Transition Cow Management to Reduce Metabolic Diseases and Improve Reproductive Management[1]

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Introduction

The dairy industry in North America has changed dramatically with a trend in direction of larger herds and higher production per cow, which seems to be associated with a decrease in fertility, especially a decrease in conception rates and extension of calving intervals (Butler, 2000; Lucy, 2001; Stevenson, 2001). This decline in fertility of lactating dairy cows is observed not only in North America, but also in the United Kingdom (Royal et al., 2000) and Israel (Zeron et al., 2001). Fertility measures of lactating dairy cows have reached at an all time low in 1999 in the US, with services per conception of 3.0 in 143 dairy herds monitored by the Raleigh DHIA record system (Lucy, 2001). This is similar to results reported by Overton (2002) in California with over 100,000 cows in approximately 100 dairy herds in which conception rates averaged 30.8%.

The physiological and environmental stress of high producing dairy cows negatively affects estrus detection and conception. Early postpartum, lactating dairy cows undergo a period of negative energy balance (Butler, 2000; Butler and Smith, 1989). Negative energy balance results in extension of the period of postpartum anovulation. Prolonged periods of postpartum anovulation associated with negative energy balance in lactating dairy cows is inversely related to subsequent fertility (Butler, 2000).

Despite the increased knowledge of the reproductive biology of the lactating dairy in recent years, reproductive efficiency continues to decline in dairy herds. In the high producing dairy cow, the extended period of negative energy balance, the altered competence of follicles, the lower circulating levels of estradiol during proestrus, the lower circulating levels of progesterone during diestrus, the reduced fertilization rate, and the suboptimal cross-talk between conceptus and uterus are associated with reduced fertility.

Part of the decline in fertility in high producing dairy cows is associated with the higher incidence of metabolic disorders early postpartum. Cows affected by retained fetal membranes, metritis, hypocalcemia, ketosis, mastitis and other diseases have an increased risk for delayed conception (Gröhn and Rajala-Schultz, 2000). Also, prevalence of endometritis is still is high at 50 d postpartum and it has marked negative effects on fertility (Gilbert et al., 1998). Therefore, low fertility in lactating dairy cows results from a complex array of events and interactions between energy balance, ovarian function and uterine and overall health, which set the stage for subsequent reproductive performance.

Improvements in fertility in lactating dairy cows can be achieved by feeding management during the transition period to reduce the incidence of metabolic disorder that might directly or indirectly impact reproductive function in cows.

Nutrition of transition cows

Literature linking the effects of prepartum nutrition and subsequent fertility is scarce. Most of what is suggested to optimize future fertility is related to relationships between metabolic disorders and risk for delayed conception. Common metabolic problems that affect early postpartum cows such as retained fetal membranes, milk fever, ketosis, and displaced abomasum are know to extend the period of negative energy balance and delay resumption of ovarian cycles.

Energy

Manipulation of the energy content of the diet prepartum has been shown to affect dry matter intake (Hayirli et al., 2002) and postpartum lactational performance. Cows fed high fermentable energy diets prepartum have improved energy balance, reduced concentrations of plasma nonesterified fatty acids and ß-hydroxybutyrate, and reduced triacylglycerol infiltration in the hepatic tissue.

Hepatic lipidosis early postpartum has been linked with an extension in the postpartum anovulatory period (Reist et al., 2000) and reduced reproductive performance in lactating dairy cows (Jorritsma et al., 2000). It is difficult to determine whether the negative effect of triacylglycerol infiltration in the hepatic tissue on postpartum resumption of ovarian cycles is direct, or just a consequence of negative energy balance. Nevertheless, diets that minimize the risk for hepatic lipidosis and ketosis are expected to improve postpartum energy balance and resumption of cyclicity.

Minor et al. (1998) fed 50 multiparous and 25 primigravid Holstein cows one of five diets that consisted of a standard nonfibous carbohydrate diet beginning at 19 d prepartum, a high nonfibrous carbohydrate diet beginning at 19 d prepartum, a standard nonfibrous carbohydrate diet plus 12 g/d of niacin beginning at 19 d prepartum, a high nonfibrous carbohydrate diet plus 12 g/d of niacin beginning at 19 d prepartum, and a standard nonfibrous carbohydrate diet beginning at 19 d prepartum plus niacin beginning at 14 d postpartum. Treatments were applied during the first 40 weeks of lactation. Feeding a high nonfirbrous carbohydrate diet prepartum increased dry matter and energy intake, which improved energy balance of cows throughout the transition period. Plasma glucose concentrations tended to be higher, and nonesterified fatty acids and ß-hydroxybutyrate concentrations were lower for cows fed the high nonfibrous carbohydrate diet. Because of the positive effects on lipid metabolism, diets high in nonfibrous carbohydrates increased concentrations of liver glycogen and reduced concentrations of hepatic triacylglycerol. The positive effects of high fermentable energy diets fed prepartum are expected to minimize the incidence of subclinical ketosis and hepatic lipidosis, which might have positive effects on postpartum fertility.

Although diets high in nonfibrous carbohydrates improve energy status of transition cows, provision of adequate amounts of physically effective NDF is important to maintain rumen fill, thereby helping in the prevention of displacement of abomasum. Cows that develop displacement of abomasum have delayed postpartum insemination (Raizman and Santos, 2002), which might decrease reproductive efficiency. Diets with a net energy for lactation greater than 1.65 Mcal/kg were associated with increased risk for displacement of abomasum (Cameron et al., 1999).

Little scientific information is available for the fiber requirements of dry cows to maintain adequate rumen health and minimize the incidence of displacement of abomasum. Mertens (1997) created a system to determine the effectiveness of NDF to stimulate rumination and to maintain milk fat concentrations. According to his data, fiber sources are classified according to their ability to stimulate rumination, which was directly related to the size of particle and suggested that lactating Holstein cows should receive diets containing at least 21% physically effective NDF in order to maintain milk fat content above 3.5%.

