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The purpose of this manual is to assist agricultural field workers in identifying the common diseases of barley. No specific chemical controls are given since these may differ from country to country. Local plant pathologists should be contacted for disease identification and specific control measures.

FIELD MANUAL OF COMMON BARLEY DISEASES

including a section on
BREEDING DISEASE-RESISTANT BARLEY

Department of Plant Pathology
Montana State University
Bozeman, Montana 59717
USA

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Prepared by: H. E. Bockelman, E. L. Sharp, D. C. Sands, A. L. Scharen, D. E. Mathre, T. W. Carroll, J. Riesselman, R. F. Eslick, and other staff of the Dept. of Plant Pathology, Montana State Univ.

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NET BLOTCH
Pyrenophora teres

As the name indicates, net-like lesions typically form on the leaves. These can occur at any stage of growth from seedling to adult. The lesions are usually surrounded by a chlorotic area. Occasionally, the causal fungus produces a brown, spot-type lesion which may be confused with spot blotch caused by *Cochliobolus sativus*. In later stages the net-like lesions tend to coalesce giving a striped appearance to the leaves. Even in this stage the netting can still be observed along the leaf margins. Secondary infections can occur throughout the growing season spread by wind, splashing rain, and dew formation. The fungus survives from one growing season to the next on plant debris. The spores can be seed-borne and provide an additional source of primary inoculum. The disease is found wherever barley is grown, including semi-dry and moist areas. Relatively cool temperatures favor the disease. When the disease develops early in the growth of barley, it can cause considerable yield loss.

Control measures include seed treatments, crop rotation, and resistant varieties.

Fig. 1.
Typical net blotch lesions on seedling leaves.

Fig. 2.
Symptoms on adult leaf.

Fig. 3.
Symptoms on adult plant.

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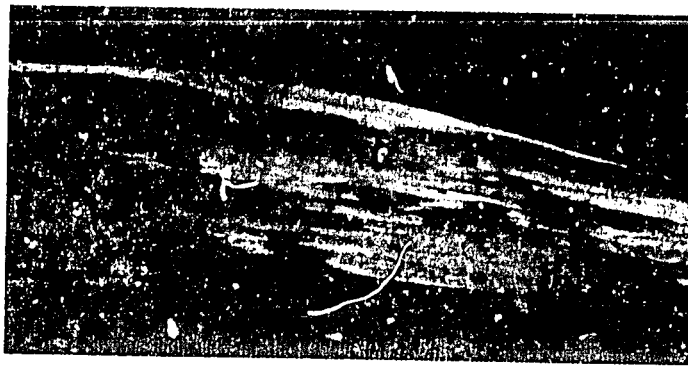

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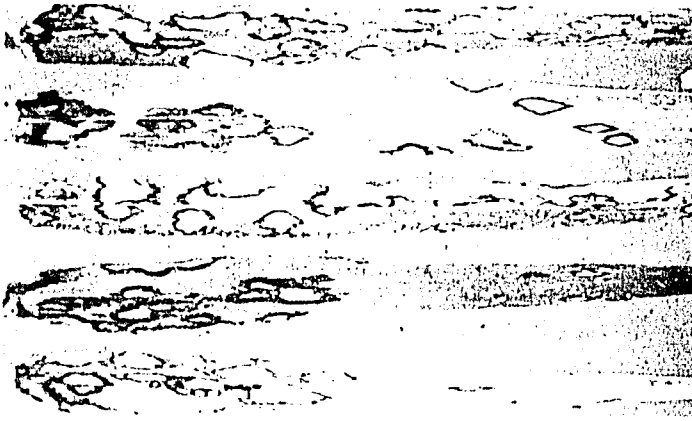
SCALD
Rhynchosporium secalis

The scald-like lesions of this disease are conspicuous on the leaf blades and sheaths. The ovate to irregular lesions are first water-soaked in appearance, but soon fade to a bleached, scalded appearance and are surrounded by a brown-pigmented ring. Spores are produced abundantly during moist periods and secondary spread occurs via wind or splashing rain. The disease may develop rapidly during cool weather and in severe cases may virtually cause defoliation by coalescing of the lesions. Yield losses can be considerable. The fungus persists on dead leaves and other plant residues to initiate primary infection. Seedborne spores may contribute to initial infections.

Means of control are crop rotation, elimination of crop residue, and resistant varieties.

Fig. 4.
Scald lesions on leaves.

Fig. 5.
Field symptoms of scald.



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POWDERY MILDEW

Erysiphe graminis f.sp. *hordei*

Superficial mycelium appears as white to light-gray spots on leaves, sheaths, and glumes. These spots become powdery in appearance as conidia are produced. Later, the spots enlarge and darken as the plants mature. Infected tissue first yellows, then turns brown and dies. In older lesions numerous black, round cleistothecia appear. Powdery mildew usually ceases to increase if the weather is dry and warm. The fungus overwinters as cleistothecia or as mycelium and conidia on green leaves in mild climates. Ascospores, the primary inoculum in cold regions, usually form within cleistothecia in the spring and are windborne to barley. Conidia are produced 10-14 days after infection and thereafter provide secondary inoculum for spread of the disease. Conditions that favor powdery mildew are cool weather with high humidity, but without free water on the leaves. The disease tends to be severe in succulent growth of a densely planted crop that has received heavy applications of nitrogen fertilizer.

The best control is to use resistant varieties.

Fig. 6.
Close-up of powdery mildew symptoms.

Fig. 7.
Symptoms in the field.

SEPTORIA LEAF AND GLUME BLOTCH

Septoria avenae f.sp. *triticea*
(syn. *S. passerinii*)
Septoria nodorum

Leaf lesions are light-brown to yellowish to green, from the center to the indefinite margin. Eventually, lesions merge to form large, necrotic areas. Pycnidia, that look like small, black specks, appear in the tan to brown necrotic area of the lesion. *S. nodorum* may cause chocolate-brown colored lesions on glumes and nodes with gray centers that contain pycnidia. The organism survives as mycelium in live plants and as pycnidia on straw and other plant debris. During cool, wet weather pycnidiospores are exuded from pycnidia and disseminated by splashing and blowing rain to barley leaves. Winter barley in temperate regions or spring barley planted during the autumn in a Mediterranean climate may become infected soon after planting. Secondary inoculum is from pycnidia that mature in 15-20 days when the weather warms. Ascospores are produced in mature barley or in crop debris and may be the source of airborne primary inoculum that causes infection of the newly emerged crop.

Resistant cultivars, destruction of infected residues, and rotation of barley with fallow or legumes, are effective control measures.

