Cows Need Both C16 and C18 Fatty Acids

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Introduction

During the past decade, fatty acid (**FA**) research has been focused on discovering the optimal combination of FA to feed to lactating cows for the purpose of improving milk yield (**MY**) and milk components. Most long chain fatty acid (**LCFA**) supplements contain either combinations of palmitic (**C16:0**) and oleic acids (**C18:1**), highly enriched C16:0 (>80%), or C16:0, stearic acid (**C18:0**), and C18:1. These supplement categories have substantial published research trials over the past 30 years. Nutritionists, researchers, and dairymen are continuing to search for the most optimal combination of these three LCFA. New information continues to enhance our knowledge of the metabolism and utilization of these LCFA for the purpose of improving MY, milk fat (**MF**), milk protein (**MP**), and reproduction by improving energy balance (**EB**) in early lactation. These LCFA are intimately involved in the metabolism of the lactating cow and have specific functions in the production of milk and milk components. Palmitic acid has been shown to improve milk fat % and yield. However, supplementing C16:0 has no effect on MY, body weight gain or body condition score (**BCS**). Stearic acid has been observed to have a positive influence on dry matter intake (**DMI**) and the yield of milk, MF, MP, and milk lactose. Combinations of C16:0 and C18:0 have been shown to improve MY, milk components, and improve EB in early lactation. Thus, the importance in discovering the proper ratio and feeding rates of these LCFA to improve performance is of interest.

Fatty Acids That Enter The Rumen Are Not What Leaves The Rumen

Palmitic and C18:0 are saturated LCFA which have little effect on ruminal microbial populations and are considered rumen inert. Wu et al. (1991) observed quantities of C18:0 leaving the rumen were several fold higher than the amount fed, while C16:0 is similar to the amount fed. Loor et al. (2004) observed that while C18:0 was only 2.1 to 2.4% of the total FA fed in a high (65%) or low concentrate (35%) diet, the amount flowing into the duodenum was ~25 times higher than the amount fed. Stearic acid accounted for 46 to 39% of the total FA flow leaving the rumen in the low- and high-concentrate diets, respectively. The flow of C18:0 from rumen to duodenum is an evolutionary phenomenon that emphasizes the importance of C18:0 to the lactating cow. Substantial microbial biohydrogenation of mono and polyunsaturated C18 fatty acids (**PUFA**) leads to the several fold increase in duodenal C18:0. While much emphasis has been placed on reducing biohydrogenation of PUFA to positively affect milk FA composition, little research has been conducted to determine just how important C18:0 is to the metabolism of the lactating cow as well as the dual presence of C16:0

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and C18:0 in the diet.

Digestibility of C16:0 and C18:0

When entering the abomasum, most FA are calcium, potassium, and sodium salts and mixed in an insoluble particulate phase of feed particles and microbial cells. These salts are dissociated and protonated to a great extent in the abomasum due to low pH, and enter the duodenum mostly as non-ionized free FA (**FFA**). These FFA, if not absorbed, may reform as salts as pH increases in the duodenum and ileum. Several research trials and reviews have reported the digestibility values for C16 and C18 LCFA. These are summarized in **Table 1**. The apparent digestibility of C16:0 and C18:0 is very similar and averages 79.6% and 78.3% respectively. Bauman et al. (2003) concluded that the rumen outflow of lipids are predominantly FFA and differences in the digestibility of individual fatty acids in the small intestine are negligible. Thus, the composition of FA absorbed in the small intestine is similar to the composition of FA leaving the rumen. Boerman et al. (2015) reported in their meta-analysis that C16:0 and C18:0 have similar digestibility averaged across published studies. However, as the quantity of C18:0 increased in the duodenum, the corresponding digestibility declined linearly. Loften et al. (2014) summarized that even if the percentage absorption of C18:0 is decreased at high flows into the duodenum, it likely has limited significance because more C18:0 is present in the small intestine than any other FA and, therefore, the quantity absorbed relative to other FA is always much greater.

Metabolism in Tissues

Ruminant adipose tissue is active in both lipogenesis and lipolysis. The major FA concentrations in adult ruminant adipose tissue are C18:1, followed by C16:0, and then C18:0. Choi et al. (2013) reported the C16:0, C18:0, and C18:1 adipose tissue concentrations as 27.9%, 10.4%, and 42.9%, respectively in feedlot steers fed a low fat basal diet. Douglas et al. (2007) reported the C16:0, C18:0, and C18:1 adipose tissue concentrations as 27.0%, 10.7%, and 48.6% in dairy cows prior to calving. The FA in adipose cells come from both diet and de novo synthesis.

