Complex disorders arise when the calcium and magnesium requirements of ruminants are not met during pregnancy and lactation. **Hypocalcaemia** and **hypomagnesemia** occur when the losses of Ca and Mg in milk cannot be supplied from the diet or mobilized from body reserves. Older cows and ewes, which cannot mobilize enough minerals from bone or body fluids, are particularly susceptible when there is reduced absorption of Ca and Mg from the gut. This may arise simply from inadequate intakes, or as a result of complex interactions that decrease Ca and Mg absorption from the rumen. Increased concentrations of ammonium ions and, particularly, potassium in the rumen act independently to reduce net Mg absorption. Intrarumen K concentrations may be increased either through dietary intake, or from reductions in salivary sodium secretion. Factors affecting salivary secretion, which is the main source of Na and P for the rumen, may indirectly affect the absorption of Mg and also of Ca. The absorption of Ca from the rumen is unaffected by intrarumen K concentration, but increases as intrarumen P concentrations increase. Ewes, which normally mobilize skeletal Ca reserves to meet the fetal demands during late pregnancy, are particularly susceptible to hypocalcaemia when intake or absorption of Ca decreases. The young lamb will maintain its plasma Ca concentration at the expense of bone structure when dietary Ca intake is inadequate during growth. Young lambs are unable to absorb sufficient Ca from pasture. Milk intakes that result in growth rates greater than 150 g/day during the first six weeks are necessary to prevent osteoporosis by the time lambs are weaned at 12 weeks. Dietary management offers the most practical way of preventing most nutritional disorders affecting Ca and Mg metabolism in ruminants.

**INTRODUCTION**

Hypocalcaemia and **hypomagnesemia** occur frequently in ruminants kept under pastoral conditions in Australia. To prevent these disorders of Ca and Mg metabolism, the nutritional management of cattle and sheep requires continual planning. In this paper some findings from recent research on the nutritional physiology of Ca and Mg in ruminants are reviewed with the object of proposing better ways to recognize and prevent hypocalcaemia and **hypomagnesemia** in cattle, and hypocalcaemia and osteoporosis in sheep.

**Regulation of plasma Ca and Mg concentrations**

Plasma Ca is maintained within narrow limits (2.0 to 2.5 mmol/l) in adult ruminants by the actions of parathyroid hormone, metabolites of vitamin D, particularly 1,25 dihydroxyvitamin D, and calcitonin on the alimentary tract, bone and kidney. Ruminants differ from monogastric animals in that absorption

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of Ca in excess of requirements rarely occurs (Braithwaite 1978).

Maintenance of plasma Mg concentrations is dependent on continual absorption from the gut. When excess Mg is absorbed, renal excretion of Mg maintains plasma concentrations less than 1.2 mmol/l. No hormonal systems have been shown to directly regulate plasma Mg concentration in ruminants.

ROLE OF THE RUMEN IN CALCIUM AND MAGNESIUM HOMEOSTASIS

Studies on sheep and cattle prepared with fistulae in the abomasum or proximal duodenum have shown that the reticulorumen is the main site for net absorption of Mg (Grace 1983; Greene et al. 1983).

Until recently, the role of the reticulorumen in Ca absorption has been unclear. Some studies had indicated there was net secretion into this region (between 0.15 and 1.44 g/day in sheep; Pfeffer et al. 1970; Ben-Ghadalia et al. 1975; Wylie et al. 1985), whereas others indicated no net Ca movement across the rumen wall (Sklan and Hurwitz 1985). In contrast, net absorption of between 0.5 and 4.3 g Ca/day from the forestomachs in sheep was observed by Grace et al. (1974), Leibholz (1974), Dillon and Scott (1979) and Grings and Males (1987). There were also conflicting findings from experiments on Ca absorption from the reticulorumen of cattle (Rogers and Van't Klooster 1969; Kemp et al. 1973; Bertoni et al. 1976). One conclusion which could be made from these observations was that Ca absorption from the rumen was variable. Furthermore, this variable absorption of Ca from the rumen might be influenced by dietary management and be important in predisposing sheep and cattle to hypocalcaemia.

Recent studies have provided explanations for the variations in Ca and Mg absorption from the rumen. Measurements of the unidirectional Ca and Mg fluxes across isolated sheets of rumen mucosa in Ussing chambers in vitro have indicated that absorption of these minerals is by active processes (Holler et al. 1988). The use of a temporarily-isolated and washed rumen technique has enabled direct in vivo studies of the mineral absorption from inorganic buffers and supernatants of rumen fluid from the reticulo-rumen of conscious sheep (Martens and Rayssiguier 1980; Grace et al. 1988; Holler et al. 1988).

