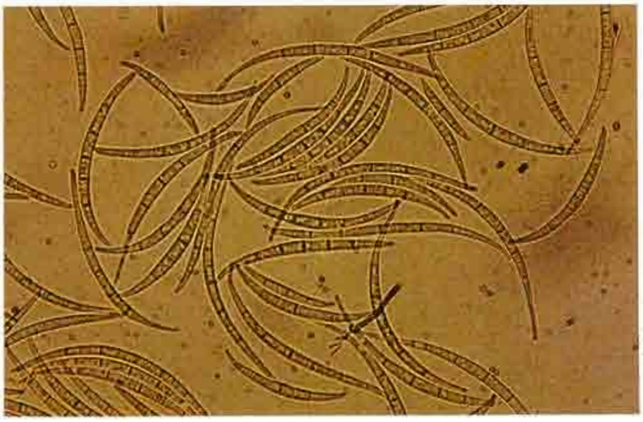


Figure 120. Conidia of *F. avenaceum* (10 x 40).

*Fusarium equiseti**

This species occurs most often as a soil fungus, forming a high proportion of the soil microflora in some areas. However, the species is not considered a serious cereal crop pathogen. Pink spore masses of *F. equiseti* occur frequently on spikes of cereal crops on the High Plateau of Mexico (Figures 121, 122). In this region, *F. equiseti* has also been observed to sporulate on the immature leaves of cereal crop plants. The macroconidia are hyaline, curved, and have elongated apical cells that bend sharply inward. The mature conidia measure 22-60 µm x 3.5-6.0 µm (Figure 123). Many strains have dark conspicuous septa. Microconidia are not produced. The perithecial state has been reported, but it seldom occurs on cereal crops or in culture.

* Perfect stage: *Gibberella intricans*

Figure 122. A closer view of a fruiting structure of *F. equiseti* on a durum wheat spikelet.

Figure 121. Conidial development of *F. equiseti* on an oat leaf.

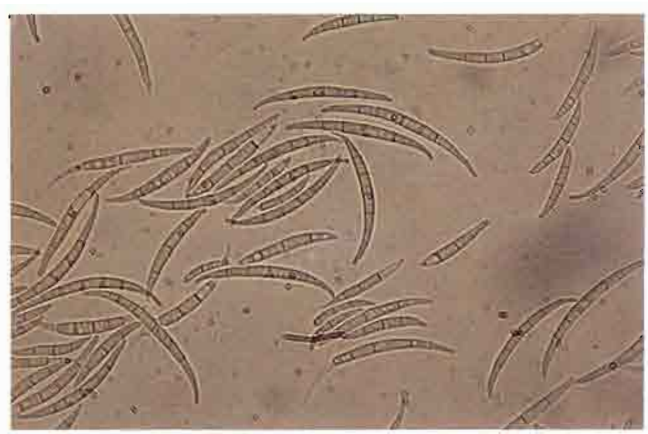


Figure 123. Conidia of *F. equiseti* (10 x 40).

Fusarium leaf blotch and snow mold

*Fusarium nivale**

Fusarium nivale is an important pathogen in cool temperate zones and causes snow mold of winter cereals. It also causes seedling infection and foot rot during cool temperatures. Much of the winter killing of winter cereals can be attributed to infection by *F. nivale*.

In the cooler areas of the subtropics, this organism causes leaf blotches on wheat, triticale and rye (Figure 124). The leaf blotching caused by *F. nivale* is prevalent on the High Plateau of Mexico, in the high valleys of East Africa, and in the Andean region of South America. Under severe attack, complete defoliation may occur. Head blight diseases do occur, but the major crop losses result from poor grain development following the leaf blotch disease. Durum wheat and triticale are generally more severely affected by the disease than wheat or rye. Oats and barley appear to be immune.

Leaf blotch symptoms usually appear at about heading time. Young lesions occur as oval or irregularly shaped, greyish green mottled areas. The lesions enlarge rapidly, develop into greyish brown, oval-shaped blotches with grey centers (Figure 125). The leaf tissue in the older lesions becomes brittle and tends to split or fold.

Fruiting structures of the fungus develop rapidly in young lesions. White to light pink sporodochia develop from the stomata. Under low magnification they appear as rows of light colored or pinkish stitches parallel to the veins of the leaf (Figure 126). As the lesion becomes older, the sporodochia turn yellowish. Numerous hyaline, sickle-shaped conidia are produced on the sporodochia (Figure 127—next page). Mature conidia are uniform in size, measuring 20-28 μm x 2.5-5 μm, with three septa. The apical cell tapers to a sharper point than does the foot cell (Figure 128).

* Perfect stage: *Calonectria nivalis*

Figure 124. Early symptoms of fusarium leaf blotch on wheat, caused by *F. nivale*.

Figure 125. The lesions of fusarium leaf blotch cause leaves to become distorted or frayed.

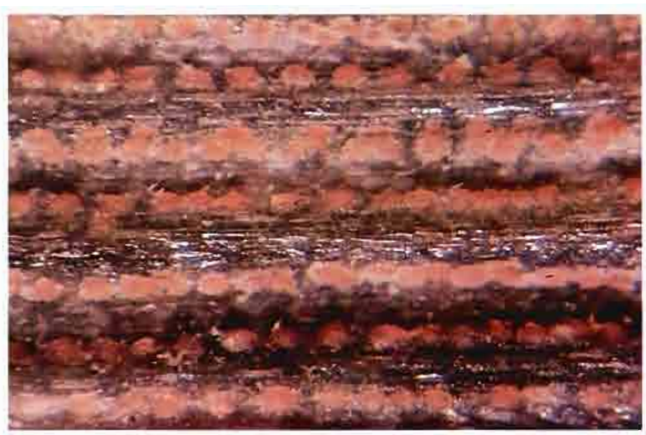


Figure 126. Pinkish-colored sporodochia of *F. nivale* develop in rows parallel to the veins of leaves.

The perfect stage (*Calonectria nivalis*) develops readily in the lesions of diseased leaves when plants are maturing, but mature ascospores are not released until some weathering has occurred (Figure 129). The perithecia are embedded in the leaf tissue. A pore through which ascospores are released protrudes through the epidermis (Figure 130). At first glance, the perithecia look like pycnidia of the septoria complex, but can be distinguished by the asci and ascospores they contain, rather than conidia. The ascospores are small, hyaline, elliptical, irregularly curved, have one to three septa, and measure 10-18 μm x 3.5 μm (Figure 131).

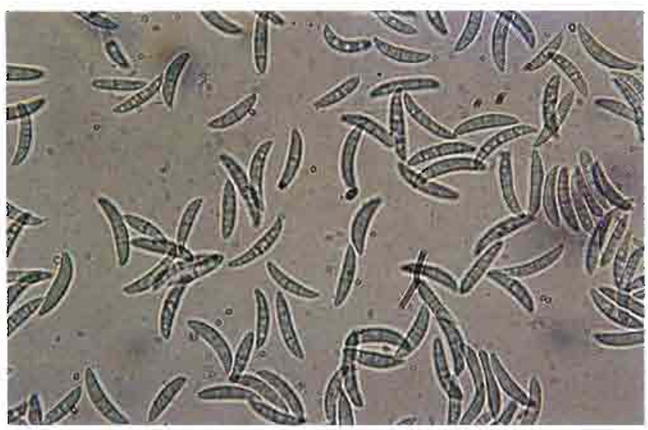


Figure 128. Conidia of *F. nivale* (10 x 40).

Figure 127. A closer look at a single sporodocium of *F. nivale*. Note the production of conidia (10 x 40).

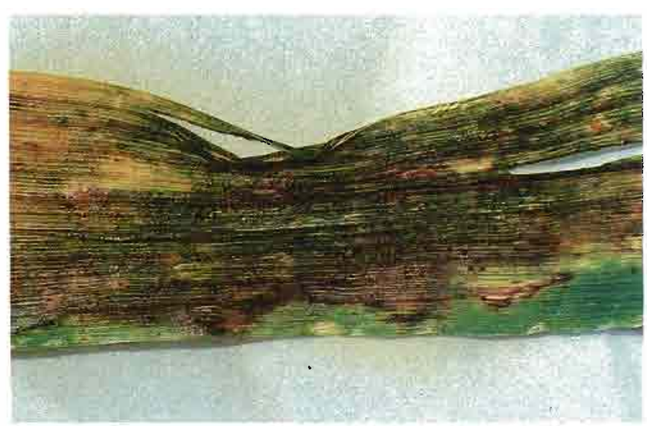


Figure 129. Perithecia of *Calonectria nivalis* (the perfect stage of *F. nivale*) developing on a triticale leaf.

Snow mold is the more common disease caused by *F. nivale*, and occurs on winter cereals in the winter wheat areas of the USA and Europe. Plants affected by snow mold become chlorotic, with pinkish-colored mycelium, sporodochia, and visible spore accumulations on the necrotic leaves. Crops are most likely to be damaged under prolonged snow cover (Figure 132). Snow mold has not been observed on winter cereals on the High Plateau of Mexico, although night temperatures below 0°C occur regularly from November to February. The leaf blotching form of the disease develops only in midsummer.

Researchers are generally more familiar with snow mold than with the leaf blotching caused by *F. nivale*. Some success in the control of snow mold has been achieved by using crop rotations that include more resistant varieties. Little research has been done on the control of the leaf blotching form. CIMMYT researchers have found that *Benomyl*, used as a foliar spray at two week intervals starting at the heading stage, kept the disease in check on cereals in the Toluca nursery in Mexico. Some progress has been achieved in developing strains of wheat, triticale and durums with resistance to fusarium leaf blotch.

Figure 130. A closer view of the perithecia of *C. nivalis*. Note that they are embedded in the leaf tissue.

Figure 131. Asci containing ascospores of *C. nivalis*, being released from a perithecium (10 x 40).

Figure 132. Winter wheat suffering from snow mold caused by *F. nivale* (photo: G.W. Bruehl).

Miscellaneous root and crown diseases

The pathogens described in this section are all soil-borne fungi. Initial infection usually occurs on the roots or at the base of the plant. Some of the fungi, such as *Ophiobolus graminis* (Figure 133), *Pseudocercosporella herpotrichoides* and *Sclerotium rolfsii*, produce lesions at or near the soil surface. Diseased plants usually wilt or develop general necrosis of the basal portions without the secondary spread of disease to other parts of the plant.

Colletotricum graminicola usually causes early lesions near the plant base, and often secondary lesions on leaf blades and sheaths. *Cephalosporium gramineum* and *Sclerophthora macrospora* invade the plant through roots or crowns and subsequently become systemic. The symptoms become conspicuous on leaf blades and sheaths, usually accompanied by dwarfing or distortion. All of the fungi in this group persist on debris of the host plants.

Figure 133. Foot rot symptoms of take-all on wheat (facing page), caused by *Ophiobolus graminis*.

Distinguishing the fungi

Conidia are less useful as diagnostic features to identify the fungi in this group. However, when possible they are included with the description of other symptoms.

Sporodochia of *Cephalosporium gramineum*

Cephalosporium gramineum (cephalosporium stripe)

This is a disease of winter cereals in temperate climates. One or two pale green to white stripes develop the full length of leaf blades and sheaths. Their color darkens to reddish brown as the stripes age. Unicellular slime spores are produced on sporodochia on the overwintered straw and stubble of diseased plants. Conidia are hyaline, oval shaped, nonseptate, and small, measuring 4-7 µm x 2-3 µm.

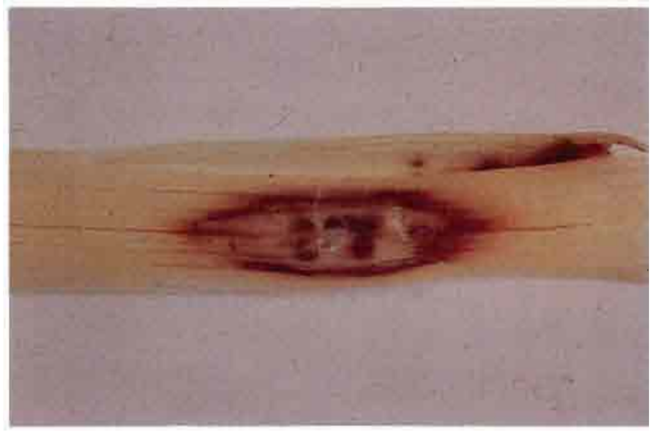
Ophiobolus graminis (take-all)

Dark brown to black dry rot occurs on roots and the bases of stems. Mats of mycelial strands develop on the surface of stems and leaf sheaths near the base of the plant. Diseased plants may develop "white heads" prior to maturity. Mature perithecia release long, narrow, hyaline and septate ascospores, measuring 70-90 μm x 2.5-3.5 μm.

Dark mycelial strands and perithecial initials of *Ophiobolus graminis*

Pseudocercosporella herpotrichoides (eyespot)

This disease usually causes lodging and premature senescence. Elliptic eyespot-type lesions develop on stems close to the soil surface. The lesions are a conspicuous light grey to tan with brown margins. Sporulation occurs on diseased stubble in the early spring. The conidia are hyaline, curved, several septate, and measure 30-60 μm x 1.5-3.5 μm.

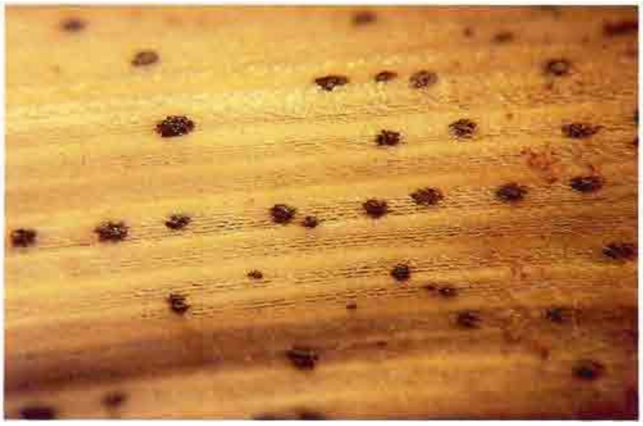


Eyespot lesion typical of those produced by *Pseudocercosporella herpotrichoides* (photo: B. Fitt)

Sclerotium rolfsii (sclerotium wilt or southern blight)

Masses of white mycelia develop on wilting or dead plants just below the soil line. Spherical sclerotia, white at first and gradually turning with age to a dark greyish brown, form on the roots and stem bases.

Spherical sclerotia and white mycelia typical of *Sclerotium rolfsii*



Fruiting structures typical of those produced by *Colletotrichum graminicola*

Colletotrichum graminicola (anthracnose)

Fruiting structures provide the most reliable diagnostic feature of this fungus. Small, black, oval to elongated fruiting structures (acervuli) develop on the stems and leaf sheaths, usually near the soil line. The fruiting structures are raised above the epidermis, and have numerous black setae (spines) protruding above the conidial layer. The conidia are sickle shaped, single celled, hyaline to light grey, and measure 20-30 μm x 4-6 μm.

Symptoms typical of those caused by *Sclerophthora macrospora*

Sclerophthora macrospora (downy mildew)

Diseased plants are stunted, yellowed and have thickened, distorted leaves. If heading occurs, the floral bracts are fleshy, elongated, and leaflike. No viable grain is produced. Oospores can be found in the dead tissue of lower leaves. They are spherical, smooth walled and 36-60 μm in diameter. The disease is most prevalent in water-logged areas.

Cephalosporium stripe

Cephalosporium gramineum

This is a common disease of fall-sown cereals. Wheat is the most common host, although oats, barley, triticale and rye are susceptible. Cephalosporium stripe occurs in the midwestern and northwestern USA, in western Europe and eastern Asia. The disease is rarely serious on spring-sown cereal crops.

