Abstract

During the transition period, the dairy cow is undergoing numerous changes in endocrine, nutritional, metabolic, and physiological status as she prepares for calving and initiation of lactation. These changes result in a dramatic decrease in dry matter intake that worsens the negative energy balance already present after calving. If the negative energy balance during transition becomes excessive, metabolic diseases, such as fatty liver and ketosis, can result. Disruption of mineral balance during the periparturient period leads to mineral balance disorders, especially milk fever. These diseases are costly in terms of their affect on milk production, reproduction, and the cow’s susceptibility to other periparturient disorders. Intensive management of the nutrition, feeding system, and environment of the periparturient dairy cow reduces the odds of disease and increases the odds of success.

The ‘100 day contract’ is a series of delicate negotiations that encompass the full impact of the transition cow. Unsuccessful negotiations at any point increase the risk of overall failure. Getting the details right and ensuring adequate intake of all nutrients are the key elements of the ‘100-day contract’.

Introduction

In evaluating the production cycle of the dairy herd, a 100-day period of critical importance exists. The ‘100-day contract’ with the dairy cow begins 30 days before calving and continues through first breeding at 70 days postpartum. The terms of the contract include the birth of a live calf, with the cow remaining healthy during the transition period, high peak milk production, controlled loss of body condition, and high fertility at first breeding (Figure 1). The momentum toward successful achievement begins in the close-up dry cow group and builds through calving to first breeding. Getting the cow off the track at any point disrupts the momentum and can lead to ‘wrecks’. Wrecks include metabolic disorders during the periparturient period that can have long-term impact on production and reproduction. This paper will focus on a phase-by-phase look at the negotiations required to successfully fulfill the ‘contract’, as well as the long-term consequences of cows getting off track.

The Transition Period

Goff and Horst (1997b) defined the transition period of a dairy cow’s productive cycle as the change from the pregnant, nonlactating state to...
the nonpregnant, lactating state during the interval from three weeks prepartum until three weeks postpartum. The transition period is characterized by numerous changes in physiological, metabolic, and endocrine status to accommodate parturition and lactogenesis (Grummer, 1995). If nutritional management does not meet these challenges, the transition cow is at risk of developing a wide range of health problems soon after parturition (Bell, 1995). These problems include milk fever, fatty liver, ketosis, retained placenta, displaced abomasum, and severely suppressed immune function (Goff and Horst, 1997b). Proper management during the transition period affects the well being of the dairy cow by decreasing the incidence of metabolic and infectious diseases, increasing production, and improving reproductive performance during the subsequent lactation. Achieving successful transition should have a positive impact on the profitability of a successful dairy farm.

To successfully manage the transition cow, we must first understand the changes the cow is experiencing and the impact of a poor transition. Then we must implement strategies that address the challenges of the transition cow.

The Transition Cow: Understanding the Challenge

During the dry period, the cow must be prepared for calving and initiation of lactation. The concept of preparing the dry cow is different from the traditional view of the dry period as a ‘rest’ phase (Gerloff, 1988). Goff and Horst (1997b) concluded that the periparturient period should adapt the rumen while maintaining normal energy and calcium metabolism, as well as supporting a strong immune system.

Changes in Endocrine Status

As parturition approaches, the transition cow undergoes a variety of changes in endocrine status. Plasma prolactin levels increase sharply the day prior to calving, resulting in initiation of lactation and increased colostrum synthesis. Progesterone concentration, which is elevated during gestation for maintenance of pregnancy, drops to nearly undetectable levels on the day before calving (Figure 2). Plasma estrogen concentration rises sharply at the same time in response to secretion of fetal cortisol. Prostaglandin F$_{2\alpha}$ (PGF$_{2\alpha}$) concentration begins to rise and peaks at parturition, causing luteolysis and further inhibition of progesterone synthesis (Goff and Horst, 1997b). High levels of estrogen are thought to contribute to the decline in dry matter intake (DMI) that occurs around parturition (Bell, 1995; Grummer, 1995). Goff and Horst (1997b) reported that DMI declines by as much as 30 to 40%, or from 2% to less than 1.5% of the animal’s body weight. Severe decreases in intake put the animal at risk for a number of metabolic disorders.

Changes in Nutritional and Metabolic Status

Although not producing milk, the prepartum cow is undergoing numerous changes that result in significantly higher nutrient requirements. Bell et al. (1995) measured energy and protein deposition in the uterus and fetus. Their research clearly illustrated the increased nutrient requirements during the final 30 days of gestation (Table 1). However, as shown in Figure 3, the increased nutrient requirements occur concurrently with declining appetite and nutrient intake.

