

Troubleshooting Milk Fat Challenges On Commercial Dairy Farms

Thomas R. Overton¹, Dale E. Bauman¹, and Adam L. Lock²

¹Department of Animal Science, Cornell University, Ithaca, NY

²Department of Animal Science, University of Vermont, Burlington

Introduction

Although the topic of milk fat depression (MFD) is not a new topic in the dairy industry, both industry and research interest in MFD in North America has intensified dramatically during recent years. Most pricing systems in the U.S. are now based on yields of milk components, with greater value placed upon yields of milk fat and milk protein. Recent data summaries indicate that approximately 38% of herds shipping milk into the Mideast Federal Order 33 (primarily Indiana, Michigan, Ohio, and Pennsylvania) experienced a short-term (one- to three-month period in any year) decrease (more than 1 SD decrease below the mean; milk fat test < 3.46%) in milk fat test (Bailey et al., 2005). Until recently, milk production quotas tied to milk fat yield in Canada resulted in perceived economic advantages in decreasing milk fat percentage while increasing kilograms of milk protein sold per cow. However, recent changes in guidelines for milk composition in Canada have dictated a minimum ratio of milk fat to milk protein in milk sold, effectively eliminating the economic advantage of purposely producing milk with lower milk fat percentage. During the past decade, substantial evolution has occurred in our understanding of the etiology of MFD. As will be discussed below, we do not fully understand all of the ruminal conditions that can result in predisposition for MFD; however, this new understanding of the mechanisms for MFD has facilitated our ability to troubleshoot milk fat problems on commercial dairy farms. These mechanisms (and the reasons why previously held theories for MFD likely are not applicable) will be reviewed briefly in this paper; however, the reader is referred to other recent reviews that describe

these in more detail (Bauman and Griinari, 2001; 2003; Perfield and Bauman, 2005; Bauman and Lock, 2006). The subsequent emphasis of this paper will be to discuss dietary and management factors that affect the predisposition of cows and herds to MFD. Our understanding of many of these remains conceptual; we expect that after research conducted during the next few years is summarized our recommendations will be more quantitative.

Theories of Milk Fat Depression

In general, theories of the cause of MFD can be divided into two broad categories — those suggesting that substrate supply for milk fat synthesis is limiting in situations in which MFD occurs and those suggesting that MFD is caused by direct inhibition of milk fat synthesis in the mammary gland (Bauman and Griinari, 2001). Theories relating to substrate limitation of milk fat synthesis that have been discussed include acetate insufficiency, beta-hydroxybutyrate (BHBA) insufficiency, and the glucogenic-insulin theory of MFD. Approximately 50% of milk fatty acids are synthesized *de novo* from acetate and BHBA (formed from butyrate produced in the rumen) in the mammary gland — these typically are the short- and medium-chain fatty acids, and approximately half of the 16-carbon fatty acids (Bauman and Griinari, 2001). Although the substrate supply theories are attractive (e.g., acetate and butyrate are required for fatty acid synthesis by the mammary gland; therefore a deficiency of ruminal production of these VFA in scenarios of low ruminal fiber digestion must cause MFD) and are still referred to in the dairy industry, it is unlikely that they explain MFD. This argument

is summarized in Table 1. Concurrent with the substantial decrease in milk fat yield when a high-grain, low forage diet was fed was a substantial decrease in the molar percentage of acetate and a small decrease in the molar percentage of butyrate in ruminal fluid. The molar percentage of propionate was increased sharply, resulting in a dramatic decrease in the acetate to propionate ratio in ruminal fluid. These types of approaches continue to be used commonly by researchers and others to imply that a change in molar percentage of a VFA in ruminal fluid must reflect a change in production rate. However, if we refer to data for ruminal production (measured using isotopic approaches) of VFA from cows fed these two types of diets at the bottom of Table 1, it is evident that the changes in molar proportions of VFA in the top part of the table were driven only by substantially increased production of propionate and that production rates of acetate and butyrate were not affected by diet.

