Special Nutritional Needs of the Transition Cow

Drs. Elliot Block and William K. Sanchez
Church & Dwight Co., Inc.

INTRODUCTION

The transition period for a dairy cow begins two to three weeks prepartum and continues until two to three weeks 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 clinical and subclinical postpartum diseases that can significantly affect productivity and 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 their importance in the productivity and health of cows. The problem research scientists face is an inability to gather enough cows of similar productivity characteristics in one place to conduct the research necessary to enhance the understanding of this animal. Consequently, animals outside this short time frame have been used in an attempt to simulate the conditions that exist in the true transition cow. This method has expanded the 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). 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 and profitability for the remainder of the ensuing lactation.

What follows is a review of the biology of the transition cow, and the phenomena that mark this period, with the goal of developing these nutritional strategies.

BIOLOGY OF THE TRANSITION COW

Dry Matter Intake

It is now well established that dry matter intake (**DMI**) decreases as calving approaches. DMI 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 day 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 increases at the rate of 1.5 to 2.5 kg per week (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 DMI decrease prepartum and the increase postpartum 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, hormonal and other physiological factors have the most important impact on this phenomenon (Grant and Albright, 1995; Robinson, 1997).

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, while 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. In addition, 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). An increase in concentration of plasma non-esterified fatty acids (**NEFA**) results from this mobilization of lipids, which increases gradually in the prepartum transition period but rapidly in the last 3 days of gestation. A portion of this increase in NEFA is obligatory and is under hormonal control, while another portion of this increase in NEFA 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 livers, 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 days in milk (**DIM**) to consume 22 kg of DM/day, of which more than half is in the form of concentrates, without posing any particular problems to the cow, provided the diet is well balanced. However, this same diet consumed by a fresh cow can cause severe ruminal acidosis. The major difference in rumen function between these two 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 two 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.

The second factor has to do with the length and number of rumen papillae. These papillae are the absorptive surface in the rumen. They primarily absorb the volatile fatty acids (**VFA**) generated during normal fermentative processes. These papillae can decrease in size by half during the dry period by reason of the low dietary NFC (Dirksen et al., 1985). 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 three principal axes:

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

In the next sections, some of the etiology and preventive measures for these problems and the nutritional considerations related to these three axes are discussed. It is necessary to mention here that these disorders are interrelated and 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 **(DA)** 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 DAs. Generally, this analysis suggests that prevention of one problem can decrease the incidence of others.

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 drives 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, a review of the literature related to specific nutrients studied in this period of the cow's production cycle follows.

Energy

Requirements and the Negative Energy Balance (NEB)

The energetic demands of gestating cows reach 1.3 to 1.5 times 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).

During both the prepartum and postpartum transition period, cows require more energy than they can consume, resulting in the NEB and the concomitant loss of body weight (condition) to supply the necessary energy. 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 days after the nadir in NEB. Energy during the transition period, therefore, has a major impact on cow performance and longevity.

Body Condition Score (BCS) at Calving

It is widely accepted that high producing cows at the beginning of lactation lose BCS due to the NEB resulting from a low but increasing DMI that provides less energy than required for production. It is not until several weeks post peak production that DMI reaches it's maximum and cows can truly regain the lost BCS (Garnsworthy, 1989). It appears logical to conclude that body energy reserves in the form of adipose have an influence on production, reproduction and health of cows at the beginning of lactation.

Many studies have demonstrated the relationship between BCS at calving and DMI, productivity, reproductive capacity and health of cows at the beginning of lactation. The BCS represents the cumulative effects of BCS at drying off, of the dry period itself and the weight changes that occur during this period (Markusfeld et al., 1997). Most recommendations are to have cows attain a BCS between 3.25 and 3.75 by the time they go dry and to maintain that score during the dry period.

DMI

A significant reduction in postpartum DMI is often observed as BCS at calving increases (Garnsworthy and Topps, 1982; Garnsworthy and Jones, 1987; Broster and Broster, 1998). Moreover, cows having high BCS at calving take a longer time postpartum to attain their maximum DMI (Garnsworthy and Topps, 1982). A negative correlation exists between BCS prepartum and DMI postpartum (Grummer, 1995).

This slow increase in postpartum DMI for obese cows negatively impacts energy balance, in that cows calving with a BCS exceeding recommendations lose more weight postpartum than cows calving at optimal BCS. Further, obese cows are more susceptible to metabolic disorders in the transition period (Boisclair et al., 1986; Ruegg and Milton, 1995; Putnam et al., 1997).

