

Managing Milk Fat Depression

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Introduction

Milk fat concentration is variable within and between farms and is modified by genetics, season of the year, and physiological state, but is especially responsive to diet. Synthesis of milk fat is an energy demanding process, but also represents a significant portion of the economic and nutritional value of dairy products. First described over one and a half centuries ago, diet-induced milk fat depression (**MFD**) is characterized by a decrease in milk fat yield of up to 50% with no change in milk yield or yield of other milk components. Milk fat depression 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 the past two decades have provided extensive knowledge of the causes and mechanisms of MFD we continue to experience the condition because of the requirement to feed high-energy diets and the desire to maintain optimal milk production. Additionally, numerous dietary factors commonly interact to cause MFD, making prediction difficult. We have investigated the time course of induction and recovery of MFD that provides insight into identifying causative factors and setting expectations for correction of MFD. We have also demonstrated that a rumen available methionine analog reduces the risk of MFD and that feeding management has an important role.

Historical Theories of Milk Fat Depression

The investigation of diet-induced MFD has a rich history that has included many theories to explain reduced milk fat synthesis. 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 including a decrease in the acetate to propionate ratio (Bauman and Griinari, 2001). This formed the basis for one of the most widely known substrate

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supply limitation theories that proposed that acetate supply was limiting milk fat synthesis during diet-induced MFD. However, the reduced ratio of acetate to propionate with highly fermentable diets is predominantly due to increased ruminal production of propionate (Davis and Brown, 1970). Overall, several decades of research has tested numerous theories based on substrate limitations and found little to no evidence for their support in classical diet-induced MFD which is associated with highly fermentable and high unsaturated fat diets (extensively reviewed by Bauman and Griinari, 2003; Shingfield and Griinari, 2007; Bauman et al., 2011). Acetate supply does not appear to be limiting during classical diet-induced MFD; however, acetate supply may have a small impact on milk fat synthesis. Sheperd and Combs (1998) increased milk fat yield 230 g/d with ruminal infusion of 2.2 kg/d of neutralized acetate. We have recently observed a 178 g/d increase in milk fat with infusion of 420 g/d of neutralized acetate (Urrutia et al., 2015). Importantly, these were increases in milk fat above normal levels (>3.55%). Acetate appears to provide an opportunity for small changes in milk fat outside of conditions of classical diet-induced MFD.

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 *trans*-FA originated from incomplete ruminal biohydrogenation of unsaturated FA and might contribute to the development of MFD. Subsequent studies have demonstrated a clear relationship between specific *trans*-FA and MFD (see reviews by Bauman and Griinari, 2003; Shingfield and Griinari, 2007; Bauman et al., 2011). Investigations over the past twenty 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 (Figure 1, 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 (reviewed by Bauman et al., 2011). In experiments abomasally infusing *trans*-10, *cis*-12 CLA, a curvilinear relationship ($R^2 = 0.96$) exists between the dose of *trans*-10, *cis*-12 CLA and the reduction in milk fat secretion; as little as 2.5 g/d delivered post-ruminally caused a 20-25% reduction (de Veth et al., 2004). A curvilinear relationship also exists between milk fat content of *trans*-10, *cis*-12 CLA and the reduction in milk fat [$R^2 = 0.93$; (de Veth et al., 2004)]. Milk FA profile provides a sensitive indicator of rumen outflow of FA as 85% of the preformed FA originate directly from chylomicrons absorbed in the gut (Palmquist and Conrad, 1971).

Ruminal Biohydrogenation

Ruminant diets are low in total fat, although forages, oilseeds, fat supplements, and some byproducts can result in a significant intake of PUFA. Dietary FA are metabolized in the rumen resulting in a large difference between the FA profile of the diet and the FA absorbed. Most FA in the diet are esterified and these are hydrolyzed in the rumen and the resulting unsaturated FA are isomerized (double bond position

changed) and biohydrogenated (double bond removed; Figure 1). The extent of biohydrogenation and the intermediates formed are determined by the properties of the fat source, retention time in the rumen, and characteristics of the microbial population (Allen, 2000, Palmquist et al., 2005). Dietary factors that modify ruminal fermentation (ex. high starch, high oil, monensin) also modify ruminal FA metabolism through associative effects that presumably result in a microbial population that utilizes the alternative pathway of PUFA biohydrogenation.

