

## **Transition Cow Research – What Makes Sense Today?**

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### **INTRODUCTION**

The transition period for a dairy cow is from 3 to 2 wk prepartum until 2 to 3 wk postpartum. The term *transition* is to underscore the important physiological, metabolic, and nutritional changes occurring in this time frame. It constitutes a turning point in the productive cycle of the cow from one lactation to the next. The manner in which these changes occur and how they are managed are of great importance as they are closely linked to lactation performance, clinical and subclinical postpartum diseases, and reproductive performance that can significantly affect profitability.

Transition cow nutrition and management have received much attention in the research and popular-based literature in recent years because of the recognition of its importance in the productivity and health of cows. The problem that our research scientists are facing is their ability to gather enough cows of similar productivity characteristics in one place to conduct the research necessary to help us to understand this animal. Because of this difficulty, we have used animals outside of this short time frame and tried to simulate the same conditions that exist in the true transition cow. This method has given us a deeper understanding of the problems faced by these cows, but has also led to some confusion in interpreting data.

Many reports exist citing the metabolic and health problems that occur with increasing productivity (Simianer et al., 1991; Jones et al., 1994; Emanuelson and Oltenacu, 1998) and with the economic

losses associated with these (Drackley, 1999). The occurrence of health problems during the transition period is clearly a major complicating factor for subsequent reproductive performance (Ferguson, 2001), resulting in additional economic losses. Poor transitions also result in milk income losses. Every pound of milk lost from peak production represents a loss of 200 lb of milk for the lactation. Poor transition periods often result in the loss of 10 to 20 lb of peak milk (e.g., Wallace et al., 1996), which could represent 2000 to 4000 lb of unrealized milk yield. It is, therefore, pertinent to elaborate nutritional strategies to facilitate the passage of the cow through this transition phase; while minimizing health problems and optimizing productivity/profitability for the remainder of the ensuing lactation.

### **TRANSITION COW BIOLOGY**

We will proceed with a review of the biology of the transition cow and the phenomena that mark this period with the goal of developing nutritional strategies.

#### **Dry Matter Intake**

It is now well established that dry matter intake (**DMI**) decreases as calving approaches. Dry matter intake can decrease from 2 % of body weight (**BW**) in the first few weeks of the dry period to 1.4 % BW in the 7 to 10 d period before calving. This 30 % decrease in DMI appears to occur very rapidly in the transition period (Bertics et al., 1992; Hayirli et al., 1998; Robinson and Garrett, 1999). During the 3 wk after calving DMI will increase at the rate of 1.5 to

2.5 kg /wk (Grant and Albright, 1995) with this increase being more rapid in multiparous cows than primiparous cows (Kertz et al., 1991; Robinson and Garrett, 1999). However, individual cow variation in the decrease prepartum and the increase postpartum in DMI is enormous (VandeHaar and Donkin, 1999).

The decrease in prepartum DMI has classically been attributed to the rapid growth of the fetus taking up abdominal space and displacing rumen volume. However, there is no doubt that hormonal and other physiological factors have the most important impact on this phenomenon (Grant and Albright, 1995; Robinson, 1997). During the last week of pregnancy, nutrient demands by the fetal calf and placenta are at their greatest (Bell, 1995), yet DMI may be decreased by 10 to 30 % compared with the early dry period.

### **Physiological Changes**

A number of profound physiologic changes occur in the transition cow that modifies her metabolism drastically. The rapidly increasing demands of the fetus and the development of the mammary glands, including the initiation of synthesis of milk components, are causing these changes. Bell (1995) estimated that the daily demands for fetal and placental growth in the last 3 wk of gestation are 360 g of metabolizable protein and 3 to 5 Mcal of net energy.

The concentration of plasma insulin continually declines in the transition period until calving and that of somatotropin increases rapidly between the end of gestation and the initiation of lactation. Concentration of plasma progesterone, which is high in gestation, rapidly falls at calving and there is a transitory elevation in estrogens and glucocorticoids in the periparturient period. These hormonal changes not only contribute to the decline in

DMI, but also coordinate the metabolic changes that favor, if not force, the mobilization of body fat reserves from adipocytes (Grummer, 1995). Resulting from this mobilization of lipids, we observe an increase in concentration of plasma non-esterified fatty acids (**NEFA**), which rise gradually in the prepartum transition period, but rapidly in the last 3 d of gestation. A portion of this increase in NEFA is obligatory and is under hormonal control while another portion is the result of an energy deficit (negative energy balance or **NEB**) (Bertics et al., 1992; Grummer, 1995; Dyk and Emery, 1996). The magnitude of the NEB prepartum, therefore, appears to be a variable that can be mitigated through nutritional management. Additionally, the NEB and resulting increase in plasma NEFA, if sufficiently high, contributes to the development of fatty liver; which itself is a contributing factor to other health problems in the postpartum period (Grummer, 1995; Dyk and Emery, 1996).

### **Rumen Function**

It is not unusual for a high producing cow in the first 100 d in milk (**DIM**) to consume 22 kg of DM/d, of which more than half is in the form of concentrates, without posing any particular problems to the cow if the diet is well balanced. However, this same diet consumed by a fresh cow can cause a severe ruminal acidosis. The major difference in rumen function between these 2 stages of lactation can explain the different responses to the same diet. During the dry period, cows generally consume a diet that is principally composed of forages and, by consequence, is more fibrous than the type of diet offered in lactation. This nuance affects rumen function in 2 ways. First, the rumen flora is adapted to a diet that is low in non-fiber carbohydrates (**NFC**) during the dry period

allowing for a large population of cellulolytic bacteria and a low population of amylolytic bacteria. As the amylolytic bacteria also generate lactic acid, their decrease is accompanied by a decrease in the bacteria that utilize lactic acid (Goff, 1999).

If the ration is changed abruptly at calving the capacity of the rumen flora to metabolize lactate, the principal acid responsible for acute rumen acidosis, is at a minimum at the initiation of lactation. The lactate producing bacteria increase in numbers rapidly as the amount of NFC in the diet increases, but the lactate-utilizing bacteria adapt more slowly (3 to 4 wk). Therefore, the risk of lactate accumulation in the rumen is high with abrupt changes from high to low fiber diets. Further, it is a known phenomenon that as DMI increases, rate of passage from the rumen increases as well. After calving, when DMI is relatively low, rate of passage is slow; allowing for greater extent of fermentation and acid accumulation in the rumen.

If dietary NFC increases abruptly at calving, with high levels of fermentable carbohydrates, the amount of VFA produced far exceeds the capacity of the rumen to absorb them leading to elevated concentrations of VFA in the rumen. This situation leads to the phenomenon known as subacute rumen acidosis (**SARA**) and contributes to reduced DMI and feed digestibility as well as laminitis in the early postpartum period.