The increased molar percentage and production rate of propionate when a high grain, low forage diet was fed that is depicted in Table 1 led some to consider the glucogenic-insulin theory of MFD. This theory suggests that large amounts of propionate produced in the rumen result in production of large amounts of glucose by the liver and subsequently increased circulating insulin concentrations. The mammary gland is considered to be somewhat insensitive to insulin compared with tissues such as adipose tissue and muscle; therefore it was proposed, milk fat synthesis decreases due to a “competition” among tissues for substrates for milk fat synthesis with diets that increase circulating insulin causing a preferential channeling of substrates to non-mammary. However, cows subjected to a long-term hyperinsulinemic-euglycemic clamp (experimental technique in which the effect of insulin can be determined without the confounding effects of hypoglycemia) did not decrease milk fat synthesis compared to control cows (McGuire et al., 1995); in fact, insulin or glucose infusion results in very different profiles of milk fatty acids compared to diet-induced MFD (Bauman and Griinari, 2001),

suggesting that this mechanism does not explain diet-induced MFD.

The second category of theories for the cause of MFD relates to the production of specific fatty acids in the rumen in situations of diet-induced MFD that directly inhibit milk fat synthesis in the mammary gland. Davis and Brown (1970) observed that MFD commonly was associated with increased concentrations of *trans*-fatty acids in milk fat. *Trans*-fatty acids are produced in the rumen as intermediates of the biohydrogenation of linoleic and linolenic acids to stearic acid (Figure 1). Linoleic and linolenic acids represent a large percentage of the fatty acids found in most forages and other plant-based feedstuffs fed to dairy cattle (cereal grains, oilseeds, etc.). Biohydrogenation of these fatty acids in the rumen by rumen bacteria is extensive, and most of the linoleic and linolenic acid consumed by cows is biohydrogenated fully to stearic acid before leaving the rumen (Lock et al., 2005); however, advances in analytical techniques during the past few years have led to the determination that varying quantities of a large number of *trans*-C18:1 monoenes and conjugated linoleic acids pass from the rumen to the lower tract for absorption (Bauman and Lock, 2006; Table 2). These findings, coupled with the finding that only certain *trans*-fatty acids and isomers of conjugated linoleic acid are associated with MFD led Bauman and Griinari (2001) to evolve the “*trans*-theory” of MFD into the “biohydrogenation theory,” through which they 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.”

Research conducted before the development of the biohydrogenation theory of MFD and that conducted during the past few years since it was advanced suggest that this theory represents a unifying theory for diet-induced MFD. The most well-studied “altered pathway” of ruminal biohydrogenation of linoleic acid is depicted in Figure 2, in which under situations of altered ruminal fermentation (commonly low ruminal pH) linoleic

acid (C18:2) is first isomerized to *trans*-10, *cis*-12 conjugated linoleic acid (CLA) and then reduced to *trans*-10 C18:1 before being further reduced to stearic acid (C18:0). Using pure isomers of CLA infused into the abomasum, Baumgard et al. (2000) determined that *trans*-10, *cis*-12 CLA was a potent inhibitor of milk fat synthesis. In contrast, infusion of *cis*-9, *trans*-11 CLA (the CLA isomer produced through normal ruminal biohydrogenation; Figure 1) into the abomasum did not affect milk fat synthesis. In subsequent experiments, it was found that the response of milk fat to infusion of *trans*-10, *cis*-12 CLA was dose-dependent (de Veth et al., 2004). Passage to the intestine of as little as 1.5 to 2 grams per day of this fatty acid isomer would be sufficient to reduce milk fat synthesis by 10 to 15%, which is within the magnitude of MFD that has economic consequences for dairy producers in North America.

Although the potent effects of *trans*-10, *cis*-12 CLA on milk fat synthesis are the most well-characterized, it is likely that other related fatty acid isomers have effects on milk fat synthesis. As mentioned above, advances in analytical techniques have enabled the characterization of a large number of isomers of *trans*-C18:1 and CLA (Table 2); the specific biological functions (if any) of many of these isomers remain uncharacterized. Recently, Perfield et al. (2005) showed that *trans*-9, *cis*-11 CLA caused a reduction in milk fat synthesis and another report indicated that the *cis*-10, *trans*-12 CLA also reduced milk fat synthesis in lactating dairy cows (Sæbø et al., 2005). It is likely that further research and advances in analytical techniques will identify other biologically potent fatty acid isomers that are produced during ruminal biohydrogenation of long-chain unsaturated fatty acids.

