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Calcium and the Dairy Cows

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Introduction

Calcium (Ca) is one of the main components of bone and is an important but often over looked factor in achieving proper growth in young cattle (Kaneko *et al*., 2008). Calcium metabolism at calving is one of the most important animal health factor influencing production, reproduction and prevention of metabolic diseases (Goff, 2000). Clinical hypocalcemia (milk fever) occurs within 24 to 48 hour of parturition and is due to the inability of the cow to meet the sudden physiological demand for Ca at calving (Chan *et al*., 2006). Dairy cows excrete a large amount of Ca during lactation due to the high Ca content in milk (Horst *et al*.,1997) and this Ca flow suddenly and markedly increases later during lactation (Horst *et al*., 2005). During the first few months of lactation, the dietary Ca intake is generally lower than the amount of Ca excreted in milk, feces and urine (Taylor *et al*., 2009).

Pathophysiology of Calcium

The sudden increase of Ca losses through the mammary gland at the onset of lactation presents a considerable challenge for the circuits regulating Ca homeostasis (Fekata 2021). Periparturient dairy cattle have a sudden increase in demand for calcium. The requirement for fetal growth in late gestation is approximately 10 g of calcium per day but at calving, this increases to 30–50 g/day for colostrum production (Mahen *et al*., 2018). For the formation of milk, parturition is accompanied by a rapid increase in calcium sequestration and the calcium requirements increase by 2 to 5 times during the lactation (Lean *et al*., 2006).

Physiological adaptations related to the dry period and the onset of lactation can influence macromineral homeostasis. Hypocalcaemia-defined as, total concentrations of Ca in blood $\leq$ 2.0 mmol/l 48 hours after calving (Reinhard *et al.,* 2011), a significant problem in the dairy industry especially the clinical cases (Murray *et al*., 2008). Hypocalcaemia has been associated with impaired reproductive performance including delayed estrus cycles, impaired response to oestrus synchronisation protocols (McNally *et al*., 2014) and impairs gastro intestinal function as well. Serum calcium levels below 5 mg/dL decrease abomasal motility and rumen function (Fekata, 2021). Likewise, intracellular calcium signaling is a key feature in immune cell activation, Kimura *et al*., (2006) hypothesized that, the increased demand for calcium in periparturient cows may adversely affect intracellular calcium stores of immune cells resulting in immunological suppression. Hypocalcaemic cows have reduced feed intake and rumination, increased plasma concentrations of cortisol (Hansen *et al*., 2003) decreased numbers of neutrophils with phagocytic activity (Ducusin *et al*., 2003) and reduced innate immune response (Martinez *et al*., 2012), which all together result in a higher susceptibility to peripartum disease (Chapinal *et al*., 2012).

Oral Calcium Supplementation Around parturition

The type of formulation, palatability and the required treatment frequency, oral Ca supplement is more or less labor intensive and invasive. Martinez *et al*., (2014) observed a reduced prevalence of subclinical hypocalcaemia in cows that received an oral Ca-bolus. The oral administration of easily absorbed Ca salts such as calcium chloride or calcium propionate (Jahani-Moghadam *et al*.,2018) provide 40 to 50 g calcium per dose as a bolus, gel, paste,or liquid, given in a single dose or repeated doses beginning 12 to 24 hours before calving and continuing to 24 hours after calving, effectively increase the plasma Ca concentrations for atleast 6 hours (Radostits *et al*., 2000). A positive response to oral Ca dosing postpartum in lame and high producing groups of multiparous cows observed by Oetzel and Miller (2012). Oral supplementation with calcium chloride and calcium sulfate in a fat-coated bolus have significant effects on improving calcium status in during calving (Sampson *et al*., 2009) but it is also caustic and may result in epithelial lesions of the mucosa and oropharyngeal region, esophagus, for estomaches or abomasum.

In contrast, Calcium propionate has the advantages that it is less irritating and safer to administer while providing a gluconeogenic substrate (propionate) but it requires a larger volume to provide a similar amount of Ca and has a more delayed effect on the plasma Ca concentration (Kara, 2013). Prophylactic treatment with oral Ca formulations in contrast to parenteral Ca administration bears the advantage that it does not disturb the Ca homeostasis, however supports it by providing oral Ca and upregulation of intestinal Ca absorption (Martin and Martens, 2014).

Concept of Dietary Cation–Anion Difference (DCAD)

Other strategies to reduce hypocalcaemia includes, feeding diets with a negative Dietary Cation–Anion Difference (DCAD) before calving (Jahani-Moghadam *et al*., 2018). Feeding ration with low Dietary Cation-Anion Difference (DCAD) to dairy cows or at least 2 weeks before calving decreases the incidence of periparturient hypocalcemia (Constable *et al*., 2009). Adjustment of such ions in the ration in such a way that DCAD favors the amounts of anions (Cl and S) can activate calcium metabolism pathway. DCAD is defined as, the strong ion difference in milliequivalents. The following equation can be used to calculate the DCAD from the percent element in the diet dry matter: mEq/100 g DM = [(%Na ÷ 0.023) + (%K ÷ 0.039)] - [(%Cl ÷ 0.0355) + (%S ÷ 0.016)](Bani *et al*.,2018).

A negative DCAD has been shown to have an effect on calcium metabolism (Goff, 2004). It can induce a more acidic intestinal pH for calcium absorption and facilitate recruitment of bone reserves, increase the excretion of calcium in urine (Cunningham 2002) and consequently activate calcium reabsorption and recruitment processes, increase production and/or receptor receptiveness to vitamin D (1,25(OH)2D3) and parathyroid hormone (PTH) (Espino *et al*., 2003).Adjusting DCAD to the recommended negative values (-100 to -150 mEq/kg) makes the feed unpalatable, leading to decreased feed intake with serious consequences related to negative energy balance. Bani *et al*., (2018) suggested that DCAD (0 +30 mEq/kg) effectively decreased parturient paresis incidence and increase ration palatability by lowering acidogenic salts in the ration. Highly positive DCAD values (via K supplementation) are important during the production period for rumen health, increased productivity and milk fat, particularly in closed intensive systems (Ma G *et al*., 2017).

Conclusion

Lowering the dietary Ca content to

between 0.8 and 0.6 g/kg dry matter increases digestive absorption of Ca of the cows at 3 weeks of lactation but marginally affected the body retention of Ca. This suggests that bone mobilization in cows at the beginning of lactation can be unaffected by the supply of Ca, as long as the source of Ca is available for absorption and exogenous Ca is not lower than 70 percent of total requirement. Oral Ca bolus administration increases serum concentrations of Ca on day 2 postpartum and increases the conception rates at first service. Special consideration should be given to DCAD measurement and adjustment at all stages of the reproductive cycle, not only during the pre and postpartum period. Based on current evidence, the diet containing DCAD value range from 10-15 mEq/100 g DM (-100 to 150 mEq/kg DM) have the lowest incidence of milk fever and such diet should be fed for 2 to 3 weeks before calving. Monitoring the urine pH can be a useful aid and a reasonable indicator of metabolic pH status which reflects the effectiveness of anionic products. Urine pH should be 6.0 to 6.5 for Holsteins and 5.5 to 6.0 for Jerseys.

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