Fig. 8.
Symptoms of *S. passerinii* infection.

SPOT BLOTCH

Cochliobolus sativus
(syn. *Helminthosporium sativum*)

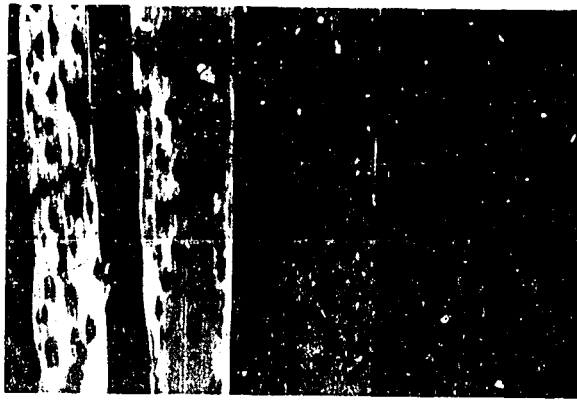
The fungus attacks all parts of the plant. It is one of the organisms involved in dryland root rot. It can attack the leaves from seedling to adult stages. Dark-brown to black discolorations of the seedling leaves can kill the plant, if severe. In later stages the spots coalesce to form blotches that can cover large areas of the leaf blade. Heavily infected leaves dry out and mature early. The fungus can also attack the kernels and cause symptoms called "blackpoint," which appear as numerous dark-brown spots on the germ end. The organism is seedborne and overwinters as mycelium in plant residue and on seedling leaves of winter barley. In the spring, conidia are produced on plant residue and are windborne to barley seedlings. The disease generally is distributed wherever barley is grown and is favored by warm, wet weather.

Resistant varieties, crop rotation, and seed and foliar fungicide treatments reduce the severity of the disease.

Fig. 9.
Spot blotch leaf symptoms.

Fig. 10.
Plants with a severe infection of spot blotch.

Fig. 11.
"Blackpoint" symptoms on kernels.



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STEM RUST (BLACK RUST)
Puccinia graminis f.sp. *tritici*

This organism infects barley, wheat, and some related grasses. Characteristically, the brick-red uredial pustules are produced throughout the growing season. The time period from germination to sporulation is about 10 days. The linear pustules containing uredospores are produced in all above-ground plant parts and eruption of the pustules causes considerable shredding of the epidermis. Extreme dessication may occur with heavy infection. If the infection first occurs before heading and environmental conditions are favorable, complete loss may occur. From a distance, plants have a gray ragged appearance and close-up examination reveals a rough appearance to the culms, which may weaken and break over. As the plant matures black teliospores are formed, which may overwinter and produce basidiospores to infect the alternate host, common barberry, on which sexual recombination and new virulence types may result. The disease is most prevalent at relatively high temperatures combined with dew periods of 8 hours or more. The spores may be disseminated for hundreds of kilometers.

The best control is use of resistant varieties.

Fig. 12.
Stem rust pustules containing uredospores.

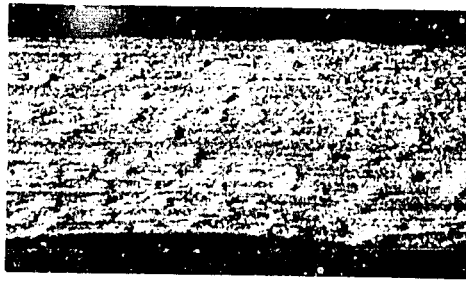
STRIPE RUST (YELLOW RUST)
Puccinia striiformis

On seedling plants the small, yellow pustules containing the uredospores are uniformly distributed over the leaves, but in mature plants the pustules occur in stripes. The disease also occurs on the leaf sheaths and often develops on the heads both inside and outside the glumes. In comparison to the other rusts, stripe rust is particularly favored by low temperatures with adequate infection occurring above 5°C. If the temperature during the dark period exceeds 20°C, disease development is inhibited. In areas of adequate snow cover the fungus may live for several months as dormant mycelium within the leaves. Sporulation occurs as the snow melts and temperatures rise. Infection may also be initiated from uredospores which may be wind-disseminated over long distances.

Resistance is the best means of control.

Fig. 13.
Stripe rust on seedling leaf.

Fig. 14.
Stripe rust on adult leaf.



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LEAF RUST (BROWN RUST)
Puccinia hordei

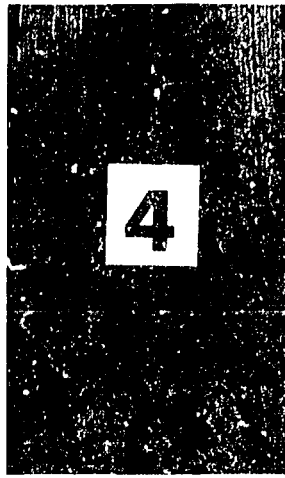
This disease is similar to stem rust in many respects but differs in having much smaller uredial pustules, circular in shape and generally confined to the leaves and sheaths. Leaf rust thrives under the same conditions as stem rust and may become epidemic with extended moist periods or conditions favorable for repeated dew deposition. The uredospores may be airborne for hundreds of kilometers to initiate primary infection. In mild climates the spores may live from one season to the next. As the plants mature or under stress conditions, the black teliospores are formed. On germination the teliospores produce basidiospores which may infect the alternate host, *Ornithogalum* spp.

A number of resistance sources are known which can be used in varietal development.

Fig. 15.
Leaf rust on seedling.

Fig. 16.
Leaf rust on adult plant.

Fig. 17.
Teliospore stage on maturing plant.



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BARLEY STRIPE

Pyrenophora graminea
(syn. *Helminthosporium gramineum)*

This disease originates from infected seed and first appears as long, pale-green stripes on the leaves. These stripes are parallel to each other and extend the entire length of the blade. As the leaves reach full development, these stripes turn brown and leaves sometimes split along the stripes. Infected plants are stunted and heads may not emerge or, if they do, are empty or shriveled. The disease is not apparent in the dormant seed. Upon seed germination, the fungus grows into the sheath that surrounds the leaves of the seedling, then into the first leaf and from it into the next leaf, and so on as the plant grows. Spores of the fungus are blown about the field during and after the flowering period. Kernels of healthy heads become infected when the spores germinate and form mycelia in the pericarp, the seed coat, and between the glumes and kernels. Humid conditions in the heading period favor the infection of kernels. The disease causes considerable damage in North Africa, the Middle East, Northern Europe, and the Southern United States.

Control is by the use of resistant varieties and/or seed treatment with systemic fungicides.

Fig. 18.
Stripe symptoms on seedling.

Fig. 19.
Close-up of leaf symptoms.

