Effects of a Monensin Controlled-Release Capsule or Premix on Attenuation of Subacute Ruminal Acidosis in Dairy Cows

T. Mutsvangwa,* J. P. Walton,* J. C. Plaizier,§ T. F. Duffield,†
R. Bagg,‡ P. Dick,‡ G. Vessie,‡ and B. W. McBride*

*Department of Animal and Poultry Science,

University of Guelph,
Guelph, ON, Canada N1G 2W1;

§Department of Animal Science,
University of Manitoba,

Winnipeg, MB, Canada R3T 2N2;

†Department of Population Medicine,
University of Guelph; and

‡Elanco Animal Health/Provel Division, Eli Lilly Canada Inc.,

Research Park Centre,

Guelph, ON, Canada N1G 4T2

ABSTRACT

The effects of monensin, administered either as a controlled release capsule (CRC) or a premix, on attenuating grain-induced subacute ruminal acidosis (SARA) and on ruminal fermentation characteristics in Holstein cows receiving a total mixed ration were investigated in two experiments. In both experiments, six multiparous, rumen-fistulated Holstein cows were used in a two-treatment, two-period crossover design with 6-wk periods. In Experiment 1, treatments were either a monensin CRC or a placebo CRC. In Experiment 2, treatments were either a monensin premix or a placebo premix. In both experiments, at the beginning of wk 4 SARA was induced in experimental cows for a 10-d period with a grain challenge model, and ruminal pH was measured continuously using indwelling pH probes. The administration of monensin either as a CRC or a premix had no effect on ruminal pH characteristics. Neither monensin CRC nor premix had an effect on ruminal volatile fatty acid concentrations, but reduced the acetate:propionate ratio. Monensin premix-treated cows were observed to have increased milk yield, largely as a result of a higher dry matter intake in monensin-treated cows compared to control cows. Milk fat content and yield were lower in monensin-treated cows compared to placebo-treated cows during SARA. In conclusion, there is no evidence that monensin was efficacious in raising ruminal pH during SARA under the conditions employed in this study.

(**Key words:** dairy cow, monensin, ruminal acidosis, milk production)

Abbreviation key: CRC = controlled-release capsule, SARA = subacute ruminal acidosis.

INTRODUCTION

In early lactation, high-yielding dairy cows cannot consume enough DM to meet nutrient demands for rapidly increasing milk production (NRC, 1989). To meet these nutrient demands, cows are often changed from a high-fiber diet to an energy-dense diet that is higher in grain and lower in fiber. This rapid change to a diet that is high in readily fermentable carbohydrates can result in a decrease in ruminal pH due to the accumulation of VFA and lactate, resulting in ruminal acidosis (Owens et al., 1998), which can be acute or subacute. In acute ruminal acidosis, a rapid increase in total acid (i.e., VFA + lactate) supply from the rumen (Owens et al., 1998), leads to a drop in ruminal pH, and a ruminal pH of 5.2 is generally considered a threshold (Owens et al., 1998). Cows with acute ruminal acidosis will exhibit clinical signs of illness, including blood acidosis, leg stiffness, laminitis, ruminal lesions, and in some cases death (Nocek, 1997). In subacute ruminal acidosis (SARA), cows do not typically exhibit any clinical signs of illness; however, feed intake and milking performance are often depressed. As in acute acidosis, ruminal pH drops during SARA, and usually lies between 5.2 and 5.6 (Owens et al., 1998).

Monensin, an ionophore, has been reported to have a variety of beneficial effects in ruminants. Monensin exerts its effects primarily by selectively altering the balance of ruminal microbes (Bergen and Bates, 1984). Monensin supplementation in ruminant diets has been reported to increase ruminal production of propionate

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Corresponding author: B. W. McBride; e-mail: bmcbride@uoguelph.ca.

