Studies have shown that claw conformation has an impact on longevity and production. The problem in dairy cattle is that hoof growth always exceeds hoof wear. When the hoof becomes too long, it becomes difficult to get much hoof wear when the animal walks. If the hoof is trimmed to a more normal shape, the animal is better able to wear the hoof down on their own. Rates of hoof growth and wear are lowest during the winter and highest during the spring and summer. Hoof growth is greatest in heifers and the least in older cows. Therefore, if one trims heifers after the season of rapid hoof growth (summer), they should maintain better hoof conformation longer than those trimmed before the warmer months.

Agriculture Canada funded a research project to investigate the impact of preventive hoof care on first lactation milk production. 252 heifers on eight Holstein dairies in Prince Edward Island were enrolled. All enrolled heifers were between the ages of 6 and 16 months. Four farms used tie-stalls for their heifers and four utilized large loafing pens. 50% of the heifers were randomly selected for preventive hoof care by a professional hoof trimmer. Approximately half of the trimmed group were trimmed in May and the other part of the trimmed group were done in November-January, depending on when the heifers were brought into the barn for winter housing. 133 heifers completed their 305 day first lactation record; 68 had been trimmed as heifers and 65 had not. The trimmed heifers produced 500 kg more milk than the control heifers only when they were trimmed in the late fall. There was no difference between the groups if they were trimmed in the spring. The heifers kept in tie-stall winter housing responded to the fall trimming treatment with higher milk production than those heifers in large loafing pens.

Trimming heifers before they calve does have an impact on their first lactation milk production. In this data, it appears that trimming should be done in heifers in the fall when they return to their winter housing. Tie-stall heifers appear to benefit most from preventive hoof care.

Efficacy of Monensin in the Prevention of Subclinical Ketosis in Lactating Dairy Cattle

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Abstract

A field study was conducted to evaluate the efficacy of a monensin controlled release capsule (Rumensin CRC) for the prevention of subclinical ketosis in lactating dairy cattle. A total of 1011 cows from 25 Holstein dairy herds in southern Ontario were enrolled in this trial. Allotment of treatment and placebo capsules were randomised within farm and the capsules were administered approximately 21 days prior to expected calving date. Blood was collected at the time of bolus administration, and both blood and milk were obtained at weeks 1, 2, 3, 6 and 9 post calving. All cows were given body condition scores at each sampling period. Serum was evaluated for a seven item profile that included beta-hydroxybutyrate (BHB), glucose, calcium, phosphorus, aspartate aminotransferase (AST), total protein and urea. Milk was analysed qualitatively for acetoacetate and acetone using a sodium nitroprusside based test (Ketocheck).

Monensin treated cows had mean values of BHB that were significantly lower than controls for weeks 1,2 and 3 post-calving ($p<.05$). Mean glucose levels in treated cows were significantly higher than controls for weeks 1 and 2 post-calving ($p<.05$). Mean serum urea values were significantly higher in treated cows for weeks 2 and 3 post-calving. Mean serum AST values were significantly lower in treated cows after freshening. No significant differences were found for serum calcium, phosphorus or total protein. Mean body condition scores were significantly higher for treated cows post-calving. Subclinical ketosis was defined, a priori, at a threshold of 1200 umol/L BHB. Monensin-treated cows had significantly less subclinical ketosis than placebo cows in every sampling week post-calving ($p<.05$). Percent of cows with positive milk ketone tests were significantly lower for treated cows ($p<.05$).

Prepartum administration of monensin significantly improved indicators of impaired energy metabolism in the postpartum period, including serum

BHB, serum glucose, serum AST, milk ketone reactions and body condition scores. The Rumensin CRC administered prepartum shows significant promise as an aid to the prevention of subclinical ketosis in lactating dairy cows.

Introduction

Subclinical ketosis has been associated with a decrease in milk production, with an increase in cystic ovarian disease, displaced abomasum, and mastitis, and with impaired reproductive performance[1,2,3,4]. Prevention of the condition should hypothetically improve health, increase milk production and enhance reproductive performance. Subclinical ketosis is characterised by an increase in circulating levels of ketone bodies without the presence of the commonly described clinical signs of anorexia, decreased milk production and firm dry feces[5]. The primary risk period is the first two months of lactation. The peak prevalence occurs during the first month[1,6,7], when the homeorrhetic drive for increased milk production draws on body fat stores to compensate inadequate energy intake . The resulting increased mobilisation of free fatty acids lead to synthesis and accumulation of ketone bodies[5]. The common ketone bodies are beta-hydroxybutyrate (BHB), acetoacetate and acetone. A range of ketone bodies and thresholds have been reported for defining subclinical ketosis, many of which have been summarised by Andersson[6]. Threshold levels of BHB used for defining subclinical ketosis have ranged from 1000 to 1400 umol/L[2,8,9].

