rots (Great Plains); seedling blight (Great Plains).

Indian ricegrass (*Oryzopsis hymenoides*): Seedling blight (far West); common root rot and crown rot (far West); rhizoctonia rot (scattered); snow molds (Pacific Northwest); seed rots (general); root necrosis and crown rots (far West).

Kentucky bluegrass (*Poa pratensis*): Dollar spot (Northeast); brown patch (general); foot rot, *Helminthosporium* (general).

Lovegrass (*Eragrostis*): Sometimes seedling blight and common root rot (Southwest and Plains).

Orchardgrass (*Dactylis*): Root necrosis (general); seed rot (general); seedling blight (scattered).

Panicum grasses: Seedling blight (Great Plains); root necrosis (Plains and South).

Redtop and bents (*Agrostis*): Seed rots (general); rhizoctonia rot (general); damping-off (coastal areas).

Ryegrasses (*Lolium*): Common root rot (general); root necrosis (general); rhizoctonia rots (general); seed rots (general).

Timothy (*Phleum pratense*): Seedling blight (general); seed rots (general); root necrosis (general).

Wheatgrasses (*Agropyron*): Seedling blight (Western States); common root rot and crown rot (Western States).

Wild-rye grasses (*Elymus*): Seedling blight (general); common root rot and crown rot (general).

Stipa grasses: Seedling blight (Plains and far West); common root rot and crown rot (Western States).

**Seed Disorders of Forage Plants**

**John R. Hardison**

Seed diseases of forage plants are relatively few, if we do not count the smuts. The four seed disorders I discuss here are different from the seed-borne diseases, in which the causative agents primarily infect leaves, stems, or roots, but also may attack seeds.

Seed diseases are particularly important when they reduce the supply of seed needed to plant forage crops, lawns, or turf. In two diseases—ergot and grass seed nematode disease—the grass seeds are replaced by the sclerotia and galls, which are poisonous to animals.

**Blind Seed Disease** of perennial ryegrass, *Lolium perenne*, apparently became established in the United States about 1940, although it has been a seed production problem in New Zealand since 1932. Poor germination of seed of domestic perennial ryegrass alarmed growers in Oregon in 1943 and led to positive identification of the disease. But three-fourths of the Oregon crop by then had become infested, and more than one-third of the 1943 seed could not be certified.

The causal fungus, *Phialea temulenta*, was identified in France on cereal rye in 1892, and was identified on perennial ryegrass in New Zealand in 1942. Since then the pathogen has been recognized on perennial ryegrass in England, Ireland, and Scotland; probably it can be found on the grass wherever climate permits infection.
Heavily diseased crops have many seeds that fail to germinate. Such dead seeds were referred to as blind seeds in New Zealand, and the malady was named blind seed disease. It is hard to tell infected seeds from healthy ones unless the lemma and palea are removed. Then one can see the shriveled, soft, pasty appearance of diseased seeds. Healthy seeds have hard, plump, purple endosperms.

Diseased seeds reach the soil by pre-harvest shattering of the crop, planting diseased seed, feeding infested seed or screenings, harvesting operations, and, in unharvested areas, by the natural dispersal of seed. The blind seeds remain dormant during the winter. In spring, when perennial ryegrass flowers, the small-stalked, cup-shaped spore-producing organs (apothecia) arise from the overwintered blind seeds and forcibly discharge the primary spores (ascospores). The ascospores are showered on the ryegrass flowers and infect the developing seeds. Asexual spores are produced in a slimy matrix surrounding infected seeds. These secondary spores can infect other developing seeds when rain and insects spread them from head to head. Badly infected seeds that overwinter at or near the surface of the soil produce spore cups the following spring and repeat the cycle. The entire life cycle is confined to the seed. Infected seeds are not toxic to livestock. The disease is important only when the grass is grown for seed.

To provide a basis for control, each sample of cleaned seed of perennial ryegrass entered for certification in Oregon is examined for disease. Growers then are advised through their county agents of the amount of disease in all fields. Plowing before May 1 is recommended for fields that appear too badly infested to produce a profitable crop of seed. The procedure makes it possible for each seed grower in Oregon to know how much disease is in his field and what to do each year. He can avoid unprofitable crops and use the land for spring grain or leave it fallow to reduce weeds. Thorough burning of the straw and stubble after harvest gives good control for a year.

Disease-free seed is selected after harvest and approved for planting in new seed-production fields. The blind seed fungus dies after 24 months in dry storage; aged seed therefore is also safe to plant. The planting of any seed more than one-half inch deep, with complete soil coverage, prevents emergence of the apothecia.

Much heavily infected seed is spread on the field with the straw during combine harvesting, as such seed is lighter in weight than healthy seed. Because the disease is perpetuated primarily by infected seed that is left on the soil, precautions should be taken during harvest so that light as well as heavy seed is removed from the field.

