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The effect of conjugated linoleic acid, acetate, and their interaction on adipose tissue lipid metabolism in nonlactating cows

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ABSTRACT

During biohydrogenation-induced milk fat depression, adipose tissue lipogenesis may be increased through nutrients spared from milk fat synthesis. However, the direct effect of *trans*-10, *cis*-12 conjugated linoleic acid (CLA) and the indirect effect of spared nutrients on adipose tissue lipogenesis during milk fat depression is not clear. The objective of this study was to determine the direct effect of CLA, spared acetate, and their interaction on adipose tissue lipogenesis using nonlactating dairy cows as an experimental model, which allows separation of the effect of CLA and nutrient sparing. Eight ruminally cannulated, multiparous nonlactating and pregnant Holstein cows were randomly assigned to treatments in a 4 × 4 Latin square design with a 2 × 2 factorial arrangement of treatments. Factors were CLA and acetate, and treatments were control (CON), rumen acetate infusions (Ac; continuous infusion of 7 mol/d adjusted to pH 6.1 with sodium hydroxide), abomasal infusion of *trans*-10, *cis*-12 CLA (CLA; 10 g/d of both *trans*-10, *cis*-12 CLA and *cis*-9, *trans*-11 CLA), and Ac + CLA (Ac + CLA). Dry matter intake was not affected by Ac, but tended to decrease by CLA. Plasma *trans*-10, *cis*-12 CLA concentration as a percentage of fatty acids was increased by 0.3 percentage points by CLA. No treatment effect was observed on plasma glucose and β-hydroxybutyrate, but an interaction was observed of CLA and Ac on plasma insulin and nonesterified fatty acids. Insulin was increased 24% by CLA, but not by Ac + CLA, and nonesterified fatty acids were increased 55% by Ac + CLA, but not by CLA alone. Lipogenesis and oxidation capacity of adipose tissue explants were not affected by treatments. Adipose expression of key lipogenic factors (peroxisome proliferator-activated receptor γ2 and sterol response element binding protein 1c) were reduced by CLA, by the interaction of Ac and CLA (sterol response element binding protein 1c), and tended to be reduced with Ac (S14 and peroxisome proliferator-activated receptor γ1). Expression of several adipose lipogenic enzymes (fatty acid synthase, acetyl-CoA carboxylase, and stearoyl-CoA desaturase 1) was reduced by CLA and Ac. An interaction was observed of Ac and CLA for fatty acid binding protein 4, which was decreased by Ac, but not Ac + CLA. In conclusion, in the nonlactating cow, adipose tissue is sensitive to the anti-lipogenic effects of *trans*-10, *cis*-12 CLA at the transcription level and acetate does not stimulate lipogenesis.

Key words: acetate, conjugated linoleic acid, adipose lipogenesis, spared nutrient

INTRODUCTION

Biohydrogenation (**BH**) induced milk fat depression (**MFD**) results in a specific decrease in milk fat synthesis in lactating dairy cows and *trans*-10, *cis*-12 CLA is the most well studied bioactive fatty acid (**FA**) that causes the condition. Milk fat originates either from de novo synthesis in the mammary gland or mammary uptake of preformed FA originating from the diet and mobilization of body fat reserves (Emery, 1973). During BH-induced MFD, yield of both preformed and de novo synthesized FA are decreased, although de novo synthesized FA are decreased to a larger extent. The mechanism involves a decrease in mammary lipogenic capacity and expression of key lipogenic factors, including sterol response element binding protein 1 (*SREBP1*) and thyroid hormone responsive spot 14 (*S14*), and lipid synthesis enzymes, including fatty acid synthase (*FASN*), acetyl-CoA carboxylase (*ACC*), stearoyl-CoA desaturase 1 (*SCD1*), lipoprotein lipase (*LPL*), fatty acid binding protein 4 (*FABP4*), and glycerol phosphate acyl transferase (*GPAT*; reviewed by Harvatine et al., 2009a).

The decrease in milk fat synthesis during BH-induced MFD decreases mammary demand for nutrients used

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for milk fat synthesis. This decrease in demand spares a large amount of acetate and also butyrate, glucose, and preformed FA, which are then available to other tissues for other metabolic uses. Spared nutrients and the associated increase in energy balance are expected to feedback to reduce intake, and Harvatine et al. (2009b) observed a small decrease in intake in a meta-analysis of short-term CLA infusion experiments (3 to 5 d). However, the decrease in intake only accounted for a portion of the nutrients spared by the reduction in milk fat observed. Additionally, Harvatine et al. (2009b) reported an increase in expression of key lipogenic factors [*SREBP1*, *S14*, and peroxisome proliferator-activated receptor gamma (*PPARγ*)], lipogenic enzymes (*FASN*, *LPL*, *SCD1*, and *FABP4*), and leptin in adipose tissue during CLA-induced MFD. Thering et al. (2009) also reported increased expression of *S14* in adipose tissue during long-term BH-induced MFD, although the experiment used a limited number of cows and failed to observe changes in other lipogenic genes.