To compensate for the negative energy balance caused by decreased DMI around parturition (Figure 4), the stress of calving, increased energy demands resulting from fetal growth and lactogen-
nesis, and other unknown endocrine-related factors, mobilization of adipose tissue increases. Adipose tissue provides energy in the form of non-esterified fatty acids (NEFA) (Grummer, 1995). Plasma NEFA concentration increases approximately two-fold during the last 17 days of gestation, peaks around parturition, and remains higher than prepartum levels until about two weeks postpartum (Figure 5). The liver oxidizes NEFA to ketone bodies and carbon dioxide via the tricarboxylic acid cycle (TCA) or esterifies them to triacylglycerols (TG), which are exported from the liver as very low density lipoproteins (VLDL) (Rukkwamsuk et al., 1998). If these changes become too dramatic, they can lead to ketosis and fatty liver disease.

In short, there is a significant increase in the cow’s nutrient requirements during the final 30 days of gestation, and thus, there is a critical need to maintain intake and provide support for a proper plane of nutrition. A key to successful transition cow management is a nutritional management system (diet and feeding system) that provides nutrients in the proper balance and maximizes intake.

Changes in Physiological Status of the Reticulorumen

Significant physiological changes also occur during the transition period. Because of significant fetal growth during the last 60 days of gestation, ruminal capacity decreases by as much as 20%, then increases again within 8 days after calving. Ruminal dry matter and fluid fill also decrease just before calving and remain low until about 20 days postpartum (Table 2). Decreased capacity of the rumen limits the amount of feed the cow can consume, however, it does not account for the magnitude of the decrease in dry matter intake that occurs around calving (Stanley et al., 1993).

Absorptive capacity of the rumen also changes during the dry and transition periods. Beginning at dry-off, cows are most often fed a high-forage, low-concentrate diet that is higher in neutral detergent fiber (NDF) and less energy dense than the lactation diet. The lower energy diet causes a decrease in length and surface area of rumen papillae (Figure 6). This physiological change in rumen papillae corresponds to a 50% loss of absorptive capacity of volatile fatty acids (VFA) during the first 7 weeks of the dry period (Dirksen et al., 1985).

Postpartum, the papillae must increase in length and surface area to achieve maximum absorption of VFA. Feeding a diet higher in fermentable organic matter stimulates development of the papillae. However, this growth process requires 4 to 6 weeks after changing to a high-energy diet (Dirksen et al., 1985). If the amount of fermentable organic matter is increased too rapidly after parturition and before the papillae have reached adequate surface area, the cow cannot absorb VFA efficiently. Volatile fatty acids can build up in the rumen, causing pH to fall, and resulting in rumen acidosis. Protozoa and some bacteria in the rumen are killed, releasing endotoxins that are absorbed into the bloodstream (Goff and Horst, 1997b). These endotoxins cause systemic changes in blood flow and affect the growth and health of the hooves. These changes can result in the painful condition of laminitis. Cows with laminitis have limited mobility, and therefore, limited intake. Lameness, combined with the rumen papillae’s limited ability to absorb VFA from the rumen, may worsen the transition cow’s negative energy balance, increasing the risk of metabolic disorders. Successful transition cow programs are designed to accommodate the endocrine and physiological changes in the cow while
minimizing costly metabolic diseases.

**Metabolic Disorders**

The transition from late gestation, non-lactating to nonpregnant, lactating presents significant challenges to the cow’s system. When nutrition management does not meet these challenges, a wide range of health problems can result. Metabolic diseases are disorders that are nutritional in origin and often result in acute symptoms, require treatment. Incidence is highest during the period just prior to calving through peak lactation (Shearer and Van Horn, 1992). As shown in Table 3, most of the periparturient diseases, such as milk fever, ketosis, retained placenta, and displaced abomasum, occur within the first 2 weeks postpartum. Many infectious diseases, such as mastitis, also become clinically apparent at this time as a result of the animal’s depressed immune function. Other health disorders that become evident later in lactation, such as laminitis, can be traced back to complications during the first two weeks after parturition (Goff and Horst, 1997b). The majority of metabolic diseases are related to either energy balance or mineral balance. The diseases commonly associated with severe negative energy balance include fatty liver and ketosis. Milk fever is the most common mineral balance disorder (Shearer and Van Horn, 1992).

**Energy Balance Disorders**

During late gestation and early lactation, the cow becomes anorexic. This condition severely limits consumption of energy in amounts necessary to meet demands for maintenance and milk production. Fatty acids are mobilized from adipose tissue as an additional energy source, however, the bovine liver has limited capacity for the amount of fatty acids that can be oxidized or exported as VLDL. When this limit is reached, TG accumulate in the liver, and acetyl coenzyme-A (from oxidation of fatty acids) that is not utilized in the TCA cycle is converted to ketone bodies, such as acetone, acetoacetate, and b-hydroxybutyrate. These ketones appear in the blood, milk, and urine (Goff and Horst, 1997b).