It is helpful also to destroy infested perennial ryegrass screenings, prevent heading in pastures until after July, plow clean to bury infected seeds deeply, have good soil drainage, and plow all ryegrass on a farm at the same time to prevent spread of disease from old fields to new plantings nearby.

Anther mold was first discovered in Ladino clover in 1947. A seed blight of white clover had been seen in Scotland before 1928, but the identity of the causal fungus was unclear until Mary Noble, of the University of Edinburgh, named it in 1948. By comparing data with Dr. Noble, we found that the seed blight of white clover in Scotland and the anther mold in Ladino clover in Oregon are different stages of the same disease.

Anther mold is caused by Sclerotinia spermophila, a fungus that can live inside the clover stems and leaves. Infected plants look the same as healthy plants. The disease is evident only in the flowers and seeds. Infected flowers bear gray, fuzzy anthers, on which the fungus produces spores that largely replace the pollen—hence the name anther mold. Bees carry the fungus spores to healthy flowers. Infection of
young seeds in the flower is the only known way the fungus can enter the plant.

Infected seeds are shriveled and have a dull-brown or gray-pink color. Healthy seeds are plump and bright yellow or reddish brown. Most infected seeds will not germinate. The fungus therefore appears to be largely self-eliminating, and the disease has not been economically important. If a less lethal strain of the fungus would appear by mutation or other genetic change, however, more diseased seeds could grow and produce infected plants, which could furnish inoculum for infecting healthy seeds. The disease might then rapidly become a serious problem in the production of seed of white and Ladino clover.

The disease seems not to affect the vegetative growth of infected plants. Its dissemination has been retarded by the use of modern seed-cleaning machinery, which removes the lighter infected seed.

Grass seed nematode disease, caused by *Anguina agrostis*, is a main disorder of chewings fescue and Astoria and Seaside bents west of the Cascade Mountains in the Pacific Northwest, where those grasses are grown for seed. A similar nematode infests seeds of buffalo grass in the Great Plains. Other related nematode species infest seeds sporadically in species of *Calamagrostis*, *Danthonia*, *Elymus*, *Holcus*, *Sporobolus*, and *Stipa* throughout the United States. I found a few heads of orchardgrass infested with a seed nematode in Oregon in 1947.

The life history of the grass seed nematodes is like that of *Anguina tritici*, which causes the eelworm disease of wheat and rye in Southeastern States. Only the seed is infested, so that utilization of a grass for lawn and turf purposes is not affected by the presence of the grass seed nematode.

Striking symptoms occur in bents of *Agrostis*. The seeds are transformed into purple to black galls that may be much longer than the healthy seed. In orchardgrass the seed panicles are greatly malformed. In chewings fescue the symptoms are less easily seen.

Each gall contains many nematodes, which leave the galls after fall rains and migrate to grass leaves. Eventually they work their way between the folded leaves near the growing points. When the grass panicles develop, the microscopic roundworms penetrate the ovaries and stimulate the plant to produce the galls, within which many eggs are laid. Thus the cycle is repeated.

Satisfactory methods have been devised for control of the grass seed nematode in chewings fescue in the Pacific Northwest. Clean seed for planting seed-production fields can be had from dry-land districts or by disinfesting seed with a specific gravity separator, using air flotation. Chewings fescue seed weighing 21 pounds per bushel from such seed-cleaning machinery contained no nematode galls. Burning fescue fields after seed harvest is effective because it destroys many of the nematode galls in the grass stubble or in the straw. Vacuum machines, which were developed to pick up seeds on bare soil, could be helpful in removing the lightweight nematode galls from a grass field if they are adapted to work in the thick stubble. Crop rotations that use crops other than susceptible grasses effectively starve out the grass seed nematode.

Many cases of fatal poisoning of sheep, cattle, and hogs have occurred in western Oregon from feeding chewings fescue screenings that contained nematode galls. When heavily infested screenings of chewings fescue were fed to sheep and rats in controlled experiments at the Oregon Agricultural Experiment Station, the animals were killed. Nervous phenomena in sheep and gangrenous developments in rats, similar to ergotism, also appeared. The poisonous principle is not known.

Several species of ergot fungi attack grasses in the United States.
Claviceps cinerea infects curly-mesquite (Hilaria belangeri) and tobosa grass (H. mutica) in the Southwest. Claviceps tripsaci occurs on eastern gamagrass, Tripsacum dactyloides, in the Southeast. Claviceps paspali is restricted to members of the genus Paspalum. The most common and important species, Claviceps purpurea, causes the ergot disease in about 150 different grasses throughout the United States.