Fig. 20.
Healthy vs. diseased plants showing stunting.

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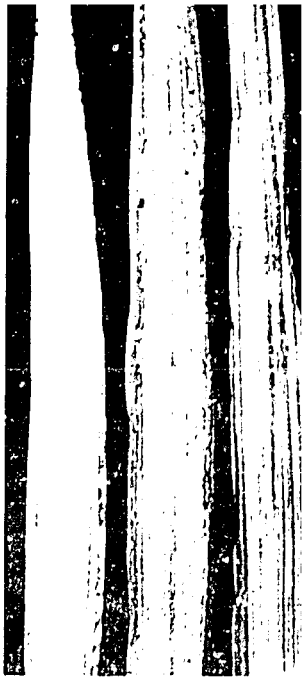

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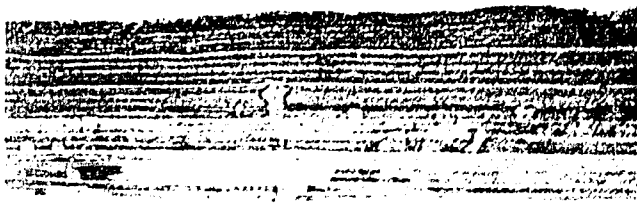
BACTERIAL LEAF STREAK (BLACK CHAFF)
Xanthomonas translucens

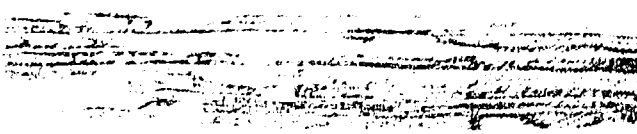
The seedling stage of this disease is an important phase, but is inconspicuous. It consists of greasy, water-soaked streaks on the primary leaf. Obvious symptoms are observed at the boot stage and later. Leaf streaks, about 2 mm wide and running 5 to 20 mm long, develop as water-soaked streaks that later turn yellow. A heavily infected field will have a brownish appearance. A key diagnostic feature is the presence of bacterial slime on the surface of the leaf. This bacterial slime dries to a thin, yellow, translucent layer that can be flaked-off. Seed from severely infected plants may have a shellacked appearance from this slime. The "black chaff" symptoms are a browning of the entire head and are not common, but result from late-season moisture. Seed transmission can be a major source of primary inoculum and sprinkler irrigation can spread the disease rapidly.

The most effective controls are to avoid planting infected seed, destroy volunteer plants and crop residue, and plant tolerant varieties. No resistant varieties are known and no seed treatments are 100% effective.

Fig. 21.
Close-up showing bacterial slime.

Fig. 22.
Field symptoms of bacterial leaf streak.





BACTERIAL LEAF BLIGHT
Pseudomonas syringae

The bacterium, *Pseudomonas syringae*, can often be associated with leaf blight symptoms, either as the only causal agent or sometimes with other pathogens. The symptoms range from small, white streaks and flecks on the flag leaves to severe and confluent water-soaking and bleaching of most leaves of a mature plant. The symptoms appear suddenly, usually following a two- or three-day rainy period with cool nights.

Control is best effected by selecting varieties that are tolerant. No true resistance has been observed.

Fig. 23.
Bacterial leaf blight symptoms on flag leaves (also illustrates bird damage on the heads).

Fig. 24.
Bleaching symptoms of bacterial leaf blight on flag leaves.

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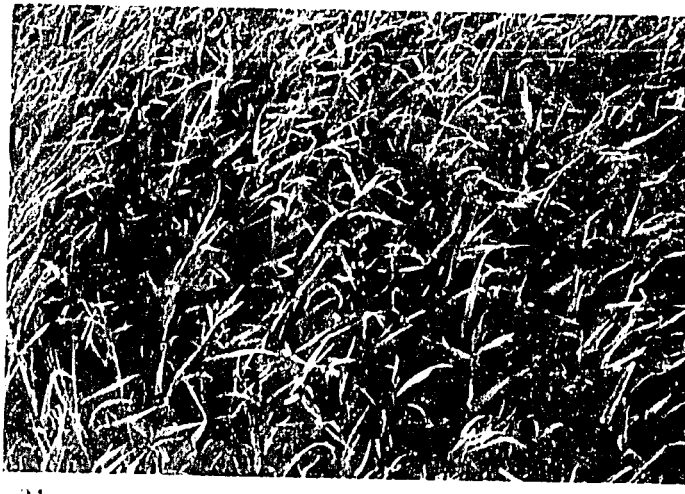

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ERGOT
Claviceps purpurea

Claviceps purpurea infects the open flower and eventually consumes the ovary, replacing the developing seed with a mass of mycelium (sclerotium) or ergot body. The initial infection can be from airborne ascospores or conidia carried by insects or windblown rain. About 7-10 days after infection, a mass of conidia is produced in a sticky, sugary substance called "honeydew." Honeydew is attractive to insects which feed on it and transmit the conidia to nearby open florets. The ergot body which eventually forms falls to the ground where it lies dormant through a 2-3 month cycle of cold, moist weather. It then germinates to produce a stroma in which are embedded perithecia with asci and ascospores. Infection by ascospores is favored by cool, moist weather, but secondary infection from conidia carried by insects can occur under dry conditions.

Control of ergot is accomplished by deep plowing or deep seeding to bury the sclerotia more than 5 cm. In addition, control of grassy weeds (e.g. *Bromus*, *Agropyron*) surrounding barley fields may reduce the quantity of secondary inoculum. Some varieties are much more susceptible than others. The susceptible varieties have a tendency to produce a few sterile florets, which are available for infection.

Fig. 25.
"Honeydew" symptoms of ergot.

Fig. 26.
Ergot bodies (sclerotia) in heads.

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COVERED SMUT
Ustilago hordei

Covered smut survives as teliospores (chiamydospores) on seed or in the soil. Infection occurs as the seedling emerges from the seed and the mycelium moves systemically with the growing point. Smutted heads emerge at about the same time as normal healthy heads. The kernels are replaced by a black mass of teliospores, but a tough membrane remains intact. This keeps the teliospores from being dispersed until harvest when the membrane is disrupted by the threshing operation. Covered smut is found wherever barley is grown, but infection is favored by cool soil temperatures (10-15°C). The spores remain viable on seed or in the soil for long periods of time.

Some varieties are available that are resistant to covered smut. However, because of the effectiveness of seed treatments, many currently-grown varieties are susceptible. The best seed treatments are those that are systemic in action. In addition, avoidance of seeding when soil temperatures are below 20°C will provide a measure of control.

Fig. 27.
Heads infected with covered smut.

