Washington State University

Ergot

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Diseases of Washington Crops.
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Ergot is a disease of cereals that has had considerable historical impact. The fungus produces toxins that cause ergotism in animals, including humans. Ergotism may cause either gangrenous or convulsive reactions and has been associated with several great historical events, such as the Salem witch trials, the invasion of France by Norsemen, and the failure of Peter the Great to capture the Dardanelles. Ergot chemicals are widely used in medicine, for example as a vasoconstricting agent in the treatment of migraine and in childbirth.

Cause

Ergot is caused by Claviceps purpurea. This fungus, an ascomycete, forms sclerotia, ascospores and conidia. The sclerotia vary in size (2-20 millimeters long), depending on the host (the host's seed size, which the sclerotia replace) and are purple-black, elongate (spur-like), with white centers. Ascospores are long, thin, and septate; conidia are small, round and one-celled.

Hosts

Ergot has a wide host range of about 60 genera, all in the grass family. Rye and triticale are particularly susceptible, because they are open-pollinated, but wheat, barley, and many grasses (including weed grasses such as quack grass, meadow foxtail, wild oat, cheatgrass, hair grass, wild barley, annual bluegrass, and green foxtail) are among the hosts.

Symptoms and Signs

At flowering a cloudy, tan, sticky "honeydew" may appear on the florets, especially if the weather is humid. The most noticeable signs of the disease, though, are the sclerotia (Photo 14), which protrude from the glumes and are produced as the host plant matures. The sclerotia that contaminate harvested grain are easily seen because they are dark.

Disease Cycle

Sclerotia remain viable for about 1 year. After over-wintering or being exposed to cold temperatures, they germinate to produce several small stalked, mushroom-shaped perithecial stroma. Ascospores from the perithecia comprise the primary inoculum and are spread by wind and splashing rain to host plant flowers. Ascospores germinate and invade the flower through the stigma to penetrate the ovary. A convolute mycelial mat forms over the developing ovary, and masses of conidia in a sticky matrix (honeydew) appear in about 5 days. These conidia constitute secondary inoculum. Honeydew attracts insects visiting the flowers, and the insects in turn disseminate the conidia. Upon host maturity, fungal mycelium replaces the ovary tissues to form a sclerotium.

Wet conditions during flowering and open flowers favor the disease. Susceptibility of different hosts relates to the length of time the flowers are open; hosts with longer flowering periods are more susceptible than those with shorter flowering periods.

Control

Cultural. Wheat cultivars whose florets remain open for short periods of time may escape infection, but sanitation measures are the most important way of controlling ergot. Remove sclerotia from seed by screening or by brine flotation. In addition, seeding deeper than 3 inches buries sclerotia and eliminates primary inoculum by preventing emergence of perithecial stroma. Alternate-year rotations allow time for sclerotial disintegration and reduce the amount of inoculum in the soil. Destroy stands of ergoted grass near seed or grain fields.

Other Interesting Ergot sites:

University of California Statewided Integrated Pest management guidelines -- pictures and descriptions.

Alberta Agriculture, Food, and Rural Development -- good description and great pictures at the bottom of their page.