



## Effect of dietary fat blend enriched in oleic or linoleic acid and monensin supplementation on dairy cattle performance, milk fatty acid profiles, and milk fat depression

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### ABSTRACT

The effect of feeding increasing levels of oleic and linoleic acid both independently and together, with or without monensin, on milk fat depression was evaluated. Fifty-six Holstein cows were blocked by parity and then were divided by milk production into 2 groups (high or low) of 14 cows each within each parity block. A cow pair of 1 high and 1 low production cow within each parity block was fed in a single electronic feeding gate. Gates ( $n = 28$ ) were considered the experimental unit and were assigned to monensin (17.5 g/t of dry matter) or control as the main plot ( $n = 14$  each). The 7 cow pairs in each of the fixed effect groups were further assigned to a sequence of fat blend diets as split plot. Seven fat blend treatments in the split plot  $7 \times 7$  Latin square were no added fat (no fat) and diets with increasing levels of oleic or linoleic acid: low C18:1 + low C18:2 (LOLL); low C18:1 + medium C18:2 (LOML); low C18:1 + high C18:2 (LOHL); medium C18:1 + low C18:2 (MOLL); medium C18:1 + medium C18:2 (MOML); and high C18:1 + low C18:2 (HOLL). Monensin feeding did not affect milk yield or concentration and yield of milk fat. Feeding monensin decreased the proportion of C <16, increased the proportion of total C18, increased the proportion and yield of *trans*-10 C18:1, and increased the proportion of *trans*-10, *cis*-12 conjugated linoleic acid in milk fatty acids (FA). As dietary C18:1 or C18:2 increased beyond the concentration present in LOLL, milk fat concentration, milk fat yield, and proportion and yield of milk C <16 all decreased, and the proportion and yield of milk *trans*-10 C18:1 increased. A quadratic effect on milk fat concentration and yield was noticed for C18:2 feeding, but not for C18:1 feeding. When dietary contents of total FA and FA other than C18:1 and C18:2 were similar, C18:2-rich diets decreased milk fat concentration and yield compared with C18:1-rich diets (LOML

vs. MOLL, and LOHL vs. HOLL), indicating that C18:2 is more potent than C18:1 for depressing milk fat. Increasing dietary FA content from no fat to LOLL, which increased primarily C18:1 and C18:2 with small increases in C18:0 and C16:0, decreased the secretion of C <16 but increased total C18 secretion in milk. This suggests that biohydrogenation intermediates act to decrease mammary FA synthesis at low levels of added C18:1 and C18:2. No significant monensin  $\times$  fat interactions were detected for the milk composition parameters analyzed; however, a monensin  $\times$  fat interaction was found for milk fat *trans*-10 C18:1 proportion.

**Key words:** oleic, linoleic, monensin, milk fat depression

### INTRODUCTION

Fatty acids occur in dairy rations due to intentional supplementation as a way to raise diet energy density or due to their inherent presence in certain feeds. However, milk fat depression (MFD) may occur when highly unsaturated oils are directly added to the diet, or when full-fat seeds or meal containing polyunsaturated FA are supplemented (Bauman and Griinari, 2001). Lipids undergo hydrolysis and biohydrogenation in the rumen, and unique FA intermediates that inhibit *de novo* FA synthesis in the mammary gland are produced under certain conditions (Bauman and Griinari, 2003). Experiments involving abomasal infusion of FA isomers have provided evidence that *trans*-10, *cis*-12 conjugated linoleic acid (CLA; Baumgard et al., 2002), *trans*-9, *cis*-11 CLA (Perfield et al., 2007), *cis*-10, *trans*-12 CLA (Sæbø et al., 2005), and possibly *trans*-10 C18:1 (Shingfield et al., 2009) inhibit milk fat synthesis, although other unidentified FA may also contribute to MFD. Critical ingredients for this phenomenon are the profile and availability of unsaturated FA supplied to the rumen and ruminal conditions that favor incomplete or altered biohydrogenation pathways.

Milk fat depression has been found in several studies when a variety of lipid sources rich in linoleic acid (C18:2) were supplemented in the diet (Lock et al.,