SEMI-LOOSE SMUT
Ustilago nigra

Semi-loose smut (black nigra or false loose smut) survives as teliospores (chlamydospores) on seed or in the soil. Infection occurs in the germinating seedling and the mycelium moves systemically with the growing point. Smutted heads emerge at about the same time as healthy heads. The mass of dark, chocolate-brown teliospores is surrounded by a membrane that breaks down rather quickly. The teliospores are spread by wind during the harvesting-threshing operation and cover healthy kernels or fall to the soil. If the teliospores germinate immediately, the mycelium may penetrate the shallow layer of the seed just under the seed coat. When the seed is sown, the resulting seedling becomes infected. Temperatures of 15-20°C and dry soil favor infection.

Semi-loose smut is easily confused with true loose smut in the field. While there are subtle differences in teliospore color, precise identification requires laboratory methods.

Control is best accomplished by use of a systemic seed treatment fungicide. Some of the protective seed treatment materials may give partial control. Resistance to semi-loose smut is known, but those varieties resistant to it are not necessarily resistant to true loose smut.

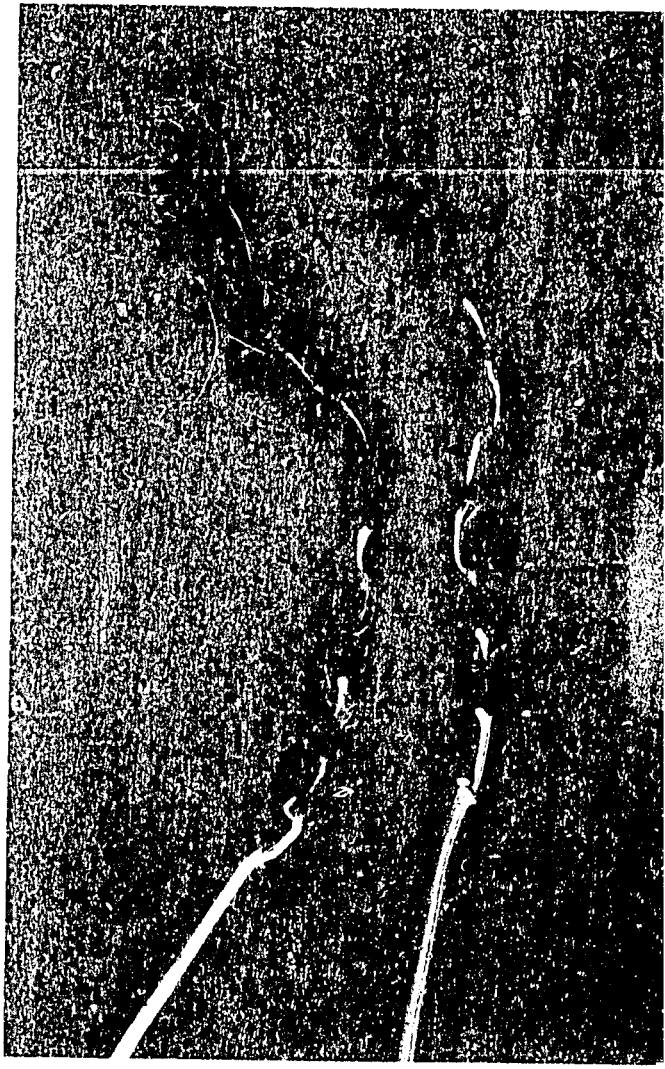
Fig. 28.
Heads infected with semi-loose smut.

TRUE LOOSE SMUT
Ustilago nuda

True loose smut, sometimes called brown loose smut, is seedborne. It survives from one growing season to the next as mycelium in the embryo of infected seed. When the seed germinates, the mycelium moves systemically within the plant and is found just behind the growing point. Infected plants head out 2-4 days prior to healthy plants. The smutted head is surrounded by a very thin membrane which breaks down soon after head emergence. This allows release of the olive-brown teliospores (chlamydospores) which are wind-blown to nearby healthy plants as they head out and flower. Infection is favored by cool, moist weather. The teliospores germinate on exposed stigmas and the mycelium soon invades the ovary where it becomes dormant in the developing embryo. Heads of smutty plants eventually are reduced to a nude rachis, which is all that remains at harvest time.

Varieties differ in susceptibility to true loose smut with some being highly resistant. Besides resistance, control can be accomplished by use of a systemic fungicide seed treatment. Hot water treatment can be used, but requires special equipment and can reduce seed viability if not done properly.

Fig. 29.
Heads infected with true loose smut.



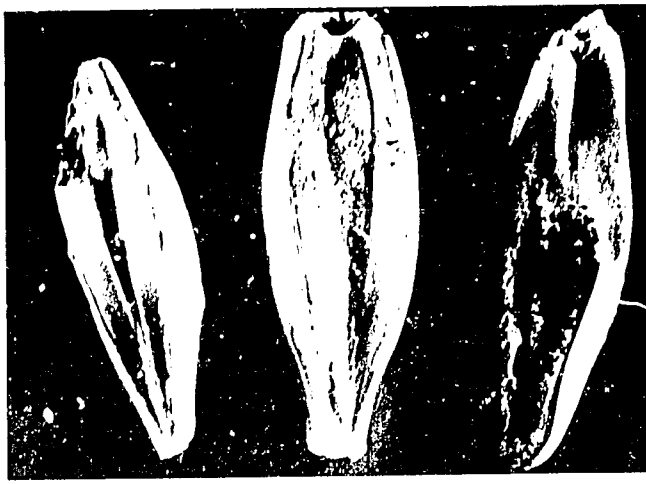
SCAB
Gibberella zeae
(syn. *Fusarium graminearum*)

This disease, sometimes called Fusarium head blight, survives between crops as mycelium and spores on seed and plant residue. In some regions, the *Gibberella* perfect stage develops, with the fungus then surviving as perithecia on residue. This is particularly true where maize is infected. Infection occurs during wet weather or under sprinkler irrigation when the spores land on dehisced anthers. Warm weather is particularly conducive to scab. The mycelium moves from the anthers into the other portions of the head, and can consume the entire head. The mycelium imparts a pinkish-red appearance to infected heads. Diseased spikelets at first are water-soaked, then die and become brown. In severe cases the kernels are shrunken and brown. The interior of the kernels becomes floury and discolored. Infected grain may be harmful when fed to animals (particularly swine) and humans.

Resistance to scab is unknown. Disease severity can be decreased by use of one or more of the following techniques: do not sow barley after maize, wheat, or barley; plow under infected residue; and sow early to avoid heading during warm, moist weather.