Factors affecting Ca absorption from the rumen. Net Ca absorption occurs when Ca concentration in rumen fluid increases above 0.7 mmol/l, and when there is an increase in P concentration from 0.6 to 8 mmol/l (Holler et al. 1988). The absorption of Ca from the rumen is unaffected by intrarumen K concentration (Beardsworth et al. 1987).

Concentrations of Ca in rumen fluid in sheep usually range between 1 and 4 mmol/l (Holler et al. 1988). In cattle we have found Ca and P concentrations in 100,000 g supernatants of rumen fluid to range between 0.5 and 1.5 mmol/l, and 2.5 and 16 mmol/l, respectively (M.B. Allworth and I.W. Caple unpubl.). Inorganic phosphorus concentrations in rumen fluid are influenced by dietary P intakes, but salivary secretion provides the main source of P for the rumen (Tomas et al. 1967). Changes in salivary P secretion in response to the nature of the diet and rumen fermentation may have more important affects on rumen P concentrations than P intake and, indirectly, on the rate of Ca absorption from the rumen. Roughage intake, which influences salivary secretion, may be particularly important in enabling ruminants to absorb sufficient Ca from the rumen to maintain Ca homeostasis when they are fed grain diets low in Ca (Langlands et al. 1967).
Factors affecting Mg absorption from the rumen. The absorption of Mg from the rumen is dependent on the concentration of Mg in rumen fluid (Martens and Rayssiguer 1980). Active Mg absorption is decreased as a result of an increased electrical potential difference across the rumen wall, and the direct effects of high K concentrations on the Mg pump (Grace et al. 1988). High ammonium ion concentrations (30 mmol/l) decrease Mg absorption (Martens and Rayssiguer 1980), and this effect is additive and independent of the effects of K (Care et al. 1984).

If K concentrations in herbage increase (from 10 to 40 g/kg DM), and/or salivary secretion decreases, this may lead to an increase in K (from 5 to 40 mmol/l) in the rumen, and a decrease in Na (from 135 to 80 mmol/l) concentrations. Simply changing the diet, such as from hay to lush pasture, which may not directly affect mineral intake, can lead to similar effects as changing mineral intake if the dietary change results in a decrease or increase in salivary secretion. For example, a decrease in salivary secretion rate may lead to decreased Na and P concentrations, and increased K concentrations in the rumen, and result in decreased absorption of Mg and Ca from the rumen.

Roughage intake is particularly important in enabling ruminants maintain Ca, P, Na and Mg homeostasis. More Na and P enters the rumen from saliva than in the diet, and recycling of these minerals between the gut and blood via saliva plays an important role in the economy of these minerals and the absorption of Ca and Mg from the rumen.

DISORDERS OF CALCIUM METABOLISM IN RUMINANTS

Hypocalcaemia in cattle. Milk fever or "parturient paresis" occurs mainly in the periparturient period in dairy cows, but a comparable condition is also seen in beef cows. The hypocalcaemia results from a rapid withdrawal of Ca from the blood by the udder at the start of lactation. Most dairy cows are expected to be in negative Ca balance in early lactation irrespective of the Ca content of the diet, and must draw on bone Ca reserves (Ramberg et al. 1975; ARC 1980). Hypocalcaemia can occur in cows in which all the known mechanisms in Ca homeostasis, such as parathyroid hormone and calcitonin secretion, and production of 1,25 dihydroxyvitamin D3 by the kidney are functioning normally. It seems that one of the most important factors in Ca homeostasis of the cow is the dietary intake of Ca. Maintenance of plasma Ca in a lactating cow depends mainly on continuous intestinal absorption of Ca (Hove and Hilde 1984).

Hypocalcaemia can also occur in dairy cows at any stage during lactation (Caple and Halpin 1985). Hove (1986) observed that young cows were able to regulate plasma Ca in a stable manner after the parturient hypocalcaemia, whereas 50% of older cows showed one or more hypocalcaemic episodes of 24 to 36 h duration at approximately 9-day intervals during the first month of lactation. If these periods of hypocalcaemia corresponded with a reduced feed intake, clinical hypocalcaemia may occur since maintenance of hypocalcaemia depends mainly on continuous intestinal absorption.