Ruminal biohydrogenation may be simply described as a function of the available FA pool size, ruminal retention time, and bacterial biohydrogenation capacity (Harvatine and Bauman, 2007). Microbial biohydrogenation is a multi-step process for which the kinetics are not well documented. Harvatine and Allen (2006b) used the pool and flux method (Firkins et al., 1998) to observe *in vivo* ruminal FA kinetics of a cottonseed-based diet that included a fat supplement. Dietary FA had a slow ruminal passage rate (6.4 to 7.4%/h) indicating a long average rumen retention time. In contrast, the fractional biohydrogenation rate of linoleic acid was high (14.6 to 16.7%/h). Interestingly, the biohydrogenation of *trans* C18:1 FA was also very high (33.4 to 48.4 %/h), although a decrease in the biohydrogenation rate of *trans*-C18:1 FA was associated with an increased duodenal flow of biohydrogenation intermediates and diet-induced MFD. *In vivo* ruminal FA kinetics clearly demonstrates that ruminal FA metabolism is responsive to associative dietary factors and that the long retention time provides ample time for metabolism of fat sources that are not rapidly available in the rumen.

Variation in Milk Fat between and within Herds

Milk fat is variable between farms because of differences in diet, management practices, and herd genetics among other factors. Bailey et al. (2005) conducted an economic analysis of the variation in milk production and composition using monthly milk records of all herds in the Mid-East milk market over two years and reported a wide distribution in milk fat concentration and that one-third of the herds experienced a reduction in milk fat for 1 to 3 months. Significant variation in milk fat composition exists within herds because of stage of lactation, genetics, physiological state, management, and their interactions. This variation is well demonstrated by a 905-cow example herd with low milk fat (herd average = 3.2%). The 25th and 75th percentiles of milk fat concentration were 2.6% and 3.6%, respectively. We also observed a relationship between milk fat concentration and milk yield with higher producing cows having lower milk fat concentration. Although significant variation is not explained by milk yield, there was a large slope to the regression line and milk fat concentration of cows less than 75 lbs averaged 3.81%, cows 75 to 95 lbs averaged 3.19%, and cows above 95 lbs averaged 2.90%. Decreased milk fat with increased milk yield may be due to dilution of milk fat in greater yields, but may also be due to some degree of diet-induced MFD.

Dietary Risk Factors For Milk Fat Depression

Prediction of the occurrence of 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. Below is a summary of major risk factors. This is not a complete list, but highlights the most important issues.

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 fermentability is important for energy intake, but care should be given to minimizing sub-acute ruminal 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. Low milk fat is commonly associated with sub-clinical and clinical 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). Providing multiple sources of starch and fiber with overlapping rates of digestion is the safest approach. Additionally, sugar substituted for dietary starch may reduce risk without loss of digestibility (Mullins and Bradford, 2010).

Diet Polyunsaturated Fatty Acids

Unsaturated fatty acids have a dual impact on ruminal biohydrogenation 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, which dictates the FA profile and rate of FA 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 (C18:2) 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 FA availability, for instance cottonseed and whole roasted soybeans are expected to have a much slower release of FA in rumen than distiller's 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 (Lundy et al., 2004; Harvatine and Allen, 2006b). 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 protection. The impact of calcium salts depends on the FA profile and interaction with other factors (Harvatine and Allen, 2006a; Rico and Harvatine, 2011).

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 varying levels of evidence supports their effectiveness. We observed that 2-hydroxy-4 (methylthio) butanoic acid (HMTBA) decreased the risk of diet-induced MFD in two separate experiments (Baldin et al., 2014; Baldin et al., 2015), although the mechanism is not clear. Additionally, a direct fed microbial product was shown to stabilize rumen biohydrogenation during a high diet fermentability challenge (Longuski et al., 2009) and others may have similar impacts.

