Effects of Monensin on Ruminal Forage Degradability and Total Tract Diet Digestibility in Lactating Dairy Cows During Grain-Induced Subacute Ruminal Acidosis

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ABSTRACT

The effects of monensin premix supplementation on ruminal pH characteristics and forage degradability, and total tract diet digestibility during grain-induced subacute ruminal acidosis (SARA) in lactating dairy cows receiving a total mixed ration were investigated. Six multiparous, rumen-fistulated Holstein cows were used in a 2-treatment, 2-period (5 wk per period) cross-over design. During wk 5 (d 29 to 35) of each period, SARA was induced using a grain challenge model, and ruminal pH was measured continuously using indwelling pH probes. Ruminal degradation of corn silage and alfalfa haylage was determined using the *in situ* (nylon bag) technique, and total tract diet digestibility was determined by total fecal collection during wk 5. Monensin supplementation did not affect dry matter intake, milk yield, and composition, and ruminal pH characteristics under these experimentally induced SARA conditions. Rates of ruminal forage fiber degradability were similar between control and monensin-treated cows; however, monensin supplementation increased total tract fiber digestion. This study indicates that monensin altered total tract nutrient digestion by increasing fiber digestion at postruminal sites.

(Key words: dairy cow, monensin, ruminal acidosis, nutrient digestibility)

Abbreviation key: SARA = subacute ruminal acidosis.

INTRODUCTION

Subacute ruminal acidosis (SARA) is a common digestive disorder that affects most high-producing dairy

herds, impairing both the health and productivity of dairy cows (Nocek, 1997). This digestive disorder is characterized by daily episodes of low ruminal pH between 5.2 and 5.6 (Owens et al., 1998). In early-lactating dairy cows, SARA is usually caused by the consumption of diets with high levels of rapidly fermentable carbohydrates and/or marginal, often deficient, levels of physically effective fiber (NRC, 2001). Acute acidosis, which is characterized by ruminal pH below 5.2 (Owens et al., 1998), is generally linked to lactic acid production, but excessive VFA production may be a more important contributor to SARA problems in many dairy herds (McGuffey et al., 2001). Potentially, ruminal acidosis can occur at any time in high-producing dairy cows, but it is most common during the transition period when cows are rapidly switched from a forage-based diet in the dry period to a concentrate-based diet in early lactation. Among its numerous manifestations, SARA impairs the activity of ruminal cellulolytic bacteria (Grant and Mertens, 1992), and various reports have demonstrated decreased fiber digestion both *in vitro* (Grant and Mertens, 1992, Calsamiglia et al., 2002) and *in vivo* (Plaizier et al., 2001, Krajcarski-Hunt et al., 2002) under SARA conditions in dairy cows.

Monensin, an ionophore, has been reported to have a variety of beneficial effects in ruminants. Reported benefits in dairy cattle include a lower incidence of ketosis and displaced abomasums, reduced loss of body condition, increased milk production and improved milk production efficiency (see review by McGuffey et al., 2001). Dennis et al. (1981) showed that monensin supplementation inhibited most lactate-producing ruminal bacteria, while most lactate-consuming ruminal bacteria were unaffected. These changes in ruminal microbial populations would be expected to result in shifts in patterns of ruminal fermentation. In beef cattle (Nagaraja et al., 1985; Burrin and Britton, 1986; Cooper and Klopfenstein, 1996) and in transition dairy cows (Green et al., 1999) consuming high-grain diets, monensin was efficacious in elevating ruminal pH. Such increases in ruminal pH would make the ruminal environ-

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ment more favorable for cellulolytic bacteria which, in turn, could potentially mitigate the decrease in fiber digestibility that has been associated with SARA; however, there are no reports in the literature in which ruminal degradability and total tract digestibility of fiber has been measured under SARA conditions. Plaizier et al. (2000) showed that prepartum administration of a monensin controlled-release capsule improved apparent fiber digestibility immediately before calving and improved apparent CP digestibility immediately after calving in transition dairy cows, but these measurements were taken under "normal" feeding conditions without induction of SARA, and ruminal pH was not monitored. Therefore, the objective of this study was to investigate the effects of a monensin premix on ruminal forage degradability and total tract diet digestibility in lactating Holstein dairy cows during grain-induced SARA. Our hypothesis was that supplementation with monensin would minimize the impairment of fiber digestion that is common during SARA in dairy cows.

