

Problem Feeds

Frozen Grain: Low in energy, but not toxic.

Sprouted Grain: Sprouting lowers the yield, bushel weight, and energy content but the grain is not harmful to livestock.

Moldy Feeds: Mold growth, accompanied by heating, takes place in most feeds when their moisture content is above 15 or 16 per cent for a period of time. The presence of mold does not necessarily mean that the feed can not be used. Most molds are not toxic when fed to livestock. However, some molds are very toxic and dangerous. Producers should be aware of the possible dangers and recognize steps to take to minimize risk.

Molds in feeds (both hay and grain) can cause problems for four reasons.

1. Many molds are known to produce one or more toxins, some of which cannot be identified at present. Symptoms vary with the particular mold and toxin. However, it helps to remember that cattle are generally more resistant to mold toxins than either swine or poultry. Young animals are more susceptible than mature animals.
2. Loss in feed value can be significant (10% +) when molds are present in a feedstuff. Adjustments to the ration should be made accordingly.
3. Some molds (Mucor, Aspergillus) can cause mycotic abortions.
4. When inhaled, mold spores can cause the lungs to become abnormally sensitive to these particular spores. Chronic respiratory disease and even death can occur if exposure to the moldy feedstuff is continued.

Moldy feeds may mean trouble, but the risks can be minimized by smart management. If moldy hay, grain, or concentrate is to be fed, observe the following precautions:

1. Send a representative sample of the feed to a feed testing laboratory for mold analysis. Grain samples should not be ground or rolled. The procedure takes two to four weeks, as a culture of the mold must be grown before examination to indicate if the mold involved produces toxins.
2. Avoid feeding moldy feeds to young, milking or gestating animals. These classes of animals are all particularly susceptible to problems caused by mold.
3. Gradually introduce feeds into the ration. Moldy hay is unpalatable, and many problems attributed to mold are actually caused by malnutrition. It takes cattle a few days to

adjust to the poor taste and dust; some cattle never adjust.

4. Balance moldy feeds with good quality ingredients.
5. Feed moldy hay outside so as to reduce the effects of dust and spores in the respiratory system.
6. Producers should be aware of the health hazard involved in working with moldy feeds and take every precaution to decrease personal exposure.
7. If problems are encountered, stop using the moldy feed and seek help from a competent source.

Red Clover:-- a mold that causes black patches, a disease of red clover, also produces excessive salivation (slobbering) when infected hay is fed to cattle. After two or three days the cattle will stop eating this spoiled hay. The chemical causing the trouble is an alkaloid and the hay should be destroyed.

Sweet Clover Poisoning:-- Poorly preserved sweet clover or hay silage may result in livestock deaths.

A substance, coumarin, is present in varying amounts in all sweet clover. However, two new varieties, Norgold and Polara have a very low coumarin content and do not pose a problem.

In poorly preserved sweet clover hay or silage from common seed or other varieties, coumarin may change to dicoumarol, a potent anti-coagulant. When eaten by farm livestock, dicoumarol causes the blood to lose its normal ability to clot. Livestock may bleed to death from the slightest injury, either internally or externally.

The first sign of sweet clover poisoning may be the death of one or more animals. Warning signs include stiffness, lameness, dull attitude and swellings beneath the skin over all parts of the body, but primarily at the hips, legs, brisket or neck. Blood may be present in the feces, urine or milk and also the nostrils.

Control

There is no laboratory test for dicoumarol available in Manitoba. Producers feeding sweet clover should observe the following management practices:

- Feed sweet clover for 7 to 10 days and then completely replace it with another forage for 7 to 10 days. It is important that the animals be taken off sweet clover

completely for the 7 to 10 days to give the body time to recover.

- Do not feed sweet clover for at least two weeks and preferably four weeks before calving and during the calving period.
- Avoid surgery (dehorning, castration, etc.) on animals consuming sweet clover.
- Make sure animals have proper mineral supplementation.
- If a problem does develop or is suspected, contact your veterinarian immediately.

Nitrate Poisoning

High levels of nitrate in feed need not be a problem if the producer is aware of the fact and manages the feeding program accordingly.

Nitrates generally build up in young plants subjected to stress conditions such as drought, hail, disease, frost or spray drift. Although high nitrate levels are generally associated with frosted green oats, high levels can also be found in wheat, barley, rye,

corn, sorghum, sudan grass and numerous weeds. Seeds do not appear to accumulate nitrate nitrogen.