Most prepartum diets contain between 30 to 40% NDF and manipulation of the fiber content of the ration should be made to accommodate the needs of the specific herd. In farms with aggressive fresh cow programs, incidence of displacement of abomasum is below 2% of the total cows calving per year, and these herds can feed prepartum diets with less than 35% NDF, therefore higher energy diets.

Fat feeding during the transition period and reproduction

Fat feeding during late gestation and the first 3 weeks of lactation has usually not been recommended because it can compromise feed intake without any clear positive effect on postpartum lactation performance. This negative effect on intake is more pronounced in primiparous than multiparous cows (Hayirli et al., 2002). However, recent evidence suggests a positive effect of fat feeding during late gestation and early lactation on subsequent reproductive performance. A study at Cornell University (Frajblat, 2000) observed that feeding of saturated fatty acids in a prilled form during late gestation improved reproductive performance of lactating Holstein cows. Cows were fed isoenergetic diets with or without supplemental fat during the last 21 d of gestation and no supplemental fat postpartum. Insemination started at 60 d postpartum, and survival analysis of days open indicated an improvement in pregnancy rates after approximately 110 d postpartum (Frajblat, 2000).

Positive effects of feeding fat on reproduction may occur through stimulation of ovarian follicular growth, improved luteal function, and hastening of uterine involution. The effects of feeding fat early in lactation on follicular population have been observed in several studies (Staples et al., 1998). Calcium salts of palm fatty acids were substituted for corn at 2.2% of diet dry matter, but energy density of the diet was not altered. Ultrasound scanning of the ovaries observed an increase in the number of medium follicles prior to d 25 postpartum. On d 25, estrous was synchronized and the number of small and large follicles increased for fat-supplemented cows (Lucy et al., 1991). Other authors (Beam and Butler, 1998; Beam and Butler, 1997) have observed similar effects of fat supplementation on follicular population of dairy cows. Increased number and size of dominant follicles due to the feeding of fat-supplemented diets on fertility has not been defined. When supplemental fat reduced DMI during early postpartum, size of the largest follicle did no influence ovulation rate (Beam and Butler, 1998). However, the same group observed in a previous study that follicles that ovulated in the first follicular wave postpartum were of greater diameter (Beam and Butler, 1997). Therefore, it is possible that increasing the number and the size of larger follicles by feeding fat can accelerate the interval from calving to first postpartum ovulation, which can be beneficial to fertility (Butler, 2000).

Increased size of the ovulatory follicle might result in larger corpora lutea, capable of maintaining higher circulating concentrations of progesterone (Vasconcelos et al., 2001). Therefore, it is plausible that fat feeding during the transition period might improve subsequent fertility due to its effects on follicle development and luteal function.

Although early resumption of ovarian cycles is important to fertility (Butler, 2000; Butler and Smith, 1989), early rise in progesterone prior to uterine involution might favor establishment of uterine infections and pyometra (Black et al., 1953). Progesterone creates a uterine environment that is inductive of establishment of infections that otherwise would be cleared from the uterus. Arcanobacterium pyogenes, a gram-positive bacterium, is the main agent of uterine infections after 2 weeks postpartum, and it is associated with the presence of an active CL and pyometra. Progesterone stimulates the closure of the cervix and reduces the influx of leukocytes to the uterine lumen, which favor the establishment of uterine infection and inflammation. A recent large field trial (LeBlanc et al., 2002a; LeBlanc et al., 2002b) with 1,865 cows in 27 dairy herds indicated a prevalence of postpartum endometritis after 3 weeks in lactation of 16.9%. Cows diagnosed with endometritis between 20 and 33 d had marked reduction in reproductive performance and were 1.7 times more likely to be culled from the herd for reproductive failure. More sensitive methods for diagnosis of endometritis such as uterine cytology have indicated an overall lactational incidence of up to 60% (Gilbert et al., 1998), and endometritis diagnosed by cytology after 40 d postpartum is associated with a decrease in PR and extended days open.

Recently, it was shown that cows not affected by endometritis have higher concentrations of plasma prostaglandin F_{2a} metabolite during the first 2 weeks postpartum (Seals et al., 2002). Fat feeding during early lactation results in increased secretion of endogenous prostaglandin F_{2a}. Intravenous infusion of soybean oil ($\cong$ 50% linoleic acid) increased the concentrations of linoleic acid and prostaglandin F_{2a} metabolite in plasma of postpartum beef heifers (Filley et al., 1999). In a subsequent study, the same group (Filley et al., 2000) demonstrated that feeding a protected fat source (9.5% linoleic acid) to postpartum primiparous beef cows increased concentrations of linoleic acid and uterine secretion of prostaglandin F_{2a} as measured by its metabolite concentration in plasma. Therefore, it is possible that increasing secretion of endogenous PGF_{2a} by feeding a diet with a protected source of linoleic acid during transition may hasten uterine involution and reduce incidence of endometritis, which in turn is expected to increase subsequent fertility.

Protein

Prior to the 2001 NRC publication, it was hypothesized (Van Saun and Sniffen, 1996) that dietary crude protein requirements of dry cows were above 12% and restrictions in protein supply in the diet would have negative impacts on subsequent lactation performance. Curtis et al. (1985) analyzed data from several dairy farms and concluded that cows that received diets with CP content higher than 11% during the last 3 weeks of gestation had a lower incidence of retained placenta and clinical ketosis. More recent information from several controlled studies indicates that supplying dietary protein above requirements has no positive impact on subsequent lactation. Current feeding practices of close-up dry cows utilize diets rich in fermentable energy (Grummer, 1998), which may have a dramatic impact on microbial protein flow to the duodenum. This will increase the supply of high quality protein (microbial protein) to the small intestine and will improve overall nitrogen utilization (NRC, 2001).

