

Milk Fat Depression: What Do We Know and What Can We Do About It?

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Introduction

Nutrition affects both the quantity and composition of milk fat, and a striking example is milk fat depression (MFD). Diet-induced MFD represents a challenging problem, and our understanding of the interrelationship between rumen lipid metabolism and milk fat synthesis has progressed significantly over the last decade. The ‘biohydrogenation theory’ represents a unifying concept to explain the basis for diet-induced MFD where intermediates of ruminal fatty acid biohydrogenation (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. The first rumen BH intermediate shown to affect milk fat synthesis was *trans*-10, *cis*-12 conjugated linoleic acid (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), with as little as 2.0 g/d being sufficient to cause a 20% reduction in milk fat production. Recently, two additional BH intermediates that regulate milk fat synthesis have been identified, *trans*-9, *cis*-11 CLA (Perfield II et al., 2007) and *cis*-10, *trans*-12 CLA (Saebo et al., 2005). MFD has been observed over a wide range of feeding situations including diets high in concentrates and low in fiber, and diets supplemented with plant or fish oils. Although the cause of all types of diet-induced MFD involves inhibition of milk fat synthesis by unique BH intermediates, troubleshooting milk fat issues on dairy farms remains one of the more challenging tasks within overall nutritional management of dairy cows. Clearly, small quantities of specific BH intermediates

produced in the rumen and subsequently taken up by the mammary gland are sufficient to induce substantial decreases in milk fat content and yield. Escape of these intermediates from the rumen is influenced by ruminal passage rate, bacterial BH capacity and dietary polyunsaturated fatty acid (PUFA) concentration and profile. Bacterial BH capacity is intrinsic to the bacterial population, and numerous factors are known to cause an altered ruminal fermentation with a propensity towards production of BH intermediates that are associated with MFD. Therefore, the induction of MFD requires both an altered rumen fermentation and the presence of PUFA in the rumen, and within each of these categories there are a number of potential risk factors and areas to address when developing nutritional strategies designed to minimize effects on milk fat production (Table 1).

The focus of the following sections will be to discuss the impact of dietary components and rumen environment interactions on milk fat. Experience indicates that MFD occurs as a result of several concurrent diet or management factors rather than as a result of a single factor. It will always be a challenge to troubleshoot MFD when the magnitude of decrease in milk fat (e.g. 3.8 to 3.2%) may be caused by 1 to 2 g/d or less of *trans*-10, *cis*-12 CLA or a related intermediate passing to the small intestine. 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.

Rumen Biohydrogenation

Since unsaturated fatty acids are toxic to many rumen bacteria, the majority of dietary lipids are biohydrogenated through a series of fatty acid intermediates that ultimately results in saturated fatty acids being produced (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 fatty acids available to the dairy cow (Lock et al., 2006a). Generally, BH of linoleic acid produces *cis*-9, *trans*-11 CLA and *trans*-11 18:1 (Palmquist, et al., 2005). Under certain dietary situations, however, 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). Therefore, dietary situations causing MFD alter the pathways of rumen BH resulting in changes in the specific TFA and CLA isomers available for uptake by the mammary gland and incorporation into milk fat. As shown in Figure 2, 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 fatty acid 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 three predominant ways in which dietary components can impact the risk of milk fat depression: 1) through increasing substrate supply of 18-carbon unsaturated fatty acids, 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. These three areas are discussed in the following sections.

Supply of Unsaturated Fatty Acids

Given that the specific fatty acids that cause MFD are intermediates produced during ruminal BH of PUFA, it is logical that the amount of initial substrate (linoleic acid and perhaps linolenic acid) may be related to the amount of the key BH

intermediates that are produced. 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, 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 fatty acid, 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 using CPM-Dairy indicates that in these situations linoleic acid intake can approach and even exceed 400 to 500 g/d (Table 2). 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 is altered. Nevertheless, this is a moving threshold which depends on the rate at which the PUFA become available to the rumen bacteria and the extent to which perturbations in rumen fermentation occur. With the increased availability of corn byproducts (e.g. distillers’ grains) an additional important consideration is their fat content, because they can contain a considerable amount of lipid, which is predominately linoleic acid. In particular, the fat content of corn distillers’ grains is highly variable (e.g. ~5 to 15% of DM), and this degree of variation can significantly alter the dietary supply of unsaturated fatty acids to the dairy cow, thereby increasing the risk of MFD.