Determining the amount of dietary FA uptake in adipose tissue is difficult due to the flux between lipogenesis and lipolysis occurring constantly in a dynamic state. Summers et al. (2000) estimated that slightly more than 10% of saturated FA were stored in human adipose tissue compared to those that were consumed. Mitchaothai et al. (2007) fed finishing swine a diet containing 5% sunflower oil for 13 weeks resulting in the consumption of 1.24 kg and 0.21 kg of C16:0 and C18:0, respectively. They observed 3.75 kg and 2.39 kg of C16:0 and C18:0 deposited in adipose tissue. The ratio of FA deposition:FA intake for C16:0 was 3:1, whereas C18:0 was 11.9:1. These results in monogastrics indicate that dietary FA may be found in adipose tissue, but the majority of FA deposited in adipose tissue is from de novo synthesis. This is true in ruminants as well. The basic building block for de novo lipogenesis is acetyl-CoA which is derived primarily from acetate and glucose (Hellerstein et al.,1996; Vernon, 1981).

Choi et al. (2013) fed either 1) no added lipid, 2) 3% palm oil, 3) or 3% soybean oil to finishing steers to determine if fat sources differing in FA composition would alter FA composition of adipose tissue. The results are illustrated in **Table 2**. Steers fed 3% palm oil or 3% soybean oil did not show an increase in C16:0 concentration in subcutaneous adipose tissue. The only significant change in FA composition was C18:0. However, C18:0 increase in adipose tissue is likely due to C16:0 elongation to C18:0, and because stearoyl-CoA desaturase activity, which converts C18:0 to C18:1, was inhibited by C16:0 (Choi et al., 2013). Synthesis of FA beyond C16:0 does not occur in ruminant adipose tissue, but through a family of elongation enzymes (**ELOV**), C18:0 is produced from C16:0. Stearic acid is then desaturated to C18:1 by the enzyme stearoyl-CoA desaturase. The primary purpose of desaturation is to regulate fluidity of adipose cells from a buildup of high melting point (solid) C18:0 and loss of membrane integrity. Thus, C18:1 is the predominant FA stored in ruminant adipose tissue. Burns et al. (2012) found C16:0 and C16:1 to be regulators of lipogenesis, desaturation, and apoptosis in adipose cells. Thus, C16:0, C18:0, and C18:1 provide the structure of the tissue while maintaining the fluidity of adipocytes preventing their premature apoptosis. The practical implication of decreased adipogenesis with higher amounts of C16:0 through either dietary sources and/or de novo synthesis in adipose cells is potential weight and body condition loss. In short term feeding studies, both Warntjes et al. (2008) and Piantoni et al. (2013) reported numerical decreases in BCS of cows fed C16:0 compared to cows fed control diets.

In the liver, shortly before and after parturition, plasma NEFA concentrations lead to increased hepatic uptake of FA, their subsequent esterification, and accumulation of triglycerides (Grummer, 1993). Douglas et al. (2007) measured the effects of prepartum nutrition on LCFA composition of total lipids in plasma, adipose tissue, and liver, and whether dry period effects persisted (**Table 3**). Hepatic triglycerides (**TG**) contents of C16:0, C18:0, and *cis* C18:1 were similar in the dry period; but, following parturition, C16:0 and *cis* C18:1 increased compared with 45 d prepartum by 58% and 11%, respectively, while C18:0 decreased 42%. Other studies, Rukkwamsuk et al., (2000) and Litherland et al. (2012) found similar results. Mashek and Grummer (2003a) observed no net uptake of C18:0 in the caprine liver when 0.3 m*M* concentrations of C16:0 and C18:0 were perfused into the caudate lobe. They observed that C16:0 uptake was significantly increased compared with C18:0. Mashek and Grummer (2003b) observed C16:0 oxidation doubled when C18:0 was added to bovine cell hepatic cultures compared with C16:0 alone. This may indicate a role for C18:0 in aiding hepatic tissue clear excess C16:0 that collects in hepatic tissue before and after parturition. Loften et al. (2014) concluded that these data indicate that C18:0 does not accumulate in tissues of cows in negative EB and cows preferentially metabolize C18:0 for energy (e.g., βoxidation) in the liver and muscle or secrete large proportions of C18:0 through milk as both C18:0 and C18:1. From these data, Linn and Loften (2015) concluded C18:0 may be better oxidized by the liver or used as an energy source during late prepartum and early postpartum periods than C16:0.

White et al. (2011) suggested that the circulating FA that are characteristically increased in transition cows may contribute to increased expression of pyruvate

carboxylase mRNA to stimulate gluconeogenesis and maintain oxaloacetate for the tricarboxylic acid cycle. Stearic acid was shown to regulate pyruvate carboxylase promoters (P1, P2, and P3) in different tissues, with C18:0 suppressing promoter P1 and enhancing promoter P3 activity simultaneously. These data suggest that C18:0 contributes to the partitioning of energy during periods of upregulated gluconeogenesis, increased hepatic FA supply, or both. This would suggest that C18:0 may spare glucose in early lactation when negative EB occurs.

