New Approaches to Control of Ruminal Acidosis in Dairy Cattle

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ABSTRACT: Eighty Holstein-Friesian cows were randomly assigned to four treatment groups of 20. The four groups of twenty cows were fed on diets that were designed to place cows at some degree of risk of ruminal and metabolic acidosis. The cows were either 10 kg of pellet and ate pasture, or a subgroup of six cows per group was fed 10 kg of pellet with ad lib forage in a Calan gate facility. Cows in Group 1 were fed the basal diet that contained no feed additives (C); Group 2 were fed the basal diet and 20 mg of Tylosin (T) per kg of feed; Group 3 were fed the basal diet and 20 mg of Tylosin and 20 mg of monensin (TM) per kg of feed, and Group 4 were fed the basal diet and 30 mg of Virginiamycin (VM) per kg of feed. The TM group had significantly lower rumen pH than the Tylosin group or the VM group. Volatile fatty acid concentrations in rumen fluid were consistently higher for the TM group.

Cows in the VM group and control cows also had significantly higher mean blood glucose concentrations than the TM group. Mean blood glucose concentrations significantly decreased over time, but concentrations for the TM group were significantly less variable than for other groups. There was a tendency for blood urea nitrogen concentrations to be higher in the TM group. Concentrations in this group increased markedly over time, though pattern of increase was not significantly different from the other groups. Plasma urea concentrations increased significantly over time. The TM group maintained a higher level of milk production over the period than any of the other groups and did not decline as much over the trial as did the other groups. There were marked differences in rumen metabolism, blood concentrations of metabolites and milk production that suggest that controlling rumen fermentation to reduce lactic acid production may be a useful means of reducing health risks from acidosis.

Key Words: Dairy Cattle, Acidosis, Monensin

INTRODUCTION

Ruminal acidosis is a clinical disorder of cattle that can result in rumenitis, metabolic acidosis, lameness, hepatic abscessation, pneumonia and death. Of greater economic importance are production losses that result from subclinical acidosis in dairy cattle, particularly those fed on pasture, that result in the decreased intake of pasture known as substitution. Studies have identified marked differences in fermentation rates of grains, which were related to feed intake. The common feeding practice in the Australian dairy industry, of offering large amounts of grain at milking times will increase the risk of ruminal disorders and substitution. Subclinical effects of acidosis in cattle include a lower fat content of milk, a reduced rate and extent of roughage degradation, lower feed conversion efficiency and lower feed intake. With improved control of mastitis, it is possible that acidosis is the most significant disorder of lactating dairy cattle.

An alternate approach to controlling acidosis under pasture based dairy conditions may be to control lactic acid formation in the rumen by use of modifying agents that control growth of Streptococcus bovis. Sodium monensin1, an ionophore antibiotic produced by Streptomyces cinnamomensis, selectively modifies the rumen flora and improves the digestive efficiency of cattle. Effects of monensin include reduced risk of acidosis in vitro (Nagaraja et al., 1987). Tylosin2 is a macrolide antibiotic and Virginiamycin3 (VM), an antibiotic with primarily gram positive activity, and both are effective in reducing lactic acid production in vitro (Nagaraja et al., 1987). Virginiamycin is effective in removing S bovis, the organism primarily responsible for lactic acid production. These agents used in combination or singly may be effective in reducing the risk of lactic acidosis. This study reports the effects of rumen modification with monensin, tylosin and virginiamycin on milk production and rumen volatile fatty acid production.

