TechNote 14

Monitor and mitigate ketosis

IN THIS TECHNOTE

14.1 Understand what causes ketosis

Ketosis is a metabolic disease that occurs when the cow is in a severe state of negative energy balance.

In early lactation, all cows are in a state of negative energy balance; however, the magnitude of this can vary. While in a negative energy balance the cow will mobilise body fat reserves (lipolysis) and the resulting free fatty acids are oxidised in the liver (via ß-oxidation) to produce energy that can be used by some of the body cells.

For more details see TechNote 7: Lipid metabolism.

When the negative energy balance is severe, the cow mobilises excessive amounts of body fat but there is a high demand for glucose synthesis in the liver, which can limit the oxidation of free fatty acids. Instead, ketone bodies (acetone, acetoacetate, and ß-hydroxybutyrate; BHBA or BOH), which are intermediates in the oxidation process, are produced and released into circulation. In small amounts, the cow can use BHBA as an alternative energy source to help meet its requirements,

Ketosis can be divided into three types:

Type 1: Sudden drop in energy intake

- Ketosis is a result of a sudden drop in energy intake.
- This can be due to underfeeding or adverse weather events (e.g. floods, snow storms) that prevent cows from eating sufficient amounts of dry matter.

Type 2: Post-calving

- Type 2 ketosis occurs to a small degree in all cows post-calving when they are mobilising body reserves to meet the energy demands of milk production.
- However, clinical ketosis only occurs when cows are in a severe state of negative energy balance.

- Cows that are too fat at calving (BCS greater than 5.0) are particularly at risk of type 2 ketosis. For example, cows that calve at a BCS 6.0 are twice as likely to suffer from ketosis than those that calve at BCS 5.5. This is due to greater BCS loss, reduced dry matter intake (DMI), and impaired liver function post-calving in over conditioned cows.
- Cows that have been overfed pre-calving (i.e. offered more than their energy requirements) are also at risk of type 2 ketosis.

For more details see TechNote 12: Feed the transition cow appropriately.

Type 3: Silage ketosis

- Silage ketosis is due to cows ingesting poor quality silage.
- The silage undergoes a secondary fermentation and when ingested increases BHBA levels and the risk of ketosis.

14.2 Know how ketosis is diagnosed

Because clinical signs may not always be present, ketosis is often diagnosed based on the level of circulating BHBA in the blood to classify cows as either clinical or sub-clinical:

- **Clinical ketosis:** blood BHBA levels greater than 2 mmol/l
- **Sub-clinical ketosis:** blood BHBA levels between 1.2 and 2.0 mmol/L

However, because butyrate is a volatile fatty acid that is produced in the rumen, the concentrations of BHBA in the blood are also affected by diet, or more specifically the carbohydrate source in the diet.

For more details see TechNote 5: Carbohydrate metabolism.

Q: What are the signs of ketosis?

A: Ketosis can be displayed in two ways: nervous form, where cows are uncoordinated, excitable and show unusual behaviours (e.g. walking in circles, licking fences); or wasting form, where cows are lethagic, have decreased DMI, milk production and often sweet smell on their breath.

Pasture-fed cows produce more ruminal butyrate than those fed a diet high in starch-based supplements or a total mixed ration. As butyrate is converted to BHBA in cells in the rumen wall, pasture-fed cows can have double the basal blood concentrations of BHBA than those fed a higher starch-based diet (Figure 1). Therefore, the incidence and prevalence of elevated blood BHBA greater than 1.2 mmol/L are relatively high in pasture-based systems.

