

Production of Low Fat Milk by Diet Induced Milk Fat Depression¹

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■ Take Home Messages

- ▶ Biohydrogenation theory may represent the unifying theory that explains the basis of diet induced milk fat depression (MFD).
- ▶ Simple feeding strategy to depress milk fat, involving dietary supplementation with one or several CLA isomers, is available.
- ▶ Optimal feeding strategies using CLAs to induce MFD are yet to be defined.
- ▶ Low roughage and high oil diets represent traditional dietary means for production of low fat milk; feasibility of these strategies may be limited due to the associated animal health and product quality concerns.
- ▶ Economics of low fat milk production depend on relative prices of milk components and the ability of lactating dairy cow to make use of the energy spared when milk fat synthesis is reduced.

■ Introduction

In many Western societies, consumption of milk fat in the form of fluid milk products and butter has decreased during the past 20 years resulting in surpluses of milk fat and consequently in low world market prices of butter. In some countries, component-pricing systems that favor production of milk protein have been established with limited success in reducing milk fat surpluses. It may not be easy to balance consumer demand and production of milk components,

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yet it is easy to agree with the statement presented in a review by Kennelly (1996): "Matching production of milk components to demand improves biological efficiency and ultimately enhances the overall profitability of the dairy industry".

Fat is the most variable milk component and is affected markedly by physiological and environmental factors (see reviews by Sutton, 1989; Grummer, 1991; Palmquist et al., 1993; Doreau et al., 1999). Physiological factors generally involve changes in energy balance and offer little potential as a practical means of manipulating milk fat. However, nutrition is the predominant environmental factor affecting milk fat and represents a practical tool to alter its yield. Previously, various aspects of dairy cow diet relative to milk fat synthesis have been discussed in this seminar (see papers by Chalupa & Sniffen, 2000; Beauchemin & Rode, 1999). Current paper discusses MFD as one of the most striking examples of diet induced regulation of milk fat synthesis. Feeding strategies that induce MFD could be used as a practical tool in dairy industry in situations that favour production of low fat milk.

MFD is a challenging biological problem involving the interrelationship between digestive processes and tissue metabolism. A number of theories have been proposed to explain the cause of diet induced MFD, but no single theory is universally accepted as the basis. Recent studies have provided new evidence for the role of specific *trans* fatty acids in the development of MFD. Their involvement was first proposed 30 years ago by Davis and Brown (1970) when they observed that *trans*-C_{18:1} fatty acids were increased in milk fat of cows exhibiting MFD. In the following sections we will review MFD and present recent studies that elucidate its possible cause.

■ Background for MFD

The earliest recognition of MFD was in 1845 when Boussingault observed reduced milk fat yield in dairy cows fed with beets. Through the first half of the twentieth century, MFD was observed for a range of diets including supplements of fish oil, diets high in grain and low in roughage (HG/LR), and diets containing plant oil supplements (see review by Bauman and Griinari 2000). Powell (1939) also established that the fat content of milk was also affected by the physical characteristics of the roughage (e.g. grinding or pelleting). This effect was demonstrated during the 1939 World's Fair exhibit of the first rotary milking parlor when the Borden Company fed 150 cows an all pelleted diet and encountered MFD (Erdman, 1996). Over the last 50 years there has been substantial research addressing MFD and practical recommendations have been developed for producers to minimize its occurrence (see reviews by Sutton 1989; Palmquist et al., 1993). This research has also identified several general characteristics that provide insight into the biology of MFD.

Diets causing MFD are commonly divided into two groups. The first group involves diets that provide large amounts of readily digestible carbohydrates and reduced amounts of fibrous components, the most common of these being the high grain/low roughage (HG/LR) diet. However, diets where the roughage content is adequate but it's ground or pelleted also fall into this group because these processes reduce fiber's effectiveness in maintaining normal rumen function. In the following sections, "low roughage diet" is used as a common description for these diets. The second group of diets that induce MFD represent dietary oil supplements high in polyunsaturated fatty acids (e.g. plant and fish oils or products containing these oils; i.e. "high oil diet"). The extent of the reduction in milk fat yield can be modified by a number of factors such as diet preparation (e.g. mixing time of TMR), the presence of other dietary components (e.g. buffers), management practices (e.g. TMR vs. separate feeding of grain and roughage, feeding frequency), and animal dimensions (e.g. stage of lactation) (see reviews by Grummer 1991; Palmquist et al., 1993). Reviews of MFD have often considered low roughage and high oil diets independently, as in fact they may represent different facets of a single pivotal process.