Application of the Biohydrogenation Theory to Troubleshooting Diet-induced MFD on Commercial Dairy Farms

The biohydrogenation theory provides an attractive unifying conceptual framework for troubleshooting milk fat issues on commercial dairy

farms for several reasons. First, changes in milk fatty acid profile caused by infusion of *trans*-10, *cis*-12 CLA into the abomasum are consistent with common dietary situations (high grain, low forage; high unsaturated oil intake) that cause MFD and are unlike the changes in milk fatty acid profile caused by glucose or insulin infusion as described above. Second, this theory helps reconcile problems with low milk fat percentage absent overt signs of ruminal acidosis (e.g., by conventional evaluation on a dairy farm, rumen health seems excellent yet the herd has low milk fat test). Third, it enables us to conceptualize the potential roles of known modifiers of the ruminal environment (e.g., monensin) in interacting with other factors of the ruminal environment to result in MFD in some cases. Finally, field experience troubleshooting milk fat issues on dairy farms suggests that MFD occurs as a result of several concurrent diet or management factors rather than as a result of a single factor, and our understanding of the biohydrogenation theory offers many opportunities for interactions of diet and management components to result in MFD.

We can divide the factors that can contribute to MFD into four general categories: 1) Factors that affect substrate supply and availability; 2) Factors that result in an altered ruminal environment; 3) Factors that influence biohydrogenation rate; and 4) Factors that influence rate of passage.

1) Factors that affect substrate supply and availability:

Given that the fatty acid isomers that cause MFD are intermediates in the pathways of ruminal biohydrogenation, it is logical that the amount of initial substrate (linoleic acid and perhaps linolenic acid) may be related to the amount of the key fatty acid intermediates that are produced and hence are subject to passage from the rumen. As indicated above, linoleic acid is the predominant long-chain fatty acid in corn and corn byproducts. Estimates of linoleic acid intake using CPM-Dairy in herds in the Northeastern US in which corn silage comprises the majority of the forage

base in the ration and oilseeds are essentially the sole source of added dietary fat can approach or exceed 400 to 500 g/d. Furthermore, ready availability of corn byproducts (e.g., distillers grains) at low-cost in the feed industry can result in substantial inclusion of these byproducts in “least-cost” rations. Commonly, book values are used for the fat content of distillers grains in ration formulation; however, interaction with a number of feed industry professionals suggests that the fat content of distillers grains can vary widely. Furthermore, the fatty acids in wet distillers grains likely are more rapidly available than in dry distillers grains, which further affects the dynamics of fatty acid biohydrogenation in the rumen. Based upon general survey of the literature, an effect of grain processing (in addition to potentially impacting the ruminal environment) may be to increase fatty acid availability in the rumen in some situations.

2) Factors that result in an altered ruminal environment:

These factors adhere most closely to those traditionally considered when troubleshooting MFD on dairy farms, although it is likely that some factors not commonly considered also may interact with diet formulation to produce an altered ruminal environment leading to the production of *trans*-10, *cis*-12 CLA or related biohydrogenation intermediates. One major factor that leads to flux of fatty acids through alternate pathways of ruminal biohydrogenation is low ruminal pH. Ruminal pH is thought to represent the balance between acid production from ruminal fermentation of carbohydrates, buffer production from salivary and dietary sources, and the rate of removal of fermentation acids from the rumen by absorption or passage (Allen, 1997). Dynamic interactions of these factors can result in marked changes in ruminal pH through any 24-h period. These factors have been well-reviewed (Shaver, 2005) and include dietary carbohydrate profile and rates of degradation of these carbohydrate fractions as affected by source, processing, and moisture; physically effective NDF (peNDF) supply as affected by source and

particle size; and production of salivary buffers as a function of peNDF supply and source. Shaver (1995) also illustrated that the amount of chewing (and salivary buffer produced) per unit of rumen-fermentable organic matter (RFOM) consumed decreases as RFOM increases. This has implications for the risk of higher producing cows (who also have higher DMI) to have lower ruminal pH or different dynamics of ruminal pH during any 24-h period. In addition to factors associated with diet formulation, practicing nutritionists are well-aware of the on-farm factors related to feeding management (DM changes, variation in mixing and measurement of ingredient quantities) that affect the translation of the ration on paper to the ration in the feedbunk. Finally, other aspects related to management or the environment (feeding frequency, stocking density, heat stress, among others) can have marked effects on meal patterns of dairy cows and hence the dynamics of ruminal pH in any 24-h period (reviewed by Shaver, 2005; Von Keyserlingk and DeVries, 2005). Despite our general understanding of these factors, the degree and duration of low ruminal pH required to cause sufficient flux of linoleic acid through alternative pathways of ruminal biohydrogenation is not known.

