

Minimising hypocalcaemia in the transition cow

There are numerous factors that affect fertility and reproductive performance of dairy cows, including genetics, environment and nutrition. The transition period is one of the most stressful and physiologically demanding times in the cow's production cycle and is the time when metabolic disturbances are common.

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One such metabolic problem occurs with the initiation of lactation when there is a tremendous increase in the requirement for calcium in order to meet demands for milk synthesis. Around 20-30g calcium per day is needed for milk production compared with 8-10g per day for foetal development just prior to calving.

Thus, metabolic adaptations must take place to support the increased need for calcium. If they do not take place soon enough or are of sufficient magnitude, the concentration of calcium in the blood drops below a critical threshold and clinical and subclinical hypocalcaemia (milk fever) can result.

Role of calcium in the body

Calcium is the most abundant mineral in the body with the vast majority being found in bone. The remainder (only ~1%) is vital for nerve function, muscle contraction and cell signalling.

There is internal regulation of blood calcium levels involving parathyroid hormone (PTH), calcitonin and vitamin D3 (1, 25-dihydroxycholecalciferol), known as calcitriol.

Regulation involves calcium moving into or out of the skeleton, as well as dietary uptake of calcium from the small intestine.

In situations of low blood calcium, PTH is secreted resulting in a greater amount of skeletal calcium that is resorbed and released into the

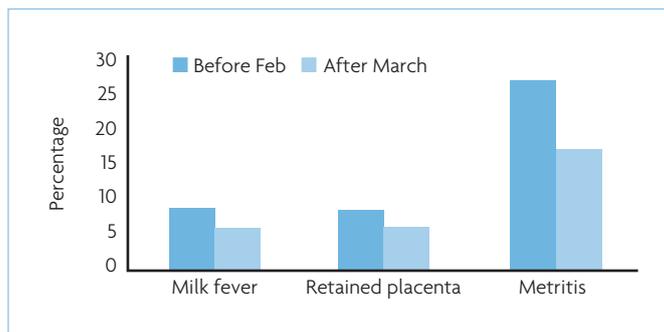


Fig. 1. Field trial, Poland 2015: Incidence of metabolic diseases.

blood. Parathyroid hormone release stimulates renal production of calcitriol, which then increases the absorption of calcium from the small intestine.

Magnesium is also involved in appropriate PTH response to lower blood calcium levels; hence the importance of adequate levels of this mineral in the diet of dry cows.

Clinical vs subclinical

Dry period diets are often high in potassium, which increases blood alkalinity, resulting in insensitivity of target tissues to PTH. This lack of response by tissues to PTH results in reduced calcium release from bones, reduced renal activation of calcitriol and reduced renal reabsorption of calcium.

Clinical hypocalcaemia (milk fever) is certainly becoming much more widely recognised in terms of the provision of treatment plans when signs are apparent and it is well known that this disease increases the cow's susceptibility to mastitis, retained placenta, displaced abomasum, dystocia and ketosis.

Subclinical hypocalcaemia is defined as low blood calcium concentrations without clinical signs and, unfortunately, because of its non-symptomatic nature, is often not dealt with as efficiently as its clinical counterpart.

Subclinical hypocalcaemia impacts fresh cow health, future milk production, and reproductive performance. Cows with lower

blood calcium concentrations within the first day after calving are more likely to have a displaced abomasum, ketosis (and fatty liver), retained placenta and resulting metritis, and mastitis.

Some studies have shown a decrease in feed intake and rumination and corresponding higher non-esterified fatty acid (NEFA) concentrations after calving.

As previously mentioned, hypocalcaemia has a range of effects on the cow that increases the risk of other diseases and issues.

Goff (2008) notes that hypocalcaemia reduces rumen and abomasal motility increasing the risk of abomasal displacement, as well as reducing feed intake so that greater body fat mobilisation occurs in early lactation. This, in turn, increases the risk of ketosis.

Goff also describes the fact that hypocalcaemia reduces all muscle

contraction including the teat sphincter muscle responsible for closure of the teat orifice after milking, thus increasing the risk of mastitis.

As if this was not enough, Kimura et al. (2006) demonstrated a direct impairment of immune cell response.

A study by Martinez et al. (2012) from the University of Florida also revealed that cows with subclinical hypocalcaemia were at greater risk of developing metritis when compared with normal cows.

These cows also showed lower pregnancy rates and a longer period of time to pregnancy. So it is clear that even subclinical hypocalcaemia has far-reaching implications for dairy cow production.

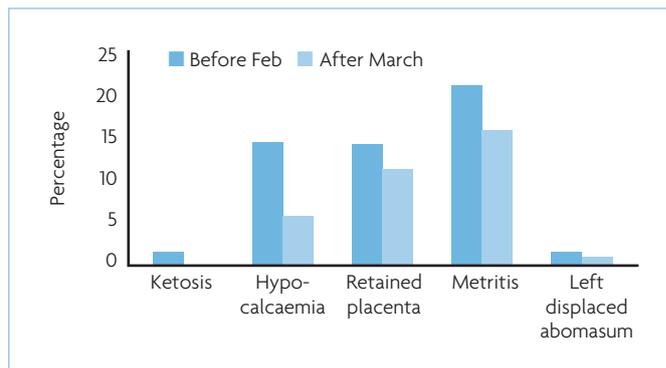
The cost to producers of subclinical hypocalcaemia can actually be much greater than that of the clinical disease. While the cost per case may be higher for the latter, the significantly greater number of affected cows, coupled with the effects on other aspects of metabolism, make the cost of subclinical hypocalcaemia potentially four times greater than that of clinical milk fever.