Low roughage diets result in changes in the rumen microbial processes (Van Soest, 1963; Davis and Brown, 1970). Powell (1939) first recognized this and he cogently observed "there is apparently a positive correlation between the activities of the rumen and the composition of the milk produced." Changes in microbial processes are characterized by changes in rumen microbial populations, by an altered pattern of fermentation end products and often by reduced rumen pH. These changes are important indicators of an altered rumen function, but low roughage diet induced change in rumen biohydrogenation appears to be a prerequisite for MFD to occur. Furthermore, presence of dietary unsaturated fatty acids appears to be in a permissive role regarding induction of MFD. Plant oil supplements provide unsaturated fatty acids, but if roughage intake is high, rumen biohydrogenation is unaltered and milk fat yield is normal (Brown et al., 1962). Likewise, a low roughage diet results in prerequisite alteration in biohydrogenation, but if the diet contains minimal unsaturated fatty acids then there is little effect on milk fat yield (Griinari et al. 1998). When combinations of grain, oilseed and byproduct feeds are fed, unsaturated fatty acids are often present in sufficient amounts MFD to be induced by low roughage content of the diet. Dietary supplementation with oils or increased level of unsaturation of the dietary fat appears to lower the threshold for low roughage diet induced MFD. Based on the above, we propose that the pivotal process affected both by low roughage and high oil diets resulting in MFD is ruminal biohydrogenation.

■ Theories of MFD

Over the last century numerous theories have been postulated to explain the cause of MFD. They can be broadly summarized into two categories: theories

that consider the reduction in milk fat yield to be a consequence of a shortage in the supply of lipid precursors for mammary gland synthesis of milk fat, and theories that attribute the reduction in milk fat to a direct inhibition of one or more steps in the mammary gland synthesis of milk fat. Theories in the former category include acetate deficiency, β -hydroxybutyrate deficiency, and the glucogenic-insulin theory, while examples in the latter category include vitamin B₁₂/methyl-malonate and *trans* fatty acids (see review by Bauman and Griinari, 2000). Several of the more widely investigated theories will be considered below.

Acetate and β -hydroxybutyrate Deficiency Theories

The theory that MFD was the result of an acetate deficiency had many supporters, and was based on the importance of acetate as a carbon source for fat synthesis and the observed shift in the rumen pattern of VFA with a low roughage diet. Specifically, the molar ratio of acetate:propionate is markedly decreased in rumen fluid, and subsequent work established a clear relationship between changes in the ruminal VFA pattern and the reduction in milk fat yield for low roughage diets (see reviews by Van Soest, 1963; Davis and Brown, 1970; Sutton, 1985). Changes in VFA proportions were associated most closely with feeding of low roughage diets. Changes in the pattern of ruminal VFA are of a smaller magnitude and less consistent when high oil diets are fed (Davis and Brown, 1970; Doreau et al., 1999).

The acetate deficiency theory has been examined by using infusions of acetate. Davis and Brown (1970) summarized these investigations and found that slight to modest increases in milk fat yield resulted from providing supplemental acetate to cows fed normal diets or diets causing MFD. They concluded that a simple deficiency in acetate could not adequately explain the reduction in milk fat yield. Most investigations of the relationship between rumen VFA and MFD have reported VFA results as molar proportions and equated these values with VFA production rates. However, molar proportions do not necessarily equate with actual production rates, particularly when comparisons are made across diets (Bauman et al., 1971; Sutton, 1985). For low forage diets, the reduction in the molar proportion of acetate is mainly due to an increase in propionate concentration rather than a reduction in acetate. The critical kinetic studies demonstrated that ruminal production rates of acetate did not differ between control and low roughage diets (Table 1). Thus, results offer little support for the acetate deficiency theory as the cause of MFD, even for low roughage diets.

A deficiency in β -hydroxybutyrate as the cause of MFD has also been proposed. This theory is based on the anti-ketogenic effect of propionate and it suggests that increased formation of propionate that occurs when on low roughage diets are fed would reduce hepatic synthesis of ketones and hence, limit the supply of β -hydroxybutyrate. While kinetic studies did establish that rates of propionate production were increased on low roughage diets (Bauman et al., 1971), whole

animal turnover rates of β -hydroxybutyrate were unaltered even though milk fat yield was substantially decreased (Palmquist et al., 1969) (Table 1). These results, together with the fact that β -hydroxybutyrate only contributes a maximum of 8% of the milk fatty acid carbon (Palmquist et al., 1969), offer little support for this theory as the basis for diet-induced MFD.

