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METABOLIC DISEASE

Milk fever – an overview

Milk fever or periparturient hypocalcaemia, is a metabolic disease of dairy cows that occurs around the time of calving. During the dry period, calcium requirements are relatively low; but at calving there is a sudden increase in calcium requirement as lactation begins. If this demand is not met quickly enough, the concentration of calcium in the blood drops below a critical threshold, resulting in either clinical or subclinical milk fever.

A cow can lose 23g of calcium in 10 litres of colostrum in one milking; yet, typically, only has 12g of free calcium available to her. This huge demand for calcium must be met from elsewhere to prevent milk fever developing. The average incidence of clinical milk fever in the UK dairy herd is four to nine per cent, while the incidence of subclinical milk fever is estimated to be between 25 to 50 per cent. The average cost of a case of milk fever is approximately £200 (Husband, 2005).

As plasma calcium is required for neuromuscular function, milk fever is characterised by decreased feed intake, decreased heart rate, inhibition of urination and defaecation, rumen and intestinal stasis and reduced rectal temperature. If it is left untreated, cows progress to lateral recumbency, and eventually coma and death.

Reduced feed intake leads to greater fat mobilisation, and one study (Reinhardt et al, 2011) has shown cows with low blood calcium levels post calving (<2mmol/L) have higher concentrations of non-esterified fatty acids (NEFAs). As muscle contraction is inhibited, the teat sphincter relaxes; which leads to an increased risk of mastitis. Hypocalcaemia post calving also directly impairs immune cell response to an activating stimulus, therefore making these cows more susceptible to disease.

As shown in **Figure 1**, the incidence of mastitis and ketosis in cows recovering from

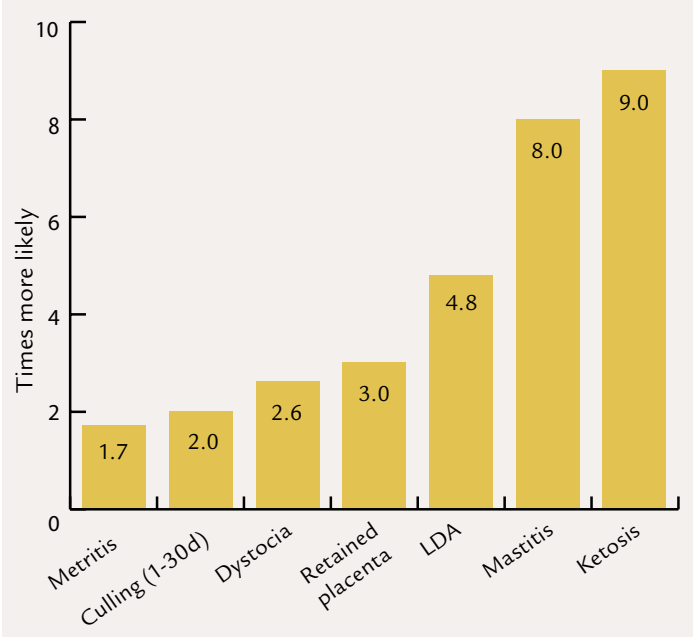

Figure 1. *The incidence of other conditions associated with milk fever (Source: EBVC, 2011).*

Figure 2. *A cow with milk fever and metritis.*

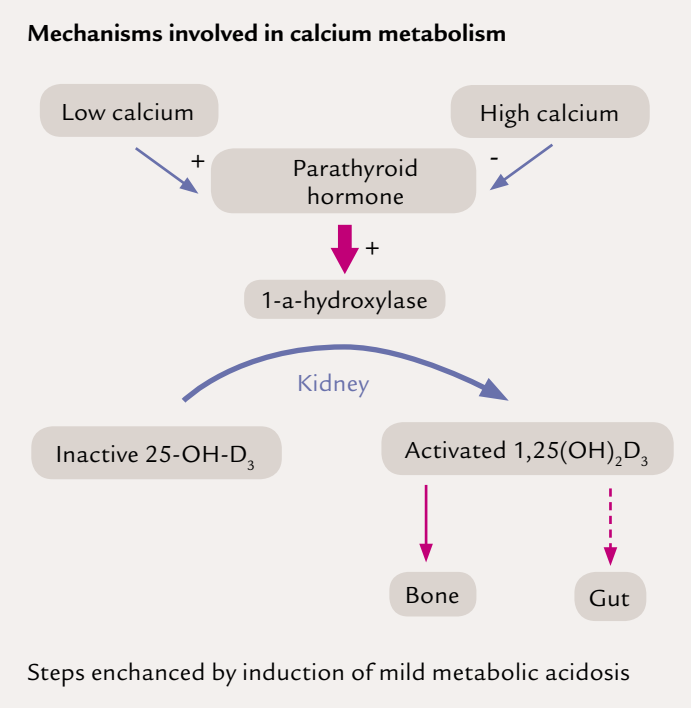

Figure 3. *The mechanisms involved in calcium metabolism in the cow (Husband J, 2005).*

