Alleviated Nutrient Imbalance by Monensin Premix (Romensin®, Rumensin®); Reduced Risk of Ketonaemia in Dairy Cows

L.J. Jonker[1], **J.I.D. Wilkinson**[2] and **M. Tarrant**[1]
[1]*Elanco Animal Science Research, Lilly Industries Ltd.*
Dextra Court, Chapel Hill, Basingstoke, Hampshire, RG21 6XA, UK
[2]*Lilly Research Laboratories*
2001 W. Main Street, Greenfield, Indiana 46140

Abstract

In a European series of studies, the effects of administering monensin premix (Romensin®, Rumensin®) to dairy cows via the feed were examined. A total of 365 cows received either 0 or 300 mg monensin per cow per day from approximately one month post calving onwards. Blood samples were examined for ketone levels (ß-hydroxybutyrate and acetoacetate).

Cows were categorized for ketonaemia risk by using their average ketone levels for up to five months of treatment. Mean ketone levels of or below 1.0 mmol/l were considered to indicate normal values. Cows with normal ketone values were categorized into three groups: low normal (≤0.50 mmol/l), medium normal (>0.50 to ≤0.75 mmol/l) and high normal (>0.75 to ≤1.00 mmol/l). Ketone levels above 1.00 mmol/l were considered to indicate a ketonaemic condition.

Cows treated with monensin were approximately twice as likely to occur in the lowest normal risk category and were approximately half as likely to occur in the highest normal category than control animals. Five percent of the control cows were found in the ketonaemic condition category versus one percent of cows which had received monensin. The difference between the two groups was significant ($p<0.001$).

These findings were most likely caused by the effects of monensin on rumen fermentation, increasing the production of propionic acid and increasing the supply of glucogenic nutrients derived from the ration. In these studies, monensin apparently alleviated nutrient imbalance and reduced the risk of ketonaemia in dairy cows during early lactation.

Keywords: monensin, dairy cows, ketones

Introduction

Feed intake level in dairy cows during early lactation is often insufficient to support the high milk yield. Peak feed intake normally occurs some weeks after peak milk production and it is a common phenomenon in early post-partum cows to produce milk partly from body nutrient reserves, mainly adipose tissue. Many tissues can derive energy directly from mobilized fat by utilizing free fatty acids or glycerol; the udder however has an absolute requirement for glucose to produce lactose. The major substrate for this glucose is propionic acid which is produced via microbial fermentation processes in the rumen.

Glucose is synthesized from propionate via the TCA-(tricarboxylic-acid) cycle. An important intermediate in the TCA cycle is oxaloacetate, which is needed for the production of glucose and for the oxidation of acetyl-CoA which may be derived from mobilized body fat (Blood and Radostits, 1989). However, when glucose or glucose precursors are in short supply, oxaloacetate is preferentially used for glucose formation and this results in the accumulation of acetyl-CoA. The accumulating acetyl-CoA is transformed into ketones (acetoacetate and subsequently to acetone or ß-hydroxybutyrate).

Ketones can be used for energy supply by various tissues. However, if their production exceeds their use, they may accumulate. As a result of this, blood ketone levels are commonly increased in early lactation dairy cows, which is a normal physiological response to the increased nutritional demands of early lactation and the insufficient supply of glucose precursors. However, increased ketone levels may present a drawback to animal performance, as this may result in ketosis which can be present in a clinical or in a sub-clinical form (Kauppinen, 1983; Dohoo and Martin, 1984; Blood and Radostits, 1989; Lean, Bruss, Baldwin and Troutt, 1991). Sub-clinical ketosis is described as the condition of in-

Presented at the XIX World Buiatrics Congress, Edinburgh, Scotland, July 8-12, 1996.

creased blood ketone levels without obvious clinical symptoms. However, the sub-clinical condition may also have a negative impact on animal performance, which includes depression of milk yield by 1-1.5 kg/day, reduced fertility and an increased risk of becoming clinically ketotic. The sub-clinical condition may be prevalent in 10-30% of early lactation cows and may rank with the clinical disease in its economic effects.

Herd incidence of ketonaemia (clinical and subclinical) may be reduced by adequate management, feeding practices and by an increased supply of glucogenic precursors in the ration (Sauer, Kramer and Cantwell, 1989).