Since ketosis occurs early in lactation, prevention has focused on nutritional management of the dry and transition cow[10]. Propylene glycol has been shown to be beneficial when administered prophylactically[11,12], however, its use requires daily administration. Monensin sodium, administered in the feed was effective in alleviating a herd problem of clinical ketosis[13], and in reducing levels of circulating total ketone bodies[14]. The Rumensin CRC administered during the first week post-calving has been shown to reduce circulating levels of BHB[15]. The Rumensin CRC capsule is a sustained release intraruminal device that delivers approximately 335 mg of monensin sodium per day for about 95 days. The Rumensin CRC offers the benefits of a sustained release that is independent of fluctuating daily feed intake. The objectives of this field study were to investigate the efficacy of the Rumensin CRC, administered prepartum, on the prevention of subclinical ketosis in lactating dairy cattle.

Materials and Methods

Twenty-five Holstein dairy herds located within 30 kilometers of Guelph, Ontario, Canada were selected, based on the herd operators= willingness to participate in the study, and the herds= enrollment in Ontario Dairy Herd Improvement Corporation (ODHIC) for the recording of milk and component measurements. Assignment of placebo or monensin capsules were randomised within farm and were administered approximately three weeks prior to the expected calving date. Capsule administration commenced in March, 1995 and was completed by December of 1995. First lactation animals were excluded (5 farms) if any ionophore was fed closer than 4 weeks prior to parturition. Every farm was visited weekly, on the same day of the week and approximately the same time of day. Blood was collected from the coccygeal vein into 10ml vacuum tubes at the time of capsule administration and during weeks 1,2,3,6 and 9 post-calving. Cows were body condition scored on a scale of 1 to 5 at the time of each sample collection. Composite quarter milk samples were obtained from each cow at the same time as blood collection post-calving. Samples were returned to the OVC within five hours post collection. Blood was centrifuged at 1200 g=s for 10 minutes. Harvested serum was subjectively scored for hemolysis on a scale of 0 to 3 and immediately submitted for determination of calcium, phosphorus, total protein, urea, glucose, aspartate aminotransferase (AST) and beta-hydroxybutyrate (BHB) levels. All biochemical tests were conducted on an automated analyser (Dacos, Coulter Electronics, Hialeah, Florida, USA). Reagents for all tests were supplied by Coulter Electronics except for beta-hydroxybutyrate which was supplied by Sigma Diagnostics, St. Louis, Missouri, USA. Milk was evaluated for ketones using a commercially available sodium nitroprusside based test (KetoCheck).

Data were stored in a commercial data base program. Analysis of the serum profile and body condition score data was conducted using a repeated measures model in SAS (16). The interaction term of sample (1 to 6) and treatment was tested for significance (p<.05). If there was significant interaction, data was analysed by sample with farm and season as covariates. Hemolysis associated with sampling has been reported to increase assay estimates of serum beta-hydroxybutyrate (17). Thus, the level of hemolysis was included as a covariate in the analysis of the treatment effect on BHB. Since we were assessing the effect of treatment on seven biochemical variables and body condition score, a Bonferoni correction of the p-value was used (p<.006 = .05/8). For evaluation of the effect of treatment on the proportion of cows with subclinical ketosis and proportion of cows with positive milk ketone tests, a logistic regression model in Stastix (18) was used. The model included the dependent variable, treatment and sample, season, herd and hemolysis. All p-values used were .05, unless otherwise indicated.

Results

A total of 504 cows were given Rumensin CRC=s and 507 received placebo capsules. Approximately 10 percent of cows were sold after treatment, but before week 9. An additional 2% were lost due to mortality unrelated to treatment. Four percent of cows regurgitated the capsule. There were no significant differences in these rates between treated and placebo cows. There were significant effects of season and herd for all analyses.

The levels of BHB, glucose, AST, urea and body condition score were significantly influenced by treatment. There were no significant effects found for calcium, phosphorus and total protein. There were significant sample*treatment interactions for BHB, glucose, and urea but not for body condition score or AST. BHB was significantly lower for monensin treated cows in weeks 1,2 and 3 post-calving after accounting for the effects of hemolysis. Glucose was significantly higher for monensin treated cows in weeks 1 and 2 post-calving. Serum urea was significantly higher in the Rumensin CRC group during weeks 2 and 3 post-calving. Mean values for BHB, glucose and urea by treatment group and sampling week post calving are shown in figures 1,2 and 3, respectively. Mean values for AST and body condition score before and after calving are outlined in table 1.