Fatty liver occurs when the rate of TG synthesis exceeds the rate of TG hydrolysis and TG export as VLDL (Grummer, 1993). Excessive accumulation of TG in the liver impairs its normal function and, in severe cases, can result in liver failure (Shearer and Van Horn, 1992). Because rate of TG synthesis is proportional to plasma NEFA concentration, fatty liver is likely to develop during periods of high plasma NEFA, such as the periparturient period. As shown in Figure 5, NEFA concentration increases approximately two-fold between 17 days prepartum and two days prepartum and increases two-fold again, reaching peak concentration by calving (Grummer, 1993). Because the accumulation of fat in the liver impairs its function, the liver of an overconditioned cow has a more limited ability to oxidize fatty acids than that of a thinner cow (Goff and Horst, 1997b). As a result, excessive body weight gain during late lactation or the dry period predisposes cows to the development of fatty liver following parturition (Rukkwamsuk et al., 1998).

Another major factor contributing to the formation of fatty liver is the inherently slow rate of VLDL secretion by the liver in ruminant animals compared to other species (Bertics et al., 1992). The elevated estrogen levels around parturition can also enhance TG deposition in the liver, escalating the problem even more (Grummer, 1993; Goff and Horst, 1997b). Fatty liver is best prevented by nutritional management during the dry period that minimizes TG deposition in the liver and maximizes liver glycogen stores (Grummer et al., 1993). This balance can be accomplished by
monitoring and managing body condition through the late lactation and dry period diets so cows approach calving in proper body condition (Shearer and Van Horn, 1992). There is also evidence that propylene glycol administration prevents fatty liver by improving energy balance during the last days of gestation and first few weeks of lactation (Formigoni et al., 1996). Data from work done by Grummer et al. (1994) revealed that 296 ml of propylene glycol given as an oral drench once daily was effective for reducing plasma NEFA concentrations.

Fatty liver is thought to precede spontaneous clinical ketosis. Fatty liver is most common by the first day after calving, but cows are most susceptible to ketosis at 3 weeks postpartum. In addition, development of fatty liver may have a direct effect on carbohydrate metabolism and influence susceptibility to ketosis. Gluconeogenic activity of liver tissue has been found to be impaired under conditions conducive to fatty liver development. Reduction in gluconeogenesis by the liver may lower blood glucose levels and decrease insulin secretion, which would support greater lipid mobilization and increased rate of fatty acid uptake by the liver and increased ketogenesis (Grummer, 1993).

Ketosis results from impaired metabolism of carbohydrates and VFA, leading to hypoglycemia. Formation of ketones is the result of incomplete metabolism of mobilized fat. Fatty acids accumulate in the liver as acetyl-CoA because the liver has reduced ability to utilize them. Excess acetyl-CoA is converted to ketones that can be metabolized by peripheral body tissues. When ketones are produced in excess of peripheral tissue’s capacity to use them, they accumulate in the bloodstream, resulting in ketosis. Cows with clinical ketosis exhibit reduced feed intake, reduced milk yield, loss of body weight, central nervous system involvement (staggering, lack of coordination, and appearance of staring or blindness), and, in severe cases, acetone odor on the cow’s breath (Shearer and Van Horn, 1992). Ketosis can be prevented by implementing the same nutritional management strategies used to prevent fatty liver. Additional prevention strategies include avoidance of fermented feeds, such as certain silages containing ketogenic precursors, increased frequency of concentrate feeding, and use of specific additives during the dry and transition periods (Kronfeld, 1982; Grummer, 1993). Additives include daily niacin supplementation, which has been shown to reduce plasma concentration of the ketone b-hydroxybutyrate (Duffield et al., 1998) and daily oral administration of propylene glycol, which provides glucose precursors (Shearer and Van Horn, 1992; Grummer et al., 1994).

Mineral Balance Disorders

A second major cause of metabolic disease is a disruption of mineral balance, primarily calcium balance, around parturition. Lactogenesis and colostrum synthesis place a large demand on calcium homeostasis mechanisms so that almost all cows develop some degree of hypocalcemia at parturition (Beede and Pilbeam, 1998). When plasma calcium concentration drops too low to support nerve and muscle function, parturient paresis, or milk fever, develops (Goff and Horst, 1997a).