Diseased plants can occur singly or in patches. The symptoms may appear at any growth stage, but are more conspicuous as the plants approach heading. One or more white to pale green stripes with diffuse edges develop along the full length of the leaf blades and sheaths. The stripes darken and turn reddish brown (Figure 134). The remainder of the leaves soon turn chlorotic or yellow. As plants approach maturity, the stem areas below the nodes become dark. Plants that are infected early are dwarfed and sterile.

The fungus *C. gramineum* is a soil-inhabiting microorganism. Sporulation occurs on the senescent stems and leaves of diseased plants. Unicellular conidia develop in yellow to straw colored slime masses from sporodochia produced on moist straw (Figure 135). These conidia are hyaline, oval shaped, nonseptate and quite small, measuring 4-7 μm x 2-3 μm (Figure 136).

The fungus persists on plant debris. Inoculum tends to build up in soils that are continuously planted to cereal crops and is becoming more prevalent where minimum tillage is practiced. Native grasses help perpetuate the fungus. The inoculum may be disseminated on seed. Following initial root infection, the disease develops systemically. Some wheat varieties display a considerable degree of tolerance to the disease. Rotations with crops other than cereals, as well as deep plowing of diseased stubble, are the recommended cultural practices to reduce the incidence of the disease.

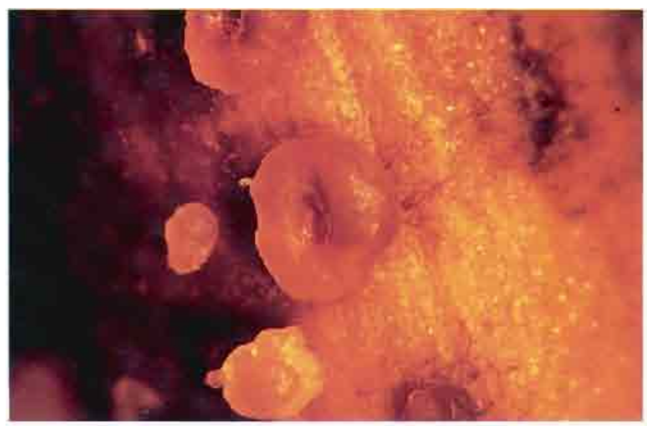


Figure 135. Sporodochia of *C. gramineum* on weathered wheat straw (photo: W.W. Bockus).

Figure 134. Leaf-striping symptoms of cephalosporium stripe on winter wheat, caused by *C. gramineum* (photo: W.W. Bockus).

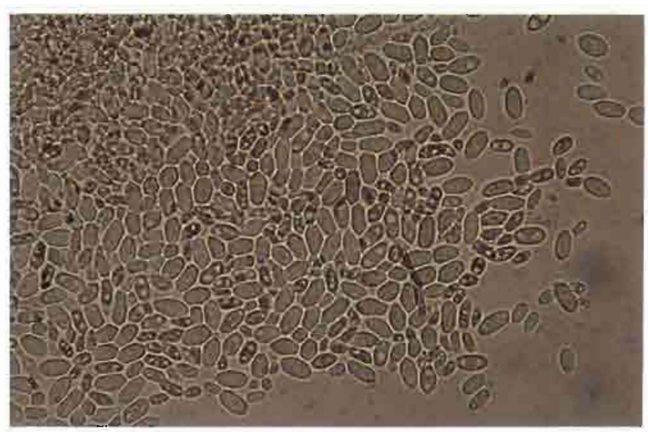


Figure 136. Conidia of *C. gramineum* (10 x 40).

Take-all

*Ophiobolus graminis**

Take-all is a root and foot rot disease of cereals and grasses. The disease is usually more destructive on winter crops, but spring-sown crops may be heavily damaged. Although infection can occur from the seedling stage onward, the disease is most often observed soon after heading. The foliage turns to pale green, and the heads become bleached and ripen prematurely (Figure 137). The heads are sterile or develop shriveled seed (Figure 138). When soil moisture is limited, the plants are more severely stunted and tillering is reduced. Diseased plants have dark brown to black dry rot or decay on roots and the basal portions of stems (Figure 139). Mats of dark mycelial strands develop superficially under the lowest leaf sheath. Stems are weakened, plants are prone to lodging, and they break easily at or near the crowns when pulled.

* Renamed: *Gaeumannomyces graminis*

Figure 137. A patch of spring wheat displaying the symptoms of take-all, caused by *O. graminis*. Note the bleached, prematurely ripe heads.

Figure 138. Wheat spikes and grain from plants infected by *O. graminis* at different growth stages. A disease-free spike on the right.

Figure 139. Lower culms and roots decayed by *O. graminis*.

In recent years, several closely related fungi have been implicated as causes of take-all. Although the fungi have remained the same, their names have been changed, resulting in some confusion. The take-all pathogen, originally identified as *Ophiobolus graminis*, develops perithecial initials among the mats of dark mycelia usually found at the base of the stem under the leaf sheath (Figure 140). Under favorable conditions, the perithecia develop and produce mature ascospores (Figure 141). The perithecia are spherical to oblong, black in color, and frequently develop a beak. The ascospores are narrow, threadlike, hyaline, septate, measure about 70-90 μm x 2.5-3.5 μm, and taper toward the ends (Figure 142). The perithecia and ascospores are excellent diagnostic features but, because they often fail to develop, the mats of dark mycelial strands that develop superficially on diseased stems serve as the more consistent distinguishing feature of the fungus.

The fungus survives as mycelia and perithecia on plant debris. Infection occurs by direct mycelial penetration of root and stem tissue. The density of inoculum in the soil is influenced by the soil environment and the presence of antagonistic microorganisms. Crop rotations that include legumes or other resistant crops reduce the amount of inoculum in the soil.

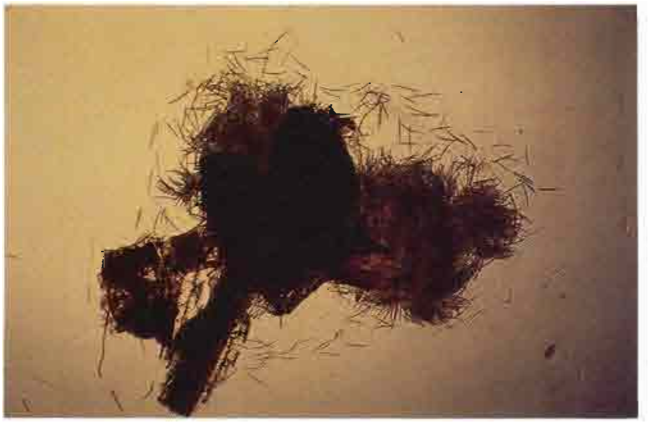


Figure 141. A mature perithecium of *O. graminis* releasing ascospores (10 x 4).

Figure 140. Dark mycelial strands and perithecial initials of *O. graminis*, developing on the basal internode of a wheat stem.

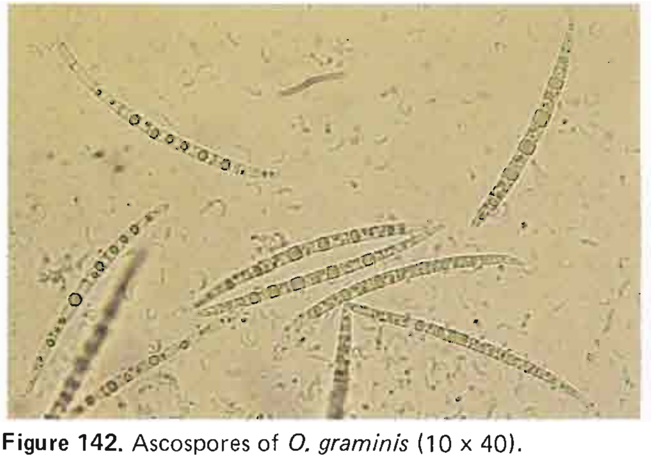


Figure 142. Ascospores of *O. graminis* (10 x 40).

Eyespot

Pseudocercosporella herpotrichoides

Eyespot is generally assumed to be a disease of winter wheat, but spring cereals in many regions of the world can be severely damaged. Usually restricted to the basal portions of the plant, symptoms seldom appear on roots or extend more than six to eight inches above the soil line (Figure 143). All of the cereals and many grasses are hosts of this fungus. Cool wet weather favors disease development.

The disease symptoms become more obvious as the crop approaches maturity. Elliptical or eyespot lesions develop on the lowest leaf sheaths and adjacent internodes just above the soil line (Figure 144). Young lesions have brown elongated borders with straw-colored centers. Eventually the whole stem base may be encircled. The center area of lesions becomes darker with age (Figure 145). Severe attacks are usually accompanied by lodging.

Sporulation occurs most frequently during the cool moist weather of early spring, and it may cease entirely during the summer. Spore production is usually difficult to induce in mature stems of diseased plants. Unbranched, short, erect conidiophores develop from the dark stromalike mycelium. The conidia taper toward the apex, are hyaline, straight or slightly curved, measure 30-60 μm x 1.5-3.5 μm and have three to seven septa.

The fungus persists as mycelia on crop residue. Infection may occur from conidia produced on diseased straw (or from active mycelia) invading the lower leaf sheaths. The disease develops inward and laterally, eventually producing cottony mycelia in the hollow center of the stem. Conidia produced on diseased plants during the early growth stages serve as a secondary source of inoculum. Suggested control measures include delayed fall seeding and rotations with crops other than cereals, thus reducing the amount of inoculum in the soil.

Figure 144. Eyespot lesions occur at or near the soil line on the base of stems (photo: B. Fitt).

Figure 143. Advanced symptoms of eyespot on triticale stems.

Figure 145. A typical eyespot lesion caused by *Pseudocercosporella herpotrichoides* (photo: B. Fitt).

Sclerotium wilt (southern blight)

Sclerotium rolfsii

Sclerotium wilt is a widely distributed, warm climate disease with a very broad host range. Among its hosts are grain legumes, numerous fruit and vegetable crops, cereals, ornamentals and weeds.

The first symptoms of the disease are the yellowing and wilting of the plants. This may happen to scattered individual plants or in small patches (Figure 146). Infection may occur at any stage from tillering to maturity. Most of the diagnostic symptoms occur within a few inches of the soil surface. Masses of white mycelia develop on the roots, stems and leaf sheaths at, or just below, the soil line (Figure 147). Fruiting bodies (sclerotia) develop after the plants begin to wilt. The sclerotia develop as small white spherical structures on the mycelia (Figure 148). As they grow in size, the color changes from white to tan, then to light brown, and finally to medium brown (Figure 149). The fruiting bodies may develop in the soil, on the roots or on the lower portion of the plant (Figure 150).

Figure 146. A barley plant showing symptoms of sclerotium wilt, caused by *S. rolfsii* (photo: H. Vivar).

Figure 147. Young sclerotia and white mycelia of *S. rolfsii*.

Figure 148. A closer view of young sclerotia caused by *S. rolfsii*. Note their white, spherical, smooth appearance.

The fungus is identified most readily by the masses of white mycelia and the white to brown sclerotia at the bases of the wilted plants. The basidial state, *Corticum rolfsii*, is occasionally observed on dead plant tissue, but is probably of minor importance in the transmission of the disease and survival of the fungus.

The fungus is a highly efficient facultative parasite. It may continue to grow extensively on dying plant tissue near the soil surface. It persists as sclerotia or mycelia in plant debris in the soil. The fungus prefers acidic soils. *Sclerotium rolfsii* may be very difficult to eradicate after it becomes established because of its wide host range, its capability to develop saprophytically, and its longevity via sclerotia and mycelia in the soil. Control measures include removal of all plant debris, deep plowing, complete weed control and liming if soil is acidic.

Figure 149. Sclerotia of *S. rolfsii* turn brown with age.

Figure 150. Sclerotia of *S. rolfsii* are sometimes produced on the lower portion of the stem, near the soil line.

Anthracnose

Colletotrichum graminicola

Anthracnose can occur on all small grains, maize, sorghum and many grasses. Symptoms usually appear on the lower portions of the plant, but may also develop on the upper leaves and heads. Although the disease is relatively common, it seldom causes serious losses in cereal crops.

During the early stages, the symptoms are obscure. Diseased plants develop a bleached appearance, ripen prematurely, and are susceptible to lodging. They may be easily confused with plants having other diseases of the foot rot complex. The fruiting structures that develop as the plants approach maturity serve as the most reliable diagnostic feature. Small, black, oval to elongated fruiting structures (acervuli) develop on the surface of stems and leaf sheaths (Figure 151). These acervuli have a raised appearance due to the presence of numerous black setae (spines) that protrude above the surface of the plant tissue (Figures 152, 153). The conidia that develop within the acervulus among the setae are sickle shaped, single celled, hyaline to light grey, and measure 20-30 μm x 4-6 μm.

The fungus persists on host residue as conidia or mycelium. Infection usually occurs at the base of the plant or on the roots from soil-borne inoculum. Fruiting structures that appear on the upper leaves and heads develop from the secondary spread of conidia. Wild grasses may serve as sources of primary and secondary inoculum.

Relatively little research has been devoted to this disease, probably because it never occurs on an epidemic scale. However, it occurs over a broad range of environments. The disease may be more prevalent in soils low in phosphorus and potash. Crop rotations that include legume crops may be useful as a control measure in areas where the disease occurs regularly.

Figure 152. A closer view of the acervuli of *C. graminicola.* Note the raised appearance, which is due to the presence of spines (satae) that protrude above the surface of the plant tissue.

Figure 151. Black fruiting structures (acervuli) on the surface of stems and leaf sheaths are symptomatic of anthracnose, caused by *Colletotrichum graminicola.*

Figure 153. A closer view of the long, dark spines that surround the acervuli of *C. graminicola* (10 x 10).

Downy mildew

Sclerophthora macrospora

Downy mildew of cereals is usually associated with water-logged or irrigated soils. The fungus, *Sclerophthora macrospora,* has a broad host range, including small grain cereals, maize, sorghum and many grasses. Disease symptoms most frequently appear during the early growth stages of host plants as leaf yellowing (Figure 154), severe stunting and excessive tillering (Figure 155). Many severely diseased plants die during the tillering to early elongation growth stages. Diseased plants may develop thickened and twisted leaves, and usually fail to head. Where heading occurs, the floral bracts are fleshy, distorted and elongated (Figure 156). These plants rarely produce viable grain.

The downy appearance, due to the development of conidiophores and conidia, occurs on many crops, but is seldom observed on small grains. Oospores can be found readily in the mesophyll tissues of lower leaves and leaf sheaths of diseased plants. The oospores are spherical, smooth walled and measure 36-60 μm in diameter (Figure 157). The oospores persist in diseased plant residue in dry soil. These oospores germinate in water or wet soil to produce sporangia, from which zoospores are released. Infection takes place by water-borne zoospores, and seedlings appear to be more susceptible than adult plants.

Figure 154. Downy mildew developing on spring wheat being grown on poorly drained soil.

Figure 156. Triticale infected by *Sclerophthora macrospora* exhibits deformed stems, leaves and spikes.

Figure 155. Wheat plants with downy mildew are dwarfed and chlorotic (center, right); a healthy plant is on the left.

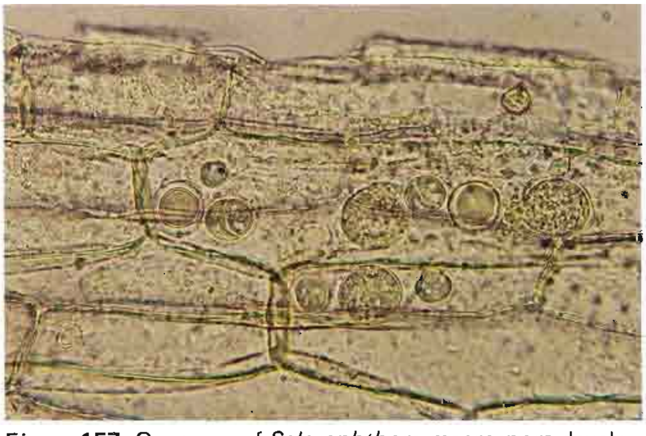


Figure 157. Oospores of *Sclerophthora macrospora* developing in diseased leaf sheath tissue (10 x 40).