Fig. 30.
Scab symptoms on seed.

Fig. 31.
Scab symptoms on head.



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COMMON DRYLAND ROOT ROT

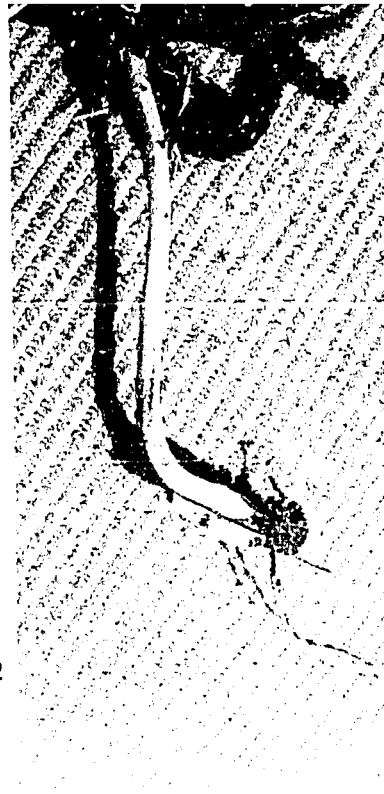
Cochliobolus sativus
(syn. *Helminthosporium sativum)*
Fusarium culmorum
Fusarium graminearum

Dryland root rot is caused by one or more of the fungi above. Without making isolations from infected tissues, it is nearly impossible to identify the pathogen involved. Inoculum is seed and soilborne, oftentimes in association with plant residue. Infection can occur anytime after seed germination and, in some circumstances, may result in seedling blight. More commonly, however, infected plants continue to grow and few above-ground symptoms are observed. Below ground, the first symptoms are brown lesions at the base of sheaths that cover the seedling leaves. As the plant continues to grow, the sub-crown internode is often infected with the appearance of elongate lesions which may become completely necrotic. Severely infected plants may be slightly stunted in height. The most severe effect on the plant is a reduction in the number of tillers. The effects of the disease are most severe when the plants are under drought stress.

Resistance varieties are unknown. The disease severity may be reduced by seed treatments to eliminate any seedborne inoculum and long rotations with non-grass crops, such as legumes.

Fig. 32.
Early stage of common root rot on seedling.

Fig. 33.
Symptoms on sub-crown internodes.



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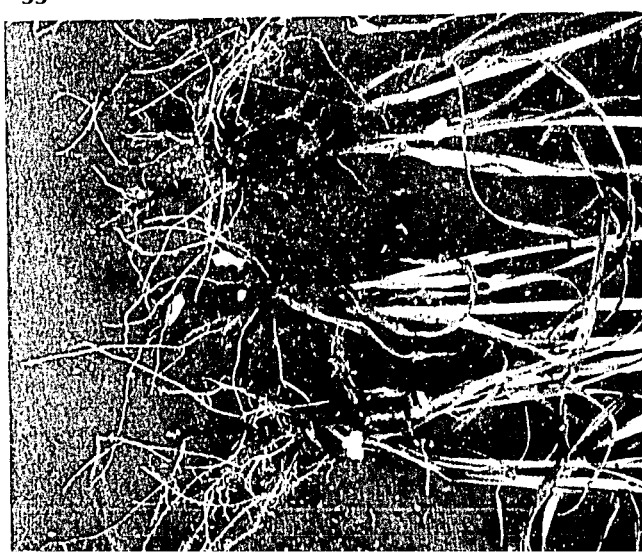


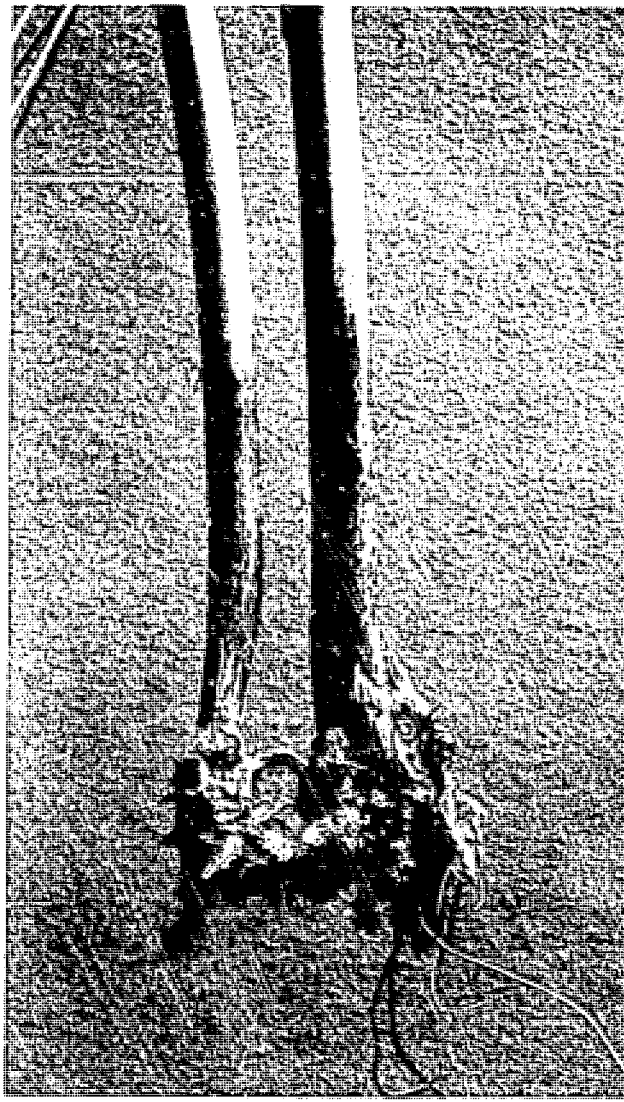
TAKE-ALL

Gaeumannomyces graminis

The take-all fungus survives from one growing season to the next as mycelium in plant residue. Seedlings become infected via their roots growing in the vicinity of infected residue. The initial symptoms are light- to dark-brown lesions on the roots. As infected plants reach the boot stage most of the roots may be dead. Plants not killed by this time may produce heads that often turn white prematurely. Any seed produced is usually shriveled. Plants showing symptoms can be easily pulled from the soil since their roots are highly decayed. A black, scurfy mycelium is often observed near the base of the culms which can be scraped off with a fingernail. Take-all is most severe where soils are alkaline and remain wet for long periods of time. Continuous cropping with members of the grass family (e.g. wheat, barley, rye) also accentuates the damage from this disease.

No resistant varieties are known. Long rotations (3-4 years or more) out of a grass crop will provide some control. However, a decline in severity is often observed if cereals are grown for long periods of time (e.g. 7-10 years or more).

