PP-551 (revised)

ERGOT

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Ergot, caused by the fungus *Claviceps purpurea*, is a disease of cereal crops and grasses. The disease causes reduced yield and quality of grains and hay and also causes a livestock disease called ergotism, if infected grains or hay are fed. The disease cycle of the ergot fungus was first described in the 1800s, but the connection with ergot and epidemics among people and animals was known several hundred years earlier. Ergot's medicinal applications and animal poisoning properties first called attention to this plant disease. Human poisoning was common in Europe in the Middle Ages when ergoty rye bread was often consumed.

Ergot occurs to some extent every year on cereals and grasses in North Dakota. The disease generally is more prevalent in rye and triticale than in other cereals, but significant losses have also been reported in spring wheat, durum, barley, and other small grains. Although the crop loss caused by this disease is important, the effects of the ergot's alkaloid toxins on man and animals is of much greater significance.

ERGOT AS A PLANT DISEASE

Symptoms and Signs

The most common sign of ergot is the dark purple to black sclerotia (ergot bodies) found replacing the grain in the heads of cereals and grasses just prior to harvest (Figure 1; a) rye field, b) wheat, c) various grasses). The ergot bodies consist of a mass of vegetative strands of the fungus. The interior of the sclerotia is white or tannish-white. In some grains, ergot bodies are larger than the normal grain kernels, while in other grains, such as wheats, grain kernels and the ergot bodies may be similar in size (Figure 2). A larger size separation between the sclerotia and the grain kernel simplifies the removal of ergot bodies during grain cleaning.

Prior to development of the sclerotia bodies, the fungus develops a stage in the open floret (flowering head part) commonly called "honey dew." The "honey dew" consists of sticky, yellowish, sugary excretions of the fungus which form droplets on the infected flower parts.

Figure 1a. Rye field.

Figure 1b. Wheat.

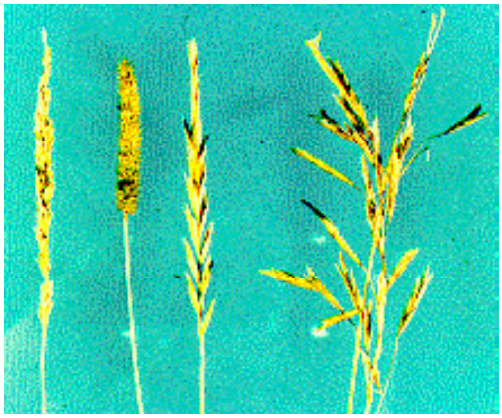

Figure 1c. Various grasses.

NDSU EXTENSION SERVICE
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JUNE 1998
Reviewed and reprinted May 2002

Disease Cycle

Sclerotia produced in small grain fields or grassy areas fall to the ground and survive on the surface of the soil. In the spring and early summer, the sclerotia germinate to produce tiny mushroom-like bodies approximately the size of a pin (Figure 3). Spores (ascospores) formed by a sexual process in these bodies are shot into the air, and wind currents may carry them to grain heads.

The first infections are from these wind-borne sexual spores. The spores land on open flowers, germinate, and the fungus then invades the embryo of the developing kernel (Figure 4). Soon a yellow-white, sweet, sticky fluid ("honey-dew") exudes from the infected flowers. The fluid contains a large number of asexually produced fungus spores (conidia). Many species of insects visit the "honey-dew" and become

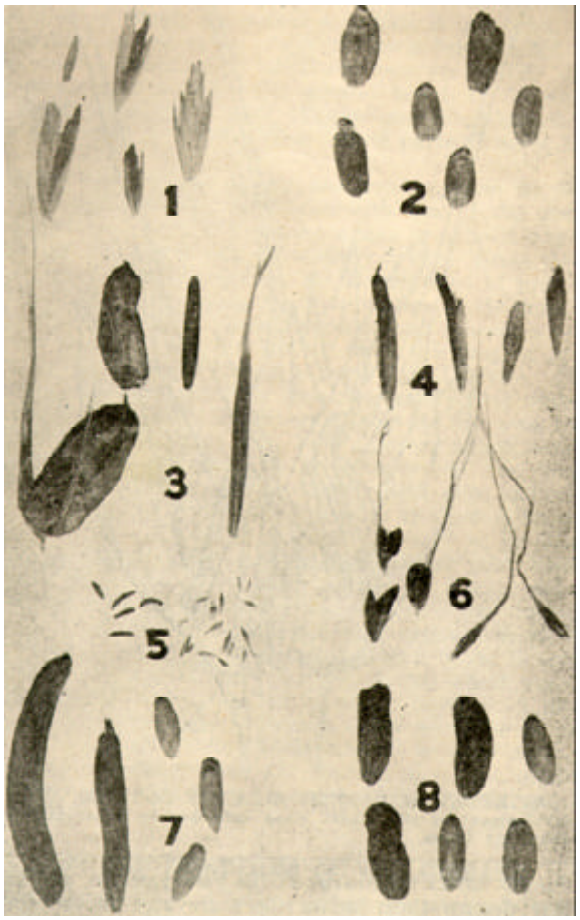

Figure 2. Ergotized and healthy seed of various grains and grasses to show relative sizes of ergot bodies. Enlarged approximately one and one-half times.