Miscellaneous leaf and head diseases

The leaf pathogens in this group cause conspicuous disease symptoms. They produce superficial fruiting structures on leaves, and occasionally on the heads. Their reproduction activity is generally restricted to living plants, although the sexual cycle may occur on dead plant tissue. *Claviceps purpurea*, which causes ergot, is strictly a head pathogen (Figure 158).

Figure 158. Symptoms of ergot: honeydew and sclerotia formation on a barley spike (facing page) infected by *Claviceps purpurea* (photo: V. Pederson).

Distinguishing the fungi

Hyphae of *Erysiphe graminis* on rye

Erysiphe graminis (powdery mildew)

Fruiting structures develop as white to light grey patches of superficial hyphae and conidia on the upper surface of leaves. The undersides of these leaves turn a discolored pale green to yellow below the affected areas. The conidia are oval, hyaline, single celled, and about 20-35 µm x 8-10 µm in size. The mycelium can be scraped off the leaf surface. As the fruiting structures age, they darken to a yellowish grey color. The older leaves develop black spherical fruiting structures (cleistothecia) within the mycelial mats.

Rhynchosporium secalis (scald)

Early lesions are a pale bluish grey, and with time enlarge to irregularly shaped blotches with dark brown borders. Conidia are produced in a thin moist layer on the surface of fertile stroma. The conidia are hyaline, single septate, vary in shape, usually have a hook or beak, and measure 16-20 μm x 3-5 μm.

Older lesions produced by *Rhynchosporium secalis* on a barley leaf

Cercospora apii (cercospora spot)

Lesions appear as dark brown, rectangular to elongated spots on leaf blades and sheaths. Clusters of conspicuous, dark brown, septate conidiophores produce thin, hyaline, tapered and septate conidia. The conidia range from 50-200 μm in length, and taper from 3-5 μm wide at the base to 1 μm at the tip.

A lesion typical of those produced by *Cercospora apii*

Claviceps purpurea (ergot)

This fungus produces dark purplish grey, hornlike sclerotia that replace the seed in diseased florets. Infection occurs at flowering time, and the infected ovaries exude droplets of sticky slime (honeydew) prior to sclerotia formation. Conidia are nonseptate, cylindrical with rounded ends, hyaline, and measure 6-9 μm x 5-6 μm.

A wheat head with the sclerotia produced by *Claviceps purpurea*

Powdery mildew

Erysiphe graminis

Powdery mildew is a common disease of cereals and some grasses, particularly in humid areas. Economic losses are greater on barley than other cereals. *Erysiphe graminis* is an obligate parasite.

Early symptoms appear as colonies of fluffy superficial mycelium, white to light grey in color, on the upper surfaces of the leaf blades (Figure 159). Under favorable conditions, the leaf sheaths and spikes may be affected. The mycelial "fruiting bodies" darken to a yellowish grey with age (Figure 160). The undersides of affected leaves have yellowish necrotic spots at the infection sites. Dry powdery conidia are produced in abundance from conidiophores among the mycelium. Late in the season, black spherical fruiting structures develop in the mycelial mats.

The mycelium develops superficially, except for haustoria that form in the epidermal cells. The conidia are ovoid, hyaline, single celled and measure 20-35 μm x 8-10 μm (Figure 161).

Figure 159. Powdery mildew on wheat, caused by *Erysiphe graminis*. Note the white superficial hyphae (photo: C. Dowswell).

Figure 160. The superficial mycelium and fruiting structures of powdery mildew turn yellowish grey with age.

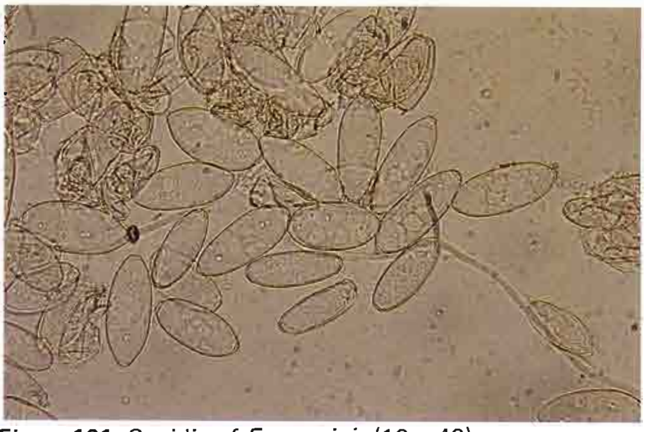


Figure 161. Conidia of *E. graminis* (10 x 40).

The sexual fruiting structures (cleistothecia) are black, spherical, measure about 150-300 μm in diameter and have a very tough outer coating (Figure 162). They usually require considerable weathering or overwintering to produce mature ascospores. Cleistothecia that develop on growing plants usually contain immature asci (Figure 163).

Erysiphe graminis has acquired a high degree of specialization. Each cereal crop apparently has its own special form. Triticale appears to be highly resistant to the wheat and rye forms of the fungus. Further specialization exists in the form of physiologic races. Reaction to the fungus varies with varieties: chlorosis and browning, accompanied by dense mycelial formation and sporulation, occurs on the more susceptible varieties, while light flecking without mycelial development occurs on the more resistant varieties.

Cleistothecia develop readily on diseased plant tissue, but mature ascospores are rarely observed. The importance of ascospores as primary innoculum is uncertain. Mycelia that survive on plant debris and on overwintering plants are probably most important in the perpetuation of the disease.

The use of resistant varieties is the most effective means of controlling the disease. However, fungicides are used as foliar sprays in some parts of Europe.

Figure 162. Cleistothecia of *E. graminis* appear as black dots in the mycelium on this barley leaf (photo: M. Moore).

Figure 163. Immature asci are released from the cleistothecia of *E. graminis*.

Scald

Rhynchosporium secalis

Scald is primarily a foliar disease of barley and rye, though it also affects several common grasses, which may serve as reservoirs for primary inoculum. The disease is reported to be most serious on barley in the cool moist areas of the temperate zone, but it is also destructive in the highland tropics. Scald is usually restricted to the leaves, but under severe conditions may also affect the glumes and awns of barley.

Scald is readily identified from its leaf symptoms. Early symptoms appear as oval, elongated or elliptical lesions, having bluish grey centers with dark brown to reddish brown borders (Figure 164). With time, the lesions enlarge and merge, producing patterns of various shapes and sizes. The centers of the lesions progress through several color changes, from bluish grey (Figure 165) through light grey to straw brown (Figure 166).

Sporulation is favored by cool moist weather. Spore production diminishes, or may cease entirely, during hot dry weather, or as the plants approach maturity. Scald lesions provide suitable sites for facultative or saprophytic organisms to develop. The conidia develop in a thin slime layer on the surface of the lesion from a mat of spore-producing mycelia. They appear as a wet, compact layer of colorless yeast cells. The conidia are hyaline with a single septum, and vary considerably in shape and size. Most spores have a hook or beak, and measure 16-20 μm x 3-5 μm (Figure 167).

The fungus survives as mycelia on plant debris and on diseased cereals and grasses. Spore production begins early in the season during cool wet weather. Primary and secondary infections occur from conidia. Control is achieved with resistant varieties and by rotations involving crops other than barley or rye.

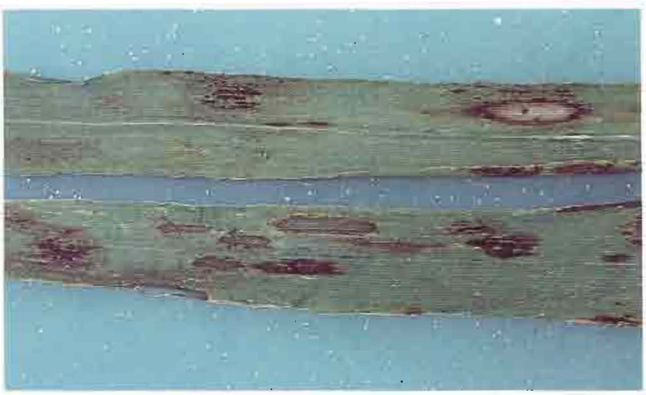


Figure 164. Early leaf symptoms of scald on barley, caused by *Rhyncosporium secalis*.

Figure 166. Scald lesions merge and become more brown with age.

Figure 165. An early scald lesion (photo: S. Fuentes).

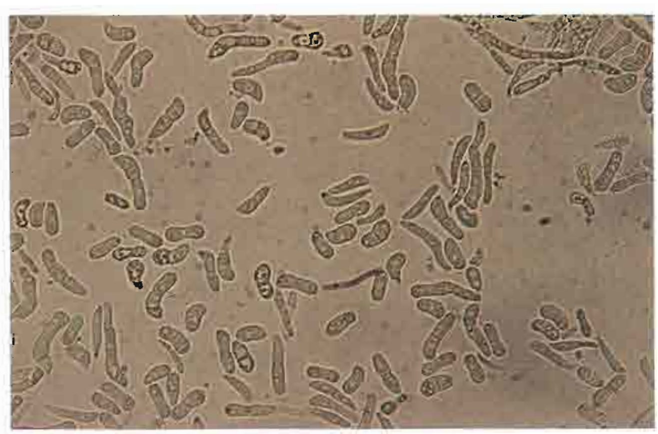


Figure 167. Conidia of *Rhyncosporium secalis* (10 x 40).

Cercospora spot

Cercospora apii

Leaf spots caused by *Cercospora apii* are common on a wide range of herbaceous plants, but seldom cause serious economic losses on cereal crops. The fungus usually behaves as a weak parasite, but occasionally causes leaf spot disease on small grain cereals when conditions are favorable. Cercospora spot is more prevalent on wheat and triticale under warm humid conditions.

The disease develops as small brown rectangular to elongated necrotic spots with darkened areas in the center of the lesions (Figure 168). The fruiting structures develop superficially as clusters of dark conidiophores, usually emerging through stomata from mycelia embedded in the leaf tissue (Figure 169). The conidiophores are usually unbranched and slightly swollen at the base. The basal portions of conidiophores are darker than the upper portions. They measure about 30-70 µm long, 3-4 µm thick at the apex, and 5-9 µm at the base (Figure 170). The conidia are clavate, straight or slightly curved, 3-5 µm thick near the base, and gradually taper to about 1 µm at the apex. They are smooth, hyaline, 9-17 septate, and vary from 50 µm to 200 µm in length (Figure 171). Research on varieties resistant to Cercospora spot has not been reported.

Figure 168. A dark elongated cercospora spot lesion on wheat, caused by *Cercospora apii*.

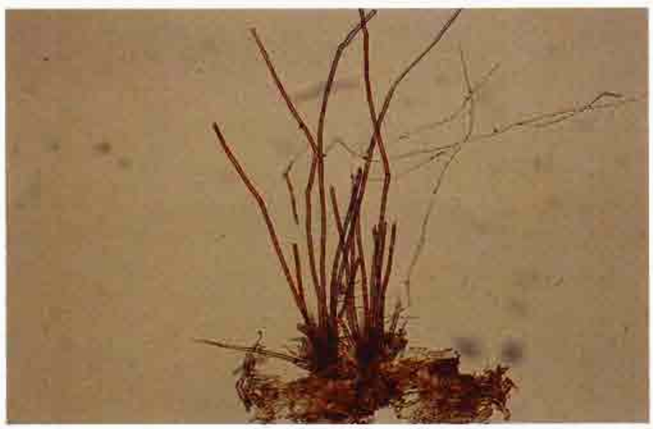


Figure 170. A closer view of a fruiting structure of *Cercospora apii* (10 x 10).

Figure 169. Clusters of dark conidiophores give the lesions produced by *Cercospora apii* a dark, bushy appearance (50 x).

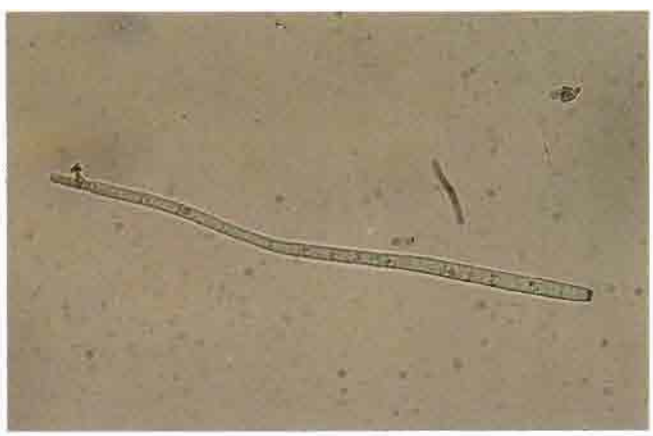


Figure 171. A conidium of *Cercospora apii*.

Ergot

Claviceps purpurea

Ergot is usually considered a disease of rye, but it also occurs on wheat, triticale, barley, oats, millet and many grasses. The fungus attacks only the seed-producing organs, and usually the yield losses caused by the disease are less serious than the losses from discounted grain quality. The ergot bodies that replace the kernels are toxic to humans and animals, and they are difficult to remove from harvested grain without special equipment.

Ergot is readily identifiable. The first symptoms appear during flowering as yellowish droplets of sticky exudate on the infected florets (the honeydew stage—Figure 172). As plants approach maturity, some of the kernels are replaced by purplish black sclerotia that protrude from the spikelets (Figure 173). The dark ergot sclerotia can be detected in the threshed grain as well.

The fungus overwinters on the soil surface or as sclerotia in plant debris. In the spring and early summer, small stalklike stromata grow out of the sclerotia (Figure 174). Perithecia buried in the mushroom-shaped heads of the stromata release ascospores that may infect the stigmas and ovaries of small grains and grasses. Within a few days, honeydew that contains conidia is exuded from infected ovaries. The honeydew is disseminated among the flowers by insects, and the conidia cause floret infection similar to the ascospores. Mature conidia are nonseptate, hyaline, cylindrical with rounded ends, and measure 6-9 μm x 5-6 μm. As the flowering stage passes, the production of honeydew ceases and infected ovaries form fungal sclerotia instead of grain. By the time the plant has matured, the sclerotia have developed into conspicuous dark, hornlike structures, two or three times as large as normal kernels.

Ergot is more prevalent in cool, temperate climates. The prevalence of the disease may be related to the susceptibility of native grasses in these areas. The level of incidence among varieties usually depends more on flowering habits and floral structures than on genetic resistance to the fungus. Crops in which the florets remain open for extended periods during the flowering stage are more likely to become infected. The incidence of disease may be reduced by clipping the grasses along the roadways and borders of the fields.

Figure 172. Honeydew being exuded from the florets of a barley spike infected with *Claviceps purpurea* (photo: V. Pederson).

Figure 173. Ergot bodies (sclerotia) of *Claviceps purpurea*. (photo: V. Pederson).

Figure 174. Stromata developing from an ergot sclerotium after weathering (photo: M. Moore).

Saprophytic and weakly pathogenic fungi

Often referred to as field fungi, these organisms are aggressive spore producers. They are widely distributed, reproducing on diseased, dead, or ripe plant material, especially during periods of high humidity. Field fungi often occur on plants affected by other diseases or stresses, such as root rots, leaf blights, lodging, aphid attacks, and bacterial or viral diseases. The failure of field fungi to appear on individual plants in a plot or nursery where a particular disease is prevalent indicates that either these plants escaped the primary pathogen or were resistant.

The more common genera, such as *Alternaria, Stemphylium, Epicoccum, Cladosporium* and *Torula* invade leaves, stems and heads of ripening or severely stressed cereal crop plants. The grey or black discoloration on heads and seed due to such fungal infections is referred to as sooty molds, black point, or smudge (Figure 175). The development of mycelia and discoloration of grain (black point) may reduce yield and grain quality.

