Update on Dietary and Management Effects on Milk Fat

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Summary

- Low milk fat percentage and yield is an important economic issue to dairy farms across North America.
- Available evidence indicates that all situations of milk fat depression **(MFD)** are due to changes in rumen biohydrogenation **(BH)** of unsaturated fatty acids **(FA)** and the passage of specific intermediates (e.g. *trans*-10, *cis*-12 conjugated linoleic acid) out of the rumen that subsequently reduce milk fat synthesis in the mammary gland.
- These changes in ruminal microbial processes are an essential component for the development of MFD and are centered on both an altered rumen environment and an alteration in the rumen pathways of polyunsaturated fatty acid (PUFA) BH.
- The typical magnitude of decrease in milk fat (e.g. 3.8 to 3.4%) may be caused by 1 to 2 g/day or less of *trans*-10, *cis*-12 conjugated linoleic acid or a related FA leaving the rumen and subsequently taken up by the mammary gland.
- In general, no single dietary factor is responsible for MFD, and this paper highlights the interactions between various dietary components that can increase the rumen outflow of BH intermediates associated with MFD.
- Dietary components can increase the risk of MFD by increasing substrate supply, altering rumen BH pathways, and altering rates of BH. With the latter, it is important to consider factors that alter rates of BH as not being causative for MFD per se; rather they interact with a predisposing condition (e.g., altered ruminal BH pathways) to accentuate the effects on milk fat.
- Further research is required to better understand the ruminal conditions that promote the formation of BH intermediates that may trigger MFD. An improved understanding of these events will provide the critical framework with which to better troubleshoot MFD.

Background

Milk components and not milk volume continue to be the principal driver of producer milk prices. The concentration and yield of milk fat is driven by the nutrition of the dairy cow; therefore, diets that allow for an improvement in milk fat output would potentially be economically advantageous. Equally, low (or reduced) milk fat percentage and yield is also an important economic issue to dairy farms across the U.S. For example, using a recent milk pricing structure, the change in milk price when milk fat dropped from 3.8 to 3.4% was equivalent to $0.60/cwt. Such reductions in fat are common, with a 2005 assessment of the Midwest Federal Order reporting that over one-third of dairy farms were experiencing a significant decrease in milk fat test at any one time (Bailey *et al.*, 2005).

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As previously reviewed at this Conference, we have improved our understanding of the biology of MFD with currently available evidence indicating that all situations of MFD are because of changes in rumen metabolism of unsaturated FA and the subsequent passage of specific intermediates of ruminal FA metabolism out of the rumen (e.g., *trans*-10, *cis*-12 CLA) that reduce milk fat synthesis in the mammary gland (Bauman and Lock, 2006). Although our understanding of the interrelationship between rumen lipid metabolism and milk fat synthesis has progressed significantly over the last decade, troubleshooting milk fat issues on dairy farms remains one of the more challenging tasks within overall nutritional management of dairy cows. The following sections of this paper will focus on highlighting our improved understanding of lipid metabolism in the dairy cow and how this impacts milk fat production. Finally, practical solutions to alleviating low milk fat tests will be discussed.

Lipid Metabolism in the Dairy Cow

Since unsaturated FA are toxic to many rumen bacteria, the majority of dietary lipids undergo BH through a series of FA intermediates that ultimately results in saturated FA being produced (Figure 1; Palmquist *et al*., 2005). Accordingly, there is an extensive metabolism of lipids in the rumen, and this has a major impact on the profile of FA available to the dairy cow (Lock *et al*., 2006a). In vivo studies have revealed a vast array of *trans*-18:1 and conjugated linoleic acid **(CLA)** isomers present in digesta contents of cattle and sheep (Bauman and Lock, 2006); as many as 14 CLA isomers have been identified in ruminal contents taken from cattle (Jenkins *et al*., 2008). Most published pathways of BH, however, account for the synthesis of only one or two CLA isomers. Particularly pertinent to the following discussion is that under certain dietary situations, the rumen environment is altered and a portion of BH occurs via a pathway that produces *trans*-10, *cis*-12 CLA and *trans*-10 18:1 (Figure 1).