Although research has evaluated reproductive parameters when cows are fed diets differing in protein content, much of the published data originates from studies having nutritional rather than reproductive objectives. Data on the effects of prepartum protein feeding and subsequent reproductive performance are scarce. We compared the effects of two levels of crude protein fed during late gestation on the performance, blood metabolites, and ovarian activity of Holstein cows (Santos et al., 2001). One-hundred and six cows (42 primigravid and 64 multiparous) 32 d before calving were divided into two groups and fed diets containing moderate (12.7% CP, 36% rumen undegradable protein) or high (14.7% CP, 40% rumen undegradable protein) protein. Higher prepartum protein diet increased yields of milk and milk components during early lactation in primigravid cows, but had no effect on multiparous cows. Incidence of postpartum disorders and plasma concentrations of glucose, nonesterified fatty acids and ketones were unaffected by prepartum protein concentration. Interval from calving to first postpartum ovulation, complete uterine involution, first postpartum detected estrus, and first insemination were not altered by prepartum dietary protein concentrations. Conception rate at first insemination, proportion of pregnant cows at the end of the study, days open, and services per conception were similar for cows receiving prepartum diets differing in protein concentrations.

There is little indication in the published literature that supplying dietary protein in excess of requirements during late gestation would have biological effects that reflect in benefits in reproductive performance of lactating dairy cows. Grummer (1998) estimated the dietary crude protein concentration required to meet the protein requirements of most cows based on expected feed intake during the last 21 d of gestation (Figure 1). He suggested that prepartum diets with 12% CP may be adequate for multiparous cows, but he pointed out that primiparous cows may require an additional amount of protein to accommodate their growth and the lower DM intake relative to body weight. The NRC (2001) suggests that prepartum diets for multiparous cows contain 12% CP and for primigravid cows approximately 14% CP. These protein concentrations are expected to provide enough metabolizable protein in diets rich in fermentable energy to meet the cow's protein requirements.

Dietary cation anion balance (DCAB)

Balancing diets for the mineral profile is critical to prevent milk fever, hypomagnesemia, and concurrent diseases associated with mineral imbalances early in lactation. Cows that experience clinical or subclinical hypocalcemia are more likely to develop retained placenta, metritis, ketosis, displaced abomasum, and mastitis. A great deal of epidemiological evidence exist that links milk fever with increased incidence of retained placenta and endometritis. Hypocalcemia presumably prevents the uterine contractions that are necessary for expulsion of the placenta in those cases where the placenta is free from the caruncles. However, cows with retained placenta have stronger and more protracted uterine contractions than cows that shed the placenta normally (Horst et al., 1997). Chemotactic activity of leukocytes has an important role in the process of detaching and expelling the placenta. During transition, cows are immunossupressed, and nutritional factors might be involved in this process. Development of hypocalcemia is associated with higher plasma cortisol concentrations and reduced phagocytic activity of leukocytes during the puerperal period. Such changes in the immune system favor establishment of bacterial infections in the uterus. Therefore, the mechanism by which hypocalcemia affects retained placenta may be associated with poor uterine tone, reduced DMI and enhanced immunosuppression, which reduces leukocyte activity in the uterine lumen.

In dairy cows, milk fever is often considered a major determinant of reproductive performance in herds. Beede et al. (1991) demonstrated that prevention of clinical and subclinical hypocalcemia through dietary cation-anion balance improves reproductive performance of dairy cows (Table 1). Although dietary cation-anion balance is more associated with dietary levels of sodium, potassium, chloride and sulfur, both calcium and phosphorus can interfere with blood calcium concentrations during the periparturient period, which might affect milk fever incidence and reproductive performance of cows.

A technology that has been developed to manipulate the acid base status of transition cows to improve calcium metabolism is the feeding of acidogenic salts. These are salts whose strong anions are absorb to a greater extent than the cations in the salt, resulting in retention of H^+ to maintain electrical neutrality of cells. The resulting acidification from H^+ retention is thought to enhance tissue sensitivity to the parathyroid hormone by enhancing binding of the hormone to receptors in the bones, kidneys and digestive tract (Goff, 2000). These receptors, when activated, stimulate the activity of osteoclasts, which in turn promote bone resorption and increased blood ionized calcium.

In order to easy the utilization of acidogenic diets and target cows that will benefit from them, grouping cows prior to calving become important. Primigravid cows are capable of maintaining adequate plasma ionized calcium throughout the transition period. Despite feeding diets with different cation anion balance, Moore et al. (2000) demonstrated that primigravid cows did not benefit from lowering the DCAB by adding acidogenic salts. In fact, dry matter intake was suppressed in those cows. Therefore, prevention of hypocalcemia with acidogenic salts should target only multiparous cows, and primigravid and multiparous cows should then be grouped and fed separately during the prepartum period.

Successful acidogenic diets depend upon the initial DCAB of the basal diet. In many instances, high dietary potassium is the culprit for diets causing hypocalcemia. Forages such as alfalfa hay and silage, oat hay, wheat silage, and high fibrous by-products such as almond hulls and beet pulp with added molasses tend to be high in potassium. Such ingredients should be added to the diet at limited amounts, otherwise large quantities of acidogenic salts are required to lower the DCAB. Horst et al. (1997) suggested that the DCAB of the basal diet should be lower than 250 mEq/kg in order to avoid adding large quantities of acidogenic salts. When corn silage and low potassium hay is available, it is possible to formulate diets with only 2 to 3 equivalent of anions to prevent hypocalcemia. Corn silage is high in fermentable energy, has low crude protein, and usually contains less than 1.6% potassium. These characteristics make corn silage the ideal forage for prepartum cows. However, corn silage does not provide large feed particles that help to maintain rumen fill and prevent displacement of abomasum. In California and some other states, a niche market for low potassium, high chloride alfalfa hay has been created and these hays are marketed to close-up dry cows to add additional protein and provide physically effective NDF for adequate rumen fill, with only small increases in the DCAB.

