

Strategies to Optimize Feed Intake in Lactating Cows

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■ Take Home Messages

- Feed intake is affected by the interaction of diet characteristics, physiological state of animals, and environmental stressors.
- The dominant signals controlling feed intake likely change throughout lactation. Control of feed intake is likely dominated by hepatic oxidation of non-esterified fatty acids during transition and propionate in late lactation, while ruminal distension likely controls feed intake of peak lactation cows. Thus, optimizing feed intake requires different diets through lactation (i.e. grouping cows).
- Controlling mobilization of body fat stores during transition and limiting diet fermentability are keys to maximize feed intake during transition.
- Peak milk yield is maximized by feeding low-fill diets that are highly fermentable. The filling effect of diets is affected most by concentration, digestibility, and fragility of forage NDF.
- Diets should be formulated to limit diet fermentability to provide consistent supply of fuels as milk production declines post-peak and plasma insulin concentration and insulin sensitivity of tissues increase.

■ Introduction

Feed intake is determined by many interacting factors and prediction of feed intake is the “Achilles heel” of diet formulation. Many different diet characteristics interact with environment and physiological state of cows, making it difficult to predict feed intake accurately. However, understanding the factors controlling feeding allows us to manipulate diets to optimize feed intake. Eating is controlled by the integration of peripheral signals in brain feeding centers. Dairy cow diets must contain a minimal concentration of relatively low-energy roughages for proper rumen function and signals from

ruminal distension can control feed intake when the drive to eat is high and metabolic control of feed intake is diminished (e.g. cows at peak lactation). Signals derived from metabolism of fuels dominate the control of feed intake when signals from distension diminish (e.g. cows in late lactation). Therefore, effects of diet on feed intake vary with the physiological state of the animal. Furthermore, they interact with environmental stressors such as social (e.g. overcrowding) and thermal stress. The objective of this article is to discuss factors controlling feed intake in lactating cows and how they can be manipulated to optimize feed intake. "Optimal" feed intake might mean the maximum to attain higher milk yields for high-producing cows or less than maximum to increase efficiency of feed conversion for lower producing cows.

■ **Hepatic Oxidation Theory (HOT)**

There is a large body of evidence (mostly in non-ruminant species) 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 ATP (a form of energy currency within cells) concentration in the liver with satiety occurring as fuels are oxidized and ATP is produced, and hunger occurring as oxidation decreases and 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.

■ **Physiological Changes Through Lactation**

Because fatty acids are readily oxidized in the liver, the supply of non-esterified fatty acids (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 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 limited, 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.

Further increases in lipolysis following parturition, combined with higher starch diets, likely suppress feed intake because rapid production and absorption of propionate stimulates oxidation of acetyl CoA (see below). Because feed intake of fresh cows is likely controlled primarily by hepatic oxidation, diets with moderately high forage fiber concentrations might benefit cows. Forage fiber increases rumen fill, decreasing the risk of abomasal displacement, and increases acetate production, sparing glucose utilization by extrahepatic

tissues. While research is needed to evaluate effects of concentration and fermentability of starch on feed intake response, starch sources with moderate ruminal fermentability and high digestibility in the small intestine such as dry ground corn will likely provide more glucose precursors by increasing feed intake.

Milk yield increases rapidly following parturition and, over the next several weeks, increasing plasma glucose stimulates insulin secretion, thereby decreasing lipolysis and plasma NEFA concentration. Because fewer NEFA are 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 limit ATP accumulation in the liver, and satiety signals to the brain decrease. As milk yield increases further and feed intake control by hepatic oxidation diminishes, control is dominated by distension from gut fill and cows should be offered a diet that is less filling and more fermentable. This change in the dominant mechanism of intake regulation might occur only 7 to 10 days after calving for some cows in the herd or more than 3 weeks for others; the best signs that hepatic oxidation is less limiting are lower plasma NEFA and ketone concentrations and steadily increasing feed intake.

As energy requirements decrease following peak milk yield, control of feed intake by gut distension gradually diminishes and control by hepatic oxidation increases. Plasma insulin concentration and sensitivity of tissues to insulin increase as lactation progresses and affect the feed intake response to highly fermentable diets. Higher plasma insulin concentrations that are indicative of adequate nutritional status likely provide negative feedback on hepatic gluconeogenesis. This relationship is consistent with HOT because decreased use of propionate for glucose production leads to greater propionate oxidation and decreased feed intake. Individual cows with an adequate supply of glucogenic precursors may respond to a further increase in supply by decreasing DMI. Greater sensitivity of tissues to insulin likely increases clearance of fuels from the blood sooner, partitioning more energy to body reserves and decreasing the interval between meals.

