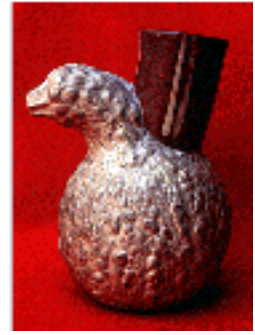




Ethnobotanical Leaflets



Ergot

By Halima Ibrahim

Ergot (*Claviceps purpurea* Tul.) is a poisonous fungus that is hazardous to animals and humans. It appears to be a small, hard, black, elongated structure that grows on and contaminates grains, which is called sclerotium. The appearance tends to look like mold. When eaten in quantity or small amounts over a period of time it can be highly toxic. Furthermore, it is a major problem for humans in North America but even more severe for animals. Humans are susceptible to being contaminated with this poisonous fungus because ergot can grow on mushrooms, rye, wheat, and barley. These are products that humans ingest everyday. Ergot is the most notorious poison because it includes several species of ascomycete fungi with complex life cycles that parasitize the grains of various cultivated and wild grasses. Therefore, it is necessary to check for ergot contamination, especially if they are home-grown grains.

The epidemic or spreading phase of the ergot fungus occurs at the same time the grasses are in flower. Fungal spores infect the ovaries of the mature flower of the host plant. These develop mycelia (fine threadlike filaments). At this initial infection an excretion of honeydew (moist, sticky secretion) stage, develops and the mycelia continue to grow within the ovaries of the host and become sclerotia (fruiting bodies). A sclerotium appears as a cylindrical, three sided body, slightly curved, hornlike, hard, grooved on one or both sides, dark purple next to black that is 1/3 to 1 1/2 inches long and 1/8 to 1/4 inches wide. There is a very unpleasant aroma (odor), which is somewhat fishy. The developed sclerotia finally falls to the ground to germinate in the summer at once or may wait until after the winter and germinate the following spring. While germinating, each sclerotia grows into a long stalk with globular fruit heads that may be formed from wind born to infect a new crop of grass flowers. The infection can occur by insects also.

According to Fuller and McClintock, ergotism has caused so many occurrences in epidemic and the middle ages was known as St. Anthony's fire. In France many people died from this disease, which was caused mainly from eating bread made from rye grains contaminated with ergot (p. 20).

Plant Toxin/Derivative Drug

Researchers state over fifty alkaloids are within the ergot sclerotia, but mainly ergonovine (also known as ergometrine, ergobasine, ergotocine, ergotstetrine C₁₉H₂₃N₃O₂, ergotamine C₃₃H₃₅N₅O₅, ergotryptine C₃₂H₄₁N₅O₅, ergocornine C₃₁H₃₉N₅O₅, ergocristine C₃₅H₃₉N₅O₅, ergosine C₃₀H₃₇N₅O₅, and ergovalide) is present in ergot. Most of the alkaloids contain thirty to thirty-five carbon atoms, although some have a smaller number. Therefore, these alkaloids can be classified as polypeptides. All ergot alkaloids form lysergic acid. Lysergic acid diethylamide, LSD, is the most powerful compound known that produces imitations of severe mental disorders. Ergot alkaloids not only have the indole structure but the other ring system and side chains as well.

Symptoms

Humans and animals common acute symptoms is a convulsion stimulation of CNS (central nervous system). Chronic symptoms include damage to the lining of capillaries and constriction of the smaller arterial blood vessels resulting in gangrene of the extremities (Fuller and McClintock,19). Some symptoms involve tingling of fingers, vomiting, and diarrhea. Other symptoms involve losing the sensation in fingers and limbs of developing gangrene of fingers where they rot away, which causes painful spasms of legs and arms.

In general, animals are more susceptible to obtaining this poisonous fungus because they eat grass in pastures that may contain ergot. Acute poisoning to the cattles display intermittent blindness, deafness, and variation of sensitivity of skin. More severe cases display comas, temporary paralysis, stiff-legged walking, falling in unusual positions. Cows have many problems after consuming this poisonous fungus. Whereas, sheeps have been experimentally tested with ergot and do not display many problems. They do not develop gangrene of extremities, but they do suffer lining of the mouth, rumen, and small intestine. Pregnant sheep and pigs fed ergot may lose their embryo or suffer a high mortality rate of embryo (Morton,7).

Symptoms associated with involvement of the smooth musculature of the digestive system usually precede of accompany the above. They include nausea, abdominal pain, and constipation or diarrhea. In some cases the oral mucous membranes are inflamed or display shallow superficial erosions from 1/4 to 1 inch in diameter. Pregnant animals may abort.

Symptoms and lesions in the human being are similar. In mild cases only the nails are lost. Extreme cases result in the loss of the hands or feet and occasionally in gangrene of internal organs. Gastrointestinal discomfort and headache usually precede and accompany any of these lesions.

Gross necropsy findings in both the gangreous and convulsive syndromes, other than the obvious lesions of gangrene, are meager and not characteristic. Degenerative changes in the central nervous system of sheep have been described histologically after convulsive ergotism. It was postulated that the central nervous system lesions may have resulted from increased pressure of the cerebrospinal fluid such as is associated with hypovitaminosis A. Low body levels of vitamin A have been reported more than once in cases of ergotism.

It is worth emphasizing that undergrazing, a practice opposite to that usually resulting in poisoning of animals by plants, in this instance has the opposite effect. It allows pasture grasses to develop an inflorescence and become potentially dangerous (86-87).

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Last updated: 15-May-98 / du