Effects of Prepartum Administration of a Monensin Controlled Release Capsule on Rumen pH, Feed Intake, and Milk Production of Transition Dairy Cows

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ABSTRACT

Effects of prepartum administration of a monensin controlled release capsule (CRC) on rumen pH, dry matter intake, and milk production during the transition period and early lactation were determined in 16 multiparous Holstein cows. Cows were divided into blocks of 2 depending on calving date. Cows were fed either a close-up dry cow or a lactating cow total mixed ration *ad libitum*. Rumen pH was monitored continuously using indwelling probes. Monensin did not affect average daily rumen pH, time below pH 6, time below pH 5.6, area below pH 6, and area below pH 5.6 throughout the experiment. Average daily pH, time below pH 6, and time below pH 5.6 before calving were 6.62, 65.6 min/d, and 17.6 min/d, respectively, and did not differ among the weeks before calving. Average daily pH, time below pH 6, and time below pH 5.6 were 6.19, 443.3 min/d, and 115.5 min/d, respectively, during the first week after calving, and were 6.36, 204.3 min/d, and 52.4 min/d, respectively, during the sixth week after calving. In the weeks after calving, average daily pH showed a quadratic increase, time below pH 6 showed a quadratic decrease, and time below pH 5.6 showed a linear decrease. Monensin did not affect dry matter intake and daily yields of milk, milk fat, and milk protein. Results suggest that prepartum administration of a monensin CRC did not increase rumen pH in multiparous cows fed the experimental diets during the transition period and early lactation.

Key words: transition dairy cow, monensin controlled release capsule, rumen pH, milk production

INTRODUCTION

A monensin controlled release capsule (CRC; Rumensin) has been approved in Canada as an aid in the

prevention of subclinical ketosis in lactating dairy cows. The monensin CRC has a positive effect on energy indicators, as it lowers blood BHBA and increases blood glucose in early lactation dairy cows (Duffield et al., 1998a; Green et al., 1999). This effect on energy indicators reduces the incidence of subclinical ketosis during this period (Duffield et al., 1998b). In herds at increased risk of ketosis, monensin increased projected 305-d milk production (Duffield et al., 1999a). Plaizier et al. (2000) have shown that the monensin CRC increases the apparent digestibility of nitrogen and improves the nitrogen balance in fresh cows.

Sauer et al. (1989) found that monensin reduced feed intake, without reducing milk production. Van der Werf et al. (1998) and Phipps et al. (2000) reported that monensin tended to reduce feed intake. The effect of monensin on milk production is variable. Sauer et al. (1989) found that addition of 15 ppm of monensin to the diet of dairy cows reduced milk fat percentage, whereas the addition of 30 ppm of monensin had no effect. Van der Werf et al. (1998) showed that monensin at a dose of 450 mg/d decreased milk fat percentage, but did not affect milk protein percentage. Duffield et al. (2003) found that supplementation with monensin in doses ranging from 9 to 23 mg/kg reduced milk fat in herds that received diets with a low nonstructural carbohydrate content (below 40.2% DM), but not in herds that received diets with a higher nonstructural carbohydrate content. Duffield et al. (2003), therefore, demonstrated an interaction between diet composition and the effect of monensin on milk fat percentage.

Monensin increases rumen pH and attenuates ruminal acidosis in cattle on high grain diets containing over 75% concentrate (Nagaraja et al., 1982; Burrin and Britton, 1986). In contrast, Mutsvangwa et al. (2002) monitored rumen pH of lactating dairy cows continuously using indwelling pH probes, and observed that administration of monensin as a premix at a dose of 22 mg/kg (DM basis) or as a CRC did not attenuate grain-induced subacute ruminal acidosis (SARA). Green et

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al. (1999) monitored rumen pH in transition dairy cows by collecting daily samples with a stomach tube, and found that the monensin CRC increased rumen pH in primiparous, but not in multiparous, transition cows. Due to the diurnal variation in rumen pH, continuous monitoring of rumen pH provides for a more accurate means of quantifying rumen conditions than a single daily measurement in rumen fluid. Therefore, at present, the effect of monensin on rumen pH of transition dairy cows remains ambiguous. The main objective of this study was, therefore, to determine if prepartum administration of a monensin CRC has effects on rumen pH that can be detected when using continuous monitoring of rumen pH in transition and early lactation dairy cows. Additional objectives were to determine the effects of prepartum administration of this CRC on DM intake and milk production in dairy cows between 3 wk before calving until 6 wk after calving and the changes in rumen pH during the transition period and early lactation.