Milk Production

The relationship between BCS at calving and milk production is less clear. Most studies have not shown a correlation between BCS at calving and quantity of milk produced (Garnsworthy and Topps, 1982; Boisclair et al.,1986; Ruegg and Milton, 1995). Nonetheless, some reports show a positive relation between the two (Land and Leaver, 1980; Markusfeld et al., 1997). A recent compilation of studies by Broster and Broster (1998) shows 2 to 4 kg/day more milk was obtained when BCS at calving was 3.25 compared to cows with a BCS at calving of less than 2.0. The results indicate that this effect can only be observed when the difference between the two BCS groups is large, which explains the confusion in the literature with smaller sized trials. Waltner et al. (1993) took a different approach to evaluating their data. They observed a quadratic response of BCS at calving and cumulative production to 90 days postpartum of 322 kg of milk when BCS at calving increased from 2 to 3 but only a 33 kg response when BCS at calving increased from 3 to 4; a decrease in production to 90 days was observed when the value was above 4.

Compared to cows with low BCS at calving, cows with a high score tend to produce milk with more fat at the beginning of lactation (Holter et al., 1990; Markusfeld et al., 1997). This is likely due to a combination of their reduced milk volume and the greater amount of mobilized body fat (due to reduced DMI) used directly for milk fat synthesis. The overall result is that milk fat production is not necessarily changed (Holter et al., 1990).

Milk protein does not appear to be affected by BCS at calving. There is not unanimity on this subject but the results indicate a minor decrease in milk protein concentration in cows with excessive condition at calving (Broster and Broster, 1998). According to the same researchers, the negative relation between BCS at calving and milk protein content are more pronounced in cows producing milk with high protein concentrations. This phenomenon is likely related to the reduced DMI in overconditioned cows.

Reproduction

The effect of higher than recommended BCS at calving on reproduction is reduced fertility with a delayed first estrus and more breedings per conception (Garnsworthy and Topps, 1982; Garnsworthy and Jones, 1987; Ruegg et al., 1992; Markusfeld et al., 1997; Heuer et al., 1999).

Energy balance and BCS have a positive relationship with the concentration of plasma progesterone (Macmillan et al., 1996). This helps explain the relationships between BCS, DMI and NEB with reproduction.

Health

Studies have demonstrated that cows in higher than recommended BCS at calving are more susceptible to ketosis (Fronk et al., 1980; Boisclair et al., 1987; Dyk and Emery, 1996), fatty liver syndrome (Treacher et al., 1981; Rukkwamsuk et al., 1999a), milk fever (Fronk et al., 1980; Heuer et al., 1999), mastitis (Fronk et al., 1980), DAs (Dyk and Emery, 1996; Cameron et al., 1998), and locomotion problems (Gearhart et al., 1990).

Regulation of Energy Metabolism and Glucose Homeostasis

Glucose is a substance that plays a fundamental role in all living beings. In the last weeks 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 per day 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 reestablishment 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 weeks 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 disequilibrated 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 DA. Ruminal acidosis is also a frequent problem for cows at the beginning of lactation because of highly fermentable rations and insufficient rumen adaptation 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 three 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; or 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 β 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 two values are termed subclinical ketosis (Drackley, 1997). Duffield (1997) established that when BHB reaches 1400 μmol/l (14.6 mg/dl) in blood, the incidence of metabolic disorders increases and when the value is above 2000 μmol/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. (1999b) 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 days 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 days after calving while the normal cows liver fat increased 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 post- partum transition period by minimizing the magnitude of the NEB. The weight loss, fat mobilization, NEB, increases in plasma NEFA and liver fat accumulation is inevitable in all high-producing cows. However, it is imperative to minimize these. 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 must be made.

Rumen acidosis

When acid production in the rumen resulting from fermentation of organic matter exceeds the capacity for the animal to neutralize or absorb these acids, 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) daily.

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 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 (Yokoyoma and Johnson, 1988; Nordlund, 1994; Allen and Beede, 1996; Hall, 1999).

Based on the above discussion it appears prudent to begin to:

1. Increase NFC in the prepartum transition period to adapt the rumen wall to VFA,
2. Feed as frequently as possible postpartum or to offer TMR to reduce acid accumulation at any time during the day,
3. Verify that the proper amounts of effective fiber are being fed (and consumed), and
4. Ensure 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 post- partum diets. Including rumen buffers in the postpartum diet can also serve the cow well. Table 1 gives some guidelines on ration specifications.

Table 1: Guidelines for nutrient specifications in the total ration dry matter for transition cows.