Increased expression of lipid synthesis genes in adipose tissue during *trans*-10, *cis*-12 CLA infusion in the cow is in contrast to its well-described inhibition of lipid synthesis and adiposity in other animal models including the pig, rodents, and humans (Foote et al., 2010). Additionally, CLA decreased lipogenic capacity in ex-vivo treated adipose tissue explants from beef steers (Choi et al., 2014). Importantly, these models are different from CLA-induced MFD as they do not have the confounding effect of spared nutrients and also used higher doses of CLA.

Harvatine et al. (2009b) proposed that increased expression of lipogenic genes in adipose tissue during CLA-induced MFD might represent a short-term response to nutrients spared from milk fat synthesis or that adipose tissue sensitivity to *trans*-10, *cis*-12 CLA might differ from that of mammary tissue in the lactating cow. However, significant confusion exists about the effect of *trans*-10, *cis*-12 CLA and spared nutrients on adipose tissue lipogenesis during MFD. It is not possible to separate the direct effect of CLA from spared nutrients in the lactating cow; thus, nonlactating cows were used in the current experiment. The objective of this study was to determine the direct effect of CLA, spared acetate, and their interaction on adipose tissue lipogenesis using nonlactating dairy cows. Our hypothesis was that the *trans*-10, *cis*-12 CLA dose that causes MFD in the lactating cow would reduce adipose tissue lipogenesis in the nonlactating cow and that acetate infusion mimicking the amount of acetate spared during MFD would increase adipose tissue lipogenesis and expression of lipogenic genes.

MATERIALS AND METHODS

Experimental Design and Treatments

All experimental procedures were approved by the Pennsylvania State University Institutional Animal Care and Use Committee (#41727). Eight ruminally cannulated multiparous Holstein cows (305-d energy-corrected milk yield of previous lactation: 11,206 ± 3,191 kg, average ± SD) were randomly assigned to treatments in a 4 × 4 Latin square design with a 2 × 2 factorial arrangement of treatments testing the effect of ruminal acetate and abomasal *trans*-10, *cis*-12 CLA infusion. Treatments included the following: control (**CON**), rumen acetate infusions (**Ac**), abomasal *trans*-10, *cis*-12 CLA infusions (**CLA**), and Ac + CLA (**Ac + CLA**). During treatment periods, cows were housed in a tie-stall metabolism unit located at the Pennsylvania State University Dairy Production Research and Teaching Center. Experimental periods were 10 d in length and included 3 d of treatment and 7 d of washout. Cows were arranged in 4 blocks based on expected calving date (between March 2013 and July 2014). Average dry period length was 115 ± 54 d and calf weight was 41.5 ± 3.1 kg (average ± SD).

The Ac treatment provided 7 mol/d of acetate and was prepared by diluting 400 mL of acetic acid in 7 L of distilled water and then adjusted to pH 6.1 using 269 g of sodium hydroxide pellets (J. T. Baker, Phillipsburg, PA). The CON and CLA treatments received an equal volume of distilled water. Acetate or distilled water were continuously infused through the rumen cannula using acid-resistant tubing (Norprene L/S 14, Cole-Parmer, Vernon Hills, IL) and peristaltic pumps (Masterflex L/S drive 7520–35, Cole-Parmer) similar to Sheperd and Combs (1998). The CLA treatment consisted of abomasal infusion of 34 g/d of a CLA methyl ester stock (Lutalin, BASF, Lampertheim, Germany) that contained 30% *trans*-10, *cis*-12 CLA and 30% *cis*-9, *trans*-11 CLA to provide 10 g/d of *trans*-10, *cis*-12 CLA. The CLA stock was infused in equal doses every 4 h through an abomasal infusion line [0.5 cm (i.d.) polyvinyl chloride tubing (Spires et al., 1975)] placed through the rumen cannula. The infusion lines were inserted the day before initiation of treatments and placement in the abomasum was checked daily. The lines were rinsed with 50 mL of warm distilled water before and after CLA infusion and with 20 mL of 70% ethanol before the final water rinse. In addition, CON and Ac treatments received 100 mL of warm distilled water and 20 mL of 70% ethanol abomasally as a handling control.

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