Biology of Milk Fat Depression

The 'biohydrogenation theory' represents a unifying concept to explain the basis for diet-induced MFD where specific intermediates of ruminal FA BH escape the rumen, are absorbed, and signal a decreased expression of lipogenic enzymes and a reduction in milk fat synthesis in the mammary gland (Bauman and Griinari, 2003). The first rumen BH intermediate shown to effect milk fat synthesis was *trans*-10, *cis*-12 CLA; (Baumgard *et al*., 2000). Effects are specific for milk fat, and subsequent studies demonstrated a curvilinear relationship between increasing *trans*-10, *cis*-12 CLA dose and the reduction in milk fat yield (de Veth *et al*., 2004). It is important to note that only small quantities of specific BH intermediates produced in the rumen and subsequently taken up by the mammary gland are required to induce substantial decreases in milk fat content and yield, with as little as 2.0 g/day of *trans*-10, *cis*-12 CLA being sufficient to cause a 20% reduction in milk fat production. Two additional BH intermediates that regulate milk fat synthesis have also been identified, *trans*-9, *cis*-11 CLA and *cis*-10, *trans*-12 CLA (Bauman and Lock, 2006).

As shown in Figure 1, this '*trans*-10 shift' in BH pathways, and the associated increase in the *trans*-10 18:1 content of milk fat, is indicative of the complex changes in ruminal BH pathways characteristic of MFD. Although *trans*-10 18:1 does not directly inhibit mammary synthesis of milk fat (Lock *et al*., 2007), it is relatively easy to analyze compared to *trans* 10, *cis*-12 CLA and other CLA isomers. Therefore, in general, this FA can serve as a surrogate marker for the type of alterations in rumen BH that characterize diet-induced MFD. Also shown in Figure 1 are the 3 predominant ways in which dietary components can impact the risk of MFD: 1) through increasing substrate supply of 18-carbon unsaturated FA, 2) by altering the rumen environment and BH pathways, and 3) via changes in the rate of BH at various steps in the BH process.

Experience indicates that MFD occurs as a result of several concurrent diet or management factors rather than as a result of a single factor (Overton *et al*., 2006). Although we do not fully understand all of the ruminal conditions that may trigger MFD, an improved understanding of the impact of dietary components and their interaction during rumen fermentation will provide the critical framework with which to better troubleshoot this issue.

When one considers on-farm situations of dietary-induced MFD, it is important to note the timescale(s) through which MFD may be induced, as well as recovery times from a MFD situation. These are highlighted in Figure 2 which indicates that once the specific FA are produced in the rumen in sufficient quantities that cause MFD (in this example *trans* 10, *cis*-12 CLA was directly infused into the abomasum), reduced fat synthesis in the mammary gland is rapid with a response seen within 1 to 2 days of infusion starting. Therefore, once the rumen formation of *trans* 10, *cis*-12 CLA (and/or a related intermediate) increases, resulting in sufficient amounts of these BH intermediates entering the small intestine, the onset of MFD will be rapid. When considering trying to correct a low milk fat situation, under the experimental situation shown in Figure 2, an increase in milk fat concentration and yield can be observed 12 to 24 hours once infusion had been terminated. For a diet-induced MFD situation, a return to 'normal milk fat' levels will be dependant: first, on whether the dietary change reduces rumen production of *trans* 10, *cis*-12 CLA; and second on how much *trans* 10, *cis*-12 CLA was being produced in the rumen in the first place and when the last of this 'washes out' of the rumen. Once this occurs, a response will be seen within a few days similar to that shown in Figure 2. Under typical situations, assuming the implemented dietary and/or management change reduces *trans* 10, *cis*-12 CLA production in the rumen, one would expect to see a milk fat response within 5 to 10 days.