Mammary Tissue

Palmitic acid and C18:0 are intimately involved in the synthesis of milk and milk fat. Both FA can be oxidized to supply energy for overall synthesis of milk and milk components. Numerous studies in the literature have evaluated fat supplementation to lactating dairy cows; however, most of these studies were with supplements containing mixtures of FA. Very few studies have looked at feeding only a single purified form of a FA. The classic studies of Steele and Moore (1968a,b), Noble et al. (1969), and Steele (1969) were some of the first to look at effects of feeding a purified source of C16:0 on milk yield and milk components. Steele and Moore (1968a) fed 578 g/d of highly purified C16:0, which increased milk fat percentage by 0.86% units and increased the amount of C16:0 in milk fat almost 2-fold, but had no effect on milk yield of 12.2 kg/d for control and 11.8 kg/d for C16:0-supplemented cows. In a later study (Steele, 1969), when 448 g/d of C16:0 was fed as a replacement for starch in diets of lactating cows, milk yield increased by 1 kg/d for cows fed 16:0 compared with control cows. In all 3 studies, feeding C16:0 increased milk fat percentage and yield of C16:0 in milk fat, but concentration and yield of C4 to C14 FA, along with C18:0 and C18:1 in milk fat, decreased. **Table 4** illustrates the effects of C16:0 and C18:0 on milk FA yield. The observed effects of feeding C16:0 at high levels are suppressed de novo synthesis and reduced C18 in milk fat. Noble et al. (1969) concluded that acetyl CoA carboxylase is inhibited by the mammary uptake of LCFA, in this case, primarily C16:0. The more recent studies show similar responses in reducing de novo synthesis of milk FA. The basic effect of feeding highly enriched C16:0 is the 2-4 fold increase in C16:0 in milk fat at the expense of de novo synthesis and C18:0 and C18:1 in milk fat.

Several recent studies have shown improvements in MF% and yield when C16:0 was fed to lactating cows across different production levels. **Table 5** includes an average of 8 research trials with similar design, period length, and diets. The average response measured in these studies reveals that highly enriched palmitic acid improved fat test from 3.70% to 3.87%, reduced DMI by 1.5 lb/d, did not increase MY(0.04 lb./d), reduced MP% from 3.20% to 3.16%, and reduced milk lactose from 4.75% to 4.71%. These data show the ability of feeding highly enriched C16:0 to improve MF% and MF yield. However, the reduction of DMI aids in the explanation of the absence of a MY increase and a reduction of milk lactose % and MP%. In these studies, the absence of improved MY when 428 g/d of C16:0 were fed causes the economic return to be based solely on MF yield.

There have been two recent trials (Boerman and Lock, 2014; Piantoni et al., 2015) where highly enriched C18:0 was fed to lactating cows. Both studies observed significantly higher DMI when C18:0 was fed from 200-700 g/d. The results of the Piantoni et al. (2015) study are shown in **Table 6**. Feeding 500 g/d of a 98% highly enriched C18:0 resulted in significant increases in DMI and yields of milk, MF, MP, lactose, 3.5% FCM, and ECM while not affecting milk component concentration. The intake of enriched C18:0 resulted in a significant increase in de novo, mixed, and preformed FA yields, quite in contrast to previously mentioned C16:0 trials. The authors also observed a significant interaction between production level and C18:0 supplementation. Lactating cows yielding < 60 lb/d of milk showed very little increase in 3.5% FCM, while those producing > 120 lb/d were observed to increase in excess of 10 lb/d. This illustrates the potential glucose sparing effects of C18:0 as indicated by White et al. (2011). Lactating cows requiring higher energy intake and circulating glucose responded with the highest increases in 3.5% FCM when fed enriched C18:0, while low producing cows partitioned energy from milk and MF production to other body functions.

Conclusions

The importance of C16:0 and C18:0 in the production of milk and milk components has been discussed. Each FA has separate functions, metabolism, and utilization. Feeding either FA separately in an enriched form results in different improvements in performance. Results of these studies illustrate the need for both C16:0 and C18:0 in the LCFA supplement to elicit maximum response to lactating cows. Research is underway to determine the optimal ratio of C16:0 to C18:0 in early, mid, and late lactation.

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Table 1. Apparent digestibility of long chain fatty acids from 6 published studies and reviews.