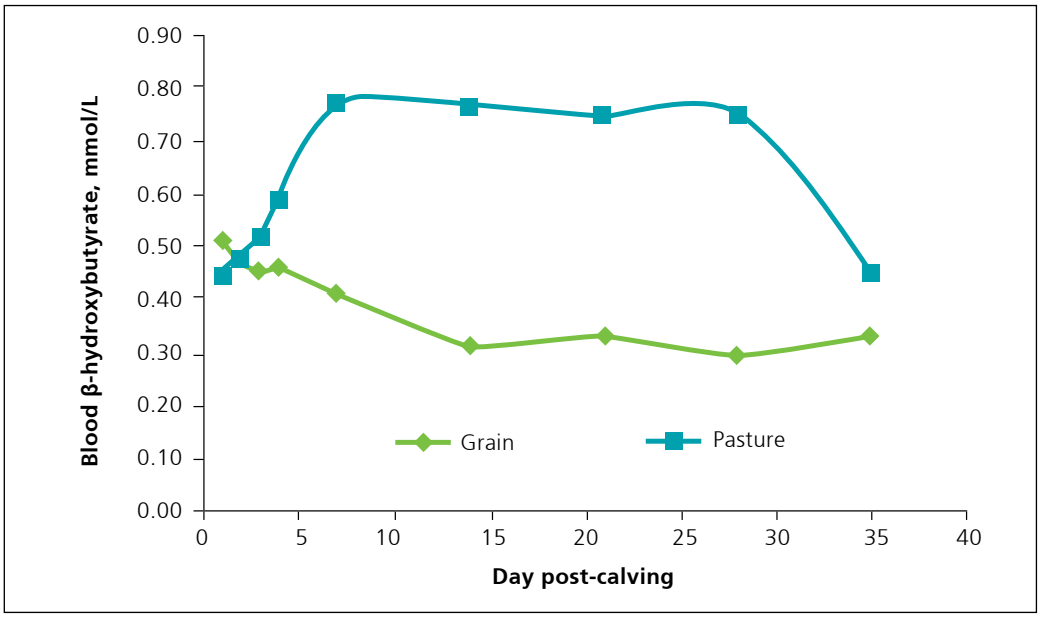

Figure 1. Blood BHBA concentrations in dairy cows fed the same energy as pasture, or pasture plus grain. Milk energy output and BCS change were not different between groups (adapted from Roche et al., 2010).

This effect may explain, at least in part, inconsistent outcomes between studies investigating associations between blood BHBA and animal performance. For example, previous NZ research indicated that, on average, 68% of cows had subclinical ketosis (using a threshold of BHBA greater 1.2 mmol/L) at least once during the first 5 weeks of lactation; however, there was large variation between herds. Furthermore, although cows with BHBA greater than 1.2 mmol/L had a 7% lower 6-week incalf rate than those that remained below 1.2 mmol/L, there was no negative effect on milk production or final pregnancy rate.

In contrast, a more recent study indicates that a threshold of 1.2 mmol/L BHBA in blood is not always associated with poor animal performance in a pasture-based system. An intensive sampling regime of testing 1000 cows in 3 herds for blood BHBA three times per week over the first 5 weeks post-calving, indicated large differences between herds in the pattern of blood BHBA and its association with various reproductive and milk production parameters. For example, although elevated BHBA was associated with a lower 6-week incalf rate in one herd, the opposite relationship occurred in the other 2 herds.

Based on this research, and because diet can influence blood BHBA concentration, we recommend that:

- **In our pasture-based systems, sub-clinical ketosis should not be diagnosed on BHBA concentrations alone.**

Although BHBA concentrations greater than 1.2mmol/L are associated with reduced reproductive performance and health outcomes in housed systems, these relationships are inconsistent between herds under grazing conditions.

Therefore, BHBA concentrations greater than 1.2 mmol/L are not necessarily indicative of sub-clinical ketosis.

- **Additional indicators of energy balance and other factors should be considered.**

A combination of variables should be considered alongside blood BHBA, such as blood non-esterified fatty acids (NEFA) and glucose concentrations, BCS and feeding levels, clinical symptoms, and days post-calving. If NEFA concentrations are greater than 1.0 mmol/L and glucose concentrations are lower than 3.0 mmol/L, then the risk of clinical and sub-clinical ketosis is increased.

For example, during spring in pasture-based systems, if there are high BHBA concentrations but NEFA concentrations are within the optimal range, then this is unlikely to represent ketosis. If high BHBA concentrations are associated with high NEFAs, then this may reflect a severe negative energy balance and is likely to represent ketosis.