Dietary and Management Factors Impacting Milk Fat Concentration and Yield

Alteration of the ruminal environment

Factors that alter the rumen environment are traditionally first considered when troubleshooting MFD on dairy farms (Lock *et al*., 2006b). One major change in the rumen environment that may lead to a flux of FA through alternate pathways of ruminal BH is low ruminal pH. Although data are limited, changes in rumen pH are most likely associated with MFD because they cause a change in the bacterial population, favoring those that have alternative BH pathways. A common misconception, however, is that acidosis is a prerequisite for MFD to occur. This is not the case, and in most situations, rumen health appears excellent and there are no overt signs of ruminal acidosis (Overton *et al*., 2006). For example, Harvatine and Allen (2006a) reported increased duodenal flow of BH intermediates and MFD with no change in ruminal pH measured every 5 seconds over 4 days. Again, this highlights the fact that only small changes in the rumen environment can lead to increased risk of MFD.

Diet, and particularly carbohydrate fermentability, in the rumen is an important factor that can result in changes in BH pathways and specific intermediates. For example, a cursory review of the literature highlights the impact of different dietary carbohydrates on the risk of MFD as affected by source, processing, and moisture, presumably as a result of differences in the rate of rumen fermentation. Therefore, careful consideration should be given to the fermentation rate of starch sources when troubleshooting MFD issues. For example, a number of studies have reported an effect of corn processing method on the risk of MFD. As we have highlighted previously, however, no single factor tends to result in low milk fat, and an example of the impact of some of these dietary interactions is highlighted in Table 1. This study (0ba and Allen,

2003), diets containing high moisture and dry ground corn were fed at either a high or low starch concentration. At the low starch level, there was no significant effect of grain processing on milk fat parameters, whereas at the high starch level, high moisture corn significantly reduced milk fat yield by 15% compared to dry ground corn (Oba and Allen, 2003).

It is worth noting that risk of MFD can also be increased not only by changes in dietary components, but also via changes in how the diet is presented to the cow, subsequently impacting the rumen environment. An example of this is shown in Table 2 in which the effect of forage particle size on risk of MFD is shown (Grant *et al*., 1990). Cows were fed total mixed rations containing either fine, medium, or coarse ground alfalfa silage as 55% of dietary DM. Intake of DM and NDF was not influenced by particle size of the ration. Milk production also was unaffected, but milk fat decreased from 3.8% for cows fed the coarse ration to 3.0% for cows fed the fine ration. The decrease in milk fat secretion with reduced size of silage particles was also associated with reduced rumination and total chewing times and a lower rumen pH.

It is also clear that cows consuming diets that contain corn silage as the only or major forage source appear to be more susceptible to MFD when unsaturated fats are supplemented. Partial substitution of corn silage with another forage, such as alfalfa, has been reported to alleviate this negative effect. For example, it has been shown that changing the forage in the diet from predominantly corn silage to alfalfa silage offset the depressing effect that tallow can have on milk fat (Ruppert *et al*., 2003). This is supported by another study (Onetti et al., 2004) which observed that replacing half the dietary corn-silage with alfalfa silage negated the negative effect of tallow on milk fat yield (Table 3).

In some situations, it has been reported that monensin can affect BH rates through altering rumen fermentation and the bacterial species present (Fellner *et al*., 1997), thus potentially increasing rumen outflow of BH intermediates. In some cases during lactation, monensin supplementation can result in decreased milk fat percentage and yield (Duffield and Bagg, 2000). These effects are likely the result of interactions with other dietary or management factors that predispose cows to experience MFD. It is important to remember that an increased rumen outflow of BH intermediates will not be a problem if typical BH pathways are present. However, even if a small proportion of dietary PUFA are undergoing BH through pathways that produce *trans*-10, *cis*-12 CLA and related intermediates, monensin can potentially increase the passage of these to the small intestine and increase the risk of MFD (Lock *et al*., 2006b). Similarly, dietary FA can also modify ruminal fermentation such that BH rates and pathways may be altered. For example, Harvatine and Allen (2006b) reported that fat supplements affected fractional rates of ruminal FA BH and passage in dairy cows; increasing the unsaturation of the fat supplement slowed down the BH of 18:1 to 18:0, while causing a significant reduction in milk fat yield. It is also well known from experimental diets that the addition of fish oil alters ruminal fermentation towards increased production of BH intermediates. Long chain n-3 PUFA present in fish oils appear to affect rumen bacteria that catalyze the terminal step in BH, thereby increasing the rumen outflow of these intermediates. In vitro studies with mixed cultures of rumen bacteria have established that docosahexaenoic acid is a specific n-3 PUFA responsible for this effect (AbuGhazaleh and Jenkins, 2004), though it is likely that similar FA will have similar effects.

