CAUSES OF MILK FAT DEPRESSION IN DAIRY COWS

Mustafa Selcuk ALATAS[1], O Baris CITIL[2], Oguzhan KAHRAMAN[1], Abdullah OZBILGIN[1]

[1]Department of Animal Nutrition and Nutritional Disorders, Faculty of Veterinary Medicine,
Selcuk University, Konya 42100, Turkey
[2]Department of Veterinary, KarapinarAydoganlar Vocational School of Higher Education,
Selcuk University, Karapinar/Konya, Turkey

Corresponding author email: selcukalatas@gmail.com

Abstract

Fat is typically the most variable component in milk, and is affected by many physiological and environmental factors. In dairy cattle, both the concentration and composition of milk fat are influenced by the diet. Concentration is reduced by feeding diets that contain large proportions of readily-fermentable carbohydrates (starch) and unsaturated fat. Conversely, the percentage of fat in milk can be increased by feeding rumen-inert fats.
Milk fat depression is due to changes in rumen biohydrogenation of unsaturated fatty acids and the passage of specific intermediates of biohydrogenation out of the rumen (e.g. trans-10, cis-12 conjugated linoleic acid) that subsequently reduce milk fat synthesis in the mammary gland. Low milk fat tests typically occur as a result of several concurrent diet or management factors rather than as a result of a single factor. Low rumen pH is a key change in the rumen environment that may lead to flux of fatty acids through alternate pathways of ruminalbiohydrogenation. In this situation, high concentrate/low forage diets or dietary supplements of plant oils or fish oils cause a dramatic decline in milk fat secretion, whereas yields of milk and other milk components remain unchanged. If the feeding of unsaturated fats reduces the numbers or activity of fiber-digesting bacteria in the rumen, then feed intake can decrease, milk production can decrease, and milk fat concentration can decrease.
This review contains the information about the milk fat depression which is caused by improper feeding. And also its huge economic losses in farm animals.

Key words: Milk Fat Depression, Dairy Cattle, Concentrate Diet, Biohydrogenation.

1. INTRODUCTION

Fat is the major energy component in milk and accounts for many of the physical properties, manufacturing characteristics, and organoleptic qualities of milk and milk products. Dairy producers have long been interested in milk fat in ruminants because of its economic value, and research has been directed toward understanding the biosynthesis of milk fat and the factors that influence its quantity and fatty acid composition.

Several factors can contribute to change the proportion and total amount of fat in the milk. The most common include genetic makeup, environment, health, season of the year, and physiological state; but fat content and composition of milk can be markedly affected by diet. The fat content in milk can be altered positively or negatively by dietary changes. Synthesis of milk fat is an energy demanding process, but also represents a significant portion of the economic and nutritional value of dairy products.

Milk fat depression (MFD) is classically observed in ruminants fed highly fermentable diets or diets high in plant oils. Varying levels of MFD are commonly experienced today in both intensively and extensively managed dairy herds, and this represents a level of milk fat production below the genetic potential of the cow. Milk fat depression is also a useful variable for evaluating herd management. In many cases onset of diet-induced MFD is an indication of modified ruminal fermentation and in more pronounced cases this can be associated with ruminal acidosis and reduced efficiency. Therefore, maintaining optimal milk fat synthesis has value beyond the milk fat sold. Although we know extensively the cause of MFD we continue to experience MFD because of the high-energy requirements of cows and

the desire to maintain optimal milk production. Numerous dietary factors commonly interact to cause MFD, making prediction difficult.

2. MILK FAT SYNTHESIS

Milk fat is a major component in milk, which is played an important role in supplying energy and accounts for many physical properties and manufacturing characteristics of milk and milk products (Bauman and Griinari, 2001). The content of lipid present in cow milk is usually about 4% (Garton, 1963). In milk fat, the most predominant lipid class (more than 95%) is TAG, followed by approximately 2% of diacylglycerol (DAG), while other lipids include small amounts of phospholipids and cholesterol, about 1 and 0.5%, respectively, and a very small fraction of free FA (about 0.1%) (Jensen and Newberg, 1995). In addition, trace amounts of ether lipids, hydrocarbons, fat-soluble vitamins, flavor compounds and compounds introduced by the feed are present in milk fat (Parodi, 2004).