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 four times higher rate after delivery of fresh feed and during the late afternoon (Niu et al., 2014). We have observed increased milk fat with feeding cows in four equal meals every six hours compared to twice per day (Rottman et al., 2014). Fresh feed delivery is a strong stimulus for feeding and feeding times may be selected to move intake into lower intake periods of the day.

Interaction of Milk Production Level and Response to Diet

In several experiments we have observed variation in individual cow response to a MFD induction diet and that high-producing cows were more susceptible to MFD risk factors. For example, Harvatine and Allen (2006a) compared saturated (highly saturated prilled free FA; Energy Booster 100) and unsaturated (calcium salts of FA; Megalac R) FA supplements to a no supplemental fat control in low and high producing blocks of cows (control 39.4 vs 47.0 kg/d, respectively). When fed the same control diet in the same barn, the low producing cows averaged 3.45% milk fat while the high producing cows averaged 3.05%. Additionally, the response to treatment differed with low producing cows having a non-significant 6% decrease in milk fat when fed the calcium salt of unsaturated FA, while the high producing cows decreased milk fat over 20%. A similar response was observed by (Rico et al., 2014) when comparing a high palmitic acid supplement (87% C16:0; Berga-Fat F100) to calcium salts of palm FA (Megalac) where low producing cows numerically increased milk fat with both treatments, but high producing cows decreased milk fat and increased *trans*-10 C18:1 in milk fat when fed the unsaturated palm FA. Collectively these studies demonstrate that there is a strong correlation between the level of milk production and diet-induced MFD. The exact

mechanism is unclear, but high producing cows also have higher intakes. Increased intake is expected to increase rumen passage rate, which may modify the microbial population and increase ruminal outflow of *trans* intermediates before complete biohydrogenation has occurred. Additionally, high producing cows may differ in feeding and ruminating behavior and increased meal size or higher amount of intake after feed delivery may result in rumen acidosis.

How to Predict the Occurrence of Milk Fat Depression

The complexity of 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 compare nutrient concentrations and model predictions to benchmarks and experience with similar diets. However, even with the best feed analysis, software, and experience the interaction of diet ingredients and risk of the diet is best determined by the cow and observed by titration and observation. Book values are expected to reasonably represent the FA profile of most feedstuffs. The FA concentration of byproducts should be closely monitored and we have also observed significant variation in the FA concentration of corn silages. If MFD is experienced on the farm it may be advisable to conduct a FA analysis of major forage grain sources.

The Time Course of Induction and Recovery

Traditionally, dietary factors that cause low milk fat have almost exclusively been studied through induction of MFD. This is useful because it tells us what dietary factors cause MFD, but it does not directly tell how to recover or accelerate recovery once you have MFD. The mammary gland is acutely sensitive to absorption of CLA with reduced milk fat synthesis observed within 12 h of abomasal infusion (Harvatine and Bauman, 2011). We have conducted time course experiment to characterize the timing of induction and recovery of diet induced MFD and repeatedly observe that milk fat yield decreases progressively and reaches a nadir in 7 to 10 d when feeding high risk diets [Figure 2; (Rico and Harvatine, 2013)]. When cows are returned to a recovery diet, milk fat progressively increases with a major portion of recovery occurring in approximately 10 d. Knowing the time course is very important in identifying what may have caused MFD and setting expectations and monitoring recovery from the condition.

Rapidly Recovering Milk Fat

When milk fat moves below the herd's goal, the logical approach is to systematically remove risk factors. The challenge is which risk factors to remove without loss of milk or energy intake. A multi-step approach may be best. First, determine the diet unsaturated FA level and availability. In the short term, minimizing unsaturated FA intake may be the best first step. Secondly, determine if diet fermentability is higher than optimal. In some cases reducing fermentability may reduce

sub-clinical acidosis and improve rumen function without loss of milk yield. If diet fermentability appears within safe limits a reduction may result in decreased milk yield, so monitor production closely after making modifications. Lastly, determine if a rumen modifier can be added to stabilize fermentation. It is important to have reasonable expectations on the time-course of recovery. Dietary changes are expected to result in observable improvements in 10 to 14 d, but complete recovery will require nearly 3 weeks and maybe longer with more modest dietary changes.

