Concepts in Lipid Digestion and Metabolism in Dairy Cows

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Introduction

Fat and fatty acid metabolism and digestion in the dairy cow are of considerable interest, both to scientists and the dairy industry. This renewed interest is based on several reasons; first, the use of dietary fat supplements has increased, and will continue to do so, as nutritionists strive to increase the energy density of diets to meet requirements of the high producing dairy cow; second, we now recognize that fatty acids, both of dietary and rumen origin, can have specific and potent effects on ruminant metabolism and human health; and third, we now recognize that specific fatty acids produced in the rumen are potent regulators of milk fat synthesis in the mammary gland. Our objective in this review is to provide an overview of lipid metabolism in the dairy cow. Our focus will include the biological processes and quantitative changes occurring during the metabolism of fatty acids in the rumen and their subsequent absorption in the small intestine. In addition, we will discuss the interrelationship between rumen lipid metabolism and milk fat synthesis, and dietary factors that result in milk fat depression.

Lipid Metabolism in the Rumen

Extensive metabolism of lipids occurs in the rumen and this has a major impact on the profile of fatty acids available for absorption and tissue utilization. The two major processes that occur are hydrolysis of ester linkages in lipids found in feedstuffs and the biohydrogenation of unsaturated fatty acids (Figure 1). Hydrolysis of dietary lipids is predominantly due to rumen bacteria, and although the extent of hydrolysis is generally high (>85%), a number of factors that affect the rate and extent of hydrolysis have been identified. For example, the extent of hydrolysis is reduced as the dietary level of fat is increased or when factors such as low rumen pH and ionophores inhibit the activity and growth of bacteria (see reviews by Doreau et al., 1997; Harfoot and Hazlewood, 1997). Unsaturated fatty acids are toxic to many rumen bacteria, so the second major transformation that dietary lipids undergo in the rumen is biohydrogenation of polyunsaturated fatty acids **(PUFA)**. Biohydrogenation requires a free fatty acid to proceed; as a consequence, rates are always less than those of hydrolysis, and factors that affect hydrolysis also impact biohydrogenation. Classical pathways of ruminal biohydrogenation were established using pure cultures of rumen organisms and the bacteria involved in biohydrogenation have been classified into two groups, A and B, based on their metabolic pathways (Kemp and Lander, 1984). To obtain complete biohydrogenation of PUFA, bacteria from both groups are generally required. Although Group A contains many bacteria that can hydrogenate PUFA to *trans* 18:1 fatty acids, only a few species characterized as Group B can carry out the last step and hydrogenate the *trans* 18:1 fatty acid to stearic acid (Harfoot and Hazlewood, 1997). This feature of biohydrogenation explains why increased feeding

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of PUFA simultaneously causes an increase in the rumen concentration of monounsaturated fatty acids and a decrease in the concentration of saturated fatty acids.

The rates of rumen biohydrogenation of fatty acids are typically faster with increasing unsaturation, and based on available data from lactating dairy cows, linoleic and linolenic acids are hydrogenated to the extent of 70 to 95% and 85 to 100%, respectively (Lock et al., 2005; 2006). These averages are in agreement with values by Doreau and Ferlay (1994) who reviewed data from all ruminant species. Therefore, the extensive metabolism of dietary unsaturated fatty acids in the rumen results in stearic acid being the major fatty acid entering the duodenum. Figure 2 illustrates this based on linoleic acid intake (separated into tertiles) and compares changes in intake and duodenal flow (i.e. rumen output) of linoleic and stearic acids. Linoleic acid is generally the most common fatty acid present in diets for U.S. dairy cows and the intake varies widely; however, only a fraction of the linoleic acid consumed is actually available for absorption. On the other hand, typically very little stearic acid (18:0) is consumed, but we see a reciprocal increase in stearic acid flow to the duodenum (Figure 2) as a result of it being the end product of biohydrogenation of all 18-carbon unsaturated fatty acids (oleic, linoleic, and linolenic). Despite the dramatic changes that occur in the rumen, outflow of total fatty acids is very similar to dietary intake of fatty acids and this is true across a wide range of diets with different fatty acid intakes (Doreau and Ferlay, 1994; Lock et al., 2005). Therefore, an accurate determination of fatty acid intake will allow for a reasonable approximation of duodenal flow of total fatty acids, although the profile of the fatty acids will be vastly different (Figure 2). While simple in principle, an accurate determination of fatty acid intake can present some challenges, often due to the overestimation of total fatty acid content of forages and the difficulty in obtaining complete lipid extraction of highly saturated fat supplements (see review by Palmquist and Jenkins, 2003).

Fat supplements are used as a means to increase the energy density of the diet and many of these are referred to as inert. In this case, inertness simply means that the fat or fatty acid supplement has minimal affects on rumen fermentation. Although deemed inert at the level used, they can still be hydrolyzed if a triglyceride, or hydrogenated if unsaturated (Figure 1). Often, calcium soaps of palm fatty acids or canola are referred to as 'protected'. However, these are not protected from ruminal biohydrogenation, but they are rather ruminally inert with regard to their effects on the microbial population (Palmquist, 2006). Factors such as low rumen pH and increased unsaturation of the fatty acid can lead to dissociation of the Ca-fatty acid complex, allowing biohydrogenation to occur (Demeyer and Doreau, 1999). Thus, the feeding of a Ca-salt of unsaturated fatty acids will "protect" against adverse effects on microbial fermentation, but in most cases, it will not increase either the bypass of these fatty acids to the duodenum (Lundy et al., 2004) or their content in milk fat (Castañeda-Gutiérrez et al., 2005) compared with the feeding of the parent oil.

