INTRODUCTION

Genetic improvement programs have done a great job selecting animals with superior genetics for milk production. Selection with emphasis on milk production is generally recognized as the single most important objective of breeding programs in dairy cattle worldwide. As a result of this improvement, the dairy cow has changed significantly over the past 7 decades with a remarkable increase in milk production/lactation (Butler, 2000). Comparing the shape of lactation curves from a dairy cow from the 1940s with today’s dairy cow, it is evident that the milk yield/lactation is almost 5 fold higher than in the 1940s. However, comparing reproductive performance, the modern dairy cow has decreased fertility (Butler, 2000) which has become the number one reason for involuntary culling (NAMHS, 2007).

Several reasons for reproductive failure exist including: nutrition, management, disease, and intrinsic physiological factors. In order to maintain high levels of milk production, modern dairy cows are under a constant challenge to meet nutritional demands and are more likely to experience adverse postpartum diseases and reproductive failure. Numerous recent studies have reported this negative correlation between high levels of production and reproductive performance. Lucy (2001) attributes this decrease in fertility not only to increased milk production, but also to adverse events surrounding the periparturient period. These events do not impair reproduction directly, but affect animal health with undesirable effects carried over throughout the entire lactation.

Nutritional management of a dairy cow, mainly during periods of high levels of stress, is an important tool to improve animal health and reproduction. During late pregnancy and early postpartum, the nutritional requirements of a dairy cow significantly increase to support fetal growth, mammogenesis, and lactogenesis; at the same time that dry matter intake (DMI) decreases. After parturition nutritional requirements remain elevated to support incremental increases in milk yields until lactation peak. During this period, diets must be designed to minimize the losses in body condition score (BCS) observed during the negative energy balance (NEB) period, which is the period when nutrient requirements for maintenance and lactation exceed the ability of the cow to consume sufficient energy from feed. It is well documented in the literature the negative effect NEB has on animal health and reproduction. When cows experience a period of NEB, blood levels of nonesterified fatty acid (NEFA) increase as a consequence of body energy reserve mobilization (adipose tissue), at the same time insulin-like growth factor-I (IGF-I), glucose, and insulin are low. These metabolic changes might compromise animal health, ovarian function, and fertility.

In each period of lactation, nutritional strategies must be customized to improve animal health and reproduction without interfering with lactation performance. These strategies, when used wisely, can positively impact animal performance and improve the efficiency of a dairy operation. However, good management practices must be in place in order to observe these diet effects.

POSTPARTUM DISEASES AND THE NUTRITION OF TRANSITION COWS

Postpartum Uterine Diseases

The postpartum period is one of the most critical stages of lactation for a high producing dairy cow. It is characterized by drastic metabolic changes, immunosuppression, NEB, and elevated levels of stress; which can lead to increased incidence of diseases and decreased animal efficiency. Most of the events observed in this period (ketosis, milk fever, retained placenta, metritis, etc.) have some effect on animal performance, mainly on subsequent fertility (Ferguson, 2000). Cows diagnosed with postpartum uterine diseases (metritis, clinical or subclinical endometritis) had decreased odds to conceive at the first AI and experienced extended periods until conception (Ferguson, 2000; Bruno et al., 2007). These postpartum reproductive diseases are linked to the nutritional status of animals. Recent studies have linked postpartum uterine diseases with reduced
feed intake (Hammon et al., 2006; Huzzey et al., 2007). In both studies researchers observed that cows developing metritis (severe or mild) or endometritis had decreased DMI beginning 2 wk prior to calving. A similar study, evaluating behavior of dairy cows during the periparturient period, indicated that cows experiencing acute metritis spent less time eating than cows without acute metritis (Urton et al., 2005). Not only are clinical uterine diseases negatively correlated with reproduction, subclinical endometritis has also been linked with impaired fertility (Bruno et al., 2007). Subclinical endometritis is characterized by a large intrauterine influx of neutrophils without clinical signs. Cows identified with subclinical endometritis at 35 d postpartum had lower conception rates at first AI and experienced an extra 27 d to conception compared to cows classified as having normal uterine health (Bruno et al., 2007).