It is probable that other factors can also cause changes in the rumen bacterial population, resulting in an increased flow of FA through alternate pathways of ruminal BH. It has been hypothesized 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 BH required to cause MFD; however, these factors remain unstudied in a controlled manner (Overton *et al*., 2006). We are currently investigating the possible effects of high dietary wild-yeast counts on rumen BH and subsequent risk of MFD. Additional issues that warrant further attention include environmental factors (e.g. heat stress), management issues (e.g. stocking density), and possible dietary components that may aid the maintenance of 'normal' BH pathways (e.g. antioxidants).

Rumen unsaturated fatty acid load ***(RUFAL)***

The FA content of most cereal seeds and forages typically ranges from 10 to 30 g/kg DM (1 to 3%), with the majority of the FA classified as unsaturated (predominately oleic, linoleic, and linolenic acids). Among the unsaturated FA, linolenic acid is the predominant FA in most forage species, followed by linoleic acid. In cereal seeds, FA are comprised mainly of linoleic acid, followed by oleic acid. Stearic acid is low in plant oils, but present in higher amounts in animal fats, particularly in fats obtained from ruminant species, such as beef tallow. Oleic acid is the predominant FA in animal fats and some plant oils, such as canola and palm oil. Linoleic acid is the predominant FA in many plant oils, including cottonseed oil, soybean oil, and corn oil. Table 4 highlights some important feed sources and their respective FA profiles.

Elevating FA concentration in ruminal contents may cause a number of changes in ruminal fermentation characteristics and microbial population distribution. Ruminal changes are the result of the antimicrobial nature of unsaturated FA. Because some bacterial species are more susceptible than others, the result is a microbial shift in the rumen. The FA-induced microbial shift can alter fermentation of carbohydrates and fiber digestion in the rumen (Jenkins, 2002). As previously mentioned, this microbial shift also can redirect the pathways of FA BH, causing accumulation of CLA isomers linked to MFD. In attempting to predict ruminal fermentation changes caused by dietary lipid, it is often assumed that the fat load is contributed only by the fat supplement and that free-FA concentration is low. Both assumptions can be wrong. The FA from the TMR and forage can significantly contribute to total rumen fat load. Also, free-FA concentration may be elevated in some feed ingredients, such as whole cottonseed or distillers' grains stored in warm, humid conditions (Cooke *et al*., 2007), or in forages resulting from hydrolytic cleavage of esterified lipids during hay-making (Yang and Fujita, 1997). Recent evidence with cottonseeds suggests that increased free FA concentrations in the diet may impact rumen BH and subsequent MFD risk more than the same FA being present as triglycerides (Figure 5).

Since various lipids and FA can trigger a number of changes in rumen lipid metabolism, the feeding of supplemental fat can be challenging. In general, as you increase the degree of unsaturation of supplemental fat and/or the availability of the FA present (e.g. extruded vs. roasted oilseeds), the chances of MFD occurring will increase. For example, Relling and Reynolds (2007) showed that as the unsaturation of a supplemental fat increased, this was associated with reduced milk fat content and yield. This will not happen in all cases but will depend on interactions between the supplemental fat and the basal diet (Table 6). Furthermore, with the increased availability of corn byproducts (e.g. corn distillers' grains; **DDGS**) an additional important consideration is their fat content because they can contain a considerable amount of lipid that is predominately linoleic acid. The fat content of DDGS is highly variable, and this degree of variation can significantly alter the dietary supply of unsaturated FA to the dairy cow, thereby increasing the risk of MFD. For example, we recently analyzed 20 individual samples of DDGS with total FA content ranging from 10 to 18% DM; little to no variation was observed in the FA profile of these samples.