MATERIALS AND METHODS

Animals and Experimental Design

Six ruminally fistulated, multiparous Holstein dairy cows (4.0 ± 0.5 parity, 135.8 ± 36.7 DIM) were used in this experiment. The study was run as a crossover design experiment with 2 treatments and 2 periods of 35 d each. At the beginning of the study, the cows were blocked in pairs based on DIM, and each pair was randomly assigned to receive either a monensin premix or a placebo premix. After the first 35-d experimental period, the treatments were then switched. Animals were cared for and handled in accordance with the Canadian Council on Animal Care regulations, and the University of Guelph Animal Care Committee approved their use for this experiment. The experiment was conducted at the Elora Dairy Research Center (EDRC, University of Guelph, Guelph, ON) from July 1 to September 9, 2002. The animals were housed in the main barn at the EDRC until approximately 7 d prior to each SARA week (d 29 to 35) when they were moved into the Physiology Wing and put into individual tie stalls fully equipped for metabolism studies and for continuous measurement of ruminal pH.

Experimental Treatments and Feeding

Experimental treatments were a monensin premix (Rumensin Premix, Elanco Animal Health, Division Eli Lilly Canada Inc., Guelph, ON, Canada) or a placebo premix. For the monensin premix, monensin was incorporated into soybean hulls, which acted as the carrier.

Table 1. Ingredients and chemical composition of the total mixed rations fed to lactating dairy cows.

Ingredient	%, as-fed basis	
Corn silage	50.7	
High moisture corn	11.9	
Mixed haylage	25.5	
Second-cut alfalfa hay	3.4	
Lactating cow supplement ¹	8.5	
Chemical composition	Monensin	Control
DM, %	42.5	42.5
	% of DM	
CP (N × 6.25)	17.7	18.0
ADF	25.9	24.8
NDF	40.6	39.0
NFC	41.8	43.0
Crude fat	3.4	3.4
Ash	6.4	6.5
Ca	0.78	0.90
P	0.40	0.41
K	1.50	1.47
Mg	0.26	0.27
Na	0.17	0.19
NE _L , ² Mcal/kg	1.47	1.49

¹Contained (DM basis): 41.0% CP, 43.2% rumen undegradable protein (% of CP), 56.8% rumen degradable protein (% of CP), 3.07% Ca, 1.23% P, 0.62% Mg, 1.25% K, 0.74% S, 1.04% Na, 1.56% Cl, 660 mg of Fe/kg, 496 mg of Zn/kg, 124 mg of Cu/kg, 495 mg of Mn/kg, 1.27 mg of Se/kg, 8.4 mg of Co/kg, 5.72 mg of I/kg, 52,890 IU of vitamin A/kg, 13,230 IU of vitamin D/kg, 190 IU vitamin E/kg.

²Estimated using equations and values according to NRC (1989).

The placebo (control) premix consisted of soybean hulls. The monensin premix was mixed with the TMR daily to achieve a rate of 22 mg of monensin/kg of TMR (DM basis). The placebo premix was mixed with TMR at the same rate as the monensin premix. Dietary composition and chemical analysis are shown in Table 1. Animals were fed TMR at 0700 and 1500 h for ad libitum consumption. For the initial 7 d of each experimental period, cows received unmedicated TMR (no monensin). Cows were then introduced to dietary treatments, with a 3-wk adaptation period (d 8 to 28) and a 7-d measurement period (d 29 to 35). During the measurements period, individual cow feed intake was recorded daily. Feed (TMR) samples and orts were collected daily, stored at -20°C , and composited weekly. Pooled TMR and orts samples were analyzed for DM by drying in an oven at 60°C for 48 h (AOAC, 1990), ADF (AOAC, 1990), NDF (Goering and Van Soest, 1970), and CP using the macro-Kjeldahl procedure (AOAC, 1990). Experimental cows were milked twice daily at 0500 and 1500 h, and milk weights were recorded. Milk samples were collected daily from morning and afternoon milkings and preserved with 2-bromo-2-nitropropane-1-2-diol. Milk samples were then pooled daily based on milk yield, and pooled samples were immediately submitted to the Central Milk Testing Laboratory (Laboratory Services Division, University of Guelph, Guelph, ON).

