Effects of rumensin anti-bloat capsules on plasma magnesium concentration and aspects of health and performance of pastured dairy cows

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ABSTRACT

One hundred and twenty three Friesian or Friesian x Jersey mature dairy cows were used to examine the effects of Rumensin Anti-bloat Capsules (ABC), containing sodium monensin on plasma magnesium, glucose and ß-hydroxybutyrate concentrations. Forty one cows received the ABC 3-8 weeks prior to calving, 41 immediately after calving and 41 were control animals. The experiment was undertaken early in the spring prior to the period during which bloat normally becomes a problem.

The ABC increased plasma magnesium concentrations relative to the control cows prior to calving (ABC 0.783 v Control 0.733 m mol/l; $P < 0.04$), but not after calving. Plasma glucose concentrations were raised significantly ($P < 0.05$) by the ABC during the first two months of lactation (ABC 3.209 v Control 3.045 m mol/l), but not prior to calving. Plasma ß-hydroxybutyrate concentrations were lowered significantly ($P < 0.01$) by the ABC both before (ABC 1.320 v Control 1.583 m mol/l), and after calving (ABC 1.510 v Control 1.777 m mol/l).

Differences between treatments in body weight, milk production and reproductive parameters were not significant, but there was a suggestion that ABC protected the cows from clinical mastitis.

It is concluded that use of the Anti-bloat capsules should help improve the health of dairy cows.

Keywords: Sodium monensin, plasma magnesium, pastured dairy cows, Rumensin anti-bloat,capsules.

INTRODUCTION

Rumensin[1] Anti-bloat capsules (ABC) are intra-ruminal devices designed to administer a controlled and continuous supply of a biologically active compound to cattle, over a period of about 100 days. The active ingredient in the ABC is sodium monensin, a (carboxylic polyether) ionophore produced by the bacteria *Streptomyces cinnamonensis*. Monensin is a rumen modifier in that it modulates selected pathways of rumen metabolism and is toxic to some bacteria (gram positive), protozoa and fungi. Its mode of action is complex (Schelling, 1984) and potential effects on animal physiology and production are extensive.

While monensin has been widely used since 1975 to increase feed conversion efficiency of beef cattle in feedlots in North America, it has more recently been used to control bloat in dairy and beef cattle in Australia and New Zealand. Overseas, it has also been reported to have been used as a preventative for coccidiosis, to be anti-ketogenic (eg Sauer *et al.*, 1989) and to improve the absorption of magnesium (Greene *et al.*, 1988).

The primary objective of the present experiment was to investigate the potential of ABC to reduce the incidence of hypomagnesaemia in pasture fed dairy cows in early lactation. Magnesium deficiency is recognised as a major limiting factor to good health and production on a high proportion of dairy farms in New Zealand.

EXPERIMENTAL DESIGN AND METHODS

One hundred and twenty three Friesian or Friesian x Jersey cows, aged 3 to 10 years, were used in the experiment. On 5 July, approximately 4 weeks prior to the planned start of calving, the cows were bled by tail venipuncture into EDTA vacutainers, plasma samples were prepared by centrifugation and were stored at -12°C prior to metabolic analysis. On 12 July cows were randomly allocated to three groups of equal size on the basis of expected calving date, age, initial plasma magnesium concentration and previous season's milk production.

The three treatments were as follows:

Group A (After)	: ABC administered on day 1, 2 or 3 after calving
Group B (Before)	: ABC administered 12 July, 3-8 weeks prior to calving
Group C (Control)	: No capsules administered

The ABC contained an 11 cm core of monensin in a hexaglycerol distearate matrix and the 32 g of monensin was expected to be released in approximately 100 days (ie 320 mg/d). The ABC were administered by balling gun and cows were held for a 1 h period on concrete to ensure that no capsules were regurgitated.

The cows were fed and managed as part of a 270 cow commercial herd, receiving some pasture silage in addition to

[1] Rumensin ABC is marketed by Elanco animal Health, Auckland, New Zealand.

grazed pasture prior to calving, but pasture alone after calving. Further plasma samples were obtained from a representative group of cows (34 and 33) from the B and C groups prior to calving on 8 August, and from all cows after calving on five occasions (at two weekly intervals) between 21 August and 21 October.

Milk yields and fat and protein concentrations in milk were measured on six individual days between 22 August and 14 November. Body weights were measured regularly, immediately prior to blood sampling, and body condition scores assessed by eye at the start and end of the experiment.