1. **Quack grass** *(Agropyyron repens)*
2. **Common wheat**
3. **Wild rice** (*Zizania aquatica*)
4. **Brome grass** (*Bromus inermis*)
5. **Red top** (*Agrostis alba*)
6. **Feather grass** (*Stipa viridula*)
7. **Rye**
8. **Durum wheat**

Source: Ergot and Its Control (Bulletin 176, 1924)

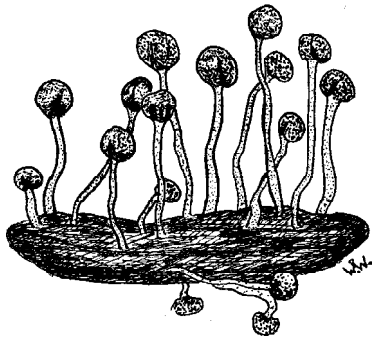

Figure 3. Germinated ergot sclerotium. The rounded heads on these stalks produce spores that infect flowers in summer. Source: Ergot and Its Control (Bulletin 176, 1924)

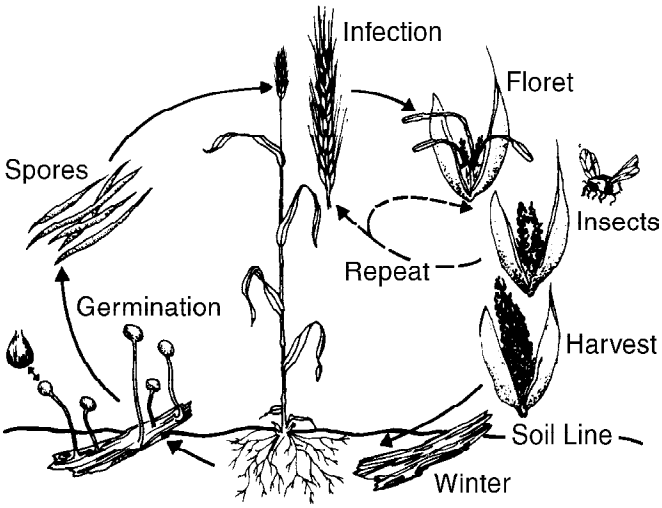

Figure 4. Disease cycle of ergot.

contaminated with the fungus spores. These insects visit other grass flowers and spread the fungus, in a repeating cycle that continues as long as the florets are open. Spores may be transferred to other grain heads by rain-splash and direct contact, as well.

Once the fungus becomes established in the florets, it grows throughout the embryos and replaces them, later producing the dark sclerotia. Many sclerotia fall to the ground before harvest and overwinter on the soil surface, serving as potential sources of spores the following year (Figure 4).

Environmental Conditions that Favor the Disease

Ergot develops more abundantly during wet seasons. Wet weather and wet soils favor germination of the ergot bodies, and cool, wet weather during flowering favors development of the "honey-dew" stage. Dry weather during flowering is detrimental to spore production and germination. Susceptibility to infection among crops increases with any condition that may prolong flowering or cause sterile florets.

Host Range

The same fungus, *Claviceps purpurea*, can produce ergot in many hosts. Rye, triticale, wheat, durum, barley, oat, quack grass, crested wheat grass, brome grass, foxtail, rye grass, orchard grass, timothy, wild rye, and other grasses serve as ergot hosts. Differences in susceptibility among cultivars within a grain crop may exist but are not well documented.

Economic Importance

Ergot can cause a direct yield loss in proportion to the number of kernels infected. In North Dakota, as much as 10 percent loss has been reported in wheat, while losses of 5 percent are common in rye.

Ergot also may affect the market grade of a grain. In the commercial grain trade, wheat or durum is graded as "ergoty" when it contains more than 0.05 percent by weight of the ergot sclerotia; rye is graded as "ergoty" when it contains more than 0.3 percent by weight of ergot sclerotia, and triticale, oat, or barley are "ergoty" when they contain more than 0.1 percent.

Most of the sclerotia can be removed from ergoty grain with modern cleaning machinery, unless broken pieces are present or the sclerotia are similar in size to the grain. However, it is costly and often difficult to remove enough sclerotia to meet the legal standards, and traces which have proven toxic to livestock are left.