(Bergen and Bates, 1984; Weiss and Amiet, 1990), reduce methanogenesis (Armentano and Young, 1983; Schelling, 1984), increase diet digestibility (Schelling, 1984; Plaizier et al., 2000), reduce ruminal degradation of dietary protein and increase flow of AA to post-ruminal sites (Russell and Strobel, 1989; Yang and Russell, 1993), and increase N retention (Schelling, 1984; Plaizier et al., 2000). In early-lactating dairy cows, monensin reduced blood concentrations of β -hydroxybutyrate (BHB) and increased blood glucose concentrations (Duffield et al., 1998a, Green et al., 1999), and this was associated with a reduced incidence of subclinical ketosis (Duffield et al., 1998b). In several studies with dairy cows, monensin reduced DMI (Ramanzin et al., 1997; Sauer et al., 1998; Van der Werf et al., 1998) and increased milk production (Hayes et al., 1996; Van der Werf et al., 1998; Duffield et al., 1999), suggesting that monensin might increase the efficiency of feed utilization for milk production. Some studies (Sauer et al., 1998; van der Werf et al., 1998) have observed that monensin decreased milk fat content in dairy cows, while others (Duffield et al., 1999; Green et al., 1999) have not. Milk contents of protein and lactose were not affected by monensin (van der Werf et al., 1998; Duffield et al., 1999).

In previous experiments with beef cattle consuming high-grain diets, monensin reduced the characteristic post-prandial decline in ruminal pH (Nagaraja et al., 1985; Burrin and Britton, 1986). In a study by Cooper and Klopfenstein (1996) in which they monitored ruminal pH and feed intake continuously in beef steers on high grain finishing diets, monensin was efficacious in elevating average ruminal pH and decreasing the area of ruminal pH below 5.6. Few trials have evaluated the effects of monensin on ruminal pH in dairy cows. Green et al. (1999) reported that monensin increased ruminal pH in transition dairy cows, but other workers (Haimoud et al., 1995; Ruiz et al., 2001) did not detect an increase in ruminal pH in monensin-treated dairy cows. A shortcoming with these studies was that ruminal pH was measured in spot samples obtained via ruminal cannula or by stomach tube at various intervals after feeding, and this is not necessarily representative. Additionally, there was the possibility of salivary contamination with stomach tubing, which might have elevated ruminal fluid pH in the study by Green et al. (1999). The objectives of this study, therefore, were to determine the efficacy of monensin, administered either as a controlled-release capsule (CRC) or as a premix, on attenuation of experimentally induced SARA, and its effects on ruminal fermentation characteristics in Holstein dairy cows.

MATERIALS AND METHODS

Experiment 1

Animals and experimental design. Six multiparous Holstein dairy cows (630.5 ± 35.4 kg BW; 81.2 ± 49.6 DIM) were used in this study. The experiment was run as a two-treatment, two-period crossover design with 6-wk periods. At the beginning of the experiment, cows were randomly assigned to either a monensin CRC ($n = 3$) or a placebo CRC ($n = 3$). After the first experimental period, treatments were then switched. The experiment was conducted in the Physiology Wing at the Elora Dairy Research Center (University of Guelph, Guelph, ON) from February through May 2001. 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.

Experimental treatments and feeding. Experimental treatments were a monensin CRC (Rumensin CRC; Provel, Division Eli Lilly Canada Inc., Guelph, ON) or a placebo CRC. The monensin CRC contained 32 g of monensin sodium blended into a hexaglycerol distearate matrix core. According to the manufacturer, the monensin CRC delivered approximately 335 mg of monensin on a daily basis. The placebo CRC was identical to the monensin CRC except that it did not contain monensin sodium in the core. The monensin and placebo CRC were administered via the rumen cannula at the beginning of the first experimental period and then removed at the end of the first recovery period. Treatments were switched at the beginning of the second experimental period when new monensin and placebo CRC were administered via the rumen cannula of each cow. For the initial 21 d, experimental animals were fed a TMR ad libitum twice daily at 0700 and 1300 h. Diet composition and chemical analysis are shown in Table 1. Individual cow feed intake was recorded daily during the experimental period. The 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), and for ADF (AOAC, 1990), NDF (Goering and Van Soest, 1970), and CP using the macro-Kjeldahl procedure (AOAC, 1990).

