Increasing Milk Fat Yield: Opportunities Beyond Fat Supplements

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Abstract

Increasing milk fat yield is a central goal of dairy nutritionists and producers. Milk fat synthesis is responsive to many factors providing substantial opportunity to modify milk fat both in the short and long term, but complex interactions make optimization difficult. The first recommendation is to consider genetic potential and season of the year to accurately and precisely set goals. In balancing diets, it is important to manage the risk of diet-induced milk fat depression, but minimizing the risk likely reduces energy intake and milk yield. De novo synthesis of milk fat is supported by increasing acetate supply that can be best accomplished through high quality forages and stable rumen fermentation. Dietary fat is also needed for milk fat synthesis and although a specific requirement cannot be stated, consideration of all sources and the impact of individual fatty acids both in the rumen and post-ruminally is important.

Introduction

Many cow, diet, and environmental factors interact to determine milk fat yield **(Figure 1)**. There is a large impact of nutritional and management factors that provide the opportunity to have substantial impact in the short term, although genetic factors that can be modified over the long-term should not be overlooked.

Milk fatty acids **(FA)** originate both from *de novo* synthesis in the mammary gland and uptake from plasma that come either from dietary absorption or export from other body tissues [Illustrated in **Figure 2**; (see reviews by Bauman and Davis, 1974; Bauman and Griinari, 2001; Palmquist and Harvatine, 2020)]. Nearly all FA <16 carbons originated from mammary *de novo* synthesis and nearly all FA >16 carbons originated from uptake from the plasma. The 16 carbon FA originated from both sources with roughly an equal contribution from *de novo* synthesis and plasma uptake. Synthesis of d*e novo* FA and uptake of preformed FA and their incorporation into triglycerides and secretion from the mammary epithelial cell are impacted both by substrate availability and physiological regulation of metabolic processes.

Milk Fat Impacts Profitability

As a commodity industry, dairy producers are generally price takers, but the "mailbox price" received is mainly dependent on milk fat and protein concentration in solids/cheese markets and on milk fat concentration in fluid milk markets. Milk fat has been a consistent contributor to milk value over the past two decades. For example, a 0.1 unit increase in milk fat concentration in a herd averaging 85 lb of milk would increase milk value $0.19/day, or $70/cow/year, based on the average milk fat price over the past five years ($2.26 /lb).

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Importantly, decisions should be made on the pounds of components produced and overall value of milk produced. Small decreases in milk yield can overwhelm increases in milk fat, resulting in little or no improvement in milk income. For example, a 0.1 unit increase in milk fat can be entirely offset by a 2 lb decrease in milk yield. When considering fat plus protein yield, a 0.1 unit increase in fat is offset by a 1.1 lb decrease in milk yield.

Non-Nutritional Factors Impacting Milk Fat Synthesis

Non-dietary factors include season of the year, milking and feeding management, stage of lactation, and genetics among others. We have recently characterized the strong seasonal rhythm to milk fat concentration and yield that is conserved across milk markets, herds, and cows (Salfer et al., 2019; Salfer et al., 2020). Interestingly, work from the Miner Institute has also reported a seasonal rhythm in the concentration of FA < 16 C (Dann et al., 2019). Milk fat synthesis also follows a daily rhythm with lowest milk fat in the morning and highest milk fat at the evening milking (Quist et al., 2008). The timing of feed intake impacts the daily rhythm of milk fat synthesis, with 4 x/day feeding increasing milk fat and decreasing the amplitude of the rhythm by ~50% (Rottman et al., 2014) restricting feeding to the day vs the night (Salfer and Harvatine, 2020). Lastly, milk fat is one of the most heritable production traits and cows and herds differ in their genetic potential for milk fat synthesis [heritability of 0.45 and 0.29 for concentration and yield, respectively (Welper and Freeman, 1992)]. Genomic selection and changes in selection indexes have applied additional selection pressure and milk fat percent has increased ~0.30 units (107 lb/lactation) over the past 10 years alone (Council on Dairy Cattle Breeding, https://uscdcb.com). Average milk fat concentration

has been on a linear increase in most US milk markets, likely due to improved genetic potential of the cow as well as improved nutrition and management.