Improvements in analytical techniques have revealed an impressive complexity in the pattern of fatty acids that are produced during rumen biohydrogenation and subsequently incorporated into milk fat. Table 1 summarizes published data for lactating dairy cows and illustrates the range of positional and geometric isomers of *trans* 18:1 and conjugated linoleic acids **(CLA)** identified in the lipid material leaving the rumen. The established major pathways of biohydrogenation describe the formation of *trans*-11 18:1 and *cis*-9, *trans*-11 CLA but do not account for the fatty acid intermediates arising from minor pathways of rumen biohydrogenation. This is an area of increasing interest because of the recognition that some of these biohydrogenation intermediates have specific and

potent effects on ruminant metabolism and human health. For example, both *trans*-11 18:1 and *cis*-9, *trans*-11 CLA present in milk fat have been shown to have anticarcinogenic and antiatherogenic properties in animal models of human health (Bauman et al., 2006), while the role of *trans*-10, *cis*-12 CLA as a regulator of milk fat synthesis in dairy cows will be discussed later in this review. For further information on the biology of hydrolysis and biohydrogenation, and the effects of diet on these processes, the reader is referred to our recent review (Palmquist et al., 2005), as well as the classic review by Harfoot and Hazlewood (1997).

Fatty Acid Absorption in the Small Intestine

Since there is no significant absorption or modification of long and medium chain fatty acids in the omasum or abomasum, the lipid material available for absorption in the small intestine is similar to that leaving the rumen (Moore and Christie, 1984). This lipid material consists of approximately 80 to 90% free fatty acids attached to feed particles; the remaining lipid components are microbial phospholipids plus small amounts of triglycerides and glycolipids from residual feed material, which are hydrolyzed by intestinal and pancreatic lipases (Doreau and Ferlay, 1994). Before fatty acid absorption can occur, it is necessary for the fatty acids adsorbed on feed particles to be solubilized into the aqueous milieu. In all species, micelle formation is the key to this solubilization process, and therefore, key to efficient fatty acid absorption. In ruminants, both bile and pancreatic secretions are required for this process, and these are added to the digesta in the duodenum. Bile supplies bile salts and lecithin, and pancreatic juice provides the phospholipase enzymes to convert lecithin to lysolecithin and the bicarbonate to raise the pH. Lysolecithin, together with bile salts, desorb the fatty acids from feed particles and bacteria, allowing the formation of the micelle (Figure 3). The critical role of lysolecithin and bile salts in this process is illustrated in studies with sheep where fatty acid absorption was virtually abolished when bile secretion into the duodenum was blocked (Moore and Christie, 1984). Once micelles are formed, they facilitate transfer of water-insoluble lipids across the unstirred water layer of intestinal epithelial cells of the jejunum, where the fatty acids and lysolecithin are absorbed. Within the intestinal epithelial cells, the fatty acids are re-esterified into triglycerides and then packaged into chylomicrons for transport in lymph to the blood.

To allow for efficient intestinal absorption, ruminants have evolved a number of key differences and features in fatty acid absorption compared with non-ruminants. First, ruminant bile is characterized by an excess of taurine-conjugated bile acids. In the majority of herbivores, glycine-conjugated bile acids predominate, but in the mature ruminant, taurine-conjugates exceed glycine-conjugates approximately 3:1 (Noble, 1981). This is of significance because under the acidic conditions of the ruminant upper-small intestine, taurine-conjugated bile acids remain in a partially ionized condition and in the micellar phase where they are able to effect solubilization of fatty acids (Noble, 1981). Even at pH 2.5, taurine-conjugated bile acids remain soluble and partly ionized, while glycine-conjugated bile acids are insoluble in much less acidic conditions (pH 4.5) and unable to effect solubilization (Moore and Christie, 1984). Second, there are significant differences between ruminants and non-ruminants in the source of amphiphile or 'swelling agent', which promotes micelle formation. In ruminants, lysolecithin is the amphiphile involved in micelle formation, whereas monoglycerides plus bile salts interact with the fatty acids to form the micelle in non-ruminants (Davis, 1990). Freeman (1969) examined the amphiphilic properties of polar lipid solutes and found that lysolecithin had a pronounced effect on the micellar solubility of stearic acid (Table 2). In fact, lysolecithin's ability to increase the solubility of stearic acid is ~2-fold greater than that of other amphiphiles, including oleic acid which has been quoted recently as having

important amphiphilic properties when fed as a Ca-salt to ruminants (Moate et al., 2004; Block et al., 2005). Lysolecithin was, furthermore, the only amphiphile examined which was shown to significantly increase the distribution of stearic acid into the micellar phase and away from the particulate phase (Table 2). Considering that most fatty acids leaving the rumen are saturated and the predominant fatty acid is stearic acid, perhaps it is not surprising that the ruminant has evolved such an efficient system involving lysolecithin for solubilizing this fatty acid.