### **Health Problems Associated with the Transition Period**

The conditions described above favor the occurrence of health problems during the transition period. The principal metabolic problems gravitate around 3 principal axes:

1. Disorders related to energy metabolism (fatty liver, ketosis, subacute and acute ruminal acidosis);
2. Disorders related to mineral metabolism (milk fever, sub-clinical hypocalcemia, udder edema); and
3. Problems related to the immune system (retained placenta, metritis, mastitis).

A factor that has not received a lot of specific attention and is superimposed on all of this is the issue of protein requirements. This will be discussed subsequently as well.

We will see in the next sections some of the etiology and preventive measures for these problems and the nutritional considerations related to these 3 axes. It is necessary to mention here that these disorders are interrelated and that the conditions conducive to the development of one problem in one category can lead to a disorder in another category. For example, milk fever or subclinical hypocalcemia can lead to loss of muscular tonicity resulting in an increased risk of retained placenta and/or displaced abomasums, in spite of retained placenta being classified as a problem related to the immune system.

Curtis et al. (1985) conducted a retrospective analysis of the risk factors associated with metabolic problems. Their analysis revealed that older cows were more at risk for retained placenta, milk fever, and mastitis. Cows having retained placentas were more at risk for developing mastitis and ketosis. Cows with ketosis were 12 times more likely to develop displaced abomasums. Generally, this analysis suggests that the prevention of one problem can decrease the incidence of others from developing.

## **NUTRITIONAL CONSIDERATIONS**

The transition period is marked by major hormonal changes. While these hormones are causing a reduction in DMI there is an increase in nutrient requirements by the cow to support fetal growth, mammogenesis, and lactogenesis (Bell, 1995; Grummer, 1995). This increase in nutrient demand is partially met by the DMI and partially by the mobilization of body tissues. Although the hormonal milieu will drive a certain amount of this body mobilization, excessive body catabolism is undesirable for health, reproduction, and milk production. It is, therefore, essential to pay particularly close attention to the formulation of rations in this transition period, both pre- and post-partum.

Few published studies exist on transition period nutrition on a holistic basis. However, we will try to review the literature related to specific nutrients studied in this period of the cow's production cycle.

### **Requirements and the Negative Energy Balance (NEB)**

The energetic demands of gestating cows reach 1.3 to 1.5 times the maintenance requirements by the end of gestation (Quigley and Drewry, 1998). The growth of fetal tissues follows an exponential curve beginning in the third trimester of pregnancy (Bell et al., 1995; House and Bell, 1994). For those using the Cornell Net Carbohydrate and Protein System (CNCPS) version 6.1; the fetal, placental, and mammogenesis requirements for nutrients are now more dynamic and added into requirements.

During both the prepartum and postpartum transition period cows require more energy than they are able to consume resulting in the NEB and the concomitant loss of body weight (condition) to supply the

necessary energy even in healthy cows (Bell, 1995). Grummer (1995) demonstrated that the NEB and body weight loss begins in the prepartum transition period, but that the NEB is greatest in the first week postpartum. However, there is much variation in the magnitude of the NEB after calving depending upon body condition score at calving (Garnsworthy, 1989), the severity of the depression in DMI (Bertics et al., 1992), the quality of the ration, and season.

A severe NEB in the transition period can aid in the development of metabolic diseases, prolong the interval between calving and first ovulation and decrease fertility. Butler et al. (1981) showed that first ovulation in cows occurs 10 d after the nadir in NEB. Energy during the transition period; therefore, has a major impact on cow performance and longevity. Severe NEB also can lead to fatty livers and compromised liver function essential in all metabolic functions including detoxification.

### **Regulation of Energy Metabolism and Glucose Homeostasis**

Glucose is a substance that plays a fundamental role in all living beings. In the last week of fetal development, the fetus uses an estimated 46 % of maternal glucose taken up by the uterus (Bell, 1995). Additionally, a cow producing 30 kg of milk/d uses at least 2 kg of blood glucose to synthesize lactose for milk (Bell, 1996). The end of pregnancy and the beginning of lactation; therefore, represent a time when there is a massive increase in need for glucose. This poses an enormous challenge for the liver that has to synthesize all of this glucose from propionate and amino acids as well as a challenge for other tissues and organs that have to adapt to a reduction of glucose availability. Glucose is an equally important energy source for the ovary and

the reduced glucose availability in the beginning of lactation can negatively impact the re-establishment of ovarian activity after calving (Rabiee et al., 1999).

The concentration of plasma insulin decreases dramatically as calving approaches and remains low in the first week postpartum. The concentration of plasma somatotropin increases dramatically and rapidly postpartum. This decrease in insulin combined with a decrease in sensitivity of adipose tissue to insulin and the increase in somatotropin results in a decrease in the synthesis of triglycerides in adipose and favors their mobilization (Bell, 1995; Lanna and Bauman, 1999). Therefore, there is a resulting increase in plasma NEFA at calving. The somatotropin, in concert with elevated cortisol, stimulates the liver to produce more glucose.

These coordinated changes in metabolism permit the cow to respond to the accrued nutrient demands for milk synthesis. However, due to many factors, including those previously mentioned, the metabolic processes do not adjust sufficiently and a disequibrated metabolism leads to many of the typical problems associated with transition cows.

### **Metabolic Problems Associated with Energy Nutrition**

The mobilization of lipids in the beginning of lactation is a normal and required process to help the cow meet her energy demands for lactation. However, when the quantity and/or the speed of mobilization are exaggerated, the incidence of metabolic problems increase significantly. It is not uncommon to find a ketotic cow also having problems with fatty liver and displaced abomasum. Ruminant acidosis is also a frequent problem for cows

at the beginning of lactation, because of highly fermentable rations and insufficient rumen adaptation (slow passage rates) to these rations.

### ***Ketosis and Fatty Liver Syndrome***

Fat, or lipid, deposited in adipose is in the form of triglycerides that are mobilized as NEFA plus the glycerol backbone of the triglyceride. The liberated, or mobilized, NEFA have one of 3 desirable fates:

1. To be utilized by the mammary gland for milk fat synthesis;
2. To be used by peripheral tissues as a source of energy; and
3. To be reesterified by the liver into triglycerides and exported as triglycerides incorporated into very low density lipoproteins (**VLDL**).

Complete oxidation of NEFA generates metabolites (acetyl coenzyme A) that can be used to generate energy via the Krebs Cycle. However, if the Krebs Cycle gets overloaded the acetyl Co A is shunted off to produce ketones (acetoacetic acid, acetone, and  $\beta$  hydroxybutyrate or **BHB**). Obviously, if ketones reach a high concentration in the blood the cow becomes ketotic.

The normal concentration of plasma ketones (using BHB as an indicator) is less than 10 mg/dl. Clinical ketosis is defined as a concentration greater than 35 mg/dl. Concentrations between these 2 values are termed subclinical ketosis (Drackley, 1997). Duffield (1997) established that when BHB reaches 1400  $\mu\text{mol/l}$  (14.6 mg/dl) in blood, the incidence of metabolic disorders increases and when the value is above 2000  $\mu\text{mol/l}$  (20.8 mg/dl) milk production begins to suffer.

