

Optimizing Transition Cow Diets

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Abstract

Diet formulation recommendations for transition cows vary widely due to conflicting experimental results, wide variation in feeding programs among well-managed herds, and lack of understanding of the effects of digestion characteristics of feeds on animal response. Physiological changes over the transition period greatly affect feed intake response to diet. Application of a new theory of feed intake control can be used to understand the effects of diet on feed intake of transition cows. The basic premise of the theory is that feeding behavior is controlled by the oxidation of fuels in the liver; increased oxidation results in a satiety signal decreasing meal size, and decreased oxidation results in a hunger signal causing an eating response. Fat mobilization in the transition period increases oxidation in the liver, likely suppressing feed intake. Considerations to control mobilization of fat reserves and increase feed intake of cows after calving are discussed. Also discussed is the importance of maintaining gut fill during this period to maintain buffering, prevent displaced abomasum, and to extend the supply of absorbed fuels when intake decreases at calving. Understanding the function of feeds beyond their energy and nutrient concentrations will help to optimize diets for transition cows.

Introduction

High incidence of metabolic disorders and infectious disease continues to impact animal well-being and dairy farm profitability. This is despite intense interest and considerable research over the last 20 years that has contributed greatly to our understanding of physiological changes during the transition from pregnancy to lactation. The suppression of appetite and the immune system during the transition period increases the risk for fatty liver, displaced abomasum, retained placenta, metabolic disorders such as ketosis and milk fever, and infectious disease such as mastitis and metritis. Sustained depression in feed intake decreases fertility and increases the risk of culling. The overall goal of transition cow management is to ease the transition from pregnancy to lactation by optimizing health and improving long-term milk yield, reproductive success, and farm profitability. The specific goal is to control fat mobilization throughout the transition period to decrease the depression of feed intake and immune-suppression from elevated non-esterified fatty acids (NEFA). The objective of this paper is to discuss the application of a new theory of feed intake control to formulate diets for transition cows. Consideration of the dramatic physiological changes occurring through the transition period and the physiological response to diets will help us to optimize diets to control fat mobilization and increase feed intake of fresh cows, improving animal health, and increase profitability of dairy farms.

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Hepatic Oxidation Theory (HOT)

There is a large body of evidence (mostly in non-ruminant species) indicating that food intake is controlled by oxidation of fuels in the liver. This has been reviewed previously (Allen et al., 2005; Allen and Bradford, 2006) and will be only briefly discussed here. The liver is “hardwired” to feeding centers in the brain via the hepatic vagus nerve. Feeding behavior is controlled by the firing rate of the nerve, which is determined by oxidation of fuels in the liver; increased firing rate is associated with hunger and decreased firing rate is associated with satiety. Feeding behavior has been linked to adenosine triphosphate (ATP) concentration in the liver with satiety occurring as fuels are oxidized and ATP is produced, and hunger occurring as oxidation decreases and the pool of ATP is depleted. The mechanism by which ATP concentration affects the firing rate of the hepatic vagus nerve has not yet been determined. Fuels oxidized in the liver vary across species, but for ruminants, they include fatty acids (from the diet or mobilized from body reserves), propionate (produced by microbial fermentation in the gut), lactate (produced by muscle and gut tissues from glucose), and amino acids (from protein degradation). It is important to realize that the pattern of oxidation of fuels (minute to minute) is what affects feeding behavior because the amount of oxidation over longer periods of time (hours or days) is relatively constant and determined by the energy requirements of the liver.

Physiological Changes Through Transition

Because fatty acids are readily oxidized in the liver, the supply of NEFA from mobilization of body fat reserves likely suppresses feed intake in the transition period. The degree of fat mobilization is affected by changes in plasma insulin concentration and sensitivity of tissues to insulin. Plasma insulin concentration signals tissues to synthesize fat if elevated or to mobilize fat if lowered. Changes in sensitivity of tissues to insulin through

the lactation cycle modify this signal; decreased sensitivity (increased resistance) results in greater fat mobilization and increased sensitivity results in greater fat deposition at the same insulin concentration. Plasma insulin concentration decreases 50% or more by calving, beginning several weeks prepartum. Plasma NEFA concentration increases because fat is mobilized in response to decreased plasma insulin concentration. In addition, tissue sensitivity to insulin decreases in late pregnancy, contributing to increased fat mobilization. Decreased plasma insulin concentration and sensitivity help the cow maintain constant plasma glucose concentration, despite declining feed intake in the last week or so before calving. This is because utilization of glucose by tissues decreases, and utilization of NEFA by muscle increases, sparing glucose.