Induction of SARA and continuous measurement of ruminal pH. Between d 22 and d 32 of each experimental period, SARA was induced using a nutritional model developed by Keunen et al. (2000). This experimental model consisted of restricting the intake of TMR to 85% of ad libitum intake (as determined in the preceding 7-d period) and substituting 15% grain pellets consisting of 50% wheat and 50% barley. The feeding schedule during this period was as follows: 1)

Table 1. Ingredient and chemical composition of the TMR fed to lactating dairy cows in Experiment 1.

Ingredient	%, as fed basis
Corn silage	51.7
High-moisture corn	14.9
Mixed haylage	13.1
Mixed hay	4.4
Lactating cow supplement ¹	15.9
Chemical composition	
DM, %	49.9
	% of DM
CP (N × 6.25)	17.9
ADF	20.3
NDF	38.7
Ca	0.79
P	0.44
K	1.56
Mg	0.36
Na	0.16
NE _L , ² Mcal/kg	1.57

¹Contained (DM basis): 35.1% CP, 85.5% RUP (% of CP), 14.5% RDP (% of CP), 2.13% Ca, 0.73% P, 0.82% Mg, 2.02% K, 0.61% S, 1.09% Na, 0.72% Cl, 257.9 mg/kg Fe, 365.2 mg/kg Zn, 67.6 mg/kg Cu, 177.5 mg/kg Mn, 0.97 mg/kg Se, 0.87 mg/kg Co, 3.23 mg/kg I, 33,380 IU/kg of vitamin A, 13,350 IU/kg of vitamin D, 120.2 IU/kg of vitamin E.

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

At 0700 h, cows were fed 2 kg of the TMR; 2) At 0900 h, cows were fed two-thirds 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 remainder (one-third) of their daily grain pellet allowance; 5) At 1500 h, cows were offered TMR for 30 min; and 6) At 1700 h, cows were fed the remainder of the daily allowance of TMR. Cows were allowed 30 min for complete consumption of the grain pellets after which any grain pellets not consumed were directly introduced into the rumen via the rumen cannulae. Days 33 to 42 were considered a recovery period during which cows were maintained on treatment and had ad libitum access to the TMR. The rationale for including a recovery period was that, assuming that we observed significant effects of monensin on ruminal pH characteristics during SARA, we wanted to determine if monensin treatment would improve the prognosis for recovery. Ruminal pH was recorded on a continuous basis using indwelling pH probes during the SARA and recovery periods as described by Cumby et al. (2001). A pH reading was taken every second, averaged over every 60 s, and then stored. The position of the pH electrode was checked daily in each cow, and the pH electrodes and pH transmitters were calibrated with pH 4 and 7 buffer solutions (Fisher Scientific, Fairlawn, NJ) once weekly. The continuous ruminal pH data were summarized for each 24-h period by calculating the mean pH, the amount of time below pH 6 and 5.6, and the area (time × pH) below pH 6 and 5.6.

Sample collection. Experimental cows were milked twice daily at 0500 and 1500 h, and milk weights were recorded. During the 10-d SARA and recovery periods, 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, Hillerod, Denmark). Urea N was assayed colorimetrically using the diacetyl monoxime method (Sigma kit no. 535; Sigma Chemical Co., St. Louis, MO). On d 24 and 31 of the experimental period, approximately 300 ml of ruminal fluid was collected at 0900, 1100, 1300, 1500, 1700, and 1900 h from the ventral sac through the cannula using a probe and a vacuum pump. Ruminal fluid samples were centrifuged at 4000 × g, supernatants obtained, and then stored frozen at -70°C until later analysis. Ruminal fluid VFA concentrations were determined following protein precipitation using the copper tungstate method on a Varian GC3400 using an 8000 autosampler and a Varian 654 data system (Varian Canada, Mississauga, ON, Canada). A fused silica capillary column was used (15 m × 0.53 mm, i.d.; Nukol; Supelco Canada Ltd., Mississauga, ON, Canada). The injection and ionization temperature was 200°C and was reached by gradually increasing the temperature at a rate of 5°C/min with a holding time of 1 min.