Understanding and predicting the non-nutritional factors that impact milk fat concentration and yield is essential for accurately and precisely setting expectations and determining if a herd or cow is meeting their potential.

Milk Fat is Commonly Decreased by "Milk Fat Depression"

Milk fat depression (MFD) is defined as a decrease in milk fat associated with disrupted rumen fermentation and is more accurately called "Diet-induced" or "Biohydrogenation (BH)-Induced" MFD (Griinari et al., 1998; Bauman and Griinari, 2003). It is important to note that this is a specific condition and not simply any change in milk fat yield. Up to a 50% reduction in milk fat concentration and yield can be observed with no decrease in milk or milk protein yield. Extensive work over the past 20 years has demonstrated that diet-induced MFD is caused by unique bioactive conjugated linoleic acid (CLA) isomers that are made during rumen biohydrogenation of unsaturated FA by an altered rumen microbial community. Investigating this condition has provided insight into the regulation of milk fat synthesis and management strategies to reduce inhibition of milk fat synthesis (Reviewed by Harvatine et al., 2009) which has greatly aided management of the condition in the field.

Milk fat depression is caused by disrupted rumen fermentation that results in a shift in the rumen microbial population. The specific causative microbes are not clear, but a decrease in microbial diversity is apparent in microbiome analysis (Pitta et al., 2018; Pitta

et al., 2020). Prediction of the occurrence of BH-induced MFD is complex because it is not directly caused by a single dietary factor; rather it is the result of the interaction of numerous factors that reduce the rate of biohydrogenation and shift biohydrogenation to the alternate pathway. It is preferable to think of dietary risk factors that move a diet along a continuum from low to high risk. Extensive work has highlighted dietary factors that increase and decrease risk. Briefly, risk is increased by increasing diet fermentability and unsaturated fatty acids, decreasing effective fiber, ionophores, poorly fermented silages, slug feeding/eating, and other factors that decrease rumen pH or disturb normal rumen fermentation. Milk fat depression is also more likely in higher producing cows. Risk is decreased by increasing DCAD (Iwaniuk and Erdman, 2015) and feeding 2-hydroxy-4 (methylthio) butanoic acid **(HMTBA)** (Baldin et al., 2018; Baldin et al., 2019).

Large decreases in milk fat (>15%) are almost undoubtably BH-induced MFD, but this mechanism does not explain many other smaller changes in milk fat synthesis. The occurrence of BH-induced MFD is best diagnosed by milk fat concentration of *trans*-10 C18:1 (Matamoros et al., 2020), although this requires analysis by gas chromatography.

Recently there has been proposed that diet-induced MFD may be less of an issue on farms. This may be because the condition was commonly diagnosed as average milk fat below ~3.4%. It is important to remember that genetic potential and average milk fat has increased from ~3.6 to 4.0% over the past decade. The diagnosis criteria for the condition need to be continually updated during this transition. Some risk factors, such as high fat distillers grains, have been decreased, but some risk factors, such as diet fermentability, cannot be decreased without sacrificing energy intake and milk yield.

It is difficult to definitively say if the occurrence of MFD has decreased, but it is highly unlikely that it has been entirely eliminated and likely still decreases milk fat yield in some herds and some cows within many herds.

Increasing Milk Fat by Increasing Acetate Supply

Acetate is a two-carbon volatile fatty acid **(VFA)** originating from rumen fermentation. VFA provide over 70% of the energy requirement of the cow, 45% of which is contributed by acetate alone (Bergman, 1990). The amount and profile of VFA produced are dependent on diet composition, the rate and extent of fermentation, the microbial population, rumen environment, numerous other factors, and interactions between these factors, making model predictions difficult (Dijkstra et al., 1998). Acetate production is variable, but dependent on the extent of ruminal digestion, diet composition, and rumen environment, with higher acetate production from fiber sources and stable fermentation conditions. Interestingly, acetate has not been well-investigated over the past 40 years beyond measuring concentrations in rumen fluid. Before identification of bioactive conjugated linoleic acid **(CLA)** isomers as the causative factor of BH-induced MFD, acetate deficiency was proposed to limit milk fat synthesis. However, it was shown that acetate production did not decrease during MFD and investigation of acetate received little to no attention after the 1970's. Five years ago, we conducted ruminal infusions of acetate to study the effect of nutrients spared during diet-induced MFD on adipose tissue and surprisingly observed an increase in milk fat yield (Urrutia and Harvatine, 2017a). This was followed by a dose titration experiment, and we observed a linear increase in milk fat yield with a 217 g/day increase in milk fat with 600 g/day of acetate, a remarkable 36% apparent transfer (Urrutia