In south-eastern Australia, where milk production is mainly from cows at pasture, parturient paresis occurs more frequently in cows given unlimited access to lush pastures in the periparturient period than in cows fed mainly on hay (Harris 1981). Dairy cows grazing improved fertilized pastures during early winter and spring appear to have a limited ability to respond to abrupt changes in Ca metabolism. Pregnant cows sent to saleyards or meatworks
commonly develop hypocalcaemia within 12 hours after removal from such pastures (Warnock et al. 1976). These hypocalcaemic disorders in grazing dairy cows may arise from a number of different causes such as: (a) an inadequate Ca intake to maintain plasma and extracellular Ca concentrations when mobilisation of Ca from bone reserves was maximal (Hove and Hilde 1984; Van de Braak et al. 1987), (b) an adequate Ca intake according to nutritional requirements, but inadequate absorption of Ca from winter and spring pastures (Hutton et al. 1967), and (c) concurrent hypomagnesemia impairing Ca mobilisation (Sansom et al. 1983).

Traditionally, attempts have been made to propose universal preventive methods for parturient paresis. These include (a) reducing the alkaline alkalinity of the diet by feeding calcium chloride and magnesium sulphate (Dishington 1975), (b) reducing the Ca intake to less than 50 g/day before parturition, then increasing the intake to 100 g/day after parturition (Pickard 1975) or (c) administration of vitamin D$_3$, or 1,25(OH)$_2$D$_3$ or its analogues to cows before parturition in an attempt to increase Ca absorption (Sachs et al. 1987).

Preventive measures for parturient paresis in a particular dairy production system must be designed to overcome the factors predisposing hypocalcaemia. The current management practices in south-eastern Australia are aimed at providing lactating cows green rapidly growing pastures for as long as possible to maximise milk production. Such practices may not enable lactating cows to restore their overall Ca balance rapidly. Older cows may be particularly vulnerable to hypocalcaemia when any factor further reduces Ca intake or absorption. The usual practice to prevent parturient paresis in this system is to feed the cows on hay and restrict pasture intake until a few days after parturition (Harris 1981). The effectiveness of this control procedure may achieved by having Ca intakes less than 50 g/day, and enabling improved Ca absorption from the rumen on the roughage diet. Cattle can absorb Ca with high efficiency from poor quality tropical hays that supply Ca intakes just above and below maintenance requirements (Blaney et al. 1982).

Urine Ca excretion, which represents the overflow of Ca not required for metabolic demands, shows a seasonal pattern in grazing dairy cows (Caple and Halpin 1985). Urine Ca excretion is lowest during the winter and early spring months, and the highest values are observed during the late spring and summer months. This seasonal pattern appears to be unrelated to either the stage of lactation, age of cow or her milk production, or Ca intake. It may simply reflect the increased absorption of Ca from the rumen as pasture digestibility decreases. For cows fed pastures, a period of grazing dry pastures over the summer or hay feeding may be critical in maintaining overall Ca balance.

Calcium nutrition and disorders of Ca metabolism in sheep

Pregnancy and lactation present major challenges to Ca homeostasis in sheep in flocks kept under pastoral conditions. Ca and P contents in herbage and supplements may not meet requirements for a substantial part of the year (Caple et al. 1988c; Grant et al. 1988; Larsen et al. 1986). Underwood (1981) concluded that Ca deficiency was unlikely in ruminants grazing herbage, but was likely when sheep were fed wheat or other low Ca concentrate diets (1.0 gCa/kg DM) for survival during droughts.

The Ca requirements of the developing fetus put considerable demands upon Ca homeostasis of the ewe. During pregnancy, plasma Ca concentration is maintained by increasing mobilisation of Ca from bone and absorption from the intestine. These effects are probably mediated via an increase in plasma
1,25-dihydroxyvitamin D$_3$ [1,25(OH)$_2$D$_3$] as a result of increased secretion of parathyroid hormone (PTH). Other hormones may also be important. Plasma osteocalcin, a protein which is produced by osteoblasts, decreases in ewes by day 35 of pregnancy indicating there may be a reduction in maternal osteoblastic activity before there is any marked increase in Ca transfer to the fetus (Wark et al. 1987).