Milk fat is mainly composed of triacylglycerol (TAG) with 3 fatty acids (FA) esterified into the glycerol-3-phosphate backbone. Fatty acids are classified according to carbon chain length and saturation. Based on chain length, FA are grouped as short-chain FA (4-8 carbons), medium-chain FA (10-14 carbons), and long-chain FA (16 and more carbons). Fatty acids are also classified by desaturation, including saturated FA (no double bond), monounsaturated FA (one double bond), and polyunsaturated FA (more than one double bond).

There are two sources of FA for milk fat synthesis, the *de novo* FA synthesis in mammary epithelial cells and preformed FA uptake from blood circulation derived from either diet or mobilized body fat (Barber et al., 1997). To synthesize milk fat, many enzymatic activities are involved in the pathways, including FA activation, transport, desaturation, TAG synthesis, milk fat globule formation and secretion (Clegg et al., 2001).

3. THE MILK FAT SYNDROME

Most of these theories postulated that limitations in substrate supply for milk fat synthesis caused MFD, generally based on changes in absorbed metabolites as a consequence of alterations in ruminal fermentation. For example, the alterations in the ruminal environment typically include decreased pH and decreased acetate to propionate molar ratio (Bauman and Griinari, 2001). This formed the basis for one of the most widely known substrate supply limitation theories that proposed that acetate supply was limiting milk fat synthesis. However, the reduced ratio of acetate to propionate with highly fermentable diets is predominantly due to increased ruminal production of propionate (Bauman and Griinari, 2001, 2003), and ruminal infusion of acetate to cows during MFD has only a marginal impact on milk fat yield (Davis and Brown, 1970). Overall, several decades of research has tested numerous theories based on substrate limitations and found little to no evidence in their support (Bauman and Griinari, 2003; Bauman et al., 2011; Shingfield and Griinari, 2007).

Davis and Brown (1970) recognized that trans-C18:1 fatty acids (FA) were increased in milk fat of cows with low-milk fat syndrome. They suggested that these trans-FA originated from incomplete ruminalbiohydrogenation of unsaturated FA and might contribute to the development of MFD. Subsequent studies have demonstrated a clear relationship between trans-FA and MFD (Bauman and Griinari, 2003; Bauman et al., 2011; Shingfield and Griinari, 2007). Investigations over the past dozen years have clearly established that diet-induced MFD is associated with rumen production of unique FA from ruminal metabolism of dietary polyunsaturated fatty acids (PUFA). Referred to as the *biohydrogenation theory*, the basis for diet-induced MFD relates to an inhibition of mammary lipid synthesis by specific FA that are intermediates in the biohydrogenation of dietary PUFA, and these are only produced under certain conditions of altered ruminal fermentation (Bauman and Griinari, 2003). Trans-10, cis-12 conjugated linoleic acid (CLA) was the first of these to be recognized and it has been extensively investigated at the whole animal and molecular level (Bauman et al., 2011).

4. DIETARY RISK FACTORS FOR MILK FAT DEPRESSION

Milk fat depression has been associated with a reduction in the acetate to propionate ratio and increased insülin (Byers and Schelling, 1988; Bauman and Griinari, 2003), and the production of trans-octadecenoic acids in the rumen (Griinari et al., 1998; Bauman and Griinari, 2003). Induction of SARA by adding grain pellets or by adding alfalfa pellets to the diet reduced both milk fat and the acetate to propionate ratio in the rumen fluid, but the decrease in this ration was due to an increase in propionate and not due to a decrease in acetate (Gozho et al., 2006; Fairfield et al., 2007; Khafipoor et al., 2007). Griinari et al. (1997) found that a hyperinsulinemic–euglycemic clamp did not depress milk fat. An increase in insulin decreases lipolysis (Bauman and Griinari, 2003). This might explain why stage of lactation and energy balance could affect the milk fat depression, as the contribution that body fat makes to milk fat is much greater in cows in a negative energy balance compared to cows in a positive energy balance. The results obtained by Griinari et al. (1998) support the theory that a low rumen pH caused by feeding a low fiber diet results in incomplete biohydrogenation of fatty acids and increases in trans-octadecenoic acids, and especially the trans-10 isomer of transoctadecenoic acid, that cause milk fat depression. It is not yet understood why experimentally induced SARA increases milk protein, but an increase in rumen digestible organic matter, which increases microbial protein synthesis in the rumen (NRC, 2001) might play a role.