and Harvatine, 2017b). Recent experiments feeding sodium acetate in a TMR also observed an increase in milk fat concentration (Urrutia et al., 2019; Matamoros et al., 2021). Importantly, these observations represent an increase in milk fat under normal conditions, and not alleviation of MFD as controls were near breed average and had low concentrations of BH intermediates associated with diet-induced MFD.

Sodium acetate is not economical for dairy diets, but we expect that acetate supply can be increased by feeding highly digestible fiber and maintaining stable rumen conditions. Importantly, this is not alleviating diet-induced MFD and is separate from historical acetate deficiency mechanisms. Harvesting and storing high quality feed and managing diets and feed mixing and delivery are obvious factors but are not easy to optimize. It is important to remember that acetate is the predominant nutrient needed for *de novo* FA synthesis. When milk fat yield and FA < 16C are high, like is expected during the winter season or feeding lower fat diets, the demand for acetate is especially high and may become a limiting factor for overall fat yield.

Increasing Milk Fat by Feeding Fat

Preformed FA taken up from the blood are expected to account for half of the 16-carbon FA and all of the longer-chain FA (>16 carbons) in milk fat **(Figure 2)**. Taken together, this represents approximately 55% of the total FA. Dietary fat is not an essential nutrient as the cow has a high capacity to synthesize fat both in mammary and adipose tissues. However, it is logical that there is a level of fat intake that maximizes milk fat yield. This has not been specifically investigated and most recent work has focused on impacts of supplementing specific FA. The response to increasing dietary fat is variable. In some cases, increasing dietary fat increases preformed FA in milk fat but does

not increase milk fat yield because of an equal decrease in *de novo* synthesized FA. In these cases, mammary and adipose tissue lipogenic capacity likely had overcome deficiencies in absorbed FA when not supplementing fat. If lipogenic capacity or substrate was limited, increased milk fat is expected with fat supplementation. There is also a clear effect of FA profile, with palmitic acid supplements most consistently increasing milk fat likely due to less impacts on yield of FA < 16 C.

Palmquist et al. (1993) reported a quadratic relationship between intake and milk 18 C FA, with an intercept of 80 g/day that was interpreted as adipose synthesis, a linear term of 0.58 that was interpretted as the transfer coefficient and a quadratic term of 0.000186 that was interpreted as the maximal response that they proposed might have been due to limitations in digesibility. Abomasal infusion studies also provide key insight into tranfer of individual FA to milk fat as it elminates the confounding effects of ruminal BH and issues with quantifying duodenal flow. During abomasal infusion,, LaCount et al. (1994) reported linear transfers of increasing oleic and linoleic acids, with slopes of ~0.53. Moallem et al. (2012) also reported 45% transfer of α-linolenic acid (**ALA**, C18:3 n-3) to milk fat. These transfer efficiencies set expectations on the potential for dietary fat to increase milk fat yield if de novo synthesis is not decreased.

Early recommendations by Palmquist and others over 40 years ago proposed feeding cows the same amount of fat as they are producing (Reviewed by Palmquist and Jenkins, 2017). We recently revisited this concept when contemplating the very high levels of milk fat achieved by some cows and herds. Since milk fat is generally ~55% preformed FA and FA are expected to have roughly a 54% transfer to milk (discussed above), we arrive at a calculated

feeding rate nearly equal to milk fat yield [Intake = (0.55 * milk fat) / 0.54)]. However, this would require feeding diets >8% fat to many high producing cows, which is well above what will inhibit rumen fermentation and challenge FA digestibility. This led us to explore transfer efficiency outside of infusion experiments. Khiaosa-Ard et al. (2015) reported greater than the expected 54% transfer of 18 C FA to milk fat in the majority experiments with 18 C FA < 2.5%, with apparent transfers above 100 and 200% common. We have observed a similar relationship in our own meta-regression, and the relationship is even more clear when points within the same experiment are joined **(Figure 3)**. Interestingly, the average dairy TMR in the Cumberland Valley Forage Labs database of 8,180 samples was 2.3% 18 C FA (Std Dev = 0.7; 2019 personal communication), indicating that half of diets fed would be resulting in higher than expected transfer efficiency. We have also observed apparent transfer efficiencies at or above 100% of 18 C FA in low fat diets that was decreased with increasing dietary fat in high producing cows.