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2006; AlZahal et al., 2008; He and Armentano, 2011). Oleic acid (C18:1) is also common in many lipid sources, including canola oil, peanut oil, and high-oleic safflower oil. Contrary to C18:2, some studies suggest that dietary C18:1 intake does not inhibit milk fat synthesis. Selner and Schultz (1980) supplemented up to 500 mL of C18:1 per cow per day and found no significant change in milk fat concentration and yield. In another study, feeding 150 mL of C18:1 twice daily also had no significant effect on milk fat concentration (Shaw and Ensor, 1959). Jenkins (1998) and Rego et al. (2009) also fed C18:1-rich canola oil, which depressed milk fat synthesis and inhibited *de novo* FA synthesis, but the canola oil used in both studies contained greater than 10% C18:2 of the total FA, which could contribute to MFD. However, AbuGhazaleh et al. (2005) confirmed the conversion of C18:1 by mixed ruminal microbes to a multitude of *trans*-C18:1 isomers having double bond positions from C6 through C16. *Trans*-10 C18:1 is also potentially inhibitory to milk fat synthesis (Shingfield et al., 2009), and *trans*-6 to 8 C18:1 are negatively correlated with milk fat concentration (Kadegowda et al., 2008), although no evidence exists showing that these other monoene *trans* FA directly cause MFD. These facts suggest that both C18:1 and C18:2 may negatively affect milk fat synthesis concurrently.

A curvilinear relationship was reported between milk fat concentration and milk *trans*-10 C18:1 proportion or milk *trans*-10,*cis*-12 CLA proportion in milk FA, characterized by a steeper initial drop in milk fat (Bauman and Griinari, 2003). Lock et al. (2007) also found a similar curvilinear relationship between rumen outflow of *trans*-10 C18:1 ( $X$ , g/d) and milk fat concentration ( $Y$ , %) ( $Y = 4.59X^{-0.14}$ ). Because *trans*-10 C18:1 and *trans*-10,*cis*-12 CLA are both FA intermediates from an altered pathway of C18:2 rumen biohydrogenation (Bauman and Griinari, 2003) and *trans*-10 C18:1 can also be synthesized from C18:1 under certain conditions (AbuGhazaleh et al., 2005), it is reasonable to believe that the MFD dose response to dietary C18:2 is nonlinear and more potent at lower C18:2 concentration. An objective of the current study was to evaluate the effects of level of dietary C18:1 and C18:2, independently and in combination, on milk production and composition of dairy cattle. A particular interest was the effects of these fats on milk FA production and composition.

Monensin was approved to be used in lactating dairy cattle for improving milk production efficiency by the US FDA in 2004 (Hamilton and Mitloehner, 2008). Monensin is a carboxylic polyether ionophore naturally produced by the bacterial strain *Streptomyces cinnamomensis* (Haney and Hoehn, 1967). Monensin functions as an ion antiporter that modifies monovalent cation transport across cell membranes of gram-positive bac-

teria. Therefore, the ruminal bacteria population is altered and, as a consequence, monensin has numerous beneficial effects on dairy cattle, including less ruminal methane production (Odongo et al., 2007), improved milk production efficiency (Duffield et al., 2008a), decreased risk of ketosis and mastitis (Duffield et al., 2008b), and attenuated ruminal acidosis in cattle fed a high-concentrate diet (Bergen and Bates, 1984).

However, similar to unsaturated oils, monensin has also been associated with MFD by inhibiting unsaturated FA biohydrogenation within the rumen, as evidenced by altered milk FA composition (Sauer et al., 1998). A decrease in milk fat concentration (Phipps et al., 2000; Benchaar et al., 2006) or a decrease of both milk fat concentration and yield (Broderick, 2004; AlZahal et al., 2008) was observed when monensin was fed to lactating dairy cattle. Feeding monensin resulted in a more significant decrease in milk fat concentration when a greater amount of C18:2-rich soybean oil was present in the diet, which suggested possible monensin  $\times$  soybean oil interaction (AlZahal et al., 2008). Their study also found greater milk fat proportion of total *trans*-18:1, including *trans*-6 to 8 and *trans*-10 C18:1 and a greater proportion of total CLA isomers including milk fat-depressing *trans*-9,*cis*-11 and *trans*-10,*cis*-12 CLA with monensin feeding when soybean oil was present (AlZahal et al., 2008).

A recent meta-analysis also indicated a possible enhanced effect of monensin on decreasing milk fat yield with increased dietary C18:1 intake (Duffield et al., 2008a). Thus, the effect of feeding vegetable oils rich in unsaturated FA, especially C18:1 and C18:2, on MFD may be amplified by monensin feeding, and the second objective of this study was to evaluate possible interactions between dietary C18:1 or C18:2 level and monensin supplementation on dairy cattle performance, especially milk fat synthesis.

## MATERIALS AND METHODS

### Animals and Treatments

The basic design of this trial was to test the monensin effect in continuous design, with 28 cows receiving monensin throughout the trial and 28 control cows. Within these treatment groups, a Latin square design was used to apply the fat blend treatments so that each animal received each of the 7 fat blend treatments but only 1 monensin level. This design was more statistically powerful to test the fat blend effects but gave less power for the effect of monensin. However, possibility still exists that results in this study could be influenced by accretion and remobilization of body fat from period to period. Fifty-six lactating Holstein cows

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