for compositional analysis. Milk samples were analyzed for CP, fat, and lactose using a near-infrared analyzer (Foss System 4000, Foss Electric, Hillerd, Denmark) according to AOAC (1990).

Induction of SARA and Continuous Measurement of Ruminal pH

During wk 5 (d 29 to 35) of each experimental period, SARA was induced in all cows on a daily basis using a grain challenge experimental model that was developed by Keunen et al. (2002). This experimental model consisted of restricting TMR intake to 85% of ad libitum intake (as determined in the preceding 7-d period) and substituting with 15% grain pellets consisting of 50% wheat and 50% barley. The feeding schedule during SARA induction was as follows: 1) At 0700 h, cows were fed 2 kg of the TMR; 2) At 0900 h, cows were fed 2/3 of their daily grain pellet allowance; 3) At 1100 h, cows were offered TMR for 30 min; 4) At 1300 h, cows were fed the remaining 1/3 of their daily grain pellet allowance; 5) At 1500 h, cows were offered TMR for 30 min; 6) At 1700 h, cows were fed the remainder of the daily allowance of TMR. Any grain pellets not consumed within 30 min of feeding were directly introduced into the rumen via the rumen cannulae. During the SARA period, ruminal pH was recorded on a continuous basis using indwelling pH probes as described by Cumby et al. (2001). Briefly, a pH probe was suspended in the ventral sac of the rumen through a rumen cannula and linked directly to a computer. A pH reading was taken every second, averaged over every 60 s, and then stored. The pH probes and pH transmitters were calibrated with pH 4 and 7 buffer solutions (Fisher Scientific, Fairlawn, NJ) twice per week (i.e., on d 29 and 33), and the position of the pH probes in the rumen was checked daily for each cow. The continuous ruminal pH data were summarized for each 24-h period by calculating the mean pH, the amount of time below pH 6.0 and 5.6, and the area (time $\times$ pH) below pH 6.0 and 5.6.

Fecal pH during SARA was monitored. Fecal rectal grab samples were collected on d 34 at 4-h intervals between 0900 and 2100 h. Fecal samples (80 g) were combined with distilled water (40 g) in cups, then sealed and shaken by hand to make the feces an even consistency. Measurements of pH were taken immediately using a Corning Benchtop pH meter (Corning 220, Corning Inc., Corning NY) and an Accumet Gel-filtered Polymet Body Combination Electrode (Fisher Scientific) calibrated with pH 4.0 and 7.0 buffer solutions (Fisher Scientific).

Ruminal Forage Degradability and Total Tract Diet Digestibility

Ruminal forage degradability and total tract diet digestibility were measured during the SARA week (d 29 to 35) of each experimental period. Ruminal forage degradability was determined using the in situ (nylon bag) technique (Ørskov et al., 1980), and test feeds were corn silage and alfalfa haylage. After oven drying at 60°C and grinding through a 1-mm screen (Thomas Wiley, Philadelphia, PA), test feeds were weighed (5-g sample) into nylon bags. Starting on d 31 of each experimental period, duplicate nylon bags containing test feeds were incubated in the rumen of each cow for 3, 6, 12, 24, 48, and 72 h. After each incubation time, duplicate bags of each test feed were withdrawn from the rumen and hand-washed in lukewarm water for 5 min. Nylon bags containing test feed residues were then oven-dried for 48 h at 60°C, and DM loss was determined. Dried feed residues were then analyzed for NDF (Goering and Van Soest, 1970). To determine DM losses during bag washing, 3 nylon bags (5-g sample) of each test feed (nonincubated) were hand-washed in lukewarm water for 5 min and then oven-dried for 48 h at 60°C. Ruminal DM and NDF degradabilities were fitted to an exponential equation using the SAS nonlinear regression procedure (SAS, 1990). Parameters that were calculated were: A, washing loss; B, potential degradability after washing; (A + B), potential total degradability; and c, degradability rate constant (Ørskov et al., 1980).