Table 1. Volatile fatty acids (VFA) and the low-fat milk syndrome.

Variable	Diet	
	Control	HG/LR
Milk fat yield, g/d ^a	683	363
Rumen VFA, molar percent ^a		
Acetate	67	46
Propionate	21	46
Butyrate	11	9
Acetate:propionate ratio	3.2	1.0
Rumen production, moles/d		
Acetate ^b	29.4	28.1
Propionate ^c	13.3	31.0
Whole body entry, mg/min·kg ⁷⁵		
b-hydroxybutyrate ^d	3.40	4.43

^aAveraged from Davis (1967) and Bauman et al. (1971).

^bDavis (1967).

^cBauman et al. (1971).

^dPalmquist et al. (1969).

Glucogenic-Insulin Theory

Endocrine status can alter the partitioning of nutrients to specific tissues. The glucogenic-insulin theory is based on tissue differences in the endocrine regulation of nutrient utilization. Insulin has acute effects on adipose tissue rates of lipogenesis (stimulatory) and lipolysis (inhibitory), but lipid metabolism in the ruminant mammary gland is unaffected by changes in circulating insulin. This forms the basis for the glucogenic-insulin theory as the cause of the MFD that occurs with diets high in readily digestible carbohydrates (characteristic feature of low roughage diets). Propionate and glucose are secretagogues for pancreatic release of insulin, and low roughage diets result in increased ruminal production of propionate (Bauman et al., 1971) and increased hepatic rates of gluconeogenesis (Annison et al., 1974). In turn, elevated circulating concentrations of insulin, often two- to fivefold, would enhance uptake of lipogenic precursors and decrease the release of fatty acids by adipose tissue. Thus, adipose tissue use of acetate, β -hydroxybutyrate and diet-derived long chain fatty acids would increase, and mobilization of long chain fatty acids from body reserves would be reduced. According to the theory, these changes have an overall effect of preferentially channeling nutrients to adipose tissue, thereby

causing a shortage of lipogenic precursors for milk fat synthesis by the mammary gland.

The glucogenic-insulin theory has been widely accepted as the basis for MFD. The theory can be examined by several different approaches, including altering circulating insulin by exogenous infusions of propionate or glucose. Davis and Brown (1970) summarized 13 trials with propionate infusion and found that the reduction in milk fat yield was quite variable ranging from 0 to 14%. As presented in Figure 1, we have summarized 24 studies involving glucose infusions, and also observed variable results with responses in milk fat yield ranging from +4 to -16% (Bauman and Griinari 2001).

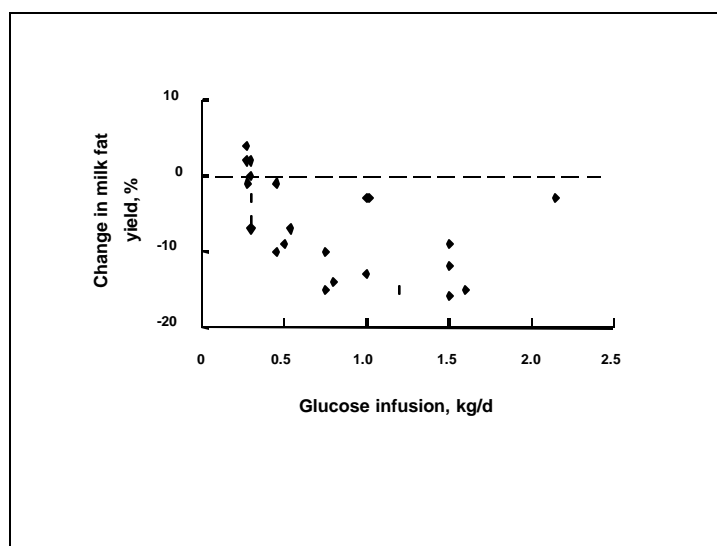


Figure 1. Summary (n = 24 studies) of the effects of abomasal infusion of glucose on milk fat yield in lactating cows. Percent change calculated from studies by Storry and Rook (1965b), Fisher and Elliot (1966), Vik-Mo et al. (1974), Spires et al. (1975), Clark et al. (1977), Frobish and Davis (1977a), Orskov et al. (1977), Rogers et al. (1979), Peel et al. (1982), Whitelaw et al. (1986), Dhiman et al. (1993), Dhiman and Satter (1993), Oldick et al. (1997), Lemosquet et al. (1997), Léonard and Block (1997) and Hurtaud et al. (1998). Adapted from Bauman and Griinari, 2001.

