Introduction

Acidosis is the major problem associated with feeding grain to cattle and is probably the cause of many secondary problems encountered during the first 40 days in the feedlot. Acute acidosis results in death; but the normal consequences include severe inappetence; lameness and lost production.

The cause of acidosis rapid fermentation of starch leading to the accumulation of acid in the rumen and/or the hindgut (colon and caecum). Acidosis can occur in the rumen from fermentation of ingested feed as well as in the hindgut a result of starch passing through the small intestine without complete digestion. The site of starch digestion and risk of acidosis is therefore a combination of feeding management and the animal’s digestive capacity.

Once lactic acid starts to accumulate there is a significant increase in acidity and a decrease in pH. For this reason, many of the successful strategies for managing acidosis focus on reducing lactic production or increasing its rate of conversion to volatile fatty acids as these acids are more rapidly absorbed from the rumen and hindgut and do not cause severe acidosis.

In addition to nutritional management and manipulation of fermentation to control acidosis it may also be possible to select those animals that are resistant to acidosis and for this reason it is important to determine if susceptibility to acidosis is under genetic control.

This paper deals with practical management options for managing acidosis as well as introducing some new possibilities that are still at the research stage.

1. **Choice of grain** can play an important part in reducing the severity of acidosis. Wheat and barley ferment far more rapidly than sorghum or maize. On the other hand oats ferment rapidly but do not have the high starch content that makes barley and wheat high-risk grains. Price and availability often dictate the choice of grain and we then need to now how to manage the situation. The project entitled ‘Premium grains for livestock’ supported by GRDC and MLA amongst other R&D corporations, has
developed assays to determine site of starch digestion and risk of acidosis. Figure 3 shows how variable fermentation of wheat and barley can be. It also shows the very high intestinal digestibility of triticale cultivars “Abacus” and “Madona”. As we develop confidence in the in vitro assays and adapt them to NRI it is likely that we will be able to test grains prior to feeding in order to determine risk of acidosis and modify management accordingly.

2. **Grain processing** involves either cooking (eg steam flaking) or cracking leading to reduced particle size (milling or rolling). The finer the particles the more rapid the fermentation and a higher risk of acidosis in the rumen. On the other hand steam flaking often does not dramatically reduce particle size but it does make the starch much more digestible in the small intestine. High intestinal digestibility of starch is likely to reduce the risk of acidosis in the hindgut.

3. **Method of feeding** and amount of grain are very important tools that are often overlooked. The first thing is to ensure that all animals are properly fed before going onto a grain feeding regime. Apart from being less likely to over eat, the fact that they have a full gut will help protect them from acidosis. The need for gradual introduction (step up) of grain is well understood. The choice of level of roughage is often set by the difficulty of grinding and mixing and a little extra roughage does not have a major negative effect on performance. Consider use of long roughage to overcome grinding and mixing. Feeding separate hay and roughage can work well in opportunity feedlots. Monensin can be considered as a tool to reduce feed intake and prevent over eating in some situations.

4. **Reducing lactic acid production** is most effectively done with virginiamycin as this is specifically active against S. bovis as a major acid producer. We should do more research on the short-term strategic use of virginiamycin during introduction and diet change. An alternative that shows promise but needs further work is the process of vaccination against key lactic acid producers. This novel approach has been tested in both sheep and cattle trials. Although vaccination shows some promise, results under extreme conditions of acidosis have not been conclusive. There appears to be a good case for testing the vaccine under more “normal” forms of sub-clinical acidosis.

5. **Probiotics** have been investigated as a way to increase lactic acid utilisation. This approach also shows some promise but there are costs and logistical difficulties in delivery of large amounts of specific probiotic organisms to the digestive tract prior to grain feeding.

6. **Buffers and clays** are not considered to play a significant role in reducing acidosis. They act too late in the process of acidosis development. In the case of buffers they don’t
provide enough alkali to reduce the acidity. Even in dairy cattle it appears that the same effects are achieved by using sodium chloride as sodium bicarbonate. Clays tend to reduce digestibility as well as binding water and ions and thus reduce diarrhoea. Therefore although they appear to provide benefits because faecal consistency improves it is unlikely that they produce significant benefits in terms of animal production.

7. Genetic selection for “resistance to acidosis” has been investigated by Channon et al. (2002) and shown to have some promise. It is well known that only some animals suffer from grain poisoning when a group of cattle is exposed to too much grain. Although the reason behind differences between animals is not clear it is likely that variation in intestinal enzyme digestion could be important. Poor starch digestion in the intestine will result in fermentable starch entering the hindgut and causing acidosis, diarrhoea and reduced production. Channon and his colleagues used a simple measure of faecal pH to look for genetic differences between cattle and found small but measurable differences between sires. While faecal pH has a few inaccuracies there may be other simple measurements that we can use to investigate genetic differences between animals and select for this important trait in feedlot animals.

Summary
There is no single “silver bullet” to remove the risk of acidosis but a combination of old and new techniques can improve the safety of grain feeding quite dramatically.

- Make sure that animals are well fed (full) of hay or pasture before starting them on grain;
- Manage the step-up process, increasing grain content, carefully;
- Commercial tests for selection of grains for low fermentability and efficient intestinal digestion may be available in the future – for the present some cultivars of triticale (eg “Abacus”) and oats offer this characteristic;
- Keep the grain particle size as big as possible to reduce the risk of rumen acidosis, tempering, reconstitution and steam flaking achieve this goal;
- Steam treatment is an excellent way of increasing small intestinal starch digestion and reducing the risk of hind gut acidosis;
- Virginiamycin reduces the risk of acidosis by specifically removing lactic acid producing bacteria – further work on more strategic use is justified;

- Vaccination as an alternative to using antibiotics holds promise but is not yet available;
- Leaving out bicarbonate and clays such as bentonite is not likely to increase the risk of acidosis and will reduce costs;
- Genetic selection for resistance to acidosis is likely to be effective provided that we identify a cheap and accurate way of assessing “resistance”.

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