

Ergot of Small Grain Cereals and Grasses and its Health Effects on Humans and Livestock

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Ergot is a fungal disease of the inflorescence, or seed heads, of cereals and grasses. Ergots, compact masses of fungal mycelium also known as sclerotia (singular: sclerotium), are produced instead of normal grain (*Figure 1*). This Extension Circular (EC) provides information on the disease and its consequences. The EC is divided into two sections. Section 1 describes the disease as it relates to wheat production including management recommendations. Section 2 presents information on the health effects of ergot on humans and livestock including recommendations on how to mitigate these health effects.



Figure 1. Ergot sclerotia protruding from rye heads.

■ 1. Ergot in Wheat Production

1.1 Economic importance

Ergot can cause 5-10 percent yield loss in small grain cereals. It

can also cause losses in forage grasses grown for seed production. Apart from actual yield reduction in small grain cereals, additional losses are incurred when contaminated grain (*Figure 2*) is discounted or rejected at the elevator. Wheat or durum wheat is graded as “ergoty” when it

contains more than 0.05 percent by weight of ergot sclerotia; barley, oat, and triticale are graded as “ergoty” when they contain more than 0.1 percent by weight of ergot sclerotia; and rye is graded as “ergoty” when it contains more than 0.3 percent by weight of ergot sclerotia. Extreme



Figure 2. Ergot-contaminated wheat grain.

losses in wheat due to ergot are rare. In 1921, significant losses occurred in durum wheat in the northwestern United States. In 2011, ergot caused economic losses in south central and southeast Nebraska. The major significance of the disease, however, is that the sclerotia are poisonous to humans who eat contaminated food, and animals that consume contaminated feed.

1.2 Causal agent

In small grain cereals and grasses, ergot is caused by the fungus *Claviceps purpurea*, which belongs to a group of fungi known as ascomycetes. Several other species of *Claviceps* also are causal agents of ergot in cereals. For example, *C. sorghi*, *C. sorghicola*, and *C. africana* are causal agents of sorghum ergot. Sclerotia of *C. purpurea* vary in size (2-20 millimeters or 0.08-0.8 inches) and may be up to 10 times larger than normal grain. They have a hard, protective, black to dark purple rind on the outside and a white to gray interior. Moisture content of sclerotia collected from rye and durum wheat in the U.S. was found to range from 3.3 percent to 6.7 percent. Sclerotia also contain

chemical compounds known as alkaloids that render them poisonous.

Sclerotia can survive on the soil surface or in the soil for at least one year. The ability of sclerotia to germinate is considerably reduced after one year. Research showed that under field conditions, germination was 83-

92 percent in one-year-old sclerotia and 43-47 percent in two-year-old sclerotia.

C. purpurea produces two types of spores. Sexual spores known as ascospores are produced in stromata (singular: stroma; compact masses of specialized vegetative hyphae) formed when sclerotia germinate. During germination, each sclerotium forms 1-60 stromata. Each stroma consists of a flesh-colored stalk 0.5-2.5 centimeters (0.2-1.0 inch) tall, the tip of which produces a spherical head (Figure 3). Numerous sexual fruiting structures known as perithecia (singular: perithecium) develop at the periphery of each head. Each perithecium contains many sac-like cells known as asci (singular: ascus). Meiosis (cell division) results in eight long, multicellular ascospores in each ascus. Asexual spores known as conidia are produced following infection of host plant ovaries by ascospores. Both types of spores are spread by wind, splashing rain, or insects.



Figure 3. An ergot sclerotium that has germinated to produce stromata that will produce sexual spores (ascospores). Courtesy of Robin Morrall, Department of Biology, University of Saskatchewan.