Placental transfer of Ca appears to be controlled by the fetus. Recently, evidence for a parathyroid hormone-related protein (PTHrP) in the fetal parathyroid gland and placenta has been obtained (Loveridge et al. 1988; Rodda et al. 1988). PTHrP, but not parathyroid hormone (PTH), is thought to regulate active placental transfer of Ca to the fetal lamb, and maintain its plasma Ca higher than that in the ewe during gestation. Parathyroid hormone concentrations in fetal plasma are normally undetectable in most immunoassays, and this might be expected in view of the high plasma Ca. However, fetal plasma PTHrP concentrations are elevated (Loveridge et al. 1988). The high fetal plasma Ca and PTHrP may maintain osteoblastic activity and promote skeletal mineralization during fetal growth (Caple et al. 1988b). A similar PTHrP is associated with hypercalcaemia produced by certain human cancers (Rodda et al. 1988), but it is not yet known if PTHrP has a role in normal maternal Ca metabolism during pregnancy and lactation in any species.

Irrespective of their Ca intake, ewes do not absorb sufficient dietary Ca to meet their requirements during pregnancy, and for milk secretion in early lactation. Ewes may mobilize up to 20% of their skeletal Ca reserves during this time (Braithwaite 1983), with the proportion mobilised depending on the pre-existing degree of mineralisation, and on the Ca content of the diet (Sykes and Dingwall 1975). Replacement of lost skeletal reserves is usually complete by 130 days after parturition on diets providing plentiful Ca (250 mg Ca/kg liveweight/day) and P (160 mg P/kg liveweight/day). Because the rate of accretion of Ca onto bone decreases with age, the replenishment of the partly-depleted skeleton will take longer in older ewes. If skeletal reserves are not replaced ewes may become susceptible to hypocalcaemia during subsequent pregnancies.

Hypocalcaemia in sheep is a well-known problem associated with drought periods when there is insufficient roughage available, and when grain is fed without the addition of Ca supplements. However, hypocalcaemia in pregnant ewes occurs more commonly when they are grazing green grass pastures following drought periods (Larsen et al. 1986), and may also occur in normal years when they have been mustered for shearing or other management purposes (Grant et al. 1988). Lambs less than 6 months of age, and particularly those with osteoporosis, may develop hypocalcaemia if they are grazed on green oat pastures during winter and spring (Caple et al. 1988a; Caple 1989).

Clinical hypocalcaemia (plasma Ca < 1.7 mmol/l) in pregnant ewes occurs when there is insufficient intake and absorption of Ca, and resorption from skeletal reserves, to meet the demands of the fetus (up to 2.8 gCa/day). We estimate that up to 3% of pregnant ewes die from hypocalcaemia each year in Victoria (Caple et al. 1988c). Although the most dramatic losses occur when pregnant ewes graze green winter and spring pastures following drought periods (Larsen et al. 1986), losses still occur during winter and spring in normal years and when nutritional intake seems adequate. Pregnant and lactating ewes seem to be unable to maintain plasma Ca as high on green winter and spring pastures as they do when grazing poorer quality summer and autumn pastures (Grant et al. 1988). The efficiency of Ca absorption from green pastures with high water contents may be less than that from dry roughage and concentrate.
diets. While clinical hypocalcaemia can occur in undisturbed grazing ewes, it is commonly precipitated by management procedures, such as mustering for shearing, which involve short-term starvation.

Young lambs at weaning, and pregnant and lactating ewes are susceptible to hypocalcaemia when fed grain diets which are low in Ca (Franklin et al. 1948). Drought periods extending from two to 12 months are not uncommon in Australia. Phosphorus concentration in herbage decreases as herbage matures, and a low P intake may limit repletion of skeletal reserves. The concentration of Ca in herbage increases as herbage matures and dries during summer. When the available dry herbage becomes limiting for survival, sheep are usually fed grains such as wheat, oats and lupins. The ability of sheep to maintain Ca homeostasis on these diets depends on the roughage intake (Langlands et al. 1967) and whether Ca supplements (1% CaCO₃) are added to the grain (Franklin et al. 1948). Little attention has been given to how well different classes of sheep maintain or restore their skeletal Ca stores during droughts and periods of grain feeding. Usually only bodyweights of sheep are monitored and the feed intake adjusted accordingly.

Osteoporosis in lambs Recent studies have shown that the relative intakes of milk and pasture in lambs less than 12 weeks influences lamb growth rate and bone structure (Heath and Caple 1988). Osteoporosis in lambs aged from 10 weeks to 15 months is a relatively common condition in sheep flocks in south-eastern Australia. Lambs growing less than 150 g/day in the first 6 weeks after birth may develop osteoporosis if they are unable to obtain sufficient Ca from pasture (Heath and Caple 1988).