MATERIALS AND METHODS

Cows and Feeding Management

Eighty Holstein-Friesian cows were randomly assigned to four treatment groups of 20. The four groups of twenty cows were fed on diets that were designed to place cows at some degree of risk of ruminal and metabolic acidosis. Before the study commenced, cows were grazed on pasture, predominantly ryegrass (Lolium perenne), oats (Avena sativa) and clover (Trifolium repens) and were fed 4 kg per day of a commercial 18% dairy pellet (Millmaster Feeds, Sydney, Australia). For two days before study commencement the pellet allocation was increased by 2 kg per day, that is to 8 kg of standard pellet per day. On day one of the study, two kgs of treatment pellet were substituted for the standard pellet and over the next two days the treatment pellets were increased incrementally to 10 kgs per cow. Cows received 10 kgs of pellet for the remainder of the study. Cows were fed on trial for 24 days after the 4 day adaptation

The cows were fed either 10 kgs of pellet and ate pasture, or a subgroup of six cows per group was fed 10 kgs of pellet with ad lib forage in a Calan gate facility (American Calan, Northwood, N.H., USA). The cows fed in the Calan gates were adapted to the feeding system in a previous study. These cows had no access to other feeds, but had constant access to water and shade. The cows held at pasture could access to shade and water. Intakes of pellet were recorded at each milking and daily feed intake was monitored for cows in the Calan gates.
The base pellet was formulated on an as fed basis as follows; wheat 30.34%; sorghum 13%; wheat mill run 30%; cottonseed meal 22%; salt 0.66%; limestone 3.5%; vitamin and mineral premix 0.5%. Cows in Group 1 were fed the basal diet that contained no feed additives (C); cows from Group 2 were fed the basal diet and 20 mg of Tylosin per kg of feed; cows from Group 3 were fed the basal diet and 20 mg of Tylosin and 20 mg of monensin (TM) per kg of feed, and cows from Group 4 were fed the basal diet and 30 mg of VM per kg of feed. These additives displaced an equal quantity of wheat millrun. The operators involved in the trial were blinded from knowledge of the contents of the feed when feeding and during the statistical analysis. The study design was a double-blind, randomised, controlled clinical trial.

**Samples and Measurements**

Cows were weighed at study commencement and again at study completion. Body condition score was assessed using a 1 to 5 point scale (Edmondson et al., 1989). In the grazing group, pellets that were not consumed at milking were collected for each cow and weighed. Feed intakes were calculated daily for cattle feeding in the Calan gates. Both groups were milked twice daily and milk volume was measured and recorded at each milking.

Eight cows from each treatment group were randomly selected for sampling of rumen fluid, milk and blood each week. Cows were sampled over two consecutive days with 4 cows sampled from each group each day. Milk samples were obtained on day 3, 10, 17 and 24 of trial and were analysed for fat, protein and lactose content and for somatic cell count. Blood samples were obtained on the same ±1 day) as milk samples and were analysed for free fatty acids, glucose, 3-hydroxybutyrate, D-lactic acid and L-lactic acid. Rumen samples were taken using a shielded stomach tube and were assessed immediately for rumen pH and to determine volatile fatty acid concentrations. Cows were carefully evaluated daily for health including assessments for lameness, general health, normality of faecal output and ruminal distension.

**Laboratory Methods**

Blood samples were obtained by coccygeal venipuncture. Lactic acid and 3-hydroxybutyrate samples were collected into a sodium heparin tube, free fatty acid samples were collected in a plain tube and glucose samples were collected using a sodium fluoride tube. Blood samples were immediately placed in ice before centrifuging at 3000 rpm and storing at -40°C.

**Lactic acid:** L- Lactic acid content was measured using a Cobas-Bio (Roche Diagnostic Systems) and a Stat Pack Rapid Lactate Test kit (Behring Diagnostics Inc. Cat No. 869218). D-Lactate was assayed using a Boehringer Mannheim Test Kit (Cat No. 149 993). Volatile Fatty acid content in rumen fluid was analysed using a gas chromatograph with 3-methyl-n-valeric acid as an internal standard, using the method of Opatpatanakit (1994). Blood lactate was measured from plasma using a Cobas-Bio (Roche Diagnostic Systems) using a Stat Pack Rapid Lactate Test kit (Behring Diagnostics Inc. Cat No. 869218). Milk samples were collected into plastic sample jars containing bronopol were analysed for fat, protein and lactose content and somatic cells (Pacific Analysis, Chippendale NSW). Rumen fluid pH was measured by placing a Piccolo 2® ATC pH meter (Hanna Instruments) into the fluid.