With the background given above it might be curious to ask why the liver would accumulate fat if it can process the NEFA. When fat mobilization is high and the liver is reesterifying NEFA into triglyceride it cannot produce VLDL fast enough to export the fat and accumulation begins. This is because the liver has a very limited ability to produce VLDL due to a limited capacity to produce a key component (apoprotein B) (Marcos et al., 1990; Grummer, 1995). Once deposited, the fat accumulated in the liver will remain there until the end of the NEB (Grummer, 1993).

Pronounced accumulation of fat in the liver with liver damage and/or reduced liver function (Van den Top et al., 1996) compromises, among other things, glucose synthesis. Most of the glucose that the cow requires is synthesized by gluconeogenesis in the liver from propionic acid, amino acids, and the glycerol liberated from adipose.

Rukkwamsuk et al. (1999) evaluated the capacity of gluconeogenesis by livers of fat and normal cows at calving. The fat cows had an increase of 446 % blood NEFA at 3 d postpartum compared to their prepartum NEFA concentration, while normal cows exhibited an increase of 123 %. Similarly, the concentration of fat in the liver of fat cows had increased 514 % by 3 d after calving while the normal cows had an increase in liver fat by only 97 %. The activity of liver enzymes indicated that gluconeogenic capacity of the fat cows was impaired.

To minimize a predisposition of cows to ketosis and fatty livers it is necessary to avoid excessive weight loss in the pre- and postpartum transition period by minimizing the magnitude of the NEB. The weight loss, fat mobilization, NEB, increased plasma

NEFA, and liver fat accumulation is inevitable in all high-producing cows. However, it is imperative to minimize these. We must make all attempts to maximize DMI, increase the energy density of the diets without sacrificing rumen function, and maintain BCS near recommended levels at this critical time.

### ***Rumen acidosis***

When acid production in the rumen resulting from fermentation of organic matter exceeds the capacity for the animal to absorb these acids or be neutralized, ruminal pH falls. This acidosis, even at the subclinical level, will cause a reduction in cellulolytic bacteria in the rumen and contribute to an overall reduction in feed digestibility. Furthermore, the acidosis reduces rumen motility and efficacy of mixing rumen contents, which reduces the amounts of VFA near the rumen wall. Consequently, VFA absorption is reduced (Allen and Beede, 1996). As rumen motility and mixing decline so does rumination, which reduces the amount of saliva flowing into the rumen. The secretion of buffers in the saliva is equivalent to more than 3 kg of sodium bicarbonate and 1 kg of disodium phosphate (Erdman, 1988).

When NFC present in the rumen increases abruptly and rapidly, the production of VFA increases proportionately (Owens et al., 1998). If these VFA are not absorbed rapidly they will accumulate in the rumen (Schultz et al., 1993). Different problems are caused by rumen acidosis depending on the quantity of acids that accumulate in the rumen. When there is a mild accumulation, DMI and production can decline in spite of the cow appearing to be in good health (Owens et al., 1998). Also, the milk produced could have reduced fat. Certain species of bacteria and protozoa in

the rumen are extremely sensitive to acidosis and release endotoxins and histamine. These substances can contribute to laminitis (Nocek, 1996). As the acid accumulation continues more severe problems can occur with DMI and production as well as with overall health (Schultz et al., 1993).

It appears that this subclinical rumen acidosis is insidious and omnipresent in transition cows, as well as cows in early lactation. It is beyond the scope of this presentation to adequately describe this subject area, however, there are numerous publications dealing with its description and detection (Yokoyama and Johnson, 1988; Nordlund, 1994; Allen and Beede, 1996; Hall, 1999).

Based on the above discussion it appears prudent to begin to increase NFC in the prepartum transition period to adapt the rumen wall to VFA, to feed as frequently as possible postpartum or to offer TMR to reduce acid accumulation at any time during the day, to verify that the proper amounts of effective fiber are being fed (and consumed), and that sufficient dietary buffers are offered in the postpartum diet. Direct microbial supplements are also being investigated with the aim of controlling lactic acid production (Kung, 1999).

### ***Displaced abomasum***

Displaced abomasum (**DA**) is frequently observed in high producing herds (Robertson, 1968; Robb et al., 1986). This disorder increases the risks of early culling, other disorders, and reduces productivity (Geishauser et al., 1998). It is logical that other disorders can also increase the risk of DA.

Epidemiological studies have shown a correlation between DA and breed, age, parity, and season (Markusfeld, 1987;

Constable et al., 1992). Hypocalcemia increases the risk of DA (Massey et al., 1993) probably because of the role of calcium in muscular tonicity. Nutrition has been implicated in the incidence of DA and these nutritional factors are controllable (Coppock, 1974; Cameron et al., 1998). These factors are: high BCS at calving; excessive weight loss; elevated plasma NEFA (Cameron et al., 1998); reduced DMI in the transition period (Constable et al., 1992); and reduced forage-to-grain ratio (Shaver, 1997).

Consequently, feeding management designed to mitigate a decline in DMI prepartum and rapidly increase DMI postpartum will reduce the risks associated with DA. Logically, reduction of any of the postpartum production diseases (milk fever, ketosis, metritis, and retained placenta) will reduce the risk for DA.

Increasing the energy density of the prepartum diet with grains equivocally affects the risk of DA. The reason for the equivocal nature of this long-held assumption is that if NFC is not increased, the rumen wall is not prepared for the abrupt increase in concentrates postpartum which will increase the risk for DA (Shaver, 1998). Moreover, if the prepartum transition diet is too low in energy, the NEB will be large leading to the other risk factors for DA as discussed previously.

### **Nutritional Strategy**

From the discussion above, it is obvious that preventing the decline in DMI prepartum, increasing DMI rapidly postpartum, and making certain that energy density is as high as possible in both transition phases are the most important control points for these cows.

There are many non-nutritional modulators that affect DMI (Mertens, 1994; Rosseler, 1993). The nutritional factors include NDF content and digestibility and NFC content and fermentability, in both the pre- and postpartum diets. Including rumen buffers in the postpartum diet can also serve the cow well.

### ***Sources of dietary energy***

The principal energy sources in a diet are carbohydrates and fat. Carbohydrates include both fiber (NDF) and non-fiber (NFC) categories. The proportion of NDF and NFC in the transition period (both pre- and postpartum) is a key factor to maximizing DMI. Too high a content of NFC predisposes a cow to acidosis, reducing DMI; too low a content prepartum will also predispose cows to acidosis postpartum; too much NDF will also limit DMI (Mertens, 1994). A cow at the end of gestation should receive a diet with a minimum of 35 % NDF in the total DM (Shaver, 1993). For the postpartum transition period the recommendations are in the order of 28 to 32 % NDF in the total DM (Hutjens, 1995; Drackley, 1998). Following the transition period NDF recommendations can be reduced to 26 % with 3/4 of this NDF being contributed by high quality forages (Drackley, 1998).