Milk fever affects up to 9% of dairy cows around calving (Joyce et al., 1997). Risk of milk fever increases with age and parity. Cows of third or greater parity are at the highest risk, while milk fever is rare in first-calf heifers. Increased risk is also likely related to higher milk yield (Shearer and Van Horn, 1992; Horst et al., 1997; Rajala-Schultz et al., 1999).
The most widely used treatment for milk fever is intravenous infusion of 23% calcium borogluconate solution. However, this treatment can cause cardiac arrest by raising plasma calcium concentrations to dangerous levels. Also, approximately 25% of cows treated for milk fever relapse and require additional treatment (Horst et al., 1997). Prevention can be a more cost-effective alternative in managing milk fever.

Traditionally, limiting calcium intake during the dry period was used to prevent milk fever. The goal of this strategy is keep dietary calcium low enough so that calcium mobilization mechanisms move calcium from body stores and are functional at calving when calcium demand for milk synthesis suddenly increases. Dietary calcium intake should be limited to less than 50 g/day; however, diets containing such a low calcium concentration are often difficult to formulate because many forages commonly used in dairy diets, especially legumes, contain a substantial amount of calcium (Shearer and Van Horn, 1992).

Another concept in milk fever prevention is utilization of dietary cation-anion difference (DCAD). When the amount of calcium in the blood drops below normal, parathyroid hormone (PTH) is secreted to stimulate release of calcium from body tissues into the blood pool. Cows that have a relatively high blood pH are less responsive or non-responsive to secretion of PTH, but cows that have relatively low blood pH are more responsive to PTH. The number of equivalents of cations and anions present ultimately determines blood pH. Blood pH decreases when more anions than cations enter the blood from the diet and digestive tract. The goal in utilizing DCAD in diet formulation and anion supplementation should be to reduce blood pH enough to affect calcium mobilization in response to hypocalcemia. An appropriate DCAD can be achieved by reducing the number of cations or increasing the number of anions in the diet. The number of cations in the diet can be controlled by selecting feeds, especially forages, that are as low in potassium and other cations as practically possible (Beede and Pilbeam, 1998). Goff and Horst (1997a) have provided evidence that increasing potassium in the prepartum diet increases the incidence of milk fever. However, current guidelines for forage production are inadequate for providing dairy producers with low potassium forages.

Sources of anions include Cl⁻ and SO₄²⁻ salts of calcium, ammonium, and magnesium. Phosphate salts are weakly acidifying and are not commonly used. However, only a limited amount of anionic salts can be added to the diet because of palatability problems that can affect intake (Horst et al., 1997). When DCAD is sufficiently reduced, increased calcium content of 180 to 210 g per cow per day does not cause milk fever and appears to have some benefit to the cow (Beede and Pilbeam, 1998).

Diets containing anions must be properly mixed in order for each cow to receive the correct amount of anions to affect blood pH. Anions are ineffective in component feeding situations because the cow’s DCAD cannot be controlled. For the DCAD approach to be most effective, anion/cation content of feedstuffs, intake of the diet, and urine pH (an indicator of blood pH and acid-base status) of cows must be closely monitored (Beede and Pilbeam, 1998). If management requirements cannot be met, other options for preventing milk fever are available. These include feeding a prepartum diet low in calcium (less than 50 g/day) and administration of readily available calcium sources at calving to increase plasma calcium (Horst et al., 1997). Sources of calcium include commercially available oral supplements, such as gels and pastes.
Parturition Disease Complex

Severe losses of body stores or a more general lack of properly balanced nutrients increase the risk of the cow experiencing a number of metabolic diseases. Markusfield (1993) describes these as a parturition disease complex. It is important to understand that these disorders are not independent but are related. For example, milk fever is a significant risk factor for several other transition cow problems, including dystocia, ketosis, retained placenta, mastitis, and displaced abomasum.

Grohn et al. (1995) reported the incidence of these diseases for Holstein cows in New York (Table 3). As the median day of occurrence indicates, these diseases are most likely to occur during the period immediately after calving. However, these disorders have an impact on production and reproduction during the entire lactation. Cows experiencing any one of these disorders are at much greater risk of suffering from a number of the other periparturient dysfunctions. Furthermore, these peripartuient disorders disrupt the cow’s metabolic momentum toward high peak milk yields and also have negative carryover effects on reproductive performance.

Effects of Metabolic Diseases

The culmination of periparturient disorders is lost milk production and decreased reproductive efficiency, both of which reduce income. Cows with fatty liver exhibit depression, loss of appetite, rapid loss of body weight in severe cases, and marked decrease in milk production. Fatty liver is frequently associated with most of the other periparturient disorders, including ketosis, milk fever, displaced abomasum, retained placenta, and metritis. Fatty liver cases do not respond well to treatment, with mortality rates of up to 50% (Shearer and Van Horn, 1992) Cows that do recover have a lengthened interval to first estrus and days to first service (Morrow, 1975).