Some weak pathogens are prevalent on grasses and weeds, but may infect cereal crops if conditions are suitable. Among this group are *Phoma, Pleospora* and *Cercosporidium* species. Their occurrence on cereals is usually as a secondary infection but they may also behave as primary pathogens by producing spots or blotches on leaves and floral bracts. Economic losses due to these fungi are usually insignificant, but they complicate disease identification by occurring with the primary pathogens in the same lesions.

Saprophytic fungi may alter the appearance of invaded tissue, but seldom produce distinctive symptoms. They are identified by the morphology of their fruiting structures, particularly the spores.

Figure 175. Typical sooty mold on wheat spikes (facing page), caused by *Torula* species.

Alternaria species

Figure 176. Leaf blight on wheat, caused by *Alternaria triticina.*

A large number of *Alternaria* species have become established throughout the world. Most species exist as saprophytes or as pathogens of crops other than cereals. Some species of *Alternaria,* such as *A. triticina,* cause severe blights on the leaves (Figure 176) and spikes of wheat and triticale, but they do not infect barley or oats. These diseases were first reported in India, and have recently occurred on wheat in western Asia and North Africa.

The fungal colonies are dark grey to black in color, and develop from immersed or partially superficial mycelia. The conidiophores are dark to olivaceous brown, arise singly or occasionally in small groups, may be simple or branched and are usually 3-6 µm thick (Figure 177). The conidia of *Alternaria* species have unique morphological characteristics by which this genus can be readily identified. However, similarities among species within the genus make species identification very difficult. The conidia of most of the saprophytic *Alternaria* species that occur on cereals develop in chains, are ovoid or ellipsoidal, and often taper to a beak at one end. They are medium to dark brown in color. They have smooth or slightly roughened walls, several transverse and longitudinal or oblique septa, and measure 20-90 µm x 8-20 µm (Figure 178).

The species that cause leaf spots develop singly or in chains, have somewhat larger conidia, up to 100 µm long and 30 µm wide, but are very similar in other characterisitcs (Figure 179). They can be distinguished from the other saprophytic forms by their ability to parasitize wheat.

Figure 178. Conidia of (wild) saprophytic *Alternaria* species (10 x 40).

Figure 177. Conidiophores of *Alternaria* species (50x).

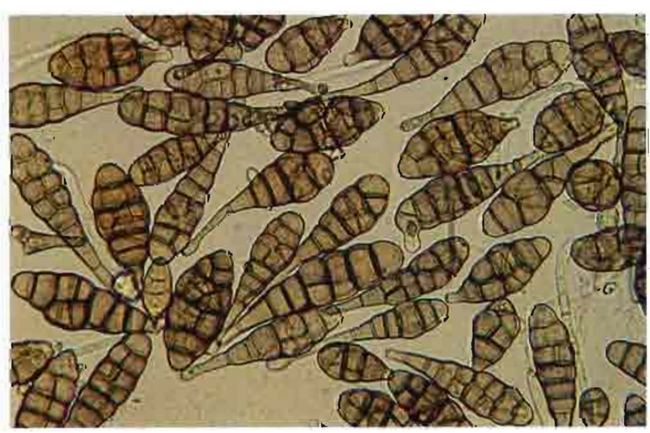


Figure 179. Larger conidia of *Alternaria triticina* (10 x 40). Compare with Figure 178.

Stemphylium botryosum

This species occurs everywhere as a common saprophyte on dead plant material. Some species occur as parasites on legume and vegetable crops. Fruiting structures are scattered in colonies, and short conidiophores arise singly from submerged mycelia or stroma, each producing a single conidium (Figures 180, 181). The conidiophores develop dark terminal swellings one after another, successive swellings appearing as each new conidium is produced. The conidia vary in shape, from globose-ovoid to cylindrical with rounded ends. They have several transverse and longitudinal oblique septa, but lack the prominent beak that characterizes *Alternaria* species. Conidia are dark brown to black in color, have a slightly rough surface and measure 25-40 μm x 20-30 μm (Figure 182).

Figure 180. Lesion on a wheat leaf in which the fruiting structures of *Stemphylium* species are developing.

Figure 181. A dense, sporulating colony of *Stemphylium botryosum* on a wheat leaf (50x).

Figure 182. Conidia of *Stemphylium botryosum* (10 x 40).

Cladosporium species

This genus includes a large number of forms that display a considerable variation in morphological characteristics. Two of the most common species especially abundant on dead plant material are *Cladosporium herbarum* and *C. macrosporum*.

The fruiting structures develop as bushy, grey to olive green to dark brown clusters (Figure 183). Conidiophores are usually branched, particularly at the upper end (Figures 184, 185). The conidia are ovoid, cylindrical or oblong with rounded ends, medium brown to olivaceous brown, have smooth to rough walls, and are zero to three septate. Conidia size varies considerably within and between species (Figure 186).

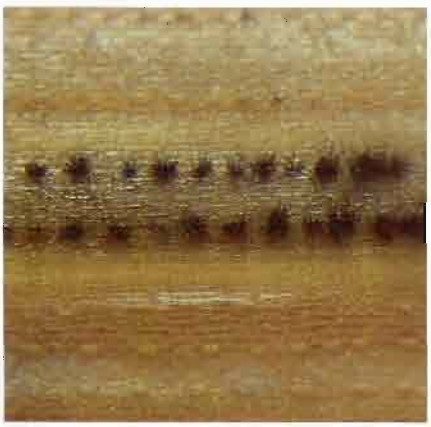


Figure 183. Fruiting structures of *Cladosporium* species (50x). Note the dark, bushy appearance.

Figure 184. A cluster of conidiophores of *Cladosporium* species developing from submerged mycelia.

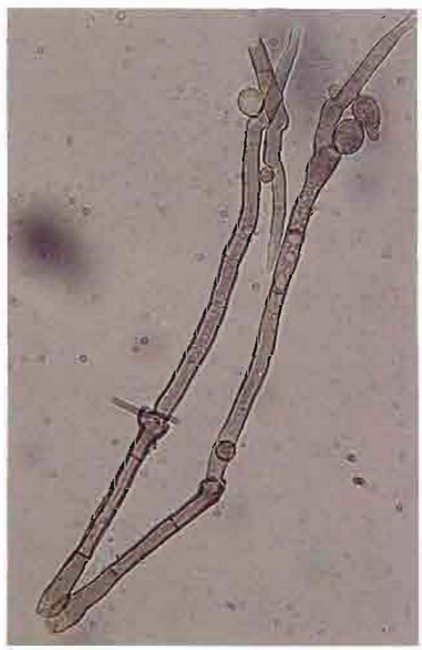


Figure 185. Two conidiophores of *Cladosporium* species showing conidial development at the upper ends.

Figure 186. Conidia of *Cladosporium* species (10 x 40). Note the variation in size and shape.

Pleospora species

Several leaf-spotting *Pleospora* species develop perithecia on cereals and grasses (Figure 187). These fungi occur as parasites or saprophytes, and either produce leaf spots or inhabit lesions produced by other parasites. The fruiting structures (perithecia) occur in small scattered colonies in necrotic areas (Figure 188). The ascospores vary in size, shape and color, but all have transverse and longitudinal septa (Figures 189, 190). Some of the species inhabiting cereal crops have yellow to brown ascospores. *Pleospora herbarum* is the sexual stage of *Stemphylium botryosum.*

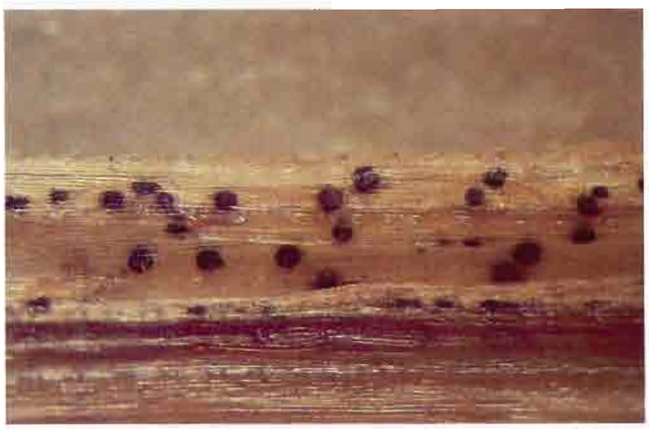


Figure 187. A colony of perithecia of *Pleospora* species in oats (50x).

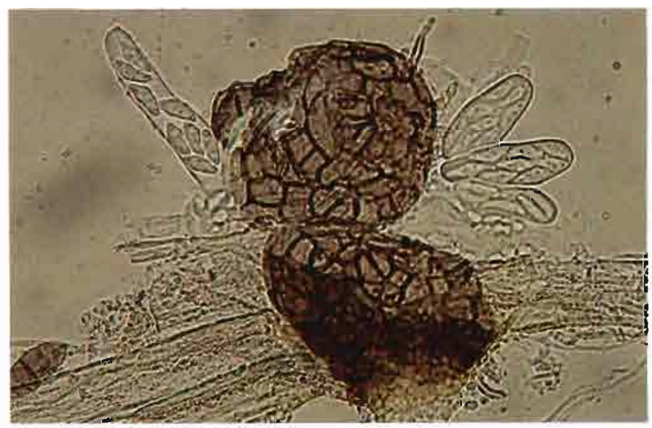


Figure 189. Two perithecia and the asci (containing ascospores) of *Pleospora* species (10 x 10).

Figure 188. Perithecia of *Pleospora* species developing in the green leaf tissue of wheat (50x).

Figure 190. Ascospores of *Pleospora* species typically develop transverse and longitudinal septa (10 x 40).

Torula species

Torula species are typical sooty mold fungi. They invade heads, upper stems and leaf sheaths of cereal crop plants, which become enveloped in a velvety, dark grey to brownish black fungal growth (Figure 191). Surface development of mycelia is restricted, and conidiophores are inconspicuous. Conidia develop in long chains, and appear to arise from segments of superficial mycelia. When mature, the conidial chains break into segments of from one to many cells. The single-celled conidia are spherical, about 6 μm in diameter, minutely echinulate and dark brown to black in color (Figure 192). The fungal growth frequently develops on cereals in the High Plateau of Mexico during wet harvests.

Figure 191. Stems, spike and grain of wheat affected by *Torula* species (left). Healthy wheat on the right.

Figure 192. Dark, thick-walled conidia of *Torula* species form in chains and are covered with an oily film when dry (photo: K.A. Mujeeb—magnification 10 x 16 x 1.25).

Phoma species

Phoma species are widely distributed and usually occur as pathogens on many herbaceous and woody plants. Their pathogenic activity is considerably restricted on cereal crops and grasses. *Phoma glomerata* has been observed to cause leaf diseases on wheat and triticale in Mexico and in Central and South America. Its appearance as a pathogen usually occurs after prolonged periods of humid weather. *Phoma* species are frequently observed as secondary invaders of the leaves (Figure 193) and floral bracts (Figure 194) of cereals during maturation.

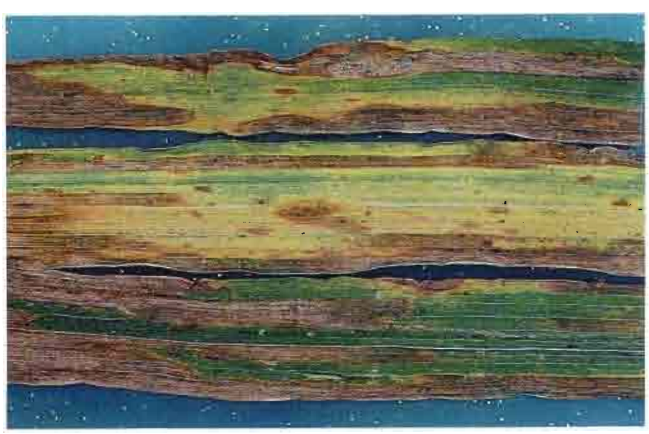


Figure 193. *Phoma* species developing on leaves already under stress.

The fruiting structures are dark, spherical pycnidia, which either develop superficially or are embedded in the host tissue. A conspicuous beak is present on the pycnidia of most species that occur on cereals (Figures 195, 196). The conidia are ovoid to elongate, unicellular and subhyaline. Unicellular conidia distinguish *Phoma* species from the pycnidial fungi of the septoria complex. The conidia of *P. glomerata* are subhyaline at first, become light greyish brown when they mature and measure 4-8 μm x 3 μm (Figures 197, 198).

Figure 196. The pycnidia of *Phoma* species often develop in compact colonies and produce spores in profusion (10 x 10).

Figure 194. Wheat spikelets covered with the pycnidia of *Phoma glomerata*.

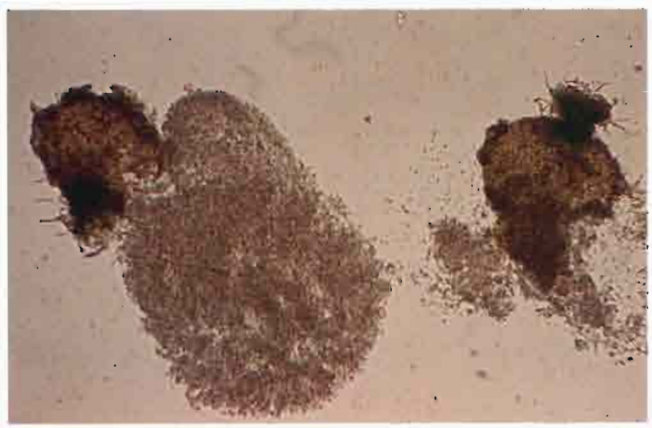


Figure 197. Two pycnidia of *Phoma* species releasing conidia. Note the prominent beak on the right (10 x 10).

Figure 195. The pycnidia of *Phoma* species are partially embedded in host plant tissue. Note the spherical shape and pronounced beaks (50x).

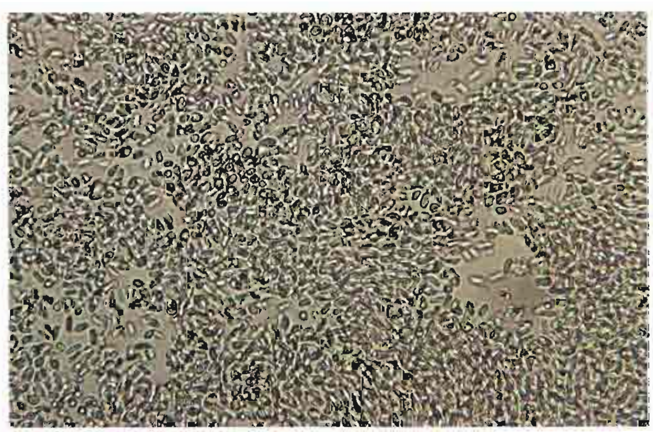


Figure 198. Unicellular conidia of *Phoma glomerata* (10 x 40).

Epicoccum nigrum

This fungus occurs everywhere as a saprophyte on dead plant material and frequently occurs on leaf spot lesions caused by other fungi or bacteria. The fruiting structures appear as small hemispherical clusters of black spores developing from sporodochia (Figures 199, 200). The mycelia are embedded in plant tissue. Conidiophores are compact, very short, smooth and produce a single terminal spore. Mature conidia are dark brown to black, globose or somewhat angular and irregularly septate (Figure 201). The septa are often obscured by the thick rough spore wall. Other than variation in spore size, there is a considerable degree of uniformity in morphological characteristics among *Epicoccum* species throughout the world. The black sporodochia may be confused under field observations with the pycnidia of *Septoria* species. However, the sporodochia of *Epicoccum nigrum* are easily rubbed off with a finger, while septoria pycnidia remain embedded in plant tissues.

Figure 200. A closer view of a sporodochium releasing the conidia of *Epicoccum nigrum* (10 x 40).

Figure 199. Sporodochia of *Epicoccum nigrum* developing on triticale straw (50x).

Figure 201. Conidia of *Epicoccum nigrum* (10 x 40).