Plasma samples were analysed for magnesium (Mg), glucose (G) and ß-hydroxybutyrate (BOHB) concentrations and urea concentration (only on 8/10) using autoanalyser methods (Rosevear *et al.*, 1969; Williamson and Mellanby, 1974; Marsh *et al.*, 1965) and for calcium (Ca) concentration (using 15-19 cows/treatment) on samples taken on 21 August, using atomic absorption spectroscopy.

Breeding records obtained included calving date, days to first oestrus and mating dates (which commenced 21 October) and age of foetus assessed manually in early February. Records were kept of the incidence of metabolic disease, clinical mastitis and somatic cell counts in the milk from individual cows.

The individual cows were not "scored" for bloat severity because all cows remained free of bloat throughout the experiment.

Statistical analyses of the data were undertaken using analyses of variance and covariance in the SAS (1985) statistical package.

RESULTS

The mean plasma Mg concentration at each of the sampling dates are shown in Table 1. The ABC administered before calving resulted in a significant improvement in plasma Mg ($P < 0.04$) prior to calving. The covariance adjusted means (using data obtained before treatment as the covariate) were 0.784 and 0.731 m mol/l for the B and C groups, respectively, and these were significantly different ($P < 0.01$). There was also a significant difference ($P < 0.01$) between the adjusted means for the B and A groups at the first sample date (21/8) after calving (A 0.805, B 0.879, C 0.835) which indicates a carryover effect into early lactation from treatment before calving.

Plasma Ca concentrations, measured during the first 12 days after calving from 15-19 cows/treatment, were 93.0, 88.3 and 95.7 mg/l (SE 2.6) for treatments A, B and C, respectively, and the values did not differ significantly.

The plasma glucose concentration (Table 2) decreased following calving and tended to recover slowly as lactation proceeded. The ABC had no effect on concentrations before calving but increased values by 6% after calving ($P < 0.05$). Time of treatment (B v A) was not important in influencing the size of the effect.

Plasma BOHB concentrations were significantly (P 0.01) reduced (25% lower) by ABC both before and after calving (Table 3).

Plasma urea concentrations were measured on one occasion in early October and mean values were: A 5.45; B 5.44 and C 5.19 (± 0.14) m mol/l (differences NS). Mean liveweights, which averaged 511 kg just prior to calving and 451 kg on 21 October, did not differ significantly between treatment groups. CS at the end of the experiment were: A 4.97; B 4.84 and C 4.78.

TABLE 1: Plasma magnesium concentrations (m mol/l) for control cows (C) and for cows treated with Rumensin ABC before (B) and immediately after (A) calving.

	Before calving			After calving			
Date	5/7*	8/8	21/8	6/9	24/9	8/10	21/10
A	0.670	-	0.800	0.849	0.752	0.730	0.893
B	0.695	0.783	0.883	0.885	0.770	0.778	0.904
C	0.705	0.733	0.838	0.874	0.768	0.736	0.918
SEM	0.013	0.017	0.020	0.018	0.018	0.021	0.021
Sign. diffs	-	P < 0.04	-	-	-	-	-

* Pre-treatment

TABLE 2: Plasma glucose concentrations (m mol/l) for cows treated with Rumensin ABC before (B) and immediately after (A) calving (C, control).

	Before calving			After calving			
Date	5/7*	8/8	21/8	6/9	24/9	8/10	21/10
A	3.900	-	3.151	3.166	3.331	3.698	3.521
B	3.932	3.553	3.148	3.185	3.276	3.615	3.523
C	3.753	3.545	2.984	3.010	3.141	3.544	3.418
SEM	0.058	0.043	0.055	0.050	0.037	0.035	0.047
Sign. diffs	-	-	A>C,B>C	A>C,B>C	A>C,B>C	A>C	-
P	-	-	0.03,0.04	0.03,0.01	0.001,0. 0.003	-	

* Pre-treatment

TABLE 3: Plasma ß-hydroxybutyrate concentrations (m mol/l) for cows treated with Rumensin ABC before (B) and immediately after (A) calving (C, control)