Several steps should be followed when implementing the use of acidogenic diets. Chloride salts are unpalatable and when added in excess they depress feed intake. The starting point is to analyze the feed ingredients and select those with a low DCAB. Special attention should be placed to potassium and sodium content of the basal diet. A suggestion that has worked extremely well is to follow these simple steps:

1- formulate a basal diet with a DCAB below 150 mEq/kg;
2- adjust magnesium content (0.35 to 0.4%) by adding magnesium sulfate and magnesium oxide. Magnesium sulfate can be anhydrous or heptahydrated, so specify which of the two is to be utilized;
3- adjust the sulfur content (0.35 to 0.4%) with calcium sulfate;
4- adjust the phosphorus content (0.35 to 0.4%) with Biophos or dicalcium phosphate;
5- adjust the calcium content (1.0 to 1.1%) with limestone; and
6- lower the DCAB to -50 to -100 mEq/kg by adding a chloride source.

In some cases, the chloride source can be calcium chloride, which will also affect the calcium content of the diet. Calcium chloride can be presented in the anhydrous or dihydrated forms, therefore, it is important to specify which of the two is to be utilized. Because only limited amounts of sulfur can be added to the diet to avoid negative interactions with other minerals (copper and selenium) and the risk for polioencephalomalacia, and also because sulfur has lower availability than chloride, the latter is usually chosen as the anion to adjust the DCAB. Typical mineral profile of prepartum diets with added acidogenic salts is shown in Table 2.

Several commercial acidogenic salt products are available in the market, some of them containing hydrochloric acid and others containing fermentation by-products high in chloride. Claims have been made suggesting that such products are less unpalatable than the typical chloride sources (calcium chloride, magnesium chloride, ammonium chloride). However, the few studies available in the literature do not substantiate such claims. Therefore, it is important that the minimum amount of acidogenic salts be added to the diet in order to be effective in preventing hypocalcemia and to avoid drastic reductions in dry matter intake prepartum.

Monitoring acidogenic salt programs can be easily accomplished by continuous evaluation of urinary pH (Jardon, 1995). Most cows require at least 24 to 48 h for their urine pH to adjust to the DCAB. Therefore, evaluating urine pH should be done in cows that have received the diet for more than 2 days. Most Holstein cows should have a urine pH between 5.8 and 6.8, which indicates adequate acidification by the diet (Jardon, 1995). Over acidification can depress dry matter intake without beneficial effects on calcium metabolism. In fact, metabolic acidosis caused by excessive feeding of acidogenic salts has been shown to depress the insulin response to glucose infusion (Bigner et al., 1996), which might compromise the metabolic status of the cow. Constant monitoring of urine pH allows for quick changes in the total amount of acidogenic salts added to the diet to avoid over and under-acidification.

Feeding to prevent mastitis

Beyond the negative impact on yields of milk and milk components, mastitis has recently been reported to have a detrimental effect on reproductive performance of lactating dairy cows. The first published report suggested that clinical mastitis caused by Gram negative (–) bacteria could alter interestrus interval (Moore et al., 1991). Cows with clinical mastitis due to Gram – bacteria were 1.6 times more likely to have an altered interestrus interval than cows without mastitis. More recent reports have indicated that cows that develop clinical mastitis early in lactation are less likely to conceive (Barker et al., 1998). Furthermore, intramammary infections of subclinical nature were also associated with reduced reproductive efficiency (Schrick et al., 2001). Schrick et al. (2001) collected milk samples from 752 cows every 4 to 8 weeks during an entire lactation, at drying off, near calving, and when clinical mastitis was diagnosed. After grouping cows based on mastitis type as no mastitis, subclinical mastitis, and clinical mastitis, they observed that cows with clinical or subclinical mastitis before first service had increased days to first AI, days open, and increased services per conception compared to cows classified as not having mastitis. The most severe impact of mastitis on reproductive performance was observed when cows developed subclinical mastitis followed by clinical mastitis.

Acute mastitis caused by Gram – bacteria can result in bacteremia in over 30% of the affected cows (Wenz et al., 2001), and inflammation of tissues can trigger the release of inflammatory mediators, one of them being the 2 series of prostaglandins. It has been suggested that clinical mastitis caused by Gram – pathogens can alter the interestrus interval by the release of bacterial endotoxins with premature luteolysis due to PGF_{2a} secretion (Moore et al., 1991).

We studied the effects of clinical mastitis on reproductive efficiency in 1,001 high producing Holstein dairy cows in 2 commercial dairy farms in central California. Cows were followed for an entire lactation (320 d) and divided into 4 treatment groups: control (C) or no mastitis, first clinical mastitis prior to first postpartum insemination (G2), first clinical mastitis between first postpartum AI and pregnancy diagnosis (G3), and first clinical mastitis after diagnosed pregnant (G4). Within each dairy, every cow in the mastitis groups was matched with a control cow that was in the same lactation, calved in the same month and had a similar 305-d milk yield in the previous lactation. Mastitis diagnosis was performed at every milking by the herd personnel. A fore sample of milk was collected from every clinical case for microbiological culture. Reproductive management consisted of estrus synchronization with PGF_{2a} prior to 70 d in lactation and timed AI afterwards for the first postpartum AI. Cows diagnosed as open at rectal palpation were re-inseminated following a timed AI protocol. Pregnancy was diagnosed 35 to 48 d after AI and reconfirmed either at 160 d pregnant or at 300 d in lactation. Conception rate at first postpartum AI was decreased by mastitis (28.7 vs 17.7%, $P < 0.001$). The effect of mastitis on first service conception rate was more pronounced when the first clinical case was diagnosed after the first postpartum AI (10.2%) compared to prior to first postpartum AI (22.1%; $P < 0.01$). Pregnancy rate at 320 d postpartum was also decreased for cows with mastitis (C, 85.4 vs G1, 72.3 vs G2, 58.5%; $P < 0.001$), which extended days open (Figure 2). Incidence of abortions was 5.8, 11.8, 11.6, and 9.7% for C, G2, G3, and G4, respectively (Figure 3; $P = 0.04$). We concluded that mastitis either prior to or after first postpartum AI compromises fertility in dairy cows, and management decisions must be taken to minimize the incidence of mastitis throughout lactation.