**Statistical Methods**

After data examination to assess normality, using Statistix (NIH Analytical Software), data were evaluated using a repeated measures analysis of variance with covariates (BMDP-2V, BMDP Statistical Software, Los Angeles). Covariates that were examined included previous milk production (for milk production), days in milk (for milk production, volatile fatty acids, and milk constituents) and site (Calan Gates or pasture). Somatic cell count data were log transformed for analysis and lactic acid data were square root transformed before analysis. Significance was noted at P < 0.05 and trends in the data (P < 0.1) were identified.

**RESULTS**

There was no evidence of illness in the study period that was related to treatment. Days in milk differed among groups with; controls (C) 118.1 (±140.8), Tylosin 165.4 (±107.4), Tylosin / monensin (TM) 133.6 (±106.4), virginiamycin (VM) 189.1 (±128.1). The TM group had significantly lower rumen pH than the Tylosin group or the VM group, but no significant time or time by group effects for rumen pH were found (Table 1). Volatile fatty acid concentrations in rumen fluid were consistently higher for the TM group. Group by time effects were not significant for any volatile fatty acid (P > 0.14), indicating that the pattern of change in VFA was not different between groups, but all VFA showed a significant quadratic response over time, with groups decreasing in VFA concentrations at the second sampling and increasing thereafter. There were no between group differences in the percentage of rumen VFA’s, however, there was evidence of a slightly higher (P = 0.1) iso-butyrate percentage in the Tylosin and VM group than the control group. Site was a significant covariate for a number of the VFA evaluations.

Blood lactate in all groups tended to be lower for the tylosin containing groups and differences between these groups and VM approached significance (Table 1). Lactate concentrations (D, L and total) increased significantly over time. Blood glucose concentrations differed among groups as mean blood glucose concentrations were significantly lower in the Tylosin and TM groups than for the VM group and control cows also had significantly higher mean glucose concentrations than the TM group.
Mean blood glucose concentrations significantly decreased over time, but concentrations for the TM group were significantly more stable than for other groups (Table 1). There was a tendency for lower blood urea nitrogen concentrations to be higher in the TM group. Concentrations in this group increased markedly over time, though pattern of increase was not significantly different from the other groups. Plasma urea concentrations tended to be lower in the Tylosin group (Table 1). There was a tendency for blood urea nitrogen concentrations to be higher in the VM group. Plasma urea concentrations tended to be lower in the Tylosin and control groups compared with the VM and TM groups. Somatic cell concentrations in milk did not differ among groups. While intake of the Tylosin pellet tended to be lower for cows at pasture, the feed intake of cows on the feedpad was highest for this group, suggesting that there was little difference in palatability of feeds (Table 1). The intake of pellet was high for all groups of cows on pasture. Bodyweight gain tended to be significantly greater for the TM and the VM group than for the control and Tylosin groups.

**DISCUSSION**

Cereal grains are included in ruminant diets to improve the fermentation characteristics of the ration. Feeding grains, high in starch and readily digested can result in digestive disruption and disorders of health. An end product of the process of rumen fermentation is lactic acid, and if this accumulates the lower pH can lead to suboptimal rumen function, a risk of acute or chronic acidosis in the rumen or gut and systemic acidosis resulting from the disturbance of the acid base balance.

The experimental diet was designed to create subclinical acidosis. Despite this, rumen pH observed during the trial were relatively high. This may a consequence of the sample time being close to feeding or the diet not providing sufficient acidotic challenge. The energy density of the pasture and chaff during the trial period was not high and the acid detergent fibre content of the ration, both in chaff and pasture, was sufficiently high to maintain rumen pH in a range where marked acidosis was unlikely. Cattle on pasture, however, can select diets higher in protein and energy.
than those analysed. Statistical analysis of the effect of site indicated that the basal diet did influence, in some cases, quite markedly, rumen pH and VFA percentages, milk fat content and blood lactate. All these results, with the exception of milk fat content indicated that the carbohydrate content of the diet was insufficient to fully challenge the cattle. Despite this, there are a number of findings that suggest that the TM combination was effective in providing favourable modifications to the rumen environment.