4.1. Diet Fermentability

The microbial population is driven by the substrate available and by the rumen environment and is directly dependent on the concentration of starch and NDF and the rates and extent of ruminal digestion. Maximizing fermentablity is important for energy intake, but care should be given to minimizing subacuteruminal acidosis. Milk fat depression more commonly occurs with corn silage compared to haylage based rations and with more rapidly digested starch sources such as high moisture corn compared to dry ground corn. Providing multiple sources of starch and fiber with overlapping rates of digestion is the safest approach. Additionally, sugar substituted for dietary starch reduces risk without loss of digestibility (Mullins and Bradford, 2010).

Low milk fat is commonly associated with subacute and acute ruminal acidosis, but MFD is frequently observed without a reduction in rumen pH (Harvatine and Allen, 2006a). Rumen pH is dependent on the VFA profile, rate of production, and rate of absorption; buffer secretion; and presence of dietary buffers and varies by approximately 1 to 1.2 pH units over the day (Allen, 1997). It appears that the microbial shift causing MFD occurs before changes in rumen pH are apparent, but may be related to more subtle changes such as the timing of low pH.

4.2. Diet Polyunsaturated Fatty Acids

Unsaturated FA have a dual impact on ruminalbiohydrogenation in that they modify the microbial population and increase the amount of substrate that must be biohydrogenated. It is important to know the total amount of unsaturated fat and also the source, since this dictates the FA profile and rate of ruminal availability. Fish oil has the greatest impact, but is not commonly found in excessive amounts in diets. Cotton, soy, corn, and many other plant oils are high in linoleic acid and incorporation of these grains, oils, and their byproducts increases the risk of MFD. The concept of Rumen Unsaturated Fatty Acid Load (**RUFAL**; Jenkins, 2011) is a simple and insightful calculation that is complemented by consideration of the fat source. There are significant differences in the rate of ruminal availability, for instance cottonseed and whole roasted soybeans are expected to have a much slower release of FA in rumen than distillers grains, ground sources, or oil supplements. Fat is commonly supplemented to increase diet energy density and many protected fat supplements are available. Supplements that are high in saturated fat (palmitic and stearic) do not increase the risk of MFD; however calcium salts of FA are available in the rumen and can reduce milk fat (Harvatine and Allen, 2006b; Lundy et al., 2004). The calcium salt

slows the release of unsaturated fat in the rumen and does reduce the impact of these oils compared to free oil, but does not provide a high level of rumen inertness. The impact of calcium salts depends on the profile of the fat supplement and interaction with other factors. For instance, we have observed in two experiments that calcium salts of palm FA reduced milk fat in high producing cows, but not in low producing cows; presumably because of differences in intake, passage rate, and rumen environment (Harvatine and Allen, 2006a; Rico and Harvatine, 2011).

4.3. Rumen Modifiers

Many supplements have a large impact on the rumen microbial population. Monensin is the most common rumen modifier associated with MFD (Jenkins, 2011). However, it is only a risk factor and can be safely used in many diets. Other rumen modifiers may reduce risk, although their effectiveness generally has not been specifically tested. For example, there may be some potential for 2-hydroxy-4 (methylthio) butanoic acid (**HMB**) to modify milk fat yield (St-Pierre and Sylvester, 2005); although its role in rumen biohydrogenation has not been specifically investigated. Additionally, a direct fed microbial product was shown to stabilize rumen biohydrogenation during a high diet fermentability challenge (Longuski et al., 2009).

4.4. Feeding Strategies

Slug feeding grain is commonly associated with sub-clinical rumen acidosis and MFD. Many assume that TMR feeding eliminates this issue since every bite has the same nutrient composition. However, the rate of intake of fermentable organic matter is very variable over the day due to sorting and variable rates of intake. Generally, cows sort for more fermentable feed particles early in the day, but also consume feed at approximately a three times higher rate after delivery of fresh feed. We recently compared feeding cows once per day or in four equal meals every six hours (Rottman et al., 2011). The frequent feeding treatment decreased the concentration of alternate biohydrogenation FA and increased milk fat yield and concentration. This experimental treatment highlights the potential to increase milk fat through management of feed delivery.