MATERIALS AND METHODS

Experimental Procedures

Sixteen second- and third-lactation Holstein cows were blocked in pairs based on their expected calving dates. Rumen fistulations were carried out between 2 and 3 mo precalving (Duffield, 1999b). Cows within each block were randomly assigned to one of two treatments: a monensin controlled release capsule (Rumen-sin CRC Provel, Division Eli Lilly Canada Inc., Guelph, ON, Canada) or a placebo CRC (Provel). The monensin CRC contained 32 g of monensin sodium blended into a hexaglycerol distearate matrix core. The monensin CRC delivers (mean $\pm$ SD) 335 ± 33 mg/d of monensin for approximately 95 d. The placebo CRC was identical to the monensin CRC, but it contained no monensin sodium in the core. The CRC was administered approximately 3 wk before the expected calving date. Animal care and use procedures were approved by the University's Animal Care Committee and were conducted in accordance to the guidelines of the Canadian Council for Animal Care.

Cows entered the experiment between February 1998 and January 1999. Calving dates were evenly distributed throughout the experimental period. Approximately 4 wk before the expected calving date animals moved to the physiology wing of the Elora Dairy Research Station (EDRC), and were housed in individual tie stalls. Animals were fed a TMR ad libitum twice daily at 0700 and 1300 h. At calving, animals were switched from a close-up dry cow diet to a lactating cow

Table 1. Ingredient composition of the dry cow and lactating cow diets (SE in parentheses)

Ingredient	Close-up dry cow diet	Lactating cow diet
	(% of DM)	
Corn silage	28.4 (3.0)	24.0 (1.8)
High-moisture corn	6.5 (1.7)	22.0 (0.8)
Haylage	28.5 (2.2)	22.0 (1.1)
Hay	20.2 (4.0)	8.2 (1.3)
Soybean meal	5.1 (0.8)	11.6 (1.6)
Lactating cow supplement ¹	—	12.2 (1.4)
Dry cow pellets ²	11.3 (3.5)	—

¹Contained (on a DM basis) 20.61% CP, 85.47% undegraded protein (% of CP), 14.70% degraded protein (% of CP), 18.20% soluble protein (% of CP), 29.21% NDF, 20.13% NFC, 3.69% total fat, 4.38% Ca, 1.61% P, 1.45% Mg, 1.25% K, 0.92% S, 2.48% Na, 1.64% Cl, 302.21 ppm Fe, 919.79 ppm Zn, 158.63 ppm Cu, 420.83 ppm Mn, 2.46 ppm Se, 2.31 ppm Co, 8.88 ppm I, 92.40 kIU/kg vitamin A, 36.96 kIU/kg vitamin D, 332.63 IU vitamin E.

²Contained (on a DM basis) 22.74% CP, 80.92% undegraded protein (% of CP), 19.08% degraded protein (% of CP), 64% soluble protein (% of CP), 25.25% NDF, 36.19% NFC, 3.28% total fat, 4.84% NaHCO₃, 1.61% P, 0.79% Mg, 0.95% K, 0.84% S, 0.07% Na, 0.13% Cl, 143.44 ppm Fe, 163.45 ppm Zn, 31.45 ppm Cu, 109.02 ppm Mn, 1.35 ppm Se, 0.37 ppm Co, 1.23 ppm I, 1.23 kIU/kg vitamin A, 4.92 kIU/kg vitamin D, 760.14 IU vitamin E.

diet (Tables 1 and 2). Diets were adjusted 10 times during the experiment, which is reflected by the standard errors given in Table 1. For the first 3 wk after calving, cows also received 1.8 kg of mixed grass/alfalfa hay once daily. Cows had unlimited access to fresh water. Animals remained in the physiology wing until 1 to 2 d before calving, and then moved to a maternity pen until 3 d postcalving. Between d 25 and 42 after calving additional high-moisture corn (HMC) was offered in an attempt to lower rumen pH. To achieve this, the total mixed diet was removed from the cows at 1100 h and HMC was offered. The total mixed diet was reintroduced by 1300 h. If the HMC had not been eaten at that time, the remainder was placed into the rumen through the rumen fistula. The initial amount of HMC offered was 0.25 kg, but this amount was increased by increments of 0.25 kg until 2 kg was offered on d 39, 40, 41, or 42 after calving. On the subsequent day, no HMC was offered and cows were fed as normal. Two days after the initial offering of 2 kg of HMC, another 2 kg of HMC was fed. The following day animals completed the experiment and were disconnected from the pH monitoring equipment. Two cows were excluded from the precalving determinations due to earlier than expected calving dates, resulting in an insufficient collection period length.

