



POPULAR ARTICLE

Nutritional management of transition dairy cow: strategies to optimize metabolic health

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Intricacies of the subject

Transition period in dairy cows is critical period in which the cows undergo large metabolic adaptations in energy and mineral metabolism to support lactation and avoid metabolic dysfunction. The practical goal of nutritional management during this timeframe is to support these metabolic adaptations. Increasing the amount of energy supplied through dietary carbohydrate during the prepartum period results in generally positive effects on metabolism and performance of transition cows. Attempts to increase energy supply by feeding dietary fat sources or decrease energy expenditure by supplying specific fatty acids such as trans-10, cis-12 conjugated linoleic acid to decrease milk fat output during early lactation do not decrease the release of non-esterified fatty acids (NEFA) from adipose tissue. Although the view that nutritional means have limited

ability to enhance hepatic export of NEFA as triglycerides in lipoproteins in ruminants has become dogma, recent evidence suggests that nutrients such as choline or specific fatty acids may enhance this process in transition cows. Adaptation of calcium metabolism to lactation is facilitated by nutritional strategies to decrease the cation-anion difference (DCAD) of the diet fed prepartum, although the degree to which the DCAD must be decreased to sufficiently prevent hypocalcemia remains controversial. Recent research also has provided possible physiological links between the associations of primary infectious disease with the occurrence of secondary metabolic disorders, thereby enabling investigation of factors affecting variation in response to nutritional management programs for transition cows on dairy farms.



Over the past 15 years, the transition period in dairy cows (just before and after calving) has become a key focus of research due to its strong link with metabolic disorders like ketosis, milk fever, and retained placenta. Early studies (Curtis *et al.*, 1985) showed that diet composition before calving, especially energy and protein levels, significantly affects the risk of these disorders. Surprisingly, calcium levels in the prepartum diet were not directly linked to milk fever in those findings. Despite extensive research, transition cow management remains challenging, and metabolic disorders still cause economic losses due to cow health issues and early culling. Studies have shown that about 25% of cows leave herds within the first 60 days of lactation, largely due to transition-related problems. This article aims to review the key metabolic changes (in energy, protein, and mineral metabolism) that dairy cows undergo during the transition phase. It also updates past knowledge (NRC 2001) with recent findings and explores how immune system function interacts with metabolism crucial for creating effective transition cow management strategies.

Glucose metabolism

The primary homeorhetic adaptation of glucose metabolism to lactation is the concurrent increase in hepatic gluconeogenesis (Reynolds *et al.*, 2003) and decrease in oxidation of glucose by peripheral tissues (Bennink *et al.*, 1972) to direct glucose to the mammary gland for lactose synthesis. Reynolds *et al.*, (2003) reported that net flux of glucose across the portal-drained viscera of cows was zero to slightly negative during the transition period and early lactation; the 267% increase in total splanchnic output of glucose from 9 d before expected parturition to 21 d after parturition resulted almost completely from increased hepatic gluconeogenesis. The major substrates for hepatic gluconeogenesis

in ruminants are propionate from ruminal fermentation, lactate from Cori cycling, amino acid (AA) from protein catabolism or net portal-drained visceral absorption, and glycerol released during lipolysis in adipose tissue (Seal and Reynolds, 1993). The maximal calculated contribution of propionate to net glucose release by liver ranged from approximately 50 to 60% during the transition period; that for lactate ranged from 15 to 20%; and that for glycerol ranged from 2 to 4% (Reynolds *et al.*, 2003). By difference, AA accounted for a minimum of approximately 20 to 30% during the transition period; the maximal contribution of alanine increased from 2.3% at 9 d prepartum to 5.5% at 11 d postpartum. These results are consistent with those of Overton *et al.*, (1998), who reported that hepatic capacity to convert (1-14 C) alanine to glucose was approximately doubled on 1 d postpartum compared with 21 d prepartum. Although AA are not likely to be quantitatively important in terms of the amount of milk that the AA pool will support during early lactation, these results lend support to the use of AA as an adaptational substrate pool for glucose synthesis during the immediate postpartum period.