In the logistic regression model for subclinical ketosis, the variables for treatment, herd, season, hemolysis and each sampling week post-calving were all significant. The prevalence of subclinical ketosis by week of sample for each treatment group is plotted in figure 4. There was a significant reduction in subclinical ketosis (BHB $ 1200 umol/L) by monensin treatment at each of the sampling weeks post-calving. In the model for positive milk ketones, sample week was not significant. Again, treatment had a significant protective effect on the percent of cows showing positive milk ketones (figure 5).

Discussion

The seasonal and herd variation found for subclinical ketosis in this study are consistent with previous research[3,19,20,21,22]. The significant treatment effects of Rumensin (monensin) in reducing serum BHB have been demonstrated in earlier studies. Sauer *et al* (1989) reported reduced BHB and total ketone bodies for cows treated with either two dose levels of monensin (15 and 30 ppm).[14] Based on Sauer observed feed intakes, the dose delivered by the CRC capsule would approximate the higher dose level (30 ppm).[14] In an Australian study by Abe *et al* (1994), the CRC reduced BHB, but showed no significant effects on urea or glucose.[15] There was a trend towards higher glucose for treated cows.[15] By contrast, significant effects of Rumensin on glucose have been demonstrated in the current trial. The mean BHB levels for both treatment and control cows were much higher in the Australian study. This could be a function of a much smaller sample size (16 vs 1011), and by the use of only one herd. The decrease in AST in treated cows post-calving supports the beneficial effects of increased glucose and reduced BHB. Although the enzyme is not specific to liver damage,[23] it may be that the observed lower levels of AST in treated cows demonstrate some improvement in liver function in these animals. The higher mean urea levels in treated cows were not reported by Abe *et al* .[15] The mean values of urea for both treated and placebo animals in this study are within suggested reference ranges for lactating cows .[24]

The higher urea levels could be the result of a greater feed intake in treated cows, which would cause more absolute protein to be ingested. Rumensin has a reported protein Asparing@ effect that causes a reduction of protein degradation in the rumen and provides more protein to the small intestine.[25] Absorbed non-essential amino acids can be used for gluconeogenesis, and the subsequent deamination would result in increased levels of circulating urea .[26] Rumensin studies in steers and lambs have demonstrated higher urea and lower rumen ammonia in treated animals.[25,27,28] In the trial by Sauer *et al.* (1989), feed intake was significantly lower for monensin treated cows. However, no decrease in milk production was found.[14] Abe *et al.* (1994) suggested that a trend of lower body weights and reduced milk production in treated cows was due to a lower feed intake; although dry matter intake was not measured .[15] The current experiment was a field study and therefore feed intake data were not available. Mean body condition scores were significantly higher in monensin treated cows post-calving. The mean body condition scores were significantly higher by week nine but the difference amounted to 0.1 of a body score. This difference obviously is not clinically detectable, and may reflect statistical rather than biological significance. However, it certainly suggests that there was no additional loss of body weight in monensin treated cows compared to placebo animals.

Using the threshold of 1200 umol/L BHB, defined a priori, the risk of subclinical ketosis was reduced by half when cows received a Rumensin CRC three weeks prepartum (Odds Ratio = .49".07). In this trial, treatment also reduced the risk of a cow developing a positive milk ketone test by approximately one half (Odds Ratio = .46 " .14). The prevalence of subclinical ketosis identified by the milk ketone test was considerably less than that of serum BHB, however, the treatment effects and sample prevalence pattern were consistent between tests. The relative difference in levels of subclinical ketosis between that identified by serum or the milk test, suggest that either the threshold for serum was too low or that the milk ketone test was not sensitive. The threshold chosen for defining subclinical ketosis was a mid-point of reported threshold ranges of serum BHB.[2,8,9] Future analyses from this study will evaluate the effect of the reduction of subclinical ketosis on milk produc-

tion, health and reproduction.

Based on the results of this field study, the Rumensin CRC administered prepartum, has a marked positive influence on energy indicators of dairy cattle in the early post partum period and shows significant promise as an aid in the prevention of subclinical ketosis in lactating dairy cattle. Further work needs to be done to evaluate the health and performance effects of this observed energy balance improvement in Rumensin treated cows.

Table 1: Chart of the number of cows, mean AST levels and mean body condition scores for cows administered either monensin (treatment) or placebo controlled release capsules at 3 weeks prepartum

	Number of Cows		Mean AST(U/L)		Mean Body Condition Scores	
Time of Sample	Treatment	Placebo	Treatment	Placebo	Treatment	Placebo
Bolus Administration	504	507	66.7	67.8	3.4	3.4
Week 1	486	489	105.8	111.3	3.3	3.2
Week 2	478	485	101	110.8	3.1	3.0
Week 3	473	467	87.1	95	2.9	2.8
Week 6	456	451	76.1	79.8	2.7	2.6
Week 9	431	424	81.2	83	2.6	2.5

Note: Treatment significantly lowered mean AST and reduced mean body condition score loss post-calving ($p<.05$).

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