Fig. 34.
Decayed roots caused by take-all.



BARLEY YELLOW DWARF (BYD)

Barley Yellow Dwarf Virus (BYDV)

Ordinarily, leaves of infected barley develop yellowish stripes, blotches, or mottles starting at the leaf tips. Occasionally, the leaf symptoms may be reddish or a mixture of reddish and yellowish colors. Some leaves may have edges that are serrated. Most infected plants are stunted, especially if they become infected when young. Blasting of the florets may also occur if the plants become infected when young. Infections prior to heading may cause severe losses while later infections have little effect on yield. *BYDV* is only transmitted by cereal- or grass-feeding aphids. The virus not only infects barley, but also over 100 additional species of cereals and grasses, including wheat, oats, rice, and maize. Although BYD is worldwide in distribution, it appears to be most destructive in North and South America, Europe, Australia, and New Zealand.

Commercial cultivars and breeding materials with good resistance (tolerance) are available for the control of BYD. Adjusting the time of seeding to avoid aphid activity when the plants are young is also an important means of reducing the severity and losses due to the disease.

Fig. 35.
Yellow and red leaf symptoms of BYD.

Fig. 36.
Yellowing symptoms on leaves.

Fig. 37.
Aphids, vectors of *BYDV*.

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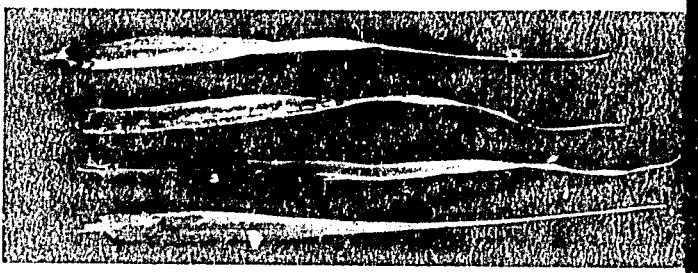

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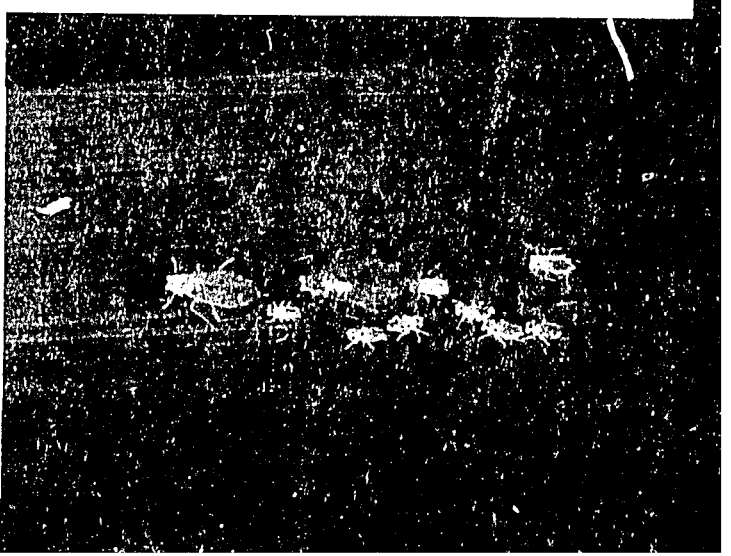


BARLEY STRIPE MOSAIC (BSM)

Barley Stripe Mosaic Virus (BSMV)

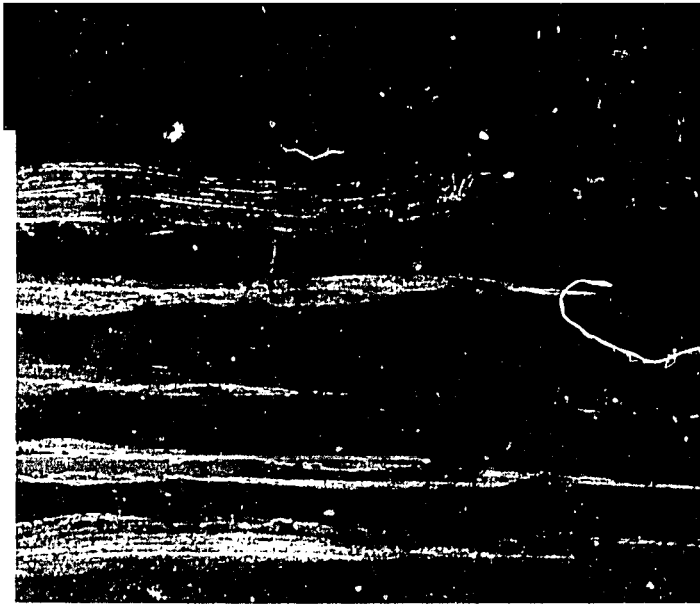
Leaf symptoms due to *BSMV* consist of yellow and brown striping and streaking. Sometimes two diagonal stripes occur together on a leaf. These stripes extend outward from the stem and meet towards the tip of the leaf, forming a V-shape or chevron. Leaf symptoms on most seedlings consist of yellow-green mottles or mosaics, and yellowish streaking and striping. Many *BSMV*-infected plants are dwarfed, if they become infected prior to heading. In some barleys, both male and female sterility occur in infected plants. Yield losses are usually greatest if plants become infected when young. Actually, most plants become infected during germination and emergence since the virus is seedborne. *BSMV* is also mechanically transmissible, and can be spread from plant to plant by leaf rubbing. The virus infects primarily barley; occasionally it infects wheat and oats. *BSMV* occurs worldwide, but has been most severe in Canada, Denmark, Japan, and the United States.

The most practical control measure is to plant virus-free seed. Roguing field plants with symptoms of BSM is also helpful in controlling the disease.

Fig. 38.
Striping, streaking, chlorosis, necrosis symptoms of *BSMV*.

Fig. 39.
Yellow and brown striping and streaking symptoms of *BSMV*.

Fig. 40.
BSMV symptoms on seedling.