Although the implications of low ruminal pH for production of the MFD-causing intermediates have been well-considered, it is not known which other factors can also cause changes in the rumen bacteria population resulting in an increased flow of fatty acids through alternate pathways of ruminal biohydrogenation. We hypothesize that factors such as ensiled feeds with abnormal fermentation profiles (particularly high acetic acid corn silages) or moldy feeds may also cause the changes in biohydrogenation required to cause MFD; however, these factors remain unstudied in a controlled manner.

3) Factors that influence biohydrogenation rate:

It is also logical that factors that affect the rate of biohydrogenation of fatty acids in the rumen

may change the likelihood that intermediates responsible for MFD will pass from the rumen to the lower tract where they can be absorbed and directly inhibit milk fat synthesis in the mammary gland (Harvatine and Allen, 2006). It is important to think of these factors as not being causative for MFD — rather they interact with a predisposing condition (e.g., altered ruminal environment) to accentuate the effects on milk fat.

Although it is likely that other factors exist that affect biohydrogenation rates of fatty acids, the effects of monensin are among the more well-characterized. Monensin increases maintenance requirements of gram positive bacteria in the rumen by altering ion channels, which renders these bacteria less competitive in the ruminal environment (Duffield and Bagg, 2000). The net result is changes in the ruminal bacterial population that appear to decrease rates of biohydrogenation of linoleic acid in the rumen (Fellner et al., 1997). Monensin supplementation does not appear to have marked effects on milk fat during early lactation (Duffield and Bagg, 2000) — these data are consistent with those that suggest that milk fat is refractory to the effects of low doses of *trans*-10, *cis*-12 CLA until several weeks into lactation (Bernal-Santos et al., 2003; Castaneda-Gutierrez et al., 2005). During established lactation, monensin supplementation can result in decreased milk fat percentage and yield (Duffield and Bagg, 2000) — likely as a result of interactions with other dietary or management factors that predispose cows to experience MFD. Duffield et al. (2003) reported that both increasing dose of monensin and low particle size in the TMR were risk factors for MFD. They also reported that component-fed herds and herds feeding higher concentrations of nonstructural carbohydrates were at lower risk for low milk fat test when monensin was fed. We currently are conducting a large field study using herds in the Northeast and Upper Midwest in which we are relating dietary factors (including fatty acids) and management factors to milk fatty acid composition in order to learn more about the interactions of monensin with other factors and subsequent MFD on dairy farms.

Dietary components can also have effects on specific steps in the biohydrogenation process. For example, fish oil affects those rumen bacteria catalyzing the terminal step in biohydrogenation and as a result the rumen outflow of *trans*-fatty acids increases (Bauman and Griinari, 2003). In vitro studies with mixed cultures of rumen bacteria have established that docosahexaenoic acid, one of the long chain omega-3 fatty acids in fish oil, is responsible for this effect (AbuGhazaleh and Jenkins, 2004).

4) Factors that influence rate of passage:

A fourth area that influences the likelihood that biohydrogenation intermediates responsible for MFD may pass from the rumen to the lower tract is rate of passage. This has been less well-characterized than the other factors, but the possibility is logical. As described above, cows consuming greater amounts of RFOM have less chewing activity and buffer production per unit of RFOM than cows consuming smaller amounts of RFOM. Cows that are consuming larger amounts of RFOM are those cows with higher DMI, hence those cows also will have greater rates of passage from the rumen. This simply means that those cows (or herds) with higher DMI likely will be more at risk for MFD, and thus the margin of error is smaller in those herds.

In addition to DMI, another factor that may be related to rate of passage (particularly liquid rate of passage) is consumption of buffer salts or salt, particularly in free-choice form. These have not been studied in a controlled manner; however, Russell and Chow (1993) suggested that sodium bicarbonate may increase ruminal pH in part by increasing water intake and therefore passage of fermentation acids from the rumen. Salt also promotes water consumption; however, it is unlikely that these factors will be of consequence unless unusually large amounts of free-choice salt or sodium bicarbonate are consumed at the herd level.