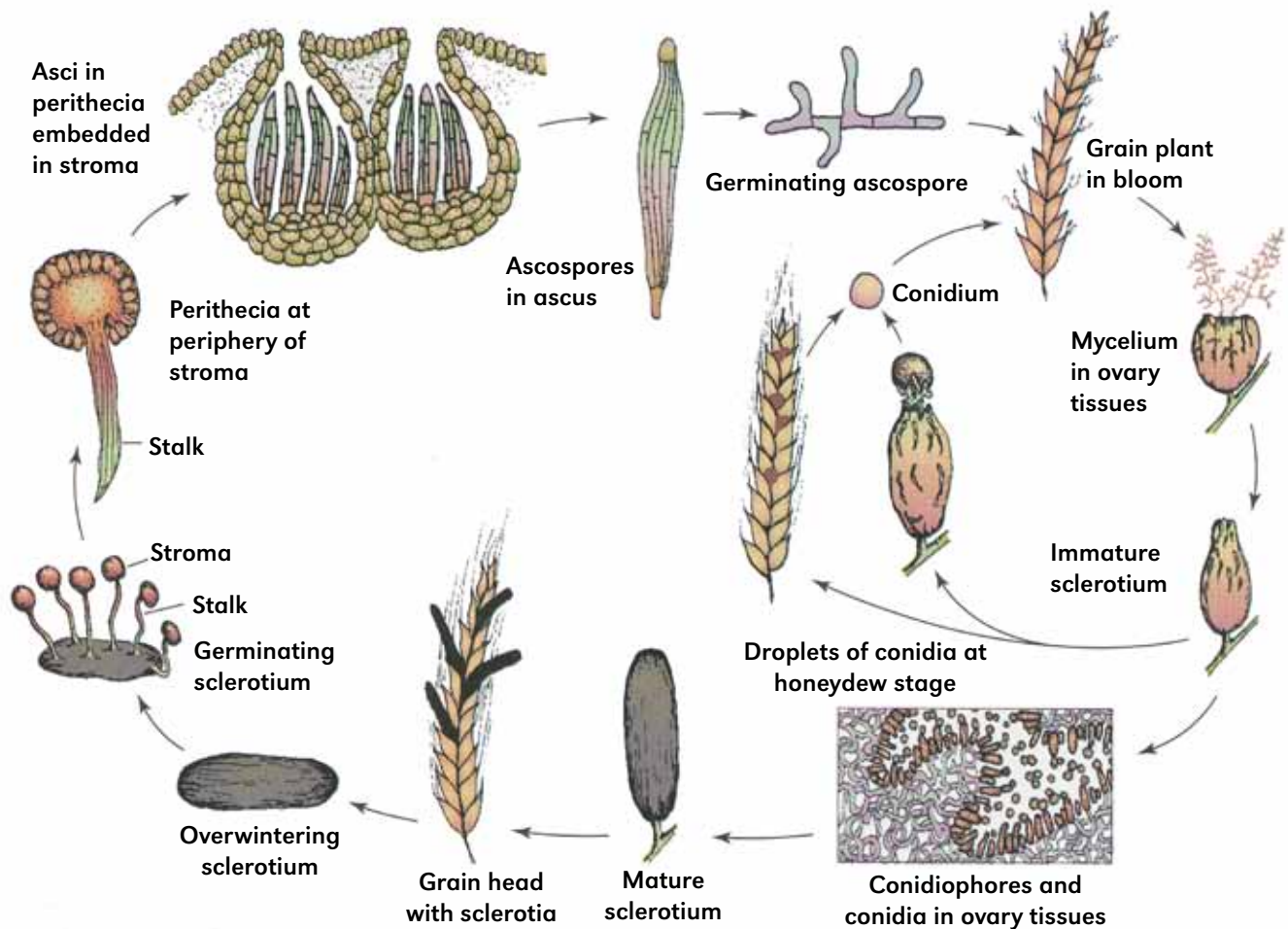


Figure 4. Disease cycle of ergot of small grain cereals and grasses. This figure was published in *Plant Pathology Fifth Edition* by George N. Agrios, page 503, Copyright Elsevier (2005).

1.3 Occurrence and host range

Ergot occurs worldwide. The causal fungus is endemic to the Great Plains wheat-producing region of North America. It occurs to some extent every year in cereal grains, pastures, and roadside grasses in Nebraska. Among the cultivated cereals, ergot is more common in rye and triticale than in wheat, barley, and oat. Open-pollinated crops such as rye, triticale, and some grasses are more susceptible because of the easy access of pathogen spores into the flowering head. Many wild and cultivated grasses are also susceptible. They include annual bluegrass, cheatgrass (downy brome), green foxtail, hairgrass, meadow foxtail, quackgrass, wild barley, and wild oat.

1.4 Symptoms

The first sign of infection by the ergot fungus is the appearance of a sugary yellowish slime (honeydew) on the heads at or soon after flowering. Later, ergots (sclerotia) develop and are the most noticeable and characteristic sign of ergot. Ergots are horn-like, purple-black, and 4 to 10 times the normal size of grain. On the head, they replace developing grain and protrude beyond the glumes (*Figure 1*).