Research has indicated that boosting the immune status of transition cows with high levels of dietary vitamin E might reduce incidence of new intramammary infections after calving. Immediately prior to calving, concentrations of plasma a-tocopherol decrease by 50% and remain low for the first 20 to 30 d postpartum (Weiss et al., 1990). However, when cows wee fed 4,000 IU of vitamin E, plasma a-tocopherol was maintained above 3.0 µg/ml (Weiss et al., 1997), which is indicative of normal blood levels.

A series of experiments conducted at Ohio State University have indicate that supplemental vitamin E at 1,000 or 4,000 IU/cow/day and dietary selenium at 3 mg/kg of diet dry matter reduces the incidence of clinical mastitis during early lactation (Smith et al., 2000). Supplemental selenium without vitamin E reduced incidence and duration of clinical mastitis, but response was enhanced with supplemental vitamin E. Weiss et al. (1997) fed transition dairy cows different amounts of vitamin E pre- and postpartum. Feeding 4,000 IU/d of vitamin E during the last 2 weeks of gestation and 2,000 IU during lactation maintained plasma a-tocopherol above 3.0 µg/ml, reduced incidence of clinical mastitis, and decreased prevalence of new intramammary infections at parturition. Cows with plasma a-tocopherol below 3.0 µg/ml were 9.4 times more likely to develop clinical mastitis during the first 7 d of lactation than those with concentrations above 3.0 µg/ml. It is possible that minimizing the incidence of mastitis, especially during the first 3 or 4 months in lactation, it might improve conception rates early postpartum and minimize incidence of abortions.

Management of Body Condition Score

Evaluation of body condition score (BCS) is a useful management tool to assess body fat reserves in dairy cows. Excessive BCS prior to calving has been recognized as a risk factor for the development of metabolic problems, especially those related to intermediary metabolism (Cameron et al., 1998). At the same time, very low BCS after calving has been associated with increased incidence of anestrous and anovulatory cycles and reduced conception rates.

Generally, BCS is classified on a 5-point scale (1to 5), with 0.25 units of increment. Ferguson et al. (1994) designed a very consistent method to assess BCS with high repeatability within and between people when BCS ranges from 2.5 to 4.0. Utilization of this method has indicated that cows with low BCS at 70 d postpartum are more likely to have anovulatory cycles or be in anestrus (Figure 4). Cows that do not experience a luteal phase prior to first postpartum insemination have reduced fertility (Stevenson, 2001; Santos et al., 2003a). Furthermore, cows with low BCS at breeding have reduced estrus detection after a luteolytic dose of prostaglandin F2a (Santos et al., 2003b) and reduced fertility when enrolled in timed insemination protocols (Moreira et al., 2000). Therefore, minimizing the loss of BCS during the first weeks postpartum is critical for adequate reproductive performance. This can only be achieved by minimizing the incidence of postpartum problems and optimizing nutrient intake during the first 4 to 8 weeks in lactation.

Conclusions

Nutritional management of transition cows impact health and incidence of postpartum metabolic disorders. Diseases such as retained fetal membranes, metritis, ketosis, hepatic lipidosis, displacement of abomasum, milk fever, and mastitis are interrelated, and they all affect reproductive performance of lactating dairy cows. Mineral unbalances resulting in hypocalcemia can be easily corrected with the utilization of low sodium and potassium diets associated with feeding of acidogenic salts. Mastitis has recently been recognized as a major impediment to adequate reproductive efficiency of dairy cows. Improving the immune status of cows might reduce intramammary infections and improve fertility. As the production potential of dairy cows increase, special attention has to be given to the transition period to minimize incidence of health disorders. Feeding diets that are balanced to meet the nutritional requirements of prepartum and early postpartum cows is expected to minimize the period of negative energy balance, reduce losses of BCS and improve cyclicity during the breeding period, which in turn improves conception rates and overall reproductive efficiency of the herd.

References

Barker, A.R., F.N. Schrick, M.J. Lewis, H.H. Dowlen, and S.P. Oliver. 1998. Influence of clinical mastitis during early lactation on reproductive performance of Jersey cows. J. Dairy Sci.81:1285-1290.

Beam, S.W. and W.R. Butler. 1998. Energy balance, metabolic hormones, and early postpartum follicular development in dairy cows fed prilled lipid. J. Dairy Sci. 81:121-131.

Beam, S.W. and W.R. Butler. 1997. Energy balance and ovarian follicle development prior to first ovulation postpartum in dairy cows receiving three levels of dietary fat. Biol. Reprod. 56:133-142.

Beede, D.K. 1992. The DCAD concept: Transition rations for dry pregnant cows. Feedstuffs. 64(53):12.

Bigner, D.R., J.P. Goff, M.A. Faust, J.L. Burton, H.D. Tyler, and R.L. Horst. 1996. Acidosis effects on insulin response during glucose tolerance tests in Jersey cows. J. Dairy Sci. 79:2182-2189.

Black, W.G., L.C. Ulberg, H.E. Kidder, J. Simon, S.H. McNutt, and L.E. Casida. 1953. Inflammatory response of the bovine endometrium. Am. J. Vet. Res. 14:179-183.

Butler, W.R. 2000. Nutritional interactions with reproductive performance in dairy cattle. Anim. Reprod. Sci. 60:449-457.

Butler W.R. and Smith R.D. 1989. Interrelationships between energy balance on postpartum reproductive function in dairy cattle. J. Dairy Sci. 72:767-783.

Cameron, R.E.B., P.B. Dyk, T.H. Herdt, J.B. Kaneene, R. Miller, H.F. Bucholtz, J.S. Liesman, M.J. Vandehaar, and R.S. Emery. 1998. Dry cow diet, management, and energy balance as risk factors for displaced abomasum in high producing dairy herds. J. Dairy Sci. 81:132-139.

Ferguson, J.D., D.T. Galligan, and N. Thomsen. 1994. Principal descriptors of body condition score in Holstein cows. J. Dairy Sci. 77:2695-2703.

