

THE ROLE OF MAGNESIUM IN MILK FEVER (HYPOCALCAEMIA)

About Milk Fever

Milk fever around the time of calving is the most common metabolic disease of dairy cattle. Most cases occur within 48 hours of calving, but cases can also be seen in the week before calving and up to 10 days after calving.

Mineral homeostasis in cattle is very complex and is influenced by many factors.

Homeostasis of serum calcium (Ca) involves the interaction of three hormones, parathyroid hormone (PTH), 1,25-dihydroxyvitamin D (1,25-DHD) and calcitonin. In simple terms, PTH and 1,25DHD increase serum Ca, and calcitonin lowers it.

The parathyroid gland monitors the level of Ca in carotid arterial blood. Parathyroid hormone is released in response to low Ca level.

PTH has three actions to restore the level of Ca in the blood:

1. Stimulates receptors in bone to increase turnover of bone, thereby releasing Ca into serum
2. Stimulates receptors in kidney so that more Ca and Mg is reabsorbed in the tubules
3. Up-regulates the enzyme responsible for activation of vitamin D, which increases absorption of Ca ions from the intestine

The net effect of PTH release is increased calcium levels via Ca resorption (bone), reabsorption (kidney) and absorption (intestine).

Milk fever results when increased demand for calcium overcomes an animal's ability to maintain Ca homeostasis.

At calving, calcium requirements increase dramatically as milk production begins. This drain on serum calcium by the udder must be met by mobilisation of calcium stores in the body to prevent hypocalcaemia. This requires the cow's hormonal regulating system (via PTH) to 'switch on' rapidly during the peri-calving period.

Role of Magnesium

Magnesium (Mg) is also an essential element in a cow's diet, and has many roles within the body. Magnesium is an essential part of the structure of DNA, a catalyst for many enzymes, critical for the activity of ATP, for correct functioning of nerves, bone development, health of rumen microbes and for parathyroid hormone (PTH) secretion and activity.

Low serum Mg inhibits PTH secretion, as well as causing resistance of PTH receptors to PTH. This means that a cow's ability to maintain Ca homeostasis is reduced. The net impact is an inability to respond effectively to low serum Ca, potentially leading to hypocalcaemia. It is therefore essential to ensure adequate Mg dietary intake so that the action of PTH is optimised.

There is no mechanism for mobilising Mg within the body, so daily requirements must be met by dietary intake. The site of Mg absorption in cattle is the rumen. Magnesium is absorbed as Mg⁺⁺, so dietary Mg, and Mg supplements must be broken into soluble components for Mg absorption to occur.