1.5 Disease cycle

The disease cycle of ergot is illustrated in *Figure 4*. Sclerotia from previous cereal crops or indigenous grasses survive in the soil or on the

soil surface. When subjected to cold temperature treatment and moisture in the spring, they germinate and form stromata bearing fruiting structures (perithecia) that release ascospores. The ascospores are dispersed by wind and splashing rain. When their release coincides with flowering, the ascospores land on the florets and infect the ovaries. One or many ovaries may be infected per head. A few days following infection, the ovary is replaced with a grayish white mycelial mass (stroma) that occupies the space between the glumes (bracts) of the flower. As the stroma grows and develops, the host plant produces a sticky, sweet exudation (honeydew) that covers the stroma. Conidia are budded off the stroma and float to the surface of the honeydew. Insects

are attracted to feed on the honeydew. The conidia are spread to other florets by insects, direct contact, and rain splash, and infect the ovaries. The honeydew stage lasts for several days and is prolonged by humid and cloudy weather. The disease cycle is completed when the stroma that replaced each ovary on the head matures into a sclerotium. Sclerotia fall to the ground before or during harvest or are introduced when contaminated grain is planted.

1.6 Favorable conditions

Cool, wet weather during flowering favors infection by ascospores and conidia. Prolonged cool, wet, and cloudy weather extends the flowering period and hence the window of infection. This type of weather prevailed in Nebraska in the spring of 2011 and largely contributed to the ergot outbreak in wheat fields in the south central and southeastern parts of the state. The most susceptible hosts are those with prolonged flowering periods and more open florets. Often, indigenous grasses are the main source of spores that infect wheat and other cereals. Many of the ergots in wheat grain may be from weedy grasses. These ergots are usually smaller and more slender than those produced on wheat.

The risk of infection is increased by conditions that delay maturity, such as poor fertility and herbicide injury. Excessive tillering results in uneven flowering which exposes the crop to infection for a longer period.

1.7 Management of ergot

- Scout fields before harvesting to determine heavily affected parts of the field such as headlands. Harvest heavily affected parts of the field separately and take the grain to the elevator separately or destroy it by burying or burning

if the sclerotia content is very high. Typically, high levels of ergot occur around the edges of the field because of proximity to surrounding grasses that often are the source of inoculum.

- Most, but not all, ergots can be removed from grain by cleaning it with gravity-type cleaning equipment. Small, high-value seed lots can be cleaned by flotation in brine (20 percent salt solution). The sclerotia float and the grain sinks in the brine solution.
- Use sclerotia-free seed to plant the next cereal grain crop. Purchasing certified seed greatly reduces the risk of planting ergots with the seed.
- Treating seed with a triazole fungicide will reduce the viability of sclerotia. However, viable sclerotia will germinate and produce spores under favorable environmental conditions even if the seed was treated with a fungicide.
- Ensure uniform stands by using seed with good germination, seeding at a consistent depth, and using a balanced fertilizer program. This will result in uniform crop development that will in turn prevent a prolonged flowering period and thus reduce the risk of infection.
- Mow grasses in headlands, roadsides, and waterways before they head. Cut or graze hay before flowering if the risk of ergot is high.
- Control grassy weeds in the field throughout the crop rotation cycle.
- Survival rate of sclerotia on the soil surface is about one year, therefore, crop rotation for at least one year with nonhost crops

such as legumes and corn reduces the risk of carryover infections from sclerotia within the field.

- Plow fields with heavy ergot infestations to bury sclerotia. Burying sclerotia more than 1-inch deep will prevent them from germinating.
- Avoid excessive irrigation during cool weather at flowering.
- Some small grain cultivars are resistant to ergot. Cultivars with short flowering periods and more closed florets are least susceptible. Avoid planting cultivars known to be highly susceptible.

2. Ergot Alkaloids and their Health Effects on Humans and Livestock

The ergot sclerotia contain poisonous compounds known as alkaloids. Ingestion of ergots in grain and flour can cause illness or death in humans and domesticated animals. Ergotism in humans is known as St. Anthony's fire or holy fire, and has occurred several times in human history. Ergotism was described by the Chinese in 1100 B.C. and by the Assyrians in 600 B.C. It has caused serious illnesses and resulted in thousands of deaths throughout human history. It is thought that the Salem witchcraft trials of 1692 resulted from hallucinations and insanity caused by ingestion of ergoty flour.