Prevention strategies

As with most things, prevention is the key. Most prevention strategies are concerned with manipulation of the close-up diet.

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Fig. 2. Field trial, Romania 2014-2015: Incidence of metabolic diseases.



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The idea behind them is to try to ensure the cow's metabolism is ready to deal with the rapid and substantial increase in calcium demand following calving.

● Dietary calcium restriction in the close-up dry period

This has been the traditional approach and has included such practices as feeding diets very low in calcium and adding materials to the diet, such as zeolite, in order to prevent calcium from being absorbed.

However, it is difficult to formulate low calcium diets unless whole crop or maize silage is being fed and the animal still requires a certain level of dietary calcium to maintain bone integrity.

● Dietary potassium restriction in the close-up dry period

This may also be difficult to achieve under practical conditions and may result in reduced intake of forage, which is undesirable in the period close to calving. One of the keys to preventing hypocalcaemia is to keep dietary potassium as close to National Research Council (NRC) requirements as possible during the dry period (~1% of the diet).

● Additional oral calcium supplementation

Oral calcium drenches post-partum have shown benefit in reducing the incidence of milk fever.

● Vitamin D3

Dosing with vitamin D3 for a short period of time prior to calving has had mixed results.

The amount required to elicit a response often has a negative impact elsewhere in the body and there remains problems associated with timing of administration.

● Dietary cation-anion difference (DCAD)

A more effective and practical approach has been to look at the overall balance between key cations

and anions that determine blood pH. Dairy cows are often in a state of metabolic alkalosis, which predisposes them to hypocalcaemia.

The idea behind DCAD is to reverse the alkalosis and generate mild metabolic acidosis during, at most, the three weeks prior to calving.

This is done by monitoring dietary levels of sodium, potassium, chloride and sulphur and can be calculated as shown in the box below.

Target DCAD for a close-up dry cow is -100mEq/kg DM. Animals on DCAD diets must be closely monitored via urine pH and must receive sufficient dietary magnesium (-0.4%) and dietary calcium at 1%. Close-up dry cows on a DCAD diet should have a target urine pH of <7, preferably nearer 6 (optimal is 6.2-6.8 for Holstein cattle).

Anionic salts are a common method of manipulating the DCAD but can have issues with palatability.

Again, close monitoring of urine pH will show whether the salts have been effective, including whether too many anions are being fed resulting in a more acidic pH (<5.5).

Prepartum transition diets

Alltech has developed a specific solution for the close up period (three weeks before calving), called Calving Care, in order to reduce the risk of metabolic diseases at calving. It brings different advantages to a diet other than promoting a negative DCAD:

● Helps minimise costly metabolic disorders for increased postpartum performance.

● Helps promote efficient calcium

Calculation for monitoring dietary levels of sodium, potassium, chloride and sulphur.

$$\text{DCAD (mEq/kgDM)} = (\text{Na}\% \times 434.98) + (\text{K}\% \times 255.74) - (\text{Cl}\% \times 282.06) + (\text{S}\% \times 623.74)$$

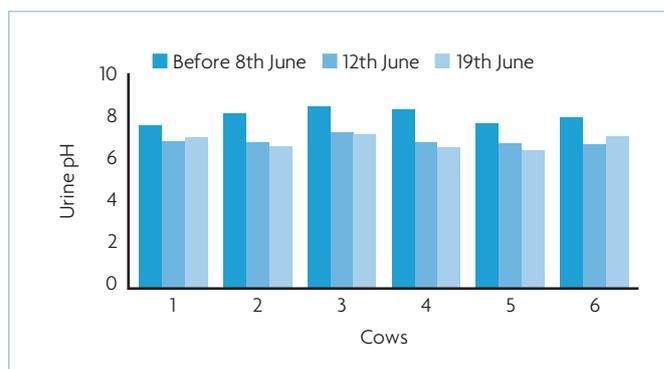


Fig. 3. Field trial, Italy 2015: Urine pH.

metabolism leading to fewer associated metabolic issues.

● Supports dry matter intake, which aids in enhancing earlier peak milk production.

● Provides macroelements to maintain homeostasis for maximum performance.

Calving Care has the advantage over other anionic supplements of delivering the desired anions in a palatable form. Its inclusion into the close-up diet shows an improvement of health status.

Trials showed a reduction of metabolic diseases (Figs. 1, 2 and 3).

Subclinical hypocalcaemia can be diagnosed by blood calcium levels. However, monitoring of the metabolic 'state' of the animal with regards to blood pH, which affects susceptibility to milk fever, can be carried out more practically using urine pH analysis). Animals on Calving Care diets showed a reduction of urine pH after four days and achieved the expected result.

Urine has also been used to estimate mineral status taking into con-

sideration factors such as hydration status and regulation of urinary excretion for certain minerals, when interpreting the results.

Conclusion

Metabolic diseases are costly to dairy operations, none more so than hypocalcaemia.

Cows with both clinical and subclinical forms are at greater risk of suffering from other metabolic and infectious diseases compared with non-affected animals, further increasing cost.

While clinical hypocalcaemia is generally easier to spot, subclinical hypocalcaemia may actually cost the producer more due to the higher number of cows affected.

Therefore, prevention should be the aim of all dairy producers. Numerous dietary strategies exist to try to reduce the risk of hypocalcaemia, including low calcium and potassium diets and the DCAD diet.

Calving Care represents a valid strategy to minimise this risk.

Accurate monitoring and urine pH analysis is a key tool in determining, not only an animal's risk of hypocalcaemia but also whether prevention strategies are working. ■

References are available from the author on request