Common nutritional toxicities that impact bovine reproduction
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Mycotoxins

These are a group of the most common toxicities affecting bovine reproduction. Caused by molds and fungi, they are found in feedstuffs. They develop in crops in the field and in storage. The most common examples are zeralanone, deoxynivalenol (vomitoxin), and T-2 toxin. Many other types exist, but these three are likely to have the greatest impact on reproduction. Nonetheless, routine diagnostic testing should cover 20 or more different mycotoxins to determine which are at work.

Mycotoxins date back to the Dark Ages, when Claviceps grew on cereal grain crops, producing ergot, which afflicted humans with gangrene and convulsions and caused animals to slough their hooves and abort.

Any one of these toxins extracted from a crop and experimentally added to a diet causes less severe symptoms than mycotoxins that grow naturally in feedstuffs. Scientists postulate that more significant symptoms result from synergistic action with other toxic agents in the diet.

Mycotoxins that negatively affect reproduction are most likely to come from ensiled forages, corn, and small grains.

Signs of mycotoxin toxicity include reduced feed consumption, gastroenteritis, diarrhea, lower milk production, reduced reproductive performance, cystic ovaries, abortions, and, in extreme cases, death. A confirmed diagnosis depends upon multiple clinical signs, laboratory tests of multiple feed samplings, and direct microscopic analysis of feedstuffs to identify mold such as Aspergillus, Fusarium, or Penicillium.

Mycotoxins may occur in isolated pockets of a feed storage facility, depending on the nature of the storage system, areas of oxygen exposure, crop quality in the field, processing of the feedstuff, and moisture content. Because of this, sampling errors may lead to a negative laboratory analysis even though toxin is present in unsampled locations of the storage facility.

The ideal management response to a confirmed diagnosis of mycotoxicosis is to remove the toxin-contaminated feed from the diet or to at least dilute the mycotoxin-carrying feedstuff. Toxin binders such as bentonite, Neutrotox™, or Omnigen™ are another solution. Omnigen™ also has positive neutrogenic effects. No antidotes to these toxic agents exist.

Molybdenum

Molybdenum (Mo) is part of an enigma related to copper and cobalt utilization. Molybdenum uptake by forage plants increases in soils with pH over 6.7. High soil pH also decreases the uptake of cobalt (Co) by forage plants. The increased uptake of Mo at higher soil pH combined with available sulfur creates a biologically active complex, thiomolybdate. Thiomolybdate binds copper (Cu) in the gastrointestinal tract, preventing absorption. High soil pH, while causing higher Mo uptake, simultaneously reduces Co uptake. So, a dual deficiency of Cu and Co is common.

This conundrum results often in legume forages with high organic content, high pH soils, and aggressive soil liming.

Bovines require small amounts of Mo as an essential component of xanthine oxidase, aldehyde oxidase, and sulphite oxidase, which are involved in intracellular ATP utilization.

The pre-eminent indication of Mo toxicity is a secondary Cu deficiency with a primary Co deficiency. Molybdenum levels above 1.5 mg/kg, with increased incidence of ketosis, is considered the threshold for the interaction with Cu and Co.

Treatment options include injectable Multimin™. Neutralize the effects of Mo toxicity by raising dietary copper levels with a chelated copper source or copper sulfate. The level should be adjusted from a range of 6:1 to 8:1 Cu:Mo ratio, plus 10 mg/kg base supplementation. Use B12 injections after calving for cows exhibiting ketosis caused by cobalt/vitamin B12 deficiency.
High blood urea nitrogen (BUN)

Dairy cows, because of high milk production, have a higher protein requirement than beef cows or dairy heifers. By having a rumen, cows utilize rumen degradable protein (RDP) and rumen undegradable protein (RUP) for maintenance, growth, and milk production. A 60:40 RDP-to-RUP ratio is recommended. Water is the first limiting nutrient of ultimate importance, just ahead of the energy requirement. Energy is necessary for the rumen microflora to break down RDP into ammonia, then re-assimilate it into usable amino acids for absorption in the abomasum and the small intestine.5

Diets with excessive RDP and/or inadequate energy reduce the rumen microflora’s ability to efficiently re-assimilate ammonia into amino acids. This causes ammonia to escape through the rumen wall into the portal blood stream. The liver conjugates ammonia to plasma urea nitrogen (PUN), which causes higher blood PUN. When ammonia concentration exceeds the liver’s capacity, excess ammonia can also build up in tissues including the uterus and ovaries.

Plasma urea nitrogen directly correlates to milk urea nitrogen (MUN). High PUN also increases urea and ammonia in follicular fluid. This causes lower embryo cellular cleavage rates. Milk urea nitrogen analysis is a very inexpensive test readily available through Dairy Herd Improvement (DHI) record systems and can also be sampled from bulk milk. Generally, MUN levels should be below 14 mg/dl. Levels of >16.0 is a red flag that PUN is negatively affecting reproductive performance.

One elevated herd bulk milk tank or DHI herd analysis, however, should rarely be cause for action. The diagnositician should check monthly for a trend in PUN.

Cows on high protein diets (23%) or exclusively legume forage diets have higher urea nitrogen in uterine fluid than cows on 12% crude protein (CP) rations. Uterine pH normally increases from 6.8 to 7.1 on day seven of the estrous cycle (luteal phase), but pH does not increase when cows have excessive RDP.6 Pregnancy rates are lower in recipient heifers after transfer of embryos recovered from high PUN cows vs. moderate PUN cows (11% vs. 35%). Urea acts on oocyte maturation but does not impede the developing embryo.7 Pregnancy rates are not affected by high PUN when good quality embryos from normal PUN dams are transferred to recipients with high PUN.8

A diagnosis of the effect of PUN on pregnancy rates must be based on PUN or MUN analysis and a ration analysis. The ration analysis should include CP and predictions of RDP and RUP, as contrasted to dietary energy. A nutritional modeling program is helpful in ration evaluation.