The addition of NFC to the diet is necessary in the transition period but should be done gradually. Furthermore, NFC fermentation will produce more propionic acid; thereby increasing the supply of glucose precursors and minimizing the use of glycogen reserves normally observed in the transition period. One can further speculate that if more glucose precursors are available in the form of propionate, then fewer amino acids will be used for gluconeogenesis in supplying energy to the

cow. It was also suggested that by increasing NFC in the diet that a greater microbial mass would be present producing more microbial protein (Nocek and Russell, 1988).

The recommendations for NFC for prepartum transition cows is 30 to 35 % of total dietary DM (Shaver, 1993). For postpartum transition cows NFC should be 38 to 40 % of total dietary DM (Drackley, 1998).

It has been speculated that feeding supplemental fat would reduce fatty acid mobilization from body stores and reduce liver fat accumulation (Kronfeld, 1982). Fats in the diet are incorporated into lipoproteins in the intestine and absorbed into the circulation. As they are already in the form of lipoproteins they can be used directly by the tissues and, theoretically, not contribute to fat accumulation in the liver. The tissues produce an enzyme called lipoprotein lipase that will break down the lipoproteins (from the intestine or exported from the liver) to NEFA for utilization. The liver of ruminants lacks any appreciable quantities of lipoprotein lipase and hepatic lipase (Emery et al., 1992). This makes perfect sense in that the liver is producing lipoproteins for other tissues and should not be able to extract them from the blood when it has an alternative, i. e. NEFA, as an energy source. Note that Kronfeld's (1982) speculation was that dietary fat would REDUCE, not halt, fat mobilization. We must recognize that adipose tissue can synthesize fat (use lipoproteins to make fat) and mobilize NEFA (hydrolyze body fat). However, the transition cow is likely to be in a state of lipolysis predominantly because of her energy balance status and her hormonal profile (low insulin and high cortisol) that does not favor body fat deposition. Therefore, it is unlikely that dietary fat will reverse the status of the adipose tissue from

being catabolic (mobilization of fat) to anabolic (deposition of fat).

Research results have been mixed in terms of feeding fat to transition cows; where some show a benefit and others do not (Skaar et al., 1989; Grum et al., 1996; Douglas et al., 1998; Bertics and Grummer, 1997). There are a variety of factors that we should consider in assessing a position on feeding fat to transition cows prior to seeing more definitive work on the subject. These are: 1) the experimental models used to assess feeding fat; 2) the quantities of fat fed; and 3) the fatty acid profile of the dietary fats. Let us discuss each of these and then try to synthesize a strategy.

*1. The experimental model(s) used to assess feeding fat to transition cows.*

At best it is extremely difficult to amass groups of transition cows suitable to conduct research. Cows need to be of similar body condition score, parity, expected calving dates, and genetic potential at the onset of a trial; as all of these factors will affect the outcome and the reaction of the cow to any nutritional modification. As an example, thin vs. obese dry cows will have different dynamics in fat metabolism, DMI patterns, and nutrient needs both pre- and postpartum. These different cows will react differently to supplemental fat (or other nutrients). High producing cows postpartum will react differently to supplemental nutrients in the pre- and postpartum periods from lower producing cows as their needs, metabolic processes, and DMI differ from one another.

Research trials conducted to date have been victims of some of the circumstances mentioned above. In an attempt to circumvent these disparate conditions, some researchers have used far-off dry cows fed restricted quantities of DM (and nutrients) to

emulate the transition cow prepartum. While this model is a necessary evil to begin to evaluate transition cows, it is far from a viable description of the real situation; therefore results should be viewed with caution. An imposed feed restriction of a given percentage is not representative of a transition cow that declines in DMI gradually in the first phases and dramatically as calving becomes more eminent. Furthermore, cows of different body condition scores and parities will have different degrees of this reduction in DMI. Maybe the most important point to consider is that the metabolic, physiologic, and hormonal status; as well as the nutrients that are required by the prepartum transition cow, is vastly different from the far-off dry cow.

Another consideration is the nutrient density of the emulated transition diet. In the normal (compensated transition) diet DMI is decreasing, but concentration of nutrients is increased to attempt to offset this. When a diet is simply restricted, all nutrients are collectively restricted, which can lead to conclusions that do not represent the true transition phase.

While the published research must be commended for giving us initial insights into transition cows, we must strive to look for more appropriate and applicable data.

*2. The quantity of fat fed.*

Most of the trials investigating transition cows have fed between 225 and 454 grams (1/2 to 1 lb) of fat per cow/d. Some have fed as much as 1.5 lb/d. The rationale for the quantity fed is obscure.

The theoretical basis for feeding fat to prepartum transition cows is to reduce the energy deficit prepartum and to supply an

alternate source of lipoproteins to the tissues (i. e., intestinal source instead of originating from the liver). This is aimed at **reducing** adipose lipolysis and reducing the load of NEFA presented to the liver for processing. The emphasis here is on reducing adipose lipolysis, not stopping or reversing it. As pointed out earlier, all cows just prior to calving show an elevation of NEFA in blood that continues into early lactation. Furthermore, all cows will accumulate triglycerides (fat) in the liver. The hormonal profiles leading up to lactogenesis force the animal to increase lipolysis and reduce lipogenesis from adipose almost irrespective of their dietary energy intake.

If we consider why we are feeding fat to transition cows prepartum then it makes no sense to offer cows 1 lb of fat. Superficially, this may be the amount of body weight she can lose in a day, but this quantity makes no sense in terms of metabolism. The hormonal profile and metabolic status of the cow is forcing catabolism (breakdown of body stores). There is not only lipolysis of adipose but also a mobilization of storage proteins aimed at gearing up the metabolic machinery for the ensuing lactation. Again, this cannot be stopped, but should be minimized to absolute necessity if the proper nutrient profile is supplied in the diet. The only metabolite that is being deposited is liver and muscle glycogen (storage form of glucose), which is impeded by fatty liver.

The cow is going to have a certain amount of lipolysis occurring, NEFA available, lipoprotein production, and fat accumulation in the liver. The suggestion has been made by Bertics and Grummer (1999) that feeding fat elevates blood NEFA because the intestinally produced lipoproteins are not completely used by the tissues. When a tissue hydrolyzes lipoproteins, the resulting NEFA are partly

used for energy and the rest *spill* into the blood and are available for liver deposition. What is referred to as *spilling into the blood* may actually be the result of an oversupply of lipoproteins to the tissues. Most tissues do not accumulate fat. Excess fat in circulation can only be deposited in adipose storage sites or the liver. Since there is general catabolism in the animal at this time, it is unlikely that she will deposit appreciable amounts of adipose. Hence, the liver will accumulate the excess fat.

The above discussion is to suggest (or theorize) that high levels of fat inclusion in the diet of transition cows are contraindicated. While added fat may not be required from an energy standpoint, per se, it seems beneficial from a metabolic standpoint. Quantities of less than 1/2 lb/d have a good chance of being more beneficial than no fat or than more than 1/2 lb of fat in terms of optimizing lipoprotein use by tissues and reducing adipose lipolysis. Moreover, 1 lb fat/d in a diet when DMI is severely restricted results in a very high concentration of dietary fat, especially in relation to other dietary nutrients.