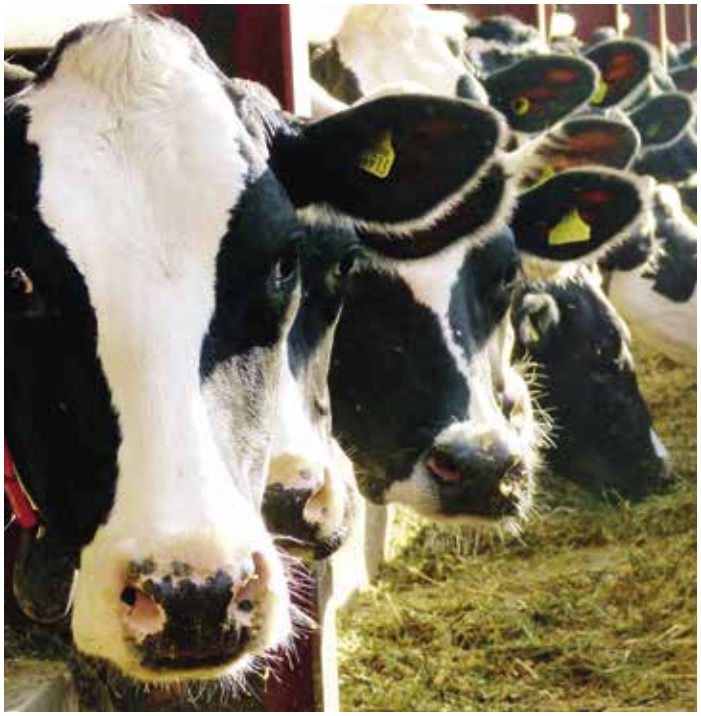

Figure 4. *Milk fever is a disease that can largely be prevented and controlled through careful transition cow feeding.*

milk fever is eight and nine times greater, respectively. The incidences of dystocia, retained foetal membranes, displaced abomasum and uterine prolapse are all greater in cows with milk fever (**Figure 2**).

During the dry period, calcium requirements for skeletal development and maintenance are low – at less than 30g/day. At calving, the demand for calcium is much increased – 2.3g Ca/L of colostrum produced and 1.2g Ca/L milk produced. Calcium quantity in milk increases as butterfats increase. The cow does not have enough calcium in her bloodstream to meet these increased demands, and so it must be replaced by the activation of homeostatic mechanisms to increase its intestinal absorption, reduce renal excretion and increase resorption from the bone (**Figure 3**).

Low blood calcium causes the parathyroid glands to secrete parathyroid hormone (PTH). This hormone increases renal re-absorption of calcium from glomerular filtrate within minutes. Although the amount of calcium available for recovery from the urine is small, this process may still be sufficient to compensate for small decreases in plasma calcium. However, in many cases this mechanism alone is insufficient and continued PTH secretion stimulates calcium resorption from bone. The bones are the source of 95 per cent of the cow's calcium, which is available to the cow in two forms.

There is a soluble, readily available pool of calcium in the fluid surrounding bone cells. PTH acts on bone lining cells to transfer this soluble calcium into the extracellular pool. There is approximately 6-10g of calcium available here, which can be increased by 6-8g in cows under metabolic acidosis.

PTH also activates the renal enzyme responsible for producing the vitamin D metabolite, 1,25 $(OH)_2 D_3$. This stimulates osteoclastic bone resorption activity to mobilise more calcium from the skeleton – cows typically lose nine to 13 per cent of skeletal calcium in first month of lactation. 1,25 $(OH)_2 D_3$ also increases renal tubular reabsorption of calcium and stimulates active transport of dietary calcium across the intestinal wall.

These mechanisms take between 24 and 48 hours to correct the negative calcium balance and both are magnesium dependant. The nadir in blood calcium occurs at 12 to 24 hours post calving. Normal blood calcium for an adult cow is 2.1-2.8mmol/L, with clinical or subclinical milk fever occurring below this (*Merck Veterinary Manual*).

Certain breeds are more susceptible to milk fever, including Jerseys, Channel Islands and Swedish red and whites. It is thought this is a consequence of the increased loss of calcium in their milk with increasing butterfat, and that there are fewer intestinal receptors for 1,25 $(OH)_2 D_3$ in these breeds.

