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Effect of monensin on recovery from diet-induced milk fat depression

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ABSTRACT

The objective of the present experiment was to investigate the effect of monensin (MN) on the time course of recovery from diet-induced milk fat depression. Milk fat depression was induced in all cows (n = 16) during the first phase of each period by feeding a low-fiber, high-unsaturated fat diet [25.3% neutral detergent fiber (NDF), 6.9% fatty acids (FA), and 3.24% C18:2] with MN (450 mg/cow per day) for 10 to 14 d. A recovery phase of 18 d followed, where cows were switched to a higher-fiber and lower unsaturated fat diet (31.2% NDF, 4.3% FA, and 1.7% C18:2). According to a crossover design, treatments during recovery were (1) control (no MN supplementation) or (2) continued MN supplementation. Milk yield, milk composition, and milk FA profile were measured every 3 d during recovery. No effect was observed of MN on dry matter intake or yield of milk, milk protein, and lactose. Milk fat concentration and yield increased progressively during recovery in both treatments. Monensin decreased milk fat yield from d 6 to 15, but it was the same as the control on d 18. A treatment by time interaction on milk fat concentration was detected, which was decreased by MN only on d 3 and 6. The yield of milk de novo synthesized FA increased progressively in both treatments and was not affected by treatment. Similarly, yield of 16-C FA increased progressively, but was decreased by MN on d 6 and 9. Preformed FA yield was lower in the MN group from d 6 to 15, but was not different from the control on d 18. Importantly, milk FA concentration of *trans*-10 C18:1 and *trans*-10,*cis*-12 conjugated linoleic acid rapidly decreased in both groups; however, MN slightly increased *trans*-10 C18:1 concentration above baseline on d 15 and 18. In conclusion, MN supplementation had minimal effect on recovery of normal rumen biohydrogenation and de novo FA synthesis during recovery from milk fat depression by correction of dietary starch, NDF, and polyunsaturated FA concentration, but moderately decreased recovery of preformed FA in milk.

Key words: monensin, milk fat depression, dairy cow, conjugated linoleic acid

INTRODUCTION

Milk fat depression (**MFD**) is characterized by a specific reduction in milk fat synthesis, and is caused by bioactive *trans* FA arising from altered ruminal fermentation (Bauman and Griinari, 2001). Many dietary factors affect the formation and ruminal outflow of bioactive FA, including feeding methods, dietary concentration of highly fermentable feeds, PUFA, effective fiber, and ionophores. The interaction of these risk factors sometimes results in modification of the rumen environment, the microbial population, and the pathways of FA biohydrogenation (**BH**), which leads to increased formation of the bioactive FA that cause MFD (Jenkins et al., 2003; Weimer et al., 2010).

Ionophores are lipophilic molecules toxic to many bacteria, protozoa, and fungi (Russell and Strobel, 1989). Monensin (**MN**) is an ionophore that has been shown to increase milk yield and feed efficiency in lactating dairy cows (Ipharraguerre and Clark, 2003; Duffield et al., 2008b). Monensin alters the ion flux and ATPase systems of sensitive bacteria, resulting in an increase in maintenance energy expenditure and compromised growth (Ipharraguerre and Clark, 2003). Thus, MN supplementation results in selection of ruminal bacteria that produce less H_2 and acetate and more propionate and ATP (Russell and Strobel, 1989). In addition, under some circumstances, MN leads to reduced BH capacity and utilization of alternate pathways, resulting in bioactive FA formation (Fellner et al., 1997; Jenkins et al., 2003).

The effect of MN on milk fat yield and concentration is not consistent across studies, as some have reported reductions (Phipps et al., 2000; Odongo et al., 2007; AlZahal et al., 2008), whereas others have reported no effect (Lean et al., 1994; Duffield et al., 1999; He et al., 2012). It is likely that the variation in response to MN feeding is related to interactions with diet type. For example, starch source and level of dietary PUFA have been previously shown to reduce the rates of FA BH (Fellner et al., 1997; Duffield et al., 2008b) and increase the concentration of alternate BH intermediates, such

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as *trans*-10 C18:1, in rumen fluid and milk fat (Jenkins et al., 2003; AlZahal et al., 2008; He et al., 2012). In addition, MN and PUFA synergistically increased *trans*-10 C18:1 formation in vitro (Jenkins et al., 2003) and in vivo (AlZahal et al., 2008; He et al., 2012).