Ergot has retarded seed production of grass desirable for regrassing programs. Seed production of big bluegrass, Poa ampla, was discontinued in Union County, Oreg., because of such heavy infestations that harvesting and cleaning were difficult. Production of seed of some grasses in the Great Plains is often a failure because of sterility due to ergot. Most of the seed often is blighted, although only a few sclerotia may develop.

Ergot poisons livestock that graze heavily diseased grass inflorescences and feed on contaminated seed screenings. Substances in the ergot sclerotia cause abortion, nervous disorders, blindness, and paralysis. In gangrenous ergotism there is a sloughing of the hoofs, tips of ears, and tail; shedding of teeth and hair; and death.

Ergotism in man is no less severe. The disease is contracted usually by eating rye bread made from contaminated flour. Epidemics in humans were common in the Middle Ages.

Modern methods of grain cleaning permit the removal of ergot to within the tolerance of 0.3 percent by weight set by the Federal grades and pure food laws. This has almost eliminated the disease in man, although local epidemics occurred in France and India in 1951.

The ergot fungus attacks only the developing seeds of grasses. It has economic importance when the susceptible grasses are grown for seed or are allowed to flower and seed before grazing or cutting for hay. Ergot is of no importance in seed for planting lawns or other clipped turf.

The first sign of ergot infection appears at flowering time when a sweet exudate, "honeydew," is noticeable. Infested heads feel sticky when drawn through the hand. The exudate, which contains the asexual spores (conidia) of the fungus, attracts flies and other insects. Conidia carried by such insects to healthy heads or spread by rain in the same head start new infections. As the infection progresses, purple or blue-black horny bodies (sclerotia) develop in place of seeds. These sclerotia may fall to the ground or be harvested with the seed. When planted or left on the soil they produce specialized sporebearing organs that eject spores into the air. The spores are then carried by wind to grass flowers and infect the young seeds, thereby repeating the disease cycle the following spring.

Many grasses are susceptible to ergot, Claviceps purpurea, and wild grass may serve as sources of infection for grass seed or grain crops.

Control of ergot involves avoidance of the airborne primary spores or the secondary spores spread by rain and insects. This can be accomplished by planting nonsusceptible crops, deep plowing to bury the sclerotia too deeply for emergence of the spore organs, clipping grass before seed heads bloom, and eradication or preventing the heading of grasses in fence rows or other adjacent waste areas. Deep planting, permissible with the larger grass seeds, will prevent emergence of the sporebearing organs from the ergot sclerotia. However, selection of ergot-free seed, removal of ergot sclerotia by seed cleaning with specific gravity separators, or aging the seed for 2 years would be preferable.

Some grasses, such as the bents, produce their flowers in midsummer when the soil is too dry in many areas for development of spore organs from sclerotia. Under such conditions, infection depends entirely on spores carried by insects that have visited the spore slime on grasses infected earlier; control of the offending insect carriers could prevent ergot infection.
Ergot in perennial ryegrass has been materially reduced in western Oregon by burning the fields after the seed harvest. The fire destroys many of the ergot sclerotia in the straw and stubble and also prevents regrowth head formation in the late summer and fall. Such late-formed spikes are usually abundantly infected even in the years that are dry.

Although infection starts in the spring on the early-flowering grasses, the heaviest ergot infestations occur in the fall in many areas. The phenomenon is due to recurrent heading of many grasses, which become infected by insect-carried spores. This is important, because of the increase in sclerotia that furnish inoculum for infection of grass-seed crops the following spring. Such heavy infestation in late-grass heads is especially dangerous to livestock, because leaf growth is at a low ebb and grazing is heavy on the infected inflorescences.

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Endothia parasitica: a, Cross-section of a fungus mass (stroma) showing perithecia; b, saclike membranes (asci), in which ascospores, c, are produced.

Some of the 125 Rusts of Grasses

George W. Fischer

About 125 different species of rusts attack grasses in the United States. Nearly 400 species of grasses are among the hosts of the rusts.

Some of the rusts attack only one or a few grasses. Others can attack a great many. Stem rust, Puccinia graminis, for example, has been recorded on nearly 200 species of grasses in the United States. Among the rusts, which have such large numbers of hosts, we find innumerable strains or races, which look alike, even under the microscope, but differ in their comparative ability to attack species or varieties of grasses.

Some of the rusts of grasses are often destructive on the cereals. Most of the so-called cereal rusts have numerous grass hosts; probably the cereal rusts originally were grass rusts that found susceptible hosts among the cereals during the centuries they have been cultivated by man. However, since the cereals—wheat, oats, barley—are really only grasses whose seeds are large enough and nutritious enough to warrant intensive cultivation as food, it is only to be expected that there would be a great deal of similarity between the rusts that attack grasses and those that attack cereals.

The rusts are microscopic fungi and are strictly parasites. They are among that parasitic classification known as obligate parasites; that is, they can grow only on a living plant. Indeed,