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Palmitic and Stearic Acids: Metabolism and Tissue Utilization

The use of palmitic acid (C16:0), stearic acid (C18:0), and oleic acid (C18:1) in long chain fatty acid (LCFA) inert supplements for dairy cattle is a common practice. Until recently, there has been a shortage of knowledge of how these fatty acids (FA) are metabolized and utilized by the lactating cow in the public domain. Several recent short term production studies involving feeding highly concentrated C16:0 supplements (>80%) have reported improved milk fat tests and feed efficiency while others have not. Our understanding of how the cow metabolizes and utilizes these individual FA is required to help dairymen make an informed decision on the FA composition of their LCFA supplement.

In the cow, adipose tissue contains 27% C16:0, 11% C18:0, and 48% (C18:1). It is interesting to note that nearly all of the C18:0 and C18:1 in adipose tissue is from the elongation of C16:0 to C18:0, and then the C18:0 is desaturated by stearoyl CoA desaturase to form C18:1 (Smith et al., 2006). C18:0 is a poor source for lipogenesis in adipose tissue, while C16:0 is an excellent source. In cows' milk, C16:0 is the most plentiful at 27-30%, while C18:0 is 9-12%, and C18:1 is 26-28% (Table 1). However, nearly all of the C18:1 is derived from the desaturation of C18:0 in mammary tissue. This would indicate that nearly 40% of the total milk FA are from C18:0. This establishes C18:0 as a very important FA in milk fat synthesis. Milk also contains about 27% medium and small chain FA from C4-C15 (Palmquist et al. 1993). C16:0 can be either absorbed from the gut and directly transported into milk fat by plasma triglycerides (PTG), or about 50% of the C16:0 in milk fat can be from de novo synthesis. The C18:0 and C18:1 as well as the remainder of LCFA in milk fat come directly from PTG transfer. The mammary tissue does not have the ability to elongate C16:0 to C18:0 as it does in adipose tissue due to the absence of necessary enzymes. That is why C16:0 is a terminal FA when it comes to milk fat synthesis.

Table 1. Milk fatty acid composition of cows' milk from 50 different processing plants across the U. S.

Milk FA	C4:0	C6:0	C8:0	C10:0	C12:0	C14:0+ C14:1	C16:0	C16:1	C18:0	C18:1	C18:2
% of Total FA	3.3	2.3	1.2	2.8	3.4	14.0	29.5	3.4	9.8	27.4	2.8

Adapted from Palmquist et al., 1993.

Table 2. Fatty acid composition of tissues in pre- and post-partum dairy cows[1]

	Day relative to parturition			
Tissue, g /100 g FA	-45	1	21	65
Adipose				
C16:0	27.0	27.5		
C18:0	10.7	10.8		
C18:1 *cis* 9	49.4	48.1		
Liver triacylglycerols				
C16:0	26.8	42.3a	39.0a	26.0b
C18:0	25.5	10.6b	12.2b	24.7a
C18:1 *cis* 9	23.9	26.6a	26.6a	17.2b
Plasma				
C16:0	16.7	18.2a	14.5b	12.2c
C18:0	16.5	15.6a	13.9b	13.7b
C18:1 *cis* 9	18.0	19.6a	20.1a	14.5b

[1]Adapted from Douglas et al. (2007).

While C16:0 and C18:0 only differ by 2 carbon units, their metabolism and utilization in tissues of dairy cows is quite different. Douglas et al. (2007) found proportions of C16:0, C18:0 and C18:1 differed among blood and body tissues and changed with the onset and progression of lactation (Table 2). Weight percentages of C16:0, C18:0 and C18:1 in plasma were similar during the dry period, but following parturition, C16:0 and C18:1 increased whereas C18:0 decreased (Table 2).

The key differences in C16:0 and C18:0 utilization by the cow can be found in liver and mammary tissue. Prior to and shortly after parturition, plasma NEFA concentrations lead to increased hepatic uptake of FA, their subsequent esterification, and accumulation of TAG in the liver (Grummer, 1993). Drackley (1999) stated the liver is prone to increased NEFA esterification around calving. Research by Contreras and Sordillo (2011) suggested that excessive accumulation of lipid components in hepatocytes and other cells could cause physical damage including compression and reduction in size and number of organelles in transition cows. Several research trials have shown that after parturition, C16:0 increases in liver tissue while

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C18:0 does not (Rukkwamsuk et al., 2000, Mashek and Grummer, 2003, Sato and Inoue, 2006, and Douglas et al., 2007). These data indicate that C18:0 does not accumulate in tissues of cows in negative energy balance and cows metabolize C18:0 for energy, e.g. beta oxidation, in the liver and muscle and/or secrete large proportions of C18:0 through milk as both C18:0 and C18:1. There is the possibility that a high C18:0 and a low C16:0 LCFA supplement may be advantageous in the close up period and early lactation. C16:0 appears to have a greater chance to increase in liver tissue postpartum than C18:0.

C18:0 may be better oxidized by the liver or used as an energy source during late prepartum and early postpartum periods compared to C16:0. Karcagi et al. (2010) reported that feeding a diet containing hydrogenated palm oil (HTG; 69% C18:0 and 23% C16:0) provided a better energy supply for high-yielding dairy cows in negative energy balance than calcium salts of palm fatty acid distillate (CaS; 33% C16:0 and 4% C18:0). Diets were fed for 25 days prepartum and at 5 days prepartum there was significantly less triglycerides in the liver of cows fed HTG than either the control-no fat or CaS. Cows fed HTG consumed an average of 2.1 kg/d more diet DM and produced 7.6 kg/d more 4% FCM during the first 100 d postpartum than cows fed CaS.

Feeding supplemental C16:0 can increase body tissue reserves of cows when fed during positive energy balance, but the energy stored will be in the form of C18:0 rather than C16:0. More research with lactating dairy cows is required to establish this hypothesis. Most of the C18:0 in adipose tissue likely comes from elongation of C16:0 and not from direct uptake and esterification of C18:0 into triacylglycerol. The preferred substrate for synthesis of triglycerides is C16:0, whereas C18:0 itself is a poor substrate for triglyceride synthesis in adipocytes (Sampath and Ntambi, 2005).

The differences in metabolism and utilization of C16:0 and C18:0 by the lactating cow have been discussed. The major issues concerning C16:0 is the accumulation of this FA in liver triglycerides after calving and the potential for damage to liver cells if fatty liver syndrome develops. C18:0 does not accumulate in the liver and it appears that C18:0 is preferentially utilized as an energy source by the early lactation cow in negative energy balance. The excess C16:0 that is not incorporated into milk fat or oxidized for energy in the liver is deposited in adipose tissue primarily as C18:1 through elongation and desaturation. A higher C18:0 LCFA supplement in early lactation may be warranted.

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