Evaluating the involvement of insulin in MFD is complicated by the central role of this hormone in glucose homeostasis, and stimulation of insulin release by use of secretagogues leads to counter-regulatory changes. We examined insulin's role in milk fat synthesis using a chronic hyperinsulinemic-euglycemic clamp to avoid hypoglycemia and counter-regulatory changes in glucose homeostasis. During a 4-day insulin clamp, milk yield was maintained and there was no evidence of insulin resistance based on the constancy of circulating insulin concentrations (~4-fold elevation), the exogenous glucose required to maintain euglycemia (2 to 3 kg/d), and the reduction in circulating nonesterified fatty acids (antilipolytic effect) (McGuire et al., 1995; Griinari et al., 1997b; Mackle et al., 1999). We observed minimal effects on milk fat synthesis during the insulin clamps (Table 2).

Table 2. Effect of hyperinsulinemic-euglycemic clamp on milk fat yield (g/d)¹

Reference	Standard Diet		Supplemental Protein ²	
	Control	+ Insulin	Control	+ Insulin
McGuire et al. (1995)	1260	1300	-	-
Griinari et al. (1997)	910	850	1010	940
Mackle et al. (1999)	860	770	860	830

¹ During the 4-day hyperinsulinemic-euglycemic clamps circulating insulin was approximately 4-fold over basal levels. Values represent milk fat yield on day 4 of the insulin clamp.

² Supplemental amino acids were provided by abomasal infusion of casein or casein plus branched chain amino acids.

Circulating insulin concentrations are related to energy balance and several studies report dietary situations where insulin is elevated but the yield of milk fat is unaltered. As previously discussed, induction of MFD requires alterations in the rumen microbial processes as well as the dietary presence of unsaturated fatty acids. To test this further we formulated a low roughage diet with minimal unsaturated fatty acids. This diet caused a doubling of circulating insulin, decreased plasma non-esterified fatty acids and a reduction in the rumen ratio of acetate:propionate, but effects on milk fat yield were minimal (Griinari et al., 1998). Thus, the changes in circulating insulin and lipogenic enzymes in adipose tissue would appear to be a response to energy balance (more positive) characteristic of cows fed low roughage diets rather than a cause of the MFD.

Trans Fatty Acid Theory

Direct inhibition of milk fat synthesis by *trans* fatty acids was first proposed by Davis and Brown (1970) and elaborated by Pennington and Davis (1975), based on observed increases in *trans*-C_{18:1} fatty acids in milk fat during MFD. *Trans* fatty acids are formed as intermediates in rumen biohydrogenation and *trans*-11 C_{18:1} is the predominant *trans* isomer produced, as illustrated by the pathway for the biohydrogenation of linoleic acid (Figure 2). However, analysis of positional

isomers demonstrated the presence of *trans*-C_{18:1} isomers ranging from *trans*-4 to *trans*-16 in both rumen contents and milk fat (see review by Bauman and Griinari 2000).

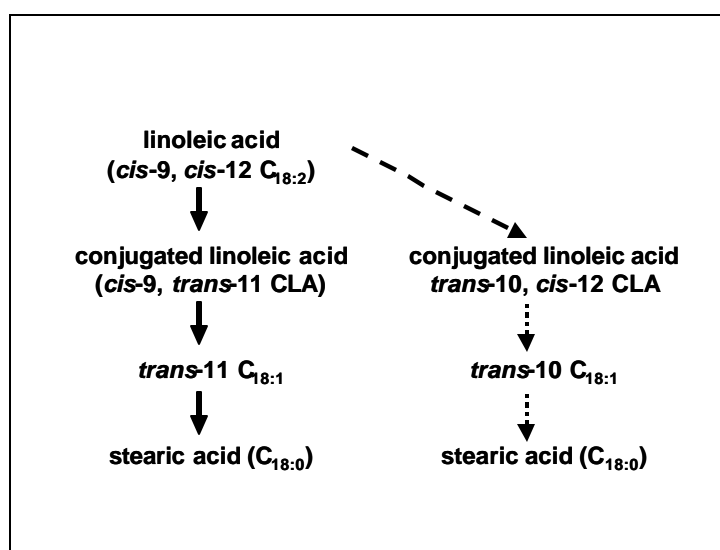


Figure 2. Pathways of the ruminal biohydrogenation of linoleic acid. Adapted from Griinari and Bauman (1999).

