



ERGOT OF CEREALS AND GRASSES

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Ergot attacks cereals and both wild and cultivated grasses throughout the world. Only the flowering parts and developing kernels are affected. The disease is caused by numerous species and races of fungi in the genus *Claviceps*, the most common and damaging one being *Claviceps purpurea*.

Ergot is common on rye. Most varieties of barley, wheat, and triticale are susceptible. Recent outbreaks have occurred on the open-floreted, male-sterile wheat lines used to produce hybrid seed. Oats are the least susceptible of the small grains. Some 200 or more wild and cultivated grasses may be infected, especially where they are permitted to form seed.

Symptoms and Disease Cycle

Ergot bodies or sclerotia develop in place of certain seeds or kernels in a spike or panicle. These sclerotia are dark purplish-black, horny, spur-like structures, up to four times as large as normal kernels, that protrude well beyond the glumes (Figure 1 and 2a). Because affected flowers produce ergot sclerotia instead of kernels, the grain yield is reduced. The heads containing sclerotia commonly have blasted kernels and empty florets. Such heads are usually shorter and weigh less than healthy ones. Certain grasses in the Great Plains occasionally fail to produce seed because of sterility caused by ergot.



Figure 1. Ergot of wheat.

The first sign of ergot infection appears at or soon after flowering, when infected florets exude a sugary slime called "honeydew." The creamy to golden-colored, sticky droplets that ooze from infected flowers contain large numbers of asexual spores (conidia) of the *Claviceps* fungus (Figure 2h). The honeydew, with its rotten-smelling odor, attracts a wide variety of flies and other insects that congregate about infected heads. After feeding on it, the insects become contaminated with conidia and inadvertently transport the microscopic spores to healthy flowers, where new secondary infections take place. More than 40 species of insects are known to frequent rye flowers and transport or feed on the conidia of the ergot fungus. Splashing rain and contact also carry the conidia to healthy florets in the same spike or panicle and to adjacent plants. New infections occur as long as the cereal and grass plants bloom and are susceptible.

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As infection proceeds, sclerotia develop in place of kernels. These sclerotia are large, purplish-black, horny, and somewhat curved (Figure 2a). The sclerotium or ergot consists of a hard compact mass of fungal tissue which in cross section appears white or faintly purple. The sclerotia mature about the same time as the healthy seeds. The sclerotia may fall to the ground around harvest time or be combined with the grain. When sown with the seed or left on the soil surface, the sclerotia germinate during the spring and early summer producing one to 60 slender, flesh-colored stalks on top of which round stromatic heads form in which perithecia are produced (Figure 2b). Large numbers of long (0.5 to 2.5 millimeters tall) sexual spores (ascospores) (Figure 2e-g) form in the perithecia (Figures 2c-d). The ascospores are forcibly discharged into the air and are disseminated by wind, insects, and splashing rain. Spores contacting a flower stigma of a grass or cereal plant germinate and penetrate the ovary within 24 hours. Within 5 days, conidia form on the ovary surface (the "honeydew" stage), completing the disease cycle.