Nitrates in the feed are converted during digestion to nitrite, a much more toxic form. When this is absorbed into the blood, the bloods' oxygen carrying capacity is reduced, with the animal's death being comparable to asphyxiation.

A dangerous quantity of nitrate in forage can be detected only by chemical analysis. Any amount of nitrate nitrogen over 0.5 per cent of the total ration (on a moisture-free basis) is regarded as a potential source of trouble.

The signs of nitrate poisoning appear suddenly. Rapid and weak heart beat, body temperature below normal and muscular weakness develop quickly. The animal may die in convulsions within an hour, but normally death results after three to four hours. Most animals that show signs but live, fully recover within 10 to 14 days. Pregnant females frequently abort following recovery from nitrate poisoning. Low daily intake of nitrate apparently causes no evidence of disease.

Control: Nitrate poisoning can be controlled by management. If you suspect your feed may be high in nitrates, have it analyzed **before** feeding. High nitrate forages can be mixed with low nitrate level forages, reducing the overall toxicity of the ration. Feeding adequate levels of energy and Vitamin A reduces the risk of nitrate toxicity. Ensiling forage

tends to result in a 40 to 60 per cent reduction in nitrate level, as fermentation reduces some of the nitrates to gas.

Note: *When nitrate troubles are suspected, consult your veterinarian or agricultural representative.*

Prussic Acid Poisoning

Under certain conditions some plants cause the death of stock from prussic acid, or hydrocyanic acid poisoning. Two native species, arrowgrass and chokecherry and two cultivated plants, flax and the sorghum - sudan grass crops, all common to many areas of Canada--may contain toxic quantities of prussic acid. Because arrowgrass also contains a fairly high salt concentration it is often eaten by salt-deficient cattle and sheep. Chokecherry leaves are not very palatable, but when normal feed is sparse cattle will eat them.

Flax and sorghum-sudans in the early vegetative stage and/or stunted by drought or frost may contain toxic quantities of prussic acid. The effect of the toxin may be reduced when plants are cured as hay or stored for several weeks as silage.

Stock affected by the poison often die a few minutes after eating only a small quantity of the dangerous forage. Normally, animals stagger and struggle before they go down. There is salivation and a gradual increase in the respiratory rate. Death from respiratory paralysis occurs during severe convulsions and the animal usually dies within 30 to 45 minutes. Most animals that live for two hours after the onset of signs will recover. Horses are rarely affected.

If a poisoned animal is discovered, call a veterinarian. Should there be any doubt at all about the safety of feed, have it tested for prussic acid levels at a feed analysis laboratory.

Ergot

Ergot bodies have the same general shape as the seed but they are larger, purple to black in color, and hard. They are caused by a fungus which replaces individual seeds or kernels on the head of the plant. Although it is most common in rye, ergot also infects triticale, wheat, barley, oats, and grasses such as wheat grass, quack grass, smooth brome, wild rye and blue grasses. Ergot is most prevalent during wet seasons and may be present in greater concentrations near the edge of a field than in the center.

Ergotized seed may cause serious problems for animals when consumed in either forage or grain. Ergot causes two types of poisoning. One form causes convulsions, staggering, muscle spasms of the hind legs and sometimes temporary paralysis. This

form of ergotism is usually found in sheep, horses and carnivores but seldom in cattle.

The second, or gangrenous, form of ergotism causes lameness that may be followed by the loss of the end of the tail, ears and perhaps hooves. Loss of these parts is due to a dry form of gangrene caused by impaired circulation and reduced blood supply to the body's extremities. This form of ergotism occurs in cattle and pigs. It may take two weeks to three months for symptoms to develop. In the meantime, rate of gain, milk production, and reproduction suffer. Sows fed ergot generally have their litter but no milk.

Research is not exacting in regard to tolerance levels. The problems with ergot are caused by alkaloids in the ergot body. These alkaloids vary in toxicity. Therefore, ergot bodies vary in toxicity depending on the amount and type of alkaloid they contain. No practical test has been devised to determine the toxicity of ergot samples.

Generally speaking, if grain contains more than 0.1 per cent ergot (10 ergot bodies per litre of grain) it should not be fed to livestock. If more ergot is present, contaminated grain can be mixed with clean grain to reduce the concentration. Grain infected with any amount of ergot should never be fed to pregnant or lactating animals.