The newborn and growing lamb maintains a higher plasma Ca concentration than the ewe for several months after birth. A high plasma Ca is required for stimulation of osteoblasts and bone development in the fetal lamb (Caple et al. 1988b), and a similar requirement may persist after birth. Inadequate Ca intake and absorption, combined with excessive mobilization of skeletal Ca in order to maintain plasma Ca concentration, may result in osteoporosis in lambs. This may be due to decreased Ca intake, reduced efficiency of absorption, and increased endogenous Ca losses on herbage diets. It is the rapidly-growing proximal limb bones such as the femur which are most affected (Hodge et al. 1973).

During the first 3 weeks after birth the rumen is essentially non-functional in lambs, and there is an absolute requirement for milk for survival. The level of milk intake influences the time at which the young lamb commences grazing, and the quantity of pasture ingested before weaning. A lamb growing from 5 kg liveweight at birth to 15 kg liveweight at 10 weeks, and having an intake of about 1 litre milk/day plus herbage (4 g Ca/kg DM) before weaning, would have Ca intakes ranging from 320 to 200 mg/kg liveweight/day. The Ca requirements of lambs for appetite (50 mgCa/kg/day), and growth and maintenance of Ca homeostasis (250 mgCa/kg/day), are much lower than for maximum mineralization of the developing skeleton (450 mgCa/kg/day). The Ca concentrations in herbage diets are unlikely to limit appetite and growth of lambs, but may limit skeletal mineralization (Hodge 1973; Heath and Caple 1988).

Lambs with osteoporosis are particularly susceptible to hypocalcaemia when grazing green oat crops, or when mustered for shearing. The main preventive method for osteoporosis in lambs aged less than 4 months may be to improve milk production of ewes. In south-eastern Australia, lambs born in autumn are most likely to be at risk to osteoporosis because the available
pasture and energy intake of ewes is often limiting for milk production during late autumn and early winter. One simple solution for improving nutrition of ewes during lactation is to change the time of lambing from autumn to late winter and spring. This would involve a major management adjustment for the sheep industry in Victoria where 80% of wool-producing flocks lamb in the autumn or early winter (Caple et al. 1989). Pasture management and improvement to increase the legume content of winter and spring herbage would also increase the Ca intakes of ewes and lambs. Intestinal parasitism may be important in lambs in the winter resulting in reduced food intake, decreased Ca and P absorption, and osteoporosis (Sykes et al. 1975). Effective control of intestinal parasites after the lamb is weaned is also required to maintain growth and skeletal development when lambs are grazing green pastures. Vitamin D₃ nutrition may also be limiting in winter (Caple et al. 1988a). However, vitamin D₃ deficiency is usually associated with rickets and not osteoporosis: rickets occurs mainly in lambs older than 6 months which have compensatory bone growth while grazing oat crops in autumn and winter (Caple 1989).

MAGNESIUM NUTRITION, HYPOMAGNESEMIA AND GRASS TETANY

Inadequate Mg absorption predisposes lactating ruminants to grass tetany, a nervous disorder characterised by hypomagnesaemia, muscle spasms, tetany convulsions and death. The disorder is the main cause of death of lactating beef cows in south-eastern Australia (Shiel et al. 1981, Harris et al. 1983) and it also occurs in lactating dairy cows (Allen and Caple, 1981), but rarely in lactating ewes.

The loss of Mg in milk (0.17 g Mg/l) may predispose lactating ewes to hypomagnesaemia, but the clinical signs of hypomagnesaemic tetany usually do not occur unless there is concomitant hypocalcaemia (Hemingway and Ritchie 1963; Herd 1966). Lowering both the concentration of Mg and Ca in the cerebrospinal fluid produces more severe convulsions than reducing Mg concentration alone. The disorder has normally occurred in flocks where older lactating ewes were grazing short (<500 kg/DM/ha) green grass pastures which were low in Mg (<0.15 g Mg/Kg DM). Supplementation of the ewes with oats treated with MgO has been the method recommended for prevention of the disorder.