Filley, S.J., H.A. Turner, and F. Stormshak. 2000. Plasma fatty acids, prostaglandin F_{2a} metabolite, and reproductive responses in postpartum heifers fed rumen bypass fat. J. Anim. Sci. 78:139-144.

Filley, S.J., H.A. Turner, and F. Stormshak. 1999. Prostaglandin F_{2a} concentrations, fatty acid profiles, and fertility in lipid-infused postpartum beef heifers. Biol. Reprod. 61:1317-1323.

Frajblat, M. 2000. Metabolic state and follicular development in the postpartum lactating dairy cow. Ph.D. dissertation, Department of Animal Science, Cornell University.

Gilbert, R.O., S.T. Shin, C.L. Guard, and H.N. Erb. 1998. Incidence of endometritis and effects on reproductive performance of dairy cows. Theriogenology 49:251. (Abstr.).

Goff, J.P. 2000. Pathophysiology of calcium and phosphorus disorders. In The Veterinary Clinics of North America: Metabolic disorders of ruminants. 16: 319-337.
Gröhn, Y.T and P.J. Rajala-Schultz. 2000. Epidemiology of reproductive performance in dairy cows. Anim Reprod Sci. 60-61:605-614.
Grummer, R.R. 1998. Transition cow energy, protein nutrition examined. Feedstuffs. 70(38):11.
Hayirli, A, R.R. Grummer, E.V. Nordheim, and P.M. Crump. 2002. Animal and dietary factors affecting feed intake during the prefresh transition period in Holsteins. J. Dairy Sci. 85:3430-3443.
Horst, R.L., J. P. Goff, T.A. Reinhardt, and D.R. Buxton. 1997. Strategies for preventing milk fever in dairy cattle. J. Dairy Sci. 80:1269-1280.
Jardon, P.W. 1995. Using urine pH to monitor anionic salt programs. In The Compendium – Dairy Production Management. 6:860-862.
LeBlanc, S.J., T.F. Duffield, K.E. Leslie, K.G. Bateman, G.P. Keefe, J.S. Walton, and W.H. Johnson. 2002a. Defining and diagnosing postpartum clinical endometritis and its impact on reproductive performance in dairy cows. J. Dairy Sci. 85:2223-2236.
LeBlanc, S.J., T.F. Duffield, K.E. Leslie, K.G. Bateman, G.P. Keefe, J.S. Walton, and W.H. Johnson. 2002b. The effect of treatment of clinical endometritis on reproductive performance in dairy cows. J. Dairy Sci. 85:2227-2249.
Lucy M.C.2001. Reproductive loss in high-producing dairy cattle: where will it end? J. Dairy Sci. 84:1277-1293.
Lucy, M.C., R.C. Staples, F.M. Michel, and W.W. Thatcher. 1991. Effect of feeding calcium soaps to early postpartum dairy cows on plasma prostaglandin $F_{2\alpha}$, luteinizing hormone, and follicular growth. J. Dairy Sci. 74:483-492.
Mertens, D.R. 1997. Creating a system for meeting the fiber requirements of dairy cows. J. Dairy Sci. 80:1463-1481.
Minor, D.J., S.L. Trower, B.D. Strang, R.D. Shaver, and R.R. Grummer. 1998. Effects of nonfiber carbohydrate and niacin on periparturient metabolic status and lactation of dairy cows. J. Dairy Sci. 81:189-200.
Moore, S.J., M.J. VandeHaar, B.K. Sharma, T.E. Pilbeam, D.K. Beede, H.F. Bucholtz, J.S. Liesman, R.L. Horst, and J.P. Goff. 2000. Effects of altering dietary cation-anion difference on calcium and energy metabolism in peripartum cows. J. Dairy Sci. 83:2095-2104.
Moore, D.A., Cullor, J.S., BonDurant, R.H., Sischo, W.M. 1991. Preliminary field evidence for the association of clinical mastitis with altered interestrus interval in dairy cattle. Theriogenology 36, 257-265.
Moreira, F., C. Risco, M.F. Pires, J.D. Ambrose, M. Drost, M. DeLorenzo, and W.W. Thatcher. 2000. Effect of body condition on reproductive efficiency of lactating dairy cows receiving a timed insemination. Theriogenology. 53:1305-1319.
Overton, M.W. 2002. Economics of bovine reproduction. In Dairy Reproduction. Dairy Performance Series. Continuing Education Course for Practicing Veterinarians, University of California Davis, Tulare, CA, March 7 to 9.

Raizman, E. and J.E.P. Santos. 2002. Effect of left displacement of abomasum corrected by toggle pin suture on lactation reproduction and health of Holstein cows. J. Dairy Sci. 85: 1157-1164.

Reist, M., A. Koller, A. Busato, U. Kupfer, J.W. Blum. 2000. First ovulation and ketone body status in the early postpartum period of dairy cows. Theriogenology. 54: 685-701.

Royal M, G.E. Mann, and A.P. Flint. 2000. Strategies for reversing the trend towards subfertility in dairy cattle. Vet. J. 160:53-60.

Santos, J.E.P., S.O. Juchem, R.L.A. Cerri, C. Dei, R. Chebel, W.W. Thatcher, and C. Bilby. 2003a. Effect of bST and reproductive management on reproductive and lactational performance of Holstein cows. J. Dairy Sci. 86(Submitted January).

Santos, J.E.P., M. Villasenor, E.J. DePeters, P.H. Robinson, and C.H. Holmberg. 2003b. Type of cottonseed and gossypol in diets of lactating dairy cows: Plasma gossypol, reproduction, and health. J. Dairy Sci. (In Press).

Santos, J.E.P. Santos, R.L.A. Cerri, M.A. Ballou, G. Higginbotham, and J.H. Kirk. 2002. Effect of clinical mastitis incidence on lactational and reproductive performance of Holstein dairy cows. J. Dairy Sci. 85(Suppl. 1):375. (Abstr.).

