Recent Outbreaks of Ergot of Rye Poisoning in Sheep and Cattle Have Further Clarified the Historical Confusion that Surrounds this Problem

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Ergotism is a microbial poisoning caused by the ingestion of either ergot (Claviceps purpurea) of rye, or endophyte (Acremonium spp) infected grasses, notably tall fescue. It can also refer to poisoning by ergot (Claviceps paspali) of paspalum. The first two disorders are similar and involve ergot alkaloids, but the third is different and involves tremorgenic indole alkaloids. Literature accounts of ergotism refer to a convulsive nervous syndrome and a peripheral gangrenous syndrome. However there has never been a convulsive nervous disorder in livestock that has been demonstrated to be caused by ergot alkaloids. An unusual nervous disorder was reported in some outbreaks of human ergotism but it was neither convulsive nor caused by ergot alkaloids. Peripheral gangrene, which typically affects the feet and results in lameness, is caused by ergot alkaloids. A third form of ergot alkaloid poisoning involves a sub-lethal, hyperthermic, ill thrift syndrome.

In Australia hyperthermia has been referred to as an ‘atypical’ form of ergot of rye poisoning; it has been associated with high morbidity but usually no mortality. In North America it is regarded as a ‘typical’ form of tall fescue poisoning and again is not associated with mortality. During 1998 and 1999 outbreaks of ergot of rye poisoning occurred in sheep and cattle in central New South Wales. Ergot had been consumed either directly from ergotised annual rye grass pastures or indirectly in barley grain contaminated with ergotised annual rye grass seeds. A lethal form of hyperthermia characterised these outbreaks. There were no concurrent cases of peripheral gangrene or so-called nervous ergotism. These outbreaks, together with other reports of ergot of rye poisoning in Australia since 1983, would suggest that hyperthermic ergotism is typical, rather than atypical, of ergotism in the Australian environment. They also confirm that gangrenous ergotism is rare and so-called ergot alkaloid nervous ergotism non existent.

The recent outbreaks were unusual in that they involved high mortalities and these occurred in both Summer and Winter regardless of the ambient temperature. Mortality was associated with conditions of bright sunlight in the absence of shade and occurred following the ingestion of as little as 200mg of ergots per kg of feed. In two herds deaths from ergot induced hyperthermia reached 10%. In one of these herds they occurred during two periods of only 6 daylight hours each, in summer, when cattle were deprived of shade, in the other herd deaths occurred sporadically over 25 days in winter when sunlight was abundant but shade unavailable. The morbidity and mortality in sheep was much less than that in cattle, but similar clinical signs were observed. These included: reduced weight gain or even weight loss, reduced milk production, diarrhoea in some animals, inappetance with a tendency to only feed at night, constant shade seeking and water seeking, standing in dams and water troughs, abnormal breathing, excessive panting, excessive drooling of saliva, depression, protracted recumbency, a vague hind limb weakness with an uncoordinated gait, and a rectal temperature of between 41 and 43°C. In some herds an increase in calving problems and late term abortions, together with a decrease in conception rates, were reported.

It would seem that hyperthermic ergotism can occur as either lethal or sub-lethal outbreaks, this could indicate two different mechanisms of poisoning are possible. Rye ergots contain both ergot alkaloids and ergot pigments. Sub-lethal outbreaks of hyperthermic ergotism seem to occur with either ergot of rye poisoning or tall fescue endophyte poisoning, probably require ambient temperatures of 30°C or greater and are probably an indirect result of the peripheral vaso constrictive effect of ergot alkaloids. Conversely lethal hyperthermia outbreaks may be restricted to ergot of rye poisoning, can occur at either higher or lower ambient temperatures and possibly involve the photo-chemical activation of circulating ergot pigments in affected animals. These light activated pigments may directly effect the hypothalamic body temperature control centre in the brain causing a lethal hyperthermia.

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