■ 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 immune function. 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, 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 (Holtenius et al., 2003). 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 CFIA, 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, elevating plasma NEFA concentration further. Therefore, great attention should be paid to reducing all potential stressors of cows including stressful interactions with farm workers, management procedures, and facilities (e.g. bedding, ventilation, 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. Propionate can be produced and absorbed at very high rates and 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. Some acetyl CoA is exported as ketones, but it is also readily oxidized when propionate 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 diet formulation options that help prevent the depression in feed intake, including manipulating the 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.

■ Altering Propionate Flux To The Liver

Rate of propionate production can be decreased by reducing starch concentration and fermentability and by increasing efficiency of microbial protein production from organic matter, while absorption rate is likely to be reduced by inhibiting ruminal motility.

Dietary Starch Concentration

Starch concentration of diets is often reduced by substituting forage or non-forage fiber sources (NFFS) such as beet pulp or soyhulls for cereal grains. Dilution of starch in the diet has the added benefit of reducing the fermentation rate of the starch remaining when starch concentration is decreased by adding forage or NFFS, reducing the rate of propionate production. The optimal strategy depends upon the relative cost of ingredients, efficiency of feed utilization, and animal production response. For instance, longer fiber particles from forage compared to NFFS might increase fiber digestibility by increasing ruminal pH through stimulation of rumination and by increasing ruminal retention of fiber; however, forage fiber is very filling and forages might limit feed intake compared to NFFS. Therefore, when ruminal distension contributes to the control of feed intake, substitution of NFFS for grain might be a better choice than substitution of forage.

Site of Starch Digestion

Substitution of a less fermentable starch source is an option when feed intake is depressed by a rapidly fermented starch source. Altering dietary starch

fermentability will likely be more desirable than replacing starch with fiber for ruminants with high glucose demand, such as early lactation cows, because postruminal starch digestion yields more glucose precursors than ruminal fermentation of fiber. It is important to note that the fraction of glucose precursors provided by starch fermentation in the large intestine is much lower than in the rumen or small intestine because microbial cells containing starch 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.

Rate of Propionate Absorption

Ruminal motility affects the rate of propionate absorption because mixing of ruminal contents is required to replenish its supply at the ruminal epithelium where it is absorbed. Therefore, ruminal motility likely affects the rate at which propionate stimulates oxidation within meals. Ruminal motility is affected by diet and is likely increased by physically effective fiber and decreased by long-chain fatty acids and butyrate. Butyrate production increases when feed ingredients containing sugars are consumed. Therefore, other diet components can alter feed intake by affecting flux of propionate to the liver.

Nitrogen Metabolism

Consumption of ruminally degraded protein or total protein in excess of that required can decrease feed intake. Hepatic oxidation of ketogenic amino acids can contribute to satiety according to HOT and urea production from excess ammonia produces a carbon skeleton that can be oxidized. However, greater dietary protein concentration can also increase feed intake by reducing propionate production. Increasing protein concentration could dilute diet starch concentration and decrease energy spilling by ruminal microbes, thus converting a greater fraction of fermented organic matter into microbial cells and less into VFA.

■ Gut Fill

As feed intake increases in early lactation, control of feed intake is dominated by ruminal distention and the extent to which ruminal distention limits feed intake is linearly related to milk yield (Voelker Linton and Allen, 2007). High

producing dairy cows should be fed diets with lower filling effect to maximize feed intake. The filling effect of a diet is determined primarily by the initial bulk density of feeds as well as their filling effect over time in the rumen. The overall filling effect is determined by forage NDF content, forage particle size, fragility of forage NDF determined by forage type (legumes, perennial grasses, annual grasses), and NDF digestibility within a forage family (Allen, 2000). Forage NDF is less dense initially, digests more slowly, and is retained in the rumen longer than other diet components. Feed intake of high producing cows is often dramatically reduced by increasing the forage NDF concentration of the diet. Several studies in the literature reported a decrease in DMI of up to 4 kg/d when diet NDF content was increased from 25 to 35% by substituting forages for concentrates. Although most studies reported a significant decrease in DMI as forage NDF increased, the DMI response was variable, depending upon the degree to which intake was limited by ruminal fill. Higher producing cows are limited by fill to the greatest extent and the filling effect of forage fiber varies depending upon particle size and fermentation characteristics.