### *3. The fatty acid profile of the fat fed to transition cows.*

Recently, attention has been paid to the digestibility of different fat sources. Fat sources that are high in stearic acid (i. e., hydrogenated fats) have a lower digestibility than the unsaturated C:18 fatty acids. A reduced digestibility implies that the energetic value of different fats will differ based on their degree of hydrogenation and their ability to be biohydrogenated in the rumen (i. e., free fats vs. calcium salts of long chain fatty acids). Obviously, the lower the digestibility of the fatty acids, the less chance there is to improve energy balance or reduce lipolysis.

Furthermore, although the research is in its infancy there are strong indications that saturated and unsaturated fatty acids will cause different blood and liver profiles in fat metabolism and cause different rates of ApoB synthesis in the liver (for lipoprotein production). It appears that unsaturated fatty acids are more desirable in this case. (Leplaix-Charlat et al., 1996).

Interestingly, reported trials that used more saturated fats or fats that are active in the rumen seemed to have negative impacts on transition cows. Longer term feeding of fat supplements that were rumen protected, had a content of long chain mono- and poly-unsaturated fatty acids, and were fed at less than 1/2 lb/d had more positive effects when fed prepartum.

The subject of feeding fat in the postpartum transition phase also makes ultimate sense from the standpoint of energy balance, as well as productive and reproductive performance. Allen (1998) showed compiled data indicating that DMI is reduced when fat is fed postpartum. However, closer examination of the data reveals that this finding is true only when levels higher than recommended are included or when productivity is not considered. This makes sense in that once energy demands are met or exceeded the cow will reduce DMI to compensate for an oversupply of nutrient. When the data is examined for recommended inclusion rates of dietary fat from high producing cows fed normal amounts of fat, a depression in DMI is not apparent.

Some recent research has also been pointing to the inclusion of unsaturated fatty acids and essential fatty acids to the intestine (via the use of calcium salts) in improving immune function and reproductive function during the transition period. Staples et al.,

(1998) and Jones et al. (2008) both show that fat in general (Staples et al., 1998) and more specifically calcium salts of essential fatty acids (Jones et al., 2008) improve reproductive and immune function leading to potentially higher pregnancy rates and less endometritis.

### **Protein and Amino Acids**

Bell (1995) had shown that the uterus extracts 72 % of amino acids in circulation at the end of gestation. When there is insufficient protein and amino acids, the cow will mobilize her limited protein reserves in peripheral tissues and muscle (Bell, 1995). Even though the synthesis and efficiency of protein synthesis is increased during this period the diet should theoretically require a higher concentration of protein because of the reduced DMI (Bell, 1995). Moreover, it should be beneficial to maintain rather than use protein reserves to support milk production postpartum.

The literature on dietary protein in the transition period is contradictory. Some studies show a benefit to increasing dietary protein prepartum for body gain (Van Saun et al., 1993) and milk protein production postpartum (Van Saun et al., 1993; Moorby et al., 1996). However, other studies did not detect a benefit above the NRC (1989) (Wu et al., 1997; Carson et al., 1998; VandeHaar et al., 1999). While primiparous cows may require a higher dietary protein concentration at 14 to 16 % of total DM, dietary protein requirements for multiparous cows at 12 % (NRC, 1989 specification) appear only valid when DMI in the transition period is only marginally declining (Bell, 1995; VandeHaar and Donkin, 1999). Most of this cited work does not indicate a necessity to increase the undegradable protein (**UIP**) in the diet beyond NRC (1989). However, use of non-

protein nitrogen sources should be avoided (VandeHaar and Donkin, 1999; Schwab, 1998).

It has been recommended, contrary to NRC (1989), to increase the crude protein (**CP**) in the prepartum transition diet to 14 to 16 % (Shaver, 1993; Dyk and Emery, 1996) with a UIP of 33 to 38 % of the total protein (Shaver, 1993). It is interesting that the studies that show a response to prepartum protein and/or increased UIP were feeding diets that were marginal in energy in the form of NFC (Van Saun et al., 1993; Huyler et al., 1999). It is probable that a considerable portion of absorbed amino acids were oxidized for use as energy; thereby creating a protein (amino acid) deficit and allowing the cows to respond to extra protein or UIP.

When the energetic density of the prepartum transition diet is high (> 1.48 Mcal/kg DM) with an NFC of 33 to 35 % there appears to be no benefit for multiparous cows to have a dietary protein concentration more than 14 % (Putnam and Varga, 1998; VandeHaar et al., 1999; Santos et al., 1999a). Primiparous cows in the prepartum transition period seem to need 15 to 16 % protein in the total DM (VandeHaar and Donkin, 1999; Santos et al., 1999b). Supplementing protected amino acids prepartum is receiving more attention with promising results (Fredeen et al., 1999; Rode et al., 1999) but dosages and requirements need more definition (Garthwaite et al., 1998). In the case of amino acid excess, an oversupply of methionine in the intestine causes a disequilibrium of the methionine-to-lysine ratio and can have profound effects on further reducing DMI (Schwab, 1998).

Specific amino acids can influence other aspects of metabolism not related to tissue

formation. For example, methionine is needed for VLDL production by supplying metabolites for apoprotein B and/or lecithin synthesis. This should result in less fat accumulation in the liver and less circulating ketones (Beauchart et al., 1998).

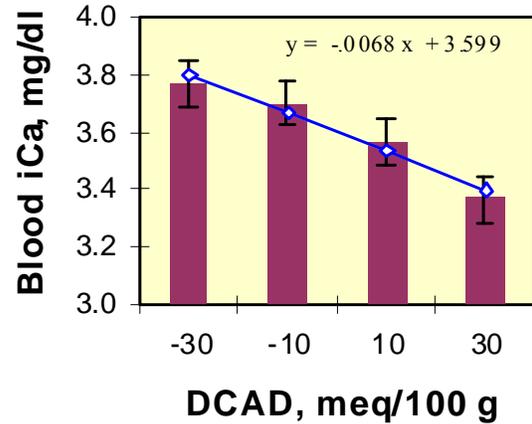
A typical diet in the beginning of lactation has between 18 and 19 % CP and 35 to 40 % UIP (Nocek, 1995; NRC, 1989). Lysine and methionine are recognized as the 2 most limiting amino acids for production of milk protein (Schwab et al., 1992). It would, therefore, be advantageous to add these in the form of rumen protected amino acids or in a protein supplement of known rumen bypass and amino acid content. A compilation of studies shows that when bypass amino acids are added to the diet to meet the requirement, supplementation in the postpartum transition phase yields a positive response of 0.5 kg/day more milk and 68 g/day more milk protein and when supplementation commenced in the prepartum transition phase the response was greater at 1.7 kg/day more milk and 79 g/day more milk protein (Garthwaite et al., 1998; Schwab, 1998).