Rumen pH (Table 1) was lower and rumen lactate and rumen volatile fatty acid concentrations higher in the TM group. Rumen pH is influenced by the total VFA present and by the relative proportion of the three main VFA present (Dijkstra, 1994). Lactic acid has approximately 10 times more influence on rumen pH than the VFA (Russell and Hino, 1985). Total blood lactic acid concentrations even following a challenge to increase accumulation were low in rumen fluid and were low, suggesting that this did probably not greatly contribute to the lower pH. While total VFA concentrations were significantly higher in the TM group, these were not as high as at the first sampling. While rumen VFA concentrations may not reflect the clearance of VFA from the rumen (Dijkstra, 1994), the higher VFA concentrations of the TM group are reflected in other biologically important outcomes. Milk yields in this trial were very satisfactory for the farm used. Milk production in the TM group was highest, but not significantly higher, after days in milk was used as a covariate. Blood glucose concentrations were lower and blood β-hydroxybutyrate (BHB) tended to be lower in the TM group, findings consistent with higher levels of milk production. These findings are not consistent with other studies in lactating dairy cattle, that have identified a glucogenic effect of monensin (Abe et al., 1994). However, the significantly higher plasma urea concentrations in the TM group may reflect the improved flux of microbial protein to the small intestine identified with monensin use. Plasma urea concentrations increased markedly in the last two weeks of treatment, when blood glucose concentrations also fell and suggest that the significantly sustained higher milk production in the TM group may possibly reflect improved protein availability. Further, both the TM and VM groups gained an additional 7 to 10 kg more weight, that could be converted to an additional 238 to 340 MJ of ME. This would reflect approximately 1 kg per day of extra dry matter feed intake for these groups. However, feed intakes for the cattle in the Calan gates were not significantly different and intakes for the TM group were lowest suggesting that the responses were mediated in part through improved rumen function and, in part, through the pastured cattle, rather than cows fed in the Calan gates. While effects of fewer days in milk needs to be considered for the TM group, the evidence of more sustained milk production, greater weight gain, significant changes in rumen and blood metabolites suggests that the responses to this treatment were consistent. All groups should have decreased in milk production at a similar rate, but this did not occur and milk production increased in the TM group during the time when blood urea concentrations increased and glucose concentrations decreased.

The significantly higher blood lactate concentrations of the VM group are difficult to evaluate. Blood lactate concentrations do not simply reflect rumen concentrations of lactate, but also reflect endogenous lactate production from glucose metabolism in anaerobic pathways and from deamination of amino acids.

New methods of controlling acidosis will derive from improved understanding of the pathogenesis of acidosis. Factors that influence ruminal pH include; access to preformed acids in feeds; a failure to produce buffering with endogenously derived buffers such as salivary bicarbonate; production of weak volatile fatty acids acetate, butyrate and propionate; and production of lactic acid in the rumen. Control mechanisms for acidosis used to date in dairy production consisted of dietary buffers and neutralising agents. There is a limited capacity for NaHCO3 to buffer the rumen and studies that have examined the effects of sodium bicarbonate on rumen pH on pasture- or alfalfa-based diets (Staples and Lough, 1989) found no significant effect of NaHCO3 on the pH of rumen fluid. However, acidosis can occur on pasture only diets. There is a need to focus on the control of rumen fermentation, rather than on rumen pH per se. The rumen modifiers, especially TM or VM can achieve this (Nagaraja et al., 1987). Cows produce 2.75 to 7.22 mols/day of volatile fatty acids per kg of dry matter intake and increasing the production of these is a positive outcome that may result in a lower pH.

REFERENCES


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