Cercosporidium graminis

Cercosporidium graminis usually occurs as a leaf pathogen on grasses and occassionally infects rye, triticale and oats. Lesions develop in narrow elongated spots or streaks. The spots appear bleached, yellow-green to light grey, with numerous black specks scattered throughout the necrotic areas. Lesions on oat leaves have a yellowish red tinge (Figure 202).

The fruiting structures appear as tiny black tufts raised above the leaf surface. The conidia are formed singly on the apices of compact clusters of conidiophores protruding through the epidermis (Figure 203). The conidia are smooth, pale grey to dark brown, usually have one or two septa, taper gradually to a rounded apex, and are 8-12 µm wide near the base and up to 60 µm long (Figure 204).

Figure 202. Yellowish red lesion on an oat leaf, caused by *Cercosporidium graminis* (50x). Note the scattered clusters of fruiting structures (see Figure 203).

Figure 203. Dense clusters of the fruiting structures of *C. graminis* erupt through the epidermis of leaves (10 x 10).

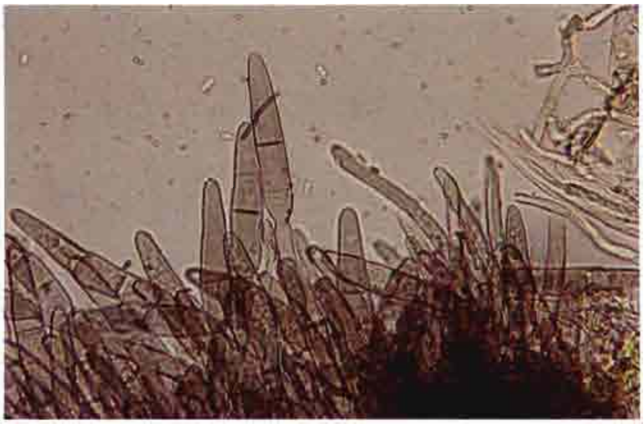


Figure 204. Greyish conidia of *C. graminis* usually have one to several septa (10 x 40).

Bacterial diseases

Bacterial plant pathogens are small unicellular rods from 1 to 3 μm in length. They do not have a well-defined nucleus, nor a nuclear membrane. Bacteria are spread by insects, air currents, splashing rain, and by mechanical means. Free moisture is usually necessary for infection, and penetration of host tissue occurs through wounds or stomatal openings. These pathogens invade the vascular system or intercellular spaces in host tissue, and necrosis results from toxins produced or enzymatic activity of the bacteria (Figure 205).

Symptoms on cereal crops appear as water-soaked streaks, spots or rots. Disease identification in this guidebook is limited to the symptoms produced and crop species attacked. Some bacterial pathogens form droplets of bacterial ooze on the surface beyond the area of the lesion under humid conditions (Figure 206). These bacteria are capable of movement between the cells in a gelatinous matrix. Some other species of bacteria do not form exudate on the surface, and the spread of disease is thus more or less restricted to the lesions surrounding the initial infection.

Figure 205. Bacterial stripe on barley leaves (facing page), caused by *Xanthomonas translucens* (photo: S. Fuentes).

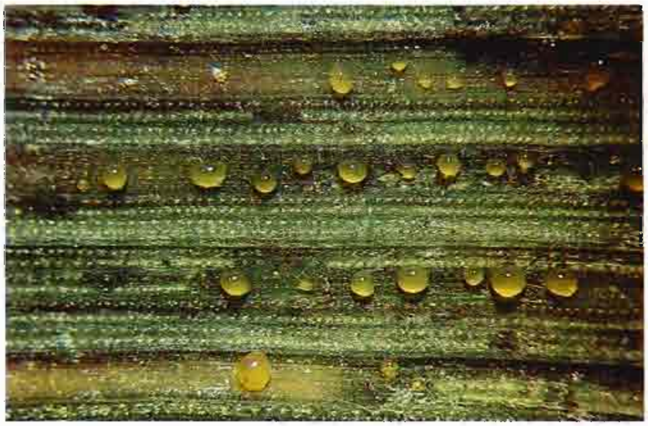


Figure 206. Droplets of bacterial exudate can develop under conditions of high humidity on the surface of diseased leaves. Some bacterial pathogens produce a clear yellow exudate; others, such as *X. translucens* (shown here) produce a cloudy yellow exudate.

Pathogenic bacteria are most commonly spread by air currents, water and insects. They also persist on seed, plant debris and in the soil. An important control measure is to use disease-free seed or seed that has been disinfected. Disease-resistant varieties are available for some bacterial diseases. Bactericide sprays are used effectively on vegetable and ornamental crops, but are usually uneconomical on cereals.

Bacterial stripe and black chaff

Xanthomonas translucens

Bacterial stripe and black chaff, caused by *X. translucens*, are the most common bacterial diseases of cereal crops and occur in all of the cereal-growing regions of the world. The pathogen attacks wheat, barley, rye, triticale and many grasses. All of the above-ground parts of the plant may be affected, but the disease occurs most commonly on the leaves and glumes.

When it attacks leaves and stems, the disease is referred to as bacterial stripe. The early symptoms appear as small, linear, light brown, water-soaked spots or streaks. The lesions tend to develop longitudinally between the veins during the early stages (Figure 207), but eventually expand and merge, producing irregularly shaped grey-brown blotches. Under high humidity, droplets of yellow bacterial exudate form along the lesions (Figure 208). Small yellowish granules or thin shiny scales form on the surfaces of leaf blades when the exudate dries. The disease progresses to the leaf sheaths and adjacent culms, causing dark staining and weakening of the stems.

Figure 207. *Xanthomonas translucens* on a barley leaf. Note how the early lesions tend to develop longitudinally between the veins.

Figure 208. Cloudy, yellowish bacterial exudate, typical of *X. translucens* (50x).

The disease is called black chaff when the heads and neck are affected. Black chaff is easily recognized by the dark, linear, water-soaked streaks on the glumes and lemmas (Figure 209). Usually the symptoms appear first on the upper parts of the glumes. As the disease develops, the lesions merge producing a dark staining of the glumes, lemmas and peduncles. Under severe conditions the kernels may become stained and shriveled.

This disease is often confused with brown necrosis (page 132) and glume blotch (page 41). Bacterial stripe of oats, reported to be caused by *Pseudomonas striafaciens*, develops symptoms very similar to those caused by *X. translucens* (Figure 210).

Figure 209. Symptoms of black chaff on a wheat glume, caused by *X. translucens*. Note the narrow dark stripes beginning at the base of the awn and the slimy appearance, which is due to bacterial exudate.

Figure 210. Bacterial stripe blight lesions on oat leaves, caused by *Pseudomonas striafaciens*. Note how the lesions are found at or near the edges of the leaves (photo: M. Moore).

Basal glume rot

Pseudomonas atrofaciens

Pseudomonas atrofaciens occurs on wheat, barley, rye and triticale. It is widespread, but usually of minor economic importance. The disease affects the floral bracts and grain, and can be recognized by light grey-brown discolored areas on the lower part of the glumes (Figure 211). The dark staining is more pronounced on the inner sides of glumes and lemmas, and the staining may extend to the rachis and kernels (Figure 212). During extended wet periods, a whitish grey bacterial exudate may form on the discolored lesions. Early infections of basal glume rot cause shriveling and discoloration of the kernels.

Figure 211. Symptoms typical of basal glume rot, caused by *Pseudomonas atrofaciens*. Note the grey-brown discoloration of the lower part of the glumes.

Figure 212. The staining caused by *P. atrofaciens* is more pronounced on the inside of the glumes. At right is a seed totally destroyed by the bacteria.

Halo blight of oats

Pseudomonas coronafaciens

Halo blight occurs in most oat-growing regions of the world. Cultivated species of oats and several grasses are susceptible. The disease occurs most frequently on leaf blades, but the leaf sheaths and panicles may also be affected. The early lesions are small, oval-shaped, water-soaked spots, straw to light brown in color (Figure 213). The borders surrounding the spots gradually turn light yellow and become water soaked (Figure 214). Under moist conditions, bacterial exudate may form in the lesions, but not to the same extent as on plants infected by *Xanthomonas translucens* or *Pseudomonas striafaciens*. The disease lesion tends to be restricted to the brown area surrounding the initial point of infection (Figure 215).

Aphids and other insects carry the bacteria from plant to plant and, through their feeding, provide the means by which the bacteria may enter the host tissue. The disease may be seed borne, but because the bacteria survive on plant debris and other grass hosts, seed treatment does not ensure freedom from the disease.

Figure 213. Typical early lesion of halo blight of oats, caused by *Pseudomonas coronafaciens*. Note the water-soaked appearance.

Figure 214. As the lesions of halo blight of oats age, the borders surrounding the lesions turn a yellowish green (photo: M. Moore).

Figure 215. The lesions caused by *P. coronafaciens* tend to be restricted to the brown areas surrounding the infection sites.

Bacterial spike blight

Corynebacterium tritici

Spike blight is a disease of wheat, and is also referred to as "tundu" and yellow ear rot. The disease occurs in North Africa, Asia and Australia, and is usually associated with a gall-forming nematode, *Anguina tritici*. The gall nematode has been observed in some areas of North America, but without the spike blight bacterial disease.

The disease is generally restricted to the heads, and appears as a sticky, yellow slime on wheat spikes. Spikes emerge from the sheath covered with a mass of sticky bacterial exudate. The leaves and spikes are distorted and produce little or no viable seed (Figure 216). If the gall nematode is associated with the disease, the kernels are replaced by seed galls (Figure 217). The disease is dispersed through contaminated seed, in soil or in association with seed galls. Larvae of *Anguina tritici* are released from the gall in the soil and may serve as vectors for the bacteria with which they have become contaminated (Figure 218).

Figure 217. A wheat spike, normal seed and the seed galls (cockles) caused by an infestation of the nematode, *Anguina tritici*. These nematodes can carry the inoculum of *C. tritici*.

Figure 216. Distorted flag leaves and the formation of sticky exudate on the spikes are symptoms typical of bacterial spike blight, caused by *Corynebacterium tritici* (photo: E.E. Saari).

Figure 218. A closer view of a seed gall (cockle) from a wheat spike. The gall has been cut open to reveal the mass of dormant nematodes.

Bacterial leaf blight

Pseudomonas syringae

This species is a common pathogen on maize, sorghum and beans in many areas of the world, but only recently has it been recognized as a serious pathogen of small grain cereals. The disease is prevalent and destructive on bread wheat, durums and triticale in the Great Plains of the USA, and probably other cereal growing areas as well.

The early lesions appear as small, bleached green, water-soaked spots that expand rapidly under favorable conditions to produce irregular yellow-brown blotches (Figure 219). The disease develops most rapidly under warm, humid conditions, and disease lesions produce bacterial ooze in moisture. *Pseudomonas syringae* does not produce the leaf stripe or black chaff symptoms that characterize *X. translucens*.

Little is known regarding the etiology of the disease or its control. Some wheat varieties have been reported as being resistant to the bacteria.

Figure 219. Lesions typical of those produced on wheat leaves by *Pseudomonas syringae*.

Viral and mycoplasmal diseases

Viruses are very small life forms capable of infecting living cells and causing injury to their hosts. Mycoplasmas, since they cause viruslike diseases, were at one time considered to be viruses, but more recently have been given a category of their own between bacteria and viruses. Mycoplasmas somewhat resemble bacteria, but are smaller and do not have a rigid cell wall. They have a very fine outer membrane, which allows greater flexibility than bacteria.

Viruses and mycoplasmas that cause diseases in cereal crops may be transmitted by aphids, leafhoppers, planthoppers, mites, fungi, seed or mechanically. They are identified by the symptoms produced, the host range, vectors, and by more complicated laboratory procedures, including electron microscopy and serological techniques.

Viral diseases generally cause symptoms that distinguish them from other infectious diseases. The most common symptoms are dwarfing, excessive tillering, and various forms of leaf streaking, spotting, chlorosis and necrosis (Figure 220). Viruses do not produce visible fruiting structures or ooze. Many viral diseases produce somewhat similar disease symptoms and are difficult to identify with certainty. Determining the mode of transmission helps considerably in reducing the number of possibilities. Beyond this, the plant scientist may determine the crop specificity and consider the geographic distribution of a particular virus or vector. To assist in identification, the viral diseases discussed here are grouped by vector.

Figure 220. Streaking, general chlorosis and yellowing of leaves, stunting, and sterile spikes (see Figure 236) are symptomatic of many viral diseases. Here, the symptoms of aster yellows, a leafhopper-transmitted viral disease (photo: E. Banttari).

Successful control of viral diseases depends upon the vectors involved and their relationships with the hosts in transmitting disease. Seed-borne viruses can be controlled by the use of virus-free seed. Soil areas contaminated by fungal vectors should be sown to resistant crops in long-cycle rotations. Insect vectors, particularly aphids, may transmit a virus during very brief feeding periods. Insecticides are not very effective against aphids, but can be successfully used against leafhoppers, which require longer feeding periods to become viruliferous and to transmit the virus. Systemic insecticides are more effective against those insect vectors that cause leaves to curl, thus forming a protective covering against predators and contact insecticide sprays. Genetic resistance against the vector or the pathogen is the most effective means of control, but is not always available.

Aphid-transmitted viral diseases

Aphids are among the most common and efficient vectors of viral diseases. Disease development on cereal crops requires infestations of viruliferous aphids. Normally, small populations of aphids migrate from neighboring crops or grasses. Diseased plants occur singly or in small patches, usually starting at the borders of the field. Under certain conditions, the aphid population builds up to the point where extensive windborne migrations may infest large areas almost simultaneously.

General chlorosis, yellowing and dwarfing have been considered the typical disease symptoms of aphid transmitted viruses on cereal crops. In recent years, however, cereal crop viral diseases with mosaic and striping symptoms have been reported as having aphid vectors.

Barley yellow dwarf virus (BYDV)

This disease is among the most widely distributed viral diseases on cereals and can cause serious economic losses. Wheat, barley, oats, rye, triticale and grasses are hosts of BYDV. Symptom expression on barley and oats is quite conspicuous—bright yellowing (on wheat and barley—Figure 221) or reddening (on oats—Figure 222) of the leaves starting from the tip and developing toward the base, stunting, excessive tillering, the development of white sterile spikes, and the presence of aphids are common diagnostic characteristics. Many species of aphids are vectors for barley yellow dwarf, but among the most common are *Rhopalosiphum, Metopolophium* and *Macrosiphum* species (Figure 223). *Schizaphis graminis*, the greenbug, may also transmit the virus (though somewhat less efficiently) and can also produce

Figure 222. Severe dwarfing, sterility and a reddish yellow discoloration of leaves in oats may be due to BYDV.

Figure 221. A wheat leaf showing symptoms of barley yellow dwarf virus (BYDV). Note the yellow, bleached appearance of the leaf. Symptoms are similar on barley (photo S. Fuentes).

Figure 223. Several aphid species serve as common vectors of BYDV, including *Rhopalosiphum maidis* (shown here). The main vector by far is *R. padi*, which is similar in appearance to *R. maidis* but with reddish hindquarters.

spots or blotches that develop on the leaves due to toxins released during feeding (Figure 224). Considerable research has been focused on the development of varieties resistant to barley yellow dwarf virus.

Figure 224. Greenbugs (*Schizaphis graminis*) act as vectors of BYDV, and also cause damage when feeding.

Figure 225. Aphid vectors of Free State streak are found inside the rolled upper leaves of affected plants. Note the pale green stripes caused by the virus.

Free State streak

Free State streak is an aphid-transmitted disease that occurs in Mexico and in southern Africa. It is found on wheat, barley, rye, triticale and some annual brome species, but does not produce symptoms on oats. Diseased plants develop one to three pale green stripes on the leaves (Figure 225). The upper leaves tend to roll lengthwise, developing a rigid "onion leaf" appearance. A pale reddish or purple color occasionally develops among the stripes on wheat and triticale. Spikes are usually fleshy and distorted, and rarely produce viable grain (Figure 226). The aphid vectors, known as *Diuraphis noxia* in Africa (Figures 227, 228–next page) and *D. mexicana* in Mexico, are found inside the rolled upper leaves. Young barley plants tend to recover when the aphids are removed or killed.