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

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

where

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

For dependent variables that had repeated measurements (i.e., ruminal VFA measurements), 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.

Table 2. Ingredients and chemical composition of the total mixed rations fed to lactating dairy cows in Experiment 2.

Ingredient	Composition	
	Placebo	Monensin
	— (% , as fed basis) —	
Corn silage	39.8	39.8
High-moisture corn	17.6	17.6
Mixed haylage	27.6	27.6
Mixed hay	3.0	3.0
Lactating cow supplement ¹	11.8	11.8
Chemical composition		
DM, %	47.1	47.6
	— (% of DM) —	
CP (N × 6.25)	17.7	17.1
ADF	23.1	23.4
NDF	37.9	39.4
Ca	0.83	0.84
P	0.39	0.38
K	1.71	1.76
Mg	0.36	0.33
Na	0.15	0.14
NE _L , ² Mcal/kg	1.52	1.50

¹Contained (DM basis): 35.1% CP, 85.5% RUP (% of CP), 14.5% RDP (% of CP), 2.13% Ca, 0.73% P, 0.82% Mg, 2.02% K, 0.61% S, 1.09% Na, 0.72% Cl, 257.9 mg/kg Fe, 365.2 mg/kg Zn, 67.6 mg/kg Cu, 177.5 mg/kg Mn, 0.97 mg/kg Se, 0.87 mg/kg Co, 3.23 mg/kg I, 33,380 IU/kg of vitamin A, 13,350 IU/kg of vitamin D, 120.2 IU/kg of vitamin E.

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

Experiment 2

Six multiparous Holstein dairy cows (667.7 ± 46.2 kg BW; 150.2 ± 53.2 DIM) were used in this study. Experimental design, cow feeding management, induction of SARA and continuous measurement of ruminal pH, sample collection, and statistical analysis of data were all similar to Expt. 1. Experimental treatments were a monensin premix (Rumensin; Elanco, Division Eli Lilly Canada Inc., Guelph, ON) or a placebo premix. In the monensin premix, monensin was incorporated into soybean hulls, which acted as the carrier. The placebo premix was identical to the monensin premix except that it did not contain monensin sodium. Both the monensin and placebo premixes were mixed with the TMR daily to achieve a rate of 22 mg/kg of monensin (DM basis). Diet composition and chemical analysis are shown in Table 2. The experiment was conducted in the Physiology Wing at the Elora Dairy Research Center (University of Guelph, Guelph, ON) from August through November 2001.

RESULTS AND DISCUSSION

Continuous pH Measurements and Ruminal Fermentation Characteristics

The primary objective of this study was to determine the efficacy of monensin in attenuating SARA in dairy

Table 3. Ruminal pH characteristics during subacute ruminal acidosis (SARA) and recovery periods in Holstein dairy cows as affected by supplementation with a monensin controlled release capsule in Experiment 1.

