



## Influence of conjugated linoleic acid and vitamin E on performance, energy metabolism, and change of fat depot mass in transitional dairy cows

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### ABSTRACT

The objective of this experiment was to determine the effects of conjugated linoleic acid (CLA) and vitamin E as well as their interaction on performance variables and lipomobilization during late pregnancy and early lactation (wk 6 antepartum until wk 10 postpartum). For this purpose, 59 pluriparous German Holstein cows were assigned to 4 dietary groups in a  $2 \times 2$  design with the factors CLA and vitamin E at 2 levels. For this trial, we selected cows with a high body condition score because they are more likely to mobilize fat and consequently are at a higher risk of developing ketosis. Furthermore, concentrate proportions were adjusted to provoke ketosis. Lactation performance variables were analyzed in 3 periods (d 42 antepartum until calving, 1 to 21 d in milk, 22 to 70 d in milk). Dry matter intake and net energy intake were reduced in animals receiving CLA. Milk fat content was reduced in the CLA group compared with the control group (4.83 vs. 5.46% in period 2; 3.36 vs. 4.57% in period 3). In the vitamin E and the CLA + vitamin E groups, reduction of milk fat content was observed in period 3 (3.76 vs. 4.57% compared with the control group). Milk yield was not affected by treatment.  $\beta$ -Hydroxybutyrate concentrations and liver lipid contents were not influenced by CLA or vitamin E. Moreover, longitudinal changes of adipose tissue depot mass were not affected by dietary treatments. Results suggest that the effects CLA had on milk composition were compensated by an increased milk yield and a decreased dry matter intake. Reduced milk energy output in CLA-treated animals was compensated by a reduced dry matter intake. Therefore, the net energy balance was not affected by either treat-

ment. Consequently, we found no group effect on the mobilization of adipose tissue.

**Key words:** dairy cow, conjugated linoleic acid, vitamin E, fat depot

### INTRODUCTION

During the transition period, dairy cows are confronted with profound changes in their metabolic status. The increased nutrient demand caused by the growth of the fetus and the onset of lactogenesis cannot be compensated due to a reduced feed intake (Grummer et al., 1995). This results in a negative energy balance, which is partly compensated by the mobilization of fatty acids from adipose tissue and  $\beta$ -oxidation with the help of oxaloacetate in the liver. In a state of energy deficit, oxaloacetate is mainly needed for gluconeogenesis; therefore, a relative lack of oxaloacetate exists in the citrate cycle. As a consequence, the fatty acids can only partly be metabolized by the liver and ketone bodies (acetate, acetoacetate, and BHB) are produced from acetyl-CoA. Although ketone bodies can serve as an energy source for skeletal muscle, heart, kidney, and mammary glands in ruminants, Heitmann et al. (1987) showed that the extraction percentages of ketone bodies from blood by these organs are constant. Consequently, a higher ketone body production in the liver results in higher blood levels. Levels of BHB higher than 1.2 mmol/L are defined as the threshold for subclinical ketosis (Nielen et al., 1994). According to Schulz et al. (2014), a high concentrate proportion before calving, a high BCS at calving, and a delayed increase of concentrate proportion after parturition stimulate lipolysis; as a consequence, cows are more sensitive to develop a subclinical ketosis. Subclinical ketotic cows show no clinical signs of ketosis, but their reproductive health and economic productivity might be impaired (Drackley, 1999; Janovick et al., 2011). Therefore, the main objective of feeding in the transitional phase is the reduction of

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the negative energy balance. As milk fat is the major source of energy in milk, a reduction of milk fat percentage leads to a reduced milk energy output and, consequently, might partly counterbalance the negative energy balance. Baumgard et al. (2000) showed that the *trans*-10,*cis*-12 isomer of CLA is specifically responsible for the reduction of milk fat content and total milk fat yield (milk fat depression) by reducing the de novo milk fat synthesis in the udder, which results in a more efficient use of the ME. However, according to Bauman et al. (2008), supplementation of CLA in the state of early lactation or underfeeding also leads to a higher total milk yield and, therefore, repartitioning of energy. Consequently, CLA might have no significant effect on the total energy balance of the animal. Energy-dense diets antepartum (**a.p.**) lead to an increased DMI and increased accretion of internal fat depots without influencing carcass weight or BCS (Drackley et al., 2014). Pronounced lipomobilization caused by high fat depot mass is a risk factor for fatty liver and ketosis (Grummer, 1993; Bobe et al., 2004) and enhances the sensitivity to oxidative stress (Bernabucci et al., 2005), whereas vitamin E acts as an antioxidant (Rimbach et al., 2002; Nakamura and Omaye, 2010). Conjugated linoleic acid has been shown to have a decelerating influence on the mobilization of the retroperitoneal adipose tissue depot (von Soosten et al., 2011). To the contrary, Akter et al. (2011) reported a lipolytic or antilipogenic effect of CLA on the adipose tissue. This makes it necessary to evaluate the influence of CLA on lipomobilization. Literature indicates possible interactions between CLA and vitamin E, as CLA enhances  $\alpha$ -tocopherol concentration in muscle tissue (Schlegel et al., 2012), probably by preventing the degradation of vitamin E in the liver (Chao et al., 2010). Furthermore, Pottier et al. (2006) observed an influence of vitamin E on biohydrogenation pathways in the rumen, where vitamin E counteracted the *trans*-11 to *trans*-10 shift.