Given that the specific FA that cause MFD are intermediates produced during ruminal BH of PUFA, it is logical that the amount and/or concentration of unsatured FA may be related to the amount of the key BH intermediates that are produced. Linoleic and linolenic acids represent a large percentage of the FA found in most forages and other plant-based feedstuffs fed to dairy cattle, with linoleic acid representing the predominant PUFA in corn and corn byproducts. As a result, under typical US situations, linoleic acid is the major dietary FA, particularly when corn silage comprises the majority of the forage base in the ration and oilseeds are the major source of added dietary fat. Estimates of linoleic acid intake indicates that in these situations, linoleic acid intake can approach and even exceed 400 to 500 g/day. Therefore, it would appear that typical rations have more than enough substrate as linoleic acid to meet the required presence of PUFA for MFD to occur if rumen fermentation and BH pathways are altered. The example shown in Table 3, however, raises a number of interesting questions relating to substrate supply of unsaturated FA. Since it appears to be 18:2 BH intermediates that are responsible for MFD, we have typically only looked at PUFA when considering substrate supply (linoleic and linolenic acid). However, data in Table 3 indicate that we should more broadly consider overall 'unsaturated load' in the rumen when troubleshooting MFD. Increasing the dietary supply of oleic acid from tallow, or other sources of this fatty acid (e.g. palm fatty acid distillate), will not directly increase the rumen outflow of 18:2 BH intermediates; however, in some circumstances, it would appear that the increase in unsaturated load from increasing oleic acid supply is sufficient to alter BH pathways to favor the production of *trans*-10, *cis*-12 CLA and related intermediates from the PUFA already in the diet. This concept is supported by a continuous culture study using ^{13}carbon-labeled oleic acid (Abu-Ghazaleh *et al.*, 2005). As expected, lowering culture pH to 5.5 reduced the concentration of *trans*-11 18:1 and increased *trans*-10 18:1 concentration. However, the ^{13}carbon enrichment of *trans*-10 18:1 was lower at pH 5.5 compared with pH 6.5, indicating that more of the *trans*-10 at this low pH originated from sources other than oleic acid . This must come from PUFA sources and will presumably be driven through BH pathways that also promote the formation of *trans*-10, *cis*-12 CLA or related intermediates, thereby increasing MFD risk.

Based on this discussion, we coined the term "rumen unsaturated FA load" **(RUFAL)** to reflect the total dietary unsaturated FA supply entering the rumen each day. As defined, RUFAL accounts for intakes of unsaturated FA from all feed ingredients rather than FA intake coming only from fat supplements. The RUFAL can be calculated as the sum of the three primary unsaturated fatty acids consumed by dairy cows, namely oleic (C18:1), linoleic (C18:2), and linolenic (C18:3) acids. It is proposed that RUFAL will be a better indicator of fermentation disruption in the rumen and risk of MFD rather than relying just on the percentage of fat added to the diet or only the dietary linoleic acid supply.

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Table 1. Effect of corn grain processing method and starch intake on milk fat synthesis (Oba and Allen, 2003).

	High starch (32% of DM)		Low starch (21% of DM)	
	High moisture corn	Dry ground corn	High moisture corn	Dry ground corn
Milk yield (lb/day)	85.4	84.5	73.5	75.5
Milk fat (%)	3.05[b]	3.59[a]	3.95[a]	3.73[a]
Milk fat yield (lb/day)	2.57[b]	2.97[a]	2.93[ab]	2.79[ab]

[ab]Treatment significance ($P < 0.05$) indicated by differences in superscript letters.

Table 2. Influence of ration particle size on rumen fermentation and milk fat synthesis (Grant et al.,1990).[1]

	Treatment[2]			
	Fine	Medium	Coarse	P
Dry matter intake, lb/day	49.3	48.4	48.8	0.88
Milk yield, lb/day	69.3	70.6	68.4	0.56
Milk fat, %	3.0	3.6	3.8	0.001
Milk fat yield, lb/day[3]	2.08	2.55	2.60	---
Rumen pH	5.3	5.9	6.0	0.10
Rumination time, min/24 h	374	466	531	0.001
Total chewing time, min/24 h	570	671	735	0.001

[1]Arithmetic mean particle size of the fine and course silages used in the study were 2.0 and 3.1 mm, respectively.
[2]Rations formulated on 55:45 silage:concentrate basis.
[3]Calculated from reported values.