Moore and Williams (1963) were among the first to report an increase in the *trans*-C_{18:1} fatty acid content of milk fat when cows were fed a low roughage, high cottonseed oil diet. Storry and Rook (1965b) also observed an increase in *trans*-C_{18:1} with a classical low roughage diet. As the database grew, it became evident that MFD was related to an increase in the *trans*-C_{18:1} content of milk fat across a wide range of diets and these were summarized in our earlier papers (Griinari et al. 1995; Griinari et al. 1998). In addition, feeding or abomasal infusion of partially hydrogenated vegetable oil (PHVO) as a source of *trans*-C_{18:1} caused a 14 to 25% reduction in milk fat yield (Selner and Schultz (1980); Gaynor et al 1994; Romo et al 1996). The fatty acid patterns of the infused PHVO were not reported, but commercial preparations typically contain a range of *trans*-C_{18:1} isomers and would also contain other intermediates from chemical hydrogenation. The only studies to examine pure *trans*-C_{18:1} isomers observed no effect on milk fat yield with abomasal infusion of 25 g/d of *trans*-9 C_{18:1} (Rindsig and Schultz, 1974) or 25 g/d of an equal mixture of *trans*-11 and *trans*-12 C_{18:1} (Griinari et al., 2000). In addition, Selner and Schultz (1980) fed 500 ml

of oleic acid and observed a 3-fold increase in the *trans*-C_{18:1} content in milk fat with no MFD, indicating that the *trans*-C_{18:1} isomers resulting from dietary oleic acid supplementation did not influence milk fat yield.

Our recent work has demonstrated that the pattern of *trans* isomers rather than total *trans* fatty acids is a key dimension in diet induced MFD. We initially observed that the increase in *trans*-C_{18:1} fatty acids in milk fat was associated with MFD only in cows fed a low roughage and not in cows fed high forage diets (Griinari et al., 1995). We speculated that this discrepancy could be explained by differences in the profile of *trans*-C_{18:1} fatty acids produced in the rumen. Subsequent analysis showed that the decrease in milk fat yield was associated with an altered *trans*-C_{18:1} profile characterized by an increase in the proportion of *trans*-10 C_{18:1} (Griinari et al 1998). Others have confirmed these results (Newbold et al., 1998; Piperova et al., 2000), and in subsequent work we demonstrated that a specific increase in *trans* -10 C_{18:1} also occurred with other types of diets that cause MFD (Griinari et al., 2000a). Based on this, we postulated that *trans*-10 C_{18:1} or related metabolites could be the cause of MFD (Griinari et al. 1997a; Griinari et al. 1998).

The putative biohydrogenation pathway for the formation of *trans*-10 C_{18:1} is presented in Figure 2. Consistent with this, we found a linear relationship between the concentration of *trans*-10 C_{18:1} and *trans*-10, *cis*-12 CLA in milk fat (Griinari et al. 1999). Milk fat concentrations of *trans*-10, *cis*-12 CLA varied between 0.02 and 0.2% of total fatty acids and represented <5% of the levels of *cis*-9, *trans*-11 CLA. It is of particular importance, that there was also a curvilinear relationship between the reduction in milk fat yield and the increase in milk fat content of *trans*-10, *cis*-12 CLA in cows fed a low roughage diet supplemented with sunflower oil (Figure 3). Moreover, we have observed a similar curvilinear relationship across a range of diets that cause MFD (Griinari et al., 2000a). Thus, the altered rumen biohydrogenation associated with MFD results in an increased ruminal formation and milk fat content of *trans*-10, *cis*-12 CLA. Our recent studies examined specific CLA isomers and found that *trans*-10, *cis*-12 CLA markedly reduced milk fat secretion whereas *cis*-9, *trans*-11 CLA had no effect. Thus, the specific CLA isomer that increases in milk fat during MFD is a very potent inhibitor of milk fat synthesis.