14.3 Understand how to reduce the risk of ketosis

The risk of ketosis can be reduced by managing feed allocation and BCS (both pre- and post-calving) and by paying attention to cow behaviour and adverse weather conditions. Ensure feed supply meets feed demand. Consider the following:

- Ensure cows calve at recommended BCS targets (BCS 5.0 for mixed aged cows and 5.5 for heifers and second calvers).
- If at or above target BCS, feed springers 90% of their energy requirements during the last two to three weeks precalving.
- Ensure cows have adequate feed allocated post-calving by using the spring rotation planner. Target post-grazing residuals of 1500 – 1600 kg DM/ha, taking into account weather conditions and pasture utilisation.
- Avoid sudden feed shortages, if possible. Allocate pasture accurately and use supplementary feeds if there is a pasture deficit. If feed restrictions are unavoidable, try to introduce the feed deficit gradually and consider using once-a-day milking to improve cow energy balance.
- Ensure any silage fed is of high quality and stored correctly.

- Cows that have had difficulty calving or metabolic issues, such as milk fever, can benefit from a starter drench that provides immediate energy, particularly if they have a poor appetite.

For more details on transition cow feeding see TechNote 12: Feed the transition cow appropriately.

14.4 Treatment of clinical ketosis

If a cow shows clinical signs of ketosis, seek advice from your vet. Successful treatment of clinical ketosis will involve providing cows with oral drenches or drugs that stimulate an increase in blood glucose.

Treatments that have been used in severely affected cows include intravenous metabolic solutions (e.g. 4-in-1; Ca, Mg, P, glucose), intravenous dextrose and multivitamin injections.

If the affected animal is still able to stand, the energy content of the diet should be increased including using oral drenching (twice daily) of glucose precursors such as monopropylene glycol (e.g. Ketol™).

14.5 Is there value in treating subclinical ketosis?

Recent US studies have investigated the effects of monopropylene drenching in cows that have sub-clinical ketosis. These studies indicate that metabolic status, survival, and reproduction can be improved when housed cows are drenched daily until they test below 1.2 mmol/L BHBA in blood.

However, recent NZ research indicates that although this strategy reduces blood BHBA, the effects vary between herds under pasture-based systems, and in some cases reproductive performance may actually be reduced (Figure 2).

Therefore, using a threshold of 1.2 mmol/L BHBA in blood to treat cows with monopropylene glycol drench (without other symptoms of metabolic illness) is not recommended to improve cow performance in pasture-based systems.

For more info see dairynz.co.nz/pillars-bhb

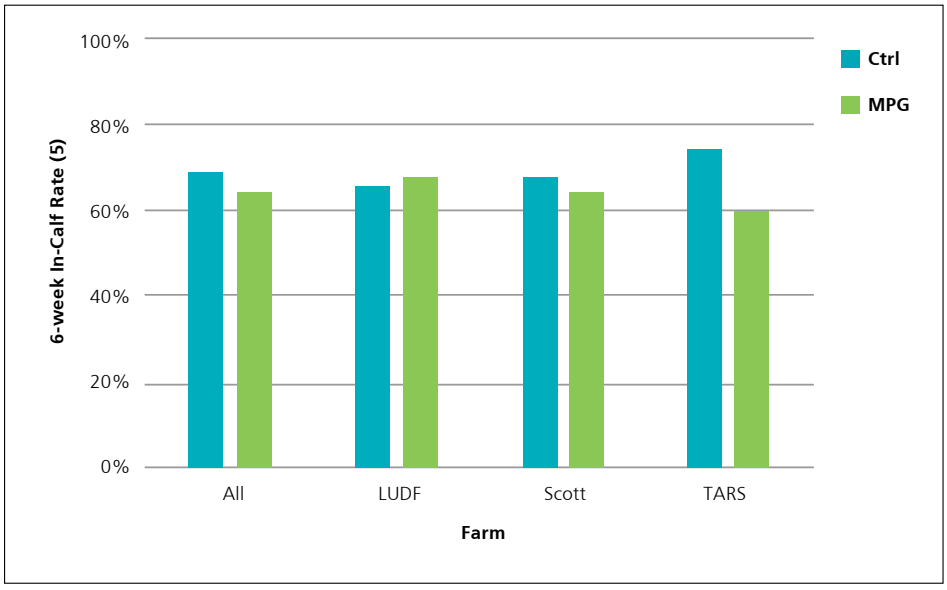

Figure 2. The effect of using monopropylene glycol (MPG) drench to treat cows diagnosed with moderate hyperketonaemia (blood BHBA between 1.2 and 3.0 mmol/L) on 6-wk In-Calf rate in 3 herds (Scott Farm, Waikato; Taranaki Agricultural Research Station (TARS), Taranaki; Lincoln University Dairy Farm (LUDF), Canterbury). Controls (Ctrl) remained untreated.