The risk of milk fever also increases with age, rising by nine per cent per lactation. (DeGaris et al, 2009). There are several reasons for this – there is usually an increasing milk yield and, therefore, demand for calcium with increasing age. Older cows are also less able to mobilise calcium from the bone, and have reduced active transport of calcium from the intestines owing to reduced production of the steroid hormone, 1, 25 $(OH_2)D_3$ and reduced receptors on target tissues.

Prevention of milk fever

Milk fever is a disease that can largely be prevented and controlled through careful transition cow feeding (**Figure 4**). Some of the key principles are discussed overleaf.

"A cow can lose 23g of calcium in 10 litres of colostrum in one milking; yet, typically, only has 12g of free calcium available to her"

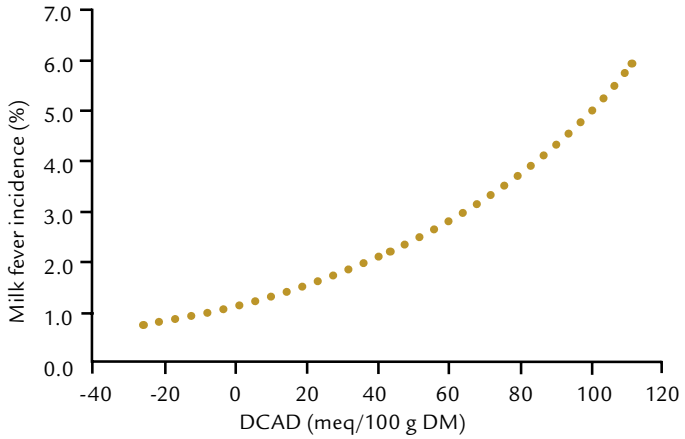

Figure 5. *The linear relationship between DCAB and milk fever incidence (DeGaris PJ and Lean IJ, 2009).*

Dietary cation anion balance (DCAB)

The transition diet can be manipulated to push the cow into mild metabolic acidosis, which has been shown to reduce the risk of clinical milk fever. Reducing the DCAB reduces the risk of milk fever – the two have a linear relationship (**Figure 5**).

DCAB (meq/kg DM) = (sodium + potassium) - (chlorine + sulphur)

Feeding a dry cow diet higher in anionic salts, chloride and sulphur, reduces the DCAB, making the cow acidotic and the blood and urine pH decrease. These acidotic conditions increase the tissues' responsiveness to PTH as the receptors for PTH on bone and kidney cells function better, as well as increasing the production of 1,25 $(OH)2D_3$ and, therefore, increasing active transport of calcium across the intestine. Cows in metabolic acidosis are thus more able to mobilise more calcium and meet the increasing requirements without suffering from milk fever.

A low DCAB dry cow diet can be achieved by two means.

1. Manipulating the diet to achieve a low DCAB

By manipulating the diet, it may be possible to achieve a DCAB of <50-100 without adding anionic salts. The two cations in the DCAB equation, sodium and potassium must be kept at low levels to achieve a low DCAB. Potassium in UK diets is usually the most difficult to control. Grass, grass silage and hay can all be very high in potassium, while maize, whole crop silages and straw are usually low in potassium.

It is crucial to have all forages fed to dry cows analysed for their mineral content, because there can be great variation (**Figure 6**). As a rule, potassium must be kept <1% DM. Potassium in grass can be reduced by having dry cow paddocks with no manure spread on them and using just N fertiliser at moderate to heavy applications. Potassium levels in grass are highest in spring and autumn, when the grass growth is greatest, and the potassium antagonises magnesium, which causes seasonal peaks in staggers or hypomagnesaemia.

2. Adding anionic salts to achieve a low DCAB

It can be difficult to achieve a low enough DCAB just by manipulating forages and concentrates fed. Anionic salts can be added to the diet to reduce the DCAB further. Typical choices are magnesium or ammonium chloride. Care must be taken when adding anionic salts not to acidify the diet and the cow too much. If high levels of anionic salts are fed, palatability is reduced and, therefore, so is dry matter intake (DMI).

Feed component	DCAB (mEq/kg DM)	Comments
Grass silage (2003 season to date)	+390	Extremely variable (up to +1,000), higher with legumes
Grass silage (2002 season)	+391	
Maize silage	+194	Less variable than grass silage
Whole crop silage	+158	Consistently low DCAB
Straw	+139	
Barley	+25	
Brewer's grains	-220	Can be useful to 'dilute' high DCAB silages
Fodder beet	+300	
Kale	+806	
Maize gluten	+137	
Molasses	+681	
Potatoes	+343	
Rape extract	-201	
Wheat	-6	
Sugar beet pulp	-123	
Soya extract	+203	
Wheat feed	+177	

Data supplied by Thomson & Joseph, Norwich.