Our review of the available data from lactating dairy cows indicates that fatty acid absorption is relatively constant with no significant decline when fatty acid duodenal flow was high (Lock et al., 2005). Total fatty acid digestibility averaged 74% with a range (95% confidence interval) of 58 to 86%. These data are in agreement with Doreau and Ferlay (1994), who carried out an extensive review of the literature for all ruminant species and reported values for fatty acid digestibility ranging from 55 to 92%; again, this range was not related to fatty acid intake. One consideration is whether differences exist in the digestibility of individual fatty acids, with the digestibility of stearic acid in dairy cows in relation to the digestibility of other fatty acids being of particular interest. In general, the ability of ruminants to absorb fatty acids is much higher than that of non-ruminants (Noble, 1981). In non-ruminants, there is a wide divergence in the digestibility of fatty acids (Freeman, 1984), with the digestibility of individual fatty acids decreasing when chain length increases and increasing as the number of double bonds increases (Lessire et al., 1992). In particular, free palmitic and stearic acids are poorly absorbed in non-ruminants (Noble, 1981). However, as illustrated in Figure 4, although similar patterns are observed in ruminants, relative differences in the digestibility of individual fatty acids are modest; mean digestibilities for 16:0, 18:0, 18:1, 18:2, and 18:3 were 75, 72, 80, 78, and 77% (Lock et al., 2005). These data are in agreement with the review of Doreau and Ferley (1994), which reported that mean digestibilities were 77, 85, 83 and 76% for 18 carbon fatty acids with zero, one, two, and three double bonds, respectively.

Recent improvements in analytical techniques will allow the digestibility of individual fatty acids to be more thoroughly examined. However, application in feeding systems still requires accurate information on the profile of fatty acids leaving the rumen. Figure 4 also illustrates the considerable variation in the digestibility of individual fatty acids across studies. The overall conclusion is that differences in digestibility among individual fatty acids contribute very little to the extensive variation reported in the literature (range ~60 to 90%). Rather, the majority of this variation reflects differences among individual experiments, and thus relates to experimental approaches and analytical techniques as well as differences in diets and specific feed components. As emphasized earlier, stearic acid is the predominant fatty acid in the digesta and consequently is the major contributor to total absorbed fatty acids. Therefore, any discrimination against the absorption of stearic acid relative to the other fatty acids may be hardly noticeable since this is the predominant component in the digesta and more is absorbed than of any other fatty acid (Noble, 1981). Consequently, the composition of absorbed fatty acids is close to the composition of fatty acids entering the duodenum.

Milk Fat Depression

Nutrition is the predominant environmental factor affecting milk fat and represents a practical tool to alter its yield and composition. One of the most striking examples of nutritional effects on milk fat is the low fat milk syndrome, typically referred to as milk fat depression **(MFD)**, and our understanding of its etiology has advanced significantly in recent years. The MFD has been observed over a range of feeding situations, including

diets supplemented with fish oils or plant oils, and diets high in concentrates and low in fiber **(HC/LF)** (Bauman and Griinari, 2001). The fat content of milk can also be affected by the physical characteristics of the roughage (e.g. grinding or pelleting) or use of ionophores such as Rumensin® (Elanco, Greenfield, IN).

The MFD is properly diagnosed by an observed reduction in milk fat yield, as milk fat percentage can be influenced by a change in milk volume with no actual change in milk fat produced. Several general characteristics have been identified that provide insight into the biology of MFD (Bauman and Griinari, 2003). First, the changes that occur with diet-induced MFD are specific for milk fat; fat yield can be reduced by 50% or more with little or no change in milk yield or the yield of lactose or protein. Second, the yield of most of the different fatty acids in milk fat is reduced, but the decline is greatest for *de novo* synthesized fatty acids. As a result, milk fat composition shifts toward lower proportions of short chain and medium chain fatty acids (<16 carbons) and a greater concentration of longer chain fatty acids (>16 carbons). Third, changes in ruminal microbial processes are an essential component for the development of MFD. These changes in the rumen environment are often associated with a decrease in rumen pH and a shift in the acetate:propionate ratio. Fourth, for MFD to occur, the diet must contain unsaturated fatty acids and the pathways of their biohydrogenation in the rumen must be altered. Thus, the induction of MFD is centered on both an altered rumen environment and an alteration in the rumen pathways of PUFA biohydrogenation.

Davis and Brown (1970) were among the first to recognize that increases in the milk fat content of *trans* fatty acids **(TFA)** was associated with MFD caused by feeding HC/LF diets. As the database grew, it became evident that MFD was often related to an increase in the TFA content of milk fat across a wide range of diets (Griinari et al., 1998). However, there were also many situations where increases in milk fat content of TFA did not correspond to changes in milk fat production, and thus, the basis for MFD had to be more complex than a simple relationship to the ruminal production of TFA. A key development in understanding diet-induced MFD occurred when we utilized improved analytical techniques and discovered that it was the pattern of *trans* 18:1 isomers rather than total TFA that was correlated to MFD. Specifically, we demonstrated that MFD was associated with a marked increase in the milk fat content of *trans*-10 18:1 (Griinari et al., 1998). Thus, under certain dietary situations, a portion of the linoleic acid undergoes biohydrogenation via a pathway that produces *trans*-10 18:1 (Figure 5). *Trans*-10, *cis*-12 CLA is also an intermediate in this pathway, and we found that the milk fat content of this unique CLA isomer also increased in many dietary situations associated with MFD (Bauman and Griinari, 2001). Over the same interval, we were also conducting studies with pure CLA isomers and discovered that *trans*-10, *cis*-12 CLA was a potent inhibitor of milk fat synthesis (Baumgard et al., 2000). We established that the dose response relationship was curvilinear and found that as little as 2.5 g/day of *trans*-10, *cis*-12 CLA delivered post-ruminally was sufficient to cause a 25% reduction in milk fat (deVeth et al., 2004). Effects of *trans*-10, *cis*-12 CLA are specific for milk fat and its mechanism and that for diet-induced MFD involves coordinated reductions in key mammary enzymes involved in the regulation of milk fat synthesis (Griinari and Bauman, 2006).

