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A Wide Range of Nutritional and Non-Nutritional Factors

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Description

A wide range of nutritional and non-nutritional factors influence milk fat synthesis and explain the large variation observed in dairy herds. The capacity of the animal to synthesize milk fat will largely depend on the availability of substrates for lipid synthesis, some of which originate directly from the diet, ruminal fermentation or from adipose tissue stores.

The mobilization of non-esterified fatty acids from adipose tissues is important to support the energy demands of milk synthesis and will therefore have an impact on the composition of milk lipids, especially during the early lactation period. Such mobilization is tightly controlled by insulin and catecholamines, and in turn, can be affected indirectly by factors that influence these signals, namely diet composition, lactation stage, genetics, endotoxemia, and inflammation. Environmental factors, such as heat stress, also impact adipose tissue mobilization and milk fat synthesis, mainly through endotoxemia and an immune response-related increase in concentrations of plasma insulin. Indeed, as proposed in the present review, the central role of insulin in the control of lipolysis is key to improving our understanding of how nutritional and non-nutritional factors impact milk fat synthesis. This is particularly the case during early lactation, as well as in situations where mammary lipid synthesis is more dependent on adipose-derived fatty acids.

Regulation of Milk Fat Synthesis

A better understanding of all factors involved in the regulation of milk fat synthesis may help maximize milk fat yield under commercial conditions. This review highlights the central role of insulin in controlling the flux of adipose-origin fatty acids into the mammary gland. Dietary strategies that increase insulin secretion should receive particular attention, especially in early lactation. Immune system activation by diet and heat stress can increase insulin; therefore, factors able to modulate the immune response may increase milk fat yield. Lastly, the multifactorial nature of milk fat synthesis regulation and its implications on milk fat depression theories are discussed.

Milk fat is arguably the most complex of all edible fat in human diets, providing a wide range of lipid types with varying molecular configurations, Fatty Acid (FA) chain length, and degree of saturation (Jensen, 2000). In addition, fat is the most variable component of milk, and given its important role in determining milk quality for processing, it plays a fundamental part in determining the production efficiency and profitability of farms; thus, it is possible to improve production efficiency on dairy operations via the implementation of strategies able to maximize milk fat yield. Numerous factors are known to affect milk fat synthesis in dairy animals, including diet composition, lactation stage, and genetic potential. Among these factors, nutritional factors represent an important short-term control of milk fat synthesis. However, response to these factors may depend on interactions with others, such as the lactation stage.

Milk Fat Depression

Milk fat is composed of a variety of lipids with diverse origins including diet, rumen microbiota, neutral lipid reserves from Adipose Tissue (AT), and de novo synthesis in the mammary gland. Excellent reviews have addressed the origin of milk FA and how dietary factors can impact milk fat synthesis. Diet-induced Milk Fat Depression (MFD) was first recognized almost 200 years ago and has been the subject of multiple studies spanning decades. As reviewed by Bauman and Griinari, bioactive compounds, such as rumen biohydrogenation (BH)-derived trans-10, cis-12 conjugated linoleic acid (CLA), undoubtedly play a key role in the onset of MFD. Notwithstanding, milk fat is also known to vary in response to other factors, such as the provision of dietary lipids, ruminal fermentation by-products, and FA flux from adipose tissue through insulin action. For this reason, the role of insulin in MFD, defined as the glucogenic-insulin theory, is often described as insufficient or invalid, considering that a specific decrease in the flux of AT-origin FA is not observed in many cases of diet-induced MFD. However, as it will be discussed in the present review, BH and insulin need not be competing theories, but complementary mechanisms each explaining a portion of the observed variation in milk fat under different conditions. Indeed, the notion that both trans-10, cis-12 CLA and insulin additively favor nutrient flow to adipose reserves and away from mammary fat synthesis has been previously discussed by others.

Insulin plays a central role in nutrient partitioning across tissues. Indeed, low insulin action during early lactation is an essential adaptation to support milk production demands by allowing AT lipolysis and increasing the flux of Long-Chain Fatty Acids (LCFAs) to mammary tissue. Importantly, several nutritional and non-nutritional factors can in turn affect insulin concentrations and thus affect milk fat synthesis.