Palmitic acid supplements are the most consistent to increase milk fat, although milk fat increases have been observed with other fat sources with more variable responses. For example, de Souza et al. (2018) increased milk fat from 3.34 to 3.87% (0.13 kg/day) by feeding 10% whole cottonseed in substitution for soyhulls, while Rico et al. (2017) in the same research group saw no response to a similar increase in cottonseed in cows making 3.86% milk fat. In a fat supplement study, Relling and Reynolds (2007) reported a 187 g/day increase in milk fat (3.37 to 3.86%) in cows fed a blend of palmitic and stearic acids substituted for corn grain. Lastly, we observed that increasing roasted soybeans from 5 to 10% of the diet increased milk fat 92 g/day (3.35 to 3.56%) in one study (Bomberger et al., 2019),

but increasing roasted soybeans from 0 to 15% of the diet had no effect in another study when milk fat averaged 4.08% in the low fat control (Khonkhaeng et al., 2020). When milk fat response were not observed, milk 18 C FA were increased, but *de novo* FA were decreased, counteracting an increase in milk fat yield, which is a well described response (Palmquist et al., 1993).

The cost of energy feeds has drastically increased over the past year with strong increases in both corn grain and multiple oil and FA sources. It is difficult to predict future prices, but economic incentives for biofuels and carbon credits may continue to pressure the cost of energy feeds. The first recommendation is to accurately account for all FA sources in the diet, including corn silage, that can vary considerably in FA concentration. Oilseeds may be an economical and home-grown source of FA in some cases and high oleic soybeans provide a lower-risk of diet-induced MFD. Lastly, it will be important to determine the total FA feeding level and FA profile that optimally balances energy intake, milk and milk fat yields, and body weight gain.

Conclusions

- Milk fat is impacted by many dietary, genetic, and environmental factors and their interactions make it difficult to manage.
- It is important to consider non-nutritional factors, such as genetic potential, season of the year, and milking sampled when setting goals and interpreting data.
- BH induced MFD explains large decreases in milk fat and is caused by fundamental issues with stable rumen fermentation.
- Increasing acetate supply by increasing fiber digestibility supports higher milk fat yield.
- Increasing dietary fat can increase milk fat but is most consistent when feeding enriched palmitic acid supplements.