Summary

Low milk fat percentage and yield is an important economic issue to dairy farms across North America. Research conducted during the past decade has markedly heightened our understanding of the etiology of milk fat depression, and this understanding can be translated into conceptual approaches for troubleshooting milk fat issues on commercial dairy farms. Ongoing university- and industry-based research will further enhance our ability to diagnose the causes of milk fat issues on individual farms and to provide detailed guidelines for preventing or troubleshooting milk fat problems on dairy farms.

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Table 1. Acetate and butyrate shortage theories and milk fat depression¹

| Item | Normal diet | High grain, low forage diet |
|--|-------------|-----------------------------|
| Milk yield | | No change |
| Milk fat, g/d | 683 | 363 |
| Ruminal VFA, molar percentage | | |
| Acetate | 67 | 46 |
| Propionate | 21 | 46 |
| Butyrate | 11 | 9 |
| Acetate/propionate | 3.2 | 1.0 |
| Ruminal VFA production, moles/d | | |
| Acetate | 29.4 | 28.1 |
| Propionate | 13.3 | 31.0 |
| Whole-body entry of butyrate (moles/d) | 7.0 | 9.1 |

¹ Data compiled from Davis et al. (1967); Bauman et al. (1971); Palmquist et al. (1969). Adapted from Bauman and Griinari, 2001.

Table 2. Range of positional and geometric isomers of *trans*-18:1 and conjugated linoleic acids (CLA) and their ruminal outflow (g/d) in lactating dairy cows¹

| <i>Trans</i> -C18:1 | | | Conjugated Linoleic Acids | | |
|-----------------------|-----------------|-------|------------------------------------|-----------------|------|
| Isomer | Ruminal Outflow | | Isomer | Ruminal Outflow | |
| | Min | Max | | Min | Max |
| <i>Trans</i> -4 | 0.4 | 2.0 | <i>trans</i> -7, <i>cis</i> -9 | <0.01 | 0.01 |
| <i>Trans</i> -5 | 0.4 | 3.4 | <i>trans</i> -7, <i>trans</i> -9 | <0.01 | 0.02 |
| <i>Trans</i> -6-8 | 0.4 | 16.2 | <i>trans</i> -8, <i>cis</i> -10 | <0.01 | 0.3 |
| <i>Trans</i> -9 | 1.4 | 13.1 | <i>trans</i> -8, <i>trans</i> -10 | <0.01 | 0.10 |
| <i>Trans</i> -10 | 1.5 | 114.0 | <i>cis</i> -9, <i>trans</i> -11 | 0.31 | 2.86 |
| <i>Trans</i> -11 | 17.0 | 148.0 | <i>trans</i> -9, <i>trans</i> -11 | 0.14 | 0.29 |
| <i>Trans</i> -12 | 1.9 | 20.8 | <i>trans</i> -10, <i>cis</i> -12 | 0.02 | 1.84 |
| <i>Trans</i> -13 + 14 | 4.2 | 60.3 | <i>trans</i> -10, <i>trans</i> -12 | 0.05 | 0.23 |
| <i>Trans</i> -15 | 2.0 | 29.0 | <i>cis</i> -10, <i>trans</i> -12 | 0.08 | 0.29 |
| <i>Trans</i> -16 | 2.3 | 18.2 | <i>cis</i> -11, <i>trans</i> -13 | 0.01 | 0.33 |
| | | | <i>trans</i> -11, <i>cis</i> -13 | <0.01 | 0.46 |
| | | | <i>trans</i> -11, <i>trans</i> -13 | 0.09 | 2.02 |
| | | | <i>cis</i> -12, <i>trans</i> -14 | 0.12 | 0.85 |
| | | | <i>trans</i> -12, <i>trans</i> -14 | 0.07 | 0.19 |

¹ Adapted from Bauman and Lock, 2006. Data were 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; Looor et al., 2004; 2005).