Control of Ergot is Small Grain Crops and Grasses

- Rotate cereals and grasses with nonsusceptible crops for one year or longer. The ergot sclerotia usually do not survive in the soil for more than one year. Therefore, summer fallow or crop rotation to a non-cereal crop for at least one year will help reduce ergot.
- Deep-plow fields which have a severe ergot infestation to bury the sclerotia. The ergot sclerotia will not germinate if buried more than 1 inch deep.
- Plant only ergot-free seed to avoid introducing or reintroducing the fungus into the field.
- Eradicate or prevent wild grasses from reaching heading in fields, rocklands, headlands, ditches, and fence rows. Mow wild and escaped grasses and pastures, or graze pastures before they flower, to prevent ergot infections.
- Resistant commercial varieties of wheat, barley, rye or cultivated grasses are not available. However, some differences among varieties may occur, and those with long flowering periods may be more frequently infected.

EFFECT OF ERGOT ON ANIMALS

Ergot is toxic to animals. Animals consume ergot by eating the sclerotia present in contaminated feed. All domestic animals are susceptible, including birds. Cattle seem to be the most susceptible.

Two well known forms of ergotism exist in animals, an acute form characterized by convulsions, and a chronic form characterized by gangrene. A third form of ergotism is characterized by hyperthermia (increased body temperature) in cattle, and a fourth form is characterized by agalactia (no milk) and lack of mammary gland development, prolonged gestations, and early foal deaths in mares fed heavily contaminated feed. Which form of ergotism is manifested depends on the type of ergot consumed and the ratio of major toxic alkaloids present in the ergot: ergotamine, ergotoxine, and ergometrine. *Claviceps purpurea*, the common cause of ergot in North Dakota, is usually associated with gangrenous ergotism. *Claviceps paspali*, an ergot of *Paspalum* spp. of plants, is most commonly associated with central nervous derangement. *Paspalum* is a water grass distributed in pastures in southern states, but is not commonly found in North Dakota.

The responses of animals consuming ergot are usually quite variable and are dependent on variations in alkaloid content, frequency of ingesting ergot, quantity of ergot ingested, climatic conditions under which ergot grew, the species of ergot involved, and the influence of other impurities in the feed such as histamine and acetylcholine.

Animal Clinical Symptoms

Symptoms of convulsive ergotism include hyperexcitability, belligerence, ataxia or staggering, lying down, convulsions and backward arching of the back. Symptoms of gangrenous ergotism involve the extremities of the animal including the nose, ears, tail, and limbs . Early signs usually start in the hind limbs (Figure 5). Lameness may appear from two to six weeks after first ingesting ergot. There may be pain, stamping of the feet, and coolness of the affected areas. If ergot consumption continues, sensation to pain is lost in the affected areas and an indented line appears between normal tissue and gangrenous tissue. This gangrenous tissue is called 'dry gangrene.' Eventually all tissue below this line will slough. Besides the limbs, other extremities involved can include the tail and ears. The tips and distal areas of the extremities will also slough.

Animals fed large amounts of ergot over time lose portions of their hooves, ears (Figure 6), tails, combs, and wattles. Spontaneous abortion and loss of milk has occurred in cows and sows fed even small amounts of ergot. Ergoty feed should not be fed to breeding females.

Figure 5. Early signs of gangrenous ergotism usually start in the hind limbs.

Figure 6. Animals fed large amounts of ergot over time lose portions of their ears or other extremeties.

Diagnosis of Ergot Poisoning

A diagnosis of ergot poisoning is based on finding the sclerotia in the feed or pasture and whether the animals are exhibiting symptoms of ergotism. Extraction and detection of ergot alkaloids also may be done if ground feed is suspect.

Treatment of Ergot Poisoning

The only treatment for erotism is to remove the ergot-contaminated feed or remove the animals from the contaminated pasture. If nervous signs are present, call your veterinarian for medical advice and supplemental therapy such as pain control. If dry gangrene is present, the affected portion will be sloughed. There is nothing to reverse this process. If secondary infections or open wounds are present, call your veterinarian for appropriate therapy.

Prevention and Control of Ergot Poisoning

Prevention is based on feeding feed and forage that are free of ergot. For pastures, graze the infected fields before seed heads begin to flower. The ergot is contained in the ovary of the flower. Commercially prepared feeds will rarely contain ergot. Non-commercial grain (home grown) should be screened prior to feeding (and planting to prevent contamination of future crops). Special care should be taken in feeding screenings. If feeding ergoty grain is unavoidable, the amount of sclerotia should be reduced to an amount less than 0.1% (by weight) of the feed through mixing with 'clean' grain. Again, ergoty feed should not be fed to breeding females.

Photo Credits

Figure 1. a) Marcia McMullen b) Vernyl Pederson
c) R. W. Stack; NDSU Plant Pathology Dept.
Figure 2. Wanda Weniger
Figure 3. Wanda Weniger
Figure 4. NDSU Ag. Communication
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Figure 6. NDSU Veterinary and Microbiological Sciences

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