We realize that cows really require intestinally available protein from bacterial and RUP sources and that this requirement for metabolizable protein (**MP**) can be unrelated to dietary CP concentration. If we re-evaluate the research cited above, it appears that these prepartum transition cows require between 1100 and 1300 g/d of MP. In calculating the trial by Van Saun et al. (1993), primiparous cows can need MP in excess of 1300g/d. This disconnect between MP and CP becomes even stronger as more byproducts become available for use. It has probably been more typical for some diets to contain 14 to 15 % CP that deliver less than 1000g/d of MP. We must be cognizant of the fact that MP is absolutely required for

mammary gland development, fetal development, milk protein yield, immune function, reproductive function, etc. Further, the cow has only limited reserves of protein available. Metabolizable protein might, in fact, be one of the more significant bottlenecks to successful transition periods.

### DIETARY CATION-ANION DIFFERENCE (DCAD) AND BLOOD CALCIUM AT CALVING

The onset of lactation causes a severe and rapid drain on blood calcium required to produce milk. If this blood calcium is not replaced as rapidly as it is reduced via bone calcium release (resorption) or intestinal absorption of calcium, cows will become hypocalcemic with some developing clinical milk fever. Reducing DCAD to negative values has been shown by many authors to prevent this rapid decline in blood calcium at calving. This is best illustrated in Figure 1 and Table 1. Giesy et al. (1997) showed that when different DCAD were fed to cows that were challenged with an infusion of EDTA (ethylene-diamine-tetraacetic acid) – to remove calcium from blood—that cows maintained their blood calcium better as the DCAD was reduced (Figure 1). In a trial with periparturient cows, Leclerc and Block (1989) showed a highly significant negative correlation between DCAD and concentration of blood calcium, which was strongest from 12 hr pre- to 12 hr postpartum (Table 1). Therefore, as DCAD is reduced prepartum, blood calcium concentration is maintained at a higher level around parturition.



**Figure 1.** Blood ionized Ca response to 4 levels of DCAD following infusion with EDTA to mimic hypocalcemia (Giesy et al., 1997).

### Clinical and Subclinical Hypocalcemia (Milk Fever)

In a 2007 survey, (USDA, 2009) 83.5 % of all dairies in the United States reported clinical milk fever as a health problem with an incidence rate of 4.9 %. This figure is only slightly below the estimated incidence in 1993 of 5 to 7 % (Jordan and Fourdraine, 1993). Research indicates that cows with clinical milk fever produce 14 % less milk in the subsequent lactation and their productive life is reduced approximately 3.4 yr when compared to non-milk fever cows (Block, 1984; Curtis et al., 1984).

Furthermore, cows that recover from milk fever have an increased risk of ketosis, mastitis (especially coliform mastitis), dystocia, left displaced abomasum, retained placenta, and milk fever in the subsequent lactation (Curtis et al., 1984; Wang, 1990; Oetzel et al., 1988). Guard (1996) estimated the average cost per case of milk fever to be

**Table 1.** Correlation between blood calcium concentration and DCAD from 48 hr prepartum to 36 hr postpartum (Leclerc and Block, 1989)

Time (hours)	Coefficient of Correlation	Probability of significance (P > F)
48 prepartum	-0.47	0.048
36 prepartum	-0.38	0.121
24 prepartum	-0.33	0.162
12 prepartum	-0.46	0.046
PARTURITION	-0.55	0.015
12 postpartum	-0.59	0.013
24 postpartum	-0.27	0.248
36 postpartum	-0.46	0.058

\$334. In addition, cows with milk fever are also susceptible to secondary disorders, such as ketosis, mastitis, retained placenta, and displaced abomasums (Curtis et al., 1985).

In addition, and maybe of greater importance, is the prevalence of subclinical hypocalcemia (i.e. milk fever) which may be as high as 66 % for multiparous dairy cows following calving (Beede et al., 1992). Subclinical milk fever occurs when the clinical symptoms of the disease are not seen, but blood calcium still decreases substantially around parturition. Just as with clinical milk fever, subclinical low blood calcium can lead to low dry matter intake (**DMI**) postpartum, dystocia, ketosis, and retained placentas.

The occurrence of hypocalcemia (clinical and subclinical) is a multifactorial disease that is not solely dependent on DCAD. In a comprehensive meta-analytical model, Lean et al. (2006) described the 4-mineral DCAD equation as a major contributor to hypocalcemia, but also found other dietary and management factors had independent contributions (prepartum dietary calcium, magnesium, and phosphorus as well as breed and number of days fed the prepartum transition diet). Most of the prepartum diets included in this manuscript were relatively high in starch and low in fiber compared to the traditional far-off dry cow diet.

However, it was recently shown that the positive effects of DCAD on blood calcium and postpartum milk production (discussed subsequently) are prevalent as well when higher fiber, reduced starch diets are fed (Sicilliano-Jones et al., 2008)

#### **Prepartum DCAD and its Relation to Pre- and Postpartum DMI and Subsequent Milk Production**

The negative effects of DCAD on DMI have been reported and appear obvious when more than 300 meq/kg (30 meq/100g) of anionic salts are added to a diet. Heifers seem more sensitive to the anionic supplements than cows. Most of the reported research has been with anionic mineral supplements, which might pose palatability problems when less than 300 meq/kg are needed. Research reported with non-mineral based anionic supplements have not shown the DMI reduction prepartum as occurs with mineral salt supplementation (DeGroot, 2004; Sicilliano-Jones et al., 2008).

Obviously, the prevention of milk fever will increase milk yield by about 14 % in those cows that would have succumbed to the disease, will extend their productive life (Block, 1984; Curtis et al., 1984), and will reduce the incidence of other postpartum disorders (Wang, 1990; Oetzel et al., 1988).

In those trials that have measured postpartum milk production, multiparous cows produce more milk after being fed a prepartum diet with a negative DCAD irrespective of the occurrence of milk fever (Block, 1984; Joyce et al., 1997; DeGroot, 2004; Penner et al., 2008; Siciliano-Jones et al., 2008). It is not clear if this increase in production performance is directly related to improving blood calcium at calving or indirectly through reduction of other clinical and subclinical postpartum diseases. Estimated increases in milk yield when cows are fed prepartum diets with a negative DCAD range from 1,800 lb (Degaris et al., 2004; Degaris and Lean, 2008) to 3,200 lb (DeGroot, 2004).

Controlled experiments have not yet determined the optimal prepartum DCAD. The recommended target DCAD of -10 to -15 meq/100g dietary DM may be lower than needed to achieve the desired changes in acid-base status and subsequent increases in blood Ca. However, this range of DCAD provides a margin of safety to account for varying concentrations of the minerals in feeds and K consumed from pasture or free-choice hay.

To illustrate the point of varying mineral contents of forages and to stress the need for routine forage mineral analyses by wet chemistry, Arm & Hammer Animal Nutrition has been conducting a DCAD forage testing program over the past few years. The results are shown in Table 2.