Other Important Impacts on Milk Fat

Seasonal Variation in Milk Fat

Most dairy producers and nutritionists recognize a seasonal change in milk fat that is sometimes attributed to changes in forage sources, weather, or herd days in milk. A very repeatable seasonal pattern is observed in milk fat and protein concentration in all milk markets including Florida (Figure 3). In most regions of the country, fat concentration peaks in December or January and reaches a nadir in July. The range for annual cycle is approximately 0.25 percentage units. This highly repeatable pattern is reasonably independent of year-to-year differences in forage quality and weather. This seasonal variation should be incorporated into the expected milk fat concentration when setting production goals and troubleshooting milk fat production.

Circadian Patterns

Circadian rhythms are daily patterns and the dairy cow has a daily pattern of feed intake and milk synthesis. Dairy producers commonly recognize that morning and evening milking differ in milk yield and composition. Gilbert et al. (1972) reported 1.4 lbs higher milk yield at the morning milking, but 0.32 and 0.09 percentage unit higher milk fat and protein, respectively, at the evening milking in cows milked at 12 h intervals. More recently, Quist et al. (2008) conducted a large survey of the milking-to-milking variation in milk yield and composition on 16 dairy farms. Milk yield and milk fat concentration showed a clear repeated daily pattern over the 5 d sampled in herds that milked 2X and 3X/d. We have also observed milk yield and milk composition at each milking while milking every 6 h and feeding cows 1X/d at 0800 h or in four equal feedings every 6 h (Rottman et al., 2014). We observed a daily rhythm of milk fat synthesis and feeding 4x/d decreased the amplitude of the rhythm. This demonstrates that the daily rhythm is partially dependent on the timing of intake. We are exploring nutritional opportunities based on these rhythms including the timing of feed deliver and feeding multiple diets over the day.

Conclusions

Milk fat depression results from an interaction between ruminal fermentation processes and mammary tissue metabolism. Investigation of milk fat synthesis over the past 100 years has resulted in numerous theories. To date, the biohydrogenation theory is the only proposed mechanism that has provided causative evidence and

withstood rigorous examination. The mechanism by which biohydrogenation intermediates reduce milk fat synthesis has and will continue to provide insight into the regulation of milk fat synthesis. Milk fat depression 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.

Acknowledgements

This work is partially supported by the Agriculture and Food Research Initiative Competitive Grant No. 2010-65206-20723 and 2015-67015-23358 from the USDA National Institute of Food and Agriculture [PI Harvatine], USDA Special Grant 2009-34281-20116 [PI Harvatine], Berg-Schmidt, ELANCO Animal Health, Novus International, and Penn State University.

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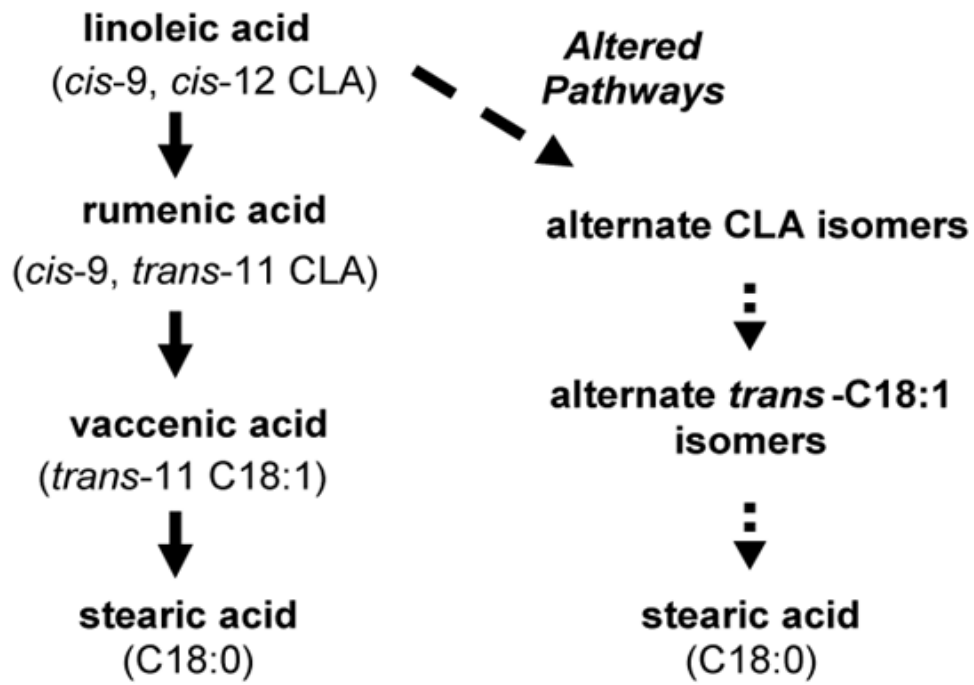