Item	Control	Monensin	SE	P value
SARA period				
Mean pH	6.17	6.20	0.03	0.68
Minimum pH	5.25	5.14	0.07	0.34
Maximum pH	6.89	6.94	0.07	0.65
Minutes pH <6, min/d	406.0	450.0	24.1	0.27
Area pH <6, min pH/d	129.4	152.7	9.9	0.17
Minutes pH <5.6, min/d	128.0	161.6	13.3	0.15
Area pH <5.6, min pH/d	30.4	34.8	3.2	0.39
Recovery period				
Mean pH	6.24	6.26	0.03	0.71
Minimum pH	5.32	5.51	0.15	0.41
Maximum pH	6.84	6.85	0.08	0.93
Minutes pH <6, min/d	341.8	405.6	70.4	0.56
Area pH <6, min pH/d	96.7	89.6	10.9	0.67
Minutes pH <5.6, min/d	91.7	52.0	17.9	0.19
Area pH <5.6, min pH/d	21.2	5.9	7.7	0.23

cows, an area that has received little attention. In previous studies that reported the effects of monensin on ruminal pH in dairy cows, a drawback was that ruminal pH was measured in spot samples obtained via ruminal cannula (Ruiz et al., 2001) or by stomach tube (Green et al., 1999) at various intervals after feeding, and this might not necessarily be representative. In addition, there is the possibility of salivary contamination in the study (Green et al. 1999) that could have elevated ruminal fluid pH. A methodological improvement in the current study is that ruminal pH was measured continuously during a 24-h feeding cycle for 10 d, thereby yielding more comprehensive data on diurnal profiles of ruminal pH. Additionally, this allowed calculation of duration of time (min) and area (min × pH/d) when ruminal pH was below certain thresholds, thereby providing a more accurate measure of ruminal acidosis. Data on ruminal pH characteristics during the grain challenge and recovery periods are presented in Tables 3 and 4 for Expt. 1 and 2, respectively. The minimum amount of time per day that pH was below 6 (≥ 7 h) and 5.6 (≥ 2 h) during the grain challenge indicates that cows in both experiments experienced prolonged periods of low ruminal pH that are indicative of SARA. Due to the difficulty involved in obtaining ruminal fluid samples for diagnosis of SARA under field conditions, limited information is available in the literature on the prevalence of this digestive disorder in commercial dairy herds (Garrett et al., 1999). For this reason, it is somewhat difficult to assess how closely the SARA conditions experienced in our study simulate field conditions. In a field study conducted by Oetzel et al. (1999) in 14 Holstein dairy herds feeding typical dairy diets in Wisconsin, 20.1% of the cows were detected to have ruminal pH less than or equal to 5.5 (the benchmark

Table 4. Ruminal pH characteristics during subacute ruminal acidosis (SARA) and recovery periods in Holstein dairy cows as affected by supplementation with a monensin premix in Experiment 2.

Item	Control	Monensin	SE	<i>P</i> value
SARA period				
Mean pH	6.15	6.12	0.02	0.45
Minimum pH	5.33	5.23	0.05	0.22
Maximum pH	6.81	6.83	0.02	0.43
Minutes pH <6, min/d	415.0	461.0	31.1	0.36
Area pH <6, min pH/d	126.8	144.2	15.9	0.48
Minutes pH <5.6, min/d	123.7	154.2	22.4	0.39
Area pH <5.6, min pH/d	25.1	29.1	4.7	0.58
Recovery period				
Mean pH	6.24	6.22	0.07	0.85
Minimum pH	5.48	5.39	0.13	0.66
Maximum pH	6.91	6.84	0.07	0.49
Minutes pH <6, min/d	323.4	329.4	67.4	0.95
Area pH <6, min pH/d	112.5	79.9	36.9	0.57
Minutes pH <5.6, min/d	131.5	67.8	53.5	0.44
Area pH <5.6, min pH/d	33.2	11.9	12.9	0.31

that is indicative of SARA). In the current study, ruminal pH was less than or equal to 5.6 for prolonged periods, and minimum ruminal pH attained ranged from 5.14 to 5.33 during SARA. This indicates that we achieved a simulation of SARA that is typical of dairy herds under practical conditions.