Total tract diet digestibility was determined using the total collection technique (Plaizier et al., 2000). Between d 29 and 35, representative TMR samples were obtained daily and stored frozen at -20°C until later analysis. Urine was collected using indwelling bladder catheters (Bardex Foley catheter, 75-mL capacity balloon; C. R. Bard Inc., Covington, GA) as described by Wright et al. (1998) to avoid feces contamination, and was discarded on a daily basis. All feces were collected in large steel trays positioned over the gutter behind each cow. On a daily basis at approximately 1100 h, all feces were removed from the trays and placed in large plastic tubs. Feces were weighed and thoroughly mixed, and subsamples (approximately 1 kg) were taken and stored frozen at -20°C. Before chemical analysis, frozen fecal samples were thawed and oven-dried for 48 h at 60°C. After drying, samples were pulverized, and pooled by weight (using oven DM values) for each cow and period. After drying again, the pooled samples were ground through a 3-mm screen on the Retsch SM 2000 (Denmark) and then ground through a 1-mm screen on the Retsch ZM 100 (Denmark). Pooled samples of feeds, orts, and feces were analyzed for CP using the macro-

Table 2. Ruminal pH characteristics and fecal pH during subacute ruminal acidosis (SARA) in lactating Holstein dairy cows as affected by supplementation with a monensin premix.

Item	Monensin	Control	SE	<i>P</i> value
Ruminal pH characteristics				
Mean pH	6.11	6.07	0.05	0.57
Minimum pH	5.39	5.27	0.13	0.58
Maximum pH	6.77	6.79	0.08	0.87
Minutes pH < 6, min/d	566.3	591.4	39.8	0.69
Area pH < 6, min pH/d	199.6	276.8	46.0	0.32
Minutes pH < 5.6, min/d	198.1	301.5	66.6	0.36
Area pH < 5.6, min pH/d	53.9	100.5	29.3	0.35
Fecal pH	6.46	6.34	0.07	0.31

Kjeldahl procedure (AOAC, 1990), ADF (AOAC, 1990), NDF (Goering and Van Soest, 1970), crude fat (AOAC, 1990), ash (AOAC, 1990), and gross energy using a C-5000 calorimeter (IKA Analysetechnik, Heitersheim, Germany).

Statistical Analysis

The ANOVA was conducted using the SAS general linear models procedure (SAS, 1990) using the following general model:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + \tau_k + \varepsilon_{ijk},$$

where

- Y_{ijk} = the dependent variable,
- μ = overall mean,
- α_i = effect of cow ($i = 1, 2, 3, 4, 5, 6$),
- β_j = effect of period ($j = 1, 2$),
- τ_k = effect of treatment ($k = 1, 2$), and
- ε_{ijk} = random residual error.

For dependent variables that had repeated measurements (ruminal pH), the repeated measurement option within the SAS (1990) GLM procedure was used. Effects were considered significant at a probability $P < 0.05$, unless otherwise indicated.

RESULTS AND DISCUSSION

SARA and Ruminal Forage Degradability and Total Tract Diet Digestibility

Data on ruminal pH characteristics during SARA are presented in Table 2. The minimum amount of time per day that ruminal pH was below 6 (≥ 9 h) and 5.6 (≥ 3 h), and minimum ruminal pH values of 5.27 to 5.39 (Table 2) that were observed for both control and monensin-treated cows during the grain challenge indicate that cows experienced prolonged periods of low ruminal pH that are indicative of SARA. This is consis-

tent with the findings of other researchers (see Keunen et al., 2002; Mutsvangwa et al., 2002) who used a similar grain challenge model to induce SARA. Mean ruminal pH, amount of time per day that pH was below 6 and 5.6, and the area (time $\times$ pH) that pH was below 6 and 5.6 were not different ($P > 0.05$) between control and monensin-treated cows during SARA (Table 2). This is in agreement with a previous study from our laboratory (Mutsvangwa et al., 2002), indicating that monensin was not efficacious in increasing ruminal pH under the SARA conditions used in this study. Supplementation with monensin had no effect ($P > 0.05$) on mean fecal pH during SARA (Table 2), and this is consistent with the lack of effect of monensin on ruminal pH.