Figure 1. Biohydrogenation pathways during normal and altered ruminal fermentation. Adapted from Griinari and Bauman (1999).

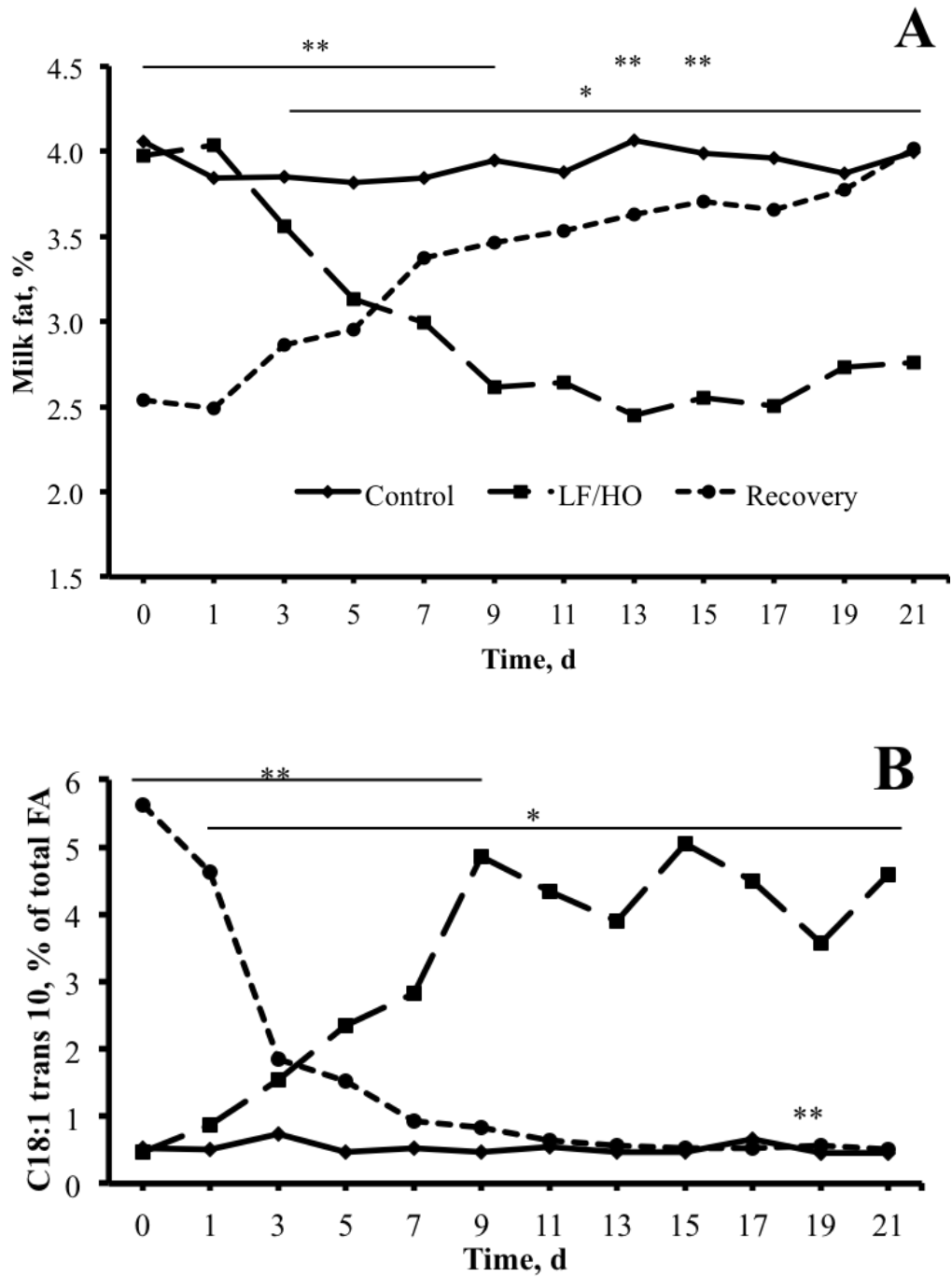


Figure 2. Temporal changes during induction of and recovery from milk