Ketosis also causes appetite depression, decreased milk yields, and weight loss (Shearer and Van Horn, 1992). Deluyker et al. (1991) reported that clinical ketosis caused losses in milk production of 557 lb during the first 119 days in milk for cows that were diagnosed within the first 21 days postpartum. These cows also had peak milk production of nearly 6 lb less than healthy animals. Clinical ketosis has been associated with increased risk of metritis, displaced abomasum, and mastitis. Subclinical ketosis has been associated with decreased milk yield, increased risk of clinical ketosis, metritis, and cystic ovarian disease, and impaired reproductive performance (Duffield et al., 1998).

Energy balance disorders, such as fatty liver and ketosis, indicate that the parturient cow is in a state of severe negative energy balance. During this period of negative energy balance, luteinizing hormone pulse frequency and growth rate and size of the dominant follicle are decreased. As a result, cows have a longer interval to first ovulation, which causes an increase in days to first service, days open, and services per conception, as well as decreased first service conception rate (Table 4). Achieving high energy intake during the transition period is critical to normal resumption of ovulation and normal corpus luteum development, and therefore, high reproductive efficiency (Roche et al., 2000).

Milk fever is another important periparturient disorder. Rajala-Schultz et al. (1999) found that milk fever alone caused a milk loss of between 2.42 and 6.38 lb/day during the first 4 to 6 weeks following parturition. It can also reduce the productive life of the cow by as much as 3.4 years.
The average cost per case of milk fever has been estimated at $334, based on direct treatment cost and estimated production losses (Horst et al., 1997).

Milk fever also increases the risk of other metabolic diseases, primarily because it has a detrimental affect on smooth muscle function. Muscle tone decreases in most body systems, particularly in the cardiovascular, reproductive, and digestive systems, and possibly in the mammary system. Blood flow to the extremities is reduced, causing the characteristic cold ears of a cow suffering from milk fever. Jonsson and Daniel (1997) found that there was also a significant reduction in blood flow to the ovaries of sheep with induced hypocalcemia. This would result in suppressed ovarian function, including progesterone synthesis and follicular development. Unfortunately, the highest incidence of hypocalcemia is during the first 6 weeks after calving, a critical time for resumption of ovarian activity.

As shown in Table 5, hypocalcemia also predisposes the cow to calving disorders, including retained placenta, dystocia, and metritis, as well as other periparturient disorders. Calving disorders are detrimental to postpartum reproductive function because they slow the rate of uterine involution and resumption of a normal estrous cycle (Risco, 1992). Reproductive efficiency is decreased as a result of a longer interval to first service and first conception and a lengthened calving interval.

Hypocalcemia affects the digestive system by reducing rumen contractility and increasing the risk of displaced abomasum. As a result, feed intake may be suppressed, worsening the negative energy balance already present around parturition and putting the cow at a greater risk for ketosis (Goff and Horst, 1997b). Hypocalcemia may also put the cow at greater risk for mastitis by affecting the teat end sphincter. If the teat end cannot close sufficiently following milking, the cow is more susceptible to bacterial invasion that causes mastitis. In addition, hypocalcemic cows have increased plasma cortisol concentrations that may worsen the immunosuppression normally present at parturition. This leaves the cow with decreased ability to fight infectious diseases, including mastitis (Goff and Horst, 1997b).

Transition Cow Management

Dry Cow Nutrition

The decreases in milk production and reduced reproductive efficiency associated with the periparturient diseases indicate that the incidence of these diseases must be closely monitored. Retained placenta and related reproductive tract infections are often assumed to be caused by nutritional deficiencies. More specifically, since researchers reported the relationship between vitamin E, selenium, and retained placenta, many producers first react to cows calving with retained placenta by increasing vitamin and mineral supplementation of the dry cow diet. Vitamin E and selenium are antioxidant substances that aid in the removal of reactive oxygen metabolites (ROM), or free radicals, that are generated during normal metabolism. When ROM are not effectively removed, they can impair the health and productivity of the cow by damaging cells and tissues, altering metabolism and inducing changes in steroidogenesis. Membrane permeability, enzyme function, and muscle tone can be affected by reactions involving ROM. In addition, ROM alter metabolism by reducing the supply of essential cofactors, such as nicotinamide adenine dinucleotide phosphate (NADPH), and diverting glucose from the important metabolic pathways. The ROM also cause inactivation of steroidogenic en-
zymes that are necessary for the synthesis of reproductive hormones, such as progesterone and estrogen. Vitamin E is a chain-breaking antioxidant that terminates reactions involving ROM by reacting directly with the radicals after they have been formed. Glutathione peroxidase, an enzyme containing selenium, prevents the formation of ROM by removing the reactants $\text{O}_2^-$ and $\text{H}_2\text{O}_2$.