Mean 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 affected ($P > 0.05$) by monensin during both the grain challenge and recovery periods in the two experiments, indicating that monensin was not efficacious in increasing ruminal pH under the dietary conditions used in this study. Green et al. (1999) reported that monensin increased ruminal pH in periparturient dairy cows. Monensin inhibits lactate-producing bacteria, which proliferate in abundant starchy conditions, and a reduction in ruminal lactate concentration 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). However, in the nutritional model employed in this study to induce SARA, ruminal lactate concentration is less than 0.02 mM (see Keunen et al., manuscript accepted), 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 rumi-

Table 5. Ruminal pH and VFA characteristics during grain-induced subacute ruminal acidosis in Holstein dairy cows as affected by supplementation with a monensin controlled release capsule (CRC) or premix in Experiments 1 and 2.¹

Item	Control	Monensin	SE	<i>P</i> value
CRC (Expt. 1)				
VFA, mM				
Acetate (A)	105.0	97.2	2.7	0.10
Propionate (P)	39.1	41.0	1.7	0.48
Butyrate	11.7	10.9	0.3	0.12
Isobutyrate	0.9	0.8	0.03	0.10
Valerate	2.1	1.9	0.09	0.36
Isovalerate ²	1.5	1.4	0.04	0.10
Total	160.2	153.2	4.4	0.33
A:P	3.18	2.72	0.09	0.02
Premix (Expt. 2)				
VFA, mM				
Acetate (A)	109.5	107.7	1.8	0.53
Propionate (P)	36.8	39.6	1.4	0.23
Butyrate	12.7	13.5	0.9	0.53
Isobutyrate	0.8	0.8	0.04	0.88
Valerate	2.1	1.7	0.1	0.08
Isovalerate	1.5	1.5	0.1	0.88
Total	163.4	164.8	1.6	0.56
A:P	3.29	2.83	0.11	0.04

¹Ruminal fluid spot samples were obtained at 0 (0900 h, grain pellet feeding), 2, 4, 6, 8, and 10 h.

²3-Methyl-butanolic acid.

nal pH in the current study. The lack of a significant effect of monensin on ruminal pH observed in the present study agrees with the results of Ruiz et al. (2001).

Mean ruminal VFA concentrations are shown in Table 5 for Expt. 1 and 2. In both experiments, monensin had no effect on ruminal VFA concentrations ($P > 0.05$); however, numerically, monensin decreased ruminal acetate and increased propionate concentrations. Both monensin CRC and premix reduced the acetate-to-propionate ratio, and this is in agreement with results reported by Green et al. (1999) and Ruiz et al. (2001) in dairy cows. Several studies have reported a characteristic shift in rumen fermentation, as demonstrated by changes in VFA pattern, in beef steers (Richardson et al., 1976), and dairy cows (Sauer et al., 1989). In these studies, animals treated with monensin had consistently higher ruminal concentrations of propionate, but changes in ruminal concentrations of acetate and butyrate were not consistent. These findings on ruminal VFA patterns are in agreement with the study by Green et al. (1999) with transition dairy cows fed similar diets as those fed in this study. Other studies have reported that monensin premix does not affect total VFA levels in lactating dairy cows (Haimoud et al., 1995), and in beef steers (Muntifering et al., 1980; Armentano and Young, 1983). However, a decrease in total VFA levels in vitro (Whetstone et al., 1981) and in beef steers (Lemaguer et al., 1978) has also been reported. Reasons for these discrepancies between studies may include differ-

Table 6. DMI, milk production, and milk composition of Holstein dairy cows as affected by supplementation with monensin controlled release capsule (CRC) in Experiment 1.