Santos, J.E.P., E.J. DePeters, P.W. Jardon, and J.T. Huber. 2001. Effect of prepartum crude protein level on performance of primiparous and multiparous Holstein dairy cows. J. Dairy Sci. 84:213-224.

Schrick, F.N., M.E. Hockett, A.M. Saxton, M.J. Lewis, H.H. Dowlen, and S.P. Oliver. 2001. Influence of subclinical mastitis during early lactation on reproductive parameters. J. Dairy Sci. 841407-1412.

Seals, R.C., I. Matamoros, and G.S. Lewis. 2002. Relationship between postpartum changes in 13, 14-dihydro-15-keto-PGF$_{2a}$ concentrations in Holstein cows and their susceptibility to endometritis. J. Anim. Sci. 80:1068-1073.

Smith, K.L., W.P. Weiss, and J.S. Hogan. 2000. Role of vitamin E in optimizing mammary gland health and productivity of dairy cows: The next generation. In Proc. Southwest Nutr. Manag. Conf., University of Arizona, Tucson, AZ. pp 1-8.

Staples, C.R., J.M. Burke, and W.W. Thatcher. 1998. Influence of supplemental fats on reproductive tissues and performance of lactating cows. J. Dairy Sci. 81:856-871.

Stevenson, J.S. 2001. Reproductive management of dairy cows in high milk-producing herds. J. Dairy Sci. 84(E. Suppl.):E128-E143.

Van Saun, R.J. and C.J. Sniffen. 1996. Nutritional management of the pregnant dairy cow to optimize health, lactation and reproductive performance. Anim. Feed Sci. Tech. 59:13.

Vasconcelos, J.L., R. Sartori, H.N. Oliveira, J.G. Guenther, and M.C. Wiltbank. 2001. Reduction in size of the ovulatory follicle reduces subsequent luteal size and pregnancy rate. Theriogenology. 56:307-314.

Weiss, W.P., J.S. Hogan, D.A. Todhunter, and K.L. Smith. 1997. Effect of vitamin E supplementation in diets with low concentrations of selenium on mammary gland health of dairy cows. J. Dairy Sci. 80:1728-1737.

Weiss, W.P., J.S. Hogan, K.L. Smith, and K.H. Hoblet. 1990. Relationships among selenium, vitamin E, and mammary gland health in commercial dairy herds. J. Dairy Sci. 73:381-390.

Wenz, J.R., G.M. Barrington, F.B. Garry, K.D. McSweeney, R.P. Dinsmore, G. Goodell, R.J. Callan. 2001. Bacteremia associated with naturally occurring acute coliform mastitis in dairy cows. J. Am. Vet. Med. Assoc. 219:976-981.

Zeron, Y., A. Ocheretny, O. Kedar, A. Borochov, D. Sklan, and A. Arav. 2001. Seasonal changes in bovine fertility: relation to developmental competence of oocytes, membrane properties and fatty acid composition of follicles. Reproduction 121:447-454.

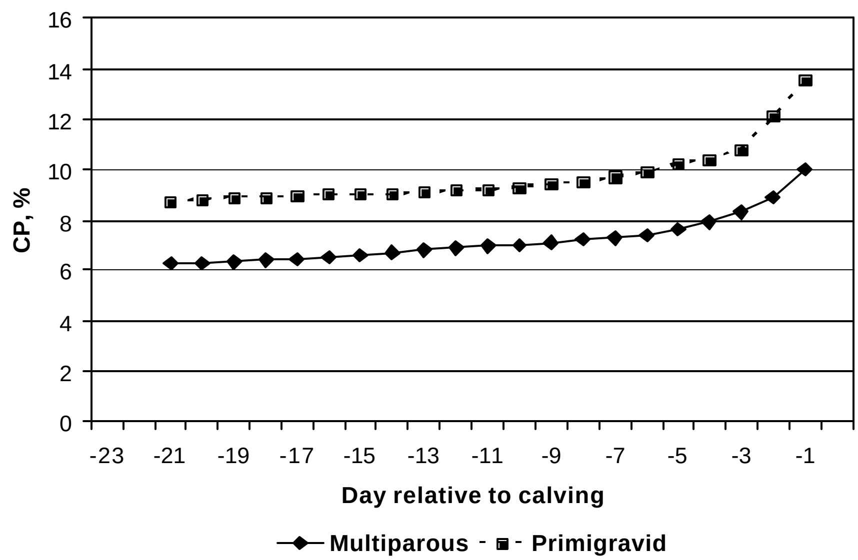

Figure 1. Estimated crude protein requirements of prepartum Holstein cows during the last 21 d of gestation (Grummer, 1998).

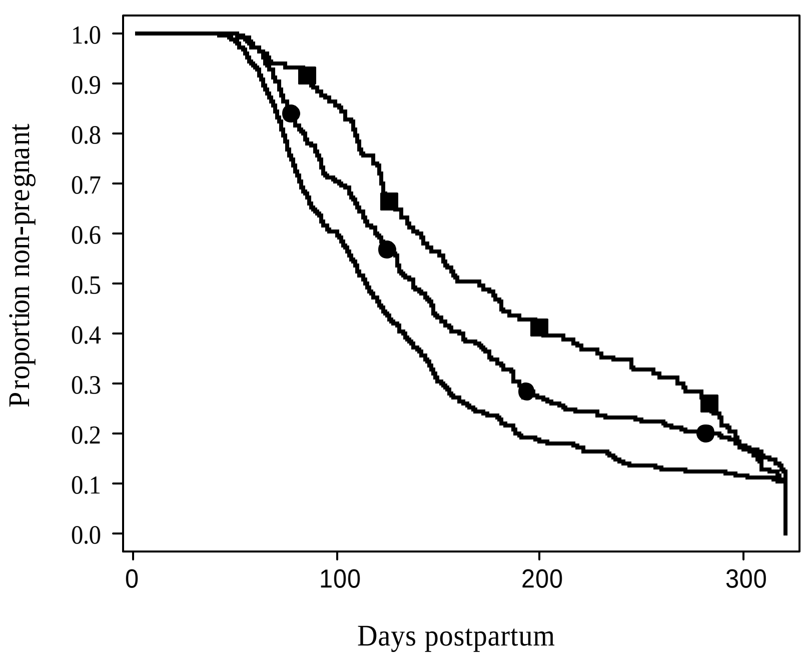

Figure 2. Survival curves for the interval from calving to conception in control cows (—), cows that developed their first clinical mastitis case prior to the first postpartum AI (G1; —?—), and cows that developed their first clinical mastitis case between the first postpartum AI and pregnancy diagnosis (G2; —¦ —). Median days to conception were: control = 114; G1 = 137; G2 = 169. Effect of treatment: $P < 0.0001$.