Other studies (Green et al., 1999) have reported that monensin increased ruminal pH in periparturient dairy cows. Monensin increases ruminal pH primarily by inhibiting lactate-producing bacteria, which would otherwise proliferate in abundant starchy conditions. This reduction in ruminal lactate concentration, which is the major "driver" of acidosis when high grain diets are fed, raises ruminal pH (Russell and Hino, 1985; Callaway and Martin, 1997). In feedlot experiments in which monensin was potent in preventing extreme drops in ruminal pH following consumption of readily fermentable carbohydrates, ruminal lactate concentrations were generally high, exceeding 5 mM (Owens et al., 1998). We did not measure ruminal lactate concentrations in the present study; however, in the nutritional model employed in this study to induce SARA, ruminal lactate concentration does not exceed 1 mM (Plaizier et al., unpublished data), and this level is much lower than the threshold of 5 mM above which monensin has been reported to increase ruminal pH (Owens et al., 1998; Oetzel et al., 1999). For this reason, we can surmise that SARA in our experimental cows was not lactate-induced, and ruminal pH declined primarily as a function of VFA concentrations and, therefore, the inhibitory effect of monensin on lactate production would have played only a minor role in the regulation of ruminal pH in the current study.

Table 3. Ruminal degradability of DM and NDF for corn silage and haylage during subacute ruminal acidosis (SARA) in lactating Holstein dairy cows as affected by supplementation with a monensin premix.

Item	Monensin	Control	SE	P Value
DM degradability				
Corn silage				
A + B, ¹ %	80.9	83.7	3.0	0.56
B, ² %	30.7	34.7	4.3	0.56
c, ³ % h ⁻¹	2.1	2.4	0.2	0.40
24-h degradability, %	61.0	63.1	0.9	0.21
48-h degradability, %	69.0	73.0	2.0	0.27
Haylage				
A + B, %	72.4	71.8	0.6	0.53
B, %	39.6	38.7	1.0	0.59
c, % h ⁻¹	3.3	3.8	0.3	0.39
24-h degradability, %	53.7	56.2	1.9	0.43
48-h degradability, %	66.1	68.7	2.3	0.48
NDF degradability				
Corn silage				
A + B, %	49.6	54.2	3.5	0.42
B, %	43.7	47.2	3.9	0.58
c, % h ⁻¹	2.2	2.3	0.1	0.50
24-h degradability, %	16.7	21.3	1.9	0.20
48-h degradability, %	33.5	42.3	5.2	0.32
Haylage				
A + B, %	59.2	60.8	0.4	0.07
B, %	49.3	49.5	0.5	0.77
c, % h ⁻¹	3.2	3.3	0.3	0.85
24-h degradability, %	33.9	36.9	2.5	0.46
48-h degradability, %	51.5	55.3	4.1	0.56

¹A + B = Potential degradability; A = washing losses.

²B = Potential degradability minus washing loss.

³c = Degradation rate constant.