Item	Control	Monensin	SE	P value
Day 22 to 32 (SARA)				
DMI, kg/d	22.2	22.1	0.2	0.65
Milk yield, kg/d	37.0	35.7	0.6	0.20
Fat, %	3.29	3.03	0.06	0.04
Protein, %	3.10	3.13	0.04	0.64
Lactose, %	4.85	4.85	0.02	0.80
Fat, kg/d	1.20	1.06	0.02	0.01
Protein, kg/d	1.15	1.12	0.01	0.06
Lactose, kg/d	1.80	1.73	0.03	0.22
Milk urea N, mg/dL	12.4	12.6	0.38	0.73
Day 33 to 42 (RECOVERY)				
DMI, kg/d	22.6	23.0	0.2	0.29
Milk yield, kg/d	35.7	36.4	0.9	0.62
Fat, %	3.54	3.22	0.13	0.20
Protein, %	3.14	3.17	0.04	0.61
Lactose, %	4.81	4.83	0.01	0.33
Fat, kg/d	1.23	1.15	0.04	0.29
Protein, kg/d	1.12	1.15	0.02	0.31
Lactose, kg/d	1.72	1.76	0.05	0.56
Milk urea N, mg/dL	12.9	13.2	0.4	0.55

ences in dietary inclusion levels of monensin, and interactions between feed intake and composition, and monensin.

Feed Intake and Milk Production

In Expt. 1, monensin CRC did not affect ($P > 0.05$) DMI or milk yield (Table 6). In Expt 2, monensin-treated cows consumed 1.5 to 2.8 kg/d more DM compared with control cows during the SARA and recovery periods, and this difference was significant ($P < 0.05$; Table 7). Concomitantly, monensin premix-treated

Table 7. DMI, milk production, and milk composition of Holstein dairy cows as affected by supplementation with a monensin premix in Experiment 2.

Item	Control	Monensin	SE	P value
Day 22 to 32 (SARA)				
DMI, kg/d	19.8	21.3	0.3	0.03
Milk yield, kg/d	27.0	31.0	0.9	0.04
Fat, %	3.38	3.18	0.04	0.03
Protein, %	3.48	3.37	0.06	0.22
Lactose, %	4.60	4.63	0.03	0.50
Fat, kg/d	0.88	0.98	0.04	0.12
Protein, kg/d	0.91	1.04	0.03	0.05
Lactose, kg/d	1.25	1.44	0.04	0.04
Milk urea N, mg/dL	10.2	10.9	0.56	0.41
Day 33 to 42 (RECOVERY)				
DMI, kg/d	20.9	23.7	0.4	0.01
Milk yield, kg/d	25.9	29.5	0.5	0.006
Fat, %	3.44	3.13	0.12	0.15
Protein, %	3.58	3.44	0.05	0.14
Lactose, %	4.62	4.66	0.03	0.42
Fat, kg/d	0.83	0.91	0.04	0.18
Protein, kg/d	0.90	0.99	0.02	0.02
Lactose, kg/d	1.20	1.37	0.02	0.01
Milk urea N, mg/dL	10.7	12.8	0.5	0.06

cows yielded 3.6 to 4 kg/d more milk compared to control cows, and this difference was significant ($P < 0.05$; Table 7). 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. However, the lack of response in DMI and milk production in CRC-treated cows is in agreement with earlier trials (Lean et al., 1994; Hayes et al., 1996; Green et al., 1999; Vallimont et al., 2001).

In Expt. 2, the likely mechanism for the increased milk yield observed was that monensin-treated cows ate more DM. Based on dietary analysis (Table 2), we calculated that the additional 1.5 to 2.8 kg of DMI would correspond with 2.3 to 4.2 Mcal of additional NE_L intake. In the present study, mean milk fat, protein, and lactose contents were 32.6, 33.8 and 46.1 g/kg milk for monensin-treated cows (Table 7), and if we assume caloric values of 9.3, 5.9, and 4.0 kcal/g of fat, protein, and lactose, respectively (Arieli et al., 2001), then the extra NE_L intake could correspond to an additional 1.5 to 2.9 kg/d of milk in monensin-treated cows. Monensin-treated cows did produce 2 to 4 kg/d more milk compared to placebo cows. The magnitude of this increase (7 to 14%) in milk production is similar to that recorded in previous studies using similar dose rates of monensin (Wade et al., 1996; Phipps et al., 2000). However, the milk yield response is higher compared with that of 4 to 6.5% generally reported in the literature (Van der Werf et al., 1998; Duffield et al., 1999; Ruiz et al., 2001). In Expt. 2, an interesting observation is that both DMI and milk yield were not affected by monensin during the initial 3-wk adaptation period (data not shown), and significant responses were only detected when SARA was imposed. We speculate that there was some unknown interaction between monensin and SARA. It is plausible that during SARA, monensin provided a prophylactic effect that allowed a more rapid adaptation to grain feeding, thereby minimizing or avoiding ruminal digestive disturbances. Additional research is needed to elucidate the effects of monensin on digestive function, specifically ruminal fiber degradation and total tract diet digestibility during SARA.

