Ruminal Mg transport:A Tool for Prediction of Mg Requirement (Intake)

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Abstract

Magnesium is an essential mineral with numerous physiological functions and, hence, Mg deficiency and hypomagnesaemia cause a variety of clinical symptoms including neurological disorders such as tetany in ruminants. Since Mg homeostasis in the extracellular compartment (ECC) and blood is not regulated by hormones, the steady-state concentration of Mg in the blood depends on Mg *efflux* from the (ECC) into milk or tissues including bones and on absorption of Mg (*influx*) from the gastrointestinal tract (GIT). Mg absorption (influx) from the rumen is a) active, b) essential for Mg homeostasis and c) includes two transport mechanisms: a potential difference (PD) dependent or K-sensitive and potential difference independent or K-insensitive mechanism. The kinetic data (K_m and V_{max}) of the two parallel working uptake mechanisms are not known but there are convincing evidence for a "job sharing" between these two mechanisms: Mg uptake with high affinity/low capacity via the PD-dependent mechanism at low Mg concentrations, and Mg uptake with low affinity/high capacity for high Mg concentrations (PD-independent) (for details see review Martens and Schweigel 2001).

If the proposed model of "job sharing" of the two Mg uptake mechanisms is correct, two consequences regarding possible effects of K can be predicted. (i) An increase of K should reduce Mg absorption to a large extent at low Mg concentrations, because Mg absorption at low ruminal Mg concentration primarily depends on PD-dependent or K-sensitive uptake. (ii) An increase of Mg intake could compensate for the possible negative effects of high K intake, because the PD-independent or K-insensitive uptake is mainly active at high ruminal Mg concentrations. These assumptions are indeed confirmed by experimental observations. Ram et al. (1998) fed increasing amounts of Mg at two levels of K-intake. It was interesting to learn that the absolute amount of reduced Mg absorption was almost identical at all Mg intakes. However, the relative change decreased with increasing Mg intake (table 1).

Table 1: Effect of K intake (1 or 3.6 %) on Mg absorption in sheep at increasing Mg intake (Ram et al. 1998).
Number in parenthesis: Apparent digestibility of Mg

Mg-Intake g/d	Mg-Absorption (g/d) K 1 %	K 3.6 %	Decrease Mg-Absorption (g/d)	Change Decrease %
1.64	0.58 (35)	0.27 (16)	0.31	54
3.14	1.17 (37)	0.81 (26)	0.36	31
4.66	1.56 (33)	1.14 (24)	0.42	27

This observation was confirmed by a meta-analysis of Weiss (2004). The magnitude of Mg-digestibility depends on Mg intake and K content, which takes into account the two Mg transport mechanisms as explained above, and is described by the equation: Digestible Mg = 4.5 (± 4.0) + 0.24 (± 0.07) x Mg intake (g/d) – 4.4 (± 2.2) x K (% dry matter). Hence Mg requirement (digestible Mg) can be calculated according to this equation.

Introduction

Magnesium is an essential mineral with numerous physiological function hence, Mg deficiency and hypomagnesaemia cause a variety of symptoms including neurological disorders such as tetany in ruminants. Mg homeostasis in the extracellular compartment (ECC) and blood regulated by hormones, the steady-state concentration of Mg in the extrac space (ECS) and blood depends on Mg absorption from the gastrointestina (*influx*). Mg in the ECS is used for covering Mg requirement of the tissues growth, for secretion into milk and endogenous secretion into the gut (e Physiological blood Mg concentrations are maintained as long as (absorption) > *efflux* (covering of requirement). The difference between *Mg* – *Mg efflux* is exactly excreted via urine and therefore Mg excretion in urir be considered as a regulator of Mg homeostasis. This simple type of regu "*Mg influx – Mg efflux = Mg (surplus) in urine*" for Mg homeostasis in EC blood clearly indicates that if "*Mg influx* < *Mg efflux* Mg in urine appro almost zero. Furthermore hypomagnesaemia is induced as a po consequence, because Mg cannot be mobilized from tissues or bone. H physiological Mg concentrations in blood and ECS can only be maintained *Mg influx* exceeds *Mg efflux*. *Mg influx* can be insufficient at low Mg intak deficiency) or at disturbed Mg absorption which is very often the ca ruminants (Fontenot et al., 1973; Martens and Schweigel, 2000). It intention of this short review to analyse the reasons of disturbed Mg abso and it consequences for Mg requirement (intake).