Research has shown that the levels of antioxidants in the blood are higher for cows that shed the placenta within 12 hours of parturition (Figure 7). Several other studies have shown that supplementation of vitamin E and selenium reduced the incidence of retained placenta. In addition, supplementation seems to be more effective when vitamin E and selenium are both added to the diet than when one or the other is lacking (Table 6) (Miller and Brzezinska-Slebodzinska, 1993).

Correct vitamin and mineral supplementation to enhance immunity is certainly a goal of proper transition cow management. However, French researchers more completely described retained placenta as an under-nutrition disease. Chassagne and Chacornac (1994) reported that cows that retained the placenta were on a lower plane of nutrition prior to calving. Blood metabolite measurements showed higher fat mobilization and lower blood glucose, as well as lower blood calcium and amino acids (Table 7). These results show the importance of the overall nutritional balance of the transition cow.

Levels of crude protein (CP) and amino acids in the dry cow diet also affect performance in the subsequent lactation. During pregnancy, the cow requires protein for maintenance, fetal growth, and, in the case of a primiparous heifer, growth of the dam. The National Research Council (NRC, 2001) recommends feeding 12.4% CP in the late dry period, or 2.8 lb of CP for a mature 1500-lb (without conceptus) cow consuming 22.2 lb/day of DM. Approximately 9.6% of the diet should be in the form of rumen degradable protein (RDP). Levels above or below these recommendations can have detrimental effects. Greenfield et al. (2000) found that cows fed 12% CP for 28 days prepartum had a higher DM intake and produced more milk during the first 56 days in milk when compared to cows fed 16% CP. On the other hand, lower protein levels in the dry cow diet can restrict the growth of the fetus, resulting in low calf birth weight. In addition, amino acids from protein can be oxidized for energy during the late dry period, when energy demands for fetal growth are high and DM intake is depressed (Greenfield et al., 2000). Without this additional energy source, the transition cow’s negative energy balance may worsen.

There are also a variety of feed additives available to help make the transition period more successful. Anionic salts and oral calcium supplements can be given to alleviate milk fever problems. Daily oral doses of propylene glycol and/or daily niacin supplementation during the transition period help decrease the severity of negative energy balance. Other feed additives, such as yeast culture and probiotics, have been used to aid cattle in the transition from low starch diets fed to dry cows to high starch diets fed to lactating cows.

**Feeding Management**

The environment in which cows are fed is important when evaluating the transition program and the ability to successfully achieve the 100-day contract. Much has been written pertaining to the feeding environment of lactating cows, but comparatively, little information is available relative to the periparturient cow. Adequate bunk space to allow all cows equal access at feeding time is important, as is the availability of water.
relative to distance from feed (less than 50 feet) and the number of animal spaces. In managing the transition cow group, there can be large fluctuations in the number of cows on a day-to-day basis. The amount of feed delivered must be carefully monitored as group size changes when fresh cows are moved out after calving and late gestation cows are added. Age and body weight of the cows entering and leaving the transition group will also affect the amount fed. These details of where and how feed is offered to the transition cow group can determine the success or failure of the early lactation cow.

Environment

Another factor critical to a successful transition cow contract is housing. The dry cow experiences significant stress with calving and initiation of lactation. The housing system is key to minimizing exposure to environmental stress. Housing should protect the animal from injury and disease. This is especially important for the dry cow in late gestation. Harmon and Crist (1994) reported that the incidence of environmental mastitis is highest during the first two weeks and the last two weeks of the dry period. Voermans (1997) recommended evaluating the housing system in terms of ability to reduce exposure of the animals to pathogens. Furthermore, Voermans (1997) concluded that the important benefits of good housing in minimizing animal stress were manifested in improved immune function and increased resistance to challenge by pathogenic microorganisms. Clean, dry bedding is essential to improved animal health, especially in the periparturient transition phase.

High environmental temperatures result in significant thermal stress for the transition cow. Exposure to heat during the third trimester of gestation shifts blood flow to the extremities and away from the uterus, compromising placental and fetal growth. Calves often have lower than normal birth weights, putting them at higher risk for mortality (Shearer and Beede, 1990). In addition, researchers in Georgia found that the incidence of retained placenta increased from 12% during the warm, humid months of May through September to 24% during the cooler months (Dubois and Williams, 1980). Hormone alterations due to heat stress affect mammary development and lactogenesis, reducing milk yield in the subsequent lactation (Table 7) (Shearer and Beede, 1990). Strategies to keep cows cool and comfortable during the transition period include providing shade for cows on pasture or utilizing sprinklers, misters, and/or fans in free-stall structures. Cows should also be provided with an easily accessible source of clean drinking water.