Treatment and prevention of a negative reproductive impact caused by dietary protein requires the ration to be reformulated to lower CP and RDP and correlate them to energy.

Subclinical nitrate toxicity

Nitrates are noted for causing several rare, severe health conditions in ruminants and, at high levels, even death. The rare but acute form of nitrate toxicity is most common from consuming nitrate fertilizers, grazing heavily fertilized fields, or drinking runoff from heavily fertilized fields. Plants such as Brassica, cereal grains, or sweet clover may also contain high levels of nitrates. The purpose of this discussion is to describe subclinical cases of nitrate toxicity rather than clinical life-threatening cases. In acute cases, symptoms include abdominal pain, weakness, drooling, blue discoloration of the mouth, mouth breathing, collapse, coma, and death,9 though not with lower levels of dietary nitrates.

Cows fed high nitrate corn silage (0.78% NO3) as half their total digestible nutrient do not demonstrate clinical signs of nitrate toxicity. They also exhibited no noticeable effect on vitamin A levels, milk production, or reproduction.10 Nonetheless, low level nitrate toxicity may occur in certain instances. Pubertal heifers fed daily diets containing 0, 440, and 660 mg/kg of body weight, starting three estrous cycles prior to breeding, were impacted at the highest dietary level.11 Conception rate was lower in heifers fed 660 mg of dietary nitrates. One abortion occurred in heifers fed the lowest level of nitrates (which was not significant). Two abortions and two deaths occurred in heifers on the highest dietary level of nitrates. There were no differences in estrous cycles, length of gestation, birth weight, or performance of the calves. Vitamin A and carotene levels were unaffected. Milk production postpartum was the same for all groups.
Gossypol toxicity

Gossypol is a naturally occurring phenolic compound produced by pigment glands in cotton stems, leaves, seeds, flower buds and also contained in cottonseed meal, hulls, and cottonseed cake. Gossypol is toxic to most monogastric animals, but somewhat less so to ruminants because of their ability to detoxify it in the rumen. In the bovine, gossypol-containing cottonseed and other cottonseed products cause a male fertility issue at dietary gossypol levels as low as eight grams per day. It appears dairy cows are not affected until approaching 20 gm/day of gossypol consumed orally.

Gossypol levels vary depending upon the extracted product produced. For instance, cottonseed hulls have much safer and lower levels than cottonseed cake.

Signs of toxicity first appear in bulls on a whole cottonseed diet. A bull can be rendered infertile within a couple of months of consuming no more than 2.5 kg of cottonseed containing as little as one percent gossypol.

Quantity purchases of whole cottonseed for dairy cattle should be made contingent on analysis of the gossypol content. If not being provided to bulls, whole cottonseed fed to cows is acceptable with a level of two percent gossypol. The diet for the cows should not exceed 2.5 kg/hd/day. If the whole cottonseed exceeds 12 gm/cow/day, vigilance is needed. It has been confirmed that 20 gm/cow/day of gossypol may be fatal to dairy cows.

Natural plant toxicities

A number of natural plant toxins may also impact the bovine species. Many bovines, however, will not touch these plants until pastures are short and they are searching for something to eat. The following tables categorize these natural plant toxins.

<table>
<thead>
<tr>
<th>Toxic Plant</th>
<th>Toxin</th>
<th>Effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veratrum californicum (False hellebore)</td>
<td>Jervine, cyclopamine</td>
<td>Facial &amp; skeletal defects, Tracheal stenosis</td>
<td>James et.al., 1992</td>
</tr>
<tr>
<td>Lupinus spp.</td>
<td>Anagyrine quinolizidine alkaloid</td>
<td>Cleft palate</td>
<td>James et. al. 1992</td>
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<tr>
<td>Poison hemlock</td>
<td>Piperidine alkaloids</td>
<td>Skeletal defects</td>
<td>James et.al. 1992</td>
</tr>
<tr>
<td>Locoweed</td>
<td>Indolizidine alkaloid</td>
<td>Fetal edema, enlarged rt. heart ventricle</td>
<td>James et.al. 1992</td>
</tr>
<tr>
<td>Tree tobacco</td>
<td>Piperidine alkaloid</td>
<td>Skeletal defects, cleft palate</td>
<td>James et.al. 1992</td>
</tr>
<tr>
<td>Selenium-accumulators</td>
<td>Selenium</td>
<td>Deformed hooves</td>
<td>James et.al. 1992</td>
</tr>
<tr>
<td>Ponderosa pine</td>
<td>unknown</td>
<td>Light birth weight</td>
<td>James et.al. 1992</td>
</tr>
</tbody>
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Table 2. Plants causing abortion or embryonic deaths in livestock

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Locoweed</td>
<td>Indolizidine alkaloid</td>
<td>Abortion, embryonic death</td>
<td>Delayed placentation</td>
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<tr>
<td>Broom snakeweed</td>
<td>unknown</td>
<td>Abortion, premature birth</td>
<td>Kingsbury, 1984</td>
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<tr>
<td>Ponderosa pine</td>
<td>unknown</td>
<td>Abortion, premature birth</td>
<td>James et. al. 1989</td>
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<tr>
<td>Little leaf horsebrush</td>
<td>unknown</td>
<td>Abortion</td>
<td>Johnson, 1974</td>
</tr>
<tr>
<td>Veratrum californicum</td>
<td>Jervine</td>
<td>Embryonic &amp; fetal death</td>
<td>Binns et. al. 1963</td>
</tr>
</tbody>
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References