As a result of these advances, Bauman and Griinari (2001) proposed the "biohydrogenation theory" to explain MFD and hypothesized that "under certain dietary conditions the pathways of rumen biohydrogenation are altered to produce unique fatty acid intermediates which are potent inhibitors of milk fat synthesis." Clearly, *trans*-10, *cis*-12 CLA represents one example, and results from several recent studies have led investigators

to suggest the existence of additional fatty acid intermediates that inhibit milk fat synthesis (Perfield and Bauman, 2005). Dietary situations causing MFD result in alterations in the biohydrogenation pathways, and as a consequence, changes in many fatty acid intermediates occur and most are correlated with MFD (Loor et al., 2005a; Shingfield et al., 2006). Since correlation does not imply causality, it is important to directly examine the biological activity of specific fatty acids. Of particular interest, milk concentrations of *trans*-10 18:1 are highly correlated with the extent of diet-induced MFD. However, the limited availability of *trans*-10 18:1 has precluded a direct examination of its effect, and its presence could simply be an indication of the change in rumen fermentation associated with diet-induced MFD, rather than a significant cause of the reduction in milk fat synthesis. We recently showed that *trans*-9, *cis*-11 CLA caused a reduction in milk fat synthesis (Perfield et al., 2005), and another report indicated that the *cis*-10, *trans*-12 CLA also reduced milk fat synthesis in lactating dairy cows (Sæbø et al., 2005). Therefore, three CLA isomers have been identified as regulators of milk fat synthesis, and the production of these is increased in different types of diet-induced MFD. Further identification and characterization of rumen-derived inhibitors of milk fat synthesis and the conditions which result in their formation will enable us to more effectively troubleshoot problems in low fat test on commercial farms.

We are seeing more problems with MFD in the last few years. This increased occurrence of MFD is likely due to a number of reasons; for example, changes in rumen biohydrogenation pathways may have been caused by poor silage making conditions the past several growing seasons, increased occurrence of sorting of TMR due to attempts to increase effective dietary fiber, the increased use of unsaturated fat sources in diets, and Rumensin supplementation of certain diets. In addition, higher DMI will increase passage rates from the rumen, potentially increasing washout of biohydrogenation intermediates, including those that could cause MFD (Overton and Bauman, 2003). Of particular interest is the increased use of by-products feeds and Rumensin in dairy cow diets. By-product feeds can contain a considerable amount of lipid, which is predominately linoleic acid. In particular, corn distillers' grains have relatively high lipid content which is highly variable (~9 to 18% of DM). Such variation can significantly alter the dietary supply of unsaturated fatty acids to the dairy cow, thereby increasing the risk of dietary-induced MFD. In addition, Rumensin supplementation of certain diets appears to impact rumen fermentation in such a manner that MFD is sometimes observed. Results are inconsistent and not well described, but they appear to be associated with the classical factors affecting MFD. Duffield et al. (2003) identified that herds fed a TMR low in fiber were more prone to Rumensin-related milk fat problems as compared to TMR-fed herds with adequate fiber or component-fed herds. As in other situations of diet-induced MFD, the associative effects of feed ingredients in the rumen ultimately affects the production of unique biohydrogenation intermediates. Table 3 lists a number of potential risk factors for reduced milk fat and areas to address when troubleshooting low milk fat tests. Further research is needed to fully evaluate these interrelationships and to develop nutritional strategies designed to avoid dietary-induced MFD problems in today's high producing dairy cows.

Summary

Digestion and metabolism of dietary lipids is complex, and in this paper, we have provided an overview of the biology of these processes in dairy cows. Dietary lipids undergo extensive hydrolysis and biohydrogenation in the rumen, resulting in the lipid material leaving the rumen consisting primarily of free fatty acids that are highly saturated. Although lipid hydrolysis and classical pathways of fatty acid biohydrogenation are well established, analytical

improvements have revealed the complexity of these processes. Clearly, several minor biohydrogenation pathways exist, and many factors related to diet and rumen environment affect these processes; as a consequence, there are numerous fatty acid intermediates produced during rumen biohydrogenation, and some of these affect biological processes in the cow, including rates of milk fat synthesis. The dairy cow has evolved a number of key differences in fatty acid absorption compared with non-ruminants; these allow for efficient absorption of fatty acids and include differences in both bile salt composition and the amphiphile involved in micelle formation, as well as the slow and continuous release of relatively small amounts of fatty acids into the duodenum. Consequently, in general, the ability of ruminants to absorb fatty acids, particularly saturated fatty acids, is much higher than that of non-ruminants. Available data from lactating dairy cows indicate that relative differences in the digestibility of individual fatty acids are modest and contribute little to the extensive variation reported in the literature. Rather, this variation likely reflects differences in diets, specific feed components, and methodology among individual experiments.