Figure 226. Distorted, chlorotic barley spikes are symptomatic of Free State streak.

Other aphid-transmitted viral diseases

Several other aphid-transmitted viral diseases occur in central and eastern Asia. Wheat yellow leaf occurs on wheat and barley in Japan. The symptoms are somewhat similar to barley yellow dwarf virus. The corn leaf aphid, *Rhopalosiphum maidis*, is the vector. Barley mosaic and Cardamon streak mosaic are viral diseases that commonly occur in India. *Rhopalosiphum maidis* and other aphid species serve as vectors. Wheat, oats, barley and some grasses are susceptible.

Figure 227. A winged adult Russian grain aphid (*Diuraphis noxia*), a vector of Free State streak.

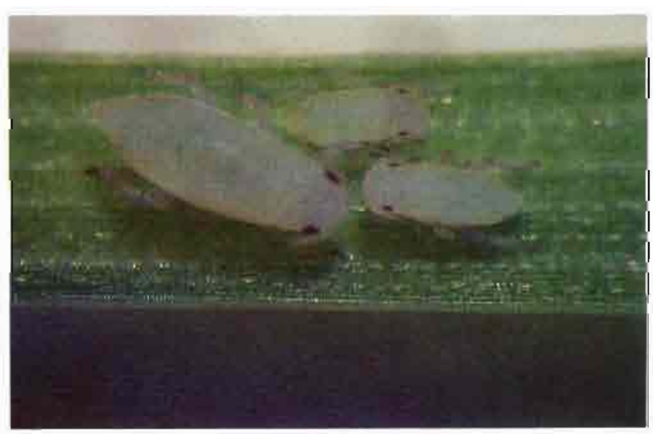


Figure 228. Young Russian grain aphids (*D. noxia*).

Leafhopper-transmitted viral diseases

Leafhoppers serve as vectors for several cereal crop viruses and at least one mycoplasma. Several wheat striate mosaic diseases produce rather similar symptoms, making disease identification based on symptoms very difficult. The symptoms occur as fine discontinuous chlorotic streaks along the veins of leaves (Figure 229). Later, the leaves develop necrotic spots (Figure 230). The plants may become stunted, with considerable head sterility. Wheat, oats, barley, rye and various grasses are hosts to most of these viruses. Winter crops often are more seriously damaged than spring crops, but delaying fall seeding by several weeks usually reduces leafhopper infestation. Disease tolerant varieties also help to reduce losses.

American wheat striate mosaic

This disease occurs in the central and northern USA and in Canada. The leafhopper vectors are *Endria inimica* (Figure 231) and *Elymana virescens.* Winter wheat is the most commonly affected crop, but other small grains are also susceptible.

Chlorois striate mosaic virus (Australian wheat striate mosaic)

The leafhopper *Nesoclutha obscura* is the vector for this virus. The vector overwinters on corn and grasses, which also serve as hosts for the virus. The disease is relatively unimportant on small grain crops.

Russian wheat mosaic

This disease can be serious on both spring and winter wheats in eastern Europe and Russia. It also occurs on oats, barley and rye. The symptoms are typical of those reported for the American wheat striate and chlorois striate mosaic viruses. Severe stunting occurs following seedling infection. Two leafhoppers, *Psammotettix striatus* and *Macrosteles laevis*, are vectors.

Figure 230. Later symptoms of wheat striate mosaic diseases on barley (photo: J. Slykhius).

Figure 229. Typical leaf symptoms of wheat striate mosaic diseases on wheat. Note the fine, discontinuous chlorotic streaks or striations.

Figure 231. The leafhopper, *Endria inimica*, a vector of American wheat striate mosaic.

Enanismo de Nariño

This is the name given to a cereal dwarfing virus that occurs in highland areas of Colombia and Ecuador. The disease is associated with the leafhopper *Cicadulina pastusae* (Figure 232). Serious losses have occurred in wheat, oats and barley in some areas. Diseased seedlings are severely stunted. Plants infected after elongation develop leaf enations or galls on the new leaves (Figure 233). It is suspected that a toxin produced by the leafhopper *C. pastusae* is responsible for the gall formation.

Aster yellows

Aster yellows is a minor disease of wheat, barley and some other Gramineae, but may cause serious economic losses to vegetable and ornamental crops. It occurs in many areas of North America, Europe and Asia. Leafhoppers of the *Macrosteles* (Figure 234) and *Endria* genera are the most common vectors. The pathogen is now classified as a mycoplasma, although the disease symptoms and transmission are similar to those associated with some viral diseases. Diseased plants develop general chlorosis and yellowing of the leaves, stunting and sterile spikes (Figures 235, 236). The symptoms on cereal crops are quite similar to those caused by BYDV.

Figure 233. Leaf enations and distortions symptomatic of enanismo de nariño

Figure 232. A leafhopper nymph of the genus *Cicadulina* (50 x).

Figure 234. The leafhopper, *Macrosteles fascifrons*, a common vector of aster yellows. (photo: E. Banttari).

Figure 235. General chlorosis and yellowing are symptoms typical of aster yellows (photo: E. Banttari).

Figure 236. Barley spikes affected by aster yellows (center and right). Note the sterility and the chlorotic, distorted appearance (photo: Dr. Banttari).

Planthopper-transmitted viral diseases

Planthopper-transmitted viral diseases occur on wheat, barley, triticale, oats, rye and various grasses. Disease symptoms are similar to those of other viral diseases. White to yellow streaks on leaves, stunting, excessive tillering and sterility are fairly common. Viral diseases transmitted by planthoppers appear most frequently in Europe and East Asia. The most efficient vectors appear to be *Laodelphax* and *Javesella* species.

Hoja blanca of rice

Hoja blanca, or white leaf, is primarily a disease of rice, but may affect all of the small grain cereals. Its distribution is restricted to the presence of the vectors, *Sogatodes* species, occurring on cultivated rice. The "white leaf" characteristic is more pronounced in the upper leaves and spikes. The lower leaves may display mottling and striping. This disease occurs in the rice-growing areas of the southern USA, the Caribbean, and Central and South America.

African cereal streak

This disease is common on wheat in Kenya and Ethiopia. It may occur on all small grain cereals and local grasses. The disease usually occurs on scattered plants or in small patches, and is more prevalent on crops grown during the dry season. The virus is transmitted by *Toya catilina*. Early symptoms appear as thin pale streaks on leaves. These tend to expand, particularly on upper leaves, producing pale yellow stripes or general leaf chlorosis (Figures 237, 238, 239). Crop losses are of little economic importance.

Figure 238. Dwarfing that is characteristic of African cereal streak (photo: D. Harder).

Figure 237. Leaves and spike of wheat with African cereal streak. Note the pale yellow stripes and general chlorosis.

Figure 239. A closer view of the symptoms typical of African cereal streak.

European wheat striate mosaic

This viral disease developed to epidemic proportions in western Europe in the late 1960s. It occurs on wheat, oats, barley, rye, maize and some grasses. Two or more *Javesella* species are efficient vectors. Plants infected during the seedling stage do not survive (Figure 240). Those infected later produce broad, pale yellow stripes on upper leaves (Figure 241). Seed production on such plants is very low and of poor quality. Late fall seeding of winter cereals reduces exposure to viruliferous planthoppers.

Other planthopper-transmitted viral diseases

Several other cereal viral diseases transmitted by planthoppers, particularly *Laodelphax* species, occur in Europe. Among these are barley yellow striate mosaic, wheat chlorotic streak and cereal tillering. Oat sterile dwarf (also occurring in Europe) is transmitted by the planthoppers *Javesella pellucida* (Figure 242) and *Dicranotropis hamata*. Most grain crops are susceptible to these viruses.

Figure 240. A comparison of the effects of European wheat striate mosaic when infection occurs early in the life cycle of the host plant (left) with the symptoms produced when infection occurs somewhat later (center). A healthy plant is on the right (photo: R. Plumb).

Figure 241. Leaf symptoms produced on wheat by European wheat striate mosaic (photo: R. Plumb).

Figure 242. The planthopper, *Javesella pellucida*, a vector of oat sterile dwarf (photo: R. Plumb).

Mite-transmitted viral diseases

At least three mite-transmitted viral diseases of cereals occur in North America and other cereal-growing areas of the world (Figure 243). Two species of mites, *Aceria tulipae* (Figure 244) and *Abacarus hystrix*, are common vectors of the viruses. The diseases are more serious on wheat than on barley and rye.

Wheat streak mosaic

Wheat streak mosaic is a serious disease of winter wheat in the winter wheat belt of the central USA. The disease is widely distributed in North America, Europe and the Near East. The wheat curl mite, *Aceria tulipae*, is the common vector. Diseased plants develop mottled chlorotic streaks (Figure 245). Stunting is more pronounced on plants infected during the seedling stage.

Figure 244. Wheat curl mites (*Aceria tulipae*) are common vectors of several viruses (photo: T. Harvey).

Figure 243. Leaf symptoms of three different mite-transmitted viral diseases: wheat streak mosaic (top), agropyron mosaic (center), and wheat spot mosaic (bottom).

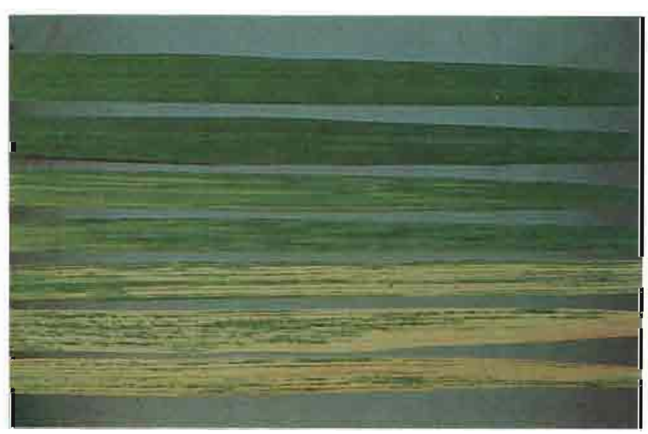


Figure 245. Leaf symptoms of wheat streak mosaic on wheat (photo: J. Slykhius).

Agropyron mosaic and rye grass mosaic

Agropyron mosaic and rye grass mosaic are viral diseases transmitted by the mite species *Abacarus hystrix*. The primary hosts are the grass species, but cereal crops are occasionally infected. Yellowish green mottling and narrow chlorotic streaks develop on the leaves of affected wheat and barley plants (Figure 246). Oats are susceptible to rye grass mosaic (Figure 247), but not to agropyron mosaic.

Wheat spot mosaic

Wheat spot mosaic is also transmitted by the mite *A. tulipae*. The symptoms are distinct, light green spots that eventually enlarge into yellow necrotic areas starting at the leaf tip (Figure 248). The pathogen that causes wheat spot mosaic has not yet been identified. The disease may occur along with wheat streak mosaic in some areas. Crop losses from wheat spot mosaic are not economically important.

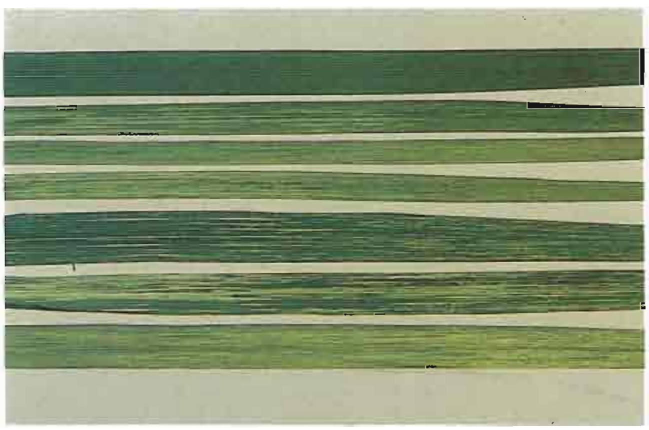


Figure 246. Leaf symptoms of agropyron mosaic on wheat (photo: J. Slykhius).

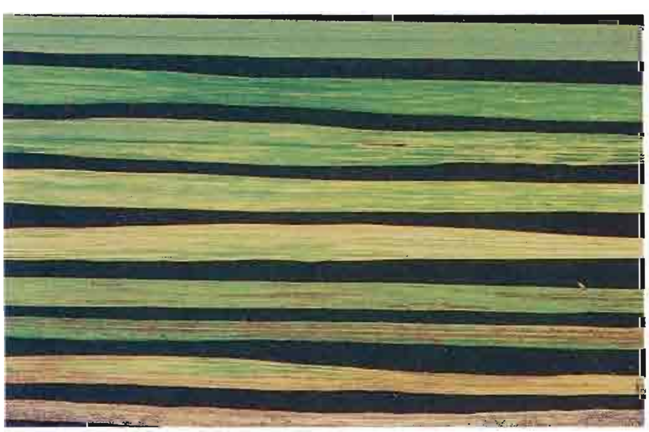


Figure 247. Leaf symptoms of rye grass mosaic (photo: J. Slykhius).

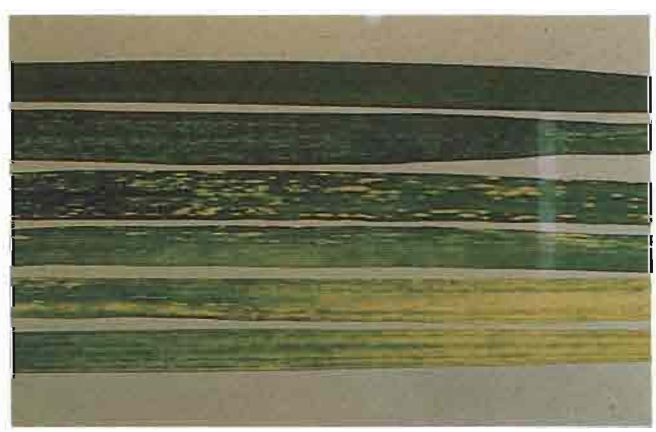


Figure 248. Leaf symptoms of wheat spot mosaic (photo: J. Slykhius).

Soil-borne viral diseases

The occurrence of soil-borne viral diseases is worldwide. Those found on cereal crops in North America are transmitted primarily by the primitive soil-inhabiting fungus *Polymyxa graminis* (Figure 249). Nematodes and certain other soil-inhabiting organisms serve as vectors for some viruses. Wheat, barley and rye are common hosts to soil-borne viruses, but oats are either resistant or do not produce symptoms. The diseases tend to appear in the same area each year and usually spread slowly. The fungal vector is soil inhabiting and is difficult to control. Soil fumigation is effective, but not economical on cereal crops.

Wheat soil-borne mosaic

This disease causes serious losses to winter wheat crops in North America. Barley and rye may also be affected. The disease is transmitted by the fungus *Polymyxa graminis,* and appears to be more common in wet-soil areas. Diseased plants are stunted and develop leaf streaks or various chlorotic patterns (Figures 250, 251). When infected in the seedling growth stage, susceptible varieties develop a "mosaic-rosette" and do not produce heads. Resistant varieties of winter wheat are available.

Wheat spindle streak mosaic

Wheat spindle streak is another viral disease transmitted by the fungus *P. graminis.* The disease occurs in the Great Lakes and other regions of the USA and Canada. Diseased plants develop more distinct symptoms during cool weather (Figure 252). Diseased plants tend to recover during warm weather.

Figure 250. Wheat soil-borne mosaic on wheat (photo: S. Fuentes).

Figure 251. Fleshy, distorted and sterile wheat spikes are symptomatic of wheat soil-borne mosaic virus.