	Before calving		After calving				
Date	5/7*	8/8	21/8	6/9	24/9	8/10	21/10
A	0.989	-	1.514	1.529	1.526	1.125	1.180
B	0.904	1.320	1.462	1.450	1.578	1.103	1.149
C	1.031	1.583	1.816	1.829	1.687	1.107	1.199
SEM	0.060	0.043	0.058	0.052	0.067	0.040	0.061
Sign. diffs	-	B<C	A<C,B<C	A<C,B<C	A<C	-	-
P	-	0.001	0.001,0.001	0.001,0.001	0.10	-	-

* Pre-treatment

The mean milk yields and fat and protein concentrations for treatment groups over the 6 sampling dates are presented in Table 4. While there were no statistically significant differences between groups for milkfat concentration, the values for ABC treated cows were consistently lower than those for the control group on all sampling dates.

TABLE 4: Productivity, reproduction and health status of cows treated with Rumensin AB

	After (A)	Before (B)	Control(C)
Milk production:			
Mean milk yields (l/cow daily)	22.24	22.92	22.54
Mean milkfat concentration (%)	4.41	4.44	4.58
Mean protein concentration (%)	3.52	3.54	3.56
Metabolic diseases:			
Grass staggers (cows)	1	0	0
Milk fever (cows)	0	1	1
Ketosis	0	0	0
Somatic cell counts in Oct (x000)	132	178	337
Mastitis treated (cows)			
1/8 - 20/10	0	1	5
20/10 - 30/11	0	3	0
Reproduction:			
Average days to first oestrus	51.6	52.4	51.8
No. cows holding to 1st service	28/41	25/39	26/40
Services/conception	1.34	1.36	1.43

The occurrence of metabolic diseases and the reproductive parameters (days to first oestrus, numbers holding to first service and conception rates) are presented in Table 4 and were similar for the three groups. There was a strong indication that the incidence of mastitis may have been reduced by ABC. Only one out of 82 cows which received the ABC had mastitis during the life of the capsules and 3 of the B treatment developed mastitis within 5-6 weeks of the expiration of the treatment. The control group of 41 cows showed 5 clinical cases.

DISCUSSION

Monensin administered during the late pregnancy period significantly increased plasma magnesium concentrations, which supports the work of Greene *et al.* (1988), who reported an increased absorption of Mg from a concentrate/cottonseed hull diet fed to steers. Following treatment with ABC the higher plasma values established before calving in the current experiment tended to persist into early lactation. This suggests that farmers should pay more attention to monitoring and raising plasma concentrations during the dry period than is currently the practice, in order to reduce hypomagnesaemia after calving.

Surprisingly, monensin administered after calving did not affect plasma Mg concentrations. The most likely reason for this is that the cows were better fed, with respect to Mg requirements, after calving than before calving. This suggestion is made on the basis of the control plasma Mg concentrations which were generally higher after calving than at the initial sampling during the dry period. Apart from herbage potassium concentrations, plane of feeding is probably the most important factor which affects plasma Mg levels in New Zealand cows. Magnesium supplements will only raise plasma Mg values (and increase production) if cows are initially severely hypomagnesaemic and this did not apply during this particular experiment.

Administration of monensin (before and after calving) had a significant positive effect on plasma glucose and a significant negative effect on ß-hydroxybutyrate concentrations in early lactation. These results are consistent with those of Sauer *et al.* (1989), who demonstrated glycogenic and antiketogenic effects of monensin in a concentrate and corn silage ration.

It has been found that monensin increases the supply of propionate at the expense of acetate in the rumen (Schelling, 1984). Maas (1990) measured the effect of monensin sodium on rumen fermentation in sheep (dose/kg liveweight daily equivalent to 320 mg/day given to cows) fed spring pasture (September) from the same farm as used in the current experiment. Mean (± standard deviation) ruminal molar proportions of acetic, propionic, butyric and minor volatile fatty acids in control and monensin treated sheep were as follows: 66.3 (2.3), 20.2 (1.7), 10.0 (1.2), 3.4 (0.8) and 64.6 (3.2), 24.2 (3.8), 8.2 (1.7), 2.9 (0.7) respectively. The ratio of lipogenic (acetic and butyric) to glycogenic (propionic, iso-butyric, valeric and iso-valeric) volatile fatty acids were 3.27 (0.17) for the control sheep and 2.74 (0.20) for the treated sheep. Similar changes in the rumen of the cows used in the present experiment would account for the glycogenic and antiketogenic effects observed in the plasma.