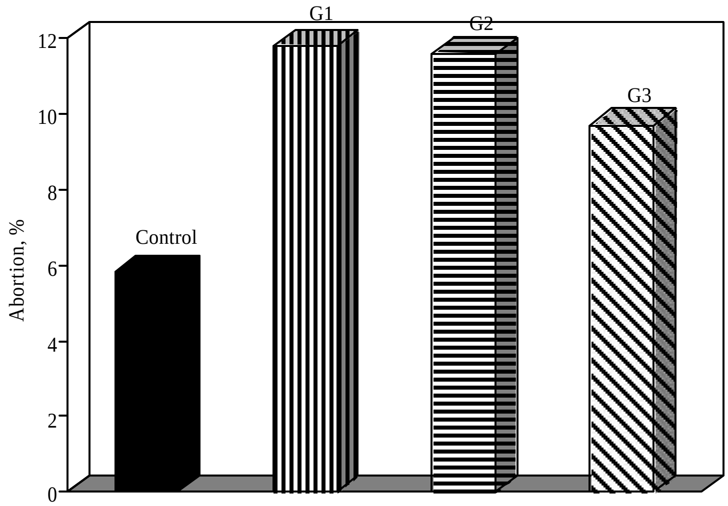

Figure 3. Incidence of abortions in control cows (black solid bar), cows that developed their first clinical mastitis case prior to the first postpartum AI (G1; bar with vertical shading), cows that developed their first clinical mastitis case between the first postpartum AI and pregnancy diagnosis (G2; bar with horizontal shading), and cows that developed their first clinical mastitis case after diagnosed pregnant (G3; bar with diagonal shading). Effects: treatment ($P < 0.04$); control vs mastitis ($P < 0.01$); and mastitis prior to first AI vs mastitis after first postpartum AI ($P = 0.97$).

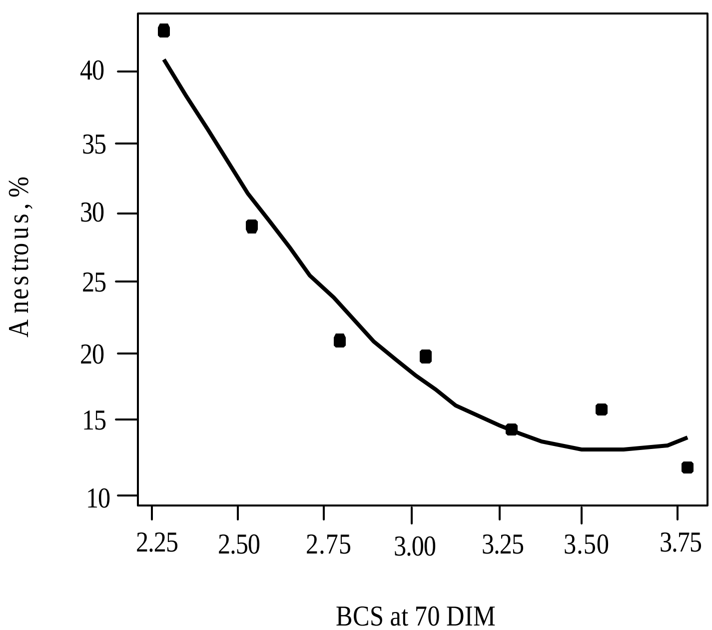

Figure 4. Relationship between the frequency of cows classified as anestrous/anovulatory based on plasma progesterone (< 1.0 ng/ml) and body condition score (BCS) at 70 d in milk (DIM) in lactating dairy cows (Santos et al., 2003). Frequency of anestrous = $223.114 - 118.914 X + 16.8457 X^2$, where X is the BCS at AI ($P < 0.02$).

Table 1. Effect of feeding acidogenic salts to prevent milk fever on reproductive performance of Holstein cows.

Item	Acidogenic salts	Control	*P* <[1]
Clinical hypocalcemia, %			
≤ 2 lactations	0	0	NS
≥ 3 lactations	5	12	.01
All cows	4	9	.01
Subclinical hypocalcemia, %			
≤ 2 lactations	2	16	.01
≥ 3 lactations	28	66	.01
All cows	19	50	.01
Pregnancy rate, %			
150 d postpartum	55	42	.03
200 d postpartum	71	54	.01
250 d postpartum	77	66	.06
Services/pregnancy	3.0	3.4	.16
Average days open	124	138	.10
Milk yield, kg (ME 305 d)	9,376	9,049	.01

Adapted from Beede et al. (1991).
[1] NS: not significant

Table 2. Mineral profile of diets fed to late prepartum and postpartum cows

	Period	
	Close up period	Postpartum
Mineral	--------------------- DM basis	---------------------
Ca, %	1.0 – 1.2	0.8
P, %	0.35 – 0.4	0.4
K, %	< 1.2	1.5 to 1.7
Mg, %	0.35 to 0.4	0.35 to 0.4
S, %	0.3 to 0.4	0.2
Na, %	< 0.1	0.3 – 0.4
Cl, %	0.5 to 0.8	0.25 to 0.3
DCAB, mEq/kg	-50 to -150	+ 300 to + 400
Urine pH	5.8 to 6.8	8.0 to 8.5