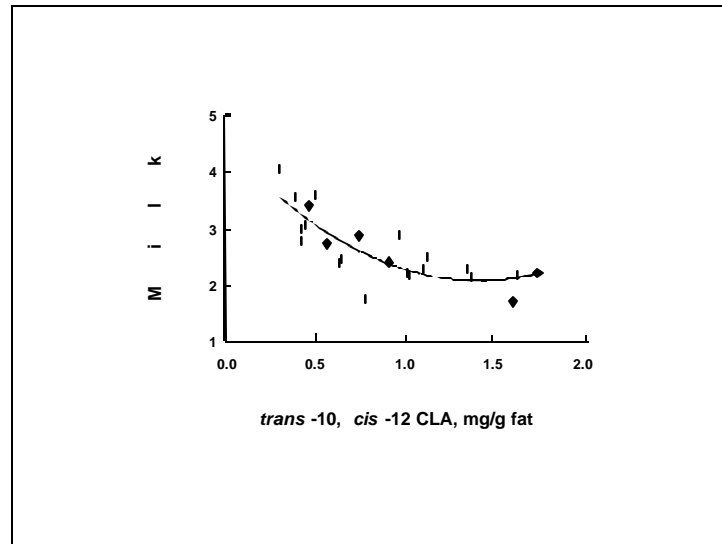


Figure 3. Relationship between milk fat content of *trans*-10, *cis*-12 conjugated linoleic acid (CLA) and milk fat percentage for cows fed a low-fiber diet supplemented with sunflower oil. Adapted from Griinari et al. (1999).

Biohydrogenation Theory

Recent advances have substantially increased our understanding of the cause of diet-induced milk fat depression. Based on our results, the trans theory of MFD needs to be modified. We suggest it be referred to as the “biohydrogenation theory” of MFD because under certain conditions rumen biohydrogenation results in unique fatty acids that are potent inhibitors of milk fat synthesis. Our work has clearly shown the inhibitory effects of *trans*-10, *cis*-12 CLA, and based on studies with different CLA enrichments we have suggested *cis/trans* 8,10 CLA may also be inhibitory (Baumgard et al. 2000). We further speculate that other unique biohydrogenation intermediates might also be inhibitory, e.g. *cis*-6, *trans*-10, *cis*-12 and *trans*-10, *cis*-12, *cis*-15 conjugated octadecatrienoic acids, intermediates formed by the putative biohydrogenation pathways involving isomerization of the *cis*-9 double bond of γ -linolenic acid and α -linolenic acid, respectively (Griinari and Bauman 1999). The original trans theory was based on rumen-derived *trans*-C_{18:1} fatty acids acting as inhibitors of milk fat synthesis, but as previously discussed studies to date have failed to observe MFD with infusions of *trans*-9 C_{18:1}, *trans*-11 C_{18:1} or *trans*-12 C_{18:1}. Other specific *trans*-C_{18:1} isomers have not been examined and *trans*-10 C_{18:1} would be of particular interest.

Could the biohydrogenation theory for MFD represent a unifying theory that would explain the basis for diet-induced MFD? The two conditions needed for MFD across all diets, altered rumen microbial processes and the dietary presence of unsaturated fatty acids, are essential features of the biohydrogenation theory. Thus, under certain rumen conditions microbial processes are altered so that a portion of biohydrogenation involves production of *trans*-10, *cis*-12 CLA from linoleic acid and possibly related intermediates from linolenic acid. Our recent investigations of diet-induced MFD have established the relationship between the decrease in milk fat percentage and the increase in *trans*-10, *cis*-12 CLA content of milk fat. Moreover, the dramatic reductions in milk fat correspond to relatively small changes in milk fat content of *trans*-10, *cis*-12 CLA. Consistent with these results our studies with *trans*-10, *cis*-12 CLA demonstrate that this CLA isomer is an extremely potent inhibitor of milk fat synthesis. It is clear that under certain conditions rumen biohydrogenation produces unique fatty acid isomers that are central to the development of a unifying theory to explain diet-induced MFD. Several different fatty acid isomers could be involved and our results with *trans*-10, *cis*-12 CLA provide an example of dramatic effect such isomers can have in regulating milk fat synthesis.

■ Dietary Applications to Induce MFD

Based on our current understanding, a number of different MFD strategies can be used in production of low fat milk. However, many strategic questions remain to be answered. First of all, the question of short-term profitability of low fat milk production needs to be addressed. Also, possible effects on animal health and welfare as well as effects on product quality require further study.