mg absorption (influx) and its disturbance

Mg absorption from the gastrointestinal tract (GIT) and its possible disturb are of paramount importance for Mg homeostasis and consequently requi a better understanding knowledge about a) the site of Mg absorption in th b) mechanism of Mg absorption, c) factors which may impair Mg absorpti d) the underlying mechanisms of impaired Mg absorption.

Site of Mg absorption

Studies about the flow rate of minerals in the GIT of ruminants show surprising observation that Mg disappeared before the intestine an intestinal absorption of Mg was even larger than total absorption of Mg fr whole GIT (see review; Martens, 1978). This finding was further substantia the observation that Mg absorption before the duodenum was essen maintaining physiological blood Mg concentrations (Pfeffer and Rahman, and that disturbed Mg absorption before the intestine was not compensa absorption from the small and large intestine (Tomas and Potter, 1976).

The disappearance of Mg before the duodenum opened the discussion the site of Mg absorption within the forestomachs and it turned out that ru the main site of Mg absorption in the GIT of ruminants and essential homeostasis (Martens, 1978).

Mechanism of Mg absorption

An early assessment of the driving forces for Mg absorption across the epithelium realized that there is a chemical gradient from the rumen to blo 6 mmol/l Mg or even more in the rumen and 0.8 – 1.0 mmol/l Mg in blood) would favour passive absorption by diffusion. However, the chemical gra opposed by an electrical gradient (blood side positive 20 – 70 mV), whic driving force is in most cases larger than the chemical gradient. Hence,

passive driving force for Mg would favour a secretion into the rumen and not absorption. For this reason Mg absorption must be an active transport, which has been demonstrated *in vivo* (Care et al., 1984; Martens and Blume, 1986) and *in vitro* (Martens et al., 1987; Leonhard- Marek and Martens, 1996). Further characterization of this active transport demonstrated two transport mechanisms for Mg uptake across the luminal membrane or the rumen epithelium: a potential difference (PD) dependent or Ksensitive and potential difference independent or K-insensitive mechanism. The kinetic data (Km and Vmax) of the two parallel working uptake mechanisms are not known but there are convincing evidence for a "job sharing" between these two mechanisms: Mg uptake with high affinity/low capacity via the PD-dependent mechanism at low Mg concentrations, and Mg uptake with low affinity/high capacity for high Mg concentrations (PD-independent) (for details see review Martens and Schweigel 2000; Martens in press).

Impaired Mg Absorption from the rumen

It is well established since decades that Mg absorption from the GIT can be impaired (Fontenot et al., 1973) and it was a breakthrough when Tomas and Potter (1976) demonstrated that the site of reduced Mg absorption caused by high K intake is the forestomach region and that the disturbed Mg absorption within the forestomachs is not compensated by the small and large intestine. Similarly Na deficiency caused a reduction of Mg from the forestomachs (Martens et al., 1987b). Both feeding conditions high K intake and Na deficiency (replacement of Na by K in saliva) are changing the same parameters within the rumen (table 1).

Table 1: Effects of high K intake and Na deficiency on ruminal K and Na concentration, the transmural potential difference, PDt, and Mg absorption from the rumen (see Tomas and Potter, 1976; Martens et al., 1987b)

Parameter	High K Intake	Na Deficiency
Ruminal K Concentr	↑	↑
Ruminal Na Concentr.	↓	↓
PDt	↑	↑
Mg Absorption	↓	↓