Lipid metabolism

The primary homeorhetic adaptation of lipid metabolism to lactation is the mobilization of body fat stores to meet the overall energetic requirements of the cow during a period of negative energy balance in early lactation. Body fat is mobilized into the bloodstream in the form of NEFA. The NEFA are utilized to make upwards of 40% of milk fat during the first days of lactation (Bell, 1995). Skeletal muscle uses some NEFA for fuel, particularly as it decreases its reliance on glucose as a fuel during early lactation. Given that plasma NEFA concentrations increase in response to increased energy needs accompanied by inadequate feed intake, DMI and plasma



NEFA concentrations usually are inversely related. Available evidence suggests that the liver takes up NEFA in proportion to their supply (Pullen *et al.*, 1989; Reynolds *et al.*, 2003), but the liver typically does not have sufficient capacity to completely dispose of NEFA through export into the blood or catabolism for energy; therefore, cows are predisposed to accumulate NEFA as triglycerides within liver when large amounts of NEFA are released from adipose tissue into the circulation (Emery *et al.*, 1992). It is likely that some triglyceride accumulates in liver of almost all high-producing cows during the first few weeks postpartum. What is uncertain is the threshold at which fat begins to have detrimental effects on other hepatic processes. Piepenbrink and Overton (2003) reported that there is a negative correlation ($r = -0.4$) between triglyceride accumulation in liver and the capacity of liver slices to convert propionate to glucose *in vitro*. Cadorniga-Valino *et al.*, (1997) demonstrated that lipid infiltration of isolated hepatocytes decreased gluconeogenic capacity from propionate. A subsequent experiment using a “physiological” mixture of fatty acids determined that lipid infiltration did not affect rates of gluconeogenesis but decreased ureagenic capacity (Strang *et al.*, 1998). The implications of decreased ureagenic capacity are not clear, but limited evidence suggests that this phenomenon may occur in dairy cows during the transition period. Zhu *et al.* (2000) determined that peripheral concentrations of ammonia doubled when liver triglyceride concentrations increased during the first 2 d postpartum. *In vitro* incubation with ammonium chloride strongly inhibited capacity of isolated hepatocytes to synthesize glucose from propionate (Overton *et al.*, 1999). Therefore, it is conceivable that inhibition of gluconeogenesis may occur *in vivo* when triglycerides accumulate in liver. Perhaps the mechanism is modulated by

ammonia supply to liver. Potential implications of this research for the management of transition dairy cows centers around carbohydrate and protein nutrition. As discussed previously, significant quantities of AA are needed for gluconeogenesis. However, we hypothesize that excess protein or asynchronous supply of ruminal N relative to carbohydrate supply may increase the ammonia load on the animal, and thereby affect the capacity of a triglyceride-laden liver to synthesize glucose. Furthermore, the specific nature of the potential link between impaired gluconeogenic capacity and ureagenic capacity has yet to be elucidated.

Calcium, phosphorus and magnesium metabolism

Calcium (Ca) and phosphorus (P) homeostasis in dairy cows is tightly regulated, especially during the transition period. The skeleton stores 99% of Ca and 80% of P. Hormonal control via parathyroid hormone (PTH) and vitamin D regulates Ca absorption, bone resorption, and renal reabsorption. Calcitonin, secreted in response to high Ca levels, lowers blood calcium. Studies (Goff *et al.*, 2002) show that the mammary gland is responsible for periparturient hypocalcemia. However, hypophosphatemia is influenced by other factors. Despite high hormone levels during hypocalcemia, issues may arise from receptor-level dysfunction or signaling problems. Nutritional strategies aim to stimulate Ca metabolism mechanisms before calving to prepare cows for lactation demands.

Strategies to minimize the transition period metabolic disorders

Nutritional grouping strategy

A two-group feeding scheme during the dry period is effective. Lower-energy diets early on (1.25 Mcal/kg NEI) help prevent excessive body condition gain, while higher-energy



diets (1.54–1.62 Mcal/kg NEL) during the last 3 weeks support lactation needs. Overfeeding energy too early can impair metabolic function post-calving. Ideal BCS at dry-off is around 3.0; cows with lower BCS (2–2.5) tend to have better feed intake and milk yield.