5. HOW TO PREDICT THE OCCURENCE OF MILK FAT DEPRESSION

The complexity of predicting dietary fermentability and associative effects makes prediction of MFD difficult. It is arguably impossible to balance a diet that maximizes milk yield and energy intake without incorporation of numerous risk factors. Ruminant nutrition is best practiced as a continuous experiment that monitors cow response to diet modification (Allen, 2011). It is important to monitor nutrient concentrations and model predicted benchmarks that are applicable to your region and logical based on previous experience with similar diets. However, even with the best feed analysis, software, and experience the interaction of diet ingredients and effectiveness of the diet is best determined by the cow and observed by titration and observation.

Diet fermentability is much more extensively handled by feed analysis and software prediction than dietary fat. Dietary FA have typically been consolidated in ration balancing and simply reported as total ether extract or fat concentration. More recently the FA profile of feedstuffs has been included in feed libraries and a more detailed approach of FA nutrition has been taken (Moate et al., 2004). Effectively utilizing this information in diet formulation represents a challenge because of rumen alterations of dietary FA and the fact that individual FA isomers differ in their biological effect. Thus, based on the current understanding of bioactive FA, effective models must predict ruminal outflow of individual FA, including specific trans-FA isomers. Secondly, the metabolism of FA by rumen bacteria is extremely dynamic and difficult to integrate into prediction algorithms. Ruminal FA models must account for dietary associative effects that modify the predominant pathways and rates of ruminalbiohydrogenation; thereby altering the

pattern of FA outflow. This may require a mechanistic rather than empirical approach to adequately model. Book values are expected to accurately represent the FA profile of forages and grains and testing of individual lots should not be required for most feedstuffs. However, more variability exists in byproducts, which may require frequent testing of FA concentration and profile depending on the byproduct and source. An understanding and quantification of all factors that induce altered ruminal fermentation is not currently available and development of prediction equations that consider dietary risk factors will require further experimentation and more advanced modeling.

6. CONCLUSIONS

Milk fat depression results from an interaction between ruminal fermentation processes and mammary tissue metabolism. MFD continues to be a real-world condition that reduces the efficiency and productivity of dairy cows, but understanding its fundamental basis will allow for effective management and intervention strategies. Management of the risk factors associated with MFD is required to reach both milk and milk fat yield goals. The time course of induction and recovery can be utilized to both identify contributing factors and set expectations for recovery. Lastly, the seasonal and circadian pattern of milk fat synthesis explains variation observed between summer and winter and between milkings and should be considered in monitoring and setting production goal.

REFERENCES

Allen, M. S. 1997. Relationship between fermentation acid production in the rumen and the requirement for physically effective fiber. J. Dairy Sci. 80:1447-1462.

Allen, M. S. 2011. Mind over models. Pages 29-44. *In:* Proc. 20th Ann. Tri-State Dairy Nutr. Conf., Fort Wayne, IN.

Barber, M.C., Clegg, R.A., Travers, M.T., Vernon, R.G., 1997. Lipid metabolism in the lactating mammary gland. Biochim. Biophys. Acta 1347, 101–126.

Bauman, D. E., and J. M. Griinari. 2001. Regulation and nutritional manipulation of milk fat: low-fat milk syndrome. Livest. Prod. Sci. 70:15-29.

Bauman, D. E., and J. M. Griinari. 2003. Nutritional regulation of milk fat synthesis. Ann. Rev. Nutr. 23:203-227.

Bauman, D. E., K. J. Harvatine, and A. L. Lock. 2011. Nutrigenomics, rumen-derived bioactive fatty acids, and the regulation of milk fat synthesis. Ann. Rev. Nutr. 31:299-319.

Byers, F.M.,Schelling, G.T., 1988. Lipids in ruminantnutrition. In: Church, D.C. (Ed.), The Ruminant Animal, Digestive Physiology and Nutrition. Prentice Hall, Englewood Cliffs, NJ, pp. 298–312.

Clegg, R.A.,Barber, M.C., Pooley, L., Ernens, L., Larondelle, Y., Travers, M.T. 2001. Milkfat synthesis and secretion: molecular and cellular aspects. Livstk. Prod. Sci. 70, 3–14.

Davis, C. L., and R. E. Brown. 1970. Low-fat milk syndrome. Pages 545-565. *In:* Physiology of Digestion and Metabolism in the Ruminant. A. T. Phillipson, ed. Oriel Press, Newcastle upon Tyne, UK.