Table 2 shows that DCAD and individual minerals can and do have large variation. Therefore, the chances for DCAD remaining negative may not be achievable if you balance it at, near, or just below zero. A DCAD of -10 to -15 meq/100g dietary DM would be more desirable to ensure that all cows always receive the low DCAD diet.

## **DCAD and the Post Fresh Transition Cow**

As production increases, so does the acid load in the cow. With insufficient blood buffering capacity the cow will not be allowed to fully express her production potential and will reduce her DMI and production accordingly. It makes sense, therefore, that cows require more blood buffering capacity if we expect high DMI and productivity.

White et al. (2008) utilized cows in their first 12 wk of lactation to investigate the effect of increasing DCAD. Table 3 shows the nutrient composition of the experimental diets used in this trial. The control diet had the addition of DCAD Plus<sup>®</sup>, which is a feed grade potassium carbonate product (Church & Dwight Co., Inc., Princeton, NJ) to increase dietary K from 1.2 to 2 % of the total DM and to increase DCAD from 22 to 44 meq/100g DM. All other nutrients were held fairly constant between the 2 diets.

Table 4 shows the performance parameters and statistical analyses for this trial. With the exception of milk protein, all performance parameters were significantly and positively affected by increasing DCAD using DCAD Plus<sup>®</sup>. Body weight was not affected by treatment and, although DMI was numerically increased by 1.32 lb/d (600 g/d), this was not significantly different from cows offered the control diet.

Other interesting observations in Table 4 are the milk production efficiency values. While gross efficiency (kg milk/kg DMI) only tended ( $P = .09$ ) to be improved with higher DCAD, the efficiencies based on fat corrected milk (**FCM**) and energy corrected

**Table 2.** Results from 2008 Arm & Hammer Animal Nutrition forage testing program for the Northeastern and North Central United States – DCAD variation.

NORTHEAST (ME, VT, CT, NY, PA, VA, MD, OH)					
SAMPLE TYPE	n	Average DCAD	MIN	MAX	STDEV
Legume Haylage	28	24.24	-5.28	63.30	17.72
Grass Haylage	4	33.16	13.48	42.93	13.34
Balage	10	29.13	9.50	52.93	12.73
Corn Silage	47	24.84	-13.93	77.54	19.77
Small Grain Silage	8	42.45	18.44	76.27	21.36
Legume Hay	18	22.82	8.66	49.13	11.13
Grass Hay	18	31.11	0.61	51.96	16.76
NORTHCENTRAL (MI, WI, IL, IN, IA, NE)					
SAMPLE TYPE	n	Average DCAD	MIN	MAX	STDEV
Legume Haylage	166	35.54	-9.21	82.62	18.37
Grass Haylage	5	46.74	8.73	105.55	36.52
Corn Silage	85	12.26	-0.21	46.94	8.53
Small Grain Silage	17	35.10	4.24	63.43	14.19
Legume Hay	75	35.76	2.96	97.52	19.27
Grass Hay	67	27.08	-9.50	119.28	20.52
Straw	7	14.03	-3.25	33.11	11.06

**Table 3.** Nutrient content of experimental diet fed to cows for their first 12 wk of lactation in a trial conducted by White et al. (2008).

Nutrient	CONTROL	DCAD+
	Composition (dry basis)	
Dry Matter (%)	60.1	59.4
Crude Protein (%)	16.1	16.1
Soluble Protein (% of CP)	34.1	31.1
ADF (%)	19.8	19.3
NDF (%)	35	34.7
Calcium (%)	0.76	0.76
Phosphorus (%)	0.4	0.39
Magnesium (%)	0.4	0.4
Potassium (%)	1.21	2.05
Sulfur (%)	0.26	0.25
Sodium (%)	0.51	0.51
Chloride (%)	0.54	0.54
DCAD (meq/100g)	21.52	43.68

**Table 4.** Body weight (BW), dry matter intake (DMI), and milk and milk component production parameters when low and high DCAD diets were fed to cows in their first 12 wk of lactation (White et al., 2008).

Item	Treatment		trt	wk	p< trt x wk	Linear	Quadratic		
	Control	DCAD +							
BW, kg	669 ± 20	674 ± 20	0.49	0.002	0.93	0.18	0.28		
DMI, kg/d	26.2 ± 0.9	26.8 ± 0.9	0.20	<0.0001	0.09	0.26	0.004		
Milk, kg/d	39.3 ± 1.4	40.8 ± 1.4	0.01	0.01	0.46	0.48	0.01		
ECM, kg/d	41.3 ± 1.4	44.3 ± 1.5	0.00	0.24	0.72	0.55	0.45		
3.5% FCM, kg/d	42.2 ± 1.6	46.1 ± 1.6	<0.0001	0.09	0.70	0.61	0.51		
Fat, kg/d	1.55 ± 0.06	1.75 ± 0.06	<0.0001	0.03	0.37	0.50	0.75		
True protein, kg/d	1.16 ± 0.03	1.14 ± 0.03	0.38	0.12	0.78	0.76	0.06		
Milk composition									
Fat, %	3.96 ± 0.09	4.31 ± 0.09	<0.0001	<0.0001	0.27	0.47	0.42		
True Protein, %	2.97 ± 0.04	2.79 ± 0.04	<0.0001	<0.0001	0.91	0.36	0.09		
Casein, %	2.42 ± 0.04	2.30 ± 0.04	<0.001	<0.001	0.57	0.49	0.01		
Lactose, %	4.85 ± 0.05	4.92 ± 0.05	0.04	<0.0001	0.71	0.06	0.35		
Solids, %	12.60 ± 0.14	12.79 ± 0.14	0.02	<0.0001	0.38	0.32	0.69		
SNF, %	8.32 ± 0.08	8.18 ± 0.08	<0.0001	0.0001	0.40	0.03	0.04		
MUN, mg/dl	10.38 ± 0.25	10.62 ± 0.25	0.21	<0.0001	0.30	0.15	0.62		
SCC, 1,000/ml	23.1 ± 113.7	265.4 ± 113.4	0.83	0.40	0.63	0.12	0.44		
Feed Efficiency,									
kg milk/kg DMI	1.53	0.03	1.57	0.03	0.09	<0.0001	0.63	0.45	0.12
Kg FCM/kgDMI	1.61	0.02	1.72	0.02	<.0001	<.0001	0.72	0.62	0.26
Kg ECM/kgDMI	1.58	0.03	1.65	0.03	<.001	<.0001	0.68	0.52	0.31