Figure 6. *The DCAB of common feedstuffs (Husband J, 2005).*

Urine pH is a good indicator of whether the dry cow diet is acidified enough or too much. Target urine pH is 6.2-6.8. If average urine pH is 5-5.5, the diet is too acidic and DMI will be suppressed. Urine pH can be checked 48 hours after dietary changes and should, ideally, be measured after one to three weeks.

Magnesium

Magnesium – although not included in the DCAB equation – is important for the prevention of milk fever. It increases the supply of PTH in response to hypocalcaemia and increases the tissue sensitivity to PTH. Magnesium is primarily absorbed from the rumen and cows need a constant supply of it in the diet. As the rumen pH increases above 6.5 – and on higher forage diets, conditions common to dry cows – magnesium solubility declines sharply.

High potassium concentrations may also reduce magnesium absorption in the rumen. Recommended levels of magnesium are 0.4% DM. Increasing dietary magnesium from 0.3% DM to 0.4% DM can reduce milk fever by approximately 62 per cent (Lean et al, 2006).

"Treatment of milk fever should raise the blood calcium levels for long enough until the homeostatic mechanisms are functional"

Calcium

Calcium requirements for a dry cow are around 30g/day. Calcium-restricting dry cow diets – where calcium in the diet is less than 20g/day for three weeks pre-calving and the cow is in true negative calcium balance – work to reduce the risk of milk fever at parturition. The mechanisms to increase calcium production once lactation starts are already activated and the cow can meet increasing calcium demand once she calves. However it is not usually possible to get the calcium supply in the diet this low, with most ranging from 50g-100g calcium per day.

Calcium binders, such as zeolite, can be added to diets to achieve these low dietary calcium levels. These products bind calcium in the intestines and it is passed out in faeces. They often have to be fed in large quantities of 0.5kg-3kg per day for at least two weeks. They can bind phosphorus as well, so extra phosphorus may sometimes be required.

The basics!

Ensure all dry cows have access to fresh food and clean water. Dry cows need 80cm of feed trough space per individual. Making sure that food troughs are cleaned out and fresh food is presented daily will ensure palatability is maximised. Current recommendations are to provide at least 10cm of drinking space per cow and ensuring there are enough water troughs for 10 per cent of the group to drink at once (AHDB Dairy, 2018).

Treatment of milk fever

Treatment of milk fever should raise the blood calcium levels for long enough until the homeostatic mechanisms are functional (**Figure 7**).

Intravenous calcium borogluconate

Each 400ml of 40% $^{w}/_{w}$ solution provides 12g calcium – cows need approximately 2g calcium per 100kg bodyweight to correct hypocalcaemia. Intravenous (IV) calcium should be administered slowly, at 1g/minute to avoid cardiac complications and arrhythmias. Increases in blood calcium following IV administration last approximately four hours.

Figure 7. *A recumbent cow following treatment for milk fever.*

Subcutaneous calcium salts

Absorption of calcium from preparations administered by this route is variable as blood flow to the periphery is compromised.

Oral calcium

Large amounts of soluble calcium can be given orally. This forces calcium across the intestinal tract by passive diffusion between intestinal epithelial cells. Oral solutions contain approximately 50g of calcium; and many of these products are available for administration at calving and again 24 hours later. ■

PPD Questions

1. What is the normal blood calcium range for a dairy cow?
2. How much calcium is required a) per litre of milk produced and b) per litre of colostrum produced?
3. Which hormones are released when blood calcium levels are too low?
4. What is the recommended level of potassium in a dry cow diet to prevent milk fever?
5. When urine pHs are used to monitor acidification of the diet, what is the target range?

Answers
1. 2.1-2.8mmol/L
2. 1.2g and 2.3g respectively
3. PTH and 1,25 (OH_2) D_3
4. <1% DM
5. 6.2-6.8.

References

DeGaris PJ and Lean IJ (2009). Milk fever in dairy cows: A review of pathophysiology and control principles The Veterinary Journal, 176: 58-69.

Goff JP (2008). The monitoring, prevention, and treatment of milk fever and subclinical hypocalcaemia in dairy cows. The Veterinary Journal 176: 50-57.

Horst RL et al (1997). Strategies for Preventing Milk Fever in Dairy Cattle. J Dairy Sci, 80: 1269-1280.

Husband J (2005). Strategies for the control of milk fever. In Practice, 27: 88-92.

Lean IJ et al (2006). Hypocalcaemia in Dairy Cows: Meta-analysis and Dietary Cation Anion Difference Theory Revisited. Journal of Dairy Science, 89(2): 669-684.

Merck Veterinary Manual. https://www.msdvetmanual.com

Reinhardt TA et al (2011). Prevalence of subclinical hypocalcaemia in dairy herds. The Veterinary Journal, 188: 122-124.