Underlying mechanisms of impaired Mg absorption

Further studies *in vitro* (Martens et al., 1987a) revealed that electrophysiological changes of the epithelium, PDt, are causing decreased Mg absorption and not the increase of K or the decrease of Na concentration. These observations with the proposed model of "job sharing" of the two Mg uptake mechanisms led to the following conclusions (details see Martens and Schweigel, 2001): (i) An increase of K should reduce Mg absorption to a large extent at low Mg concentrations because Mg absorption at low ruminal Mg concentration primarily depends on PD-dependent or K- sensitive uptake. (ii) The PD-independent or K-insensitive uptake is mainly active at high ruminal Mg concentrations and hence possible effects of K at high Mg intake should be of minor importance.

prediction of mg requirement (intake)

The assumptions about the physiological consequences of the two Mg uptake mechanism are indeed confirmed by experimental observations. Ram et al (1998) fed increasing amounts of Mg at two levels of K-intake. It was interesting to learn that the absolute amount of reduced Mg absorption was almost identical at all Mg intakes. However, the relative change decreased with increasing Mg intake (table 2).

Table 2: Effect of K intake (1 or 3.6 %) on Mg absorption in sheep at increasi Mg intake (Ram et al. 1998). Number in parenthesis: Apparent digestibility of Mg.

Mg-Intake g/d	Mg-Absorption (g/d) K 1 %	K 3.6 %	Decrease Mg-Absorption (g/d)	Change Decrease %
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Digestible Mg = 4.5 (± 4.0) + 0.24 (± 0.07) x Mg intake (g/d) – 4.4 (± 2.2) x K (dry matter). Hence Mg requirement (digestible Mg) can be calculated accordi to this equation.

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References

Care, A.D., R.C. Brown, A.R. Farrar & D.W. Pickard (1984). Magnesium absorption from digestive tract of sheep. Q.J. Exp. Physiol. 69, 577-587.

Leonhard-Marek, S. and H. Martens (1996). Effects of potassium on magnesium transport ac rumen epithelium. Am. J. Physiol. 271, G1034-1036.

Fontenot, J.P., M.B. Wise & K.E. Webb (1973). Interrelationships of potassium, nitrogen, magnesium in ruminants. Fed. Proc. 32, 1925-1928.

Martens, H. (in press) Diseases of Dairy Animals: Non-infectious Diseases: Grass Tetany Encyclopedia of Dairy Science. Elsevier – Kidlington/UK.

Martens, H. (1978). Lokalisation und Mechanismus der Magnesiumresorption bei Wiederkä Uebers. Tierernaehrg. 6, 75-89.

Martens, H. and Blume, I. (1986). Effect of intraruminal sodium and potassium concentrations a transmural potential difference on magnesium absorption from the temporarily isolated rum sheep. Q. J. Exp. Physiol. 71, 409-415.

Martens, H., G. Gäbel & H. Strozyk (1987a). The effect of potassium and the transmural pote difference on magnesium transport across an isolated preparation of sheep rumen epithe Quart J Exp Physiol 72, 181-188.

Martens, H., Kubel, O.W., Gäbel, G., and Honig, H. (1987b). Effects of low sodium intak magnesium metabolism in sheep. J. agric. Sci. 108, 237-243.

Martens, H. and Schweigel, M. (2000). Grass Tetany and other Hypomagnesaemias. In: Herc (eds) Veterinary Clinics of North America: Food Animal Practice: Metabolic Disorde Ruminants. 16, 339-368. W. B. Saunders – Philadelphia.

Pfeffer, E., Rahman K.A. (1974) Untersuchungen zur Lokalisation der Magnesiumabsorption Wiederkäuer. Zeitschrift fuer Tierphysiologie, Tierernaehrung und Futtermittelkunde 33, (Abstract).

Ram, L., Schoneville, J. Th., Van't Klooster, A. Th., Martens, H. and Beynen, A. C. (1998). J. Sci. 81, 2485-2492.

Tomas, F. M., Potter, B.J. (1976). The effect and site of action of potassium upon Mg absorpti sheep. Austr. J. Agricult. 27, 873-880.

Weiss, W. P. (2004). Macromineral digestion by lactating dairy cows: Factors affecting digestibil magnesium. J. Dairy Sci. 24, 2167-2171.