Strategies to rescue milk fat synthesis after MFD has occurred are important to reduce the duration of the condition. We have previously validated a model of MFD induction and recovery, which provided the basis for the experimental design of the current experiment (Rico and Harvatine, 2013). Briefly, during induction of MFD, milk fat yield was near the nadir by d 11 and during recovery, a higher-fiber and low-PUFA diet completely rescued milk fat yield by d 19. Previous investigations of MN have predominantly focused on the long-term or steady-state effects of supplementation; however, long-term changes in the microbial population after termination of MN have been reported (Weimer et al., 2008). The objective of the current study was to investigate the effect of MN on the rate of recovery of milk fat synthesis and milk FA profile after induction of MFD while supplementing MN in dairy cows. The hypothesis was that MN would minimally affect the rate of recovery of milk fat synthesis.

MATERIALS AND METHODS

Experimental Design and Treatments

The experiment was conducted from February to May of 2012. All experimental procedures were approved by the Pennsylvania State University Institutional Animal Care and Use Committee (University Park). Sixteen multiparous Holstein cows (183 ± 21 d postpartum; mean ± SD) were randomly assigned to treatment sequences in a balanced crossover design. Cows were housed in a tie-stall barn located at the Pennsylvania State University Dairy Production Research and Teaching Center. All cows received recombinant bST (rbST; Posilac; Elanco Animal Health, Greenfield, IN), administered every 14 d for the duration of the experiment. As reviewed by Bauman (1992), bST treatment has minimal effect on milk components, whereas milk yield is gradually increased, with a maximal response observed approximately 6 d after dosing. All animals received bST on the same day, allowing the cyclic response to be modeled as a main effect of day.

A 21-d pretrial and washout period were used to allow adaptation to MN (Table 1). Commercially sourced MN (Rumensin 90; Elanco Animal Health) was topdressed at a rate of 280 mg/cow per day (in 0.6 kg of cookie meal, DM basis) from d 1 to 10 and at 450 mg/cow per day (in 1.0 kg of cookie meal, DM basis) from d 11 to 21 of the pretrial and washout periods. Each experimental period was divided into an MFD induction phase (14 d in period 1 and 10 d in period 2) and a recovery phase (18 d). In period 1, the expected magnitude of decrease in milk fat percentage was not observed by d 7 of induction, so the induction diet was slightly modified and the induction phase extended to 14 d. During induction, all cows were fed a low-fiber, high-unsaturated fat diet with MN (25.3% NDF, 6.9% FA, and 3.24% C18:2; 450 mg of MN/cow per day; Table 2; Supplemental Table S1; http://dx.doi.org/10.3168/jds.2013-7486). During recovery, cows were fed a higher-fiber and lower-unsaturated fat diet (31.2% NDF, 4.3% FA, and 1.7% C18:2). Treatments were applied during the recovery phase only, and treatments were control (no MN) or continued supplementation with MN topdressing (450 mg/cow per day).

Cows were fed individually once daily (0800 h) at 110% of expected intake and intake was observed daily. Forage and base diet DM was determined weekly for diet adjustment and DMI determination (72 h at 55°C in a forced-air oven). All individual feed ingredients were sampled weekly and stored at −20°C, thawed at room temperature, composited by period, and DM content determined as described above. Individual feeds were ground in a Wiley mill through a 1-mm screen (A. H. Thomas Co., Philadelphia, PA). Individual forages and a representative mixture of concentrate feeds were analyzed for nutrient composition by wet chemistry procedures (Cumberland Valley Analytical Services Inc., Maugansville, MD). Briefly, assays conducted were DM and CP according to AOAC International (2000); NDF and ADF according to Van Soest et al. (1991), using heat-stable amylase and sodium sulfite;

Table 1. Treatment sequences in a crossover design evaluating the effect of monensin (MN) removal on recovery from diet-induced milk fat depression (MFD)

Sequence	Pretrial (21 d)	Period 1 (14 d[1] → 18 d)	Washout (21 d)	Period 2 (10 d →18 d)
1	MN; step-up[2]	Induction +MN → recovery +MN[3]	MN; step-up	Induction +MN → recovery −MN
2	MN; step-up	Induction +MN → recovery −MN	MN; step-up	Induction +MN → recovery +MN

[1]The induction phase was extended in period 1 when milk fat was not reduced to expected levels by d 7 of induction.

[2]Monensin was fed at 280 mg/cow per day for 10 d and then at 450 mg/cow per day for 11 d.

[3]Induction of MFD by feeding a low-fiber and high-unsaturated fat diet supplemented with MN. Recovery = a higher-fiber and lower-unsaturated fat diet with (+MN) or without (−MN) monensin supplementation for 18 d.