Figure 249. The soil-inhabiting fungus, *Polymyxa graminis,* in wheat root tissue. This fungus can serve as a vector of certain soil-borne viral diseases (photo: D.J.S. Barr).

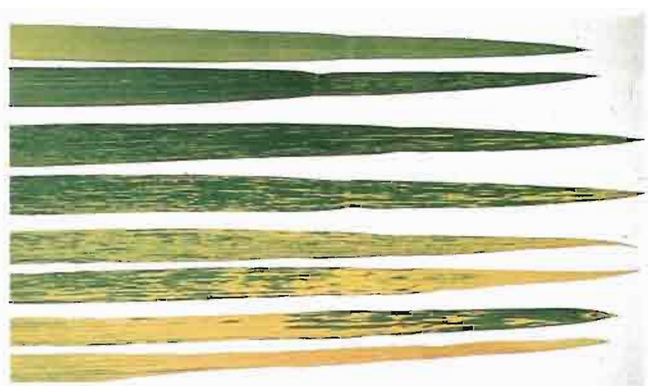


Figure 252. Leaf symptoms of wheat spindle streak mosaic, transmitted by *P. graminis* (photo: J. Slykhius).

Seed-borne viral diseases

Barley stripe mosaic

Barley stripe mosaic is one of the few cereal crop viral diseases known to be seed transmitted. The disease occurs principally in barley, but may also affect wheat, oats, maize and several grasses. The disease is distributed worldwide. The virus can be transmitted mechanically by plants brushing together in the field, or may be pollen transmitted.

Disease symptoms from seed-borne viruses tend to appear early in the season. Diseased barley plants develop chlorotic streaking or striping of the leaves (Figure 253). Severe stunting and excessive tillering occur on the more susceptible varieties (Figure 254). The stripe symptoms are somewhat similar to the early symptoms of barley stripe caused by *Helminthosporium gramineum* (page 30). Symptoms on wheat are usually moresubdued, although susceptible varieties develop typical mosaic symptoms. The use of disease-free seed and the limiting of barley volunteers from previous crops usually provide adequate control.

Barley mosaic

Barley mosaic, an uncommon viral disease reported in India, is usually transmitted by the aphid *Rhopalosiphum maidis*, but may also be transmitted through seed and mechanically to barley, wheat and oats. The disease has not been reported outside of India. Diseased plants are stunted, their leaves displaying mosaic symptoms late in the growth cycle.

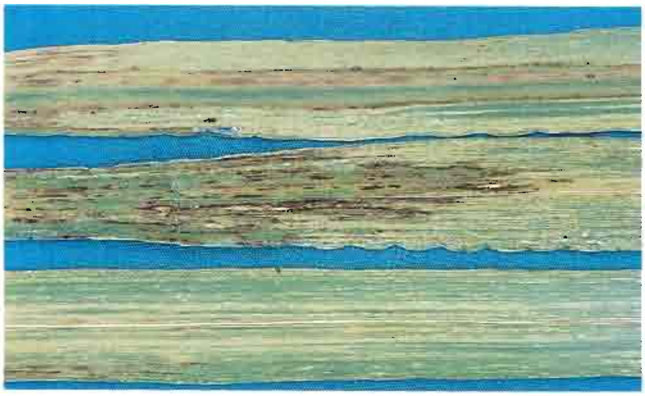


Figure 253. Leaf symptoms typical of those produced by barley stripe mosaic virus. Note how the necrosis tends to scribe a "V" or "W" across the leaf.

Figure 254. Stunting and excessive tillering are symptomatic of barley stripe mosaic virus.

Injury from nematodes and other pests

Injury to cereal crop plants from nematodes and other pests is usually associated with feeding and mechanical damage. However, such parasites as nematodes and insects may transmit disease pathogens. The damage caused by pests can be as serious as from disease organisms. Pests vary greatly in size, form and activity. With larger animal pests, the nature of the injury inflicted and the presence of the pest or its debris provide clues as to the injurious agent involved. With smaller forms, such as nematodes and small insects, it is frequently necessary to examine symptoms or signs under magnification to identify the cause. Only a very brief introduction to some of the most important groups of cereal crop parasites and pests is included in this section.

Nematodes (eelworms)

Nematodes have been recognized as cereal crop pests for over 300 years, but their ability to transmit viral diseases was discovered only recently. Nematodes occur in large numbers, and most live in soil, water or on plant and animal debris close to the soil surface. They are small, transparent worms that can be observed under low magnification (Figure 255). The species parasitizing cereal crop plants cause mechanical injury, rotting, root knots, galls and the distortion of plant parts (Figure 256). Nematodes affect the yield and quality of the crops they infest. They may act as vectors for some viruses and provide bacteria and fungi with a means of entry through mechanical injury to the plant.

Identification of the injurious agent can be made from symptoms on the roots or above-ground parts of affected plants, and by the presence of galls, cysts and nodules in which the eggs are found.

Among the more important parasitic forms infesting cereal crops are the seed gall nematode (*Anguina tritici*), the oat cyst nematode (*Heterodera avenae*), and root knot nematodes (*Meloidogyne* species).

Figure 255. Nematodes are small transparent worms that are visible under low magnification (10 x 10). This one (facing page) is typical of those taken from infested wheat nodes on the High Plateau of Mexico (see Figure 256).

Figure 256. Distorted wheat nodes infested with nematodes.

Seed gall nematodes *(Anguina tritici)* were among the first to be recognized as a cause of disease in wheat. This parasite also infests rye, triticale and related species. Seed gall occurs on wheat chiefly in the northern part of the Eastern Hemisphere, particularly throughout the Near and Middle East. The symptoms appear before heading as distorted leaves and lower stems. As the plants approach maturity, galls are formed in the florets and replace the seed. The galls are similar in size to the seed they replace, dark brown in color, and more difficult to crush than bunt balls (Figure 257). Large numbers of motile nematode larvae are contained in the seed galls (Figure 258) and become active after the galls have been moistened. The seed galls, when mixed with seed to be planted, provide a means of propagation.

Oat cyst nematodes (*Heterodera avenae*) are widespread in the cereal-growing areas of the world. Wheat, oats and rye are susceptible. The roots of infested plants develop frequent branches and swellings (knots). Cysts can be observed clinging to the roots (Figure 259). The cysts are light grey when young and dark brown when old. The female of *H. avenae* has a characteristic saclike body containing large numbers of eggs that remain in the sac even after her death.

Figure 257. A comparison of seed galls (cockles), caused by the nematode *Anguina tritici* (left), with normal wheat seed (right).

Figure 258. Seed galls are filled with dormant nematodes (*A. tritici*), which become active in moisture.

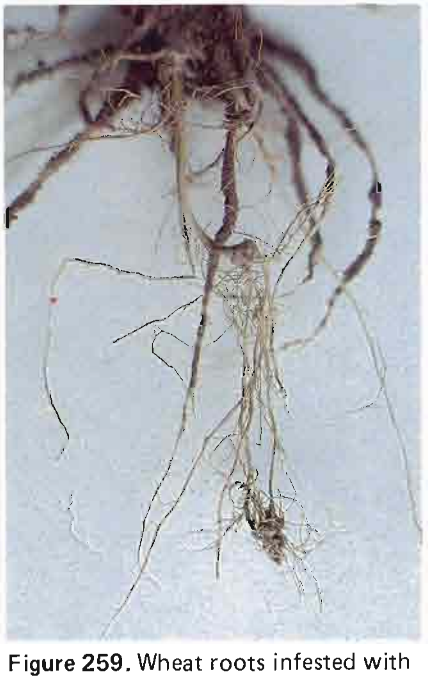


Figure 259. Wheat roots infested with oat cyst nematodes. Note the cysts clinging to the roots.

Figure 260. Symptoms of a root knot nematode infestation. Note the excessive branching of the roots and the stunted, yellow appearance of the above-ground parts (photo: S. Fuentes).

Root knot nematodes (*Meloidogyne* species) have a wide range of hosts, including all of the cereal crops. *Meloidogyne naasi* is the most common species attacking cereal crops and some grasses. Root knot nematode infestations are recognized by small knots or galls, usually curved or twisted, near the tips of the roots. Above ground, the infested plants are stunted and yellow (Figure 260). Excessive branching of infested roots sometimes occurs. Nematodes usually invade plants in the spring or early summer. Each knot envelops one to several females within the root tissue. The females produce large egg masses within their saclike bodies.

Nematicides and soil fumigants are effective in controlling most nematodes, but are seldom economical on cereal crops. The most effective control is through crop rotations that include resistant crop species. *Anguina tritici* larvae, which persist in seed galls, are released when contaminated seed is planted in wet soil. Thus, use of clean seed is one important precaution.

Insects

Figure 261. Greenbugs on a triticale leaf. Note the discolored blotch caused by toxins emitted by the greenbugs as they feed.

The number of insect species that feed on cereal crops is very large. Only a few of the most common pests are mentioned here. Insects cause injury by feeding, and predispose weakened plants to disease. They produce wounds that serve as infection sites, and often carry pathogens with them from plant to plant. Insects are by far the most numerous and most efficient vectors of viruses and mycoplasmas. Some insects, such as greenbugs, produce toxic substances or byproducts that cause further injury to plant tissues (Figure 261).

Careful observation of the type of injury, and the presence of the insects or their droppings help to identify the injurious agent. Insects that have chewing mouth parts remove tissue or cut off young plants. Wire worms, cut worms and June bug (May beetle) larvae either feed under the soil surface or return below the surface after feeding. The worms can usually be found below the soil surface close to the damaged plants.

The larvae of Hessian flies and shoot flies are very destructive to cereal crops in many areas of the world. Individual tillers of young plants are killed, and the remaining tillers of infested plants often develop root rot (Figure 262). The larvae and pupae live and feed inside the succulent stems of young tillers (Figure 263). Sawfly larvae (Figure 264) and wheat stem maggots live inside the hollow stems, causing head sterility and lodging. The type of damage, and the presence of the larvae, pupae and debris help to identify the source of the problem.

Figure 263. Pupae of Hessian flies in wheat (photo: S. Fuentes).

Figure 262. Damage caused by Hessian fly larvae.

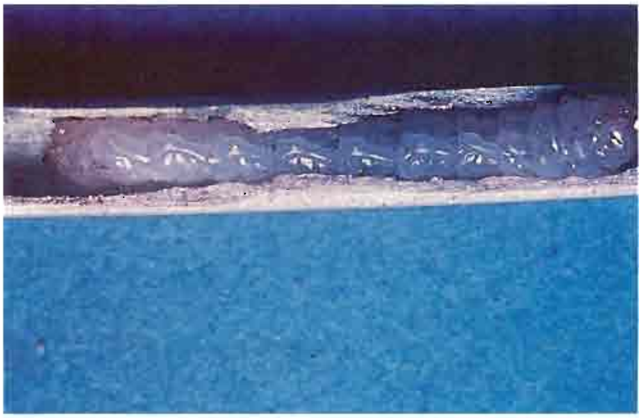


Figure 264. Sawfly larva in a wheat stem (photo: S. Fuentes).

Insects that feed on the surface of leaves, spikes or stems of cereal crops are usually more easily identified. Grasshoppers (Figure 265), army worms, aphids (Figure 266) and leafhoppers may infest crops in large numbers, or their numbers may build up rapidly. Smaller, less conspicuous species, such as thrips (Figure 267), mites and cereal leaf beetles (Figures 268, 269, 270 — next page), are often overlooked, particularly when populations are low.

Grain-destroying insects do their damage during grain filling and at maturity. In some areas of Africa and Asia, grain losses are caused by ants and termites. Ants usually climb the stems and remove grain from the florets, allowing it to drop to the ground where other ants carry it away. Termites burrow into the crown and cause the plants to lodge. Clay tunnels are then built along the stems to underground storage areas (Figure 271). Weevils and grain borers are particularly destructive to stored grain in areas where dry, tight storage facilities are unavailable (Figures 272, 273). These insects are less of a problem in areas having low winter temperatures.

Figure 265. Grasshopper feeding on a durum wheat spike (photo: S. Fuentes).

Figure 266. Corn leaf aphids on a barley leaf (photo: S. Fuentes).

Figure 267. Thrips on barley (photo: S. Fuentes).

Figure 268. Cereal leaf beatle larva feeding on a wheat leaf (photo: C. Dowswell).

Figure 269. Cereal leaf beatle larvae spray their backs with fecal material (photo: S. Fuentes).

Figure 270. Typical damage (feeding pattern) of cereal leaf beatle larvae (photo: S. Fuentes).

Figure 271. Clay tunnels built by termites along lodged wheat stems.

Figure 272. Grain weevils taken from infested triticale seed.

Figure 273. A sample of damaged triticale grain, infested with grain weevils. Note the holes in the grain and loose "flour dust."

Birds and mammals

Birds are attracted to cereal crops from the milk stage to maturity. Grain is removed from the spikelets, leaving the heads damaged and the glumes and lemmas scattered on the ground. Stems are broken from the weight of the feeding birds (Figure 274). In Asia and Africa, parrots and similar birds clip off the heads completely, leaving only barren stems. Bird damage is usually most severe along the edges of the fields.

Rodents cause serious economic losses to cereal crops, both in the field (Figure 275) and in storage. Gophers, groundhogs and some species of rats dig burrows in the field. In some countries the losses due to burrowing rodents are serious enough to preclude cereal crop production. Larger animals clip the plants while grazing. Tracks, droppings and the type of damage are means by which the culprits may be identified.

Figure 274. Damage typical of that caused by birds. Note the broken stems.

Figure 275. Durum wheat nursery plots damaged by rodents.

Physiological and genetic disorders

Numerous forms of abnormal plant development not caused by infectious disease organisms occur in cereal crop plants. These disorders may be due to genetic anomalies or environmental stresses and irritants.

Physiological leaf spots, blotches and chlorosis of leaves frequently occur on cereal crop plants. Barley varieties are especially prone to spotting and blotching under some conditions (Figure 276). Some appear as responses to irritants or other environmental factors, but many are completely unpredictable. Varieties differ greatly in their predisposition to develop spotting, and in the physical appearance of the spots. Some forms of spotting and necrosis result from chromosomal instability or genetic combinations, such as hybrid necrosis in wheat and triticale (Figures 277, 278). The disorders are usually observed in the early generations of breeding programs, and affected progenies are eliminated as varietal development progresses.

Figure 276. Physiological leaf spots on barley leaves.

Figure 277. An example of chromosomal instability in triticale (facing page). Note the chlorotic streaks.

Figure 278. A mild form of hybrid necrosis in triticale.

Brown necrosis occurs on some wheat varieties that have derived their resistance to stem rust from such progenitors as Hope and H44. Under certain environmental conditions, a dark brown pigmentation develops on the glumes and peduncles (Figure 279). This condition may be confused with black chaff, caused by the bacteria *Xanthomonas translucens*, or glume blotch, caused by *Septoria nodorum* (Figures 280, 281—also, see pages 102 and 41).

Brittle rachis and preharvest sprouting are genetic problems associated with some strains of triticale. Certain lines tend to have a much higher number of plants with brittle rachis (Figure 282). Delaying selection until 10 days to 2 weeks after the plants have ripened is very effective in eliminating the problem.

Figure 279. Brown necrosis on wheat spikelets. Compare these symptoms with those shown in Figures 280 and 281 (photo: S. Fuentes).

Figure 281. Symptoms of glume blotch, caused by *Septoria nodorum*. Note the brownish grey discoloration that progresses from the base of the awns downward, the distinct brownish border, and the embedded pycnidia.

Figure 280. Symptoms of black chaff, caused by *Xanthomonas translucens*. Note the distinct brownish streaks that begin at the base of the awn.

Figure 282. Brittle rachis, resulting in shattering of the spikelets, in a triticale line having *Secale montonum* as a proginator.

The tendency to preharvest sprouting varies considerably; some strains even tend to sprout on the spike as soon as they mature (Figure 283). Screening for nonsprouting lines among segregating populations (especially during extended, moist harvest periods) has increased the degree of dormancy.