DMI and Feed Analyses

The amounts of TMR and alfalfa hay offered and refused each day were recorded for all animals. Repre-

Table 2. Nutrient composition (DM basis) for experimental diets (SE in parentheses)

Item	Close-up dry cow diet	Lactating cow diet	Hay	High-moisture corn
DM (%)	46.2 (0.71)	50.4 (0.43)	86.7 (1.19)	69.1 (0.76)
CP (N $\times$ 6.25, %)	13.7 (0.67)	15.3 (0.53)	16.3 (0.59)	9.2
Soluble protein (% of CP)	35.1 (3.00)	25.6 (2.65)	38.6 (1.58)	ND ¹
Undegradable protein (bypass est., % of CP)	47.1 (2.22)	46.3 (1.31)	32.0 (0.73)	ND
NDF (%)	39.4 (1.51)	33.9 (0.83)	47.5 (0.88)	10.3
ADF (%)	23.6 (0.95)	20.4 (0.47)	35.0 (1.60)	3.6
ADF – CP (%)	1.06 (0.09)	1.59 (0.33)	1.06 (0.06)	1.8
NFC ² (%)	36.2	39.1	30.4	69.2
Calcium (%)	0.96 (0.09)	1.02 (0.05)	0.96 (0.05)	0.03
Phosphorus (%)	0.47 (0.03)	0.52 (0.01)	0.28 (0.02)	0.30
Potassium (%)	1.48 (0.07)	1.60 (0.03)	2.18 (0.09)	0.43
Magnesium (%)	0.35 (0.02)	0.36 (0.01)	0.23 (0.02)	0.12
Sodium (%)	0.20 (0.02)	0.40 (0.04)	0.05 (0.01)	0.01

¹ND = Not determined.²NFC estimated as 100 – CP – NDF – ether extract – ash.

sentative feed and weigh-back (orts) samples were taken each day. The feed samples collected were pooled by experimental week for DM analysis. The orts samples collected were pooled by weight for each cow for each experimental week and analyzed for DM. The DM content of feeds and orts were determined by oven-drying in a 60°C oven for 48 h.

Dried feed and orts samples were ground using a Wiley mill with a 1-mm screen (Arthur H. Thomas, Philadelphia, PA) and were subsequently stored at –20°C until analysis could occur. The pooled samples of feeds were analyzed for CP using the macro-Kjeldahl procedure (method 988.05; AOAC, 1990), soluble protein (Licitra et al., 1996), RDP (Licitra et al., 1999), ADF (method 973.18; AOAC, 1990), NDF (Goering and Van Soest, 1970), Ca, P, K, Mg, and Na. Minerals were measured using inductively coupled plasma spectroscopy (method 968.08; AOAC, 1990) with a Perkin-Elmer Optima 3000 spectrophotometer.

Continuous Rumen pH Measurement

Rumen pH was monitored continuously using an adaptation of the method used by Dado and Allen (1993). A Sensorex Combi pH Electrode 450 CD (Sensorex, Stanton, CA) was placed through the rumen cannula and suspended into the ventral sac of the rumen. The electrode was protected by a wire shield and attached to a weight of approximately 0.5 kg. Electrodes were connected to a Jenco Digital pH Transmitter model 691N (Jenco Inc., La Jolla, CA). The output of the pH transmitter was captured by a Universal Analog Input Multiplexer EXP-16 (Omega Engineering Inc., Stamford, CT) and DAS-8 analog input board (Omega Engineering Inc.) installed in a Pentium personal computer. The software used in the data capturing was Labtech Notebook Version 10 (Laboratory Technologies Corpo-

ration, Wilmington, MA). A pH reading was taken every second, averaged over every 60-s period, and subsequently stored for further analysis. The position of each pH electrode was checked daily and the electrodes and pH transmitters were calibrated with pH 4 and pH 7 buffer solutions (Fisher Scientific, Fairlawn, NJ) at least once weekly. These pH data were summarized for each 24-h period and expressed as average daily pH, the amount of time below pH 6 and pH 5.6, and the area (time $\times$ pH) below pH 6 and pH 5.6.