Fairfield, A.M.,Plaizier, J.C., Duffield, T.F., Lindinger, M.I., Bagg, R., Dick, P., McBride, B.W., 2007. Effects of a prepartum administration of a monensincontrolled release capsule on rumen pH, feedintake, and milk production of transition dairy cows. Journal of DairyScience 90, 937–945.

Garton, G. A. 1963. The composition and biosynthesis of milk lipids. Journal of lipid research 4:237-254.

Gozho, G.N.,Krause, D.O., Plaizier, J.C., 2006. Effects of gradual adaptation to concentrate and subsequentinduction of subacuteruminalacidosis in steers on ruminallipopolysaccharide and acute phase proteins. Journal of DairyScience 89, 4404–4413.

Griinari, J.M.,McGuire, M.A., Dwyer, D.A., Bauman, D.E., Palmquist, D.L., 1997. Role of insulin in theregulation of milkfatsynthesis in dairycows. J DairySci 80, 1076–1084.

Griinari, J.M.,Dwyer, D.A., McGuire, M.A., Bauman, D.E., Palmquist, D.L., Nurmela, K.V., 1998. trans-Octadecenoicacids and milk fat depression in lactating dairy cows. Journal of DairyScience 81, 1251–1261.

Harvatine, K. J., and M. S. Allen. 2006a. Effects of fatty acid supplements on milk yield and energy balance of lactating dairy cows. J. Dairy Sci. 89:1081-1091.

Harvatine, K. J., and M. S. Allen. 2006b. Fat supplements affect fractional rates of ruminal fatty acid biohydrogenation and passage in dairy cows. J. Nutr. 136:677-685.

Jenkins, T. C. 2011. Managing the rumen environment to control milk fat depression. Pages 31-37. *In:* Penn State Dairy Nutr. Workshop, Grantville, PA.

Jensen, R. G. and D. S. Newberg. 1995. Bovine milk lipids. Handbook of milk composition. Academic Press, San Diego, CA.

Khafipoor, E.,Krause, D.O., Plaizier, J.C., 2007. Induction of subacuteruminalacidosis (SARA) by replacingalfalfa hay with alfalfapellets does not stimulate inflammatory response in lactating dairy cows. J Anim Sci 85 (Suppl. 1), 654.

Longuski, R. A., Y. Ying, and M. S. Allen. 2009. Yeast culture supplementation prevented milk fat depression by a short-term dietary challenge with fermentable starch. J. Dairy Sci. 92:160-167.

Lundy, F. P., E. Block, W. C. Bridges, Jr., J. A. Bertrand, and T. C. Jenkins. 2004. Ruminal biohydrogenationin Holstein cows fed soybean fatty acids as amides or calcium salts. J. Dairy Sci. 87:1038-1046.

Moate, P. J., W. Chalupa, T. C. Jenkins, and R. C. Boston. 2004. A model to describe ruminal metabolism and intestinal absorption of long chain fatty acids. Anim. Feed Sci. Tech. 112:79-105.

Mullins, C. R., and B. J. Bradford. 2010. Effects of a molasses-coated cottonseed product on diet digestibility, performance, and milk fatty acid profile of lactating dairy cattle. J. Dairy Sci. 93:3128-3135.

National Research Council, 2001. National Research Council. Nutrient Requirements of DairyCattle. 7th rev. ed. National Academies Press, Washington, DC.

Parodi, P. 2004. Milk fat in human nutrition. Australian J. Dairy Technol. 59:3-59.

Rico, D. E., and K. J. Harvatine. 2011. Effect of a high palmitic acid fat supplement on ruminal fermentation and milk production in high- and low-producing dairy cows. J. Dairy Sci. 94(E-Suppl. 1): 202(Abstr.).

Rottman, L. W., Y. Ying, and K. J. Harvatine. 2011. Effect of timing of feed intake on circadian pattern of milk synthesis. J. Dairy Sci. 94(E-Suppl. 1): 750(Abstr.).

Shingfield, K. J., and J. M. Griinari. 2007. Role of biohydrogenation intermediates in milk fat depression. Eur. J. Lipid Sci. Technol. 109:799-816.

St-Pierre, N. R., and J. T. Sylvester. 2005. Effects of 2-hydroxy-4-(methylthio) butanoic acid (HMB) and its isopropyl ester on milk production and composition by Holstein cows. J. Dairy Sci. 88:2487-2497.