The ergot alkaloid content of sclerotia varies in amount and alkaloid make-up, depending upon the *Claviceps* species causing the infection, the type of plant infected, and environmental conditions during the host plant's growing season. Research published in 1945 showed that the

Table 1. Percent sclerotia (by weight) and alkaloid content (in parts per billion) in 10 wheat grain samples^a from the 2011 crop in south central and southeast Nebraska

Sample No.	Cultivar	Sclerotia ^c (%)	Alkaloid content ^b (ppb)					
			Ergosine	Ergotamine	Ergocornine	Ergocryptine	Ergocristine	Total
1	Postrock	0.0174	NDA ^d	NDA	NDA	NDA	NDA	NDA
2	Overland	0.0000	NDA	NDA	NDA	NDA	NDA	NDA
3	Everest	0.0046	30	10	5	5	60	110
4	Armour	0.0000	NDA	NDA	NDA	NDA	25	25
5	Everest	0.0222	NDA	NDA	NDA	NDA	NDA	NDA
6	Postrock	0.0252	20	5	20	25	15	85
7	Thunderbird	0.0044	NDA	15	5	NDA	20	40
8	Santa Fe	0.0933	80	45	45	40	90	300
9	2137	0.0004	15	NDA	5	15	NDA	35
10	Karl 92	0.4300	685	750	795	1,105	1,425	4,760

^aGrowers were asked to submit samples for analysis of sclerotia and alkaloid content. The data presented in the table are not from a planned, replicated field experiment.

^bAdverse effects on livestock performance are generally observed when total ergopeptine alkaloid concentrations in the diet exceed 100 to 200 ppb. Clinical signs observed depend upon the species and physiologic state of the exposed animal, environmental conditions during exposure, the individual ergopeptine alkaloids present and their interactions, and duration of exposure. Late gestational mares have been reported to suffer from agalactia (failure to produce milk) when consuming diets with ergopeptine alkaloid concentrations as low as 50 ppb.

^c7 CFR 810 (Title 7 Part 810 - Official United States Standards for Grain) defines ergoty wheat as containing > 0.05% ergot sclerotia.

^dNondetectable amount.

alkaloid content of sclerotia collected from rye and durum wheat in the U.S. varied from 0.131 percent to 0.301 percent. Analysis of 10 wheat grain samples from the 2011 wheat crop in south central and southeast Nebraska where significant levels of ergot occurred showed that percent sclerotia (by weight) in the samples ranged from 0.0000 percent to 0.4300 percent and total alkaloid content (in parts per billion) ranged from nondetectable amounts to 4,760 ppb (Table 1).

The sclerotia are usually visible to the naked eye. However, they may be difficult to detect if they were fragmented or ground up during the processing of flour or feed. Therefore, ergot alkaloids may be detectable in flour or feed even though no visible sclerotia are evident.

Although the various ergot alkaloids share some common poisoning characteristics, the effects they cause after ingestion of ergot-contaminated flour or feed may vary depending on the specific alkaloids present. Humans and all species of animals are susceptible to poisoning, but not necessarily to the same extent. Ergot poisoning has been reported in cattle, sheep, pigs, horses, and poultry.

Diseases caused by ingestion of ergot-contaminated feed or food have been called by various names, including ergotism, ergot poisoning, ergot toxicosis, and St. Anthony's fire. There are four forms of ergot toxicoses in animals: gangrenous or cutaneous, hyperthermic, reproductive, and convulsive, described as follows.

2.1 Gangrenous toxicosis

Gangrenous or cutaneous toxicosis is associated with longer-term ingestion of ergot-contaminated feed. Ergot alkaloids can cause small blood vessels to constrict, which reduces or stops blood flow through them. If the blood flow is restricted or stopped for a significant length of time, the tissues behind the restriction become starved for oxygen and other nutrients and can die. The medical term for tissue death is necrosis. This form of the disease is usually evident when digits of any of the limbs, ear tips, ends of tails, combs, or beaks become necrotic. This condition is called "dry gangrene." Distinct lines may separate normal from necrotic tissues and the odor of rotting flesh may be noticed. Affected areas may slough

off. Animals whose digits are affected may show signs of lameness. Cold temperatures can exacerbate this form of the disease.

2.2 Hyperthermic toxicosis

Hyperthermic toxicosis is also associated with longer-term exposure. How ergot alkaloids cause this syndrome isn't well understood, but it involves various mechanisms used to regulate body temperature. This form of toxicosis is exacerbated by any environmental condition that reduces the ability of animals to stay cool, such as higher temperatures or humidity, reduced availability of shade, increased animal density, and reduced air movement. Affected animals may pant, mimicking the signs of respiratory disease. Animal performance often is compromised when they suffer from elevated body temperature, also known as hyperthermia. Feed intake is reduced and consequently, weight gain or milk production may drop significantly. Reproductive performance may also decline (See the discussion of the reproductive form of the disease that follows).