Therefore, the current experiment aimed to investigate the effect that CLA has on lactation performance, energy metabolism, and lipomobilization, as well as the interactions between CLA and vitamin E in dairy cows during the transition period and the first 10 wk of lactation. Our objective was to clarify the influence that CLA has on lipomobilization of fat depots and resulting subclinical ketosis. Furthermore, we wanted to evaluate whether treatment with vitamin E reduces the milk fat-decreasing effect of CLA.

## MATERIALS AND METHODS

The study was conducted at the experimental station of the Friedrich Loeffler Institute in Brunswick, Ger-

many. The experiment was carried out in accordance with the German Animal Welfare Act approved by the LAVES (Lower Saxony State Office for Consumer Protection and Food Safety, Germany).

## Experimental Design and Diets

The experimental design was a  $2 \times 2$  factorial design with CLA and vitamin E as main factors, resulting in 3 intervention groups (CLA, vitamin E, CLA + vitamin E) and 1 control group. We used the experimental strategies as proposed by Schulz et al. (2014) to generate cow groups suited for testing of possible protective effects of CLA and vitamin E. Sixty-four pluriparous German Holstein cows were allocated to these groups 8 wk prior ( $-42$  d a.p.) to the calculated calving date based on their BCS (Edmonson et al., 1989), which was targeted higher than 3.5 with a standard deviation of 0.5 in each group. Further criteria were milk yield and milk composition of the previous lactation, BW, and number of lactation. In the control group, cows were in the second ( $n = 10$ ), third ( $n = 4$ ), fourth ( $n = 1$ ), and fifth ( $n = 1$ ) lactation. For the CLA group, cows in the second ( $n = 9$ ), third lactation ( $n = 3$ ), fourth ( $n = 1$ ), sixth ( $n = 1$ ), eighth ( $n = 1$ ) and ninth ( $n = 1$ ) lactation were included. In the vitamin E group, 10 cows were in their second ( $n = 10$ ), third ( $n = 3$ ), and fourth ( $n = 2$ ) lactation. Cows in the CLA + vitamin E group were in their second ( $n = 5$ ), third ( $n = 4$ ), fifth ( $n = 1$ ), and eighth ( $n = 2$ ) lactation. The study was divided into 3 periods: period 1 from 42 d a.p. until calving, period 2 from calving until d 21 postpartum (**p.p.**), and period 3 from d 21 until 70 p.p.

The animals were fed a standardized partial mixed ration (**PMR**) during the whole experiment, which was provided ad libitum by self-feeding stations (type RIC, Insentec B.V., Marknesse, the Netherlands). Concentrate was administered at 3 kg/d per animal via computerized self-feeding stations (Insentec B.V.).

The components and the chemical composition of the feedstuffs are presented in Table 1. During the first period the ration was composed of 60% concentrate and 40% silage (50% maize, 50% grass silage on a DM basis). After parturition the concentrate proportion increased from 30 to 50% until d 21 p.p., where it remained until d 70 p.p. The treatment groups received either conjugated linoleic acid (BASF Lutrell, Lampertheim, Germany) containing 8.4 g of *trans*-10,*cis*-12 and 8.4 g of *cis*-9,*trans*-11 per day per animal (**CLA**,  $n = 16$ ), 2,327 IU of vitamin E/d per animal (BASF Lutavit E 50; **Vit. E**,  $n = 15$ ), or both supplements (**CLA + Vit. E**,  $n = 12$ ) from d 42 a.p. to 70 p.p. The control group ( $n = 16$ ) as well as the Vit. E group received a

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