Summary

During the transition period, the dairy cow is undergoing numerous changes in endocrine, nutritional, metabolic, and physiological status as she prepares for calving and initiation of lactation. These changes result in a dramatic decrease in DM intake that worsens the negative energy balance already present after calving. If the negative energy balance during transition becomes excessive, metabolic diseases, such as fatty liver and ketosis, can result. Disruption of mineral balance during the periparturient period leads to mineral balance disorders, especially milk fever. These diseases are costly in terms of their affect on milk production, reproduction, and the cow’s susceptibility to other periparturient disorders. Intensive management of the nutrition, feeding system, and environment of the periparturient dairy cow reduces the odds of disease and increases the odds of success.
The ‘100 day contract’ is a series of delicate negotiations that encompass the full impact of the transition cow. Unsuccessful negotiations at any point increase the risk of overall failure. Getting the details right and ensuring adequate intake of all nutrients are the key elements of the ‘100-day contract’.

References


Table 1. Energy and protein deposition in the uterus and fetus during pregnancy in Holstein cows.¹

<table>
<thead>
<tr>
<th>Gestation (days)</th>
<th>Energy (kcal/day)</th>
<th>Protein (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Uterus</td>
<td>Fetus</td>
</tr>
<tr>
<td>210</td>
<td>631</td>
<td>500</td>
</tr>
<tr>
<td>230</td>
<td>694</td>
<td>601</td>
</tr>
<tr>
<td>250</td>
<td>757</td>
<td>703</td>
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<td>270</td>
<td>821</td>
<td>805</td>
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</tbody>
</table>

¹Adapted from Bell et al., 1995.

Table 2. Periparturient changes in ruminal water-holding capacity and fill.¹

<table>
<thead>
<tr>
<th>Average days from calving²</th>
<th>-61</th>
<th>-48</th>
<th>-34</th>
<th>-20</th>
<th>-6</th>
<th>+8</th>
<th>+22</th>
</tr>
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<tbody>
<tr>
<td>Rumen Capacity, gal</td>
<td>33.5</td>
<td>31.4</td>
<td>28.5</td>
<td>28.0</td>
<td>26.9</td>
<td>37.5</td>
<td>35.1</td>
</tr>
<tr>
<td>Total fill/capacity, %</td>
<td>46.5</td>
<td>51.9</td>
<td>57.3</td>
<td>55.5</td>
<td>53.1</td>
<td>51.0</td>
<td>58.9</td>
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<td>DM fill/capacity, %</td>
<td>6.7</td>
<td>6.2</td>
<td>6.6</td>
<td>6.0</td>
<td>6.2</td>
<td>6.4</td>
<td>7.4</td>
</tr>
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<td>Fluid fill/capacity, %</td>
<td>39.9</td>
<td>45.7</td>
<td>50.7</td>
<td>49.5</td>
<td>47.0</td>
<td>44.6</td>
<td>51.5</td>
</tr>
</tbody>
</table>

¹Adapted from Stanley et al., 1993.
²Negative values indicate days prior to calving; positive values indicate days after calving.
Table 3. Lactational incidence risks and median days postpartum of disorders in 8070 multiparous Holstein cows in New York state.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Lactational Incidence</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Risk (%)</td>
</tr>
<tr>
<td>Retained placenta</td>
<td>7.4</td>
</tr>
<tr>
<td>Metritis</td>
<td>7.6</td>
</tr>
<tr>
<td>Milk fever</td>
<td>1.6</td>
</tr>
<tr>
<td>Ketosis</td>
<td>4.6</td>
</tr>
<tr>
<td>Displaced abomasum</td>
<td>6.3</td>
</tr>
<tr>
<td>Mastitis</td>
<td>9.7</td>
</tr>
</tbody>
</table>

1Adapted from Grohn et al., 1995.

Table 4. The effects of early postpartum energy status on reproductive performance.1

<table>
<thead>
<tr>
<th>Energy Status</th>
<th>Days to first service</th>
<th>Days open</th>
<th>Services per conception</th>
<th>First service conception rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>70.5</td>
<td>80</td>
<td>1.2</td>
<td>75</td>
</tr>
<tr>
<td>Subclinical ketosis</td>
<td>75.8</td>
<td>102</td>
<td>2.0</td>
<td>44</td>
</tr>
<tr>
<td>Ketotic</td>
<td>78.0</td>
<td>100</td>
<td>1.9</td>
<td>40</td>
</tr>
</tbody>
</table>

1Adapted from Miettinen, 1990.