Monensin is widely used in various parts of the world, including the EU area, to improve weight gain and feed efficiency in beef cattle. The mode of action of monensin has been researched intensively and it is known for a considerable time that monensin influences the microbial fermentation in the rumen. Effects published in scientific literature include: reduction of energy losses via fermentation gases, reduction of protein breakdown in the rumen, reduced de-amination of amino acids for energy supply, an inhibition of lactic acid producers and a reduction in gas production and fluid viscosity. A major effect of monensin however is the shift it causes in the production of volatile fatty acids: propionic acid production is increased, at the expense of acetic and butyric acid (Bergen and Bates, 1984). It was felt therefore that addition of monensin to the ration may also be beneficial for dairy cows.

Materials and Methods

In order to examine the effects of monensin administration to dairy cattle under European conditions, a series of eight studies was initiated in September, 1993. Studies were run in France, the Netherlands and the UK.

Various dosages were included in the studies, but common dosages in all eight studies were 0 and 300 mg/head/day. A total of 365 cows were included in these two groups. The study design included a pre-treatment period of approximately 1 month after calving. Cows were then blocked and randomly allocated to the treatment groups. The treatment duration was 4 to 5 months or more. A variety of performance parameters were examined in these studies, which included regular blood samplings and assay of ketone levels (ß-hydroxybutyrate and acetoacetate). Blood samples were taken following the morning milkings from the jugular or tail vein. Ketones were assayed in plasma or serum by enzymatic/spectrophotometric analysis.

Cows were classified for ketonaemia risk by using their average ketone levels for up to five months of treatment. Mean ketone levels of or below 1.0 mmol/l were considered to indicate normal values (Blood and Radostits, 1989; Lean *et al.*, 1992; Lean, 1994). Cows with normal ketone values were categorized into three groups: low normal (≤0.50 mmol/l), medium normal (>0.50 to ≤0.75 mmol.l) and high normal (<0.75 to ≤1.00 mmol/l). Ketone levels above 1.00 mmol/l were considered to indicate a ketonaemic condition.

Results and Discussion

The following numbers of cows per ketone level category were found:

Ketone level category	Control Group	300 mg Group
Low normal	27	53
Medium normal	95	107
High normal	48	24
Ketonaemic	9	2

(4-category classification, Chi-squared P<0.001)

The findings of these studies demonstrated that a relatively large proportion of cows had elevated ketone levels in early lactation. Combined over the two treatment groups, a total of three percent of cows were found in the ketonaemic condition category while 20 percent of cows were in the highest normal category.

Cows treated with monensin were about twice as likely to be in the lowest risk category. Numbers of cows in the medium normal group were about equal for both treatments. Twice as many controls were found in the highest normal group compared to monensin treated animals (27 versus 13 percent of cows respectively). The number of cows in the ketonaemic condition category was higher in the control group than in the treated group (five versus one percent of cows respectively). The difference between the two groups was highly significant.

These findings were most probably caused by the effects monensin is reported to have on rumen fermentation, thereby increasing the production of propionic acid and increasing the supply of glucogenic nutrients derived from the ration. In these studies, monensin apparently alleviated nutrient imbalance and reduced the risk of ketonaemia in dairy cows during early lactation.

References

Bergen, WG and Bates, DB, 1983. Ionophores: their effect on production efficiency and mode of action. *J Anim Sci,* 58:1465-1483. Blood, DC and Radostits, OM, 1989. Metabolic diseases. In: *Veterinary Medicine*, 1100-1149. Bailliere Tindall, London. Dohoo, IR and Martin, SW, 1984. Subclinical ketosis: prevalence and associations with production and disease. *Can J Comp Med* 48:1-5. Kauppinen, K, 1983. Prevalence of bovine ketosis in relation to number and stage of lactation. *Acta Vet Scand* 24:349-361. Lean, IJ, Bruss, ML, Baldwin, REL and Troutt, HF, 1991. Bovine ketosis: A review I Epidemiology and