In Expt. 1, treatment with monensin consistently reduced milk fat content by 0.26 to 0.32 percentage units, and these effects were significant ($P = 0.04$) during SARA (Table 6). In Expt. 2, treatment with monensin reduced milk fat content by 0.20 to 0.31 percentage

units, and these differences were significant ($P = 0.03$) during SARA. The observed decrease in milk fat content due to the administration of monensin has been reported in other studies (Van der Werf et al., 1998; Phipps et al., 2000; Vallimont et al., 2001), but not others (Duffield et al., 1999; Green et al., 1999). In Expt. 1, treatment with monensin CRC reduced ($P = 0.01$) milk fat yield during SARA (Table 6). Even though monensin treatment reduced milk fat content in Expt. 2, milk fat yield was unaffected ($P > 0.05$; Table 7), primarily because monensin elevated milk yield. During the recovery period, milk fat content in control cows returned to pre-SARA levels (data not shown), whereas milk fat content remained depressed in monensin-treated cows, perhaps indicating that there is an additive effect of monensin and grain feeding on milk fat content. Further studies are needed to compare the effects of monensin and different forage-to-concentrate ratios on milk fat production.

In Expt. 1, milk contents and yields of protein were unaffected ($P > 0.05$) by monensin (Table 6). In Expt. 2, monensin treatment had no effect ($P > 0.05$) on milk protein content (Table 7); however, primarily because of higher milk yield in monensin-treated cows, milk protein yield was higher due to monensin treatment during SARA ($P = 0.05$) and recovery ($P = 0.02$) periods (Table 7). Phipps et al. (2000) reported that monensin treatment significantly reduced milk protein content in a study with a much larger sample size than the current study. However, in other studies (Van der Werf et al., 1998; Duffield et al., 1999; Green et al., 1999; Ruiz et al., 2001), monensin had no effect on milk protein content.

In Expt. 1, milk lactose content was unaffected ($P > 0.05$) by addition of monensin (Table 6). In Expt. 2, milk lactose content was unaffected ($P > 0.05$) by monensin treatment; however, milk lactose yield was higher in monensin-treated compared with control cows during the SARA ($P = 0.04$) and recovery ($P = 0.01$) periods (Table 7). This response was primarily due to higher milk production as a result of monensin treatment in Expt. 2. Other studies did not detect any effects of monensin on milk lactose content or yield (Green et al., 1999; Phipps et al., 2000; Vallimont et al., 2001). In both experiments, milk urea N was unaffected by treatment with monensin and this agrees with earlier studies (Vallimont et al., 2001).

CONCLUSIONS

In summary, the administration of monensin either as CRC or premix had no effect on ruminal pH characteristics under conditions of SARA. In addition, monensin CRC had no effect on milk production, but monensin

premix was observed to increase DMI and milk yield. Even though monensin did not increase ruminal pH during SARA, it is conceivable that there is an interaction between monensin and SARA that allowed monensin-treated cows to adapt faster to additional grain feeding, thereby maintaining DMI and milk yield. Additional research characterizing the effects of monensin on ruminal function, specifically fiber degradation, is needed to aid us in further understanding this interaction.

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