Table 5. Influence of hypocalcemia on risk of other periparturient disorders.1

<table>
<thead>
<tr>
<th>Disease</th>
<th>Odds ratio</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dystocia</td>
<td>2.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Retained placenta</td>
<td>6.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Left displaced abomasum</td>
<td>3.4</td>
<td>0.06</td>
</tr>
<tr>
<td>Ketosis</td>
<td>8.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mastitis</td>
<td>8.1</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

1Adapted from Table 2 in Curtis et al., 1983.
Table 6. Incidence of placental retention in dairy cows fed diets containing > 0.12 ppm of Se with or without 1000 IU of supplemental vitamin E during the last 40 days of gestation.¹

<table>
<thead>
<tr>
<th>Year</th>
<th>Reference</th>
<th>Treatment</th>
<th>Control</th>
<th>Vitamin E</th>
</tr>
</thead>
<tbody>
<tr>
<td>1988</td>
<td>Mueller et al., 1988</td>
<td>26.7</td>
<td>6.9*</td>
<td></td>
</tr>
<tr>
<td>1989</td>
<td>Mueller et al., 1989</td>
<td>34.4</td>
<td>10.8**</td>
<td></td>
</tr>
<tr>
<td>1990</td>
<td>Thomas et al., 1990</td>
<td>52.9</td>
<td>22.0*</td>
<td></td>
</tr>
<tr>
<td>1991</td>
<td>Brzezinska-Slebodzinska and Miller, 1992</td>
<td>32.3</td>
<td>21.9</td>
<td></td>
</tr>
</tbody>
</table>

*P < 0.05
**P < 0.01
1Adapted from Miller and Brzezinska-Slebodzinska, 1993.

Table 7. Measurements of blood metabolites and nutrients between normal cows and cows with retained placenta.¹

<table>
<thead>
<tr>
<th>Item</th>
<th>Retained</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, ng/dl</td>
<td>59.6</td>
<td>61.8</td>
</tr>
<tr>
<td>NEFA², meq/dl</td>
<td>0.494</td>
<td>0.340*</td>
</tr>
<tr>
<td>Amino acids, moles/dl</td>
<td>2.34</td>
<td>2.48*</td>
</tr>
<tr>
<td>Calcium, mg/dl</td>
<td>96.3</td>
<td>98.5*</td>
</tr>
<tr>
<td>Monocytes, 10³/ml</td>
<td>225</td>
<td>310*</td>
</tr>
</tbody>
</table>

*P < 0.05
¹Adapted from Chassagne and Chacornac (1994)
²NEFA = non esterified fatty acids.
Table 8. Effect of prepartum heat stress on postpartum milk yield.\textsuperscript{1}

<table>
<thead>
<tr>
<th>Production</th>
<th>Cooled</th>
<th>Heat stressed</th>
<th>Difference (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>305-d milk yield, lbs.\textsuperscript{2}</td>
<td>5878.4</td>
<td>5623.2</td>
<td>255.2 (4)</td>
</tr>
<tr>
<td>150-d milk yield, lbs./d</td>
<td>89.5</td>
<td>81.8</td>
<td>7.7 (8.5)</td>
</tr>
<tr>
<td>Peak milk yield, lbs./d\textsuperscript{3}</td>
<td>91.0</td>
<td>87.4</td>
<td>3.6 (4)</td>
</tr>
</tbody>
</table>

\textsuperscript{1}Adapted from Shearer and Beede, 1990.
\textsuperscript{2}305-d predicted yield adjusted for age, month of calving, and Estimated Relative Producing Ability (ERPA).
\textsuperscript{3}Means of peak milk production taken from three herds.

Figure 1. Terms of the 100-day contract

1. Birth of a live calf
2. Healthy cow during the transition period
3. High peak milk production
4. Controlled loss of body condition
5. High fertility at first breeding
Figure 2. Changes in serum concentrations of hormones in cows during the periparturient period (adapted from Bell, 1995).
Figure 3. Dry matter intake of transition cows (Adapted from Underwood, 1998).
**Figure 4.** Estimated prepartum energy balance of transition cows (Adapted from Grummer, 1995).

**Figure 5.** Serum non-esterified fatty acid concentration of transition cows (Adapted from Underwood, 1998).
**Figure 6.** Changes in the area of cross sections of rumen papillae of cows fed low-energy diets prepartum and high-energy diets postpartum (Adapted from Dirksen et al., 1985).
**Figure 7.** Total antioxidants in bovine plasma as measured by their protection of phycoerythrin fluorescence in vitro (Adapted from Miller and Brzezinska-Slebodzinska, 1993).