The feeding of supplement fat can be challenging since various lipids and fatty acids can trigger a number of changes in rumen metabolism. Space does not permit a detailed discussion of specific fat sources, but readers are directed to a recent review by Staples (2006) which discusses the influence of different fat supplements on milk fat. In general, as you increase the degree of unsaturation of supplemental fat and/or the availability of the fatty acids present (e.g. extruded vs. roasted oilseeds), the chances of MFD occurring will increase. Recently, Relling and Reynolds (2007) examined the impact of feeding rumen-inert fats differing in their degree of saturation on performance of lactating dairy cows. Cows were fed a control

mixed ration ad libitum, and treatments were the dietary addition (3.5% of ration dry matter) of 3 rumen-inert fat sources differing in fatty acid profile. As shown in Table 3, as the unsaturation of the supplemental fat increased, this was associated with reduced milk fat content and yield.

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 may alleviate this negative effect. For example, Ruppert et al. (2003) showed 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. The concentration of *trans* 18:1 BH intermediates in milk fat tended to increase to a greater extent when tallow was fed in the corn silage-based diets than in the alfalfa silage-based diets. Although not reported in this study it is most likely that the profile of *trans* 18:1 fatty acids also shifted to favor *trans*-10 18:1 with the corn silage-based diets. This is supported by a study by 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 4). Furthermore, the addition of alfalfa silage to the diet attenuated the tallow-induced increase in *trans*-10 18:1 formation in the rumen and subsequent incorporation into milk fat (Table 4).

The example shown in Table 4 raises a number of interesting questions relating to substrate supply of unsaturated fatty acids. 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. These data, however, suggest that it may be appropriate to more broadly consider overall 'unsaturated load' in the rumen when troubleshooting MFD. Increasing the dietary supply of oleic acid (*cis*-9 18:1) from tallow or other sources (e.g. palm fatty acid distillate), will not directly increase the rumen outflow of 18:2 BH intermediates because these fat supplements supply very little PUFA and, as

we showed previously, under some circumstances we can feed high levels of oleic acid without inducing MFD (Hinrichsen et al., 2006). In some circumstances, however, 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 hypothesis is supported by a recent study using continuous cultures and ¹³carbon-labeled oleic acid. As expected, lowering culture pH to 5.5 reduced the concentration of *trans*-11 18:1 and increased *trans*-10 18:1 concentration. The ¹³carbon enrichment of *trans*-10 18:1, however, was lower at pH 5.5 compared with pH 6.5 indicating that more of the *trans*-10 at low pH originated from sources other than oleic acid (Abu-Ghazaleh et al., 2005). 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 (Lock et al., 2006b).

Alteration of the Ruminal Environment

Factors that alter rumen environment are traditionally first considered when troubleshooting MFD on dairy farms. One major change in the rumen environment that leads to flux of fatty acids through alternate pathways of ruminal BH is low ruminal pH. Factors that can result in marked changes in ruminal pH through any 24-h period include: dietary carbohydrate profile and rates of degradation of the 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, 2005). Despite our general understanding of these factors, the degree and duration of low ruminal pH required to cause sufficient flux of PUFA through alternative pathways of ruminal BH is not known. 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 d. Again, this highlights the fact that only small changes in the rumen environment can lead to increased risk of MFD.

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. A somewhat extreme example was reported by Jurjanz et al. (2004) which compared the effect of different starch sources (potato vs. wheat) on rates of milk fat synthesis; although there were no significant differences in milk yield, the wheat diet significantly reduced milk fat yield by 11%. Of greater relevance, a number of studies have reported an effect of corn processing method on risk of MFD. For example, Guyton et al. (2003) reported a 10% reduction in milk fat yield when steam-flaked corn replaced dry-ground corn. Clearly, careful consideration should be given to the fermentation rate of starch sources when troubleshooting MFD issues. 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 5. Oba and Allen (2003) fed diets containing high moisture and dry ground corn at either a high or low starch level. 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.

The preceding paragraphs have discussed situations in which changes in dietary components and their interactions have resulted in alterations in the rumen environment and BH pathways. It is worth noting, however, 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. An example of this is shown in Table 6 in which the effect of forage particle size on risk of MFD is reported (Grant et al., 1990). Cows were fed total mixed rations containing either fine (2.0 mm), medium (2.6 mm), or coarse (3.1 mm) 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 (Table 6).