The primary objective of this study was to determine the effects of monensin supplementation during grain-induced SARA on ruminal forage degradability and total tract diet digestibility. Several *in vivo* studies have investigated the effects of monensin supplementation (Muntifering et al., 1981; Haimoud et al., 1995; Plaizier et al., 2000) or grain-induced SARA (Krajcarski-Hunt et al., 2002; Plaizier et al., 2002) on ruminal forage degradability or total tract digestibility; however, to our knowledge, this is the first study to investigate the effects of monensin supplementation during grain-induced SARA on ruminal forage degradability and total tract digestibility. In the present study, monensin supplementation did not affect ($P > 0.05$) 24- or 48-h ruminal DM and NDF disappearance when haylage or corn silage was incubated in the rumen during SARA (Table 3). Because monensin has been reported to attenuate SARA in both beef cattle (Nagaraja et al., 1985; Cooper and Klopfenstein, 1996) and dairy cattle (Green et al., 1999), we anticipated that dietary supplementation with monensin in dairy cows subjected to SARA would make the ruminal environment more favorable for the proliferation of cellulolytic bacteria by increasing ruminal pH, thereby reducing the characteristic decrease in fiber digestion that has been observed during SARA (Krajcarski-Hunt et al., 2002; Plaizier et al., 2002).

Somewhat surprisingly, ruminal fiber digestion was unaffected by the addition of monensin (Table 3). All indicators of SARA that we measured in the present study, including mean ruminal pH, minimum ruminal pH, and duration of time when ruminal pH < 6 and < 5.6, were not different between control and monensin-treated cows (see Table 2), suggesting that monensin was ineffective in attenuating SARA. In addition, average ruminal pH during SARA, i.e., 6.07 and 6.11 for control and monensin-treated cows, respectively, indicate that only a mild ruminal acidosis might have been induced, likely driven by higher ruminal VFA concentrations, rather than higher ruminal lactate concentrations. Ruminal fiber digestion is depressed when ruminal pH declines below 6.2 (Grant and Mertens, 1992), and the average ruminal pH values observed in the present study for both control and monensin-treated cows were fairly close to this threshold ruminal pH. It is plausible, therefore, that ruminal pH conditions might not have been detrimental to fiber digestion in the present study. For these reasons, any potential benefits of monensin supplementation on ruminal fiber digestion might be expected to be small.

In the present experiment, cows were under conditions of experimentally induced SARA (albeit mild) and, for that reason, any direct comparisons of the effects of

Table 4. Total tract apparent diet digestibilities during subacute ruminal acidosis in Holstein dairy cows as affected by supplementation with a monensin premix.

Item	Monensin	Control	SE	<i>P</i> Value
Apparent digestibility, %				
DM	65.5	62.2	1.4	0.19
CP	66.0	62.8	1.3	0.19
NDF	54.8	45.8	2.6	0.09
ADF	51.4	44.1	1.7	0.05
Crude fat	78.0	75.9	2.7	0.62
Ash	37.9	33.5	2.2	0.26
NFC	75.7	76.8	1.3	0.59
GE	65.4	61.6	1.2	0.10

monensin on diet digestion with published studies in which monensin was supplemented under “normal” feeding conditions might be somewhat misleading. However, the average ruminal pH observed in the present study was > 6 , similar to average ruminal pH of 6 observed for control and monensin-treated cows in the study by Haïmond et al. (1995), so direct comparisons with the present study are valid. In contrast with results from our study, Haïmond et al. (1995) observed that monensin supplementation altered dietary nutrient digestion by decreasing ruminal fiber digestion; however, postruminal (small intestinal) fiber digestion was higher with monensin supplementation, such that total tract fiber digestion was not changed. Monensin, therefore, shifted the site of fiber digestion from the rumen to postruminal sites. In the present study, monensin did not affect ruminal fiber digestion (Table 3), but it increased ($P = 0.05$) total tract ADF digestion and tended to increase ($P = 0.09$) total tract NDF digestion (Table 4). We did not measure small intestinal fiber digestion in the present study, but we can surmise from our results that dietary addition of monensin increased postruminal fiber digestion, thereby increasing total tract fiber digestion even though ruminal fiber digestion was unaffected. Perusal of the literature indicates that the influence of monensin on ruminal or postruminal nutrient digestion has not been consistent. Monensin supplementation reduced ruminal nutrient digestion in some studies with cattle or sheep fed concentrate or

forage diets (Owens et al., 1978; Simpson, 1980; Muntifering et al., 1981), but not in others (Morris et al., 1990; Rogers et al., 1991). Reasons for these discrepancies between studies may include differences in dietary inclusion levels of monensin, and interactions between feed intake and composition, and monensin (Mutsaers et al., 2002). Total tract digestion of DM, CP, crude fat, ash, nonfiber carbohydrates, and gross energy were unaffected ($P > 0.05$) by the dietary addition of monensin (Table 4).