pathogenesis. *Veterinary Bulletin* 61:1209-11218. Lean, IJ, Bruss, ML, Baldwin, RL and HF Troutt, 1992. Bovine ketosis: A review II Biochemistry and prevention. *Veterinary Bulletin* 62:1-14. Lean, IJ, 1994. Bovine ketosis and somatotropin: risk factors and effects of ketosis on health and production. *Research in Veterinary Science* 57:200-209. Sauer, FD, Kramer, JFG and Cantwell, WJ, 1989. Antiketogenic effects of monensin in early lactation. *J Dairy Sci* 72:436-442. Carruthers, VR, O'Connor, MB, Feyter, C, Upsdell, MP, Ledgard, SF, 1987. Results from the Ruakura bloat survey. In *Proc. Ruakura Farms Conference,* 44-46. Johnson, DE, Bramine, M and Ward, GM, 1991. Methane emission in livestock. In *Proc. Amer Feed Ind Assoc Nutr Symp* - Animal Agriculture in the 90's, p33-55. Katz, MP, Nagaraja, TG and Fina, LR, 1986. Ruminal changes in monensin - and lasalocid - fed cattle grazing bloat - provocative alfalfa pasture. *J Anim Sci* 63:1246-1257. Laby, RH, 1973 The anti-bloat capsule and detergents for bloat control. In *Bloat, Reviews in Rural Science No. 1,* University of New England, 81-83. Lowe, LB, Ball, GJ, Carruthers, VR, Dobos, RC, Lynch, GA, Moate, PJ, Poole, PR and Valentine, SC, 1991. Monensin controlled-release intraruminal capsule for control of bloat in pasture dairy cows. *Aust. Vet. Jr.* 68:17-20. Moate, PJ, Robinson, IB, O'Brien, GN, Rogers, GL and Stockdale, CR, 1992. The value of legumes for dairy production. *Proc Aust Soc Anim Prod* 19:345-347. Morris, CA, Cockrem, FRM, Carruthers, VR, McIntosh, JJ and Cullen, NG, 1991. Response to divergent selection of bloat susceptibility in dairy cows. *NZ J Agri Res* 34:75-83. Potter, EL, Cooley, CV, Raun, AP, Richardson, LF and Rathmacher, RP, 1974. Effect of monensin on daily gain of cattle on pasture. *Proc West Section, Amer Soc Anim Sci* 25:343. Quinn, AH, Austin, JA and Ratcliff, K, 1944. A new approach to the treatment of bloat in ruminants. *J Amer Vet Med Assoc* 114:313-314. Sakauchi, R and Hoshino, S, 1981. Effects of monensin on ruminal fluid viscosity, pH, volatile fatty acids and ammonia levels and microbial activity and population in healthy and bloated feedlot steers. Z Tierphysiol. *Tierenhrg u Futtermittelkde* 46:21-33. Strobel, HJ, Chow, JM and Russell, JB, 1989. Rumen ionophores: manipulating fermentation and control of acidosis. *Proc Cornell Nutr Conf.* Wolfe, EC, 1968. Cattle bloat in southern New England, NSW, 1961 - 66 *Proc Aust Soc Anim Prod* 7:123-128.

Abstract

Vitamin B_{12} Responses to Cobalt Pellets in Beef Cows

G. J. Judson[ab], J. D. McFarlane[c], A. Mitsioulis[a] and P. Zviedrans[a]
[a]Primary Industries SA, GPO Box 1671, Adelaide, South Australia, 5001
[b]Present address: South Australian Research and Development Institute
33 Flemington Street, Glenside, South Australia, 5056
Author for correspondence.
[c]Primary Industries SA, PO Box 618, Naracoorte, South Australia 5271

Aust Vet J 1997;75:660-662

Objective

To assess the effectiveness of cobalt pellets in maintaining adequate vitamin B_{12} in beef cows on pasture of low cobalt content.

Design

A field experiment in a herd grazing cobalt deficient pasture.

Animals

Mature Murray Grey cows.

Procedure

Cows were given a single oral dose of 0,1,2 or 4 cobalt pellets (30 g pellets containing 30% by weight cobaltic oxide) with a selenium pellet and a grub screw. Samples of blood, liver, faeces and milk for chemical analyses were collected at intervals over a period of 2 years after treatment.

Results

A single cobalt pellet raised liver vitamin B_{12} concentration of cows above that of untreated cows for at least 28 weeks, and 2 or 4 pellets for 57 weeks. Plasma vitamin B_{12} concentration was an unreliable indicator of the effectiveness of cobalt pellet therapy. Milk vitamin B_{12} and faecal cobalt concentrations increased in response to cobalt pellet therapy.

Conclusion

These studies show that one cobalt pellet will prevent vitamin B_{12} inadequacy in beef cows for between 28 and 57 weeks; two or four pellets will prevent inadequacy for 57 to 75 weeks. Milk vitamin B_{12} concentration may be a useful indicator of the effectiveness of cobalt pellets in increasing the vitamin B_{12} supply in lactating cows.