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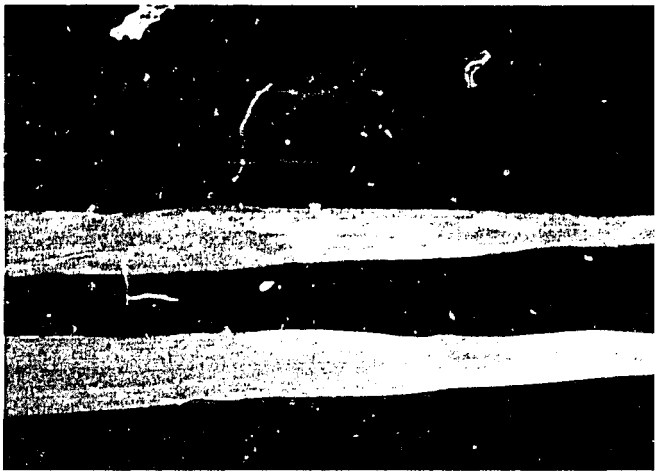
ASTER YELLOWS

Aster yellows is presumably caused by a mycoplasma-like organism. Symptoms caused by the aster yellows agent in infected barley consist of yellowish blotches on the leaves, sterility, and stunting. These symptoms resemble those of barley yellow dwarf, except that they usually develop more slowly. The aster yellows agent is only transmitted by certain species of leafhoppers. Moreover, only low percentages of feeding insects transmit the agent. It infects over 300 different plant species, including wheat, oats, other grasses, vegetables, and ornamentals. Aster yellows is known to occur in North America, Europe, and Asia. It is generally not economically important.

No control measures have been recommended for aster yellows. All barley genotypes tested for reaction have been susceptible. Insecticides to kill the leafhopper vectors have not given good control of the disease.

Fig. 41.
(Top to bottom) Healthy, aster yellows symptoms, yellow dwarf symptoms.

Fig. 42.
Healthy plant vs. aster yellows-dwarfed plant.



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NEMATODES

Several different nematodes are capable of parasitizing barley. These include: root gall nematode, *Subanguina radiciola;* oat cyst nematode, *Heterodera avenae;* root knot nematodes, *Meloidogyne* spp.; and root lesion nematodes, *Pratylenchus* spp.

Barley infected with nematodes appears yellow and stunted, often mimicking water stress. Usually, only isolated pockets or low areas in fields are affected. Root gall and root knot infestations can cause noticeable swellings or thickening of roots. Microscopic examination of the swellings or galls reveals the presence of nematodes or larvae. Root lesion nematodes penetrate the root system and move through cortical cells. These wounds provide entry for fungi, which hastens root decay.

Controlling established nematode populations is difficult. While soil fumigation is effective the cost is prohibitive for lower value crops, such as barley. Rotation with non-host crops will reduce nematode populations. Since many nematodes utilize weeds as a secondary host, a good weed management program will help minimize damage.

Fig. 43.
Roots of plant infested with *H. avenae.*

NON-PATHOGENIC BARLEY DISORDERS

Disorders caused by non-pathogenic entities are very common in barley. Environmental stresses, nutritional imbalances, genetic abnormalities, physical injuries, and non-target chemical damage are among the common causes of abiotic barley diseases.

Non-parasitic leaf disorders are more common in barley than in other small grains. These disorders are often triggered by environmental conditions unfavorable for the variety being grown. The actual expression of non-parasitic leaf diseases varies considerably depending upon the weather pattern, nutrient availability, and the particular variety.

Unfavorable weather conditions also cause non-pathogenic disorders. Frosts can cause necrotic banding on young leaves or sterility in older plants. Hot weather can result in blasting, poor seed production, stunting, and leaf burning.

Misapplication of pesticides is another common problem. Injury induced by the volatile phenoxy herbicides can occur if applied at the wrong growth stage. Symptoms include trapped heads, sterility, and wrinkled leaves.

Properly managed barley fields, which includes good fertility, excellent water and residue management, the use of adapted varieties, and proper planting dates and procedures, will normally encounter few non-pathogenic disorders.

Fig. 44. Frost damage on seedlings.

Fig. 45. Hail damage.

Fig. 46. Salt damage.

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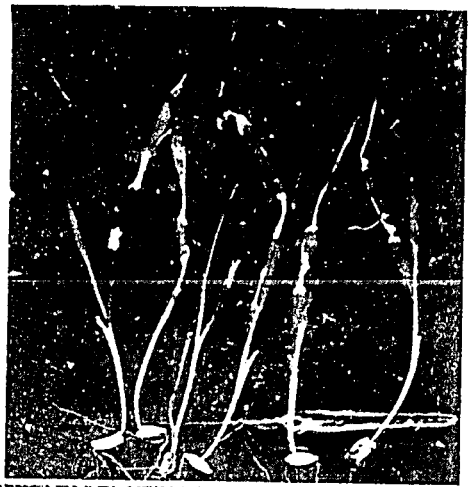

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BREEDING DISEASE-RESISTANT BARLEY

Barley, an important food and feed crop in many regions of the world, is subject to numerous diseases that limit yields and reduce the quality of the grain harvested. Genetic resistance is usually the most satisfactory solution to a disease problem in barley. Disease-resistant varieties add little or nothing to the cost of production, and require no action or additional knowledge on the part of the farmer.

Breeding for disease resistance can be conveniently divided into two general phases: (1) germplasm resource development, and (2) exploitation (variety development).

Germplasm Resource Development

Development of germplasm resources is often given a low priority by the urgent need to develop varieties. However, adequate attention to germplasm resources, which are the parental materials for variety development, is essential for the long-term success of a breeding program.

A search through the diversity of germplasm represented in world collections, regional collections, and local breeding programs is almost always successful in locating adequate sources of resistance. This material is often useable directly in variety development by backcross or pedigree breeding (discussed later). However, this germplasm can be modified and made more valuable by recurrent selection procedures. A method commonly employed in cross-pollinated crop plants, recurrent selection utilizes populations of plants to intensify particular characters through cycles of selection and recombination. In order to use this method with a

self-pollinated crop, such as barley, an easy means is needed to obtain large numbers of crosses. In barley this is accomplished with genetic male sterility (a single, recessive gene). The other components of a recurrent selection population for disease resistance are lines with resistance and varieties with good agronomic traits. The lines with resistance are chosen to include as many different genes as possible. Varieties with good agronomic traits are necessary to compensate for the generally poor plant types of the resistant lines and, ultimately, to allow selection of desirable plants from the population. These varieties are chosen on the basis of a proven performance record or for some specific agronomic trait. The recurrent selection cycle generally consists of two parts: selection for resistance and recombination. Selection for resistance should be conducted in disease nurseries in a number of locations, if possible. This diversity of locations is important to the development of multi-genic (polygenic) resistance. Assuming that the virulence of the pathogen is different in each location, then selection of resistant plants in these locations will be selection for different resistance genes. Seed harvested from the disease nurseries is mixed and planted in a recombination nursery (preferably in isolation). The F_1 seed is harvested from the male sterile plants which are open-pollinated at random. This effectively scrambles the population and combines genes selected in the various locations. With continued cycling of the population a high percentage of the plants will be multi-genic for resistance. Large numbers of plants (and genes) can be handled in a population with a small input of labor. Recurrent selection populations should be considered long-term germplasm resources to be continually exploited and continually improved.