The higher blood glucose concentrations in the treated cows was not associated with any differences in liveweight between treatment groups. During the first month of lactation a total of 26 cows had "high" plasma ß-hydroxybutyrate

concentrations (2-2.5 m mol/l) at one or more sampling dates. While these values were well below the clinical "ketosis" range, there were marked differences in distribution across treatments (7 cows in treatment A, 19 in C and none in B). Therefore, the Rumensin ABC should, on the basis of the present results, be a useful means of helping to prevent (and probably treat) ketosis in New Zealand dairy herds. Again, it seems that treatment before calving may result in a useful carry-over effect in reducing ketosis after calving.

There were no clear cut differences between treatment groups in milk production, although there was a tendency for ABC to reduce milkfat concentration. Rumensin ABC have been shown to increase milk production of pasture fed cows by 6-8% in trials in Australia and New Zealand (Lowe *et al.*, 1991; Lynch *et al.*, 1990). Milkfat concentration was decreased, and protein maintained or increased in these experiments, effects which are consistent with an increased proportion of propionic acid/acetic + butyric acid in the rumen fluid, found by Maas (1990).

In this particular season the cows were relatively well fed, as judged by mean production levels. Because most evidence suggests that milk production responses to magnesium supplements will only occur if feeding levels (and blood plasma Mg levels) are low it is tempting to hypothesise that monensin might only give increases in milk production under poor feeding conditions and that such effects could (at least partially) be mediated through providing extra magnesium. Previous studies (Wilson, 1980) have shown that the effect of supplementary magnesium on production is mainly mediated via an increase in the efficiency with which pasture is digested by the cow.

There were no obvious differences in the reproductive parameters measured for cows in the three treatment groups. A positive effect might have been expected in view of the known effect of plasma glucose on fertility (McClure, 1972) but it should be noted that the ABC treatment effect had dissipated by the last sampling date, which coincided with the commencement of mating.

A potentially important finding from this experiment could be that the Rumensin ABC appeared to protect the cows from clinical mastitis. The mechanism of action that may be involved is not clear as no residues of monensin have been found in milk of treated cows (Lowe *et al.*, 1991).

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REFERENCES

Greene, L.W.; May, B.J.; Schelling, G.T.; Byers, F.M. 1988. Site and extent of apparent magnesium and calcium absorption in steers fed Monensin. *Journal of Animal Science* **66**: 2987-2991.

Lowe, L.B.; Ball, G.J.; Carruthers, V.R.; Dobos, R.C.; Lynch, G.A.; Moate, P.J.; Poole, P.R.; Valentine, S.C. 1991. Monensin controlled-release intraruminal capsule for control of bloat in pastured dairy cows. *Australian Veterinary Journal* **68**: 17-20.

Lynch, G.A.; Hunt, M.E.; McCutcheon, S.N. 1990. A note on the effect of monensin sodium administered by intraruminal controlled release devices on productivity of dairy cows at pasture. *Animal Production* **51**: 418-421.

Maas, J.A. 1990. The effects of monensin sodium on lactational performance of autumn and spring calving cows. MAgrSc thesis, Massey University, Palmerston North, New Zealand.

Marsh, W.H.; Fingerhut, B.; Muller, H. 1965. Automated and manual direct methods for the determination of blood urea. *Clinical Chemistry* **11**: 624-627.

McClure, T.J. 1972. Blood glucose and female fertility. *Veterinary Record* **91**: 193.

Rosevear, J.W.; Pfaff, K.J.; Service, F.J.; Molnar, G.D.; Ackerman, E. 1969. Clinical Chemistry 15: 690.

S.A.S. 1985. Statistical analysis systems. SAS Institute Inc. North Carolina, USA.

Sauer, F.D.; Kramer, J.K.G.; Cantwell, W.J. 1989. Antiketogenic effects of Monensin in early lactation. *Journal of Dairy Science* **72**: 436-442.

Schelling, G.T. 1984. Monensin mode of action in the rumen. *Journal of Animal Science* **58**: 1518-1527.

Williamson, D.N.; Mellanby, J. 1974. Methods of enzymatic analysis. Bergmeyer, H.E. ed. Verlaz. Chemie, Wenheim, pp 1836-1839.

Wilson, G.F. 1980. Effects of magnesium supplements on the digestion of forages and milk production of cows with hypomagnesaemia. *Animal Production* **31**: 153-157.