Plasma glucose concentration drops precipitously at calving and partially recovers over the course of the next several weeks. Plasma insulin concentration and sensitivity of tissues to insulin remain low in early lactation, so plasma NEFA concentration remains elevated for several weeks or more. The length of time that NEFA remains elevated varies greatly among cows and depends upon the rate of mobilization and removal from the blood by the liver and mammary gland. Transfer of NEFA to milk fat by the mammary gland is highly desirable because storage of NEFA as triglycerides in the liver results in fatty liver, compromising glucose production, and oxidation of NEFA in the liver likely decreases feed intake according to HOT. This, in turn, delays the increase in plasma glucose concentration following calving, extending intake suppression. This is because glucose stimulates insulin secretion by the pancreas, and plasma insulin concentration remains low, extending the period of fat mobilization, and therefore extending the period that feed intake is suppressed by oxidation in the liver. In addition, low plasma glucose likely limits milk yield because glucose is required by the mammary gland for the production of milk lactose, the primary determinant of milk volume.

Hepatic oxidation of NEFA is a two-stage process; long carbon chains of fatty acids are partially oxidized to acetyl CoA, a two-carbon molecule, which is either completely oxidized or exported as ketones. The ability of the liver to completely oxidize NEFA is limiting, so ketones are exported and their concentration in plasma is elevated when fat mobilization is high. Ketones can be beneficial because they can be used by some tissues for energy, sparing glucose, but can cause keto-acidosis if concentrations are very high.

Optimizing Fat Mobilization

Plasma NEFA are used as an energy source by maternal and fetal tissues, thereby sparing glucose and also enrich the fat content of milk. However, plasma NEFA concentrations should be limited because elevated NEFA can depress feed intake and suppress the immune system. To limit plasma NEFA concentrations, rate of fat mobilization must be controlled. Rate of fat mobilization is dependent upon the amount of fat reserves available for mobilization, as well as insulin concentration, tissue sensitivity to insulin, and stress. The importance of controlling body condition at calving is well recognized. Cows with excessive body condition generally mobilize fat very rapidly through transition because their tissues are more insulin resistant and they have greater fat stores to mobilize. Therefore, it is very important to manage body condition to limit over-conditioned cows by reproductive management, grouping lactating cows, diet formulation, use of recombinant bovine somatotropin (**rbST**) to partition energy to milk, etc. Recent research indicates that allowing cows to consume more energy than required during the dry period results in increased NEFA concentrations in early lactation. Controlling energy intake by feeding high-fill diets during this relatively short period might reduce depots of readily mobilized fat, reducing the rate of fat mobilization after calving. Fat mobilization will be reduced by increasing sensitivity of fat tissues to insulin (decreasing insulin

resistance). Niacin decreases fat mobilization but likely needs to be supplemented at higher concentrations than currently recommended unless provided in a protected form. Chromium increases insulin sensitivity, and supplemental chromium has been demonstrated to decrease plasma NEFA concentrations in lactating cows. While chromium supplements are restricted for use in lactating dairy cattle diets by the Food and Drug Administration (**FDA**), the chromium concentration of feeds varies. A more rapid increase in plasma glucose following calving will likely increase insulin and decrease NEFA concentrations sooner. However, increasing insulin sensitivity of fat tissue is preferable to increasing insulin concentration because insulin can reduce glucose production by the liver. Hormones released during stress increase fat mobilization, further elevating plasma NEFA concentration further. Therefore, great attention should be paid to reduce all potential stressors of cows, including stressful interactions with farm workers, management procedures, and facilities (e.g., bedding, ventilation, and bunk space).