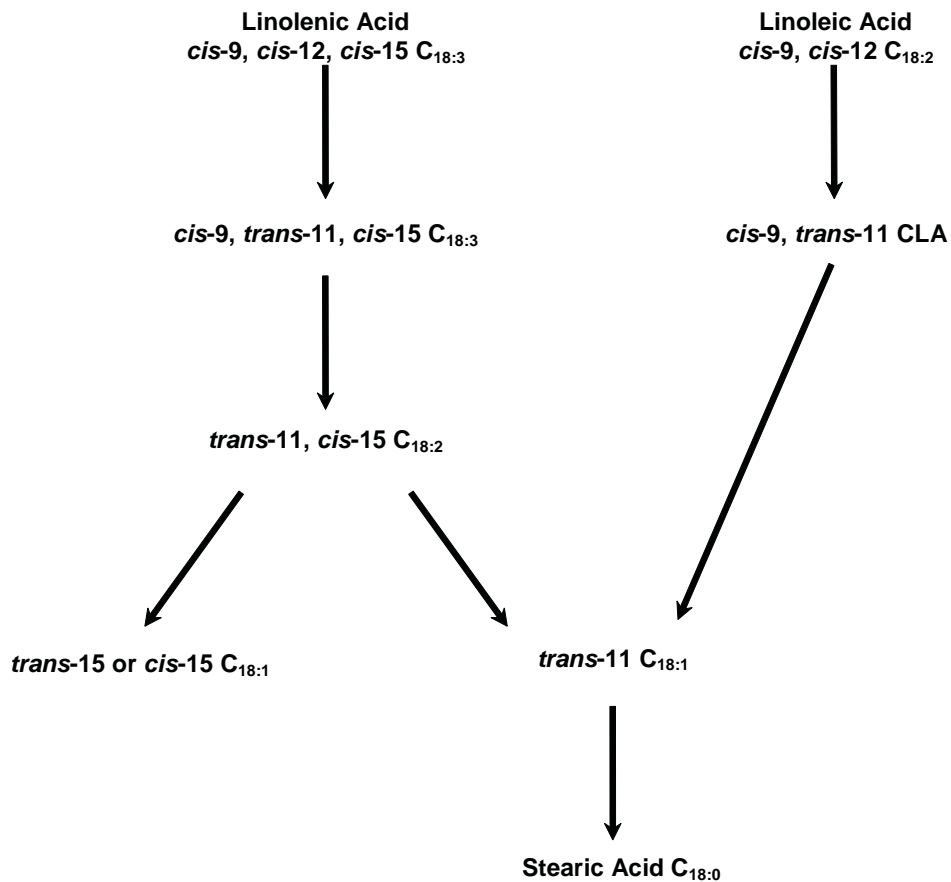


Figure 1. Generalized scheme of ruminal biohydrogenation of linoleic and linolenic acids to stearic acid. Adapted from Harfoot and Hazlewood, 1997.

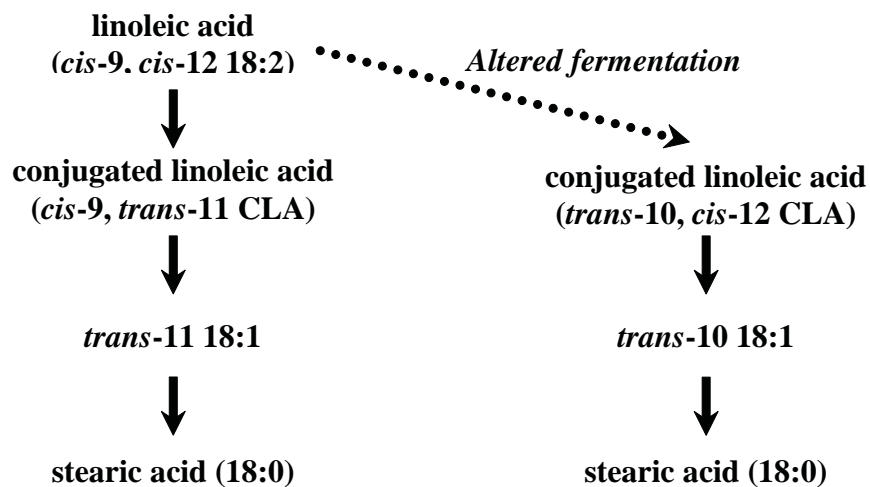


Figure 2. Generalized scheme of ruminal biohydrogenation of linoleic acid under normal conditions and during diet-induced milk fat depression (dotted line). Adapted from Griinari and Bauman (1999).