Exploitation

To most effectively utilize resistance a certain amount of basic information concerning the host and the pathogen is required. It is desirable to know the extent of variation in the pathogen and the amount and kinds of resistance available in the host. A collection of isolates of the pathogen from a number of locations can be tested against a group of host lines that differ in resistance to determine the variability in the pathogen. This, in turn, gives important information on the effectiveness of particular resistance genes. In other words, does a gene provide resistance to all isolates or only some or few? This information is then used to determine which sources of resistance would be most useful in a breeding program. Likewise, it is useful to determine inheritance of resistance by crossing a resistant line to a susceptible variety and to other resistant lines and then examining segregation in the F_1, F_2, F_3, and BC generations. This type of information is helpful in determining the best way to incorporate the resistance in varieties.

Often, obtaining basic information (as described above) is not feasible or desirable when expertise, equipment, and manpower are limited. Even with limited resources, however, it is still possible to breed disease-resistant varieties. The single-most important requirement is the consistent presence of the disease at high enough levels to allow separation of susceptible and resistant plants. Some diseases that cause significant damage one year, may be absent or present in only small amounts another year. In order for a breeding program to proceed with expediency, the disease must be present every year. Therefore, it may be necessary to induce disease by artificial inoculation techniques. Much can be done in this regard with limited manpower and without large amounts of equip-

ment. With a basic understanding of the life cycle of the pathogen and the environmental conditions required for the disease, relatively simple procedures can be utilized to obtain epiphytotics. For example, some foliar diseases, such as scald and net blotch, can be enhanced by harvesting infected straw and spreading it in next year's disease nursery.

There are basically two breeding methods to utilize disease resistance genes: (1) backcross breeding and (2) pedigree breeding. The choice of a method will depend on the number of genes controlling resistance and the agronomic qualities of the resistant parent.

(1) **Backcross Breeding.** When adequate resistant is not found in an adapted variety, but only in lines that are unsuitable agronomically, the method of choice is the backcross. The recurrent parent is an adapted variety and the backcross parent is a line with demonstrated high levels of resistance. Backcross breeding is most useful in transferring resistance governed by one, two, or maybe three genes. However, there can be exceptions. If the resistance character can be precisely identified in segregating populations, then the number of genes becomes less important. In practice, however, identification can seldom be precise enough. For instance, one gene may be masked by another. Therefore, in the transfer process one or more genes may be lost unless special care is taken.

Generally, six backcrosses are sufficient to return to the phenotype of the recurrent parent. The number of backcrosses can be reduced to four or five if F_2 and F_3 populations are grown after the first and third backcrosses. The advantage of this alternative are, first, that selection for the type of the recurrent parent, if based on F_2 and F_3 populations of moderate size, is equiva-

lent to one or two additional backcrosses, and second, selfing provides the large populations required for effective selection of the resistance being transferred.

(2) **Pedigree Breeding.** When both parents can contribute to adaptation, yield, or quality, then the pedigree method of handling segregating generations should be used. In the pedigree method superior types are selected in successive segregating generations and a record maintained of parent-progeny relationships. Selection, based on individual plants, begins in the F_2 generation. Every F_2 individual will be different from every other. In the F_3 and F_4 generations many loci will be homozygous and family characteristics will begin to appear. In these generations selection is practiced for both the best plants and the best families. In the F_5 and F_6 generations most loci will be homozygous, thus, selection will be mainly among families. In subsequent generations selection is almost exclusively among families and requires increasing use of replicated testing.

Application of Backcross and Pedigree Breeding to the Development of Varieties with Multiple Gene Resistance

Backcross- and pedigree-derived varieties that are resistant due to one or a few genes are frequently rendered susceptible by a changing pathogen. A single resistance gene employed in a widely-grown variety gives a selective advantage to mutants of the pathogen that are virulent. The usual procedure has been to replace a variety that has succumbed to a new race with a variety that is resistant to the new race, once again forcing the pathogen to mutate. This move-countermove aspect, analogous to a

chess game, is disturbing since the number of resistance genes is limited. This problem can be alleviated by deploying numerous genes for resistance simultaneously, as added obstacles to the pathogen. This can be accomplished by varieties with multi-line or multi-genic resistance.

A multi-line variety is one with several components differing only in the resistance genes. They are most useful against foliar, air-borne pathogens, such as the rust fungi. Although one or two components may be susceptible to a particular race of the pathogen, the remaining resistant components will reduce the spread and severity of the disease by reducing the quantity of inoculum available for reinfection. A multi-line variety is developed through a series of backcrosses. The source of each selected resistance gene is backcrossed to the same recurrent parent, which results in components that are essentially identical except for the resistance genes. These components are combined mechanically to produce the multi-line variety. If one or more components are kept in reserve, then a multi-line variety can be quickly changed if the virulence of the pathogen changes.

A variety with multi-genic resistance contains numerous genes for resistance in every plant. The usefulness of this type of resistance is based on the supposition that many genes present a formidable obstacle to the pathogen. While a typical pathogen reproduces in such large numbers that multiple mutations for virulence to overcome any number of resistance genes can occur, this often upsets the homeostatic balance in the pathogen population, thus preventing such multiple mutations from become widespread. There is, also, evidence to indicate that multi-genic resistance often has a "horizontal" component, which results in a resistance broader than the total contributions of the indi-

vidual genes.

As discussed earlier, recurrent selection populations are an effective means of producing plants with multi-genic resistance. To utilize material in these populations single plant selections can be made each generation. The pedigree method is the ideal way to evaluate these selections. As a population is improved agronomically and with continuing selection for resistance, the chances of selecting good material also improves.

PHOTO CREDITS

Figures 1,2,3,4,5,6,9,11,12,13,14,15,16,17,18,19, 20,21,22,23,24,25,26,27,29,30,31,32,33, 34,35,36,37,38,39,40,44,46, Front Cover, Back Cover: Dept. of Plant Pathology, Montana State Univ.

Figure 7: P. S. Baenziger, USDA, Beltsville, MD.

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Figure 45: L. Hockett, USDA, Montana State Univ.

COVER PHOTOS

Front: Scald, net blotch, and bacterial leaf streak on field plants.

Back (Top Left): Teliospore stage of leaf rust.
(Top Right): Leaf showing symptoms of bacterial leaf streak.
(Middle): Heads infected with covered smut, semi-loose smut, and true loose smut (top to bottom).
(Bottom): Plant with barley yellow dwarf symptoms.

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