The problem of diet-induced MFD has challenged producers and scientists for over a century, and in the last few years, we are seeing many more problems with low milk fat tests. We now recognize that MFD involves the interrelationship between digestive processes in the rumen and the synthesis of milk fat by the mammary gland; specific biohydrogenation intermediates produced in the rumen under certain dietary situations are potent inhibitors of milk fat synthesis in the mammary gland. Consequently, our ability to predict and troubleshoot commercial problems related to milk fat is dependent on a complete understanding of the dynamic interactions in the fermentation of feedstuffs in the rumen and the biological activities of the fatty acid intermediates produced under these different conditions. Obviously, our knowledge of lipid digestion and metabolism is rapidly advancing, and the opportunity and challenge is to effectively apply this knowledge in the feeding and management of today's high producing dairy cows.

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Table 1. Range of positional and geometric isomers of *trans* 18:1 and conjugated linoleic acids **(CLA)** and their ruminal outflow (g/day) in lactating dairy cows.[1]

Trans 18:1			Conjugated Linoleic Acids		
	Ruminal Outflow			Ruminal Outflow	
Isomer	Min	Max	Isomer	Min	Max
trans-4	0.4	2.0	*trans*-7, *cis*-9	<0.01	0.01
trans-5	0.4	3.4	*trans*-7, *trans*-9	<0.01	0.02
trans-6-8	0.4	16.2	*trans*-8, *cis*-10	<0.01	0.3
trans-9	1.4	13.1	*trans*-8, *trans*-10	<0.01	0.10
trans-10	1.5	114.0	*cis*-9, *trans*-11	0.31	2.86
trans-11	17.0	148.0	*trans*-9, *trans*-11	0.14	0.29
trans-12	1.9	20.8	*trans*-10, *cis*-12	0.02	1.84
trans-13 + 14	4.2	60.3	*trans*-10, *trans*-12	0.05	0.23
trans-15	2.0	29.0	*cis*-10, *trans*-12	0.08	0.29
trans-16	2.3	18.2	*cis*-11, *trans*-13	0.01	0.33
			trans-11, *cis*-13	<0.01	0.46
			trans-11, *trans*-13	0.09	2.02
			cis-12, *trans*-14	0.12	0.85
			trans-12, *trans*-14	0.07	0.19

[1]Data derived from five studies where samples were collected from either the omasum or duodenum of lactating dairy cows (Piperova et al., 2002; Shingfield et al., 2003; Qiu et al., 2004; Loor et al. 2004; 2005b).

Table 2. Amphiphilic properties of some polar lipids. Adapted from Freeman (1969) and Freeman (1984).

Amphiphile	Amphiphilic Index[1]	Increase or decrease (%) in $K_{m/o}$ of stearic acid[2]
Oleic Acid	0.138	-11
Monoglyceride (1-Mono-olein)	0.138	+37
Linoleic Acid	0.154	---
Lauric Acid	0.164	---
Lysolecithin	0.280	+115

[1]The amphiphilic index is defined as the increase in stearic acid solubility in bile salt solution per unit increase in amphiphilic concentration.

[2]Distribution coefficient describing the distribution of stearic acid between the particulate oil phase and the micellar phase; a positive (+) value indicates that an amphiphile increases the distribution of stearic acid into the micellar phase, which would favor absorption.

Table 3. Partial list of potential risk factors for reduced milk fat and areas to address when developing nutritional strategies designed to avoid dietary-induced milk fat depression.[1]

Altered Rumen Environment	Supply of PUFA
• Low rumen pH/low peNDF	• Amount (esp. linoleic acid intake)
• Feed particle size	• Availability
• Fiber	• PUFA:SFA
• Starch (NSC)	• Feeding Pattern
• Rumensin®[2]	• Variation in fat content and FA composition of feed ingredients
• Feeding Pattern	

[1]FA = fatty acids, NSC = nonstructural carbohydrates, peNDF = physically effective neutral detergent fiber, PUFA = polyunsaturated fatty acids, and SFA = saturated fatty acids.
[2]Elanco, Greenfield, IN.