Propionate Control of Feed Intake

Propionate, produced by microbial fermentation in the gut, is a primary fuel controlling feed intake in ruminants fed diets containing high grain concentrations. It is a primary endproduct of starch fermentation and production rates vary greatly among diets because of great differences in starch concentration and fermentability. Propionate can be produced and absorbed at very high rates and is very rapidly taken up by the liver, where it is a major fuel used to produce glucose. However, when propionate is absorbed faster than it can be utilized to produce glucose in the liver, it will likely be oxidized, generating ATP and a satiety signal to the brain. The capacity of the liver to produce glucose is affected by glucose demand (the difference between glucose required and glucose produced) because limiting enzymes in the liver are up-regulated to meet demand. Because of this,

propionate is less likely to be oxidized (and decrease feed intake) at peak lactation, when glucose demand is high, than in late lactation when glucose demand is lower. Although propionate might be expected to have little effect on feed intake of fresh cows because they have high glucose demand, decreasing oxidation of propionate *per se*, propionate also stimulates oxidation of acetyl CoA. Fresh cows have a large supply of acetyl CoA in the liver from partial oxidation of NEFA, which is exported as ketones, but its oxidation is stimulated by propionate when it is taken up by the liver, quickly generating ATP and a satiety signal (see Allen and Bradford, 2006 for more details). This is an apparent conundrum: propionate is a primary fuel used to produce glucose, which is needed to increase insulin and decrease NEFA, thereby alleviating the depression in feed intake by NEFA oxidation in fresh cows, but propionate suppresses feed intake by stimulating oxidation of acetyl CoA in fresh cows. However, there are options for diet formulation, including the manipulation of rate of propionate production, to extend meal length, supplying other glucose precursors that stimulate oxidation of acetyl CoA to a lesser extent, and providing alternate energy sources for tissues to spare glucose. The goal is to maximize the amount of glucose produced or spared per unit of ATP generated in the liver over time. Manipulating the pattern of oxidation of fuels in the liver can increase plasma glucose and insulin concentrations, decreasing fat mobilization and the period of time feed intake is suppressed by oxidation of NEFA in the liver.

Propionate Production and Absorption

The rate at which propionate is produced and absorbed is easily manipulated. Propionate production increases as starch concentration and starch fermentability of diets increase and ruminal pH decreases, altering microbial populations. Starch fermentability varies with type of grain (wheat > barley > corn > sorghum), endosperm type (floury or vitreous), processing (grinding, rolling, or heat

treatment), ensiling (moisture concentration or length of time), and increases as more grain is fed. The rate of propionate absorption increases with increased blood flow and rumen motility. Rumen motility is affected by diet and is likely reduced by salts, fatty acids, and butyrate. Butyrate is produced at higher concentrations when sugars are fed. Although decreased motility might increase meal size by slowing rate of propionate absorption, fat can depress feed intake, and butyrate also spares propionate oxidation by the ruminal epithelium, counteracting this effect.

Glucose Production

Glucose production is dependent upon availability of precursors, which is affected by diet composition and feed intake. In addition, the capacity of the liver for glucose production changes with its size, degree of fat storage, and hormonal regulation. Liver size increases greatly after calving, increasing the capacity for glucose production in response to increased glucose demand. However, excessive mobilization of fat reserves in the transition period increases storage of fat as triglycerides in the liver, inhibiting glucose production.

According to HOT, slowing the rate of oxidation in the liver during meals will delay satiety, increasing meal size and feed intake of fresh cows. Choosing less fermentable starch sources, such as dry ground corn compared to high moisture corn, will decrease rate of production of propionate and slow the rate of oxidation of acetyl CoA within meals. Decreasing the rate of starch digestion in the rumen will also likely shift the site of starch digestion post-ruminally. Although efficiency of starch utilization is likely greater for more fermentable starch sources, fewer glucose precursors are produced. When starch is fermented in the rumen, other products besides propionate are produced (e.g., acetate, butyrate, and compounds in microbial cells) that cannot be utilized for glucose production. In addition, if rate of propionate production is high,

feed intake might be depressed. When starch is digested to glucose in the small intestine, most of the glucose is oxidized to lactate (an important precursor for glucose production in the liver) by intestinal microbes and tissues. Starch digested in the small intestine is much less likely to stimulate oxidation of acetyl CoA in the liver during meals than propionate because of the time delay for passage through the rumen and because fractional uptake of lactate by the liver is much lower than for propionate. The fraction of glucose precursors provided by starch fermentation in the large intestine is much lower than in the small intestine and even less than in the rumen because precursors within microbial cells are lost in the feces. Therefore, careful consideration of site of starch digestion is very important to maximize the yield of glucose precursors over time. Starch sources with lower ruminal digestibility should be highly digestible in the small intestine to provide the greatest yield of glucose precursors. For instance, dry ground and cracked corn both slow the rate of propionate production in the rumen compared to high moisture corn, but the ground corn will provide more glucose precursors because of greater digestibility in the small intestine and total tract.