Table 3. Effect of feeding tallow on rumen fermentation and milk fat synthesis in dairy cows fed diets based upon corn silage or alfalfa silage with or without tallow supplementation (Onetti et al., 2004).

	Treatment[1]		
	CS	CST	AST
Milk, lb/day	98.8	97.5	95.9
Milk fat, %	3.12	2.68	3.32
Milk fat, lb/day	3.04	2.57	3.19
trans-10 18:1, % of fatty acids	0.75	2.15	0.78

[1]CS = 50% corn silage + 50% concentrate; CST = 50% corn silage + 50% concentrate + 2% tallow; AST = 25% corn silage + 25% alfalfa silage + 50% concentrate + 2% tallow.

Table 4. Fatty acid composition of typical feedstuffs (Data from CPM Feed Library; Cornell University, Ithaca, NY).

Feed Name	Fatty Acid (g/100 g fatty acids)					
	C14:0	C16:0	C18:0	C18:1 cis	C18:2	C18:3
Corn silage	0.46	17.83	2.42	19.24	47.74	8.25
Alfalfa silage	0.66	18.81	3.35	2.05	15.91	38.71
Grass hay	0.43	16.44	1.33	2.53	23.38	49.90
Corn grain (ground fine)	2.33	13.21	1.99	24.09	55.70	1.62
Corn high moisture, 78% DM	0.26	13.57	1.83	25.99	55.08	1.64
Tallow (beef)	3.00	24.43	17.92	41.62	1.09	0.53
Soybean oil	0.11	10.83	3.89	22.82	53.75	8.23
Corn distillers grains (ethanol)	0.14	14.05	2.39	24.57	56.11	1.68
Cottonseed (whole, with lint)	0.69	23.91	2.33	15.24	56.48	0.19

Table 5. The effect of whole cottonseed with elevated concentrations of free fatty acids (FA) in the oil on dry matter intake (DMI), milk yield and milk fat synthesis in dairy cows (Cooke *et al*., 2007).[1]

Item	Control[1]	HFFA1	HFFA2	P-value
DMI, lb/day	47.5	48.4	51.7	0.07
Milk, lb/day	77.0	74.8	77.2	0.39
Fat, %	4.22[a]	3.64[b]	3.58[b]	0.008
Fat, lb/day	3.04	2.75	2.84	0.11

[ab]Means in the same row with superscripts differ (P < 0.01).

[1]Control = normal cottonseed (WCS); HFFA1 = WCS with 24.1% free-FA and normal color; and HFFA2 = WCS with 22.3% free-FA and discolored.

Table 6. The effect of rumen-inert fats containing mostly saturated fatty acids (SFA), mostly monounsaturated fatty acids (MUFA), or mostly polyunsaturated fatty acids (PUFA) on dry matter intake (DMI), milk yield and milk fat synthesis in midlactation dairy cows (Relling and Reynolds, 2007).

	Diet				
	Control	SFA	MUFA	PUFA	*P*[1]
DMI, lb/day	52.4	50.8	48.6	48.4	0.12
Milk, lb/day	81.2	82.1	78.8	76.7	0.44
Fat, %	3.37	3.86	3.32	2.61	0.03
Fat, lb/day	2.75	3.16	2.61	2.01	0.02

[1]Probability comparing the difference between saturated and unsaturated fat supplements (SFA vs. MUFA and PUFA).