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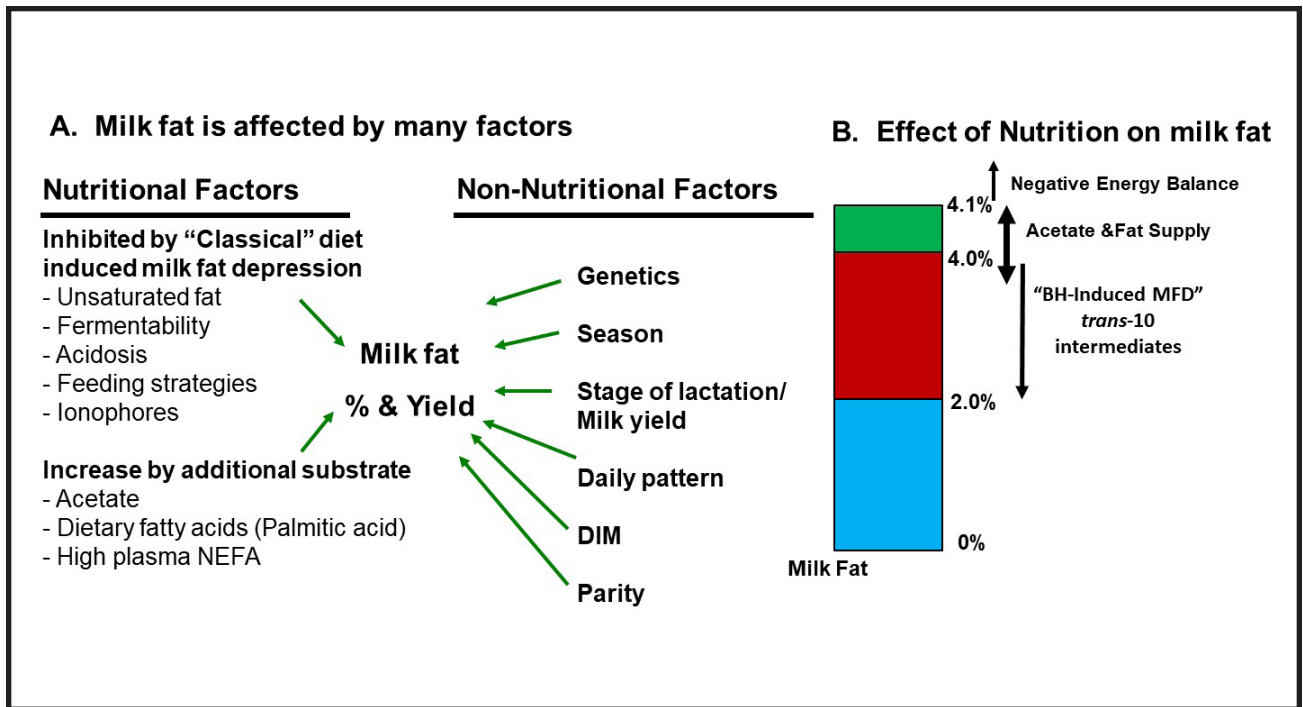
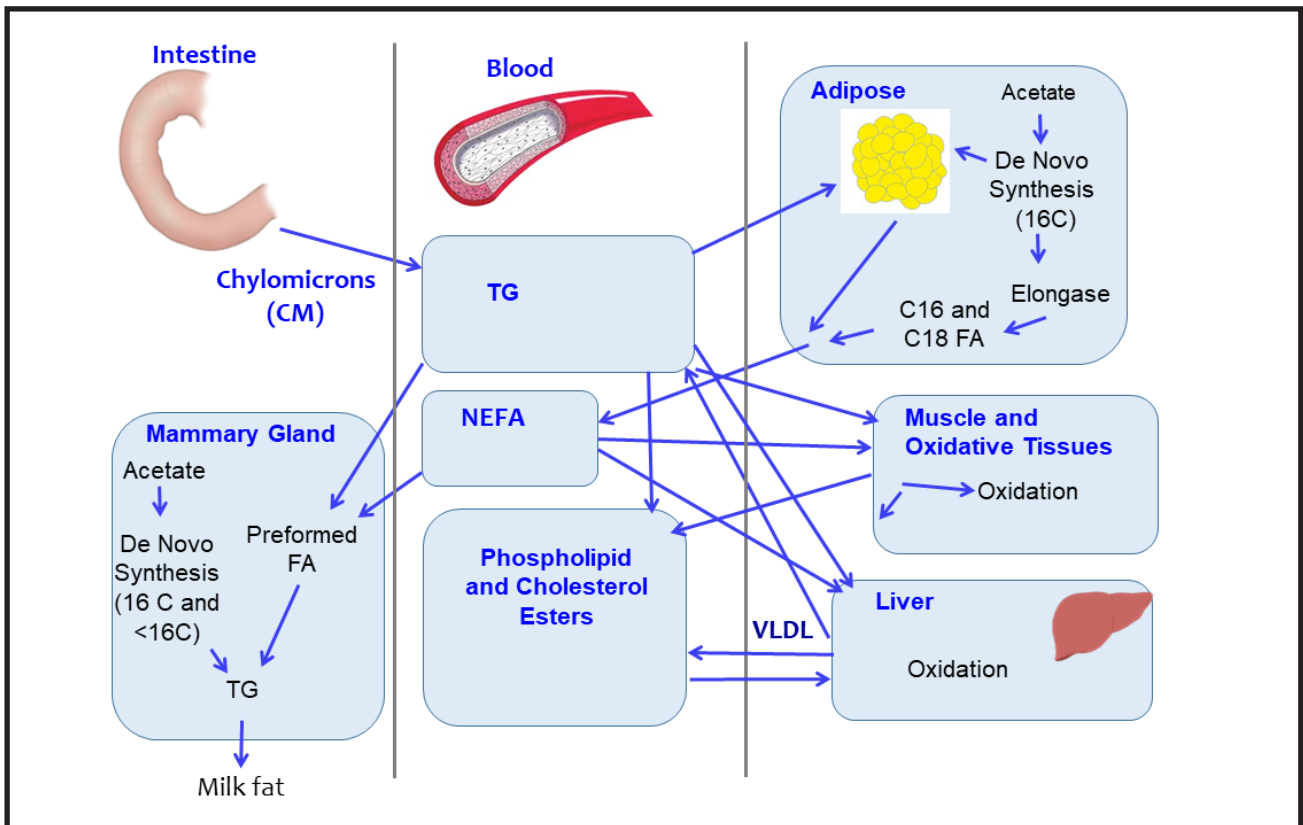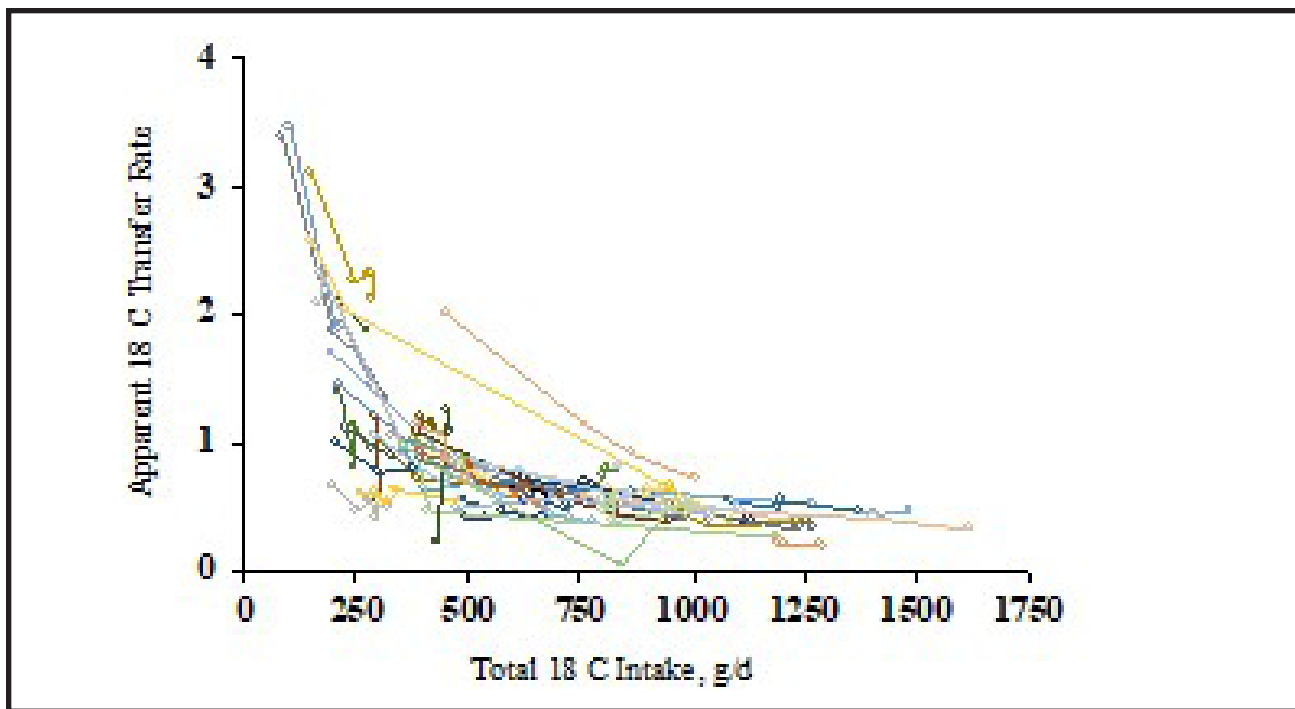**Figure 1.** Illustration of the factors impacting milk fat synthesis.

Figure 2. Illustration of sources of fatty acids found in milk fat.

Figure 3. Relationship between total 18 carbon FA intake and apparent transfer efficiency of 18 C FA to milk fat from a preliminary meta-analysis. Treatment means are connected within experiment.