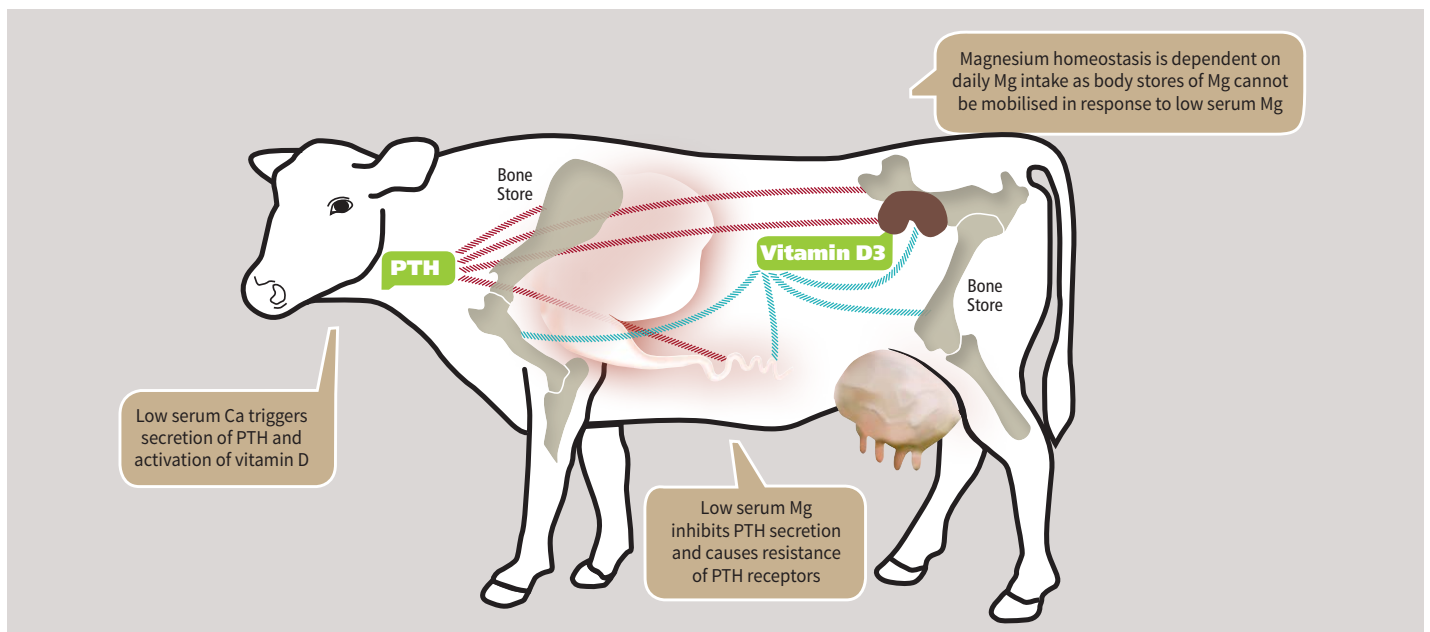


Fig 1. PTH and vitamin D3 as regulators of Ca metabolism

Magnesium intake is not the same as Mg absorption. The availability of Mg (amount of Mg able to be absorbed in the rumen) in pasture and many supplements is very low and can vary.

Cow requirements for Mg are well known, however, meeting these levels requires calculating content and availability of both dietary and supplementary forms of Mg. Interactions with

other minerals in the diet (eg. K, N, Na) can also influence Mg absorption.

Maintaining an adequate level of Mg in the cow is essential for the prevention of both grass tetany (hypomagnesaemia) and milk fever (hypocalcaemia) in periparturient cows. It is important that supplementary Mg is provided to cows on a daily basis from a month prior to calving until after peak lactation.

THIS TABLE ILLUSTRATES LIKELY MAGNESIUM REQUIREMENTS FOR A SPRING CALVING NEW ZEALAND DAIRY COW VERSUS DIETARY INTAKE FROM SEASONAL NZ PASTURE

Stage of Lactation	Mg in NZ pasture						Cow [#] Mg Requirements*				Deficit / Surplus (g Mg)
	Milk production (L)	Pasture level Mg (g/kg DM)	Pasture intake (kg DM)	Total Mg in consumed pasture (g)	Mg availability (%)	Mg absorbed (g)	Maintenance (g Mg)	Pregnancy (g Mg)	Milk (g Mg)	Total (g Mg)	
Dry	0	1	10	10	10	1	2	0.3	0	2.3	- 1.3
Calving	10	1	12	12	10	1.2	2	0	1.5	3.5	- 2.3
Early - Peak Lactation	30	1	18	18	15	2.7	2	0	4.5	6.5	- 3.8
Mid lactation	23	2	16	32	20	6.4	2	0.1	3.5	5.6	+ 0.8
Late Lactation	10	4	14	56	30	16.8	2	0.2	1.5	3.7	+ 13.1

#Cow in this example is a 450kg cow, producing 30L (2.4kg MS) in peak lactation (Holmes, et al)

*Mg requirements for liveweight (LW) gain are not included in this example. Liveweight gain typically requires 0.45g Mg / kg LW gain.

DAILY SUPPLEMENTATION WITH MAGNESIUM SALTS IS REQUIRED IN COWS FROM ONE MONTH BEFORE CALVING TO MID LACTATION. THIS TABLE PROVIDES AN EXAMPLE OF MAGNESIUM PROVIDED BY VARIOUS COMMONLY USED SUPPLEMENTS

Mg form	Mg content (%)	Recommended dose (g)	Mg dose (g)	Mg availability (%)	Daily Mg absorption (g)
Mg oxide	55	22 g (drenched) 44 g (dusted)	12.1	33	4
Mg chloride	10	122 g	12.2	Not published (est 33%*)	4*
Mg sulphate	12	100 g	12	Not published (est 33%*)	4*
Mg pidolate	8.5	60 g (= 100mL)	5.1	Not published (est 50%*)	2.6*
Rumetrace	90	One capsule for 9-12 weeks	189 (slow release technology)	100%	2

* Availability of Mg in this salt is unpublished so estimated % has been used to calculate daily Mg absorption on this basis.

THE APPROPRIATE MG SUPPLEMENT(S) FOR LACTATING DAIRY COWS

Stage of Lactation	Deficit / Surplus (g Mg)	Recommended Supplement
Dry	- 1.3	Any (provided daily)
Calving	- 2.3	Any (provided daily)
Early - Peak Lactation	- 3.8	Mg oxide, chloride, sulphate (or Rumetrace with additional Mg supplement)
Mid lactation	+ 0.8	Generally not required
Late Lactation	+ 13.1	Not required

References

Dairy NZ FarmFact 3-1. Magnesium Supplementation. October 2009.
 Holmes CW, Brookes IM, Garrick DJ, MacKenzie DDS, Parkinson TJ and Wilson GF. Milk Production from Pasture. Massey University. 2002.
 Martens H and Schweigel M. Magnesium homeostasis in ruminants and grass tetany. In: Advances in Magnesium Research: Nutrition and Health. John Libbey & Co Ltd. 2001.
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