Milk Collection and Analysis

Milk yields during all milkings were recorded. Milk samples were collected from the morning and afternoon milkings on each Monday and Thursday, preserved with 2-bromo-2-nitropropane-1-2-diol, and pooled daily based on production. Pooled samples were sent immediately for composition analysis to the Laboratory Services Division of the University of Guelph (Guelph, Ontario, Canada). Concentrations of fat, protein, and lactose were determined by near infrared analysis with the Foss System 400 (Foss Electric, Hillerød, Denmark).

Statistical Analysis

Data on the response variables were analyzed using PROC MIXED of SAS (SAS Institute, 1997) as recommended by Wang and Goonewardene (2004) for the analysis of animal experiments with repeated measures using the model:

$$Y_{ijkl} = \mu + T_i + B_j + D_k + (T \times B)_{ij} + (T \times D)_{ik} + \varepsilon_{ijkl}$$

where Y_{ijkl} is the dependent variable; μ is the overall mean; T is the effect of treatment (monensin or control); B_j is the effect of block ($j = 1, 2, \dots, 8$); D_k is the effect

Figure 1. Weekly averages of average daily rumen pH in cows receiving a placebo (control) or a monensin controlled release capsule precalving. Note: error bars are for comparison within week relative to calving.

of day relative to calving ($k = -21, -20, \dots, 41, 42$); $(T \times B)_{ij}$ is the effect of the interaction of T_i and B_j ; $(T \times D)_{ik}$ is the effect of the interaction of T_i and D_k ; and ε_{ijkl} is the random residual error. The effects of concentrate T_i and D_k and their interaction were analyzed as fixed effects. The effect of B_j and interactions of other factors with B_j were considered random. Day relative to calving was the repeated measure. The effect of D_j was tested for significance using the $(T \times B)_{ij}$ as error term. Covariance structures relative to daily measurements that were tested included simple, compound symmetry, first-order autoregressive, first-order ante dependence, and unstructured (Wang and Goonewardene, 2004). Final mixed models were accepted only if the convergence criteria were met, the estimated G matrix was a positive definite, and the degrees of freedom for hour were the same as those obtained by running the same model using the GLM procedure (SAS Institute, 1997). The covariance structure resulting in the lowest value for the fit statistic was chosen (Wang and Goonewardene, 2004).

Differences among weeks relative to calving were tested for significance using orthogonal contrasts (SAS Institute, 1997).

RESULTS AND DISCUSSION

Rumen pH

Monensin did not affect average daily rumen pH ($P = 0.54$), time below rumen pH 6 ($P = 0.52$), time below pH 5.6 ($P = 0.40$), area below pH 6 ($P = 0.33$), and area below pH 5.6 ($P = 0.21$) across the days of the experiment (Figures 1, 2, and 3). Day relative to calving affected average daily pH ($P < 0.0001$), time below pH 6 ($P < 0.0001$), and time below pH 5.6 ($P < 0.0001$). Day relative to calving did not affect the area below rumen

pH 6 ($P = 0.69$) and area below rumen pH 5.6 ($P = 0.99$). The interactions between monensin and week relative to calving on average daily rumen pH ($P = 0.57$), time below pH 6 ($P = 0.84$), and time below pH 5.6 ($P = 0.29$) were not significant. Average daily pH was lower ($P < 0.0001$), time below pH 6 was higher ($P < 0.0001$), and time below pH 5.6 was higher ($P < 0.01$) after calving compared with before calving. Average daily pH, and times below pH 6 and pH 5.6 did not differ among the weeks before calving. Throughout the weeks after calving, average daily pH showed a quadratic increase ($P < 0.005$), time below pH 6 showed a quadratic decrease ($P < 0.001$), and time below pH 5.6 showed a linear decrease ($P < 0.05$).