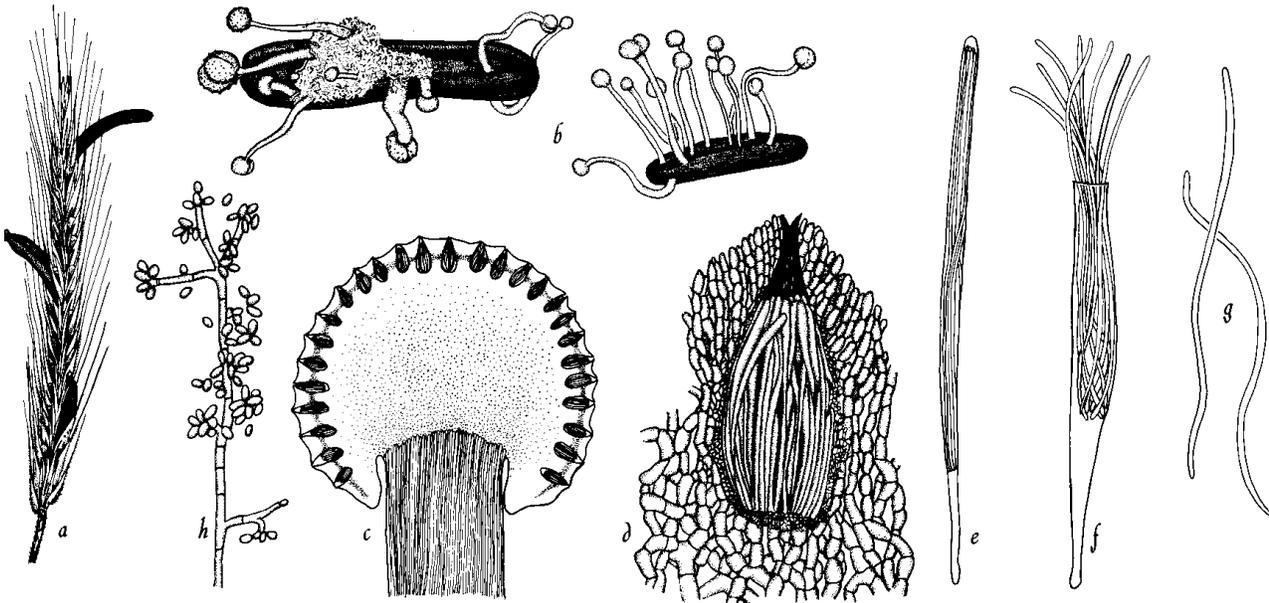


Figure 2. *Claviceps purpurea* the ergot fungus. A) Sclerotia (ergots) protruding from a head of rye; b) two germinated sclerotia with a number of perithecial stromata; c) longitudinal section through a perithecial stroma showing numerous embedded perithecia around the outer margin; d) section through a perithecium showing numerous asci; e) ascus with eight ascospores; f) ruptured ascus discharging ascospores; g) two ascospores; h) conidia produced on mycelium. Drawing by Lenore Gray.

Plants infected with another disease (e.g., barley stripe mosaic virus) are more susceptible than healthy plants to infection by the ergot fungus. Ergot is favored by cool, wet weather which prolongs the flowering period and the formation of conidia. The susceptibility of the florets is greatest just before anthesis, decreasing immediately afterward.

Control

1. **Rotate cereals and grasses with nonsusceptible crops such as corn, legumes or sorghum – plants outside the grass-grain family – for one year or longer.** Fortunately, the ergot sclerotia do not remain viable for more than one year.
2. **Plow under grass and cereal stubble (2 inches or more) deeply and cleanly after harvest.** Plowing buries the sclerotia too far down for the spore-bearing organs (perithecia) to form and reach the soil surface to liberate their ascospore.

3. **Select and plant only ergot-free seed.**
4. **Remove ergot sclerotia by cleaning the seed – using modern, gravity-type, grain-cleaning equipment Or, plant high-germinating seed that is at least one year old.**
5. **Mow wild, escaped, and cultivated grasses before flowering.** Eradicate grasses or prevent the heading of grasses in grain fields, fence rows, ditch banks, headland, roadsides, or other similar areas adjacent to cultivated fields. Heavy grazing of grass pastures before heading also reduces the threat of ergot poisoning among livestock.
6. **No commercial varieties of barley, rye, wheat, triticale, or cultivated grasses have been developed that are resistant to ergot.** Varieties with exposed florets which remain open for relatively long periods are the most susceptible ones.

The grasses growing in Illinois that are quite susceptible to ergot include various bluegrasses, fescues, brome-grasses, mannagrass, meadow foxtail, orchardgrass, quackgrass, reedgrasses, redtop, ryegrasses, squirrel tail, timothy, wheatgrasses, and wild rye.

ERGOTISM

The disease is of unusual importance because of the medicinal properties of extracts of the sclerotia and because of the relation of ergot to a disease of man and animals known as ergotism.

Grain that exceeds market tolerances for ergot is discounted in value, since it presents a toxicity risk if eaten by humans or animals. Ergot poisoning primarily affects cattle that eat the diseased grass heads or cleanings from contaminated grain. The ergot sclerotia contain a number of alkaloids in varying amounts. These alkaloids induce a contraction of the smooth (nonstriated) muscle fibers and the small blood vessels, particularly in the hind legs. The first sign is lameness, which may appear 2 to 6 weeks after exposure to an ergot-infested pasture or field of grain. A high body temperature with an increased pulse and respiration rate may accompany the lameness. The joint may swell and become tender. After a few days, a loss of feeling and then dry gangrene develop. One or both hoofs or any lower part of the limb may slough off. The tip of the tail and the tips of the ears may be affected in the same way. Feeding livestock with ergoty grain can also lead to reproductive failure. The ergot toxins, some of which are related to LSD, accumulate in the body. Hence, their detrimental effects sometimes occur after long periods of low-level ingestion.

With the increased acreage of fescue seeded for grazing in Illinois, lameness in the hind feet of cattle is becoming frequent. The toxic substance appears to have a vasoconstrictive (contractive) effect like that of ergot poisoning. When ergot poisoning or fescue foot is suspected, a veterinarian should be consulted.

Ergotism is no less severe in man than in cattle, but modern methods of cleaning grain make it possible to remove ergot to within the tolerance of 0.3 percent by weight, as established by federal grades and pure food and drug laws. In the United States, grain is classed as "ergoty" when it contains more than this percentage of ergot sclerotia by weight. Although most of the sclerotia can be removed with modern grain-cleaning equipment, it is often difficult and expensive to reduce the percentage to the permitted level.

People usually contract ergotism by eating rye bread made from contaminated flour. Human epidemics were common in the Middle Ages, when ergotism took thousands of lives. At present, the disease has almost been eliminated in man, although local epidemics are reported every few years somewhere in the world.

MEDICAL USES

The ergot sclerotia contain a group of alkaloids that in controlled dosages are of special medicinal value in stopping blood flow, in the postpartitive contraction of the uterus just after childbirth, and in the treatment of migraine. Ergot is supplied commercially for pharmaceutical companies from the drier, west-central states or is imported from the Mediterranean area. The only ergot used in medicine comes from rye. In epiphytotic years, a rye crop may be more valuable for its ergot content than for its grain.