Although the implications of low ruminal pH for production of the MFD-causing intermediates have been considered, it is probable that other factors can also cause changes in the rumen bacteria population resulting in an increased flow of fatty acids through alternate pathways of ruminal BH (Palmquist, et al., 2005). Overton et al. (2006) 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. Additional issues that warrant further attention include environmental factors such as heat stress as well as management factors such as stocking density. Finally, when considering factors related to rumen environment, the impact of changes in rate of passage out of the rumen should also be considered; cows with higher DMI have higher rates of passage which potentially will ‘flush’ more BH intermediates out of the rumen. Cows with slower rates of fermentation, potentially increasing DMI and passage rate to compensate, and cows with higher DMI in general (e.g. higher producing cows) are, therefore, more likely at risk of MFD, and thus the margin of error is less in these animals (Overton, et al., 2006).

Alteration of Rates of Biohydrogenation

Under some circumstances specific feed components can alter rumen fermentation in a manner that results in changes in BH rates of fatty acids. Altering these rates can potentially increase the rumen outflow of *trans*-10, *cis*-12 CLA and related intermediates responsible for MFD, thereby increasing risk of MFD. This is a facet of troubleshooting MFD which is not typically considered when thinking about the traditional 'supply of PUFA' or 'altered fermentation' groupings, even though these changes are a result of changes in the rumen environment. Monensin is an example of a feed ingredient that can affect BH rates through altering rumen fermentation and the bacterial species present. In some cases during established 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. Monensin increases maintenance requirements of gram positive bacteria in the rumen, 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 BH of PUFA in the rumen. Very few species of bacteria have been identified that can convert *trans*-18:1 fatty acids to stearic acid (18:0), and most of these have been identified as being gram positive. Thus the final step in BH is already the 'rate-limiting' step; therefore decreasing the number of bacteria that can carry out this process can potentially lead to a 'build-up' of BH intermediates in the rumen thereby increasing their passage to the small intestine. This was highlighted by Fellner et al. (1997) when they examined the effect of monensin on the formation of BH products when linoleic acid was infused continuously into rumen fermentors. With an unsupplemented diet the rate of 18:0 formation was 7.5 mg/L/hr whereas this decreased to only 2.7 mg/L/hr when monensin was supplemented (Fellner et al., 1997). It is important to remember, however, 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 being biohydrogenated 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.

Dietary fatty acids can also modify ruminal fermentation and may shift BH towards the production of intermediates that cause MFD. For example, Harvatine and Allen (2006b) reported that fat supplements affected fractional rates of ruminal fatty acid 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 to the diet alters ruminal fermentation towards increased production of BH intermediates. Long chain omega-3 PUFA present in fish oils appear to affect rumen bacteria catalyzing 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 omega-3 PUFA responsible for this effect (Abu-Ghazaleh and Jenkins, 2004), though it is likely that other fatty acids may have similar effects. We have previously taken advantage of the effects of fish oil on rumen lipid metabolism as a method to facilitate the production of *cis*-9, *trans*-11 CLA-enriched milk (e.g. Lynch et al., 2005). Interactions are once again key; if normal BH pathways are maintained then the rumen outflow of *trans*-11 18:1 and *cis*-9, *trans*-11 CLA will increase. Small changes, however, in rumen fermentation as a result of fish oil feeding can alter these pathways thereby increasing the rumen outflow of intermediates that cause MFD. This is highlighted by recent studies by our group that emphasize the impact of feeding pattern of fish oil on MFD risk. In our first study we infused fish oil into the rumen 4X / day and observed a 24% decrease in milk fat yield. However, a follow up study which utilized a similar basal diet but infused the fish oil 6X / day resulted in no MFD (McConnell, Lock and Bauman, unpublished). Due to these

multifaceted interactions it has proven difficult to experimentally distinguish the effect of PUFA as increased substrate vs. its potential role as a modifier of rumen fermentation.

Conclusions

Low milk fat percentage and yield is an important economic issue to dairy farms across North America. The available evidence indicates that all situations of MFD are due to changes in rumen BH of unsaturated FA and the passage of specific intermediates 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 PUFA BH. In general, no single dietary factor is responsible for MFD, and this paper has highlighted 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 (e.g. monensin) 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. Finally, our understanding of the effect of specific BH intermediates on milk fat synthesis in the mammary gland has advanced at a much greater rate than our knowledge of their production in the rumen. Therefore, 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.