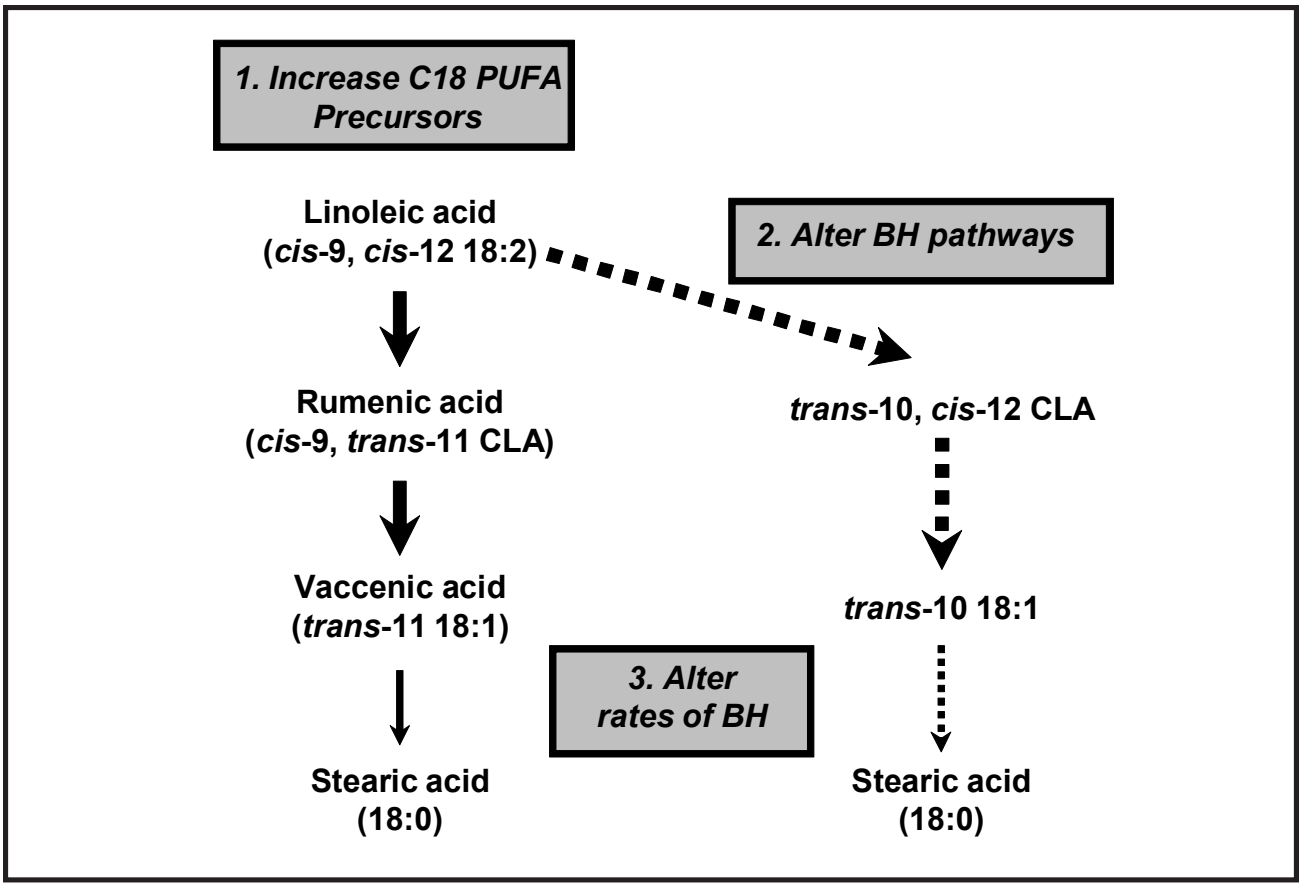

Figure 1. Generalized scheme of ruminal biohydrogenation (BH) of linoleic acid under normal conditions (left side) and during diet-induced milk fat depression (dotted lines, right side) (CLA = conjugated linoleic acid and PUFA = polyunsaturated fatty acids). The grey boxes highlight 3 potential means by which dietary components can increase the risk of milk fat depression (Bauman and Griinari, 2003).