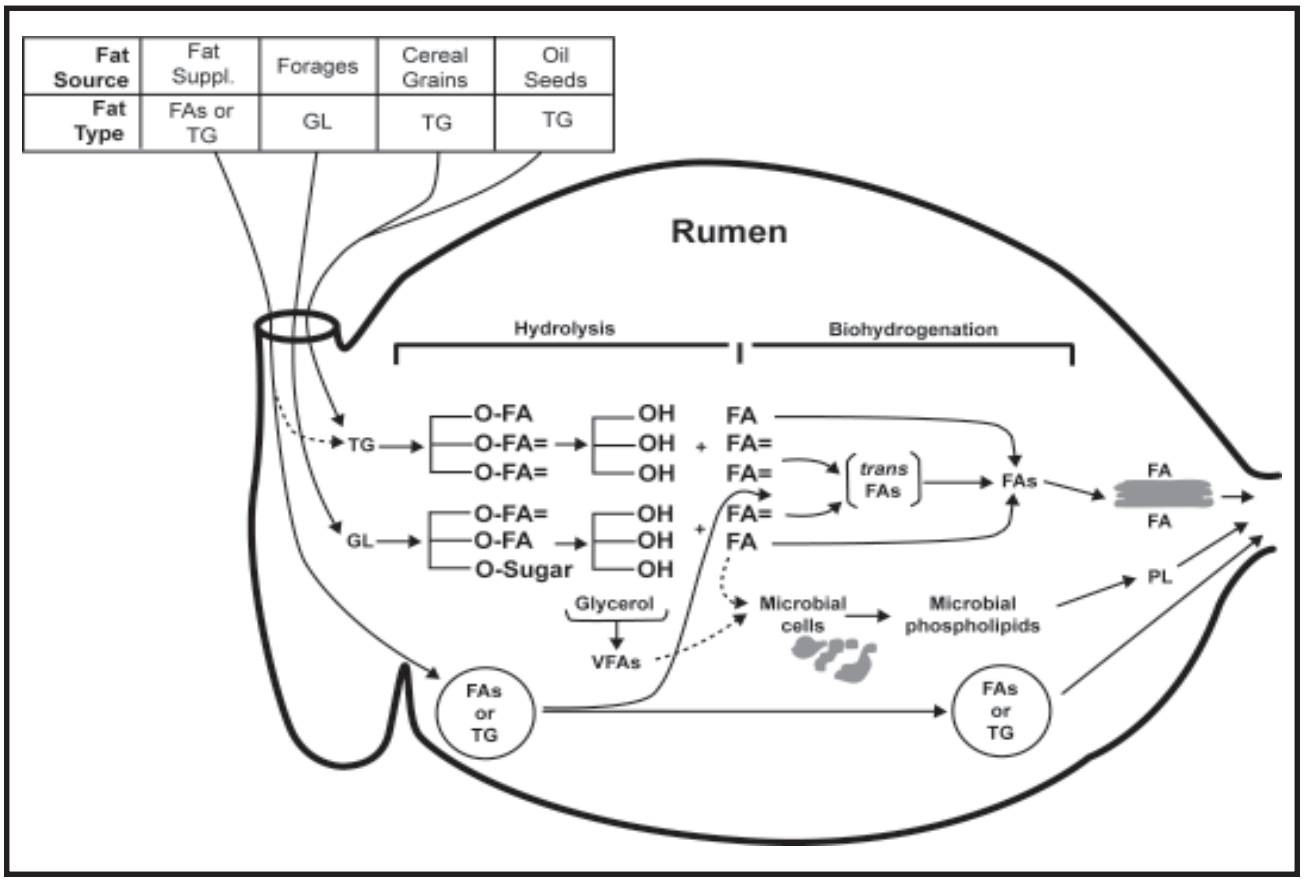

Figure 1. Lipid metabolism in the rumen. Also shown are the predominant fat types in common feedstuffs (TG = triglycerides, GL = glycolipids and FA = fatty acids). Adapted from Davis (1990).