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Table 1. Partial list of potential risk factors for reduced milk fat and areas to address when developing nutritional strategies designed to avoid diet-induced MFD¹

Altered Rumen Environment	Supply of PUFA
<ul style="list-style-type: none"> • Low rumen pH/low peNDF • Feed particle size • Fiber • Starch (NSC) • Rumensin • Feeding pattern 	<ul style="list-style-type: none"> • Amount (esp. linoleic acid intake) • Availability • PUFA:SFA • Feeding pattern • Variation in fat content and FA composition of feed ingredients

¹Adapted from Bauman and Lock (2006) and Overton et al. (2006).

Table 2. Modified CPM Dairy lipid submodel output showing the sources of dietary fatty acids from a diet formulated for a cow producing 100 lbs milk/d

	Fatty Acid (g/d)					Total
	C16:0	C18:0	C18:1c	C18:2	C18:3	
Alfalfa Silage	15	3	2	13	31	80
Corn Silage	30	4	32	79	14	166
Soybean Hulls	3	1	3	8	3	20
Corn Grain Ground	28	4	51	117	3	211
Soybean Meal	14	3	10	42	7	78
Blood Meal	2	2	2	1	0	7
Cottonseed Whole	92	9	58	217	1	384
Megalac	107	9	75	15	0	210
Ration	290	35	234	496	59	1161

Table 3. 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 cows¹

	Diet				P ²
	Control	SFA	MUFA	PUFA	
DMI, kg/d	23.8	23.1	22.1	22.0	0.12
Milk, kg/d	36.9	37.3	35.8	34.8	0.44
Fat, %	3.37	3.86	3.32	2.61	0.03
Fat, g/d	1,249	1,436	1,184	911	0.02

¹Adapted from Relling and Reynolds (2007)

²Probability comparing the difference between saturated and unsaturated fat supplements (SFA vs. MUFA and PUFA).

Table 4. Effect of feeding tallow on rumen fermentation and milk fat synthesis in dairy cows fed diets based upon corn silage (CS) or alfalfa silage (AS) with or without tallow supplementation¹

	Treatment ²		
	CS	CST	AST
DMI, kg/d	27.6	25.9	26.5
Milk, kg/d	44.9	44.3	43.6
Fat, %	3.12	2.68	3.32
Fat, kg/d	1.38	1.17	1.45
<i>trans</i> -10 18:1, %	0.75	2.15	0.78

¹Adapted from Onetti et al. (2004).

²CS = 50% corn silage + 50% conc; CST = 50% corn silage + 50% conc + 2% tallow; AST = 25% corn silage + 25% alfalfa silage + 50% conc + 2% tallow.

Table 5. Effect of corn grain processing method and starch intake on milk fat synthesis¹

	High starch		Low starch	
	High moisture corn	Dry ground corn	High moisture corn	Dry ground corn
Milk yield, kg	38.8	38.4	33.4	34.3
Milk fat, %	3.05 ^b	3.59 ^a	3.95 ^a	3.73 ^a
Milk fat yield, kg	1.17 ^b	1.35 ^a	1.33 ^{ab}	1.27 ^{ab}

¹Adapted from Oba and Allen (2003). Treatment significance ($P < 0.05$) indicated by differences in superscript letters.

Table 6. Influence of ration particle size on rumen fermentation and milk fat synthesis^{1,2}

	Treatment ³			<i>P</i>
	Fine	Medium	Coarse	
Dry Matter Intake, kg/d	22.4	22.0	22.2	0.88
Milk Yield, kg/d	31.5	32.1	31.1	0.56
Milk Fat, %	3.0	3.6	3.8	0.001
Milk Fat Yield, g/d ⁴	945	1156	1182	---
Rumen pH	5.3	5.9	6.0	0.1
Rumination Time, min/24 h	374	466	531	0.001
Total Chewing Time, min/24 h	570	671	735	0.001

¹Adapted from Grant et al. (1990).

²Arithmetic mean particle size of the fine and coarse silages used in the study were 2.0 and 3.1, respectively.

³Rations formulated on 55:45 silage:concentrate basis.

⁴Calculated from reported values.