The increased incidences of periparturient diseases also are associated with physiological postpartum immunosuppression. Causes of this impairment of the immune status around calving are not completely elucidated yet, but it is the consequence of multiple factors observed during this period such as fetal corticoid release, NEB, increased blood levels of NEFA, imbalance of Ca and glucose metabolism, and decreased DMI. This immunosuppression is characterized by reduced neutrophil function and decreased production of lymphocytes beginning up to 2 wk prior to calving and extending until 3 to 4 wk postpartum (Kehrli et al., 1989). Hammon et al. (2006) evaluated neutrophil function during the postpartum period of 83 periparturient cows, studying the killing ability of neutrophils correlated to blood levels of NEFA during the transition period. In this study researchers observed that cows diagnosed with puerperal metritis (within 14 d postpartum) or subclinical endometritis (within 28 d postpartum) had significantly lower neutrophil activity than cows classified as having normal uterine health. They also observed that the cows diagnosed with puerperal metritis or subclinical endometritis had significantly lower DMI and increased levels of NEFA and B-hydroxybutyrate (BHBA) starting prior to calving. These changes in the metabolic profile of lactating dairy cows are commonly observed during NEB period, which has been associated with postpartum immunosuppression.

These data clearly identify suppressed nutrient intake prior to calving as a major risk factor for postpartum uterine diseases and consequently impairment of subsequent fertility. Nutritional management in the periparturient period should be designed to prevent or minimize DMI suppression in order to improve uterine health.

**Metabolic Diseases**

Negative energy balance and mineral imbalance early postpartum has been attributed to several postpartum disorders compromising dairy cow fertility. Deficiency of Ca during this period is a major problem in high producing dairy cows. There is no other period in a dairy cow’s lactation in which Ca requirements are higher than the period around calving. Calcium is required for numerous vital functions, for example acting as a second messenger or co-factor for intracellular metabolic pathways, milk synthesis, and muscle contractions in organs such as the diaphragm, rumen, lungs, mammary gland, liver, and uterus. As parturition approaches, a high demand for Ca is required for colostrum production as well as for contraction of all the muscles involved in the parturition process. Reviewing briefly peripartum Ca requirements, shortly before calving the mammary gland extracts a large amount of Ca from the blood and incorporates it into colostrum. The normal blood level of Ca is approximately 8.5 to 10 mg/dL, which means there is approximately 3.5 g of Ca, (ionized Ca) in the entire plasma pool of an adult Holstein cow weighing 600 Kg. The Ca concentration in the colostrum ranges from 2 to 3 g/L of colostrum. A cow producing 15 L of colostrum has approximately 30 g of Ca, which was transferred from the blood to the mammary gland; meaning 10 times more Ca was required than the free form of Ca present in the cow’s plasma. With this deficit of Ca, subclinical or clinical signs of hypocalcemia can be observed.

Several studies have reported the deleterious effect of hypocalcemia on subsequent fertility. Whiteford and Sheldon (2005) reported that cows experiencing hypocalcemia had increased incidence and severity of uterine diseases and delayed return of ovarian cyclic activity. Moreover, cows experiencing hypocalcemia were 3 times more likely to experience retained placenta than cows not experiencing this disease (Curtis et al., 1983). Hypocalcemia can also affect dairy cow fertility indirectly. Hypocalcemic cows at parturition had 4.8 times greater risk of developing left displacement of abomasum (LDA) than normocalcemic cows (Massey et al., 1993) and cows experiencing LDA had delayed postpartum first service (Raizman and Santos, 2002).

Nutritional efforts to prevent clinical or subclinical hypocalcemia may improve reproductive efficiency of high producing dairy cows. Several
nutritional strategies have been proposed to minimize the Ca imbalance in the periparturient period. One of these strategies, widely used in large dairy operations, is dietary cation-anion difference (DCAD) diets fed throughout the last 21 d before the expected calving date. The DCAD diet consists of addition of anionic salt to the prepartum diets, which creates a slightly metabolic acidosis status; therefore increasing Ca mobilization. Although this strategy is very effective in preventing and minimizing the incidence of hypocalcemia, adding anionic salts to prepartum diets can inhibit DMI due to palatability issues leading to different metabolic distresses, which also have negative effects on animal health and reproduction. Monitoring DMI and urine pH often are necessary tools for the success of DCAD diet.