The absence of a monensin effect on rumen pH in the current study contrasts with several earlier studies. Administration of 1.3 mg of monensin/kg of BW was able to prevent experimentally induced lactic acidosis by increasing rumen pH and total VFA and reducing rumen lactate (Nagaraja et al., 1981). Feeding either 150 or 300 ppm/d of monensin to steers that were switched from an all-forage diet to a diet containing 75% grain (DM basis) reduced the drop in rumen pH and the increase in total rumen VFA (Burrin and Britton, 1986). Adding 25 mg/g of monensin to a 92.5% concentrate finishing diet increased average daily rumen pH and area below pH 5.6 in beef steers in which rumen pH was monitored continuously with indwelling pH probes (Cooper and Klopfenstein, 1996). Green et al. (1999) showed that the monensin CRC increased rumen pH in primiparous, but not multiparous, dairy cows. Mutsavangwa et al. (2002) observed that administration of monensin either as a CRC or as a premix at a rate of 22 mg/kg (DM basis) did not affect rumen pH during experimentally induced SARA and the subsequent recovery period in multiparous cows. The differences between the current study and those obtained by Nagaraja et al. (1981), Burrin and Britton (1986), and Cooper and Klopfenstein (1996) might be explained by differences in the dietary inclusion of concentrates and nonstructural carbohydrates. In the studies in which an effect of monensin on rumen pH was observed, the concentrate inclusion ranged from 75 to 92.5% DM, whereas in the current study concentrate inclusion did not exceed 65% DM. When very high grain diets are fed, such as in the experiment of Nagaraja et al. (1981), rumen lactate will accumulate, but this is not the case when the concentrate inclusion does not exceed 65% DM (Burrin and Britton, 1986). Monensin supplementation may only be effective for controlling reduction in rumen pH when diets are likely to produce significant amounts of accumulated lactic acid.

Green et al. (1999) demonstrated that monensin increased rumen pH in primiparous cows, but not in mul-

Figure 2. Weekly averages of the durations of rumen pH below pH 6 and below pH 5.6 of cows receiving a placebo (control) or a monensin controlled release capsule precalving. Note: error bars are for comparison within week relative to calving.

tiparous, cows. This parity effect could be the result of a greater adaptation of rumen microbial populations, the rumen papillae, and the absorptive capacity of VFA in multiparous cows compared with primiparous cows. Also, in the study from Green et al. (1999), a stomach tube was used to monitor rumen pH by spot sampling. This is a less accurate technique for the monitoring of rumen pH than continuous pH measurement with indwelling probes due to the diurnal variation of rumen pH and the possibility of saliva contamination of rumen fluid samples collected using a stomach tube (Duffield et al., 2004).

Between d 25 and 42 after calving, a grain challenge model was used to determine if the monensin CRC would attenuate the drop in rumen pH due to a grain

overload. In our attempt to lower rumen pH with the feeding of an increased level of HMC, we found no significant changes in rumen pH from the weeks before the challenge compared with the challenge period. This may have been due to the moderate level of grain that was gradually added to that already included in the TMR that was introduced to the cows. The total percentage of HMC fed was increased to approximately 20% DM of the total ration. In a previous experiment (Cooper and Klopfenstein, 1996), steers received diets containing up to 92.5% concentrate, whereas in our study, concentrate inclusion did not exceed 60% of DM. We chose to be more conservative in our experiment to minimize health risks and to mimic on-farm grain feeding practices. The cows in the current experiment al-

Figure 3. Weekly averages of the areas of rumen pH below pH 6 and below pH 5.6 of cows receiving a placebo (control) or a monensin controlled release capsule precalving. Note: error bars are for comparison within week relative to calving.

ready experienced a drop in rumen pH after calving of on average 0.43 to 0.49 pH units, compared with a drop in rumen pH of one pH unit caused by the ruminal acidosis induced by Nagaraja et al. (1981) and Burrin and Britton (1986). Follow up studies (Cumby et al., 2001; Keunen et al., 2002) showed that lactating cows fed a TMR similar to the one used in the current experiment needed to receive at least an additional 6 kg/d of a rapidly fermented grain source (e.g., wheat or barley) to induce SARA.

The differences in average daily rumen pH, time below pH 6 and time below pH 5.6 between the periods before and after calving, could be explained by differences in nutrient composition between the dry cow diet and the lactating cow diet (Tables 1 and 2). Because the lactating cow diets contained more grain, less NDF, and less forage NDF than the dry cow diet, a drop in rumen pH would be expected (Pitt et al., 1996). The gradual increases in rumen pH after calving could have been due to microbial and ruminal adaptation to the lactating cow diet. As observed by Dirksen et al. (1985), the capacity to absorb VFA decreases during the dry period when high forage diets are fed, and increases only slowly when cows are changed to a high concentrate diet. Reynolds et al. (2004) also found that rumen papillae mass was higher in dry cows than in lactating cows. Hence, a rapid increase in concentrate in the diet can result in insufficient absorption of and accumulation of VFA, and a reduction in rumen pH after a change to a higher concentrate diet at calving.