Feed Intake and Milk Production

Monensin supplementation did not affect ($P > 0.05$) DMI and milk yield under experimentally induced SARA conditions (Table 5). Considerable research has been devoted to determining the effects of monensin on DMI and milk yield in dairy cows. A preponderance of data indicate that the well-documented effects of adding monensin to dairy cow diets have been increased milk production in conjunction with no change or a decrease in DMI (van der Werf et al., 1998; Phipps et al., 2000; Ruiz et al., 2001), thereby suggesting that monensin improved the efficiency of feed utilization. However, in the current study dairy cows were under conditions of experimentally induced SARA and direct comparisons with published studies in which monensin was supplemented under “normal” feeding conditions would be somewhat misleading. In previous research

Table 5. Dry matter intake, and milk production and composition of Holstein dairy cows during subacute ruminal acidosis (SARA) as affected by supplementation with a monensin premix.

Item	Monensin	Control	SE	<i>P</i> Value
DMI, kg/d	17.5	16.9	1.3	0.77
Milk yield, kg/d	25.6	25.2	1.2	0.84
Fat, %	3.26	3.78	0.25	0.24
Protein, %	3.09	3.32	0.06	0.09
Lactose, %	4.61	4.60	0.10	0.96
Fat, kg/d	0.82	0.94	0.06	0.23
Protein, kg/d	0.78	0.82	0.05	0.62
Lactose, kg/d	1.19	1.16	0.08	0.79

from our laboratory in which SARA was induced by feeding additional grain (see Mutsvangwa et al., 2002), we observed that cows fed a monensin premix ate more DM (+1.5 to +2.8 kg/d) and, consequently, produced more milk (+3.6 to +4.0 kg/d) compared with control cows. Reasons for discrepancies between that previous study and the present study are unclear but could be related to differences in stage of lactation between the 2 studies. However, the lack of response in DMI and milk production due to monensin supplementation that we observed in the present study is in agreement with earlier trials (Lean et al., 1994; Hayes et al., 1996; Green et al., 1999; Vallimont et al. 2001).

Treatment with monensin had no effect ($P > 0.05$) on milk content of fat and protein, and milk fat and protein yields (Table 5). However, milk fat content in monensin-treated cows was 0.52 percentage units lower than control cows (Table 5). The magnitude of the decrease in milk fat content due to monensin supplementation was higher than that reported to be significant in other studies (e.g., 0.26 to 0.32 percentage units reported by Mutsvangwa et al., 2002). Differences in milk fat content in the present study were not significant, likely because standard errors were larger compared with the study of Mutsvangwa et al. (2002), i.e., 0.25 vs. 0.04 to 0.06. However, the observed trends in decreasing milk fat content due to the administration of monensin have been reported in other studies (van der Werf et al., 1998; Phipps et al., 2000; Vallimont et al., 2001). Milk contents and yields of protein were unaffected ($P > 0.05$) by dietary supplementation with monensin (Table 6), and this is in agreement with other studies (van der Werf et al., 1998; Green et al., 1999; Ruiz et al., 2001; Mutsvangwa et al., 2002).

CONCLUSIONS

In summary, monensin premix supplementation had no effect on ruminal pH characteristics, indicating that monensin was not efficacious in attenuating SARA. In addition, adding monensin to the diet did not affect ruminal fiber digestion during SARA, likely because only a mild ruminal acidosis was induced and, therefore, ruminal pH conditions might not have been detrimental to fiber digestion. However, total tract fiber digestion was increased by adding monensin to the diet, suggesting that monensin supplementation increased fiber digestion at post-ruminal sites. These results suggest that monensin might be a potential tool for improving nutrient digestion during grain-induced SARA in dairy cows.

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