Energy metabolism during the transition period plays an important role on animal health and reproduction. Energy requirements at the end of the gestational period and initiation of lactation always exceed the amount of energy the cow obtains from her diet. It is estimated that in the last week of fetal development the fetus uses approximately 46% of maternal glucose taken up by the uterus (Bell, 1995) and the onset of milk production makes this energy shortage even more remarkable. The mammary gland drains a large amount of glucose to synthesize milk lactose through its predominant glucose transporter GLUT-1; which does not require insulin for glucose uptake. Rigout et al. (2002), studying glucose metabolism in dairy cows, concluded that the mammary gland consumes 60 to 70% of the whole body glucose, mainly for lactose synthesis. In this case, a cow producing 30 Kg of milk per day uses at least 1.5 Kg of blood glucose to synthesize milk lactose. The high demand of energy during this period of glucose shortage triggers a compensatory process of nutrient partitioning and adipose tissue mobilization. However, there is a limit to the amount of fatty acids that can be metabolized by the liver in order to be converted to energy. When this limit is reached, fats are no longer oxidized and start to accumulate as triglyceride in hepatocytes leading to hepatic lipidosis (fatty liver syndrome). Cows experiencing fatty liver syndrome experience extended periods to first ovulation (Reist et al., 2000) and reduced reproductive performance (Jorritsma et al., 2000). Ketone bodies are formed as a result of β-oxidation of fat, which is used as energy. The excess ketone bodies are eliminated in the urine and milk and are observed during clinical and subclinical cases of ketosis. A meta-analysis indicates that clinical ketosis is associated with an increase of 2 to 3 d to first service and with 4 to 10% fewer pregnancies per AI at first service (Fourichon et al., 2000).

Duffield (2000) reported that fat cows (BCS ≥ 4.0) had elevated blood levels of BHBA postcalving and were at higher risk of developing subclinical ketosis compared to cows classified as moderate or thin BCS prior to calving. In addition, subclinical ketosis has also been linked with increased incidence of ovarian cysts (Duffield, 2000).

Because increased energy demand is observed even before calving, strategies to prevent metabolic disease have focused on the nutritional management of the dry and transition cow. The goals of these diets are basically to provide all required nutrients and to adapt the rumen for diet changes as cows advance to different stages of lactation. Diets must be formulated in order to accomplish this goal and also minimize DMI change to prevent metabolic and uterine diseases. Managing BCS towards the end of the lactation is another important management practice in order to minimize postpartum diseases.

NUTRITION INTERACTION WITH POSTPARTUM OVULATION RESUMPTION

In all mammalian species, resumption of cyclicity requires that all organs and tissues associated with reproduction recover from the previous pregnancy and parturition:

- Ovaries must re-establish full follicular development with ovulation;
- Hypothalamus/pituitary glands must secrete gonadotropin hormones in an appropriate manner to stimulate follicle growth; and
- Liver must be adapted to support heavy metabolic loads (Butler, 2005).

However, in dairy cows the re-establishment of all these functions is negatively correlated to NEB. After parturition, nutritional requirements increase dramatically with each increment of milk production and the resulting NEB can extend up to 8 to 10 wk after calving, delaying resumption of ovulation to 10 to 14 d after the nadir of NEB (Butler, 2003).

During the NEB period, homeorrectic controls assure nutrient partitioning and adipose tissue mobilization to support milk production. During this period it is common to observe losses in body weight and BCS. Santos et al. (2009), evaluating changes in BCS, reported that severe weight and BCS losses due to inadequate nutrition or diseases are associated with anovulation or anestrus status in dairy cows. In fact, cows with poor BCS are more likely to be anovular, which compromises dairy cow fertility (Bruno et al., 2005). Reasons for anovulation during the NEB
period include attenuation of luteinizing hormone (LH) pulse frequency and low levels of blood glucose, insulin, and IGF-I that collectively limit estrogen production by dominant follicles (Butler, 2005).

Fertility of dairy cattle is affected by NEB by inhibition of ovulation in early lactation, reduction of estrous expression and impairment of conception and embryo survival during the breeding period (Santos et al., 2004). Several studies have demonstrated improvement in estrous expression, conception rate, and embryo survival when resumption of ovulation is observed prior to the initiation of synchronization programs for first postpartum AI (Bruno et al., 2005; Santos et al., 2004).

Resumption of ovarian activity in high producing dairy cows is determined by energy status of the animal. Nutritional management, to minimize the inhibitory effects of NEB on resumption of ovulation and to prevent postpartum metabolic diseases, should begin during the prepartum period. These strategies should include maintaining moderate BCS and energy intake throughout the transition period.

**NUTRITION INTERACTIONS WITH THE BREEDING PERIOD**

Most dairies start re-breeding their cows between 50 and 70 d after calving. It is expected that by this time the majority of cows are completely recovered from their previous calving and are ready to conceive again. However, reproductive and nutritional management during this period is important for a successful reproductive program.