	Published studies						
	Doreau and Chilliard 1997	Enjalbert et al. 1997	Scollan et al. 2001	Lock et al. 2006	Glasser et al. 2008	Boerman et al. 2015	Average
	Fatty acid digestibility %						
C16:0	79	76	92	75	NA	76	79.6
C18:0	77	79	95	72	74	73	78.3
C18:1	85	78	89	80	79	82	82.2
C18:2	83	66	73	78	72	78	75.0
C18:3	76	63	72	77	70	79	72.8

Table 2. Fatty acid composition of subcutaneous adipose tissue of feedlot steers fed palm oil or soybean oil.[1]

Fatty acid %	Control	3% Palm Oil	3% Soybean Oil	*P* value
C16:0	27.9	27.0	26.7	0.09
C18:0	10.4[a]	12.6[b]	12.6[b]	0.02
C18:1	42.9	42.7	42.9	0.22
C18:2	1.84	1.90	2.04	0.18

[a,b] Means in rows not bearing a common superscript differ, $P < 0.05$.
[1] Adapted from Choi et al. (2013).

Table 3. Fatty acid composition of tissues in pre- and post-partum dairy cows.[1]

Tissue	Day relative to parturition			
g/100 g of FA	-45	1	21	65
Adipose				
C16:0	27.0	27.5		
C18:0	10.7	10.8		
C18:1	49.4	48.1		
Liver TG				
C16:0	26.8	42.3[a]	39.0[a]	26.0[b]
C18:0	25.5	10.6[b]	12.2[b]	24.7[a]
C18:1	23.9	26.6[a]	26.6[a]	17.2[b]
Plasma				
C16:0	17.7	18.2[a]	14.5[b]	12.2[c]
C18:0	16.5	15.6[a]	13.9[b]	13.7[b]
C18:1	18.0	19.6[a]	20.1[a]	14.5[b]

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

Table 4. Effects of feeding C16:0 and C18:0 on milk fatty acid yield.

		Fatty acid fed, g/d	Milk fatty acid yield, g/d		
			De novo	Mixed	Preformed
Steele and Moore 1968a					
	Control	0	82	152	78
	C16:0	578	62	297	86
	C18:0	564	83	133	103
Noble et al. 1969					
	Control	0	143	206	106
	C16:0	448	110	338	112
	C18:0	448	110	138	228
Recent trials[1]					
	Control	0	338	127	436
	C16:0	428	305	543	414

[1] Trials included reported milk fatty acid composition and yield (Piantoni et al., 2013; Lock et al., 2013; Rico et al., 2014; Boerman et al., 2015; and de Souza et al., 2017.)

Table 5. Effect of feeding palmitic acid supplements to lactating cows and milk composition from 8 trials utilizing similar design. **[1]**

		Measures				
Treatment	C16:0 Intake lb/d	DMI lb/d	Milk yield lb/d	Milk fat %	Lactose %	Milk protein %
Control	0	58.3	84.24	3.70	4.75	3.20
Palmitic acid	428	56.8	84.28	3.87	4.71	3.16
Palmitic acid minus Control	428	-1.5	0.04	0.17	-0.04	-0.04

[1] Studies included Lock et al., 2013; Piantoni et al., 2013, Rico et al., 2014; Garver et al., 2015; Boerman et al., 2015; DeSouza et al., 2017.

Table 6. The effects of feeding highly enriched stearic acid on milk yield, milk components, and milk fatty acid yield in lactating dairy cows.[1]

Item		Control	Stearic acid[a]	+/-	*P* value
DMI	lb/d	55.4	57.4		<0.01
Yield					
Milk	lb/d	84.7	88.4	3.7	0.02
Milk fat	g/d	1350	1420	70	<0.01
Milk protein	g/d	1140	1190	50	0.02
Lactose	g/d	1870	1960	90	0.02
3.5% FCM	lb/d	84.0	89.1	5.1	<0.01
ECM	lb/d	84.0	88.2	4.2	<0.01
Composition					
Milk fat	%	3.60	3.59	-0.01	NS
Milk protein	%	3.00	2.99	-0.01	NS
Lactose	%	4.83	4.86	0.03	NS
Milk fatty acids					
De novo	g/d	344	359	15	<0.0001
Mixed	g/d	451	461	10	<0.01
Preformed	g/d	352	393	41	<0.001
TOTAL	g/d	1147	1213	66	<0.01
Transfer efficiency	%		12.9%		
Total FA digestibility	%	76.1	56.6	-19.50	<0.0001
16 C	%	76.2	75.8	-0.40	0.79
18 C	%	79.1	55.3	-23.80	<0.0001

[a] Included in diet at 2% of the DMI or 522 g/d of 98% C18:0 per day.
[1] Adapted from Piantoni et al. (2015).

SESSION NOTES