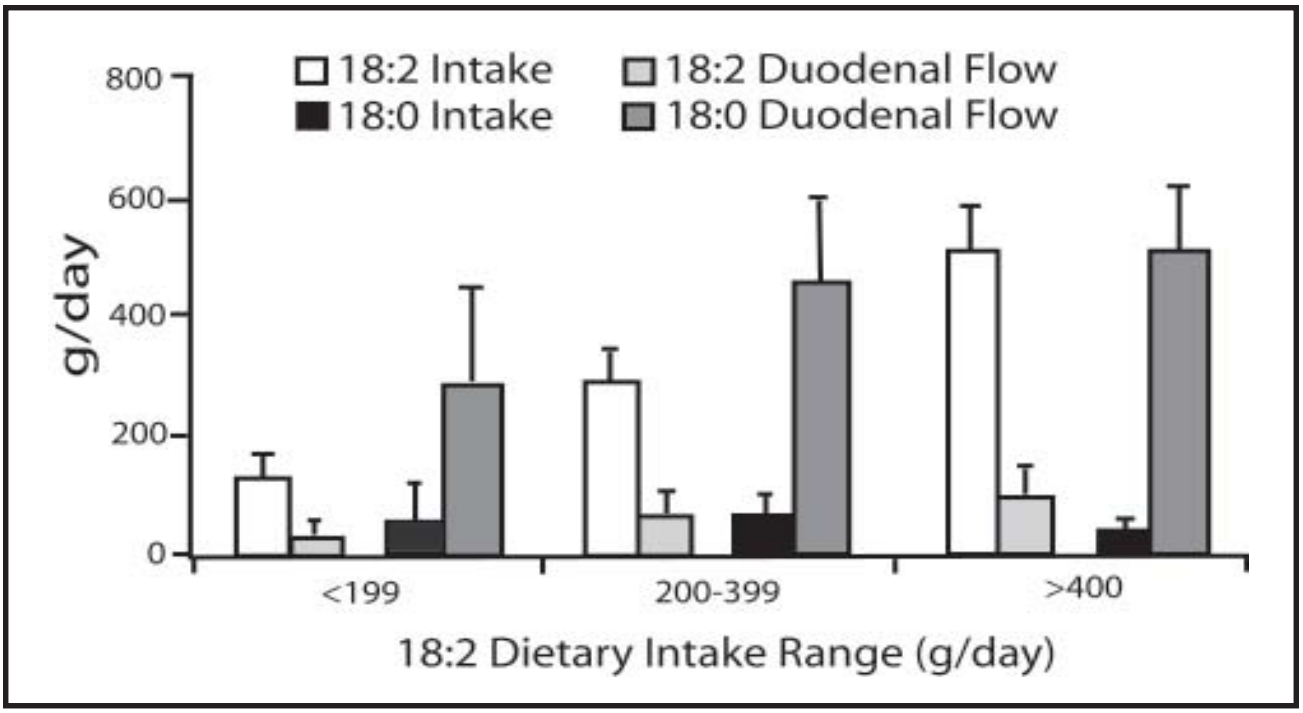

Figure 2. Relationship for linoleic acid (18:2) and stearic acid (18:0) intake and duodenal flow. Values represent means ± SD for the data obtained from 20 published studies involving 80 treatments reporting individual fatty acid intakes and duodenal flow; data are separated into tertiles based on the dietary intake of linoleic acid. Adapted from Lock et al. (2006).

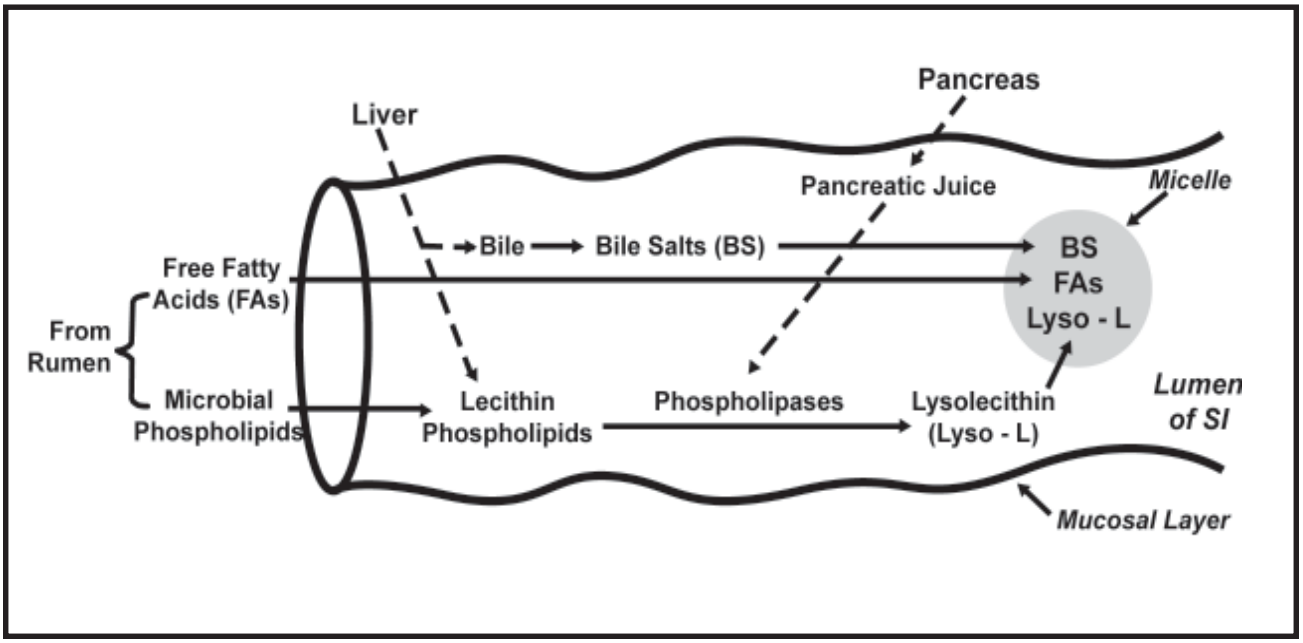

Figure 3. Fat digestion in the small intestine (SI) of the dairy cow. Adapted from Davis (1990).