The beginning of the breeding season coincides with the period in which cows are reaching or are about to reach the peak of milk production, which generally occurs around 6 to 9 wk postpartum. Targeting higher peak milk production, diets offered at this stage are designed to maximize DMI. However, increased DMI is associated with an increased rate of metabolism of reproductive hormones (Rabiee et al., 2001) impairing reproductive performance. High producing dairy cows have increased DMI due to lactation requirements. Lopez et al. (2004) reported that cows with milk production above herd average (39 Kg/d) had shorter duration and lower intensity of estrus than cows with lower milk production. This negative effect of DMI on estrous expression is explained by the increased liver blood flow decreasing the concentration of plasma hormones, including steroids and consequently changing estrous behavior. Identification of feedstuffs that potentially can block hepatic steroid metabolism is currently under investigation at the University of Wisconsin-Madison and is not ready to use on dairy farms. Another potential strategy to overcome poor estrous expression is through supplementation of steroid hormones to improve reproduction, although none are approved for use.

On the other hand, cows not consuming enough until peak lactation lose BCS and have decreased reproductive performance. Santos et al. (2009) evaluating changes in BCS in early lactation concluded that cows losing one or more units of BCS from calving to the time of first AI were less likely to conceive and were at higher risk of losing their pregnancy. Moreover, cows with BCS lower than 3.0 at the time of AI had the lowest risk for pregnancy compared to cows with better BCS (Santos et al., 2009).

Nutritional strategies to improve reproductive performance during this period include increasing the energy density of the diet, which can be done with fat supplementation. Fats in the diet of a dairy cow can positively influence reproduction by altering both ovarian follicle and corpus luteum function via improved energy status and by increasing precursors for the synthesis of reproductive hormones, such as steroids and prostaglandins (Mattos et al., 2000). However, different degrees of saturation might influence fertility outcomes. Juchem (2010) evaluated the effect of feeding protected fat as Ca salts and observed increased conception rates in cows fed Ca salts of linoleic and monoenoic C18 trans fatty acids (unsaturated fatty acids) compared with cows fed Ca salts of palm oil (highly saturated fatty acid). This improvement in fertility was attributed to higher fertilization rates (87.2 % versus 73.3 %), and the greater proportion of embryos graded as 1 and 2 (73.5 % versus 51.5 %), for saturated and unsaturated fatty acid diets respectively (Cerri et al., 2009).

**NUTRITION AND LATE LACTATION INTERACTIONS**

Towards the end of lactation it is expected that all cows in the herd are pregnant; therefore the nutritional goal during this period is to maintain pregnancy and prepare cows for the next lactation. After 90 d of gestation, few cows lose their pregnancy (Santos et al., 2004) unless a major stressor such as a disease outbreak, heat stress, mycotoxin from spoiled feed, etc. are present. During
this period diets must be formulated to support fetal growth and milk production, dependent upon stage of lactation, without allowing cows to gain excess BCS. Cows overconditioned prior to calving are at a higher risk of developing metabolic diseases and are more likely to be culled involuntarily at the beginning of the next lactation. During this period providing animal comfort, clean water, adequate nutrition, and minimal stress are important features in minimizing pregnancy loss; which is critical for cows in late lactation.

CONCLUSION

Genetic selection in dairy cows during the last decades has focused mainly on milk production. As cows became more specialized in producing milk, an increased likelihood of health disorders and reproductive failure has been observed. Studies have shown that nutritional management during the transition period can improve uterine health and minimize metabolic distress in the postpartum period.

Managing negative energy balance by increasing energy intake during the early postpartum and breeding period may improve resumption of cyclicity and reproductive outcomes. Supplementation with unsaturated fat can improve fertility, as long as it is protected from rumen biohydrogenation of fatty acids. Nutritional management during the late lactation period must meet nutritional requirements for production and fetal development without allowing cows to become obese, which negatively impacts animal performance in the following lactation.

Managing a high producing herd and maintaining high reproductive performance is an enormous challenge for the dairy producer. Nutritionists and veterinarians must work together to ensure that producers are applying all the currently known facts about nutrition, herd health, and reproduction on a daily basis. Proper nutrition is part of a successful reproduction program; but absence of any one of the other key management components likewise can result in a poor herd reproduction program.

LITERATURE CITED