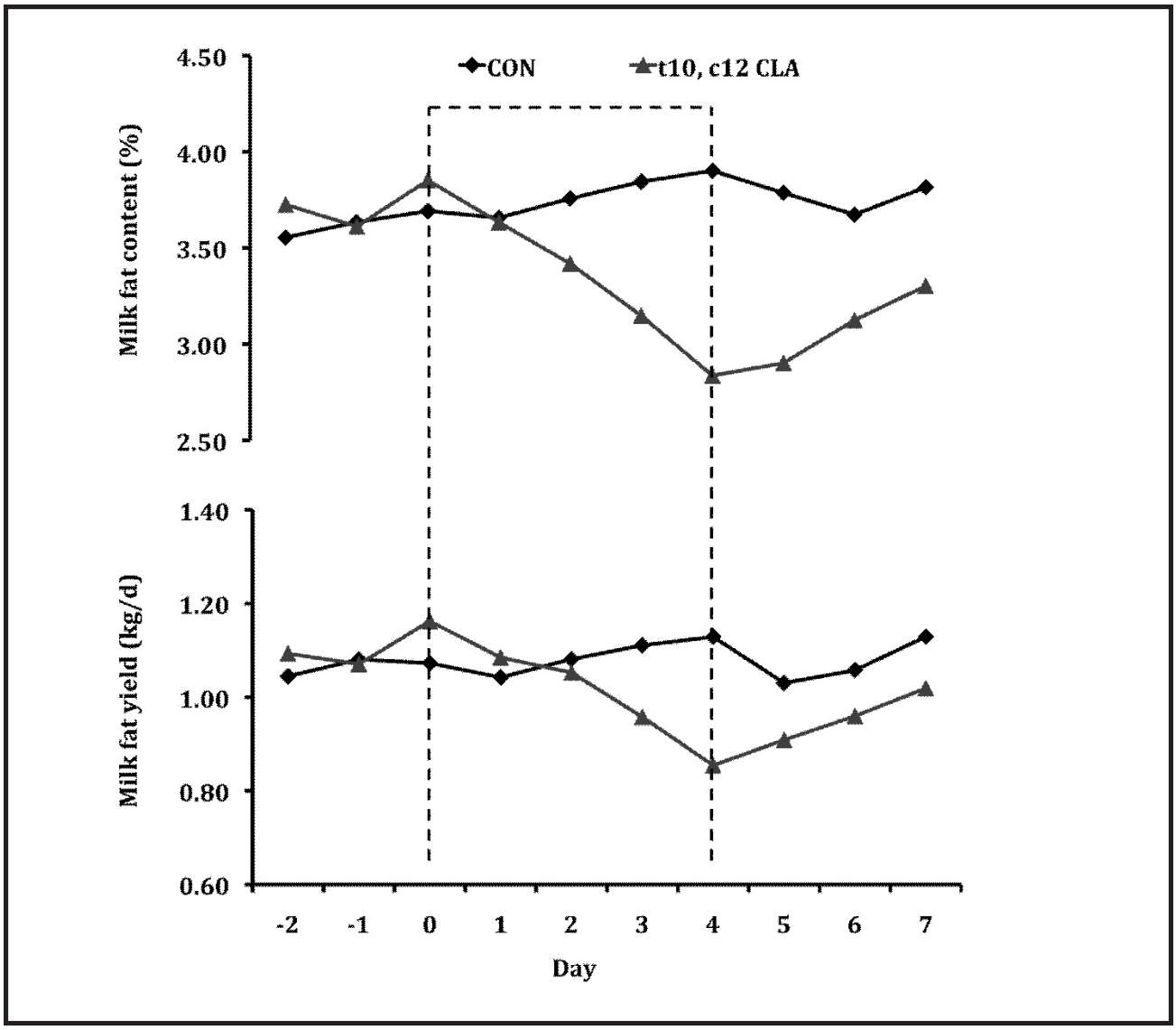

Figure 2. Temporal pattern of milk fat content and yield during abomasal infusion of *trans*-10, *cis*-12 conjugated linoleic acid (CLA) (▲) or control (CON; carrier only; ◆) in lactating cows. Animals received supplements for 4 days (dotted lines; Lock *et al.,* 2007).