THE UNIVERSITY OF MAINE Cooperative Extension

Bulletin #1014

Ergot of Barley and Other Small Grains

By Steven B. Johnson, Extension crops specialist, University of Maine Cooperative Extension

Ergot is a disease of cereal crops and grasses caused by the fungus *Claviceps purpurea*, which infects developing grains. *Claviceps purpurea* has a very broad host range of more than 300 species in over 50 genera of grasses, including wheat, rye, barley, and all of the cool-season forage grasses. Rye and triticale are very susceptible to the pathogen, wheat, and barley less so, and oats very rarely. Many common weed grasses are common hosts of the pathogen and serve as a pathogen reservoir.

The greatest concern regarding ergot is the alkaloid toxins produced by the fungus and their effect on humans and animals. "Ergotism" develops after ingesting ergot-contaminated grain, or grain products (like bread), processed from ergot-contaminated grain. Convulsions, severe pain, hallucinations, gangrenous limbs, and more, have been reported as symptoms in severe cases. In the Middle Ages, regular outbreaks of ergotism occurred in which many people died. Ergotism was later known as "St. Anthony's Fire," as monks of the newly formed order of St. Anthony built hospitals for ergotism treatment. *Claviceps purpurea* has been used during childbirth and for migraine treatment and was the source for the first isolation of the alkaloid LSD.

Barley and other small grains represent important rotation crops for Maine potato producers. Such crops are grown for livestock feed, malt production, and other uses. Owing to the toxicity of the *Claviceps purpurea* fungus, ergot infection results in crop loss and crop rejection. The presence of ergot could render a farmer's entire grain crop worthless.

Biology

The ergot pathogen overwinters as black ergot bodies that are actually sclerotia (rough black masses of hardened fungal mycelium) of *Claviceps purpurea*. Ergot bodies may be introduced from planted seed or neighboring grasses or may remain from a previous cereal crop. Moist soil is required for germination of sclerotia, which generally begins in spring. Once sclerotia germinate, dry conditions may interrupt the process, but germination will continue when wet conditions resume. As the ergot bodies germinate, they form tiny spore-producing, mushroom-like structures. These structures produce wind-dispersed spores that infect grass or grain flowers. Grain and grass are most susceptible just before the flowers are fully open.

Ergot bodies in grain heads.

Within 5 days, infected flowers produce "honeydew," the secondary phase of the disease. The honeydew is a sticky asexual spore-filled liquid that oozes from infected flowers and contains a large number of ergot spores. Rain splash, visiting insects, and direct contact all spread the infective spores for as long as flowering continues. Cool weather favors increased disease levels by extending the flowering period and thereby increasing the period of infection. Moisture is not required for pathogen transmission during the honeydew phase of the disease.

Within 10 days of flower infection, sclerotia begin to form and the infected seed ovary is replaced with a hardened dark ergot body. Grain heads may have more than one ergot body. At this point, the typical ergot symptoms are evident, including the presence of an elongated ergot body, frequently sticking out farther than the unaffected grain in the head.

Symptoms

The easily recognizable sign of the disease is the presence of dark sclerotia or ergot bodies. These replace the grain in cereal and grass heads and can be readily identified at harvest and in infested grain seed. There are tolerances for ergot levels and they vary from crop species to crop species. Grain lots above the ergot tolerance are described as "ergoty" and may be rejected.

Management

Little can be done to control ergot once it is present in the crop. Prevention is the best management, but it must be done before ergot symptoms have been observed. No barley varieties are resistant to *Claviceps purpurea* and there are no fungicides to control the disease. Planting ergot-free seed from reputable sources will prevent introduction or re-introduction of the fungus into the field. Sclerotia of *Claviceps purpurea* can be cleaned from seed, but broken pieces of sclerotia still contain toxic alkaloids.

Tillage that buries residue at least 4 inches below the soil surface keeps the sclerotia from germinating. Since sclerotia don't survive for more than a year, crop rotation with a nonsusceptible host will help control ergot. Broadleaf plants are not affected by the pathogen and make a good rotation crop.

Weed grasses in or near fields are can be a source of inoculum. Mow or otherwise destroy stands of grasses adjacent to cereal fields. This is most effective if done before grasses flower. If ergot is higher on field edges, these areas could be harvested separately and the grain destroyed.

Recent efforts have correlated ergot with copper-deficient soils. In such soils, the addition of copper may help control ergot infection. The addition of copper is far more effective with wheat or barley than with rye.

Call 800.287.0274 (in Maine), or 207.581.3188, for information on publications and program offerings from University of Maine Cooperative Extension, or visit extension.umaine.edu.