Feed Intake, BW, and BCS

Average BW and BCS decreased from 712 to 629 kg and from 3.71 to 2.81 on a scale from 1 to 5 (1 = thin, 5 = obese; Edmondson et al., 1989), respectively, during the experiments; that is, from 4 wk before calving until 6 wk after calving.

Monensin did not affect DMI across the days of the experiment ($P = 0.51$), but days relative to calving significantly affected DMI ($P < 0.0001$; Figure 4). Between the third week before calving and the week before calving DMI decreased linearly ($P < 0.0005$). From the week after calving until the sixth week after calving DMI showed a quadratic increase ($P < 0.0001$). The interaction between monensin and days relative to calving on DMI was not significant ($P = 0.22$).

The reduction in DMI before calving and the gradual increase in DMI during the first weeks of lactation observed in the current study are common in dairy cows (NRC, 2001). Earlier studies have reported conflicting results on the effect of monensin on feed intake. Studies with beef cattle have demonstrated that monensin can decrease in DMI (Goodrich et al., 1984). Sauer et al.

Figure 4. Weekly averages of DMI of cows receiving a placebo (control) or a monensin controlled release capsule precalving.

(1989) found that supplementing 15 and 30 mg/g of DM of complete diets starting 1 wk prepartum and continuing for 3 wk postpartum reduced average daily DMI from 14.5 to 13.9 and 13.3 kg/d, respectively. Phipps et al. (2000) observed that supplementing monensin to cows fed a TMR at a rate of 450 mg/d tended to decrease DMI. Ramanzin et al. (1997) found that supplementation of 300 mg/d of monensin to early and mid lactation cows fed diets with a 70:30 or a 50:50 forage-to-concentrate ratio tended to reduce DMI. Van der Werf et al. (1998) also concluded that supplementation with 300 and 450 mg/d of monensin to dairy cows between 4 and 24 wk after calving tended to reduce DMI. Addition of 22 mg/kg of monensin to TMR (DM basis) did not reduce DMI during experimentally induced SARA in mid lactation dairy cows (Osborne et al., 2004). Mutsvangwa et al. (2002) found that administration of monensin as a CRC to mid lactation cows did not affect DMI during experimentally induced SARA and during the recovery from SARA. However, inclusion of monensin premix in a TMR at a rate of 22 mg/kg (DM) increased DMI from 19.8 to 21.3 kg/d during the experimentally induced SARA and from 20.9 to 23.7 kg/d during the recovery period. Differences among studies included doses of monensin, animal numbers, stage of lactation, and diet, making it difficult to conclude which of these factors are responsible for the differences among these studies. The stage of lactation and the dose of monensin of the study from Sauer et al. (1989) are the closest to those of the current study. That the study from Sauer et al. (1989) observed that monensin reduced DMI, whereas the current study did not, might be explained by the larger number of animals and the lower DMI in the earlier study. The larger number of cows in the earlier study would have resulted in a greater statistical power. The lower DMI suggests that feed intake might have been constrained by other factors in the study from Sauer et al. (1989) than in

Figure 5. Weekly averages of daily milk yields of cows receiving a placebo (control) or a monensin controlled release capsule precalving.

our study and that monensin may not affect these factors similarly.

Milk Production

Monensin did not affect daily milk yield across DIM ($P = 0.36$), but DIM significantly affected milk yield ($P < 0.0001$; Figure 5). Across treatments, daily milk yield showed a quadratic increase ($P < 0.05$) between the first week and the sixth week of lactation. The interaction between monensin and DIM on milk yield was not significant ($P = 0.84$). Monensin did not affect milk fat percentage ($P = 0.93$) and milk fat yield ($P = 0.94$) across DIM, but DIM significantly affected milk fat percentage ($P < 0.0001$; Figure 6) and milk fat yield ($P < 0.0001$). Across treatments, daily milk fat percentage showed a quadratic decrease ($P < 0.001$) between the first week and the sixth week of lactation. The interaction between monensin and DIM on milk fat percentage and milk

Figure 6. Weekly averages of milk fat percentage of cows receiving a placebo (control) or a monensin controlled release capsule precalving.