Amino acids mobilized from tissue are important contributors to glucose production following calving. While some absorbed amino acids are utilized as glucose precursors, feeding excess protein for this purpose is expensive, increases nitrogen excretion, and might depress feed intake by stimulating oxidation in the liver. Glycerol is a glucose precursor that has a more direct pathway to glucose and is expected to stimulate oxidation of acetyl CoA less than propionate. Increased availability of glycerol from biofuel production will increase its competitiveness as a feed ingredient. Fructose is a simple sugar that is a component of sucrose and is in many feedstuffs at low concentrations. Unlike glucose, fructose does not directly promote secretion of insulin. It can be converted to glucose more directly than propionate

without stimulating oxidation of acetyl CoA in the liver and stimulates glycogen production, thereby providing a more even supply of plasma glucose throughout the day. While many feeds contain fructose in low concentrations, it comprises up to 30% of DM of temperate grasses stored as fructosans. However, fructose and fructosans are partially metabolized by microbes during ensiling and ruminal fermentation. Sorbitol is converted to fructose in the liver and is likely metabolized more slowly by ruminal microbes, possibly increasing its effectiveness compared to fructose. The extent to which fructose supply to the liver can be manipulated by diet formulation is unknown and requires further research.

Glucose production by the liver is controlled by hormones; it is stimulated by glucagon and that stimulation is blocked by insulin. Therefore, a low ratio of insulin to glucagon is desirable to stimulate the production of glucose. Although plasma glucagon concentration is high in fresh cows, administration of glucagon has been effective at increasing plasma glucose and insulin and decreasing plasma NEFA concentration. Feeds vary in their ability to stimulate glucagon, and future research might provide information on how to formulate diets to increase glucagon further. While stress hormones stimulate glucose production in the liver, stress should be minimized because they also mobilize fat, increasing NEFA.

Glucose Sparing

Although glucose production must increase in early lactation, glucose can also be spared by providing alternative energy sources for tissues; these fuels should not be overlooked when formulating diets for transition cows. Acetate can be used by tissues to spare glucose and adequate acetate production should be insured by preventing low ruminal pH, including digestible fiber sources in the diet and providing adequate effective fiber to increase ruminal retention of small fibrous particles,

increasing their digestibility. As previously mentioned, glucose is spared by utilization of plasma NEFA by muscle, but inclusion of fat in transition cow diets is not generally recommended because plasma NEFA concentration is already high and some fat sources can depress feed intake. However, individual fatty acids have physiological effects that might be beneficial. For instance, specific fatty acids depress fat synthesis in the mammary gland, sparing glucose.

Ruminal Digesta

While ruminal distention becomes a primary limitation to feed intake as milk yield increases, it likely has little effect on feed intake during the transition period if feed intake is controlled primarily by oxidation in the liver. Diets can be formulated to meet requirements for energy and nutrients with large differences in the amount and turnover rate of ruminal digesta. Formulating diets to maintain gut fill with ingredients that are retained in the rumen longer, have moderate rates of fermentation, and high ruminal digestibility will likely benefit transition cows several ways. The ruminal digesta will provide more energy over time when feed intake decreases at calving or from metabolic disorders or infectious disease. This will help maintain plasma glucose and prevent even more rapid mobilization of body reserves compared to when diets are formulated with ingredients that disappear from the rumen quickly. Ruminal digesta is very important to buffer fermentation acids, and buffering capacity is directly related to the amount of digesta in the rumen. Therefore, diets formulated with ingredients that increase the amount of digesta in the rumen will have greater buffering capacity and will maintain the buffering capacity longer if feed intake decreases. Inadequate buffering can result in low ruminal pH, decreasing fiber digestibility and acetate production, and increasing propionate production, possibly stimulating oxidation in the liver and thereby decreasing feed intake. Low ruminal pH also increases risk of health problems, such as ruminal

ulcers, liver abscess, and laminitis, and causes stress, likely increasing mobilization of body reserves even further. Diets formulated with ingredients that maintain digesta in the rumen longer when feed intake decreases will likely decrease the risk of abomasal displacement.