Nutrient	Transition prepartum	Transition postpartum
DMI (% Body weight)	1.7	2.5 - 3.0
NDF intake (% Body weight)	0.7 - 0.9	0.8 - 0.9
NE (Mcal/kg)	1.45 - 1.55	1.62 - 1.67
NFC (%)	33 - 35	35 - 38
Total fat (%)	3 - 5	4 - 6
Added fat (%)	0 - 1.5	0 - 3
NDF (%)	32 - 36	29 - 33
ADF (%)	25 - 29	20 - 21
Crude protein (%)	14 - 15	18 - 19
Undegradable protein (% of CP)	33 - 35	35 - 38
Degradable protein intake (%)	9 - 10	10 - 12
Ca (%)	1.2 - 1.5	0.8 - 1.1
P (%)	0.32 - 0.4	0.4 - 0.45
Mg (%)	0.32 -0.35	0.33 - 0.5*
Na (%)	0.1 - 0.2	>0.3
K (%)	<1.6	>1.5
Cl (%)	0.5 - 0.7	0.25
S (%)	0.2 - 0.45	0.25
DCAD (meq/100g DM	0 - (-15)	>35
Zn (mg/kg)	45 - 55	45 - 55
Mn (mg/kg)	45 - 55	40 - 50
Cu (mg/kg)	12 - 14	12 - 14
Se (mg/kg)	0.3	0.3
Vitamin A (1000IU/day)	100 - 150	120 - 200
Vitamin D (1000IU/day)	24 - 40	30 - 50
Vitamin E (1000IU/day)	1000	600 - 800

*Mg should be adjusted according to the level of dietary K such that there is a 4-to-1 ratio between K and Mg.

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 post- partum) 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. As previously mentioned, besides the added benefit of energy density, rumen papillary development is also favored (Dirksen et al., 1985). Furthermore, NFC fermentation produces 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 NEFAs 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 Kronfeld (1982) speculated 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), which 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, 1999). There are a variety of factors to 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.

Discussion of each of these and a synthesized strategy follows.

Experimental models used. 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. For example, thin and obese dry cows have different dynamics in fat metabolism, DMI patterns and nutrient needs both pre- and post-partum. Cows in these different conditions react differently to supplemental fat (or other nutrients). High producing cows postpartum react

differently to supplemental nutrients in the pre- and post-partum 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 and results should be viewed with caution. An imposed feed restriction of a given percentage is not representative of a transition cow, which declines in DMI gradually in the first phases and dramatically as calving becomes more eminent. Furthermore, cows of different body condition scores and parities have different degrees of 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.

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 per day. Some have fed as much as 1.5 lbs./day. 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 one pound 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; Martinet and Houdebine, 1993). There is not only lipolysis of adipose but also mobilization of stored 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, NEFAs 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 NEFAs 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. Quantities of less than 1/2 lb. per day have a good chance of being more beneficial than no fat or more than 1/2 lb. of fat in terms of optimizing lipoprotein use by tissues and reducing adipose lipolysis. Moreover, one pound of fat per day in a diet when DMI is

severely restricted results in a very high concentration of dietary fat especially in relation to other dietary nutrients.

Fatty acid profile of the fat. 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 it's 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 the unsaturated fatty acids are the more desirable in this case (Leplaix-Charlat et al.,1996a,b).

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 less than 1/2 lb./day of rumen protected fat supplements with a content of long chain mono- and poly-unsaturated fatty acids, had more positive effects when fed prepartum (Douglas et al., 1998).

The subject of feeding fat in the postpartum transition phase also makes ultimate sense from the standpoint of energy balance, 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 *high*. This makes sense in that once energy demands are met or exceeded the cow reduces DMI to compensate for an oversupply of nutrient. When the data is examined for recommended inclusion rates of dietary fat from high producing cows, there is no DMI depression.

Because of the way in which dietary fats are digested (as explained above), the rate of fat mobilization from adipose should be reduced as intestinally absorbed lipoproteins can be used directly by tissues (specifically the mammary glands in this case), thus sparing the need for mobilizing body condition.

We believe that there are sound reasons for offering dietary fat to transition cows. The case becomes stronger for rumen protected long chain mono- and poly- unsaturated fatty acids plus palmitate. These fatty acids have the highest intestinal availability compared with other long and short chain saturated fatty acids (Firkins, 1994; Pantoja et al., 1995). A source of rumen protected methionine available in the intestine should assist in lipoprotein production by the liver by correcting a methionine imbalance or deficiency. The recommended quantities to optimize health, production and reproduction are 1/4 to 1/2 lb. per cow per day in the prepartum transition period and 1/2 to 1 lb. per cow per day in the postpartum transition period and into lactation until body condition score objectives are reestablished. Of course careful attention must be paid to the rest of the diet in terms of NFC, NSC, peNDF, UIP, DIP, amino acids, DCAD (see below), buffering, etc. to optimize the effects and utilization of all nutrients. The ultimate goal is to make the *easiest transition* as possible from the dry state through parturition and the onset of lactation.