Blast is a common problem in oats, and results in the production of empty spikelets under some environmental conditions (Figure 284). Unbalanced nitrogen-phosphorous ratios, unfavorable temperatures and high soil moisture are among the factors that have been implicated as causes of blast. Varieties differ considerably in the degree of blasting that occurs.

Figure 283. Preharvest sprouting in triticale spikes.

Figure 284. Blast in oats (right) compared with a healthy plant (photo: M. Moore).

Nutrient deficiencies and environmental stresses

Nutrient deficiencies in plants may occur either as a result of low levels of nutrients in the soil, or because nutrients are present in a form that makes them unavailable to the plants. This latter condition may be due to the soil pH, or other abnormal chemical or physical properties of the soil. Some of these can be corrected by the application of commercial fertilizer or lime, while others require foliar sprays or seed treatments with specific chemical compounds.

Nitrogen, phosphorous, and potassium are the three most common nutrients depleted by growing crops. Deficiencies may be determined from soil analysis or from agronomy trials. Nitrogen-deficient crops appear pale green, chlorotic or yellow during early growth stages (Figure 285). As plants develop, their lower leaves die prematurely. Phosphorous deficiency is usually expressed as red, purple, or brownish discolorations on leaves; necrosis starts at the tips of the leaves. Also symptomatic of phosphorous deficiency are less developed root systems (Figure 286). Potassium deficiency symptoms appear somewhat similar to those caused by nitrogen deficiency. The yellowing occurs on the tips of older leaves first, and develops into streaks or general necrosis.

Minor element deficiencies are usually more difficult to identify than N, P, or K deficiencies. Several minor element deficiencies occur that may substantially reduce crop yields. Manganese, copper, and zinc are the most common minor element deficiencies in cereal crops.

Manganese deficiency is relatively easily detected in such crops as oats or peas. Symptoms are most apparent at the seedling stage, especially when growing during cool weather. Irregular spots, light green to grey in color, occur on the leaves. These enlarge, merge and turn in color to straw to light brown (Figure 287). The dying leaves tend to twist or become distorted. This condition is referred to as grey speck, and rapid recovery may be obtained from foliar sprays with manganese sulfate.

Figure 286. Test plots in phosphorous deficient soil. The effects of applied phosphorous on the right (photo: P. Wall).

Figure 285. Symptoms of nitrogen deficiency in wheat. Note the general yellowing.

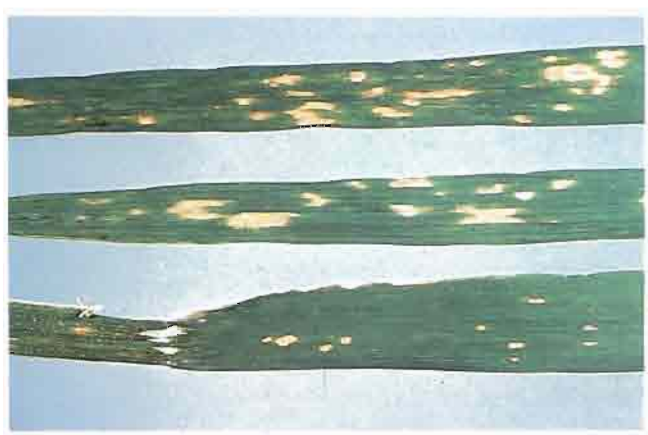


Figure 287. Symptoms of manganese deficiency on oat leaves (photo: V. Cooke).

Copper deficiencies are common in areas of Australia, Europe, Asia and East Africa. The deficient seedlings develop a characteristic necrosis and curling at the tips of leaves (Figure 288). Plants become stunted, pale green and produce bleached spikes. Seed treatment with copper compounds are quite effective in controlling the problem.

Zinc deficiency often occurs on alkaline soils, but is not restricted to them. The problem tends to increase under continuous high phosphate fertilization. Zinc-deficient plants are stunted, chlorotic and have characteristic white stripes on the midribs of new leaves. A reddish brown speckled discoloration occurs on the leaves of some crops. Foliar sprays or seed dressings with zinc sulphate are effective corrective measures.

Soil pH

Many soil related problems result from abnormal pH levels. Cereal crops generally are sensitive to soil acidity, although some varieties of rye tolerate moderate acidity levels (pH 6.0-4.5) quite well. Acid soils tend to reduce the amount of available phosphorous and increase the levels of free aluminum and manganese. Toxic levels of both elements may occur in acid soils (Figure 289).

Soil acidity may be corrected to some extent by the application of agricultural lime. Alkaline and calcareous soils usually have pH levels above 7.5. Though barley is probably the most tolerant, cereal crops as a group generally are not tolerant to alkaline soils (Figure 290). The salt concentration of soil can usually be reduced by providing drainage and leaching.

Figure 288. Symptoms of copper deficiency in wheat. Note the curling of the leaf tips.

Figure 289. Acid soils can drastically retard the growth of certain small grain crops.

Figure 290. Poor drainage can result in a build-up of salts, which inhibit the growth of most small grains (photo: T. Harris).

Soil toxicity

Several mineral elements may occur in soils at levels that are detrimental to plant growth. Aluminum, selenium, copper and boron are among the more common elements to occur in toxic concentrations. Aluminum toxicity usually occurs in acidic soils. Many of the weathered laterite soils have a high aluminum content. Cereal crop varieties differ in their reaction to aluminum (Figures 291, 292). Rye and triticale are usually more tolerant than other cereals. Lime and phosphorous help to reduce the effect of aluminum toxicity. The uptake of copper can be reduced through application of calcium phosphate, and liming reduces the availability of boron.

Drought

Moisture stress is probably the single most limiting crop production factor (Figures 293, 294). Very little genetic improvement in the drought resistance of cereal crops has occurred as a result of plant breeding. Water conservation by improved tillage practices, and the use of drought-escaping techniques (such as early maturing varieties and the careful timing of seeding) have been effective in increasing yields.

Figure 291. Differing reactions of wheat to a high level of aluminum in the soil.

Figure 292. A wheat test plot showing susceptibility to aluminum toxicity (photo: S. Fuentes).

Figure 293. Wheat spikes stressed by severe drought early in the growing season.

Figure 294. Leaf necrosis caused by drought, high temperatures and wind.

Excessive moisture

Water logging, resulting from poor drainage, reduces the productivity of cereal crop plants in several ways (Figure 295). Reduced oxygen availability, reduced nutrient uptake, low soil temperature and high incidence of soil-borne diseases are some of the main factors associated with high levels of soil moisture. Wet soils also tend to build up high levels of soil salts. Improved drainage is the best means of controlling water logging.

Chemical injury and soil residues

Pesticides used to protect crops against weeds, insects and diseases occasionally produce side effects that are injurious to crop plants (Figure 296, 297). The damage usually results when the chemicals are applied in excessive amounts, or to susceptible crops, or at improper stages of plant development. Fertilizers applied too closely to the seed at planting or directly on the foliage causes injury to seedlings of the growing crop. Atrazine is an effective herbicide in maize production, but leaves residues in the soil that are detrimental to small grain cereals (Figure 298).

Figure 295. Early symptoms of water logging in irrigated wheat. Note the yellowing of the leaves.

Figure 297. Spikelet deformation in wheat due to excessive application of 2,4-D herbicide (photo: S. Fuentes).

Figure 296. Grey blotch on wheat, caused by nonselective herbicide spray.

Figure 298. Yellowing and leaf necrosis in wheat, caused by residual herbicide (atrazine).

Frost and hail damage

Several factors influence the degree of damage caused by frost (Figures 299, 300). They include the physiological condition of the plants when the frost occurs, the frost tolerance of the plants and the stage of plant development. Fall-sown crops require hardening at temperatures close to freezing before being able to withstand severe frosts. The inherent frost tolerance of winter cereals is greater in rye and wheat than in barley or oats. Small grain crops are most sensitive to frost damage after heading and during the soft dough stage, and are least sensitive during the tillering stage.

Hail is a hazard in most small grain production areas. Cereal crops tend to recover from hail damage incurred during early growth stages, but the capacity for recovery diminishes after stem elongation. Little regrowth can be expected if hail damage occurs after flowering. Hail damage appears as randomly broken stems, bleached spots on stems and leaf sheaths, and bruised or distorted leaf blades and spikes (Figure 301). Hail damage predisposes cereal crops to outbreaks of various leaf, stem and head blights, especially those caused by bacteria and *Septoria* species.

Figure 300. Sterility of the upper portions of wheat spikes, caused by freezing temperatures during the early growth stages (photo: S. Fuentes).

Figure 299. Frost damage on wheat leaves (photo: S. Fuentes).

Figure 301. Damaged spikes, broken stems and split leaves are typical of the damage caused by hail (photo: S. Fuentes).

Glossary and selected references

acervulus (pl. acervuli)—saucer-shaped, exposed fruiting structure that produces conidia from a compact layer of conidiophores, often surrounded by spines
aeciospores—spores in a chainlike series within an aecium
aecium—the fruiting body of rust fungi
alternate host—a second host species required by some rusts and other organisms to complete their life cycle
apical cell—the cell located at the tip or apex
apices—plural of apex
ascocarp—a mature fungal fruiting body containing asci and ascospores
Ascomycete—a class of fungi that produces ascospores within a saclike ascus
ascospore—a spore produced within a saclike structure (the ascus)
ascus (pl. asci)—an oval or tubular spore sac containing ascospores
aseptate—without cross walls (septa)
asexual spore—a spore produced by cell division, which is capable of developing without conjugation into a new individual
avirulent—nonpathogenic
basal cell (foot cell)—the cell at the foot or base of a conidium or conidiophore
basidial state—the part of the life cycle of Basidiomycetes during which haploid spores are formed
Basidiomycete—a class of fungi that produce sexual spores on a basidium
basidiospore—a haploid sexual spore produced on a basidium
basidium—a short threadlike structure, produced by germinating teliospores, which gives rise to basidiospores or sporidia; also called promycelium in the smut fungi
chlamydospore—a thick-walled, resting spore
chlorosis —yellowing or whitening of the normally green tissue of plants
cirrhus (pl. cirrhi)—a ribbonlike column of spores extruded from the ostioles of pycnidia or perithecia
clavate—club shaped
cleistothecium—a closed fruiting structure containing asci
conidiophore—a threadlike stalk upon which conidia are produced
conidium (pl. conidia)—any asexual spore formed on a conidiophore
diploid—having two sets of chromosomes
echinulate—having short projections on the surface of spores
ellipsoid—shaped like an ellipse or oval
enation—a disease condition in which deformed tissue or galls develop on plant leaves or stems
epiphytotic—the sporadic recurrence of a disease, usually over a wide area and affecting large numbers of susceptible plants
erumpent—the ability of some fungi to grow vigorously from beneath the leaf surface, bursting through and rising above the epidemis
etiology—all the factors contributing to the cause of a disease
extrude—to release or discharge spores
exudate—accumulation of spores or bacterial ooze
facultative parasite—a saprophytic organism capable of behaving as a pathogen
filiform—threadlike
filamentous—threadlike
flexuous—having bends or waves
foot rot—disease symptoms, such as discoloration, necrosis and decay, affecting the roots and basal portions of the plant
fruiting body—the structure from which spores are produced
fungicide—a chemical or toxin that kills or inhibits fungi
fusoid (fusiform)—tapering toward the ends
gall—an abnormal growth or swelling, usually caused by pathogenic organisms, nematodes, or insects
germ tube—the hypha of a germinating spore
globose—more or less spherical in shape
haploid—having one set of chromosomes
haustorium (pl. haustoria)—a specialized structure for extracting nutrients that is formed on some fungal hyphae following plant cell penetration
herbicide—a chemical that is toxic to some herbaceous plants
honeydew—a sticky exudate (containing conidia) produced during one stage of the life cycle of *Claviceps purpurea*
hyaline—transparent or colorless
hymenium—the layer of a fruiting body that produces spores

hypha (pl. hyphae)—a tubular threadlike filament of fungal mycelium
immune—not affected by pathogens
imperfect state—the asexual period during the life cycle of a fungus
inoculum—spores or other disease material that may cause infection
isolate—a culture of an organism
latent—the lack of visible symptoms following infection
lesion—a visable area of diseased tissue on an infected plant
macroconidia—the larger, and usually more common, of the conidia produced by fungi
microconidia—the smaller of the conidia produced by fungi
morphology—referring to the form or structure of an organism
mosaic—a pattern of disease symptoms displaying mixed green and white patches
motile—capable of movement
multiseptate—having several septa (crosswalls)
mycelium (pl. mycelia)—a mass of hyphae that form the body of a fungus
mycoplasma—minute unicellular organisms that are smaller than bacteria, variable in shape, and lack rigid cell walls
necrosis—death of plant tissue, usually accompanied by discoloration
obligate parasite—an organism that develops only on living host tissue
oospore—thick-walled, resting spore of Phycomycetes
ostiole—a pore or opening on pycnidia or perithecia through which spores are released
ovoid—egg shaped
pathogen—a microorganism that causes disease
pathogenic—capable of causing disease
pathogenicity—capacity for causing disease
perfect stage—sexual stage of reproduction
perithecium (pl. perithecia)—a closed ascocarp having an ostiole or opening
persistant—pertaining to viruses that remain infectious in insect vectors for long periods
Phycomycetes—a large class of fungi that range in form from an undifferentiated mass to a well-developed mycelium with many branches
physiological forms—groups within species that are morphologically similar, but differ in pathogenicity or other characteristics
primary inoculum—spores or fragments of mycelium capable of initiating a disease
promycelium—hypha of a germinating teliospore on which basidiospores are produced
pustule—a spore mass developing below the epidermis, usually breaking through at maturity
pycnidium (pl. pycnidia)—a flask-shaped fruiting body producing asexual spores
race—a group of organisms within a species that is distinguished by its pathogenicity
resistance—inherent capacity of a host plant to prevent or retard the development of disease
resting spore—a spore that remains dormant for a period of time before germination
reticulate walls—spore walls having a pattern of superficial lines or ridges
rosette—a very short, bunchy growth
saprophyte—an organism that uses dead organic matter as food
sclerotium (pl. sclerotia)—a dense, compact mycelial mass capable of remaining dormant for extended periods
senescence—the phase of plant growth that extends from full maturity to death
septum—a cross wall or partition
serology—a technique for identifying antigens and antibodies
seta (pl. setae)—short hairlike tufts
sexual spore—a spore produced during the sexual stage of the fungal life cycle
sign—fruiting structures or other features of the pathogen that are visible on the host plant
sorus (pl. sori)—a spore mass erupting through, or replacing, host tissue
sporangium (pl. sporangia)—a compact fungal mass within which spores that are usually asexual are produced
spore—a minute reproductive unit in fungi and lower plant forms
sporidium (pl. sporidia)—the haploid sexual spore developing from a basidium; a basidiospore
sporodochium (pl. sporodochia)—a cushionlike fruiting body producing conidiophores and conidia over its surface
sporulation—active spore production
striate—displaying narrow parallel streaks or bands

stroma (pl. stromata)—a mass of mycelium from which spores develop

susceptible—being subject to infection or injury by a pathogen

symptom—a visible response of a host plant to a pathogenic organism

systemic—the capacity of pathogens or chemicals to spread throughout plants, rather than remaining localized

telium (pl. telia) —pustule containing telisopores

teliospore—a thick-walled resting spore produced by rust and smut fungi

tolerant—the ability of a host plant to develop and reproduce fairly efficiently while sustaining disease

toxin—a poison produced by an organism

transmission—the spread of a virus or other pathogen among individual hosts

urediospore—an asexual spore of the rust fungi

vector—an organism capable of transmitting inoculum

vermiform—worm shaped

virulence—the relative ability of a microorganism to overcome the defenses of a host

viruliferous—carrying a virus

water soaked—appearing wet, darkened, and partially translucent

zoospore—a fungal spore that is motile in water

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