Figure 7. Weekly averages of milk protein percentage of cows receiving a placebo (control) or a monensin controlled release capsule precalving.

fat yield were not significant ($P = 0.14$ and $P = 0.66$, respectively). Monensin did not affect milk protein percentage ($P = 0.96$) and milk protein yield ($P = 0.83$) across DIM, but the interaction between DIM and milk protein percentage ($P < 0.0001$) and the interaction between DIM and milk protein yield ($P = 0.0006$) were significant (Figure 7). Milk protein percentage and milk protein yield were higher in control cows than in monensin cows in the first week of lactation, but not in any other week of the study. Days in milk significantly affected milk protein percentage ($P < 0.0001$) and milk protein yield ($P < 0.0001$). Across treatments, milk protein percentage showed a quadratic decrease ($P < 0.001$) between the first week and the sixth week of lactation.

The decreases in milk fat and milk protein percentages in early lactation in the current study are typical for Holstein cows (Stanton et al., 1992). Earlier studies have reported conflicting results on the effect on monensin on milk production. Supplementation with 15 or 30 mg/g (DM) of diets with monensin starting 1 wk prepartum and continuing for 3 wk postpartum did not affect milk yield (Sauer et al., 1989). However, in this study the monensin supplementation of 15 mg/g reduced milk fat percentage, but did not affect milk protein percentage in the first 4 wk after calving. Supplementation of dairy cows with 150, 300, and 450 mg/d of monensin between 4 and 24 wk tended to increase milk yield, but did not affect milk protein content (Van der Werf et al., 1998). In the same study, supplementation with 450 mg/kg of monensin reduced milk fat content. Prepartum administration of a monensin CRC did not affect milk fat percentage and milk protein percentage on the first 3 milk tests after calving at approximately 30 DIM, 60 DIM, and 90 DIM (Duffield et al., 1999a). Similarly, Green et al. (1999) found that prepar-

tum administration of a monensin CRC did not affect milk yield and milk composition in primiparous and multiparous cows. Supplementation of 300 mg/d of monensin to early and mid lactation dairy cows fed diets with a 70:30 or a 50:50 forage-to-concentrate ratio did not affect milk yield and milk protein percentage, but tended to reduce milk fat percentage. Phipps et al. (2000) observed that increasing the supplementation of monensin to cows fed a TMR with 150, 300, or 450 mg/d increased milk yield, reduced milk fat percentage and milk protein percentage, but did not affect milk fat yield and milk protein yield. Duffield et al. (2003) observed that supplementation with between 9 and 14 mg/kg (DM basis) of monensin reduced milk fat in TMR fed herds, but that supplementation with between 9 and 23 mg/kg (DM basis) of monensin did not reduce milk fat in component fed herds. These authors also found that the milk fat depression caused by monensin was lower in herds that fed sufficient coarse fiber than in herds fed insufficient coarse fiber, which suggests that the effect of monensin on milk production depends on dietary factors. The earlier studies are difficult to compare, because they differed in doses of monensin, animal numbers, stage of lactation of cows and diets, and each of these factors could have affected the observed impact of monensin on milk production. Hence, it is not possible to isolate which of these factors is responsible for the discrepancies among these studies. In the studies in which monensin decreases milk fat and milk protein percentages, an increase in milk yield was also observed (Van der Werf et al., 1998; Phipps et al., 2000), which resulted in an absence of effects of monensin on milk fat yield and milk protein yield. The study from Green et al. (1999), which did not observe an effect of monensin on milk production, was conducted with cows in a similar stage of lactation and similar administration and dose of monensin compared with the current study, but with a larger number of cows. Hence, the results from the current study confirm those obtained by Green et al. (1999).

CONCLUSIONS

Prepartum administration of a monensin CRC did not affect rumen pH characteristics, DMI, or milk production of Holstein cows that were switched from a dry cow diet to a lactating cow diet at calving. The lack of a monensin effect on rumen pH might be explained by the maximum inclusion rate of concentrate, which did not exceed 65% of DM. Average daily rumen pH decreased and time below pH 6 and pH 5.6 increased after calving. In the weeks after calving average daily pH showed a quadratic increase, time below pH 6 showed a quadratic decrease, and time below pH 5.6 showed a

linear decrease. Area below pH 6 and area below pH 5.6 were not affected by day relative to calving. Changes in rumen pH characteristics during the experiment were due to dietary changes, but adaptation of rumen microbes, rumen papillae, and the VFA absorption capacity of the rumen might also have contributed.

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