Experiments that have evaluated effects of forage particle size have generally shown small effects on DMI (Allen, 2000). However, one experiment showed little effect of particle size of alfalfa silage when fed in high grain diets but a large reduction in DMI for the diet containing longer alfalfa silage when fed in a high forage diet (Beauchemin et al., 1994). Feed intake might have only been limited by ruminal fill in the high forage diet, which could explain the interaction observed.

Increasing diet NDF content by substituting non-forage fiber sources for concentrate feeds has shown little effect on DMI in studies reported in the literature (Allen, 2000). Non-forage fiber sources include byproduct feeds with significant concentrations of NDF such as soyhulls, beet pulp, cottonseeds, corn gluten feed, and distiller's grains. Fiber in non-forage fiber sources is probably much less filling than forage NDF because it is less filling both initially (smaller particle size) and over time in the rumen because it digests and passes from the rumen more quickly.

Forage NDF has a much longer ruminal retention time than other major dietary components. Retention time in the rumen is longer because of longer initial particle size and greater buoyancy in the rumen over time, which differs greatly across forages. As forages mature, the NDF fraction generally becomes more lignified. Lignin is a component of plant cell walls that helps stiffen the plant and prevent lodging. It is also essentially indigestible by ruminal microbes and limits fermentation of cellulose and hemicellulose. Within a forage type, the degree to which NDF is lignified is related to the filling effects of the NDF. Fiber that is less lignified clears from the rumen faster, allowing more space for the next meal. However, ruminal retention time of NDF from perennial grasses is generally longer than for legume NDF

in spite of being less lignified. Because of this, it is more filling and should not be included in high concentrations in diets of cows for which feed intake is limited by ruminal fill, unless it is of exceptionally high quality. Corn is an annual grass, and corn silage NDF digests and passes from the rumen quickly and can be an excellent source of forage NDF for high producing cows.

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, and 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 buffer 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 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 risk of abomasal displacement.

■ Unsaturated Fatty Acids

Feed and energy intake can be depressed by supplementation of fat and the extent of depression is dependent upon fat type (Allen, 2000). Fat sources with more unsaturated fatty acids reduce intake to the greatest extent and fatty acids that are highly saturated have less effect.

■ Recommendations

Limit mobilization of body fat by controlling body condition during mid to late lactation and limiting feed intake of dry cows by feeding diets with high forage

NDF concentration. A low concentration of highly fermentable starch might reduce NEFA concentration prepartum.

Maintain rumen fill through transition. Diets with high concentrations of grain, non-forage fiber, and finely chopped forages fed through the transition period should be avoided. 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 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 TMRs 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. However, grass with high potassium concentrations might require anionic salts in prepartum diets to reduce milk fever following calving.

Supplemental fat should not be fed through the transition period because it can depress feed intake by stimulating gut peptide release and increasing the supply of fatty acids to be oxidized. An exception might be the use of supplemental CLA to suppress fat production in the mammary gland, benefiting fresh cows by sparing glucose.

Avoid feeding highly fermentable starch sources to 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.

Feed a less filling and more fermentable diet 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, visual observation of cow gut distension, 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. Because feed intake is limited by ruminal fill, feed ingredients that can depress ruminal motility such as fat and sugar sources should be limited.

Feed a more filling, less fermentable diet as milk yield declines. As lactation progresses past mid-lactation, the highly fermentable diet that is optimal for high-producing cows can depress feed intake as milk yield and glucose demand decreases. Propionate is likely oxidized when it is produced faster than it can be utilized, generating ATP and a satiety signal. Therefore, cows should be switched to a less fermentable and more filling diet as milk yield declines. This will increase feed intake and provide a more consistent supply of fuels, reducing insulin and partitioning more energy to milk rather than body condition. Furthermore, the less fermentable, more filling diet will decrease risk of milk fat depression and late lactation abomasal displacement. Unsaturated fats likely decrease feed intake and should be limited. Limit highly fermentable starch sources (e.g. high moisture corn, ground barley) by substituting less fermentable feeds such as dry ground corn or non-forage fiber sources.

■ Conclusions

Consideration of physiological changes occurring through lactation and the physical and digestion characteristics of feeds beyond their nutrient composition is required to optimize feed intake for lactating 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.

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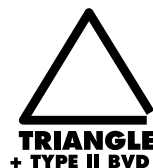
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¹ Data on File, Wyeth Animal Health

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