14.6 Rumensin™ (or other ionophores)

Rumensin™ contains monensin sodium, an ionophore that alters the population of micro-organisms in the rumen. Rumensin™ targets the microbes that are responsible for the fermentation of structural carbohydrates and thus results in an increased population of the microbes that convert non-structural carbohydrates to propionate. This has the effect of increasing ruminal production of propionate relative to butyrate.

Accordingly, Rumensin™ lowers blood BHBA concentrations and thus may reduce the risk of ketosis in grazing dairy cows; however, it does not improve BCS or reproductive performance.

Rumensin™ also attacks some of the microbes that hydrolyse (break down) protein in the rumen. This results in a greater amount of protein that by-passes the rumen and reaches the small intestine.

For more details see TechNotes 7: Protein metabolism, and 5: Carbohydrate metabolism.

These outcomes should theoretically increase milk protein and reduce milk fat yields. However, responses to Rumensin™ in pasture-based systems are inconsistent and may be related to the quality of the pasture and/or effects on DMI. On average, grazing cows supplemented with Rumensin™ produced 15 - 20 g more milk protein than those that were not supplemented, with a range in the milksolids response of -80 to +80 g. The greater responses tended to occur when diet digestibility declined (e.g. summer pastures), and lower responses occurred when high quality spring pastures were fed. More experiments are necessary to gain a better understanding of the effects of Rumensin™ in pasture-fed dairy cows and the interactions with dietary quality and composition.

Rumensin™ can be an effective bloat control agent; however, it does not prevent acute/severe bloat.

14.7 Further reading

Duffield, T. F., S. LeBlanc, R. Bagg, K. Leslie, J. Ten Hag, and P. Dick. 2003. Effect of a monensin controlled release capsule on metabolic parameters in transition dairy cows. Journal of Dairy Science. 86: 1171 – 1176.

Duffield, T. F., A. R. Rabiee, and I. J. Lean. 2008. A meta-analysis of the impact of monensin in lactating dairy cattle. Part 3. Health and reproduction. Journal of Dairy Science. 91: 2328 – 2341.

NRC. 2001. Nutrient requirements of dairy cattle (7th rev. ed). Washington, United States of America: National Academy Press.

Oetzel, G. R. 2007. Herd-level ketosis – diagnosis and risk factors. Proceedings of the 40th Annual conference of Bovine Practitioners, Vancouver, Canada.

Guillund, P., O. Reksen, Y. T. Grohn, and K. Karlberg. 2001. Body condition related to ketosis and reproductive performance in Norwegian dairy cows. Journal of Dairy Science 84. 1390 – 1396.

Phyn C.V.C., B. Kuhn-Sherlock, S-A. Turner, T.M. Grala, C.R. Burke, J.R. Roche. 2017. Contract session: An overview of postpartum hyperketonaemia and its associations with cow health and performance in pasture-based diary systems. Proceedings of the New Zealand Society of Animal Production, 77:104-106

Roche J.R., J.K. Kay, C.V.C. Phyn, S. Meier, J.M. Lee, C.R. Burke. 2010. Dietary structural to nonfiber carbohydrate concentration during the transition period in grazing dairy cows. Journal of Dairy Science 93:3671-3683

Roche, J. R., 2012. Avoiding metabolic diseases around calving. DairyNZ Technical series. June 2012: 13 - 18.

Schultz, L. H. 1971. Milk fever and ketosis. Digestive Physiology and the Nutrition of Ruminants. D. C. Church, ed. Illinois, United States of America: Waveland Press, Inc