Considerations for Diet Formulation

Conflicting research results and failure of surveys of successful dairy herds to reveal common feeding strategies for transition cows indicate that interactions among many different factors affect animal health and profitability. Because of this, the same diets are not likely to be optimal for all farms. Diets likely interact with many management factors, including body condition, feed bunk space, cow comfort (stress), length of dry period, and grouping strategies for dry cows and lactating cows. Body condition should be controlled during mid to late lactation and stress should be minimized to limit fat mobilization. Diets should be formulated to decrease plasma NEFA concentration and maintain rumen fill throughout the transition period. Suggestions to accomplish this goal are mentioned below.

Diets with high concentrations of grain, non-forage fiber, and finely chopped forages fed through the transition period should be avoided because of increased risk of displaced abomasum and acidosis. Feeding high-fill diets prior to calving to control feed intake might reduce depots of readily mobilized fat and provide energy to help sustain plasma glucose through calving. Increased amounts of ruminal digesta also decrease the risk of displaced abomasum and increase buffering capacity, decreasing risk of acidosis. Forage fiber is much more filling than non-forage fiber or other diet components, but the filling effect of forage fiber varies greatly. Some long fiber particles are necessary to form a mat and increase digesta retention in the rumen, but excessive length of cut can increase sorting and can decrease feed intake. Digestion characteristics of forage fiber vary greatly

by forage type and maturity and have a large effect on retention time in the rumen. Wheat straw digests and likely passes from the rumen slowly, and it has been used to dilute energy density of corn silage in TMR for dry cows. Grass silage or hay is likely more beneficial because the fiber is more digestible and it provides energy for a longer time when feed intake decreases at calving. Some fructose stored in grasses as fructosans might survive ensiling and ruminal digestion and decrease oxidation in the liver, helping to increase feed intake following calving and increase glycogen stores to reduce the decrease in plasma glucose following calving. However, grass with high potassium concentrations might require anionic salts in prepartum diets to reduce milk fever following calving.

Specific fatty acids reaching the small intestine stimulate release of gut peptides that affect insulin and glucagon concentrations as well as gastrointestinal motility. However, until future research suggests a clear benefit, supplemental fat should not be fed through the transition period because it can depress feed intake by stimulating gut peptides and increase the supply of fatty acids to be oxidized. An exception might be supplemental conjugated linoleic acid (CLA) to suppress fat production in the mammary gland to benefit fresh cows by sparing glucose.

Optimal starch fermentability for cows immediately prior to calving is unclear and requires more research. However, limited research suggests that increased starch fermentability prior to calving decreases fat mobilization, tending to increase feed intake after calving. Highly fermentable starch sources should not be included in diets for fresh cows because rapid production and absorption of propionate will stimulate oxidation of acetyl CoA and suppress feed intake. Starch sources with moderate ruminal fermentability and high digestibility in the small intestine, such as dry ground corn, will provide glucose precursors and less propionate to stimulate oxidation and suppress feed intake. Milk

yield increases rapidly following calving, and over the next several weeks, plasma glucose increases, insulin increases, and fat mobilization decreases, decreasing plasma NEFA. Because less NEFA is available for oxidation, the acetyl CoA concentration in the liver decreases, decreasing ketone output by the liver. Lack of acetyl CoA and high glucose demand limits oxidation in the liver, and satiety signals to the brain decrease.

As lactation proceeds towards its peak, feed intake is controlled more by effects of gut fill on distension and less by oxidation of fuels in the liver. Cows should be switched from the more filling, less fermentable fresh cow diet to a diet that is less filling and more fermentable as gut fill begins to dominate the control of feed intake. This might be only 7 to 10 days after calving for some cows in the herd or more than 3 weeks for others and is likely indicated by lower plasma NEFA and ketone concentrations and steadily increasing feed intake. While group housing prevents measurement of feed intake for individual cows, kits are available to measure NEFA and ketones concentrations on the farm.

Conclusions

Consideration of physiological changes occurring through the transition period and the physical and digestion characteristics of feeds beyond their nutrient composition is required to optimize diets for transition cows. Understanding the control of feed intake is critical to diet formulation, and the Hepatic Oxidation Theory is exciting for its potential contribution to our ability to formulate diets. While more research is needed to better understand animal response to diets, the theory and concepts presented in this paper will help to formulate diets to improve animal health and farm profitability.

References

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