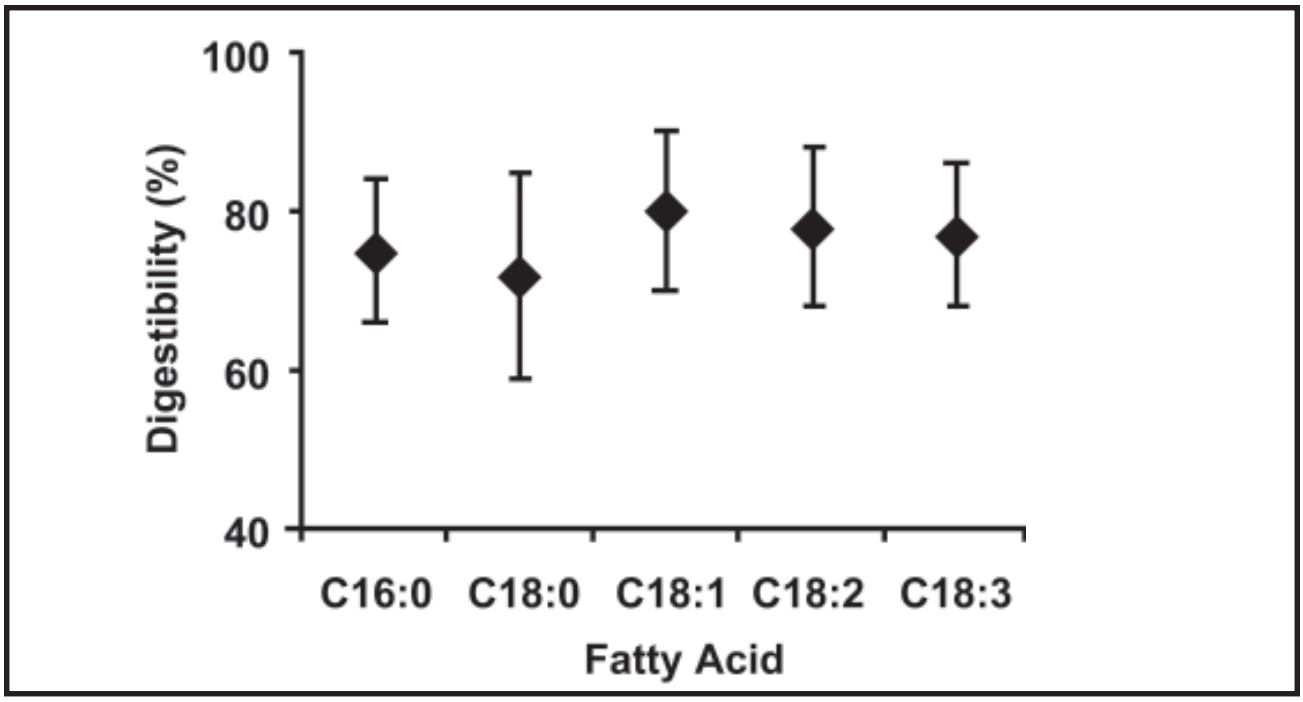

Figure 4. Comparison of individual fatty acid digestibilities in lactating dairy cows. Values represent means ± SD for the data obtained from 14 published studies involving 70 treatments. Digestibilities were calculated by differences between duodenal/omasal and ileal/fecal samples; total fatty acid digestibility averaged 74 ± 9%. Adapted from Lock et al. (2005).

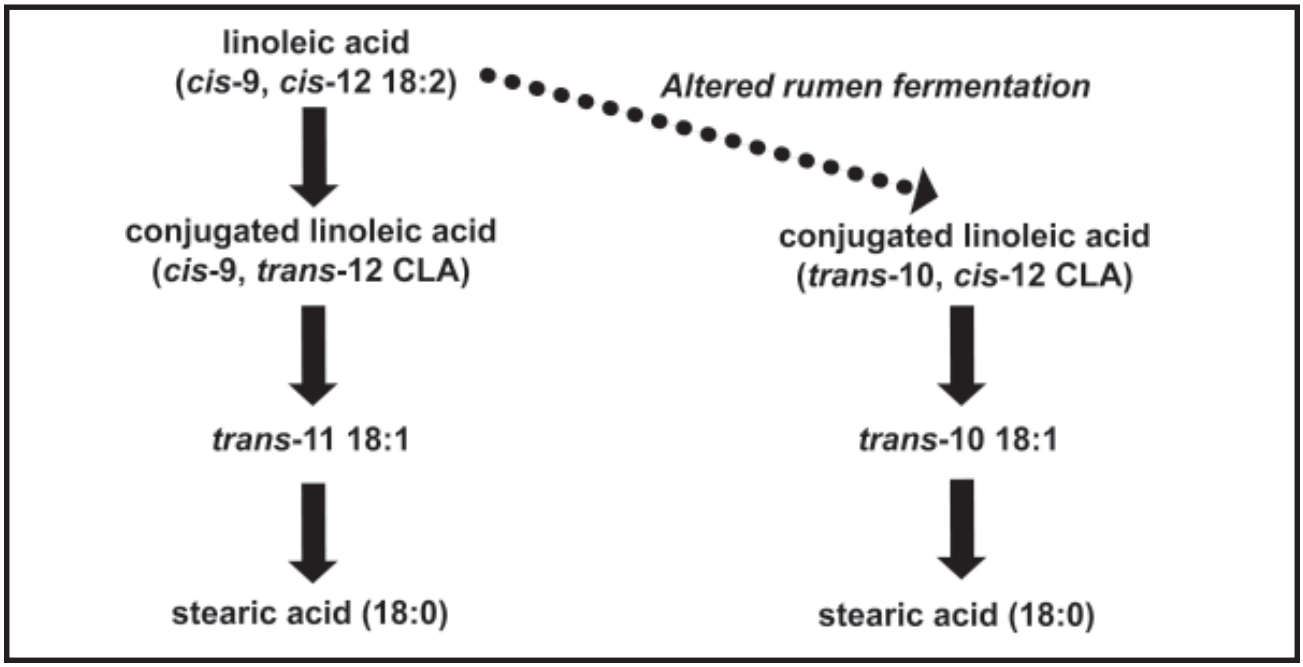

Figure 5. Generalized scheme of ruminal biohydrogenation of linoleic acid under normal conditions and during diet-induced milk fat depression (dotted line). Adapted from Griinari and Bauman (1999).