fat depression. Panel A: Milk fat percent. Panel B: *trans*-10 C18:1 in milk fat.

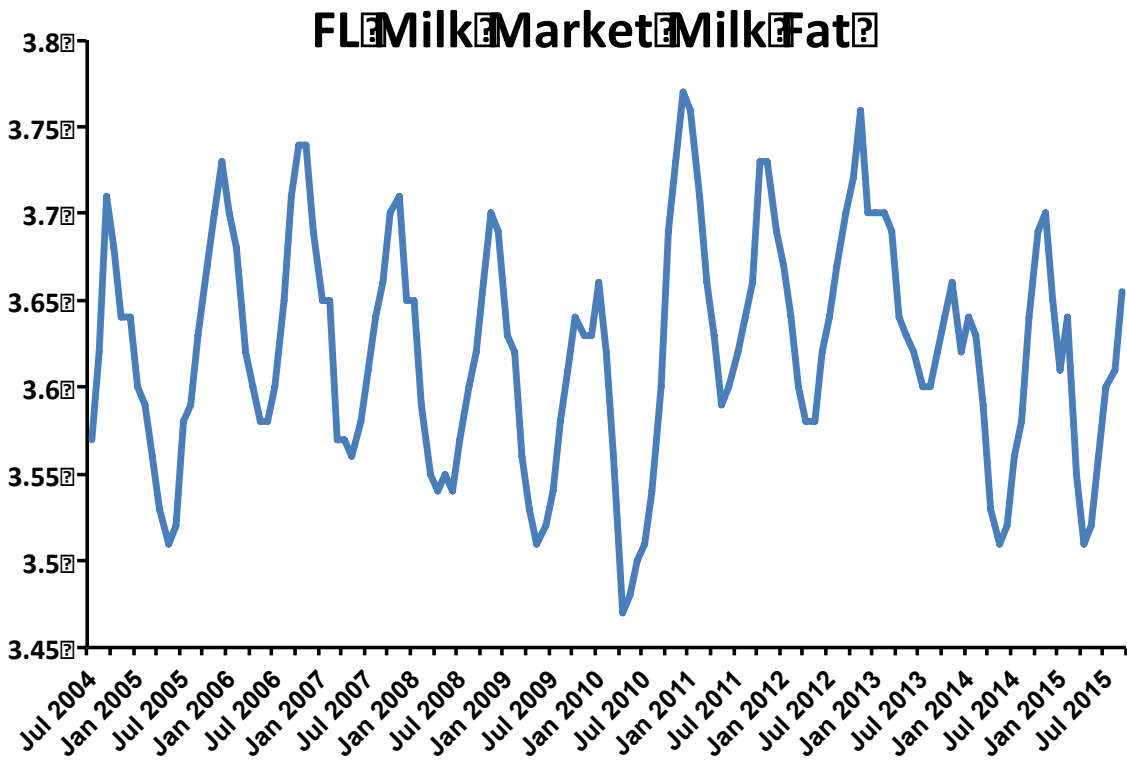


Figure 3. Seasonal pattern of milk fat in the Florida Milk Market from July 2004 to July 2015.

SESSION NOTES