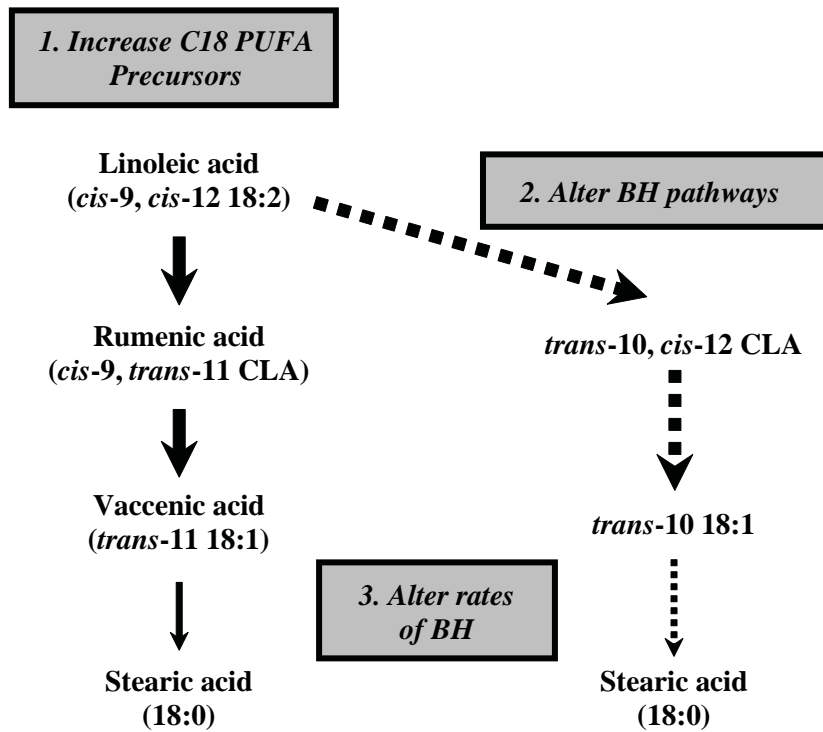


Figure 1. Generalized scheme of ruminal biohydrogenation of linoleic acid under normal conditions (left side) and during diet-induced milk fat depression (dotted lines, right side). Adapted from Bauman and Griinari (2003). The grey boxes highlight three potential means by which dietary components can increase the risk of milk fat depression.

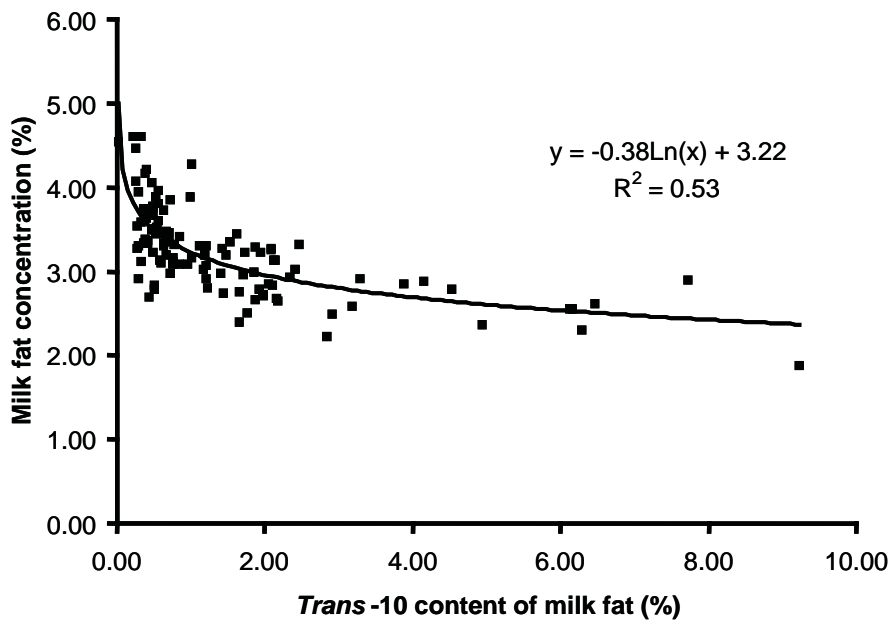


Figure 2. The relationship between the content of *trans*-10 18:1 in milk fat and milk fat percent. Adapted from Hinrichsen et al. (2006).