Glucose and NEFA management

Carbohydrates in Prepartum Diet: Feeding higher non-fiber carbohydrate (NFC) diets prepartum helps adapt rumen microbes and increases propionate, a key glucose precursor. Studies show higher NFC diets improve dry matter intake (DMI) and reduce postpartum metabolic issues. Monensin Supplementation: Monensin improves energy metabolism by boosting ruminal propionate production, enhancing glucose levels, and reducing ketosis. Its impact on NEFA levels varies, but it consistently improves milk yield in overconditioned cows. Administering it via controlled-release capsules (CRC) is more reliable than mixing it into feed due to intake variability.

Added fat in transition diets

Dietary fat may help decrease NEFA concentrations and prevent ketosis in cows. Long-chain fatty acids are absorbed into the lymphatic system and provide energy for peripheral tissues and the mammary gland. However, research indicates that added fat during the prepartum period does not decrease plasma NEFA concentrations. Cows fed fat during the dry period had decreased DMI and reduced liver triglycerides. High-energy diets during the prepartum period had decreased periparturient NEFA concentrations and postpartum liver triglyceride concentrations. Some dairy practitioners have reported beneficial effects of administering dietary fat by oral drench to cows during the postpartum period. However, fat sources are commonly

included in commercially available mixtures administered orally to fresh cows.

Effects of specific fatty acids on NEFA supply

Nutritional strategies can be used to decrease the supply of circulating nitric oxide (NEFA) in dairy cows and the rate at which NEFA are converted to triglycerides within the liver. Choline, a quasi-vitamin, plays a significant role in mammalian metabolism and is required for the synthesis and release of triglycerides in the liver. Choline deficiency in rats resulted in a sixfold increase in liver triglyceride content. Feeding rumen-protected choline to transition dairy cows decreased the rate of accumulation of esterified products in liver slices, suggesting that VLDL export was sensitive to choline supply. Yields of milk and fat-corrected milk have generally increased in response to feeding rumen-protected choline during the transition period.

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Milk fever preventive management

Controversy exists regarding whether sufficient alleviation of hypocalcemia can occur by decreasing the cation content of the diet fed during the prepartum period alone without adding anions through mineral- or acid-based sources. Existing research is equivocal about whether a reduction in dietary K and a moderate DCAD are sufficient to avert milk fever or if herds predisposed to milk fever might benefit from diets with a DCAD of -10 to -15 meq/100 g of DM. Research on calcium metabolism in dairy cattle has sparked interest in the inclusion of sodium aluminum silicate Zeolite A in their diets. Zeolite A potentially binds calcium in the digestive tract, making it unavailable for intestinal absorption and restricting calcium entry rate. Initial results suggest that dietary supplementation with Zeolite A prepartum prevents milk fever and subclinical hypocalcemia in Jersey cows. However, more research is needed to determine the actual *in vivo* Ca binding capacity and potential negative effects on micronutrient bioavailability. Fatty liver and metabolic perturbations negatively impact immune function in dairy cattle. The immune system may also impact normal adaptations during the transition period, potentially leading to secondary metabolic disorders. Attenuation of immune sensitivity during the transition

period may be a normal and protective homeorhetic adaptation to lactation, but it also allows for more severe infections and greater inflammatory reactions. These concepts must be evaluated in periparturient cows and the biological magnitude of the potential interface of nutrient metabolism and immune function determined.

Conclusions

Advances in understanding dairy cow metabolic adaptations during the transition from nonlactating to lactating stage have led to precise nutritional strategies. Evidence supports two-group dry cow feeding to avoid overfeeding early and enhance energy supply prepartum, with total carbohydrate intake being more critical than its source. Efforts to improve energy status and reduce NEFA mobilization with added fat or trans-10, cis-12 CLA show inconsistent results, whereas choline and essential fatty acids may enhance hepatic NEFA export as VLDL triglycerides. Lowering prepartum DCAD aids calcium mobilization, though optimal levels remain debated. Variation in farm responses highlights the multifactorial nature of transition cow management, where nutrition, metabolism, and immune function interact. Future research should adopt integrative biological approaches to optimize health and productivity during this period.