2.3 Reproductive toxicosis

Reproductive toxicosis is caused by several mechanisms, including stimulation of smooth muscle surrounding the uterus, inhibition of the release of milk-producing hormones, and disruption of hormone secretion necessary to maintain pregnancy. This form of the disease may cause reproductive failure or abortion. Animals that should be lactating will not do so, a condition called agalactia, which can threaten viability of infants that are to be nursed. Sows and mares have experienced agalactia. There are reports of births of small, unthrifty, or stillborn offspring, and offspring that showed no suckling reflex, especially in horses.

2.4 Convulsive toxicosis

Convulsive toxicosis is associated with having ingested high amounts of ergot alkaloids over a relatively short period of time. It is relatively rare. Animals suffering from this form of toxicosis become excited, exhibit bizarre behaviors, convulse, and may die.

2.5 Diagnosis of ergot toxicoses

The diagnosis of ergot toxicosis is best done with veterinarian assistance. If animals are exhibiting clinical signs of any form of ergot toxicosis, the following considerations can help confirm or rule out the disease.

Gross lesions: Lesions visible to the naked eye; tissue necrosis and sloughing of extremities with a distinct line between healthy and necrotic tissue.

Post mortem: Fat necrosis present within the mesentery surrounding the intestines in ruminants chronically exposed to ergot alkaloids. Grossly, fat appears as hard, caseous (cheese-like), yellowish, or chalky white nodules or masses.

2.6 Diagnostic testing

Detection of ergot sclerotia in components of the diet: Detection of ergot sclerotia in components of the diet supports an assessment that exposure may have occurred. Sclerotia are often large enough to be seen with the naked eye; however, they can fragment during harvesting and processing so that microscopic examination of the feed may be necessary to detect them.

Chemical analysis of feed for ergot alkaloids can detect those chemicals in the presence or absence of visible sclerotia.

Detection of ergot alkaloids in animal specimens: Detection of ergot alkaloids in rumen or stomach content establishes that exposure to ergot alkaloids has occurred. Analytical services to detect the alkaloids in body fluid or tissue specimens aren't commonly available.

2.7 Treatment for ergot toxicosis

Stop exposure: Remove contaminated feed from feed bunks. The sooner exposure stops, the more likely animals will recover. Early recognition of clinical signs can aid in timely cessation of exposure. However, lack of alternative feed may make removal of contaminated feed impractical. Some people may turn to the use of mycotoxin binders, which are supposed to help prevent and treat mycotoxicoses including ergot toxicoses. No such products have been approved for use in the U.S., and their efficacy and possible side effects haven't been well established.

Nutrient supplementation: Nutrient supplementation for energy, protein, amino acids, selenium or copper has been tried to reduce the effects of ergot alkaloids, but with limited success.

Drugs: Various drugs have been tried with mixed results. Use of drugs to treat animals suffering from ergot toxicosis hasn't been approved for use in the U.S. Withdrawal times for use to treat food production animals have not been established. Consult a veterinarian if drugs are to be used.

2.8 Prognosis

Prognosis depends upon the severity of the clinical signs and timely cessation of exposure. Complete recovery is to be expected for mild cases of ergot poisoning. Animals with severe cases of the cutaneous form may be salvaged for slaughter.

Euthanasia may be necessary for very severe cases.

2.9 Prevention and control of ergot toxicosis

Recognition of the potential for exposure to contaminated feed and actions to prevent or minimize exposure are the best ways to reduce risks of developing ergot toxicosis.

The safest course of action would be NOT to use contaminated grain or straw. If circumstances require their use, then continually monitor the grain or straw for the presence of ergot using two methods.

The first method is **continual** inspection of the grain or straw for the presence of sclerotia as the product is used. The more vigilant the inspection, the more likely sclerotia, if present, will be detected.

The second method is chemical analysis of specimens of feed or straw. Ideally, a representative specimen from every batch or bale should be collected and submitted for analysis so that results are available in advance of use. That way, if hazardous concentrations of ergot alkaloids are detected, time will be available to take whatever action is needed to control the risk of feeding the contaminated feed. Contact the UNL Veterinary Diagnostic Center at 402-472-1434 for assistance if chemical analysis is desired.

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