Recent surveys indicate that losses of lactating beef cows from grass tetany are similar to those experienced 20 years ago (Harris et al 1983). The losses continue to occur despite the past investment of large amounts of money into research on hypomagnesaemia, and the development of commercial products (magnesium oxide (Causmag), magnesium capsules (Cheetham Rural)) which are readily available to producers. The losses also occur despite government extension activities, and attempts at developing "district warning systems". For these reasons the current state of knowledge concerning grass tetany will be summarised.

Grass tetany can be a more complex metabolic disorder than simply a deficiency of Mg. An analysis of the past losses on a property can indicate which age group of cows are most at risk, and can substantially reduce the costs for labour and supplements required for prevention. There are several types of grass tetany syndromes which can be diagnosed according to the ages of cows affected, and the aetiological factors inducing the fatal nervous disorder. Cows older than six years are most commonly affected with grass tetany, particularly if they are overfat at calving and lose liveweight during
lactation. Younger, two and three year-old, cows may be affected in herds with the more complex types of grass tetany syndromes.

The important factors associated within grass tetany include:

(a) low Mg intake which can arise simply through a reduction in food intake when cows are grazing short grass dominant pastures.

(b) high potassium and low sodium intakes which have important implications for Mg absorption from the rumen. Soils naturally high in potassium or fertilized with potash, and low in sodium are high risk areas (Jolly and Leaver 1974). Grass dominant pastures with low Mg concentrations (<2g/kg DM [dry matter]), low Ca concentrations (<3g/kg DM), low sodium (<1.5g/kg DM), and high potassium (>20g/kg DM); and high nitrogen concentrations (>50g/kg DM) predispose the development of grass tetany in cattle.

(c) the cow's ability to maintain Ca homeostasis. Cows with low blood Mg concentrations do not develop grass tetany until blood Ca levels decrease (Hemingway and Ritchie 1963). Hay feeding is an important control measure in herds where hypocalcaemia precipitates grass tetany in hypomagnesaemic cows. Phosphorus deficiency may predispose cows to hypocalcaemia, and we have found this to precipitate grass tetany in lactating two-year old heifers in herds grazing pastures with less than 1.5 gMg /kg DM.

Other important factors in grass tetany include the body condition of older cows in a herd, grazing management and provision of shelter, and husbandry procedures which involve a reduction of food intake in high risk cows (Harris et al. 1983).

There are several reasons why grass tetany continues to be a problem for the cattle industry in southern Australia. Most herds are grazed on improved pastures, which contain mainly grass species during autumn and winter. Grass pastures are usually have lower Mg and Ca concentrations than legume pastures (Jolly and Leaver 1974). Cows are calved in the autumn with the object of producing vealer calves which are finished on spring pastures. Undoubtedly an autumn calving is essential for vealer production but, apart from reducing the risks for grass tetany, the relative efficiencies of calving at different times on whole farm profitability is a high priority for research.

The autumn-calving beef cow is always going to be at risk to grass tetany. Cows invariably became fat over spring and summer and, after calving in the autumn, lose up to 1.0 kg liveweight/day during lactation between May and September. This is the main risk period for grass tetany. The main loss of Mg in cows is via milk, and essentially no Mg is obtained from the tissues mobilised during loss of liveweight to support lactation. The selection of beef bulls based on growth rate of calves means that cows are being bred for high milk yields. Cows which maintain milk yield by losing liveweight in early lactation are predisposed to hypomagnesaemia and grass tetany if they do not receive additional Mg in the diet, or hay or other supplements to prevent the weight loss.

The traditional method of feeding cows Mg oxide treated hay for grass tetany prevention is often difficult, and farmers have demanded that researchers produce a one-shot treatment. The intra-ruminal Mg capsules developed by CSIRO and produced and marketed by Cheetham Rural are a useful aid to grass tetany control in some situations where cows are fed hay, and provided these devices release at least 2 grams of Mg daily. A knowledge of the factors
which are associated with grass tetany in beef cows on a property enables an appropriate "on-farm" control programme to be developed. This can reduce the labour, costs and worry associated with control of grass tetany.

CONCLUSION

In adult ruminants, Ca and Mg metabolism may be compromised when dietary intakes do not meet the requirements for pregnancy and lactation, and for restoration of mobilised bone Ca reserves. As the rumen plays an important role in Mg and Ca absorption, adequate roughage intakes to promote salivary secretion may be as important as adequate dietary mineral concentrations in preventing hypomagnesemia and hypocalcaemia. In young lambs, milk intakes to promote growth rates of 150 g/day are required to prevent osteoporosis while they are grazing pastures with their ewes.

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