Traditional MFD Diets

As discussed above, MFD can be induced in cows by feeding a “low roughage” diet. A diet that provides large amounts of readily digestible carbohydrates and reduced amounts of fibrous components and/or forage in form that does not maintain normal rumen function. The second group of diets that induce MFD is represented by dietary oil supplements high in polyunsaturated fatty acids (“high oil diet”). More detailed description on these diets can be found in previous reviews (Sutton 1989; Erdman 1996; Palmquist et al., 1993).

Feeding cows a low roughage diet is associated with increased rumen acidity, which has been linked to an increased occurrence of lameness. Also, feeding a low roughage diet could lead to over-conditioning as well as increased incidence of ketosis and displaced abomasum (Kennelly 1996). Controlled studies evaluating long-term effects of feeding MFD diets have not been conducted. However, a survey among New York DHIA low fat herds revealed no adverse consequences of feeding diets that resulted in significantly lower rolling herd average fat percent (3.2 vs. 3.7%). Comparisons were made using available

data including parameters of milk production, health and reproduction (Griinari et al. 1995). Although the minimum level of forage fiber to maintain healthy rumen function is lower than the minimum level to prevent MFD (Beauchemin and Rode 1998), maintenance of normal rumen function with low fiber diets can be a rather difficult "balancing act" (Stone 1999). High oil diets that induce MFD will modify milk fat and this may result in altered milk fat stability. Fish oil supplementation has been evaluated for routine use as a MFD agent with rather disappointing results on milk fat oxidative stability organoleptic quality of milk products (Lacasse et al. 1998; Offer et al., personal communication).

As discussed in the Background -section, a low roughage diet appears to be the prerequisite component of MFD-induction and oil supplementation appears to be in a permissive role. If this is true, it should be possible to optimize the levels of these two components in a way that minimizes any adverse effects on rumen function, animal metabolism and milk fat quality. Dietary oils could be used at relatively low levels to lower the threshold for MFD when moderately low roughage diets are fed.

MFD Induced by CLA(s)

Recent studies using post-ruminal infusions of mixtures or relatively pure preparations of CLA isomers have established that *trans*-10, *cis*-12 CLA is a potent inhibitor of milk fat synthesis. Baumgard et al. (2000) obtained 44% reduction in milk fat yield when CLA was infused at 10 g/d. Baumgard and Bauman (unpublished) also conducted a dose response study infusing *trans*-10, *cis*-12 CLA into abomasum and observed 27% reduction in milk fat yield already at daily 3.5 g dose. These studies demonstrate the range of effective doses to induce MFD.

Effects of dietary CLA in lactating animals appear to be specific for the fat component of milk. Milk yield and milk protein have generally been unchanged in short term studies involving abomasal infusion. However, the reduction in milk fat reduces the energy requirement for milk synthesis and as a consequence dietary CLA could increase protein and lactose yields if synthesis of these components was limited by energy availability. This situation would often occur in grazing dairy cows, and Medeiros et al. (2000) demonstrated that milk protein percentage and yield were increased 11 and 16% respectively, when rotationally grazed cows received 90 g/d of Ca-salts of CLA. An energy limitation might also occur in early lactation thus limiting milk synthesis, and Giesy et al. (1999) observed a 6% increase in milk yield when cows received 50 g/d of Ca-salts of CLA. Decrease in milk fat percentage was similar in these two studies (23 to 28%). Based on these results, it is evident that improvements in milk production may result when MFD is induced in early lactation and/or when energy supply is limiting. Also, differences in milk fat responses relative to CLA dose between studies using abomasal infusion and oral administration of Ca-salts of CLA

suggest that improved methods of dietary delivery would help to bring down the cost of CLA supplementation.

The key issue in determining whether potential for profitable low fat milk production exists is the production response. Milk yield or protein yield or both have to increase enough to cover reduced income from fat and to cover any additional feed costs. A number of factors need to be accounted for in determining where the breakeven profitability of low fat milk production will occur. These factors include milk production, relative values of fat, protein and milk volume, feed cost and other input costs. With current component pricing policy in Finland (relative values 1.25 and 3.0 for fat and protein, respectively), 0.1% decrease in milk fat would be compensated by 0.2 kg increase in milk with no change in protein content.

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