Protein and amino acids

Bell (1995) has 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 in terms of BCS (Van Saun et al., 1993) and milk protein production postpartum (Van Saun et al., 1993; Moorby et al., 1996). However, other studies had not detected a benefit above the NRC (1989) recommendation (Wu et al., 1997; Carson et al., 1998; Van deHaar et al., 1999). While primiparous cows may require a higher dietary

protein concentration of 14 to 16% of total DM, dietary protein requirements for multiparous cows of 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) guideline. However, use of non-protein nitrogen sources should be avoided (VandeHaar and Donkin, 1999; Schwab, 1998).

It has been recommended, contrary to NRC (1998), to increase the crude protein 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 greater 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 disequilibration 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% crude protein and 35 to 40% UIP (Nocek, 1995; NRC, 1998). Lysine and methionine are recognized as the two 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. 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).

Dietary Cation-Anion Difference (DCAD)

The reader is directed to two papers published at last year's Mid-South Conference (Sanchez, 1999a,b) for a complete description of the DCAD theory and research results in pre- and post- partum cows. For this paper we will only highlight the issues with DCAD that were described by Sanchez (1999a,b).

The term DCAD refers to the numerical difference between the sum of certain dietary cations (positively charged minerals) and certain dietary anions. Primarily, the cations to consider are sodium (**Na**) and potassium (**K**) while the anions are chloride (**Cl**) and sulfur (**S**). Various equations have been suggested but the two primary ones are [(Na + K) - (Cl + S)] and (Na + K - Cl) expressed as milliequivalents (**meq**) per 100g DM (see Sanchez, 1999a,b). When the value is negative (i. e., more anions than cations) there is a depletion of blood buffering capacity and a mild metabolic acidosis ensues. As DCAD becomes more positive, blood-buffering capacity also increases.

Reducing DCAD in the prepartum transition period dramatically reduces the risk for milk fever and subclinical hypocalcemia by improving calcium dynamics for the cow. The reduction of these diseases reduces the risk factors associated with DA, retained placenta and ketosis postpartum and has been shown to increase DMI postpartum.

Postpartum, the cow is faced with high quantities of metabolic acids, which, if not buffered should lead to reduced performance, DMI and

increased risks of other diseases including laminitis. Elevating DCAD to 35 to 40 meq/100g DM has been shown beneficial to all production parameters. Furthermore, elevating DCAD above 40 using the combination of Na and K sources (without Cl or S) has been shown to be more beneficial for production from early-lactation cows than using either Na or K alone. Data was also introduced by Sanchez (1999a,b) that showed elevated DCAD in heat-stress and reducing Cl to minimum requirements can improve production and DMI.

The only new information to add to Sanchez (1999a,b) is two field trials that were recently completed by Church & Dwight Co. In the first trial dietary K was held constant at 1.3% of total DM but DCAD was increased by reducing Cl in one diet (i.e., substitution of KCl by K_2CO_3). The DCAD comparison was 19 vs. 25 meq/100g DM. Sodium and K concentrations in both diets were equivalent at 0.4 and 1.3% DM, respectively, and Cl was 0.66 and 0.39% DM for the 19 and 25 meq DCAD diets, respectively. Fifty cows between calving and 200 DIM per treatment were followed for four DHI tests. Statistical analysis of production data showed an increase ($P < .10$) in milk yield by 2.98 lbs./d (86.49 vs. 89.47 lbs./d) by the cows fed the higher DCAD. Feed intake could not be evaluated. In a second field trial 70 cows per treatment between calving and 200 DIM were fed diets with a low (38 meq/100g DM) or high (43 meq/100g DM) DCAD adjusted by the simple addition of K_2CO_3 to one of the diets. Sodium, K and Cl concentrations in the diets were 0.48, 1.52 and 0.26% for the low DCAD diet and 0.43, 1.8, and 0.26% for the higher DCAD diet. Again, the cows fed the higher DCAD produced 2.4 lbs./d more ($P<.05$) fat corrected milk (109.4 vs. 111.8 lbs./d) compared to cows fed the lower DCAD.

CONCLUDING COMMENTS

There are certainly other dietary factors that appear promising to aid the transition cow. Direct microbial supplements, yeast, choline and certain B-vitamins are being investigated. Unfortunately, it is beyond the scope of this paper to consider these individually.

We hope that the above discussion emphasized the importance of the transition period in the cow's production cycle. It may not be too bold to state that these are the most important six weeks in determining lactation performance, veterinary and early culling costs and longevity in the herd.

There is much speculation and theorizing in the preceding pages, however, it is all well-founded in scientific fact and argument. Much debate will continue on this subject until more controlled studies using appropriate animal models and realistic diets are conducted and published. Until then, the guidelines presented in Table 1 summarize many of the points discussed.

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