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How to Recognize and Control Ergot of Small Grains and Grasses

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Claviceps purpurea, the fungus which causes ergot, attacks cereals and grasses throughout the world. Ergot occurs most commonly on rye but may also infect wheat, barley, oats, and wild and cultivated grasses.

Ergot occurs to some extent every year in Michigan. While it is more prevalent on rye, it has been reported on wheat and forage grasses. Some losses in yield can be attributed to the disease, but its main importance is the fungal fruiting structures (ergot sclerotia) that replace the grains and are poisonous to humans and animals that eat them.

Symptoms of Ergot Disease

Ergot first appears as a creamy to yellowish droplet of sticky liquid exuding from young florets of infected heads. The "honey dew" stage may go unnoticed, but it is soon replaced by hard, horn-shaped, purplish-black structures called ergot bodies or sclerotia (Fig. 1). The sclerotia grow in place of the kernel and consist of a hard, compact mass of fungal tissue which appear white or faintly purple when cut open. Sclerotia are usually 1/16 to 1/8 inch in diameter and may be 1/4 to 1 inch long (Fig. 2).

Life Cycle of the Fungus

Claviceps purpurea survives the winter as sclerotia mixed with the seed or on and in the soil. Germination of sclerotia occurs in the spring and corresponds to the time the cereals are in bloom. Flesh-colored stalks 1/4 to 1 inch long with spherical heads form on each sclerotium. Within the heads are cavities in which spores are formed. The spores are discharged forcibly into the air where they are carried by the wind or insects to young open flowers. Within a week to ten days after infection, the honey dew stage appears. The syrupy liquid contains another type of spore (conidia) which is spread to uninfected florets by insects attracted to the sugary liquid. As the secretion of the honey dew ceases, the infected flower is replaced by the ergot sclerotium. The sclerotia mature about the same time as the healthy seeds and either fall to the ground or are harvested with healthy grain.

Fig. 1. Ergot sclerotia as they would appear on an infected plant in the field.

Fig. 2. Ergot sclerotia of different sizes compared with kernels of rye.

Economic Importance

Ergot is more important as an animal disease than a plant disease. Grain is classified as ergoty when it contains more than 0.3 percent by weight of the ergot sclerotia. While most of the sclerotia can be separated from the grain by modern cleaning equipment, it is costly and often difficult to remove enough sclerotia to meet the legal standards. Often, traces of sclerotia are left which can be toxic. When buying rye for both human and animal consumption from neighbors or at county fairs, buy only grain certified as ergot free or when visual examination of the unground grain reveals no sclerotia (Fig. 3). There is no statewide inspection for ergot in rye sold in health food stores at this time. Ergot toxins are not destroyed by baking.

How Ergot Causes Disease In Animals and Man

Ergot poisoning (ergotism) in humans occurs less often and less severely than in the past because most grain used for human consumption is inspected, but it does continue to be an important disease of animals. Sclerotia contain active chemicals known as alkaloids which cause ergotism. The alkaloid content varies from season to season, and with the type of cereal infected. Variation can also occur within a season and some sclerotia may be highly toxic while others are only mildly so.

Alkaloids cause constriction of the small blood vessels, shutting off the blood supply to body appendages such as legs, ears and tails. The surrounding tissue, deprived of nutrients and oxygen, begins to die. The tissue becomes cold and insensitive and later, gangrenous.

The alkaloids also give rise to abortions by causing the contraction of uterine muscles. The general symptoms usually associated with ergotism include abortion, signs of nervous system involvement, and gangrene. Long term feeding of low levels of ergot sclerotia reduces feed intake and weight gain.

Control

The best way to prevent ergot poisoning is to avoid feeding grains and forages that are ergoty. A zero tolerance should be assumed for pregnant animals. Ergot sclerotia do not survive more than one year in the soil, therefore, a one-year rotation to a non-cereal crop will reduce the incidence of the disease. Because sclerotia fail to germinate when they are buried at least one inch deep, fields with severe infestations should be deep plowed.

Plant only clean seed which contain no sclerotia. Wild grasses can serve as sources of inoculum and should be eradicated or prevented from heading in and around fields of cereals (Fig. 4). No ergot-resistant commercial varieties of barley, rye, wheat or cultivated grasses have been developed.

Fig. 3. A mixture of ergot sclerotia and rye kernels as they would appear after harvest. The sclerotia are sometimes broken into smaller fragments.

Fig. 4. Ergot infection on a wild grass species collected from the margin of a rye field.

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