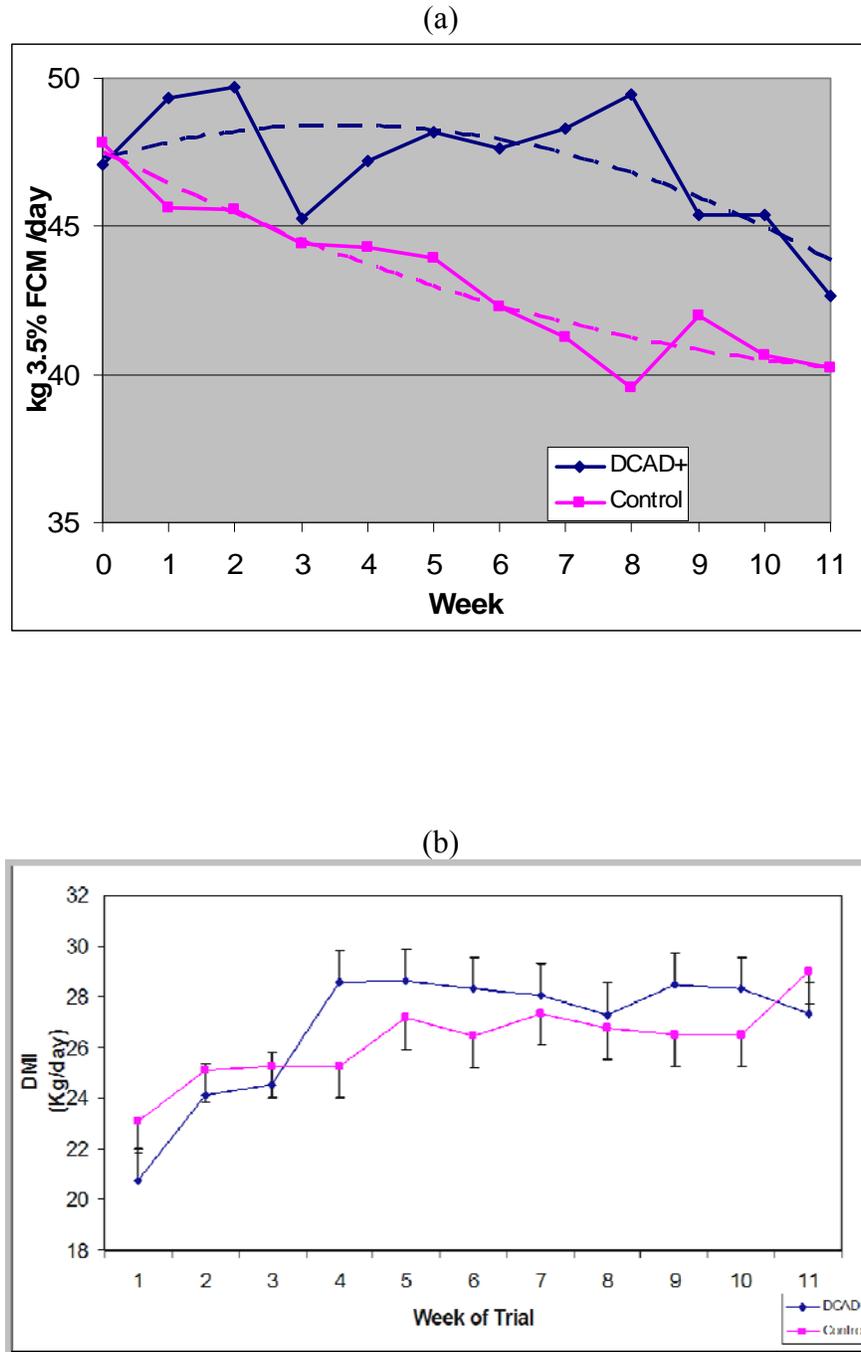
milk (ECM) were significantly ( $P < .001$ ) improved with higher DCAD. There was a 7 % increase in FCM efficiency and 4.5 % increase in ECM efficiency shown in this trial by simply increasing DCAD. This indicates that the utilization of nutrients improved in an environment that was well buffered and/or not stressed by a high acid load.

The temporal patterns for FCM production and DMI are shown in Figures 2a and 2b. Cows began their lactation at very similar FCM production levels, but quickly separated according to treatment with the high DCAD group outperforming control cows consistently throughout the trial. Although DMI was not significantly affected by DCAD, Figure 2b does show a strong tendency for the cows fed the higher DCAD to have higher DMI during most of the trial.

## CONCLUSIONS AND TAKE HOME MESSAGES

While we feel that we have a grasp of a transition cow's needs, it is not clear how we should meet those needs. Obviously there are many different successful manners to feed the transition cow. While these appear divergent, there are several commonalities that, when combined with the discussion above, lead to the following messages:

1. Protein Needs
  - a. Protein should be evaluated on a metabolizable protein (MP) basis. Parturient transition cows need about 1100-1200 g/d of MP, while first and second lactation animals need 1200-1400 g/d. Underfeeding will result in low start-up milk and reduced peaks, as well as compromising the immune system.



**Figure 2.** Temporal pattern of 3.5 % fat corrected milk (FCM) production (a) and DMI (b) when cows were fed a Control diet with a DCAD of 22 meq/100 g DM or a diet with a DCAD of 44 meq/100 g DM (DCAD Plus<sup>®</sup>) (White et al., 2008).

- b. Diets that are low in fermentable carbohydrate (starch) will need a significant amount of rumen bypass MP with a good amino acid profile.
2. Carbohydrates
- a. To mitigate the reduction in DMI in the prepartum transition period, starch levels should be kept at or below 22 %. The lower the starch the more critical will be the bypass MP due to reduced rumen microbial fermentation.
  - b. Postfresh cows should not be fed starch levels that would be more normal later in the early lactation phase (25+ %) because rumen rate of passage is slower and there is more chance for acidosis. Keep starch below 25 % and total NFC in the range of 35 – 38 %.

3. Fat

- a. Since there is no specific need to increase energy density prepartum by using fat instead of carbohydrate, but there appears to be a metabolic benefit to adding unsaturated fatty acids to the intestine, some amount of fat appears warranted. A source of rumen inert or bypass unsaturated fatty acids, not exceeding ¼ lb/cow/d will help with immune function and overall energy metabolism postpartum.
- b. In the post-fresh period, research shows that again, these unsaturated fatty acids in the intestine can improve immune

and reproductive function. However, it is still not ideal to replace carbohydrates at this point with a lot of fat. Keep levels to ¾ lb/cow/day and be careful of rumen active fats in oilseeds and corn byproducts so as not to elicit a milk fat depression.

4. Minerals

- a. Dietary magnesium (Mg) should be maximized in the pre- and post-fresh rations to 0.4 % of the DM.
- b. Prefresh calcium (Ca) should be lower than typical (less than 0.6 % if possible) if managing for a 21-day pre-fresh transition period and increased to 0.8 to 1 % post-fresh.
- c. DCAD should be less than zero pre-fresh (-10 to -15 meq/100 g DM if forages are variable in potassium levels).
- d. DCAD should be highly positive post-fresh, which can be achieved by:
  - i. Keeping Cl and S to minimal levels,
  - ii. Removing excess salt (sodium chloride),
  - iii. Maximizing sodium (Na) buffers to .5 lb (or more) per cow per day (dietary Na should not exceed 0.8 % of the diet), and
  - iv. Increasing dietary potassium (K) without adding KCl or K<sub>2</sub>SO<sub>4</sub> to reach the desired DCAD of 35-45 meq/100g DM.

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