Biography:
Dr. Colin Palmer is an Associate Professor of Theriogenology (Animal Reproduction) at the Western College of Veterinary Medicine. Originally from Nova Scotia, Dr. Palmer worked in mixed practices in Ontario and British Columbia and has owned/operated a practice in Saskatchewan. Dr. Palmer along with his wife Kim and children Lauren, Emily and Carter run a herd of purebred Red Angus cattle under the KC Cattle Co. name.

A Breeder's . . . . Veterinary Perspective

Ergot and fungal mycotoxins

The effects of ergot and fungal mycotoxins on cattle is a hot topic in western Canada. Veterinarians and feed reps are busy fielding calls and assessing toxin levels in feed samples. I have been involved in assessing the effects on reproductive performance. This article will focus on ergot, my next one will cover mycotoxins. I hope I can answer a few questions.

Ergot is certainly not a new disease with reports of its effects in both humans and animals extending back to the middle ages. Cool wet conditions during the growing season increase the likelihood that the fungus, Claviceps purpurea, will infect cereal grains and grasses. Ergot appears as dark brown, black or purple bodies protruding from the grain head and are easily identified in unprocessed grain samples. These bodies represent the overwintering stage of the fungal lifecycle and are typically much larger than a kernel of grain. Despite being shaped like a grain kernel the entire ergot body is fungal in origin that has been formed in place of the grain. Some types of grasses may also become infected, however, ergot infection is less obvious appearing as small, dark and slender bodies. Cool wet conditions in the spring promote the germination of the ergot bodies whose spores may then be spread through the air to land on flowering cereals and grasses. Ergot bodies usually do not survive for more than one winter in the soil and can be destroyed if cultivated to more than 4 cm below the soil surface. Crops become infected from previous cereal crops, or from spores blowing in from adjacent cereal crops or grasslands; hence, the reason that ergot tends to be more prevalent in grain harvested from the outside rounds. (Source – Ergot of Cereals and Grasses, ***https://www.saskatchewan.ca/)***. Cereals and grasses vary in their susceptibility to ergot. Rye, followed by wheat, barley and triticale are much more susceptible than oats. Many commonly grown grasses are at risk including brome grass, wheat grasses, rye grass, orchard grass, blue grass, fescues and quack grass. Intensive grazing or mowing to prevent grasses from heading outare good practices to prevent or control ergot in grasses.

Ergot bodies contain potent alkaloids that are essentially toxic chemicals on par with any synthetic agent that could be produced in a modern laboratory. If sufficient quantities are consumed, two types of conditions may appear in affected animals. The most common is the gangrenous type – blood vessels become constricted limiting blood flow to the body's extremities which will eventually cause the tissues to die. Hind limb soreness is the first sign – lameness, stiffness, and reluctance to get up and move appear first. Loss of sensation followed by sloughing of tissues for which there is no recovery occur last. During cold weather blood flow is naturally limited to the extremities which will worsen the effects of ergot. Sloughing of the hooves at the coronary band and loss of the tail and ears occur most commonly during cold weather, but have also been seen during the summer. Sores on teats and sloughing of teats has been seen in cows. In the US Midwest, ergot is the cause if "summer slump" - rough haircoats, weight loss and standing in the shade or water. Vasoconstriction impairs the animal's ability to cool off effectively reducing their tolerance for heat. Hyperexcitability and tremors are associated with the less common, nervous, form of ergot poisoning. Convulsions and even paralysis are rarely observed in cattle. The central nervous system signs may stem from restricted blood flow to parts of the brain or the direct effect of the toxins. Ergot alkaloids have anti-prolactin effects which lowers the amount of prolactin hormone in the body. Prolactin is required for udder development and milk production in cattle; therefore, reduced milk production or complete suppression of milk production may occur. Gestating cows may not develop an udder. Abortion in cattle has also been linked to ergot. A number of abortions occurred in cattle 7 to 10 days following introduction to a heavily contaminated rye grass pasture with the cause being attributed to ergot.

Ergot alkaloids are incredibly potent toxins. Higher levels of intake are associated with an onset of signs occurring within several days; lower levels may require several weeks. In most situations time to occurrence of clinical signs is 4 to 6 weeks. For example, total dietary levels between 0.3% and 1.0% of the diet resulted in gangrenous disease when fed for several weeks. Levels between 0.1% and 0.3% (100 to 300 parts per billion) can even cause symptoms of poisoning.

Moist growing conditions have increased the risk of ergot contamination in recent years. Pelleted rations made from screenings have been one source of poisoning leading to increased scrutiny when buying grain by livestock feed processors. Home grown feeds and pastures can also be contaminated and have resulted in some devastating losses.

Direct effects of ergot on reproductive performance in cattle are not well documented. Even the link between ergot and abortion is not 100 percent conclusive. There are some reports suggesting that ergot may decrease sperm motility and increase the proportion of sperm defects in semen; however, any direct effects were not evident. Many things can influence sperm motility and morphology. Changes in diet are as likely to cause variations in semen quality as great as those suggested to be caused by ergot. At best, prolactin plays a minor role in ovarian or testicular function in cattle so it is not likely to be involved. New directed research is likely to shed light on the effects of ergot on reproductive performance, but until then it is my opinion that any negative effects are secondary to stress and illness associated with the vasoconstrictive effect.